NATARAJAN'S

Textbook of Orthopaedics and Traumatology

Seventh Edition

Mayil Vahanan Natarajan

Wolters Kluwer Health | Lippincott Williams & Wilkins

Dedicated To

Maataa

Pitaa

Guru

Ishwar

Commissioning Editor: Ms. P. Sangeetha
Development Editor: Dr. Munish Khanna
Production Editor: Ms. Tamali Deb
Compositor: HT Media Ltd.
Assistant Manager Manufacturing: Mr. Anil Kumar Gauniyal

10th Floor, Tower C
Building No. 10
Phase – II
DLF Cyber City
Gurgaon
Haryana - 122002

The publisher is not responsible (as a matter of product liability, negligence, or otherwise) for any injury resulting from any material contained herein. This publication contains information relating to orthopaedic care and intervention for orthopaedic disorders that should not be construed as specific instructions for individual patients. Manufacturers' product information and package inserts should be reviewed for current information, including contraindications, dosages, and precautions.

Care has been taken to confirm the accuracy of the information presented and to describe generally accepted practices. However, the authors, editors, and publishers are not responsible for errors or omissions or for any consequences from application of the information in this book and make no warranty, expressed or implied, with respect to the currency, completeness, or accuracy of the contents of the publication. Application of this information in a particular situation remains the professional responsibility of the practitioner. Readers are urged to confirm that the information, especially with regard to drug dose/usage, complies with current legislation and standards of practice. Please consult full prescribing information before issuing prescription for any product mentioned in the publication.

The publishers have made every effort to trace copyright holders for borrowed material. If they have inadvertently overlooked any, they will be pleased to make the necessary arrangements at the first opportunity.

Seventh Edition, 2011

ISBN-13: 978-81-8473-443-0

Published by Wolters Kluwer (India) Pvt. Ltd., New Delhi.

Printed and bound at Sanat Printers, Haryana.

For product enquiry, please contact – Marketing Department (marketing@wolterskluwerindia.co.in).

Foreword

It is my pleasure to comment on this basic yet extremely important work on the principles of Orthopaedic Surgery. Orthopaedic practice has witnessed dramatic changes over the past decade. Newer imaging technologies have revolutionised the way orthopaedic disorders are diagnosed and managed. However, the basic principles remain the same even today.

This book will be a boon to the discerning undergraduate students who are faced with two Himalayan tasks, one their final examination and the other their post-graduate entrance examination. My teacher and mentor Professor M. Natarajan's *Textbook of Orthopaedics and Traumatology* has been providing comprehensive and well-organised information on the basics of the subject for the past three decades. Dr. Mayil Vahanan Natarajan is a well-known and experienced orthopaedic surgeon who has meticulously updated his father's textbook, keeping in mind the current requirements of the student community.

This seventh edition is going to be a ready-reckoner for the students as the language is clear and precise thus providing the necessary practical points. Each chapter ends with a set of key points that summarises the important concepts. All aspects of orthopaedic disorders are touched upon, laying stress on the areas that require explanation. The tables and charts have been updated, while all the figures are of excellent quality, making it easier to organise and remember. The artistic diagrams have been made visually appealing with more clarity and an easy-to-understand format. Full colour has been used throughout the text enhancing the book's attraction.

I congratulate Dr. Mayil Vahanan Natarajan on bringing out this thoroughly updated edition and hope this book finds wide acceptance and use.

Dr. (Maj.) D. Raja
M.S. (ORTHO)
Former Vice Chancellor
The Tamil Nadu Dr. M.G.R. Medical University

Preface to Seventh Edition

'If I have seen further it is only by standing on the shoulders of giants.'

Sir Isaac Newton

Every new edition of a successful book is an improvement on its predecessors. In no other field of medicine is change such a constant as in the vast and ever-expanding field of orthopaedics. In my three-decade long career, the principles of orthopaedics have evolved from being predominantly non-operative to the current trend of judicious use of principles of operative techniques.

This book was originally written by my beloved father and illustrious academic, Professor M. Natarajan, who had made his lectures and practical demonstrations the basis of his work. No wonder that this book, which was from such a renowned teacher, received such an exceptional response from the students. The basic aim of the book was to provide the essence of orthopaedics to a beginner along with an overview of the conditions prevailing in our country. It also provided to the students an overview of the management of trauma.

Now, after three decades the textbook is being relaunched with an international format. While retaining the unique flavour of the earlier editions, the new edition provides completely updated and revamped content. The book mainly aims to serve as an authentic ready-reckoner for undergraduates and entrant post-graduates in orthopaedics as well as a useful reference source for students of nursing and allied health specialities.

As the academic head of The Tamil Nadu Dr. M.G.R. Medical University, I am aware of the impending changes in the medical curriculum of our country. These changes are based on the need to align medical education more closely with the current practice of medicine and also to prepare students for upcoming changes in the health care system. This book has been amply updated keeping in mind the current undergraduate reforms.

Important newer trends such as minimally invasive surgeries, limb salvage and improvements in joint replacement principles have been included in this edition to keep the medical student updated. Such advanced surgeries have made orthopaedics one of the most sought-after specialities.

This edition also includes new chapters on Rehabilitation and Disaster Management in order to provide students a more comprehensive understanding of orthopaedic practice.

The format of the book has also been completely revamped with the first ever colour design. In addition, several new clinical photographs and radiographs along with easily comprehensible illustrations have made this edition much more appealing and readable. Finally, Key Point summaries and Multiple Choice Questions have been included at the end of various chapters in order to make the book more useful from the post-graduate entrance point of view.

I hope all these changes prove useful to the student fraternity and the book serves as a friendly and authentic text for medical undergraduates. I would warmly welcome suggestions and ideas from both students and faculties for the further enhancement of the book.

I place on record my sincere thanks to Dr. S. Krishnamurthi and Dr. V. Shanta, who are renowned oncologists, for guiding and encouraging my work on Orthopaedic Oncology at the Cancer Institute.

Finally, before closing this, I would like to express my deep sense of gratitude to my lord for being the guiding force in my life and all my endeavours and to my parents for my disciplined upbringing and for giving me what I needed rather than what I wanted.

Mayil Vahanan Natarajan

Preface to First Edition

It is indeed a pleasure to present to the medical students the first edition of my *Textbook of Orthopaedics and Traumatology* as my continuing service to medical education.

The text is based on the lectures and practical demonstration given by me both at the Stanley Medical College which is my Alma mater and the Madras Medical College. The book is meant to serve as a textbook for the undergraduate medical students, post-graduates in general surgery, nursing, physiotherapy and occupational therapy students. The text has been updated to include recent developments and pruned to delete obsolete matter. I have maintained my basic philosophy of biological approach to Orthopaedics and Traumatology.

The training of the medical student to assume the role of primary care doctor has been the subject of debate in our country. The explosion of medical knowledge on the one hand and the development of sophisticated technology in the delivery of medicare on the other hand have made the task of defining the exact goal of the undergraduate medical curriculum rather difficult. However, it must be admitted that at the level of the individual doctor facing an individual patient, the medical practitioner has to be knowledgeable, skilled and with the right attitudes in diagnosing, treating and advising the sick patient. As a medical graduate of the 3rd millennium he has to show awareness of many conditions which were not recognised earlier. Knowledge of the emergency management of major injuries and primary care of most other injuries is essential for every doctor.

I recall with pleasure the meeting of 15 pioneers of Indian Orthopaedics in December 1954 at Hyderabad which laid the foundation for the development of Orthopaedic Section of the Association of Surgeons of India as a prelude to the later formation of Indian Orthopaedic Association. The historic event is reproduced as the paper bearing the signatures of those founder leaders.

It has been a pleasure to highlight contributions made by my orthopaedic colleagues all over India, Dr. B. Mukopadhaya (Patna), Dr. K.T. Dholakia (Mumbai), Dr. S.M. Tuli (Varanasi), Dr. A.J. Selvapandizr (Vellore), Dr. Vyagreswarudu (Vizag), Dr. T.K. Shanmugasundaram (Chennai), Dr. B.B. Joshi (Mumbai), Dr. M.K. Goel (Lucknow) and Dr. V. Chacko (Manipal). The contributions by Dr. S. Jay Kumar (USA), Dr. S.S. Yadav (Delhi), Dr. P.K Dave (Delhi), Dr. P.D. Bakshi (Calcutta), Dr. K. Sriram (Chennai), Dr. S.V Sharma (Varanasi), Dr. J.C. Taraporvala (Mumbai), Dr. R.L. Mittal (Patiala), Dr. S. Pandey (Ranchi), Dr. M.M. Ray (Kolkata) and Dr. K.R. Daga (Sholapur) have also been acknowledged in the appropriate places.

It is my privilege to record my deep sense of gratitude to my teachers who instilled in me a great desire for teaching, particularly to my respected Professor of Surgery Dr. C. Raghavachari, who taught me surgery including orthopaedics at Stanley Medical College, Chennai. I record with pride that Dr. M.G. Kini, the Father of Orthopaedics in Tamil Nadu, was my teacher and Chief, who

created my interest in Orthopaedics. I must thank my former teacher and Chief Dr. R. Mahadevan whose encouragement to write two chapters in his *Textbook of Surgery* gave me the confidence to embark on this project. I am grateful to Professor N.S. Narasimha Iyer of revered memory who had always been a source of encouragement to me. I feel thankful to Dr. V.R. Thayumanaswamy, the first independent head of the Department of Orthopaedics at Government General Hospital whom I succeeded, for the good set-up of the department. I am extremely grateful to Professor B.N. Sinha, the doyen of Indian Orthopaedics, whose personal affection for me has been most stimulating. I must make a special mention of Professor T.K. Shanmugasundaram, my successor at Madras Medical College, for his Foreword.

It is the most self-effacing and stimulating memory of my loving wife Janaki which really made the whole work a pleasure and I have dedicated this work to her loving memory. I am thankful for the divine blessings of Lord Muruga, Lord Venkateswara and Adi Parasakthi which made this work possible.

Chennai

Professor M. Natarajan

Acknowledgements

It is my privilege and honour to place on record my wholehearted gratitude to my beloved father who had put together a book of this calibre way back in 1981 when no other treatise in orthopaedics was available in India.

I record with pride my sincere gratitude to the stalwarts in orthopaedics and my teachers Dr. T.K. Shanmugasundaram and Dr. S. Soundarapandian, who taught me the basics in orthopaedics.

I acknowledge the training given by my teachers Professor Robert Roaf, Mr. I.W. Young, Professor R. Owen and Mr. J.F. Taylor in UK and my teachers Dr. (Maj.) D. Raja, Dr. S. Rajagopal, Dr. T.S. Xavier, Dr. K. Sriram and Dr. K. Annamalai in Chennai.

I thank Dr. Raja Venkatakrishnan, Dr. K. Selvam, Dr. R.H. Govardhan, Dr. P.V. Jayasankar, Dr. Sudhakar Williams and Dr. B. Mahender who were involved in the earlier editions of this book and whose valuable suggestions have been incorporated. I thank my assistants Dr. R.M. Alagappan, Dr. K.P. Manimaran, Dr. M.D. Kumar and Dr. Jose Chellan who have been with me in my endeavours.

I thank Dr. N.O. Samson and Dr. R. Emmanuel for the compilation of clinical radiographs and photographs which have greatly enhanced the appeal of this edition.

My sincere thanks to Dr. Jaheer Hussain and Dr. Mohammed Sameer who have helped me greatly in my administrative and academic endeavours.

Dr. T.S. Rajagopal, Dr. G. Rajkumar, Dr. A. Sivaseelam and Dr. Raja Marimuthu in UK were my post-graduates who have helped me during my earlier days and in the previous editions of the book.

I would like to thank Dr. Kunal Dheep for being instrumental in bringing out this edition and for assisting me in all ways with his ideas.

This edition would not have been possible without the assistance of my post-graduate students Dr. R. Anand Kumar, Dr. V. Anjan Ramachandranath, Dr. R. Uma Shankar, Dr. Navin Balasubramanian, Dr. J. Vishwanathan, Dr. D. Kamalasekaran, Dr. M. Yuvaraja, my administrative officer Mrs. K. Kousalya and my secretary Mrs. Preetha Bobby who have spent hours of their time compiling my thoughts on each topic.

The presentation has been made in an easy-to-read format and for this I will have to thank the team at the publishing house, Wolters Kluwer, Ms. Sangeetha and Mr. Rajiv Banerji who have ably aided me with their valuable suggestions in this regard.

I am thankful to my wife Mrs. Thenmozhi for being the lucky charm in my life and for standing by me during the trials and tribulations in the past, my daughter Devi who has taught me to love all and serve all, my charming daughter Swetha who by her intelligence and beauty has been a source of immense joy to me and my family members Dr. Ramona and Dr. Chakkaravarthi, Mrs. Sashikala and Mr. Shanmugavel, Dr. Madhivanan and Dr. Chalini, Mrs. Premila and Dr. Jayarajah who have been the source of great support in my life.

My heartfelt gratitude and prayers to God Almighty for his abundant blessings and my parents who have been the anchor of my life.

Mayil Vahanan Natarajan

Publisher's Acknowledgements

The publisher would like to thank Dr. Anand J Thakur, a distinguished orthopaedician at Irla Nursing Home, Mumbai, for his valuable help in organising several clinical photographs and radiographs for this edition.

The publisher would also like to thank Dr. Rujuta Mehta and Dr. Ajay Puri for providing clinical images from their collection for this book.

Wolters Kluwer (India) Pvt. Ltd.

Table of Contents

CHAPTER 1

Orthopaedics: General

DEFINITION

Orthopaedics is the science and art of the diagnosis and treatment of all diseases and disorders of the human locomotor system.

EVOLUTION

The word 'orthopaedics' was coined by Nicholas Andry, a physician in Paris, in the year 1741 from two Greek words 'Ortho' meaning straight and 'Pedio' meaning child.

When Nicholas Andry defined it, the subject dealt with the problem of straightening deformities in children. This was due to the fact that in the eighteenth century, the incidence of deforming diseases such as rickets and skeletal infections were very high among children in Europe.

The scope of the subject gradually increased to include all deforming conditions, not only in children but also in adults. In early days, badly managed traumatic conditions contributed the maximum number of deformities in adults. Preventive orthopaedics was thus born when the specialists in orthopaedics started managing fractures and dislocations, to achieve better results and minimise deformities and disablement.

INTERNATIONAL ORTHOPAEDICS

Till the middle of the eighteenth century, orthopaedic treatment consisted mainly of manipulation, splinting and re-educative exercises. The eighteenth century saw Sir John Hunter's contribution by the addition of closed surgery in the form of tenotomies for the correction of deformities. With the advances in anaesthesia, antiseptic and aseptic techniques in the nineteenth century, open surgical methods were added to the armamentarium and orthopaedic surgery became a branch of the science of surgery.

Hugh Owen Thomas, the father of British Orthopaedics, was the first qualified doctor in a family of traditional bone setters to practice orthopaedics in Liverpool, UK. His nephew Sir Robert Jones spread his teachings among the medical professionals and promoted orthopaedic training as a speciality in Liverpool in the early twentieth century.

ORTHOPAEDICS IN INDIA

Orthopaedics was recognised as one of the specialities of surgery in the first quarter of the twentieth century. Due to historical reasons, the influence of British Orthopaedics, particularly from Liverpool and London, was a major one in Indian Orthopaedics. Dr. M.G. Kini and Dr. N.S. Narashimhan of Chennai, Dr. J. Katrak of Mumbai, Dr. S.R. Chandra and Dr. G.C. Sen of Calcutta were the early pioneers who belonged to the first generation of orthopaedic surgeons in the 1930s. The second generation of orthopaedic surgeons who were the leaders in their respective geographical

FIGURE 1.1 Emblem of orthopaedics.

areas—in our country—were Dr. B.N. Sinha, Dr. B. Mukopadhaya, Dr. V.R. Thayumanasamy, Dr. A.K. Duraiswamy, Dr. M.D. Desai, Dr. A.K. Saha, Dr. M. Ranga Reddy and Dr. A.K. Gupta.

EMBLEM OF ORTHOPAEDICS

The emblem of orthopaedics depicts a tender young plant with a deformity in the trunk being tied to a straight pole used as a splint, to gradually correct the curvature in the stem (Fig. 1.1). This typifies the conservative management of the deformities, which succeeds well if it is done when the patient is young.

More recently, surgical management of many orthopaedic deformities is being increasingly used and accepted. This is symbolically represented by a modified emblem, showing a second plant where the bent stem has been cut at multiple levels and straightened using a straight central rod.

USEFUL TERMINOLOGIES IN ORTHOPAEDICS

Arthrotomy

Surgical opening of a joint is called arthrotomy and it is indicated (1) to inspect the interior or perform a synovial biopsy, (2) to drain a haematoma or an abscess, (3) to remove a loose body or damaged structure, for example a torn meniscus, and (4) to excise inflamed synovium.

Arthrodesis

Fusion of a joint and preventing any motion is termed 'arthrodesis'. This is the most reliable operation for a painful or unstable joint, where stiffness does not seriously affect the function.

Arthroplasty

The surgical refashioning of a joint, aiming to relieve pain and to restore movement, is known as arthroplasty. The varieties are as follows:

- *Excision arthroplasty:* Sufficient bone is excised to create a gap at which movement can occur, for example Girdlestone hip arthroplasty.
- *Partial replacement:* Only one articular component is replaced, for example, Austin Moore's prosthesis for a fracture of femoral neck.
- *Total replacement:* Both articular bone ends are replaced by prosthetic implants.

Osteotomy

The surgical breaking (cutting) of bone is named osteotomy. When the bone is broken manually for correction of deformity, it is called osteoclasis.

Other Terms in Orthopaedics

Other terms in orthopaedics include the following:

- *Osteogenesis:* New bone formation
- *Arthrography:* Imaging of a joint by using dye inside it
- *Neurolysis:* Releasing a tight nerve
- *Neurotomy:* Cutting a nerve
- *Neurorrhaphy:* Repairing a nerve
- *Tenotomy:* Cutting a tendon
- *Tenodesis:* Attaching a tendon to another tendon or bone

The following Latin terminologies are frequently used in orthopaedics: Cubitus (elbow), Manus (hand), Coxa (hip), Genu (knee), Pes (foot), Pollex (thumb) and Hallux (great toe).

Acquired Deformities

The common causes of acquired deformities are trauma, infections or metabolic disorders affecting the various structures of the locomotor system. The posture of an individual can also lead to various types of deformities.

Postural Deformities

Upper Motor Neuron Lesions

These deformities are defects due to a habitual assumption of bad postures and are correctable by voluntary efforts and exercises, for example postural kyphosis, postural scoliosis and postural flat feet.

Traumatic Conditions Affecting Posture

These include the following:

- *Injuries to bones:* Malunited fractures
- *Injuries to joints:* Unreduced dislocations
- *Nerve injuries:* Claw hand: ulnar and median nerve injury
- *Vascular injuries:* Volkmann's ischaemic contracture
- *Soft tissue injuries:* Contractures due to burns

Neural Lesions Affecting Posture

Paralytic Deformities

Deformities in paralytic conditions may be due to lesions of upper motor neurons, lower motor neurons or peripheral nerves.

Spastic paralysis is seen in (1) cerebral palsy and (2) infantile or adult hemiplegia. Deformities in spastic paralysis (e.g. cerebral palsy), are due to spasticity of certain groups of muscles. The most common are equinus of the foot, flexion of the knees, flexion adduction of the hips and pronation deformity of the forearm. Long-standing spasm in muscles results in fixed contractures.

Lower Motor Neuron Disease

- *Flaccid paralysis:* Residual neglected anterior poliomyelitis results in multiple deformities due to chronic muscle imbalances and contractures.

Peripheral Nerve Disease

- *Leprosy:* In India, neural leprosy is one of the most common causes of deformities of the limbs. The common deformities are claw hand due to ulnar nerve paralysis and equinus deformity due to lateral popliteal nerve paralysis.

Infections and Other Diseases of Joints Affecting Posture

Joint disease is one of the most common causes of crippling deformities. In India, tuberculosis and septic infections of the joints cause the largest number of deformities. In the Western countries, rheumatoid arthritis is far more common.

Deformities Due to Metabolic and Other Generalised Disorders

Certain metabolic and hormonal disorders may cause skeletal deformities, for example rickets which is due to Vitamin D deficiency, hyperparathyroidism, senile osteoporosis and Paget's disease.

Deformities Due to Diseases of Muscles and Subcutaneous Tissues

Various myopathies in children manifest themselves as deformities and muscle weakness. A child with pseudo-hypertrophic muscular dystrophy is usually brought for bilateral equinus deformities.

Dupuytren's contracture is a good example of deformities in the fingers produced by contractures of the deep palmar fascia.

Idiopathic Deformities

The best example of this is idiopathic scoliosis, a lateral deformity of the spine occurring in adolescents.

Hysterical Deformities

Deformities of functional origin are common in injured workmen expecting compensation and in women and young men with psychological maladjustments.

General Principles of Management

Assessment

When a patient has a particular deformity, it is essential to determine whether it is primary or secondary due to some other condition which is not obvious. For example, an equinus deformity may be primary due to an imbalance of muscles

acting on the ankle or it may be secondary or compensatory to a shortening of the leg due to conditions in the hip or the knee.

We must also determine whether the deformity is mobile and passively correctable or fixed. Again, in assessing the nature of the deformity, it is also important to decide whether a deformity is disabling or only disfiguring.

Methods of Correction

Conservative Methods

These must always be tried first to correct deformities.

- *Physiotherapy:* Corrective exercises and passive stretching correct many deformities of a mild degree.
- *Splinting and use of appliances:* This is the most common method used to correct deformities in children.
- *Traction procedures:* This is used in the early stages of joint deformities caused by muscle spasm, which will yield to continuous traction.
- *Plaster casts and wedging:* Flexion deformities of knees which are not rigid can be corrected by repeated 'wedging' of plaster casts.
- *Manipulation under anaesthesia:* This method must be used carefully without using excessive force to correct some deformities which are not too rigid. Mild adhesions are broken by this method. It must be followed by physiotherapy.

Surgical Methods

When conservative measures fail and in cases where the deformity is producing definite disability or excessive disfiguring, surgical measures will be required. Surgical methods used are (1) soft tissue operations and (2) bone operations.

1. *Soft tissue operations:* Operations on the soft tissues are always tried first to correct the deformities. This will be possible in the younger age groups when permanent bone changes have not yet occurred. The following are some common examples.
   - Posteromedial soft tissue release in clubfoot
   - Tendo achilles lengthening and posterior capsulotomy in an equinus foot
2. *Bone operations:* When severe deformities have been long-standing, they will need the following types of operations on the bone, in addition to operations on the contracted soft tissues.
   - Osteotomy: Osteotomy is a division of the bone to correct deformities, for example supracondylar osteotomy of the femur to correct severe genu valgum or flexion deformity of the knee.
   - Excision and arthrodesis of joints, for example triple arthrodesis, to correct deformities of the foot. Recently, Ilizarov's technique is being used in the correction of deformities.

GAIT

The sequence of events, heel strike, stance phase, toe off and swing phase helping in forward propulsion of the body is defined as gait.

Observation of the patient's gait is a very important part of the examination of an orthopaedic patient with a locomotor disorder. The normal human gait basically requires the normal biomechanical functioning of the musculoskeletal system of the limbs and spine. It also needs good sensory feedback from the proprioceptive sensations from the feet and the joints. The visual and labyrinthine sensory inputs and their coordination add to the smoothness, rhythm, grace and elegance of the human gait (Fig. 1.2).

The biomechanical factors affecting the gait are due to the defects in the mechanics of propulsion, antigravity supports and stoppage. Finally, the normal swinging of the arms contributes to the elegance of the gait and hence the loss or paralysis of one arm affects the cadence of the gait.

Biomechanically, in all phases of the walk cycle, the simple law of physics must be obeyed, that is, the centre of gravity of the body mass should fall within the base of support to retain its stability. The line of gravity should also be passing through the joints of the supporting leg.

WALK CYCLE

One complete walk cycle extends from the heel strike of one leg to its next heel strike. It consists

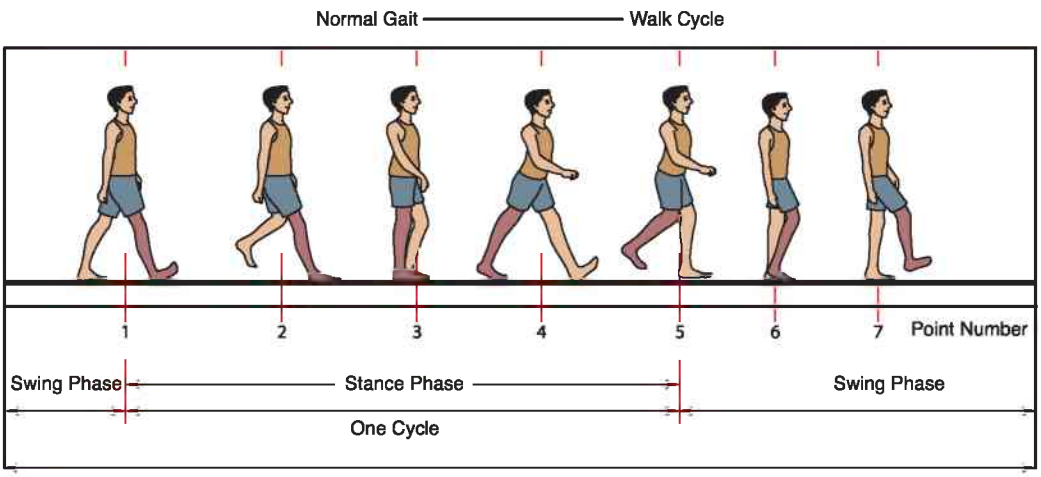


FIGURE 1.2 Normal human gait.

of a long *stance phase* and a shorter *swing phase* in the ratio of 60 and 40% of the cycle.

In the stance phase the foot is on the ground supporting the body and in the swing phase the leg swings forwards alone with the forward movement of the body. Therefore, walking itself can be described as a series of steps preventing the falling forward of the human body.

Events in the Walk Cycle

Each phase consists of the following events.

Stance Phase

Stance phase, also known as support phase, lasts from heel strike to toe off of one leg. The events occurring in this phase are as follows (Fig. 1.3):

1. *Heel strike:* The first event is the heel striking the ground.
2. *Foot flat:* In this, the whole foot is flat on the ground.
3. *Mid-stance:* With the foot flat on the ground, the body swings forwards. There is an active extension of the hip.
4. *Heel off:* The heel is lifted off the ground with an active extension of the hip and knee.
5. *Push off:* The whole foot except the toes is raised up as the calf muscles start pushing the body up and forwards.
6. *Toe off:* In this last part, the toes also go off and the whole foot leaves the ground.

The heel strike and foot flat, that is, stages 1 and 2, of one foot correspond to the push off and the toe off, that is, stages 5 and 6 of the other foot. This span of time when both feet are simultaneously on the ground is called the period of double support. For the smoothness of walk, the stance leg must support the centre of gravity and provide an active extension of the hip and knee to help and swing leg to clear the ground.

Swing Phase

The swing phase consists of the following:

1. *Acceleration:* This is the phase of acceleration of the swinging leg by the hip flexors taking it to the front of the body
2. *Mid-swing:* The leg continues to swing forwards
3. *Deceleration:* Slowing down of the swing to get the foot ready for the heel strike
4. *Heel strike:* Heel striking the ground again

Each leg alternately goes through a stance (support) phase and swing phase. The repetition of such cycles rhythmically carries the body forwards in normal walking.

Normal Walk

The normal walk of an adult of 70 kg weight has a speed of 3 miles per hour and 90–100 steps per minute. The centre of gravity of the body

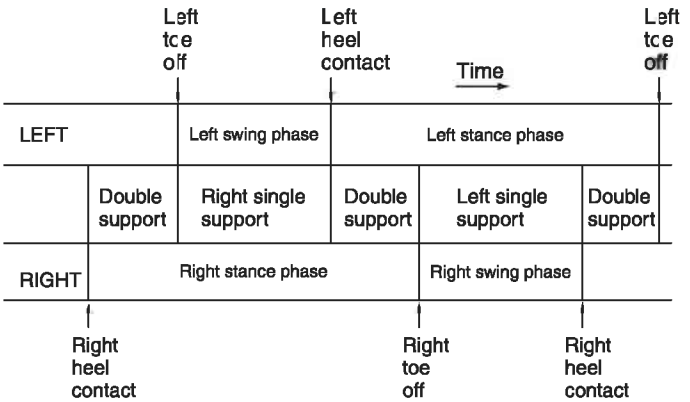


FIGURE 1.3 Phases of gait cycle.

rises and falls about 2 inches with each step. The normal gait is performed with the least expenditure of energy and hence it is mechanically a very efficient type of motion.

Running

In running, the sequences are faster and there is a moment in the cycle when both feet are off the ground.

ABNORMAL GAIT

The characteristic features of the gait in neurological disorders of the cerebellum and the spinal tracts are well described in textbooks of medicine. The gaits due to common orthopaedic disorders will be briefly described here.

The following are the common pathological gaits noticed in patients with orthopaedic disorders:

1. Painful hip gait (antalgic gait)
2. Stiff hip gait
3. Unstable hip gait
   - Trendelenburg gait
   - Gluteus medius gait
4. Gluteus maximus gait
5. Quadriceps gait
6. High stepping gait
7. Short leg gait
8. Scissoring gait
9. Stiff knee gait

Antalgic or Painful Hip Gait

Antalgic or painful hip gait is the gait of a person with a painful condition in the hip joint. To minimise the pain, the person shortens the time duration of the stance phase on the painful side and quickly transfers the weight to the painless leg. Thus, there is a longer stance on the normal leg and a much shorter stance on the painful leg. This antalgic gait is also seen in painful conditions in the knee or the foot.

Stiff Hip Gait

When one hip is ankylosed, it is not possible to flex at the hip joint during walking to clear the ground in the swing phase. Hence, the person with a stiff hip lifts the pelvis on that side and swings the pelvis with the leg in circumduction and moves it forwards.

Unstable Hip Gait

The stability of the hip in walking is provided by the bony components of the joint being kept in a stable position by the muscles and ligaments around the joint and the normal alignment of the line of gravity through the joint. The abductor muscles (gluteus medius, minimus and tensor fascia lata) of the joint play a very important role in the stability during the stance phase. For the effective action of these muscles, the fulcrum is at the hip joint.

When a normal person stands on one leg (e.g. right leg), the centre of gravity has to shift

to the right side and the line of gravity has to pass through the right hip and leg to the ground within the base of support. The right gluteus medius muscle contracts from its insertion below and pulls the pelvis down on the right side and thus shifts the centre of gravity to the right side and the body is stabilised. Any disruption of the stabilising system can disturb the stability during the stance phase of the gait.

The disruption can be anatomical or physiological.

Anatomical disruption occurs due to any discontinuity in the femoral neck or the hip joint. This occurs in non-union of the fracture neck of femur or dislocation of the hip joint.

Physiological or functional disruption occurs when the hip abductor muscles are weakened or paralysed as in poliomyelitis.

The disruption in the right hip can cause instability on the right side as follows.

Trendelenburg Gait

This gait is typically seen in the following conditions:

- Anatomical disruption on the right side
- Non-union fracture neck of femur and dislocation of hip joint

Under the above circumstances, the action of the gluteus medius in pulling the pelvis downwards in the stance phase is ineffective or weak due to the lack of a stable fulcrum. The pelvis drops on the opposite (i.e. left) side causing instability. To restore the stability and prevent fall to the left, the body lurches to the right side and shifts the centre of gravity over the stance side right hip. The top of the greater trochanter telescopes upwards and mechanically hitches on the ilium, to stabilise the joint. This movement at every stance phase is called the Trendelenburg gait.

Gluteus Medius Gait

This gait is typically seen in the following condition:

- Physiological disruption on the right side

When the right gluteus medius is paralysed, it is unable to pull down the pelvis on the right due to a functional deficiency of the abductor mechanism in the stance phase. The shifting of the centre of gravity is done by the patient actively swaying the whole body to the right. This lurching of the body to the right stabilises the person. While walking, there is lurch of the body to the right during every stance phase on the right leg. This is called the gluteus medius gait.

The biomechanical cause of the limping gait in both cases is the failure of the abductor mechanism of the hip. In the first it is the failure of the fulcrum and in the second it is the weakness of the power arm.

Gluteus Maximus Gait

The gait of a person with paralysis of gluteus maximus muscle has a typical backward lurch during every step forwards. This is seen typically in anterior poliomyelitis.

Normally, when the body moves forwards in the mid-stance phase, the hip is extended by the gluteus maximus acting from its insertion below and tilting the pelvis backwards to retain the centre of gravity over the supporting leg.

When the gluteus maximus muscle is paralysed, the stabilising factor is lost and the patient leans backwards at the hip to passively extend it and keep the centre of gravity over the stance leg. This causes the backward lurch in the gluteus maximus gait.

Quadriceps Gait

Quadriceps gait is seen typically in patients with paralysis of the quadriceps muscle due to poliomyelitis. Normally, during the last stages of the stance phase in walking, the knee is actively extended fully and is locked by the full power of the quadriceps. When the quadriceps is weak or paralysed, the locking is done by passively pushing the knee backwards by the patient putting his hand over the front of the lower thigh. This results in a limp and may even cause a genu recurvatum in course of time. This limping gait with the hand on the hyperextended knee is typical of the quadriceps gait. The leg is also kept in external rotation with the foot pointing outwards to broaden the base of support and to improve the stability.

High Stepping Gait

When there is a foot drop, the foot slaps on the ground on heel strike and then drops in the swing phase. To get the foot off the ground, the hip is flexed more and this causes the high stepping gait.

Short Leg Gait

Inequality of the legs is obvious when the shortening of one leg is more than 1 inch. Up to 1/2 inch, the shortening is totally masked by the pelvic tilt and hence can be ignored. Shortening up to 11/2 inch could be masked by a slight equinus position of the foot. Gross shortening of more than 2 inch leads to a gait with a marked pelvic tilt downwards and an equinus deformity at the foot.

Scissoring Gait

Scissoring gait is the characteristic gait of a spastic child with marked bilateral adductor spasm at the hips and equinus spasm in the ankle. The child needs support to walk and the legs go into marked abduction in the swing phase so that the foot with equinus goes across to the opposite side. Such repeated crossings of the leg while walking give a scissoring appearance and hence are called the scissoring gait.

Stiff Knee Gait

The pelvis is raised during the swing phase, in the case of ankylosed or stiff knee.

KEY POINTS

- Orthopaedics is the science and art of the diagnosis and treatment of all diseases and disorders of the human locomotor system.
- Origin of word—'ortho' means straight and 'pedios' means child.
- Important terms used in orthopaedics include arthrotomy: opening of joint, arthrodesis: fusion of joint, arthroplasty: surgical refashioning of joint and osteotomy: surgical cutting of bone.
- Deformities can be broadly grouped as congenital deformities and acquired deformities.
- Deformities can be corrected by conservative or surgical methods.
- Surgical methods include soft tissue operations and bone operations.
- Normal gait includes the following sequence of events: Heel strike, stance phase, toe off and swing phase.
- A normal walk cycle extends from the heel strike of one leg to its next heel strike.

MULTIPLE CHOICE QUESTIONS

1. The term 'orthopaedics' was coined by
   a. Nicholas Andry
   b. Hugh Owen Thomas
   c. Thomas Bryant
   d. Sir Rober Jones
2. Trendelenberg test is positive in injury of
   a. Superior gluteal nerve
   b. Pudendal nerve
   c. Inferior gluteal nerve
   d. Obturator nerve
3. Stance phase muscle among the following is/are
   a. Quadriceps
   b. Hamstring
   c. Gastrocnemius-soleus
   d. Tibialis anterior
   e. Peroneus longus
4. The paralysis of which of the following causes lurching gait
   a. Adductor muscles
   b. Gluteus maximus
   c. Iliopsoas
   d. Gluteus medius and minimus

CHAPTER 2

Congenital Deformities

AETIOLOGY OF CONGENITAL DEFORMITIES

The exact aetiology of congenital deformities is still not established. There are several causative factors. Some are intrinsically genetic factors. Others are factors operating on the developing foetus during its intrauterine development. There are also combinations of the above factors causing some defects (Table 2.1).

The teratogenous influence of drugs and chemicals on the developing chick embryos and experimentally produced club feet, spina bifida and other congenital malformations in chicken has been demonstrated.

The atom bombs dropped in 1945 in Hiroshima during the Second World War did result in an enormous number of congenital deformities in children, born to mothers who were pregnant and living in the periphery of those cities, as witnessed by a senior author in 1947. Nuclear radiation from accidental leaks from nuclear power stations can cause larger disasters even now.

The congenital and developmental deformities and disorders can be classified into groups according to the stage of development of the foetus at which the causative teratogenic factors operate as shown in Table 2.1.

In many cases, a combination of factors cause congenital disorders and deformities and it is not possible to pinpoint the exact pathogenesis; for example, the paralysis in lower limbs due to spina bifida and meningomyelocele causes further deformities in the legs and feet due to muscle imbalance.

LOWER LIMB

The common congenital deformities of the lower limb are as follows:

- Congenital talipes equinovarus
- Congenital dislocation hip
- Congenital genu recurvatum
- Congenital pseudarthrosis tibia

Congenital Talipes Equinovarus (Club Foot)

Congenital talipes equinovarus (CTEV) is one of the most common congenital deformities in India compared to Western countries. The word 'Talipes' is derived from 'Talus' and 'Pes' and was applied to those walking on their neglected deformities wherein the talus rested on the ground as the foot (Pes).

It is characterised by a foot plantarflexed at the ankle (equinus), inverted at the subtalar joint (varus) and adducted at the forefoot. The deformity is bilateral in approximately 50% of cases and is more common in males. Incidence of CTEV in India is 2 per 1000 live births.

TABLE 2.1 Aetiology of Congenital Deformities

Stage of Development	Aetiological Factor	Congenital Deformity/ Defect Produced
Cellular stage	Genetic	Achondroplasia Polydactyly. Vicious club foot Arthrogryposis
Embryonic stage	Chromosomal Malformations Teratogens	Down's syndrome
Early foetal stage of genesis of limb and organs	Drugs Thalidomide Aminopterin Alcohol Steroids Irradiation X-ray Radiotherapy Maternal diseases German measles Diabetes	 Congenital limb deficiencies Club foot Spina bifida Developmental dysplasia hip Amelia Phocomelia Hemimelia
Late foetal stage	Combination of factors Position in uterus Mechanical factor Postural factor	Structural deformities Club foot Genu recurvatum

Aetiology

The exact cause of CTEV deformity is not known.

- Mechanical theory: According to Hippocrates, the talipes deformity is of mechanical origin and is caused by an abnormal intrauterine position of the foetus.
- Germ plasm theory: Another theory is that a primary germ plasm defect in the talus causes hypoplasia of this bone with subsequent soft tissue changes in the joints and musculotendinous complexes.
- The third hypothesis is that primary soft tissue abnormalities within the neuromuscular units cause secondary bony changes.

Anatomy

It is essential to know about the normal anatomy of the foot before going to the pathology.

The foot is divided into forefoot, midfoot and hindfoot. The hindfoot comprises subtalar joint and calcaneocuboid joint. Midfoot includes talonavicular and midtarsal joints. Forefoot includes tarsometatarsal joint and the others beyond them.

The soft tissues around the foot include tendons such as tibialis posterior, flexor hallucis longus, flexor digitorum longus, abductor hallucis and ligaments such as spring ligament, plantar ligament and deltoid ligament.

Pathology

The basic pathology is a medial talonavicular dislocation. All pathological changes in the joints and bones of hindfoot are due to this basic pathology.

Medially dislocated navicular is found close to medial malleolus and is rotated in its longitudinal axis. The head of talus is uncovered and looks downwards and laterally. It is also plantarflexed. The spring ligament between the sustentaculum tali of calcaneum and navicular is contracted.

The calcaneum is inverted so that its medial tuberosity approaches the medial malleolus. This

leads to medial shift of the tendocalcaneus thereby accentuating the deformity.

The soft tissues on the posterior aspect of the ankle and medial aspect of the foot are contracted. The tendo achilles, tibialis posterior, plantar fascia, spring ligament, deep plantar ligaments, flexor hallucis longus, flexor digitorum longus and abductor hallucis are contracted. The capsules of the subtalar, talonavicular and naviculo cuneiform joints are also contracted.

In long-standing cases, callosities develop on the lateral aspect of the dorsum of the feet.

Clinical Features

The shape and contour of the foot are characteristic (Fig. 2.1). There is equinus deformity at the ankle, varus deformity at the subtalar joint and adduction deformity at the tarsometatarsal joints. The deformity is graded as mild, moderate or severe. The foot on the whole is smaller than normal and the heel is poorly developed. There is a crease across the medial border of the foot. The lateral malleolus is very prominent while the medial malleolus is buried in depression. The foot cannot be passively dorsiflexed and everted. There is wasting of the calf due to atrophy of gastrosoleus muscles.

Differential Diagnosis

Equinovarus deformity at birth may be associated with spinal defects such as meningomyelocele (neurogenic), congenital abnormalities of muscle (myogenic) or congenital skeletal defects such as tibial hemimelia (osteogenic). In a child seen after the neonatal period, the possibility of the deformity being secondary to lateral popliteal nerve palsy, poliomyelitis or cerebral palsy must be thought of.

Radiological Features

- *Foot–anteroposterior view:* Normally, the line along the axis of the talus roughly passes anteriorly through the navicular and the shaft of the first metatarsal bone. In the club foot, this line is deviated lateralwards and passes along the third or fourth metatarsal bone, due to the inward displacement of the navicular and metatarsal bones (Fig. 2.2).
- *Foot–lateral view*

Lines drawn through the long axis of the talus and calcaneus normally cross at an angle of 20–40°. But in club foot, the two lines may be almost parallel.

The lateral film is taken with the foot in forced dorsiflexion. Lines drawn through the midlongitudinal axis of the talus and the lower border of calcaneum should meet at an angle of about 40°. Anything less than 20° shows that the calcaneum cannot be dorsiflexed as in CTEV.

Treatment

The main principle is to correct the deformity and hold the foot in the corrected position. The best

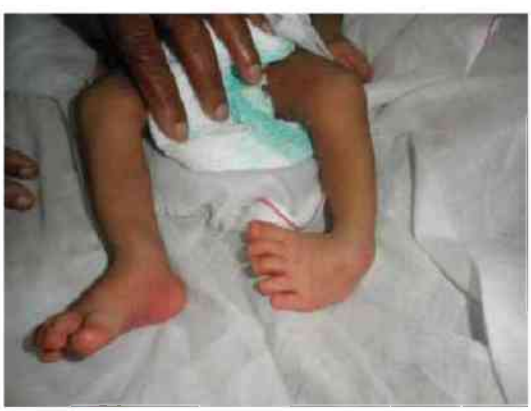


FIGURE 2.1 Clinical photo showing club foot deformity in left lower limb.
Courtesy: Dr Rujuta Mehta.

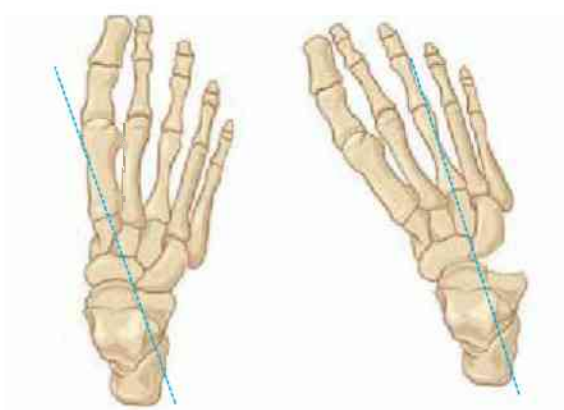


FIGURE 2.2 Line drawing of (a) normal foot and (b) club foot. Note the medial talonavicular dislocation in (b).

results will be obtained if the following principles are followed:

- The treatment should begin in the first few weeks of life.
- The different components of the deformity should be corrected in a proper sequence: first, the forefoot adduction; then, the inversion and finally, the equinus. Failure to follow the correct sequence may break the foot in the midtarsal region, creating a highly refractory 'rocker bottom' deformity.
- The use of minimum force in the correction gives a better functioning foot. Forcible manipulation should not be employed.

Conservative Treatment

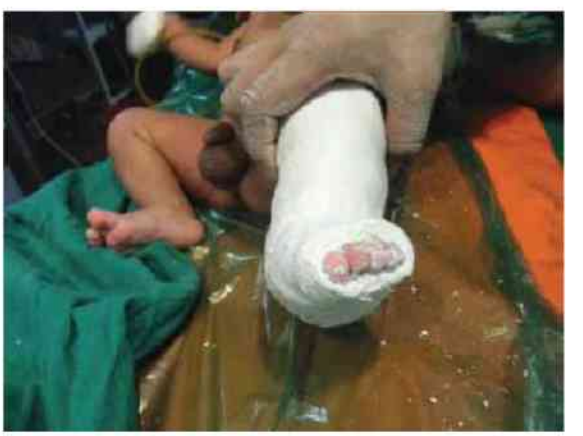
- *In the first 3 weeks:* The mother is taught to mould the infant's foot twice a day.
- *Three weeks to 3 months:* The surgeon manipulates the foot once in 2 weeks and the position is maintained by plaster of Paris cast (Fig. 2.3).
- *Maintenance of correction:* After four or five manipulations, the foot is usually in normal shape but it needs a retentive splint or shoe. Correction is maintained by below-knee splints with straps to hold the foot in position. Special club foot boots maintain the correction and prevent relapse.

Surgical Treatment

In cases where progressive correction of deformity is not achieved by casting, surgery is indicated. The following are the surgical procedures used:

1. Soft tissue operations
   - Posteromedial soft tissue release
   - Complete subtalar release
2. Bone operations
   - Lateral wedge resection
   - Calcaneocuboid fusion

(a)



(b)

FIGURE 2.3 (a, b) Clinical photos of corrective plaster cast applied to correct club foot.
Courtesy: Dr Rujuta Mehta.

1. Soft tissue operations
   - *Posteromedial soft tissue release:* This is the most common surgical procedure performed in children of an age group from 5 months to 4 years. In this operation, in addition to the lengthening of the tendo achilles, the tibialis posterior, flexor hallucis longus and flexor digitorum longus tendons are also lengthened. All the ligaments and soft tissues on the plantar and medial surfaces of the calcaneum, talus and navicular bones are divided. The talonavicular joint is reduced.
   - *Complete subtalar release:* In severe cases of club foot, a complete release of all tight

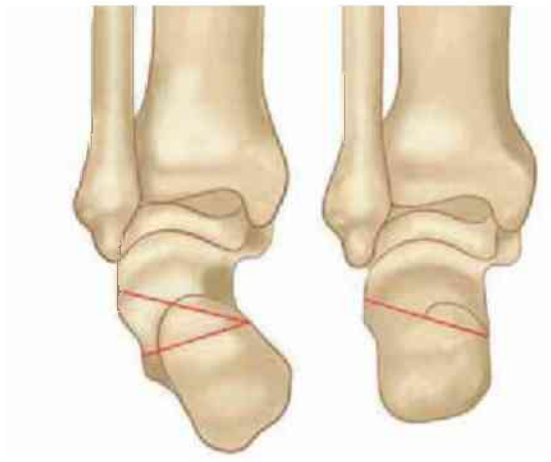


FIGURE 2.4 Diagrammatic representation of Dwyer's osteotomy.

structures in the posterior, medial and lateral aspects of the subtalar and ankle joint is done.

2. *Bone operations:* Between the ages of 4 and 8 years, soft tissue operations alone will not succeed in correcting the foot. In addition to the posterior and medial release, a lateral wedge osteotomy of calcaneum (Dwyer's osteotomy) (Fig. 2.4) and calcaneocuboid fusion (Dilwyn–Evan's procedure) by stapling will be necessary to get full correction.

Non-Surgical Treatment: External Fixators

In old age and recurrent club feet, the fibrosis is severe and open surgery is difficult to perform. These are best treated by Ilizarov external fixation or Joshi apparatus. These are non-invasive techniques. The principle of controlled differential distraction is applied to correct all the aspects of the deformity by gradual sequential stretching of soft tissues.

Neglected Club foot

Cases of neglected club feet presenting themselves even at the age of 8–10 years are occasionally seen in our country (Fig. 2.5). For these cases, resection of suitable wedges of bone and a triple arthrodesis gives a good plantigrade foot. In this operation, the talocalcaneal, talonavicular and the calcaneocuboid joints are fused.

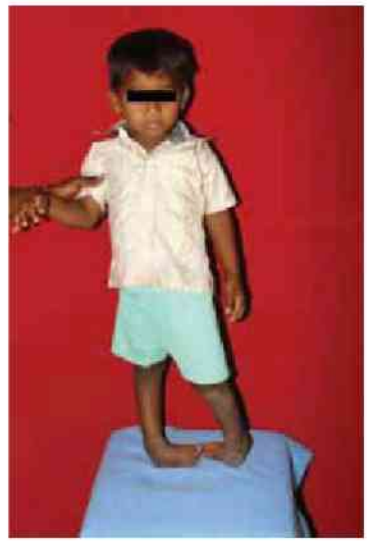


FIGURE 2.5 Clinical photo of neglected bilateral club feet.

Developmental Dysplasia of the Hip

Developmental dysplasia of the hip (DDH) is a condition where there are various degrees of displacement of the femoral head from the acetabulum. An incomplete displacement of the head of the femur is called ***subluxation*** and a complete displacement is called ***dislocation***. In earlier days, it was known as congenital dislocation of the hip.

Incidence

Developmental dysplasia of the hip is an uncommon condition in India but is very common in Europe and America. It is not clear why this condition is rare in India and in the Eastern countries. Probably, many cases of subluxation of the hip get reduced spontaneously when the child is carried on the side or back of the mother or elder sister, as in India (Fig. 2.6). It is more common in girls and usually unilateral.

Aetiology

Genetic and environmental factors appear to contribute to its causation.

- Genetic factor may play a part in the aetiology for DDH as it seems to run in families.
- Hormonal factor—high levels of maternal

(a)

(b)

FIGURE 2.6 (a, b) Photos showing the posture adopted by children when carried in their mother's hip. *Courtesy:* (b) Dr Rujuta Mehta.

oestrogen, progesterone and relaxin in the last few weeks of pregnancy may produce ligamentous laxity with dislocation of hip.

- Intrauterine malposition may be another factor in the causation of dislocation of hip.
- Primary dysplastic development of the acetabulum.

Types

There are two types of DDH. In the more common type, excessive laxity of the hip joint capsule and ligaments results in subluxation and dislocation. This type is described below. In the other rarer type, dysplasia or poor development of the acetabulum is the cause for the dislocation. This is the teratological type and is more difficult to treat.

Pathology

The main changes are seen in the capsule of the hip joint. The capsule is excessively loose in the newborn.

The capsule is thickened where it is in contact with the upper part of the head. The lower part of the capsule, as it crosses the empty acetabulum, gets adherent to it. There is hourglass contracture where the iliopsoas tendon passes between the head and the acetabulum (Fig. 2.7). The acetabular labrum is inverted into the joint (inverted limbus) and the ligamentum teres is enlarged.

Ossification of head is delayed. The head is small and flattened. The neck shows increased valgus and anteversion. The iliopsoas and adductor muscles get shortened. The acetabulum shows varying degrees of dysplasia.

Clinical Features

In a newborn child suspected to have DDH, there will be obvious shortening of the leg. Additional creases in the posterior and medial aspects of the

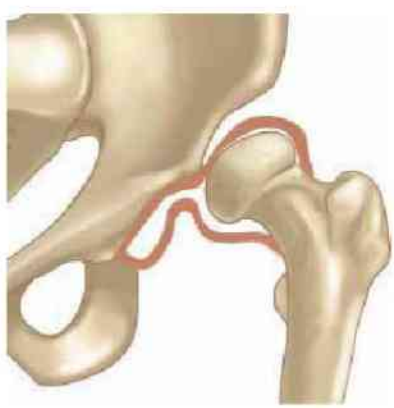


FIGURE 2.7 Diagrammatic representation of hourglass contracture of the hip joint capsule.

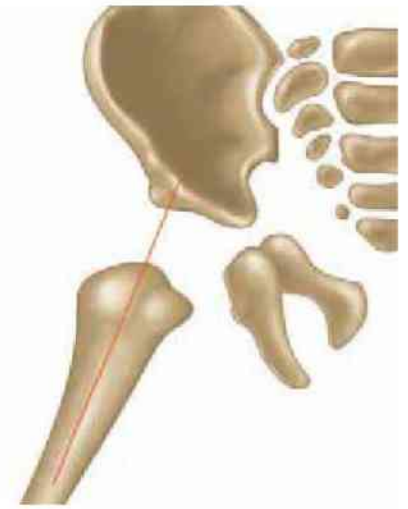


FIGURE 2.10 Diagrammatic representation of Von Rosen's line.

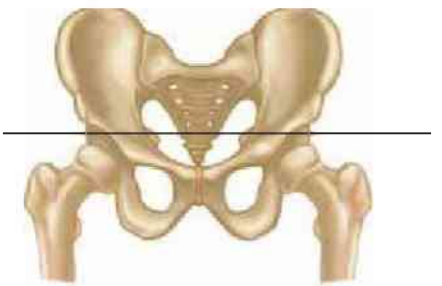
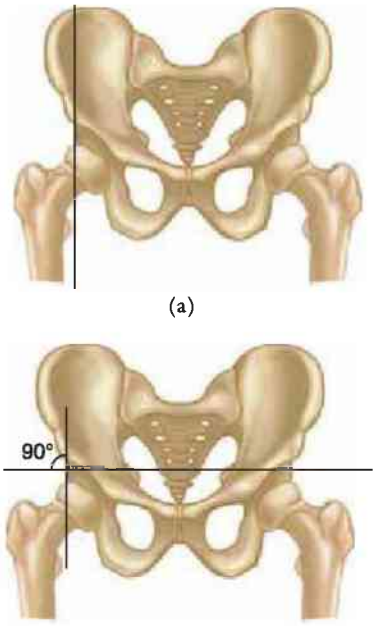


FIGURE 2.11 Diagrammatic representation of Hilgenreiner's line.



(a)

(b)

FIGURE 2.12 (a, b) Diagrammatic representation of Perkin's line.

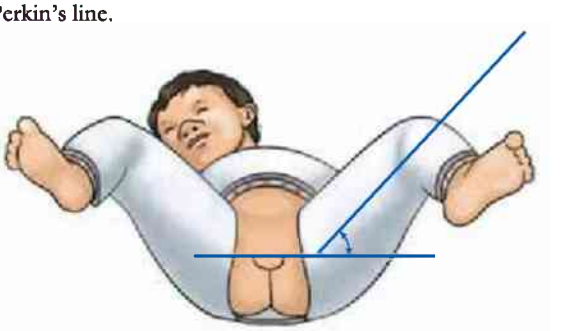


FIGURE 2.13 Diagrammatic representation of human position for hip spica application.

stable. The treatment is age related and depends on the age at which the child is brought.

Treatment in Infants Below 3 Months

Splints such as Von Rosen's splints and Pavlik harness are applied to retain the position of the head till stability is achieved.

Treatment in Children From 3 to 6 Months

The dislocation is reduced by gentle manipulation under general anaesthesia and immobilised in plaster cast, with the hip at 90° flexion and 45° abduction. This is the 'human position' as described by Kumar (USA) (Fig. 2.13).

Treatment in Children From 6 to 12 Months

A preliminary traction and gradual abduction is done for 2–3 weeks before reduction under general anaesthesia. Children will need adductor tenotomy if there is resistance to reduction by its tightness. The *plaster spica* is then applied.

Treatment in Children From 1 to 3 Years

In this age group, after preliminary traction, surgical open reduction will be required to reduce the head of femur into the acetabulum. In cases with excessive valgus and anteversion of the femoral neck, ***femoral varus derotation osteotomy*** is done for stability.

Treatment in Children From 3 to 6 Years

In this age group, there are acetabular changes present. After open reduction of the hip and

femoral osteotomy, an osteotomy of the innominate bone such as *Salter's* or *Pemburton's osteotomy* is also done. This is to alter the alignment of the acetabulum to retain the head in stable position.

Neglected Developmental Dysplasia of the Hip

Neglected DDH may present after the age of 10 years with pain in the hip and unstable gait. These will require pelvic stabilising operations such as *Schantz osteotomy.*

Complications

Avascular necrosis of the head of the femur is a complication of forceful reduction of the hip in childhood. Neglected cases develop painful instability of the hip and in later years osteoarthrosis of the hip.

Congenital Genu Recurvatum

Congenital genu recurvatum is a congenital hyperextension of the knee with inability to flex the knee. This is due to contracture of the quadriceps muscle. In its severe form, the knee will be subluxated or dislocated anteriorly. Mild forms are treated by gentle manipulation and plaster in flexion. More severe forms will need surgery in the form of quadriceps lengthening.

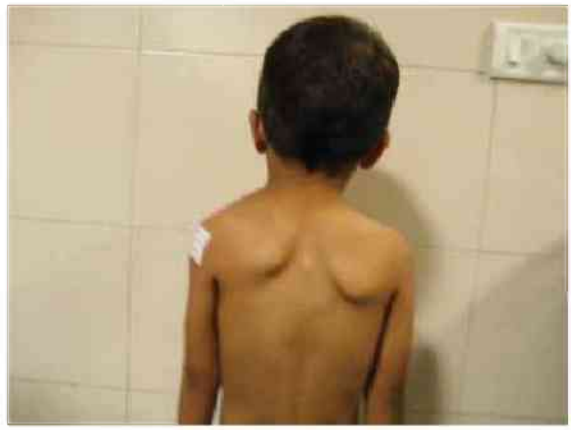
UPPER LIMB

The following are some important congenital deformities of the upper limb:

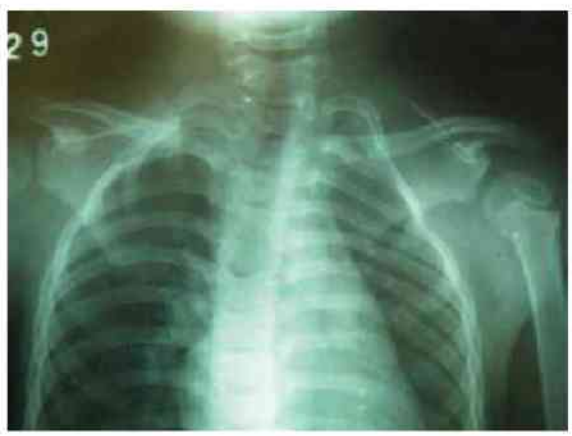
- *Shoulder:* Sprengel's shoulder
- *Elbow:* Congenital superior radioulnar synostosis
- *Wrist:* Madelung's deformity
- *Hand:* Syndactylism, polydactylism

Sprengel's Shoulder

Sprengel's shoulder is a condition where there is a congenital elevation of the scapula (Fig. 2.14a). Developmentally, the scapula has failed to descend from its embryological position in the neck. Scapula is higher in position than normal and is usually small and prominent.



(a)



(b)

FIGURE 2.14 (a) Clinical photo of Sprengel's shoulder (left side). (b) X-ray of the chest showing Sprengel's shoulder (left side).
Courtesy: Dr Rujuta Mehta.

The condition is treated by surgical release of muscles and repositioning the scapula, at an earlier age, usually within 6 years.

Clinical Features

The patient presents with an elevated shoulder on the affected side. The superior angle of the scapula is seen and felt prominently in the neck under the upper border of the trapezius muscle. The vertical height of the scapula is smaller and medial border appears prominent. There is only slight limitation of abduction (especially after 90°) of the shoulder joint and minimal interference with function.

There may be associated abnormalities such as fusion of two or three cervical vertebrae resulting in congenital brevicollis or short neck with a low hairline in the neck. This combined abnormality is called '*Klippel–Feil syndrome*'.

Radiological Features

Radiograph shows the elevated position and diminished size of the scapula (Fig. 2.14b). It may also show a bony bar connecting the lower cervical vertebra with the superomedial angle of the scapula and is called the *omovertebral bone.*

Treatment

In mild cases, no treatment is necessary. In severe cases, surgery can be done with surgical release of muscles and repositioning the scapula, at an earlier age, usually within 6 years of life.

Klippel–Feil Syndrome

The clinical features of this syndrome (Fig. 2.15) are as follows:

- Bilateral high riding scapula
- Short neck with low hairline
- Webbing of neck
- Cervical spine abnormalities

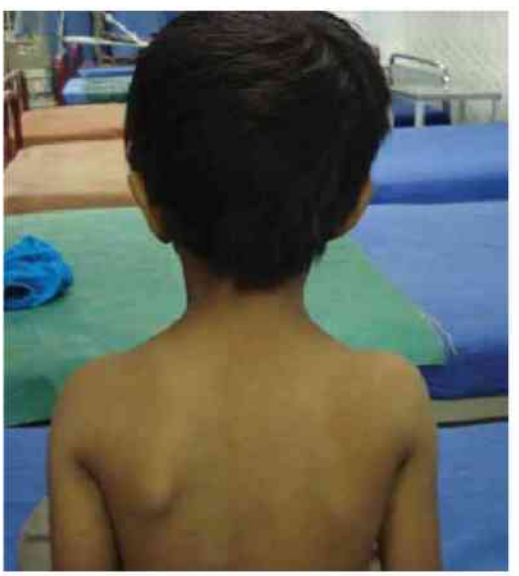


FIGURE 2.15 Clinical photo of Klippel–Feil syndrome. Note the low hairline and short neck.

Winging of Scapula

Weakness or paralysis of serratus anterior causes this deformity. This may occur following injury to brachial plexus, long thoracic nerve or muscular dystrophy.

Congenital Radioulnar Synostosis

Congenital radioulnar synostosis is a condition where there is fusion of the superior radioulnar joint. It is often bilateral. The condition is detected only at the age of 3 or 4 when the child uses the hand for various activities.

Clinical Features

There is an inability to supinate the forearm as the fusion is mostly in the fully pronated position. Eating with the hand is awkward, as the food tends to drop down when the hand is taken to the mouth. On attempted supination and pronation, there is no movement palpable over the head of the radius.

Radiological Features

The proximal end of the radius is found deformed. It is fused to the upper end of the ulna for about 1–3 cm. Both elbows must be radiographed (Fig. 2.16).

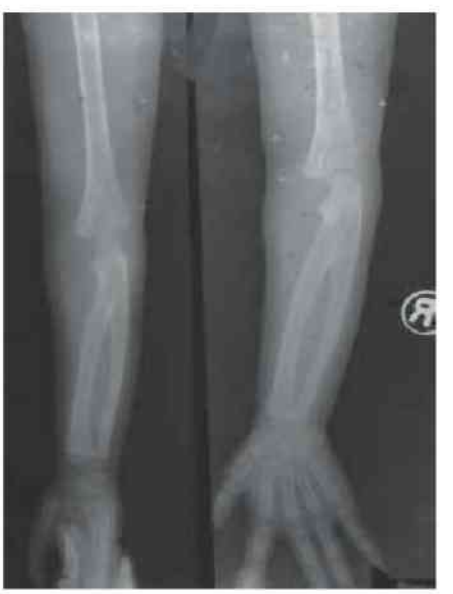


FIGURE 2.16 X-ray of the right forearm lateral and anteroposterior views showing congenital radioulnar synostosis.

Treatment

This is a difficult condition to treat. Excision of the upper end of the radius is only partially successful as the condition may be associated with other congenital defects such as absence of supinator muscle. In cases where the forearm is in full pronation, the function may be improved by a derotation osteotomy of radius.

Madelung's Deformity

Madelung's deformity is a deformity at the wrist where there is dorsal and lateral bowing of the distal radius (Fig. 2.17), shortening of the radius and subluxation of the inferior radioulnar joint.

This is due to a growth disorder of the ulnar half of the distal radial epiphysis. It is usually bilateral and more common in girls.

Clinical Features

Clinically, the wrist is deviated to the ulnar side and there is a prominence of the distal end of the ulna due to dorsal dislocation of the head of ulna. The range of dorsiflexion of the wrist is diminished and palmar flexion is increased.

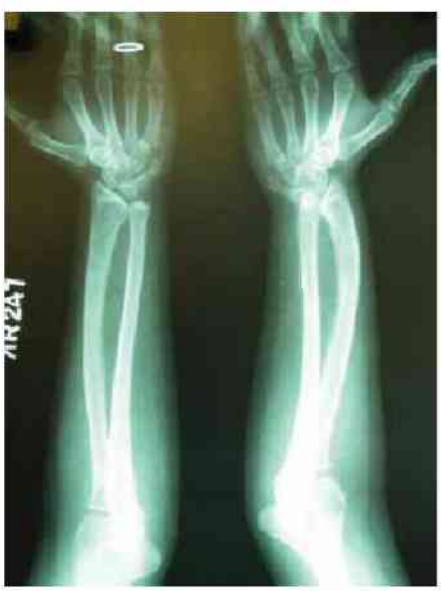


FIGURE 2.17 X-ray of the forearm anteroposterior and lateral views, demonstrating Madelung's deformity. Note the dorsal and lateral bowing of radius. *Courtesy:* Dr Rujuta Mehta.

Radiological Features

Radiograph of wrist shows arrested development of the ulnar half of the epiphyseal plate, and the articular surface of the radius faces ulnarwards and anteriorly.

Acquired Form

There is also an acquired form where a similar deformity is caused by trauma or infection destroying the distal radial epiphyseal plate. This is due to the arrested development of the ulnar and volar part of the distal end of radius.

Surgery

Surgery may be needed for pain and limitation of movement. Excision of the lower end of the ulna is only partially helpful (Darrach's procedure). In severe cases, corrective osteotomy of the radius to realign the wrist is done.

Syndactylism

Syndactylism is a condition characterised by the fusion of two or more fingers. The fusion of two fingers is usually total. The treatment is by surgical separation of the digits and skin grafting of the raw areas.

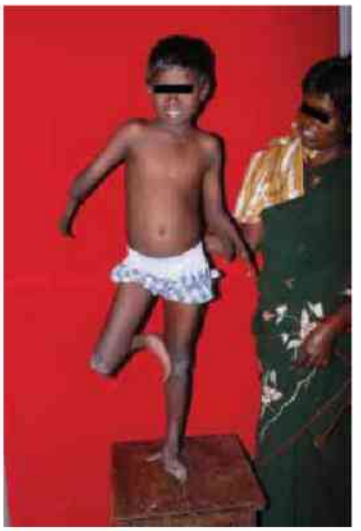
Polydactylism

Polydactylism is the congenital presence of an extra finger or part of a finger. It is attached to a normal digit or to the radial or ulnar aspect of the hand. If the main finger has functioning tendons and nerves, the extra digit may be excised to improve the appearance of the hand.

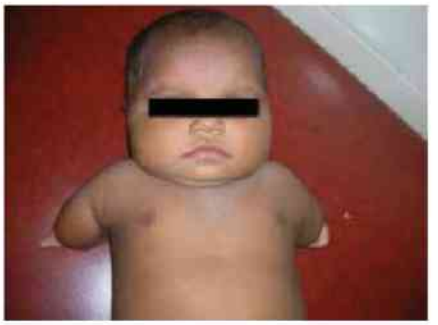
CONGENITAL SKELETAL LIMB DEFICIENCIES

Congenital skeletal limb deficiencies are deficiencies of a part or whole of the limb of a child at birth. These deficiencies have been classified for clinical purposes as follows:

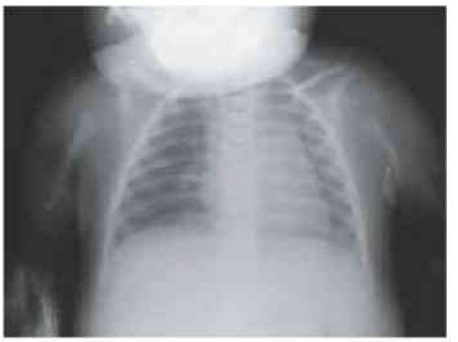
- *Terminal deficiencies:* Congenital amputation at elbow, wrist, knee or ankle (Fig. 2.18a)
- *Intercalary deficiencies:* The intermediate portion of the limb is absent



(a)



(b)



(c)

FIGURE 2.18 (a) Clinical photo of terminal deficiency. (b) Clinical photo of phocomelia. (c) X-ray of the chest with both arms demonstrating phocomelia. Note the absence of forearm bones.
Courtesy: (b, c) Dr Rujuta Mehta.

Each of the above deficiencies is further subdivided into transverse or longitudinal (axial) deficiencies. Some important examples are as follows:

- *Amelia:* The term indicates absence of the whole limb (terminal transverse deficiency)
- *Phocomelia:* Absence of the proximal part of the limb (intercalary transverse deficiency; Figs 2.18b and c, for example the hand is found attached to the trunk. The tibia with foot is attached to the pelvis)

Hemimelia (Longitudinal Deficiency)

- Absence of one of the forearm bones and the corresponding portion of the metacarpals and fingers
- Absence of the fibula and the outer two rays of the foot

Congenital Absence of the Radius (Radial Club Hand)

Radial club hand is a type of congenital hemimelia characterised by a partial or total absence of the radius and the thumb (Fig. 2.19).

This results in gross radial deviation of the hand and wrist with overgrowth and curvature of the ulna. The muscles on the radial side of the

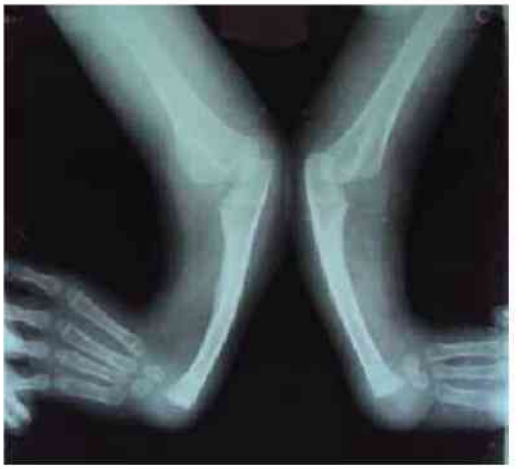


FIGURE 2.19 X-ray of both forearms showing congenital absence of radius and thumb (radial club hand).

forearm are also defective. Radial nerve will have anomalous course.

In infancy, the deformity is corrected by repeated splinting of the forearm and wrist.

When the soft tissues are stretched out, the carpus is surgically centralised on the lower end of the ulna, with the wrist in the corrected position. The pollicisation of the index finger (i.e. reconstruction of the index into a thumb) is done at the age of 1 year to obtain a functioning hand.

Congenital Absence of the Fibula

Fibula is the most frequently absent long bone. The salient features of this deficiency are as follows:

- Complete or partial absence of the fibula
- Anteromedial bowing of the tibia with shortening
- Equinovalgus deformity of the foot
- Absence of the lateral two rays of the foot

The severe forms are bilateral, associated with proximal femoral deficiency. These patients need correction of the tibial and foot deformity and also procedures for equalising the length of the limb. Very severe and useless rudiments may need amputation and prosthetic restoration.

NECK AND SPINE

The following are the common deformities in the neck and spine:

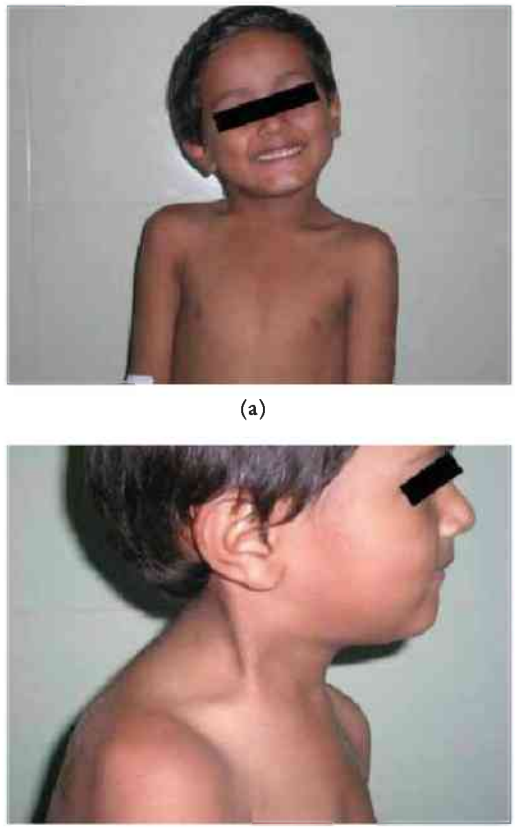
- Congenital torticollis
- Spina bifida
- Congenital scoliosis

Congenital (Infantile) Torticollis

Congenital torticollis is a deformity of the neck characterised by tilting of the head to one side. There is a rotation of the occiput to one side and the chin to the opposite side (Fig. 2.20).

Aetiology

The basic feature of this condition is a contracture of the sternomastoid muscle on one side.



(a)

(b)

FIGURE 2.20 (a, b) Clinical photographs showing right side torticollis.
Courtesy: Dr Rujuta Mehta.

The exact pathology leading to the contracture is not known. It may be a myodysplasia of genetic origin. In some cases, it is due to a birth trauma causing either a haematoma or arterial ischaemia in the lower third of the muscle. This condition is commonly associated with breech presentation.

Secondary causes of torticollis (secondary torticollis) are as follows:

- Bone anomalies
- Trauma
- Retropharyngeal abscess
- Ocular problems

Clinical Features

The child may be brought soon after birth when the mother notices the tilting of the head to one

side and often a swelling in the neck. There may be a firm swelling at the junction of middle and distal third of the sternomastoid muscle. This is called the sternomastoid tumour. In cases due to birth trauma, it may be associated with fractures of the clavicle or Erb's palsy. In a left sternomastoid torticollis, the chin is tilted upwards to the right side and the occiput is tilted down on the left side.

Radiography may show congenital anomalies in the cervical spine such as hemivertebrae or partial fusion of the cervical vertebrae in cases where the sternomastoid is normal. In neglected cases, the contracture is severe and cannot be stretched passively. There is facial asymmetry, as the face in the affected side is not well developed. A new macula develops in the fundus to make the vision level in the tilted position of the head.

Treatment

In the newborn stage, the sternomastoid is stretched passively by the physiotherapist. The treatment in later cases is mainly surgical tenotomy of the lower end but sometimes at the upper end or at both ends of the sternomastoid. After 4–6 weeks, the child is taught neck exercises.

SPINA BIFIDA

Spina bifida is a congenital condition of the spinal cord where the spinal cord lies exposed without the complete closure of the vertebral arches, most commonly seen in the lumbosacral region. The incidence of this disease has drastically come down due to the introduction of folate supplementation before conception and in early pregnancy.

Embryology

Once the neural canal is formed from the surface ectoderm, there is a sheet of mesoderm anterior to it that develops into the notochord. The vertebral bodies develop around the notochord. Two projections arise from the body that grow around the neural canal to form the vertebral arch. The fusion of the two halves occur behind the thoracic region first, from where the fusion extends cranially and caudally. Spina bifida results when this fusion fails and it results in a gap in the vertebral neural arch.

Types

Spina bifida is classified into two types:

1. Spina bifida cystica
2. Spina bifida occulta

Spina Bifida Cystica

Spina bifida cystica is the more severe of the two. The defect involves the vertebral arches and also the overlying meninges, soft tissues and skin. They can be classified according to the severity of the defect as follows:

- Meningocele
- Myelomeningocele
- Syringomyelocele
- Myelocele

Meningocele

Meningocele involves protrusion of meninges through the bony arch defect but there is no involvement of spinal cord. The protruded sac contains only cerebrospinal fluid. As it does not involve spinal cord, there is no paralysis.

Treatment

Surgical closure is indicated.

Myelomeningocele

Majority of spina bifida cystica is of myelomeningocele type. It is characterised by protrusion of meninges along with a plaque of neural tissue. Infection may set in and death may ensue due to meningitis.

Treatment

Surgical closure is mandatory.

Various Other Deformities Seen in Myelomeningocele

- Spine—kyphoscoliosis
- Hip—flexion, abduction deformities
- Knee—flexion deformities
- Foot and leg—equinovarus (club foot) and valgus deformities

Syringomyelocele

In the condition of syringomyelocele, there is distention of the central canal of the cord (syringomyelia) associated with protrusion of spinal cord out of the bony defect.

Myelocele

In myelocele, the neural canal itself is open and there is an elongated fissure with a gross spinal defect on the back.

Spina Bifida Occulta

Spina bifida occulta is a less severe defect among the two and the defect may go unnoticed. However, there is a defect in the bony arches but the spinal cord and meninges are normal and do not protrude out. The main cause of spina bifida occulta is failure of fusion as mentioned earlier. Other causes are as follows:

- Diplomyelia—duplication of the spinal cord
- Diastematomyelia—bony septum that divides the spinal cord
- Intraspinal-associated tumours—lipoma, chondroangioma and dermoid
- Myelodysplasia—developmental defect of the spinal cord
- Errors in skeletal segmentation

Clinical Features

- Neurological impairment occurs due to attachment of dura to the skin by a fibrous band known as *membrane reunions.*
- The membrane reunions do not increase in size but as the child grows, they produce a traction on the cord leading to paralysis of the lower limb or bladder.

There is a skin marker in the form of tuft of hair in some patients (Fig. 2.21).

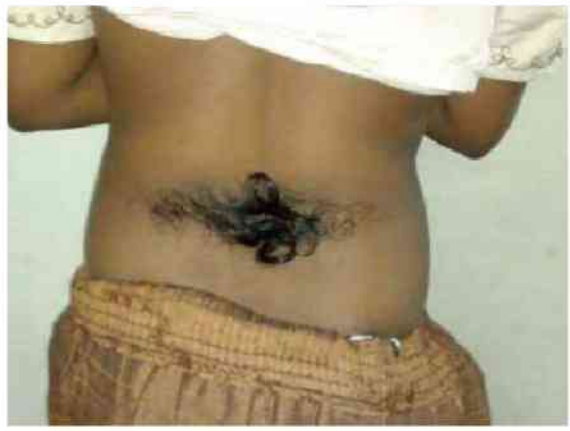


FIGURE 2.21 Clinical photo of tuft of hair in the back—a marker spina bifida occulta.

Investigations

Prenatal detection of spina bifida can be done by:

1. Amniocentesis
   - 16–18 weeks of pregnancy
   - Increased α-fetoprotein
   - Increased acetylcholinesterase
2. Ultrasound examination
3. MRI

Treatment

1. 60–100% reduction in risk of neural tube defects has been attributed to adequate intake of folate by pregnant women.
2. The aim of treatment in meningomyelocele is to achieve independence of the individual as far as possible.
   - Surgical closure within first 48 hours of birth
   - Prompt recognition and treatment of hydrocephalus
   - Urinary tract care by drugs and surgical methods
   - Orthopaedic management includes spinal support to enable surgical correction of deformities of lower limbs

Anterior Spina Bifida

Anterior spina bifida is a rare condition in which the segments of vertebral body fail to fuse.

CONGENITAL SCOLIOSIS

Congenital scoliosis is a lateral curvature of the spine due to congenital vertebral anomalies. (Refer to Chapter 12.)

KEY POINTS

- Teratogenic factors act at different stages of development of the foetus to produce congenital and developmental deformities.
- The structural deformity of foot that is seen in congenital talipes equinovarus include plantar flexion at the ankle, inversion at the subtalar joint and adduction of foot at the level of forefoot.
- Medial talonavicular dislocation is the basic pathology in congenital talipes equinovarus.
- Correction of the deformity in CTEV should be done in the following sequence; first, the forefoot adduction is corrected, followed by inversion and, finally, the equinus is corrected.
- Age of the child and the severity of deformity decide the type of treatment modality (conservative or surgical) to be followed.
- Developmental dysplasia of the hip can be either due to laxity of ligaments and capsule (more common) or due to dysplastic acetabulum.
- Ortolani's test and Barlow's test are used for diagnosis of developmental dysplasia of the hip.
- Treatment modalities used for dislocation of hip include manual reduction, spica application, tenotomy, femoral osteotomy and innominate osteotomy.
- Sprengel's shoulder is a developmental anomaly characterised by congenital elevation of scapula.
- Sprengel's shoulder may be associated with other anomalies such as fusion of two or three cervical vertebrae. This combined anomaly is called Klippel—Feil syndrome.
- Congenital radioulnar synostosis is due to fusion of the superior radioulnar joint and is associated with restricted supination.
- Madelung's deformity is a growth disorder of the ulnar half of the distal radial epiphysis.
- Congenital skeletal limb deficiencies are characterised by deficiency of a part or whole of limb. These deficiencies are classified clinically into terminal and intercalary deficiencies.
- Congenital torticollis is associated with breech presentation.
- Spina bifida is congenital anomaly whereby the spinal cord lies exposed to the external environment. This anomaly can be closed or open.

MULTIPLE CHOICE QUESTIONS

1. Club foot (CTEV) in a newborn is treated by
   a. Surgery
   b. Manipulation by mother
   c. Dennis brown splint
   d. Strapping
2. Treatment of chronic cases of club foot
   a. Triple arthrodesis
   b. Dorso medial release
   c. Amputation
   d. None
3. In a newborn child, abduction and internal rotation produces a click sound. It is known as
   a. Ortolani's sign
   b. Telescoping sign
   c. McMurray's sign
   d. Lachman's sign
4. All of the following are true about developmental dysplasia of the hip (DDH) except
   a. It is more common in females
   b. Oligohydroamnios is associated with a higher risk of DDH
   c. The hourglass appearance of capsule may prevent a successful closed reduction
   d. Twin pregnancy is a known risk factor
5. Sprengel's deformity of scapula is
   a. Undescended/elevated scapula
   b. Undescended neck of scapula
   c. Exostosis scapula
   d. None of the above

CHAPTER 3

Developmental Disorders of Bones

The anatomical and structural features of an adult bone are easily understood, when it is considered as the skeletal framework and supporting structure, for the human body. But the bone in the living body is much more than a mechanical frame for the body. It may be said that the bone of a skeleton in the anatomy museum is only the skeleton of the bone in the living body. The living bone with its periosteum, endosteum, bone marrow, vascular and neural supply is a *functioning tissue* and an *organ* with multiple functions through its hematopoietic and reticuloendothelial tissues and mineralised bony tissue.

As the largest storehouse of calcium, the skeleton plays an important role in the calcium metabolism and reflects the changes in the absorption, storage and excretion of this mineral in the body. As a mineralised structure is easily visualised in the radiographic pictures, many disorders have been recognised mainly from their radiological appearances.

Many disorders of development and growth of the bone are characterised by the formation of swellings in the bones. Such swellings cast radioopaque shadows in radiographs. Many of these are clinically palpable and were described as tumours with names such as chondromas and osteomas, although they are not true neoplasms. The bone as seen in radiographs can show only two types of reactions, *osteosclerotic* and *osteolytic*. Thus, many different pathological processes show similar radiological features. Hence, the diagnosis of bone lesions needs the integrated understanding of the pathological, radiological and clinical manifestations of the diseases. Modern advances in imaging sciences such as CT scan and radionuclide imaging have helped us to understand the structure and function of bone tissue with greater accuracy.

DYSPLASIA AND CLASSIFICATION OF DEVELOPMENTAL DISORDERS OF BONE

Disturbance of growth and development is called *dysplasia*. *Aplasia* denotes absence of development, and *hypoplasia* and *hyperplasia* mean diminished or excessive growth.

Dysplasia may affect the physis (epiphyseal plate), metaphysis or diaphysis of the bone and cause various growth disorders of the bone and cartilage.

A large number of such dysplasias have been described as various disease entities.

1. Cartilaginous dysplasias
   It is due to disturbance in chondroid formation.
   a. Heterotropic proliferation of chondroblasts of the growth plate
      - Multiple exostoses (diaphyseal aclasia)
      - Enchondromatosis (Ollier's disease)
   b. Abnormal maturation of chondroblasts of the growth plate
      - Achondroplasia
      - Metaphyseal dysostosis

c. Abnormal epiphyseal centre
   - Multiple epiphyseal dysplasia (Fairbank)
   - Spondyloepiphyseal dysplasia

d. Abnormal mucopolysaccharide metabolism (mucopolysaccharidosis, chondro-osteodystropy)
   - Morquio–Brailsford disease
   - Hurler's disease (Gargoylism)

2. Bony dysplasias
   Dysplasias that are due to disturbance in osteoid production are as follows:
   a. Deficient osteoid production
      - Osteogenesis imperfecta
   b. Excessive osteoid production
      - Osteopetrosis (Albers Schonberg disease)
      - Osteopoikilosis
   c. Abnormal osteoid production
      - Fibrous dysplasia
      - Neurofibromatosis
      - Pseudarthrosis
3. Miscellaneous dysplasias
   a. Cleidocranial dysostosis
   b. Nail patella syndrome
   c. Marfan's syndrome
   d. Chromosomal abnormalities causing skeletal dysplasia

CARTILAGINOUS DYSPLASIAS

Multiple Exostoses (Diaphyseal Aclasia)

Multiple exostoses is a growth disorder due to a disturbance of endochondral ossification. The characteristics of this condition are as follows:

- Hereditary transmission (autosomal dominant)
- Presence of multiple exostoses
- Dwarfism
- Failure of remodelling of bone ends

Clinical Features

The patient is short in stature and presents with bony swellings near the growing ends of the long bones, such as the lower end of femur, upper end of tibia (Fig. 3.1a) and fibula, and lower end of radius. The other sites are the ribs, scapula and ilium. The swelling is first noticed during childhood but is asymptomatic. The exostosis increases in size during the growth period.

In the forearm, there is a bowing of the radius and shortening of the ulna resulting in the ulnar deviation of the wrist. At the knee, the exostosis at the lower end of the femur and upper end of the tibia grow away from the joint. There are two types of exostosis:

1. Pedunculated (with a narrow pedicle)
2. Sessile (with a broad pedicle)

The apex is covered with a cap of cartilage. At the time of fusion of the epiphyseal plate, the cartilage cap also gets ossified and there is no further growth.

Pathology

Structurally, the exostosis has a core of cancellous bone, surrounded by cortical bone, and the top is covered by a layer of hyaline cartilage.

Complications

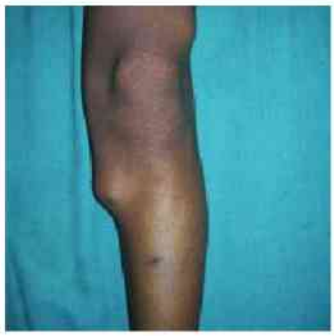
- Mechanical restriction of movements of the adjacent joint
- Adventitious bursa with bursitis
- Compression of the adjacent nerves or vessels and slipping of the adjacent tendons
- Fracture at the pedicle
- Malignant transformation (about 2%) into chondrosarcoma

Radiological Features

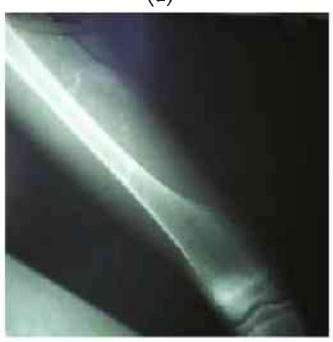
Radiography will show pedunculated or sessile exostosis pointing away from the joint (Fig. 3.1b).The metaphyseal area remains broad and barrel shaped without the normal remodelling. In the wrist, there is a shortening of the ulna and bowing of the radius with subluxation of the inferior radioulnar joint.

Treatment

Surgery is needed only in symptomatic cases. Excision of an exostosis is done only when there are complications. It is not necessary to excise every exostosis. If a particular one interferes with the joint function or is embarrassingly large, it should be excised. If there is rapid growth of the exostosis, malignant changes should be suspected and it should be excised.



(a)



(b)

FIGURE 3.1 (a) Clinical photograph showing exostosis in proximal tibia. (b) X-ray of femur with knee anteroposterior view showing exostosis at the distal end of femur.

Enchondromatosis (Ollier's Disease)

Enchondromatosis (dyschondroplasia) is a condition due to the abnormal proliferation of the cartilage cells of the growth plate and their migration within the metaphysis of several bones. They cause thinning of the overlying cortex and distortion of growth. It is not a hereditary disease.

Clinical Features

Clinically, the condition presents as multiple swellings of the bones of the hands and feet and in the long bones near the knee. It has a unilateral distribution. This condition produces shortening of the limb and deformities such as genu varum and genu valgum. Rarely, these swellings could become malignant chondrosarcoma. It may be discovered when a patient presents with a pathological fracture in a finger. Low-grade malignancy rarely occurs.

Maffucci syndrome is a rare type of disease in which there is associated multiple cavernous hemangiomata and phleboliths in soft tissues.

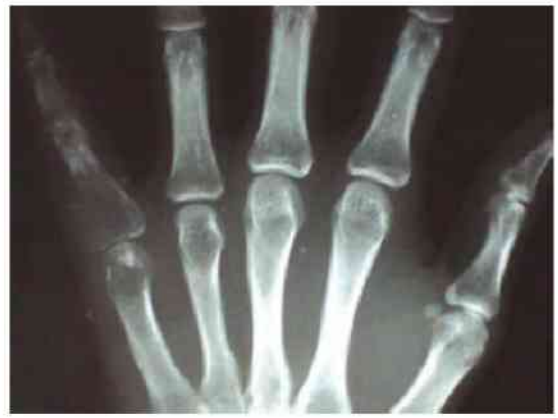


FIGURE 3.2 X-ray of hand anteroposterior view showing enchondroma of the fifth proximal phalanx.

Radiological Features

They are seen as multiple translucent areas in the short long bones of the hands and feet (Fig. 3.2). In long bones, there are radiolucent streaks from physis to metaphysis.

Treatment

If the patient presents with pain and pathological fractures, the lesion may be curetted and bone grafted. Deformities will need corrective osteotomy.

Achondroplasia (Achondroplastic Dwarfism)

Aetiopathology

Achondroplasia is a hereditary (autosomal dominant) and congenital condition. It is caused by an abnormal maturation of growth plate chondroblasts. The membranous ossification is normal.

As the defect occurs in the foetal stage of development, the child is a dwarf even at birth. This is the most common type of dwarfism, and most of the circus dwarfs belong to this category.

Clinical Features

The achondroplastic dwarf is a short-limbed dwarf, wherein the shortness is due to reduction in the length of the legs (Fig. 3.3). The upper limbs are also short and the finger tips do not reach below the upper one-third of the thigh. In a normal person, the finger tips reach the distal third of the thigh on

FIGURE 3.3 Clinical photograph of an achondroplastic dwarf. Upper and lower limbs are short.

standing. The fingers are short and stumpy and the hand is called ***trident hand***. The head is large and the bridge of the nose is flattened.

The length of the trunk is not much affected. As they grow into adult life, they develop cervical lordosis and increased lumbar lordosis as they have to look upwards all the time to see normal people. Still later, they develop spinal canal stenosis and other spinal deformities. Intelligence and sexual development are normal.

Radiological Features

There is a shortening of the long bones with flaring of the ends. The radiograph of the pelvis shows a characteristic quadrilateral shape of the ilium with flattening of the acetabular roof and coxa valga.

Treatment

Orthopaedic and neurological complications require treatment as they arise. Increased height can be obtained by leg-lengthening operations.

Chondro-Osteodystrophy (Morquio–Brailsford Disease, MPS IV)

Chondro-osteodystrophy is one of the hereditary dysplasias of the growing bone, caused by abnormality in the mucopolysaccharide metabolism which causes excessive storage of mucopolysaccharide in the cells. There is defective maturation of the chondroblasts of the epiphyseal plate. It causes dwarfism due to defective growth in the spine.

Clinical Features

The child is normal at birth and is usually brought for a kyphotic deformity of the spine and a waddling gait at the age of 2 or 3 years. Dwarfing is a marked feature. The head is enlarged and is sunk between high shoulders. The sternum is convex forwards with increased manubriosternal angle and the chin seems to rest on it. There is also abnormal laxity of ligaments of the big joints. The hands are short and the fingers appear thickened. The lower limbs show limitation of movements in hip as well as genu valgum and flat feet.

Atlantoaxial subluxation due to odontoid hypoplasia is common.

The trunk is short and the spine shows a thoracolumbar kyphosis. The kyphosis is maximum at the dorsolumbar region, and it is smoothly rounded and mobile without any rigidity or spasm.

Laboratory Findings

Mucopolysaccharide (keratan sulphate) excretion in the urine is increased.

Radiological Features

The typical radiological features are seen in the lateral view of spine. The vertebral bodies are flattened (platyspondyly) and have a tongue-shaped projection from the middle of the anterior surface. The upper femoral epiphysis shows flattening and patchy fragmentation (Fig. 3.4). The hand shows shortening of the metacarpals with conical bases.

Treatment

No treatment is indicated in childhood. Severe genu valgum can be corrected by osteotomy. In the later age, these patients will need treatment for osteoarthrosis in the joints.

Hurler's Syndrome (Gargoylism)

Gargoylism resembles Morquio's disease very closely and is also due to a defect in the mucopolysaccharide metabolism. In addition to the skeletal changes, there is evidence of mental retardation, corneal opacities and hepatosplenomegaly and dwarfism. The milestones of development such as sitting, standing and walking are delayed. The head is large,

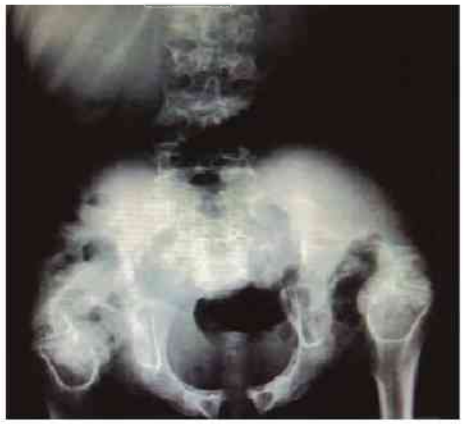


FIGURE 3.4 X-ray of pelvis showing hip joint changes in Morquio's disease. Femoral epiphysis is flattened.

the eyes are set wide apart with a broadened bridge of the nose and the ears are set low on the head giving a frog-like appearance. There is no specific treatment. Complications will require treatment.

BONY DYSPLASIAS

Osteogenesis Imperfecta (Fragilitas Ossium)

Osteogenesis imperfecta is a familial disease characterised by marked fragility of the long bones, osteopenia and ligamentous laxity.

The condition is caused by defective osteoid formation due to congenital inability to produce adequate intercellular substances such as osteoid, collagen and dentine. There is failure of maturation of collagen in all connective tissues. Growth in the thickness of the bones is more affected leaving a thin cortex which permits bending and breaking. Cartilage develops normally.

Clinical Features

Clinically, the disease is of two types:

1. Osteogenesis imperfecta congenita
2. Osteogenesis imperfecta tarda (mild and severe types)

In the congenital (prenatal) type, the child may be born alive or dead with numerous fractures.

In the tarda type, the occurrence of repeated fractures in childhood is the most typical feature. Due to the bending of the femora or malunited fractures, the child is dwarfed. The fractures heal normally. Occasionally, they heal with exuberant callus which may be mistaken for sarcoma. The tendency to fracture gets less as the child grows into adult life.

The extremities are long and thin; the muscular development is poor. The fingers are hypermobile at the joints due to excessive ligamentous laxity. The sclera is blue in these children due to its thinness and translucency. The teeth are of poor quality and translucent due to imperfect dentine. This is called *dentinogenesis imperfecta*. Many patients develop otosclerosis and deafness at the adolescent age. There is no biochemical abnormality.

Radiological Features

Radiographs of long bones show marked thinning of the cortex, osteoporosis and broadening of the medullary canals. The long bones of the lower and upper limbs show curvatures due to previous fractures (Fig. 3.5).

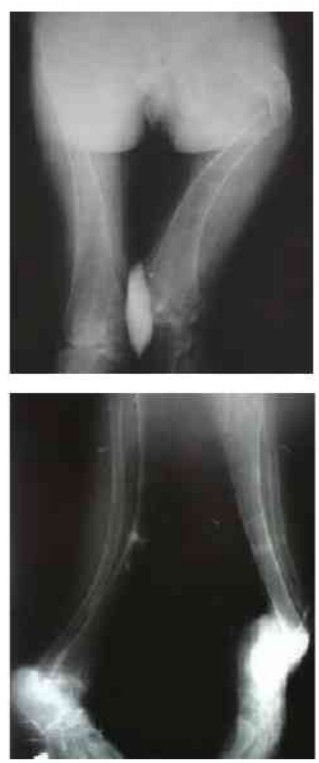


FIGURE 3.5 X-ray of bilateral femur and leg showing features of osteogenesis imperfecta. Note osteopenia, broadened medullary canals and deformed long bones.

Treatment

The mild cases need treatment for the fractures by cautioning the parents to avoid trauma. Established deformities of long bones such as the femur or tibia are corrected by multiple osteotomies, realignment and intramedullary nail fixation. Severe degrees of osteogenesis imperfecta are most disabling and crippling.

Osteopetrosis (Marble Bone Disease, Albers Schonberg Disease)

Osteopetrosis is a hereditary condition characterised by increased generalised density of all the bones in the body. It may be due to a failure of resorption of the calcified osteochondroid tissue during growth. The bone is hard but chalky and brittle. There is also failure of remodelling. There are two types in this condition: (1) the more severe congenita type (autosomal recessive) and (2) the less severe tarda type (autosomal dominant).

Clinical Features

Clinically, the case is often detected during childhood by the features of aplastic anaemia due to the replacement of the medullary cavity by the bone. The spleen and liver are enlarged. The child may present with a pathological fracture due to the brittleness of the bones. Osteosclerosis affects the foramen in the skull resulting in the compression and paralysis of the optic and other cranial nerves. The patients develop blindness and deafness. The patients also develop osteomyelitis of the mandible easily due to severe caries in the teeth.

Radiological Features

The whole bone shows uniformly increased density. The skeletal survey demonstrates such changes in all the bones (Fig. 3.6). The vertebral bodies show increased sclerosis of the upper and lower ends giving a sandwich appearance (rugger jersey spine).

Treatment

Minor cases survive to adult age and will require treatment for fracture, infection and anaemia. The prognosis is poor in severe cases.

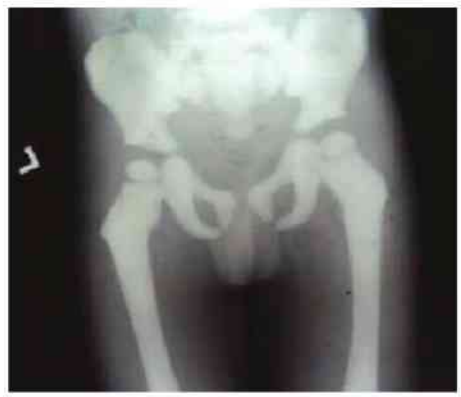


FIGURE 3.6 X-ray of pelvis with both hips showing osteopetrosis. Note the increased bone density on plain X-ray.

FIBROUS DYSPLASIA

Fibrous dysplasia is a disorder of normal bone mass maintenance by resorption and new bone formation.

The exact cause of fibrous dysplasia is unknown. Normal bone is not static in structure. There is a continuous dynamic process of bone absorption and replacement by the activity of the osteoblasts and osteoclasts. There is a balance between the two processes in normal adult bones. In fibrous dysplasia, there is a disorder in the bone maintenance; the resorbed bone is replaced by a fibro-osseous tissue instead of by normal bone. As the mass of fibrous tissue proliferates, it erodes the cortical bone from within, while the periosteum lays down a thin shell of bone outside and hence the bone appears expanded.

Clinical Features

There are three types of fibrous dysplasia. These may involve a single bone (monostotic fibrous dysplasia), a single limb (monomelic) or many bones (polyostotic fibrous dysplasia).

Clinically, this condition is noticed in childhood or adolescence. The only symptom is pain in the metaphysis or diaphysis of the long bones. The affected bone may undergo gradual deformity or may have a pathological fracture. The bones affected are the femur, tibia, humerus and pelvis. Some cases present with asymmetry of the face due to involvement of the orbit and mandible. In some cases, there

may be patches of cutaneous pigmentation. The localised lesion in a single bone may simulate a bone tumour.

A polyostotic type occurring in girls and associated with endocrine dysfunction, precocious puberty and cutaneous pigmentation is known as McCune–Albright's disease.

Radiological Features

The affected bone shows areas of translucency which has a ground glass appearance. The cortex may be expanded and sometimes has a multilocular appearance (Fig. 3.7).

It sometimes resembles an osteoclastoma and hence is a giant cell variant. In the upper end of femur, it produces a coxa valga deformity and a bowed shaft called *Shepherd's crook* deformity.

Laboratory Findings

The blood serum calcium level is normal, and this differentiates the condition from hyperparathyroidism where it is raised. The serum alkaline phosphatase may be increased if the lesion is extensive.

Biopsy

Biopsy is necessary to confirm the diagnosis. The curetted material is gritty. Microscopically, the tissue consists of spindle cells of loose fibrous tissue interspersed with bony trabeculae of immature bones. A few giant cells are also seen. A small percentage of the polyostotic variety may undergo malignant transformation.

Treatment

When pain is a prominent symptom, treatment is done by operative curettage and packing the cavity with cancellous bone grafts. Deformities will need corrective osteotomy.

NEUROFIBROMATOSIS (Von Recklinghausen's Disease)

Neurofibromatosis is a hereditary familial condition involving the nerves, skin, soft tissues and bones characterised by multiple neurofibromatous swellings over the body surface, pigmented spots and skeletal lesions (Fig. 3.8).

Clinical Features

The patient presents with pigmented cafe-au lait spots or patches in the skin. More than four such patches of 1.5 cm are diagnostic.

Neurofibromata of various shapes and sizes are present all over the body involving the peripheral nerve endings and the nerve trunks. But they rarely cause motor or sensory loss. They may

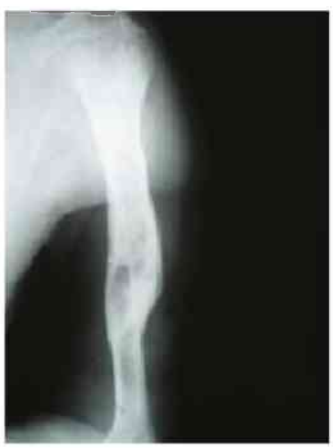


FIGURE 3.7 X-ray of humerus anteroposterior view showing fibrous dysplasia. This is located in diaphysis, with groudglass appearance and cortical expansion.

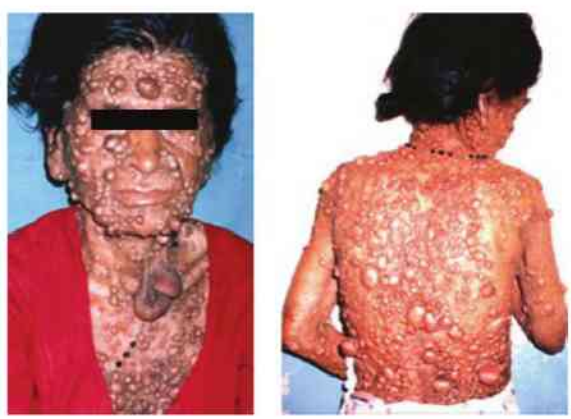


FIGURE 3.8 Clinical photograph showing neurofibromatosis. Multiple neurofibromas are seen throughout the body.

also involve the spinal nerves near the spinal cord causing pressure symptoms. They may involve the cranial nerves such as the eighth nerve (e.g. acoustic neuroma) causing intracranial pressure symptoms. The plexiform type of the cutaneous neurofibromata causes gross thickening of the skin which hangs as thick folds. Sometimes, local gigantism of a finger or part of the limb occurs in this disease due to diffuse soft tissue and vascular hypertrophy.

Skeletal manifestations are common in about 30–50% in this condition. Severe scoliosis and kyphoscoliosis of the spine in children are due to neurofibromatous intraosseous cystic lesions resulting in pathological fractures.

Radiological Features

The intervertebral foramen is enlarged. In scoliosis, the curve is sharp and short. In the long bones such as tibia and ulna, intraosseous cystic lesions, bowing, fracture and osteolysis of a segment of bone may be seen.

Treatment

Treatment must be confined to excision of any lesion which shows abnormal growth or causes pressure symptoms. Spinal deformities will need surgery.

CONGENITAL PSEUDARTHROSIS OF THE TIBIA

Congenital pseudarthrosis of tibia is a familial condition characterised by an area of deossification at the junction of the middle and distal third of the tibia leading to bending, fracture and nonunion. There is an inherent inability of the bone to form callus at the site of the fracture.

Classification

Boyd has classified this condition into six types:

- *Type I:* Pseudarthrosis with anterior bowing and a defect in the tibia at birth
- *Type II:* Pseudarthrosis with anterior bowing and an hourglass constriction
- *Type III:* Pseudarthrosis which develops in a congenital cyst
- *Type IV:* Pseudarthrosis which originates in a sclerotic segment of bone
- *Type V:* Pseudarthrosis tibia with dysplastic fibula
- *Type VI:* Pseudarthrosis occurs as an intraosseous neurofibroma

Clinical Features

Clinically, the child presents with angulation of the tibia or abnormal mobility in the bone at the junction of the middle and lower third.

Radiological Features

The site may show a cystic area with continuity of the cortex. In some cases, there is established non-union with tapering of the proximal and distal fragments of the tibia.

A similar condition may be seen in the fibula at the same level. In some cases, there is only anterior bowing with sclerosis of the cortex (Fig. 3.9).

Treatment

This is one of the most difficult conditions to treat. The principles of treatment are excision of the fibrous mass, massive bone grafting and

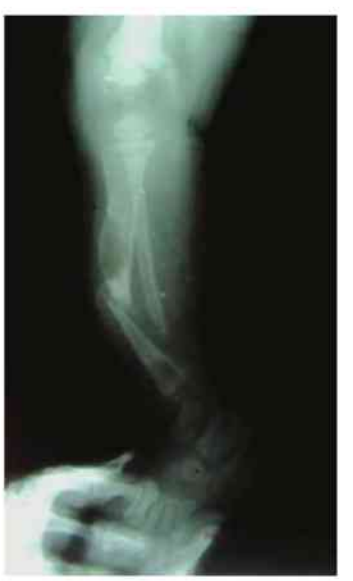


FIGURE 3.9 X-ray of leg showing congenital pseudarthrosis tibia. Note anterior bowing and sclerosed bone ends.

internal fixation. More recently, Ilizarov ring fixation and bone transport are being used for union.

MISCELLANEOUS DYSPLASIAS

Cleidocranial Dysostosis

Cleidocranial dysostosis is a developmental and familial disorder of membranous ossification with involvement of the clavicle and the cranial bones. In the typical case, the skull is larger and the face is small. The mandible is large and prognathous, but the maxilla is poorly developed. There is bossing of the parietal and frontal bones. The closure of fontanelle is delayed.

The clavicle may be totally or partially absent. This may be bilateral so that both the shoulders could be brought forward towards the midline to touch each other. The pelvic bones also show poor development with non-fusion of the symphysis pubis and poor development of the sacrum. Complications such as coxa valga will need treatment.

Nail Patella Syndrome (Onycho-Osteodysplasia)

Nail patella syndrome is characterised by a triad of congenital anomalies of the nails, elbows and knees in addition to iliac horns from the outer side of the iliac bones. The thumb nails are short and dystrophic. At the elbows, there is cubitus valgus deformity due to hypoplasia of the lateral condyle. At the knees, there is hypoplasia or absence of patella.

Chromosomal Abnormalities Causing Skeletal Dysplasia

Certain abnormalities of chromosomal configuration result in skeletal abnormalities in addition to other defects such as mental retardation. The common ones of orthopaedic interest are as follows:

- Down's syndrome (mongolism)
- Turner's syndrome

Alteration in the number of chromosomes results in certain genetic disorders. An increase in chromosomes by 1, so that it contains three chromosomes in the 21st pair, is called *Trisomy* 21. This pattern is found in the clinical syndrome called Down's syndrome or mongolism. In these individuals, the total number of chromosomes is 47 instead of the normal 46. A decrease in the sex chromosomes by 1, namely the X chromosomes, results in the condition called Turner's syndrome.

Down's Syndrome (Mongolism/Trisomy 21)

Mongolism is a trisomy syndrome, characterised by skeletal abnormalities in the pelvis and the skull. The patient is mentally retarded and is a dwarf. The head is small and oval and the forehead is sloping. The bridge of the nose is absent or poor. The eyes are slanting upwards with prominent medial epicanthal folds. The hand is broad and ape like with a single palmar crease and the little finger is small and curved. There is generalised ligamentous laxity. The management of these children is mainly educational and occupational training.

Turner's Syndrome

Clinically, there is sexual infantilism due to gonadal aplasia, short stature and cubitus valgus deformity. The hand shows a short fourth metacarpal bone. There is often a hypoplasia of the posterior arch of C1 vertebra. Treatment is mainly for gynaecological problems.

Dwarfism

Dwarfism is a condition of abnormally short stature. An adult of 90 cm height could be considered a dwarf according to Indian standards. The dwarfism is of two types:

1. *Proportionate dwarfism:* In proportionate dwarfism, there is a generalised reduction of the height of the trunk as well as the limbs. This could be caused by chromosomal, endocrine or metabolic disorders which cause stunted growth of the whole person, for example cretins, pituitary dwarfism and severe rickets.
2. *Disproportionate dwarfism:* In disproportionate dwarfism, the dwarfism is due to a shortened

trunk alone, shortened limbs alone or a combination of both. This dwarfism could be grouped into the following:

a. Short-limb dwarf
   - Achondroplasia
   - Diastrophic dwarfism

b. Short trunk dwarf
   - Chondro-osteodystrophy (Morquio's disease)
   - Multiple congenital vertebral anomalies with fusions, hemivertebrae, etc.
   - Congenital spondylocostal dysplasia

c. Combination-type dwarf
   - Spondyloepiphyseal dysplasia
   - Osteogenesis imperfecta

The orthopaedic problems of dwarfism are commonly related to the dysplastic hips and deformities at the knees. In the spine, odontoid dysplasia, scoliosis and kyphoscoliosis cause neurological symptoms.

Treatment

Treatment is on the same principles as followed in the management of the deformities due to other causes. In recent years, limb-lengthening operations by the Ilizarov technique have been done successfully to increase the height of the dwarfs.

A person with marked diminution of height not only has physical and functional problems but also has to contend with psychological and social problems in life.

KEY POINTS

- Disturbance of growth and development (dysplasia) can be cartilaginous or bony. A group of dysplasias are kept in miscellaneous category.
- Multiple exostoses is an autosomal-dominant growth disorder of endochondral ossification.
- Exostosis can be pedunculated and sessile.
- Enchondromatosis (dyschondroplasia) is characterised by abnormal proliferation of the cartilage cells of the growth plate and their migration within the metaphysis. It is not a hereditary disease.
- Achondroplasia is an autosomal-dominant disorder and is caused by an abnormal maturation of growth plate chondroblasts. This is the most common cause of dwarfism.
- Achondroplastic dwarf is a short-limbed dwarf.
- Mucopolysaccharidoses cause defective maturation of chondroblasts of the epiphyseal plate. Dwarfism is a striking feature.
- Osteogenesis imperfecta is caused by defective osteoid formation due to congenital inability to produce adequate intercellular substance such as osteoid, collagen and dentine. Clinically, two types are of congenita and tarda.
- Osteopetrosis is due to a failure of resorption of the calcified osteochondroid tissue during growth and also failure of remodeling.
- Fibrous dysplasia is a disorder in the bone maintenance; the resorbed bone is replaced by a fibro-osseous tissue instead of by normal bone. Three types are monostotic, monomelic and polyostotic.
- Congenital pseudarthrosis of tibia is characterised by an area of deossification at the junction of the middle and distal third of the tibia. Boyd classification has six types.

MULTIPLE CHOICE QUESTIONS

1. The treatment of enchondroma is
   a. Amputation
   b. Irradiation
   c. Local excision
   d. Curettage and bone grafting
2. The following is false of achondroplasia
   a. Autosomal dominant
   b. Mental retardation
   c. Due to gene mutation
   d. Shortening of limbs present
3. Not associated with osteogenesis imperfecta is
   a. Blue sclera
   b. Cataract
   c. Deafness
   d. Fractures
4. Musculoskeletal abnormalities present in neurofibromatosis
   a. Hypertrophy of limbs
   b. Scoliosis
   c. Pseudarthrosis
   d. All
5. Pseudarthrosis of tibia is best treated by
   a. Internal fixation
   b. Internal fixation and bone grafting
   c. Above-knee PoP cast
   d. Below-knee PoP cast

CHAPTER 4

Infections of Bones and Joints

OSTEOMYELITIS

Osteomyelitis is defined as inflammation of the bone and bone marrow caused by infective organisms. Osteitis indicates inflammation of the bone. This can be caused by the following types of organisms:

- Non-specific pyogenic organisms such as *Staphylococcus* and *Streptococcus*
- Specific organisms causing tuberculous osteomyelitis, syphilitic osteomyelitis, typhoid osteomyelitis
- Mycetomal (fungal) infections causing actinomycosis or maduromycosis
- Parasitic infection—echinococcus

PYOGENIC OSTEOMYELITIS

Infective organisms causing infections reach the bone from a septic foci, usually situated somewhere away or in close vicinity of bones but mostly not detectable. The major portal of entry of microbes into the bones is haematogenous and they traverse through the medullary arteries and reach metaphysis of long bones. Infection can reach the bone by the following routes:

- Through the blood stream from a focus of infection elsewhere (haematogenous)
- Direct invasion from the atmospheric air as in open fractures
- Spread from a neighbouring focus, for example mastoiditis from middle ear infection, osteomyelitis of mandible from dental root abscess

Classification

Clinically, pyogenic osteomyelitis can present as follows:

1. Acute osteomyelitis
   - Primary subacute osteomyelitis
2. Chronic osteomyelitis
3. Acute flare-up of chronic osteomyelitis

Acute Pyogenic Osteomyelitis

Incidence and Aetiology

Acute haematogenous osteomyelitis is still common in India. This is a disease of childhood and adolescence, but even adults are not spared. It occurs in undernourished children whose general resistance is poor. The focus of infection in the skin such as impetigo, septic tooth or tonsil predisposes to this condition. Such a focus produces a bacteraemia and the organisms are carried to all parts of the skeleton. A mild trauma to a bone tends to localise the infection and initiates the infective process in the bone. The most common sites are the distal end of the femur and the proximal end of the tibia. The most common causative organism is *Staphylococcus aureus* (*S. aureus*).

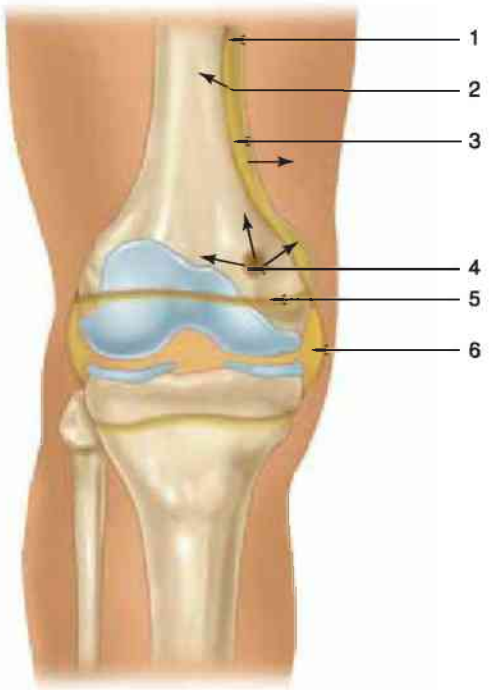


FIGURE 4.1 Diagrammatic representation of the lower end of femur showing the spread of the infection. (1) Cortex of the shaft, (2) periosteum, (3) subperiosteal abscess, (4) initial focus in the metaphysis, (5) epiphyseal plate and (6) capsule of the joint.

Pathology

The initial focus is in the metaphyseal region of the bone. This is due to slowing of the circulation in the capillary loops in the metaphysis. This is why osteomyelitis is more common in the metaphyseal region. The infection starts in the medullary tissues resulting in a focus of suppuration and spreads outwards to the surface and forms a subperiosteal abscess (Fig. 4.1). Further spread involves the overlying soft tissues and results in a subcutaneous abscess. Eventually, the periosteum is lifted and at some point of time it is perforated. The infection then spreads into the adjacent soft tissue and tracks out of the skin and forms a draining sinus. Infection can also spread subperiosteally towards the middle of the shaft raising the periosteum of the bone. It also spreads along the medullary canal to the middle of the diaphysis.

In the upper end of the tibia and the lower end of the femur, the epiphyseal plate is extra-articular and the firm attachment of the periosteum near the epiphyseal plate prevents the spread of infection into the joint. In certain situations such as the upper end of the femur and the upper end of the humerus, the metaphyseal region is partly intra-articular as the joint capsule is attached distally. In these regions, a focus of osteomyelitis in the intracapsular part of the metaphysis, on extension to the surface of the bone, directly involves the joint and causes septic arthritis of the joint.

Clinical Features

The patient is usually a child brought with fever, acute pain and swelling just above or below the knee joint. The constitutional disturbances are severe due to toxaemia.

On examination, there is diffuse swelling, warmth and acute tenderness over the area of bone involved. The child is unable to move the limb. This is called *pseudo-paralysis*. On palpation, a fluctuant abscess may be palpable. The knee joint may be swollen with a 'sympathetic' effusion with sterile fluid.

Differential Diagnosis

- *Acute poliomyelitis:* The inability to move the limb and fever mimics the paralysis of acute poliomyelitis and has to be differentiated from it.
- *Acute septic arthritis:* This acute stage will also have to be differentiated from septic arthritis of the joint. In this condition, the movements will be extremely painful and tenderness will be on the joint line.
- *Acute rheumatic fever:* The clinical picture can also simulate acute rheumatic fever, but there will be joint line tenderness rather than bone tenderness in rheumatic fever.

Radiological Features

In the early stages, the radiograph will not show any changes in the bone. When the condition has lasted for about 2 weeks or more, the radiograph may show patchy areas of destruction and slight periosteal reaction. Tc 99 bone scan will show increased uptake in early cases even when no X-ray changes are present.

Laboratory Findings

WBC count will show a marked increase in total count with high polymorphs. The ESR will be raised. Blood culture will reveal the causative organism.

Treatment

- *If the child presents before the formation of abscess:* General treatment for septicaemia must be instituted urgently. The affected part must be rested in a splint. A combination of antibiotics with a broad spectrum of bactericidal activity is given. The drugs, cefotaxime and amikacin, are given parenterally. With this, the inflammation will subside in mild cases and the whole condition will resolve. If the infection settles, continue the antibiotics for 6 weeks.
- *If the child presents after the formation of abscess or if the infection is virulent:* If the infection is virulent, it may proceed uncontrolled and form an abscess. As soon as there is evidence of an abscess, ***surgical drainage*** must be instituted under general anaesthesia. The soft tissue abscess is drained by an incision. If there is subperiosteal collection of pus, it is incised and drained. A few drill holes are then made in the cortical bone to drain any pus in the medullary cavity. Pus is sent for smear, culture and sensitivity to antibiotics. The appropriate antibiotics must be continued for at least 4–6 weeks.

  The limb is immobilised in plaster without weight bearing. Once the condition resolves, mobilisation of the knee and gradual weight bearing are started.

Complications

Septic Arthritis

Under the age of 1, acute osteomyelitis can spread from the metaphysis to the epiphysis and the adjacent joints as vascular channels are open across the epiphysis. In the knee of children above this age, the epiphyseal plate acts as a barrier to the spread of infection, since there is no vascular connection between the metaphysis and the epiphysis.

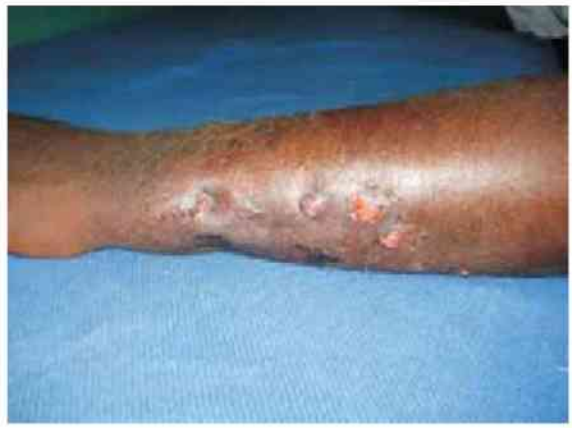


FIGURE 4.2 Clinical photograph of forearm showing multiple sinuses.

Destruction of the Epiphyseal Plate

This may be incomplete or complete. This causes later deformity or limb length discrepancy.

Chronic Osteomyelitis

Chronic osteomyelitis is a persisting pyogenic infection of bone of more than 6 weeks' duration characterised by recurrent attacks of inflammation with sinuses discharging pus (Fig. 4.2).

Aetiology

An acute osteomyelitis becomes chronic due to one of the following reasons:

- Improper drainage of the pus in the acute stage
- Formation of an undrained non-collapsible cavity in the bone
- Presence of sequestrum
- Presence of foreign bodies in the case of osteomyelitis following open injuries
- The causative organisms are usually *S. aureus*, epidermidis and *Streptococcus pyogenes*

Pathology

The thrombosis of vessels in the medullary canal and the stripping of the periosteum result in the

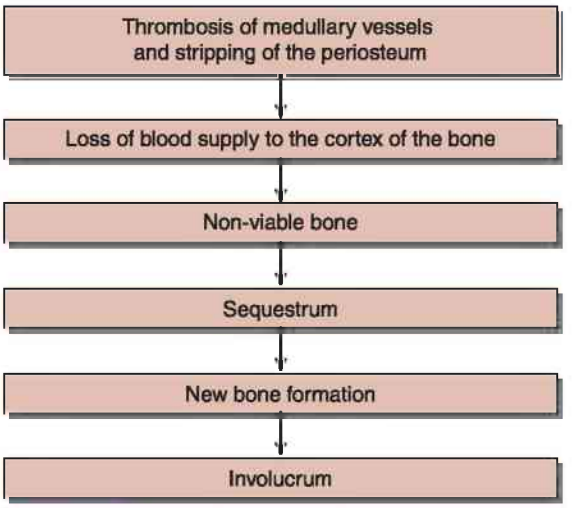


FIGURE 4.3 Flow chart depicting pathology of chronic osteomyelitis.

loss of blood supply to the cortex of the bone. The involved portion of the cortex becomes non-viable and forms a sequestrum. Usually, it is a segment of the cortex of the bone. It is called *cortical sequestrum*. Occasionally in children, when the infection is fulminant, the whole shaft of the bone forms a *diaphyseal sequestrum*. Further, pathological processes take place to provide an outlet for the pus and for the discharge of the sequestrum. The demarcation by granulation tissue and separation of the non-viable bone mark the beginning of chronic osteomyelitis (Fig. 4.3).

Sequestrum is a piece of dead bone separated from healthy bone. The area of dead bone gets demarcated by granulation tissue and gradually separates and forms a loose piece of sequestrum. The *cortical sequestrum* is flat with serrated edges (Fig. 4.4). The surface in contact with the granulation tissue is rough and the other surface is smooth and shiny. The healing response of the bone consists of new bone formation under the raised periosteum. This new bone is called 'involucrum'. This new bone forms the wall of the cavity with holes called *cloacae* for the discharge of pus and small sequestra.

The other types of sequestra encountered are *sandy sequestrum* in tuberculosis, *black granular sequestra* in actinomycosis and *ring sequestrum* around pin tracts or amputation stumps. The diaphyseal sequestrum is seen in children.

FIGURE 4.4 Clinical photograph showing typical cortical sequestrum.

Clinical Features

The patient presents with one or more sinuses discharging pus over the involved bone with a history of an acute attack earlier. Granulation tissue will be protruding at the sinus. The sinus is often adherent to the bone underneath. The whole bone is thickened and irregular. The patient may also sustain a pathological fracture at the site.

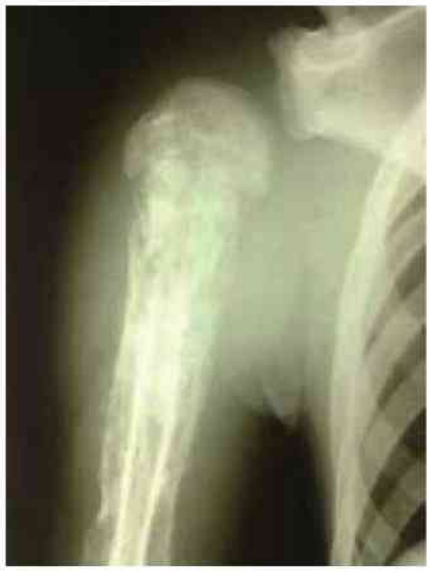
There will be periodic acute flare-up of the infection with severe pain, swelling, fever and discharge of pus and small sequestra from the sinus.

Radiological Features

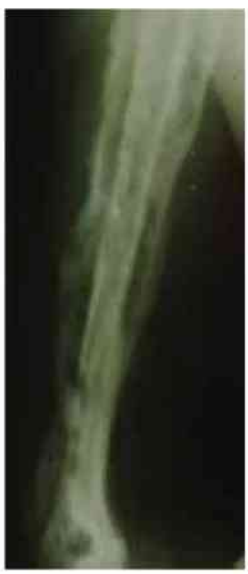
Radiology will reveal sclerosis of the cortical bone with a cavity which appears as an osteolytic area. Inside the cavity, the sequestrum lies as an irregular sclerotic piece of bone (Fig. 4.5). The sequestrum is denser than normal bone because decalcification in normal bone does not occur here. In children, sometimes the whole diaphysis might separate as a sequestrum (Fig. 4.6). A sinogram is useful to identify the entire tract and the multiple communicating sinuses.

General Management

It is essential to identify the causative organism by culture, from a swab of the pus. The sensitivity of the organism to antibiotics is also established and appropriate antibiotics are given. A general assessment of the condition of the patient is made regarding Hb percentage and RBC count. Measures, such as correcting anaemia, are taken to improve the general condition of the patient.



(a)



(b)

FIGURE 4.5 (a, b) X-ray of humerus showing subperiosteal thickening in osteomyelitis.

Surgical Management

When the sequestrum appears well separated in the radiograph, surgery is indicated. When the presence of the cavity and sequestrum are established, surgical removal of the sequestrum

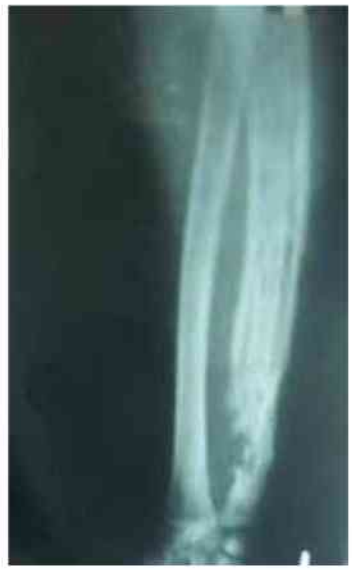


FIGURE 4.6 X-ray of forearm anteroposterior view showing osteomyelitis of ulna with diaphyseal sequestrum.

(*sequestrectomy*) and drainage by deroofing of the cavity (saucerization) are done (Fig. 4.7).

Other measures include the following:

- Curettage of bony lesion
- Sinus tract formation
- Foreign body removal, if present
- Excision of the infected bone followed by Ilizarov's fixation

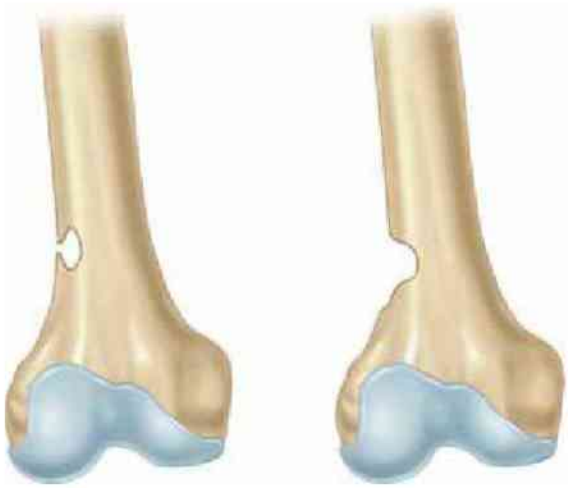


FIGURE 4.7 Diagrammatic representation of the lower end of femur showing how a saucerization is done.

More recently, the cavities are filled with muscle flaps from the neighbouring region to improve the blood supply to the area. Antibiotic impregnated implants are left in the cavity.

Complications

- *Pathological fracture:* This occurs in the bone weakened by chronic osteomyelitis.
- *Deformity:* The focus of osteomyelitis sometimes destroys a part of the epiphyseal plate. This results in the arrest of growth in that part of the epiphyseal plate. The cortical growth of the rest of the epiphyseal plate produces a deformity at that level.
- *Shortening:* Destruction of the whole epiphyseal plate due to osteomyelitis results in the arrest of growth and shortening of the limb.
- *Lengthening:* In some cases, the focus of infection near the epiphyseal plate causes stimulation of growth due to hyperaemia. This results in lengthening of the limb.
- *Bone defects:* Loss of large sequestrum will result in a gap in the shaft of long bone. This produces abnormal mobility and instability.
- *Muscle fibrosis:* Quadriceps fibrosis with stiff knee following osteomyelitis of femur.
- *Adjacent joint stiffness*
- *Sinus tract malignancy*
- *Amyloidosis*

Chronic Pyogenic Osteomyelitis in Open Fracture

Chronic pyogenic osteomyelitis is caused by the direct invasion of bone by pyogenic organisms in open fractures. Improper primary treatment of open fractures results in this type of osteomyelitis. The usual site is the shaft of tibia injured in road accidents (Fig. 4.8). The most common cause of delayed union or non-union of open fracture is osteomyelitis. The fracture will heal only if the sequestra are removed and infection cleared.

Primary Subacute Osteomyelitis

Primary subacute osteomyelitis is the infection of bone of insidious onset and slow progression,

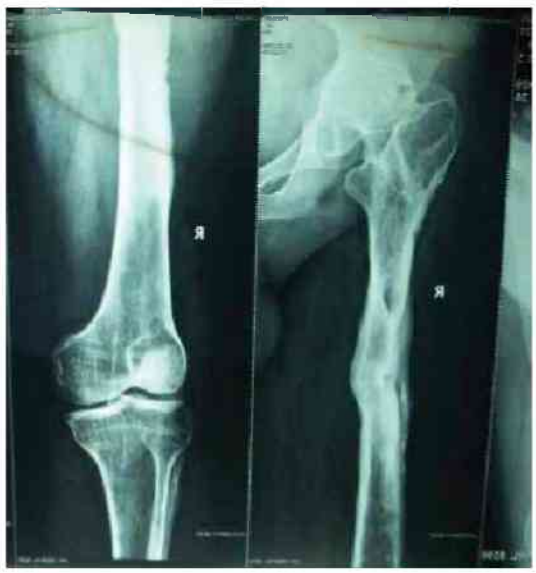


FIGURE 4.8 X-ray of femur anteroposterior view showing chronic osteomyelitis in open fracture.

caused by a less virulent type of organism or by a high resistance in the host.

There are three main types of this lesion: (1) Brodie's abscess, (2) Garre's osteomyelitis and (3) Salmonella osteomyelitis.

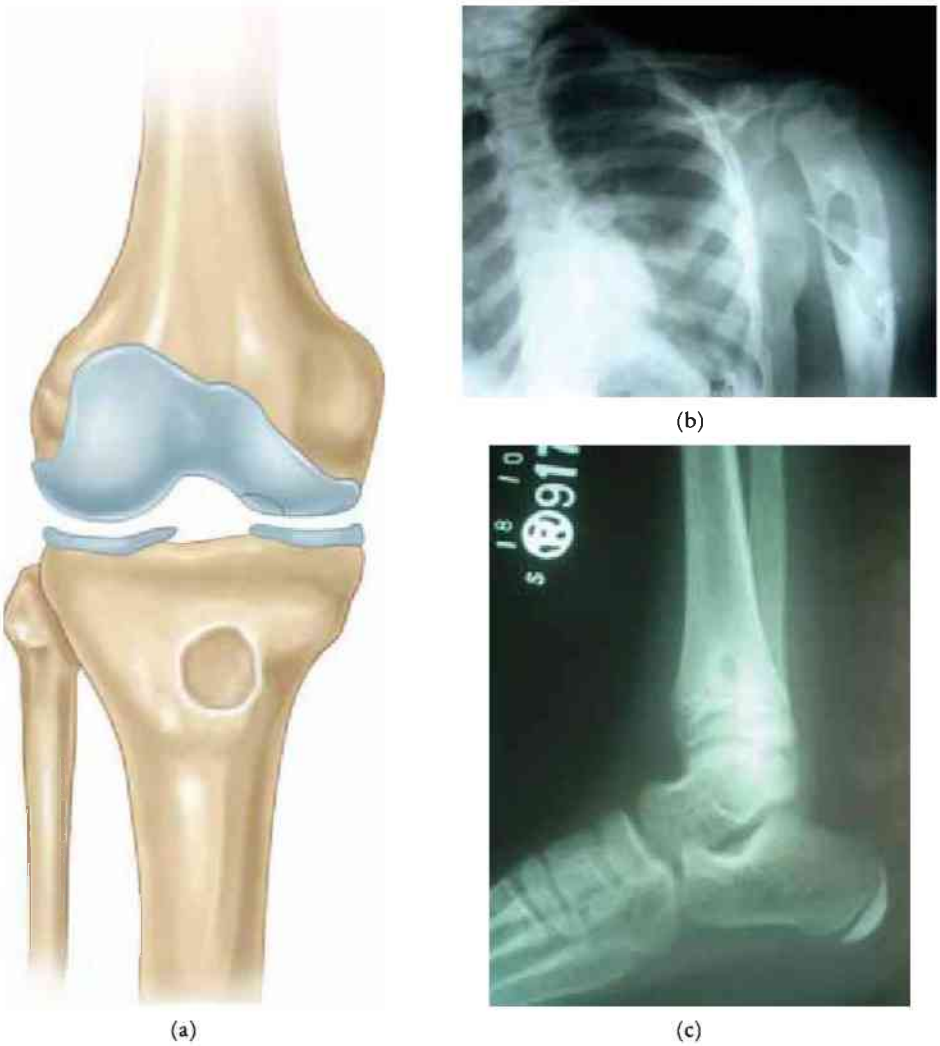
Brodie's Abscess

Brodie's abscess is an abscess in the medulla of the bone as a result of a low-grade infection by staphylococcus.

It commonly occurs in the metaphyseal area of the proximal end of the tibia or distal end of the femur. Clinically, the patient gets intermittent attacks of pain and swelling in the region. Locally, there will be tenderness and swelling on palpation. Radiograph shows an osteolytic area in the bone with a ring of dense sclerosis (Fig. 4.9). Treatment is surgical. The cavity is deroofed and curetted.

Garre's Osteomyelitis

Garre's osteomyelitis is a diffuse sclerosing inflammatory lesion of the shaft of long bone or the jaw. It is a non-suppurating and granulating lesion. Clinically, the patient presents with complaints of diffuse dull aching pain over the involved limb/area. There is tenderness on deep palpation. It causes low-grade symptoms and signs. Radiologically, there is marked thickening and increased density of the cortex of



(a) (b) (c)

FIGURE 4.9 (a) Diagrammatic representation of Brodie's abscess in the upper end of tibia, (b) X-ray of humerus anteroposterior view showing Brodie's abscess in its upper end, (c) X-ray of distal tibia lateral view showing Brodie's abscess. Note the central radiolucent area surrounded by a sclerotic margin.

bone with partial obliteration of the marrow space. Treatment is by antibiotics and surgery.

Salmonella Osteomyelitis (Typhoid Osteomyelitis)

Typhoid osteomyelitis is a subacute type of osteomyelitis usually occurring in the ulna, rib and vertebra. It occurs some months or years after the attack of typhoid or paratyphoid fever. An abscess often forms in the middle of the shaft of the ulna. Salmonella osteomyelitis also occurs as a complication of sickle cell anaemia.

Typhoid infection of the spine closely mimics caries spine. Radiograph shows a central or cortical area of rarefaction with periosteal new bone formation. Blood widal tests are done. Surgery is always required. The focus is curetted out thoroughly.

Osteomyelitis of Spine (Septic Spondylitis)

Pyogenic osteomyelitis of the spine is called septic spondylitis. It starts by blood stream infection of the vertebral body by *S. aureus* secondary to a focus elsewhere. It can also start as an infection

of the intervertebral disc space following lumbar puncture.

Clinically, its onset is very acute with pain, severe spasm of spinal muscles and restriction of movements. There is localised tenderness. Haematology shows leucocytosis. Radiologically, it shows early destruction of the body with *reactive new bone formation*. Breaking of the corners of the vertebral bodies with sclerosis and their fusion is a characteristic appearance.

This has to be differentiated from tuberculosis and brucellar infection of the spine.

Treatment

Treatment is by rest, immobilisation in plaster shell and massive doses of antibiotics. Occasionally, surgical drainage and curettage of the lesion will be needed.

Brucellar Osteomyelitis

Brucellar osteomyelitis is due to infection by brucella abortis or brucella suis. It is an infectious disease of animals that is transmitted to man by drinking unpasteurised infected milk or by direct contact with infected cows or pigs.

Clinical Presentation

The clinical presentation may be acute or chronic. The site of the disease is usually the spine or knee. In the spine, the focus is in the subchondral area of the vertebral body adjacent to the intervertebral disc. Clinical features simulate tuberculous infection. There is also lymph node enlargement, splenomegaly and hepatomegaly.

Radiological Features

The radiological changes in joints are minimal. The spine shows narrowing of the disc space with erosion of the adjacent vertebral bodies and sclerosis. The sclerosis and new bone formation differentiate it from tuberculosis of the spine. High titres on agglutination tests are diagnostic. Tetracycline is the drug of choice.

FUNGAL (MYCOTIC) INFECTIONS OF BONE

Mycotic infection is caused by certain types of fungi which are normally saprophytes in the human body. When the infection gets a foothold in the bone or other soft tissues such as cervicofacial area, jaw or ileocaecal area, it forms a chronic granulomatous lesion.

Mycotic infection of the foot was first described in 1832 by Gill from Madurai, a district in the present Tamil Nadu state in South India. Hence, the condition was called *Madura foot*. As the condition forms a tumour-like mass, it was called *mycetoma*.

The causative fungus is one of the following two types:

1. Actinomyces which causes the lesions in the jaw, ileocaecal area, lungs and spine
2. Maduromycosis which causes the lesion in the foot (Madura foot) or hand

Madura Foot

Mycetoma of the foot starts as a nodular swelling either in the dorsum or on the sole of the foot. Infection enters through a minor injury to the foot like a thorn prick. The initial nodule bursts, discharging thin pus. The indurated swelling spreads and blisters form and burst, resulting in multiple sinuses. Clinically, the patient presents with a large diffuse *indurated* swelling of the foot with multiple discharging sinuses. Pain is not severe unless there is secondary pyogenic infection, which causes extensive destruction of the tarsal bones. The mycotic infection itself does not involve the bone till the last stage.

The pus contains small black granules and on microscopic examination shows the fungus. The marked induration of the involved area and comparatively less pain differentiate this condition from tuberculosis of the tarsal bones and joints which is a much more common condition (Fig. 4.10).

In the early stage, the lesion responds to massive doses of penicillin or dapsone over prolonged periods. Localised lesions respond to

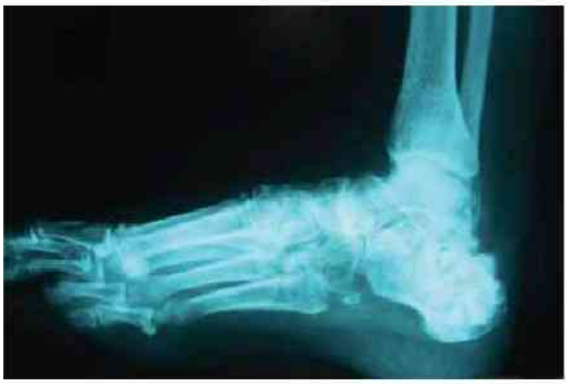


FIGURE 4.10 X-ray of lateral view of foot showing mycotic infection of calcaneum. Note the marked erosion and destruction of bones.

localised debridement or excision. When the foot is completely disorganised and there are multiple persistent sinuses, amputation may be necessary. X-ray of the involved foot shows multiple erosions of the bones and joint.

SYPHILITIC INFECTION IN BONE

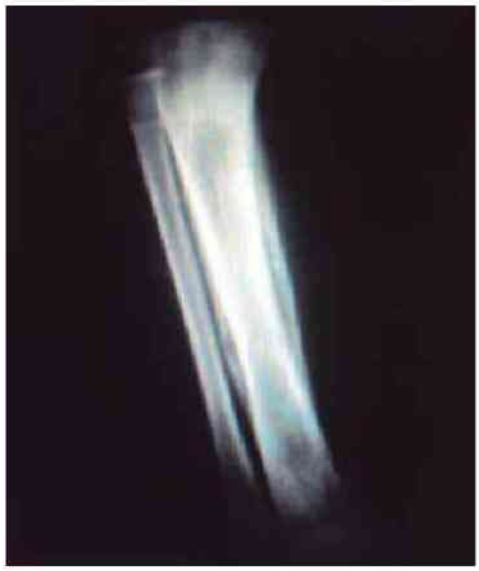
Syphilitic lesions in bone are much less common now due to the early detection and efficient treatment of syphilis. Osseous lesions occur in congenital as well as in acquired syphilis.

Congenital Syphilis

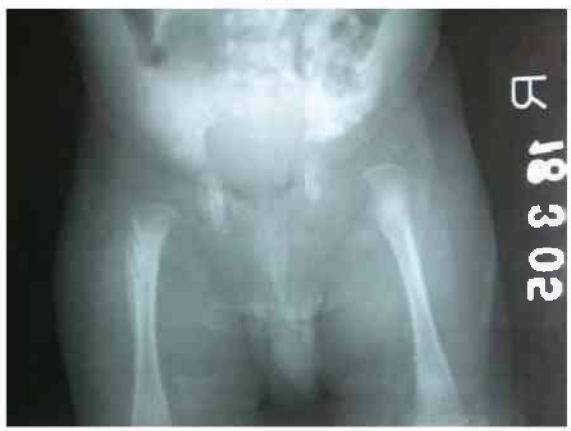
Early lesions occur in infants as multiple symmetrical epiphysitis at the ends of the long bones. The most common sites are at the distal end of the femur, proximal end of the tibia and distal ends of radius and ulna. The area is swollen and the child keeps the limb immobile due to pain (pseudo-paralysis). There may be other evidences of congenital syphilis in the child. Radiograph shows metaphyseal rarefaction and often a separation of the epiphysis. It can also occur as dactylitis involving the phalanges of the finger.

Acquired Syphilis

The osseous system is affected in the secondary and tertiary stages of syphilis.



(a)



(b)

FIGURE 4.11 (a) X-ray of leg showing syphilitic osteoperiostitis of tibia and (b) X-ray of pelvis with bilateral femur showing syphilitic osteoperiostitis. Note the thickening of the cortex to sclerosis.
Courtesy: Dr Rujuta Mehta.

In the secondary stage, periostitis occurs in the tibia and subperiosteal nodes may form, producing thickening of the shaft. The patient complains of pain, which is worse at night. Radiograph shows thickening of the cortex due to subperiosteal new bone formation and sclerosis (Fig. 4.11). Antisyphilitic treatment relieves the symptoms.

The tertiary lesion called *gumma* is now rare. It occurs in the skull, sternum and hard palate. The lesion starts as a granulomatous focus in the subperiosteal region. The underlying bone undergoes necrosis and is limited by an area of reactive sclerosis. The lesion bursts on the surface and forms a typical gummatous ulcer; secondary pyogenic infection is also superadded. Treatment is with antisyphilitic drugs and local dressings to prevent secondary infection.

PARASITIC INFECTION IN BONE (Hydatid Cyst)

Aetiopathology

Causative organism: Hydatid cyst is caused by infestation with a dog tapeworm, *Echinococcus granulosus.*

Life cycle: Dog is the definitive host and sheep is the intermediate host. Man gets the infestation by fondling dogs or by eating contaminated vegetables. In South India, it is found in the Madurai district of Tamil Nadu and Guntur in Andhra Pradesh. In the life cycle of the hydatid, the cyst stage forms in the bone or brain. There is a fibrous ectocyst and an endocyst consisting of the germinal layer. The parasite forms the cyst in the bone. This cyst is filled with an opalescent fluid containing multiple daughter cysts. Under the microscope, one can see hooklets and scolices.

Clinical Features

The common sites in the bone are the ilium, vertebra and occasionally long bones such as the femur and tibia. The patient presents with pain and a swelling in relation to the involved bone or the paraspinal region. The cyst in the vertebra can present with cord compression and paraplegia.

Investigations

Radiological examination shows a cystic lesion in the bone with scalloped margins (Fig. 4.12). It may also show a pathological fracture.

FIGURE 4.12 X-ray of lateral view of the lower end of femur showing hydatid cyst with pathological fracture.

The blood shows eosinophilia. Casoni's skin sensitivity test is positive wherein the patient's skin shows hypersensitivity reaction to the cyst fluid. A positive complement fixation test is also diagnostic.

Treatment

The only treatment is surgical removal of the cyst under very careful precautions to prevent spilling of the daughter cyst into the tissues. Wide excision of bone should be considered in bony lesions followed by stabilisation with graft or prosthesis.

ARTHRITIS (Infections of Joints)

Arthritis is defined as inflammation of a joint. Infections of joints are still common in India, leading to prolonged morbidity and disability. The inflammatory arthritis caused by infection can manifest in an acute or a chronic form:

1. Acute arthritis
   - Pyogenic arthritis
   - Gonococcal arthritis
   - Rheumatic arthritis
   - Smallpox arthritis
2. Chronic arthritis
   - Chronic pyogenic arthritis
   - Tuberculous arthritis

- Syphilitic arthritis
- Parasitic—guinea worm arthritis

Acute Pyogenic (Septic) Arthritis

Acute pyogenic arthritis is an inflammation of the joint due to infection by pyogenic organisms such as staphylococcus (most common), streptococcus or pneumococcus, gonococcus and meningococcus. It commonly occurs in children and adolescents. The common joints involved are the knee, hip and shoulder. The most common causative organism found in adults is *S. aureus*. In children, haemophilus influenza is common.

Route of Spread

The infection spreads most commonly by the haematogenous route. A primary focus may lie elsewhere in the form of tonsillitis, otitis, pyoderma, etc. Septic arthritis can also occur secondarily by extension from a neighbouring focus of pyogenic osteomyelitis. In adults, it is commonly caused by a penetrating wound at the time of injury; a compound wound at the time of injury can also result in the same. Umbilical cord sepsis is a specific route to neonatal pyogenic arthritis.

Pathology

The severity of this condition depends on the virulence of the organism and the resistance of the patient. Depending on its severity, the condition can present as a serous, serofibrinous or purulent type of arthritis.

In the serous type, the synovium is hyperaemic and the effusion is serous. In the serofibrinous type, the synovium is more inflamed and oedematous and the effusion is serofibrinous with a fibrinous deposit on the surfaces of the joint cavity.

In the most serious and virulent infection, there is frank thick pus in the joint cavity. There are areas of necrosis in the capsule and the synovium. The articular gets destroyed by the proteolytic enzymes in the exudate.

A pannus of the granulation tissue forms over the articular cartilage and destroys it further. The infection spreads to the periarticular and subcutaneous tissues and forms an abscess which may burst through the skin. The inflammatory exudates contain plasmin and other lysosomal enzymes and they destroy the cartilages. This later on results in ankylosis of the joint, that is, complete loss of the joint movement.

Clinical Features

The patient complains of severe throbbing pain, redness and swelling in the affected joint with the acute onset. There is high fever, malaise and constitutional disturbances such as tachycardia due to the toxaemia. The joint is swollen, distended with fluid (joint effusion), hot to touch and extremely tender. There is marked spasm of the muscles and painful limitation of all movements (pseudo-paralysis). The patient resists any attempt to move the joint. It passes through the following stages:

- Stage of synovitis with effusion
- Stage of reversible arthritis with minimal destruction of the articular cartilage
- Irreversible arthritis with total destruction of the articular cartilage and exposure of the underlying subchondral cancellous bone
- Stage of bony ankylosis

When the hip joint is involved, the limb is kept in flexion, abduction and external rotation. This is the position of comfort and maximum capacity of the joint cavity. In the case of the knee, it is kept in flexion.

Radiological Features

In the first week, the radiograph will not show any bony change. The distension of the joint with fluid is shown by an increase in joint space. Later, there will be narrowing of the joint space due to destruction of the articular cartilage.

Laboratory Findings

There will be polymorphonuclear leucocytosis and elevated ESR. Blood culture may be positive and can identify the organism. Aspiration of fluid from the joint and its analysis on Gram staining and culture sensitivity will reveal the causative organisms.

Differential Diagnosis

Acute septic arthritis has to be differentiated from acute osteomyelitis, traumatic synovitis or haemarthrosis, haemophilic bleed into joints, acute rheumatic fever (fleeting arthritis, elevated C-reactive protein) and irritable joint in children.

Management

The management of acute septic arthritis depends on the severity of the infection. The treatment specific to individual joints is discussed below.

For Knee Joint

Conservative Treatment

The leg is immobilised in a Thomas splint with skin traction applied from below the knee. The traction keeps the bony surfaces apart, relieves pain and also prevents further destruction of the articular surfaces.

Aspiration is done and the pus sent for smear, culture and sensitivity to antibiotics. Appropriate antibiotics are given parenterally. The swelling and pain will subside and the warmth also reduces. The splint is then removed and gradual active movements of the knee in bed are started. Later, weight bearing is permitted.

Surgical Treatment

If the aspirated fluid is frankly purulent, immediate drainage by arthrotomy must be performed. The joint is flushed with sterile saline and local antibiotics instilled. This regime along with parenteral broad spectrum antibiotics clears the infection with minimal destruction of the joint surfaces, leaving a joint with a useful range of movement.

When the destruction of articular surfaces has been gross, the joint is immobilised in the functional position. The inevitable end result will be bony ankylosis (Fig. 4.13).

For Hip Joint

A similar procedure with splinting with traction in a Thomas splint and antibiotic therapy is followed. Aspiration or arthrotomy and drainage are done according to the severity of the infection.

For Other Joints

In the elbow and wrist, splinting is done by a plaster slab and other measures are the same as above.

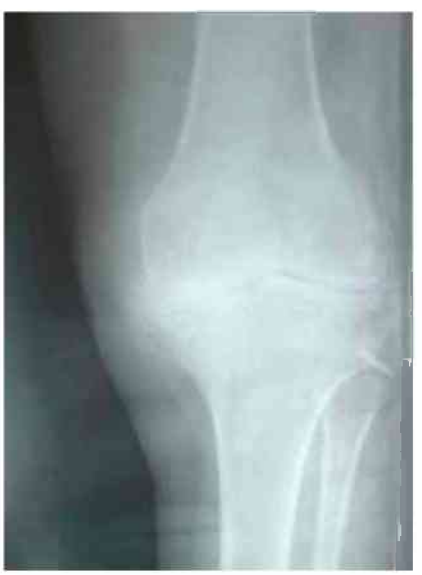


FIGURE 4.13 X-ray of knee anteroposterior view showing bony ankylosis following septic arthritis knee.

Septic Arthritis of Infancy (Tom Smith's Arthritis)

The septic infection of the hip joint in infants below 1 year is a distinct clinical entity. It can occur in the neonatal period due to the spread of infection from a septic umbilicus.

The condition has an acute onset with high temperature and swelling in the hip region which rapidly goes in for an abscess formation. The abscess may burst. As most of the head of the femur is cartilaginous, the infection completely destroys the head and dislocation occurs. In this stage, as the cartilaginous head is radiolucent, diagnostic ultrasonography is useful in demonstrating the condition of the head. If the condition is diagnosed in the acute stage, vigorous treatment by aspiration and antibiotic instillation or open drainage may save the head of the femur and resolve the infection.

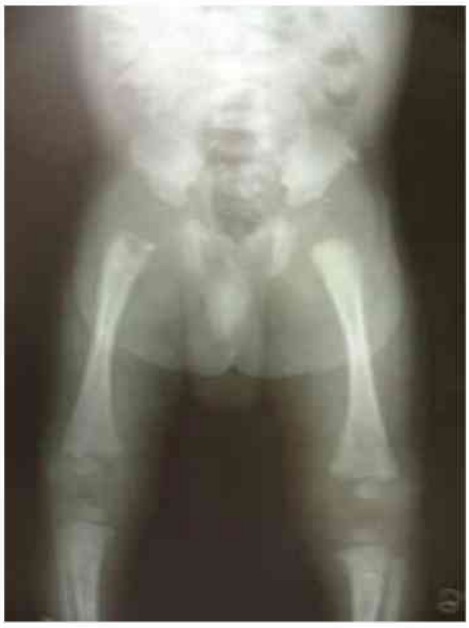


FIGURE 4.14 X-ray of pelvis with bilateral femur showing Tom Smith arthritis. Note the abscess of head and neck of femur.
Courtesy: Dr Rujuta Mehta.

Delayed Presentation

Very often, the child is brought months later when it begins walking with a painless limp. The affected leg is shorter and the child has an unstable hip gait. On examination, the hip has full range of movement and also shows telescopy. Clinically, the condition at this stage closely resembles the congenital dislocation of the hip. Radiograph shows complete absence of the head and neck of the femur with upward displacement of the trochanter (Fig. 4.14). The acetabular roof is normal.

This end result of a hypermobile joint instead of bony ankylosis is characteristic of septic arthritis in infancy. If the head and neck are completely destroyed, reconstructive surgical procedures are indicated for stabilising the hip.

Smallpox Arthritis

Arthritis following smallpox is not seen now as smallpox has been successfully eradicated in India. Patients who had suffered from smallpox in the younger years still present with the late effects of this crippling arthritis.

The condition affects multiple joints and is often symmetrical in distribution. It involves both elbows, ankles or knees. There is extensive destruction of the articular ends of the bones causing disorganisation of the joint. The end result is often a disorganised joint with some useful range of movements. In older children, involvement of elbows often leads to bilateral bony ankylosis.

Acute Gonococcal Arthritis

The incidence of acute gonococcal arthritis has considerably diminished due to the more efficient treatment of acute gonorrhoea by antibiotics. The acute arthritis usually involves a single large joint such as the knee (most commonly affected) or the ankle. The inflammation is confined to subsynovial layers and does not invade the joint. It may start a few weeks after the onset of the gonorrhoeal urethritis. Periarticular oedema is a common feature of gonococcal infection. The general condition of the patient is well maintained. Treatment is by rest and antibiotics and aspiration, if necessary; penicillin is the drug of choice.

Acute Rheumatic Arthritis

Acute rheumatic arthritis occurs usually in children with a sudden onset of fever and constitutional disturbances. The most characteristic feature is the fleeting and flitting nature of the joint pain; one day, one joint may be painful and swollen and the next day it subsides and some other joint is swollen and painful. The condition rapidly responds to salicylate therapy.

Chronic Arthritis (Pyogenic)

Chronic pyogenic arthritis may present as a persisting, grumbling arthritis with features of swelling, low-grade warmth, tenderness and painful movement. It also occurs in the iatrogenic form, following intra-articular hydrocortisone

injection given with insufficient aseptic facilities and precautions. Treatment is by rest, antibiotics and arthrotomy. Its late sequelae is bony ankylosis.

The most common chronic arthritis in India is tuberculous arthritis. This is dealt with in detail in Chapter 5.

Syphilitic Lesions of Joints

Children with congenital syphilis sometimes develop bilateral serous symmetrical synovitis in the knee joints. They present with painless swelling of both knees with marked effusion. These are called Clutton's joints.

In the secondary stage of syphilis, patients complain of fleeting joint pains. This is called syphilitic arthralgia and is worse at nights. Diagnosis is by the concomitant presence of other secondary syphilitic manifestations.

In the tertiary stage of syphilis, patients with tabes dorsalis may manifest a neuropathic joint lesion called Charcot's joint. This is characterised by painless swelling in the knee with gross disorganisation and instability. It is an indirect consequence of syphilis.

Acquired Syphilis

Polyarthritis and gummatous arthritis are consequences of the acquired syphilis.

Guinea Worm Arthritis

Guinea worm arthritis is a condition where a parasitic infestation with guinea worm (*Dracunculus medinensis*) manifests as arthritis. This infestation is endemic in certain areas in South India. This type of nematode infection occurs in areas where step wells are used as sources of water supply, where people get down into the water. This condition is now rare due to the improvement in the well construction in rural areas.

The condition is usually seen in the knee or the ankle. The joint is swollen and tender, and there is an abscess near the joint around the site of the adult worm. When the worm presents through the burst abscess, the patient tries to extract it by pulling and twisting it around a match stick. Snapping of the worm during this manoeuvre gives rise to an exacerbation of the arthritis. When the condition subsides, the worm gets calcified and is seen in the radiograph as a cork screw-like shadow.

KEY POINTS

- Osteomyelitis is inflammation of the bone and bone marrow caused by infective organisms. Osteitis indicates inflammation of bone.
- The most common cause of acute pyogenic osteomyelitis is *Staphylococcus aureus* and most common sites are distal femur and proximal tibia (metaphysis).
- In early cases of acute pyogenic osteomyelitis, no radiologic changes are seen. In these cases, Tc 99 bone scan will show increased uptake.
- Antibiotics are given for 4–6 weeks to manage cases of acute osteomyelitis.
- Chronic osteomyelitis is persistent pyogenic infection for more than 6 weeks.
- Sequestrum is a piece of dead bone separated from healthy bone. Involucrum is the reactive new bone formed subperiosteally.
- Brodie's abscess is an abscess in the medulla of the bone, due to a low-grade infection by *staphylococcus*.
- Garre's osteomyelitis is a diffuse sclerosing inflammatory lesion of the shaft of the long bone or the jaw.
- Salmonella osteomyelitis is subacute osteomyelitis that also occurs as a complication of sickle cell anaemia.
- Brucella osteomyelitis is infectious disease of animals that is transmitted to human being.
- Mycetoma is caused by *Actinomyces* or *Maduramyces*.
- Mycetoma presents with a large diffuse indurated swelling of the foot with multiple discharging sinuses (sulphur granules).
- The most common causative organism of acute pyogenic arthritis in adults is *Staphylococcus aureus*. In children, *haemophilus influenza* is common.
- Depending on the severity of infection, the condition can present as a serous, serofibrinous or purulent type of arthritis.
- Tom Smith arthritis is septic infection of the hip joint in infants below 1 year of age.
- Children with congenital syphilis present with painless swelling of both knees with marked effusion—Clutton's joints.

MULTIPLE CHOICE QUESTIONS

1. Bony ankylosis results from
   a. Pyogenic arthritis
   b. Tuberculosis arthritis
   c. Rheumatic arthritis
   d. All of the above
2. Which is true regarding acute osteomyelitis?
   a. Staphylococcus is the usual organism
   b. Rest and elevation relieve the pain
   c. Parenteral antibiotics relieves the pain
   d. All of the above
3. What is Brodie's abscess?
   a. Long-standing localised pyogenic abscess in the bone
   b. Cold abscess
   c. Subperiosteal abscess
   d. Soft tissue abscess
4. All are seen in chronic osteomyelitis except
   a. Sequestrum
   b. Amyloidosis
   c. Myositis ossificans
   d. Metastatic abscess
5. Tom Smith arthritis manifests as
   a. Increase hip mobility and unstability
   b. Hip stiffness
   c. 'a' and 'b'
   d. Shortening of limb

CHAPTER 5

Bone and Joint Tuberculosis

SKELETAL TUBERCULOSIS

Skeletal tuberculosis is still a common orthopaedic problem in India and in many other developing countries of the world. It affects nearly 30 million people world over. The incidence of tuberculosis in a community reflects the standard of nutrition and housing in the society, and hence tuberculosis can be called a 'social disease'. In an individual, the skeletal lesion is a local manifestation of a systemic infection, the source often being a lesion in the lung. The incidence of tuberculosis has had a dramatic fall in the Western countries due to marked improvement in the socioeconomic status and the availability of extremely effective antitubercular drugs. It is more prevalent in lower socioeconomic groups. Osteoarticular tuberculosis, that is, tuberculosis of the bones and joints, constitutes about 1–3% of all cases of tuberculosis.

Incidence

The incidence of skeletal tuberculosis is high in the first 3 decades of life. Spine is the commonest site of bone and joint tuberculosis, followed by hip, knee and other joints (Table 5.1).

TABLE 5.1 Prevalence of Tuberculosis in Different Parts of the Body

Parts of Body	Prevalence (%)
Spine	42.0
Hip	8.0
Knee	7.0
Sacroiliac joint	6.0
Elbow	4.5
Tarsal bones	4.0
Ankle	4.0

Aetiopathology

The human type of tubercle bacillus, *Mycobacterium tuberculosis*, is invariably the causative organism in India. The organism enters mainly via the nasal route, and the initial focus is set up in lungs, from where the infection spreads haematogenously and reaches the bone.

The focus in the bone is always secondary to another focus, usually in the lungs or in the mediastinal glands. The spread is by the blood stream and the infection settles in the bone usually near the epiphyseal cartilage where the synovial membrane gets attached.

The 'tubercle' is the microscopic pathological lesion with a central necrosis surrounded by epithelioid cells, giant cells and round cells. Two types of microscopic lesions can be recognised:

1. The *caseating exudative type* where necrosis, caseation and cold abscess formation predominate (Fig. 5.1)
2. The *proliferating type* where the cellular proliferation predominates with minimal or no

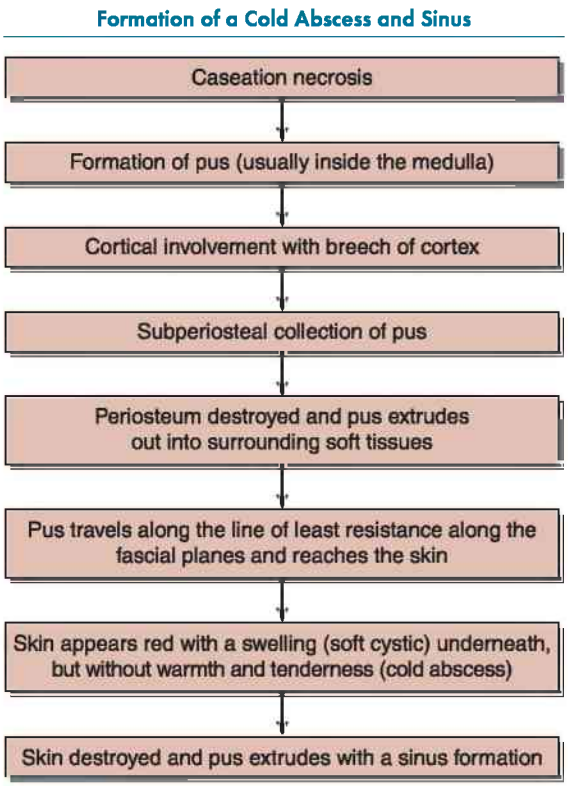


FIGURE 5.1 Flowchart depicting the formation of cold abscess and sinus.

caseation; the extreme form of this type is the tuberculous granuloma. ('Caries sicca' is a lesion of the dry proliferating type.) There is minimal bone destruction, and this type is more commonly seen in the adults.

The lesion in children is commonly the caseating type with rapid and extensive destruction of bone and cartilage. The lesion in the adult is more commonly of the proliferating type with minimal bone destruction.

Clinical Features

Clinically, the onset of tuberculosis in any bone or joint is insidious. In the early stage, there are constitutional symptoms such as loss of weight, loss of appetite and low-grade evening temperature. The localising symptoms and signs then appear in the affected part. There may be pain, swelling and local tenderness of involved bone and joints with associated muscle spasm. Acquired deformities may be present (kyphosis in the spine and triple deformity in the knee). Cold abscess formation may also occur (Fig. 5.2).

Investigations

1. *Radiograph:* Anteroposterior and lateral views are required in most cases. A radiograph of the chest (posteroanterior view) must be taken for possible pulmonary/mediastinal involvement. CT and MRI may also be done to look for soft-tissue involvement, cold abscess and lymphadenitis. The earliest sign in an X-ray examination is decalcification of the bones related to the affected joints. Later, films show joint destruction and bone rarefaction.
2. *Blood:* The following tests are usually done:
   - RBC count
   - Hb percentage may be decreased
   - Erythrocyte sedimentation rate increased
   - WBC (total and differential) count—lymphocytes predominate in the differential leucocyte count
3. *Mantoux test:* Mantoux test is useful in children and adults. A negative Mantoux test does not rule out bone/joint tuberculosis.
4. *Sputum examination:* This is done for acid fast bacillus (AFB) by Ziehl–Neelson staining and culture in the Lowenstien–Jensen medium.
5. *Aspiration of abscess:* Aspiration of thin pus from cold abscess often confirms the diagnosis. The pus is sterile on culture for pyogenic organisms. Sometimes, tubercule bacilli can

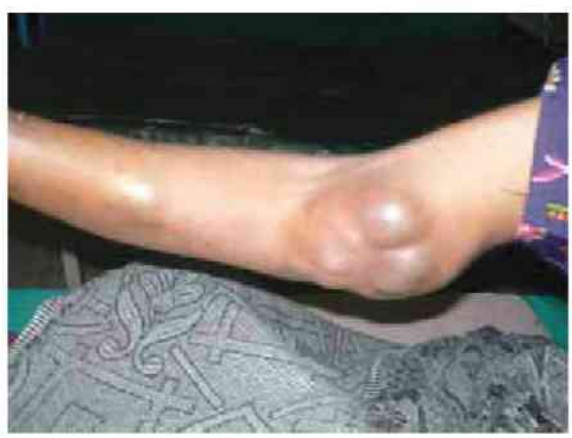


FIGURE 5.2 Clinical photograph showing cold abscess over the elbow.

be demonstrated in smears, culture of the pus and by guinea pig innoculation.

6. *Biopsy:* In early stages, when the signs and symptoms are minimal, synovial biopsy or a biopsy from the bony focus will establish the diagnosis before there are obvious signs of articular destruction. The tissue is stained for AFB and culture is done for bacilli.

Management

In skeletal tuberculosis, the skeletal focus is only a local lesion in a patient with systemic tuberculosis. The management should be comprehensive, including the general and local treatment for the disease. The availability of the modern antituberculous drugs has certainly improved the prognosis but has not changed the basic principles of treatment of this crippling disease.

General Treatment

The patient's general condition should be improved by rest, hygienic environment, high protein and nutritious diet including milk, eggs, vitamins and haematinics.

Chemotherapy

The advent of the modern antituberculous chemotherapeutic drugs has revolutionised the medical management of skeletal tuberculosis. Chemotherapy has shortened the duration of the treatment to 6–9 months from 1.5–2 years. It has made surgery safer and possible than what it was earlier. It has also markedly reduced the incidence of complications.

It is preferable to give multidrug antituberculous therapy in the following dosage schedule with modifications according to the age and weight of the patient. The drugs which are now available for short-term chemotherapy are Rifampicin, Isoniazid (INH), Ethambutol, Streptomycin and Pyrazinamide (Table 5.2).

It is very essential to watch for symptoms of toxicity while using these powerful drugs. Rifampicin, which is a bactericidal antibiotic, is noted for its hepatotoxicity and hence liver function must be monitored during therapy. Peripheral neuritis is the common toxic symptom while using INH. Ethambutol can cause optic neuritis. One must watch for vertigo, tinnitus and deafness while using Streptomycin. Hyperuricaemia, arthralgia and hepatitis are the toxic effects of Pyrazinamide.

Local Treatment

The aim of local treatment is to correct deformities due to muscle spasm, minimise destruction of articular surfaces and preserve the maximum mobility in the affected joint.

Role of Surgery

The aim of surgery in skeletal tuberculosis is clearance of the lesion wherever possible and the preservation of function of the joint.

In lesions such as caries of the rib, excision of the affected bone is done. It is possible to excise or curette a tuberculous focus in joints in the active stage of the disease under cover of antituberculous drugs. Surgery is done to manage the complications of the disease. It is also indicated to treat the end results of the disease, such as deformity and ankylosis in the peripheral joints.

TABLE 5.2 Dosage of Antituberculous Drugs and Their Side Effects

Drugs	Daily Dose (mg/kg)	Thrice-Weekly Dose (mg/kg)	Side Effects
Isoniazid	5	10	Peripheral neuritis, hepatitis
Rifampicin	10	10	Hepatitis
Pyrazinamide	25	35	Hepatitis, hyperuricaemia
Ethambutol	15	30	Optic neuritis
Streptomycin	15	15	Ototoxicity, nephrotoxicity

TUBERCULOSIS OF THE SPINE (Pott's Disease)

Incidence

Tuberculosis of the spine is the commonest site of bone and joint tuberculosis. Tuberculosis of the spine forms 40–45% of the total incidence of skeletal tuberculosis. It is a disease of childhood and adolescence, 50% of cases occurring in the age group of 1–20 years. The most common level of the lesion is in the thoracolumbar level. This is because movement and the stress of weight bearing are maximum at this level. The percentage-wise regional distribution of tuberculosis lesion in the vertebral column is given in Table 5.3.

Anatomy

There are 7 cervical, 12 thoracic, 5 lumbar, 5 sacral and 4 coccyx vertebrae in human body.

A classical vertebra has a wide body anteriorly, lamina and spinous process posteriorly attached to the body by the pedicles. The lumbar vertebra has transverse processes, each on either side laterally (Fig. 5.3). The focus in the spine may be found in one of the above-mentioned sites.

The length of the vertebral column is greater than the length of the spinal cord. The spinal cord ends at the level of the lower border of the first lumbar vertebra due to disproportionate growth of the vertebral column and spinal cord.

Beyond the level of L1, the dural sac contains only a bunch of nerve roots, namely cauda equina (horse tail). The relationship between the vertebral column and the spinal cord segment is given in Table 5.4.

TABLE 5.3 Regional Distribution of Tuberculosis in the Spine

Part of the Spine	Percentage Distribution
Cervical	12
Cervicodorsal	5
Dorsal	42
Dorsolumbar	12
Lumbar	26
Lumbosacral	3

In 7% of cases, more than one level is involved.

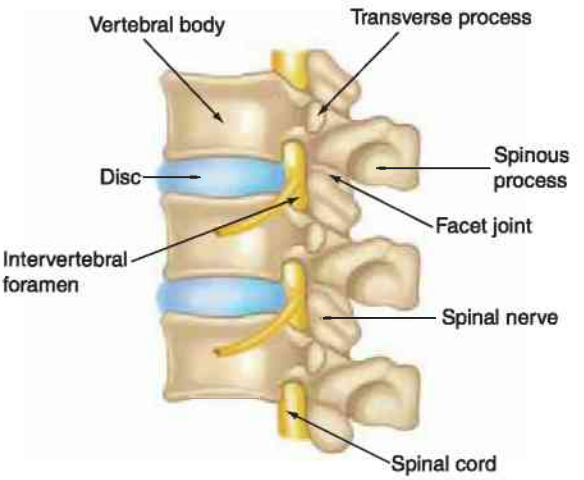


FIGURE 5.3 Parts of typical lumbar vertebra. Also shown are the spinal cord and spinal nerve exiting from the intervertebral foramen.

TABLE 5.4 Relationship Between the Vertebral Column and Spinal Cord Segment

Vertebral Column	Spinal Cord Segment
Cervical	Add 1 to the vertebral level
Upper dorsal	Add 2 to the vertebral level
Lower dorsal	Add 3 to the vertebral level
D10	All dorsal segments over
D12	All lumbar segments over
L1	All sacral segments over

Pathology

Spinal tuberculosis is always secondary to infection elsewhere in the body, such as lungs, gastrointestinal tract or genitourinary tract. The route of infection is haematogenous. Paravertebral Batson's venous plexus has free communication with visceral organs leading to haematogenous spread.

The focus in the spine may be found in one of the following sites (Fig. 5.4):

- *Paradiscal lesion* in the subchondral area of the vertebral body. This is the common site.

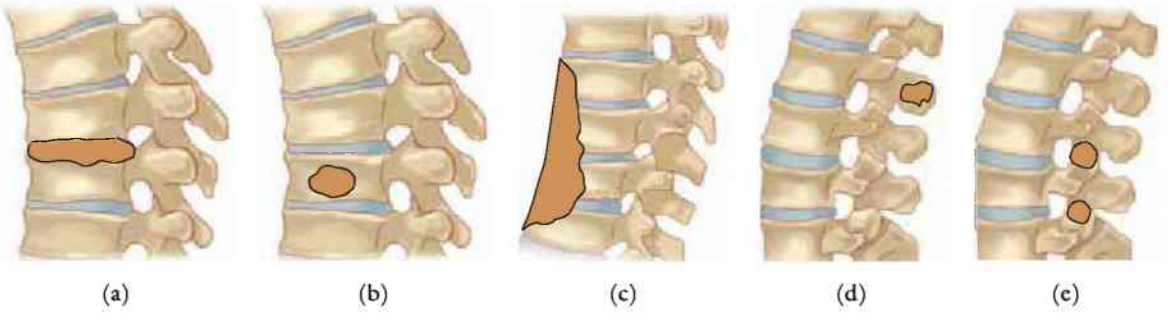


FIGURE 5.4 Sites of lesion. (a) Paradiscal, (b) central, (c) anterior, (d) appendiceal and (e) articular.

This is because the two adjacent vertebrae (lower half of the upper vertebra and the upper half of the lower vertebra), along with the intervening disc, develop from single sclerotome. These structures therefore have common blood supply and hence the paradiscal infection

- *Central body lesion* in the centre of the vertebral body leading to the collapse of the vertebra
- *Anterior type* in the anterior surface of the vertebral. Infection starts below the anterior longitudinal ligament and the periosteum of the anterior part of the body
- *Appendiceal lesion* in the pedicle, lamina, transverse process and spinous process
- *Articular lesion* in the posterior intervertebral facet joint

In the affected vertebra, there is granulomatous inflammation. This lesion soon destroys the intervertebral disc and the adjacent surface of the vertebral bodies which slowly collapse and obliterate the intervertebral space. There is anterior wedging of the vertebra, and this wedging is more common in the dorsal vertebra than cervical and lumbar vertebra. Destruction of the framework of the vertebral bodies results in their collapse and the development of an angular kyphosis called *gibbus*. The disease commonly involves two vertebrae, but in children it can rapidly destroy three or more vertebrae and cause gross deformities. Skip lesion involvement in more than one vertebral level must be looked for. The histological changes in the bone are typical of tuberculous lesions elsewhere. Spreading caseation results in osteolysis of the bony trabeculae, leading to the formation of cold abscess.

Healing of the lesion occurs with the formation of the new bone at the areas of lysis, thereby leading to bony ankylosis. This is most commonly seen in the paradiscal type of lesion.

Tuberculoma

Compared to the total incidence of spinal tuberculosis, tuberculoma formation in the spinal cord is a rare phenomenon; it presents like an intramedullary spinal tumour causing cord compression and paraplegia.

Clinical Features

The classical symptoms of tuberculosis of the spine as described by Percival Pott are back pain, rigidity, deformity, cold abscess and paraplegia, along with the constitutional symptoms:

- *Back pain:* Pain in the back will be localised by the patient to one region of the spine. It is diffuse, dull aching but later it can become radicular in nature. Localised tenderness over one vertebral spine is diagnostic of the level of the lesion. The disease can also present as *referred pain*. Disease in the *cervical spine* can present as pain in the ear or pain down the arm. *Upper thoracic spine* lesion can present as pain in the chest and as intercostal neuralgia. *Lower thoracic spine* can cause referred pain in the abdomen.
- *Rigidity:* Rigidity is caused by the spasm of the paravertebral muscles due to the disease in the spine. There may be associated night cries. A *cervical lesion* causes rigidity of the

neck which at times may be asymmetrical, producing torticollis. In *lumbar lesions*, there is marked rigidity of the back and the spine moves in one piece when the patient attempts to bend forwards. This is demonstrated by the *coin test*. The patient is asked to pick up a coin from the floor. He bends at the knee and hip and picks up the coin holding the spine rigid and straight all the time.

- *Deformity:* Deformity is a typical kyphotic deformity or gibbus due to wedging of one or two vertebra with forward angulation. Forward wedging of one or two vertebral bodies produces small kyphosis—*knuckle kyphosis*. Wedging of three or more bodies would produce an *angular kyphosis* (gibbus) and moderate wedging of a large number of vertebrae would cause a *round kyphosis*. In the *cervical and lumbar spine*, the loss of the normal lordosis occurs first followed by the gibbus. In the *thoracic spine*, angular kyphosis (gibbus) is characteristic. The prominence of the gibbus depends on the number of the vertebrae involved.
- *Cold abscess:* The formation of cold *abscess* is an invariable feature of tuberculosis of the spine. The abscess is called cold abscess since it shows no sign of inflammation. The pus and debris formed from the affected vertebra courses along the fascial planes or neurovascular bundles. As a result, the abscess from the cervical and dorsal regions can present far away from the vertebral column. The pus can go anterior to vertebra forming *prevertebral abscess* or the side of the vertebral body forming *paravertebral abscess* (Fig. 5.5).

The abscess in the paravertebral areas soon tracks downwards due to gravity and towards the surface following the tracks of nerves and blood vessels.

In the *cervical spine*, the cold abscess can point retropharyngeally producing dysphagia or show up in the neck behind the sternomastoid. In the *thoracic spine*, the cold abscess fills up the posterior mediastinum and tracks along the intercostal nerves to point either in the lateral chest wall or in the anterior chest wall. The abscess reaches the posterior surface also under the sacrospinalis muscle. The cold abscess sometimes enters the spinal canal causing pressure on the spinal cord resulting in paraplegia. Thoracolumbar cold abscess can point either in the back or enter the

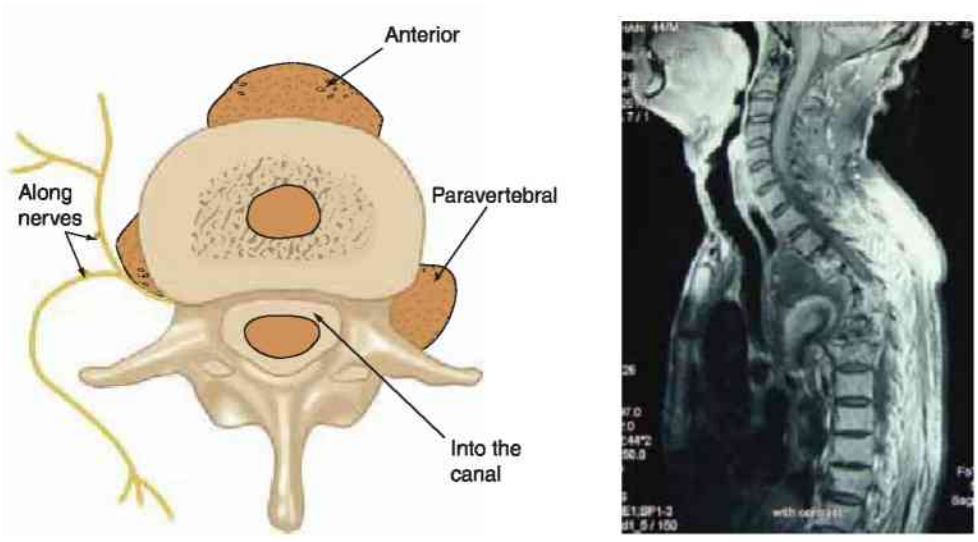


FIGURE 5.5 (a) Diagrammatic representation of a typical lumbar spine showing sites of cold abscess formation. (b) MRI scan of the cervicodorsal spine showing extensive tubercular involvement of the dorsal vertebrae D2-6 with gibbus and large pre-vertebral cold abscess tracking down till D10.

psoas sheath and track down as psoas and iliac abscesses. These abscesses collect as lumps in the iliac fossa and point above the inguinal ligament or track down behind the inguinal ligament, and point in the femoral triangle or even lower down.

- *Paraplegia:* If neglected, the destruction of the vertebral body will invade and involve the neural structures causing *Pott's paraplegia.*
- *Constitutional symptoms:* These include evening rise of temperature, loss of weight, loss of appetite, etc.

Radiological Features

1. *Plain X-ray film:* To know the level of the lesion, minimum anteroposterior and lateral views of the affected areas of the spine are needed. The following can be detected in the plain X-rays; however, these fractures can also be viewed in an MRI:
   - *Reduction of disc space:* This is the earliest radiological sign of spinal tuberculosis. It is well evident in the lateral views of the spine. Later, there is erosion of the adjacent surfaces of the vertebral bodies (Fig. 5.6). After that, there is destruction and collapse of the vertebral bodies with obliteration of the intervertebral space.
   - *Kyphosis:* As the disease is progressing, due to increased destruction of the margins, of the affected vertebrae, an angular kyphotic deformity develops. The severity of the deformity depends on the number of vertebrae involved. Sound healing usually ends in bony fusion of adjacent vertebrae. Neglected cases in children result in gross kyphotic deformities (Fig. 5.7).
   - *Paravertebral shadows:* They are produced by extension of tuberculous granulation tissue and the collection of an abscess in the paravertebral region (Fig. 5.8). Retropharyngeal abscess in the cervical region usually presents as a soft tissue shadow between the vertebral bodies and the pharynx and trachea. Retropharyngeal abscess in the dorsal region produces a *fusiform-shaped* shadow. An abscess under tension will produce a *globular shadow.* Widened mediastinum is a feature of dorsal cold abscess. The psoas shadow on an X-ray of abdomen may show a bulge if there is psoas abscess.

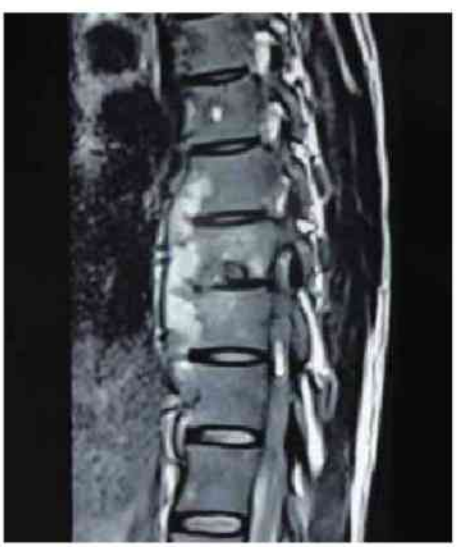


FIGURE 5.6 MRI scan of the dorsolumbar spine showing early tubercular lesion, D11 and D12 with narrowing of intervertebral disc space and paradiscal erosion of the body of D11.

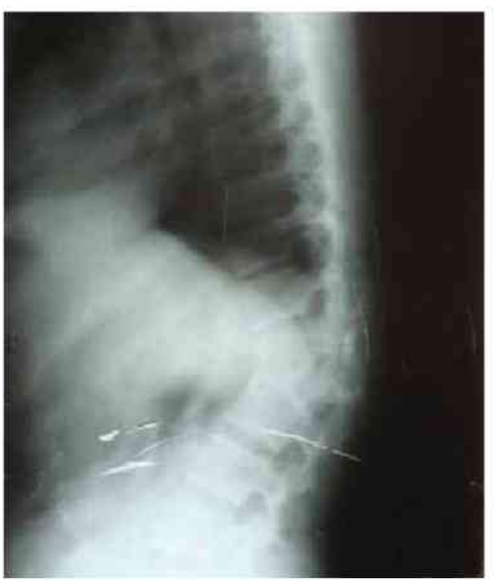


FIGURE 5.7 Lateral view X-ray of the thoracolumbar spine showing a neglected case of tuberculosis with kyphotic deformity at the D12 level.

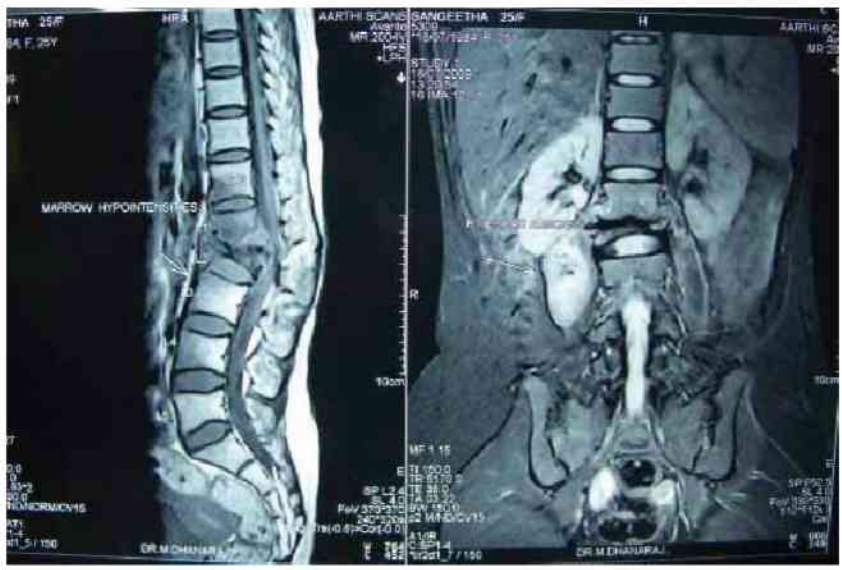


FIGURE 5.8 MRI scan of the dorsolumbar spine showing destruction of L1 and L2 vertebrae with a large psoas abscess on the right side.

- *Aneurysmal sign:* In the region of thoracic spine, the tense paravertebral abscess of long-standing duration may cause concave erosions along the anterior margins of the vertebral bodies causing *aneurysmal sign* in lateral films.

2. *CT scan:* The following features can be seen on a CT scan:
   - Extent of destruction of the vertebral body
   - Small paravertebral abscess
   - Sequestra and bony debris of vertebral bodies
   - Detection of spinal tumour syndromes
3. *MRI:* Additionally, an MRI can reveal the following details:
   - Extent of cord compression (Fig. 5.9)
   - Soft tissue involvement of the disease
4. *Myelography:* It is performed in the following conditions:
   - Multiple level lesions
   - Failed surgical debridement
   - Detection of spinal tumour syndromes
   - Detection of tuberculosis arachnoiditis

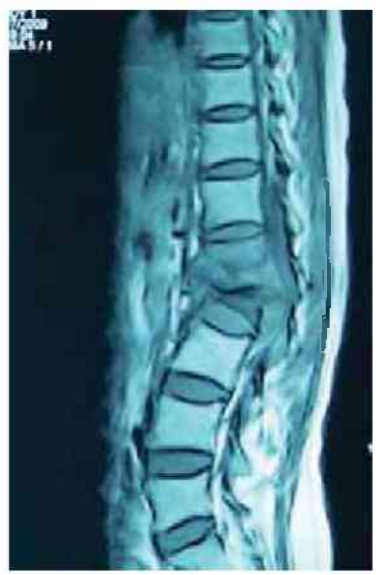


FIGURE 5.9 MRI scan of the dorsolumbar spine showing late tuberculosis lesion with destruction and collapse of L2 vertebra with partial destruction of L1 vertebra.

Biopsy

Biopsy is done for confirming the doubtful diagnosis.

Other basic investigations such as chest X-ray, ESR, Mantoux test and ELISA for tuberculosis antibody can also be done.

Differential Diagnosis

1. *Trauma:* This diagnosis can be made if there is
   - no fever or abscess,
   - no paravertebral shadow on X-ray,
   - fracture dislocation is seen in an X-ray.

2. *Secondaries:* The secondaries are suspected if the following are present:
   - Old age group
   - Disc space normal
   - Pedicles involved
3. *Disc prolapse:* Disc prolapse is suspected if there is a radiating pain with a positive Straight Leg Raising Test (SLRT).
4. *Ankylosing spondylitis:* The following clinical features can point to the above clinical condition:
   - Reduced chest expansion
   - Bamboo spine appearance in X-ray

Other conditions such as primary malignant conditions, osteoporotic conditions and spondylolisthesis should be differentiated from the spinal tuberculosis.

Treatment

The aim of treatment is as follows:

- Healing of the disease
- Prevention, early detection and prompt treatment of complications such as paraplegia

The other modalities used are as follows.

1. *Rest:* Rest is essential for pain relief and also to prevent further collapse and pathological dislocation of the spine and antituberculous chemotherapy is started. If the patient is noncompliant, then plaster of Paris body cast can be given to immobilise the spine. The patient is periodically assessed clinically, radiologically and haematologically
2. *Antitubercular treatment:* Antitubercular treatment regimen consists of the following:
   - *Intensive phase:* INH, rifampicin and ofloxacin for 5–6 months
   - *Continuation phase:* INH and pyrazinamide for 3–4 months followed by INH and rifampicin for 4–5 months
   - *Prophylactic phase:* INH and ethambutol for 4–5 months
3. *Gradual mobilisation:* Mobilisation with suitable spinal braces is advised as soon as the pain at disease site permits or when the lesion is quiescent. After 3–9 weeks, back extension exercise, 5–10 min, three to four times a day, is started. Spinal brace is continued for about 18 months to 2 years when it is gradually discarded.
4. *Aspiration of abscess:* Abscesses are aspirated when near the surface, and 1 g of streptomycin with or without INH in solution is instilled at each aspiration.

POTT'S PARAPLEGIA (Spinal Tuberculosis with Neurological Involvement)

The paraplegia in spinal tuberculosis is called Pott's paraplegia. The overall incidence of this complication is between 10 and 30%. The highest incidence of paraplegia is seen in lesions of the thoracic spine. This is because of the fact that spinal canal is narrowest in this area and even a small compromise can lead to neurological deficit.

Aetiopathology

Pott's paraplegia is due to pressure effect on the neural tissues within the canal due to diseased vertebra. The usual causes of paraplegia in spinal tuberculosis are as follows:

1. Mechanical/extrinsic causes
   - *Tubercular debris*
   - *Sequestra from vertebral body and disc:* Sequestra from the devascularised part of the vertebra or intervertebral disc can cause compression.
   - *Vertebral canal stenosis:* Stenosis can occur due to fibrosis of the canal or during bony fusion. Surgical decompression is required.
   - *Internal gibbus:* Angulation of the diseased spine may lead to the formation of a bony ridge or spur called *internal gibbus* on the anterior wall of the spinal cord.
   - *Extradural granuloma:* The extradural mass is the commonest mode of affection. It may be in form of extradural granuloma or tuberculoma or peridural fibrosis. The tuberculosis osteitis of the vertebral bodies along with formation of an abscess in the extradural space causes compression of the cord from the anterior aspect. Meningeal changes such as extradural granulation

tissue and peridural fibrosis may be responsible for some cases of recurrence of paraplegia.

2. Intrinsic causes
   - *Prolonged stretching of the cord:* Stretching of the cord may cause reversible changes such as compression, posterior displacement or irreversible changes such as atrophy of the cord.
   - *Infarction of the spinal cord:* The infarction of the spinal cord can be caused by endarteritis, periarteritis or thrombosis of an important tributary to the anterior spinal artery caused by inflammatory reaction. The paralysis caused by infarction is irreparable
   - *Pathological dislocation of the spine:* This condition occurs because of the rough manipulation by native bone setters or indiscriminate laminectomy for caries spine
   - *Tuberculous meningomyelitis:* Involvement of the spinal cord and its leptomeninges results in incomplete recovery.
   - *Syringomyelic changes:* Tuberculosis has been found to cause widening of the spinal canal and neurological deficit. This results in poor recovery.
   - *Spinal tumour syndrome:* Diffuse extradural granuloma or tuberculoma in the spinal cord or peridural fibrosis can mimic spinal tumour and hence is known to cause spinal tumour syndrome.

Depending on the severity of the paralysis, paraplegia is graded as Grades I, II and III, Grade I being a partial paralysis (paresis) and Grade III being a total paraplegia.

Classification

Pott's paraplegia is classified into the following types:

- *Early onset paraplegia* occurring during the active stage of the disease usually within 2 years of onset
- *Late onset paraplegia* occurring in patients whose lesion has reactivated after long years of quiescence, usually at least 2 years after the onset of the disease

The early onset paraplegia is due to the pressure on the cord by (1) cold abscess, (2) granulation tissue, (3) necrotic debris and sequestra from the bone or the intervertebral disc tissue and (4) occasionally, a vascular thrombosis of the spinal arteries which produces a sudden total paraplegia.

In the late onset type, the paraplegia could be either due to the reactivation of the disease causing cold abscess or due to mechanical pressure or stretching of the cord by acute angulation in the spine. The prognosis is worse in the late onset paraplegia.

A rare type is the gradual onset paraplegia due to a granulomatous mass developing inside the spinal cord. This is called the *spinal cord tumour syndrome.*

Clinical Features

In the clinical examination look for every early sign of pressure on the cord, such as slight spasticity of the legs causing unsteady gait, exaggerated knee and ankle jerks, and extensor plantar response. The progressive severity of neural deficit due to cord compression can be staged as follows (Table 5.5).

- *Stage I:* The patient is able to walk normally and he/she is not aware of any motor weakness.

TABLE 5.5 Classification of Tuberculous Paraplegia/Tetraplegia (Predominantly Based on Motor Weakness)

Stage	Clinical Features
I (Negligible)	Patient unaware of neural deficit Plantar extensor reflex/ankle clonus present
II (Mild)	Patient aware of deficit but manages to walk
III (Moderate)	Non-ambulatory because of paralysis (in extension) sensory deficit less than 50%
IV (Severe)	III + flexor spasms/paralysis in flexion/flaccid/sensory deficit more than 50%/sphincters involved

- *Stage II:* The patient presents with the complaints of clumsiness or spasticity of the limbs while walking. He/she is able to walk with or without support. Clinical examination reveals all signs of spastic paresis.
- *Stage III:* The patient is bedridden and cannot walk because of severe weakness. Examination reveals spastic ***paraplegia in extension***.
- *Stage IV:* Severe form of the disease. The patient has paraplegia with flexor spasm or ***paraplegia in flexion***. Flaccid paralysis due to very severe cord compression or sudden compression is also included in stage IV.

Investigations

- Plain radiography can reveal the site and level of bony destruction with involvement of disc space.
- CT scan can detect the type of destruction, presence of paravertebral abscess and any pus or sequestra.
- MRI is the investigation of choice.

Management

The initial treatment of the case is the same as before. The patient is immobilised in the plaster shell and chemotherapy started. A neurological chart is maintained and the clinical status of the paraplegia recorded once a week.

With this regimen, more than 50% of the cases with paraplegia recover in a few months. This is due to the resorption of the intraspinal cold abscess resulting in a medical decompression of the spinal cord.

Conservative Treatment

- *Rest:* By traction for cervical disease and bed rest or plaster of Paris body cast for dorsal and lumbar disease
- *Antitubercular drugs:* Patients are started with four drugs of antitubercular chemotherapy as soon as the diagnosis is made

The patient is periodically assessed clinically, radiologically and haematologically. When the lesion is quiescent, the patient is given a spinal brace (Fig. 5.10) and made ambulant. If the paraplegia improves, the conservative treatment is continued. The patient is allowed to move with the help of braces. Bracing is continued for a period of about 18 month to 2 years.

FIGURE 5.10 Clinical photograph showing a patient wearing spinal brace—Taylor's brace.

Surgical Treatment

If paraplegia does not improve or if it actually deteriorates, surgical intervention is indicated. Following are the indications for surgical treatment:

- No progressive recovery to a satisfactory level after a period of 3–4 weeks of conservative therapy
- The patients for whom neurological complications develop during conservative treatment
- Patients with neurological complications which become worse while undergoing conservative treatment
- Patients who have a recurrence of neurological complications
- Patients with prevertebral cervical abscess with neurological signs and pressure symptoms

- Patients with advanced disease with flaccid paralysis or severe flexor spasms
- Spastic paraplegia with severe and uncontrollable spasms of the legs

Operative Procedures for Pott's Paraplegia

The aim is to remove the agents causing compression on the neural structures. The operations for decompression are as follows.

Anterolateral Decompression *(Fig. 5.11a)*

This is the most commonly done surgery. Access is made from the front and sides of the cord. In this operation, in addition to costotransversectomy, the pedicles and part of the vertebral bodies are excised to achieve decompression. However, lamina or facet joints are not removed.

Costotransversectomy *(Fig. 5.11b)*

In this operation, the posterior ends of one or two ribs and the corresponding transverse processes of the vertebrae are excised and the cold abscess evacuated. It is indicated in children and also in cases of tense abscess.

Radical Debridement and Arthrodesis

This surgery prevents progress of the kyphosis and heals the disease early.

Laminectomy

In rare cases where the disease presents as a spinal (cord) tumour syndrome and in those where paraplegia has resulted from posterior spinal disease, laminectomy is done and the granuloma is removed.

Anterior Decompression and Spinal Fusion (Hong Kong Operation)

Through a standard thoracotomy, the abscess is evacuated and debridement done. The diseased vertebral bodies are excised (vertebrectomy) and the cord decompressed. Autologous bone grafts are placed between the vertebral bodies to promote anterior spinal fusion.

The prognosis of spinal tuberculosis is given in Table 5.6.

A summary of management of tuberculous paraplegia/quadriplegia is given in Figure 5.12

TUBERCULOSIS OF THE HIP JOINT

Tuberculosis of the hip joint constitutes about 8% of all cases of osteoarticular tuberculosis and is the most common site for involvement by tuberculosis second only to the tuberculosis of the spine. This also occurs most commonly in children, the highest incidence being between 5 and 15 years of age.

Pathology

Infection of the hip is secondary to some primary focus in either the lungs or the mediastinal nodes

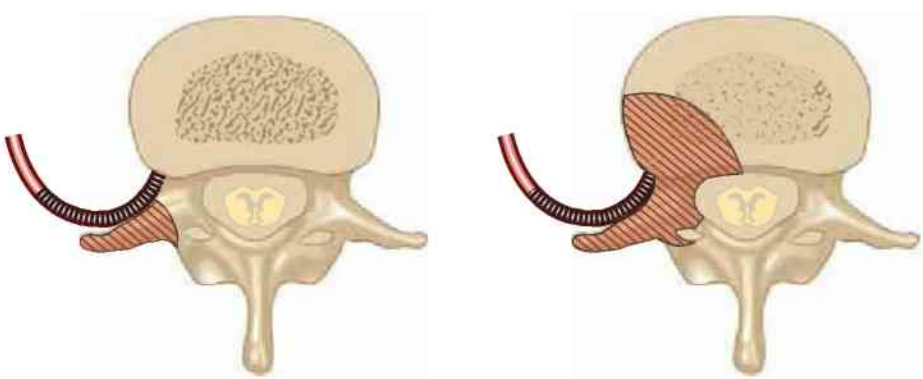


FIGURE 5.11 Diagrammatic representation of a vertebra showing operative procedures for Pott's paraplegia. (a) Costotransversectomy and (b) anterolateral decompression.

TABLE 5.6 Prognosis of Spinal Tuberculosis

Cord Involvement	Better Prognosis	Poor Prognosis
Degree	Partial	Complete (Stage IV)
Duration	Shorter	Longer (>12 months)
Type	Early onset	Late onset
Progression	Slow	Rapid
Age	Younger	Older
Neural condition	Good	Poor
Vertebral disease	Active	Healed
Kyphotic deformity	<60°	>60°
Cord on MRI	Normal	Myelomalacia
Preoperative	Wet lesion	Dry lesion

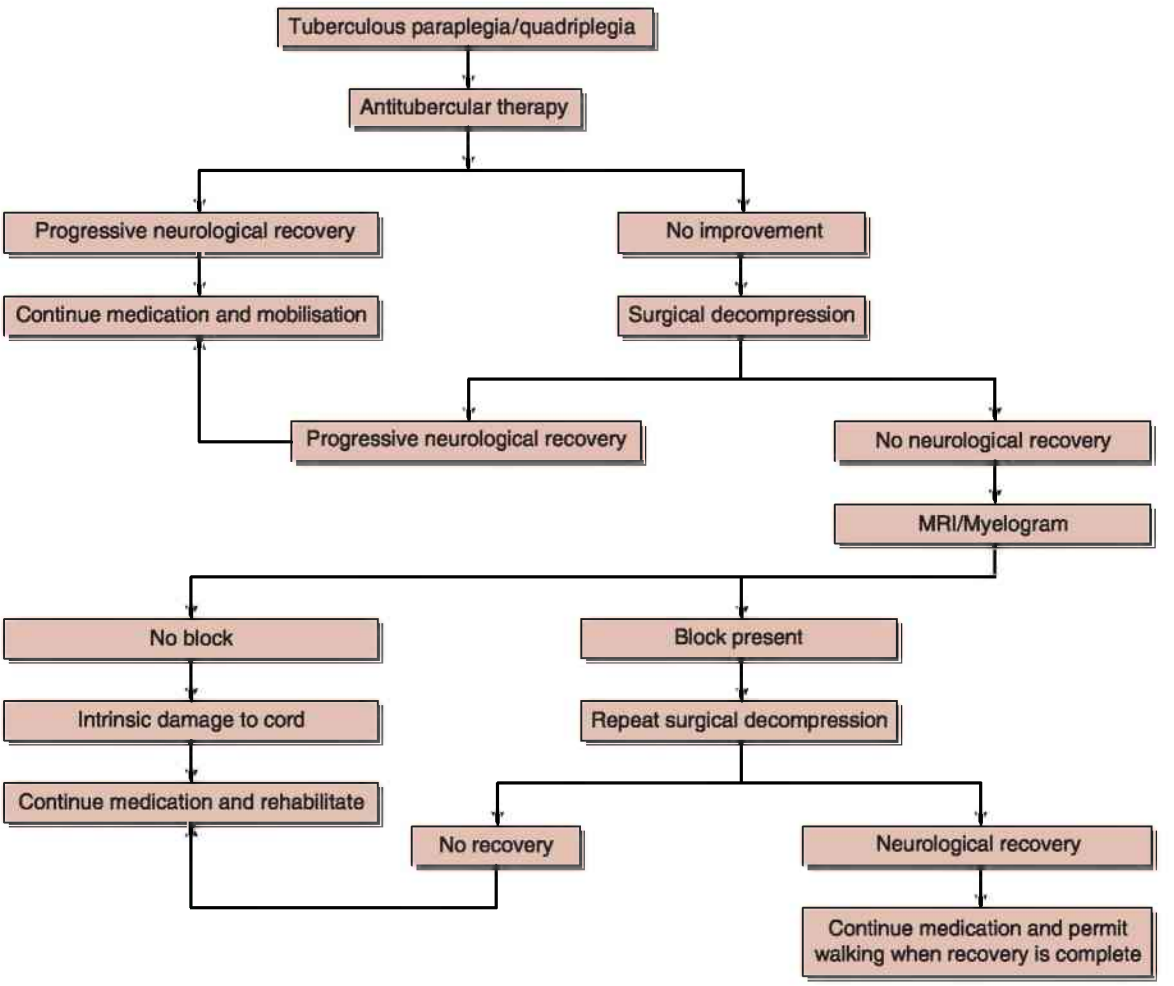


FIGURE 5.12 Management of tuberculous paraplegia/quadriplegia.

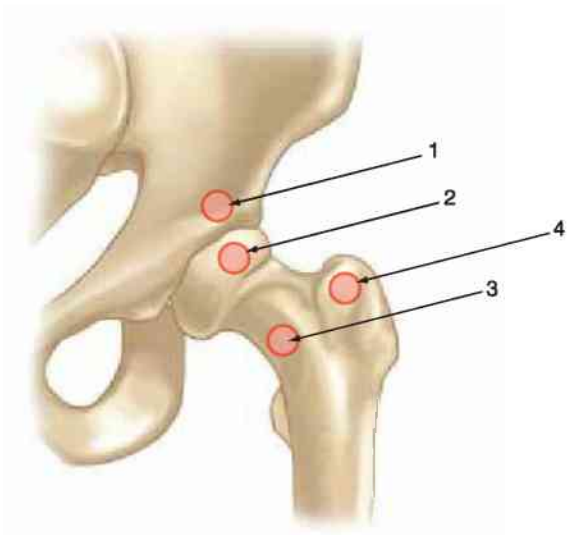


FIGURE 5.13 Diagrammatic representation showing sites of lesion of tuberculosis around the hip joint.

and spread to the hip by the blood stream. In the earliest stages, the disease is synovial but soon a bony focus appears.

The common sites in the hip region are as follows (Fig. 5.13):

1. Acetabular roof
2. Head of the femur
3. Neck of the femur
4. Greater trochanter
5. Bursa over the greater trochanter (not shown in the figure)

The initial lesion is a tubercle which enlarges with necrosis and caseation. At this stage, there is hyperaemia and thickening of the synovial membrane with effusion. It should be noted that initially the disease is synovial, but soon bony involvement occurs. The tuberculous granulation tissue spreads over the articular cartilage, erodes and destroys it. Another layer of granulation tissue spreads deep to the articular cartilage separating it from the underlying cancellous bone. The destructive process soon involves the adjacent articular surfaces. The destruction of the acetabular roof permits an upward displacement of the femoral head. A new depression forms in the dorsum ilium on which the head of the femur lodges. This is called the '*travelling acetabulum*'. Multiple cavitations in femoral head and acetabulum are typical of tuberculosis.

A cold abscess invariably forms inside the joint and the pus escapes through the weak inferior capsule of the joint. It then tracks along blood vessels or nerves to reach the surface. Tracking forwards, the abscess often presents in the femoral triangle. The pus travels along the circumflex femoral vessels and points on the outer aspect of the joint.

Occasionally, the floor of the acetabulum gets destroyed and the cold abscess becomes intrapelvic. Depending on whether the intrapelvic spread is above or below the attachment of the levator ani muscle, the abscess tracks upwards to point in the inguinal region or downwards into the ischiorectal fossa. The final stages of the disease are marked by fibrosis of the hip, leading to fibrous ankylosis.

Clinical Features

The patient is usually a child or an adolescent and the disease is of insidious onset. Pain and swelling in the region of the hip and limping are the usual presenting symptoms. Sometimes the child complains of pain in the knee. This is *referred pain* and is often misleading. There will be constitutional symptoms such as loss of appetite, loss of weight, low-grade fever and a sense of tiredness. There may be discharging sinus and puckered scars over and around the involved hip. Wasting of thigh/gluteal muscles may be present. The gait is usually stable, unaided, painful or antalgic, and stiff hip gait.

For purposes of description, the clinical picture of tuberculous disease of the hip is described as showing three stages (Table 5.7). These stages occur when the disease is allowed to progress without any treatment.

1. *Stage I (synovitis) (stage of apparent lengthening):* This is the stage when the disease is a synovitis with effusion into the cavity. The hip joint assumes the position of flexion, abduction and external rotation. There is a pelvic tilt downwards which causes an apparent lengthening of the affected limb. There is an increased lordosis in the

TABLE 5.7 Clinical Stages of Tuberculosis of the Hip Joint

Stage	Attitude	X-Rays	Treatment
Stage I: Stage of synovitis (stage of apparent lengthening)	Involvement of the synovial membrane with effusion makes the hip assume an attitude of flexion, abduction and external rotation (FAbEr) There is a pelvic tilt downwards, resulting in apparent lengthening of the involved limb	No specific radiological sign seen X-rays show generalised rarefaction of bones Joint space appears widened	Deformity (flexion, abduction and external rotation) is corrected by traction in a Thomas splint over 2–3 weeks Later, the involved hip is immobilised in the functional position (15° abduction and neutral rotation). This is continued till the disease gets controlled
Stage II: Stage of arthritis (stage of apparent shortening)	If the progression of the disease continues, it involves the articular surfaces and causes destruction Typically, this is associated with adductor muscle spasm, making the hip to assume an attitude of flexion, adduction and internal rotation (FAdIr) This causes an upward tilt of the pelvis in the affected side and hence apparent shortening of the involved limb	Erosion of articular surfaces seen Joint space appears narrowed	Hip is immobilised in the functional position with traction. This helps in overcoming adductors spasm and hence prevents articular surface erosion The end result may be a hip with a good range of movement after a period of physiotherapy or an immobile fibrous ankylosed hip
Stage III: Stage of pathological dislocation (stage of true shortening)	Further, if left untreated, the disease process destroys much of the acetabulum and leads to dislocation of the hip joint Hence, there is a real shortening with hip in flexion, adduction and internal rotation (FAdIr)	Destruction of the femoral head Travelling acetabulum with dislocation (pseudo-acetabulum on the ilium) of the hip Break in Shenton's line Acetabulum may get deepened with deformed femoral head shifted medially → pestle and mortar appearance	Surgical management (refer to Table 5.8)

lumbar spine. There are also other local signs of warmth, tenderness, muscle spasm and painful limitation of all movements of the joint.

At this stage, the condition will have to be differentiated from traumatic synovitis of hip, rheumatic arthritis, non-specific synovitis of hip, early Perthes disease and deep iliac adenitis with psoas spasm.

2. *Stage II (arthritis) (stage of apparent shortening):* When the disease is untreated and the patient is bedridden for some time, the destructive process spreads to the articular surfaces. In this stage, the spasm of the abductors predominates and the limb assumes the position of flexion, adduction and internal rotation. The adduction at the hip joint causes an upward tilt of the pelvis

on that side and this produces an apparent shortening of the leg. At this stage, there is marked wasting of the gluteal muscle and the local signs become more prominent. Cold abscesses appear and may point either on the lateral aspect or anteriorly.

3. *Stage III (pathological dislocation) (stage of true shortening):* When the condition is neglected, the destruction spreads in the acetabulum and pathological dislocation of the hip joint occurs. The position of the adduction, flexion and internal rotation gets exaggerated due to the dislocation. There is real shortening of the limb. The cold abscess bursts and there are sinuses discharging thin pus. The sinuses show thin undermined edges with pigmentation in the surrounding area. When secondary pyogenic infection supervenes, there is thick purulent discharge from the sinus.

Radiological Features

- *Stage I:* At this stage, radiographs show only generalised rarefaction of bones. No bony focus will be seen. The joint space appears widened due to the effusion.
- *Stage II:* Radiographs at this stage show erosion of the articular surface and narrowing of the joint space (Fig. 5.14).
- *Stage III:* Radiographs show destruction of the head of the femur, travelling acetabulum with dislocation of the hip and a break in Shenton's line (Fig. 5.15).

Shanmugasundaram (Chennai) has classified tuberculosis hip into the following clinicoradiological types:

- Normal hip type
- Travelling acetabulum type
- Dislocation hip type
- Perthes type
- Protrusio acetabular type
- Atrophic hip type
- Mortar and pestle type (Fig. 5.16)

This is of prognostic value and helps in the choice of management.

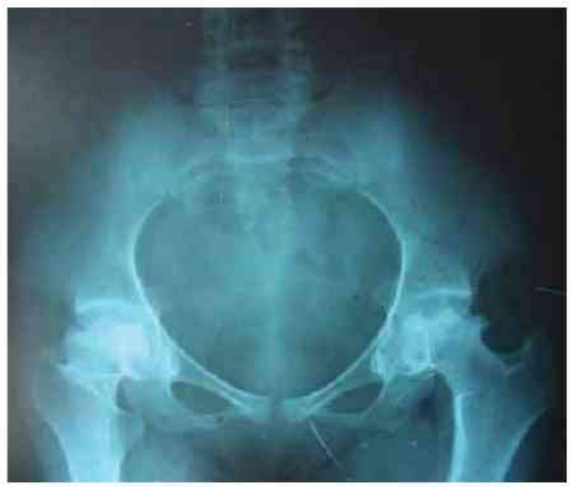


FIGURE 5.14 X-ray anteroposterior view of the pelvis with both hips showing tuberculosis hip Stage II in the right hip—articular surface erosion with joint space narrowing—and tuberculosis hip Stage III in the left hip—bony destruction of the head of femur and acetabulum.

Management

The principles of management are to

- eliminate the infection,
- control the disease progression,
- manage the sequelae,
- rehabilitate the patient.

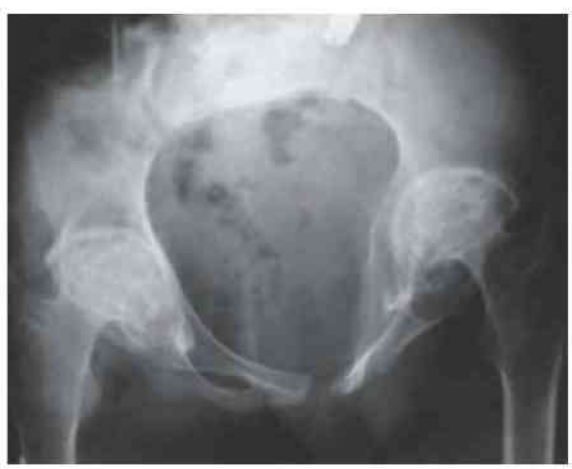


FIGURE 5.15 Anteroposterior view of pelvis showing bilateral arthritis of the hip—tuberculosis hip Stage III. The left hip shows travelling acetabulum (pseudo-acetabulum onileum). There is uniform articular erosion and narrowing of joint space.

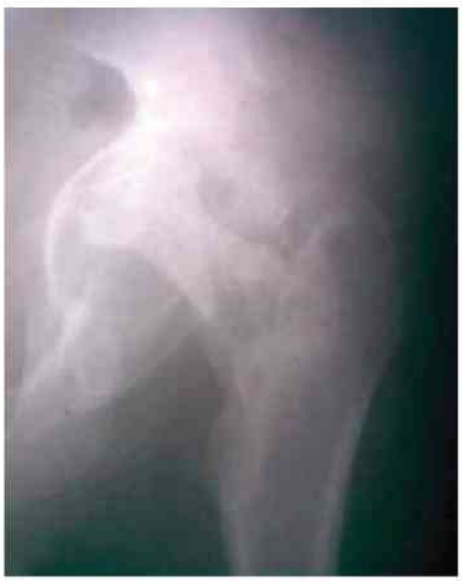


FIGURE 5.16 Anteroposterior X-ray of the left hip showing pestle and mortar type of tuberculosis (deepened acetabulum with a deformed femoral head with medial shifting).

Conservative Treatment

The conservative measures are adopted to improve the general condition of the patient. These measures include

- high nutritious diet,
- hygienic environment.

Antituberculous chemotherapy is started. The method of local treatment depends on the stage of the disease.

In Stage I, the deformity of flexion abduction and external rotation is corrected by gradual continuous skin traction in a Thomas splint over a period of 2–3 weeks. When the deformity is corrected, the hip is immobilised in the position of function in 15° abduction and neutral rotation. This immobilisation with traction is continued till the disease gets controlled.

In Stage II, the hip is immobilised with skin traction in the position of function. The traction is meant to overcome the muscle spasm and prevent erosion of the articular surfaces by lessening their contact.

When the disease is stabilised, the traction is discarded and the hip is immobilised in a full plaster spica. When the stage of quiescence is reached, the plaster is removed, the hip is mobilised in bed. At this stage, the end result may be a hip with a good range of movement or in more severe cases, fibrous ankylosis.

Surgical Treatment

1. Early surgery
   - *Surgical curettage:* When the disease is stabilised, the presence of a well-localised cavity in the neck of the femur or the acetabulum is an indication for surgical curettage.
   - *Surgical debridement of the joint:* When there is progressive destruction of the articular surfaces, surgical debridement helps in eradication of the disease and obtain a mobile joint.
2. Management of sequelae: Further management depends on the end result (sequelae) reached which may be as follows:
   - *Sound ankylosis in good position:* The hip in this position needs only protection from sudden strains and weight bearing for a short period. It can be left with little protection to hip during the initial period of weight bearing.
   - *Sound ankylosis in bad position:* A hip in this position needs a corrective *subtrochanteric osteotomy* but the joint itself is left alone.
   - *Unsound ankylosis in good position:* An unsound fibrous ankylosis is a painful condition and leads to increasing deformity on walking. There is also a risk of reactivation. Hence, it needs active intervention to convert it into a sound ankylosis. This is done by arthrodesis (fusion) of the joint by surgery.
   - *Unsound ankylosis in bad position:* Such a hip needs correction of the deformity as well as arthrodesis of the joint.
3. Surgical procedures: Table 5.8 gives an overview of various surgical procedures that can be performed to manage the sequelae arising due to tuberculosis of hip joint:
   - *Excision arthroplasty (Girdlestone):* Adult patients with ankylosed hip joint have difficulties in squatting on the ground

TABLE 5.8 Surgical Procedures for Management of the Tuberculosis Hip

Post-Operative Outcome	Arthrodesis	Excision Arthroplasty	Replacement Arthroplasty
Stability of hip after surgery	Stable hip	Unstable hip	Stable hip
Squatting	Not possible	Possible	Not possible
Pain after surgery	Painless	Painless	Painless

for social purposes. These patients can be helped by excision arthroplasty, wherein the head and neck of femur are excised to create a pseudo-joint.

Differential Diagnosis

- *Low-grade septic arthritis:* Constitutional symptoms may be present.
- *Rheumatoid arthritis:* Joint space is uniformly narrowed.
- *Developmental dislocation of hip:* Usually diagnosed at an earlier age. The child presents with a painless limp with telescopy test being positive.
- *Congenital coxa vara:* The patient presents with painless limp with limitation of abduction and internal rotation.
- *Perthes disease:* It usually occurs in children of an age group of 4–8 years. The child usually presents with painful limp, little limitation of abduction and internal rotation. Joint space is widened in X-ray films and the X-ray changes are out of proportion to the clinical findings.
- *Psoas abscess:* It presents with flexion deformity of the hip and all movements except extension are painful.

TUBERCULOSIS OF THE KNEE JOINT

The knee is the third common site for tuberculous infection in the skeletal system. The incidence is high in children and young adults as in other forms of tuberculosis. The disease process in knee joint may be purely synovial, and such a condition is not so common in hip.

Pathology

The lesion in the knee can be (1) synovial, (2) osseous, involving the articular surface, and (3) extra-articular osseous focus. The knee is one place where, in a good percentage of cases, the lesion starts and remains purely synovial. The infection then spreads from the synovium as a pannus over the articular cartilage and destroys it. Effusion and pus formation may occur within the joint and cause distension of the joint. Further damage to the joint surface and ligaments produces subluxation of the tibia. Eventually, the pus tracks outside to form a cold abscess and then a discharging sinus. In the final stages of the disease, *fibrous ankylosis* of the knee joint usually occurs.

Clinical Features

The onset is insidious. The patient presents with pain and swelling in the knee joint for a considerable period of time without any preceding history of trauma. The patient also has some constitutional symptoms.

On examination, the knee is swollen with prominence of the suprapatellar pouch and filling of the fossae on either side of the quadriceps tendon. There is wasting of the thigh and calf muscles. On palpation, there is warmth and patellar tap due to effusion into the joint. There is synovial thickening, felt best at the lateral and medial edges of the suprapatellar pouch. There is tenderness along the medial arid lateral joint line. Movements elicit marked spasm of the muscles, and there is painful limitation of both flexion and extension.

- The joint is warm and tender.
- Cold abscess may present as a swelling around the joint.
- Sinuses, either discharging or healed with puckering of the skin, may be present.

In advanced cases, the tibia is subluxated posteriorly, displaced laterally and rotated externally. This

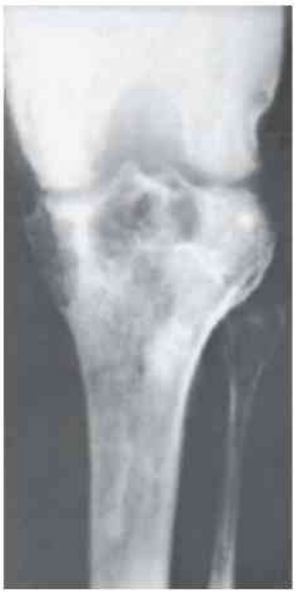


FIGURE 5.17 Anteroposterior X-ray of the knee showing articular erosion and joint space narrowing as seen in chronic tuberculosis of the knee.

is called *triple dislocation*. This is due to the spasm of the hamstring, biceps and the iliotibial band.

Radiological Features

In a typical case, radiograph shows generalised rarefaction and erosion of the articular surface (Fig. 5.17). There is also narrowing of the joint space. Late cases may show triple dislocation.

Investigations

In the knee, synovial biopsy is a very useful investigation to confirm the diagnosis.

Differential Diagnosis

The condition will have to be differentiated from monoarticular rheumatoid arthritis, chronic recurrent traumatic synovitis and subacute infective synovitis.

Management

The principles of management include

- improving the general condition of the patient,
- drug therapy with antituberculous chemotherapy.

Conservative

The knee joint is immobilised with below-knee skin traction in a Thomas splint and antituberculous chemotherapy started. When the lesion is stabilised, the knee is protected from strain and immobilised in a plaster of Paris cast. When the lesion is quiescent, active movements of the knee are started.

Surgical Treatment

- *Synovectomy:* It is a useful procedure to remove the focus when the synovial thickening is gross and symptoms are persisting. Debridement and curettage of a localised focus can be done in some cases.
- *Arthrodesis:* When there is destruction of the articular surface, healing is by fibrous ankylosis which can lead to increasing deformity and reactivation on weight bearing. In an adult, when there is marked destruction of the articular surfaces or fibrous unsound ankylosis, arthrodesis (fusion) of the joint is indicated (Fig. 5.18).

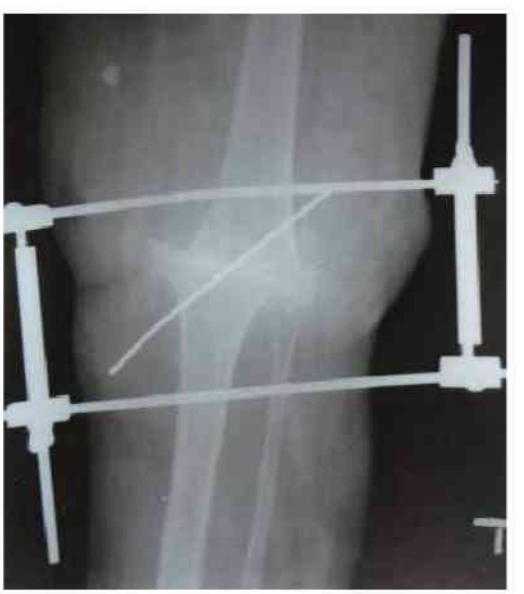


FIGURE 5.18 X-ray of knee showing knee arthrodesis using Charnley's compression arthrodesis device.

TUBERCULOSIS OF OTHER JOINTS

The following joints are discussed in this category:

- Sacroiliac joint
- Tarsal joint
- Shoulder joint
- Elbow joint
- Wrist joint

Tuberculosis of the Sacroiliac Joint

This is more common in women and presents as chronic pain and tenderness over the joint. Radiograph shows erosion of the joint line (Fig. 5.19).

Management

The treatment of this is on the same lines as that for tuberculosis of the spine. When the lesion is quiescent, a sacroiliac support is prescribed for some months. Persistent pain due to unsound healing will require fusion of the joint. Rest is given to the joint with a sacroiliac support and antituberculous chemotherapy.

Tuberculosis of Tarsal Joints

Tuberculosis of the tarsal bones involves the midtarsal joints and forms superficial cold abscesses and sinuses. The disease usually manifests as superficial cold abscesses and sinuses. There is severe pain and the soft boggy swelling in the dorsum of the foot which differentiates this condition from actinomycosis of the foot. Treatment is by immobilisation in a below-knee plaster cast and antituberculous chemotherapy till the lesion is quiescent.

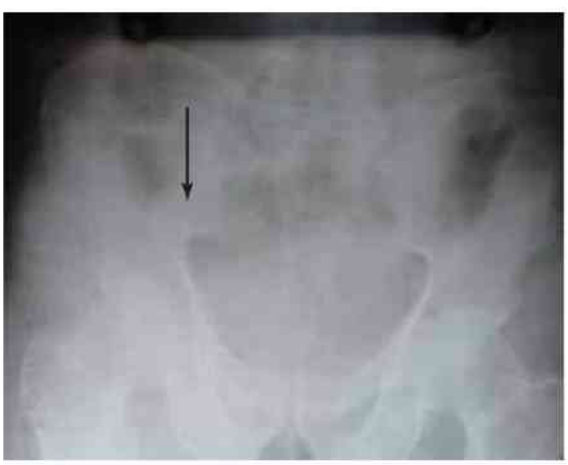


FIGURE 5.19 X-ray of the anteroposterior view of the pelvis showing tuberculosis of the right sacroiliac joint with articular erosion and reduced joint space.

Tuberculosis of the Shoulder Joint

Shoulder tuberculosis in this joint is characterised by a dry type of lesion 'Caries sicca'. Cold abscess formation is very rare. It is treated by rest and immobilisation is by cuff and collar with a pad in the axilla and antituberculous chemotherapy.

Tuberculosis of the Elbow Joint

Tuberculosis in the elbow usually starts as a focus in the olecranon or the lower end of the humerus and soon involves the joint. The elbow is immobilised in an above-elbow plaster cast and full course of antituberculous drugs given. Involvement of the joint may result in ankylosis fibrous. When the lesion is quiescent with ankylosis, excision arthroplasty of the joint may be done.

Tuberculosis of the Wrist Joint *(Fig. 5.20)*

The patient presents with swelling around the wrist joint and limitation of movements of wrist. Abscess formation is common and there may be involvement of flexor tendon sheaths in the palm. The abscess presents in the palm and may extend above the wrist deep to the transverse carpal ligament. This swelling cross-fluctuates and is called *compound palmar ganglion*. This is one of the causes of carpal tunnel syndrome. Treatment is by immobilising the wrist in functional position in a below-elbow plaster cast and antituberculous chemotherapy. Arthrodesis of the joint is done at a later stage when the healing is unsound.

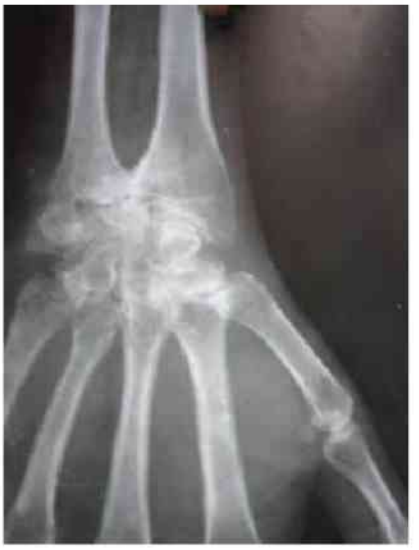


FIGURE 5.20 X-ray of the right wrist showing tuberculosis lesion of the wrist with articular joint erosions and reduced joint space.

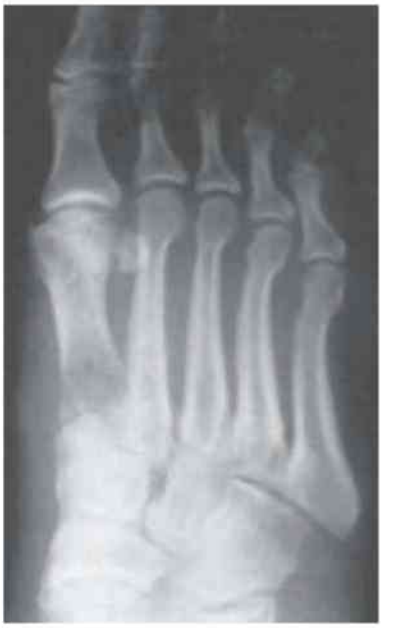


FIGURE 5.21 X-ray of the foot showing tuberculosis of the first metatarsal bone.

TUBERCULOSIS OF BONE (Tuberculous Osteomyelitis)

Tuberculosis of bone as an isolated infection of the bone occurs in the phalanges, metacarpal and metatarsal bones (Fig. 5.21), short long bones, carpal and tarsal bones, ribs and pubis.

Tuberculous Dactylitis

Tuberculous dactylitis occurs in children and young adults. The infection starts in the shaft of the phalanx and causes erosion and gradual destruction of the bone. There is subperiosteal new bone formation and thickening of the bone.

This phenomenon is peculiar to the tuberculous infection in the short long bones. The surrounding soft tissues also swell up and cold abscess often forms and bursts to form chronic sinuses. The patient presents with a painful spindle-shaped swelling of the finger which is called *spina ventosa*.

Tuberculosis of the Rib

In the rib, tuberculous infection forms a localised destructive focus with a cold abscess. Excision of the affected segment of the rib is the treatment.

BCG Osteomyelitis

BCG osteomyelitis is a very rare complication of BCG vaccination. It occurs in children from a few months to a few years after the vaccination. The lesions are localised to the metaphysis or epiphysis of long bones.

Poncet's Tubercular Rheumatism

Poncet described cases of polyarthritis resembling rheumatoid occurring in patients with tuberculosis. When the tuberculous focus is treated, the polyarthritis symptoms disappear.

KEY POINTS

- Pathologically, two types of microscopic lesions are recognised in tuberculosis: caseating exudative type and proliferative type.
- Spine is the commonest site of bone and joint tuberculosis.
- The most common level involved in tuberculosis of spine is the thoracolumbar level.
- Paradiscal region is the most common site of pathologic lesion in tuberculosis of spine.
- In late stages, anterior wedging leads to angular kyphosis called gibbus.
- Classical symptoms of tuberculosis of spine are back pain, rigidity, deformity, cold abscess and paraplegia.
- Reduction of disc space is the earliest radiological sign of spinal tuberculosis.
- Modalities of treatment are rest, antitubercular treatment, gradual mobilisation and aspiration of abscess.
- The common sites of tuberculosis in the hip region are acetabular roof, head of the femur, neck of the femur, greater trochanter and bursa over the greater trochanter.
- Travelling acetabulum is a classical sign of the tuberculosis hip.
- Fibrous ankylosis occurs in the fi nal stages of the tuberculosis hip.
- Three stages of tuberculosis disease of the hip are (1) synovitis, (2) arthritis and (3) pathological dislocation.
- The disease process in knee joint may be purely synovial, and such a condition is not so common in hip.
- In advanced stages of knee joint tuberculosis, the tibia is subluxated posteriorly, displaced laterally and rotated externally. This is called 'triple dislocation'.
- Tuberculosis of tarsal bones should not be confused with mycetoma.

MULTIPLE CHOICE QUESTIONS

1. The commonest site of skeletal tuberculosis is
   a. Tibia
   b. Radius
   c. Humerus
   d. Vertebrae
2. The earliest manifestation of spinal tuberculosis is
   a. Cold abscess
   b. Paraplegia
   c. Gibbus
   d. Muscle spasm
3. The commonest site of tuberculosis spine is
   a. C8-T2
   b. T2-T6
   c. T10-L1
   d. L1-L4
4. The early feature of Pott's paraplegia is
   a. Flexor spasm
   b. Increased tendon jerk
   c. Ankle clonus
   d. Sensory loss
5. The most common site of Pott's spine is
   a. Paradiscal
   b. Posterior
   c. Anterior
   d. Central
6. Spina ventosa is caused by
   a. Tuberculosis
   b. Leprosy
   c. Brucellosis
   d. Sickle cell anaemia

CHAPTER 6

Metabolic Bone Diseases

COMPOSITION OF BONE

Bone is formed by organic and inorganic material along with water.

Organic Components

Organic components form one-third (35%) of the total dry weight of the human bones. Except for some mucoproteins, glycoproteins and phospholipids, 95% of organic compounds are made of collagen.

Inorganic Components

Inorganic components form about two-thirds (65%) of the dry weight of the bones. Calcium and phosphate form the major portion along with magnesium, sodium and potassium. Majority of the calcium and phosphate are in crystalline–hydroxyapatite form.

CALCIUM METABOLISM AND BONE TISSUE

More than 99% of calcium is stored in the bone, in human body. Hence, changes in calcium metabolism seriously affect the body's osseous tissue. There are some regulatory mechanisms maintaining the calcium level in the body and also in the bone. The main regulators of calcium metabolism are as follows:

- Parathyroid hormone
- Vitamin D
- Calcitonin

These hormones maintain the body calcium level by

- increasing resorption of calcium from bone,
- increasing absorption of calcium,
- decreasing excretion of calcium.

Effects of Parathyroid Hormone

Parathormone increases reabsorption of calcium from the bone and in the kidney, and so the calcium levels in the blood increase.

Phosphate excretion increases through the kidney and thereby phosphate levels decrease.

Effects of Vitamin D

Vitamin D increases serum calcium and decreases serum phosphate. It also increases the alimentary absorption of calcium and phosphate and increases the resorption of calcium from bones.

Effects of Calcitonin

Calcitonin decreases serum calcium and increases serum phosphate. It decreases urinary excretion of calcium and decreases the resorption of calcium from bone.

Abnormalities in the levels of calcium, phosphorus, magnesium or vitamin D lead to clinical disorders. These disorders are reversible once the underlying defect has been treated. Generalised diseases of bones include skeletal disorders in metabolic, endocrine and reticuloendothelial diseases and diseases of bone marrow.

METABOLIC DISEASES

Metabolic disorders could be divided into four major groups.

1. Osteopenia: Due to inadequate osteoid synthesis
   - *Dietary:* Protein deficiency (e.g. malabsorption syndrome and chronic infection)
   - *Disuse:* Deficient muscular stress (e.g. muscular paralysis of poliomyelitis and immobilisation of the limb in fractures)
2. Disorders due to inadequate mineralisation of the osteoid
   - Rickets
   - Osteomalacia
3. Osteoporosis: Due to decreased osteoid synthesis and increased osteolysis
   - Senile osteoporosis
   - Post-menopausal osteoporosis
4. Osteosclerosis: Characterised by an increase in bone mass (e.g. Paget's disease, osteopetrosis)

The skeletal system stores 98% of the calcium in the human body, and hence calcium metabolism has a major influence on the structure and growth of bone. Bone formation is an orderly process in which the inorganic mineral is deposited as calcium hydroxyapatite in relation to the organic protein matrix.

HISTOLOGICAL FEATURES OF BONE GROWTH

Calcium deposition occurs at the epiphysiodiaphyseal junction or the growth plate. The normal process of growth at the epiphyseal plate could be understood by studying histological sections of the area from the epiphyseal side of the growth plate to the diaphyseal side.

In this area, the following appearances could be recognised denoting the various stages of longitudinal growth activity from the cartilage to the bone:

- Proliferation and maturation of the young chondroblasts
- Columniation of the maturing cartilage cells, with formation of chondroid in between the columns
- Provisional calcification of the chondroid tissue
- Invasion of spaces by *metaphyseal capillaries* and *osteoblasts* and absorption of the calcified chondroid
- *Osteoid formation* over the remnants of calcified cartilage
- Mineralisation of the osteoid into trabeculae of immature (spongy) bone in the metaphyseal area
- Remodelling into mature (lamellar) bone

Osteopenia is defined as the reduction in the bone mass due to diminished osteoid synthesis. Osteomalacia is the condition in adults where the osteoid formation is normal but there is a deficiency in the mineralisation of the osteoid. It is called rickets when it occurs in childhood. Osteoporosis is the reduction of bone mass per unit of bone volume due to increased bone lysis. There is normal mineralisation.

RICKETS

Rickets is a disease of growing bones in childhood. It is a classic example of a metabolic disorder, wherein the osteoid formation in the child's growing bone is normal but its mineralisation is defective. It is due to defective vitamin D metabolism or its deficiency and results in developmental deformities due to a decrease in bone strength. Vitamin D3 (cholecalciferol) is as such inactive in the body and it should be hydroxylated twice, that is, 25-hydroxylation in liver and 1-hydroxylation

in kidney thereby forming active 1, 25 dihydroxy chole calciferol.

Classification

Rickets can be classified as follows:

1. Type I
   - Due to vitamin D deficiency
   - Decreased intake, also called 'nutritional rickets'
   - Decreased absorption, also called 'celiac rickets' (intestinal rickets)
   - Due to defective vitamin D metabolism
   - Hepatic failure
   - Renal failure, included in the category of 'renal rickets'
   - Renal osteodystrophy, included in the category of 'renal rickets'
2. Type II
   - Due to decreased phosphate intake
   - Due to decreased phosphate absorption
   - Increased phosphate excretion

Rickets can also be classified as

- Nutritional rickets
- Celiac rickets (intestinal rickets)
- Renal rickets

The most common type is the nutritional rickets

Nutritional Rickets

The commonest form of rickets in developing countries is nutritional rickets. This is due to vitamin D deficiency in the diet and occurs in children below 4 years.

Pathology

A deficient intake of vitamin D causes defective absorption of calcium from the gut and lowering of calcium and phosphates in the tissue fluids. There is a disorderly proliferation of the cartilage cells and lack of columniation in the zone of proliferation. The irregular extension of these cells into the metaphysis results in broadening of the metaphysis in the transverse axis and widening of the epiphysis in the long axis of the bone. There is also poor deposition of calcium in the zone of calcification and poor mineralisation of the spongy bone. The bone thus becomes soft and pliable and deforms easily on weight bearing or other stresses.

TABLE 6.1. Clinical Features of Rickets and its Respective Pathology

Clinical Feature	Pathology of the Clinical Feature
Craniotabes	Earliest manifestation of rickets Skull bones become soft
Frontal bossing	Common in frontal and parietal bones
Pigeon chest	Protrusion of the chest and widening of the spaces between the ribs cause a pigeon chest-like appearance
Rachitic rosary	Prominent costochondral junctions that form a bead-like prominence
Harrison's sulcus	Horizontal depression along the lower chest
Hypotonia	Due to hypocalcaemia
Knock knees or bow legs	These are due to softening of long bones that easily deform on weight bearing causing *genu varum* (bow legs) or *genu valgum* (knock knees)
Kyphosis	Exaggeration of the normal curvatures occurs, resulting in kyphotic deformity of the spine

Clinical Features *(Table 6.1)*

Clinically, the condition could present in either the florid stage or the healing or healed stage.

In the florid stage, the child seems to be irritable, sweats excessively and is stunted in growth. The fontanelle remain unclosed even after 2 years of age, and there is bossing of the frontal and parietal bones. This is called hot cross bun appearance (craniotabes).

The chest shows a pigeon chest deformity. There is beading at the costochondral junction called rickety rosary. Harrison's sulcus is a groove-like deformity in the lower chest due to the muscular pull of the diaphragm. The abdomen is protuberant (Fig. 6.1).

FIGURE 6.1 Clinical photo of child with rickets. Note bilateral genu varum.

Skeletal Deformities

There is broadening at the epiphyseal region of the wrist, knees and ankles (Fig. 6.2). There will be deformities in the long bones of the upper and lower limbs if the child has crawled or walked during the active stage of the disease. There will be exaggeration of the normal curvatures in long bones. At the knees, there may be genu valgum or genu varum. At the hip, there may be clinical evidence of bilateral coxa vara. The spine will show a smooth mobile kyphosis, which is marked when the child is made to sit.

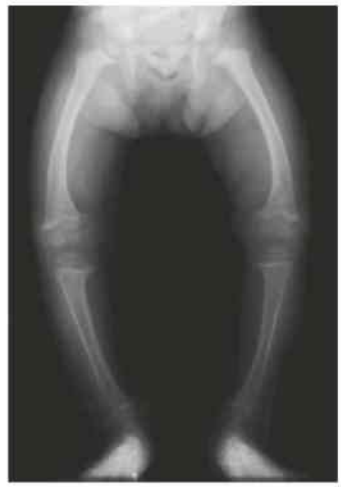
Biochemical Features

The serum calcium and phosphate levels are low. The alkaline phosphatase level is high during the active stage of the disease:

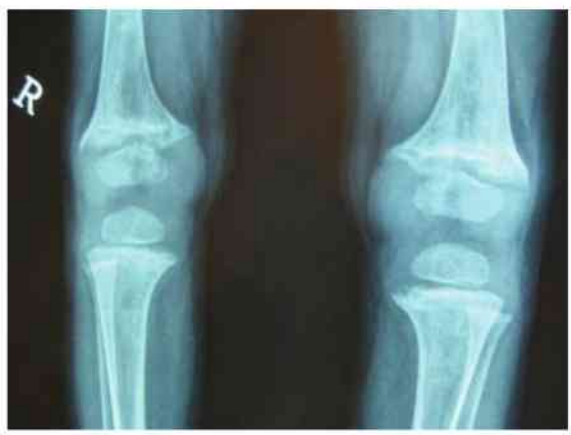
- *Serum calcium:* Normal to decreased
- *Serum phosphate:* Decreased
- *Serum alkaline phosphatase:* Increased

Radiological Features

In the florid stage, the width of the epiphyseal plate is increased markedly and its edges are fluffy and irregular. There is cupping of the metaphyseal margin. The cortex of the diaphysis is thinned and hazy and the whole bone shows rarefaction (Figs 6.2a and b).



(a)



(b)

FIGURE 6.2 (a) X-ray of both lower limbs in a rachitic child showing genu varum. (b) X-ray of both knees of a rachitic child. Note the cupping and splaying of metaphysis. *Courtesy:* (b) Dr Anand J Thakur.

In the healed stage, the long bones will show normal density with bowing of the shafts. The epiphyseal plate is again narrow with the zone of calcification seen as a dense line.

The radiological features seen in a rachitic child are as follows:

- Delayed appearance of epiphysis
- Widened epiphyseal plate
- Cupping and splaying of metaphysis
- Diaphyseal resorption

Treatment

Primary prevention of rickets in the child begins by better nutrition of the pregnant mother, followed by supply of vitamin D in the child's diet in the form of cod or shark liver oil. Bed rest, splints and high energy food are needed during early stages of deformities. In the florid stage of rickets, measures for the secondary prevention of deformities must be taken. The child should be prevented from bearing weight on the softened bones by the use of splints for the legs.

When the child has presented late with active rickets and deformities such as genu valgum or varum, surgical correction by osteotomy or osteoclasis is done along with vitamin supplementation. High dose of oral vitamin D will cause rapid healing of the affected bone. One or two doses might be needed, and there should be a maintenance dose in severe deficiency.

Celiac Rickets (Intestinal Rickets)

Celiac rickets is due to diminished absorption of calcium from the intestines in steatorrhoea, sprue, celiac disease and other malabsorption disorders. Gluten-induced enteropathy is also a cause of celiac rickets.

Renal Rickets

Renal rickets is due to various types of defects in the renal functions in children above 5 years. Many of these are hereditary and result in renal dwarfism:

1. Dysfunction of the whole kidney (glomeruli and tubules)
   - *Renal osteodystrophy:* This occurs in polycystic kidney, chronic nephritis, bilateral hydronephrosis, etc.
2. Dysfunction of the tubules
   - *Vitamin D-resistant rickets:* This is due to congenital inability of the tubules to reabsorb phosphates from the glomerular filtrate.
   - *Fanconi syndrome (cystinosis):* This is due to inability of the proximal tubules to reabsorb phosphates, glucose and amino acids.
   - *Renal tubular acidosis:* In tubular acidosis, there is defective reabsorption of bicarbonates (HCO3) from the proximal tubules. The excess HCO3 combines with calcium ion and is excreted in urine. The resulting lowered calcium in the blood causes secondary hyperparathyroidism which in turn causes osteoporosis.

Clinical Features

Clinically, the older child is seen with signs of rickets, deformity of legs and dwarfism. There is also evidence of renal malfunction. Generalised osteoporosis is prominent in radiographs in addition to the changes in the epiphyseal regions.

Treatment

It is mainly the medical management of the renal pathology.

OSTEOMALACIA

Osteomalacia is a disease of adults and is similar to rickets. Like rickets, osteomalacia occurs due to deficiency of vitamin D (Fig. 6.3). It may be due to dietary deficiency, malabsorption or lack of expose to sunlight (as in females using 'purdah').

Clinical Features

There will be non-specific symptoms such as bone tenderness and muscular weakness.

Radiological Feature

- *Looser's zone (Fig. 6.4a):* These are radiolucent zones present at the sites of stress. They are due to defective mineralisation.
- *Protrusio acetabuli (Fig. 6.4b):* This is seen in severe cases.

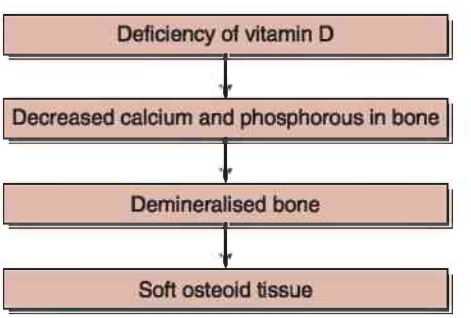
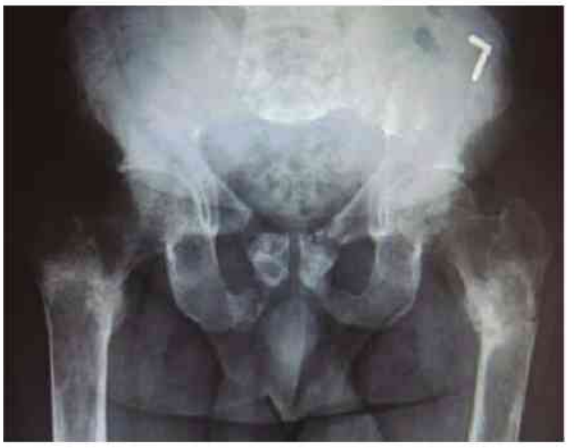
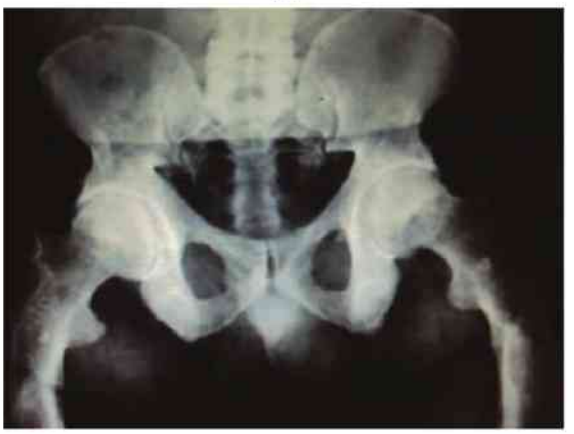


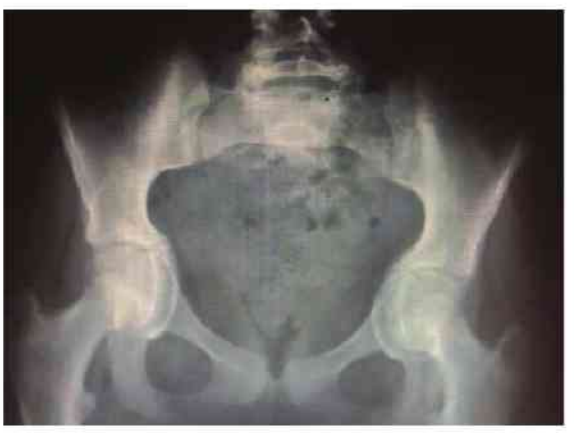
FIGURE 6.3 Aetiopathogenesis of osteomalacia.



(a)



(b)



(c)

FIGURE 6.4 (a) X-ray of pelvis with both hips showing looser's zones. (b) X-ray of pelvis with both hips showing pathological fractures. (c) X-ray of pelvis with both hips showing triradiate pelvis.

- *Triradiate pelvis (Fig. 6.4c):* This is due to softening of the pelvic bone.

Investigations

Blood investigations reveal the following status of calcium, phosphate and phosphatase:

- *Serum calcium:* Decreased
- *Serum phosphate:* Increased
- *Serum alkaline phosphatase:* Increased

Management

Higher doses of oral vitamin D or intramuscular infections can be given.

Increasing oral intake of vitamin D and calcium is helpful.

OSTEOPOROSIS

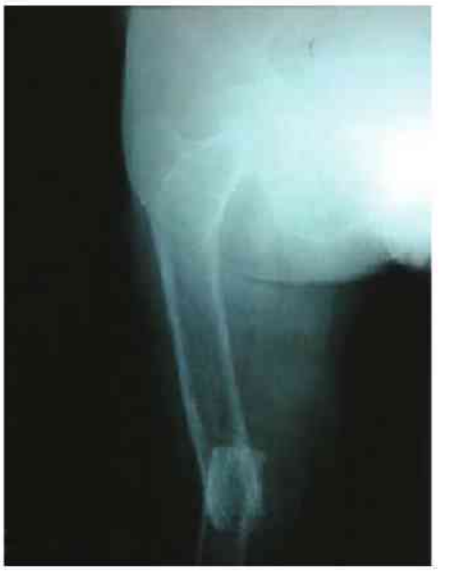
Osteoporosis is a pathological process of reduction of bone mass due to defective formation of the organic protein matrix. The mineralisation is normal. There is diffuse and abnormal diminution of bone mass per unit volume. Microscopically, there is reduction in the thickness of the cortex and a reduction in the number and size of the trabeculae. There is also a reduction in the number of osteoblasts present, and hence bone formation is severely affected.

It is classified as *primary* or *senile osteoporosis* (which is related to ageing and decreased gonodal hormones) and *secondary osteoporosis* (endocrine, drugs, neoplasm, etc.) (Table 6.2).

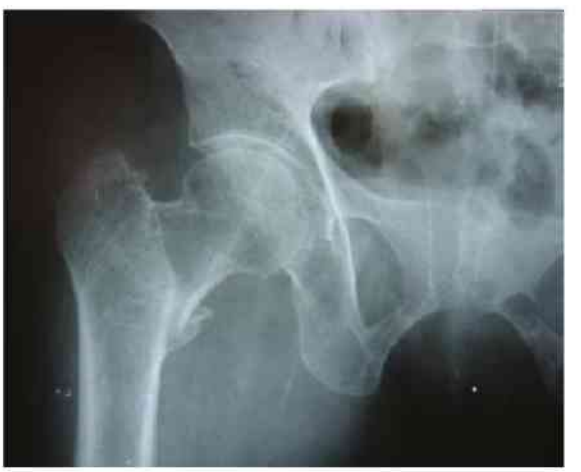
The clinical entity called 'senile osteoporosis' denotes a condition which is a combination of diminished osteoid formation and excessive osteolysis, with multiple aetiological factors. An endocrine imbalance is the usual initiating factor. Sedentary habits of old age with diminished muscular stress and diet fats contribute to the worsening of the demineralisation of the bone. In the females, the menopausal endocrine changes often cause a severe generalised osteoporosis at an age earlier than that in the senile variety. In extremely severe cases, osteomalacia may also be superadded to the condition.

TABLE 6.2 Types of Osteoporosis

Primary Osteoporosis	
Post-menopausal osteoporosis	Type I (high turnover)
Senile osteoporosis	Type II (low turnover)
Secondary Osteoporosis	
Endocrine causes	Cushing disease Hyperthyroidism Hypopituitarism Hypogonadism Diabetes mellitus
Chronic diseases	Rheumatoid arthritis Chronic liver diseases Chronic lung diseases
Drug administration	Corticosteroid Heparin Alcohol Smoking



(a)



(b)

FIGURE 6.5. (a) X-ray of femur showing pathological fracture due to osteoporosis. (b) X-ray of right hip anteroposterior view showing fracture neck of femur.

Clinical Features

The main clinical features are follows:

- Pain—radiates to the lower limbs
- Loss of weight—due to deformities of spine (kyphosis)
- Associated fractures—fractures of the neck of femur, distal tibial metaphysis, Colles' fracture, neck of the radius and vertebral body fractures are associated with osteoporosis (Fig. 6.5)

Clinically, the common presentation is the complaint of backache diffusely spread over the whole lumbar spine. The lumbar backache radiates to the lower limbs. The tenderness is also diffuse but may be localised if there is a pathological compression fracture. The patient gradually develops kyphosis in the dorsal spine (senile kyphosis). The patient may present with the fracture neck of femur (the commonest fracture in old age). Biochemically, the serum calcium, serum inorganic phosphate and alkaline phosphatase levels are normal.

INVESTIGATIONS

1. *Blood investigations:* Serum calcium, phosphate and alkaline phosphatase are within normal levels.
2. *Assessment of bone density:* These are recent advances in the diagnosis of osteoporosis. They are performed by either dual photoabsorptiometry or dual energy X-ray absorptiometry.

Bone density is also assessed by quantitative computerised tomographic scanning.

Radiological Features

Radiologically, there is extensive demineralisation with a coarse trabecular pattern in the vertebral bodies. The vertebral bodies are biconcave in shape. There may be one or more compression fractures of the vertebra, caused by minimal trauma. This has to be differentiated from multiple myelomatosis and secondary carcinomatosis. Long bones may also go for fractures following trivial trauma. X-ray of calcaneum and neck of femur is useful to evaluate the degree of osteoporosis by calcaneal index and Singh's index, respectively.

Differential Diagnosis

Osteoporosis should be differentiated from the following diseases:

- Osteomalacia
- Multiple myeloma
- Paget's disease
- Mono-ostotic fibrous dysplasia

Treatment

Osteoporosis is extremely difficult to treat; hence, prevention is best. As multiple aetiological factors may be present, the treatment has to be comprehensive. The patient must be given a high protein diet with a liberal intake of milk. The patient must be given vitamin D and calcium orally. Vitamin D increases absorption of calcium from the intestines and reabsorption from the kidneys. Oestrogen replacement therapy is indicated in premature or surgically induced menopausal women. The oestrogen stops the loss of bone mass in post-menopausal women. Calcitonin and bisphosphonates (Table 6.3) have proven to slow down post-menopausal bone loss. Adrenal anabolic hormone as a weekly intramuscular injection is very helpful in promoting synthesis of the osteoid matrix. The androgens are useful due to their anabolic effect. Use of braces will prevent pathological fractures; a spinal brace may be given for a few months. The patient should not be immobilised for too long even if there is compression fracture of vertebrae but must be encouraged to be active to promote muscular tone stress on the bones.

TABLE 6.3 Bisphosphonates Used in the Management of Osteoporosis

Bisphosphonates	Dosage
Etidronate	Not used nowadays
Pamidronate	Only IV injection available
Alendronate	Once-a-day tablets
Zolendronate	Once-a-year IV infusion

Differential Diagnosis

- Osteomalacia
- Multiple myeloma
- Paget's disease
- Mono-ostotic fibrous dysplasia

Secondary Osteoporosis

Secondary osteoporosis is due to indirect causes affecting the bone. These are potentially reversible disorders, and hence patients with osteoporosis in young age and rapidly progressive osteoporosis in old age are thoroughly investigated. Some of the causes of secondary osteoporosis are as follows:

- Nutritional (scurvy, malabsorption)
- Endocrine (hypercorticism, thyrotoxicosis)
- Drugs (corticosteroids, alcohol, heparin)
- Malignancy (multiple myeloma, leukaemia)
- Non-malignancy causes (chronic renal disease, rheumatoid disease, tuberculosis)

Clinical Features

The patients present with the primary problem, for example moon face and cushingoid features in the case of steroid therapy. Fractures following trivial trauma are common in regions of vertebra, pelvis and femoral neck. Investigations are done as for primary osteoporosis.

Treatment

Treatment is needed for the primary conditions; fractures are treated with internal fixation; immobilisation period is really shortened and kept to a bare minimum. The drug therapy is as per the treatment of primary osteoporosis.

SCURVY

Scurvy is a disease occurring in poorer children due to deficiency of vitamin C (ascorbic acid) in the diet. It occurs between 6 months and 2 years of age.

The main pathological effects of this deficiency are (1) inability to form normal intercellular collagen and organic bone matrix and (2) haemorrhagic tendency due to fragility of the capillary walls due to poor intercellular cement substance. The growth of the bone up to the point of provisional calcification is normal. The formation of capillaries and their invasion of the chondroid is defective, and hence osteoid formation is poor. Haemorrhage may occur in the subperiosteal areas of long bones such as the femur. The subperiosteal haemorrhage gets partly calcified and partly absorbed.

Clinical Features

The child is in poor general health and shows failure to gain weight. The child may be brought with a swelling in the thigh of rather sudden onset. This is caused by acute subperiosteal haemorrhage. The child will have severe pain and will not move the limb (pseudo-paralysis). The child will show other evidences of vitamin C deficiency such as gingivitis with the bleeding gums. In many cases, infantile scurvy and rickets may coexist (scurvy rickets).

Differential Diagnosis

Subperiosteal haemorrhage may simulate acute haematogenous osteomyelitis, but there will be no fever or other laboratory evidence of infection.

Radiological Features

The cortices of the long bones are thinned. The metaphyseal area is rarefied, and there is a prominent white line of calcification (Frenkel's line). The epiphysis is outlined by a prominent white line (ringing). The periosteum may be elevated and a shadow of subperiosteal haemorrhage, partly calcified, may be present (Fig. 6.6). There may be a displacement of the distal femoral epiphysis due to weakening at the metaphysis.

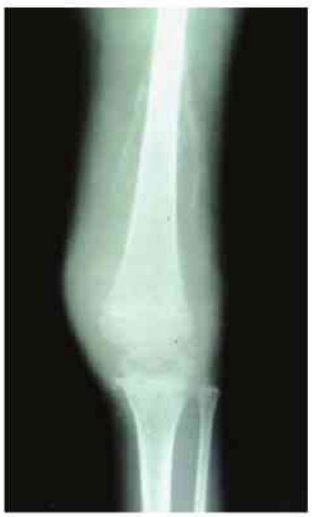


FIGURE 6.6 X-ray of left femur demonstrating subperiosteal haemorrhage.

Treatment

Scurvy is totally preventable with an adequate intake of vitamin C in the diet. High doses of vitamin C are given to cure established cases of scurvy. Signs and symptoms clear up completely in a few weeks of therapy.

BONE CHANGES IN ENDOCRINE DISORDERS

Many of the endocrine disorders have skeletal involvement as part of their clinical manifestations. Although some of them may show prominent growth defects or skeletal deformities, they are mainly problems for the endocrinologist and physician rather than for the orthopaedic surgeon. Orthopaedic care is needed only when complications such as fractures or deformities occur.

Aetiopathogenesis

1. Pituitary dysfunction
   a. Hyperfunction
      - Gigantism: Hyperfunction before epiphyseal closure
      - Acromegaly: Hyperfunction after epiphyseal closure

b. Hypofunction
   - Pituitary dwarfism

2. Thyroid dysfunction
   a. Cretinism (thyroid dwarfism): Hypothyroidism in childhood affects bone growth.
3. Parathyroid dysfunction
   a. Hyperparathyroidism: Due to
      - Generalised hyperplasia: Primary or secondary
      - Adenoma (focal hyperplasia)
   b. Hypoparathyroidism
4. Gonadal hormonal dysfunction
   - Menopausal osteoporosis
   - Senile osteoporosis

Parathyroid Osteodystrophy (Osteitis Fibrosa Cystica)

The parathyroid hormone is an 84-amino-acid polypeptide and it acts through the adenyl cyclase enzyme. Parathyroid osteodystrophy is one of the disorders due to hyperparathyroidism which manifests itself mainly as a skeletal disease. Hyperparathyroidism can be due to generalised hyperplasia of the whole gland or localised secreting adenoma of the gland, which is a more common type. Eighty-eight per cent of cases of hyperparathyroidism are due to parathyroid adenoma.

The parathyroid hormones have two actions:

1. Direct action on the bone causing osteoclastic osteolysis
2. Action on the renal tubules, inhibiting reabsorption of phosphates from the glomerular filtrate resulting in phosphaturia and hypophosphataemia

The hyperactivity of the parathyroid gland results in progressive generalised decalcification of the entire skeleton causing deformities and pathological fractures. In some cases, it may manifest as a single localised cystic lesion in the bone simulating a tumour and hence called a Brown tumour. Brown tumours are commonly seen in the facial bones and are associated with this condition. Occasionally, there is nephrocalcinosis (multiple renal stones) and soft tissue calcification.

Clinical Features

The clinical effect of hyperparathyroidism is due to its function in mobilising calcium from bone and inhibiting its renal secretion. The disease is more common in females than males. Majority of cases occur during the third, fourth and fifth decades. The disease begins insidiously with lethargy, loss of muscle tone and general weakness. There is anorexia, nausea and vomiting. These are due to increased calcium in the blood and the effect of calcium ion on neuromuscular excitability of the voluntary as well as the smooth muscles. In later stages, prolonged hypercalcaemia causes renal calculi and damage to the kidneys and the patient may finally develop renal failure. Pain usually occurs and is due to renal stones. Patients may also develop pancreatic calcification. Psychotic disturbances can also occur.

Orthopaedic Signs

The patient complains of generalised bone pains. This is the most common initial feature which is associated with weakness and hypotonia. The patient may occasionally present with a pathological fracture either through the osteoporotic bone or through a localised cystic lesion.

Biochemical Features

The most important feature of this condition is an elevated serum ionised calcium and decreased serum inorganic phosphate. The serum calcium may be increased from the normal level of 10 to more than 12 mg per 100 ml. Serum phosphorous is lowered to 2–3 mg per 100 ml. Serum alkaline phosphatase is raised. Urinary excretion of calcium is low and that of phosphate is high.

Radiological Features

There is generalised demineralisation of all the bones. The bones show broadening of medullary canals and thinning of the cortex. The femur and tibia show deformities, and there may also be pathological fractures. Localised osteolytic areas which resemble giant cell tumours radiologically are seen in long bones. Phalanges show haziness of the cortical surface due to subperiosteal resorption. The diagnostic feature of parathyroid bone disease is subperiosteal resorption of the phalanges. There

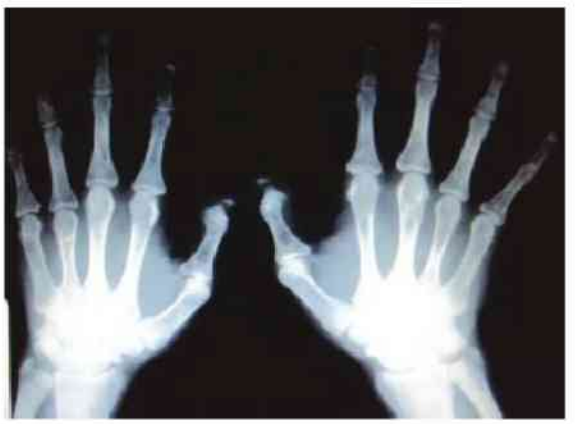


FIGURE 6.7 X-ray of both hands' anteroposterior view showing parathyroid osteodystrophy. Note the subperiosteal resorption of phalanges.

is also absorption of the tips of the terminal phalanges causing a disappearance of the terminal tuft (Fig. 6.7). The mandible shows characteristic absence of lamina dura of the teeth sockets.

The radiological features seen in hyperparathyroidism can be enumerated as follows:

- Irregular diffuse rarefaction with resorption of the compact bone
- Cyst-like degeneration
- Salt pepper appearance of the skull
- Loss of lamina dura
- Osteoporotic and collapsed vertebrae
- Erosion of the tufts of the phalanges
- Extra-osseous calcium deposits

Treatment

Surgical correction of deformities is done by doing osteotomies. Brown tumours are curetted and bone grafting done. The specific treatment for this condition is management of the basic pathology leading to hyperparathyroidism. In the case of adenoma, removal of the parathyroid adenoma by surgery is performed. Orthopaedic treatment consists of prevention of deformities and fractures in the softened bones by the use of splints, braces and appliances; bed rest is also recommended for the same reason.

Secondary Hyperparathyroidism

Secondary hyperparathyroidism is a hyperactivity of the parathyroid, secondary to chronic renal disease which causes lowering of serum calcium and raised serum phosphates. The low serum calcium stimulates the parathyroid to hyperactivity, which when prolonged causes decalcification of bones. In this condition, the serum calcium level is never high; it is either low or normal. The skeletal changes are the same as those in hyperparathyroidism; deformities in the bones and pathological fractures are common. The osteolysis is called *renal osteodystrophy* in adults. In children, it is called renal rickets and causes renal dwarfism. The treatment is mainly for the renal disease.

Growth Hormone Deficiency

Growth hormone is secreted by acidophilic, somatotrophic cells of anterior pituitary. Disorders may occur in all cells of hypothalamic-pituitary somatomedin-chondro-osseus axis and can be of any one of the following types:

- Acquired pituitary insufficiency in a growing child
- Multitropic pituitary hormone deficiency or pan hypopituitary dwarf
- With proportionate dwarfism—high-pitched voice, subcutaneous adipose tissue and sexual infantilism and infertility
- Isolated growth hormone deficiency—there is proportionate dwarfism with normal sexual development
- Congenital absence of pituitary

Radiological Features

The epiphyses remain ununited for a prolonged period. Metaphysis terminates in a line of dense bone. Histologically, there is absence of division of cartilage cells and primitive connective tissue cells.

DISEASES OF THE RETICULOENDOTHELIAL SYSTEM INVOLVING BONES (Histiocytosis)

The following diseases will be discussed in this category:

- Eosinophilic granuloma of bone
- Hand–Schuller–Christian disease

- Bone lesions in diseases of the haemopoietic system

Eosinophilic Granuloma of Bone

Eosinophilic granuloma is a condition characterised by a solitary localised granulomatous lesion in bone. It is a rare disease occurring in children and young adults. Histologically, eosinophils are very predominant in the granulomatous mass.

Clinically, it presents with vague pain referable to the site involved. The upper end of the femur, the ilium and the vertebral bodies are the common sites. Radiologically, the lesion presents as an osteolytic area. In the vertebra, it causes collapse and flattening of the vertebral body (vertebra plana). The treatment is by curettage or radiation therapy.

Hand–Schuller–Christian Disease

Hand–Schuller–Christian disease is a disorder of the reticuloendothelial system where, in addition to generalised lesions, it produces lesions in the bones.

The bone lesions involve any part of the long bone or spine and present as swellings. Cholesterol or other lipids are found phagocytosed within the macrophages of the reticuloendothelial system. The condition presents as focal granulomatous lesions in the bone, tendon sheaths, etc., and enlarges progressively. The exact aetiopathology of these conditions is not clear.

The disease begins in early childhood. The bone lesions may involve any part of the long bones or spine. The classical triad is the involvement of the orbit with exophthalmos, involvement of pituitary fossa with endocrine disturbance and lesions in other bones.

The characteristic radiological feature is the presence of sharply defined punched-out osteolytic areas. Diagnosis is confirmed by biopsy. The prognosis is bad in this disease, although it could be controlled by radiation therapy and steroids.

Bone Lesions in Diseases of the Haemopoietic System

Bone marrow being the location of the major portion of the haemopoietic system, many diseases affecting this system are reflected by lesions in the medullary cavities of the bone. Obliteration of the medullary cavities by osteosclerotic diseases such as osteopetrosis and fluorosis results in severe anaemia.

All chronic haemolytic anaemias cause a compensatory hyperplasia of the marrow resulting in the broadening of the medullary cavities and thinning of the cortical bone. These bone changes are most markedly seen in certain chronic haemolytic anaemias of genetic origin such as sickle cell anaemia.

Haemoglobinopathies are disorders due to the presence of abnormal haemoglobins (HbS. HbC.) caused by genetic mutation. This is the aetiological factor for sickle cell anaemia and thalassaemia, which cause bone changes as a characteristic clinical manifestation.

Clinical Features

The patients often present with generalised pain in the bones and joints. There is increasing anaemia. The course of the disease is characterised by the occurrence of vaso-occlusive *sickle cell crisis* which causes infarction due to intravascular thrombosis and avascular necrosis in the head of the femur and humerus. Dactylitis in the hands and feet with painful swelling in the phalanges is an important feature in this condition. Salmonella infection of the bone is also a common complication in sickle cell anaemia.

Radiological Features

The trabecular pattern of the pelvic bones and long bones is coarse. The long bones show a widened medullary cavity. In sickle cell anaemia, avascular changes are seen in the head of the femur.

Treatment

The main line of treatment is by repeated blood transfusions to maintain the haemoglobin at 10 g per 100 cc.

MISCELLANEOUS BONE DISEASES

There are some lesions in the bone which do not fit into the broad classification of diseases given earlier. They are as follows:

- Paget's disease of the bone
- Fluorosis
- Hypervitaminosis
- Infantile cortical hyperostosis (Caffey's disease)

Paget's Disease (Osteitis Deformans)

Paget's disease, first described by Sir James Paget, is a generalised disorder of the bone, occurring in the elderly, resulting in progressive deformities of the spine and limbs. This condition is uncommon in India but common in Europe and America.

Aetiopathology

The aetiology is unknown. The presence of inclusion bodies in osteoclast has suggested a viral aetiology. The basic pathology is increased bone resorption and disorganised bone formation. Both stages of increased osteoblastic and osteoclastic activities are seen. The bone changes are characterised by a slow progressive replacement of compact bone by vascular spongy bone and fibrous tissue. There is demineralisation causing coarsening of the trabeculations. Later on, dense areas appear due to excessive reparative bone formation.

Clinical Features

There are three varieties of Paget's disease: (1) monostotic form involving a single bone such as clavicle or tibia, (2) polyostotic form which affects several bones and (3) facial type affecting the jaws.

Clinically, the patient complains of severe pain in the bones or back. The skull is enlarged, and there is kyphosis of the spine. The tibia, femur, pelvis, spine and skull are affected. There is thickening and bending of the tibia and femora resulting in bowing of bones and shortening of the stature (Fig. 6.8). The bones are thicker and there is warmth over subcutaneous bones such as tibia, due to intraosseous arteriovenous (AV) shunts. The pulse pressure is increased due to the AV shunts.

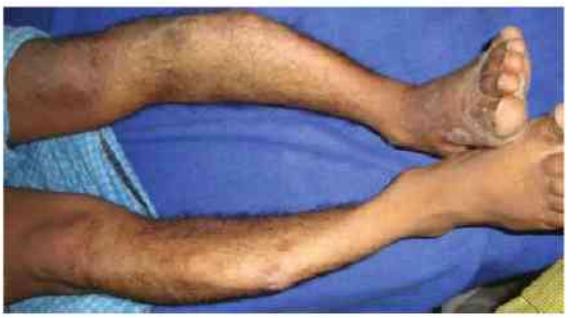


FIGURE 6.8 Clinical photo of Paget's disease showing tibial bowing.

Complications

The patient may present with complications such as pathological fracture or localised bone swelling due to sarcomatous changes. He may also get cranial nerve palsies with deafness or cord symptoms due to spinal canal stenosis. Osteoarthrosis, hypercalcaemia and high output cardiac failure are also seen.

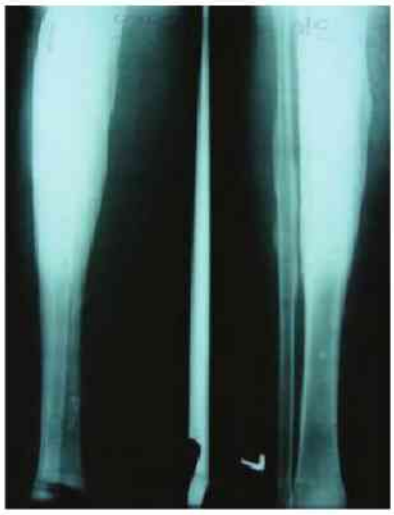
Radiological Features

Paget's disease shows varying radiological features depending on the stage of activity and the site. The most common type is the honey comb or spongy appearance which is widespread (Fig. 6.9). The pelvis and calcaneum may show a striated appearance.

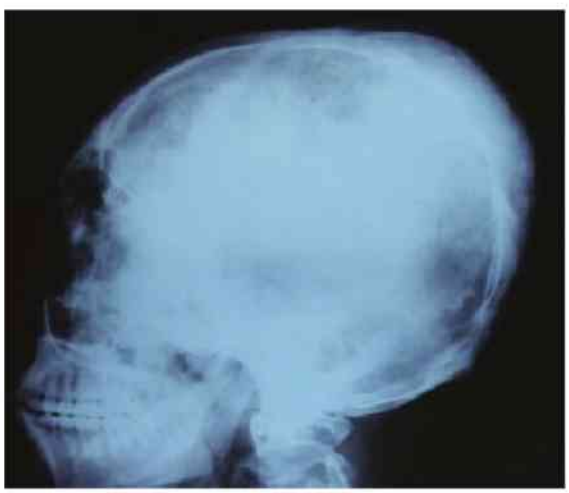
In the vertebrae, the lesion shows an appearance of uniform increased density. Flame-shaped lesion along the shaft of bone or circumscribed patch of osteoporosis in the skull (osteoporosis circumscripta) may be found.

Laboratory Investigations

Serum calcium and phosphorus are normal. Serum alkaline phosphatase is increased. There is an increase in hydroxyproline excretion in the urine indicative of rapid turnover of bone matrix.



(a)



(b)

FIGURE 6.9 Paget's disease. (a) X-ray of both legs shows dense and coarse lobe and (b) X-ray of skull shows thickened diploic.

Treatment

This is mainly symptomatic. Mild pain may be relieved by common analgesics. Calcitonin and diphosphonates are very effective in relieving pain by reducing bone turnover. Pathological fractures and bone sarcoma need active management.

Fluorosis

Fluorosis is a condition of chronic intoxication by fluorine due to excessive fluorides in drinking water. This condition is characterised by excessive deposition of fluoride in bone and soft tissues. This condition is prevalent in the Salem district of Tamil Nadu, Nellore and Guntur districts of Andhra Pradesh and in some areas in Punjab, Uttar Pradesh, Orissa and West Bengal. A fluorine content of more than 1 ppm in drinking water is liable to cause fluorosis. Ray (Kolkata) has done extensive work on fluorosis.

Clinical Features

The patient is usually middle-aged and complains of pain in the back and in the large peripheral joints. On examination, there will be marked stiffness of the spine. Other signs of fluorosis are brownish mottling in the teeth and in later stages destruction and dropping of the affected teeth and anaemia.

- *Skeletal changes:* There will be low back pain associated with stiffness of the spine. There will be compression over the cord due to calcification of soft tissue surrounding the cord. Excessive quantities of fluoride are deposited over the bones which become uneven, thick and heavy. Irregular bone is also laid down along the attachment of muscles, tendons, ligaments, capsules and interosseous membranes. Maximum changes occur in the spine with marked calcification of the paraspinal ligaments. Fluoride bones are always heavy bones.
- *Dental fluorosis:* The earliest sign is mottling of teeth followed by falling of teeth.

Radiological Features

The radiological appearance of generalised osteosclerosis is very diagnostic of fluorosis. Spine shows a marked increase in the density of the vertebral bodies with calcification in the anterior and lateral spinal ligaments (Fig. 6.10). There is also calcification of other soft tissues surrounding the axial skeleton.

There is calcification at the muscular and ligamentous attachment of the interosseous ligaments in the forearms and legs.

There will be increased density and calcification of non-osseous tissue especially surrounding the axial skeleton. There will be intraosseous membrane calcification.

Other investigations show a higher fluoride level in body fluids and drinking water.

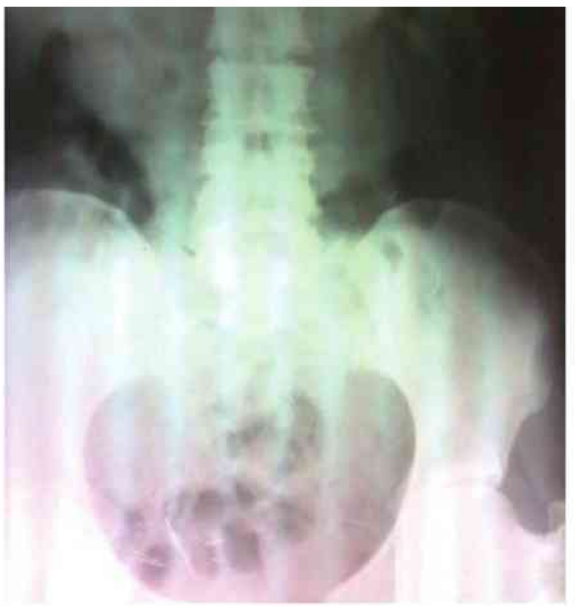


FIGURE 6.10 X-ray of lumbosacral spine in fluorosis.

Differential Diagnosis

The increased sclerosis has to be differentiated from osteopetrosis, secondaries from carcinoma prostate, diffuse idiopathic skeletal hyperostosis and Paget's disease.

Laboratory Findings

The fluorine estimation in urine shows elevation above the normal figure of 0.1 mgm per cent. Estimation of fluorine content in the bone shows a marked increase above the normal figure of 0.05–0.2 mgm per cent.

Complications

Compression of the spinal cord and the nerve roots occurs in advanced cases. Lung functions are compromised due to rigid chest cage leading to dyspnoea. Others are severe anaemia and renal complications.

Treatment

This is mainly symptomatic in the severe cases. The only effective preventive measure is to avoid drinking water containing excessive fluorine and provide protected water supply to the population. Defluorination of water (e.g. Nalgonda technique) can be done for this purpose. Skeletal deformities may need correction.

Hypervitaminosis A

Hypervitaminosis A usually results from excessive intake of preparations containing vitamin A. It may be either acute or chronic.

Clinical Features

Its orthopaedic importance arises from the fact that one of the prominent symptoms of chronic hypervitaminosis A is bone pain and swelling in the bone. The symptoms of acute toxicity are abdominal pain, nausea, vomiting, headache and irritability. Chronic toxicity is characterised by anorexia, weight loss, hair loss, bone and joint pain, benign intracranial hypertension and hepatomegaly. Bone pain is due to calcification of the pericapsular tissue and subperiosteal new bone formation. The clinical diagnosis is usually made following the appearances of tender, deep, hard swellings on the extremities and in the occipital region.

Investigations

Plasma vitamin A concentration will be elevated.

Treatment

It is merely withdrawal of vitamin A.

Hypervitaminosis D

Hypervitaminosis D is a clinical entity caused by excessive ingestion of vitamin D2 in doses of 50,000 units per day over long periods by children.

Clinical Features

The signs and symptoms of vitamin D intoxication are usually due to deranged calcium metabolism caused by excessive absorption of calcium from the gut. The initial symptoms are those associated with hypercalcaemia and its effects on the myoneural junction. They are weakness, fatigue, headache, nausea, vomiting and diarrhoea. With prolonged hypercalcaemia, there may be deposition of calcium in the kidney, periarticular tissues, joint capsules, tendon sheaths and blood vessels.

Treatment

The treatment consists in immediate withdrawal of the vitamin, a low calcium diet, administration of glucocorticoids and a generous intake of fluids.

Infantile Cortical Hyperostosis (Caffey's Disease)

Infantile cortical hyperostosis is a self-limiting disease of unknown aetiology occurring in infants. It can simulate a systemic infection with fever and leucocytosis and is characterised by soft tissue swellings over the clavicle, mandible and the long bones. The swellings are firm and tender with no features of inflammation and they do not suppurate. When some swellings subside, others may appear. The bone may thicken enormously.

The typical radiological feature is a marked periosteal new bone formation surrounding the diaphysis of long bones. The new bone becomes lamellated as the activity of the condition subsides and the bone becomes normal. It is a self-limiting condition and no treatment is indicated.

KEY POINTS

- Bones in the human body store more than 99% of calcium.
- Calcium metabolism is mainly regulated by PTH, vitamin D and calcitonin. Their physiological actions are as follows:
  PTH: ↑serum calcium, ↓serum phosphate
  Vitamin D: ↑serum calcium, ↓ serum phosphate
  Calcitonin: ↓serum calcium, ↑serum phosphate
- Rickets, depending on the cause, can be either Type I: defective metabolism of vitamin D or its deficiency, or Type II: deficiency of phosphate.
- Osteoporosis is a pathological process characterised by defective formation of the organic protein matrix, thereby leading to reduction of the bone mass.
- Osteoporosis can be classified into primary: senile/post-menopausal or secondary: endocrine/drugs/neoplasm. DEXA scan is helpful in diagnosis.
- Scurvy is a disease occurring due to deficiency of vitamin C. This disease is seen frequently in poor children. It may coexist with rickets in many cases (scurvy rickets).
- Increased activity of parathyroid hormone (hyperparathyroidism) may cause generalised bone pains, pathological fractures, brown tumours (cystic lesion in bones) and soft tissue calcification. Subperiosteal resorption of phalanges is the diagnostic feature.
- Paget's disease is a generalised disorder of bones characterised by increased bone resorption and disorganised bone formation.
- Fluorosis is characterised by excessive deposition of fluoride in bone and soft tissues. Fluorine content of more than 1 ppm in drinking water is liable to cause fluorosis.

MULTIPLE CHOICE QUESTIONS

1. Rickets in infancy is characterised by the following except
   a. Craniotabes
   b. Rachitic rosary
   c. Wide open fontanelles
   d. Bow legs
2. Osteomalacia is characterised by
   a. Compression of vertebra
   b. Cod fish vertebra
   c. Looser's zone
   d. Bamboo spine
3. The commonest site of fracture in senile osteoporosis is
   a. Neck of femur
   b. Shaft of femur
   c. Radius
   d. Vertebra
4. The characteristic feature of primary hyperparathyroidism is
   a. Swelling of lamina dura
   b. Diffuse osteopenia
   c. Salt and pepper appearance of skull
   d. Subperiosteal resorption of terminal phalanges
5. Hand–Schuller–Christian disease, which is correct
   a. Proliferation of reticuloendothelial cells
   b. Foam cells seen
   c. Punched-out lesions in X-ray
   d. Diabetes insipidus and exophthalmos present
   e. All are correct
6. Paget's disease of bone commonly affects
   a. Skull
   b. Vertebra
   c. Pelvis
   d. Femur
   e. Humerus

CHAPTER 7

Diseases of Joints

Joint diseases form one of the most important groups of crippling diseases in the world. The most common cause of arthritis in India is due to the prevailing infections of various types, whereas degenerative and metabolic joint diseases predominate in the developed countries.

DISEASES OF JOINTS AND THEIR CLASSIFICATION

Joints are subject to various types of diseases and disorders. Many lesions which are not strictly inflammatory are still loosely termed as arthritis. Diseases of joints can be classified as follows:

1. Infective arthritis: Bacterial, viral and parasite
   a. Acute infection
      - Acute pyogenic arthritis
      - Acute gonococcal arthritis
      - Acute rheumatic arthritis
      - Small pox arthritis
   b. Chronic infections
      - *Non-specific:* Pyogenic arthritis
      - *Specific:* Tuberculous arthritis, syphilitic arthritis, gonococcal arthritis
      - *Parasitic:* Guinea worm arthritis
2. Rheumatoid arthropathy
   a. Rheumatoid arthritis
      - Rheumatoid arthritis
      - Juvenile rheumatoid arthritis
   b. Seronegative spondyloarthropathy
      - Ankylosing spondylitis
      - Reiter's disease
      - Psoriatic arthritis
      - Enteropathic arthritis
3. Degenerative arthrosis (osteoarthritis)
   a. Primary osteoarthrosis
   b. Secondary osteoarthrosis
4. Neuropathic arthropathy
   a. Charcot's arthropathy
   b. Syringomyelia
   c. Leprosy
   d. Diabetes mellitus
5. Metabolic arthritis
   a. Gout
   b. Pseudo-gout
   c. Alkaptonuric arthritis
6. Arthritis in systemic disorders
   a. Haemophilic arthritis
   b. Reactive arthritis
7. Miscellaneous conditions
   a. Villonodular synovitis
   b. Synovial chondromatosis
8. Hysterical joint

Pathology

Arthritis is an inflammation of all the component structures of the joint with involvement of the synovium, articular surfaces and capsule. The following stages can be identified:

- Stage of synovitis
- Stage of reversible arthritis
- Stage of irreversible arthritis
- Stage of ankylosis

The critical stage of the disease is the involvement and destruction of the articular cartilage, as any gross damage to the cartilage is irreversible, leading to ankylosis and loss of function.

RHEUMATOID ARTHRITIS

Collagen diseases are systemic diseases affecting all connective tissues in the body. Many of these disorders have joint manifestations. The most important of these is rheumatoid arthritis. Rheumatoid arthritis is a generalised chronic multisystem disease affecting the connective tissues of the whole body with focalised involvement of the musculoskeletal system.

There is inflammatory synovitis of peripheral joints, leading to cartilage damage, bone erosions and subsequent joint changes.

Aetiology

Aetiology remains unknown. The following are put forth as possible aetiological factors:

- *Genetic predisposition:* Rheumatoid arthritis runs in families. It is associated with Class-II major histocompatability complex allele HLA-DR4 and HLA-DRBI. Genetic factors alone do not account for the disease.
- *Abnormal immune response:* Rheumatoid arthritis may be a manifestation of an immune-mediated response to infections caused by mycoplasma, Epstein–Barr virus, cytomegalovirus, parvovirus in a genetically predisposed individual.

Pathology

Rheumatoid disease is considered to be an autoimmune response to an unknown antigen and the antibody formed is the rheumatoid factor which is identified as immunoglobulin M (mostly IgM) or IgG (less commonly).

The rheumatoid factor is an IgM antibody directed against the Fc portion of IgG antibodies. It is present in about 80% of patients with rheumatoid arthritis. It is not diagnostic of rheumatoid arthritis but is an important *prognostic factor* in follow-up of rheumatoid arthritis.

Rheumatoid factor may also be present in the following:

- Systemic lupus erythematosus
- Sjogren's syndrome
- Sarcoidosis
- Tuberculosis

Other autoantibodies found in rheumatoid arthritis are antibodies to antifilaggrin and anticitrullinated proteins.

Rheumatoid arthritis is an inflammation of the synovial membrane, which becomes oedematous and thickened with inflammatory exudates. Chronic persistent synovitis is a characteristic feature of rheumatoid arthritis. The disease follows three stages of synovitis, destruction and deformity. Microscopy shows lymphoid follicles forming nodules with scattered cells. In the later stage, the synovium is more vascular and throws a fibrinous exudate which gets organised into a granulation tissue and spreads over the articular cartilage as the pannus. The articular cartilage gets lysed from the surface. A similar lytic process occurs on the deeper surface of the articular cartilage from the granulating lesion in the subchondral bone. The inflammatory process spreads into the capsule and the periarticular tissue.

During the healing process, the granular pannus becomes fibrous, uniting the joint surface and causing a fibrous ankylosis. The muscles around the joint also undergo inflammatory changes in the collagen tissue and get atrophied. Rheumatoid nodules which are palpated over bony prominences consist of a central necrotic zone surrounded by radially dispersed palisade of local histiocytes.

Stages in Pathology of Rheumatoid Arthritis

1. Stage of synovitis
   - Initial lesion occurs in the synovium, leading onto vascular stasis, infiltration of the subsynovial layers with inflammatory cells and formation of fibrinous exudate.
   - Synovial hypertrophy occurs with the thickening of capsular structures.

2. Stage of destruction
   - *Pannus formation:* Hypertrophied synovium along with granulation tissue leads to formation of pannus which encroaches the articular cartilage from its periphery.
   - *Articular cartilage destruction:* The articular cartilage gets destroyed gradually. Further the bony surfaces are involved, leading to obliteration of joint space. Joints get destroyed and deformed (see Fig. 7.1).
3. *Stage of deformity:* The extending granular pannus gets organised into fibrous tissue, which bridges the articulating bone ends, leading to fibrous ankylosis and later bony ankylosis.
   - Ligaments are involved leading to joint subluxation or dislocation.
   - Muscles, tendons and soft tissues around the joint also undergo inflammatory changes and get contracted or ruptured.
   - Juxta-articular osteoporosis occurs.
   - Not all patients progress through all three stages.

Some may have mild disease and recover, while others may suffer from chronic disease with crippling deformities.

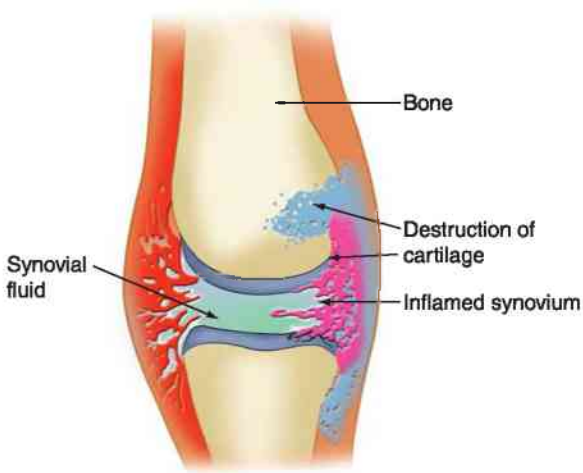


FIGURE 7.1 Diagrammatic representation of the stage of destruction in rheumatoid arthritis.

Clinical Features

Rheumatoid arthritis is more common in women and occurs between 25 and 40 years of age. It is a chronic disease with periodic acute exacerbations and remissions. Morning stiffness is very characteristic of rheumatoid arthritis. It usually involves the small joints of the hands and feet and later on spread to the proximal joints such as the knee, hips, elbow and shoulder. Bilateral symmetric polyarthritis is also a characteristic feature.

Occasionally, it may start in the knee or hip and remain as a monoarticular lesion for some time, but soon it spreads to other joints. The presenting joint is swollen and warm. There is joint line tenderness and the movements are painful and limited. There is effusion into the joint. The synovium could be felt thickened and is tender. In the knee joint, the swelling causes a fusiform appearance with pericapsular swelling. In the late stage, with progressive damage to the articular cartilage, there will be an increase in flexion deformity, ultimately ending in ankylosis.

In the hand, the small joints are swollen and the fingers assume a position of 'ulnar deviation', clinically called 'ulnar drift'. The fingers assume an 'intrinsic plus' deformity which consists of flexion at the metacarpophalangeal joints and extension at the interphalangeal joints. The end stage of the disease is marked by ankylosed joints.

The 'intrinsic plus' position of the fingers is due to the spasm of the intrinsic muscles of the hand which later becomes a contracture. The other deformities in the hand are as follows:

- *Boutonniere* (buttonhole) deformity due to the rupture of the central slip of the extensor tendon resulting in flexion at the proximal interphalangeal joint with hyperextension at the distal interphalangeal joint.
- *Swan neck deformity*, where there is hyperextension at the proximal interphalangeal joint and flexion at the distal interphalangeal joint. In the late stages, the capsular contractures become fixed, resulting in a permanent crippling deformity (Fig. 7.2).
- Elbow—flexion deformity
- Knee—flexion deformity followed by triple subluxation: flexion, posterior subluxation and external rotation

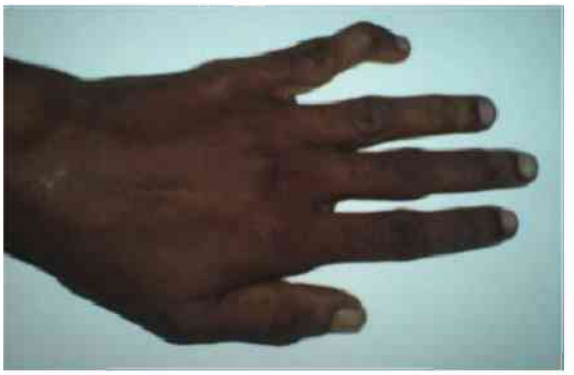


FIGURE 7.2 Clinical photograph showing swan neck deformity in the little finger.

- Ankle—equinus deformity
- In the foot, patients develop hallux valgus of the great toe (Fig. 7.3), claw toes, hammer toes and callosities.

Other joints involved in order of frequency are as follows:

- Foot
- Wrist
- Knees, elbow
- Ankle
- Hip
- Temporomandibular joint
- Interspinous facet joint (rare)

Extra-Articular Manifestation of Rheumatoid Arthritis

The patient may also present with fever, lymphadenopathy, splenomegaly, rheumatoid nodules and ocular symptoms. The following are the extraarticular manifestations of rheumatoid arthritis:

1. *Rheumatoid nodule:* It usually occurs beneath the skin over bony prominences such as olecranon (Fig. 7.4). It also occurs in synovium, tendons, sclera and other viscera.
2. Rheumatoid tenosynovitis or bursitis, for example trigger finger or thumb. Carpal tunnel syndrome can be caused by the tenosynovitis in the carpal tunnel.
3. *Atrophy of skeletal muscles:* There is general wasting of the muscles and thinning of the skin.
4. *Tennis elbow:* Epicondylitis of the lateral condyle of the humerus
5. Plantar fascitis with heel pain
6. Fibrositis of the neck and back
7. *Rheumatoid vasculitis:* It can involve any vessels. It can cause polyneuropathy, mononeuritis multiplex, cutaneous ulceration and visceral infarction.
8. *Pulmonary involvement:* It includes the following:
   - Pleural disease
   - Interstitial fibrosis
   - Pleuropulmonary nodules
   - Pneumonitis
9. *Cardiovascular system:* Asymptomatic pericarditis may occur.
10. *Neurological involvement:* This may follow cervical spine subluxation and entrapment neuropathies of peripheral nerves.
11. *Ocular involvement:* Episcleritis and scleritis may develop; keratoconjunctivitis sicca is another noted complication.

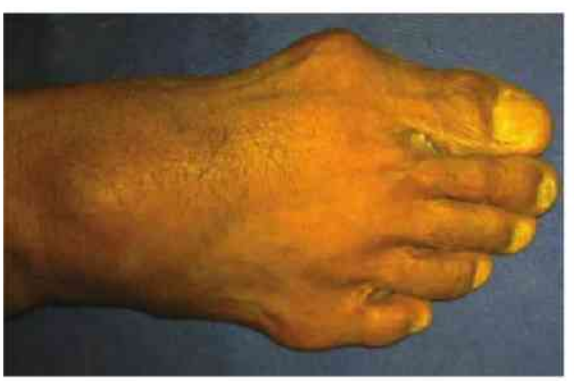


FIGURE 7.3 Clinical photograph showing hallux valgus deformity in rheumatoid arthritis.

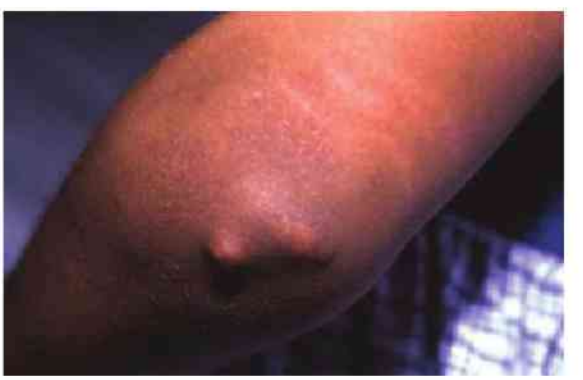


FIGURE 7.4 Clinical photograph showing rheumatoid nodule.

12. *Felty's syndrome:* Chronic rheumatoid arthritis associated with *splenomegaly* and *neutropenia* is noted in this syndrome.
13. *Osteoporosis:* This may be associated with rheumatoid arthritis itself and concomitant steroid therapy.

Differential Diagnosis

- Ankylosing spondylitis
- Systemic lupus erythematosus
- Reiter's disease
- Gout and pseudo-gout
- Osteoarthritis

Diagnosis

The essential criteria laid down by the American Rheumatism Association for the diagnosis of rheumatoid arthritis are as follows:

1. Morning stiffness
2. Arthritis of three or more joints
3. Arthritis of hand joints (wrist, metacarpophalangeal joints, proximal interphalangeal joints)
4. Symmetric arthritis
5. Rheumatoid nodules
6. Serum rheumatoid factor demonstration
7. X-ray changes—erosion/periarticular osteopenia

Presence of four or more features suggests the diagnosis of rheumatoid arthritis. The first four criteria, mentioned above, should at least be present for 6 weeks.

Laboratory Findings

Blood picture shows normochromic, normocytic anaemia during the active stage of the disease. The erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) remain elevated.

Special Tests

The majority of patients with rheumatoid arthritis show evidence of the presence of an antibody, called rheumatoid factor (seropositivity), in the serum. This rheumatoid factor is the IgM fraction of the immunoglobulin which can be detected by Rose–Waaler test and latex (agglutination) test.

TABLE 7.1 Radiological Features Seen in Rheumatoid Arthritis

Early Changes	Late Changes
Soft tissue shadows resulting from swelling	Joint dislocation
Narrowing of joint space	Deformity of joints
Bony erosion	Subluxation/dislocation of joints
Juxta-articular osteopenia	Subchondral cystic areas

Other investigations done are as follows:

1. Synovial fluid examination shows the following:
   - Turbidity
   - Reduced viscosity
   - Increased proteins
   - Normal or decrease glucose concentration
   - Increased polymorph count
2. Synovial biopsy and histological examination
3. Arthroscopic examination to evaluate damage to articular cartilage and also for synovial biopsy

Radiological Features

The X-ray of small joints of hand and other involved joints shows the following changes (Table 7.1; Fig. 7.5).

Apart from the features given in the table, there is generalised osteoporosis of the bones.

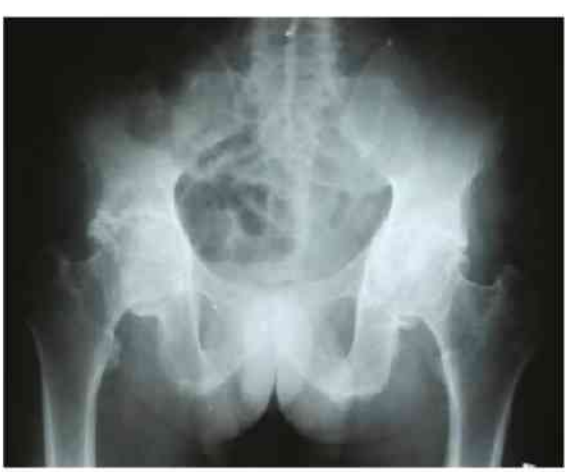


FIGURE 7.5 X-ray of pelvis with both hips' anteroposterior view showing bilateral rheumatoid arthritis. Note the symmetrical reduction in joint space.

Management

The treatment of this chronic crippling condition needs the team work of rheumatologist, orthopaedic surgeon, physiotherapist, occupational therapist and social worker to provide comprehensive management. The patient and his relatives must understand the condition fully and be well motivated to cooperate with the treatment which has to be prolonged.

The aim of the treatment is to

- relieve pain,
- keep the inflammatory process down to a minimum,
- preserve joint motion,
- maintain the tone of muscles,
- prevent deformities and stiffness of joint,
- correct deformities.

General Treatment

It is important to correct anaemia by haematinics and even blood transfusion may be necessary. A nutritious diet with a high intake of vitamin C is very essential for these patients.

Conservative Treatment

In the acute phase, the inflamed joint is kept at absolute rest by splinting the joint in the position of function. The splint must be removed as soon as the pain is diminished. Physiotherapy is given during the acute phase. Active joint mobilisation and muscle strengthening exercises are also prescribed. Heat therapy in the form of wax bath is very useful before giving exercise therapy.

Intra-articular injection of hydrocortisone is useful in the acute stage.

Drug Therapy

The drugs used are as follows:

1. Non-steroidal anti-inflammatory drugs (NSAIDs)
2. Disease modifying antirheumatoid drugs (DMARDs)
   - Methotrexate
   - Gold
   - Penicillamine
   - Antimalarials
   - Sulphasalazine
3. Steroids
4. Cytotoxic drugs
   - Azathioprine
   - Leflunomide
   - Cyclosporine
   - Cyclophosphamide
5. Newer drugs
   - TNF receptor antagonist—etanarcept
   - TNF receptor antibody—infliximab
   - IL-1 receptor antagonist—anakinra

Surgical Treatment

Surgery plays only an adjuvant role in the overall management of the disease. The role of surgery is mainly reconstructive or rehabilitative. The following surgical procedures are used in the management of rheumatoid arthritis. In the early stages of the disease,

- *Synovectomy:* This is done to remove the main focus of the disease and slow down its progress in joints such as knee and elbow, when bone destruction is minimum.

In late stages of the disease,

- Soft tissue release including capsulotomy and tenotomy are done to correct soft tissue contractures around the joint.
- Osteotomy: This is useful to correct deformities such as flexion–adduction deformities in the hip and genu valgum/varum in the knee.
- Arthroplasty: This is the operation to restore movements in joints stiffened by the disease. There are two methods of achieving this:
  1. *Excision arthroplasty:* It is excision of the articular ends of bones and shaping them leaving a gap. In India, excision of the head and neck of the femur (Girdlestone) is still a very useful method of arthroplasty in the hip joint (Fig. 7.6). The patient gets full painless movements and is able to squat on the floor for all purposes. Excision arthroplasty is also useful in the elbow joint.
  2. *Replacement arthroplasty:* It is replacement of articular ends by prosthetic implants. Replacement arthroplasty is being done with

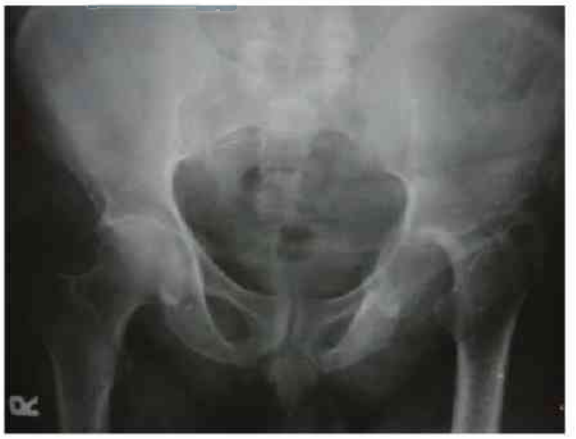


FIGURE 7.6 X-ray of pelvis with both hips' anteroposterior view showing ankylosed hip treated by excision arthroplasty on the left side.

great success in cases of severe hip involvement with stiffness. Total hip replacement (THR) involves replacement of the head of the femur by a metallic component and the acetabulum by a high density polyethylene cup (Fig. 7.7). The most common types used are Charnley's total hip prosthesis and Muller's prosthesis. The femoral and acetabular components are fixed to bone-by-bone cement (polymethyl methacrylate, Pte) (Fig. 7.7). More recently, cementless varieties of hip prosthesis are coming into use for the younger patients. Total knee prosthetic devices are also available for replacement arthroplasty for badly damaged knees. Knee prosthesis consists of the femoral component and tibial component which are fixed to the bone with bone cement (Fig. 7.8). Replacement arthroplasty is also useful in the case of metacarpophalangeal joints destroyed in rheumatoid arthritis. Prosthesis made by flexible silicon elastomer (Silastic) material is used in this operation.

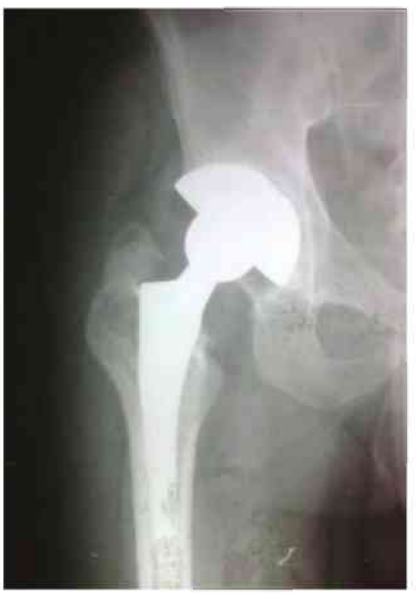
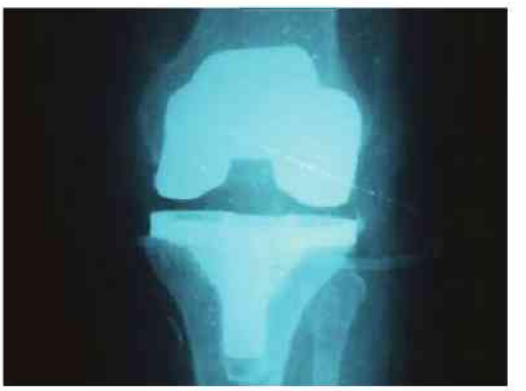
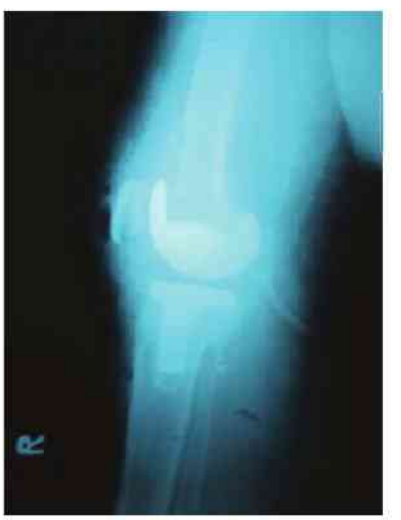


FIGURE 7.7 X-ray of hip with femur anteroposterior view showing total hip arthroplasty.



(a)



(b)

FIGURE 7.8 (a, b) X-ray of knee joint anteroposterior and lateral views showing total knee arthroplasty.

- *Arthrodesis:* This is performed to relieve pain in a joint. This is a last option where an iatrogenic fusion of joint is made, for example wrist. It provides a stable, painfree and immobile joint.
- *Rheumatoid hand:* Surgery in the rheumatoid hand is done for functional restoration. This includes the following:
  1. Tendon transfer operation for correcting deformities
  2. Excision of the metacarpophalangeal joints in case of gross deformities

JUVENILE RHEUMATOID ARTHRITIS

Juvenile rheumatoid arthritis is a clinical variant of rheumatoid arthritis in children and adolescents. This is of the following three types:

1. *Systemic type, called Still's disease:* The child is usually less than 3 years, presenting with pyrexia, malaise, rashes and polyarthritis. Other features are as follows:
   - Lymphadenitis
   - Hepatosplenomegaly
   - By puberty, stunting of growth occurs
2. *Polyarticular type:* This involves the joints of the hands and feet. Temporomandibular joints and the cervical spine are typically involved.
3. *Pauciarticular type:* This involves less than four joints, usually the large joints. This is the commonest type. Usually, medium-sized and large joints are involved.

   Clinically, the orthopaedic manifestation includes stiffening of joints, premature closure and deformities of the joints. Treatment is on the same line as for the adult type.

SERONEGATIVE SPONDYLOARTHROPATHY

Seronegative spondyloarthropathy includes the following rheumatoid-like conditions where the serum is negative for the rheumatoid factor. They are as follows:

- Ankylosing spondylitis
- Reiter's disease
- Psoriatic arthritis
- Enteropathic arthropathy

Ankylosing Spondylitis (Marie Strumpell Disease)

Ankylosing spondylitis is a chronic, progressive and crippling disease affecting the spine. The exact aetiology is unclear. Ankylosing spondylitis has been found to be more prevalent in certain races and hence shows a genetic predisposition. It is related to certain tissue types of the human leucocyte antigen (HLA) system. The majority of ankylosing spondylitis patients is found to belong to HLA-B27 group.

Aetiology

The exact aetiology of the disease is unknown. Present available evidence states that it is a genetically determined disorder with immunological abnormality.

Pathology

The disease usually starts in the sacroiliac joints and then involves spine. Synovitis of the sacroiliac joint and vertebral facet joints occurs. Other joints involved are hip and knee. In late stages, fibrous ankylosis followed by bony ankylosis occurs. Bony bridging occurs across the adjacent spines and stiff, rigid spine results. Inflammation of ligaments and tendons occurs, in particular on the site of their insertion (enthesopathy).

Clinical Features

The disease occurs in the third and fourth decades of life and is more common in males. The patients present with the following complaints:

- Diffuse back pain, worse in early morning and prolonged period of inactivity, improving with activity
- Pain and swelling of the hip/knee/ankle may occur
- In late stages, deformities of the hip and spine may be a presenting feature

On examination,

- movements of the entire spine are restricted with loss of lumbar lordosis,

- tenderness over one/both sacroiliac joints may occur.

The following clinical features are seen in the spine:

- Stiffness
- Cervical spine involvement is most commonly seen
- Wall test: When the patient stands against the wall with his back, he is unable to touch the wall with his head, due to stiff cervical spine.
- Thoracic spine if involved, chest expansion is diminished to <5 cm due to the involvement of costovertebral joints.
- Sacroiliac joints: Tenderness over both sacroiliac joints.

Extra-Articular Manifestations of Ankylosing Spondylitis

The following extra-articular manifestations are seen to be associated with ankylosing spondylitis:

- Eye—anterior uveitis, glaucoma
- Cardiovascular system—aortic valve incompetence, carditis
- Neurological—spinal cord compression may occur
- Pulmonary—restrictive lung disease, pulmonary fibrosis
- Systemic—osteoporosis, amyloidosis

Late Stages of Disease

In the late stages, the whole spine including the cervical spine is rigid and the patient is bent forwards. The classical description refers to this late stage where the patient is totally stiff and disabled with ankylosis of both hips and flexion deformity of knees. The patient is bent over with the eyes facing the ground.

One of the last joints to be affected is the temporomandibular joint.

Differential Diagnosis

1. Low backache
   - Lumbosacral strain
   - Lumbar disc disease
   - Fluorosis (posterior longitudinal ligament calcification occurs)
2. Seronegative spondyloarthritis
   - Reiter's disease
   - Psoriatic arthritis
3. Ankylosing hyperostosis (Forrestier's disease)
   - Calcification of spine, ligaments and tendons occurs
   - Sacroiliac joint involvement in minimal
   - No signs of inflammation occur

Diagnosis

Criteria for the diagnosis of ankylosing spondylitis (Rome, New York, criteria) include the following:

- Diffuse pain in the spine within the duration of some months
- Limitation of all spinal movements for some months
- Diminished chest expansion

Laboratory Findings

The blood examination shows raised ESR/CRP, and there is anaemia. The serum is negative for the rheumatoid factor. The test for HLA-B27 is positive.

Radiological Features

- X-ray examination of the cervical spine (Fig. 7.9)
- X-ray examination of the pelvis—anteroposterior (AP) view

  The earliest changes involve the sacroiliac joints. The joint margins become hazy and the joint space is widened. This is followed by subchondral erosion and sclerosis. In the final stages, the sacroiliac joints show total fusion and bony ankylosis. Calcification occurs along sacroiliac/sacrotuberous ligaments and tendinous insertions.

- X-ray examination of the lumbar spine—AP view and lateral views

  In the early stages, the lumbar spine shows, in the lateral view, filling up of the concavity of the anterior border causing a 'squaring' appearance of the vertebral body. There is loss of lumbar lordosis. Later on, extensive calcification of the

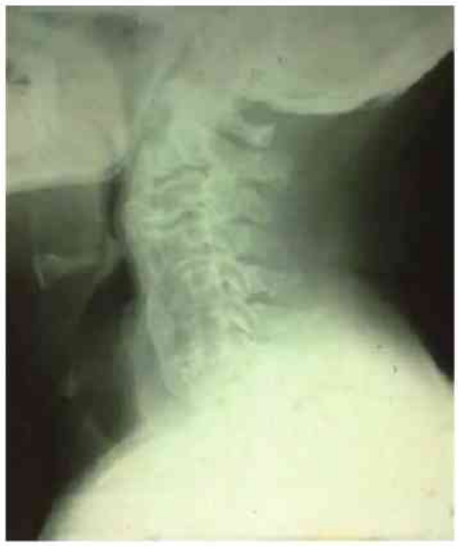


FIGURE 7.9 X-ray of cervical spine lateral view showing ankylosing spondylosis. Note the ligament calcification.

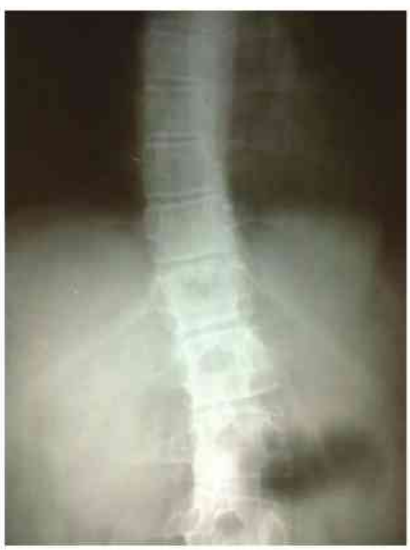


FIGURE 7.10 X-ray of dorsolumbar spine anteroposterior view showing 'bamboo spine' in ankylosing spondylitis.

anterior longitudinal ligament occurs. In the final stage, the calcification of the lateral ligaments produces the *bamboo spine* appearance in the anteroposterior view (Fig. 7.10). The interspinous ligaments are also calcified. In the later stages, the cervical spine is also involved with the fusion of posterior intervertebral joints (Fig. 7.11).

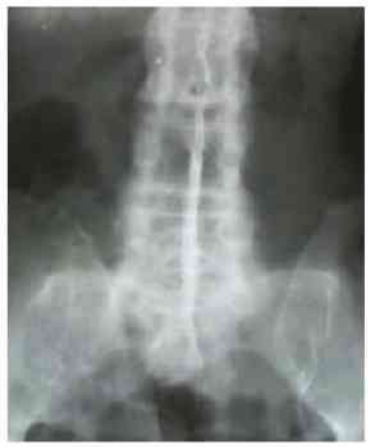
Management

Principles

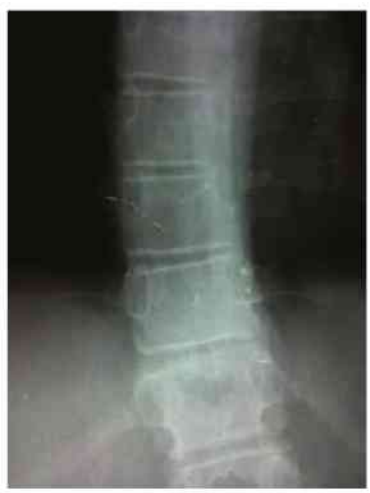
- Pain relief and control measures
- Maintenance of joint mobility
- Prevention of deformity
- Lifestyle modifications

Conservative Treatment

The patient is advised to do deep breathing exercises and exercises for the spine, be active and maintain erect posture. The patient is advised



(a)



(b)

FIGURE 7.11 (a) X-ray of lumbar spine anteroposterior view. (b) X-ray of dorsal spine anteroposterior view showing interspinous ligament calcification.

to use a hard bed and sleep supine with a single pillow. Prolonged period of inactivity is avoided. Yoga is a good option.

Surgical Treatment

When the hips are ankylosed, total hip arthroplasty or excision arthroplasty (Fig. 7.7) is indicated. When the patient has gross flexion deformity of the spine, lumbar spinal osteotomy can be done. Goel (Lucknow) has demonstrated the great benefit of osteotomies of lumbar spine for the rehabilitation of these grossly crippled patients.

Reiter's Disease

Reiter's disease is characterised by a triad of polyarthritis, urethritis and conjunctivitis. It is aseptic inflammatory arthritis associated with non-specific complaints or bowel infection. The joint condition is acute polyarthritis resembling rheumatoid arthritis. It does not cause destructive changes in the joint structures. Knee and ankle joints are commonly involved. The urethritis is non-gonococcal, but the exact organism is not known. Treatment consists of rest to the joint and anti-inflammatory drugs.

Psoriatic Arthritis

Psoriatic arthritis is polyarthritis seen in about 10% of the patients with psoriasis.

- The most common type is the one involving the distal interphalangeal joints of the hands and feet with psoriatic nail changes
- Metacarpophalangeal joints are never involved in psoriatic arthritis
- Arthritis mutilans is a severe form where there is marked destruction of joints
- Symmetrical polyarthritic type
- Oligoarthritic type
- Spondyloarthritic type

Treatment is on the same lines as for rheumatoid arthritis along with the treatment for psoriasis.

Enteropathic Arthritis

Chronic inflammatory bowel diseases such as regional enteritis (Crohn's disease) and ulcerative colitis are associated with arthritic lesions in about 10% of the cases. There is peripheral polyarthritis or involvement of the spine. The joint condition shows remissions and exacerbations along with the activity of the underlying bowel disease. Treatment of the bowel disease usually clears the joint disease also.

DEGENERATIVE ARTHRITIS

Osteoarthritis

Osteoarthritis is a chronic degenerative condition of the joints. It is not primarily an inflammatory disorder.

Aetiology

The major factor leading to osteoarthritis is 'uneven distribution of load stress' across the articulating surfaces in a joint.

Osteoarthritis can be broadly grouped into

- primary osteoarthritis,
- secondary osteoarthritis.

Primary Osteoarthritis

Primary osteoarthritis is due to the wear and tear changes that occur in old age in which weight-bearing joints such as the hips and knees are more commonly affected. It is uncommon in non-weight bearing joints such as the shoulder and elbow. Obesity is a predisposing factor.

Osteoarthritis is a progressive process affecting the articular cartilage of ageing joints. It is characterised by focal degeneration of the articular cartilage. As the articular cartilage is cyclically loaded during movements of joints, it undergoes fatigue failure leading to fragmentation of the surface and fibrillation. In the later stage, the cartilage gets completely eroded, exposing the sclerosed (eburnated) bone and subchondral cysts are also formed.

The bone undergoes reactive hypertrophy forming peripheral osteophytes. The synovial membrane undergoes hyperaemia and reactive inflammatory thickening. As there is no destructive pathology, the joint does not get ankylosed.

Secondary Osteoarthrosis

Secondary osteoarthrosis refers to arthritis occurring in a joint secondary to a previously occurring disease or disorder of the joint. It may occur in any age and involve any joint. Factors associated with secondary osteoarthritis are as follows:

1. Congenital malformation of joints
2. Traumatic alteration of articular surfaces causing articular incongruence
3. Loose bodies in the joint
4. Deformity of the joint, for example coxa vara, genu varum
5. Hip
   - Perthes disease
   - Slipped epiphysis
   - Avascular necrosis of the femoral head
6. Internal derangement of knee

Pathology

The main factor associated with osteoarthritis is an abnormal increase in the mechanical stress in some part of the articulating surfaces.

- Initial changes occur in the articular cartilage. There is an increase in the water content and a decrease in proteoglycans in the cartilage, leading to progressive cartilage destruction.
- Articular cartilage destruction leads to its softening, fraying and fibrillation.
- As soon as the articular cartilage is destroyed, the underlying subchondral bone is exposed; it looks tough, sclerotic with ivory-like smoothness (eburnation).
- Subchondral sclerosis and subchondral cysts are seen.
- Bony projections and new bone formation (osteophytes) occur within the joint.
- Thickening of the joint capsule and synovium leads to stiffness and deformity of the joint.

Clinical Features

Since the patients are usually elderly, weight-bearing joints are commonly involved.

The patient presents with

- pain that starts insidiously and increases over time,
- swelling—as a result of joint effusion,
- stiffness from the thickening of capsule and synovium,
- crepitus—while moving the joint,
- limping—due to pain and deformity of the joint.

On examination, the joint is

- tender and swollen,
- synovium—thickened,
- restricted movements,
- deformities such as genu varum/genu valgum may be present,
- joint instability may occur,
- muscle wasting around the involved joint is usually seen.

Radiological Features

X-rays of the involved joints show

- asymmetrical narrowing of the joint space,
- subchondral sclerosis,
- subchondral cysts,
- osteophytes at the joint margin,
- loose bodies within the joint,
- subluxation/dislocation at the later stage,
- deformity of joint, if present.

Differential Diagnosis

1. *Rheumatoid arthritis:* Serological testing and symmetrical joint involvement can be used for differentiation
2. *Ankylosing spondylitis:* Mainly involves spine and sacroiliac joints
3. *Reiter's disease:* Associated with urethritis and conjunctivitis
4. *Psoriatic arthritis*
   - Skin lesions are present
   - Involvement of small joints of hand and foot
5. *Avascular necrosis*
6. *Diffuse idiopathic skeletal hyperostosis*

Management

The principles of management include the following:

- Relief from pain
- Avoiding overloading of the joints

- Restoring mobility in the joint including deformity correction
- Lifestyle modification including weight reduction

Pain can be counteracted by drugs starting from paracetamol and, if not controlled, with an NSAID. Other measures such as heat therapy (waxbath and infrared ray therapy) may aid in controlling pain for short duration.

Physiotherapy

Physiotherapy is aimed at maintaining the mobility of the joints, improving muscle strength and relieving pain.

Surgical Management

- Arthroscopic debridement of the joint can be done to clear the joint of loose bodies and osteophytes
- Realignment osteotomies can be done in young patients with intense pain and marked deformity
- Replacement arthroplasty

Osteoarthritis Knee

Primary osteoarthritis of the knee is a common clinical problem in our country. The patient is usually a male above 50 years and presents with pain, swelling and restricted movements and inability to squat in the Indian toilet. The onset is insidious starting with a mild aching pain in the joint which is relieved by rest. The other main symptom is stiffness, which is maximum at the end of a long period of rest and loosens on activity for a few minutes.

The cause of secondary osteoarthritis in the knee joint is alterations in the congruity of the articular surfaces due to various lesions, such as

- malunion of fractures involving the articular surfaces of tibia, femur or patella,
- loose bodies in the joint,
- malalignment of the bones due to deformity such as genu valgum or genu varum.

Clinical Features

On examination, the joint is swollen and there may be an effusion into the joint. The synovium is thickened and tender. There is tenderness in the joint line and over the attachment of the medial collateral ligament. Movements are restricted. Crepitations are felt on movement. In late cases, there is often a genu varum deformity.

Radiological Features

The earliest change seen is the asymmetrical narrowing of the joint space and subchondral sclerosis in the medial compartment of the joint. Later, osteophytes are seen in the periphery of the articular surfaces of the femur, tibia and patella (Fig. 7.12).

Conservative Treatment

Physiotherapy including short-wave diathermy, interferential therapy or laser therapy helps in relieving the pain. *Quadriceps exercise* is very important in the treatment of the osteoarthritis of the knee joint. Intra-articular hydrocortisone may be of help in some cases.

Surgical Treatment

High tibial osteotomy is a very useful procedure and relieves pain in severe cases. It acts by realigning the weight distribution, reducing the venous stasis and improving the blood supply.

In severe cases with loss of movements, total knee replacement arthroplasty is useful (Fig. 7.8).

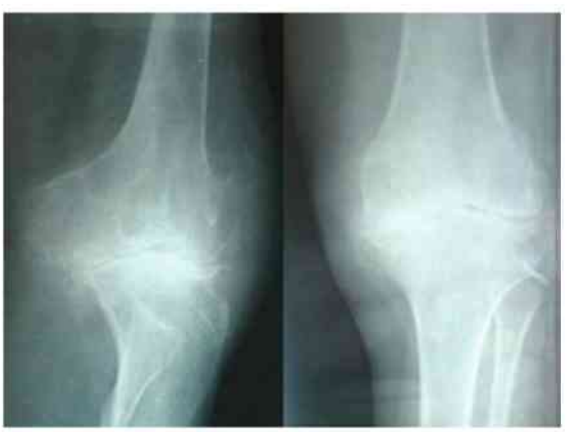


FIGURE 7.12 X-ray of knee lateral and anteroposterior views showing advanced primary osteoarthritis of the knee.

Osteoarthritis of the Hip Joint (Coxarthrosis)

Primary osteoarthritis in the hip is common in the elderly people and is often bilateral. Osteoarthritis of the hip is less common in India than in the Western countries. The lower incidence of hip involvement may be attributed to the Indian social habits of squatting and sitting cross-legged on the floor. The full physiological range of movement in the joints in squatting distributes the stresses to the whole area of the articular cartilage. The 'chair-borne' habits of the West restrict the stresses to limited areas of the articular surfaces which undergo earlier wear and tear changes leading to osteoarthritis.

Secondary osteoarthritis of the hip joint results from earlier Perthes disease, slipped epiphysis, old fracture acetabulum or a post-traumatic avascular necrosis of the head of the femur.

Clinical Features

The patient presents with pain and limitation of movements. The joint develops adduction, external rotation and flexion deformities.

Conservative Treatment

In the early stage, rest and electrotherapy followed by exercises to strengthen the abductor muscles of the hip relieve pain and improve the function. Weight reduction helps in giving symptomatic relief.

Surgical Treatment

The aim of surgery for osteoarthritis hip is to relieve pain, correct deformity and restore mobility in the joint.

- *Osteotomy:* Osteotomy at the intertrochanteric level is indicated in cases with severe pain and marked deformity but with a good range of movement.
- *Replacement arthroplasty:* When the patient is above 60 years, one should consider a hip replacement operation. THR arthroplasty has proved to be the most valuable method of relieving the pain and making the patient mobile and active.

NEUROPATHIC JOINTS (Charcot's Joint)

Aetiology

Neuropathic joint is a chronic disease of the joint characterised by extensive disorganisation/destruction of the joint with no pain or inflammatory signs. It was first described by Charcot as a complication of tabes dorsalis and hence called Charcot's joints.

The conditions that cause neuropathic arthropathy are as follows:

- Tabes dorsalis—hip, knee
- Syringomyelia—shoulder, elbow
- Leprosy—ankle and foot
- Diabetes—ankle and foot
- Iatrogenic—can involve any joints, due to repeated intra-articular injections of hydrocortisone

The cause of this gross destruction and disintegration lies in the nervous system. It is believed to result from the loss of proprioceptive and sensory impulses from the articular structure. There is loss of position sense and joint sense. The joint undergoes degeneration and destructive changes due to repeated minor trauma, when the sensory and autonomic nerve supply to the joint structures are lost (bag of bones). This explains the *total absence of pain* in the neuropathic joint.

Pathology

The joint undergoes repeated minor trauma that goes unnoticed due to lack of pain sensitivity. Three stages are described in the pathology of neuropathic joints. The first is the *stage of hydrarthrosis* with a serous effusion in the joint. In the following *stage of atrophy*, there is destruction of the affected articular cartilage and then the bone itself. The final *stage of hypertrophy* is characterised by massive hypertrophy of the bone at the periphery of the articular cartilage which can break off to form loose bodies. The joint then becomes 'a bag of bones' which is painless. Loss of stability is a very characteristic feature of the condition and later the joint is subluxated or even dislocated.

Clinical Features

In tabes dorsalis, the knee joint is commonly affected and this is described below. The patient comes with gross swelling of the knee, marked instability and abnormal movements. There is no pain in the joint. On examination, there is marked effusion in the knee. There is no tenderness in the joint. Bone ends are enlarged and irregular and multiple loose bodies may be felt. There is abnormal mobility with features of subluxation or dislocation. There will be other signs of tabes such as ataxic gait and Argyll–Robertson pupil.

Radiological Features

There is marked sclerosis of the bone ends with absorption and smoothening of the articular ends. There will be new bone formation around the joint (Fig. 7.13). These features should be differentiated from osteoarthritis.

Treatment

The main aim of the treatment is to stabilise the joint. This can be achieved by splintage of the joint and in severe cases arthrodesis can be done, not with much success.

Another better option available is bony resection and replacing it with custom mega prosthesis.

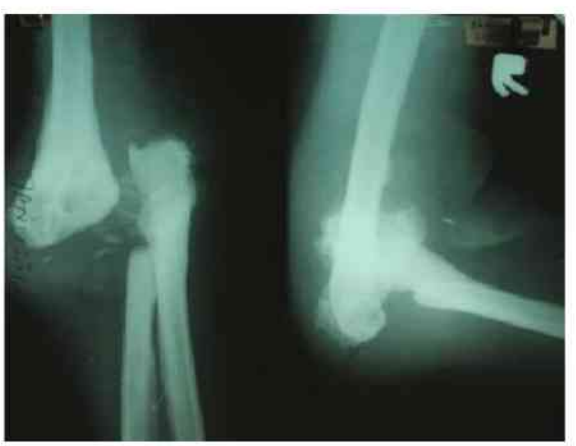


FIGURE 7.13 X-ray of elbow anteroposterior and lateral views showing neuropathic joint. Note the marked destruction of the bones.

Neuropathic Joint in Syringomyelia

Syringomyelia is a condition characterised by dissociated sensory loss in the upper limbs and spasticity in the lower limbs. This is due to cystic degeneration of the grey matter of the spinal cord around the central canal, in the cervicodorsal region.

In about 30% of the cases of syringomyelia, the joint lesion is manifested. There is a painless slow disorganisation of the shoulder joint with absorption of the upper end of the humerus. The disability is minimal and no special treatment is necessary for the joint.

Neuropathic Joint in Other Conditions

A neuropathic joint sometimes occurs in diabetes with severe neuritic symptoms. It commonly affects the small joints of the feet and the ankle. It is also seen in the joints in paralysed limbs in traumatic paraplegia.

It is seen in leprosy, where the peripheral joints in the hands and feet undergo complete disorganisation with absorption of the articular ends of the bones.

METABOLIC ARTHRITIS

Crystal Synovitis

Crystal synovitis is the synovitis caused by the deposition of crystals in the synovial membrane of joints. This includes the synovitis caused by sodium urate monohydrate (SUM) in gout and calcium pyrophosphate dehydrate crystals in pseudo-gout.

Gout

Gout is a metabolic disorder manifesting in the primary or secondary forms characterised by hyperuricaemia and joint lesions.

- *Primary gout:* It occurs in conditions where there are no obvious causes for hyperuricaemia such as drugs and myeloproliferative disorders.
- *Secondary gout:* It occurs in conditions causing secondary hyperuricaemia.

Aetiopathology

Gout is a disease due to an inborn error of uric acid (purine) metabolism. In this condition, sodium urate crystals are deposited on the articular cartilage, synovial membrane and in the periarticular tissues such as tendons and bursae. This provokes an inflammatory reaction in these tissues. These deposits often increase in size and burst through the skin to form sinuses discharging a chalky white material.

Clinical Features

It occurs mostly in men, in the third or fourth decade of life. The condition may present clinically as an acute attack or as chronic gout. The most commonly affected joint in acute gout is the metatarsophalangeal joint of the big toe. The attack is characterised by a sudden onset of excruciating pain at night with marked swelling and redness of the joint of the big toe. The attack may subside in about a week. The arthritis shows remissions and exacerbations and gradually becomes chronic.

In the chronic stage of the disease, there is a deposition of urate salts (SUM) in the periarticular tissues of the involved joint or in the subcutaneous tissues of the ear or in the olecranon bursa. These lumps are called gouty tophi. It also occurs in the knee.

After a few attacks, the patient may have persistent pain in some joints and the joints develop features of secondary osteoarthrosis. Other features are the formation of urate calculi in the kidneys with colic and haematuria ending in renal failure.

Laboratory Findings

The main finding is the raised level of serum uric acid from the normal of 3–6 mg%. The synovial fluid will show the typical slender needle-shaped urate crystals negatively birefringent on examination under the polarised microscope.

Radiological Features

Radiographs in the chronic stage may show the characteristic punched-out appearance at the periphery of the articular cartilage in the metatarsophalangeal joints. Tophi may be seen as radio-opaque shadows in the periarticular tissues. In later stages, degenerative changes will be seen in the joints with narrowing of joint spaces and sclerosis (Fig. 7.14).

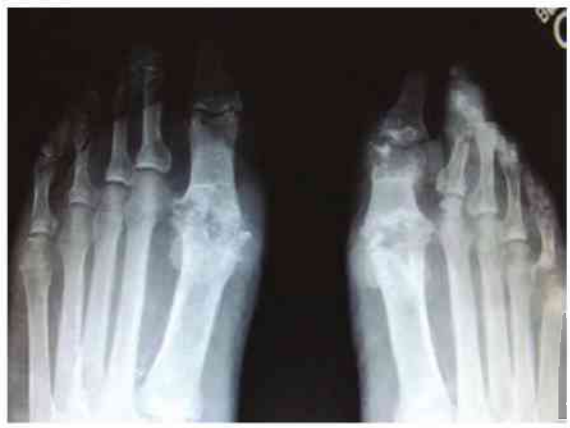


FIGURE 7.14 X-ray of foot anteroposterior view showing Gouty arthritis in big toe.

Treatment

Rest is given to the joint. Anti-inflammatory drugs such as indomethacin in high doses are given. Colchicine is a specific drug for acute gout. Drugs are given either to diminish the formation of uric acid (Allopurinol) or to act as uricosuric agents to increase the excretion of uric acid in the urine (Probenecid). At the acute stage, the joint is rested in a splint. Large gouty tophi may need surgical curetting or excision. Treatment of gout can be summarised as follows:

- *Acute gout:* NSAIDs and Colchicine
- *Chronic gout:* Allopurinol and Probenecid and NSAIDs
- *Tophaceous gout:* Excision of tophi, if necessary

Secondary Gout

Serum uric acid may be high whenever there is excessive destruction of cells and their nuclei as in multiple myeloma, leukaemia and other malignant diseases. This is found during the chemotherapeutic treatment of malignant tumours with cytotoxic drugs. The joint condition in such cases is called secondary gout.

Pseudo-Gout

Pseudo-gout is a form of arthritis due to deposition of crystals of calcium pyrophosphate dihydrate in the synovium or the articular cartilage or menisci. Clinically, the acute attacks resemble gout but the serum uric acid level is normal. The knee is the most common joint involved. Radiograph shows calcified spots in the menisci and articular cartilage. The synovial effusion shows rhomboid-shaped crystals, positively birefringent under the polarised microscope. The treatment consists of the following:

- Rest to the joint
- High-dose NSAIDs
- Aspiration of the joint and corticosteroid injection

Alkaptonuric Arthritis (Ochronosis)

Alkaptonuria is a congenital disorder of amino acid metabolism affecting the spine. The breakdown of the amino acid tyrosine does not go beyond the stage of homogentisic acid due to the absence of its oxidising enzyme. Hence, homogentisic acid appears in the urine. This condition is rare.

Clinical Features

Clinically, the first phase of the condition is simple *alkaptonuria*; the child passes normal coloured urine which, on exposure to air, turns black. In the second phase, there is a deposit of 'ochre'-coloured deposits in the bone, cartilages, tendon sheaths and the pinna of the ear. The cartilage of the pinna of the ear appears darkly pigmented. This stage is called *ochronosis*. The sclera shows characteristic slate-coloured slit-like patches on either side of the cornea. In the stage of *alkaptonuric arthritis* occurring in the fourth or fifth decade, the patient develops generalised pain and stiffening of the spine and peripheral joints such as hip and knee.

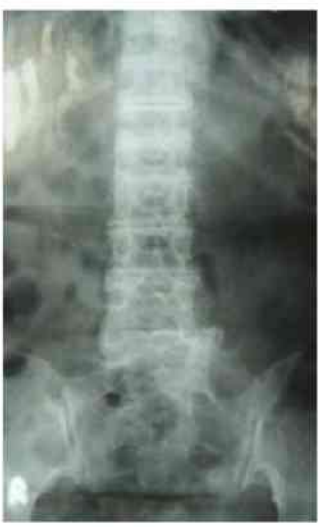
Laboratory Findings

Homogentisic acid is a reducing agent. As it gets oxidised, it forms a dark brown or black pigment. This is the basis of all the tests on urine.

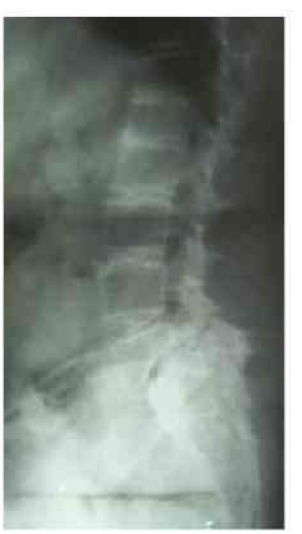
On leaving the urine to stand overnight, the top layer turns black. The urine forms a yellowish precipitate on the addition of oxidising agents such as Benedict's solution or silver nitrate. The presence of homogentisic acid in urine can be confirmed by chromatography or spectrophotometry.

Radiograph

Radiograph shows water-like calcification of the intervertebral discs (Fig. 7.15).



(a)



(b)

FIGURE 7.15 (a, b) X-ray of lumbosacral spine antero-posterior and lateral views showing intervertebral disc calcification in ochronosis.

Treatment

Treatment is mainly symptomatic as there is no specific therapy for the condition.

HAEMOPHILIC ARTHRITIS

Aetiopathology

Haemophilia is a disease characterised by a bleeding diathesis due to a defect in the clotting mechanism of the blood. It is a hereditary disease affecting males but transmitted through the females. Involvement of joints is an important complication of haemophilia.

The most common type, *Haemophilia* A, due to deficiency of factor VIII (antihaemophilia factor), forms 85% of the cases. *Haemophilia B* (Christmas disease) is due to deficiency of factor IX. Some cases show an alteration in the quality of the platelets (Von Willebrand's disease).

Clinical Features

The common orthopaedic manifestations are as follows:

- Haemorrhage into joints
- Haemorrhage into muscles
- Haemorrhage into peripheral nerves
- Haemophilic cysts

The patient is usually an adolescent boy with a history of previous bleeding episodes following cuts or tooth extraction. He presents with an acutely swollen knee or swollen hip with flexion deformity.

Haemorrhage into Joints

The joints usually involved are the knees, ankles, elbows and hips. There are sudden attacks of haemarthrosis in the joint following minimal trauma. The bleeding occurs not only inside the synovial cavity of the joint but there are also intraosseous haemorrhages occurring in the subchondral areas of the bones. After many attacks, the joint undergoes destruction of articular cartilages. In later stages, there will be fibrous ankylosis and deformities.

Haemorrhage into Muscles

Bleeding into the iliacus muscle results in a lump in the iliac fossa and flexion deformity of the hip due to ilio psoas spasm. Bleeding into the calf causes an equinus deformity. Occasionally, bleeding occurs in the forearm muscles producing a Volkmann ischaemic type of contracture.

Haemorrhage into Nerves

The nerve lesion can be caused by intraneural haemorrhage in addition to the external compression by haemorrhage into the muscle.

Haemophilic Cysts

The bleeding can form cysts (pseudo-tumour) in various tissue planes of muscles, fasciae or in the bones.

Laboratory Findings

Haematologic investigations will show a marked increase in clotting time, but bleeding time and prothrombin time are normal. It will show the deficiency of factor VIII (classical haemophilia) or deficiency of factor IX (Christmas disease). There is also anaemia, due to loss of blood.

Grading of Haemophilia

The severity of the bleeding episode corresponds to the degree of deficiency of factor VIII in blood (Table 7.2).

Radiological Features

In patients who have had many effusions of blood into the joint, there will be a generalised

TABLE 7.2 Severity of Haemophilia in Relation to Blood Factor Level

Factor VIII in Blood	Severity of Disease	Clinical Manifestation
Below 100%	Mild haemophilia	No bleeding
Below 50%	Moderate haemophilia	Bleeding after trauma or surgery
Below 5%	Severe haemophilia	Spontaneous bleeding

rarefaction of the bones with irregular cystic areas in the cancellous part of the ends of the bones. There will be epiphyseal overgrowth. In the knee, there is widening of the intercondylar notch due to splaying of femoral condyles.

Treatment

The orthopaedic management must be done in collaboration with the haematologist. The aims of the treatment are as follows:

- To stop haemorrhage
- To relieve pain
- To preserve joint mobility and prevent deformity of the joint

The mainstay of the treatment of a haemophilic patient is replacement of the deficient factor VIII. Replacement is carried out in the form of transfusion of

- fresh whole blood,
- fresh frozen plasma,
- cryoprecipitate,
- animal antihaemophilic globulin,
- human antihaemophilic globulin.

Replacement therapy by intravenous transfusion of the deficient antihaemophilic globulin has greatly improved the prognosis and has made even surgery possible and safe in these patients.

Local Treatment

In the acute stage, application of ice packs is a good first-aid measure. The joint is rested in a splint with compression bandage. When the condition is stabilised, aspiration of a tense effusion could be done. Traction to correct any deformity is given, followed by physiotherapy to mobilise the joint. Physiotherapy is given under cover of deficient coagulation factor infusions. Tendon lengthening, deformity correction and THR surgeries can be done, under cover of the coagulation factor.

Surgery

With the modern therapeutic methods available for controlling the bleeding in the haemophilic, it has become possible to perform even major surgical procedures such as arthroplasty in these patients.

REACTIVE ARTHRITIS

Reactive arthritis includes a group of miscellaneous arthritic diseases that occur as a sequence to some focus of infection in some other system. The causative organism or the antigen cannot be isolated from the synovial fluid. This includes rheumatic fever, brucellosis, post-diarrhoeal arthritis (salmonella, shigella) and post-venereal arthritis.

In rheumatic fever, the arthritis is probably a sensitivity reaction to the systemic infection with β-haemolytic streptococcus group A. Usually, one large joint is affected and it gets acutely swollen, warm and tender. It dramatically subsides in a few days leaving no residual effects on the joint and another joint gets involved. The main scarring effects of this disease occur only in the cardiac valves.

MISCELLANEOUS CONDITIONS

Villonodular Synovitis

Villonodular synovitis is an idiopathic joint lesion characterised by an overgrowth of synovial villi in the form of nodules with brownish pigmentation. It is an uncommon condition occurring in young adults, usually in the knee joint. The patient presents with swelling and marked synovial thickening in the form of nodular masses and effusion into the joint. There is no radiological abnormality. Histopathologically, there will be villous or nodular proliferation of synovial tissue and pigmentation due to presence of haemosiderin and lipid. Treatment is by synovectomy.

Synovial Chondromatosis

Synovial chondromatosis is characterised by multiple loose body formation in synovial joints. Its aetiology is unknown. It occurs commonly in the knee joint and occasionally in the hip and shoulder in young adults. The cells of the synovial membrane undergo a metaplastic change into cartilage cells, grow and get detached into loose bodies. Many of them become calcified. The patient may present with swelling of the knee with palpable masses. Radiologically, it shows numerous loose bodies. Treatment is to do a synovectomy excising most of the synovial membrane and removing the loose bodies.

Pulmonary Hypertrophic Osteoarthropathy

Pulmonary hypertrophic osteoarthropathy is a syndrome characterised by periosteal inflammation and new bone formation in the distal part of the forearm and leg, and clubbing of fingers and toes. It commonly occurs in patients with chronic intrathoracic suppuration such as lung abscess, empyema and bronchiectasis or primary malignancy in the lungs. The exact pathogenesis is not known. Radiographs show periosteal new bone formation in the distal part of the long bones. The condition is often reversed when the main disease in the lungs is treated.

Hysterical Joint

Hysterical joint is one of the manifestations of hysteria wherein the joint suddenly assumes a deformed position without any causative organic lesion. It is common in the elbow, knee and spine. It occurs in adolescent girls, young women or emotionally unstable men. In men, it often follows minimal injuries with medicolegal or workmen's compensation implications.

The joint is held rigid in flexion or extension, and any attempt to move it either way is resisted by rigidity of muscles. When the patient's attention is distracted by engaging him in conversation, the deformity can often be corrected fully but recurs promptly when left alone. The joint also relaxes into the normal position when the patient sleeps. In long-standing cases, secondary wasting of muscles and contractures of joints may develop. Treatment is by psychoanalysis and suggestion therapy.

KEY POINTS

- The stages of arthritis are synovitis, reversible arthritis, irreversible arthritis and ankylosis.
- HLA-DR4 and HLA-DRB1 are associated with rheumatoid arthritis.
- Bilateral symmetric polyarthritis is a characteristic feature of rheumatoid arthritis.
- Diagnosis of rheumatoid arthritis is based on ARA criteria.
- Seronegative spondyloarthropathy includes rheumatoid-like conditions where the serum is negative for the rheumatoid factor.
- Ankylosing spondylitis (Marie Strumpbell disease) mainly involves sacroiliac joints and spine.
- Reiter's disease is characterised by a triad of polyarthritis, urethritis and conjunctivitis.
- Psoriatic arthritis commonly involves the distal interphalangeal joints.
- The major factor leading to osteoarthritis is 'uneven distribution of load stress' across the articulating surfaces in a joint. It can be primary or secondary.
- Neuropathic joint (Charcot joint) is a chronic disease of the joint characterised by extensive disorganisation/destruction of the joint with no pain or inflammatory signs.

MULTIPLE CHOICE QUESTIONS

1. In rheumatoid arthritis, the part which is affected the most is
   a. Synovium
   b. Subchondral bone
   c. Cartilage
   d. Tendon
2. What is the pathognomic feature of rheumatoid arthritis?
   a. Rheumatoid factor
   b. Rheumatoid nodule
   c. Morning stiffness
   d. Ulnar drift of fingers
3. HLA-B27 is associated with
   a. Rheumatoid arthritis
   b. Ankylosing arthritis
   c. Rheumatic arthritis
   d. Gouty arthritis
4. In psoriatic arthropathy, the characteristic joint involved is
   a. Proximal interphalangeal joint
   b. Distal interphalangeal joint
   c. Metacarpophalangeal joint
   d. Wrist joint
5. Osteoarthritis commonly involves
   a. Proximal interphalangeal joint
   b. Distal interphalangeal joint
   c. Metacarpophalangeal joint
   d. All of the above

CHAPTER 8

Avascular Necrosis of Bone and Epiphyseal Osteochondritis

AVASCULAR NECROSIS OF BONE

Avascular necrosis of bone is defined as the death of bone tissue caused by gradual vascular impairment or sudden infarction.

General Considerations

Arrest or loss of blood supply stops the normal physiology of bone mass turnover, osteoclastic bone removal and osteoblastic bone formation. This weakens the bone and often results in structural failure.

When there is a sudden obstruction in arterial blood supply to a part of the bone, local necrosis of bone tissue occurs, as it happens in any other tissue. While the cause of the obstruction may vary, the end result is the same, giving rise to similar pathological and radiological signs but modified by varying local factors such as weight bearing and motion.

When the infarction occurs in the ends of long bones, it gives rise to asymptomatic lesions seen accidentally in radiographs as irregular opaque areas in the cancellous bone at the ends of long bones. When it occurs in the epiphysis, the avascular necrosis leads to impairment of support to the articular surfaces. Certain clinical entities have been recognised depending on the site of occurrence of the lesion.

So far, the orthopaedic diagnosis depended mainly on radiology which demonstrated gross derangements of structure, such as fractures and osteoporosis. Now it is possible to establish a *pre-radiologic diagnosis* by studying functional characteristics such as bone ischaemia or circulation disorders. This is achieved by newer diagnostic tools and procedures such as radioactive isotope study (radionuclide scintimetry) and haemodynamics of intraosseous venous and arterial circulation.

Pathogenesis

Bone ischaemia can be caused due to interruption of the arterial inflow, occlusion of venous outflow, intravascular blockage of arterioles and capillaries and increase in marrow pressure (tamponade effect). The final pathology is bone necrosis and fracture in the subchondral bone. Healing is by 'creeping substitution'.

Microscopically, the following four stages can be recognised in the lesion:

1. Stage of marrow necrosis and cell death
2. Reactive vascularisation and infiltration by new capillaries and osteoblasts over the dead trabeculae
3. Distortion of the shape by collapse and compression of the trabeculae
4. Subchondral collapse causing deformation of the articular cartilage

Clinical Features

The earliest symptom of bone necrosis is pain near the joints and limitation of movements. In the hip joint, pain of insidious onset in the groin is the presenting feature. The pain is deep

and not precisely localised but radiates down the thigh to the knee. It is worse at night. Local tenderness may be present. Restriction of hip movements may be present in some directions. In the late stage, the deformity of the femoral head and acetabulum converts the spherical joint into a cylindrical joint resulting in loss of abduction and rotations but preservation of flexion movement.

Radiological Staging

Radiologically, the typical evolution of ischaemic changes in the bone is best seen in the head of the femur as increased density in bone. This has been described by Ficat and Arlet (1980) in their classification of the four radiographic stages. Typically, the ischaemic syndrome of avascular necrosis of the head may follow a few months after reduction of a dislocation of the hip joint or treatment of fracture neck of the femur.

Stage I is a pre-radiologic stage characterised by absence of radiological signs, with the clinical features of pain and stiffness of the hip joint. MRI may show early changes. In *Stage II*, there are radiological changes such as osteoporosis or sclerotic cystic areas, but without any change in the shape of the head or joint space. *Stage III* is characterised by partial collapse and flattening of the head with a sequestrum-like appearance in a segment of the head. Increased density and deformity of the bone is the typical appearance partly due to the trabecular collapse and partly due to reactive new bone formation. *Stage IV* shows progressive secondary deterioration of the articular cartilage with joint space narrowing and osteoarthrotic changes.

Other Imaging Techniques

Radioisotope scanning is very useful in diagnosing the condition at a stage when the radiographs appear normal. *Imaging by radio nuclide* scanning with 99 Tc Antimony colloid reveals cold areas of avascular necrosis of marrow cells.

MRI shows areas of decreased bone signals where there is avascular necrosis. The hydrogen-rich fat in bone marrow sends strong signals in MRI. It is the most sensitive imaging technique to detect ischaemic necrosis, as it gives decreased signals even before X-ray changes are observed.

Functional Exploration of Bone

While radiography depicts structural changes in the bone, functional changes of the bone are recorded by a study of physiology of circulation by the following methods called functional exploration of bone:

- *Bone marrow pressure measurement:* The normal bone marrow pressure measurement is below 30 mm of mercury. Any pressure reading above 30 mm of mercury confirms the bone necrosis. This is measured by a trocar in the intertrochanteric area.
- *Intramedullary venography:* An abnormal venogram consists of absence of filling in the extraosseous venous system, diaphyseal reflux and stasis.

Core Biopsy

Core biopsy of the head helps in the detection of medullary trabecular necrosis and reactive sclerosis histologically.

Classification

Clinically, osteonecrosis is seen to occur in the following conditions:

1. Trauma
   - Fracture neck of femur
   - Fracture scaphoid (waist) fracture talus
   - Dislocation of hip, talus and lunate
   - Excessive force in reduction of the congenital dislocation of hip
2. Infection
   - Septic arthritis of hip
   - Tuberculosis of the hip (Perthes type) osteomyelitis
3. Sickle cell haemoglobinopathies
4. Caisson's disease—hip, knee, shoulder
5. Irradiation necrosis
6. Drug-induced osteonecrosis
7. Steroid therapy—immunosuppressive therapy in renal transplantation
8. Alcoholism
9. Idiopathic
   - Perthes disease

Treatment

Treatment will be described for the typical avascular necrosis occurring in the head of femur.

In early stages, bed rest and traction are used to relieve the symptoms. The use of weight relieving caliper is not very helpful. Weight reduction by dieting, withdrawal of steroids and alcohol will help to stop the progress of the disease.

In Stage II, core decompression of the head helps in pain relief and also promotes revascularisation. In late Stage II where the femoral head and joint space are normal, a muscle pedicle bone grafting operation helps to increase the blood supply and promote healing.

Late Stages III and IV with collapse and osteoarthrosis benefit by osteotomy or replacement arthroplasty operation.

SPECIFIC TYPES OF AVASCULAR NECROSIS

Fracture Neck of Femur with Osteonecrosis

Patients presenting late after the intracapsular fracture neck of femur with non-union and avascular necrosis is a common problem in our country. In the elderly, replacement arthroplasty is indicated. In adults, conservation of the avascular head with decompression, freshening the fracture ends and muscle pedicle bone grafting as advocated by Bakshi (Kolkata) have yielded good results.

Caisson's Disease

Caisson's disease is a condition occurring in deep sea divers exposed to high atmospheric pressures deep in the sea. During decompression, as the diver comes to surface, the dissolved nitrogen is released from the blood as well as from the fat globules and causes embolic vascular obstruction in various soft tissues, brain, spinal cord and bones. Extrinsic compression of the vessels within the bone marrow also causes vascular obstruction and ischaemia. The most common sites for bone infarcts are the ends of long bones. The patients develop symptoms of acute joint pain or bends, and arthritis, several months or even years after diving. Hips, shoulders and knees are commonly involved. Radiographs show areas of ischaemic necrosis in the head of the femur or near the knee.

Drug-Induced Osteonecrosis

Patients on a prolonged course of steroid therapy (for diseases such as asthma and rheumatoid arthritis) sometimes develop arthritic symptoms in the hips or shoulders. Radiographs reveal avascular changes in the heads of femur or humerus. It is also seen in immunosuppressive therapy with steroids in renal transplantation or repeated renal dialysis.

Sickle Cell Anaemia

Avascular necrosis is also seen in patients suffering from sickle cell anaemia during attacks of bone crisis. In this condition, there may be acute painful haemolytic crisis characterised by severe pain in the bones. This is due to the deformed RBC agglutinating and blocking small blood vessels in the bones.

OSTEOCHONDRITIS

Many conditions affecting the epiphyses during childhood have been recognised and described as osteochondritis. These are non-infective and non-inflammatory lesions where the exact aetiology is unclear. The radiological changes seen in osteochondritis are suggestive of vascular disturbances in the epiphyses or the ossifying centres.

Osteochondritis are considered in the following groups: osteochondritis (hip, knee, foot), osteochondritis dissecans (knee, talus, metatarsal head) and traction apophysitis (tibial tuberosity and calcaneal apophysis).

Osteochondritis in various regions bear the names of those who had described them first. The following are some of the names of the diseases and the sites involved:

- Perthes disease (upper femoral epiphysis)
- Osgood–Schlatter's disease (tibial tubercle)
- Sinding–Larsen–Johansson disease (patella)
- Sever's disease (calcaneum)

- Kohler's disease (tarsal navicular)
- Freiberg (metatarsal head)
- Scheuermann disease (vertebral epiphysis)
- Kienbock's disease (lunate)

Perthes Disease (Legg–Calve–Perthes Disease)

Perthes disease is an osteochondritis affecting the capital epiphysis of the upper end of femur. It occurs in children between 5 and 10 years and is more common in boys. It is usually unilateral but occasionally it is bilateral. Chacko (Manipal) has described a high incidence of Perthes disease in the West Coast of South India, where it is more common in girls.

The exact aetiology of this disease is still not known. The aetiological factors are believed to be either a mild trauma, a metabolic disturbance or an inflammation. The pathology is an avascular necrosis of the head of the femur resulting from interference with the blood supply, followed by the healing process of revascularisation.

Clinical Features

The child is presented with a history of periodic attacks of limping with pain. The child may have pain in the hip or referred pain in the knee. There are no signs of local inflammation. In the early stages, there will be limitation of movements with muscle spasm. Flexion will be full but the axis of movement may be altered so that on full flexion of hip and knee the thigh will rest on the outer border of the abdomen instead of directly on the anterior wall—abduction, internal and external rotations are limited. On palpation, the greater trochanter may be thickened and elevated.

Radiological Features

Radiological changes can be seen in the head of the femur, metaphysis and acetabulum. The changes in the head can be described in four stages based on the evolution of the disease. The stages are the stage of avascular necrosis (Stage I), stage of fragmentation (Stage II), stage of regeneration (Stage III) and stage of healing (Stage IV)

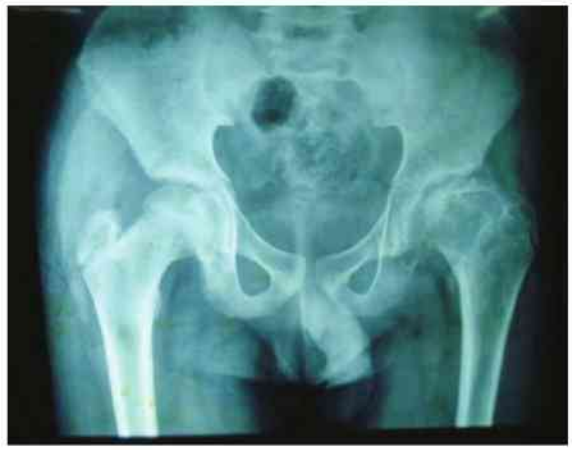


FIGURE 8.1 X-ray of the pelvis anteroposterior view showing femoral head destruction of the left hip in advanced Perthes disease of the left hip.

(Fig. 8.1). In the early stage, the joint space is widened medially (Woldenstrom's sign). In the stage of avascular necrosis, the head of femur is denser than normal and is flattened. In the stage of revascularisation, the head is flattened and fragmented. Finally, there is deformity of head with mushrooming.

The severity of radiological changes in Perthes disease is related to the extent of involvement of the femoral epiphysis. Caterall described four grades of epiphyseal involvement ranging from Group I where only the anterolateral part of the epiphysis is involved to Group IV where the entire epiphysis is avascular. He has shown that the chances of good result decrease from Group I to Group IV. He has also noted certain criteria of a 'head at risk' which indicate a worse prognosis. They are lateral displacement of the femoral head, Gage's sign (osteoporotic segment on the lateral side of epiphysis), calcification lateral to the epiphysis and horizontal orientation of the growth plate.

The radiological changes in the metaphysis are diffuse osteoporosis, cyst formation and widening of the neck. The changes in the acetabulum include osteoporosis and alteration of the acetabular contour.

Differential Diagnosis

Unilateral cases have to be differentiated from tuberculosis and juvenile rheumatoid arthritis.

Bilateral cases have to be differentiated from cretinism, multiple epiphyseal dysplasia, mucopolysaccharidosis and sickle cell anaemia.

Prognosis

The results are poor in children over 9 years of age at the time of diagnosis, when more than half of the epiphysis is avascular, when there is loss of containment of the head inside the acetabulum and in girls.

Treatment

Pathologically, the disease is a self-limiting condition. The aim of treatment should be prevention of deformity of the femoral head and promotion of containment of the head inside the acetabulum.

In the early stage of pain and muscle spasm, the child is put to bed rest with skin traction. When the muscle spasm is relieved, the child is allowed to walk with a weight-relieving caliper. Avoiding weight-bearing stresses prevents femoral epiphyseal deformation. This caliper is continued till the head of the femur shows good revascularisation.

Cases which show lateral subluxation of the head or evidence of deformation will need restoration of normal containment of the head. This is done by a special orthotic appliance which keeps the leg in abduction and internal rotation.

Surgical treatment such as femoral osteotomy or innominate osteotomy (Salter and Pemburton) aims to promote better containment of the head of the acetabulum.

Complications

In untreated cases, there is incongruity between the femoral head and the acetabulum. This results in secondary osteoarthrosis of the hip joint in the third or fourth decade.

Osgood–Schlatter's Disease

Osgood–Schlatter's disease is an osteochondritis of the apophysis of the tibial tubercle. It occurs in boys in the age group of 10–15 years. They present with pain in front of the knee with no history of injury. There is swelling and tenderness over the tibial tubercle. There are no signs of inflammation and the range of movements is full. Radiologically, there is increased density and fragmentation of the upper tibial apophysis. The condition is self-limiting and responds to symptomatic treatment.

Osteochondritis Dissecans

Osteochondritis dissecans is a condition affecting the articular surface of certain joints such as knee, ankle and elbow. There is separation of an area of the articular cartilage along with the subchondral bone. Over a period of time, this gradually separates and forms a loose body. It occurs in the age group of 15–20 years. The most common sites are the inferolateral aspect of the medial femoral condyle in the knee, the superomedial aspect of the talus in the ankle and the capitellum in the elbow. The exact cause is not clear. Traumatic and ischaemic theories have been proposed.

Clinical Features

In the knee, the patient presents with recurrent attacks of pain, swelling and a sense of the knee giving way. There may be tenderness in the medial condyle of femur, with the knee partially flexed.

In the stage of the loosened fragment, the patient presents with pain and recurrent attacks of locking of the knee. The locking episode is followed by an effusion into the joint and the history resembles that of a patient with torn semilunar cartilage. There will be no tenderness in the joint line. Very often, the patient states that he could feel something moving inside the joint and he could even demonstrate the palpable loose body. The loose body changes its position inside the joint and is called 'joint mouse'.

Radiological Features

In the early stage, a saucer-shaped defect with a fragment of denser bone over the defect is seen in the medial femoral condyle. This is best seen in the intercondylar tunnel view (Fig. 8.2). In the

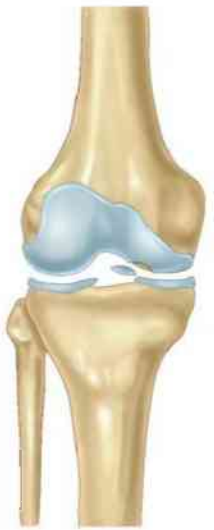


FIGURE 8.2 Diagrammatic representation of knee joint showing osteochondritis dissecans medial femoral condyle.

late stage, radiograph may show the loose body lying free and a crater defect in the articular surface of the femoral condyle.

Arthroscopy

Arthroscopy of the knee is useful to locate the site of lesion and also note the degree of separation of the fragment.

Treatment

If the fragment is only partially detached, it should be drilled and fixed with a pin. Loose fragments are best removed. More recently, this lesion is being treated by arthroscopic surgery and mosaicplasty. Delayed treatment causes damage to the articular surfaces which leads to early osteoarthrosis of the joint.

KEY POINTS

- Avascular necrosis of bone is defined as the death of bone tissue caused by gradual vascular impairment or sudden infarction.
- Radiologically, ischaemic changes in bone have been classified into four stages by Ficat and Arlet.
- Imaging by radionuclide scanning with 99 Tc Antimony colloid reveals cold areas of avascular necrosis of marrow cells.
- Perthes disease or Legg–Calve–Perthes disease is osteochondritis of capital epiphyses of the upper end of femur.
- Caterall has given four grades of epiphyseal involvement in Perthes disease.

MULTIPLE CHOICE QUESTIONS

1. Osteonecrosis of femoral head is seen in
   a. Sickle cell anaemia
   b. Caisson's disease
   c. Fracture
   d. All of the above
2. Which is not true about Perthes disease?
   a. Not painful
   b. It manifests at puberty
   c. Involves head of femur
   d. Viral aetiology
3. Perthes disease, all of the following movements are restricted except
   a. Abduction
   b. Adduction
   c. Shortening
   d. Internal rotation
4. Osteochondritis dissecans occurs at
   a. Lateral surface of lateral condyle
   b. Medial surface of lateral condyle
   c. Medial surface of medial condyle
   d. Lateral surface of medial condyle
5. Osgood Schlatters disease affects
   a. Upper tibia
   b. Lower tibia
   c. Distal femur
   d. Proximal femur

CHAPTER 9

Tumours of Bone

The incidence of bone tumours is very low (1–1.5% of the total malignancies in the body). In clinical practice, true neoplasms of bone will have to be differentiated from hamartomas and reactive bone lesions.

The group of lesions which show abnormal proliferation of cell, which soon mature and stop proliferation, is called hamartomas. These are really benign growth disorders and examples are osteochondroma, osteoma and enchondroma.

Bone also reacts to many types of injury by new bone formation, and this *reactive* or *reparative bone* can also simulate neoplasia histologically.

Clinically, certain non-neoplastic lesions present as swellings in the bone simulating a tumour, for example solitary bone cyst, fibrous dysplasia, brown tumour lesion in hyperparathyroidism.

Bone being a tissue of mesenchymal origin, the abnormality of cell growth can produce a tumour containing not only bone but also cartilage and fibrous tissue in varying degrees. All these are derived from the primitive multipotent mesenchymal cells. There is also a group of myelogenic tumours arising from the derivatives of the marrow reticular tissue.

Bone tumours can be grossly divided into true bone tumours and tumour-like conditions of bone. True bone tumours can be benign or malignant. A malignant bone tumour is progressive, invading, metastasising and non-maturing proliferation of the cells of the bone tissue.

WHO CLASSIFICATION OF BONE TUMOURS

WHO classification of bone tumours is discussed in Table 9.1.

TUMOUR-LIKE LESIONS OF BONE

1. Reactive bone lesions simulating tumours
   - Osteoid osteoma
   - Benign osteoblastoma
   - Non-osteogenic fibroma
2. Hamartomas of bone
   - Osteoma
   - Osteochondroma
   - Enchondroma
3. Cystic lesions in bone
   - Solitary bone cyst
   - Aneurysmal bone cyst

SECONDARY MALIGNANT TUMOURS IN BONE

Secondary malignant tumours primarily arise from the following:

- Thyroid
- Breast
- Bronchus
- Kidney
- Prostate

TABLE 9.1 WHO Classification of Bone Tumours

Types	Benign	Malignant	Intermediate/Indeterminate
Bone-forming tumours	• Osteoma • Osteoid osteoma and osteoblastoma	• Osteosarcoma a. Central (medullary) b. Surface (peripheral) Parosteal Periosteal High-grade surface	• Alive (malignant) osteoblastoma
Cartilage-forming tumours	• Chondroma a. Enchondroma b. Periosteal (Juxtacortical) • Osteochondroma a. Solitary b. Multiple hereditary • Chondroblastoma (epiphyseal chondroblastoma) • Chondromyxoid fibroma	• Chondrosarcoma (Primary/Secondary) • Differentiated chondrosarcoma • Juxtacortical chondrosarcoma • Mesenchymal chondrosarcoma • Clear cell chondrosarcoma	
Giant-cell tumour	• Osteoclastoma		
Marrow tumours (round-cell tumour)		• Ewing's sarcoma of bone • Neuroectodermal tumour of bone • Malignant lymphoma of bone (primary/secondary) • Myeloma	
Vascular tumours	• Hemangioma • Lymphangioma • Glomus tumour (glomangioma)	• Angiosarcoma • Malignant haemangiopericytoma	• Haemangioendothelioma • Haemangiopericytoma
Other connective tissue tumours	• Benign fibrous histiocytoma • Lipoma	• Fibrosarcoma • Malignant fibrous histiocytoma • Liposarcoma • Malignant mesenchymoma • Leiomyosarcoma • Undifferentiated sarcoma	• Desmoplastic fibroma
Other tumours	• Neurilemmoma • Neurofibroma	• Chordoma • Adamantinoma	

DIAGNOSIS OF BONE TUMOURS

Diagnosis is based on the following:

- Clinical examination eliciting the history and physical signs is the first essential step
- Imaging from plain radiography to modern imaging technologies
- Laboratory investigation
- Biopsy

Role of Imaging Techniques in Diagnoses

Plain Radiography

Plain radiography still has a big role to play in arriving at accurate diagnosis, on the basis of the following five parameters:

1. The anatomical location of the lesion: The tumour can be grouped according to the anatomical location as follows:
   - Diaphyseal (e.g. Ewing's sarcoma, lymphoma, adamantinoma)
   - Diaphyseometaphyseal (e.g. osteosarcoma)
   - Metaphyseal (e.g. osteosarcoma)
   - Metaphysioepiphyseal (e.g. giant cell, tumour [GCT], aneurysmal bone cyst)
   - Epiphyseal (e.g. chondroblastoma)
2. The anatomical borders of the tumour
   - Well-defined border, a narrow transitional area and a reactive sclerosis mean a benign lesion
   - Poorly defined margins indicate a malignant lesion
3. Pattern of bone destruction
   - Geographic pattern (slow growth)
   - Moth-eaten pattern (moderate growth)
   - Permeative pattern (rapid growth)
4. Matrix formation: New bone formation is another parameter to be observed and may vary from woolly masses to dense sclerosis.
5. Periosteal reaction is seen as non-continuous and often laminated (e.g. Sunray appearance—osteosarcoma, onion peel appearance—Ewing's sarcoma)

Computed Tomography

Computerised tomography is useful in the management at all stages, from initial diagnosis to final management and evaluation of dissemination. It helps in evaluating the integrity of the cortex and demonstrates the extraosseous extension. It also helps in early detection of pulmonary secondaries and in detecting calcification and their rim of reactive new bone formation.

Magnetic Resonance Imaging

Magnetic resonance imaging replaced CT scan as the study of choice to determine the size, extent and anatomical relationships of bone and the surrounding soft tissues. Magnetic resonance imaging is a sensitive investigation to assess intramedullary and soft tissue extension of the tumour.

Technetium Bone Scan

Tc 99m bone scan is essential for pre-biopsy staging studies and also for determining dissemination and activity of the lesion.

Angiogram

Angiogram is done before surgery for operation planning, treatment and embolisation.

Laboratory Investigations Required for Diagnoses of Bone Tumours

Blood investigations such as RBC count, Hb, ESR, VDRL, calcium, inorganic phosphates and alkaline phosphatase must be done. In patients above 40 years, presenting with osteolytic lesions in bone, serum albumin globulin estimation and electrophoretic pattern should also be done to exclude multiple myeloma. Prostate specific antigen is done for prostate carcinoma. Urine examination is done for Bence Jones protein. Serum urea, nitrogen and creatinine levels are done to rule out renal tumours.

Tissue Biopsy for Diagnoses of Bone Tumours

Biopsy is the most crucial procedure in the diagnosis of musculoskeletal lesions. The appropriate treatment cannot be initiated until the correct tissue diagnosis is available. The following types of tissue biopsy are performed to diagnose bone tumours:

- *Open biopsy:* This has been the conventional method requiring an incision under operating room conditions. The periphery of any malignant tumour is the most viable and diagnostic portion of the tumour, whereas the central region is often necrotic. Nowadays, open biopsy is being done only for selected cases with specific indications.
- *Closed biopsy:* Here no incision is required and the tissue specimen is obtained through

a skin puncture by a needle or trephine. It minimises tissue contamination. Nowadays, trocar biopsy is used widely. It cannot be done in osteosclerotic bone tumours.

- *CT-assisted needle biopsy:* Accurate localisation of the tumour in sites such as the spine and pelvis by CT enables needle biopsy of these lesions. This avoids major surgical procedures for biopsy purposes. This can also be done with an image intensifier.

SURGICAL STAGING SYSTEM OF BONE TUMOURS

Enneking has evolved a system of surgical staging of bone tumours. This is based on the histological grade (G), anatomical location (T) and the presence of secondary metastasis (M). This system (Table 9.2) provides guidelines and protocol of surgical and other therapy as well as for assessing prognosis.

Histological Grade (G)

Grade I lesions are those with low-grade malignancy. Histologically, they show few mitosis, little anaplasia and cellular atypia. Grade 2 is of high-grade malignancy. It shows poorly differentiated cells with marked atypism and pleomorphism. Cells are hyperchromatic with more frequent mitosis.

Anatomical Site (T)

Anatomical site is the anatomical location of the tumours in relation to the tissue compartments. T1 lesion is intracompartmental confined within the cortical boundaries of a bone such as femur or tibia. In extracompartmental (T2) lesions, the natural barriers are crossed and the lesion involves more than one compartment.

Metastasis (M)

MO and M1 refer to the absence or presence of metastasis.

Benign Lesions

Benign lesions may be

- latent,
- active,
- aggressive.

Arabic numerals 1, 2 and 3 are used to denote benign lesions. Stage 1 is a benign static lesion which does not progress. Stage 2 is a benign active lesion which progresses and expands bone. Stage 3 is a benign aggressive lesion which breaches bone.

Malignant Lesions

Roman numerals I and II are used for low-grade sarcomas and high-grade sarcomas, respectively. Intracompartmental lesion is denoted as A and extracompartmental lesion as B.

OPERATIVE MANAGEMENT OF BONE TUMOURS

A method of classification of surgical procedures based on the surgical plane of dissection in relation to the tumour and the method of accomplishing the removal has also been developed.

TABLE 9.2 Surgical Staging System of Bone Tumours

Stage	Grade	Site	Metastasis
lA	Low (G1)	Intracompartmental (T1)	None (MO)
1B	Low (G1)	Extracompartmental (T2)	None (MO)
IIA	High (G2)	Intracompartmental (T1)	None (MO)
IIB	High (G2)	Extracompartmental (T2)	None (MO)
IIIA	G 1 or G2	Intracompartmental (Tl)	Yes (Ml)
IIIB	G 1 or G2	Extracompartmental (T2)	Yes (Ml)

Surgical Margins

The surgical margin is defined as 'the volume of normal tissue removed with the tumour'. It can be classified as follows:

- *Intralesional margin:* The plane of surgical dissection is within the tumour.
- *Marginal margin:* The plane of surgical dissection is through the pseudo-capsule.
- *Wide margin:* The plane includes a cuff of normal tissue surrounding the tumour.
- *Radical margin:* The entire compartment in which the tumour lies is removed.

Surgical Procedures

The surgical management of the benign and malignant lesion varies accordingly.

Benign Lesion

Many benign bone tumours are treated adequately by curettage. It gives better functional result at the cost of a high rate of local recurrence. Extended curettage includes the use of adjuvants such as liquid nitrogen, phenol, bone cement or thermal cautery to extend destruction of the tumour cells.

Malignant Lesions

Amputation and limb salvage surgery are the two ends of treatment options for malignant bone lesions.

Amputation

Amputation is rarely performed nowadays. Early detection of the lesion and current concepts in the biological behaviour of tumours, clinical staging, tumour surgery, adjuvant chemotherapy and radiotherapy have made local tumour clearance possible without amputation.

Limb Salvage (Saving) Surgery

Limb-saving surgeries are currently feasible options in the management of bone tumours. The aim of limb salvage in bone tumour management is to eradicate the disease, restore the integrity of the skeletal system and preserve a limb with useful function.

Reconstruction of the skeletal defect after local tumour clearance can be done by osteoarticular allografts or prosthetic replacement. The allografts are derived from the cadavers through bone banks. Prosthetic replacement by custom mega prostheses made of stainless steel or titanium is used for replacement of the excised bone and the adjacent joint.

REACTIVE BONE LESIONS

Osteoid Osteoma

Osteoid osteoma is a reactive bone lesion simulating a tumour. The exact pathogenesis of this lesion is not clear.

Clinical Features

It occurs in children and young adults in the age group of 10–30 years. It is common in males, mostly in the cortical areas of femur, tibia and vertebra. It presents as a localised pain in the bone which progressively increases in course of time. The pain is worse at night and typically responds to salicylates. Gradually, a tender swelling develops at the site with no inflammatory signs. The lesion in the lamina or pedicle of a vertebra presents as a painful scoliosis.

Radiological Features

Radiograph shows a small osteolytic lesion in the cortex or subperiosteal region surrounded by dense sclerotic areas (Fig. 9.1). There is a small dense spot in the centre of the area called the nidus. This may be better demonstrated in a CT scan.

Histological Features

The nidus consists of osteoid tissue or woven bone lying in a fibrovascular stroma. At the periphery, there are spicules of calcified osteoid tissue.

Treatment

Treatment consists of total excision of the lesion. More recently, absolute alcohol injection or laser ablation has been used with success.

Osteoblastoma (Giant Osteoid Osteoma)

Benign osteoblastoma is a lesion similar to osteoid osteoma but of a size larger than 2 cm. It occurs

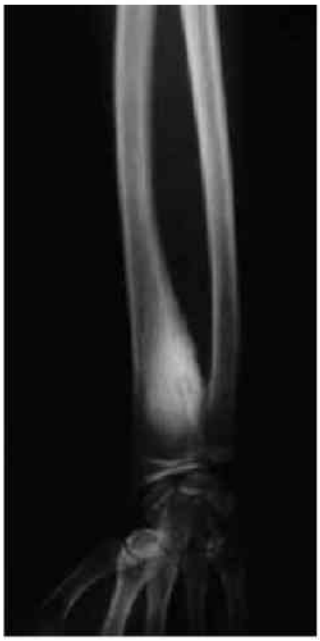


FIGURE 9.1 X-ray of right forearm anteroposterior view showing osteoid osteoma in distal radius. Note the presence of radius.
Courtesy: Dr Ajay Puri.

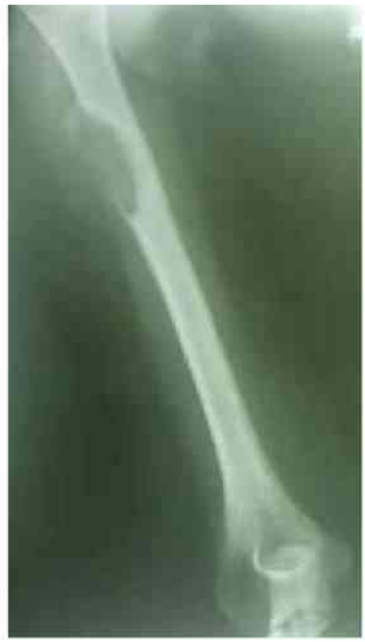


FIGURE 9.2 X-ray of humerus anteroposterior view showing non-osteogenic fibroma (note the eccentric location in the metaphyseal region).

in children and young adults in the second and third decades.

Clinical Features

Pain is the usual presenting feature. It occurs in the spine and the epiphysis of the long bones. It can present as a swelling at the end of femur. In the vertebra, it can present with neurological signs of cord or root compression.

Radiologically, there is an expanding osteolytic lesion with a thin cortex of sclerosis. It may resemble an aneurysmal bone cyst. Microscopically, there is vascular connective tissue stroma with numerous osteoblasts. These osteoblasts do not show abnormal mitotic activity. Treatment consists of curettage or excision of lesion.

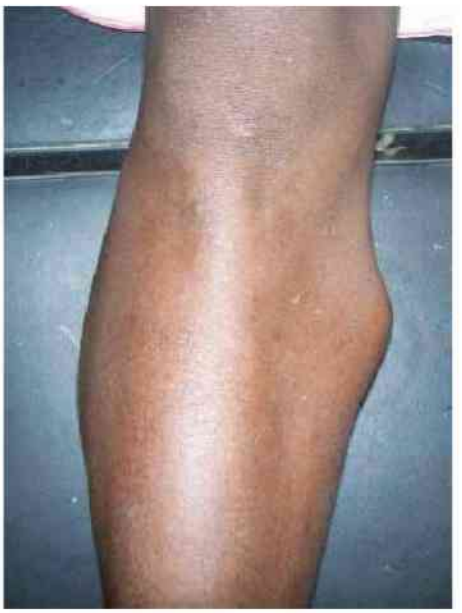
Non-Osteogenic Fibroma (Histiocytic Fibroma)

Non-osteogenic fibroma is an eccentric fibrous lesion occurring in the metaphysis of long bones in children. The lesion is found in children between 10 and 15 years in the distal femur or proximal or distal tibia. It appears first in the metaphysis and expands slowly into the cancellous tissue. Radiograph reveals an eccentrically placed osteolytic defect which is clearly demarcated by sclerotic margin (Fig. 9.2). Microscopically, the lesion is composed of cellular whorled connective tissue with spindle-shaped cells interspersed with multinucleated giant cells.

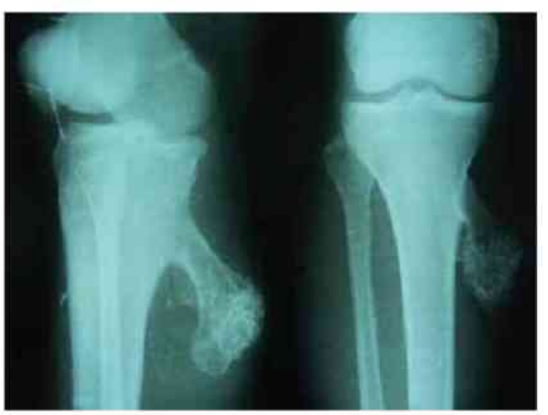
HAMARTOMAS

Osteoma

Osteoma is a benign lesion arising usually from the cortical layer of flat bones of the skull, face and orbit. It occurs in men in the fourth and fifth decades of life. The tumour is ivory hard and slow growing (ivory osteoma). It is symptomless except in rare cases when it presses on cranial nerves near the foramen. Excision is done if it produces symptoms.



(a)



(b)

FIGURE 9.3 (a) Clinical photograph of osteochondroma in tibia. (b) X-ray of right leg—lateral and anteroposterior views—showing solitary exostosis.

Osteochondroma (Solitary Exostosis)

Osteochondroma is a bony swelling arising from the metaphyseal ends of long bones (Fig. 9.3a). This is the most common among the tumour-like lesions of bone. It is first noticed during the age of 5–15 years. Its common sites are the distal end of the femur, proximal end of tibia and upper end of humerus.

Pathogenesis

Osteochondroma is a growth disorder (hamartoma). During growth, a small mass of cartilage cells from the periphery of the epiphyseal plate migrates and continues to grow in a transverse direction. It matures into bone but continues to have a cartilaginous cap which contributes to its further growth till the completion of epiphyseal fusion.

Macroscopically, there is a cartilaginous cap over the bulbous end in lesions removed before epiphyseal fusion. Microscopically, it shows normal cortical and cancellous bone with a cartilaginous covering. Structurally, osteochondroma resembles the exostosis occurring in diaphyseal aclasia (multiple exostoses) which is a developmental disorder (Fig. 3.1).

Clinical Feature

The adolescent child presents with a complaint of a hard swelling, usually near the knee. Examination shows a hard bony swelling arising from the medial or lateral aspect of the knee. The swelling is felt to arise from the bone near the metaphysis and then projects away from the joint. There are two types:

1. Pedunculated with a narrow base
2. Sessile with a broad base

Complications

The patient may also present with any of the complications. The complications include pain due to inflammation in an adventitious bursa overlying the swelling, difficulty in the movement of the neighbouring joint due to mechanical obstruction, pressure effects on nerves (e.g. exostosis of the upper end of fibula compressing lateral popliteal nerve), pressure effect on vessels causing compression, fracture of the exostosis itself and rarely malignant change of the exostosis.

Radiological Features

The characteristic radiological features of exostosis are that the cortex and the medulla of the stem of the exostosis are continuous with the cortex and medulla of the main bone. The distal end is bulbous. Radiograph shows the site and shape of the exostosis (Fig. 9.3b).

Pathological Features

Treatment

A small asymptomatic osteochondroma needs no treatment. Exostosis causing complications will need extraperitoneal excision of exostosis.

Enchondroma

Enchondroma is a benign cartilaginous growth disorder of hyaline cartilage. The lesion occurs in the short long bones such as metacarpals and phalanges; the commonest location is phalanges of the hand. It is usually symptomless. A mild trauma may result in a pathological fracture and the lesion is then discovered on the radiograph (Fig. 3.2). The lesion should be curetted and bone grafted.

The generalised manifestation of such enchondroma is called Ollier's disease (enchondromatosis) and is a developmental disorder. (Refer to Chapter 3.)

CYSTIC LESIONS IN THE BONE

Unicameral (Simple) Bone Cyst

Solitary bone cyst, otherwise known as unicameral bone cyst, is a true cystic lesion of bone where there is a fluid-filled cyst lined by epithelial-like cells. It occurs in the ends of long bones in children and adolescents. The common sites of occurrence are the proximal ends of the humerus (80%) and femur (20%).

Clinical Features

Clinically, the cyst is asymptomatic till the child sustains a pathological fracture. Occasionally, the patient presents with pain in the region.

Radiological Features

The cyst is usually seen as a radiolucent area in the metaphyseal region near the epiphyseal plate. The cyst may occupy the whole width of the bone and cause thinning of the cortex (Fig. 9.4). A fracture may be seen across the cyst. As the child grows, the cyst is seen to shift away from the metaphysis towards the middle of the shaft.

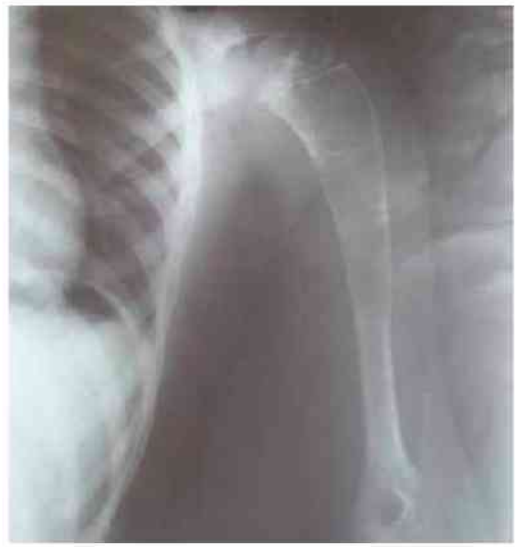


FIGURE 9.4 X-ray of left humerus anteroposterior view showing simple bone cyst. Note its central location in metaphysis and thinning of the cortex.

Pathological Features

The cyst usually contains serous or serosanguinous fluid, and the inner wall shows bony ridges and is lined by a thin membrane. Microscopically, the lining membrane shows connective tissue with scattered giant cells. The exact pathogenesis is not clear. It is believed to be a haemorrhagic cyst due to mild trauma and intraosseous bleeding.

Treatment

The conventional treatment is surgical curettage and packing with bone grafts. Small cysts sometimes heal after fracture. More recently, injection of methyl prednisolone acetate into the cyst has been found to produce healing.

Aneurysmal Bone Cyst

Aneurysmal bone cyst is a solitary rapidly progressive expansile lesion occurring in the metaphysioepiphyseal area of long bones and the pedicle and lamina of vertebra. It commonly occurs in the second and third decades. The exact pathogenesis is not known. It may be an arteriovenous malformation in the bone resulting from trauma. It may also be induced on a pre-existing neoplastic pathology.

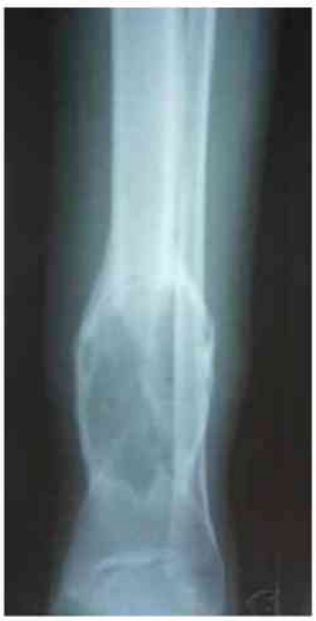


FIGURE 9.5 X-ray of left leg anteroposterior view showing aneurysmal bone cyst in tibia: expansile osteolytic lesion with thin wall, containing blood-filled cystic cavities.

Clinical Features

Clinically, the patient presents with pain and swelling in the affected bone which is tender. Pathological fracture through the cyst causes increased pain and local tenderness. A lesion in the spine can present with neurological signs.

Radiological Features

X-ray shows radiolucent ballooned-out eccentric lesion in the metaphyseal region of a long bone. There is a thin shell of cortical bone covering the lesion (Fig. 9.5).

Pathology

Macroscopically, it appears as a honeycomb of blood-filled cavities lined by fibro-osseous cystic tissue. Microscopically, there are fibrous tissue septae with some osteoid in it. There are a variable number of giant cells, xanthoid cells and altered blood pigment.

Treatment

Surgical curettage followed by adjuvant treatment with phenol or cryotherapy and bone grafting is the preferred treatment. Aggressive lesions are treated by resection and reconstruction.

OSTEOGENIC TUMOURS

Osteosarcoma

Osteosarcoma is a malignant primary bone tumour arising from the multipotent mesenchymal tissue of bone, characterised by direct formation of bone or osteoid by the proliferating tumour cells.

Incidence

It is the most common primary malignant tumour of osteoid tissue. It occurs in the young, between 10 and 20 years of age. The sites of occurrence are the distal end of the femur, the proximal end of the tibia and the proximal end of humerus in the metaphysis.

Clinical Features

Pain is the initial and dominating symptom. After some weeks, a bony swelling appears (Fig. 9.6) and progressively increases in size.

On examination, the swelling is fusiform, the skin is stretched, shiny and vascular, with prominent veins. The swelling is warm to touch and may also show pulsation if the tumour is very vascular. It is firm to hard in consistency with areas of softening where the tumour has invaded the soft tissues. In the late stages, the tumour fungates (Fig. 9.7). The patient's general health deteriorates with anaemia, loss of weight and cachexia. The patient develops pulmonary symptoms due to secondaries.

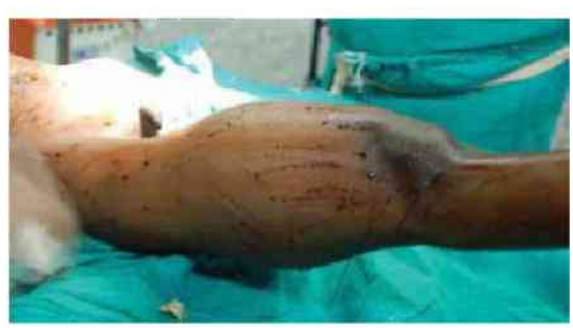


FIGURE 9.6 Clinical photo of osteosarcoma of the distal end of right femur.

FIGURE 9.7 Clinical photo of fungating osteosarcoma in the distal femur.

Radiological Features

The tumour arises at the metaphyseal region of the bone, either centrally or over the cortex. There are mottled areas of rarefaction with areas of osteosclerosis. When it extends beyond the cortex, the periosteum is raised and there is new bone formation in lines at right angles to the cortex. This causes the 'sunray' appearance in the radiograph (Fig. 9.8). There is also reactive new bone formation subperiosteally at the junction of the lifted periosteum and the normal bone. This new bone is called *Codman's triangle* (Fig. 9.9). The radiographs may also show a pathological fracture (Fig. 9.10).

Radiograph of the chest must be taken. It may show round shadows caused by secondary deposits (Cannon ball appearance) (Fig. 9.11).

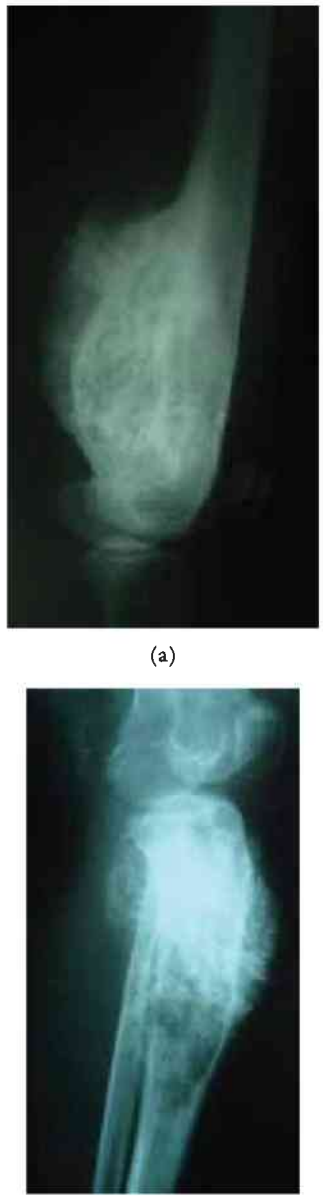
MRI scan shows the intramedullary and soft tissue extension of the tumour (Fig. 9.12).

Laboratory Findings

Serum alkaline phosphatase might show a significant increase in the level.

Biopsy

In all cases, the diagnosis should be established by a biopsy. This should be done at the growing periphery of the tumour which gives the typical microscopic appearance. Biopsy should not be taken from the centre of the lesion as it contains only necrotic cells.



(a)

(b)

FIGURE 9.8 Sunray appearance in osteosarcoma. (a) X-ray of femur lateral view and (b) X-ray of leg lateral view.

Pathology

- *Macroscopic appearance:* The tumour is located in the metaphyseal region and reaches the subperiosteal area through the cortex. It is greyish-white in colour. Its consistency may

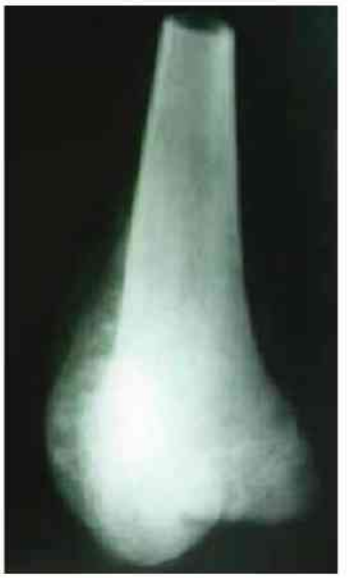


FIGURE 9.9 X-ray of distal femur demonstrating osteosarcoma with Codman's triangle.

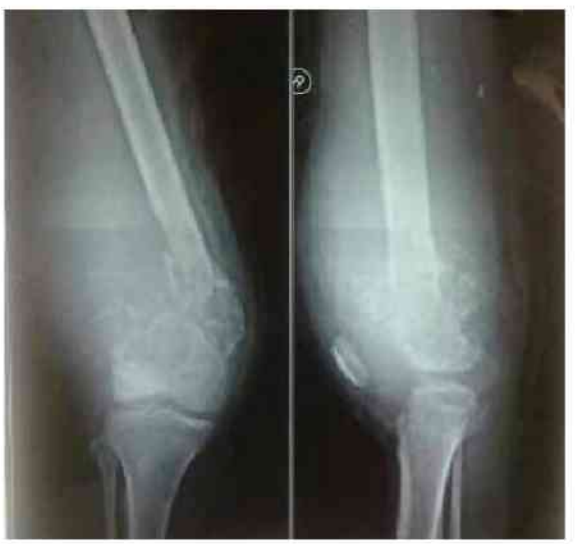
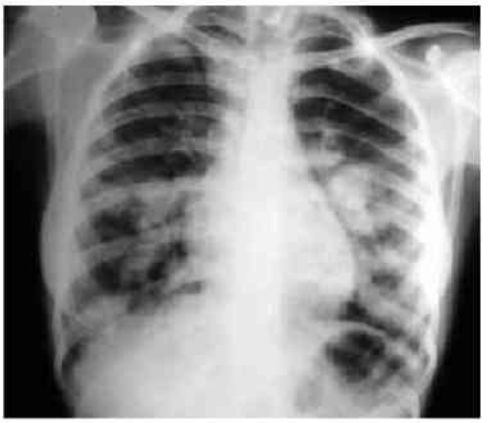


FIGURE 9.10 X-ray of right femur—anteroposterior and lateral views—demonstrating osteosarcoma with pathological fracture.



(a)

(b)

FIGURE 9.11 X-ray of chest showing (a) secondaries and (b) cannon ball deposits.
Courtesy: (a) Dr Ajay Puri.

be hard and fleshy with streaks of tumour bone or it may be soft and vascular with areas of haemorrhage (Fig. 9.13). The tumour edge stops at the epiphyseal cartilage and does not break through it. The tumour also extends into the medullary canal. In advanced cases, the tumour breaks through the periosteum and invades the soft tissue and even fungates through the skin.

- *Microscopic appearance:* The most characteristic features are the anaplastic sarcomatous stroma with newly formed woven bone and the presence of malignant osteoid. It may also show areas of malignant cartilage and fibrous tissue. The stromal cells are spindle-shaped osteoblasts showing excessive mitosis, pleomorphism and hyperchromatism. There may be areas of haemorrhage and necrosis.

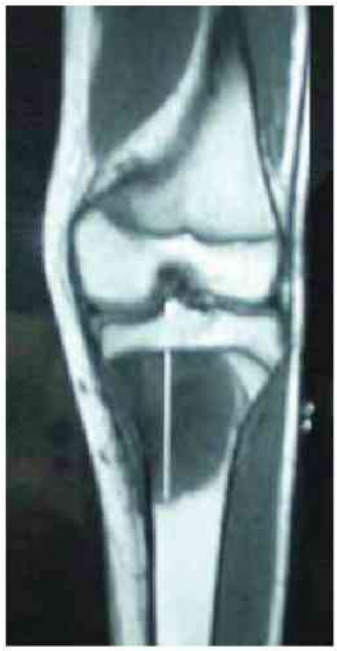
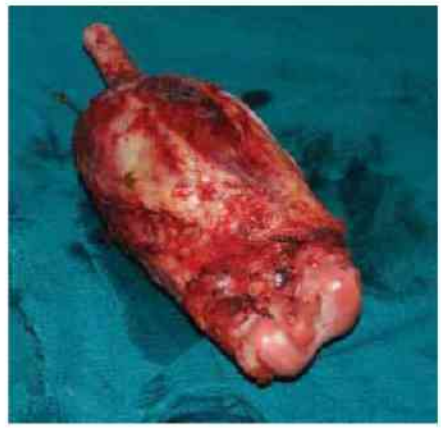
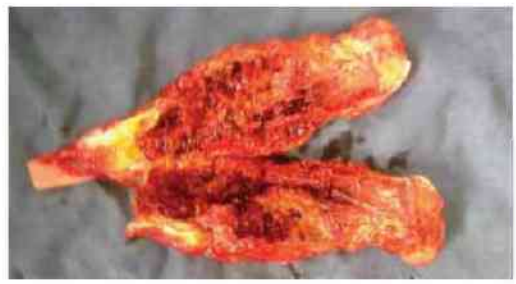


FIGURE 9.12 MRI of knee showing osteosarcoma of the proximal tibia.



(a)



(b)

FIGURE 9.13 (a, b) Clinical photo of resected specimen of osteosarcoma.

Classification

The subclassification of osteosarcoma into separate entities has been done on the basis of anatomic location, histologic subtype, cytologic grading and biologic behaviour. They are broadly classified into (1) central intramedullary type and (2) surface juxtacortical type:

1. Central (intramedullary) type
   a. Primary
      - Conventional
      - Telangiectatic
      - Small cell
      - Multicentric
   b. Secondary: Paget's disease
      - Radiation induced
      - Arising from other benign conditions such as fibrous dysplasia and osteochondroma
2. Juxtacortical (surface) type
   - Parosteal
   - Periosteal
   - Dedifferentiated

Surgical Treatment

The conventional treatment is to amputate the limb well above the upper limit of the tumour. For a tumour in the upper tibia, an above-knee amputation should be done. For a tumour in the lower end of femur, a disarticulation through the hip is done. After ablation of the limb, a suitable artificial limb is fitted and the patient made ambulant.

More recently, with the advances in chemotherapy and reconstruction methods, it has become possible to perform limb-saving surgery. In this method, wide excision of the tumour-bearing bone is done followed by reconstruction with bone grafting or custom-made metallic prosthesis.

Chemotherapy

In recent years, a great improvement in the survival rate has been achieved by the use of multidrug regimens of cytotoxic drugs. The drugs used

are high-dose methotrexate, iphosphamide, adriamycin and cisplatinum.

Parosteal Osteosarcoma

Parosteal osteosarcoma (juxtacortical osteosarcoma) is a separate clinical entity. It is less common than the usual central type of osteosarcoma. This type develops in relation to the cortex of the long bones in the lower limb. It occurs in the age group of 15–35 years and runs a slower course.

Clinically, it presents commonly as a slow-growing hard swelling on the posterior aspect of the distal end of femur. Radiologically, it appears as a densely opaque mass over the outer surface of the cortex in the popliteal surface.

The surgical treatment will be wide excision of the tumour-bearing bone and bony reconstruction. The overall prognosis is better than that in the central type of osteosarcoma.

Giant Cell Tumour (Osteoclastoma)

Giant cell tumour arises from the supporting connective tissues of the bone. As the giant cells are believed to arise from osteoclasts, this tumour is called osteoclastoma. It is a benign but locally aggressive neoplasm with a tendency for local recurrence.

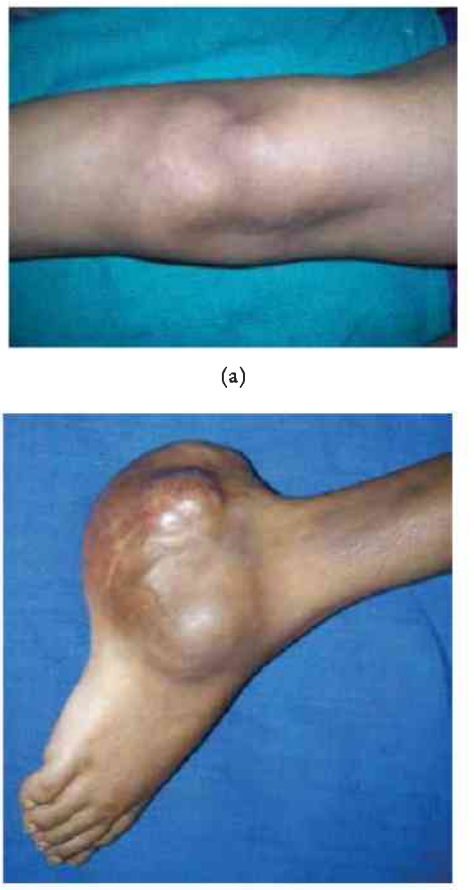
Incidence

This tumour is more common in Indians compared to the Western population, as evidenced by the large series by Vyagreswaradu (Visakapatnam) (160 cases) and by a senior author (165 cases).

This tumour occurs in patients in the age group of 20–40 years. It is more common in men although in the Western countries, it is found to be more common in women. It occurs in the metaphysioepiphyseal region of the long bones, after its fusion. The common sites of occurrence are the distal end of the femur, proximal end of the tibia, distal end of the radius and proximal humerus.

Clinical Features

The patient presents with a complaint of a bony swelling near the knee or wrist (Fig. 9.14). He gives a history of gradual growth of some months' duration and slight pain. The swelling is eccentrically placed. If the cortex of the swelling is thin, one can detect an '*egg shell crackling*' on palpation. The joint is not involved, but the movement is often restricted due to mechanical blocking by the swelling. Some cases present with a pathological fracture.



(a) (b)

FIGURE 9.14 Clinical photo of osteoclastoma of the (a) upper end of the tibia and (b) calcanuem.

Radiological Features

The radiographic appearance of this tumour shows an osteolytic lesion occupying the epiphyseal end of the bone (Fig. 9.15) with eccentric expansion of the cortex. The trabeculations in the wall of the cystic lesion show a '*soap bubble*' appearance (Fig. 9.16). It extends to the line of the articular cartilage. There is no evidence of periosteal new bone formation. MRI

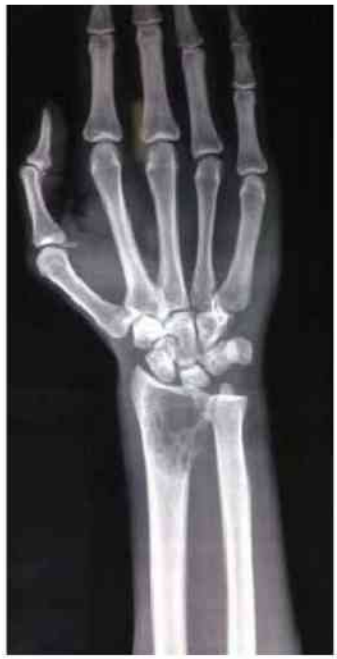


FIGURE 9.15 X-ray of left forearm with hand anteroposterior view showing giant cell tumour of the lower end of radius.

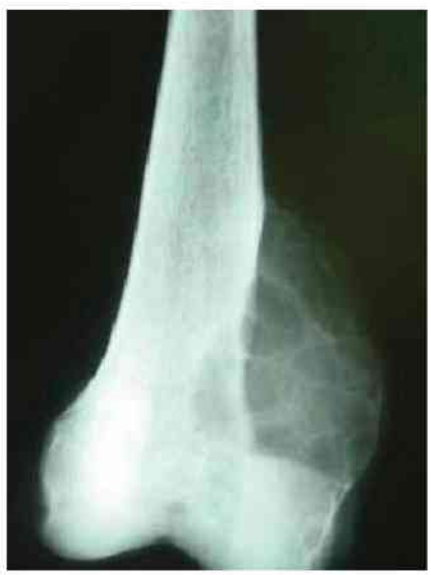


FIGURE 9.16 X-ray of femur anteroposterior view showing giant cell tumour in distal femur. Note the soap bubble appearance.

shows the intramedullary extent of the tumour and cortical discontinuity (Fig. 9.17).

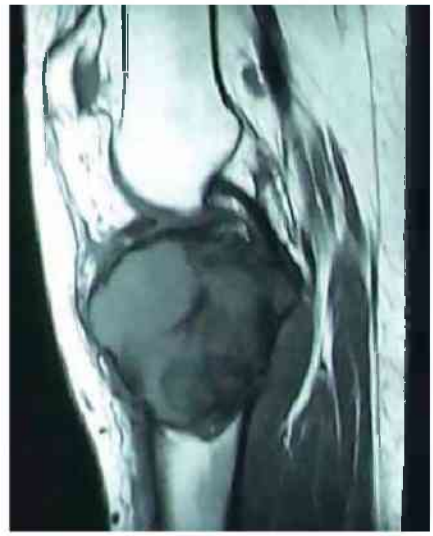


FIGURE 9.17 MRI of the knee demonstrating the giant cell tumour of the upper end tibia.

Radiologically, GCT is distinguished into three grades by Campanacci, namely the latent type, active type and aggressive type depending upon the osteolysis, cortical destruction and expansion.

Histological Features

Macroscopically, the tumour shows a fleshy greyish vascular tissue mass with some areas of haemorrhage (Fig. 9.18). Microscopically, the stroma consists of undifferentiated spindle cells which are plump and ovoid in shape. The stromal cells show varying degrees of mitotic activity. There are numerous giant cells, each containing about 10–20 nuclei.

Differential Diagnosis

Giant cell tumour has to be differentiated from the *giant cell variants* which include unicameral bone cyst, aneurysmal bone cyst, chondroblastoma, chondromyxoid fibroma, non-osteogenic fibroma, fibrous dysplasia and brown tumour of hyperparathyroidism.

Surgical Management

When the tumour is in a bone which can be sacrificed, the best treatment is surgical resection. Thus, the GCTs of the distal end of the ulna and proximal end of the fibula are best treated

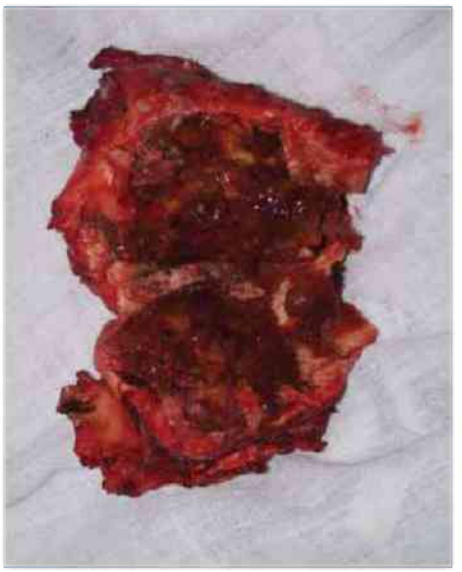


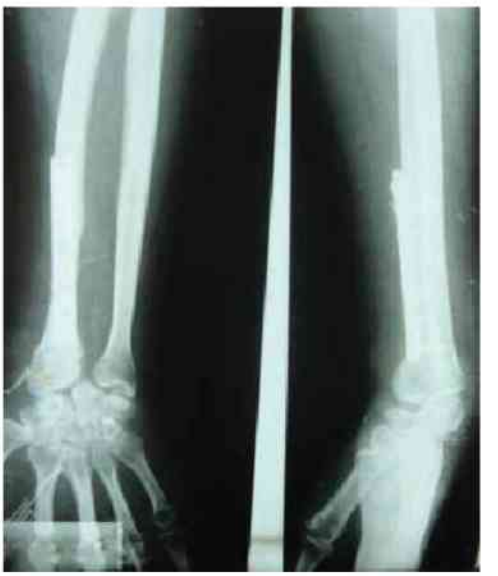
FIGURE 9.18 Clinical photo of resected specimen of osteoclastoma.

by resection of the bone including the tumour. A tumour in the distal end of radius is resected and is replaced by a fibular graft to restore function in the wrist (Fig. 9.19).

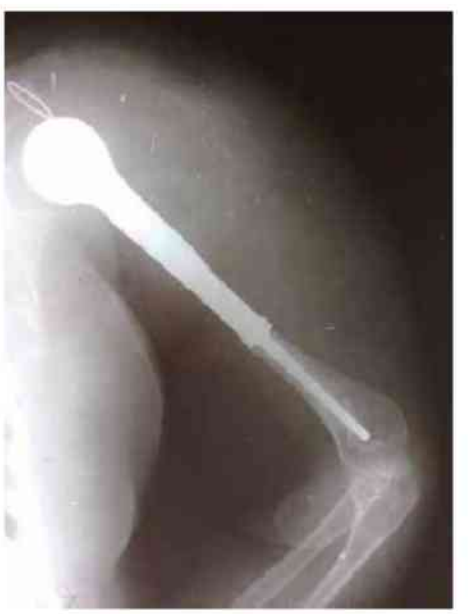
Tumours occurring in weight-bearing areas such as the distal end of the femur and proximal end of the tibia are treated by curettage of the lesion and packing with bone graft after adjuvant therapy such as phenol or liquid nitrogen (extended curettage).

More recently, bone cement is being used to pack the cavity (Fig. 9.20).

Larger lesions will require en bloc excision of the tumour-bearing bone and either biological reconstruction with bone graft or mechanical reconstruction with a custom-made metal prosthesis.



(a)



(b)

FIGURE 9.19 (a) X-ray of right forearm showing the giant cell tumour of the distal radius treated by fibula grafting. (b) X-ray of arm showing custom mega prosthesis done for proximal humerus.

CHONDROGENIC TUMOURS

Chondroblastoma

Chondroblastoma is a rare benign bone tumour of cartilaginous origin. It is more common in males and occurs in the age group of 10–25 years. It arises from the epiphysis of the long bones. The distal end of femur, proximal end of humerus and proximal tibia are the most common sites of occurrence. Pain is the presenting feature. It presents with progressive pain of the particular end of the bone and should be differentiated from any intra-articular pathologies of the involved bone.

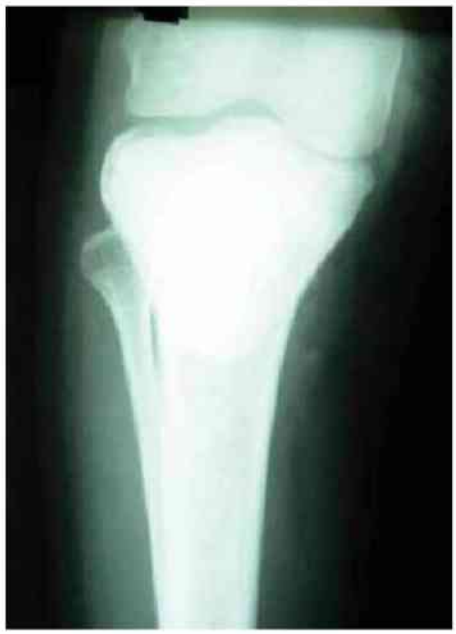


FIGURE 9.20 X-ray of right leg anteroposterior view showing the giant cell tumour of the upper end of tibia treated by curettage and cementation.

Radiographic Features

In children, a well-circumscribed epiphyseal lesion that crosses an open growth plate is virtually diagnostic of chondroblastoma. Radiograph shows a radiolucent area in the epiphysis with sclerotic margins and areas of calcification which appear hazy.

Pathological Features

Microscopically, chondroblastoma consists of sheets of chondroblasts usually with a background of chondroid matrix. Calcification is present and it may surround individual cells and give the classic 'chicken wire appearance'.

Treatment is by curettage and bone grafting.

Chondromyxoid Fibroma

Chondromyxoid fibroma is a rare benign tumour and is one of the giant cell variants. It occurs in the *metaphysis* of long bones in the lower limbs. Pain and later swelling are the presenting features. Radiograph shows an eccentric osteolytic lesion with a thin sclerotic margin. Microscopy shows areas of lobulated cartilaginous cells with myxomatous zones. Scattered giant cells are also present. Treatment is mainly by removal of the tumour including the wall and bone grafting.

Chondrosarcoma

Chondrosarcoma is a malignant tumour of cartilaginous origin.

Classification

1. Primary
   - Central (medullary) type
   - Intracortical type
   - Clear cell chondrosarcoma
   - Mesenchymal chondrosarcoma
   - Dedifferentiated chondrosarcoma
2. Secondary: Arising from pre-existing benign conditions such as
   - Chondroblastoma
   - Irradiation induced
   - Fibrous dysplasia

Histological Classification

Histologically, chondrosarcoma is classified on the basis of cellularity, pleomorphism and mitosis into the following types:

- *Grade I:* Low grade
- *Grade II:* Intermediate grade
- *Grade III:* High grade

Incidence

It is the third most common malignant tumour of bone (the first two being osteosarcoma and Ewing's sarcoma). In the long bones, it arises from the diaphyseometaphyseal region. The central type occurs commonly in the proximal end of the femur, ilium, upper end of the humerus and scapula. The common age group is between 30 and 50 years. It is common in males.

Clinical Features

The patient presents with a swelling with a history of dull aching pain. On examination, the swelling

FIGURE 9.21 Clinical photo of chondrosarcoma arising from the right proximal humerus. Note the dilated veins in the surface.

is hard in consistency with a lobulated surface and continuous with the bone (Fig. 9.21). It grows very slowly and may cause mechanical restriction of joint movements.

Radiological Features

There is expansion of the medulla in the central type and thickening of the cortex with spotty calcification (popcorn classification). The tumour may also appear as a lobulated swelling from the end of a long bone or ilium with patchy and fluffy areas of calcification (Fig. 9.22).

Pathology

Macroscopically, the tumour is lobulated and appears as a white or bluish mass of firm consistency. It often appears to have a capsule, and there are areas of myxomatous degeneration and softening. Irregular patchy areas of calcification are also characteristic.

Microscopically, masses of cartilage cells are seen, many of them with more than one nucleus which are hyperchromatic. Typically, there is formation of cartilage by the tumour cells.

Histologically, chondrosarcoma is classified as Grade I—low grade, Grade II—intermediate grade and Grade III—high grade based on its cellularity, pleomorphism and mitosis.

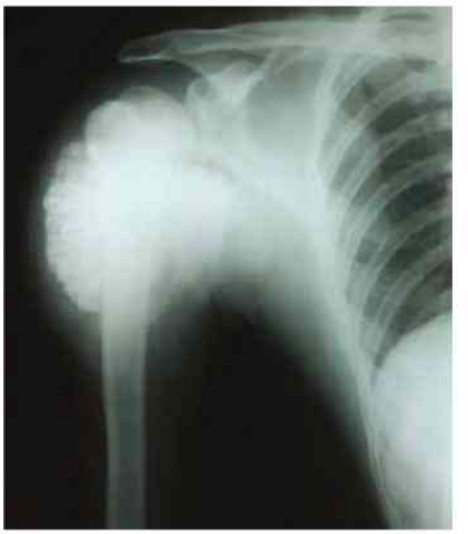


FIGURE 9.22 X-ray of right shoulder with arm anteroposterior view showing chondrosarcoma proximal humerus. Note 'popcorn calcifications'.

Treatment

Treatment of choice is surgical. It does not respond to chemotherapy or radiotherapy. Limb sparing surgery by en bloc resection and reconstruction of the skeletal defect is the treatment of choice.

High-grade massive tumours with soft tissue involvement will need amputation at the appropriate level.

FIBROHISTIOCYTIC TUMOURS

Fibrosarcoma of Bone

Fibrosarcoma is a rare malignant bone tumour arising from the fibrous elements of the medullary cavity of bone. It occurs in the metaphysis of long bones of the lower extremity in the age group of 20–40 years. It is a slow-growing tumour. The patient presents with pain or swelling in the region of the knee. Radiograph shows an osteolytic lesion in the metaphysis. There is no neoplastic osteogenesis or reactive new bone formation. Diagnosis is confirmed only by biopsy. For tumours with low-grade malignancy, wide local resection may be curative. High-grade malignant tumours need

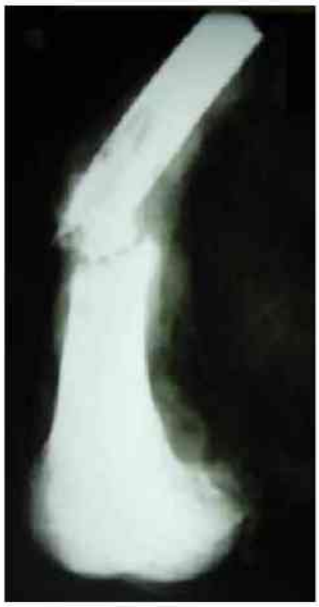


FIGURE 9.23 X-ray of femur showing malignant fibrous histiocytoma presenting as a pathological fracture.

ablational surgery. Chemotherapy and radiotherapy are not useful. Wide local resection with reconstruction is done.

Malignant Fibrous Histiocytoma of Bone (Histiosarcoma)

Malignant fibrous histiocytoma is a sarcoma of histiocytic origin. It occurs in adults in the long bones such as distal femur, proximal tibia and humerus (Fig. 9.23). It represents as pain and swelling of short duration. Radiologically, it resembles fibrosarcoma with pure osteolysis and hazy borders. Histologically, there are areas of predominating histiocytary structure or fibrocytary structure. Treatment includes chemotherapy and wide en bloc resection of the tumour-bearing bone.

MYELOGENIC TUMOURS

Ewing's Sarcoma

Ewing's tumour is an uncommon type of highly malignant bone sarcoma occurring in children. Ewing's sarcoma forms about 10% of all

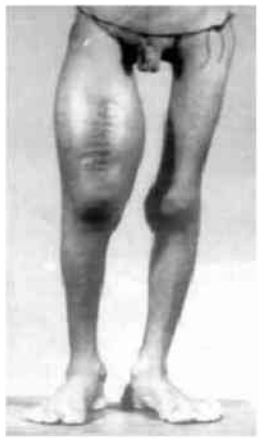


FIGURE 9.24 Clinical photo of Ewing's sarcoma of right femur.

malignant bone tumours. It occurs in the age group of 10–20 years and is more common in males. It arises from the primitive mesenchymal cells of the medullary cavity. The sites affected are the diaphyseal regions of long bones such as femur, tibia and humerus. It also occurs in flat bones such as pelvic bones.

Clinical Features

The patient presents with pain which gradually increases and is followed by the appearance of a swelling (Fig. 9.24). The swelling is firm to soft in consistency with indefinite margins. The commonest sites affected are the diaphyseal regions of the long bones such as femur, tibia and humerus. It also occurs in the pelvic bones. There is usually fever, anaemia and leucocytosis so that the condition often simulates subacute osteomyelitis. The swelling rapidly increases in size with involvement of soft tissues, and the general condition deteriorates.

The peculiar feature of Ewing's tumour is that metastasis occurs in other bones such as skull (Fig. 9.25), vertebrae and ribs in addition to the lungs, by spread through blood stream.

Radiological Features

Radiograph shows areas of mottled rarefaction in the affected bone. There will be marked destruction of the bone cortex and involvement of the soft tissues. There is also reactive new bone

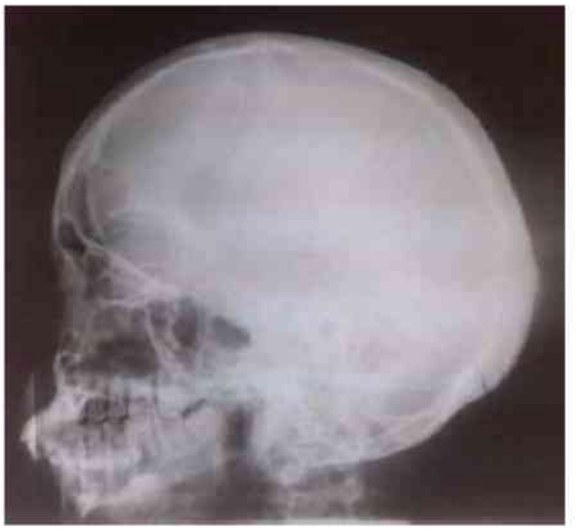


FIGURE 9.25 X-ray of skull lateral view showing secondaries arising from Ewing's sarcoma.

formation in layers under the raised periosteum producing the characteristic '*onion peel*' appearance (Fig. 9.26).

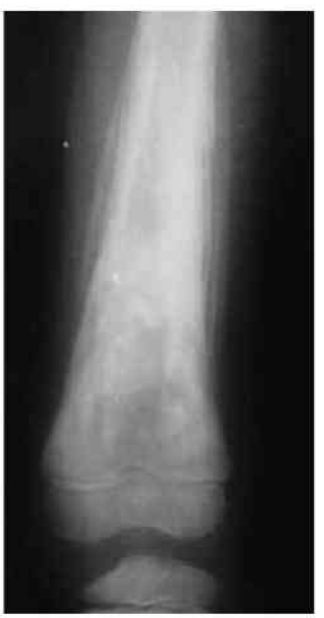


FIGURE 9.26. X-ray of femur anteroposterior view showing Ewing's sarcoma with characteristic onion peel appearance.
Courtesy: Dr Ajay Puri.

Pathology

Macroscopically, the tumour is a pale soft mass with minimal bone tissue. There are areas of degeneration and haemorrhage. There is further simulation of osteomyelitis by the presence of milky pus like fluid in the tumour tissue due to degeneration.

Microscopically, the tumour is very cellular with minimal stromal tissue. The characteristic cell is a small polyhedral cell with pale cytoplasm and large nucleus. The appearance is '*monotonously uniform*' with cells arranged in compact sheets with loose and vacuolated stroma.

In some areas, there is a pseudo-rosette formation by the tumour cells; this simulates the rosette formation of neuroblastoma. The tumour must be differentiated from lymphoma (reticulum cell sarcoma). On histochemistry, Ewing's sarcoma stains positively for glycogen.

Treatment

Multidrug chemotherapy with Vincristin, Adriamycin and Cyclophosphamide has remarkably increased the survival rate.

Currently, after pre-operative chemotherapy, surgical resection of the tumour-bearing bone is done. After skeletal reconstruction by grafting or prosthesis, post-operative chemotherapy is given.

Surgical ablation (amputation) is indicated in locally advanced cases.

This tumour is radiosensitive, and the regression following therapy is remarkable.

Lymphoma of Bone (Reticulum Cell Sarcoma)

Primary lymphoma of bone is an uncommon round cell sarcoma arising from the primitive lymphoid cells of the bone marrow. It resembles Ewing's sarcoma clinically and even histologically. It belongs to non-Hodgkin's variety of lymphoma.

It occurs in the age group of 20–40 years. It involves the femur, tibia and also occurs in the flat bones of the pelvis and scapula. The growth is rather slow and the general condition of the patient is well maintained. It metastasises to the regional lymphnodes and later to other bones and lungs. Radiography shows patchy areas of osteolysis with other areas of increased density.

Microscopically, the cells are of reticulum type. They are much larger than in Ewing's tumour, and the shape is reniform with nuclei with irregular outline and mitotic figures. The appearance is that of cellular pleomorphism. The presence of a network of reticulum fibrils surrounding single cells or small groups is characteristic.

It responds to chemotherapy and radiation therapy with megavoltage radiation. The overall prognosis is better than in other bone sarcomas.

Plasmacytoma (Myeloma of bone)

Plasmacytoma is the most common malignant tumour of the bone. It arises from the plasma cell of the reticuloendothelial tissue of the bone marrow. It can occur as solitary plasmacytoma, multiple myeloma or generalised myelomatosis.

Incidence

It occurs in persons past 40 years. It is more common in males. The common bones involved are the flat bones such as vertebrae, skull, ribs and pelvis where red marrow is predominant.

Clinical Features

The most common orthopaedic presentation of this condition is pain in the back or generalised bone pain in an elderly person. Pain may be intermittent but later becomes constant and intense. There may be a sudden increase of pain if a strain causes a pathological fracture of the vertebra. Occasionally, the patient may develop neurological signs due to cord compression. It can also present as a pathological fracture in the long bone.

The patient becomes severely anaemic, loses weight and becomes cachectic. In the late stage, the patient develops hyperazotaemia, uraemic symptoms and hypercalcaemia and hyperuricaemic syndromes and haemorrhagic diathesis.

Radiological Features

The typical feature is the presence of multiple, clear-cut, punched-out osteolytic areas without any surrounding sclerosis in the flat bones of the skull (Fig. 9.27) and pelvis.

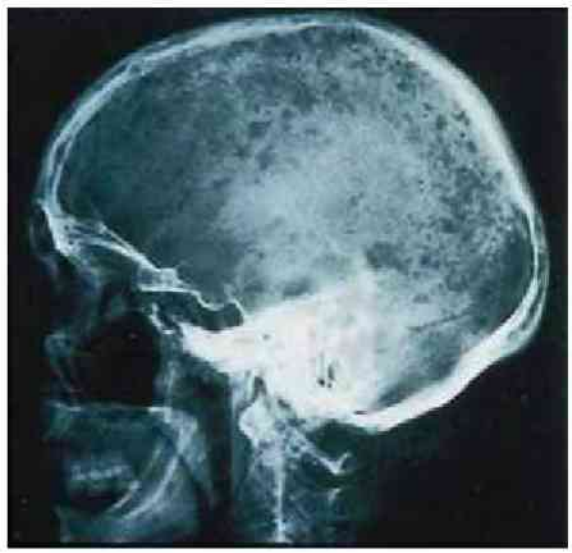


FIGURE 9.27 X-ray of skull lateral view showing multiple myeloma lesions. Note the clear-cut punched-out appearance.

The spine shows extensive osteoporosis of the vertebrae with or without compression fracture. The appearance has to be differentiated from senile osteoporosis or secondary carcinomatosis.

Laboratory Findings

Haemogram shows a low RBC count and a low value of haemoglobin due to the marrow being replaced by tumour. There is a marked increase in the erythrocyte sedimentation rate due to the increase in serum proteins.

Serum protein estimation shows a high total protein. There is a reversal of the albumin globulin ratio due to an increase in the globulin fraction. Serum electrophoresis shows an increase in the gamma fraction of the globulin called M spike. Immunoglobulin electrophoresis is useful for a more accurate diagnosis of the type of immunoglobulin affected in the disease (IgG or IgM).

The serum calcium is elevated due to the marked osteolytic action of the myeloma cells. The serum uric acid level is high due to excessive breakdown of nucleic acid. The blood urea level may show an increase depending on the extent of renal damage.

Urine examination may show Bence Jones protein in about 50% of cases. This protein precipitates when the urine is heated to 50°C but dissolves

when it is further heated to 100°C. Urinary electrophoresis and immunoelectrophoresis are more sensitive in detecting Bence Jones protein.

Bone Marrow Study

Sternal or iliac creast puncture is done. This is a fine needle aspiration cytological examination. The smear shows extensive areas of plasma cells.

Microscopic Appearance

Histologically, it shows a uniform picture of closely packed round or oval plasma cells without any intervening stroma. The cells have eccentric nuclei and clumped chromatin giving it a 'cart wheel appearance'.

Treatment

Chemotherapy is the mainstay of treatment of multiple myeloma and has markedly improved the prognosis and the quality of life. The main drugs used are Cyclophosphamide and Phenylalanine mustard. The tumour is radiosensitive, and localised lesions are treated with radiation therapy for relief of pain and pressure symptoms.

Surgery is indicated in the treatment of pathological fracture of long bone and for decompression in spinal lesions.

BONE METASTASIS (Secondary Malignant Deposits in Bone)

Metastatic bone tumour is a tumour occurring in a bone, secondary to primary carcinoma in any other organ of the body. The skeleton is one of the most common sites for metastatic deposits. The common primary tumours which give rise to metastases in bones are those arising from thyroid, breast, bronchus, kidney and prostate (Fig. 9.28). Other tumours giving rise to bone deposits are adrenal neuroblastoma and melanoma.

Sites

Metastatic bone tumour is more common in the elderly people. The common sites are the flat bones of the axial skeleton such as vertebrae, skull and pelvis. It also occurs in the proximal end of femur and humerus where there is a concentration of red bone marrow. They are very rare distal to the knee and elbow.

Mode of Spread

The cancer cells reach the skeleton through the haematogenous route. This happens through three routes, namely pulmonary, portal or caval, and then reaches the bone. Batson's vertebral venous system which communicates with the pelvic veins and the portal and caval venous system permits the spread of cancer cells from the viscera to the vertebrae, without passing through the lungs and the systemic circulation. Some tumours spread through blood stream to lungs.

Clinical Features

The main clinical presentation of a metastatic tumour in bone is pain, pathological fracture and swelling. A pathological fracture often first attracts our attention to the presence of a secondary deposit. The fracture is caused by minimal violence. Girdle pain or paraplegia may be caused by secondaries in the spine.

Sometimes the secondary in the bone is the first presenting feature, while the primary tumour itself is still clinically silent. It is therefore very important to do a thorough general clinical examination of the patient when any swelling in the bone appears in an elderly patient. Occasionally, the clinical examination may not reveal the primary. Biopsy examination of the lesion will help us to diagnose the type of tumour and may also indicate the site of the primary growth. In rare cases, the secondary deposit may show such an anaplastic picture that even the nature of the primary growth cannot be established.

Radiological Features

The deposits are usually osteolytic, and the fracture line is usually transverse. Ultimately, the cortex also is eroded and the lesion breaks through and spreads to the soft tissues. A skeletal survey of other bones should always be done to locate all the deposits. The deposits from a prostatic carcinoma are osteoblastic and cause dense shadows in the radiograph (Fig. 9.29).

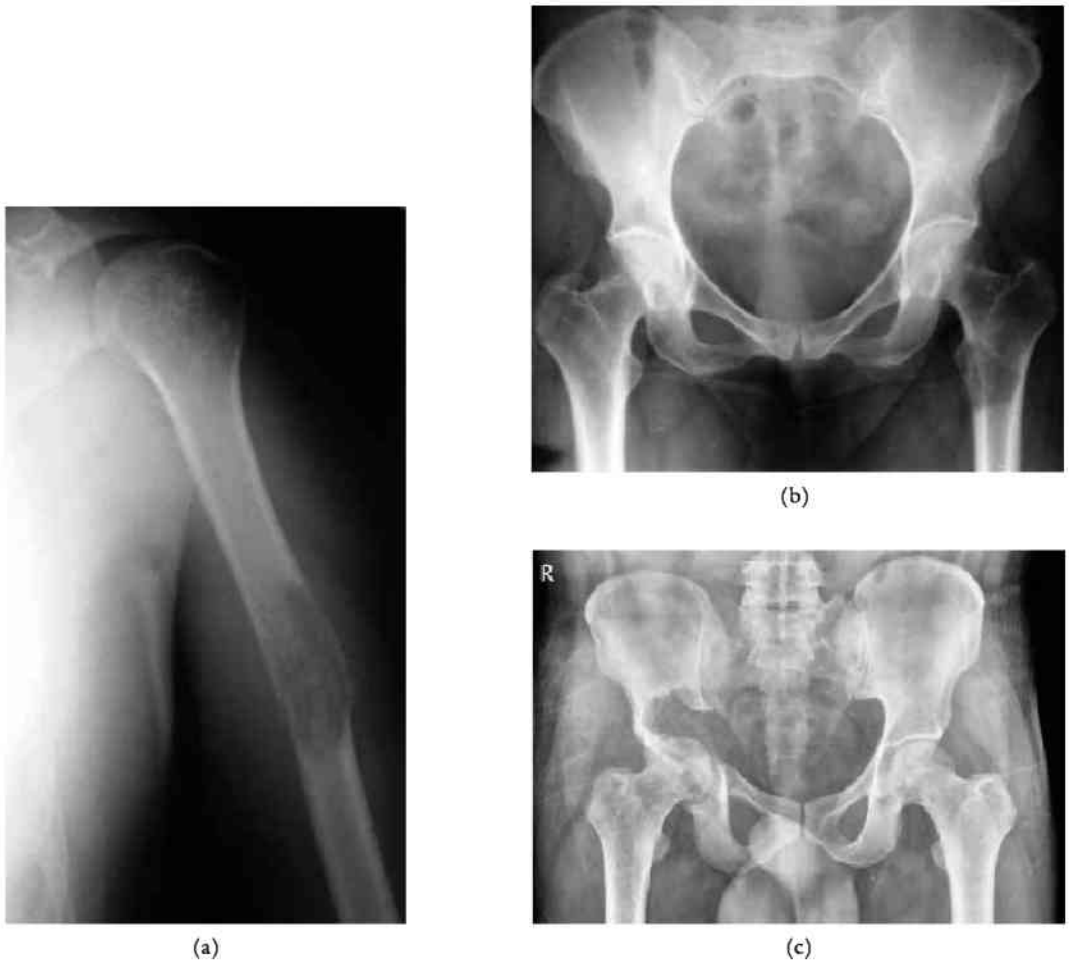


FIGURE 9.28 (a) X-ray of arm anteroposterior view showing metastasis from renal carcinoma, with pathological fracture. (b) X-ray of pelvis with both hips showing breast metastasis in the left proximal femur. (c) X-ray of pelvis with both hips showing thyroid metastasis in the right ilium and acetabulum with protrusio acetabuli.
Courtesy: Dr Ajay Puri.

Secondaries in the vertebrae may not be demonstrable in plain radiographs when the lesion is early. More recently, CT scan of the skeleton is used in visualising microlesions in the bone which are not visible in plain radiograph or tomogram.

Isotope imaging by Technetium-99 scan helps early detection of secondaries in other parts of the skeleton.

Laboratory Findings

- *Blood:* The serum alkaline phosphatase level is raised in cases of bone destruction with extensive reparative osteoblastic reaction. Blood calcium levels may be raised in cases of extensive secondary metastases in bone causing osteolysis. In case of secondaries from prostatic cancer, the serum acid phosphatase level is increased.
- *Urine:* Elevation of 24 hours excretion of hydroxy proline is an early evidence of skeletal metastasis.

Treatment

Treatment of the secondary deposit depends on the site of the primary growth and the general condition of the patient. In cases of a single secondary deposit

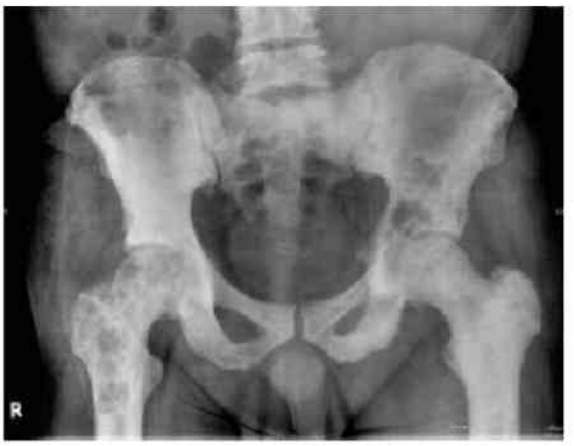


FIGURE 9.29 X-ray of pelvis with both hips showing sclerotic secondaries from prostatic carcinoma.
Courtesy: Dr Ajay Puri

with a known primary, the primary lesion is treated first. The secondary deposit, if located in an accessible site, may be surgically removed.

Pathological fracture in long bones should be treated by surgical removal of the deposit, internal fixation augmented by bone cement. More recently, prophylactic stabilisation by internal fixation is being done for osteolytic lesions in long bones to prevent pathological fractures. Secondary lesion in vertebrae with cord compression can be treated by surgical decompression and stabilisation.

Multiple secondaries with a known or unknown primary will require palliative radiation or chemotherapy.

SYNOVIAL SARCOMA

Synovial sarcoma is a sarcoma whose cells mimic the two types of cells (A and B) of the synovial membrane. It is more common in men between 15 and 35 years. The tumour occurs in the deeper tissues close to a joint adherent to the capsule, tendons, fasciae and synovial bursae. Only 10% are observed in a joint cavity. It is usually located in the region of the knee, ankle, foot, elbow and wrist.

Synovial sarcoma presents with pain and swelling in the para-articular region. The growth of the tumour is slow. Radiologically, most cases show radio-opacities. Histopathologically, synovial sarcoma is characterised by a biphasic structure—pseudo-fibroblastic spindle cells and pseudo-epithelial cells. Treatment is by wide surgical excision of the tumour or amputation. Chemotherapy and radiotherapy are useful to avoid local recurrence or metastasis.

HAEMANGIOMA OF BONE

Haemangioma of bone is a vascular hamartoma occurring in the vertebral column, flat bones of the cranium and the limb bones. In the spine, it involves the body and the posterior arch of the thoracic or lumbar vertebrae. In the cranium, it occurs in the frontal and parietal bones. The presenting clinical feature is pain or neurological deficit in the spine.

Radiological appearance is that of a striated vertebra due to bone rarefaction and vertical trabeculation. It may also present as a moth eaten or honey comb appearance. CT shows 'polka dot sign' in vertebrae. Collapse of the body is also seen occasionally. Macroscopically, in the areas of osteolysis there is a bright red tissue which is soft and bloody. Microscopically, there are newly formed and anomalous vascular spaces. These may be capillaries which are dilated until cavernous bunches or wide labyrinthic spaces are formed.

Haemangiomas, which cause pain, are treated by surgical ablation, selective arterial embolisation or radiation therapy. Haemangiomas producing neurological signs are treated by decompression and radiation therapy. Extraperiosteal resection is indicated in extensive forms in the limbs.

ADAMANTINOMA OF LONG BONES

Adamantinoma is a low-grade malignant tumour of mixed epithelial and mesenchymal origin. It resembles the odontogenic epithelium of the adamantinoma of the jaw and hence the name. It is common in adult males between 20 and 40 years. Ninety per cent of cases occur in the tibia. The other bones could be ulna, fibula and femur. It occurs in the diaphysis and occasionally metaphysis.

The patient presents with pain, swelling or pathological fracture. Radiograph shows an osteolytic area with faded margins or multilocular lucencies with a bubbly or trabeculated appearance

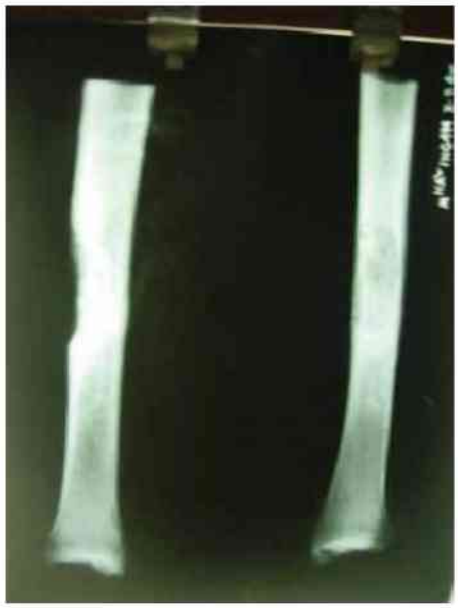


FIGURE 9.30 X-ray of both legs showing adamantinoma in right tibia.

(Fig. 9.30). Macroscopically, it is a greyish firm fibrous mass. Microscopically, some areas are alveolar or tubular with a gland-like appearance (similar to adamantinoma of jaw). Other areas are composed of spindle cells similar to synovial sarcoma. Areas of squamous cells or basal cells are also present.

The progression of the disease is very slow, and it is late to cause metastases. Treatment is by complete surgical eradication of the lesion by wide resection. Chemotherapy and radiotherapy have no role to play.

CHORDOMA

Chordoma is a rare malignant tumour of bone originating from the residues of the notochord in the cranial bone and the vertebral column. It occurs in adult males after 50 years of age. Eighty-five per cent of the cases occur in the sacrococcyx or in the spheno-occipital region of the cranial base. The remaining occur in the vertebral body.

Spheno-occipital chordomas produce symptoms of lesion of hypophysis or cranial nerves.

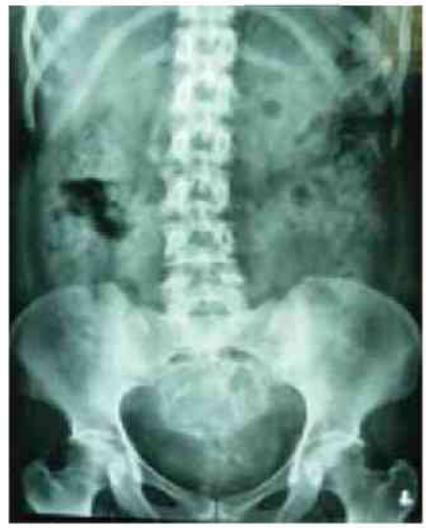


FIGURE 9.31 X-ray of lower spine with pelvis showing chordoma in the sacrococcygeal region.

The sacrococcygeal chordomas present as a palpable mass in the middle of the sacrum before the onset of neurologic symptoms. In the vertebral body, signs of myeloradicular compression occur. Radiology shows cortical destruction and poor margination (Figs 9.31 and 9.32).

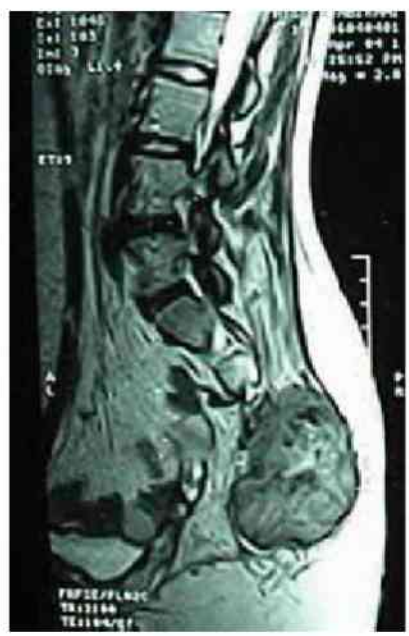


FIGURE 9.32 MRI of lumbosacral spine demonstrating sacral chordoma.

The tissue of chordoma has a dark brown colour and is semiliquid with mucinous quality. The histologic hallmark is the physaliferous cell (Greek physalis = bubble), a large polyhedral or round cell with a small centrally placed nucleus. Treatment is by wide local resection

KEY POINTS

- Features of tumours in a plain X-ray that help in accurate diagnosis include anatomical location, anatomical borders, pattern of bone destruction, matrix formation and periosteal reaction.
- Enneking staging is used for surgical staging of bone tumours.
- Osteoid osteoma occurs in an age group of 10-30 years and mostly in cortical areas. X-ray shows nidus. Pain responds to salicylates.
- Non-osteogenic fibroma is an eccentric fibrous lesion occurring in the metaphysis of long bones in children.
- Osteochondroma is the most common tumor-like lesion of the bone. It arises from the epiphyseal plate and has a cartilaginous cap.
- Aneurysmal bone cyst is a solitary rapidly progressive expansile lesion occurring in the metaphysioepiphyseal area of long bones and the pedicle and lamina of vertebra. It is eccentrically placed lesion.
- Osteosarcoma is the most common malignant tumour of primary bone tissue origin. It is metaphyseal in location. X-ray shows sunray appearance and Codman triangle.
- Osteosarcoma can be broadly classified as central intramedullary type and (surface) juxtacortical type.
- Osteoclastoma occurs in the epiphyseal region of bone, located eccentrically and has soap bubble appearance on X-ray. Campanacci grading is used radiologically.
- Chondroblastoma is an epiphyseal lesion that crosses the growth plate. Histopathological examination shows 'chicken wire appearance'.
- Chondrosarcoma is diaphyseometaphyseal in location. Shows 'popcorn calcification' on X-rays.
- Ewing's sarcoma is a diaphyseal tumour. It mimics subacute osteomyelitis in presentation. X-ray shows onion peel appearance. It is a primitive neuroectodermal tumour.
- Plasmacytoma is the most common malignant tumour of bone arising from plasma cell in bone marrow. It is characterised by hypergammaglobulinaemia.
- Secondaries (metastases) to be commonly arise from primary malignancies of thyroid gland, breast, bronchus, kidney and prostate.

MULTIPLE CHOICE QUESTIONS

1. Benign bone tumours are
   a. Osteoid osteoma
   b. Osteochondroma
   c. Chondroblastoma
   d. Chondromyxoid fibroma
   e. All of the above
2. Bone forming tumours are
   a. Osteosarcoma
   b. Osteoid osteoma
   c. Osteoblastoma
   d. All of the above
3. Bone cysts mostly occur in
   a. Spine
   b. Humerus
   c. Femur
   d. Tibia
4. Sunray appearance is seen in
   a. Osteogenic sarcoma
   b. Ewing's sarcoma
   c. Multiple myeloma
   d. Osteoclastoma
5. The commonest site for osteogenic sarcoma is
   a. Upper end of femur
   b. Lower end of femur
   c. Upper end of tibia
   d. Lower end of tibia
6. Which of the following tumours arises from epiphyses?
   a. Osteosarcoma
   b. Ewing's sarcoma
   c. Osteoclastoma
   d. Multiple myeloma
7. Ewing's sarcoma is characterised by all except
   a. Metaphysis-diaphysis in location
   b. Locally malignant
   c. Metastasis is rare
   d. Onion peel appearance is seen on X-ray

CHAPTER 10

Neurological and Muscular Disorders

CEREBRAL PALSY

Cerebral palsy (Little's disease) is a non-progressive neurological disorder of children affecting the locomotor system. The resulting morbidity includes inco-ordination, dystonia, spasticity and involuntary movements.

Aetiology

The causative factors can be classified into pre-natal, natal and post-natal:

- *Pre-natal:* Genetic factors, prematurity and maternal infection
- *Natal:* Cerebral anoxia due to difficult labour and birth trauma
- *Post-natal:* Infections such as encephalitis causing cerebral damage

Classification

Cerebral palsy is classified into the following five types:

1. Spastic type
2. Athetoid type
3. Ataxic type
4. Rigid type
5. Mixed type

The spastic type is the most common and is due to damage to the cerebral cortex. It is associated with increased muscle tone and exaggerated reflexes.

The athetoid type is due to damage in the basal ganglia and is characterised by involuntary movements of the hands and legs. Athetosis is a continuous, involuntary writhing movement.

The ataxic type is due to cerebellar involvement and is characterised by ataxic gait.

In the rigid type, the muscles are in constant state of contraction.

The mixed type has features of spastic, athetotic and ataxic types.

The spastic type which forms 70% of the cases of cerebral palsy can be grouped according to the limb involved: monoplegia, paraplegia, hemiplegia and quadriplegia.

Clinical Features

The child is brought between the age of 1 and 4 years with the complaint of inability to walk. There is a history of delayed milestones in development. On examination, there is poverty of movement. The severity of symptoms may vary from mild to severe. In mild cases, the child may be ambulant without support. In most other severe cases, walking is difficult and requires some support. Mild cases are detected by the slight spasticity of the limbs. The main signs are adductor spasm at the hips offering resistance to passive abduction. There is also equinus spasm at the feet and resistance to passive dorsiflexion of the feet.

When the child is seen a little later, the lower limbs are held with adduction at the hips,

FIGURE 10.1 Clinical photo of scissoring gait in cerebral palsy.

flexion at the knees and equinus of the ankles. The knees are in a flexed state due to hamstring spasm. The knee and ankle jerks are exaggerated, and there may be patellar and ankle clonus. The plantar response is extensor. When the child is helped to walk with support, it walks with a scissor gait in spastic palsy (Fig. 10.1). In neglected cases, there is equinus contracture at the ankle and adductor contracture at the hip. In ataxic palsy, the child characteristically walks with a broad-based gait.

If the upper limbs are affected, the shoulder is held in adduction and internal rotation, and the elbows in flexion with the forearms pronated. Further, the wrist is flexed and the thumb is drawn into the palm and the fingers are flexed at the metacarpophalangeal joints.

Along with muscle contractures, the child may also develop bony deformities such as valgus of femoral neck, external tibial torsion and scoliosis and hip subluxation.

Management

The condition should be detected even in the neonatal period by the paediatrician to initiate early therapeutic measures. The treatment and rehabilitation of the spastic children need the coordinated efforts of the orthopaedic surgeon, neurologist, psychiatrist, physiotherapist and occupational therapist. The active co-operation of the parents is very important.

The *aim of treatment* is to achieve the maximum functional ability and skill that the child can acquire. Each child requires a regime that is appropriate to its developmental age, intelligence and severity of disability.

In mild cases with good IQ, the deformities can be corrected with physiotherapy and they could be restored to normal and made fit for normal school education.

In moderate cases, in children who are educable, the aim is to correct the deformity with physiotherapy and orthotic appliances and make them walk and be independent. These children could be educated in special schools. Where children are not educable but trainable, the aim is to train them in activities which need only repetitive skills, so that they could be employed in simple occupations.

In severe cases where the patient is bedridden and depends on others for every activity, the goal is to train them to do their own 'self-care' activities such as feeding, attending to their personal toilet and dressing. These are called Activities of Daily Living (ADL) and they need custodial care in special institutions.

- *Physiotherapy:* It is the mainstay of the treatment in all cases of cerebral palsy. It consists of passive movements of the joints to correct contractures and stretch out the muscles. The patients are also taught to relax the spastic muscles. Active exercises are given to establish movement patterns and teach rhythmic contractions and relaxations of muscles. Walking calipers or below-knee orthoses are prescribed to steady their standing postures and train them in walking. Night splints are used to maintain the correct position of the knee, foot and hand.
- *Occupational therapy:* It is very useful in older children. Certain activities involving repetitive movements of the legs, hands and fingers are utilised to relax spastic muscles and establish rhythmic and coordinated movements. This also helps in training

TABLE 10.1 Specific Anatomical Deformities in Cerebral Palsy and the Surgical Procedures Performed to Correct Them

Anatomical Site	Type of Deformity	Surgical Procedure Adopted
Hip	Adduction deformity	Adductor release Medial hamstring lengthening Open tenotomy of adductor longus/division of gracilis
Hip	Flexion deformity	Psoas tendon lengthening
Hip	Internal relation deformity	Division of gluteus medius Division osteotomy of femur
Hip	Subluxation	Varus derotation osteotomy
Knee	Flexion deformity	Hamstring release
Ankle	Equinus deformity	Achilles tendon lengthening
Foot	Pes cavus	Tibialis posterior lengthening Subtalar arthrodesis

them later in some occupations in which they can be employed so that they could be economically independent and socially productive.

- *Speech therapy:* It is given to children with defective speech after proper evaluation of their IQ and speech disorder.

Surgical Treatment

The aim of surgery in cerebral palsy is to correct deformities and stabilise joints. Surgery plays only an adjuvant role in the total management of these children; the preferred age for surgery is between 4 and 8 years.

The following are the surgical procedures commonly used:

- Tendon lengthening (e.g. tendo achilles is lengthened in severe equinus contractures at the ankle).
- Tenotomy (division) of contracted tendon (e.g. in the case of severe adductor contracture, division of the adductor tendons at their origin is done).
- Tendon transfer operation: Transfer of tendon may help to improve muscle balance, particularly in the upper limb. The release of the insertion of the pronator teres helps to lessen the pronation deformity of the forearm. Its transfer into the wrist extensors lessens the flexion deformity at the wrist.
- Neurectomy: Division of the obturator nerve is done to relieve marked adductor spasm at the hip.
- Stabilisation of joints (e.g. triple arthrodesis of the foot and arthrodesis of the wrist are done to improve function).
- Procedures used for correcting the position of thumb in deformities of palm are Matev's procedure, flexor pollicis longus tendon lengthening, thenar muscle release and tendon transfer.

Refer to Table 10.1 for specific anatomical defects and their surgical corrections.

ANTERIOR POLIOMYELITIS

Anterior poliomyelitis is an acute infectious fever caused by an enteric virus affecting commonly the anterior horn cells of the spinal cord causing paralysis of the muscles of the limbs and spine.

Pathology

The disease is caused by a neurotropic virus which has an affinity for the neurons in the anterior horn of the spinal cord and also for the

motor nuclei in the brainstem. There are three types of the virus:

1. Brunhilde
2. Lansing
3. Leon

After entry, the virus travels along the nerves and blood to reach the anterior horn cells of the spinal cord and certain motor nuclei in the brainstem.

This causes damage to the motor neurons and causes lower motor neuron (LMN) type of paralysis of the corresponding muscles or muscle groups.

The two main pathological changes in the cord are oedema and chromatolysis of the motor neuron cells in the anterior horn. The first change noted is chromatolysis of Nissl's substance inside the cytoplasm. The corresponding muscles are paralysed. The changes in the neuron cells are reversible upto a point, and this explains the recovery of some paralysed muscles.

Clinical Features

- *Stage 1:* Acute stage
- *Stage 2:* Recovery stage
- *Stage 3:* Residual paralysis stage

Abortive poliomyelitis is a term used to indicate the clinical recovery that occurs in about one-third of the patients infected with polio virus, after a minor illness. This consists of fever, headache and body pain. The illness usually lasts for less than 2 days.

1. Acute stage
   It is further divided into the following:
   - *Pre-paralytic phase:* There is marked tenderness of the involved muscles with the coexisting fever and headache. A sudden onset of neck stiffness, irritability, nausea and vomiting points towards meningitis. There is spasm and hence aching of the back muscles. When there is a clinical recovery at this point, the disease is described as non-paralytic poliomyelitis.
   - *Paralytic phase:* A sudden onset of flaccid asymmetrical paralysis of muscles of limb and trunk occurs on the second or third day. There is no sensory loss, and bladder and bowel functions usually remain unaffected. Paralytic poliomyelitis occurs as two forms:

     a. Spinal form (common)
     b. Bulbar form

     In *spinal form*, muscles of trunk and extremities are affected and flaccid paralysis occurs. The lower limb is commonly involved than the upper limb and muscles commonly included are tibialis anterior and posterior, quadriceps, gluteal muscles and deltoid.

     In *bulbar form*, cranial nerves are affected. The first symptom noted is nasal intonation and gradually respiratory obstruction in majority of cases.

2. *Recovery stage or convalescent stage:* During this period, there is gradual recovery of paralysed muscles. When the muscle remains paralysed for more than 3 months, there is a meagre chance for recovery. Most of the recovery that occurs in the paralysed muscles is seen within 3–6 months and the recovery process may last for up to 2 years.

3. *Residual paralysis stage:* 'Post-polio residual paralysis' is a term to indicate the residual paralysis that has occurred after 2 years of the onset of the disease. It is important to note that
   - deformities occur due to imbalance of muscle power and bad postures,
   - disuse atrophy of muscles and shortening of the leg occur due to interference with growth.

In neglected cases, gross fixed deformities of the hip, knee and foot occur with severe wasting of muscles. Children with extensive paralysis and gross deformities have to crawl on all fours to move from place to place. The virus can be isolated from the pharynx and faeces of the patients in pre-paralytic and post-paralytic phases.

Management of Poliomyelitis

Acute Stage

In this stage, the treatment is mainly medical by the paediatric physician. General supportive

treatment for the pyrexia and meningeal irritation, prevention of secondary respiratory infection and treatment of any respiratory paralysis are the main aspects of treatment.

Orthopaedic Care

The paralysed legs are supported by plaster splints or pillows and sandbags to keep the hip joints in a 5° flexion and in neutral rotation. The knee joint is held at a 5° flexion and foot supported in a 90° position.

Recovery Stage

The treatment in this stage is mainly by physiotherapy and splinting which lies with the orthopaedic department. The aims of treatment are as follows:

- To assist in the recovery of paralysed muscles by remedial exercises
- To prevent deformities by the use of orthotic appliances

An assessment is first made of the extent of muscle paralysis by charting the power of various groups of muscles and grading them according to the international nomenclature (Medical Research Council MRC grading) as follows:

- 0—complete paralysis
- 1—slight flicker of contraction present
- 2—muscle can move a joint only when gravity is eliminated
- 3—muscle can move a joint against gravity
- 4—muscle can move a joint against gravity and resistance
- 5—full normal power

The total functional assessment of the limbs is made before planning the treatment. This will include the following:

- Charting the muscle power grades
- Extent of contractures and deformities
- Method of ambulation
- Shortening of the limb

Physiotherapy

The remedial treatment in the physiotherapy department consists of the following:

- Passive movements
- Gentle massage
- Remedial exercise
- Hydrotherapy

Orthotic Management

Appropriate orthotic appliances are prescribed to prevent deformities due to muscle imbalance (Table 10.2).

Stage of Residual Paralysis

At the end of 2 years, no further recovery of paralysed muscles can be expected. At this stage, a final assessment of the muscle power of the leg is done.

The aim of treatment at this stage is to attempt to make the best use of available muscle power and make the limb functionally as useful as possible.

Role of Surgery

- *Correction of established deformities:* The common deformities seen are the flexion abduction contracture of hip, flexion contracture of the knee and equinus contracture of the ankle.

  Marked flexion abduction contracture of the hip is corrected by Souttar's operation, consisting of division of the contracted tensor fascia and the muscle origins at the anterior superior iliac spine. Yount's procedure of division of the ilio tibial band at the lower third of thigh is also done. Fixed equinus contracture at the ankle is corrected by lengthening the tendo achilles.
- *Improvement of muscle balance:* This can be achieved by surgical redistribution of available muscle power and by tendon transfer operations (e.g. in the case of marked weakness of knee extension, the quadriceps power is improved by transferring one of the hamstring muscles into it).
- *Stabilisation of joints:* Some joints are unstable due to loss of power in one group of muscles. This is corrected by arthrodesis of such joints. The best example is triple arthrodesis of the foot in paralysis of evertors or dorsiflexors of the foot (Figs 10.2 and 10.3).

TABLE 10.2 Orthotic Appliances Used for the Prevention of Various Deformities

Muscles Paralysed	Deformity	Appliance
Foot		
Evertors	Inversion	AFO with inside bar and outside T strap
Invertors	Eversion of foot	AFO with outside bar and inside T strap
Ankle		
Dorsiflexors	Foot drop	AFO with 90° foot drop stop
Plantar Flexors	Calcaneus	AFO with reverse 90° stop
Ankle and Foot		
All muscles	Flail foot	AFO with limited motion ankle joint
Knee		
Extensor: Quadriceps	Genu recurvatum	Knee ankle foot orthosis with back knee support
Hip		
Abductors: Gluteus medius, minimus and tensor fascia	Gluteus medius gait	Hip knee ankle foot orthosis, full caliper with pelvic band

AFO—ankle foot orthosis.

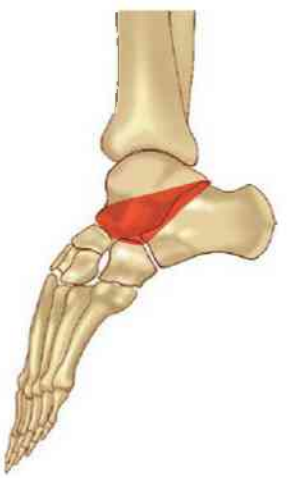


FIGURE 10.2 Diagrammatic representation of wedge-shaped area to be excised to correct foot drop.

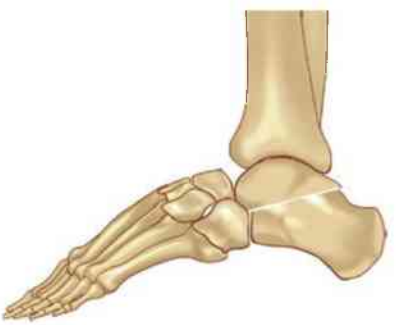


FIGURE 10.3 Diagrammatic representation of corrected foot after bone excision.

LEPROSY

Hansen's disease or leprosy is one of the common causes of disfiguring deformities in India. The disabilities produced by this disease since early times are not only physical but also social, due to the stigma attached to the disease. Management of the problem of leprosy includes not only treatment of the disease but also total rehabilitation by restoration of the person into the mainstream of social life.

Classification

According to the modern classification, they are clinically divided into two major groups:

1. Multibacillary cases, which include lepromatous and borderline lepromatous cases

2. Paucibacillary cases, which include indeterminate, tuberculoid and borderline tuberculoid cases

Clinical Features

The orthopaedic problems met with in leprosy lie in the lower and upper limbs. The common clinical problems are as follows:

1. Lower limb
   - Foot drop
   - Trophic ulcers
2. Upper limb
   - Partial claw hand
   - Total claw hand

In a patient presenting himself with any of the above, a thorough clinical examination must be made to detect the presence of other manifestations of leprosy. These include anaesthetic patches in the skin and thickening of nerves near bony canals such as ulnar nerve behind the medial epicondyle, lateral popliteal nerve near the neck of the fibula and ulceration in the mucosa of the nasopharynx. The diagnosis is confirmed by examination of skin scrapings and mucosal smears for lepra bacillus.

Prevention

The most important aspect is to prevent the development of these *nerve* palsies and their secondary lesions such as ulcer. This is done by:

1. *Primary prevention:* By early detection of the disease and the use of antileprosy drugs
2. *Secondary prevention:* Health education measures to the patient
   - Hygienic care of the anaesthetic foot with attention to cracks and fissures. Prevention of rat or insect bite by use of neem oil on feet and hands at night
   - Early detection of tender spots in the sole and adequate rest to the part
   - Awareness of injury
   - Proper footwear
   - Care of the hand and prevention of injury

Treatment

Multidrug therapy with Dapsone, Clofazimine and Rifampicin is given in appropriate doses. Treatment must be continued till the disease is clinically inactive and bacteriologically negative.

Physiotherapy

Efficient preliminary physiotherapy with active exercises, massage, passive stretching and wax bath, along with splints, must be given before undertaking any tendon transfer operations. Even after the operation, intensive physiotherapy has to be given for re-education of the transferred tendons to perform the new functions.

Surgical Treatment

Decompression of the nerve done on the swollen ulnar or lateral popliteal nerve by a longitudinal incision in the early stages arrests the progress of the paralysis and leads to recovery of the paralysis in many cases.

When the damage to the nerve is irreversible, reconstructive surgery has to be undertaken to restore or improve the function.

Lesions in the Foot

The following lesions of the foot will be discussed:

- Foot drop
- Trophic ulcer

Foot Drop

Foot drop is a condition where the patient is unable to actively dorsiflex the foot at the ankle. The foot drops down when the patient lifts the foot off the ground in the swing phase of walking.

Aetiology

The most common cause of foot drop in India is lateral popliteal nerve paralysis due to leprosy. The other causes of foot drop are residual poliomyelitis, old injuries to the lateral popliteal or sciatic nerve, cauda equina tumour and massive intervertebral disc prolapse at the L5-S1 level.

Clinical Features

On examination, the patient is unable to actively dorsiflex and evert the foot. There will be areas of diminished sensation in the foot. During walk,

the patient lifts the foot high to clear the toes of the dropped foot from dragging on the ground. This is called high-stepping gait. There will be tenderness and thickening of the lateral popliteal nerve at the neck of fibula.

Conservative Treatment

Orthotic appliances: An ankle foot orthosis with a 90° foot drop stop is prescribed.

Surgical Treatment

In the early stages of neuritis, when there is marked swelling of the nerve with tenderness, surgical decompression of the nerve at the upper third of the leg gives relief from pain and promotes recovery of the paralysis.

- *Tendon transfer operation:* In later stages, when the foot drop has not recovered, tendon transfer operation is indicated. The transfer of the insertion of the tibialis posterior tendon to the dorsum of the foot gives good results. Srinivasan's procedure of two-tailed transfer of tibialis posterior to the extensor hallucis and digitorum tendons gives good results.
- *Triple arthrodesis:* Stabilisation of the foot by triple arthrodesis can be done for cases of foot drop in leprosy as it is done for poliomyelitis.

Trophic Ulcer (Plantar Ulcer)

The anaesthetic ulcerating foot is a common complication of leprosy. The anaesthesia is not only in the dorsum but also in the plantar aspect of the foot. Trophic ulcer is an ulcer occurring in the weight-bearing area of the sole of the anaesthetic foot. It is more appropriately called the plantar ulcer. The most common sites are over the ball of the big toe and the heads of the other metatarsal bones in the forefoot. The other sites are over the heel and in the lateral border of the foot.

Pathology

The posterior tibial nerve involvement results in anaesthesia in the sole of the foot and paralysis of intrinsic muscles of the foot, resulting in clawing of the toes. Walking on the anaesthetic clawed foot causes abnormal *shearing pressures* over the heads of metatarsals, resulting in tissue destruction and blister formation. The precipitating causes for the ulcer are external trauma from stones or glass pieces, etc., on the ground or ill-fitting foot wear.

It starts as a blister and then forms an acute ulcer; if neglected, it spreads and ends up as a chronic indolent ulcer. This ulceration leads to destruction of soft tissues exposing the bones to secondary infection, osteomyelitis and destruction of the phalanges and metatarsal bones.

In addition, they develop neuropathic arthritis (Charcot's type) in the tarsal and other small joints of the foot. This leads to atrophy, destruction and absorption of the articular surfaces of the bones of the foot and ankle.

Treatment

The most important principle is the interruption of the abnormal walking cycle in the anaesthetic foot. Treatment consists of the following measures:

- Rest to the part
- Control of infection
- Special footwear
- Surgery

Rest

Rest alone will promote healing in the early blister stage and in the acute ulcer stage. In the stage of spreading and indolent ulcers, rest to the foot from weight bearing and walking contributes most to the healing of the ulcer. The rest is best given by absolute bed rest for a week or two or resting the foot in splints. A below-knee padded plaster cast gives good rest and promotes healing in most cases in 4–6 weeks.

Control of Infection

This is done by local dressing with antiseptic solutions. Ulcers with slough are dressed with Eusol solution. When the foot is grossly swollen with discharging ulcers, dressing with Glycerin Magsulph helps to reduce the swelling by its hygroscopic property. Systemic antibiotics are also given.

Special Footwear

Footwear for leprosy patients is essential to protect them from external trauma and prevent sores.

Use of microcellular rubber as insole for the footwear is necessary to prevent breakdown of scars and recurrences of the healed ulcer.

Surgery

In cases of trophic ulcers with persistent infections due to osteomyelitis, scraping the slough and sequestrectomy will clear the infection. Healing is promoted by rest in plaster and dressings. In cases with very gross destruction and deformities of the whole foot, a below-knee amputation may have to be done when all the conservative measures fail.

Deformities in the Hand

The involvement of the ulnar and median nerves in leprosy causes primary deformities due to loss of motor functions in the hand. In neglected cases, secondary deformities develop due to loss of sensation, trophic changes, trauma and absorption of the skeletal tissues.

Total Claw Hand

Claw hand is the deformity of the hand where the metacarpophalangeal joints are in hyperextension and the interphalangeal joints are in flexion.

This is due to the tuberculoid lesion involving the ulnar nerve at the elbow and the median nerve at the wrist. The intrinsic muscles normally flex the metacarpophalangeal joints and extend the interphalangeal joints. When the intrinsic muscles are paralysed, the long extensors pull the metacarpophalangeal joints into hyperextension and the long flexors hold the interphalangeal joints in flexion causing the clawing deformity. This is called intrinsic minus position of the hand.

(The opposite deformity of intrinsic plus position is seen in rheumatoid arthritis of the hand, where there is spasm of the intrinsic muscles.)

Clinical Features

In total claw hand, all the four fingers are in the clawed position (Fig. 10.4). There is wasting of the hypothenar area, hollowed-out appearance of the thumb web space and intermetacarpal spaces due to wasting of the interossei muscles. The loss of thenar muscle power of opposition makes the thumb fall back to an 'Ape thumb' position. The clinical tests for ulnar and median nerve palsies will be positive:

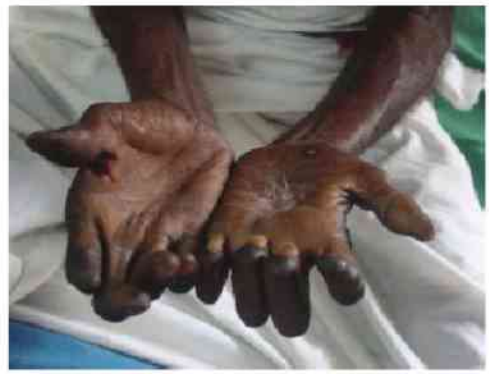


FIGURE 10.4 Clinical photo of bilateral claw hand (total claw hand on the left side).

1. Median nerve
   - Pointing index finger test (Fig. 16.7)
   - Ape thumb deformity (simian hand)
   - Tests for opponens palsy
   - Pencil test for abductor pollicis brevis
   - Inability to flex the interphalangeal joint of the thumb
2. Ulnar nerve
   - Interosseous function test of abduction and adduction of the fingers (Fig. 10.5)
   - Froment's test for abductor pollicis (Card test) (Fig. 10.6)

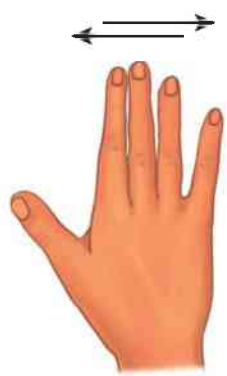


FIGURE 10.5 Diagrammatic representation of Eagwa's test.

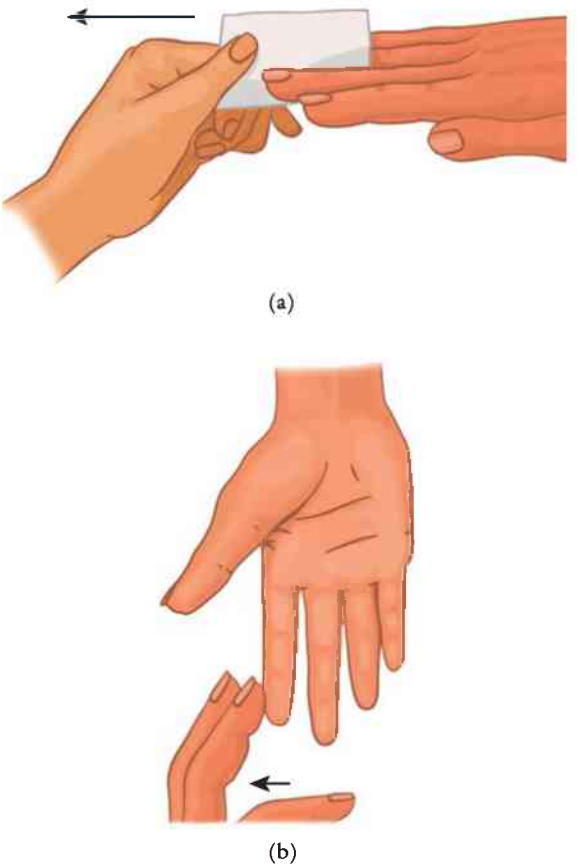


FIGURE 10.6 Diagrammatic representation of (a) card test and (b) testing abductor function of interossei.

Treatment

After physiotherapy to mobilise any stiffness in the joints, replacement of intrinsic power is done by the following tendon transfer operations.

Brand's Many-Tailed Tendon Transfer Operation (EF4T)

Brand's many-tailed tendon transfer operation was developed by Paul Brand at the Christian Medical College, Vellore, and further refined by Selvapandian. The extensor carpi radialis longus is detached from its insertion and used as a motor. A free tendon graft from the palmaris longus tendon is split into four strips and attached to the motor tendon. The four parts are attached to the extensor expansion of the respective fingers to replace the lumbrical power.

Tendon Transfer for Opponens Palsy of the Thumb

The flexor digitorum sublimis of the ring finger is detached from its insertion, rerouted to the radial side of the thumb and inserted into the lateral margin of the extensor expansion.

Ulnar Claw Hand

The partial claw hand is more common and is due to the involvement of ulnar nerve alone at the elbow. This results in intrinsic muscle paralysis as well as paralysis of flexor digitorum profundus to the little and ring fingers. There is hyperextension of the metacarpophalangeal joints and flexion deformity at the interphalangeal joints of the ring and little fingers. The tests for intrinsic muscle paralysis will be positive.

Bunnell's Operation

In this operation, the flexor digitorum sublimis tendon to the ring finger is disinserted from the middle phalanx, split into two strips rerouted and inserted into the extensor expansion on the ring and little finger.

DISORDERS OF MUSCLES AND CONNECTIVE TISSUE

The following disorders will be discussed:

- Muscular dystrophy
- Arthrogryposis multiplex congenita
- Fibrodysplastic ossificans progressiva

Muscular Dystrophies

Muscular dystrophy is a group of disorders of the muscles, resulting in difficulties in locomotion. The clinical and pathological manifestations are well described in textbooks of medicine. The detection of these conditions and their orthopaedic management alone will be described.

Types

The following are the common clinical forms of the disease:

- Pseudo-hypertrophic muscular dystrophy
- Limb girdle muscular dystrophy
- Facioscapulohumeral dystrophy

Pseudo-Hypertrophic Muscular Dystrophy (Duchenne)

Pseudo-hypertrophic muscular dystrophy is a sex-linked recessive type of muscular disorder wherein there is hypertrophy of muscles with weakness. It is more common in males. It begins in childhood before the age of 5 years.

Clinical Features

In early stages, the child is brought with the history of frequent falls and hesitation in walking. The child has difficulty in getting up from the squatting position.

On examination, the calf muscles may appear bigger than normal. Gower's sign is positive. When the child is asked to get up from the squatting position, the child gets up by climbing up on his lower limbs and supporting his weight with his hands on his legs, knees and thighs. The child develops equinus deformity. He also develops a waddling gait exaggerated lumbar lordosis due to gluteal muscle involvement.

Later, the shoulder girdle muscles are wasted and weak. It is difficult to lift the child by supporting him under his armpits as the power of the shoulder girdle is much diminished and the child is unable to hang himself on the supporting hands of the examiner.

At around 10–15 years of age, the child is unable to walk and is confined to bed. He develops deformities of ankles, knees, hips and spine. Muscles of face, respiration and swallowing are unaffected. Finally, it ends fatally by the end of the second decade by involvement of cardiac muscles.

Laboratory Diagnosis

These conditions have certain characteristic biochemical features due to the disturbance in creatine metabolism.

- *Blood:* The diagnostic feature of myopathy is increased creatine phosphokinase enzyme (CPK) in the blood. The CPK level is raised to several times the normal which is 60 IU.
- *Urine:* The excretion of creatine is increased. The excretion of creatinine is decreased.
- *Muscle biopsy:* Muscle biopsy findings confirm the diagnosis of myopathy. Macroscopically, the 'fish flesh' appearance is due to the replacement of the muscle by fat and fibrous tissue.

Electromyograph of the affected muscle is of diagnostic value. The normal fibrillation potential is absent.

Management

The main treatment is to achieve muscle balance and stabilisation and to help the patient walk independently as long as possible. When the muscles are weak, active strengthening exercises are done. Passive movements of joints are also given. When there is difficulty in walking, long-leg calipers are prescribed. Surgical measures such as tendo achilles lengthening help to correct equinus deformities.

Arthrogryposis Multiplex Congenita (Myodystrophia Foetalis; Amyoplasia Congenita)

Arthrogryposis is characterised by symmetrical contracture of multiple joints in the body. It is manifest at birth and may be generalised or localised to one limb.

Aetiopathology

The exact aetiology is unknown. There are two distinct types: the *neurogenic* type, which is more common, and the *myogenic* type. The neurogenic type is due to degeneration of anterior horn cells in certain segments of the spinal cord and intrauterine paralysis of the corresponding muscles. In the myogenic type, there is a myodystrophy in the intrauterine state with replacement of muscles by fibrofatty tissue.

Clinical Features

In the myogenic type, legs are in flexion abduction at the hips and flexion at the knees. The feet may be in gross equinovarus deformity. Typically, the skin is smooth and creases at the joint

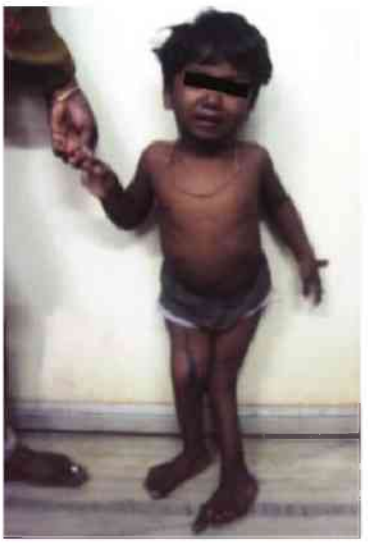


FIGURE 10.7 Clinical photo of arthrogryposis multiplex congenita.

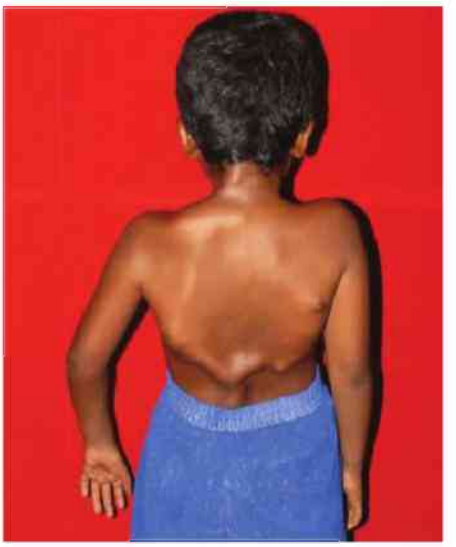


FIGURE 10.8 Clinical photo of myositis ossificans progressiva.

levels are absent (Fig. 10.7). The legs appear smooth and tapering with the joints as fusiform enlargements giving a mermaid appearance. The hips often show congenital dislocation. Intelligence is normal and there is no sensory deficiency.

In the neurogenic type, the deformities depend on the level of the lesion in the spinal cord and imbalance in muscle activity. The hips are in flexion and the knees in hyperextension (genu recurvatum) with the feet in calcaneo valgus. In the upper limbs, the elbow may be in extension or in flexion with skin webs, pronation of the forearm and flexion at the wrists.

Treatment

The aim of treatment is to make the patient ambulant by correction of the deformities. Aggressive surgical correction of the deformities must be done followed by splinting and exercise therapy. The club feet may need talectomy and hips will often need open reduction.

Fibrodysplasia Ossificans Progressiva (Myositis Ossificans Progressiva)

Fibrodysplasia ossificans progressiva is an uncommon condition characterised by progressive ossification of the connective tissue of the skeletal muscles, ligaments and fascia. It is a fibrodysplasia and is not an inflammatory condition of muscles. What was called myositis ossificans is now called fibrodysplasia ossificans progressiva.

The condition starts in childhood with painful swellings in the muscles of the back, neck and limbs. These swellings progressively harden due to calcification and ossification and lead to stiffening of the back and limitation of joint movements (Fig. 10.8). There are usually associated congenital short big toes and single phalanx. The disease shows remissions and exacerbations, ultimately totally crippling the child. Radiologically, the involved soft tissues show sheets of ossification. There are no biochemical changes in blood. Treatment is mostly symptomatic.

KEY POINTS

- Cerebral palsy (Little's disease) is a non-progressive neurological disorder of children affecting the locomotor system.
- Cerebral palsy is classified into five types: (1) spastic type, (2) athetoid type, (3) ataxic type, (4) rigid type and (5) mixed type.
- Spastic type is the most common type of cerebral palsy. It can be grouped, according to limb involved, into monoplegia, paraplegia, hemiplegia and quadriplegia.
- Clinically, there is history of delayed milestones and adductor spasm at hips.
- Gait: Scissored gait in spastic type and broad-based gait in ataxic type.
- Poliomyelitis is caused by a neurotropic virus which has affinity for the neurons in the anterior horn of the spinal cord and also for the motor nuclei in the brainstem.
- Characteristically in polio there is a lower motor neuron type of paralysis.
- Abortive poliomyelitis is a term used to indicate clinical recovery.
- Management of polio includes the following interventions: orthopaedic care, physiotherapy, orthotic management and surgery.
- Leprosy is a bacterial disease, classified clinically, into multibacillary and paucibacillary.
- Leprosy patients present with nerve palsies (e.g. lateral popliteal nerve palsy) and trophic ulcers.
- The most common cause of foot drop in India is lateral popliteal nerve paralysis due to leprosy.
- The posterior tibial nerve involvement results in anaesthesia in the sole of the foot and paralysis of intrinsic muscles of the foot.
- Pseudo-hypertrophic muscular dystrophy is characterised by hypertrophy of muscles with weakness. On examination, Gower's sign is positive.
- Arthrogryposis multiplex congenita is characterised by symmetrical contracture of multiple joints of body. There are two distinct types: neurogenic and myogenic.

MULTIPLE CHOICE QUESTIONS

1. Equinus deformity in poliomyelitis is due to weakness of
   a. Dorsiflexors
   b. Plantar flexors
   c. Evertors
   d. Invertors
2. Polio paralysis differs from paralysis due to other causes
   a. Weakness
   b. Deformity of limbs
   c. No sensory loss
   d. Full recovery is possible
3. Claw hand lesion is caused by
   a. Ulnar nerve
   b. Median nerve
   c. Radial nerve
   d. Posterior interosseous nerve
4. Clumsiness of hand in case of leprosy is due to involvement of
   a. Interosseous muscle
   b. Abductor pollicis longus
   c. Extensor carpi ulnaris
   d. Flexor carpi ulnaris
5. Trophic ulcer is seen in all conditions except
   a. Polio
   b. Leprosy
   c. Tuberculosis of the central nervous system
   d. Spinal dysraphism
6. Which nerve repair has the worst prognosis?
   a. Ulnar
   b. Radial
   c. Median
   d. Lateral popliteal

CHAPTER 11

Regional Conditions of Neck and Upper Limb

NECK

Brachial Neuralgia

Brachial neuralgia is defined as pain radiating down the arm. In many aspects, it is similar to 'sciatica' or pain down the lower limb. The presence of the brachial and lumbar plexuses at the junction of the upper and lower limbs to the trunk, respectively, and the erect posture of the human being contribute to the causation of similar pain syndromes down the upper and lower limbs.

Clinical Features

It is common in the middle aged or elderly patients. The pain usually starts as a pain in the neck or shoulder and later spreads down the arm down to the tips of the fingers. When the pain radiates to the index fingers and thumb, it is due to an irritation of C5, C6 cervical nerve roots. A similar shooting pain to the tips of the little or ring finger is due to irritation of the C8 or T1 nerve roots.

Examination of the Neck

On inspection, note the presence of stiffness and muscle spasm. On palpation, confirm the presence of spasm and palpate the spine for localised tenderness. The whole trapezius must be palpated for tender spots and tender nodules along the attachment of the muscle to the spine of the scapula. Palpate the root of the neck for any lump or tenderness. Movements of neck flexion, extension and lateral flexion must be tested and any painful limitation noted.

Examine the shoulder joint for painful limitation of movements and tender points.

Neurological Examination

The neurological examination of the upper extremity must be done first. Look for sensory deficiency or loss in the fingers and thumb. Look also for motor weakness of the muscles supplied by the nerve roots (C5, C6, C7, C8) of the brachial plexus (Fig. 11.1). The biceps, triceps and brachioradialis jerks are tested for. The sensory and motor deficiency will help to localise the root involved and the level of the causative lesion. Evidence of cord compression must be looked for, in the lower limbs. Radial pulse should be felt on both sides for any difference.

Investigations

Radiographs of the cervical spine (anteroposterior, lateral and oblique views) are taken.

Aetiological Factors

The causes of the pain down the arm can be classified as follows:

1. Causes in the neck
   a. Postural causes—drooping shoulder
      - Conditions in the muscles, fascia, ligaments and glands

b. Traumatic causes
   - Acute sprain
   - Chronic occupational strain

c. Inflammatory causes
   - Cervical lymphadenitis
   - Conditions in the cervical spine

d. Intervertebral disc prolapse
e. Lesions in the vertebral bodies
   - Trauma: Old fractures, dislocations and subluxations
   - Tuberculosis
   - Tumour deposits

f. Cervical spondylosis
g. Ankylosing spondylitis
h. Conditions in the thoracic inlet
i. Cervical rib and scalene syndromes
j. Tumours in the thoracic inlet (Pancoast tumour)
k. Intraspinal conditions
l. Cord tumours
m. Syringomyelia
n. Extradural tumours—neurofibroma

2. Shoulder lesions
   a. Periarthritis
   b. Supraspinatus tendinitis
   c. Subdeltoid bursitis
3. Referred pain
   a. Cardiac ischaemia can cause left-sided brachial neuralgia
   b. Subdiaphragmatic lesions such as gall bladder lesions cause right-sided pain
4. Systemic causes: Diabetic neuropathy

Cervical Intervertebral Disc Prolapse

The prolapse of the intervertebral disc in the lower cervical spine is a common cause for acute brachial neuralgia. This is common in young adults and middle-aged persons. There is usually a history of a sudden catch in the neck. The patient presents with an acute pain in the neck, radiating down the arm to the tips of the fingers.

Clinical Features

On examination, the neck is held stiff and the cervical muscles are in spasm. There may be tenderness over the C5, C6 or C7 spinous processes. Flexion and extension movements are markedly limited. Holding the arm in abduction at the shoulder and elbow in flexion often relieves the pain. Pain is worse on 'coughing and sneezing'.

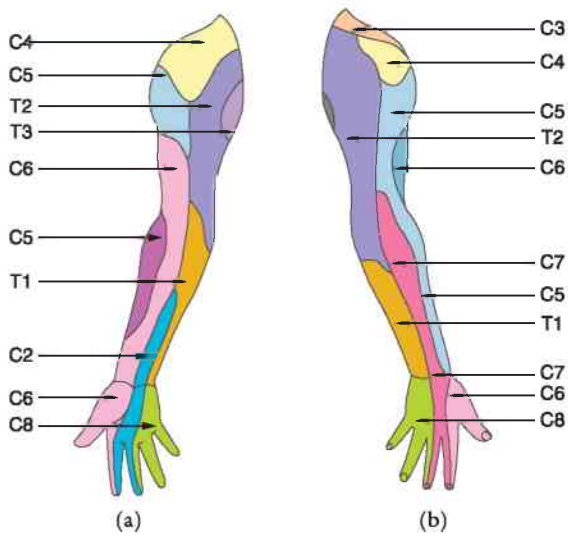


FIGURE 11.1 Diagrammatic representation of dermatomes in the upper limb. (a) Anterior view and (b) posterior view.

Neurological examination will determine the level of the disc prolapse and the root compressed. The most common level of prolapse is the C5–C6 disc which compresses the C6 nerve root. There is hypoaesthesia in the outer border of the forearm, thumb and index finger. The biceps jerk will be diminished or absent. There will be mild weakness of the biceps. Prolapse of the C6–C7 disc involves the C7 nerve root. There is diminished sensation in the index and middle fingers. There is weakness of the triceps muscle and diminished or absent triceps jerk.

Radiological Features

Radiological examination may be normal in the first episode. It shows narrowing of the affected disc space with sclerosis of the adjacent vertebral borders and anterior osteophytes (Fig. 11.2) when the patient has had a few attacks.

Treatment

In the acute stage of the attack, the patient must be put to bed rest. Intermittent cervical traction will relieve the pressure on the nerve root and the

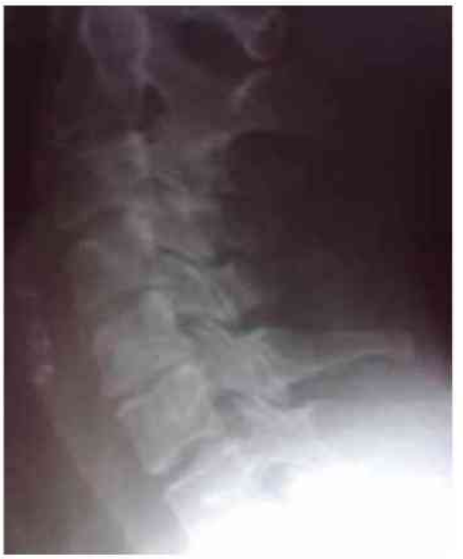


FIGURE 11.2 X-ray of cervical spine lateral view showing cervical intervertebral disc lesion at C5–C6 level.

pain will subside. The patient is also given analgesics and anti-inflammatory drugs.

Bed rest, analgesics and anti-inflammatory drugs will relieve the symptoms in a large number of cases. When the pain subsides, the patient is given a cervical collar and advised to avoid jerks to the neck. *Shoulder bracing exercises* are prescribed which help to maintain the shoulder elevated which relieves the irritation of the nerve roots. The exercises prevent further attacks of brachial neuralgia.

Massive prolapse will cause spinal cord pressure in addition to root irritation. This can be established by contrast myelography, CT scan or MRI. It is relieved by surgical decompression and disc excision.

Cervical Spondylosis

Cervical spondylosis is defined as arthrosis of the posterior intervertebral joints in the cervical vertebrae. It is common in the middle aged and the elderly.

Pathogenesis

In the early stage, it is localised to two or three cervical vertebral segments, due to degeneration of the intervertebral disc with narrowing and osteophyte formation at the anterior and posterior margins. The osteophytes cause narrowing of the intervertebral foramen resulting in nerve root irritation. In the later stage, there is a generalised degenerative arthrosis of the posterior intervertebral joints of the whole cervical spine. In the extreme form, there is compression of the spinal cord with myelopathy and symptoms of cord lesion.

Clinical Features

The patient presents with chronic pain in the neck with or without radiating pain down the arm. There will be diffuse tenderness in the cervical spine with limitation of all movements. The neurological signs will be confined to one or two roots.

Radiological Features

Radiologically, there will be narrowing of C5–C6 or C6–C7 disc space with osteophytic changes (Fig. 11.3). There may be narrowing of joint space of the posterior intervertebral joints. There will be narrowing of the intervertebral foramen which is well demonstrated in oblique views of the cervical spine.

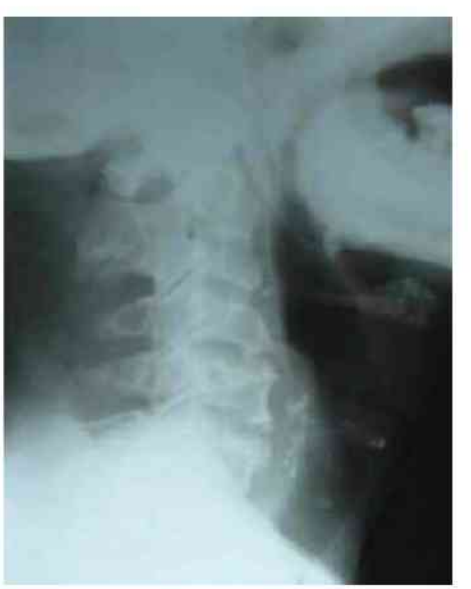


FIGURE 11.3 X-ray of cervical spine lateral view showing features of cervical spondylosis.

Treatment

The treatment includes physiotherapy with short-wave diathermy to the neck, intermittent cervical traction and analgesics. When the pain is controlled, the patient is taught shoulder bracing and neck exercises. In the acute painful stage, a cervical collar is prescribed. In the cases with cervical myelopathy, surgery will be indicated.

Cervical Rib

Cervical rib is a congenital condition characterised by the presence of an extra rib at the seventh cervical vertebral level. The condition is usually unilateral and is common on the right side.

Symptoms similar to those caused by cervical rib can also be produced by the abnormal insertion of the scalenus anterior or scalenus medius muscle, which produces compression of the lowest trunk of the brachial plexus and the subclavian vessels as they cross the first rib. These are referred to as scalene syndromes or thoracic outlet syndromes.

Pathological Anatomy

There are two types: complete and incomplete. The rib can be completely bony, extending from the C7 transverse process, passing between the scalenus anterior and medius muscles to join the anterior part of the first rib (Fig. 11.4). It can be proximally bony and distally cartilaginous or fibrous. The lower cord of brachial plexus and the subclavian artery pass above this rib instead of the first rib and can get stretched or compressed at certain positions of the shoulder.

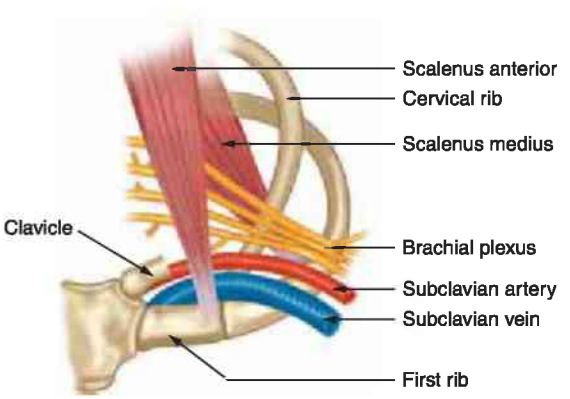


FIGURE 11.4 Diagrammatic representation of the cervical rib, seen passing between scalenus anterior and scalenus medius.

Clinical Features

Although it is present from birth, symptoms appear only in the third or fourth decade, when there is sagging of the shoulders. The presenting symptoms may be local, neural or vascular:

- *Local:* The patient presents with the complaint of diffuse pain in the root of the neck or tingling pain.
- *Neural:* There is numbness down the medial border of the arm extending to the tips of the little and ring fingers associated with small muscle wasting of the hand.
- *Vascular:* They include compression of subclavian artery leading to aneurysm formation. Tiny emboli may dislodge resulting in gangrene of fingers.

On examination, there may be some prominence in the root of the neck in the posterior triangle, just above the clavicle. Palpation will cause tenderness and even tingling down the arm. There will be diminished sensation in the ulnar nerve distribution in the hand. In late cases, there may be motor weakness of the intrinsic muscles and wasting of the hypothenar muscles.

Some vascular signs are manifested due to the pressure of the abnormal rib on the subclavian artery. Adson's test demonstrates the compression of the subclavian artery by the cervical rib. With the patient sitting or standing, he is asked to turn his head on the side of the symptom. With your fingers feeling the radial pulse, ask the patient to raise his chin upwards and take a deep breath. If the radial pulse disappears, the sign is positive.

Pulsation may be prominently palpable as a thrill above the clavicle and a bruit may be auscultated. There may be decrease in the strength of the radial pulse on applying downward pull on the hands.

Radiological Feature

The anteroposterior view of the neck will show varying degrees of the cervical rib on one or both sides (Fig. 11.5).

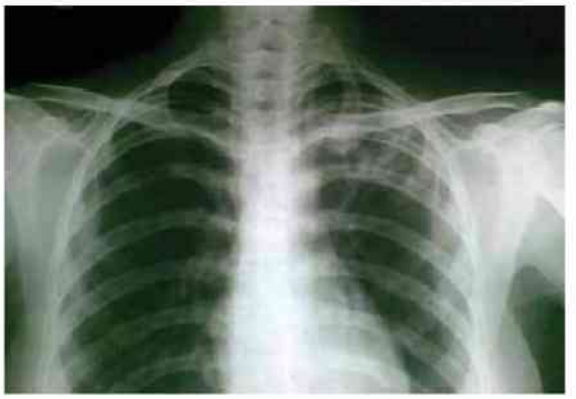


FIGURE 11.5 X-ray of chest demonstrating cervical rib.

Treatment

In most cases where symptoms are mainly neurological, they can be relieved by toning up the muscles by bracing of the shoulders by exercise therapy. Analgesics and anti-inflammatory drugs help in getting relief from the pain. In cases where the vascular signs are predominant and neurological signs are severe, surgical decompression of the brachial plexus and the subclavian vessels is done by resecting the cervical rib or the scalenus anterior muscle.

TORTICOLLIS

Torticollis is a deformity of the neck characterised by a tilt of the head with rotation of the occiput to the affected side and the chin to the opposite side. There are two types of torticollis:

1. *Congenital torticollis:* It is the more common sternomastoid type (refer to Chapter 2).
2. *Acquired torticollis:* It is also known as secondary torticollis and is due to the following causes:
   - Simple fibrositis in the neck called wryneck
   - Traumatic sprain of the neck
   - Acute cervical lymphadenitis
   - Diseases in cervical spine such as caries or tumour

In cases where torticollis is secondary to some other disease, it will get corrected when the main disease is treated.

Spasmodic torticollis is characterised by repeated uncontrolled spasm of the muscles on the side of the neck in an adult. There is a marked functional or psychogenic basis for the condition, and it is intractable to medical or surgical treatment.

UPPER LIMB

Pain around the shoulder with diffuse radiation down to the middle of the arm is a common clinical presentation in the middle-aged people. The rotator cuff and its adjacent structures are prone to inflammatory and degenerative changes. Some of the common presentations of this are lesions relating to the supraspinatus tendon.

Supraspinatus Syndrome

Supraspinatus syndrome refers to a group of conditions related to the supraspinatus tendon and the rotator cuff. The lesions include the following:

- Supraspinatus tendinitis
- Supraspinatus calcification
- Subdeltoid bursitis

Supraspinatus Tendinitis

The patient is usually 20–40 years old. He presents with acute pain in the shoulder and inability to abduct the shoulder. On examination, there is a tender spot just below the acromion laterally and anteriorly above the greater tuberosity.

On abduction, the movement up to 60° is painless. The range between 60 and 120° is acutely painful. Further range beyond 120° is painless. This painful arc of abduction is diagnostic of supraspinatus tendinitis. This is due to the impingement of the supraspinatus tendon against the edge of the acromion and coracoacromial arch.

In some cases, the subdeltoid bursa gets inflamed secondary to tendinitis. This may manifest as palpable crepitation during abduction.

Radiological Features

Radiograph will be normal in simple tendinitis. It may sometimes show an oval area of calcification

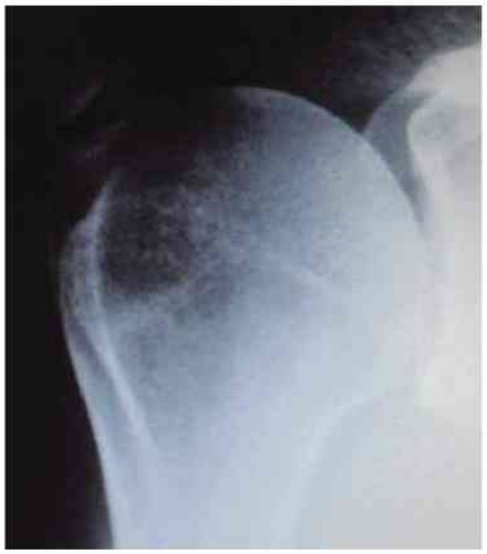


FIGURE 11.6 X-ray of right shoulder anteroposterior view demonstrating supraspinatus tendon calcification near greater tuberosity.

near the top of the greater tuberosity of humerus (Fig. 11.6).

Management

In the acute stage, the shoulder is rested in a cuff and collar sling and anti-inflammatory drugs are administered. These cases respond well to a course of ultrasound therapy to the tender area. When the pain persists, local infiltration of hydrocortisone and lignocaine is done. When the pain subsides, active exercises are started.

Rupture of the Rotator Cuff

With advancing age, the supraspinatus tendon, a part of the musculotendinous cuff, undergoes degenerative changes with diminishing subdeltoid bursal efficiency. Repeated impingement of the supraspinatus tendon against the acromion leads to tendinitis and attrition. At this stage, jerky violence may result in a rupture of the tendon which may be partial or complete. Occasionally, the rupture can also occur in a younger person due to severe violence.

The stages of rupture are classified by Neer as Stage I: oedema and haemorrhage, Stage II: fibrosis and tendinitis and Stage III: bone spur and tendon rupture.

Clinical Features

The patient presents with pain and inability to abduct the shoulder. There is tenderness over the top of the greater tuberosity. In partial tears, the patient will be able to abduct the shoulder with some assistance, but there will be a painful arc.

In complete ruptures, there will be inability to initiate abduction. When the patient attempts to abduct, the deltoid will be found to be actively contracting, but the shoulder is not abducted but goes into an elevated position. If the shoulder is passively abducted initially, further abduction will be possible.

Once the arm has been passively lifted up above 90°, the patient can actively hold it up by using the deltoid. This is the abduction paradox.

Passive abduction is full. While the arm is then brought down, he can control it down to 90° but further down the arm drops suddenly. This is called the arm drop sign.

Imaging Techniques

Plain radiograph of the shoulder will be normal. Arthrography will show a communication between the joint and the subacromial bursa.

Treatment

Incomplete ruptures are treated by rest and analgesics, followed by short-wave diathermy and exercise therapy. Total ruptures of the rotator cuff will need surgical repair.

Periarthritis Shoulder

Periarthritis shoulder is a condition characterised by pain and progressive limitation of some movements of the shoulder joint occurring in the elderly.

The patient, past middle age, presents with diffuse pain in the shoulder with radiation down to the middle of the upper arm. It occurs in two types: primary idiopathic type for which the cause is not known and secondary type occurring in patients with diabetes, tuberculosis, cardiac ischaemia and hemiplegia. On examination, there will be tenderness in the subacromial region and in the anterior joint line. There is marked limitation of abduction and external rotation of the shoulder

with free and full range flexion and extension movements.

When the condition involves the whole rotator cuff, it results in total restriction of all movements of the joint. The condition is then termed 'frozen shoulder or adhesive capsulitis'. Radiograph of the shoulder is normal.

Treatment

Pain is controlled by analgesics and short-wave diathermy or wax bath. Mobilisation is done by external rotation and abduction exercises. Resistant cases are treated by local infiltration of hydrocortisone and manipulation under anaesthesia.

Deltoid Fibrosis

Deltoid fibrosis is the contracture of the deltoid muscle fibres causing abduction deformity of the shoulder. This is a clinical entity, first described in India by Battacharya (Calcutta). It is a localised fibrosis of the middle fibres of the deltoid, resulting in an abduction contracture of the shoulder. Shanmugasundaram (Madras) has shown the causal relationship between the intramuscular injection of certain drugs such as tetracycline, quinine, paraldehyde, etc., into the deltoid in childhood and the occurrence of deltoid fibrosis during adolescence.

Clinical Features

The patient usually complains of an inability to adduct the arm (Fig. 11.7). More commonly, the patient is brought for the abnormal prominence of the scapula as he attempts to keep the elbow close to the body. On examination, the arm is held in about 30° abduction and there is a wasting of the deltoid. A tight cord-like vertical band is seen and felt in the middle fibres of the muscle. On passive adduction of the arm, the band gets further tightened and the scapula becomes more prominent. Abduction and other movements are free.

Treatment

The treatment is the surgical division of the contracted band of the middle fibres of the deltoid. Occasionally, extensive release of the deltoid origin is necessary.

FIGURE 11.7 Clinical photo of deltoid fibrosis in the left upper limb.

ELBOW

Tennis Elbow (Lateral Epicondylitis)

Tennis elbow is an inflammatory condition of the common extensor origin over the lateral epicondyle. This condition does not affect tennis players only. It often follows an injury or sudden contraction of the common extensor origin. The exact aetiology is not known. It may be a partial tear of the fibres of origin of the extensor muscles.

The patient is usually an adult complaining of pain in the lateral aspect of the elbow. The patient feels a sense of weakness when lifting even small objects. There is localised tenderness over the lateral epicondyle. The movements of the elbow and radioulnar joints are normal.

Test: Ask the patient to keep the elbow, wrist and fingers in full flexion and the forearm in full pronation. Jerky extension of the elbow causes marked pain at the lateral epicondyle.

Radiograph of the elbow is normal.

Treatment

Rest to the elbow with administration of anti-inflammatory drugs is very helpful. A course of ultrasonic therapy is given. Resistant cases will

respond to local injection of hydrocortisone. Occasionally, surgical erasure of the common extensor origin from the lateral epicondyle is indicated.

Recurrent Subluxation of the Ulnar Nerve

Recurrent subluxation of the ulnar nerve is a condition where the ulnar nerve at the elbow slips forwards over the medial epicondyle of the humerus. It is due to the congenital absence of the fibrous roof over the groove in the back of medial epicondyle in which the ulnar nerve normally lies. This condition is bilateral and often familial as well.

The patient presents with symptoms of tingling and numbness in the area of the ulnar nerve distribution in the hand, particularly on flexing the elbow. The nerve can be seen or felt slipping forwards over the medial epicondyle when the elbow is slowly flexed. The patient also feels numbness in the little finger when resting the elbow on the table during writing. If the symptoms are severe, the treatment is surgical by anterior transposition of the ulnar nerve.

Cubitus Valgus

The term 'cubitus' means elbow and 'valgus' means 'outward angulation'. Cubitus valgus is a deformity of an outward deviation of the forearm at the elbow. This is an exaggeration of the normal valgus or carrying angle present in the elbow. Normally, the forearm deviates outwards at the elbow by about 10° in the male (Fig. 16.2) and 15° in the female. Females have increased carrying angle to accommodate their pelvis. Anything that is more than the normal carrying angle is termed 'cubitus valgus'. Lateral angulation deformity of elbow is seen when the patient stands with his arms by his sides and palm facing front. This condition is termed 'cubitus valgus' (Fig. 16.2).

The usual causes are as follows:

- Non-union of a displaced fracture of the lateral condyle of the humerus is the most common cause.
- Destruction of the lateral condyle of the humerus, due to septic infection in childhood.

The patient presents with increasing deformity of an outward deviation of the forearm. Medial epicondyle becomes more prominent and may cause slow stretching of the ulnar nerve over years. Later, the patient may also present with features of a delayed ulnar neuritis and paralysis due to the stretching of the nerve. This delayed onset of ulnar nerve paralysis is called tardy ulnar palsy. The patient complains of weakness and sensory disturbances along the distribution of the ulnar nerve.

When there is evidence of ulnar neuritis, it is treated by anterior transposition of the ulnar nerve. When the deformity is gross, it is surgically corrected by a supracondylar osteotomy.

Cubitus Varus

Cubitus varus is a deformity with an inward deviation of the forearm at the elbow. Medial angulation deformity of the elbow is visible when the patient stands with his arms by his sides and the palms are facing front (gunstock deformity) (Fig. 16.8).

The most common cause is malunion of a supracondylar fracture in childhood. This is cosmetically more disfiguring than the cubitus valgus; hence, correction is indicated. The range of movements at the elbow is full.

Minimal deformities in children may get corrected by natural moulding and may be left alone. If the deformity is severe, it needs surgical correction by

- lateral closing wedge osteotomy,
- French osteotomy.

WRIST AND HAND

Anatomy

Skeleton of the hand includes eight carpal bones, five metacarpal bones and 14 phalanges. The first digit has two phalanges and the other five digits have three phalanges each. Intrinsic muscles of the hand have their proximal and distal attachments within the hand. Thenar

muscles form the thenar eminence and hypothenar muscles form the hypothenar eminence. The third group of muscles includes the interossei and the lumbricals.

The hand is supplied mainly by two arterial arches. Superficial palmar arch is contributed mainly by the ulnar artery and deep palmar arch by radial artery. The nerve supply of the hand is derived from three nerves—median, radial and ulnar nerves.

1. *Carpal tunnel:* It is an osseofibrous tunnel formed by the flexor retinaculum bridging over the carpal bones at the level of the wrist. Structures passing through the carpal tunnel include the following:
   - Median nerve
   - Four tendons of flexor digitorum superficialis
   - Four tendons of flexor digitorum profundus
   - Tendon of the flexor pollicis longus
2. *Anatomical snuff box (Fig. 11.8):* It is the depression in the posterolateral surface of the wrist that is bounded as follows:
   - Anteriorly—tendon of abductor pollicis longus and extensor pollicis brevis
   - Posteriorly—extensor pollicis longus

   Content: Radial artery

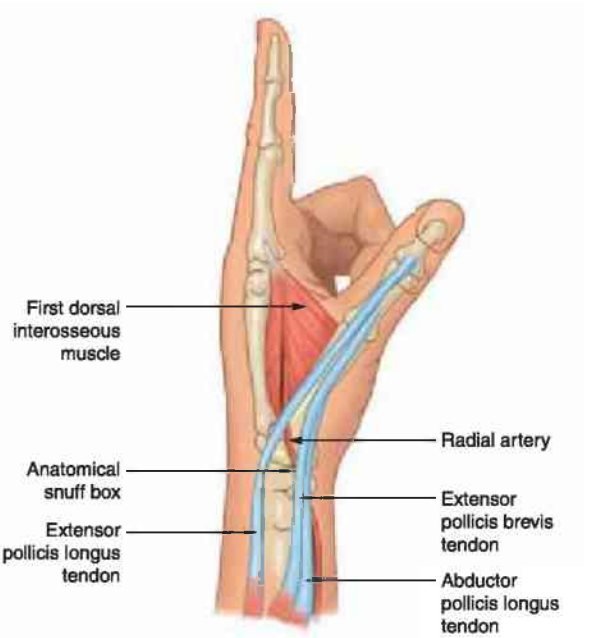


FIGURE 11.8 Diagrammatic representation of boundaries and contents of an anatomical snuff box.

Infections of the Hand

The hands are exposed and are constantly in use. They are therefore at high risk for injury and infections. Fortunately, the blood supply of the hand is excellent which acts as a protective factor in the prevention of infections and also helps in rapid healing of the infections.

Aetiology

The hand infections occur very frequently. The cause may or may not be traumatic. The causes are classified as follows:

1. Idiopathic
2. Secondary
   - Puncture
   - Burn
   - Open wound

In most of the cases, the causative organism is *Staphylococcus aureus.* In the rest of the patients, *Streptococcus* and *Enterobacter* are seen. Infection by multiple organisms is seen in 65% of cases. Opportunistic fungal infections are commonly seen in diabetics and in immunocompromised patients.

Classification

The hand infections can be classified according to anatomic areas as follows.

Nail Fold Infection

Nail fold infection is also called 'paronychia'. It is a common problem where the infection begins in the nail fold and gradually spreads to the subungual space. The patient presents with severe pain locally. On examination, swelling, redness and tenderness are the features on one or both sides of the nail fold. As the pus gets collected in the tight tissue compartment at the base of the nail, the pain becomes severe and throbbing (Fig. 11.9).

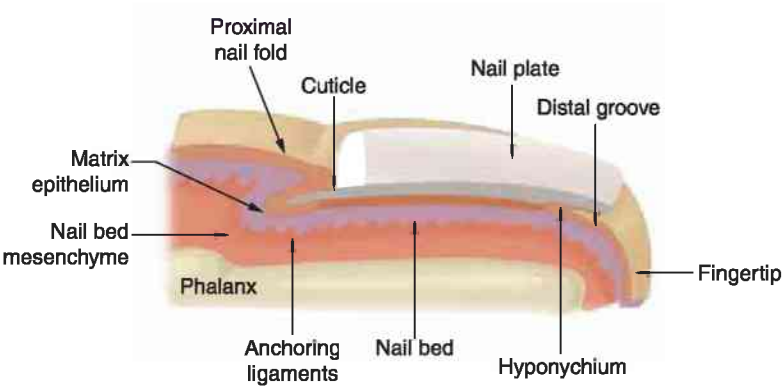


FIGURE 11.9 Diagrammatic representation of nail plate and subungual structure.

Treatment

The treatment consists of limb elevation and antibiotics initially. Later, as the pus develops and starts to point, incision and drainage are done with partial removal of the nail.

Apical Space Infection

Apical space infection occurs between the nail plate and the periosteum of the distal phalanx due to pinprick injury or a similar penetrating injury. The patient presents with severe pain under the free margin of the nail. On examination, there is pointed tenderness with pus formation.

Treatment

Treatment includes antibiotics in the early stages but once the pus develops, drainage with removal of a part of the nail overlying the pus pocket is done.

Digital Pulp Space Infection

Digital pulp space infection is also called a felon or whitlow. The infection occurs in the volar pulp of the fingertip due to a penetrating injury. The pulp of the finger has many thick septa making tight compartments. Even a small collection of pus causes severe pain. On examination, there is swelling over the palmar aspect of the fingertip, erythema, tenderness and fluctuation. In severe cases, necrosis of skin over the fingertip occurs due to compromised blood supply.

Treatment

If rest, elevation and antibiotics do not give relief, the pus is drained surgically.

Web Space Infections

Web space is triangular situated at the base of adjacent fingers. It contains loose tissue and can develop infection due to a penetrating injury. The second and third web spaces are usually affected. The patient presents with pain, swelling and fluctuation in the web space. The swelling or fullness is more pronounced on the dorsal aspect. The diagnostic feature is the fixed separation of the adjacent fingers due to abscess.

Treatment

If elevation and antibiotics do not resolve the infection, incision and drainage are done.

Tendon Sheath Infection (Synonymous Suppurative Tenosynovitis)

The flexor tendons are covered with tendon sheaths. Although there is a great variation in the disposition of these tendon sheaths, generally the tendon sheaths of the index, middle and ring fingers extend from the finger top to the base of the digits. The tendon sheath of the thumb, known as the radial bursa, extends into the forearm. The flexor tendon sheath of the little finger opens into the ulnar bursa.

Although suppurative tenosynovitis is a rare infection, but if it occurs it should be treated as an emergency to save function of the finger. The

infection is caused by a punctured wound or may be idiopathic. The patient presents with a painful swollen finger. There is tenderness along the tendon sheath. The finger is held in slight flexion due to the collection of pus within the flexor tendon sheath. Any movement of the finger causes excruciating pain. If not treated immediately, the infection can spread to the forearm and destroy the tendon completely.

Treatment

Energetic treatment is necessary. The patient is admitted and put on intravenous antibiotics with limb elevation. If there is no response within 6 hours, the tendon sheath is opened at each end and irrigated.

Infection of the Palmar Spaces

There are two spaces in the hollow of the palm: the mid-palmar space and the thenar space (Fig. 11.10). These spaces lie deep to the flexor tendons and their synovial sheaths.

The mid-palmar space extends from the middle finger radially to the little finger along its ulnar border.

The thenar space is bound radially by the adductor pollicis muscle and its ulnar border is provided by the middle finger.

Clinical Features

The infection can spread through the bloodstream, from adjacent structures or by a penetrating injury.

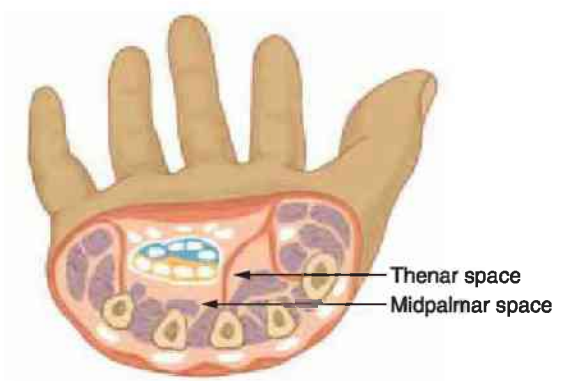


FIGURE 11.10 Diagrammatic representation of spaces of hand.

The patient presents with intense throbbing pain and swelling of the hand. The swelling is more pronounced on the dorsum of the hand (also called frog hand) than on the volar aspect. On examination, there is deep tenderness in the palm. The fingers are held in slight flexion, and any attempted extension causes severe pain.

Treatment

The patient should be admitted, arm elevated and intravenous antibiotics started. If there is no expected response with the conservative treatment, incision and drainage are undertaken.

Other Non-Infectious Conditions of the Wrist and Hand

- Ganglion
- De Quervain's disease
- Trigger finger
- Trigger thumb
- Carpal tunnel syndrome

Ganglion

Ganglion is a cystic swelling in the dorsum of the wrist. It can occur in the volar aspect also. In the dorsum, it consists of synovial protrusion from an intercarpal joint encased in a fibrous covering and containing a thick mucinous fluid. The patient presents with pain and discomfort on movement of the wrist. It is firm in consistency and is fixed to carpal bones. Multiple puncturing and injection of hyalose often cause subsidence of the swelling. If the ganglion is big and disfiguring, it is excised.

De Quervain's Disease

De Quervain's disease is a chronic constrictive tenosynovitis affecting the abductor pollicis longus and extensor pollicis brevis tendons of the thumb at the wrist. The patient complains of diffuse pain along the lateral aspect of the distal end of the radius. There is often a swelling along the tendons at that level, and it is tender on palpation. Active adduction against resistance and passive adduction of the thumb are painful (Finkelstein's test). In the early stages, splinting the wrist and thumb in full

extension and administration of anti-inflammatory drugs relieve the condition. In later stages, injection of hydrocortisone into the tendon sheath relieves the pain. In resistant cases, the sheath has to be surgically slit to release the constriction.

Trigger Finger

Trigger finger is a condition of obstruction in the free movement of the finger, with sudden release on extension. This is common in the middle and ring fingers. The condition is caused by a *constrictive tenosynovitis* of the flexor tendon sheaths occurring at the level of the metacarpophalangeal joints.

The patient complains of difficulty in flexing and extending the finger. After full flexion of the finger, active extension is not possible. On forcible passive extension, the finger extends suddenly with a palpable and audible snap. Later, a nodule in the substance of the tendon develops. As a result, there is more obstruction and the trigger effect is obvious.

This condition responds to local hydrocortisone injection into the tendon sheath and near the nodule in the tendon. When the constriction is very tight, surgical division of the tendon sheath is done.

Trigger Thumb

Trigger thumb is due to a *congenital constriction* of the tendon sheath of the flexor pollicis longus at the level of the metacarpophalangeal joint. The condition may be bilateral. The child keeps the interphalangeal joint of the thumb in flexion. Forcible extension suddenly releases the thumb into full extension with a click. A small nodule can be felt in the tendon at the level of the metacarpophalangeal joint. Treatment is by surgical division of the constricted tendon sheath.

Carpal Tunnel Syndrome

Carpal tunnel syndrome is a syndrome of compression neuropathy of the median nerve at the wrist. It is caused by elevated pressure within the carpal tunnel. The carpal tunnel is a fibro-osseous tunnel formed by the palmar hollow of the articulated carpal bones and roofed by the transverse carpal ligament (flexor retinaculum). The tunnel contains all the flexor tendons and the median nerve passing from the wrist into the palm. Any space-occupying mass or swelling of the structures in the tunnel causes pressure on the median nerve.

Pathophysiology

The increased pressure within the carpal tunnel produces ischaemia of the median nerve, leading to impaired nerve conduction. Initially, no morphological changes are seen in the median nerve and the neurologic features are reversible.

Prolonged and frequent episodes of elevated pressure lead to segmental demyelination, axonal injury and irreversible nerve dysfunction. Thickening of the synovial lining of the tendons occurs.

Aetiology

The aetiology of the syndrome may be as follows:

- Rheumatoid inflammation of the flexor tendon sheaths
- Compound palmar ganglion due to tuberculous tenosynovitis of the radial bursa
- Anterior dislocation of the lunate bone into the tunnel
- Malunited Colles' fracture stretching the transverse carpal ligament
- Myxoedema
- Pregnancy causing oedema of the tissues
- Amyloidosis
- Diabetes mellitus
- Corticosteroid use

Clinical Features

The patient complains of

- pain,
- paraesthesia over the palmar aspect of the hand,
- numbness,
- weakness and wasting of the thenar muscles.

The complaints of numbness and tingling in the thumb, index and middle fingers worsen at night. In the later stages, there may be wasting and weakness of the thenar muscles with sensory deficiency in the area.

Clinical Tests

- *Phalen's test* (wrist flexion test): Holding the wrist in acute flexion for some time worsens the symptoms.
- *Tinel's test:* It is the tingling sensation when the examiner gently taps the median nerve at the wrist.
- *Durkan's test:* Direct pressure over the carpal tunnel reproduces the symptoms.

Differential Diagnosis

This condition could be mistaken for a brachial neuritis due to cervical intervertebral disc prolapse at the C5–C6 level. Nerve conduction tests on the median nerve help to localise the lesion in the tunnel.

Treatment

Nerve conduction studies of the median nerve are useful. Initially non-operative methods, such as NSAIDS and steroid injections, are advised. If there is a specific cause for the condition, it should be treated and the nerve becomes decompressed. In severe cases, surgical division of the flexor retinaculum and transverse carpal ligament relieves the condition.

Dupuytren's Contracture

Dupuytren's contracture is a contracture of the palmar fascia of the ring and little fingers. The pathology was first described by Dupuytren. It is more common in western countries. Its exact aetiology is unclear although it is more commonly found in people with diabetes, liver disease and epilepsy.

Pathology

The changes are confined to the palmar fascia and the tissues superficial to it. There is perivascular fibroblastic proliferation which later on leads to fibrosis and contracture of the affected slips. In late stages, the skin gets adherent.

Clinical Features

It is common in males over 40 years. The patient presents with a flexion deformity at the metacarpophalangeal joint. In the earliest stage, there is a hard nodule in the deep palmar fascia at the level of the metacarpophalangeal joint. In later stages, a fibrotic band can be palpated as a cord-like thickening over the affected finger and the deformity becomes fixed.

Treatment

In early stages, subcutaneous fasciotomy may relieve the condition. In well-established cases, surgical excision of the thickened fascia is done.

KEY POINTS

- Brachial neuralgia is accompanied by neck stiffness and muscle spasm. It is common in middle-aged and elderly.
- Cervical disc prolapse is seen in young and middleaged patients. The most common level to be involved is the C5–C6 disc.
- Cervical spondylosis is characterised by arthrosis of the posterior intervertebral joints in the cervical part of vertebral column.
- Cervical rib is usually unilateral and is common on the right side. Lower cord of brachial plexus and subclavian artery are vulnerable to compression.
- Painful arc syndrome is seen in in supraspinatus tendinitis and is characterized by painful abduction between 60 and 120°.
- There are three stages in rotator cuff rupture (Neer): Stage I—oedema and haemorrhage, Stage II—fibrosis and tendinitis and Stage III—bone spur and tendon rupture.
- Periarthritis shoulder is a condition characterised by pain and progressive limitation of some movements of the shoulder joint. This condition is seen in elderly and can be idiopathic or secondary.
- The normal carrying angle is about 10° in males and 15° in females.
- Cubitus valgus causes tardy ulnar nerve palsy.
- Gunstock deformity is seen in cubitus varus.
- Trigger finger is caused by constrictive tenosynovitis of flexor tendons at metacarpophalangeal joints.
- Carpal tunnel syndrome presents with median nerve compression. Phalen's test is used for diagnosis.

MULTIPLE CHOICE QUESTIONS

1. The commonest cause of brachialgia is
   a. Spondylitis
   b. Cervical rib
   c. Avulsion injury of cervical vertebra
   d. None
2. In cervical rib, the following are seen except
   a. Cervical rib is palpable
   b. Ischaemic pain of muscles
   c. Atrophy of muscles
   d. Radial pulse is not palpable
3. Painful arc syndrome is due to
   a. Chronic supraspinatus tendinitis
   b. Subacromial bursitis
   c. Fracture greater tubercle
   d. All of the above
4. True about ganglion
   a. Common on volar aspect
   b. Seen adjacent to tendon sheath
   c. Communicates with joint cavity and tendon sheath
   d. Unilocular
5. In De Quervain's disease, the extensor pollicis brevis tendon passes to
   a. Thumb
   b. Index finger
   c. Middle finger
   d. Little finger
6. In trigger finger, the level of tendon sheath constriction is found at the level of
   a. Middle finger
   b. Proximal interphalangeal joint
   c. Proximal phalanx
   d. Metacarpophalangeal joint

CHAPTER 12

Regional Conditions of the Spine and Lower Limb

SPINE

Anatomy

A typical vertebra of the human has the following anatomical parts (Fig. 12.1):

- Vertebral body
- Neural arch, which is divided by articular processes into lamina (posterior) and pedicle (anterior)
- Articular processes, two in numbers, form facet joints with adjacent vertebrae
- Transverse processes, two in numbers
- Spinous process

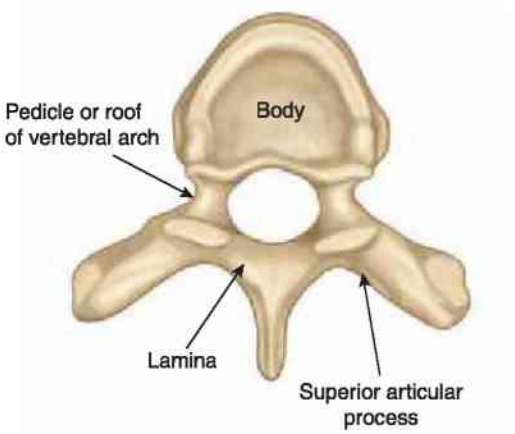


FIGURE 12.1 Diagrammatic representation of a typical thoracic vertebra.

The ligaments associated with a typical vertebra include the following:

- Supraspinous ligament
- Interspinous ligament
- Intertransverse ligament
- Capsular ligaments
- Ligamentum flavum
- Posterior longitudinal ligament

} Posterior ligament complex

- Anterior longitudinal ligament

Regarding the anatomy of spine, the following points are important to remember:

1. Human vertebral column (Fig. 12.2) has 33 vertebrae arranged in the following groups:
   - Seven cervical
   - Twelve thoracic
   - Five lumbar
   - Five sacral
   - Four coccygeal
2. Out of 33 vertebrae, 24 vertebrae are mobile.
3. Joints between adjacent vertebrae are synovial joints (zygapophyseal joints).
4. The length of the vertebral column is between 62 and 72 cm.
5. Intervertebral disc is present between two vertebral bodies. It is absent between C1 and C2.
6. Intervertebral discs are the largest avascular structures of the body.
7. Spinal cord terminates as conus medullaris at the L2 level in adults and L3 level in neonates.

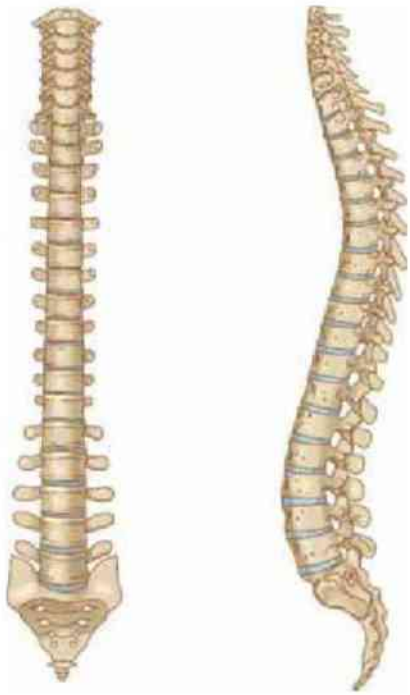


FIGURE 12.2 Diagrammatic representation of a normal spine and its curvatures.

8. Conus medullaris extends down as filum terminale upto the first coccyx segment.

The spine has certain normal curvatures at various levels (Fig. 12.2). At birth, there is a thoracic kyphosis and a sacral hollow. As the child crawls and looks up, a cervical lordosis develops. When the child assumes an erect posture, a lumbar lordosis also develops.

SPINAL DEFORMITIES

Deformities of the spine alter the shape of the whole back and occur in the sagittal plane as kyphosis and lordosis and in the coronal plane as scoliosis.

- *Kyphosis:* Kyphosis is a posterior curvature of the spine. In the thoracic level, it exaggerates the normal curve. In the lumbar and cervical levels, it obliterates the normal lordosis.
- *Lordosis:* Lordosis is an anterior curvature of the spine. It occurs commonly in the lumbar spine, most often as compensation for a kyphosis above or a flexion deformity at the hip joint.
- *Scoliosis:* In this deformity, there is a lateral curvature of the spine.
- *Combination:* Very often, there is a combination of deformities such as kyphoscoliosis.

Kyphosis

Kyphosis is of two types:

1. A smooth rounded kyphosis where several adjacent vertebrae are involved
2. Acute angular kyphosis where one or two vertebrae are involved

A smooth kyphosis can be due to adolescent kyphosis (Scheuermann's disease) or due to senile osteoporosis in the elderly. Angular kyphosis is often due to tuberculosis of spine or traumatic fusion.

Scheuermann's Disease (Adolescent Kyphosis)

Scheuermann's disease occurs in adolescents, due to extensive osteochondritis of the epiphyseal plates of the vertebral bodies. The patient complains of diffuse pain in the dorsal spine. Clinically, it is characterised by a smooth kyphosis at the dorsal spine.

Radiographs show narrowing of multiple intervertebral spaces with irregular densities and fragmentation in the epiphyses of the vertebrae. There are translucent defects in the subchondral bone known as Schmorl's nodes. Treatment is mainly symptomatic. Postural exercises and bracing will be necessary. Severe deformities with symptoms will require surgical correction.

Senile Kyphosis

Senile kyphosis occurs in old age. This is due to senile osteoporosis of the vertebrae and also due to degeneration of intervertebral discs. There are multiple anterior wedgings of the vertebrae resulting in a kyphotic deformity. This is a common cause of backache in older people.

Scoliosis

Scoliosis is defined as a lateral curvature of the spine. There is also a rotational component in

the deformity. A number of different causes may produce scoliosis which show similar clinical findings, but the difference lies essentially in the progress and prognosis of the scoliosis.

Classification

1. Postural scoliosis
2. Structural scoliosis
   a. Idiopathic
      - Infantile
      - Juvenile
      - Adolescent
   b. Neuropathic
      - Poliomyelitis
      - Cerebral palsy
      - Neurofibromatosis
   c. Myopathic
      - Muscular dystrophy
      - Arthrogryposis
   d. Osteopathic
      - Congenital hemivertebrae
      - Fused vertebrae

Postural Scoliosis

Postural scoliosis is common in the younger age group. It does not show structural changes in the spine. It corrects completely on lying down and shows no rotatory deformity on forward flexion. It does not progress and clears with spinal exercises. Another type of postural scoliosis is the one occurring as a compensatory deformity to gross shortening of one leg.

Idiopathic Scoliosis

Idiopathic scoliosis is the most common form and is classified into *infantile*, *juvenile* and *adolescent* types according to the age of the patient. In the vast majority of cases of scoliosis, the aetiology is not known.

Curves in Scoliosis

In scoliosis, there is a main curve called the primary curve. The scoliosis is named according to the level and side to which the main convexity of the curve is directed. For example, the term 'left dorsal scoliosis' denotes that the convexity of the main curve is towards the left side and is at the dorsal level.

The primary curve has compensatory curves above and below called the secondary curves. By the upper compensatory curve, nature attempts to keep the shoulders level so that the eyes are in a horizontal level. Similarly, by the lower secondary curve, nature helps to maintain the pelvis level so that the parallelism of the legs is maintained for normal locomotion.

The common patterns of scoliosis are thoracic scoliosis, thoracolumbar and cervicothoracic scoliosis depending upon the site of the primary curve.

Biomechanics

If any curved rod is bent in the lateral plane to form a second curve, it must twist in the long axis. In the case of the vertebral column, the lateral curvature causes rotation of the vertebral bodies. During such a rotation, the bodies move towards the convex side and the spinous processes move towards the concave side (Roaf).

Clinical Features

The patient, usually an adolescent, is brought with the complaint of a visible prominence of the posterior chest wall and scapula on one side. The location and extent of the primary curve are noted. The patient is made to bend forwards; in structural scoliosis, the lateral curve will persist whereas in purely postural curve it will disappear.

In thoracic curves, there is a dorsal angulation of the ribs producing the *rib hump*. In the lumbar region, there is bulging of the lumbar muscles and prominence of the pelvis on the convex side. A neurological examination is done for evidence of cord compression. General examination is done to rule out other causes such as neurofibromatosis.

Tests are done to decide whether the deformity is mobile or has become rigid. Make the patient lie in the lateral position on the concave side. The curvature is diminished in mobile cases.

Investigations

Radiography of the whole spine is done to assess the degree of the curve (Fig. 12.3).

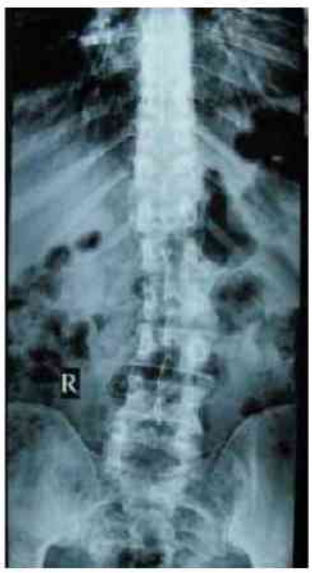


FIGURE 12.3 X-ray of dorsolumbar spine anteroposterior view showing scoliosis. Note the lateral convexity towards left.

The degree of the curve is measured by Cobb's angle. The curves are called mild, moderate or severe. Draw a line in the radiograph extending along the upper border of the uppermost vertebra. Another line is drawn extending along the lower border of the lowermost vertebra. Perpendicular lines are drawn from the above two lines. The angle subtended by the perpendiculars is Cobb's angle (Fig. 12.4). Skeletal maturity as shown by the fusion of the iliac apophysis (Risser sign) is an indicator of the end of the growth of the spine. Pulmonary function tests are done if major surgical treatment is planned. This helps in assessing the progression of infantile scoliosis.

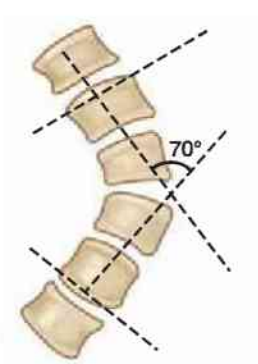


FIGURE 12.4 Diagrammatic representation illustrating Cobb's angle.

Treatment

In ***mild*** cases, the patient is kept under periodic observation to detect any rapid progression of the deformity. The patient is asked to sleep on the side of concavity. Postural correction is obtained by giving a heel-and-sole raise on the convex side of the deformity. Spinal and breathing exercises are prescribed. In ***moderate*** cases, in addition to the above, active correction by the Milwaukee spinal brace or modified plastic braces is prescribed.

Severe cases and those which show rapid progression of the deformity will require surgical correction of the deformity. Surgery consists of correction of the primary curve and spinal fusion. Correction is done by means of spinal instrumentation by the Harrington rod, Luque rod, Cotrel–Dubousset implants. Fusion is carried out using cancellous bone grafts. Sriram (Madras) and Dave (Delhi) have done extensive work in the surgical management of scoliosis in India.

Paralytic Scoliosis

Paralytic scoliosis is caused by paralysis, of some groups of spinal and abdominal wall muscles mainly due to poliomyelitis. It also occurs in cerebral palsy and muscular dystrophy. This scoliosis progresses rapidly and can result in gross deformities depending on the extent of the muscle paralysis. Severe cases will need surgical correction and fusion.

Congenital Scoliosis

Congenital scoliosis is due to congenital vertebral anomalies. It is usually associated with neural tube defects such as diastematomyelia and congenital anomalies in other systems. The vertebral anomalies may be a hemivertebra (Fig. 12.5) or fused vertebra. The deformity becomes obvious in early childhood and tends to progress rapidly. These require aggressive surgical treatment.

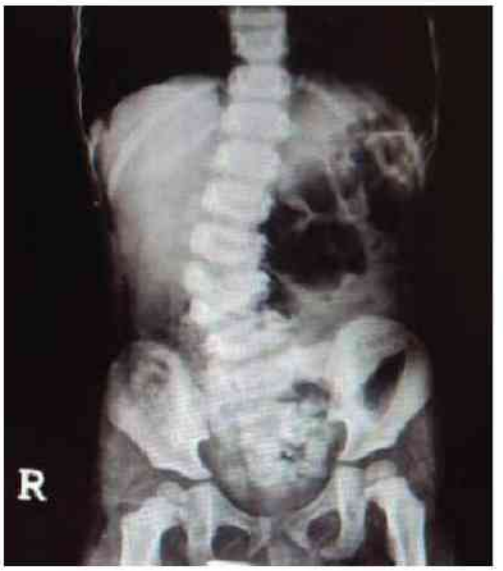


FIGURE 12.5 X-ray of dorsolumbar spine anteroposterior view showing congenital hemivertebrae.

SPONDYLOLISTHESIS

Spondylolisthesis is defined as the slipping forward of one vertebra over the next lower vertebra. It commonly occurs at the L4–L5 and L5–S1 junctions.

Classification

Spondylolisthesis has been classified into the following types according to the aetiology: congenital dysplastic, isthmic, degenerative, traumatic and pathological.

Aetiopathology

The isthmic spondylolytic type is the most common and is described below. It is of three types: lytic, elongated and traumatic.

The aetiological factor is a weakness at the pars interarticularis of the laminae of the fifth lumbar vertebra. There is a fibrous defect in the lamina between the superior and inferior articular processes, the exact cause of which is not known. Normally, there is only one centre of ossification for each half of the vertebral arch. It is believed that in this condition, there are two centres of ossification for each half of the vertebral arch. Failure of bony fusion between these two parts results in this fibrous defect. A minor trauma could then cause a separation of these two parts; the vertebral body, with the pedicle and the superior articular processes and the transverse processes slips forward, carrying the vertebra and the column above and leaving behind the inferior articular processes and the spinous process of the fifth lumbar vertebrae.

Clinical Features

The patient is usually a young or middle-aged adult complaining of low backache with diffuse pain down the back of both thighs. The pain may be of typical sciatic distribution. On examination, there is an exaggeration of the lumbar lordosis with a step-like depression above the top of the sacrum. The distance between the costal margins and iliac crest is diminished causing transverse furrows in the loin surrounding the body. There is no gross limitation of the movements of the spine. There may be neurological signs of stretching of L5 or S1 nerve roots unilaterally or bilaterally.

Radiography

Radiographs show the slipping forward of the vertebra in the lateral view. The slip may be of various grades: (1) Grade I up to 25% slip, (2) Grade II up to 50% slip and (3) Grade III up to 75% slip. Oblique views are taken to show the defect in the pars interarticularis known as 'Dog or Terrier sign' (Fig. 12.6).

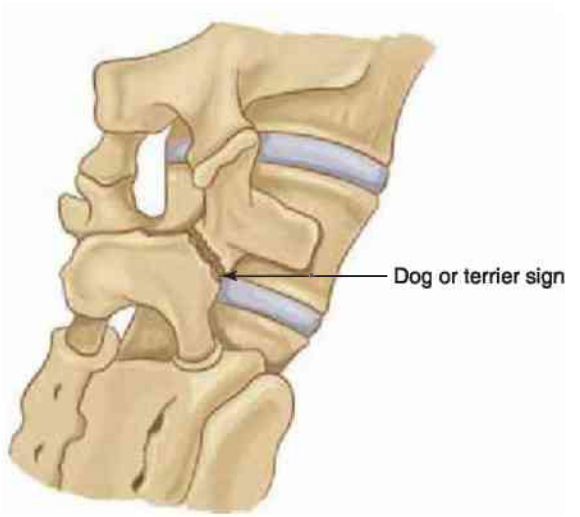


FIGURE 12.6 Diagrammatic representation of Dog sign in spondylolisthesis L4–L5. Note the decapitation of the head.

Treatment

When symptoms are mild, they may be relieved by spinal extension exercises to strengthen the sacrospinalis muscles. A lumbosacral brace is needed in some cases.

When the spondylolisthesis produces severe pain and neurological signs are not relieved by conservative treatment, surgical fusion of the lumbosacral junction is indicated. Recently, better methods of surgical stabilisation using instrumentation have enabled earlier post-operative mobilisation of the patient.

BACKACHE

Anatomical Factors

The lumbosacral region is the junction between the fixed pelvis and the very mobile lumbar spine and is therefore subject to considerable mechanical strain. It is also the site of many congenital abnormalities which weaken the site. The assumption of erect posture by man during evolution has thrown an undue strain at the lumbosacral junction. Hence, low backache is one of the most common presenting symptoms, in clinical practice. The backache may be localised in the spine or may be associated with sharp or diffuse radiation down the back of the leg, often loosely termed 'Sciatica'.

Aetiology

The causes of backache according to the site of pathology can be classified as follows:

1. *Intrinsic causes:* Causes in the various parts of the spine
   a. Conditions of the muscles, fasciae and ligaments of the low back
      - Traumatic: Acute sprain, chronic pain due to adhesions after an acute strain
      - Inflammatory: Rheumatic or rheumatoid type
   b. Lesions of the vertebral column
      - Intervertebral disc prolapse
      - Vertebral bodies: Tuberculosis of the spine, trauma—fracture, tumours—secondary deposit, multiple myeloma, general osteoporosis—menopausal, senile
      - Ankylosing spondylitis
      - Sacroiliac joint conditions: Trauma, puerperal arthritis mainly tuberculosis
      - Anatomical defects in the vertebral column: Spondylolisthesis, sacralisation, spina bifida
   c. Intraspinal conditions (e.g. cauda equine tumours)
2. *Extrinsic causes:* Causes outside the spine
   - General debility: Postural defects, obesity, chronic occupational strains
   - Shortening of the leg or deformity of the hip or the knee
   - Abdominal cause: Aortic aneurysm, duodenal ulcer
   - Genitourinary causes such as renal calculus and urinary tract infection

LUMBOSACRAL STRAIN

Lumbosacral strain is a clinical entity, which is very commonly met with in general practice. Most of the cases are due to lumbosacral strain which is due to

- extrinsic causes such as obesity and postural defects,
- anatomical defects at the lumbosacral junction such as sacralisation (Bartoletti syndrome), spina bifida and horizontal sacrum, which throw an abnormal ***mechanical strain*** at the lumbosacral junction.

Clinical Features

Low backache is an uncommon symptom in childhood but is most common after the third or fourth decade. It occurs in either sex; it is more frequent in the well-to-do and sedentary classes of people who are more prone to obesity and whose back and abdominal muscles lack tone. Pregnancy often precipitates or aggravates low backache. Very high-heeled shoes, a sagging bed or an improperly made chair, and occupations involving prolonged bending strain to the back, contribute to the higher incidence of backache.

Postural errors, either from habit or in the course of an occupation, account for quite a large proportion of cases of backache. Obesity and protuberant abdomen cause lumbar lordosis which

throws an extra strain on the ligaments of the lumbar spine.

Clinically, the patient presents with low back pain across the lumbosacral area. There is localised tenderness at the lumbosacral junction. Movements of the spine are relatively free. There are no neurological signs in the legs.

Treatment

It consists of improving the general health, reduction of weight and improving muscle tone by exercises to spinal muscles. Certain Yogasana exercises such as 'Dhanur Asana', which are spinal extension exercises, are extremely useful in strengthening the spinal muscles. Analgesics and short-wave diathermy relieve the symptoms in many cases.

FIBROSITIS BACK (Lumbago)

There is a small yet definite group of cases, where the cause of the backache is fibrositis in the lumbar fascia. Careful palpation reveals the presence of small tender nodules in the deep fascia over the sacrospinalis muscles called myofascial nodules. Pain originating in these becomes quite diffuse later on. Most of these nodules are of non-specific nature, the so-called rheumatic type. Such painful nodules respond well to a course of ultrasound therapy. The pain responds to infiltration with hydrocortisone solution with novocaine.

SACRALISATION OF THE FIFTH LUMBAR VERTEBRA

Sacralisation is the condition where the fifth lumbar vertebra is found fused with the sacrum unilaterally or bilaterally, partially or completely. This is the most common congenital abnormality (Fig. 12.7).

Although these abnormalities may be present from birth, they may produce symptoms only in the third or fourth decade. The presence of sacralisation alters the mechanics of the region and puts an extra strain on the rest of the lumbar spine. The clinical symptoms are referable to a lumbosacral strain and instability.

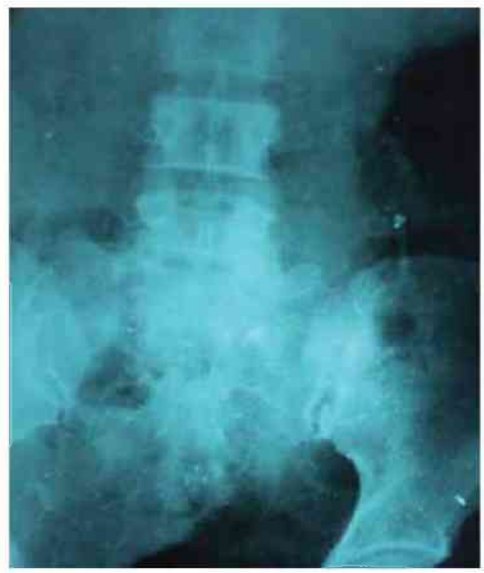


FIGURE 12.7 X-ray of the lumbosacral spine anteroposterior view showing sacralisation of L5 vertebrae.

Treatment

Many of these patients will get good relief from pain, if their spinal muscular tone is improved by spinal extension exercises which will brace up their back muscles. If exercises are contraindicated for medical seasons, a lumbosacral support will relieve the pain.

INTERVERTEBRAL DISC PROLAPSE

Intervertebral disc prolapse is the condition wherein the intervertebral disc prolapses posterolaterally or posteriorly into the vertebral canal. It is otherwise known as herniated nucleus pulposus as there is rupture of the annulus fibrosis and herniation of the nucleus pulposus. This is the most common cause of backache with sciatica. The most common level is the L4–L5 or the L5–S1 level.

Anatomical Factors

An intervertebral disc consists of a central nucleus pulposus surrounded by the annular fibrosis and covered by an end plate above and below. The prolapse can present in the following stages:

- Bulge
- Protrusion

- Herniation
- Sequestration

Clinical Features

The patient is usually a young man complaining of severe backache and sciatica which comes on after some exertion such as lifting a weight. The pain is increased on coughing or sneezing and sometimes it may be excruciating. On examination, the patient has a slight lumbar scoliosis called sciatic scoliosis and the normal lumbar lordosis is abolished. In an acute case, the spine is held rigid with very acute pain and muscle spasm. There is limitation of the movements of the spine with muscle spasm. The straight leg raising is limited on the side with sciatica. There is tenderness at the lumbosacral junction.

Neurological Examination

The prolapse is commonly posterolateral. Prolapse of the disc between L4 and L5 will compress the L5 nerve root, producing

- diminished sensation on the dorsum of the foot and anterolateral aspect of the leg,
- weakness of the extensor hallucis longus,
- ankle jerk will be normal.

The prolapse of the L5–S1 disc causes compression of the S1 nerve root. Hence, the signs will be

- diminished sensation over the S1 dermatome on the lateral aspect of the foot and leg (Fig. 12.8),
- absence of ankle jerk,
- occasionally weakness of plantar flexion of big toe and foot.

Radiological Features

Anteroposterior and lateral radiographs of the lumbosacral junction must be taken. After the first attack, there are no radiological signs. But in patients who have had recurrent attacks of sciatica for a long time, the corresponding disc space is narrowed and the adjacent vertebral surfaces are sclerosed (Fig. 12.9). The radiograph is also useful in eliminating other conditions such as tuberculosis of the spine or an old fracture.

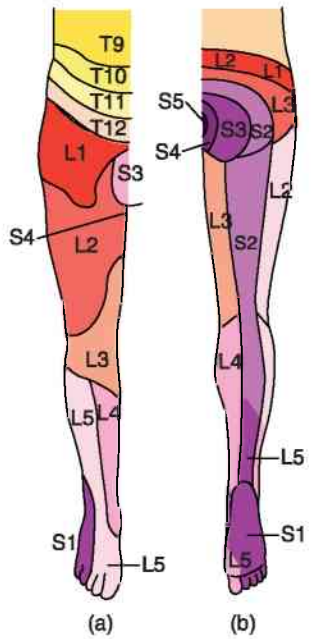


FIGURE 12.8 Diagrammatic representation showing dermatomes in the lower limb. (a) Anterior view and (b) posterior view.

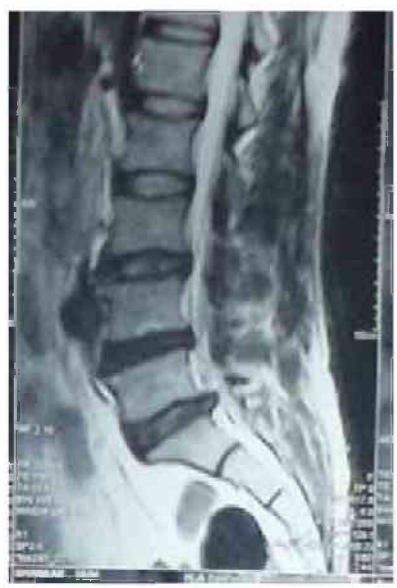


FIGURE 12.9 MRI lumbar region showing L5–S1 IVD prolapse.

MRI is most specific and non-invasive to diagnose intervertebral disc prolapse.

Treatment

The initial treatment in the stage of acute pain consists of absolute bed rest and analgesics. Pelvic traction is applied. In most cases, the pain is completely relieved within a time of 7–10 days.

In cases where conservative treatment fails, surgical excision of the prolapsed disc is done. More recently, the following procedures are being performed:

- *Fenestration diskectomy:* Prolapsed disc is removed through a space created between the lamina of two adjacent vertebrae.
- *Laminectomy and diskectomy:* The laminae of both sides and spinous process are removed to obtain a wide exposure. A wide laminectomy can disturb the stability of spine.
- *Hemilaminectomy and diskectomy:* The lamina of one side is removed and disectomy is performed. Stability of spine is not much disturbed.
- *Microdiskectomy:* Diskectomy is done through a small incision.
- Percutaneous endoscopic diskectomy is a minimally invasive recent advance.
- *Spinal fusion:* In some cases, in addition to removal of the prolapsed disc, spinal fusion may be necessary.

LUMBAR CANAL STENOSIS

Lumbar canal stenosis is a condition where there is narrowing of the lower lumbar spinal canal.

Classification

Spinal stenosis may be congenital or acquired. Congenital stenosis occurs in achondroplasia. The acquired form of the stenosis is commonly due to lumbar spondylosis. The other causes are spondylolisthesis, post-traumatic, Paget's disease, etc.

Clinical Features

Symptoms develop insidiously and the patient presents with low back pain with diffuse radiation into both the gluteal regions and the back of thighs. The pain is *neurogenic claudication*. Pain is relieved by sitting or lying down and increased by standing and walking. Root symptoms may occasionally occur simulating a disc lesion.

Radiological Features

Anteroposterior and lateral views of the lumbar spine are taken. The mid-sagittal diameter and the interpedicular distance will be diminished. MRI is done to confirm the diagnosis.

Treatment

Conservative treatment includes rest, analgesics and short-wave diathermy. Surgical treatment is by decompression. This is done by laminectomy and foraminotomy of the affected segments. Some cases with instability will also need spinal stabilisation.

LOWER LIMB

This section will deal with the conditions affecting the joints of the lower limb—hip, knee, ankle and foot and their disorders.

As movement is the main function of a joint, the grouping of conditions, based on the alterations found in the movements of the hip joint, is a logical way to arrive at an anatomical diagnosis. The case may fall into one of the following broad clinical types of hip conditions.

Clinical Conditions Affecting the Hip Joint

1. *Type I:* Limitation of all movements of the hip joint with pain
   a. Anatomical diagnosis: Arthritis
   b. Pathological diagnosis could be as follows:
      - Acute (e.g. septic arthritis)
      - Chronic (e.g. tuberculous arthritis, rheumatoid arthritis)
2. *Type II:* Loss of all movements of the hip joint
   a. Anatomical diagnosis: Ankylosis
   b. Pathological diagnosis
      - Sound ankylosis (bony, following septic arthritis)
      - Unsound ankylosis (fibrous, following tuberculous or rheumatoid arthritis)

3. *Type III:* Limitation of only some movements of the hip joint such as abduction and internal rotation
   a. Anatomical diagnosis: Coxa vara
   b. Pathological diagnosis
      - Congenital coxa vara
      - Perthes disease, slipped upper femoral epiphysis, malunited trochanteric fracture

   Rarely, an extra-articular focus of infection in the femoral trochanter, iliac adenitis with psoas spasm and myositis ossificans in a surrounding muscle could also cause limitation of some movements.
4. *Type IV:* Presence of abnormal movement
   a. Anatomical diagnosis: Telescoping hip joint
   b. Pathological diagnosis
      - Congenital dislocation of the hip
      - Old Tom Smith's arthritis of infancy
      - Non-union of the fracture neck of femur
      - Unreduced posterior dislocation hip

Deformities of Hip

Coxa Vara

Coxa Vara is a deformity wherein the angle between the neck and shaft of femur is reduced to less than 120°. Normally, the angle is between 130 and 140° in adults and 140 and 150° in children. Reduction of this neck shaft angle below 110° is called coxa vara. There are two types of coxa vara:

1. Congenital coxa vara
2. Acquired coxa vara

Congenital Coxa Vara (Infantile Coxa vara)

Congenital coxa vara is a rare condition, in which there is a development defect in the medial part of the neck of the femur. It is often bilateral and the condition is noticed by the limp, when the child begins to walk. On examination, there is a marked raising of the greater trochanter and there is limitation of abduction and rotation movements in the hip. The involved leg is usually short.

Radiograph shows the neck shaft angle to be reduced. A defect is seen in the inferior part of the neck with a separate triangular piece of bone. This is called Fairbank sign. Hilgenreiner's epiphyseal angle (angle between Hilgenreiner's line and line through proximal femoral physis) is used to diagnose congenital coxa vara from X-ray.

Treatment

Mild cases can be observed. If the deformity is severe and progressive, surgical correction by subtrochanteric valgus osteotomy is indicated.

Acquired Coxa Vara

Acquired coxa vara is classified according to the site of the disturbance causing the deformity:

1. Capital coxa vara
   - Perthes disease
   - Chondro-osteodystrophy
   - Cretinism
2. Epiphyseal coxa vara
   - Slipped upper femoral epiphysis
   - Multiple epiphyseal dysplasia
3. Cervical coxa vara
   - *Traumatic:* Malunited trochanteric fracture of femur
   - *Pathological:* Rickets, Paget's disease, fibrous dysplasia

Corrective valgus osteotomy is done if the condition is severe causing marked distress.

SLIPPED UPPER FEMORAL EPIPHYSIS (Adolescent Coxa Vara) (Epiphysiolysis)

Slipped upper femoral epiphysis is a condition where there is slipping of the epiphysis of the head of femur. It is more common in boys between 10 and 15 years of age and may be bilateral.

Aetiology

The exact aetiology is unclear. It commonly occurs in obese children with some hormonal imbalance and retarded sexual development. It occasionally occurs in thin tall children. There may be a superadded element of mild trauma which starts the slipping of the epiphysis. The head slips from the neck *inferiorly* and *posteriorly.* The slippage takes place through the hypertrophic layer of the growth plate.

Clinical Features

The onset of symptoms may be of two types: (1) a more common *chronic* slip with insidious onset of pain and limping and (2) a rarer *acute* slip with sudden and severe pain usually following a fall.

In the common type, the child is brought for the complaint of pain in the hip and slight limping. The pain is worse on walking and is unrelieved by rest. On examination, the leg lies in an externally rotated and adducted position and appears slightly shorter. There are no signs of local inflammation in the hip. The greater trochanter is at a higher level as shown by the alteration in Bryant's triangle. There is marked limitation of abduction and internal rotation movements. Flexion is free but on full flexion, the thigh tends to rotate externally. The leg is shorter by about 1–2 cm.

Radiological Features

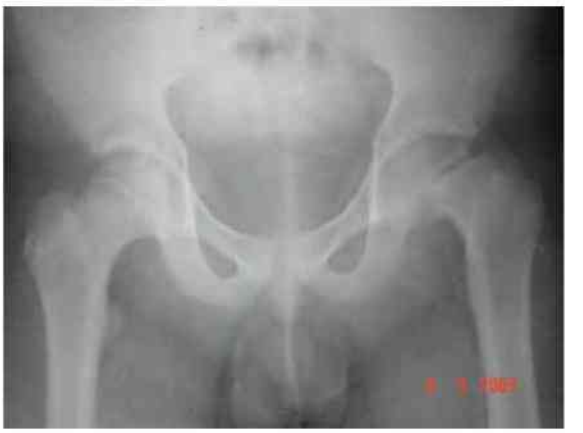
Anteroposterior radiograph shows the separation and slipping down of the epiphysis (Fig. 12.10a). A lateral view is necessary to detect minimal slip and also to show the degrees of slip—mild, moderate and severe. CT scan is useful to diagnose early slip.

- *Trethowan's sign:* Normally, a line drawn along the upper border of the femoral neck cuts a small segment of the head. In the case of the downward shift of the head, the head is found below the line. This is called Trethowan's sign.

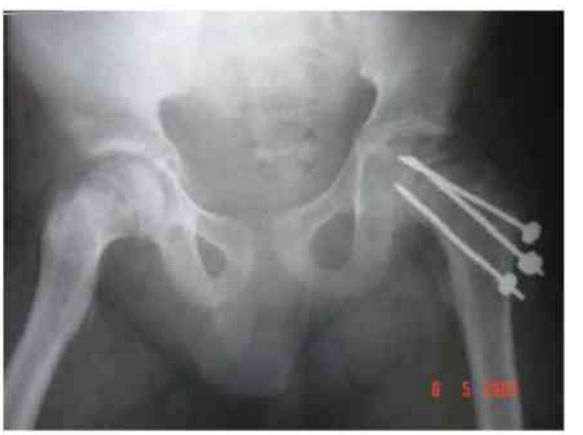
Treatment

In mild cases with less than one-third slip, surgical fixation with multiple pins is done. This prevents further slip and encourages healing. In moderate slip between one-third and two-third of the width, the slip can be reduced by applying continuous traction to the leg and gradually abducting the leg. When the slip is reduced, the best treatment is to internally fix the head of the femur by multiple pins (Fig. 12.10b).

In cases of severe slip, the coxa vara is corrected surgically by cervical or intertrochanteric osteotomy. If untreated, the epiphysis unites with the neck in a displaced position and a permanent coxa vara results. In such cases, the coxa vara must be surgically corrected by a wedge osteotomy at the trochanteric level.



(a)



(b)

FIGURE 12.10 (a) X-ray of pelvis with both hips anteroposterior view showing slipped upper femoral epiphysis (left). (b) X-ray of pelvis with both hips showing slipped upper femoral epiphysis treated with Austin Moore pins.

Complications

Early complications of slipped epiphysis are as follows:

- Acute cartilage necrosis or chondrolysis
- Avascular necrosis of the capital epiphysis

Late complication is secondary osteoarthrosis of the hip.

Idiopathic Chondrolysis of the Hip

Chondrolysis (acute cartilaginous necrosis) is an affection of the hip characterised pathologically

by features of rapid loss of cartilage and clinically by pain and restriction of movements.

Aetiology

This condition is more common in dark skinned people and has a predominance for females. It is usually secondary to a slipped epiphysis. Prolonged immobilisation in extreme positions as in the treatment of developmental dysplasia of the hip occasionally leads to chondrolysis. Causative factors hypothesised include inadequate nutrition due to either synovial fluid deficit or its improper constitution. Autoimmune factors have also been blamed.

Pathology

The cartilage is grossly eroded in both the femoral head and the acetabulum exposing the subchondral bone. Areas devoid of articular cartilage are covered by fibrous tissue or fibrocartilage.

Clinical Findings

Adolescent children are brought with the complaints of aching pain which is continuous and increased on weight bearing. Movements are rapidly reduced in all directions ending in a fibrous ankylosis.

Radiological Findings

In the initial stages, there is osteoporosis with blurring of articular margins with irregularities in both acetabulum and femoral head. As the disease progresses, the joint space becomes uniformally reduced and weight-bearing areas of the head of femur and acetabulum show sclerosis.

Differential Diagnosis

The condition has to be differentiated from tuberculosis and rheumatoid arthritis by synovial biopsy.

Treatment

A conservative approach is usually followed which includes bed rest, analgesics, traction and physiotherapy. Once the pain is reduced, non-weight–bearing crutch walking is allowed.

KNEE

The following conditions will be described:

- Genu valgum
- Genu varum
- Tibia varum
- Genu recurvatum
- Quadriceps fibrosis
- Recurrent dislocation of the patella
- Semimembranous bursitis

Genu Valgum (Knock Knee)

Genu valgum is a lateral angulation deformity of the knee with an outward deviation of the legs at the knee. The condition is otherwise called knock knees. It is an acquired deformity which can be unilateral or bilateral.

Genu valgum can be either physiological or pathological. Physiologic genu valgum is the most common form. It is a normal finding in children between 2 and 6 years of age.

Aetiology

Pathological causes include the following:

- Trauma
- Rickets
- Renal osteodystrophy

Injury or infection destroys the lateral half of the lower femoral epiphyseal plate, resulting in arrested growth of the lateral condyle of the femur. The continued growth of the medial condyle results in unilateral genu valgum. Bilateral genu valgum can result from conditions causing bone softening as in rickets, osteomalacia and rheumatoid arthritis. Epiphyseal dysplasias can also be the cause.

Clinical Features

A careful history and physical examination should be done. Femorotibial angle measurement and intermalleolar distance between the medial malleoli should be measured. On an average, when this distance is more than 8 cm, it points to genu valgum. The degree of genu valgum is measured by the *intermalleolar distance* at the ankle when the child stands or lies down with

the knees touching each other (Fig. 6.1). Genu valgum results in secondary flat foot. Excessive genu valgum often causes outward dislocation of the patella.

Treatment

In mild cases of genu valgum in young children, where the intermalleolar distance is less than 5 cm, wearing of boots with the inner side of heel raised in 3/8 inch and elongated forwards (Robert Jones heel) corrects the deformity.

When the deformity is moderate (the intermalleolar distance being 5–10 cm), correction is achieved by an orthosis consisting of boots with a long outside bar upto the level of the greater trochanter and knee straps. Any active rickets should be controlled by vitamin D therapy (Fig. 12.11).

In cases with severe deformity, a supracondylar osteotomy of the femur (Mac Ewen's osteotomy) is done to correct the deformity. Other procedures include the following:

- Varus osteotomy
- Medial hemiepiphysiodesis of the lateral femur or proximal tibia

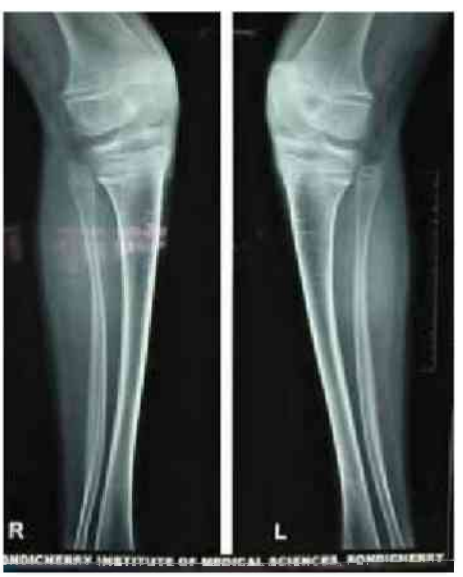


FIGURE 12.11 X-ray of bilateral knee with leg anteroposterior view showing genu valgum.

Genu Varum (Bow Leg)

Genu varum is a medial angulation deformity of the knee with lateral bowing of the legs. The condition is otherwise called bow leg. It is a common childhood deformity.

Physiological genu varum is seen in children between birth and 2 years of age. It resolves spontaneously with normal growth and development. The degree of genu varum in the standing and spine positions should be noted. The distance between two medial femoral condyles is noted, roughly when this distance is more than 6 cm in a standing child with heels touching, points to a genu varum deformity.

Aetiology

Genu varum can be either a physiological or a pathological condition. It is usually bilateral. It is commonly seen in conditions such as rickets (Fig. 6.2a) and Paget's disease. In osteoarthritis knee of a severe degree, genu varum develops.

The degree of the deformity is measured by the distance between the two medial femoral condyles (intercondylar distance) when the patient stands or lies down with the ankles together.

Treatment

Mild degrees of deformity can be corrected by wearing an orthotic appliance consisting of boots with a long inner rod extending to the groin and leather straps across the tibia and the knee, to gradually correct the bowing. Severe degrees of deformity will need corrective osteotomy operation.

Its surgical procedure includes the following:

- Valgus osteotomy
- Lateral hemiepiphysiodesis of the distal femur or proximal tibia

Tibia Varum (Blount's Disease)

Blount's disease is a condition presenting as lateral bowing of the tibia in children. It is due to a local dysplasia of the posteromedial part of the proximal epiphysis of the tibia. It is usually

bilateral and often progressive. The causes include the following:

- Physeal injury
- Metabolic disorders—rickets
- Renal osteodystrophy
- Osteogenesis imperfecta

To differentiate tibia varum from genu varum, the following test is done. With the child lying on the back, the knee is flexed fully. In tibia varum, the deformity becomes more obvious. In genu varum, the deformity disappears.

Radiology shows beaking of the metaphysis medially and defect in medial side of epiphysis. Severe cases need corrective tibial osteotomy.

Genu Recurvatum

Genu recurvatum is a hyperextension deformity of the knee with backward bowing of the knee. It can be congenital or acquired. In the congenital form, it is due to arthrogrypotic contracture of the quadriceps with or without congenital dislocation of the knee.

Aetiology

The common cause of acquired genu recurvatum in our country is weakness of the quadriceps due to poliomyelitis. This can also be secondary to walking on a fixed equinus contracture. The other causes are trauma to growth plate, malunited fracture and neuropathic joint. It also follows quadriceps fibrosis following repeated injection into the thigh in children.

Treatment

It depends on the causative factor. Recurvatum due to quadriceps contracture is corrected by quadriceps plasty operation. In cases of poliomyelitis, an above-knee caliper prevents worsening of the deformity. Severe cases need soft tissue or bony correction.

Quadriceps Fibrosis

Quadriceps fibrosis is a contracture of one or more heads of quadriceps muscle. It occurs usually in children. The most common cause of quadriceps fibrosis in India is a post-injection fibrosis following repeated injections or infusions into the thigh, in infants suffering from gastrointestinal disorders and respiratory infections. In adults, the fibrosis results from infection extending into the muscle from osteomyelitis in the shaft of femur.

There is also a congenital type found in association with congenital genu recurvatum as part of a generalised arthrogryposis multiplex.

Pathology

The common site of fibrosis is the vastus intermedius and the next common site is the vastus lateralis. A senior author's work (1963) established the relationship between the fibrosis and the following anatomical facts:

- The vastus intermedius is contained in a closed fibro-osseous compartment formed by a fascial sheath and the femur.
- The diminished vascularity of this head compared to the other heads of the quadriceps.
- Injections cause increased tension within the compartment resulting in an ischaemic type of muscular fibrosis.

Clinical Features

The child is brought with the complaint of inability to flex the knee joint and squat on the heel for toilet purposes. The girth of the thigh is less compared to the normal side. The affected head of the muscle may be palpable as a fibrotic band. The knee flexion may be 90° in mild cases and only 5 or 10° in severe cases. Some cases may present with almost complete flexion which is possible due to lateral dislocation of the patella.

Treatment

In milder cases, the knee flexion could be increased by passive stretching by the physiotherapist. In severe cases, surgery has to be done to lengthen the muscle by quadriceps plasty.

Recurrent Dislocation of the Patella

Recurrent dislocation of the patella is characterised by repeated lateral subluxation or dislocation

of the patella occurring on flexion of the knee. In ***habitual*** dislocation, the patella dislocates every time the knee is flexed and reduces on extension.

Anatomical Factors

In the normal knee, the obliquity of the line of the quadriceps muscle and its insertion in the tibia results in the valgus angle opening on the lateral side. The supplement of this angle is the quadriceps angle (Q angle) which is normally 15–20° (Fig. 12.12). Any condition tending to exaggerate the Q angle will predispose to lateral subluxation of the patella.

Aetiology

Conditions causing recurrent dislocation of patella can be grouped as follows: (1) weakening and laxity of the quadriceps expansion and the capsule on the medial side of the knee, (2) tightening and contracture of the structures on the lateral side of the knee, (3) abnormal insertion of the ligamentum patellae into a more lateral site on the tibia, (4) defective development of the lateral femoral condyle, (5) gross genu valgum and (6) patella alta and baja (infera).

In habitual dislocation of patella, there is a congenital abnormal insertion of a part of the iliotibial band in the superolateral pole of the patella. Some cases may have acquired injection fibrosis of the quadriceps.

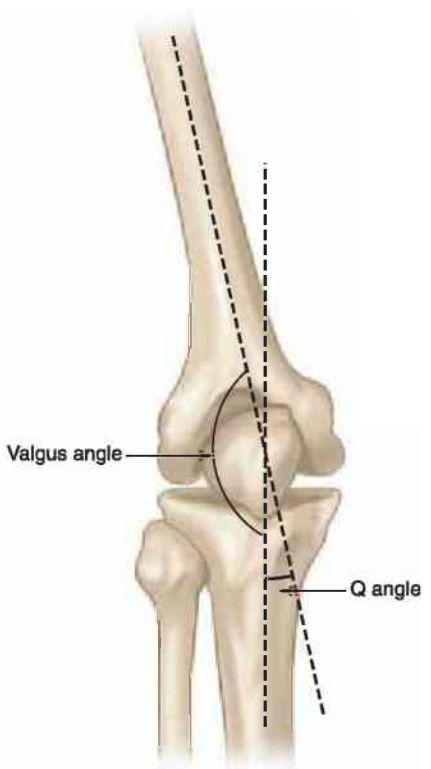


FIGURE 12.12 Diagrammatic representation of knee joint illustrating how to draw a 'Q' angle.

Clinical Features

The condition presents in childhood with the complaint of knee giving way and repeated falls and the slipping out of the patella.

On examination, gradual flexion of the knee will produce dislocation of the patella laterally. The patella may be smaller and at a higher level (alta). A tight band may be palpable at the lateral pole of the patella.

Holding the patella in the midline prevents the knee from flexion beyond 30°. Releasing of the patella allows full flexion. There is a sudden expression of fear of dislocation on attempted flexion of the knee. This is called apprehension test.

Radiograph

Radiograph shows the dislocation in the tangential view with the knee in 45° flexion (Merchant's view). The sulcus angle and Blumensaat's line are used for evaluation.

Treatment

The treatment of this condition is essentially surgical. Majority of cases are corrected by soft tissue surgery. The principles of the procedure are to release the lateral tight structures and plication of the lax medial capsule. Marked degrees of the malalignment of the extensor mechanism will need release of the insertion of the ligamentum patellae and its reattachment to a more medial site.

Bursae Around the Knee

Among the numerous bursae which normally exist around the knee, the ones causing clinical symptoms

are prepatellar bursa, infrapatellar bursa and semimembranous bursa. Inflammation of these bursae may be due to repeated mechanical irritation. They are also liable to all conditions which affect tendon sheaths and synovial membranes.

Prepatellar Bursitis

Prepatellar bursitis is called housemaid's knee due to repeated friction on the acutely flexed knee during work. It presents as a soft fluctuant swelling in front of the lower half of the patella. Milder cases will be relieved by aspiration and hydrocortisone infiltration. Larger swellings will need excision.

Semimembranous Bursitis

Semimembranous bursitis is a chronic inflammatory swelling of the semimembranous bursa, which lies in the medial part of the popliteal fossa. The patient presents with discomfort and bulge in the back of the knee. There is a cystic swelling in the medial part of the popliteal fossa, which is fluctuant and transilluminant. The swelling lies between the semimembranous tendon and the medial head of gastrocnemius. Occasionally, the swelling communicates with the synovial cavity of the knee joint. Smaller swellings could be left alone. Larger swellings will need excision.

Loose Bodies in the Knee

The knee is the most common joint for loose body formation. The other joints are the elbow and ankle. They may form due to trauma or disease. A traumatic loose body is single. Pathological loose bodies are multiple.

The common causes of loose bodies are as follows:

- Osteochondritis dissecans in adolescents
- Osteochondral fractures in adults
- Broken off osteophytes in osteoarthrosis in the elderly
- Multiple loose bodies in synovial chondromatosis
- Loose bodies in Charcot's joints

Loose bodies may be symptomless or may present with pain and locking episodes in the joint with a sensation of something moving inside the joint. It tends to slip away during palpation (joint mouse). Radiology will show the size and number of loose bodies. Symptomatic loose bodies may require removal surgically or arthroscopically.

Anterior Knee Pain

Anterior knee pain is a clinical condition in adolescents and young adults characterised by diffuse pain in the anterior aspect of the knee extending down the front of the leg. The most common cause of this pain is chondromalacia patellae.

The other causes are as follows:

- Patello femoral subluxation
- Prepatellar bursitis
- Congenital discoid meniscus
- Torn meniscus
- Plica syndrome

Occasionally, osteoarthrosis may present as diffuse pain in the anterior part of the whale leg.

Chondromalacia Patellae

Chondromalacia patellae is a condition where there are early softening and fibrillation changes in the articular surface of the patella. The exact aetiology of this condition is not known. The basic aetiopathology is probably due to mechanical overstrain of the patellofemoral joint in young athletes. The patellar articular surface goes through the stages of *softening*, *fissuring* and *fragmentation*. The patient is a teenage adolescent presenting with ***anterior knee pain***. The characteristic signs are crepitus on moving the patella over the femoral articular surface and pain on direct pressure over the patella.

Radiologically, there is a malalignment of the patella seen in the skyline view. Arthroscopy will show fibrillation in the articular surface. Early cases are treated with rest, analgesics and shortwave diathermy. Severe cases may need surgical or arthroscopic shaving of the patellar articular surface (chondroplasty).

FOOT

The Indian foot is exposed to injury as well as infection, since most people in rural India walk barefoot.

However, the encasing of the human foot in stiff shoes, worn throughout the day from childhood, has been a mixed blessing to the Western population. This is shown by the high incidence of foot problems seen among patients attending orthopaedic clinics in the west. The feet have lost their suppleness and the mobility of the toes; the crowding of the toes and the foot inside ill-fitting shoes contribute to the high incidence of hallux valgus, hallux rigidus, clawing of the toes, anterior metatarsalgia, warts, bursitis, ingrowing toe nails, etc.

Painful Heel

Pain in the heel is one of the common presenting complaints in young adults or middle-aged persons. It can also occur in adolescents. The causes of such pain are as follows:

1. Pain in the plantar aspect of the heel
   - Plantar fasciitis
   - Calcaneal spur
2. Pain in the posterior aspect of the heel
   - In adolescents: Osteochondritis of the apophysis of the calcaneum (Sever's disease)
   - Tendo achilles bursitis in adults (retrocalcaneal bursitis)
   - Calcification at the tendo achilles insertion
3. Pain in the lateral or medial aspect of the heel
   - Old fracture calcaneum
   - Subtalar arthritis
   - Tumours
   - Infections of the calcaneum

Plantar Fasciitis

Plantar fasciitis is an aseptic inflammation of the plantar fascia. It causes pain in the foot anterior to the attachment of the plantar fascia.

The patient is usually a middle-aged adult complaining of pain in one or both the heels. Typically, this pain is worse in the early morning and the patient is unable to bear weight on the foot on getting up from bed. The pain is more while walking and is relieved by rest. There may be other associated rheumatoid manifestations such as tennis elbow, joint pains or tenosynovitis. There is tenderness on pressure over the medial tuberosity of the calcaneum.

Treatment

It is conservative. Hot fomentation steroid injection can be done.

- The patient may be prescribed for soft microcellular rubber shoes.
- Operative subperiosteal elevation of the long plantar ligament may be needed rarely.

Calcaneal Spur

The calcaneal spur is not a growth but a reaction to the local inflammation of the plantar fascia and ligaments with deposition of calcium at the site of the ligamentous attachments. The condition is caused by a spike of bone at the anterior edge of the calcaneum. The symptoms are not due to the spike per se but due to the causative conditions.

Radiograph of the heel in the lateral view may show varying degrees of the calcaneal spur (Fig. 12.13).

Clinical Features

Pain is felt in the ball of the heel, which is more during long periods of standing. The severity of the pain is not proportional to the size of the spur. Even big spurs may be asymptomatic and minimal spurs may be associated with severe pain. Tenderness can be elicited on the plantar aspect of the heel and swelling is seen occasionally.

Treatment

- Rest and anti-inflammatory drugs are given.
- Shoes with soft rubber pads are prescribed (MCR shoes).
- Radiant heat therapy is also very effective in many cases.

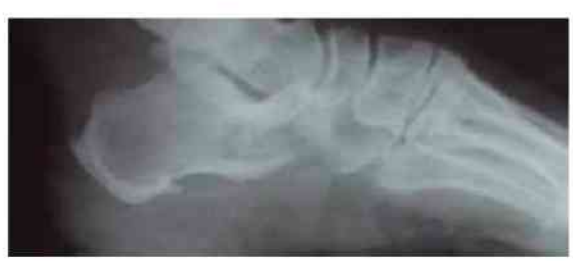


FIGURE 12.13 X-ray of foot lateral view showing calcaneal spur.

- If it does not respond to these, a local infiltration of hydrocortisone given under strict aseptic precautions will relieve the symptoms.

Surgical Treatment

In resistant cases, surgical removal of the spur is done.

Posterior Heel Pain

Pain in the posterior aspect of the heel is a common clinical condition occurring in the middle-aged persons due to retrocalcaneal bursitis and achilles tendinitis and calcification. Occasionally, it can occur in the adolescents when it is due to osteochondritis of the calcaneal apophysis (Sever's disease).

Clinically, there will be tenderness on the posterior aspect of the calcaneum. The pain is worse on forcible dorsiflexion of the foot which stretches the tendo achilles. Radiograph may show calcification at the level of insertion of tendo achilles or at the posterosuperior margin of the calcaneum. In Sever's disease, the calcaneal apophysis will show increased density and fragmentation.

These conditions respond to anti-inflammatory drugs and raising the heel of footwear by 1.5 cm. Some cases will need local hydrocortisone infiltration or ultrasound therapy.

Flat Foot (Pes Planus)

Flat foot is a condition where there is flattening of the arches of the foot. When the forefoot is pronated the foot goes into valgus, and there is flattening of the medial arch.

The following are the types of flat foot.

Congenital Flat Foot (Vertical Talus)

The feet appear flat in all newborn babies as the postural tone of the intrinsic muscles has not yet developed. But in some cases, the foot is not only flat but the undersurface is convex (Rocker bottom foot) and the whole foot is in valgus or everted position. This is due to a congenital abnormality of the talus, in which the bone lies in a vertical position, the head of the talus pointing plantarwards rather than forwards (Fig. 12.14).

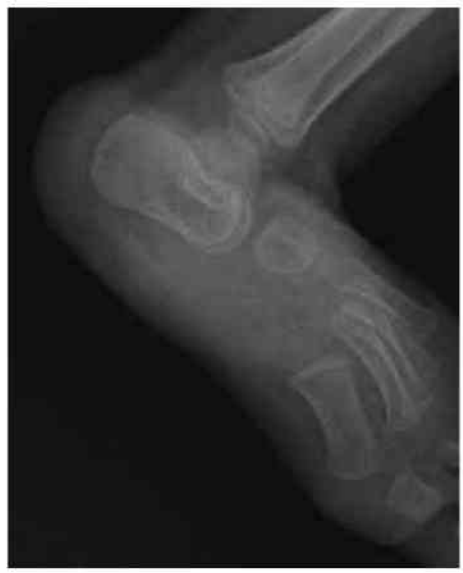


FIGURE 12.14 X-ray of ankle with foot lateral view showing flat foot due to vertical talus.

Mild cases, where the talus is oblique, are treated by modified footwear. Severe cases of vertical talus are really dislocations of the talonavicular joint and will need corrective surgery.

Infantile Flat Foot

Infantile flat foot is the physiological flat foot of children, who have just started walking. As the postural tone of the foot muscles develops, the arch becomes obvious and the flat foot disappears. There is a possibility of the flat foot being secondary to mild genu valgum or a vertical talus. This does not need any special treatment, except assurance to the parents that the condition will correct by itself.

Secondary (Compensatory) Flat Foot

In children, genu valgum at the knee can cause a secondary flat foot. In mild cases of paralytic

equinus due to polio or cerebral palsy, it is compensated by dorsiflexion at the tarsal joint and flattening of the arch.

Acquired Flat Foot

The causes of flat foot in adults are muscle weakness due to general debility, rapid increase in body weight and post-traumatic flat foot following injury to the calcaneum, metatarsal, etc., due to wasting and weakness of intrinsic muscles.

Treatment

Exercises to the intrinsic foot muscles and the use of modified footwear relieve the symptoms in most cases. Secondary flat feet are dealt with by treating the primary cause. Gross deformities may need surgery.

Spasmodic Flat Foot

Spasmodic flat foot is due to spasm of the peroneal muscles due to unaccustomed strain in adolescents, subtalar arthritis or congenital calcaneonavicular anomalies.

Acute Foot Strain

Acute foot strain occurs in adolescents who have newly taken up jobs where they have to be standing for long hours and in medical students starting the clinical ward work.

The condition is due to the failure of the muscle tone, caused by fatigue of the intrinsic muscles of the foot. Excessive load is then thrown on the ligaments of the tarsal joints. The strain and stretching of these ligaments cause the acute pain and tenderness.

The patient complains of acute pain along the inner margins of both feet. On examination, there is loss of arch in both feet and tenderness along the inner borders of the feet as well as over the medial tuberosity of the calcaneum.

If symptoms are very acute, complete rest is necessary. When the acute symptoms subside, the patient is taught foot exercises for the intrinsic muscles of the feet.

Chronic Foot Strain

Chronic foot strain occurs in middle-aged women, who put on weight and have to be on their feet cooking for long hours. The arch of the foot sags and causes a strain on the ligaments.

In such cases, in addition to diet control for reduction of weight, footwear with arch supports is prescribed and the patients are taught exercises for the intrinsic muscles of the feet. Most women in India cannot use arch supports as they do not wear shoes.

Pain in the Forefoot

The common causes of pain in the forefoot are hallux valgus, anterior metatarsalgia and march fracture.

Hallux Valgus

Hallux valgus is a deformity of the big toe, where it is turned laterally at the metatarsophalangeal joint. There are two types of hallux valgus: the congenital type and the acquired type. In the congenital type, the hallux valgus is associated with a varus of the first metatarsal bone (metatarsus primi varus). The acquired form of hallux valgus is common in western countries, due to the wearing of tight-fitting and pointed shoes.

Clinical Features

A mild degree of hallux valgus does not cause any symptoms. In severe degrees, there is actually a lateral dislocation of the big toe, with the head of the first metatarsal bone projecting medially. This subcutaneous bursa, together with the callosity of the overlying skin, forms the 'bunion'. Periodic inflammation of the bursa causes pain in that region. The outward angulation of the big toe causes crowding of the other toes with over-riding of the second over the third toe and hammer toe deformity of the lateral toes. The gross deformity of the big toe throws an extra weight-bearing strain on the transverse anterior metatarsal arch, resulting in pain in the forefoot. Neglected cases of hallux valgus result in osteoarthrosis of the metatarsophalangeal joint and stiffness at the joint (hallux rigidus).

Treatment

Minimal deformities may be left alone. If the pain and disability are severe, the deformity is corrected by operation. This is done either by excision of the head of the first metatarsal bone (Mayds operation) or by excision of the base of the proximal phalanx (Keller's operation). More recently, osteotomy of the first metatarsal is done to realign the first metatarsal and the big toe.

Anterior Metatarsalgia

Anterior metatarsalgia is pain under the heads of the metatarsal bones often due to collapse of the anterior arch of the foot. It also occurs secondary to hallux valgus, cavus foot and claw toes. The static form of metatarsalgia is due to diminished tone of the intrinsic muscles and ligaments. Foot exercises and modified footwear relieve the pain.

KEY POINTS

- The deformities of spine can lead to formation of the following pathological curvatures: (1) Kyphosis is posterior curvature of spine leading to forward bending, (2) lordosis is anterior curvature of spine leading to backward bending and (3) scoliosis is lateral curvature of spine leading to lateral bending.
- Scheurmann's disease is extensive osteochondritis of the epiphyseal plates of the vertebral bodies. It is seen in adolescents.
- Scoliosis can be postural or structural.
- Idiopathic scoliosis is the most common form of scoliosis. It can be classified into infantile, juvenile and adolescent types.
- Spondylolisthesis is the slipping forward of one vertebra over the next lower vertebra.
- Lumbosacral strain is a clinical entity which can be due to extrinsic causes or anatomical defects.
- Intervertebral disc prolapse is the condition wherein the intervertebral disc prolapses posterolaterally or posteriorly into the vertebral canal. The most common level is the L4–L5 or L5–S1 level.
- MRI is most specific to diagnose disc prolapse.
- Congenital coxa vara is a developmental defect in the medial part of neck of femur.
- Hilgenreiner's epiphyseal angle is used to diagnose this condition from X-ray.
- Slipped upper femoral epiphyses commonly occur in obese children with some hormonal imbalance and retarded sexual development.
- Blount's disease is a condition presenting as lateral bowing of the tibia in children (tibia varum).
- Recurrent dislocation of patella is its repeated lateral subluxation or dislocation on flexing the knee.
- Merchant's view (tangential view with knee in 45° flexion) is used to demonstrate this condition.
- The calcaneal spur is a reaction to the local inflammation of the plantar fascia and ligaments with deposition of calcium at the site of the ligamentous attachments.

MULTIPLE CHOICE QUESTIONS

1. The most common cause of anterior knee pain is
   a. Prepatellar bursitis
   b. Congenital discoid meniscus
   c. Plica syndrome
   d. Chondromalacia patellae
2. Beheaded Scottish terrier sign is seen in
   a. Disc prolapse
   b. Sacralisation of L5
   c. Spondylosis
   d. Spondylolisthesis
3. The most common type of spondylolisthesis is
   a. Congenital dysplastic
   b. Isthmic spondylotic
   c. Degenerative
   d. Traumatic
4. Cobbs angle is measured in
   a. Kyphosis
   b. Scoliosis
   c. Lordosis
   d. Lateral flexion
5. Prolapse disc most commonly occurs at
   a. C5 and C6
   b. C6 and C7
   c. C1 and C2
   d. L4 and L5
6. The most common cause of scoliosis in children is
   a. Unequal limb length
   b. Post-poliomyelitis
   c. Hemivertebrae
   d. Marfan's syndrome

CHAPTER 13

Advances in Orthopaedic Surgery

Orthopaedic surgery has made rapid strides in the past few decades revolutionising the speciality. The emergence of newer subspecialities such as Orthopaedic Oncology and Paediatric Orthopaedics clearly reflects the diversity of clinical problems in orthopaedics and traumatology. Improvements in biomaterials, implant and prosthetic design, better understanding of biomechanics and improved imaging methods, and newer surgical techniques have contributed greatly to this progress.

There have been newer developments in almost all the fields of orthopaedics, from fracture fixation to spinal surgery to joint replacement surgeries. It is indeed an exciting phase for the orthopaedic surgeon. But unfortunately, with all the techniques and devices at his command, there has been an increased tendency to focus more on the technique and less on the patient. The budding orthopaedicians would do well not to get carried away with the technical advances and always keep the patient's requirements in mind while planning a treatment strategy.

ARTHROPLASTY

Arthroplasty is a surgical procedure aimed at repair and refashioning of the arthritic or dysfunctional joint so as to obtain a joint with a good range of pain-free movements.

The various types of arthroplasty include the following:

- Interposition arthroplasty
- Excision arthroplasty
- Resurfacing arthroplasty
- Replacement arthroplasty

Replacement arthroplasty is further divided into the following:

- Hemiarthroplasty
- Total joint replacement or total joint arthroplasty

INTERPOSITION ARTHROPLASTY

In interposition arthroplasty, tissues such as skin, muscle or tendon are interposed between the two inflammatory articular surfaces to keep them apart. This helps in decreasing the pain and increasing the range of movements to some extent.

EXCISION ARTHROPLASTY

Excision arthroplasty aims at removing the joint surface and the bone creating the problem. Later, scar tissue grows and occupies the joint cavity. A sufficient part of the bone is removed to create an adequate gap which permits a good range of movements of the joint (Fig. 13.1) (e.g. Girdlestone arthroplasty of the hip).

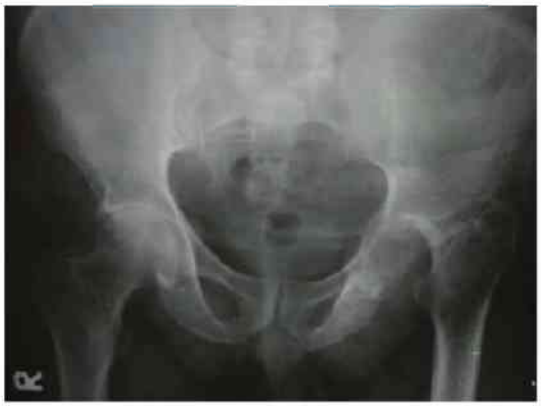


FIGURE 13.1 X-ray of the pelvis anteroposterior view showing girdle-stone excision arthroplasty of the hip.

RESURFACING ARTHROPLASTY

Resurfacing arthroplasty is a type of arthroplasty wherein the diseased articular surface alone is replaced with a prosthesis. When inflamed arthritic articular surfaces rub against each other, they produce pain. Thus, replacing the painful joint surface with an artificial implant is aimed at preventing the bones from rubbing against each other. Articular surface replacing surgery of hip is more commonly performed. The acetabular and femoral head articular surfaces are replaced by metal prosthesis. The early stage of avascular necrosis of the hip is a good indication for articular surface replacement (Fig. 13.2). The second common joint to be resurfaced is the shoulder joint.

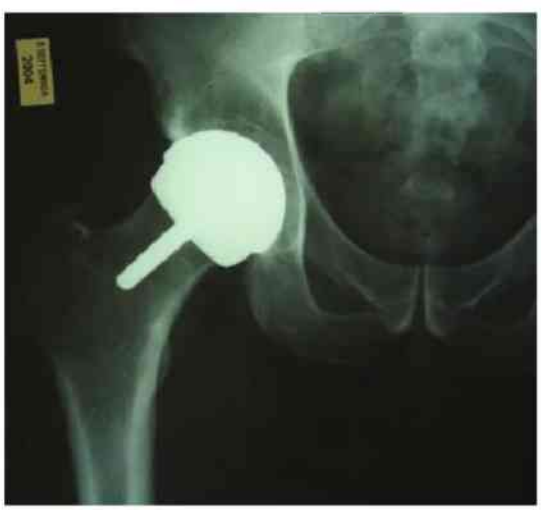


FIGURE 13.2 X-ray of the right hip anteroposterior view showing hip resurfacing surgery.

REPLACEMENT ARTHROPLASTY

Replacement arthroplasty is a surgery wherein one or both the joint surfaces are replaced with artificial prosthesis. This is further divided into

- hemiarthroplasty,
- total joint replacement arthroplasty.

Hemiarthroplasty

In hemiarthroplasty, only one end of the joint is replaced by an artificial prosthesis. This type of surgery is performed in large numbers in hip followed by shoulder and elbow. Hemiarthroplasty of the hip is done almost exclusively by replacing the femoral head and neck alone with a prosthesis (Figs 13.3 and 13.4).

The prosthesis may be either

- Thompson's prosthesis,
- Austin Moore's prosthesis,
- bipolar prosthesis.

Thompson's prosthesis is used along with bone cement, after removing the femoral head and neck. It can be used where the calcar femorale is absent. Austin Moore prosthesis is used without cement and the holes in the stem of the prosthesis allow the bone trabeculae to grow in between. It is used only when the calcar is present. Bipolar prosthesis allows movement within the prosthesis itself and also between the prosthesis and acetabulum. This can be used with or without cement. Modular bipolar prosthesis is that where the size of the 'head' of prosthesis and 'stem' of the prosthesis can be chosen from an array and assembled on the table of surgery.

Shoulder hemiarthroplasty can be done by using Neer's prosthesis.

Total Joint Replacement Surgery

Joint replacement surgery means that both the articular bone ends in a joint are replaced by prosthetic implants. It is also called total replacement

(a)

(b)

(c)

FIGURE 13.3 Prothesis. (a) Thompson's prosthesis, (b) Austin Moore prosthesis and (c) bipolar prosthesis.

arthroplasty. The goal of primary replacement arthroplasty is to re-establish the normal mechanical axis with a stable prosthesis, which is well fixed.

Total joint replacements of the hip and knee are among the most successful group of surgical procedures in the speciality; this is followed by shoulder joint. Other joints such as elbow, ankle and wrist can also be totally replaced. The present-day total hip or total knee replacements can restore near-normal function to patients with severe pain or markedly limited function in the respective joints.

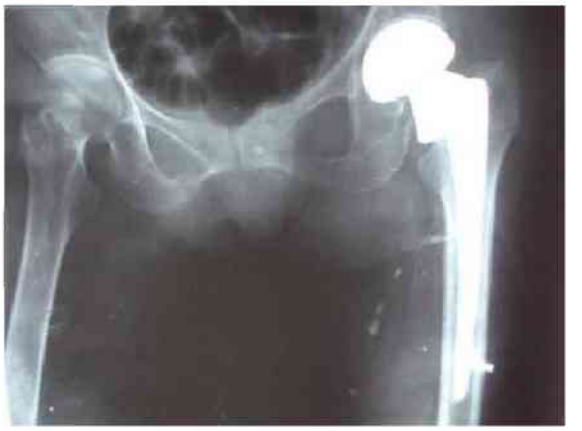


FIGURE 13.4 X-ray of the pelvis with both hips showing cemented bipolar hemiarthroplasty in the left hip.

Total Hip Replacement Surgery

The acetabulum and the head of the femur are replaced with artificial joint components (Fig. 7.7). The acetabular component has a cup made of ultra high molecular weight polyethylene (UHMWPE) and backed by a metal ring or cup. The femoral component is made of metal (stainless steel, titanium, cobalt-chromium alloy). Ceramic implants are relatively fragile and have less friction characteristics. These implants can be fixed to the patient either with acrylic cement, called 'cemented replacement arthroplasty', or with a cementless technique, termed 'uncemented replacement arthroplasty'.

Although joint replacements using bone cement have an established success rate over decades, the problem of aseptic loosening remains, requiring a revision arthroplasty. Uncemented total hip replacement is the 'State of the Art' and can be done even in young patients. Total elbow and total shoulder replacements are being increasingly done and their success rates have been good.

- *Cemented technique:* Bone cement is used to fix both the acetabular and femoral components. This is a better technique when used in osteoporotic bones.
- *Uncemented technique:* Here, the acetabular component is fixed into place with the help of screws. The femoral component is snugly fit into the femoral medullary canal. This

technique holds good in those patients with a good bone stock.

Indications for Total Hip Replacement Surgery

- Severe chronic arthritis following osteoarthritis, rheumatoid arthritis, Perthes disease, slipped capital femoral epiphysis, ankylosing spondylitis
- Avascular necrosis of the femoral head
- Malunited acetabular fractures

These are some standard indications for total hip replacement surgery.

In general, total hip arthroplasty is reserved for elderly patients and in needy young patients with destructive hip disorders and rarely in children with very severe disorders.

Complications

1. Immediate complication
   - *Sciatic nerve injury:* This is more common with a posterior approach to hip joint. Most cases recover spontaneously.
   - *Periprosthetic fracture:* This occurs as a fracture of the femur or acetabulum (perforation into the pelvic cavity), more commonly in severe osteoporotic individuals.
2. Delayed complications
   - *Dislocation of the prosthesis:* An incorrectly placed prosthesis leads on to dislocation, when the patient attempts to move the joint. This is overcome by correct and careful placement of the prosthesis during surgery.
   - *Infection:* This is the most dreaded complication that may require removal of the prosthesis.
3. Late complications
   - *Aseptic loosening of the prosthesis:* This can occur in the long term and may require revision of the surgery.
   - *Heterotopic bone formation:* This can occur around the hip and in severe cases, it is associated with pain and stiffness. Indomethacin can be given as a prophylaxis in high-risk individuals.

Total Knee Replacement Surgery

Total knee arthroplasty involves replacing the articular surfaces of the distal femur and proximal tibia that form the knee joint. Patella can also be resurfaced along with the procedure (Fig. 7.8).

The femoral and tibial components are made of metal alloy, and the spacer is made of UHMWPE. The patellar component is also made of UHMWPE and is called patellar button.

Indications

Major indications for knee surgery include the following:

- Incapacitating pain following osteoarthritis and rheumatoid arthritis
- Deformity with or without instability
- The tibial and patellar components are generally fixed with cement and the femoral component is snugly fit with the distal femur

Complications

1. *Deep vein thrombosis:* This can occur following prolonged immobilisation and so appropriate deep vein thrombosis prophylaxis must be given in the form of anticoagulants or compressive stocking.
2. *Infection:* This is a dreaded complication. Early stages of infection can be managed by debridement, wash and antibiotics. Late stages require removal of the prosthesis and arthrodesis of the joint.
   - Loosening of the implant can occur in the long term.

Total Shoulder Replacement Surgery

Shoulder is a ball and socket joint formed by a convex humeral head and concave glenoid of the scapula. Total shoulder arthroplasty is done by replacing the humeral head and glenoid.

Reverse total shoulder arthroplasty is another surgery wherein the concave glenoid is replaced by a convex prosthesis and convex humeral head is replaced by a concave prosthesis.

The indications include severe chronic arthritis, comminuted proximal humerus fractures and tumours of the proximal humerus.

Total Elbow Replacement Surgery

This surgery is indicated in severe painful arthritis following rheumatoid arthritis and in non-union of distal humerus fractures.

Majority of the patients get good pain relief and an adequate range of movements.

CUSTOM MEGA PROSTHESIS IN BONE TUMOURS

The conventional treatment of malignant bone tumour of extremity was amputation. The most recent advance in the surgical approach to skeletal defects after tumour resection involves the use of custom-made prosthetic joints for the replacement of defects near the hip, knee and shoulder. The development of metallurgy and bone cement and their successful use in joint replacement surgery gave way to the use of endoprosthesis in bridging defects in the long bones and joints. An individually designed, custom-made bone and joint replacement prosthesis is the optimum method of obtaining the best possible results for the patient. These custom-made prostheses are made of either titanium or stainless steel alloy.

Providing materials at just a fraction of the cost to the patient represent the future in the field of orthopaedic oncology in a poor country like India.

RECENT ADVANCE USING THE 'PRINCIPLE OF DISTRACTION OSTEOGENESIS'

Ilizarov Technique

A revolution in the treatment of infected non-unions, the Ilizarov fixator (Fig. 13.5) is a circular external fixator made up of rings which are connected to the bone by K-wires or pins. The rings are connected to each other by threaded rods which permit compression or distraction of fragments.

Ilizarov techniques are based on the principle of 'distraction osteogenesis' where slow distraction of bone induces regeneration of new bone. It is also called the 'tension-stress principle'. The principle involves the technique of 'corticotomy' where division of bone is done without disturbing the medullary blood supply. Subsequently, controlled distraction of the young callus or

FIGURE 13.5 Clinical photo of a child with the Ilizarov ring fixator for deformity correction.

'Callotasis' can be done after a few days at the rate of 1 mm per day. This distraction which induces new bone formation is called 'distraction osteogenesis'. It has a wide ranging use in the field of orthopaedics. It is the method of choice for infected non-unions. Limb length discrepancies can be corrected successfully by this technique. The other applications include the correction of complex bony deformities, arthrodesis of joints and bone transport to bridge defects after tumour resections.

Limb Reconstruction System

Limb reconstruction system uses the same principle of distraction osteogenesis. It is a unilateral external fixator with a distraction mechanism. The main advantage of this system over Ilizarov is the light-weight frame which is also sturdy and patient friendly. Unlike the ring-fixator assembly of Ilizarov which is multiaxial, it acts uniaxially.

ARTHROSCOPY

Arthroscopy is a procedure which uses an optical instrument called the arthroscope to visualise joints (Fig. 13.6). Professor Kenj Tokag of Tokyo University used a cystoscope for the first time to view the interior of knee joint. Almost any joint can be reached but the procedure is mostly employed in the knee, shoulder, wrist, ankle and hip. The arthroscope has dramatically changed

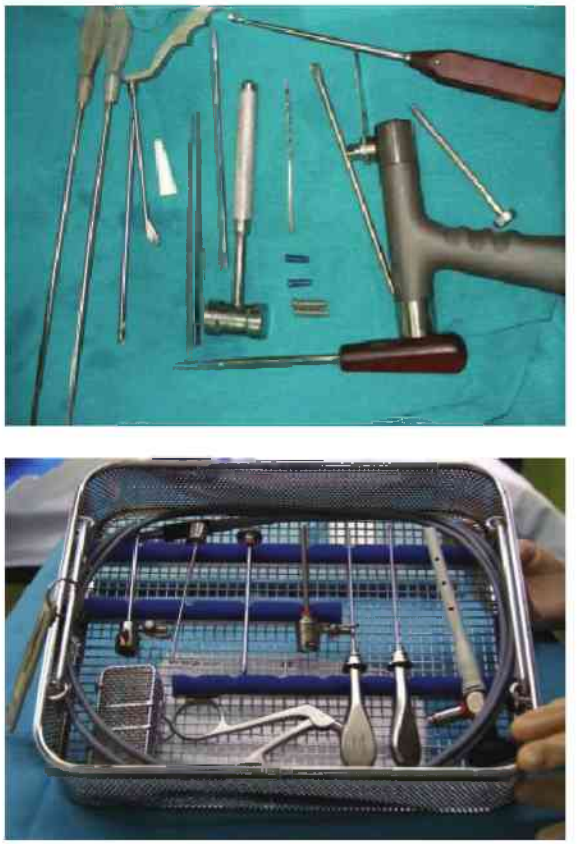


FIGURE 13.6 Instruments used in arthroscopic surgeries.

the orthopaedic surgeon's approach to a variety of joint ailments. Arthroscopy is not only diagnostic but can also have therapeutic applications. A high degree of clinical accuracy, combined with low morbidity, has encouraged the use of arthroscopy to assist in diagnosis, to determine prognosis and often to provide treatment. It is a relatively safe procedure with the commonest complications being haemarthrosis, thrombophlebitis, infection and joint stiffness.

Arthroscopy of Knee Joint

Knee is the commonest and most accessible joint subjected to arthroscopy and various arthroscopic surgeries. Arthroscopic procedures can be carried out under spinal or general anaesthesia with tourniquet control. Arthroscopy can delineate internal derangements of the knee. It also allows differentiation between inflammatory and non-inflammatory lesions, and destructive and non-destructive lesions. Meniscal tears can be diagnosed as well as repaired. It can also be used to repair cruciate ligament deficiency. Arthroscopy-assisted anterior cruciate ligament reconstruction permits earlier return to work or professional sports. It can also diagnose and remove loose bodies. Synovial thickening can be delineated and synovectomy performed.

Portals

Portals are of two types:

1. *Standard portals:* Most of the arthroscopic works are possible through these portals (Fig. 13.7).
   - Anterolateral portal, otherwise called arthroscope/viewing portal
   - Anteromedial portal, otherwise called instrument/operating portal
2. *Accessory portals:* These may be additionally created for better access and visualisation
   - Superolateral portal
   - Superomedial portal
   - Posteromedial portal
   - Posterolateral portal

Triangulation

Initially, it is difficult to bring the instruments introduced through one portal into the vision of the arthroscope introduced through the other

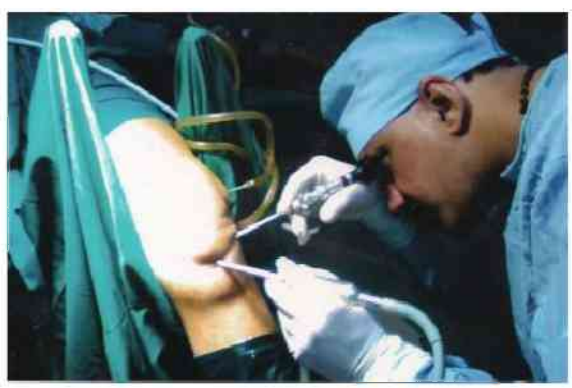


FIGURE 13.7 Photograph of a surgeon performing an arthroscopic surgery of the knee joint.

portal. This is overcome by a technique called 'triangulation' where the surgeon forms the base with instrument and the scope forming the sides of the triangle.

Procedures are carried out by synchronous movement between the three sides of the triangle:

Diagnostic Arthroscopy of Knee Joint

This includes visualisation of the interior of the knee joint as an investigatory procedure. The following structures are visualised in order:

- Suprapatellar pouch
- Patellofemoral articulation
- Medial gutter
- Medial tibiofemoral compartment
- Intercondylar notch
- Lateral tibiofemoral compartment
- Lateral gutter
- Posteromedial compartment

Diagnostic arthroscopy is most commonly combined with small procedures such as removal of loose bodies or an arthroscopic lavage of the joint.

Ligamentous (anterior cruciate ligament and posterior cruciate ligament) laxity or disruptions can be ascertained by using a probe.

Arthroscopic Surgeries of Knee Joint

The most commonly performed surgeries include the following:

- Repair of meniscal tears
- Removal of loose bodies
- Arthroscopic drilling for osteochondritis diseases
- Arthroscopic screw fixation for osteochondritis disease
- Cruciate ligament reconstruction including anterior and posterior cruciate ligaments
- Lateral retinacular release for chondromalacia patella
- Debridement and wash in pyogenic arthritis

Arthroscopy of Shoulder Joint

Arthroscopy of the shoulder joint is more difficult but is a common procedure. It is also unique in its complexity and hence the need for specialised and trained personnel. The rotator cuff injuries of shoulder are treated more by arthroscopic procedures than by open surgeries these days. General anaesthesia is preferred to regional anaesthesia, because the patient may experience distress with prolonged arthroscopic positioning in the case of regional nerve blocks.

Indications of Shoulder Arthroscopy

- Shoulder instability
- Rotator cuff tears
- Shoulder impingement
- Superior Labrum Anterior Posterior (SLAP) lesions
- Adhesive capsulitis

Portals

- Posterior portal forms the viewing portal
- Anterior inferior portal
- Anterior superior portal
- Anterolateral portal

Diagnostic Arthroscopy

Diagnostic arthroscopy includes visualisation of the following portions:

- Biceps—superior labrum complex
- Supraspinatus
- Head of humerus
- Labrum
- Glenohumeral ligament
- Glenoid
- Subscapularis

Commonly performed arthroscopic surgeries of the shoulder include the following:

- Removal of loose bodies
- Synovectomy
- Drainage and debridement
- Repair of labral tears, biceps tendon lesions
- Repair of shoulder instability
- Acromioplasty for impingement syndromes
- Rotator cuff repair

Arthroscopy of Other Joints

Arthroscopy of the wrist is useful for diagnosing torn triangular fibre cartilage and interosseous ligament rupture.

Arthroscopy of the hip is a difficult procedure but proving useful in the diagnosis of unexplained hip pain.

ADVANTAGE OF ARTHROSCOPIC SURGERIES

- Arthroscopic surgeries are more of daycare procedures, which are minimally invasive.
- Cosmetically, big surgical scars are avoided.
- Direct visualisation of the joint makes it as an excellent modality of investigation for joint pathologies.
- Post-operative morbidity is decreased.
- Reduced complication rate.

Complications of arthroscopic procedures are as follows:

1. Damage to
   - articular cartilage
   - cruciate ligaments
   - menisci and fat pad
   - extra articular structures such as vessels, tendons, ligaments
2. Haemarthrosis: It refers to collection of blood within the joint cavity
3. Thrombophlebitis
4. Infections
5. Tourniquet palsy

SPINAL SURGERY

Intervertebral disc prolapse is a common clinical problem in orthopaedic practice. Hitherto, the conventional treatment was open disc surgery. Now open disc surgery is being gradually superseded by microdiscectomy which requires a smaller opening and an operating microscope. The other options in the treatment of disc prolapse include automated percutaneous lumbar discectomy and endoscopic disc excision. These minimally invasive techniques have an advantage of needing shorter hospitalisation and shorter post-operative rehabilitation care. They also reduce spinal instability resulting from extensive laminectomy.

Better understanding of the biomechanics of the spine has resulted in the design of newer implant systems for use in spinal surgery. Use of these systems, both anteriorly and posteriorly in spinal injury along with decompression, permits earlier patient mobilisation. Pedicular screw fixation for posterior stabilisation of the thoracolumbar spine and Orion system for anterior cervical plating are examples of the newer implant systems.

With the advent of modular segmental spinal instrumentation systems and intraoperative spinal instrumentation systems and intraoperative spinal cord monitoring by somatosensory-evoked potentials, it is possible to correct spinal deformities such as scoliosis.

IMPLANTS AND INTERNAL FIXATION TECHNIQUES

Newer techniques and implants have enabled faster and better fixation of fractures leading to earlier rehabilitation and decreased morbidity.

Interlocking Nails

Conventional intramedullary nailing has gradually been replaced by interlocking intramedullary nails (Fig. 13.4). They have become the established method of internal fixation. They act as internal splints and permit locking of the nail to the bone proximally and distally, and hence provide better rotational stability. They permit early weight bearing in the lower limb fractures and thus earlier rehabilitation. In addition, bone length is maintained in comminuted fractures.

BONE GRAFTING

Bone grafting is the procedure of transplanting bone from a donor area to a recipient area. Such a grafting of bone tissue on a prepared bed in another bone is a surgical procedure often used in orthopaedic practice. The technique consists of

placing live bone pieces in close contact with a healthy raw bone surface to stimulate growth of bone tissue in the new area.

The grafted bone acts as a scaffold around which new bone tissue is laid by *creeping substitution* by vascular invasion from the surrounding tissues. It also stimulates new bone formation by the principle of *induction*.

Indications

The indications for bone grafting are as follows:

- In the treatment of non-union of fractures
- For filling cavities in bone
- For bridging gaps in the shafts of bones caused by trauma, infection or excision of tumour
- In the surgical fusion (arthrodesis) of joints

Types of Bone Grafts

The bone used for grafting may be obtained from a donor site from

- the same person (autogenous graft)
- a different person (homogeneous graft or allograft)
- a different species (heterogeneous xenograft) such as calf or pig

The best is the autogenous graft. But it is difficult to get enough bone for use in children. In such cases, the maternal homogeneous graft is the best alternative source.

Cadaver bone, stored in tissue banks, is extensively used not only to fill gaps but also to replace diseased ends of long bone or even total joints.

In terms of anatomical types, the grafts may be either a cortical bone graft in various shapes or cancellous bone pieces in the form of slivers or chips. The cancellous bone is more osteogenic as its vascularisation is quicker and bone induction better. The cortical bone functions as a fixation device and cancellous bone promotes osteogenesis.

Bone Bank

With increasing use of bone grafting procedures for various conditions, the need for large quantities of bone is great. This is met by storing bore in bone banks with special techniques of storage in sterile and viable condition.

Bone Grafting Operations

The type of bone grafting procedure depends on the biological and mechanical situation in a particular case.

Onlay bone graft is the most common type wherein a cortical graft from tibia in the rectangular shape of a plate is fixed across the prepared recipient site with metallic screws. This method is often used in the treatment of non-union of fractures. It acts mechanically to immobilise the fragments and also promotes osteogenesis.

Cancellous bone chips taken from the iliac crest are commonly used as a supplement to a cortical graft or a metallic plate in the treatment of non-union of fractures of tibia, humerus or the forearm bones. It is also used as an autogenous graft from the iliac crest or a homogeneous bank bone in filling up bone cavities after curetting or excision of cystic lesions in bone. Its function is mainly osteogenic.

Spinal fusion is the procedure which needs the largest quantity of cancellous bone graft in the operation for scoliosis. Cancellous bone chips are used to supplement Harrington or other metallic rods. A corticocancellous plate of bone taken from the iliac bone, supplemented by cancellous chips, is used as an H-shaped graft to stabilise lumbosacral junction supplemented by cancellous chips. *Pieces of ribs* are used in anterior and anterolateral spinal fusion after curetting of tuberculous lesion in the vertebra in the Hong Kong operation. The upper end of fibula is very useful as a bone graft for replacing the lower end of radius affected by giant cell tumour.

Vascularised Bone Graft

The success and the rate of integration of the bone graft with the recipient site are vastly improved when the blood supply is retained. This has been achieved by removing the graft with a vascular pedicle. This procedure requires the anastomosis of the vessels by microsurgical methods in addition to the fixation of the bone. This is used mostly while using fibula or rib as a graft.

KEY POINTS

- Arthroplasty is repair and refashioning of the arthritic or dysfunctional joint. The aim of this surgery is to obtain a joint with a good range of pain-free movements.
- The prosthesis used in total hip replacement surgery consists of acetabular and femoral components. The acetabular component has a cup made of ultra high molecular weight polyethylene and backed by a metal ring or cup. The femoral component is made of metal.
- In total knee replacement surgery, the femoral and tibial components are made of metal alloy, and the spacer is made of ultra high molecular weight polyethylene.
- Custom mega prostheses represent the future of orthopaedic oncology in India.
- Arthroscopy is visualisation of joint cavity with the help of an optical instrument called 'arthroscope'. Knee is the commonest and most accessible joint subjected to arthroscopy and various arthroscopic surgeries.

MULTIPLE CHOICE QUESTIONS

1. Arthroplasty means
   a. The joint is made of plastic material
   b. The articulating parts of bones forming the joints are excised and made to fuse together
   c. The joint is excised and bones are so kept as to avoid fusion
   d. Any of the above
2. Bone graft with maximum osteogenic potential is
   a. Fresh autograft
   b. Fresh cortical autograft
   c. Osteoperiosteal graft
   d. Vascular graft
3. The standard site for primary bone graft is
   a. Pelvis
   b. Greater trochanter
   c. Medial malleolus
   d. Tibia
4. Who is acclaimed worldwide for total joint replacement?
   a. Paul Brand
   b. John Charnley
   c. Paul Harrington
   d. Huckstep

CHAPTER 14

Injuries of Bones and Joints

Let us revise the anatomy of long bones to understand the injuries to long bones better.

STRUCTURE OF LONG BONES

Bone is a type of dense connective tissue containing cells in a mature organic material, mainly collagen and inorganic material rich in calcium and phosphate.

There are two forms of bone:

1. Compact
2. Cancellous

1. *Compact bone:* Compact bone is hard and dense. It is seen in the shafts of long bones and in the surface plates of flat bones. The compact bones contain *osteocytes* in concentric cylinders around *Haversian canals.* These canals communicate with medullary cavity and each other by *Volkmann's canals.*
2. *Cancellous bone:* Cancellous bones are found in flat bones. They consist of a sponge-like arrangement of trabeculae, arranged regularly. The direction of trabeculae is subject to constant rearrangement depending upon the physical strain it undergoes.

Parts of the Long Bone

They have a *tubular shaft*, *diaphysis* and *epiphysis* at each end (Fig. 14.1). During the growing phase, the diaphysis is separated from the epiphysis by an epiphysial cartilage or growth plate. The part of diaphysis that lies adjacent to the epiphysial cartilage is called *metaphysis.* The shaft has a marrow cavity containing bone marrow. The outer part of the diaphysis is a composed compact bone that is covered by a *periosteum.* The ends of the long bones are composed of cancellous bone surrounded by a thin layer of compact bone. The articular surfaces of the ends of bones are covered with *hyaline cartilage.*

Flat bones are found in the skull, pelvis and scapula and are composed of thin inner and outer layers of compact bone separated by a layer of cancellous bone.

FRACTURES AND BIOLOGY OF FRACTURE HEALING

A break in structural continuity of the bone is termed as *fracture.* There may be multiple fracture lines indicating comminution of the bone or it may be a single line separating two fractured bone ends. The mode of violence and the direction of transmission of force determine the site and pattern of the fracture.

It is important to understand the biological process of fracture healing and the factors influencing it, as it helps one to understand the principles of treatment. This process varies in cortical and cancellous bone.

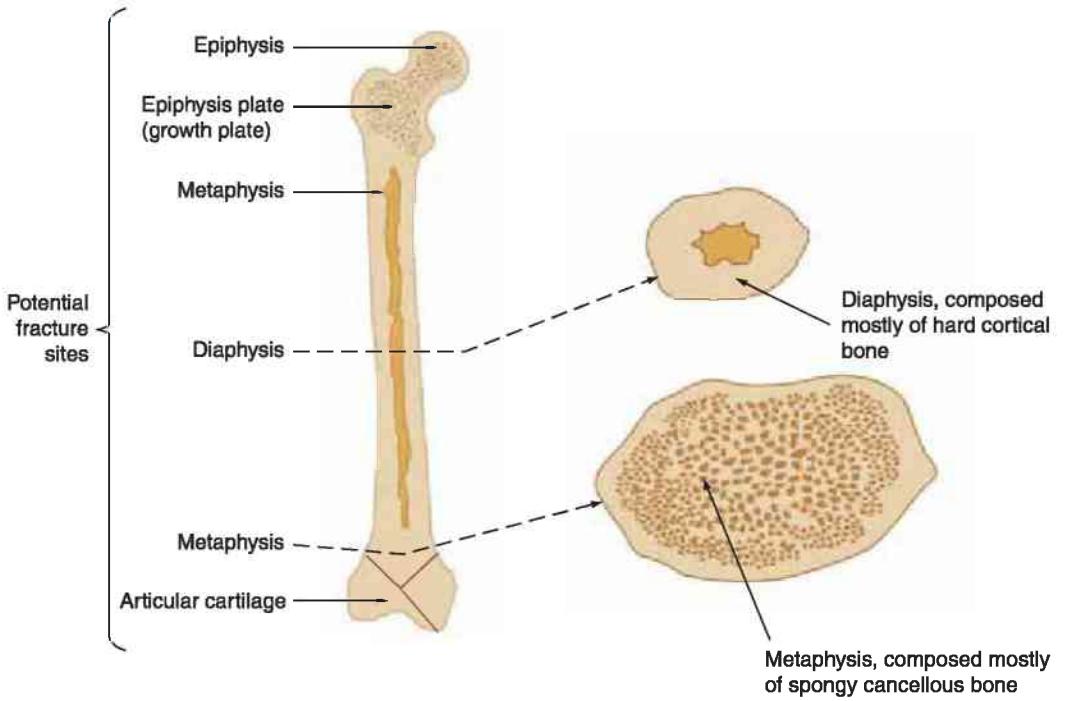


FIGURE 14.1 Diagrammatic representation of a normal long bone.

Fracture Healing in Cortical Bone

Fracture healing will be considered as a series of phases which occur in sequence but also overlap to a certain extent:

1. Inflammatory phase
   - Stage of haematoma formation
   - Stage of granulation tissue
2. Reparative phase
   - State of fibrocartilaginous callus
   - Stage of bony callus
3. Remodelling phase

Stage of Haematoma

When a bone breaks, the gap is filled with blood from the ruptured periosteal and endosteal vessels. This blood distends the soft tissues and clots to form a ***haematoma***. This process takes about 1–2 days (Fig. 14.2).

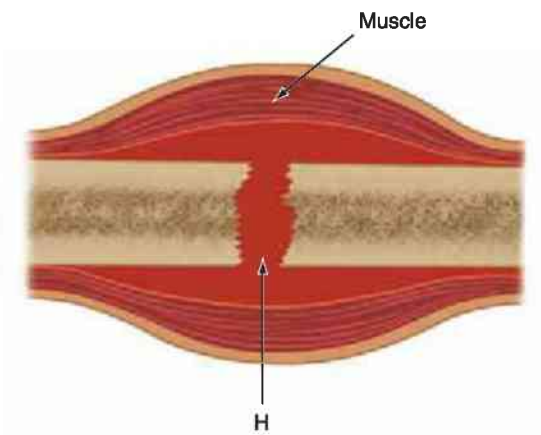


FIGURE 14.2 Diagrammatic representation of the stage of haematoma (H—haematoma).

Stage of Granulation Tissue

The soft tissues in the region undergo the usual changes of acute aseptic inflammation with vasodilatation and exudation of plasma and leucocytes. The clotted blood is invaded by fine capillaries and young connective tissue cells and converted into granulation tissue in about 2 weeks (Fig. 14.3).

Stage of Callus

The granulation tissue next matures into a fibrocartilaginous mass which holds the fragments

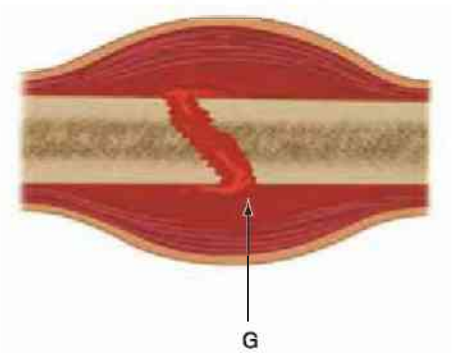


FIGURE 14.3 Diagrammatic representation of the stage of granulation tissue (G–granulation tissue).

together. Because of the peculiarities of microcirculation in cortical bone, there is some degree of cellular death in the ends of the fracture bone. The fundamental healing response of bone to injury is by the primary callus response.

According to the situation and function of callus distributed around the fracture site, the callus is described as follows: anchoring callus, bridging callus, uniting callus and sealing callus.

The fibrocartilaginous mass is first converted into ***spongy immature bone*** and later into ***mature lamellar bone***, producing bony union between the fragments in about 8–12 weeks. This conversion takes place in some areas by membranous ossification and in other areas by endochondral ossification. By this time, the clinical union of the fracture is complete.

Stage of Remodelling

Once the fracture has been satisfactorily bridged, the newly formed bone adapts to its new function. The site of fracture undergoes remodelling by muscular and weight-bearing stresses and any slight deformity gets corrected by moulding. This remodelling process takes upto a year and is seen better in children.

Primary Bone Healing

Healing of fracture has also been achieved by artificial methods of mechanical compression between the fracture fragments. In this, external bridging callus is suppressed and healing is dependent on the activity of medullary callus and direct osteonal penetration. Hence, there is no radiologically visible callus. This has been called 'primary bone healing' in the technique of compression plating of fractures.

Fracture Healing in Cancellous Bone

In fractures at the metaphyseal ends of long bones and in solid bones such as vertebrae, the healing process is different. There is no terminal bone death as in cortical fractures.

When there is direct contact of fragments, healing occurs by the process of *creeping substitution*. New trabeculae formed by intramembranous ossification are laid down on the original trabeculae to produce bone between the two fragments. No bridging callus is formed. Once union is established, remodelling occurs.

FACTORS INFLUENCING FRACTURE HEALING

The natural tendency for a fracture is to unite. When delay or failure of union occurs, the causes are either local factors at the site of fracture or defects in the methods employed in treatment. Causes interfering with the healing of fractures are as follows:

1. Imperfect immobilisation
   - Too little an extent of immobilisation
   - Too short a period of immobilisation
2. Distraction: Too heavy a pull of the distal fragment by skeletal traction
3. Surgical intervention: This empties the fracture haematoma and strips the periosteum, interfering with the blood supply and slowing the healing process

Local Causes

- *Infection:* This is the commonest cause for delayed union or non-union in open fractures.
- *Inadequate blood supply to one fragment:* Certain sites are notorious for slow union or non-union (e.g. fracture neck of femur—the blood supply to the head of the femur is poor, fracture scaphoid—the blood supply to the proximal fragment is poor). Interposition of soft tissues between the fragments

prevents bony apposition and interferes with healing.
- *Type of fracture:* Transverse fractures unite slowly compared to oblique or spiral fractures.
- *Type of bone:* Fracture at the cancellous ends of bone unites better than those in the midshaft of long bones where cancellous bone is minimal.

General Causes

Fractures in children unite very rapidly whereas delayed union is common in the aged. Other factors such as protein and vitamin deficiencies and general diseases such as diabetes play only a small part in influencing the rate of healing.

Biocompression

Biocompression at the fracture site through protected weight bearing at the proper time promotes healing of the fractures.

Types

In general, a fracture can be classified as follows:
- Traumatic fractures
- Pathologic fractures
- Fatigue/stress fractures
- Iatrogenic fractures

Mechanism of Fracture

A fracture can be caused by either direct violence or indirect violence. *Direct violence* causes a fracture at the site of impact of the force. *Indirect violence* is one that is transmitted to a bone away from the site of impact producing a fracture there.

When a man falls from a coconut tree or the top of a building and lands on his heels, he often sustains a fracture calcaneum and fracture of the spine. The fracture calcaneum is caused by direct violence and the fracture spine is caused by indirect violence.

The nature of violence can often be inferred from the radiological appearances of the fracture. Direct violence often produces a comminuted fracture. Torsion produces an oblique or spiral fracture.

TRAUMATIC FRACTURES

Traumatic fractures are commonly seen in road traffic accidents. They can be either simple (closed) fracture or compound (open) fracture:

1. *Closed/simple fracture:* Fracture in which the overlying skin is intact and the bone does not communicate with the external environment is termed as closed fractures.
2. *Open/compound fracture:* Fracture which communicate with the external environment is termed as open fracture (Fig. 14.4). This in turn can be classified as follows:
   - Compound from with in-out: The fractured bone protrudes outside, from within, piercing the skin
   - Compound from without in

The trauma causes skin laceration or skin/soft tissue loss, thereby exposing the fracture ends. Chances of infection are very high.

Table 14.1 gives the classification of open fractures.

TABLE 14.1 Gustillo–Andersons' Classification of Open Fractures

Type	Features
Type I	Clean wound of less than 1 cm in length
Type II	Wound larger than 1 cm in length without extensive soft tissue damage
Type III	Wound associated with extensive soft tissue damage; usually longer than 5 cm Open segmental fracture Traumatic amputation Gunshot injuries Farmyard injuries Fractures associated with vascular repair Fractures more than 8 hours old
Subtype IIIA	Adequate periosteal cover
IIIB	Presence of significant periosteal stripping (Fig. 14.4)
IIIC	Vascular repair required to revascularise leg

FIGURE 14.4 Clinical photograph of a leg showing Grade IIIB fracture of the both bones of leg. Note extensive soft tissue injury with periosteal stripping.

Based on the Pattern of Fracture Line

On the basis of the pattern of fracture line, the fractures can be classified as follows (Fig. 14.5):

- Transverse fracture
- Oblique fracture
- Spiral fracture
- Comminuted fracture
- Other types

Transverse Fracture

The fracture line runs either at right angles or with an obliquity less than 30° to the long axis of the bone. This type of fracture is sustained by either

- direct violence

  or
- indirect violence due to bending forces.

Oblique Fracture

The fracture line runs at more than 30° to the long axis of the bone. This type of fracture is sustained by

- direct violence,
- indirect violence due to bending forces.

Spiral Fracture

- The fracture line curves around the bone in a spiral manner.
- The type of violence is direct or indirect due to turning and twisting forces.

Comminuted Fracture

There are more than two fragments. They can be

- segmental fracture,
- with butterfly segments,
- with multiple irregular fragments.

Other Types

Impacted Fracture

Impacted fracture happens when one fragment is driven into the other. It is most commonly seen in

- fracture neck of femur,
- fracture neck of humerus,
- Colles' fracture.

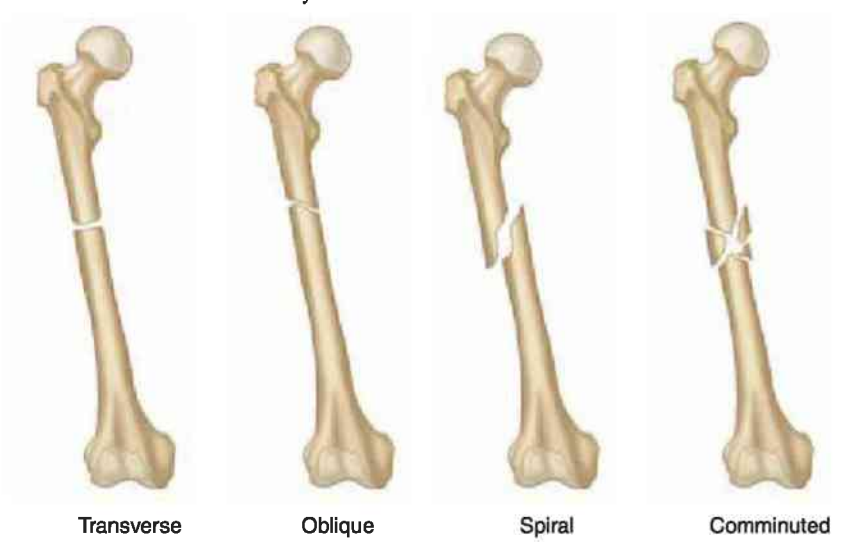


FIGURE 14.5 Diagrammatic representation of fracture types depending on the pattern of fracture line.

Compression Fracture

Compression fracture occurs usually in long thin bone where the bone is compressed by trauma. It is most commonly seen in

- vertebral body fracture,
- calcaneal fracture.

Avulsion Fracture

When there is sudden contraction of ligament/muscle, a portion of the bone to which it is attached gets pulled off, for example fracture of tibial tuberosity or fracture of upper pole of patella due to pull of quadriceps femoris muscle. Another example is fracture base of fifth metatarsal bone due to pull of peroneus brevis muscle.

Depending on the Deformity

Depending on the type of deformity, the fractures can be classified into undisplaced and displaced fracture.

Undisplaced Fracture

When the fractured ends are in anatomical positions aligned with each other.

Displaced Fracture

The direction of displacement is described by the movement of distal fragment (Fig. 14.6).

- *Shift:* It can be either upwards (overlap) or downwards (distraction).

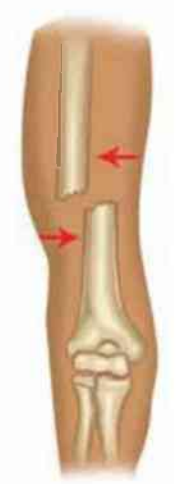


FIGURE 14.6 Diagrammatic representation of a displaced fracture of humerus.

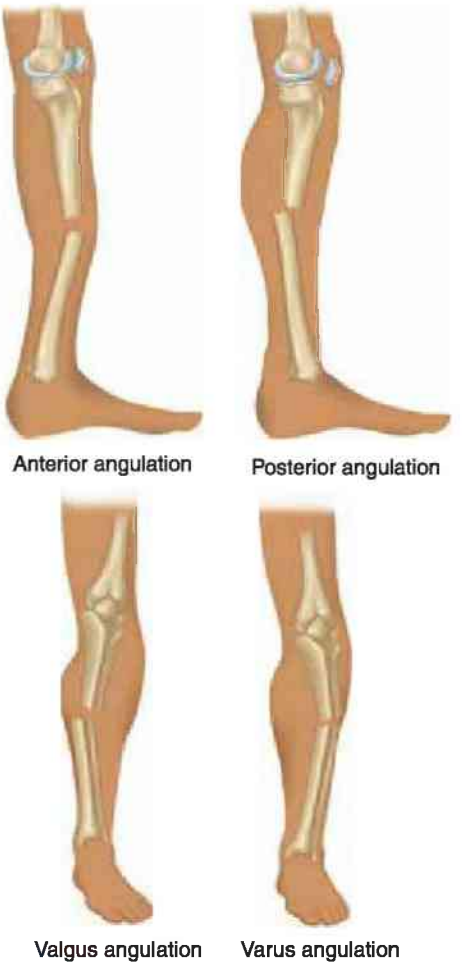


FIGURE 14.7 Diagrammatic representation of types of displacements leading to different kinds of fractures.

- *Translation:* It can be either mediolateral translation, anteroposterior translation or a continuation of both.
- *Angulation* (Fig. 14.7)*:* It depends on the relation of the distal with the proximal end. The angulation may be either varus (inward) angulation or valgus (outward) angulation or anterior or posterior.
- *Rotation:* This occurs when the fragment rotates along the long axis in relation to the others.

Clinical Types

From the clinical and treatment points of view, the following types must be recognised.

Fresh Fracture

- Closed fracture
- Open fracture
- Complicated fracture, when the fracture is associated with injuries to the neighbouring vessels or nerves

Malunited Fracture

The patient comes with an old fracture united in a bad position.

Un-United Fracture

The patient presents with an old fracture where the fragments have failed to unite.

Pathological Fracture

Pathological fracture must be suspected when an old person presents with a fracture following minimal violence.

PATHOLOGICAL FRACTURE

Pathological fracture occurs in a bone weakened by a pathological lesion following a trivial injury.

Clinical Features

When a patient presents with a fracture, with a history of minimal or trivial violence, one should suspect pathological fracture. On careful questioning, the patient may admit of having had some local pain or discomfort even before the occurrence of the fracture. The pain at the site of fracture is usually less than that in a traumatic fracture. The causative pathology may be very obvious as in the case of advanced primary malignancy, but sometimes the pathological fracture in the elderly is a secondary deposit from a primary malignancy elsewhere or multiple myeloma of bone. Pathological fracture in the vertebrae often presents as an exacerbation of backache.

Radiological Features

The fracture line is often transverse and clear-cut. The fracture line runs across a localised osteolytic area in the shaft of the bone. Sometimes, the whole bone may show evidence of generalised rarefaction or osteosclerosis.

Aetiopathology

The lesions which weaken a bone and make it susceptible to fracture can be classified as follows:

1. Generalised disorders
   a. Children
      - Osteogenesis imperfecta
      - Rickets
   b. Adults
      - Osteomalacia
      - Osteosclerosis
      - Hyperparathyroidism
   c. Old age
      - Generalised osteoporosis of bone (senile or post-menopausal)
      - Paget's disease of bone
      - Carcinomatosis
      - Multiple myelomatosis
2. Local lesions
   a. Benign
      - Solitary bone cyst in children and adolescents
      - Parathyroid lesion (localised), fibrous dysplasia
      - Enchondroma of bone in hands and feet
      - Osteomyelitis
   b. Malignant
      - Secondary deposit in bone from the primary lesion in thyroid, breast, bronchus, kidney or prostate
      - Primary malignant tumour in bone (e.g. Ewing's tumour)

Management

First, confirm the diagnosis of the pathology of the fracture. This is done by establishing the nature of the causative pathology by

- blood biochemistry including serum calcium, inorganic phosphates, serum proteins and electrophoretic pattern;
- imaging techniques: plain X-ray, tomography, computerised tomography (CT) scan and isotope bone scan, magnetic resonance imaging (MRI);
- biopsy at the site of fracture or any other accessible lesion.

Treatment

This should include treatment for the fracture (by reduction and retention of the fragments by immobilisation) and treatment for the disease. An operative curetting followed by internal fixation will help in establishing diagnosis, removal of the pathology and also treatment for the fracture. Most pathological fractures unite well after treatment. Pathological fracture through a benign lesion-like simple bone cyst or enchondroma is treated by surgical curettage and bone grafting with excellent results.

STRESS FRACTURE

Stress fractures are fractures usually occurring in weight-bearing bones, caused by repeated minimal stresses. It is seen following unaccustomed strenuous exercises in military recruits under training. It commonly occurs in the metatarsals of the foot (march fracture) upper tibia and fibula and neck of femur.

The patient presents with diffuse pain of duration of a few weeks with no history of specific injury to the site. Clinically, there is localised tenderness at the bone. Radiography shows a hairline crack in the bone and there may even be some callus around the site. Rest for a few weeks and symptomatic treatment are usually sufficient.

OTHER TYPES OF FRACTURES

- *Green stick fracture:* It is the fracture in the young bone of children where the break is incomplete, leaving one cortex intact.
- *Birth fracture:* It is a fracture in the newborn child due to injury during delivery.
- *Depressed fracture:* This occurs in the skull where a segment of bone gets depressed into the cranium.
- *Stellate fracture:* This occurs in flat bones of the skull and in patella, where the fracture lines run in various directions from one point.

MANAGEMENT OF FRACTURES

The aim of the treatment is to restore the limb functionally and anatomically so that it is indistinguishable from the normal. While anatomical restoration is certainly desirable, it is far more important to restore the function. The general principles of management of fracture patients are as follows:

- Efficient first-aid: This is done by splinting. This relieves the pain and prevents complications.
- Safe transport: This helps to minimise complications in injuries to the spine, fractures of the lower limbs, ribs, etc.
- Assessment of the general condition of the patient for shock and other injuries
- Assessment of the local condition of the injured limb regarding complications such as vascular injury, nerve involvement and injury to the neighbouring viscera
- Resuscitation
- Radiography of the part
- Reduction of the fracture
- Immobilisation of the fragments
- Early physiotherapy for the preservation of the function of the limb
- Rehabilitation after the union of the fracture to restore full muscle power and joint movements and to make the man fit for his original job

Diagnosis of Fracture

The complete diagnosis of a fracture includes recognition of the presence of a fracture and its site, the nature of the fracture, whether traumatic or pathological, recent or old, and the presence of complications. The following points help in the diagnosis of fracture.

History

In cases of children, the usual history is that of a fall but the exact nature of the fall is not known. In adults, the mechanism of the fracture can usually be made out from the nature of the violence, either a direct injury on the bone or indirect injury by a twist, fall or muscular violence.

Pain

The patient complains of pain and inability to use the limb.

Deformity

The presence of deformity in a long bone after injury is a definite sign of fracture.

Local Bony Tenderness

Local bony tenderness is the most important clinical sign for the presence of a fracture. The tenderness must be localised in a particular point in the course of the bone.

Crepitus

Although crepitus felt during the examination of the part is diagnostic of fracture, it should not be elicited purposefully as it causes severe pain and may produce further displacement and injury to soft tissues.

Abnormal Mobility

Abnormal movement in a segment of the limb denotes fracture.

Measurements

Shortening of a segment of a limb after injury indicates a fracture with over-riding of the fragments.

Radiological Features

The injured part, including the joint above and below, should be radiographed in two views. The radiograph will confirm the presence of the fracture and will also show the displacements of the fragments.

Conservative Management

The principles of conservative management are as follows:

- Closed reduction of the fracture by manipulation
- Maintenance of reduction

Reduction of Fracture

Reduction means restoration of the normal anatomical alignment of fragments in fractures. This procedure should be painless and with relaxed muscles obtained by anaesthesia.

The common types of displacements to be corrected are as follows:

- Angulation (Fig. 16.18)
- Rotation of one fragment

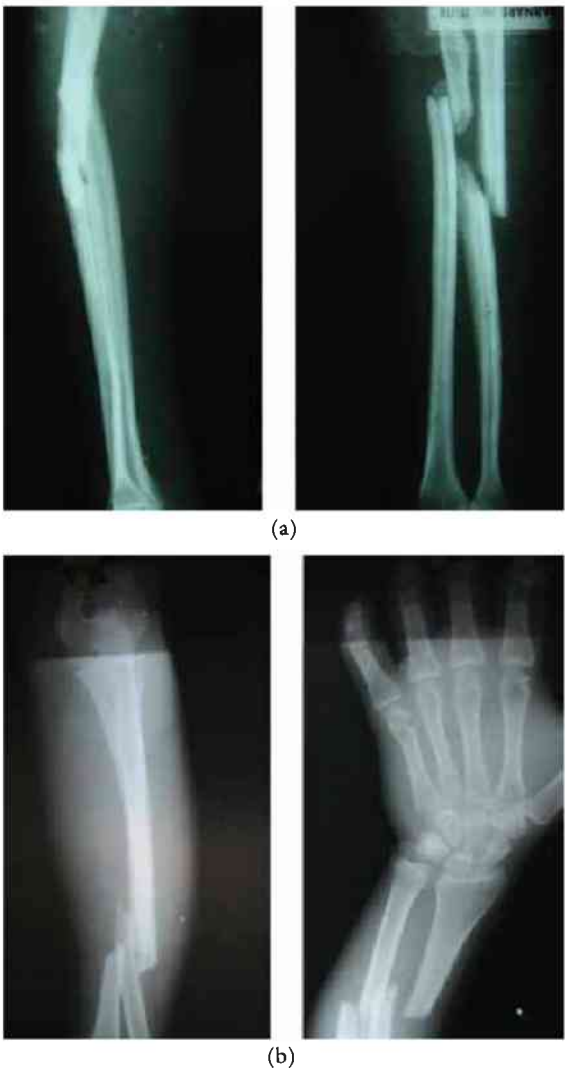


FIGURE 14.8 X-ray of forearm showing fracture of both bones of forearm with over-riding and shortening.

- Over-riding and shortening (Fig. 14.8a)
- Lateral displacement (Fig. 14.8b)

Not all fractures need reduction. Crack fractures and those with very minimal displacement need not be manipulated but need only immobilisation till union occurs.

Maintenance of Reduction

In the majority of fractures, the maintenance of the alignment of fragments is done by immobilisation with plaster of Paris casts.

Some fractures of the lower extremity such as fracture of the shaft of the femur need continuous traction to maintain the reduction and to immobilise the fragments. The methods are skin traction and skeletal traction.

Immobilisation

As a general rule, fractures must be immobilised till the union is complete and this immobilisation is done by plaster casts. The common types of plaster casts are as follows:

- Above-elbow plaster cast
- Below-elbow plaster cast
- U-slab plaster for humerus
- Below-knee plaster cast
- Above-knee plaster cast
- U-slab plaster

Post-Reduction Management

A check radiograph must be taken after the reduction to confirm the satisfactory restoration of alignment. All joints which are not immobilised should be actively exercised to prevent stiffness. This also keeps the muscles inside the plaster in good tone and minimises wasting.

Duration of Immobilisation

In children, the upper limb fractures unite in 3–4 weeks and lower limb fractures unite in 6–8 weeks. In adults, the upper limb fractures unite in 6–7 weeks and lower limb fractures unite in 12–16 weeks.

Diagnosis of the Union of Fracture

When the plaster is removed, the site of fracture is tested for clinical union. The ***absence of localised bony tenderness*** at the site is the surest evidence of the union of the fracture. The disappearance of the fracture lines in radiograph occurs much later than clinical union.

Physiotherapy and Rehabilitation

The common disabilities at the end of the fracture treatment are muscle wasting and joint stiffness, and this has been aptly named *fracture disease*. Even when the plaster is on, the patients are made to attend the physiotherapy department and are taught exercises for the joints which are free. When the plaster is removed, muscle strength is built up by graduated exercises and joints are mobilised by active and assisted exercises.

Functional Cast Bracing

Functional cast bracing is a method of conservative management of fractures which permits functioning of the joints and muscles of the limb, while immobilising the fracture.

Physiologically induced ***controlled motion*** is the single most important factor in osteogenesis (Sarmiento). This is the basic principle of all functional bracing.

The main disadvantage of the plaster cast immobilisation of limb fractures is the stiffness of joints and circulatory stagnation due to prolonged immobilisation and disuse of the limb. This is avoided in functional cast bracing.

It is generally used in the management of diaphyseal fracture of long bones such as tibia and humerus. In this method, the primary management of the fracture is reduction and immobilisation in a plaster cast. At the end of 3–4 weeks, when the soft tissue reaction has subsided and the fracture ends are sticky, the plaster cast is removed and the functional cast bracing is done as a ***second stage***. The extent of the plaster is reduced, joint movements are permitted and the muscle function is encouraged. Early weight bearing with the cast brace is allowed in the case of lower limb fractures.

Open (Surgical) Reduction and Internal Fixation

Some fractures where there is an inherent instability of the fragments or a tendency for delayed union or non-union are better treated by open reduction of the fracture by the surgical method and internal fixation.

Principles of Open Reduction

- Anatomically accurate realignment of fragments
- Rigid fixation with metallic implants

Indications

- Cases where closed methods of treatment have failed to reduce and maintain the reduction (e.g. fracture of both bones of the forearm)
- Fractures where one fragment is retracted by muscle pull (e.g. fracture patella, olecranon)
- Fractures where there is difficulty in holding the fragments rigidly in reduced position (e.g. fracture neck of femur, Monteggia fracture dislocation of the forearm)
- Fractures involving the articular surface of joints (e.g. condylar fractures of the tibia and femur)
- Fractures near a joint where a small fragment is displaced and is inaccessible for manual repositioning (e.g. fracture of the medial epicondyle of the humerus with displacement)
- Multiple limb fractures where conservative treatment is difficult in practice and cumbersome to the patient (e.g. bilateral fracture femur or tibia, fracture femur and tibia in the same leg)
- Pathological fracture
- Fracture of the distal femur or proximal tibia with vascular impairment in the leg

Methods of Internal Fixation of Fractures

After open reduction of fractures, the fragments are maintained in position by internal fixation by the following methods:

- Screws only
- Plates and screws
- Wires
- Intramedullary nails

Screw

Screws alone are used to stabilise small fragments such as medial malleolus of the ankle and lateral condyle of humerus. The types of screws available are cortical screws, cancellous screws and malleolar screws (Fig. 14.9).

Plates and Screws

Plates and screws are widely used in the fixation of diaphyseal fractures such as fracture of both bones of the forearm, fracture shaft of humerus and tibia. The plates used are dynamic compression plates and semitubular plates (Fig. 14.10).

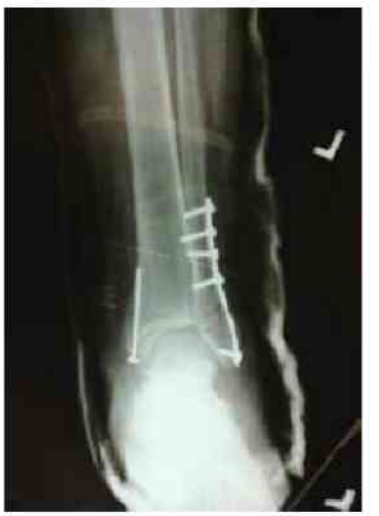


FIGURE 14.9 X-ray of ankle joint anteroposterior view showing bimalleolar fracture fixed with plates and screws.

Wires

Wires are used in the fixation of fractures of the patella and olecranon.

Intramedullary Nails

Diaphyseal fractures in the lower limbs are stabilised with intramedullary nail fixation. An example

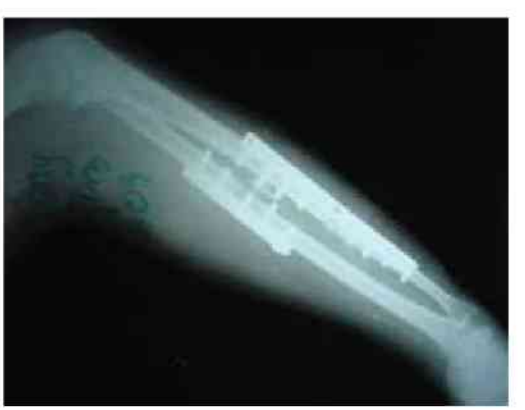


FIGURE 14.10 X-ray of forearm lateral view showing fracture of both bones of forearm fixed with plates and screws.

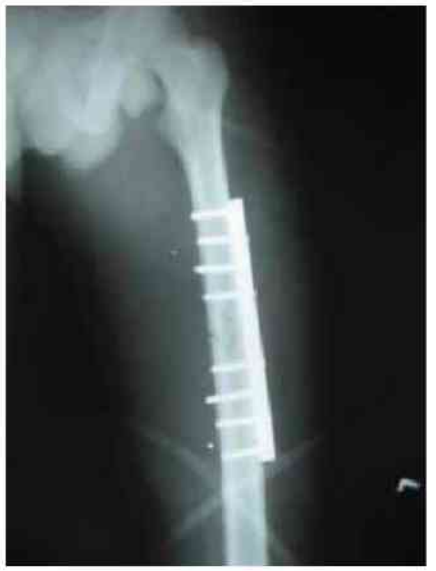


FIGURE 14.11 X-ray of femur anteroposterior view showing fracture shaft of femur fixed with plating.

is fracture shaft of femur treated by Kuntscher intramedullary nailing (Fig. 14.11).

Advantages of Internal Fixation

The main advantage of internal fixation is rigid immobilisation of the fragments which helps in earlier mobilisation and quicker restoration of function.

Disadvantages

Internal fixation involves surgical opening of the fracture site which exposes the site to infection. The stripping of the periosteum during surgery causes interference with the blood supply to the fragments and delays the healing process. Trauma to the soft tissue also contributes to joint stiffness.

Infection after an operation on bones is a disaster which could lead to chronic osteomyelitis and prolonged misery. Such an iatrogenic complication should be prevented at any cost.

COMPLICATIONS OF FRACTURE

These complications can be immediate, delayed or late. Many of these are preventable, and hence great care should be taken to minimise their incidence.

Immediate Complications

Immediate complications are usually caused by the violence producing the fracture, and these occur at the time of fracture or immediately after. These can be general complications such as shock or local complications such as injury to vessels, injury to nerves or viscera in the vicinity.

Delayed Complications

These are complications, setting in after a few days up to a few weeks. Infection in open fractures causing non-specific wound infections or specific infections such as tetanus and gas gangrene occur in the first few days. The other complications are as follows:

- Fat embolism
- Volkmann's ischaemia
- Delayed nerve injury
- Myositis ossificans

Late Complications

Late complications occur as late results of the injury or of its mismanagement. These include the following:

- Malunion
- Non-union
- Cross-union
- Stiffness and contracture of joints
- Post-traumatic osteoarthrosis
- Late nerve palsy (Tardy paralysis)
- Avascular necrosis
- Infection in open fracture

Some of the important complications are described below.

Volkmann's Ischaemic Contracture

Volkmann's ischaemic contracture (VIC) is a contracture developing in a group of muscles caused by ischaemia due to compression or spasm of arteries.

Aetiology

It is seen in the forearm and hand and less commonly in the leg and foot. It is seen in hospital practice due to the application of tight plaster bandages. It can occur in grossly displaced

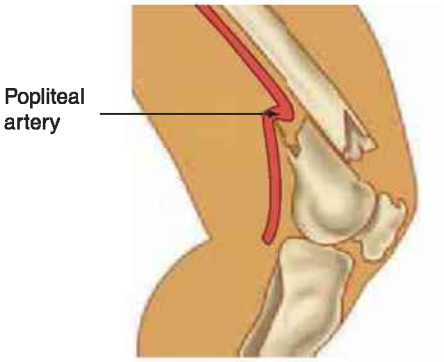


FIGURE 14.12 Diagrammatic representation of supracondylar fracture femur causing pressure on popliteal artery.

supracondylar fractures, where the sharp anterior edge of the upper fragment irritates or damages the brachial artery (Fig. 14.12).

Pathology

The condition is due to contusion, spasm or thrombosis of the brachial artery at the elbow, or the radial and ulnar arteries in the upper half of the forearm. It is also due to the oedema and increased pressure in the anterior osteofascial compartment of the forearm. There is an area of ischaemic necrosis of the pronator and flexor muscles in the forearm, followed by fibrosis and later contractures of these muscles. There may also be associated ischaemic paralysis of the median and ulnar nerves. In later stages, there are secondary capsular contractures of the wrist and the finger joints.

Clinical Features

Clinically, this can be described in the following two stages:

1. Acute Volkmann's ischaemia
2. Chronic Volkmann's ischaemia contracture

Acute Volkmann's Ischaemia

The patient who has been manipulated and plastered for supracondylar fracture elbow may present the next day with acute unbearable pain and swelling in the fingers. Capillary filling in the nail bed and radial pulse is absent. There may also be paraesthesia and paresis due to ischaemia of median and ulnar nerve. Passive extension of the fingers causes severe pain (stretch pain). The clinical features thus are pain, pallor, paraesthesia, pulselessness and paralysis.

Treatment

Treatment is emergency as the condition threatens the limb itself. The complication must be tackled with speed and vigour to prevent permanent damage.

At this stage, all constricting bandages and plaster should be removed immediately and flexion at the elbow lessened. If the fracture at the elbow has not been reduced, immediate manipulation must be done to reduce the fracture and relieve the pressure on the blood vessels.

If the pulse does not reappear after this, the artery is surgically explored and decompressed. Incision of the skin and deep fascia (fasciotomy) releases the tension in the cubital fossa and forearm and restores the pulse in the vessel. If the vessel is found damaged, it should be repaired with the help of a vascular surgeon.

Chronic Volkmann's Ischaemic Contracture

This is the established contracture of the forearm muscle in varying grades of severity. The deformity is typical. The forearm is wasted, the wrist is flexed, the metacarpophalangeal joints remain extended and the interphalangeal joints are flexed.

On passively extending the wrist, the finger flexion gets worse but on full flexion of the wrist, the fingers can be fully extended passively. This is Volkmann's sign. There will also be varying degrees of paresis of the median and ulnar nerves.

Clinically, VIC is classified as mild where there is involvement of the deep flexor compartment, moderate where both superficial and deep flexor muscles are involved and severe where all flexors and some extensors are involved with neural involvement and contracture.

Treatment

This disabling condition is difficult to treat. In minimal deformities, prolonged physiotherapy and splinting with elastic traction to the fingers will correct the deformity and improve function.

The soft tissue procedures used are the sliding of the origin of the common flexor muscles or lengthening the flexor tendons. The bony

procedures used are shortening the forearm bones or excision of the carpal bones improving the appearance and function of the hand.

Ischaemic Contracture in Leg

Volkmann's ischaemic contracture also occurs in the muscles of the leg and foot. In closed fractures of the proximal end of tibial shafts, the ***haematoma*** confined under the tense osteofascial compartment compresses the tibial arteries, causing progressive ischaemia and later contracture. If untreated, it leads to equinus contracture and clawing of the toes.

Myositis Ossificans

Myositis ossificans is a condition wherein there is new bone formation in soft tissues around joints following trauma.

The name is a misnomer as it is not an inflammation of the muscles. It is better referred to as post-traumatic ossification. This condition is quite different from the myositis ossificans progressiva which is generalised.

Aetiopathology

This complication is very common following injuries around the elbow. Myositis ossificans can occur after reduction of the dislocation of the elbow or supracondylar fracture. Sometimes, it occurs even after minimal injuries such as crack supracondylar fracture or crack fracture of the neck of the radius. In all these cases, the invariable cause is massage to the elbow and vigorous passive stretching to restore movements of the elbow.

It also occurs around hip joints following head injuries and traumatic paraplegia.

The exact mechanism of this type of new bone formation is unclear. Following trauma, there is haemorrhage around the periosteum, capsular ligaments as well as muscles surrounding the joint. In some cases, in the reactionary stage, there is formation of new bone around these tissues. This is called myositis ossificans.

Clinical Features

In the early active stage, there is slight warmth with limitation of movements due to muscle spasm. In the later consolidating stage, a firm lump is palpable in front of the elbow. In the final stages, a bony hard lump is felt surrounding the elbow with total loss of movement.

Radiological Features

In the early active stage, a fussy ill-defined radio opacity (cotton wool appearance) is seen in front of the elbow. In the later mature stage, the radiograph shows a dense irregular radio-opaque mass.

Treatment

The best treatment is preventive. In all cases of elbow injuries, strict instructions must be given not to give massage or passive stretching to the joint after removal of the plaster. In the active stage, the range or movement is recorded and the elbow is rested in a plaster slab. When the plaster is removed, it will be found that the movement has improved and the shadow is smaller but denser. The radiograph may then show a dense well-defined homogeneous calcified mass. Thereafter, active exercise is encouraged.

When the condition is well established and non-progressive, surgical excision of the myositic mass may be done to restore mobility. Some cases may benefit by arthroplasty (excision or replacement) of the elbow joint.

Fat Embolism

Fat embolism syndrome is a post-traumatic complication causing sudden respiratory distress. It occurs within the first few days after major polytrauma or fracture of the pelvis or femur. It can also occur after fracture manipulation or intramedullary fixation operation.

Pathogenesis

Free fat globules of microscopic sizes from the bone marrow escape into the blood stream and cause embolic phenomena in the lungs, brain and skin.

Clinical Features

The condition usually occurs in young adults and is of sudden onset, presenting with acute pulmonary or cerebral symptoms. The early symptoms are shortness of breath, followed by restlessness

and confusion. The clinical signs are pyrexia, tachycardia and tachypnoea with dyspnoea and cyanosis. Characteristic petechial rashes develop in the chest, axillae, foot, neck and conjunctiva. Disorientation and coma follow in more severe cases which may end fatally.

Investigations

There is arterial hypoxaemia due to pulmonary insufficiency, and PaO_2 values fall below 60 mm Hg. Thrombocytopenia also occurs. Urine may show sudanophilic granules.

Fundoscopy reveals fat emboli in retinal vessels. Chest X-ray shows a snow storm appearance.

Treatment

The only specific treatment of fat embolism is directed at improving the hypoxaemia due to respiratory distress. Oxygen is administered by nasal tubing or face mask ventilator. Accurate monitoring of blood gases, fluid and electrolyte balance is essential. The use of massive steroid therapy has been found to be helpful. Mild or moderate cases recover in 7–10 days. Massive fat embolism is most often fatal.

Non-Union

Delayed union of a fracture is one wherein healing has not progressed at the average rate for the site and type of fracture (usually 3–6 months).

Non-union is established when the fracture shows no visible progressive signs of healing for 3 months, and a minimum of 9 months has elapsed since injury.

Aetiology

The causes of delayed and non-union are as follows:

- Soft tissue interposition between the fragments
- Segmental fractures with impaired blood supply to the middle fragment
- Comminuted fractures
- Open fractures
- Infected fractures
- Pathological fractures
- Inadequate immobilisation
- Insecure fixation and premature weight bearing

Pathology

There are two types of non-union:

1. The hypertrophic type where the fracture ends are hypertrophic, sclerosed and vascular. There is a fibrous union and this has a biological capacity to unite.
2. The atrophic type where the fragments are inert and avascular. The ends of the fragments are tapering, osteoporotic and very mobile with sometimes a false joint with even a synovial lining.

Classification

Non-union is classified as follows:

1. Hypervascular non-union
   - Elephant foot type
   - Horse hoof type
   - Oligotrophic type
2. Avascular non-union
   - Comminuted
   - Gap
   - Atrophic
   - Torsion wedge

Clinical Features

It occurs in long bones such as humerus, forearm or tibia. It occurs in intracapsular fracture of the neck of femur and in the fracture scaphoid. The characteristic sign is abnormal mobility or yielding at the fracture site without pain.

Radiological Features

Radiologically, there is sclerosis of fracture surfaces and closure of the medullary canal in the hypertrophic type (Fig. 16.19). There is osteoporosis and tapering of fracture ends in the atrophic type.

Treatment

Established non-union in long bones has to be treated by operation. The fracture site is *freshened*

by excision of the scar tissue and the bone ends fixed by *internal fixation* and supplemented by *cancellous bone grafts* to promote osteogenesis.

Malunion

This means that the fracture has anatomically malunited with angulation, rotation or over-riding of the fragments. This is due to failure to reduce the fragments into proper alignment or failure to hold them in position till union.

Clinical Features

The patient presents with a deformity at the fracture site. It commonly occurs at the shaft of long bones (e.g. forearm, femur, tibia) or at the end of bones (e.g. supracondylar fracture humerus, Colles' fracture). Radiology will show the degree of angulation, rotation or over-riding of the fragments.

If the deformity is minimal and the function of the limb is satisfactory, malunion can be accepted. In young children, malunion tends to correct itself by remodelling at the fracture site. If the deformity is gross or functional disability is marked, malunion is surgically treated by osteotomy, realignment and internal fixation.

Cross-Union

This complication can occur in fractures of the shafts of the radius and ulna and in fractures of the tibia and fibula. The proximal fragment of one bone unites with the distal fragment of the other bone. When it occurs in the forearm, supination and pronation movements are lost. In the leg bones, cross-union is not of much significance.

Sudeck's Osteodystrophy

Sudeck's osteodystrophy is a post-traumatic painful stiffness of the hand and fingers. It commonly occurs as a complication of Colles' fracture or even as minor trauma to the hand. It also occurs in the foot after injuries. Its exact aetiology is unclear. It is considered to be a post-traumatic, reflex sympathetic osteodystrophy, producing vasomotor disturbances.

Clinically, the hand is swollen, painful, smooth and glossy. Skin creases are obliterated. Nails and hair are atrophic. It is associated with capillary and venous dilatation. The joints of the hands are stiff and painful.

Radiological Features

In early stages, there are generalised speckled areas of osteoporosis. Later, the cortex also gets demineralised with a glassy appearance of all bones of the forearm and hand.

Treatment

Treatment is by vigorous physiotherapy using active exercises, assisted movements, splinting and keeping the part elevated. Management is difficult and recovery is slow. In very severe cases, relief may be obtained by a cervical sympathetic block or sympathectomy operation.

FRACTURES IN CHILDREN

Fractures in children are important as the presence of epiphysis in long bones makes them vulnerable to damage to the growth plate, resulting in growth disorders and deformities. There are several differences between the bones of children and adults. Children's bones are pliable and withstand greater bending force than the rigid adult bone. As the periosteum is thick, it remains intact on one side of the cortex in moderate violence resulting in green stick fractures. Children's fractures unite much faster than those in adults. Inaccurate reduction with angulation can still heal in good shape due to remodelling. Shortening due to overlapping in long bone fractures as in femur gets corrected upto 1/2 inch due to stimulation of growth in the epiphysis.

Injuries in children occur as domestic injuries—at home and at play, mostly due to fall. The types of injuries are as follows:

- Birth fractures
- Battered baby fractures
- Epiphyseal fracture separation

Birth Fractures

These are seen in newborn babies and are more common in deliveries in breech presentation, particularly when there is difficulty in labour.

Immobilisation by simple strapping for a few weeks is sufficient for these fractures. The following are common birth fractures.

Fracture Shaft of Femur

This occurs when the obstetrician tries to bring the leg down in the breech delivery by hooking his finger around the groin of the baby.

Fracture Shaft of Humerus

This also occurs during extraction of the upper limb in breech deliveries.

Fracture Clavicle

This occurs during difficult extraction of the after coming head in breech presentation. This is often associated with birth injuries to the brachial plexus. A crack fracture clavicle may be missed at birth and the baby may be brought 2 or 3 weeks later with the lump in the clavicle due to callus formation.

Battered Baby Syndrome

Battered baby syndrome is a condition where children are brought with one or more fractures with a suspicious history of fall. The history does not correlate with the type of fracture. There may be multiple generalised contusions along with a single fracture. There may be fractures of long bones along with rib and skull fractures. Radiographs reveal multiple fractures with a subperiosteal new bone at different stages of healing. All this suggest repeated violence by the parent or attendant.

These children should be admitted in the hospital for management and also to prevent further violence. The management includes the social worker's assistance by tactful enquiries to determine the family background and to provide counselling. Family background may reveal poverty, parental separation, alcoholism and drug addiction, unwanted babies and unwed mothers. A psychologist will also be needed for parental counselling.

Epiphyseal Injuries

Fracture separation of the epiphyses is common in children and occurs in the age group of 5–10 years. Types of epiphyseal injuries (Fig. 14.13) described by Salter are as follows:

- *Type I:* Simple separation of the epiphysis (e.g. epiphysis of the medial epicondyle at the elbow)
- *Type II:* Fracture separation with a metaphyseal segment (e.g. lower radial epiphysis). The fragment often includes a small triangular metaphyseal segment, and hence it is not a pure separation of epiphysis (Thurston–Holland sign)
- *Type III:* Fracture line only through epiphysis
- *Type IV:* Fracture through metaphysis, physis and epiphysis
- *Type V:* Compression injuries of the epiphysis (e.g. at the lower end of tibia). Epiphyseal separation near the elbow often requires open reduction and internal fixation. Compression or crush injury of the epiphysis

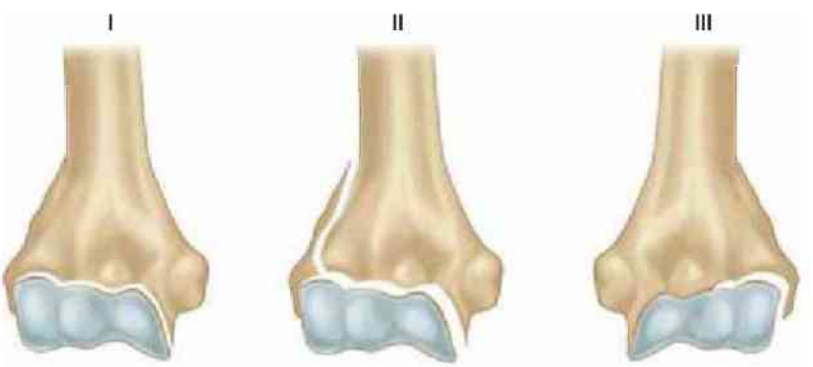


FIGURE 14.13 Diagrammatic representation of types of epiphyseal injuries.

results in premature fusion and arrest of growth at the epiphysis causing shortening or deformity.

Other common fractures in children mostly due to fall on the outstretched hand are (1) fracture clavicle, (2) supracondylar fracture and (3) fracture radius and ulna. These are dealt with in detail in other chapters.

DISLOCATION OF JOINTS

Dislocation is the total displacement of the articular end of a bone from the joint cavity. Incomplete displacement is called *subluxation*. Reduction means restoration of the normal alignment of the bones.

Classification

Dislocations are classified as follows:

- Congenital
- Traumatic
- Pathological
- Paralytic

Congenital dislocations are dealt with in Chapter 2.

Traumatic Dislocation

Clinical types of dislocation are as follows:

- Acute dislocation
- Old unreduced dislocation
- Recurrent dislocation

Acute Dislocation

The traumatic dislocations commonly occur in the shoulder, elbow and hip. The acute dislocations are further classified according to the direction of displacement of the distal bone in relation to the proximal (e.g. anterior, posterior, etc.).

Clinically, the acute traumatic dislocation is diagnosed by history and findings. There is acute pain and swelling around the joint. There is gross deformity at the joint, and the bony landmarks are distorted. The clinical signs common to all dislocations fall into two groups:

1. Signs denoting the absence of the articular end of a bone from its normal anatomical position
2. Signs denoting the presence of the displaced end of the bone in an abnormal position

One should look for associated nerve and vascular injuries. Radiographs confirm the diagnosis and detect associated fractures.

Management

Acute dislocation of a joint is an orthopaedic emergency and it requires immediate reduction under anaesthesia. After reduction, the part should be immobilised till the soft tissues such as the capsule and ligaments heal. After about 3–4 weeks, the joint is mobilised by exercise therapy.

Old Unreduced Dislocation

Patients with unreduced dislocation present themselves for treatment from weeks to months after the primary dislocation. This is unfortunately common in India due to unsuccessful treatment carried out in rural areas. These are difficult problems and need prolonged treatment.

Treatment

Closed reduction under anaesthesia is attempted in cases presenting within 3 weeks. There is a danger of fracture during the manipulation. Surgical open reduction is indicated in late cases.

Recurrent Dislocation

When a traumatic dislocation of a joint is followed by subsequent frequent dislocations by minimal trauma, it is called recurrent dislocation. This is particularly common in the shoulder joint and patellofemoral joint.

Pathological Dislocations

Pathological dislocation is caused by some disease process and is common in the hip joint. This occurs when there is destruction of the head of

the femur or excessive distention of the joint capsule. This is of two types: destructive dislocation and distensive dislocation.

Destructive Dislocation

Destructive dislocation is common under the following conditions:

- Tuberculosis of the hip when there is a travelling acetabulum
- Septic arthritis of the hip of infancy where there is total destruction of the head of femur

Distensive Dislocation

The head of the femur gets dislocated when the joint capsule is rapidly distended by an effusion of synovial fluid or pus.

Paralytic Dislocation

Paralytic dislocation occurs when there is marked imbalance of muscle power. It can occur in the hip and is always a posterior dislocation. In poliomyelitis, when the hip extensors and abductors are paralysed, the normal adductors and flexors overact and cause dislocation. In cerebral palsy, the spasm of the adductors and flexors cause the dislocation.

KEY POINTS

- Gustillo–Anderson classification is used for open fractures.
- Stress fractures commonly occur in the metatarsals of the foot (march fracture), upper tibia and fibula, and neck of femur.
- Not all fractures need reduction. Crack fractures and those with very minimal displacement need only immobilisation till union occurs.
- Functional cast bracing is a conservative method of managing fractures. It permits functioning of the joints and muscles of the limb, while immobilising the fracture.
- Open reduction involves anatomical realignment and rigid fixation.
- Volkmann's ischaemic contracture is a contracture in a group of muscles . It is caused by ischaemia due to compression or spasm of arteries.
- Myositis ossificans is very common complication following injuries around the elbow.
- Delayed union of a fracture is one wherein healing has not progressed at the average rate for the site and type of fracture (usually 3–6 months).
- Non-union is established when the fracture shows no visible progressive signs of healing for 3 months and a minimum of 9 months has elapsed since injury.
- Epiphyseal injuries are common in children. Salter–Harris classification is used for epiphyseal injuries.

MULTIPLE CHOICE QUESTIONS

1. Pathological fracture is seen in following except
   a. Radiation
   b. Anaemia
   c. Osteoporosis
   d. Osteomalacia
2. Pseudo-fractures are seen in
   a. Osteomalacia
   b. Multiple myeloma
   c. Osteoporosis
   d. Skeletal metastasis
3. Stress fracture is most often seen in
   a. Second and third metacarpal bone
   b. Talus
   c. Calcaneum
   d. Metacarpals
4. Fatigue fracture does not occur in
   a. Metatarsal
   b. Metacarpal
   c. Calcaneum
   d. Tibia
5. The earliest sign of Volkmann's ischaemic contracture is
   a. Pain during passive extension
   b. Pulselessness
   c. Necrosis of muscles
   d. Loss of adduction
6. Myositis ossificans is most common around the … joint
   a. Knee
   b. Elbow
   c. Wrist
   d. Hip

CHAPTER 15

Injuries of the Shoulder and Arm

Injuries to the bones and joints of the upper limbs are common in industrial and domestic accidents and form a high percentage of the skeletal injuries. Neglect or improper treatment leads to serious physical as well as economic disabilities due to stiffness of joints, deformities and loss of function.

One of the common causes of injuries in the upper limb is a fall on the ***outstretched hand***. This produces certain typical types of fractures in various age groups:

1. Children
   - Fracture clavicle
   - Supracondylar fracture of the humerus
   - Green stick fracture of radius and ulna
2. Adolescents
   - Epiphyseal separation of the lower end of radius
3. Adults
   - Fracture scaphoid bone at the wrist
   - Fracture of both bones' forearm
   - Fracture head or neck of radius
   - Dislocation of elbow
4. Elderly
   - Colles' fracture
   - Fracture neck of humerus

SHOULDER GIRDLE

Anatomy

The shoulder girdle is made up of three bones, namely the clavicle, the humerus and the scapula. The clavicle articulates medially with the sternum forming a *sternoclavicular joint* which is protected by a thick capsule.

Laterally, the clavicle articulates with the acromion process of the scapula forming an *acromioclavicular joint*. This joint is protected by the *coracoclavicular* ligament, which has two parts: conoid and trapezoid.

The *shoulder joint* is a ball-and-socket joint formed by the head of the humerus articulating with the glenoid cavity of the scapula. This joint is stabilised by various factors of which rotator cuff muscles are important. The rotator cuff is formed by the following muscles:

- Supraspinatus
- Infraspinatus
- Teres minor
- Subscapularis

FRACTURE CLAVICLE

Fracture clavicle is common in infants and young children. This is also one of the common birth fractures.

Nature of Violence

Fracture clavicle is caused by a fall on the outstretched hand or on the point of the shoulder. It may occur during extraction of the hand in breech delivery.

TABLE 15.1 Sites of Clavicular Fracture and Their Percentage Incidence

Sites of Fracture	Percentage Incidence (%)
Middle third fractures	80
Lateral third fractures	15
Medial third fractures	5

Site of Fracture

The most common site of the clavicle fracture is in its middle third (Table 15.1).

Clinical Features

The common site of fracture is the junction of the outer and middle third of the bone. Usually, the medial end is displaced upwards due to pull of sternocleidomastoid. The lateral end of the clavicle is displaced downwards due to pull of the pectoralis major muscle and the weight of the upper limb (Fig. 15.1). A fracture of the outer end of the clavicle is due to a direct hit on the top of the shoulder.

Diagnosis

Diagnosis is easy with a history of a fall and pain on the movements of the shoulder. The patient usually supports the flexed elbow with the normal hand. There is swelling and tenderness over the fracture site. The arm is adducted across the chest and supported by the other hand to unload the injured shoulder. The diagnosis is confirmed by taking X-ray.

Radiological Features

An anteroposterior (AP) radiograph shows the fracture. It may be a crack with no displacement or a displaced fracture with the outer fragment depressed and displaced medially. The inner fragment is displaced upwards with over-riding of the fragments (Fig. 15.2).

Conservative Treatment

Most of the undisplaced fractures unite readily with conservative treatment in the form of a figure-of-eight bandage and Jones strapping.

Surgical Treatment

Rarely open reduction and internal fixation are needed for the following:

- Displaced fractures with non-union
- Neurovascular involvement
- Floating shoulder
- Compound fractures

Floating shoulder is a condition when both the scapula and the clavicle are fractured.

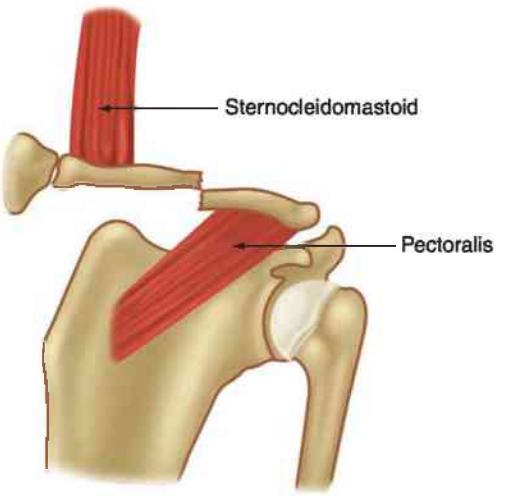


FIGURE 15.1 Diagrammatic representation of the commonest site of fracture clavicle, at the junction of outer third and middle third.

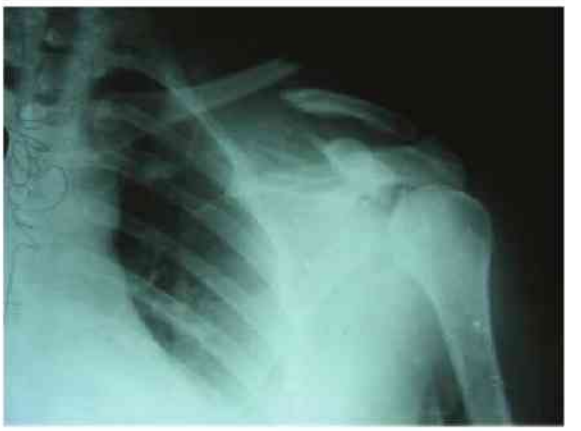


FIGURE 15.2 X-ray of the left shoulder anteroposterior view demonstrating fracture left clavicle at middle thirds.

Treatment in Infants and Children

In children below 3 years, strapping across the clavicle from the front backwards and a cuff and collar will be quite sufficient. The fracture unites in 2 or 3 weeks.

Treatment in Older Children and Adults

The principle is to lift the outer fragment upwards and maintain its alignment with the inner fragment by a firm figure-of-eight bandage with paddings in the axilla. The arm is supported in a sling. The fracture unites in about 4 weeks. Perfect anatomical reduction is not essential as fracture clavicle with moderate displacement unites well and gives good functional results.

Complications

Complications include the following:

- Malunion
- Neurovascular injuries—subclavian vessels, brachial plexus
- Shoulder stiffness
- Non-union (very rare, in distal third fractures)

Gross displacement can occasionally endanger the brachial plexus and vessels. Stiff shoulder is the commonest complication in older adults and is overcome by early mobilisation by active exercises.

Fracture of the Outer End of the Clavicle

This is often due to direct violence. The fragments are not usually displaced. A cuff and collar for 2 weeks is sufficient. When there is displacement, treatment is the same as for acromioclavicular dislocation.

DISLOCATION OF THE STERNOCLAVICULAR JOINT

This is a very rare injury. It may be anterior or posterior but the anterior is more common. The diagnosis is mostly done by clinical observation.

Treatment

Treatment is done by closed reduction by giving pressure over the area and maintenance of the reduction by strapping.

DISLOCATION OF THE ACROMIOCLAVICULAR JOINT

The acromioclavicular joint sustains subluxation or dislocation due to a fall on the outer aspect of the shoulder. Partial rupture of the coracoclavicular ligaments results in subluxation and complete rupture results in dislocation. Clinically, the patient presents with acute pain on the top of the shoulder. There is an elevation of the outer end of clavicle and tenderness at that site. There is also a history of trauma.

Radiological Features

Radiology reveals the degree of displacement at the joint. An X-ray of both the acromioclavicular joints is taken for comparison.

Diagnosis

- History of trauma and pain with swelling of the injured area
- An X-ray showing the dislocation of the acromioclavicular joint

Treatment

Mild and moderate displacements are treated by strapping. This goes around the outer one-third of clavicle above and the point of the elbow below with the elbow being kept at 90° flexion. In cases of gross displacements, open reduction may be required. Repair of the ruptured coracoclavicular ligaments and internal fixation with a vertical screw or intramedullary pin give good results functionally and cosmetically.

Types I, II and III can be managed by non-operative methods in the form of triangular slings and analgesics.

Types IV, V and VI may need surgical repair.

FRACTURE SCAPULA

Fracture scapula is not very common. It is mostly due to direct injury from the back. The fracture scapula may be fracture neck of scapula, fracture body of scapula, fracture acromion or fracture coracoid. Displacement is minimal as the bone is

well padded by muscles. A cuff and collar is given for 2–3 weeks till the soft tissue injuries heal. Active movements are then encouraged to obtain good functional recovery.

SHOULDER JOINT

Anatomy

The shoulder joint is a ball-and-socket joint which provides mobility of the whole upper extremity in all directions. As mobility is more important than stability in the upper extremity, the joint is formed with a very shallow glenoid cavity in which only a small portion of the head of the humerus lies in contact. The joint is surrounded anteriorly, superiorly and posteriorly by the *musculotendinous* cuff formed by the attachment of the tendons of subscapularis supraspinatus, infraspinatus and teres minor muscles into the capsule. The muscles together with the bigger muscles such as pectoralis and latissimus dorsi help to retain the head in the socket. The inferior aspect of the capsule is unsupported by muscles and is the weakest part of the joint.

Dislocation of the Shoulder Joint

Classification

The following clinical types should be recognised.

1. Acute dislocation
   - Anterior dislocation—commonest type
   - Posterior dislocation—rare
   - Inferior dislocation—*Luxatio erecta*
2. Old unreduced dislocation (chronic)
3. Recurrent dislocation

Mechanism of Injury

The mechanism of shoulder joint dislocation is detailed in Table 15.2.

Anterior Dislocation

The shoulder is one of the joints which easily gets dislocated by trauma (Fig. 15.3). A fall on the outstretched hand with the arm in the abducted and externally rotated positions causes the head of the humerus to slip anteriorly. Occasionally, a direct violence on the back of the shoulder dislocates the shoulder forwards.

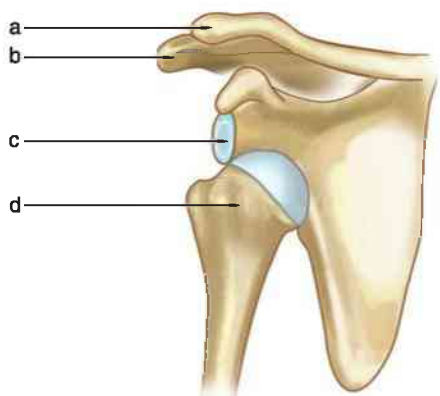


FIGURE 15.3 Diagrammatic representation of anterior dislocation in right shoulder. (a) Clavicle, (b) acromion, (c) glenoid and (d) head of humerus.

TABLE 15.2 Types of Dislocation of the Shoulder Joint and its Mechanisms

Types of Dislocation	Mechanism
Anterior dislocations	Indirect trauma to the upper extremity with the shoulder in abduction, extension and external rotation Direct trauma—anteriorly directed impact to the posterior shoulder
Posterior dislocations	Indirect trauma to the upper extremity with the shoulder in abduction, flexion and internal rotation Electric shock and convulsions usually lead to posterior dislocation Direct trauma—posteriorly directed impact on the anterior shoulder
Inferior dislocation (rare)	Hyperabduction force
Superior dislocation (very rare)	Extreme anterior and superior directed force applied to an adducted extremity

Pathology

In young adults, at the time of injury the anterior capsule with the labrum glenoidal is stripped off the anterior margin of the bony glenoid. The head of the humerus slips forwards and lies under the subscapularis muscle. The head lies commonly below the coracoid (subcoracoid type) or occasionally below the clavicle (subclavicular type). Anterior dislocation of the subcoracoid type is the most common type of shoulder dislocation. In older individuals, there is a tear in the anterior capsule through which the head slips forwards.

Bankart's Lesion

Bankart's lesion is the avulsion of the *anteroinferior labrum* off the glenoid rim.

Hill Sachs Lesion

Hill Sachs lesion is the defect caused by an impression fracture on the *posterolateral humeral head.* This is seen in 27% of the acute anterior dislocations and 73% of the recurrent shoulder dislocations.

Clinical Features

There will be history of fall on an outstretched hand. The patient is usually a young adult who typically presents with the injured shoulder held in the position of slight abduction and external rotation. The clinical features of the dislocation depend on the position of the head of the humerus in relation to the glenoid cavity. The head of the humerus may be

- absent from its normal position leaving the glenoid vacant,
- present in an abnormal position.

On inspection, the patient's arm is found to be kept away from the body and the elbow and forearm are supported by the patient's other hand. There is a flattening of the deltoid outline, and the normal rounded contour of the shoulder is lost. An abnormal palpable mass is present anteriorly, and there is fullness in the anterior axillary fold. On palpation, the emptiness of the glenoid is obvious with the normal bony resistance under the deltoid being absent. The spherical-shaped head of the humerus could be palpated deep to the pectoral muscles either below the coracoid process or sometimes below the clavicle. Due to the dislocation, it will not be possible for the patient to bring the elbow close to the body and put the hand on his opposite shoulder (*Dugas' sign*). This sign is diagnostic of fresh dislocation. One should always look for the complication of circumflex nerve paralysis. This nerve gets easily stretched as the head of the humerus slips forwards and results in paralysis of the deltoid. The following are the clinical tests to diagnose anterior shoulder dislocation:

1. Apprehension test (in recurrent dislocations, discussed below)
   - *Dugas' test:* Inability to touch the opposite shoulder (discussed above)
   - *Hamilton ruler test:* A ruler placed on the lateral side of the arm, touches acromion and lateral condyle of the humerus simultaneously when it is dislocated
   - *Callaway's test:* Vertical circumference of axilla is increased

Radiological Features

The diagnosis is confirmed by an AP X-ray view of the shoulder joint. This view of the shoulder demonstrates that the head of the humerus is displaced and the glenoid empty. There may be an associated fracture of the greater tuberosity of the humerus.

Treatment

Treatment includes closed manipulative reduction under general anaesthesia and immobilisation in an arm sling and shoulder immobiliser strapping. Mobilisation is started at 3 weeks. The anterior dislocation is easily reduced when it is a fresh one. The usual method is called *Kocher's manoeuvre.* In this method, the subscapularis muscle, which is in spasm, is tired out by gradual external rotation of the shoulder and then the head of the humerus is made to slip back into the socket.

Reduction Techniques for Shoulder Dislocation

Kocher's Manoeuvre

Traction, external rotation and adduction are done to reduce the dislocation.

Kocher's manoeuvre is the commonly used method.

The other methods used are as follows:

- Hippocrates manoeuvre
- Milch manoeuvre
- Stimson's manoeuvre

Post-Reduction Management

The arm is strapped to the body with a pad in the axilla and a short cuff and collar is applied. Check radiograph is taken. This position of internal rotation should be maintained for 3 weeks to allow the torn capsule to heal completely.

Posterior Dislocation

Posterior dislocation is a rare type which occurs during attacks of fits or electroconvulsive therapy. Here the head is displaced posteriorly, behind the glenoid, and the arm is in internal rotation. Clinically, there is restriction of abduction and no external rotation. It is classified, according to the anatomical lie of the head, as subacromial, subglenoid and subspinous. This is often missed and needs a superoinferior view radiograph also. On AP X-ray, the head of humerus stands away from the glenoid fossa, 'empty Glenoid sign'. Due to internal rotation, head looks abnormal, 'Light bulb sign'.

Luxatio Erecta

Luxatio erecta is a rare inferior dislocation of the shoulder. 'Sulcus test' is indicative of laxity of inferior glenohumeral ligaments.

Complications

Early

Injury to axillary nerve leading to paralysis of deltoid muscles presents with weakness of abduction of the shoulder and small area of anaesthesia over the deltoid region of the shoulder. Treatment is conservative. Results are good.

Late

Recurrent dislocation is the most common late complication. It needs one of the following operations to correct it:

- Putti platt operation
- Bankart's operation
- Bristow's operation

Old Unreduced Dislocation

In our country, the patients often present with a dislocation unreduced for some weeks. Manipulation under anaesthesia can be tried for dislocations up to 4 weeks old. It becomes impossible to reduce, if it is of longer duration due to soft tissue contracture. In young adults, operative reduction is done to restore the normal anatomy. In elderly patients, active movements are encouraged to get some useful range of movement.

Recurrent Dislocation Shoulder (Anterior Glenohumeral Instability)

Recurrent dislocation shoulder is a condition characterised by repeated dislocation of the shoulder joint in a person, following one episode of acute dislocation. Subsequent dislocations require less and less violence.

Clinical Features

The patient is usually a young male adult of athletic type. Sometimes, the patient is an epileptic who gets the shoulder dislocated during the fits. Usually, there is a history that after the first acute dislocation was reduced, the shoulder was not immobilised sufficiently. The subsequent dislocations of the joint are caused when the patient does ordinary external rotation and abduction movements of shoulder while dressing or playing. The subsequent dislocations are easily reduced often by the patient himself. During the examination, the patient resists any attempted movement of abduction and external rotation due to a fear or apprehension that it may get dislocated. This is the *apprehension sign.*

Pathology

The essential pathology in recurrent dislocation is as follows:

- Bankart's lesion
- Hill Sachs lesion

Bankart's Lesion

Bankart's lesion results from the failure of healing of the detachment of the anterior capsule and the labrum from the glenoid margin (Fig. 15.4).

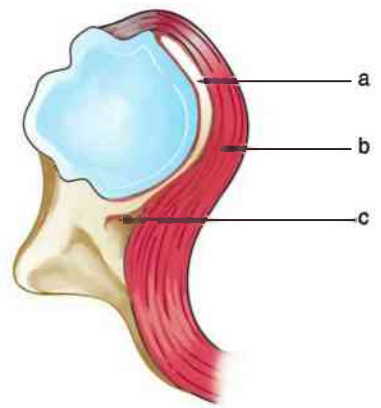


FIGURE 15.4 Diagrammatic representation of pathology in recurrent shoulder dislocations. (a) Capsule, (b) subscapularis muscle and (c) labrum glenoidale.

This leaves a pouch in front of the neck of the scapula into which the head of the humerus easily redislocates.

Hill Sachs Lesion

Hill Sachs lesion is a depression in the posterolateral aspect of the head of the humerus due to compression at the time of first dislocation. In the position of abduction and external rotation, this depression gets hitched in the posterior margin of the glenoid. Further external rotation levers the head out of the glenoid cavity and produces the dislocation.

Radiological Features

Anteroposterior views of the shoulder with the humerus in internal rotation will show the defect in the head of the humerus in some cases.

Treatment

The treatment of this condition depends upon the age, sex and occupation of the patient and the disability due to the frequency of the dislocation.

Surgical Treatment

In younger individuals leading active lives, the best treatment is to prevent excessive external rotation movement of the shoulder by surgery. Many procedures have been described, but the following procedures have proved successful:

- *Bankart operation:* In this operation, the Bankart lesion is repaired by fixing the detached labrum and capsule back to the anterior margin of the bony glenoid.
- *Putti Platt operation:* The principle of this operation is to tighten the lax anterior structures by surgically double breasting the anterior joint capsule and subscapularis. This prevents excessive external rotation of the shoulder and redislocation.
- *Birsto Helfet operation:* In this operation, the tip of the coracoid process with the attached muscles is osteotomised and reattached near the anterior margin of the glenoid in the neck of scapula to form a dynamic anterior support.
- *Saha operation:* This procedure is based on the concept of changing the direction of the articular surface of the glenoid by osteotomising the neck of the scapula.

FRACTURE PROXIMAL END OF HUMERUS

The following types of fractures occur at the proximal end of humerus:

- Fracture of the greater tuberosity
- Fracture neck of humerus

Classification

The modern classification (Neer) is based on the involvement of the four parts of the proximal end of humerus: (1) articular segment of the head, (2) greater tuberosity, (3) lesser tuberosity and (4) surgical neck. Depending on the number of parts displaced, they are named as two-part, three-part and four-part fractures.

Clinical Features

The patient presents with pain, swelling and ecchymosis in the proximal end of humerus. In impacted fractures, the patient may be able to move the shoulder because of impaction and the fracture is likely to be missed. In displaced fractures, abnormal mobility is present. In the older patient, the possibility of a pathological fracture through a secondary malignant deposit must be kept in mind.

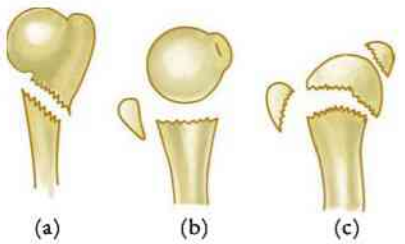


FIGURE 15.5 Diagrammatic representation of Neer's classification of fracture neck of humerus. (a) Two-part fracture, (b) three-part fracture and (c) four-part fracture.

Radiograph

Most commonly, radiology will reveal an undisplaced fracture at the neck of humerus in a bone that is osteoporotic. Displaced fractures may be two-part, three-part or four-part fractures (Fig. 15.5).

Treatment

Long periods of immobilisation in impacted fractures in the elderly will result in total stiffness of the shoulder. Cuff-and-collar rest for 2 or 3 weeks till pain subsides, and active *pendular exercises to* restore movements give good results.

In severely displaced fractures, the upper end of the broken shaft gets displaced and lies under the head with no contact with the proximal fragment. Such a type is manipulated under anaesthesia and the fragments are hitched. The shoulder is immobilised in a padded U plaster slab for 3–4 weeks and early mobilisation started.

In the younger age groups, three- or four-part comminuted fractures may need surgical reduction and internal fixation. A severely comminuted and displaced fracture may need replacement arthroplasty with prosthesis.

Complications

Complications include vascular and nerve injuries, stiffness of shoulder, malunion and avascular necrosis.

FRACTURE OF THE GREATER TUBEROSITY

This usually occurs in adults, and fall over the shoulder is the usual mechanism.

This fracture is of two types:

1. *Contusion crack fracture due to direct injury:* This needs only a cuff and collar sling for 2 weeks and active movements of the shoulder.
2. *Avulsion fracture:* Here the insertion of the supraspinatus avulses the greater tuberosity. This may sometimes occur in association with dislocation of the shoulder. With the reduction of the dislocation, the avulsed fragment usually falls into position. The arm is kept strapped to the body for 2–3 weeks.

Treatment

Immobilisation in a triangular sling is needed. If the fragment is displaced due to the pull of the supraspinatus muscle, then open reduction and internal fixation are needed.

FRACTURE SURGICAL NECK OF HUMERUS

This fracture is common in the middle-aged and the elderly but most commonly occurs in elderly women. The mechanism is a fall on the outstretched hand.

Fracture Dislocation

In some cases, the shoulder gets dislocated anteroinferiorly and the neck also breaks.

In young individuals, open reduction is done. The head is repositioned and the broken neck realigned. Internal fixation is done with screws or plate and screws.

In elderly persons, one attempt is made to reduce the dislocation and impact the fragment. If it fails, the position is accepted and early mobilisation of the shoulder is done. A reasonably good range of movement in a false joint is often obtained.

Fractures in Children

This injury is rare in children. When it occurs, it is either an impacted fracture neck of humerus or a fracture separation of the epiphysis of the head of humerus. It can also occur as a pathological fracture through a simple bone cyst.

FRACTURE OF THE SHAFT OF HUMERUS

This fracture is usually due to direct violence and is often comminuted. Occasionally, it occurs as a birth fracture in the newborn due to violent extraction of the arm in a breech presentation. This is common in all age groups. The mechanism of injury is a fall on the outstretched hand or a direct trauma over the arm. Displacement of the fracture is common and the proximal fragment goes for abduction due to the pull of the deltoid muscle.

Classification

1. Transverse fracture.
2. Oblique and spiral fracture
3. Comminuted fracture

Clinical Features

The usual signs of fracture, namely local bony tenderness and deformity, are present. Abnormal mobility at the middle of the arm will make it obvious. The most important complication to look for in this fracture is an injury to the radial nerve as it winds round the spiral groove, producing wrist drop.

Radiological Features

Radiographs will show the type of fracture and the displacement of the fragments (Fig. 15.6).

Treatment

The reduction of this fracture is easy. Without anaesthesia, the patient is kept in the sitting posture. The fracture is reduced and a U-shaped plaster slab is applied and cuff and collar is given (Fig. 15.7). Most of the fractures unite easily. Some amount of angulation and displacement is acceptable and strict immobilisation is not necessary.

Conservative Methods

This is helpful in most of the cases. Plaster slab is applied in the form of either 'U' slab or hanging cast supported with a sling. Immobilisation for 6–8 weeks is needed. In adults, early mobilisation is started by using cast brace.

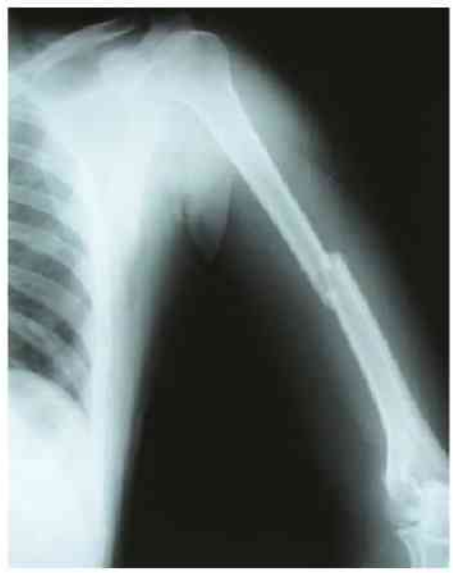


FIGURE 15.6 X-ray of the left arm anteroposterior view demonstrating fracture shaft of humerus.

FIGURE 15.7 Clinical photo of U slab applied to left arm.

Treatment

Surgery is indicated for early mobilisation and if associated injuries are present. The indications for surgery are as follows:

- Failed closed reduction
- Unstable fractures
- Fractures associated with radial nerve injury

Most fractures are fixed with dynamic plates and screws. Interlocking nail is another method of internal fixation. Compound fractures are stabilised by external fixators.

Complications

The following complications may be associated with the fracture of the shaft of humerus:

- Radial nerve injury
- Delayed union
- Non-union

Radial Nerve Injury

The radial nerve is commonly injured in the fracture shaft of the humerus when it passes in the spiral groove and laterally enter into the lateral intermuscular septum. It is usually a neuropraxia. This may lead to wrist drop with paralysis of brachioradialis and supinator and sensory change in a small area on the radial side of the hand.

Treatment

Cases of fracture shaft of humerus with wrist drop are treated conservatively adding a wrist drop splint to the U slab. The wrist drop recovers by conservative management in 4–6 weeks as the nerve injury is usually a neuropraxia. If it does not start recovering in 8 weeks, the nerve is explored and appropriate surgery is done. Other methods of treatment are as follows:

- Nerve repair or nerve grafting
- Tendon transfers

Delayed Union and Non-Union

Fractures at the lower part of the shaft are prone to non-union. This is often due to soft tissue interposition, inadequate immobilisation and distraction at the fracture site. This is treated by surgical reduction and internal fixation with compression plate and screws, supplemented by cancellous bone grafts from the iliac crest.

KEY POINTS

- Fall on outstretched hand is one of the common causes of injuries in the upper limb.
- Clavicle is most commonly fractured in middle third.
- Anterior dislocation of the sub-coracoid type is the most common type of shoulder dislocation.
- Kocher's method is most commonly used for reduction of shoulder joint.
- Apprehension sign is positive in recurrent shoulder dislocation. The essential pathology in recurrent shoulder dislocation is Bankart lesion and Hill Sachs lesion.
- Radial nerve is commonly injured in the fracture shaft of humerus.

MULTIPLE CHOICE QUESTIONS

1. The commonest type of shoulder dislocation is
   a. Subcoracoid
   b. Subglenoid
   c. Posterior
   d. Subclavicular
2. All are components of rotator cuff except
   a. Supraspinatus
   b. Infraspinatus
   c. Subscapularis
   d. Teres major
3. Treatment of anterior dislocation of shoulder is by
   a. Kocher's manoeuvre
   b. Dennis Browne splint
   c. Barlow's manoeuvre
   d. Surgery
4. Luxatio erecta
   a. Tear of the glenoid labrum
   b. Inferior dislocation of shoulder
   c. Anterior dislocation of shoulder
   d. Defect in the humeral head
5. Bankart's lesion involves
   a. Anterior aspect of the head of humerus
   b. Anterior aspect of glenoid labrum
   c. Posterior aspect of glenoid labrum
   d. Posterior aspect of the head of humerus

CHAPTER 16

Injuries of the Elbow, Forearm and Wrist

ELBOW

Anatomy

The elbow joint is a hinge joint variety made up of three separate joints, namely the humeroulnar joint, humeroradial joint and proximal radioulnar joint.

The distal end of the humerus is flattened forming two processes, namely the lateral epicondyle laterally and the medial epicondyle medially.

Medial to the lateral epicondyle is the capitulum and lateral to the medial epicondyle is the trochlea.

There are three bony points around the elbow, which are important in defining the normal anatomy and any deformity of the elbow joint—the *medial epicondyle*, the *lateral epicondyle* and the *tip of the olecranon* (Fig. 16.1).

Normally in an elbow flexed to 90°, the three bony points make an isosceles triangle. In an extended position of the elbow, these three bony prominences make a straight line. This three-point configuration is altered in various elbow injuries (e.g. the posterior dislocation of the elbow).

Carrying Angle

When the elbow is extended and supinated, the long axis of the arm and that of the forearm form an angle which is called the carrying angle (Fig. 16.2). It disappears on flexing the elbow. This is about 11° in males and 14° in females.

Ossification Around the Elbow

The clinician should be able to differentiate a fracture and normal ossification centres around the elbow. Table 16.1 shows the normal ossification centres around the elbow (Fig. 16.3).

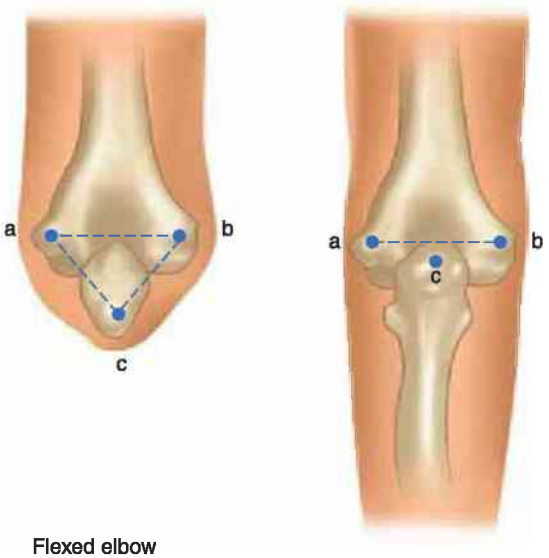


FIGURE 16.1 Illustration showing relationship of bony landmarks of the elbow seen from behind. (a) Medial epicondyle, (b) lateral epicondyle and (c) tip of the olecranon.

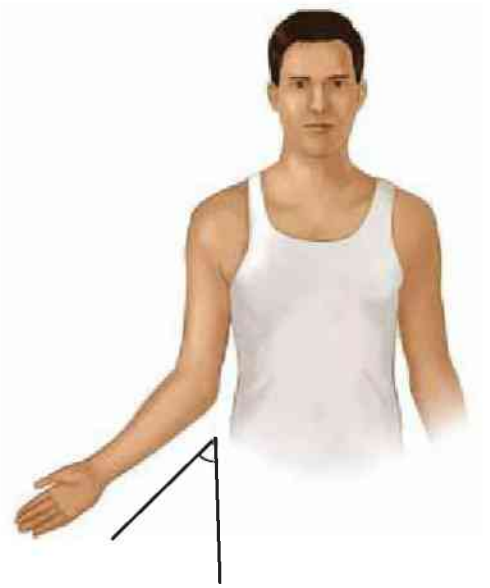


FIGURE 16.2 Illustration showing carrying angle—the angle formed between the long axis of arm and forearm when the elbow is extended and supinated.

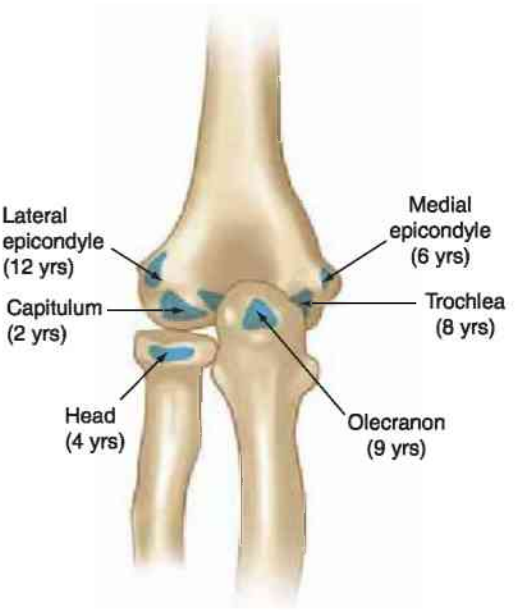


FIGURE 16.3 Illustration showing ossification centres around the elbow.

TABLE 16.1 Appearance of Different Ossification Centres

Ossification Centre	Age of Appearance (Years)
Capitulum	2
Head of radius	4
Medial epicondyle	6
Trochlea	8
Olecranon	9–10
Lateral epicondyle	12

INJURIES OF ELBOW

Elbow injuries are of great importance for the following reasons:

- They are very common in children
- Complications either due to the injury itself or due to bad treatment leading to crippling deformities such as Volkmann's ischaemic contracture and myositis ossificans

The injuries in the elbow region will be described under the following heads:

- Fractures of the distal end of the humerus
- Dislocations of the elbow
- Fractures of the proximal ends of the radius and ulna

Fractures of the Distal End of the Humerus

They include the following:

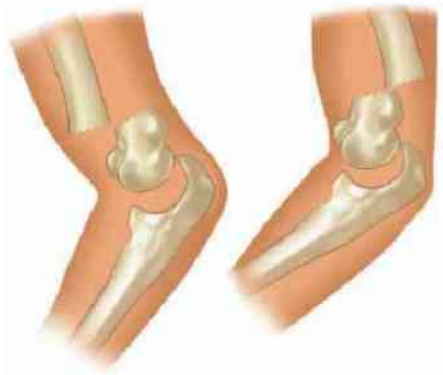
- Supracondylar fracture
- Intercondylar fracture of the humerus
- Fracture of the lateral condyle
- Fracture of the medial epicondyle
- Fracture of the capitulum

Supracondylar Fracture

Supracondylar fracture occurs most commonly in children between the age of 5 and 15 years. It is caused by a fall on the outstretched hand.

Types

- *Extension type:* The vast majority (about 98%) is of this type wherein the distal fragment is displaced backwards (Fig. 16.4).
- *Flexion type:* In this rare type, the distal fragment is displaced forwards.



(a) (b)

FIGURE 16.4 Diagrammatic representation of supracondylar fracture. (a) Extension type and (b) flexion type.

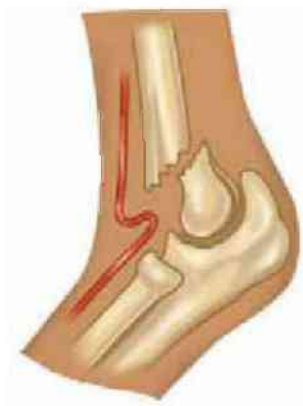


FIGURE 16.5 Illustration showing supracondylar fracture with pressure on the brachial artery.

Clinical Features

The child complains of severe pain in the elbow and holds it in the flexed position. The swelling is tense, filling up the hollows around the elbow and obscuring the bony points. Careful palpation will elicit tenderness in the distal end of the humerus. The fracture occurs just above the level of the condyles and the distal fragment is displaced backwards, upwards and laterally. Movements of the elbow are very painful and restricted.

This should be differentiated from the posterior dislocation of the elbow. As the whole distal end of the humerus carrying the elbow joint is displaced backwards in the supracondylar fracture, the normal triangular relationship between the medial epicondyle, the lateral epicondyle and the olecranon is not disturbed. In the case of posterior dislocation, the relationship is grossly disturbed and the three points may lie in a line.

In this injury, one should always feel the radial pulse to see if there is any pressure on the brachial artery (Fig. 16.5). A weaker radial pulse compared to that on the opposite side demands emergency attention to save the circulation of the forearm. One must also examine for evidence of injury to the median nerve.

Radiological Features

In the anteroposterior view, the fracture line runs transversely just above the condyles of the humerus. The distal fragment is displaced and rotated laterally. The line runs upwards and backwards in the lateral view. The lower fragment is displaced backwards and upwards and tilted posteriorly (Fig. 16.6).

Based on X-rays, Gartland has classified supracondylar fracture as follows:

- *Type I:* Undisplaced
- *Type II:* Partially displaced, but posterior cortex intact
- *Type III:* Complete displacement in both anteroposterior and lateral views

Treatment

Crack fracture without displacement needs only a posterior slab for about 2–3 weeks. Displaced

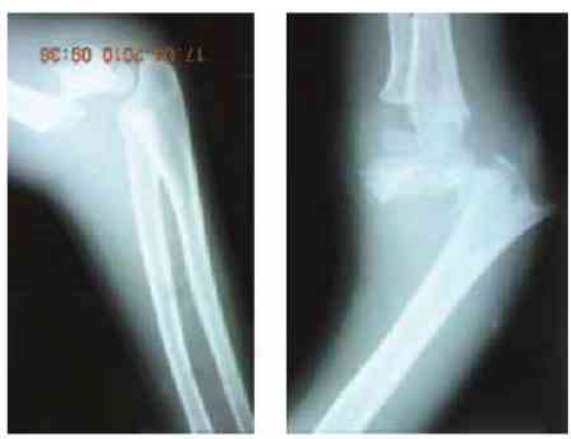


FIGURE 16.6 X ray of the arm—anteroposterior and lateral views—demonstrating supracondylar fracture showing displacement of fragments.

FIGURE 16.7 Clinical photo of the pointing index finger seen in median nerve palsy.

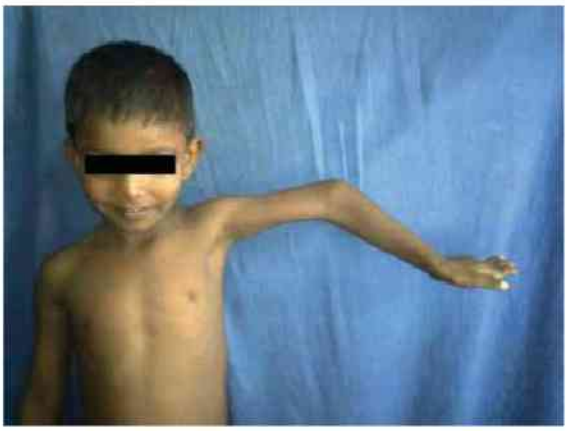


FIGURE 16.8 Clinical photo of gunstock deformity (cubitus varus).

fractures need reduction under general anaesthesia with traction, counter-traction and local pressure. A posterior plaster slab is applied with good padding. If the fragments are unstable, open reduction and internal fixation with K wires are done.

Complications

Early

Complications occur at the time of injury or immediately after the following:

- Injury to the median nerve (Fig. 16.7)
- Injury to the brachial artery (Volkmann's ischaemia): This needs emergency management

Late

- Cubitus varus deformity: The most common complication of the supracondylar fracture is the malunion of the fracture leading to the cubitus varus deformity, otherwise called the gunstock deformity (Fig. 16.8). When cubitus varus is marked, it is corrected by the supracondylar osteotomy of the humerus (French osteotomy)
- Myositis ossificans

Intercondylar Fracture of the Humerus

Intercondylar fracture of the humerus occurs in adults due to a fall on the point of the elbow. According to the shape of the fracture lines, it may be called a T or Y fracture. The fracture line involves the articular surface (Fig. 16.9). Clinically, there will be marked swelling and on palpation the lower end of the humerus is broadened. The fracture is reduced by manipulation under general anaesthesia.

A posterior plaster slab and cuff and collar are applied. As this fracture involves the articular surface, early movements of the joint are encouraged after removing the plaster in 2–3 weeks. In younger patients, gross displacement may require open reduction and internal fixation.

Complications

Elbow Stiffness

Elbow stiffness is a common complication due to intra-articular nature of the fracture. Treatment is done by physiotherapy.

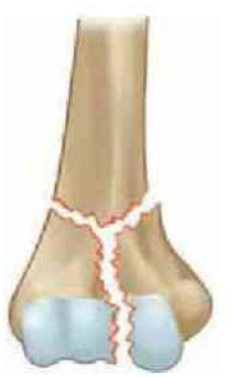


FIGURE 16.9 Illustration showing intercondylar fracture humerus.

Malunion

Malunion is a common complication leading to deformities of elbow, needing corrective osteotomies.

Fracture of the Medial Epicondyle

Fracture of the medial epicondyle is an avulsion fracture caused by a forcible valgus injury to the elbow. It occurs commonly in children and adolescents. This fracture is more common than the fracture of the lateral epicondyle. Usually, the fracture fragment is displaced. The fracture is associated with elbow joint dislocation, and associated ulnar nerve injury may be present.

The valgus violence can produce the following grades of injuries due to the avulsion force of the common flexor origin:

- Sprain or rupture of medial ligament
- Fracture of medial epicondyle with no displacement
- Avulsion fracture with downward displacement (Fig. 16.10)
- Avulsion of medial epicondyle with marked downward displacement and inclusion in the elbow joint (Fig. 16.10)

Clinical Features

Clinically, there will be swelling and tenderness over the medial side of the elbow. One should look for the signs of ulnar nerve paresis.

Radiological Features

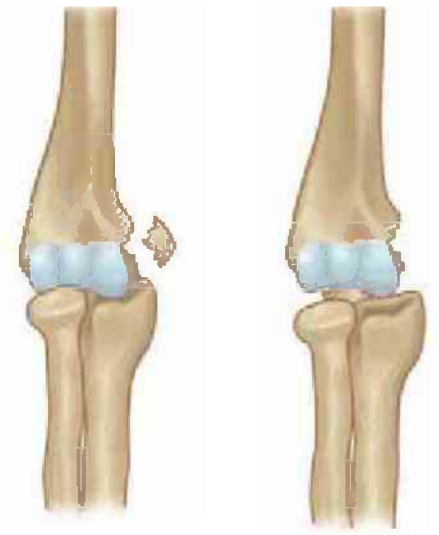
Radiograph will show the avulsed fragment of the epicondyle lying as a loose body. It is always better to radiograph the normal elbow also in children to compare the position of the epiphysis.

Treatment

In the first two grades, immobilisation in the posterior plaster slab for 3 weeks will be sufficient. In Grade III, manipulation is done to reposition the fragment. In Grade IV, manipulative reduction is attempted. In most cases, surgical repositioning will be required (Fig. 16.11).

Fracture of the Lateral Condyle of the Humerus

Fracture of the lateral condyle of the humerus is a rare but important fracture. This fracture occurs in children and is due to various types of violence (Fig. 16.12). The fractured fragment includes the epiphysis of capitellum and the lateral condyle. As the common extensor muscles origin from the



(a) (b)

FIGURE 16.10 Illustration showing fracture of the medial epicondyle of the humerus. (a) Grade III and (b) Grade IV.

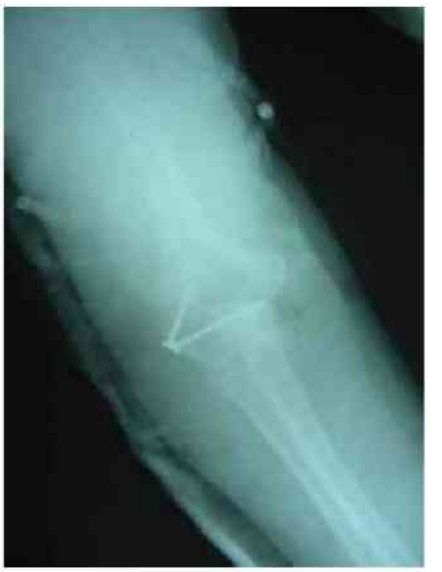
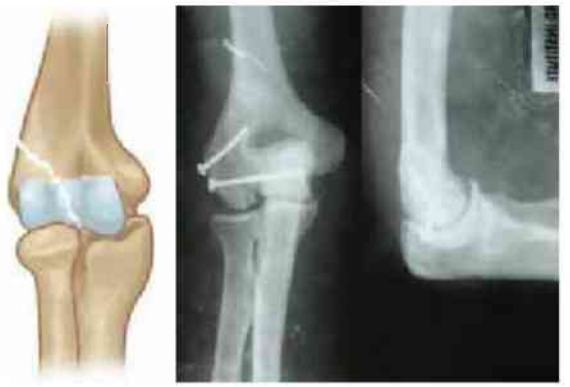


FIGURE 16.11 X-ray of the elbow anteroposterior view depicting fracture medial condyle fixed with screws.



(a) (b)

FIGURE 16.12 (a) Lateral condyle fracture of the humerus: diagrammatic representation. (b) X-ray of the elbow—anteroposterior and lateral views—depicting fracture lateral condyle fixed with screws.

lateral condyle, the fragment is pulled downwards and rotated outwards and backwards. Clinically, in addition to the swelling and tenderness over the lateral aspect, the fractured lateral condyle may be felt to be mobile on palpation. X-ray anteroposterior and lateral views of the affected elbow will show the fracture and displacement of the fragment.

Milch has classified these fractures as Types I and II based on the fracture line (Table 16.2). The displacement is also graded into four categories.

Crack fractures are treated by a simple posterior plaster slab. Displaced fractures are best treated by surgical fixation.

TABLE 16.2 Types of Fractures, Milch Classification.

Type	Extent of Fracture Line	Condition of Elbow
Type I	The fracture line extends lateral to the trochlea and into the capitulotrochear groove. This is less common.	The elbow is stable.
Type II	The fracture line extends into the apex of the trochlea. This is more common.	The elbow is unstable.

Treatment

- For undisplaced fractures, closed reduction and above-elbow slab for 3 weeks are needed.
- For displaced fractures which are more common, open reduction and internal fixation with a 'K' wire are needed (Fig. 16.12b).

Complications

Non-Union

This is very common as the fragment is pulled down and rotated. These cases often present later with a non-tender mobile bone mass on the lateral side after conservative or indigenous treatment. These are treated by surgical reduction and fixation.

Cubitus Valgus Deformity

This is due to the damage and arrested growth of the lateral part of the epiphyseal plate. The valgus deformity is progressive and later results in stretching of the ulnar nerve and its paralysis (Tardy ulnar paralysis). This will need surgical anterior transposition of the nerve. Severe deformity will need corrective osteotomy operation.

Fracture Capitellum

Fracture capitellum is a rare fracture wherein the anterior segment of the capitellum is fractured in the coronal plane and gets displaced upwards. This is caused by a direct force transmitted from the radius to the capitellum by a fall. A lateral view radiograph shows the segment of the capitellum lying displaced upwards in front of the elbow joint. Most of these cases will require surgical repositioning and fixation.

Dislocation of the Elbow Joint

Dislocation of the elbow joint is a common injury of young adults and is caused by a fall on the outstretched hand. There are two common types of dislocation:

1. *Posterior dislocation:* In this type, the proximal end of the ulna and radius are displaced

posteriorly. This is the most common type. The posterior dislocation is associated with fractures of the radial head, the coronoid process of the ulna and the medial epicondyle.

2. *Posterolateral dislocation:* In this type which is common, in addition to the posterior displacement, the radius and ulna are displaced laterally. This is often associated with the rupture of the medial ligament of the elbow, or avulsion of the medial epicondyle of the humerus and stretching and paralysis of the ulnar nerve.

Other types include the following:

1. Posteromedial (Fig. 16.13)
2. Divergent
3. Anterior

Clinical Features

The elbow is swollen but one can detect the prominence of the olecranon posteriorly with a depression just above it due to the forward displacement of the distal end of the humerus. One must always look for vascular and nerve injuries in all these cases. This injury is to be differentiated from the supracondylar fracture. In dislocation, the relative positions of the two epicondyles of the humerus and the tip of the olecranon are altered, whereas in the supracondylar fracture the relative positions are not altered (Fig. 16.1).

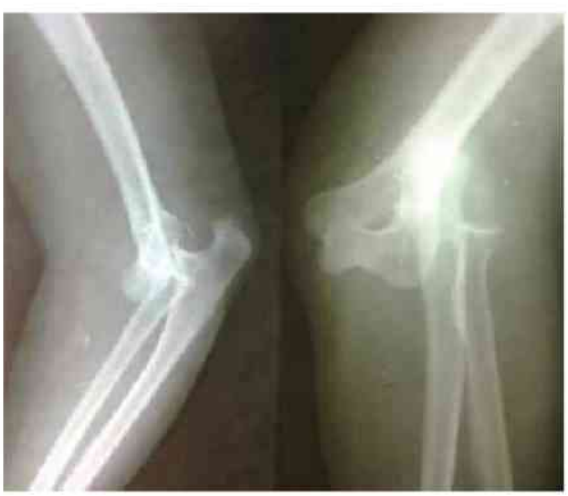


FIGURE 16.13 X-ray of the elbow—lateral and anteroposterior views—showing posteromedial dislocation of the elbow joint.

Diagnosis

The injured elbow is swollen and tender. The three-point relationship is altered. The diagnosis is confirmed by X-rays.

Radiological Features

The anteroposterior and lateral views of the elbow must be taken to confirm the type of dislocation. It also shows whether there are any associated fractures of the epicondyles or head of the radius or the coronoid process.

Treatment

Reduction is done under general anaesthesia. After reduction, a padded posterior plaster slab is applied with the elbow in a safe degree of flexion.

Complications

An immediate complication could be injury to the brachial artery or the median and ulnar nerves. There could also be fractures of the medial epicondyle or the coronoid process. Later complications could be myositis ossificans and joint stiffness.

Pulled Elbow

Pulled elbow is a traumatic subluxation of the radial head in children between 2 and 6 years of age. The injury is produced by a jerk on the forearm when the child is lifted while at play by parents or relatives.

The child presents with pain in the elbow and inability to use the whole upper limb and tenderness proximal end of the radius. The radiograph will not reveal any fracture. Awareness of this condition enables the diagnosis. Treatment is done by simple manipulation of the forearm into supination with the elbow stabilised. There is a palpable click, pain disappears and the normal movement is restored immediately.

Fractures of the Proximal End of the Radius

The following fractures occur at the proximal end of the radius:

- Fracture of the head of the radius
- Fracture of the neck of the radius
- Epiphyseal separation in children

Mechanism

These fractures are produced by forcible valgus strain which forces the head of the radius against the capitellum and causes a fracture of one or both. This is common in adults. Some small fragments can enter the elbow joint and may form loose bodies.

Classification

Mason has classified the radial head fractures as Type I—undisplaced, Type II—marginal fracture with displacement, Type III—comminuted fracture and Type IV—fracture associated with elbow dislocation (Fig. 16.14).

Clinical Features

With a history of fall on the outstretched hand, the patient complains of pain in the outer aspect of the elbow. There is tenderness over the head of the radius and swelling due to haemarthrosis. Supination and pronation are painful and restricted.

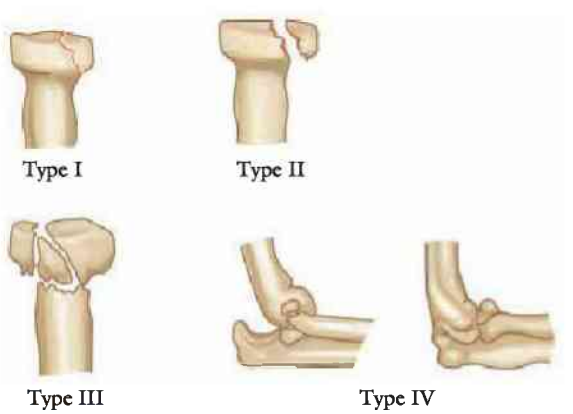


FIGURE 16.14 Illustration depicting Mason's classification of fracture head of the radius.

Radiological Features

Radiological features are of the following heads:

- Crack fracture of the head
- Fracture of the head with displacement of a segment
- Comminuted fracture of the head
- Crack fracture neck of the radius
- Fracture neck of the radius with tilting of the head
- Epiphyseal separation of the upper end of the radius in children

Treatment

Simple cracks in the head or neck without displacement or tilt are best treated with non-operative above-elbow slab immobilisation for 3 weeks. If the swelling is marked, aspiration may be done.

Other types, with comminution or gross tilt, must be treated by excision of the head. Recently, surgical replacement by silastic prosthesis of the head is being done after excision with good results.

In children with epiphyseal separation, excision should not be done as it will later on result in progressive cubitus valgus due to lack of epiphyseal growth and tardy ulnar palsy. If the tilt is marked, it is treated by surgical repositioning.

Complications

Myositis ossificans is a common complication.

Fracture of the Neck of the Radius

- This is common in children
- Results from a valgus injury
- Diagnosis confirmed by taking an X-ray (Fig. 16.15)
- Usually non-operative treatment in the form of above-elbow slab is needed for 3 weeks
- Malunion can occur

Fractures of the Proximal End of the Ulna (Olecranon Process)

Fracture olecranon occurs due to a fall on the flexed elbow. The olecranon is fractured usually near the base. The fragment gets pulled up by the insertion of the triceps tendon.

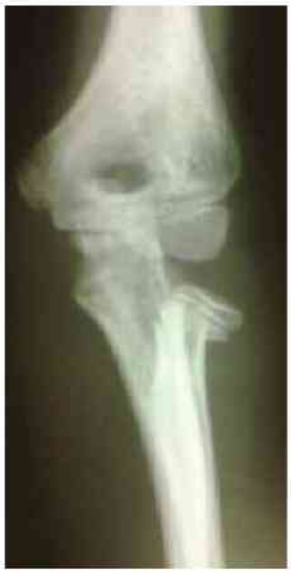


FIGURE 16.15 X-ray of the elbow—anteroposterior view—demonstrating fracture of the neck of the radius.

There is usually some abrasion in the skin over the olecranon. As the fracture line involves the articular surface of the bone, there is haemarthrosis of the elbow joint. The radiograph shows the level of fracture and the amount of separation of the fragment.

Treatment

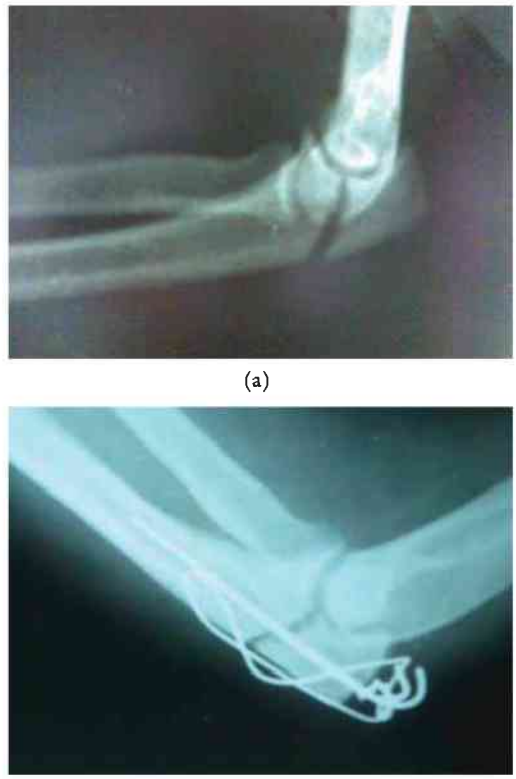
Undisplaced Fractures

Immobilisation of elbow in an above-elbow plaster cast is done with an angle of 20–30° of flexion. This position avoids the displacement of the fragment by the pull of the triceps muscle. After 3 weeks, mobilisation is started.

Displaced Fractures

Open reduction and internal fixation by tension band wiring are done. Early mobilisation is started.

It may be possible to get the fragment into good position by immobilising the elbow in full extension if the displacement is minimal. However, this is a fracture that usually needs open reduction. The olecranon is fixed to the main fragment by wiring it in position by tension band wiring (Fig. 16.16) or fixing it with a single lag screw.



(a)

(b)

FIGURE 16.16 (a) Fracture olecranon and (b) tension band wiring done.

FOREARM

Anatomy

The ulna which is relatively straight acts as the central axis around which the radius, which is laterally bowed, rotates during supination and pronation.

The *supinators* of the forearm (supinator and biceps) are attached to the proximal third of the radius.

The *pronators* of the forearm, namely pronator teres and pronator quadratus, are attached to the middle and lower third, respectively.

Hence in the fracture involving the proximal third of the bones of the forearm where only the supinators are attached the proximal part is *supinated* and the distal forearm is *pronated*, whereas in fractures involving the middle third, the proximal and distal fragments may be *pronated*. This is helpful in reducing the fractures of the forearm bones.

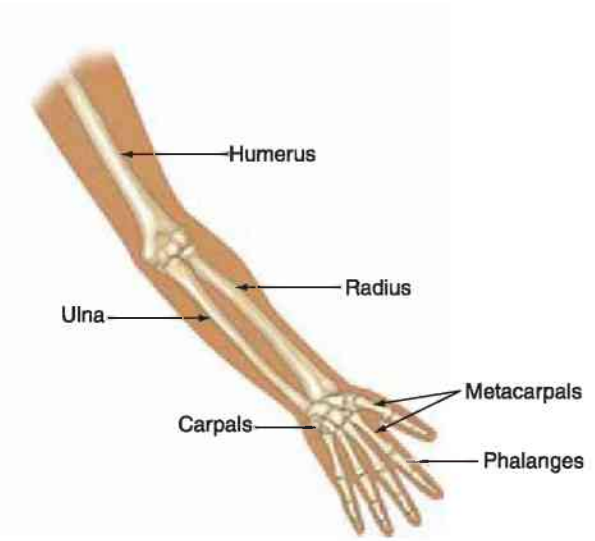


FIGURE 16.17 Illustration depicting the anatomy of forearm bones.

The radius and ulna articulate with each other both proximally and distally (Fig. 16.17).

The proximal radioulnar joint is stabilised by the annular ligament. The distal radioulnar joint is stabilised by the dorsal and volar radioulnar ligaments and the triangular fibrocartilaginous complex and by the intraosseous membrane in the middle.

Hence if there is shortening or displacement of any bone of the forearm due to fracture, it leads to dissociation of the proximal or distal radioulnar joint.

In this group, the following are included:

- Fracture of the radius and ulna
- Fracture of the radius alone
- Fracture of the ulna alone
- Fracture of the proximal third of the ulna with a dislocation of the head of the radius (*Monteggia fracture dislocation*)
- Fracture of the distal third of the radius with a dislocation of the inferior radioulnar joint (*Galeazzi fracture dislocation*)

Fracture of Both Bones of the Forearm

The radius and ulna are fractured either by a fall on the outstretched hand or by direct injury. In adults, fracture of the both bones of the forearm is common. In children, the greenstick type of fracture is very common.

In the forearm, when one of the bones is fractured and displaced, the other is also usually fractured. If only one bone shows a fracture with displacement and the other shaft is intact, one must expect a displacement at either the superior or the inferior radioulnar joint.

The axis of rotation of the forearm is the line joining the superior and inferior radioulnar joints. The *restoration of the interosseous space* by proper correction of over-riding, angulation and rotation is very important in the management of this fracture. If this is not done, there will be restriction of pronation and supination.

Clinical Features

This fracture is notorious for displacement. This displacement is due to various muscle attachments to the radial and ulnar shafts as explained previously. This is common in young adults due to a fall on the hand. The diagnosis is obvious by the deformity, swelling and abnormal mobility. In the forearm, injuries always palpate the superior and inferior radioulnar joints for evidence of injury. One should always look for evidence of vascular injury and injury to the nerves. In case of gross angulation, one fragment of the ulna often penetrates the skin causing a punctured wound making it an open fracture.

Radiological Features

Anteroposterior and lateral views of the whole forearm including the elbow and wrist joints must be taken. A radiograph will show the level of the fractures, the amount of over-riding and rotation of the fragments (Fig. 16.18). The rotation will be shown by the alteration in the shape of interosseous space.

Treatment

Non-Operative

The *undisplaced* fracture which is very rare may be treated with an above-elbow cast with elbow in 90° flexion.

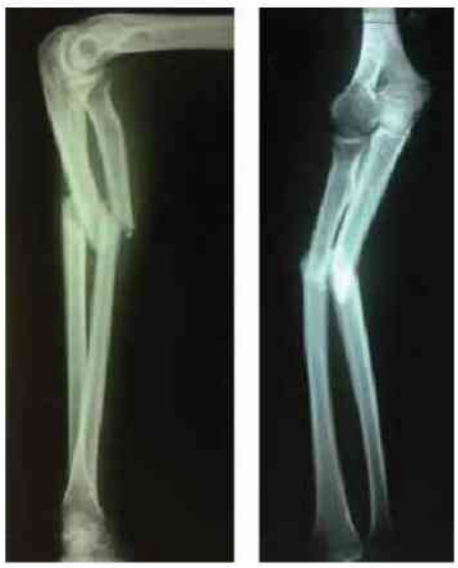


FIGURE 16.18 X-ray of the forearm—lateral and anteroposterior views—showing fracture of both bones of the forearm.

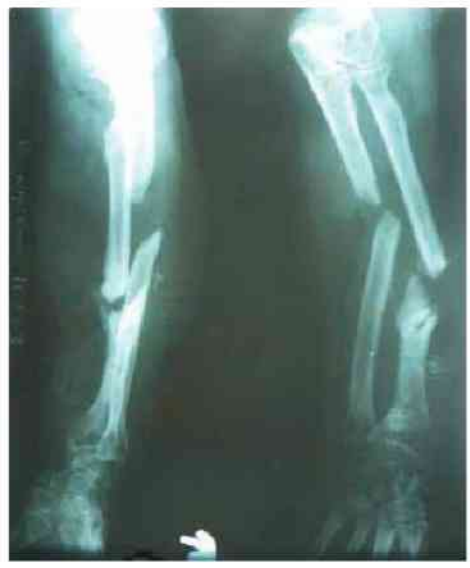


FIGURE 16.19 X-ray of the forearm—anteroposterior and lateral views—showing non-union fracture of the both bones of the forearm.

Frequent follow-ups are needed to recognise early loss of fracture reduction which is common in cases treated conservatively.

Closed Reduction and Plaster Immobilisation

Displaced fractures of the radius and ulna need manipulative reduction. The fragments are hitched in a stable position and an above-elbow plaster slab is applied with good padding in front of the elbow. Check radiographs are taken to confirm the alignment.

Open Reduction and Internal Fixation

Closed reduction may fail due to the interposition of soft tissues or instability of the reduction. In adults, most of these fractures are displaced and need open reduction and internal fixation with dynamic compression plates and screws.

The radius is approached through a volar approach and the ulna through a dorsal approach. Bone grafting may be needed in old cases.

Complications

Volkmann's Ischaemic Contracture

This is often due to tight bandaging or due to the use of unpadded plaster casts as primary treatment. This ultimately results in ischaemia of the forearm muscles.

Non-Union

This is usually due to interposition of the pronator teres tendon fibres through the site of the radial fracture. This is treated by open reduction and internal fixation supplemented by cancellous bone graft chips. Non-union of the fracture segments results from inadequate immobilisation (Fig. 16.19).

Malunion

Union of the fragments with angulation is very common. This causes a limitation of pronation and supination depending on the degree of malunion. If the deformity is marked, surgical corrective osteotomy will be required. Here, the bones are united in an unacceptable position with deformity and limited supination and pronation. Treatment is done by open reduction and internal fixation with plates and screws with bone grafting.

Cross-Union

Cross-union occurs when the fractures are comminuted and at the same level. It occurs also when too many manipulations are done to reduce the fracture. This leads to dispersal of the

fracture haematoma and cross-union. If cross-union occurs, radius unites with ulna and ulna units with radius or both are joined by a callus.

Here the forearm movement is restricted. Treatment needs open reduction, realignment and internal fixation if the united position is unacceptable.

Fractures of the Radius and Ulna in Children

Fractures of the radius and ulna are very common in children due to a fall on the hand. Most often, they are greenstick fractures with angulation only.

Clinical Features

Clinically, there is a deformity in the forearm with localised tenderness at the fracture site. A radiograph shows the level of the fractures and the degree of angulation.

Treatment

Under general anaesthesia, traction and counter-traction are applied and the angulation is corrected by pressing the forearm flat over the level surface of the table and plaster slab applied. After a few days, the swelling subsides and a complete above-elbow plaster cast is given for 4 weeks.

Fractures of the Radius

Fractures of the radius alone without displacement are rare and are immobilised in an above-elbow posterior slab for 3–6 weeks till the union is sound.

Fractures of the Ulna

Fracture of the ulna in the lower one-third is common in adults due to direct injury, when a person tries to ward off a blow on his head with his forearm. Hence, it is known as *nightstick fracture* (Fig. 16.20). Fractures of the ulna alone, without displacement, are immobilised in an above-elbow posterior slab for 6 weeks. Displaced fractures require open reduction and internal fixation with plates and screws.

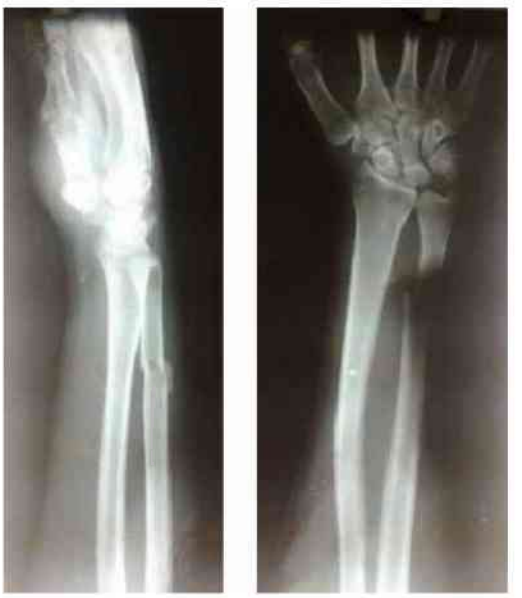


FIGURE 16.20 X-ray of the forearm—lateral and antero-posterior views—showing nightstick fracture.

Monteggia Fracture Dislocation

Monteggia fracture dislocation is a fracture of the proximal third of the ulna with a dislocation of the head of the radius.

It occurs commonly in adults. It is due to a fall associated with forcible pronation of the forearm or a direct violence on the posterior aspect of the proximal forearm.

Classification (by Bado)

- *Type I (Extension type):* Fracture of the ulna diaphysis with an anterior angulation at the fracture site and the anterior dislocation of the radial head (Fig. 16.21)

FIGURE 16.21 Illustration depicting Monteggia fracture dislocation.

- *Type II (Flexion type):* Fracture of the ulna diaphysis with a posterior angulation at the fracture site and the posterior dislocation of the radial head
- *Type III (Adduction type):* Fracture ulna at metaphysis with a lateral dislocation of the radial head
- *Type IV:* Fracture of the proximal third of the radius and ulna at the same level with an anterior dislocation of the radial head

Clinical Features

In the common extension type, the patient presents with pain and swelling in the elbow and the proximal forearm. The radial head is felt in the cubital fossa and there is tenderness in the proximal ulna with depression at the fracture site due to forward angulation. One should look for evidence of posterior interosseous nerve palsy which causes finger and thumb drop.

Radiological Features

Radiology confirms the type of fracture and displacement of the head of the radius (Fig. 16.22).

Treatment

Manipulative reduction under anaesthesia may succeed in realigning the ulna and repositioning the head of the radius. The limb is immobilised in flexion. Where closed reduction fails, open reduction is needed to reduce and internally fix the fracture of the ulna and reduce the dislocated head of the radius.

Complications

Malunion

Malunion results from conservatively treated cases. Treatment includes open reduction and internal fixation for the ulna. If the radial head is not reducing, it may require excision.

Galeazzi Fracture Dislocation

In this injury, there is a fracture of the distal shaft of the radius associated with a dislocation of the ulna. Clinically, there are deformity and tenderness at the fracture site and prominence of the dislocated head of the ulna. A radiograph will show the fracture displacement and dislocation (Fig. 16.23). It is treated by manipulation and plaster with the forearm in supination. As it is an unstable fracture, it needs open reduction and internal fixation.

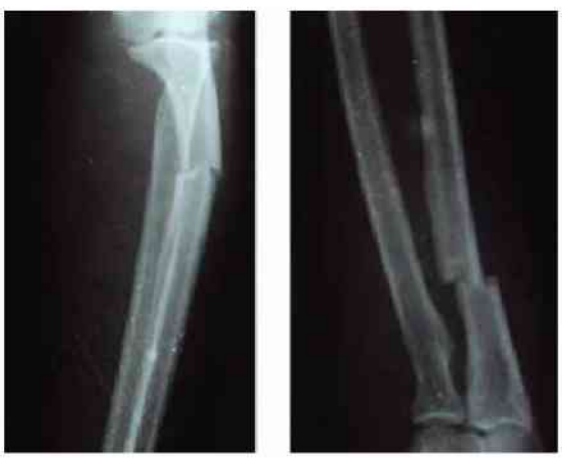


FIGURE 16.22 X-ray of the forearm—lateral and anteroposterior views—showing Monteggia fracture dislocation.

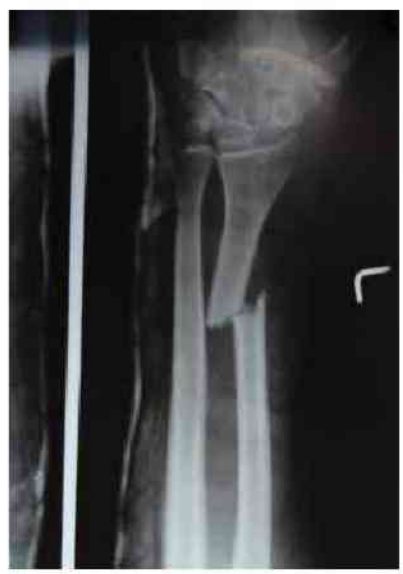


FIGURE 16.23 X-ray of the forearm—anteroposterior view—showing Galeazzi fracture dislocation.

SIDE SWIPE FRACTURE

'Don't keep your elbow out, it may reach home in another car' was the army way of educating military vehicle drivers. This illustrates the mechanism and severity of this tragic injury which often results in loss of the limb and permanent crippling. This injury is caused by the driver or passenger in a car keeping the elbow out through the window which gets hit by an oncoming vehicle.

The injury consists of a comminuted fracture of the lower end of the humerus, the fracture of the upper ends of the radius and ulna and the gross dislocation of the elbow joint (floating elbow). The injury is associated with neurovascular complications endangering the viability of the limb. Treatment is difficult and needs a judicious combination of conservative and operative management to restore useful function to the elbow.

WRIST

It is important to recognise the following injuries around the wrist joint, all of which occur due to a fall on the outstretched hand:

1. Fracture of the lower end of the radius
   - Colles' fracture
   - Fracture separation of lower radial epiphysis
   - Smith's fracture (reverse Colles' fracture)
   - Barton's fracture
2. Fracture of the scaphoid bone
3. Dislocation of the lunate bone

Colles' Fracture

Colles' fracture is a transverse fracture at the corticocancellous junction of the distal radius often associated with a fracture of the ulnar styloid process. It occurs commonly in elderly women due to post-menopausal osteopenia. The mechanism of injury is a fall on the outstretched hand.

Anatomy

The articular surface of the distal radius is biconcave in shape, and it articulates with proximal carpal and with distal end of the ulna. Normally, the radial styloid is about 10 mm distal to the ulnar styloid.

Pathology

Colles' fracture is prone for displacement. Most of the fractures will have displacement. Very few may have undisplaced crack fractures. The displacements are as follows:

- Impaction
- Dorsal shift
- Dorsal tilt
- Radial shift
- Radial tilt
- Supination

Sometimes, the fracture may extend into the articular surface leading to an intra-articular extension.

Clinical Features

The patient presents with pain and swelling near the wrist. The wrist and hand show a typical deformity, called *dinner fork deformity*, due to the dorsal displacement of the lower fragment (Fig. 16.24). On palpation, there is localised tenderness at the fracture site. The tip of the radial styloid is at the same level as or at a higher level than the ulnar styloid. Normally, it is at a lower level. There is a limitation of palmar flexion at the wrist due to the dorsal tilt of the lower fragment.

Radiological Features

Anteroposterior and lateral radiographs are taken (Fig. 16.25). This shows the following displacements—dorsal displacement, dorsal tilt, lateral displacement, lateral tilt and impaction. The lower fragment is sometimes comminuted and the fracture line may run into the articular surface.

Treatment

Most of the distal radius fractures are treated conservatively by a below-elbow cast. If the fracture

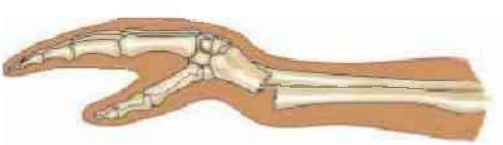


FIGURE 16.24 Illustration of dinner fork deformity in Colles' fracture.

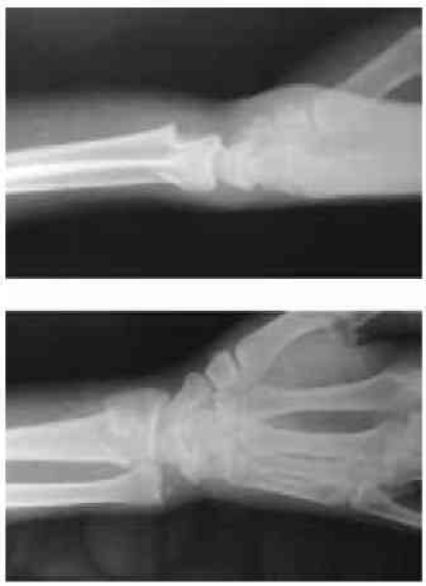


FIGURE 16.25 X-ray of the forearm with wrist—lateral and anteroposterior views—showing Colles' fracture.

is undisplaced, stabilisation in a below-elbow cast for 4–6 weeks is needed.

If the fracture is displaced, closed manipulative reduction under general anaesthesia and below-elbow cast immobilisation for 4–6 weeks are needed.

In closed reduction, the mechanism of reduction is the exact opposite of the displacement.

The technique of reduction includes the following:

- Disimpaction
- Palmar flexion
- Ulnar deviation
- Pronation

Comminuted fractures of the distal radius, in which fracture reduction is unstable, may need percutaneous 'K' wire fixation to prevent displacement, with plaster immobilisation.

Some intra-articular comminuted distal radius fractures can be treated with distal radius plates and screws. In some comminuted fractures, the fragments are distracted by putting an external fixator.

The fracture is reduced by manipulation under general anaesthesia. It is immobilised by a below-elbow plaster slab with the forearm pronated, the wrist in slight palmar flexion and ulnar deviation. Occasionally, external or internal fixation is used to maintain the length and reduction.

Complications

Malunion

Malunion is commonly due to the failure to correct the displacements fully during the manipulation. Malunion can result in dinner fork deformity and limitation in wrist movements, delayed rupture of the extensor tendon due to attrition by the rough dorsal aspect of the fracture line, and carpal tunnel syndrome.

Treatment includes osteotomy and internal fixation only in severe malunion.

Carpal Tunnel Syndrome

The median nerve is compressed in the carpal tunnel by the healing callus and requires decompression.

Distal Radioulnar Joint Subluxation

Due to impaction of the fracture radius resulting in a shortened radius, the distal radioulnar joint subluxates. The wrist and forearm movement becomes painful. In some cases, excision of the lower end of the ulna is done (Darrach's procedure).

Stiffness of Fingers and Wrist

This is due to the failure to exercise the finger joints during the period of immobilisation.

Sudeck's Atrophy

This is a rare complication characterised by severe pain in the hand and wrist, swelling and circulatory disturbance in the hand with oedema, resulting in painful stiffness of all the joints of the hands.

Fracture Separation of the Lower Epiphysis of Radius

This injury in the adolescents corresponds to Colles' fracture of the adults. The nature of violence, deformity and manipulative reduction are all similar. A radiograph shows the dorsal displacement of the epiphysis along with a chip of the radius. Immediate repositioning of the fragment by manipulation is essential.

Smith's Fracture (Reversed Colles' Fracture)

Smith's fracture is a fracture at the distal end of the radius where the displacement of the distal fragment is the opposite of that in Colles' fracture. This occurs at the same level as Colles' fracture. It is caused by a direct violence at the back of the wrist by a fall on the flexed wrist. The distal fragment is displaced palmarwards with a ventral tilt causing 'garden spade deformity'. After reduction, the wrist is immobilised in a 30° dorsiflexion position with the forearm supinated in an above-elbow plaster.

Barton's Fracture

Barton's fracture is a fracture of the distal end of the radius involving the articular surface (Fig. 16.26). The distal end is split vertically in the coronal plane with the small fragment getting displaced along with the wrist dorsalwards or palmarwards. There are two types of fracture depending upon the displacement—volar Barton's and dorsal Barton's. This is an unstable fracture and often requires surgical treatment.

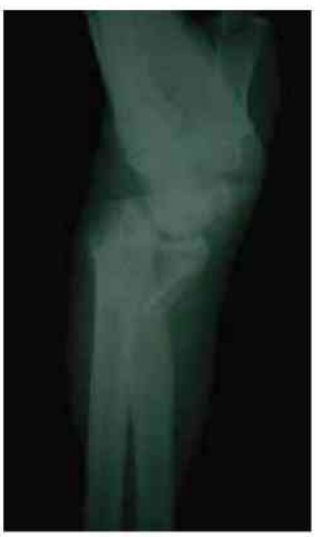


FIGURE 16.26 X-ray of the forearm—lateral view—demonstrating Barton's fracture.

Fracture of the Scaphoid Bone

There are eight carpal bones which are arranged into two rows in the wrist. Of all the carpal bones, the scaphoid is the most commonly fractured. The blood supply to the scaphoid bone enters it through its distal half. Any fracture at the waist of the bone or proximal third cuts off the blood supply to the proximal fragment. This explains the high incidence of avascular necrosis of the proximal fragment and non-union of this fracture.

Clinical Features

This occurs in young adults due to a fall on the outstretched hand. The patient presents with pain in the lateral aspect of the wrist. There is swelling and localised tenderness at the anatomical snuff box (Fig. 11.8), as the bone is normally palpable at that site.

Radiological Feature

Three views, anteroposterior (Fig. 16.27), lateral and oblique, of the wrist must be taken. The types of fracture seen are as follows:

- Proximal pole fracture
- Fracture at the waist of the bone
- Fracture of the tubercle of the scaphoid

Sometimes, the fracture line is not visible in the initial radiograph and the injury is treated as a

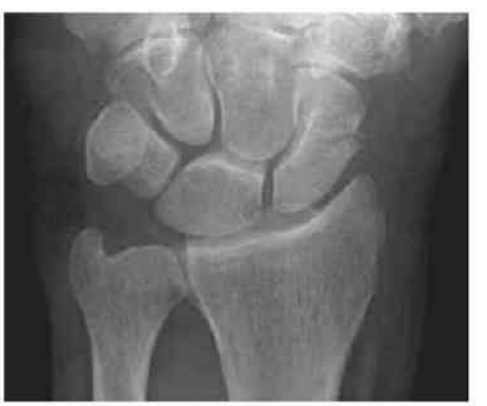


FIGURE 16.27 Scaphoid fracture as seen in the radiograph of wrist—anteroposterior view.

sprain. If the radiograph is repeated 2 weeks later, the fracture line becomes obvious, due to rarefaction along the fracture line.

Treatment

The immobilisation is done in flexion and slight radial deviation.

In displaced fractures, the fracture segment is fixed internally with Herbert's screws. Usually, there is no displacement. Hence, the wrist is immobilised in a close-fitting scaphoid plaster cast for 6 weeks.

Complications

Non-Union

Non-union occurs in fractures of the proximal pole and in a few cases of fracture waist of scaphoid due to inadequate blood supply to the proximal fragment. If the pain is persistent, surgical treatment by bone grafting can be done to promote union.

Avascular Necrosis of the Proximal Fragment

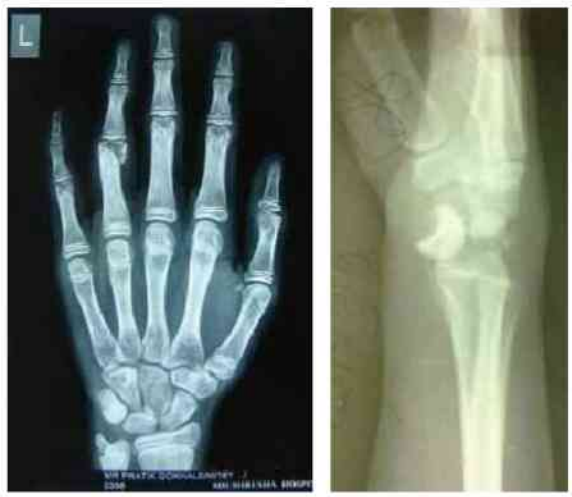
If the pain is severe, the avascular fragment may be excised. More recently, excision of the scaphoid and replacement with silastic prosthesis are seen to give good results.

Dislocation of the Lunate Bone

The lunate is the commonest carpal bone to be dislocated. This occurs due to hyperextension violence to the wrist and the bone gets dislocated palmarwards into the carpal tunnel.

Clinical Features

There is pain and swelling around the wrist. The lunate bone may be palpable under the transverse carpal ligament of the wrist with its concave articular surface facing palmarwards. One should look for the signs of median nerve pressure such as paraesthesia in the thumb, index and middle fingers due to compression of the nerve by the dislocated lunate bone inside the carpal tunnel.



(a) (b)

FIGURE 16.28 X-ray of the hand anteroposterior view. (a) Note the normal arrangement of various carpal bones. Also seen is unicondylar fracture of the proximal phalanx of second digit. (b) X-ray of wrist lateral view showing lunate dislocation.
Courtesy: Dr Anand J Thakur.

Radiograph

The lateral view of the wrist will show the lunate lying anteriorly and the capitate not articulating with the lunate (Fig. 16.28).

Treatment

Manipulative reduction is done under general anaesthesia and the wrist is immobilised in the plaster. If closed reduction fails, open reduction is indicated.

Complication

Carpal tunnel syndrome is the main late complication of an old unreduced lunate. The lunate is excised to relieve the symptom.

KEY POINTS

- Elbow joint is a hinge joint.
- Medial epicondyle, lateral epicondyle and tip of olecranon are three bony points important in defining normal anatomy of elbow.
- Carrying angle is about 11° in males and 14° in femailes.
- Supracondylar fracture occurs in children between 5 and 15 years, caused by fall on outstretched hand. The extension type is most common.
- Triangular relationship is unaltered in supracondylar fracture and it is altered in posterior dislocation of elbow.
- Pulled elbow is traumatic subluxation of the radial head. It is commonly seen in children between the age group of 2 and 6 years.
- Fracture of the olecranon process occurs due to fall on elbow which is in flexed position.
- Colles' fracture is transverse fracture at the corticocancellous junction of distal radius commonly occurring in women due to post-menopausal osteopenia.
- Smith fracture is reverse Colles' fracture in which there are distal radius fractures where the displacement of the distal fragment is opposite of that is Colles.
- Undisplaced fracture of olecranon is treated by plaster immobilisation in 20–30° of flexion for 3 weeks and displaced fractures are treated by tension band wires.
- Ulna acts on the central axis around which the radius rotates during supination and pronation.
- Monteggia fracture dislocation is fracture of the proximal third of ulna with dislocation of the radial head.
- Galeazzi fracture dislocation is fracture of the distal shaft of the radius associated with a dislocation of ulna.

MULTIPLE CHOICE QUESTIONS

1. In supracondylar fracture, the segment is often displaced
a. Laterally
b. Medially
c. Anteriorly
d. Posteriorly

2. The malunion of supracondylar fracture of humerus most commonly leads to
a. Flexion deformity
b. Cubitus varus
c. Cubitus valgus
d. Extension deformity

3. The earliest sign of Volkmann's ischaemic contracture is
a. Pain
b. Numbness
c. Paraesthesia
d. Pallor

4. The most common complication of Colles' fracture is
a. Non-union
b. Stiffness of finger
c. Vascular injury
d. Sudeks dystrophy

5. Night stick fracture is
a. Monteggia fracture
b. Galeazzi fracture
c. Fracture of neck talus
d. Fracture of shaft of ulna

CHAPTER 17

Injuries of the Hand

Industrial injuries are increasing in incidence due to the rapid industrialisation of our country. There has been a new association of Man, Machine and Management with increased industrial productivity on an unprecedented scale. The coming together of a large number of illiterate rural man power and modern machinery has resulted in a high rate of accidental injuries to the hands of the workers.

The hand is one of the most important organs in the human body. It is not only a motor organ of precision but also an essential sensor, organ and a vehicle of emotional expression.

Injuries to the hand may involve not only the bones and joints but also other structures such as skin, tendons, nerves and vessels. The aim of treatment should be to restore the hand as a functioning organ rather than just to unite a fracture of the metacarpal bone or phalanx. A badly treated cut, laceration or fracture of the phalanx of one finger can result in a useless hand with stiffness of all the joints of all the fingers. It is essential that special hand injury clinics are organised in hospitals near industrial areas. One of such clinics organised by Professor Venkataswamy at the Stanley Hospital, Chennai, is a good model for all teaching hospitals.

Classification

The injuries of the hand can be classified as follows:

1. *Closed injuries:* These can be further categorised as follows:
   - Fracture of the metacarpal bones
   - Fracture of the phalanges
   - Dislocation of the metacarpophalangeal joints
   - Bennett's fracture dislocation
2. *Open injuries:* These can be
   - cuts and lacerations,
   - crush injuries of the hand,
   - traumatic amputation.

CLOSED INJURIES

The closed injuries of hand can involve the fractures of metacarpal bones or phalanges. These injuries may also cause dislocation of metacarpal joints.

Fracture of the Metacarpal Bones

The metacarpal bones can get fractured at any level—head, neck, shaft and base (Fig. 17.1). These are mostly due to direct violence and the shafts are the common sites of the fractures. The injured metacarpal bones are normally well splinted by the neighbouring bone as well as by the interosseous muscles, and hence the fragments do not displace markedly.

Undisplaced Fractures

If there is swelling of the hand, it should be splinted in a dorsal plaster slab till the oedema subsides. After about a week, the plaster is removed and a strapping applied to the hand across the palm

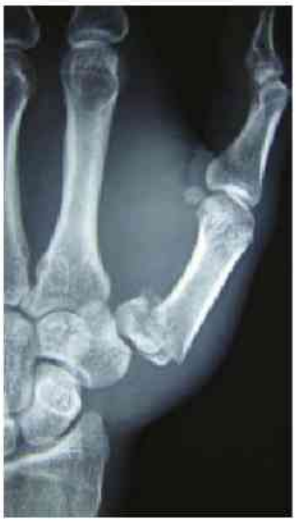


FIGURE 17.1 X-ray of hand anteroposterior view demonstrating fracture base of the first metacarpal. *Courtesy:* Dr Anand J Thakur.

and dorsum leaving the fingers and thumb free to move. Early movements should be encouraged.

Displaced Fractures

Transverse fractures of metacarpal shafts may show displacement of the fragments with overriding. Such fractures are reduced by manipulation under anaesthesia and subsequent treatment is the same as above.

Indication for Surgery

Multiple shaft fractures with displacement will need open reduction and internal fixation. Fracture of the shaft with moderate angulation or fracture neck of metacarpal bone with marked displacement will need surgical reduction and internal fixation.

Rolando's Fracture

Rolando's fracture is a comminuted intra-articular fracture through the base of the first metacarpal bone. It consists of a T or a Y configuration. Surgery is the treatment of choice which may be closed reduction and K-wire fixation or open reduction and fixation with plates and screws. Severe comminution may need external fixation.

Fractures of the Phalanges

Phalanges are fractured either due to direct trauma or due to twisting injuries. However, the distal phalanges are fractured more often due to crush injuries. These fractures may be either displaced or undisplaced (Fig. 17.2).

Crack Fractures of the Phalanges

Crack fractures may be either oblique or transverse and are stable. They are treated by strapping the injured finger to the adjacent finger.

Displaced Transverse Fractures

Displaced transverse fractures need manipulative reduction under anaesthesia. The fragments are hitched in stable position and the finger is immobilised in flexion. After 2 weeks in this position, the finger is released and strapped to the neighbouring finger and joint movements encouraged. Displaced fractures are treated by a Kirschner wire or a mini screw.

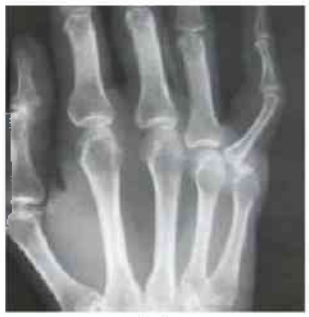
Dislocation of the Metacarpophalangeal Joint

The metacarpophalangeal joint of the index, finger and thumb is the most common joint to be dislocated. This is due to a hyperextension injury to the finger and the proximal phalanx gets displaced dorsally. Clinically, the head of the metacarpal bone is felt as a prominence in the palmar aspect. This is easily reduced if it is fresh, by manipulation under anaesthesia. After reduction, the finger is flexed and immobilised for 2 weeks till the capsule heals (Fig. 17.3).

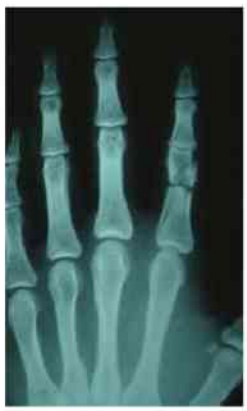
Occasionally, reduction is not possible due to the head of the metacarpal bone button holing the volar capsule of the joint. In such cases, surgery (Kaplan's procedure) is necessary to reduce the dislocation.

Bennett's Fracture Dislocation

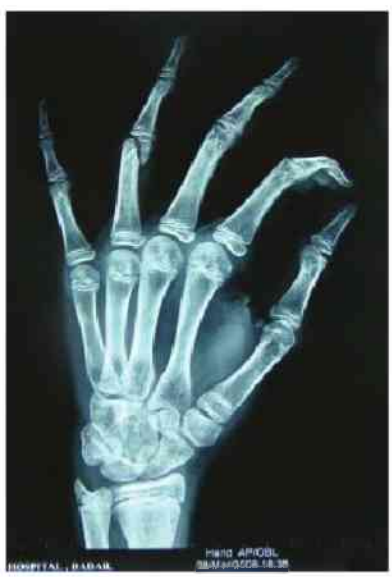
Bennett's fracture is a fracture of the base of the metacarpal bone of the thumb involving the articular surface with a dislocation of the carpometacarpal joint (Fig. 17.4). This is caused by a direct blow at the site during boxing by hitting with a



(a)



(b)



(c)

FIGURE 17.2 X-ray of hand anteroposterior view showing (a) fracture base of fifth finger proximal phalanx, (b) comminuted fracture shaft of second finger proximal phalanx and (c) fracture head of fourth proximal phalanx. *Courtesy:* Dr Anand J Thakur.

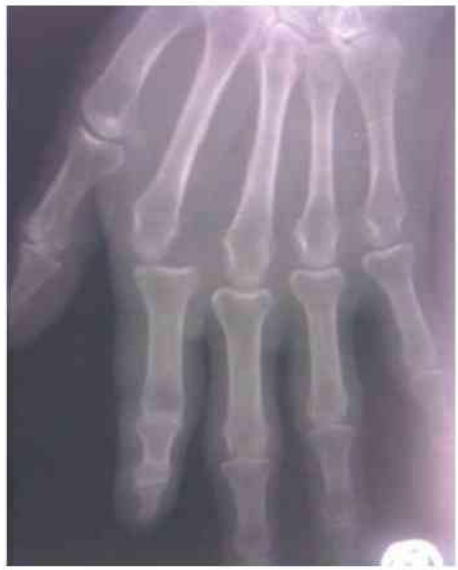


FIGURE 17.3 X-ray of hand anteroposterior view showing second metacarpophalangeal joint subluxation.

closed fist. Clinically, there is swelling over the base of the thumb and a step may be palpable at the joint level.

Radiologically, the extent of the subluxation and the line of the fracture are seen.

Treatment

It is an intra-articular fracture and accurate reduction is important. The fracture is reduced by

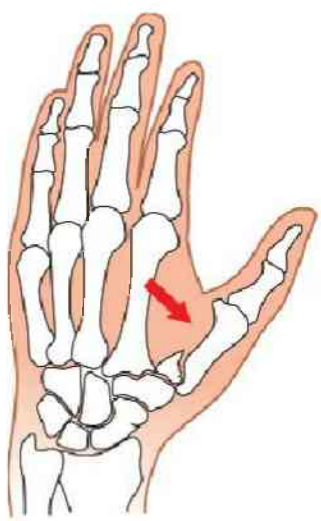


FIGURE 17.4 Illustration of Bennett's fracture dislocation.

traction on the thumb and local pressure. A below-elbow plaster is applied to the interphalangeal joint with the thumb held in extension. When closed reduction fails, open reduction and fixation with a Kirschner wire are done.

Closed reduction and PoP immobilisation may occasionally be successful but since it is an unstable fracture, it often requires internal fixation by a Kirschner wire or a screw.

Open Injuries

Open injuries have to be dealt with as surgical emergencies. They are classified for purposes of treatment as follows:

- Tidy injuries
- Untidy injuries (Table 17.1)

Management

The following are the principles of management of the open injuries of the hand.

1. Prompt first aid: It should be done at home or at the factory floor itself consisting of cleaning with running water and covering with gauze or clean cloth. The hand should be elevated.
2. Efficient primary surgical care
   - Thorough wound cleaning in sterile water to flush out all dirt, grease and foreign bodies
   - Wound debridement: A meticulous wound debridement is done under anaesthesia
   - Stabilisation of bone injuries by minimal internal fixation
   - Nerve injuries are repaired primarily in a clean-cut wound but not in a lacerated untidy wound
   - Early primary skin cover is done unless otherwise contraindicated
   - Prevention of oedema of fingers and hand by compression bandage and elevation
   - Efficient splinting of the injured hand in position of function and active exercises to all fingers
3. Secondary reparative care by reconstructive surgeons.
4. Efficient physiotherapy: Early mobilisation is the watchword in the rehabilitation of the hand.

TABLE 17.1 Differences Between Tidy and Untidy Injuries

Tidy Injuries	Untidy Injuries
Generally caused by sharp weapons	Generally caused by blunt and crushing weapons
No skin loss	Varying degrees of skin loss common
Tendons and nerves are invariably cut	Tendons and nerves are rarely injured
Bones not commonly damaged	Bones are invariably damaged

Tidy Wounds

These are clean wounds caused by cutters, knives and choppers. Most of the domestic injuries and some of the industrial injuries fall into this group. These should be surgically cleaned and sutured following the general principles laid down above.

Reimplantation

More recently, it has been possible to surgically reimplant cleanly severed fingers or even the whole hand in special centres with facilities for microvascular surgery. Clean-cut injuries with traumatic amputation of a digit can be reattached. This is possible if the amputated digit is taken in a clean condition along with the patient to hand surgery centres within 6 hours.

Proper preservation of the amputated digit is more important. The amputated part is wrapped in a clean sterile gauze and kept inside a plastic bag and closed. This is put into another bag containing ice cubes. This procedure is called dry cooling.

Untidy Injuries

These are classified as follows:

1. *Crush injuries without skin loss:* Such crush injuries need careful surgical debridement, trimming of the cut skin edges and attempt at primary closure by turning suitable flaps. Fractures and dislocations must be reduced and even internally fixed, but no repair of tendons

or nerves should be attempted. Tendon injuries are dealt with when the skin healing is satisfactory and when the fractures have united.

2. *Crush injuries with skin loss*
   a. *Single finger:* In cases of crush injuries of the finger, every attempt must be made to save the finger. Primary amputation at the site of election should not be performed as a routine, in the hand. In case of the thumb, every bit of it must be saved as even a stiff remnant of a thumb will be most useful to function as a stump with any remaining finger in the hand.

      Primary tendon or nerve repair in fingers should be done only in the following conditions by specially trained surgeons:
      - Injury caused by clean sharp instruments
      - No skin loss
      - Preferably within 6–8 hours of injury
      - No bone damage

   b. *Whole hand:* Such cases need thorough surgical debridement followed by primary skin cover. When infection is controlled and the oedema has subsided, reconstructive procedures are undertaken.

TENDON INJURIES

Tendon injuries in the hand are most disabling and hence need all the care in the primary treatment as well as at the secondary reconstructive management.

Flexor Tendon Injuries

The palmar aspect of hand is divided into five zones for purposes of treatment of cut flexor tendons (Fig. 17.5):

1. *Zone I:* From the base of the distal phalanx to the middle of the middle phalanx: contains only one tendon flexor digitorum profundus (FDP)
2. *Zone II:* From the middle of the middle phalanx to the distal palmar crease: contains two tendons, FDP and flexor digitorum superficialis (FDS)
3. *Zone III:* From the distal palmar crease to the distal margin of the flexor retinaculum

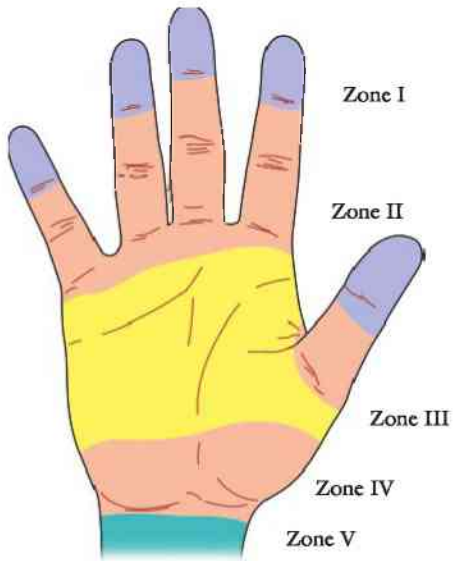


FIGURE 17.5 Illustration of hand showing various flexor tendon zones.

4. *Zone IV:* Flexor retinaculum—carpal tunnel
5. *Zone V:* Distal forearm—proximal to the wrist crease

Diagnosis

Tendon injuries are commonly caused by cuts, sharp instruments or broken glass. Clinically, when a patient presents with a cut in the flexor aspect of the finger, simple tests must be done to identify the tendon injury. Active flexion at the distal interphalangeal (DIP) joint, proximal interphalangeal (PIP) joint and metacarpophalangeal (MCP) joints are tested for identifying the cut tendons and their level.

Treatment

Tidy wounds by sharp weapons in Zones I, III and V seen within 6–12 hours with no skin loss are treated by primary suture. Injury of FDP alone does not require repair. When both tendons are cut, repair of FDP alone is done because repairing both tendons within the flexor tendon sheath leads to adhesion and loss of movements. In injuries in Zone II, occasionally both the tendons are excised and a *free tendon graft* is done to replace only the FDP, extending from the distal crease of the palm to the base of the distal phalanx.

Extensor Tendon Injuries

The following injuries will be discussed in this category:

- Mallet finger
- Injury at the PIP joint level
- Injury at the dorsum of the hand

Mallet Finger

Mallet finger is a flexion deformity of the DIP joint of the finger following injury to extensor digitorum tendon at the level of the distal phalanx, often with a chip of bone (Fig. 17.6). It results by the direct hit of a ball at the tip of the finger or a sudden flexion strain. The unopposed action of the flexor digitorum profundus tendon keeps the terminal phalanx in flexed position. Active extension at the DIP joint is not possible (Fig. 17.7).

Immediate treatment consists of immobilising the finger in a mallet finger splint with the PIP joint in flexion and the DIP joint in hyperextension. In open injuries, the tendon is resutured to the base of the distal phalanx.

Injury at the PIP Joint Level

When the central slip is cut or ruptured, the extension power to the PIP joint is lost. Hence, this joint gets flexed. The DIP joint is held in hyperextension. This is called the *Boutonniere* deformity. If the injury is detected early, repair of the central slip gives good results. Boutonniere (buttonhole) deformity also occurs in rheumatoid arthritis of the PIP joint.

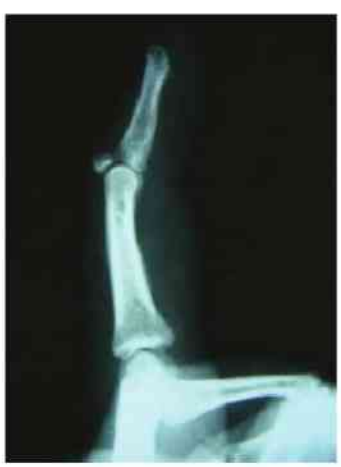


FIGURE 17.6 Radiograph of mallet finger.
Courtesy: Dr Anand J Thakur.

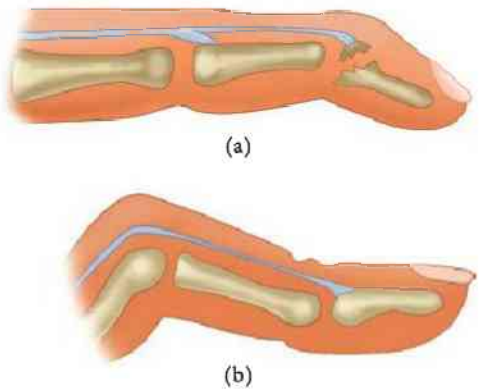


FIGURE 17.7. (a) Mallet finger with chip avulsion and (b) position of reduction.

Injury at the Dorsum of the Hand

In this area, the cut extensor tendons can be primarily repaired and the results are usually good. This could be done even in the presence of fractures in the metacarpal bones.

Game Keeper Thumb or Skier's Thumb

Game keeper thumb or skier's thumb is due to forced abduction of the thumb at the MCP joint leading to injury to the ulnar collateral ligament. In complete rupture of an ulnar collateral ligament, the adductor aponeurosis is interposed between the contents preventing healing of the ligament. This is known as Stener's lesion. Surgical repair is the treatment of choice.

ROLE OF PHYSIOTHERAPY IN MANAGEMENT OF HAND INJURIES

Early and efficient physiotherapy for hand injuries helps in reducing oedema, mobilising stiff fingers and restoring function. After the physical restoration of the injured hand of a worker, it is essential to provide for total rehabilitation of the worker by restoring him to his original work or providing alternative employment.

KEY POINTS

- Injuries of hand involve bones, joints, skin, tendon, nerves and vessels of the hand.
- The aim of treatment should be restoration of function of the hand.
- Bennett fracture dislocation is fracture of the base of metacarpal bone of the thumb involving the articular surface along with dislocation of the carpometacarpal joint.
- Open injuries are a surgical emergency and classified as tidy and untidy injuries for purpose of treatment.
- Palmar aspect of hand is divided into five zones for purpose of treatment of cut flexor tendons.
- Game keeper thumb or skier's thumb is due to forced abduction of the thumb at the metacarpophalangeal joint. This causes injury to the ulnar collateral ligament.

MULTIPLE CHOICE QUESTIONS

1. Dislocation of which one of the carpal bones can present as median nerve palsy
   a. Scaphoid
   b. Hamate
   c. Lunate
   d. Trapezium
2. Mallet finger is
   a. Avulsion fracture of extensor tendon of distal phalanx
   b. Fracture of distal phalanx
   c. Fracture of middle phalanx
   d. Fracture of proximal phalanx
3. Bennet's fracture is fracture dislocation of base of … metacarpal
   a. Fourth
   b. Third
   c. Second
   d. First
4. Reduction of Bennet's fracture is difficult to keep in position due to the pull of
   a. Abductor pollicis brevis
   b. Abductor pollicis longus
   c. Flexor pollicis longus
   d. Flexor pollicis brevis
5. Dupuytren's contracture of hand commonly starts in
   a. Thumb
   b. Index finger
   c. Middle finger
   d. Ring finger

CHAPTER 18

Injuries of the Hip and Thigh

HIP JOINT

Anatomy

The hip joint is a ball-and-socket joint of synovial variety, between the acetabular socket and femoral head.

Acetabulum is deepened by the attached acetabular labrum, thereby increasing the stability of the joint. The stability of the joint in turn depends on the following factors:

- Bony factors
- Muscular and ligamentous factors
- Congruence of the femoral head with the acetabulum

The normal angle of anteversion of the femoral neck with the shaft is 12° and the normal neck-shaft angle is around 125°.

Vascular Anatomy of the Femoral Head

The profunda femoris artery arising from the femoral artery gives medial circumflex femoral artery and lateral circumflex femoral arteries. The blood supply of the head of the femur is derived from the following:

- Medial and lateral circumflex femoral arteries, which in turn anastomoses and form an extra capsular arterial ring (Fig. 18.1).
- Retinacular vessels branch out from the arterial ring and reach the femoral head.

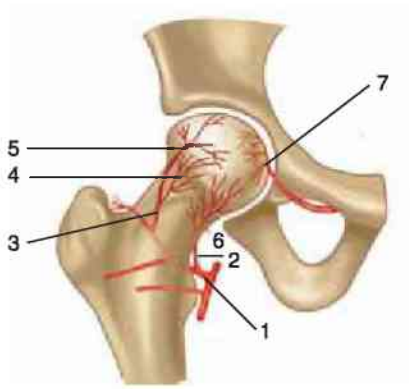


FIGURE 18.1 Vascular supply of the hip joint: (1) medial circumflex femoral artery, (2 and 3) retinacular arteries, (4) superior metaphyseal artery, (5) lateral epiphyseal artery, (6) inferior metaphyseal artery and (7) medial epiphyseal (obturator) artery.

- Foveal artery is a branch of obturator artery, which enters the head via ligamentum teres.

DISLOCATION OF THE HIP JOINT

It needs a very severe violence to cause a dislocation of a normal hip joint. The head of the femur can slip out only through the weak and unsupported inferior aspect of the joint capsule. Thereafter, it moves backwards or forwards depending on the direction of the violence and position of the lower limb at the time of violence. Traumatic dislocation occurs commonly in young adults.

Types

The hip joint can get dislocated in the following ways:

- Posterior
- Anterior
- Central fracture dislocation

Dislocation of the hip can be associated with the following:

- Acetabular fracture
- Femoral head fracture
- Femoral neck fracture

Posterior Dislocation

Mechanism

Posterior dislocation is the commonest type of dislocation. It is caused by violence applied along the femoral shaft when the hip joint is in a flexed and adducted position. It occurs in an automobile accident when the passenger sitting by the driver is thrown forwards and his knee hitting against the dashboard ('dashboard injury').

The position of the hip, in addition, determines the occurrence of associated fractures such as the femoral head fracture and acetabular fractures.

Clinical Features

A young adult is brought with a history of severe injury to the hip and inability to stand or walk.

On examination, the limb is seen to lie in the characteristic position of

- adduction,
- flexion,
- internal rotation,
- marked shortening.

There is pain and swelling around the involved hip joint.

The pulsation of the femoral artery at the mid-inguinal point is not palpable due to the absence of the femoral head in the normal position (vascular sign of Narath).

The greater trochanter is raised and the head of the femur could be felt posteriorly under the gluteal muscles. All movements of the hip are extremely painful.

One must look for the presence of *sciatic nerve paralysis* causing foot drop, as a complication. This may occur following

- stretching of the nerve by the dislocated femoral head,
- by the displaced acetabular fracture.

Radiological Features

- The anteroposterior (AP) view of pelvis to show both hips will demonstrate the head of the femur lying outside and above the acetabulum.
- Shenton's line will show a break in continuity (Fig. 18.2). Shenton's line in the radiograph is normally a continuous line running along the inferior margin of the femoral neck and the upper margin of the obturator foramen.
- The lesser trochanter of the involved limb becomes less prominent (because the limb is internally rotated).
- The femoral head, neck and acetabular fractures should be ruled out.

Occasionally, there may be a complicating fracture of the posterosuperior margin of the acetabulum. This will be visualised in a CT scan.

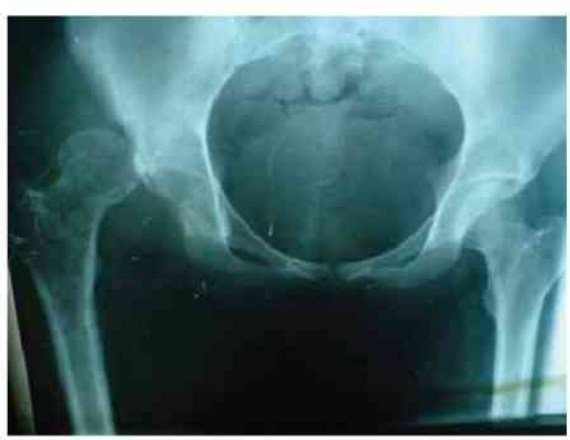


FIGURE 18.2 X-ray of the pelvis with both hips' anteroposterior view showing the posterior dislocation hip. There is a break in Shenton's line (on the right hip).

Treatment

Dislocation of hip is an orthopaedic emergency which should be reduced within 6 hours. Immediate closed manipulative reduction of the dislocation under general anaesthesia or short intravenous sedative is preferable.

The dislocation is reduced by manipulation under general anaesthesia. Reduction is done in one of the following ways:

1. *Allis method*
   - The patient is placed supine over the floor.
   - The surgeon stands on the either side of the patient, flexes the knee and applies longitudinal traction.
   - Assisting person stabilises the pelvis.
   - The leg is then gently rotated internally and externally which helps in reduction.
2. *Bigelow's method:* Steady traction, then external rotation, abduction and extension.
3. *Stimsons gravity method:* Gravity assists the reduction.
4. *Watson–Jones method:* The limb is brought to neutral position and later traction in the line of femur.

After reduction, the leg can be placed in normal position and the length will be found to be equal. The leg is immobilised in a Thomas splint. A check radiograph is taken. It is very important to continue immobilisation for 3–4 weeks to allow for the sound healing of the ruptured capsule, ligaments and other soft tissues. The patient is allowed full weight bearing in 6 weeks.

Surgical Treatment

Open reduction is indicated when there is

- failure of closed reduction,
- associated fractures such as femoral of neck or head.

A large acetabular fragment, if present, will need open reduction and internal fixation with a screw.

Complications

Immediate Complications

- *Associated fracture acetabular rim:* It may cause an unstable reduction thereby requiring open reduction and fixation of the acetabular fragment.
- *Sciatic nerve palsy:* It is due to either stretching of the nerve by the dislocated femoral head or damage caused by a posteriorly displaced acetabular fragment. In most cases (about 70%), spontaneous recovery occurs. Those with complete paralysis initially have a poor prognosis.

Late Complications

- *Avascular necrosis of the head of femur:* A delay in reduction of dislocated hip more than 6 hours leads to an increase in the incidence of avascular necrosis of the femoral head. X-ray findings may be an event within a period of 2 years. The radiograph in these cases shows a localised lesion. This is followed by collapse of the femoral head.
- *Myositis ossificans*
- *Osteoarthrosis:* It is the most common complication following hip dislocation. It occurs due to incongruity of articular surface or following avascular necrosis of the femoral head.
- *Recurrent dislocation:* Recurrence may occur due to the associated fractures of the acetabulum or the femoral head. It may also be due to early mobilisation because of the lax soft tissues.
- *Heterotopic calcification:* It is more common following open reduction of a posterior dislocation. The presenting complaints are pain and stiffness of the hip. This condition is treated by indomethacin and radiotherapy.

Anterior Dislocation of the Hip

Anterior dislocation of the hip is much less common and results from external rotation and abduction of hip. It occurs in collision accidents when a motorcyclist is hit on the medial aspect of the thigh which is in flexion and abduction and external rotation.

Two types are described as follows:

1. *Obturator type:* The limb is in an attitude of flexion, abduction and external rotation (more common).
2. *Iliac type:* The limb is in an attitude of extension, abduction and external rotation.

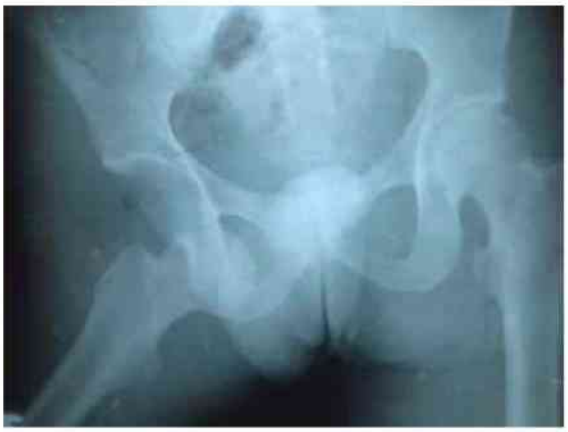


FIGURE 18.3 X-ray of the pelvis with both hips' anteroposterior view showing anterior dislocation. Note the break in Shenton's line and the attitude of the limb on the right side (see text).

The leg lies in an attitude of marked *abduction, flexion* and *external rotation* and will have *true lengthening* (Fig. 18.3). The head of the femur can be felt anteriorly in the medial aspect of the groin.

Reduction is done under general anaesthesia. After good traction, the limb is circumducted through internal rotation and adduction and then extended.

Central Dislocation

Central dislocation is a rare type and is always a fracture dislocation. Due to violent injury on the lateral aspect of the hip, the floor of the acetabulum gives way with multiple fracture lines and the head of the femur is pushed into the pelvis.

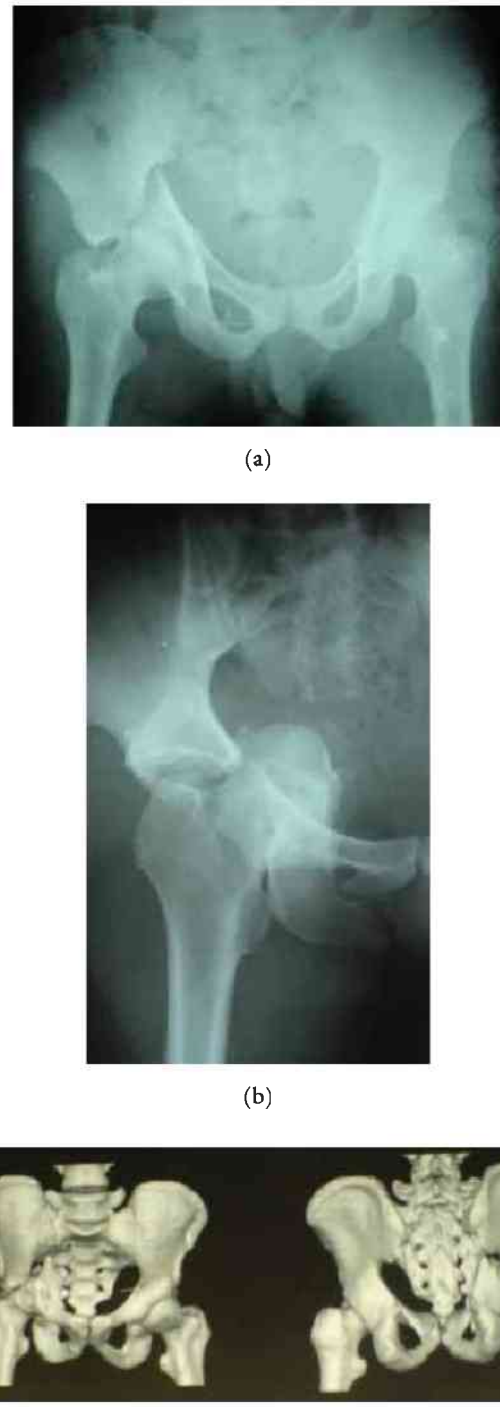
Types

The following are the two major types:

1. Fracture dislocation with an intact weight-bearing articular surface
2. Comminuted, displaced fractures of the floor of the acetabulum (bag of bones)

Clinical Features

- The patient complains of severe pain in the hip.



(a)

(b)

(c)

FIGURE 18.4 (a, b) X-rays of hip showing central dislocation of hip. (c) 3D CT reconstruction of pelvis with both hips showing central dislocation of hip.

- The limb does not show marked deformities as in other types of dislocation. The flexion, extension movements of the hip are relatively free but there is marked limitation of abduction and rotations.
- When it is suspected, a rectal examination must be done; it reveals a smooth hemispherical bulge in the lateral wall of the rectum which is diagnostic of the condition.

A radiograph will show the type of fracture in the floor of the acetabulum and the inward displacement of the head of the femur (Fig. 18.4).

Treatment

Continuous, heavy skeletal traction is applied to the limb with the leg in about 30° abduction. This will result in reduction of the dislocation. The traction is maintained for 4–6 weeks. Subsequently, active non-weight-bearing movements are encouraged. Some cases with a displaced weight-bearing articular surface will require surgical fixation.

Old Unreduced Dislocation of the Hip

In India, this is a common clinical entity. The patient with a dislocation gets treated by the traditional bonesetter with massage for some months. He then presents with a painful, stiff and deformed hip. This is a difficult problem for treatment. The radiograph will show an unreduced dislocation often complicated by myositis ossificans all-round the joint. Closed as well as open surgical reduction is not possible in most cases if it is more than 3 months old. A subtrochanteric osteotomy is done to correct the deformity and relieve pain.

THIGH

The bone in thigh is femur and the following fractures of femur bone will be discussed:

- Fracture of the neck of femur
- Trochanteric fracture of femur
- Fracture of the shaft of femur

Fracture of the Neck of Femur

The incidence of fracture neck of femur is higher in old age.

Incidence and Mechanism

Fracture of the neck of femur is common in the elderly. It does occur occasionally in young adults and even in children. It occurs more frequently in women.

- The fracture may result either from a rotational violence at the hip due to tripping over something on the floor and falling

  or
- A direct violence over the lateral aspect of the hip by a fall on the side

Classification

Based on the Anatomic Site *(Fig. 18.5)*

1. Subcapital (intracapsular)
   - Fracture immediately distal to the femoral head
2. Transcervical (intracapsular)
   - Fracture across the femoral neck
3. Basicervical (extracapsular)
   - Fracture around the base of the femoral neck

Based on Fracture Angle (Pauwel's Classification) *(Fig. 18.6)*

Three types are described as follows:

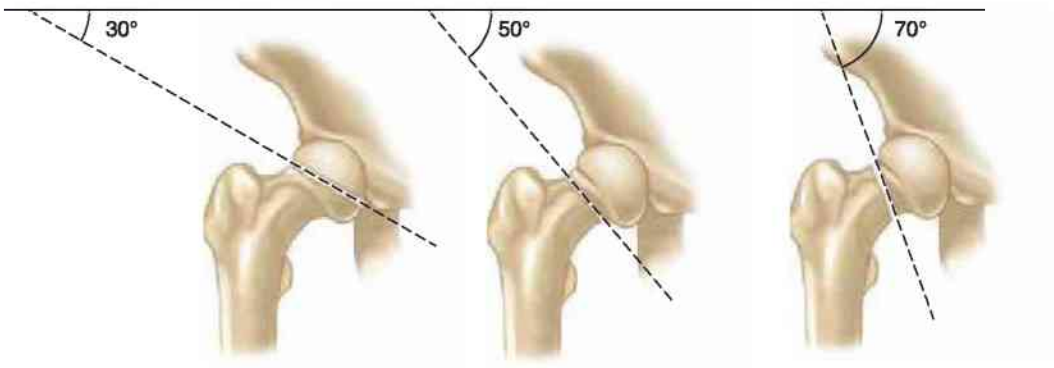
1. Fracture line 30° to the horizontal
2. Fracture line 50° to the horizontal
3. Fracture line 70° to the horizontal

Based on Displacement of the Femoral Head (Garden Classification)

- The fracture is incomplete with valgus impaction over the distal fragment. The weight-bearing trabeculae show little angulation.
- The fracture is complete without displacement. The weight-bearing trabeculae show interruption by a fracture line.
- The fracture is complete with incomplete displacement. The weight-bearing trabeculae of

(a) (b) (c)

FIGURE 18.5 Diagrammatic representation of types of intracapsular fracture of neck of femur. (a) Subcapital, (b) transcervical and (c) basal.



Type I Type II Type III

FIGURE 18.6 Pauwel's angle.

the femoral head, neck and acetabulum do not align.

- The fracture is complete with complete displacement. The weight-bearing trabeculae of the femoral head aligns with that of acetabulum and not to the femoral neck.

Clinical Features

- The patient is usually an elderly person with a history of a fall and inability to walk.
- On inspection, the injured leg lies in a position of external rotation and there is shortening of the leg. The attachment of the capsule to the distal fragment prevents excessive external rotation of the leg.
- On palpation, there is tenderness over the anterior and lateral aspects of the hip joint.
- The greater trochanter is elevated on the injured side. All movements are extremely painful except in the rare case of an impacted type of fracture.

Patients with impacted femoral neck fracture present with the following:

- History of trivial fall
- Minimal pain on walking
- Pain restricted to hip or ipsilateral knee range of movements of hip—painful and associated with muscle spasm
- Trochanteric tenderness is present

Patients with displaced femoral neck fracture present with the following:

- Inability to bear weight or walk with pain and swelling over the involved hip
- Minimal external rotation of the limb

- Minimal shortening
- Tenderness over the hip
- Painful movements of the hip
- A stress fracture of the femoral neck presents similar to undisplaced valgus impacted fractures of the femoral neck

Radiological Features

X-ray of the pelvis with the AP view of both hips shows the following (Fig. 18.7):

- Fracture line around the femoral neck
- Break in Shenton's line
- Break in the trabecular continuity

In an impacted fracture or a stress fracture, conventional X-ray may prove indecisive. It can be confirmed with an MRI of the involved hip or a bone scan.

An AP view of the whole pelvis to show both the hips must be taken.

Management

Fractures at this level have a poor capacity for union due to the following factors:

- Interference with the blood supply to the proximal fragment
- Haematoma collection in the intact capsule may impede the venous return and hence increase the already deficient vascularity
- The lack of organisation of the fracture haematoma due to the presence of the synovial fluid
- Absence of the periosteal layer required for callus formation
- Angiogenic inhibiting factors in the synovial fluid impede healing

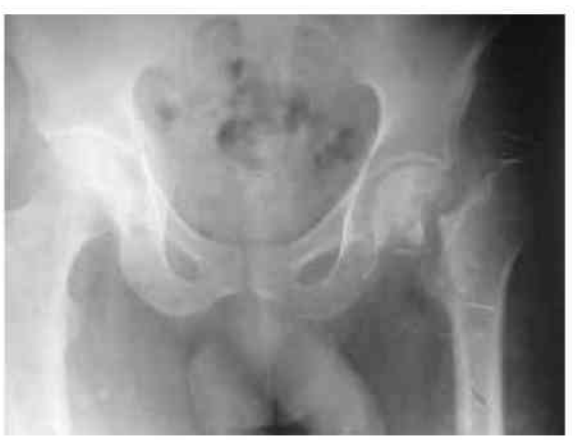


FIGURE 18.7 X-ray of the pelvis with both hips' anteroposterior view showing intracapsular displaced fracture neck of femur (left side). Note the break in Shenton's line.

Non-operative management is reserved in rare situations where the

- patient is of extremely high risk for surgery,
- patient is non-ambulatory due to various other reasons.

Surgical Treatment

Surgery is the treatment of choice for the fractures of neck femur. The surgery depends on the age of the patient and duration of fracture.

Undisplaced and Impacted Fractures

Undisplaced and impacted fractures are treated by one of the following methods:

- Multiple cancellous screw fixation, preferably three screws
- Sliding hip screw with a derotation screw

Displaced Fractures

- In young individuals, the preferred treatment is closed/open reduction and internal fixation with multiple cancellous screws or using a dynamic hip screw.
- In elderly individuals, the preferred treatment is to replace the femoral head with a prosthesis. The surgery is called ***hemiarthroplasty***, and three types of prosthesis may be used for replacement: Austin Moore's prosthesis, Thompson's prosthesis and bipolar prosthesis. ***Total hip replacement*** is also done for this fracture.

Fracture Neck of Femur in Children

The fracture neck of femur in children is classified (Delbet's) as follows:

- *Type I:* Transepiphyseal
- *Type II:* Transcervical
- *Type III:* Cervicotrochanteric (basal)
- *Type IV:* Intertrochanteric

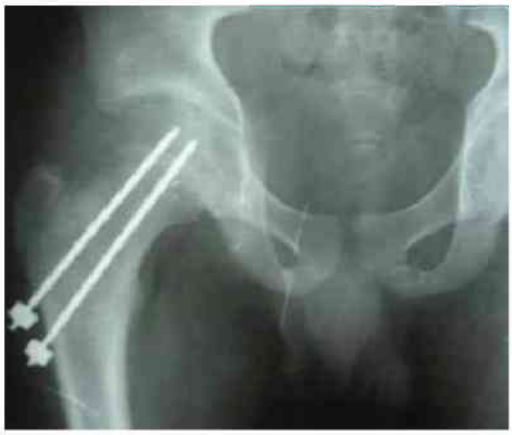


FIGURE 18.8 X-ray of the pelvis with hips showing Austin Moore pinning done for fracture neck of femur.

The fracture is reduced by manipulation and the leg is immobilised in full plaster spica in abduction for 8–10 weeks. When indicated, internal fixation could be done with multiple thin Austin Moore's pins (Fig. 18.8).

Complications of the Intracapsular Fracture Neck of Femur

The important complications are as follows:

- Non-union
- Avascular necrosis of the head of femur

Non-Union

Failure of union of this fracture still occurs due to improper reduction or imperfect internal fixation. When this occurs, the patient complains of pain and develops instability on walking.

The following points are important with regard to non-union:

- Absence of the cambium layer of periosteum (which produces callus) is an important factor in progression.
- Displaced femoral neck fractures go for non-union more commonly than undisplaced fractures.
- The patient presents with pain and difficulty in walking with history of trauma in the past.
- The Trendelenberg test is positive, and telescopy is positive in a previously unfixed hip.

Treatment

1. In elderly individuals, the treatment of choice is total hip replacement; alternatively, hemiarthroplasty can be done.
2. In young individuals, the treatment modalities include the following:
   - Pauwel's intertrochanteric osteotomy
   - Meyer's vascularised muscle pedicle graft
   - Internal fixation with cancellous screws or sliding hip screws

Avascular Necrosis

Avascular necrosis of the head of the femur is an unpredictable complication met with after any type of internal fixation. The patient presents with pain in the hip and is limping. There is limitation of all movements of the hip with muscle spasm. Radiography shows patchy areas of increased density in the head of the femur.

In early stages, rest and protected weight bearing are recommended. Surgical treatment includes core decompression of the femoral head.

In late stages, treatment options include the following:

- Osteotomy
- Arthroplasty
- Arthrodesis

Elderly patients are treated with arthroplasty, whereas young patients are treated with osteotomy or arthroplasty.

Trochanteric Fracture of Femur (Extracapsular Fracture Neck of Femur)

Basically, trochanteric fractures of the femur are extracapsular and are divided into the following:

- Intertrochanteric fractures
- Subtrochanteric fractures
- Intertrochanteric fracture with subtrochanteric extension

These are further classified as follows:

- Stable fractures
- Unstable fractures

In the trochanteric fractures, the blood supply to the proximal fragment is not interfered with and there is a greater area of contact between two fragments; hence, the fractures unite easily. While union is the rule, the common complication encountered is ***malunion with a coxa vara deformity***. The normal neck-shaft angle is about 125°. When the angle is reduced to close to 90°, the deformity is called coxa vara.

These fractures occur in the elderly, and the nature of the violence is the same as that in the intracapsular fracture.

Clinical Features

- On examination, the injured leg lies externally rotated and is obviously shorter.
- The degree of external rotation is greater than that in the intracapsular fracture.
- There is marked local swelling and ecchymoses over the trochanteric area.
- All movements of the hip are extremely painful and limited. This has to be differentiated from the intracapsular fracture (Table 18.1).

Radiological Features

- *Stable type:* There is a single fracture line and it is a two-piece fracture (Fig. 18.9).
- *Unstable type:* This is a comminuted fracture with multiple fractures at the trochanteric level.

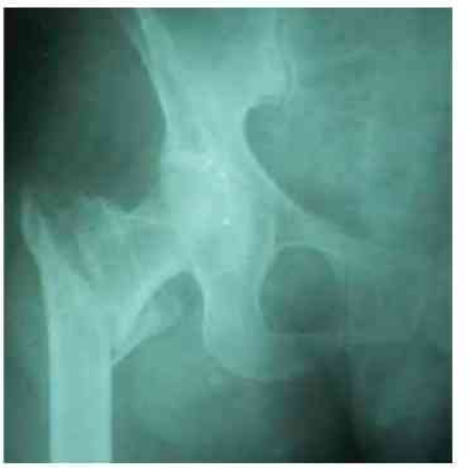


FIGURE 18.9 X-ray of the hip with femur showing trochanteric fracture of femur.

Management

The principle of the treatment is reduction of the fracture and maintenance of the fragments in good position till union occurs.

Conservative Treatment

Patients who have medical complications and at extremely high risk for surgery can be managed non-operatively

This consists of the application of continuous *skeletal traction*. For cases with marked coxa vara, continuous skeletal traction through the upper tibia is applied and the leg is immobilised in a Bohler Braun splint and the foot end of the bed is

TABLE 18.1 Differences Between the Fracture Neck of Femur and Trochanteric Fractures

	Intracapsular (Neck of Femur Fracture)	Extracapsular (Trochanteric Fracture)
Incidence	Less common	More common
Causative violence	Minimal rotation violence	Lateral violence
Clinical features		
External rotation	Minimal	Fully externally rotated
Local swelling	Nil	Marked local swelling
Treatment	Difficult	Easy
Complications		
Non-Union	Common	Does not occur
Malunion	Rare	Common

FIGURE 18.10 Clinical photograph showing Bohler Braun splint used for traction.

raised (Fig. 18.10). The fracture unites in about 12 weeks. When the coxa vara is not marked, skin traction in a Thomas splint will be sufficient.

Surgical Treatment

The use of a dynamic compression hip screw and plate system has enabled earlier mobilisation of the hip and weight bearing.

- Intertrochanteric fractures can be treated by fixing it with a sliding hip screw and plate.
- Subtrochanteric fractures can be treated by fixing it with a 95° angle blade plate or with a dynamic condylar screw and plate.

Complications

The main complication is malunion with coxa vara and shortening of the limb. If the coxa vara is gross, it can be corrected by osteotomy.

Other complications include osteoarthritis due to alteration in the normal biomechanics of the hip.

Fracture of the Shaft of the Femur

Fracture of the femur from the subtrochanteric level to the supracondylar level will be included in this group. The femur being the strongest weight-bearing bone in the body, a fracture is usually due to very severe violence. The powerful muscles surrounding the bone create problems in maintaining the fragments in position after reduction (Table 18.2).

In fractures of the *proximal third*, the proximal fragment is small and is drawn into a flexed position by the iliopsoas muscle and into the abducted position by the gluteus medius and minimus muscles (Fig. 18.11a).

In the fractures of the *middle third*, the distal fragment is displaced medially and pulled upwards by the adductors causing over-riding and outward angulation (Fig. 18.11b).

In fractures at the *supracondylar level of the shaft*, the distal fragment is flexed by the origin of the gastrocnemius muscle. In this position, the *popliteal vessels* are in great danger of injury by the sharp and irregular edges of the flexed distal fragment (Fig. 18.12). Therefore, the proximal fragment goes for flexion, abduction and external rotation. The distal fragment goes for adduction and proximal migration

Classification

On the basis of the site of fracture, the fractures of the femoral shaft can be classified as follows:

- Subtrochanteric fracture; proximal one-third
- Midshaft fracture; middle one-third
- Supracondylar fracture; distal third

On the basis of comminution, the fractures can also be classified (Table 18.3).

Clinical Features

- The patient with a fracture of the femur is likely to show symptoms of shock due to

TABLE 18.2 Effect of Muscle Action over the Displacement of Fracture Fragments

Muscles	Action
Gluteus medius and minimus	Abduction of the proximal fragment
Iliopsoas	Flexion and external rotation of the proximal fragment
Adductors	Adduction and proximal migration of the distal fragment
Gastrocnemius	Flexing the distal fragment

FIGURE 18.11 Diagrammatic representation of fracture of the shaft of humerus showing displacement. (a) Proximal third and (b) middle third.

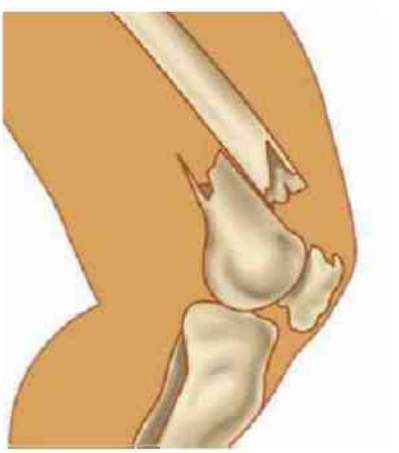


FIGURE 18.12 Diagrammatic representation of fracture of the distal third of femur.

loss of blood into the muscle planes of the thigh.

- Locally, there will be swelling of the thigh, deformity and tenderness.
- The presence of abnormal mobility and shortening of the limb will complete the clinical picture.

TABLE 18.3 Winquist and Hansen Classification

Grade	Extent of Comminution
0	No comminution
I	Minimal comminution
II	<50% comminution of the cortices
III	50–100% comminution of the cortices
IV	There is no cortical contact between the cortical segments

Additionally, the fracture may be transverse, oblique or spiral.

- One must always examine the distal pulsation in the dorsalis pedis and posterior tibial arteris. In fractures caused by runover injuries, the femoral vessels are often traumatised, leading to ischaemia and even gangrene of the leg.

Care must be taken to examine the distal neurovascular status of the limb.

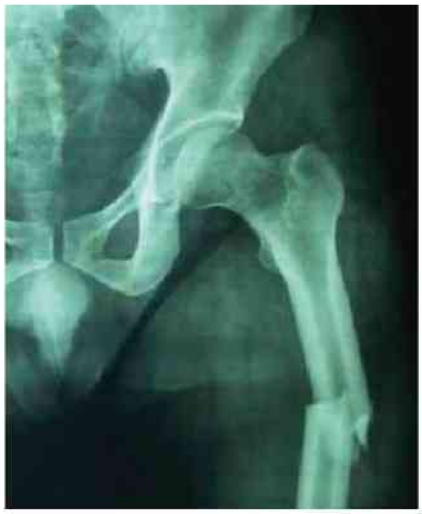
- A fracture occurring in the *distal* one-fourth shaft is notorious to cause popliteal vessel injury.
- The swelling in the thigh is due to loss of blood (approximately 1–1.5 L) into muscle compartments of the thigh.
- When this loss is high, the patient may present in a state of hypovolaemic shock.

Radiological Features

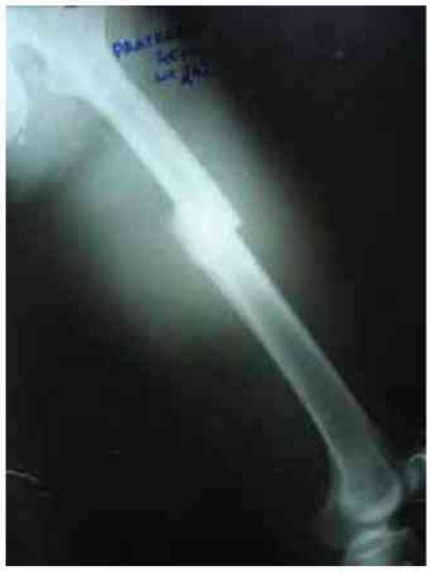
Standard AP and lateral views of the shaft of femur including knee joint and hip joint must be taken. Radiographs of the femur will show the level and type of fracture: transverse, oblique, spiral or comminuted (Fig. 18.13). They will also show other associated fractures such as the fracture neck of femur, intertrochanteric fracture and hip dislocation. A radiograph including the hip joint is mandatory as an associated dislocated hip could be missed in the clinical examination.

Management

- *First-aid treatment:* The best first-aid splint for fracture of the femur is the Thomas splint.



(a)



(b)

FIGURE 18.13 (a, b) X-ray of the left hip with femur anteroposterior and lateral view showing the fracture shaft of femur.

The thigh and leg are bandaged firmly to the splint. With this, the patient can be transported safely to the hospital.

- *Definitive treatment:* The aim of the treatment is to reduce the fracture and maintain reduction till union is complete. Any overriding or angulation must be corrected and the length of the limb maintained.

Conservative Method

It is limited only to those patients who are medically unfit and with high risk for surgery.

The patient is put on skeletal traction, for restoration of femoral length, for a period of 2–3 weeks, followed by cast bracing.

Surgical Treatment

Surgery is the treatment of choice.

Closed Fracture

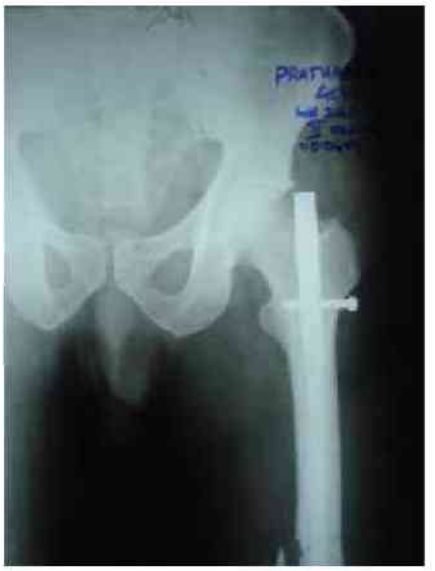
In closed femoral shaft fractures, fixation can be done with the following:

1. Interlocking intramedullary nails (Fig. 18.14)
   a. The nail is inserted inside the medullary canal of the proximal and distal fragments.
   b. The technique can be done as a closed or open procedure, depending on whether the fracture site is opened or not.
   c. The inserted nail is proximally and distally interlocked with interlocking screws. The screw is passed from one cortex into the hole through the nail to hold the other cortex.
2. Kuntscher nail
   a. It has a clover-leaf cross-section and inserted by an open technique.
   b. It has no interlocking system. It offers, stable fixation by three point-fixation.
   c. It is suited for transverse, midshaft fractures.
3. Plates and screws
   a. Femoral shaft fractures can be treated by open reduction and internal fixation with plates such as
      - Standard broad dynamic compression plate (BDCP)
      - Locking compression BDCP

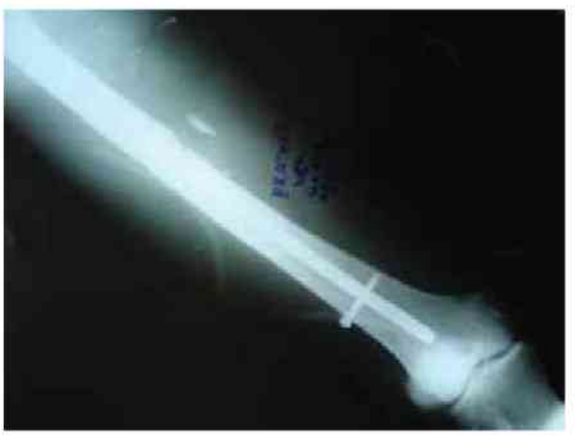
Fracture haematoma is essential for fracture healing. Hence, closed fixation methods are superior to open methods—closed intramedullary nailing is the best option for treatment of femoral shaft fractures.

Open Femoral Shaft Fractures

- These fractures up to Grade IIIA can be managed with internal fixation.



(a)



(b)

FIGURE 18.14 (a, b) X-ray of femur anteroposterior view showing the fracture shaft of femur treated with intramedullar interlocking nailing.

- Grade IIIB compound fracture is treated with an external fixator until the surrounding soft tissue heals and a wound cover is obtained.

Compound Fracture of Femur

Compound fractures of femur are usually due to blast or gunshot injuries. In addition to the management of shock and open wounds, use of external fixation maintains the stability of the reduction.

Fracture Femur in Children

The fracture of femur has been discussed below according to the age group:

- *Birth fracture:* In newborn infants with birth fracture of the femur, hip spica immobilisation is done as the thick periosteum keeps the fracture in position in most cases. The fracture unites in 2 weeks time with profuse callus formation.
- *Fracture in children under 5 years:* Hip spica application is used in undisplaced fractures. Flexible intramedullary nailing (Ender's nail and Titanium elastic nailing system) is used in displaced fractures.
- *Fracture in older children:* Flexible intramedullary nailing is used to immobilise the fracture in older children. In children of age 12 years or more, intramedullary nails can be used with locking for better stability and faster recovery. Hip spica application is rarely done in this age group.

Gallow's Traction

Traction is applied to both the legs, and the legs are pulled up till the buttocks are lifted off the bed. This is maintained for about 3 weeks after which the fracture is put in a plaster spica for another 3 weeks. This is an old method of treating fractures of the femur in children, now only of historical importance.

Complications of Fracture Femur

Malunion

The commonest complication of fracture of the femur is ***malunion*** due to the improper correction of the lateral angulation. If over-riding is not corrected, malunion occurs with marked shortening. If the shortening is more than 2.5 cm, it will need surgery by osteotomy and internal fixation. With the advent

of better internal fixation devices, incidence of malunion has become very less.

Stiffness of the Knee Joint

Knee stiffness occurs due to prolonged immobilisation, particularly in older patients. The causes are adhesions inside the joint (tibiofemoral), adhesion of patella to the femur and adhesion of quadriceps muscle to the fracture site. It should be treated by physiotherapy and, if required, surgery (arthrolysis of knee joint/quadriceps plasty).

Non-Union

Non-union is usually due the interposition of soft tissues between the fragments. This will need operative reduction and internal fixation with intramedullary nailing, supplemented with bone grafts.

Other complications are vascular injury, nerve injury and infection.

Vascular Injury

Fractures involving a distal one-third shaft may be associated with vascular injury. When a vascular injury is diagnosed, immediate repair is indicated, along with fracture fixation, preferably internal fixation.

Nerve Injury

Nerve injury is rare. Either the femoral or the sciatic nerve may get injured.

Infection

Infection may occur following open fractures or surgical site infection in closed fractures.

KEY POINTS

- Hip joint is a synovial joint of ball-and-socket variety.
- Normally, the angle of anteversion of the femoral neck with the shaft is 12° and the normal neck-shaft angle is around 125°.
- Posterior dislocation is the commonest type of hip dislocation, occurring due to automobile accident (dashboard injury).
- In posterior dislocation, limb lies in attitude of flexion, adduction and internal rotation and in anterior dislocation extension, abduction and external rotation.
- Fracture neck of femur is common in elderly and more frequently seen in women.
- Fracture neck of femur has poor capacity for union due to interference with blood supply to proximal fragment and lack of organisation of fracture haematoma due to presence of synovial fluid.
- Surgically, intertrochanteric fractures can be treated by fixing with a sliding hip screw and plate and subtrochanteric fracture can be treated by fixing with a 95°-angled blade plate or with a dynamic condylar screw and plate.
- Surgery is the treatment of choice in the fracture shaft of femur.

MULTIPLE CHOICE QUESTIONS

1. Which fracture neck of femur has a poor prognosis
   a. Intracapsular
   b. Extracapsular
   c. Both
   d. None
2. Avascular necrosis of the head of femur is not seen in
   a. Subcapital fracture
   b. Intertrochanteric fracture
   c. Transcervical fracture
   d. Central dislocation of hip
3. In fracture neck of femur in a 64-year lady, the treatment of choice is
   a. Prosthetic replacement of head of femur
   b. Conservative
   c. Austin Moore pin
   d. S.P. nailing
4. The commonest type of dislocation of the hip is
   a. Anterior
   b. Posterior
   c. Central
   d. Dislocation with fracture of the shaft
5. Dislocation of hip joint palpable on per rectal examination
   a. Congenital dislocation of hip
   b. Posterior dislocation of hip
   c. Fracture of neck femur
   d. Anterior dislocation of hip

CHAPTER 19

Injuries of the Knee and Leg

KNEE

Injuries to the knee joint can be grouped as follows:

- Fracture of the femoral condyles
- Fracture patella
- Injuries to the extensor mechanism
- Internal derangements of the knee (ligament injuries)
- Dislocation of the knee
- Fractures of the proximal tibia

Fracture of the Femoral Condyles

The condyles of femur can be fractured by direct violence or by sudden valgus or varus strains on the knee joint. They can be unicondylar involving only the medial or lateral condyle (Fig. 19.1a). They can be bicondylar extending into the articular surface in a T- or Y-shaped manner (Fig. 19.1b). When the fracture line runs into the articular surface of the knee, there is always a haemarthrosis. Radiographs will show the type of fracture and displacement.

Classification

Fractures involving distal femur are classified by Muller into three basic types, according to the extent of the involvement of the articular surface and the pattern of the fracture:

1. *Type A:* Extra-articular supracondylar fractures
2. *Type B:* Unicondylar fractures (partial articular)

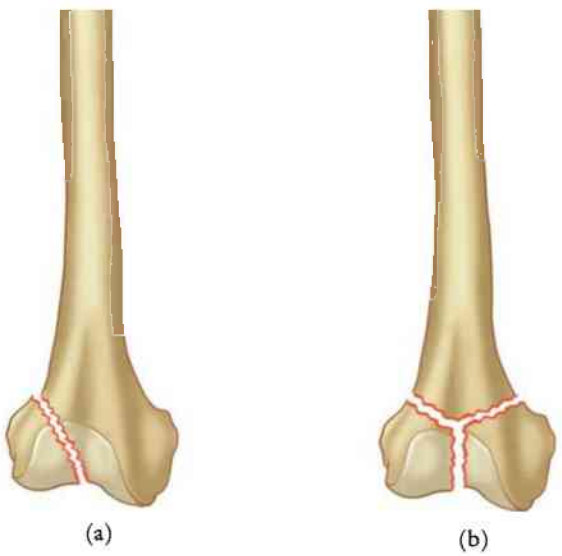


FIGURE 19.1 Diagrammatic representation of fracture of femoral condyles. (a) Unicondylar fracture femur and (b) bicondylar fracture femur.

3. *Type C:* Bicondylar fractures (complete articular)

A special type of fracture pattern is when the femoral condyles are fractured in the coronal plane, and it is referred to as *Hoffa's fracture* classified as B3.

Mechanism of Injury

Most of these injuries are sustained following severe axial loading with additional varus/valgus or rotational forces.

Radiographic Evaluation

Radiographic evaluation includes an anteroposterior view and lateral views of the distal femur and knee. This will show the type of fracture and displacement.

Treatment

Distal femur fractures are one of the complex injuries to treat. The essential principles of management of fractures involving the joint surfaces are as follows:

- Accurate anatomical reduction
- Early mobilisation of the joint
- Delayed weight bearing

Non-Operative Treatment

It has a role in the patient where

- the fracture is stable and undisplaced,
- there is medical contraindication to surgery.

The displaced fragment is reduced by manipulation.

Operative

Aim: To restore articular congruence in displaced fractures (if the fragment is large and reduction is not possible), comminuted fractures and those with vascular injury are treated by open reduction and internal fixation (ORIF) by

- cancellous screws,
- dynamic condylar screws and plate (Fig. 19.2),
- supracondylar nail,
- locking compression plates.

Open fractures (Grade IIIB compound) are treated by debridement and external fixation.

Complications

The complications associated with the fractures of the femoral condyles are vascular injury, malunion, stiffness knee and osteoarthritis.

Vascular Injury

The vascular injury can involve distal part of the femoral/popliteal artery; if present, emergency vascular repair should be done.

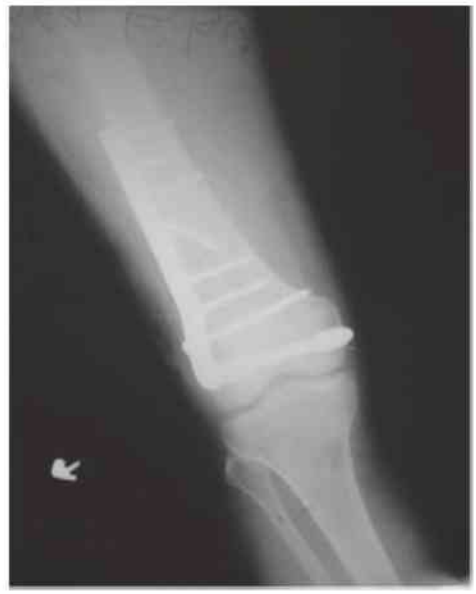


FIGURE 19.2 X-ray of the knee anteroposterior view showing supracondylar fracture femur fixed with dynamic condylar screws.

Malunion

Varus deformity is commonly seen after malunion of distal femoral fractures. Corrective osteotomy is done if the malunion is disabling.

Stiffness Knee

Stiffness knee is the most common complication. It can be treated by arthrolysis, quadriceps plasty along with adequate physiotherapy.

Secondary Osteoarthritis

Secondary osteoarthritis can happen following damage to the articular surface.

Fracture Patella

The patella is the biggest sesamoid bone in the body. Being incorporated in the quadriceps muscle, it improves the leverage for the action of its tendon by altering favourably the line of insertion of the tendon.

Classification *(Fig. 19.3)*

Depending on Displacement

- Undisplaced
- Displaced

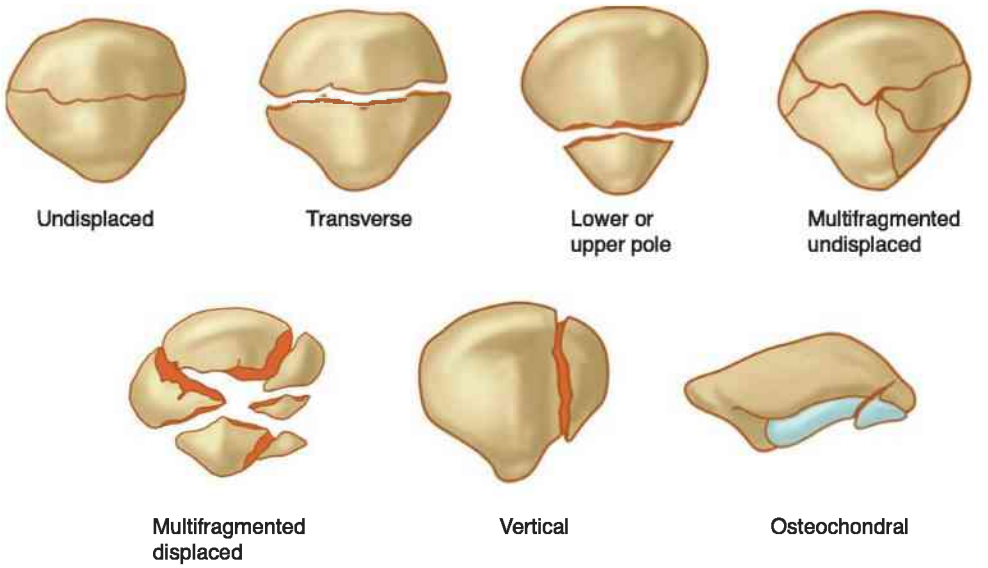


FIGURE 19.3 Diagrammatic representation showing various types of patella fractures.

Depending on the Fracture Line Pattern

- Stellate (multifragmented)
- Transverse
- Osteochondral
- Polar
- Vertical

Mechanism of Injury

Patella can be injured by one of the following two mechanisms.

Indirect Violence

A sudden contraction of the quadriceps muscle to prevent a fall causes a transverse fracture of the patella, along with a tear of the medial and lateral retinacula of the quadriceps expansion. The fracture may be across the middle of the patella or near the lower pole.

Direct Violence

A direct injury on the patella due to a fall on the knee usually produces a stellate fracture of the patella with a comminution of the bone (Fig. 19.4). In this type, the quadriceps' expansion is intact.

Clinical Features

The patient presents with difficulty or inability to walk. The patient gives a history of a feeling of something giving way in the knee, when attempting to avoid a fall or a fall directly on the knee. The knee is swollen and there is evidence of haemarthrosis in the joint. Direct violence is indicated by the presence of bruises over the surface. On palpating the bone, one could feel a transverse line of tenderness across the patella. If the fragments are separated, a gap could be felt between the two fragments (palpable patellar defect). There will be inability to actively extend the knee; extensor lag should be noted.

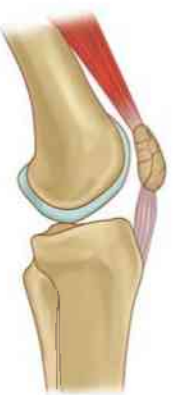


FIGURE 19.4 Diagrammatic representation of comminuted fracture patella.

Radiographs

The injured knee is evaluated by the following:

- Anteroposterior view
- Lateral view
- Skyline view

Treatment

Treatment depends upon the type of fracture and the age of the patient.

Non-Operative

Undisplaced fractures with an intact extensor mechanism can be treated by a cylinder cast for 4–6 weeks after aspiration of the haemarthrosis.

Operative

The options available in this category include the following:

- Tension band wiring
- Cerclage wiring
- Lag screw fixation

Transverse Fracture with Separation

Transverse fracture with separation always means that the quadriceps expansions are ruptured. Such cases always need operative treatment to repair and restore the quadriceps mechanism.

The patellar fragments are brought together and sutured with a stainless steel wire in a circumferential or figure-of-eight manner. The medial and lateral quadriceps expansions are carefully sutured.

Comminuted Fracture

In this type of fracture with displacement, the articular surface is badly damaged. The treatment is patellar reconstruction if feasible but as a last resort, patellectomy and repair of the quadriceps mechanism are done. Preserving the patella is of prime importance.

Fracture Lower Pole

In this case, the small lower pole fragment is excised and the ligamentum patellae is attached to the main fragment.

Injuries of the Extensor Mechanism

The extensor mechanism of the knee consists of the quadriceps muscle, patella, ligamentum patellae and the medial and lateral retinacula. Injuries will include the rupture of quadriceps muscle, fracture of the patella or avulsion of ligamentum patellae insertion (Fig. 19.5).

Rupture of the quadriceps muscle occurs due to a sudden violent contraction. The rupture usually occurs at the upper border of the patella. Rupture across the ligamentum patellae has the same effect. There is an inability to actively extend the knee. In both cases, surgical repair is indicated.

Dislocation of the Knee

Traumatic dislocation of the knee is an uncommon injury. It is caused by road traffic accidents and sometimes in sports injury. It may be *anterior, posterior* or *posterolateral* according to the direction of displacement of tibia (Table 19.1). The serious complication is injury to the popliteal artery causing grave vascular impairment to the leg. It should be treated as an emergency and immediate reduction done under general anaesthesia. The leg is immobilised in a plaster tube slab for 6 weeks in extension. In the case of unreducible dislocation or the presence of popliteal artery injury, open reduction is indicated. Injuries associated with the anterior and posterior cruciate ligaments, medial and lateral collateral ligaments and meniscus are common.

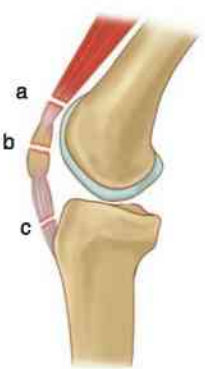


FIGURE 19.5 Diagrammatic representation of injuries of the extensor mechanism. (a) Rupture quadriceps tendon, (b) fracture patella and (c) rupture ligamentum patellae.

TABLE 19.1 Types of Knee Dislocation

Types	Mechanism	Features
Anterior	Hyperextension violence	Most common Arterial injury common
Posterior	Dashboard injury (in a flexed knee with a posteriorly directed force)	Extensor mechanism injury common Arterial injury common
Posterolateral	Abduction and internal rotation force in a flexed knee	Irreducible Peroneal nerve palsy is common

Tibial Plateau Fractures

Tibial plateau fractures involve the articulating surface of proximal tibia, thus involving the knee joint. Proximal tibial fractures are also referred to as 'bumper fractures'.

Clinical Features

- The patient presents with the history of trauma to the knee joint.
- On examination, there is swelling, tenderness and restricted knee joint movements.
- Compartment syndrome is a frequent association with proximal tibial fractures.
- Radiological evaluation includes anteroposterior and lateral views of the involved knee joint with leg (Fig. 19.6).

Tibial plateau fractures are classified by Schatzker from I to VI types.

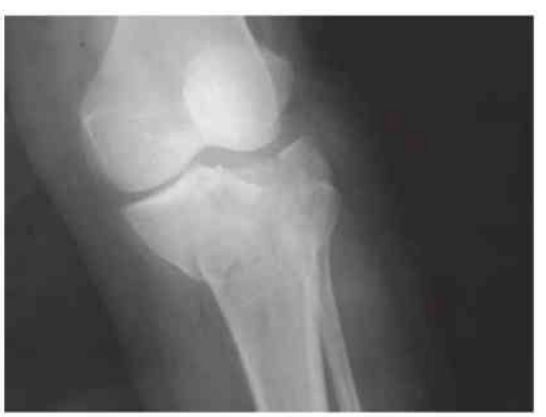


FIGURE 19.6 X-ray of the knee anteroposterior view showing Type V—bicondylar fracture of tibial plateau.

Treatment

Non-Operative

Non-displaced and stable fractures without articular depression can be treated in a plaster cast for 6 weeks, followed by partial weight bearing and then full weight bearing for 12 weeks.

Operative

Operative options include ORIF with

- screws alone,
- plates and screws (Fig. 19.7).

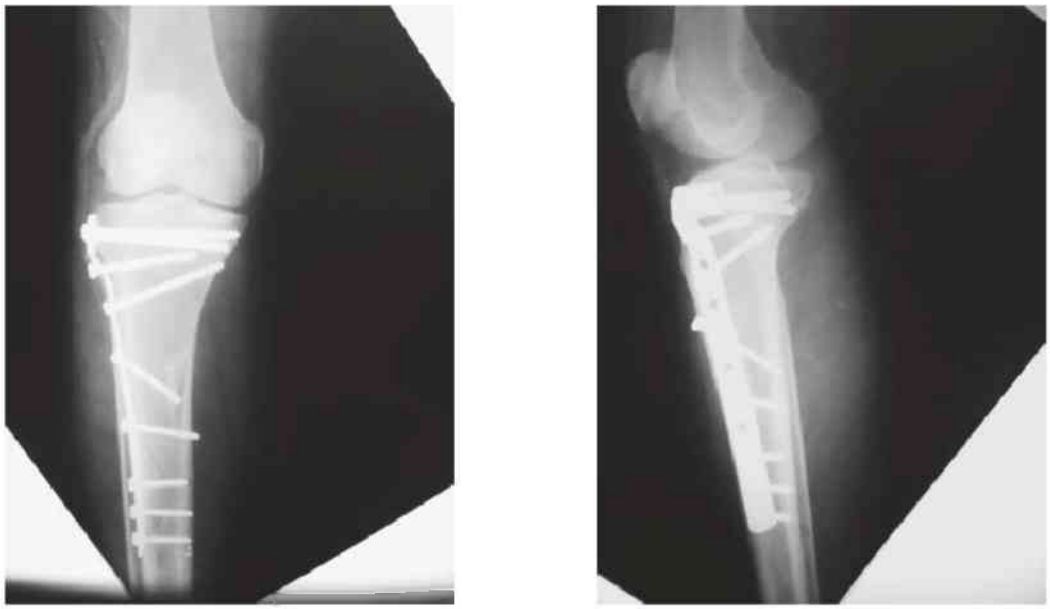
Traumatic Dislocation of the Patella

Traumatic dislocation of the patella is a rare condition where the patella gets laterally dislocated due to sudden quadriceps contraction. Mostly, it is easily reduced. It leaves the patient with a sense of knee giving way and an apprehension of instability. If the patient presents with an acute dislocation of the patella, it is reduced under general anaesthesia.

LEG

Fracture of the Tibia and Fibula

Next to the femur, the tibia is the longest and strongest weight-bearing bone in the body. Most of the bone is subcutaneous and is exposed to direct injury. The fracture is very often open, due to the subcutaneous position of the bone and hence is exposed to infection (Fig. 14.4).



(a) (b)

FIGURE 19.7 (a, b) X-ray of the knee anteroposterior view showing tibial plateau fracture treated with plates and screws.

While the upper two-third of the bone has muscular attachments in the anterolateral and posterior surfaces, the distal one-third is devoid of any muscular cover and is surrounded by tendons and neurovascular structures. Three osteofascial compartments are recognised surrounding the tibia and fibula: the anterior, lateral and posterior. Trauma and associated haemorrhage and oedema can result in acute compression in these closed spaces causing vascular insufficiency to the leg (compartmental syndrome).

The blood supply to the distal third is rather inadequate, and hence fractures at the junction of the middle and distal third of the tibia unite slowly and non-union is common at this level.

Incidence and Mechanism

The incidence of fracture of the tibia is increasing due to an increase in road traffic accidents on the highways and in the cities. It is often associated with a fracture of the fibula at the same or at a different level.

Occasionally, indirect violence due to fall from a height or in sports causes a vertical fracture in the lower third of the tibia.

Clinical Features

A patient with the fracture of the tibia and fibula presents with a swelling and severe bruises in the leg. The presence of external wounds should be noted. Deformity at the site of fracture is often obvious. Abnormal movements can be easily noted when one attempts to move the leg. The pulse in the dorsalis pedis artery and posterior tibial artery should always be palpated.

Radiological Features

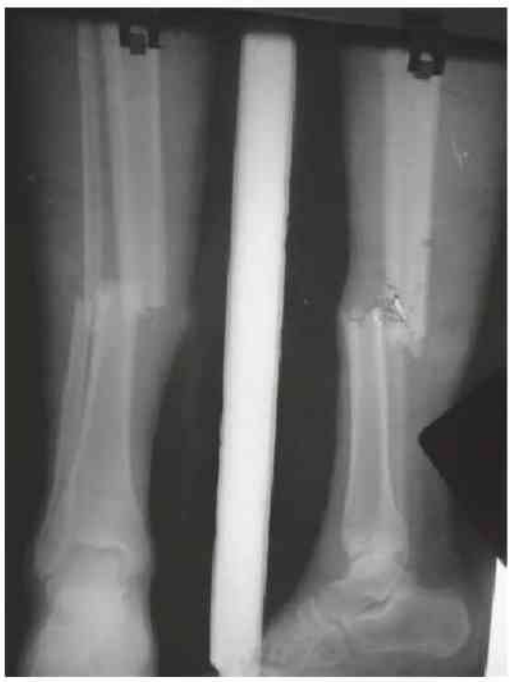
Anteroposterior and lateral radiographs of the leg and knee joint will show the level, type of fracture and displacements (Fig. 19.8). The following types may be seen:

- Transverse
- Oblique and spiral fractures
- Comminuted fracture
- Segmental fracture

Treatment

Fracture Without Displacement

These cases are immobilised in an above-knee padded PoP slab with the knee in 10° flexion. The



(a) (b)

FIGURE 19.8 X-ray of the leg with ankle (a) anteroposterior and (b) lateral views showing transverse fracture of the both bones of the leg at the distal one-third. Note the displacement at the fracture site and the resulting valgus deformity.

limb is kept elevated in bed till the oedema subsides. A week later, a close fitting above the knee plaster cast is applied.

Functional Cast Bracing

A recent advance in conservative management is the method of functional cast bracing (Sarmiento). After 3–4 weeks of the above-knee plaster cast, the patient is fitted with a functional brace or cast. The patient is encouraged to mobilise the knee and start weight bearing. This allows controlled micromovements at the fracture site and promotes callus.

Surgical Treatment

The methods of internal fixation after open reduction are as follows:

- Dynamic compression plate and screws (Fig. 19.9a)
- Intramedullary nailing: The technique of nailing is done after either closed or open reduction (Fig. 19.9b)

Open (Compound) Fractures of the Tibia and Fibula *(Fig. 14.4)*

The principles of treatment of compound fracture tibia are as follows:

- Thorough surgical debridement
- Stabilisation of the fracture by external (Fig. 19.10) or internal fixation
- Restoration of the skin cover by skin grafting or flap transfer

Complications

The complications of the fracture tibia and fibula can be immediate or late:

- Immediate: Compartment syndrome
- Late: Non-union
- Malunion

Compartment Syndrome

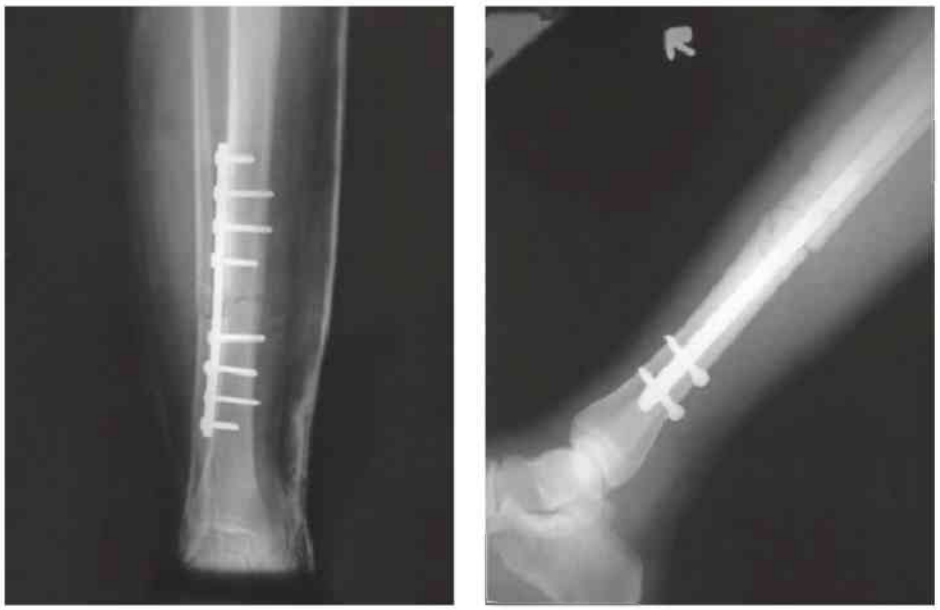
Compartment syndrome is a circulatory complication of fractures of the leg caused by a sudden increase in pressure in the osteofascial compartments due to haemorrhage and oedema. This causes ischaemia and may result in gangrene and hence must be treated as an emergency. This is similar to acute Volkmann's ischaemia in the forearm (refer to Chapter 14).

The patient with a fracture tibia presents with severe increasing pain and tense swelling of the leg, pallor and absence of distal pulses in the foot. Clinically, it can be an anterior or posterior compartment syndrome due to increased pressure in the respective compartment.

Treatment is by emergency surgical decompression of the involved compartment by incising the deep fascia (fasciotomy).

Non-Union

Non-union is common in fractures of the lower third of the tibia. This is due to the fact that the blood supply to the distal fragment is poor. If the fracture has failed to unite even after 4–6 months of immobilisation, the radiograph will show



(a) (b)

FIGURE 19.9 (a) X-ray of the leg with ankle anteroposterior view showing fracture of both bones of the leg treated with plates and screws. (b) X-ray of the leg with ankle lateral view showing fracture of both bones of the leg treated with an intramedullary nail.

sclerosis of the fractured ends and an absence of callus. The principles of treatment are open reduction, internal fixation and bone grafting.

This consists of the removal of the fibrous scar tissue between the fragments, freshening the sclerosed bone ends and securing fixation using a plate and screws. In addition, cancellous bone chips are packed around the fracture site.

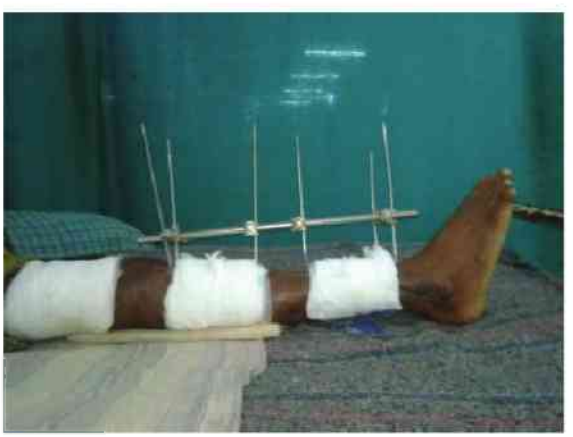


FIGURE 19.10 Clinical photograph of compound fracture of both bones of the leg treated with external fixator.

Malunion

Malunion is due to improper correction of overriding and angulation between the fragments. The patient presents with deformity and/or shortening and limping. The treatment is by corrective osteotomy, realignment and internal fixation. Untreated malunion leads to late osteoarthrosis knee and ankle.

Isolated Fracture of Fibula

Isolated injuries to the shaft of the fibula are rare. They are of no great importance as the bone is not a weight-bearing one and is well covered with muscles. However, fractures at the neck of fibula can injure the lateral popliteal nerve. Fractures of the distal end of the fibula form a part of ankle injuries and may require surgical stabilisation.

KEY POINTS

- Hoffa's fracture is fracture of femoral condyle in coronal plane.
- Vascular injury may occur with distal femur fracture requiring emergency vascular repair.
- Patella is the largest sesamoid bone in the body.
- Undisplaced fractures of patella with an intact extensor mechanism are treated conservatively. Tension band wiring, cerclage and screw fixation are available options for surgery.
- Traumatic dislocation of knee is often associated with popliteal artery injury causing grave vascular impairment to leg.
- Three osteofascial compartments are recognized surrounding the tibia and fibula-anterior, posterior and lateral.
- Fracture tibia and fibula are often compound fractures.
- Compartment syndrome is a complication of fracture tibia and fibula requiring emergency fasciotomy.
- Fracture neck of fibula can injure lateral popliteal nerve.
- Fracture of the distal end of fibula is included in the category of ankle injuries. This may require surgical stabilisation.

MULTIPLE CHOICE QUESTIONS

1. Injury to the popliteal artery in fracture lower end of femur is often due to
   a. Distal fragment compressing the artery
   b. Proximal fragment pressing the artery
   c. Tight plaster
   d. Haematoma
2. Comminuted fracture of patella is treated by
   a. Tension wire bandage
   b. Surgery and immobilisation
   c. Conservative
   d. Patellectomy
3. Proximal tibial fractures are also called
   a. Hoffa's fracture
   b. Bumper fracture
   c. Aviator's fracture
   d. Lisfranc fracture
4. Transverse fracture of patella in a young adult. What is the treatment of choice?
   a. Tension band wiring
   b. Cylinder cast
   c. Patellectomy
   d. Conservative

CHAPTER 20

Injuries of the Ankle and Foot

ANKLE

Injuries around the ankle joint are common nowadays, more so with elderly women.

Around 60% of the fractures around the ankle are malleolar fractures and they are associated with heavy soft tissue trauma.

Anatomy

The ankle joint is a complex hinge joint and is formed by distal articular surfaces of the tibia and fibula with the dome of the talus, which is its superior articular surface. The distal articular surface of the tibia is known as the plafond which, together with the medial and lateral malleoli, forms the ankle mortice.

The tibiofibular syndesmotic ligament complex exists between the distal tibia and fibula, which is strong and makes the ankle joint stable.

Ligaments Around the Ankle Joint *(Fig. 20.1)*

The medial aspect of the ankle joint is supported by the deltoid ligament which has superficial and deep intra-articular and deep (tibiotalar) extra-articular portions.

The lateral fibular collateral ligament is made of the following:

- Anterior talofibular
- Posterior talofibular
- Calcaneo fibular ligaments

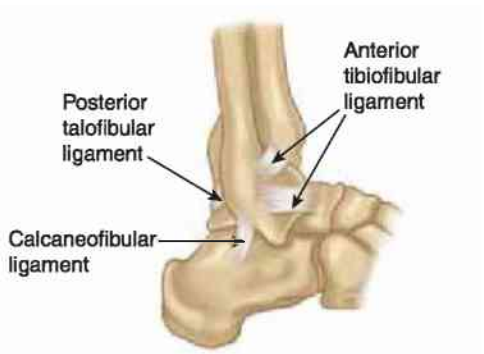


FIGURE 20.1 Diagrammatic representation of the ankle joint showing ligaments around the lateral aspect.

ANKLE INJURIES

The term 'Pott's fracture' is loosely applied to most of the severe fractures occurring around the ankle. The following are the types of violence which can cause the fracture:

- External rotation injuries
- Abduction injuries
- Adduction injuries
- Vertical compression injuries

This helps us to understand the mechanism of the fracture and to plan appropriate methods of treatment.

Each of these types of forces can produce fractures of varying degrees, according to the severity of the violence and hence we recognise first-, second- and third-degree fractures in each type.

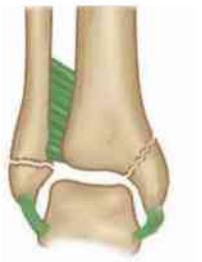


FIGURE 20.2 Supination abduction.

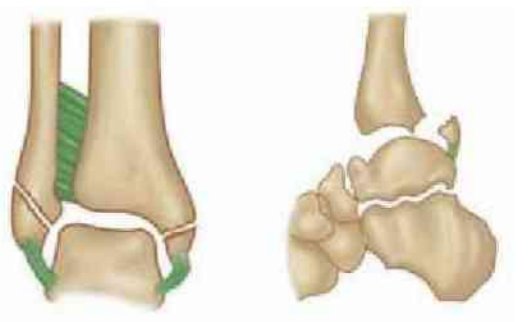


FIGURE 20.3 Supination—external rotation.

Malleolar Fractures and Their Classification

The Lauge–Hansen classification is most commonly used. This classification is based on the following:

- Position of the foot at the time of injury
- Direction of the deforming force

The types of ankle injuries according to this classification are as follows:

- Supination—abduction injuries
- Supination—external rotation injuries
- Pronation—abduction injuries
- Pronation—external rotation injuries

Supination—Abduction Injuries *(Fig. 20.2)*

The inversion force on the dorsiflexed ankle results in vertical or oblique medial malleolar fracture and transverse avulsion-type fracture of distal-fibula–associated rupture of the lateral collateral ligament. This is also associated with medial displacement of talus.

Supination—External Rotation *(Fig. 20.3)*

This is the most common type of injury. This leads to a typical oblique fracture at the distal fibula and either a transverse or avulsion type of medial malleolus fracture and rupture of the deltoid ligament.

Pronation—Abduction Injuries *(Fig. 20.4)*

In this type of injury, there will be deltoid ligament disruption and transverse fracture of the medial malleolus with short oblique fracture of fibula at the level of syndesmosis with lateral comminution fracture.

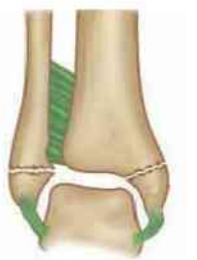


FIGURE 20.4 Pronation abduction.

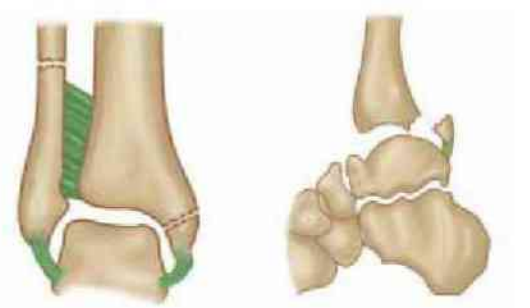


FIGURE 20.5 Pronation—external rotation

Pronation—External Rotation *(Fig. 20.5)*

In this type of injury, transverse fracture of the medial malleolus and/or rupture of the deltoid ligament may occur. This can be associated with disruption of the tibiofibular ligament and fracture of the posterior part of the distal articular surface of the tibia, that is, posterior malleolar fracture.

Special Types

Pott's fracture was described by Percival Pott in 1765. This is caused by a combined abduction

external rotation violence. It includes rupture of the medial ligament/fracture medial malleolus, fracture of the lateral malleolus and lateral displacement of the ankle.

Dupuytren's Fracture

Dupuytren's fracture again is due to abduction external rotation violence. In a typical case, there is fracture of the medial malleolus, diastasis of the inferior tibiofibular syndesmosis and fracture of the distal fibula.

Maisonneuve's Fracture

Maisonneuve described an injury where there is a partial diastasis of the inferior tibiofibular syndesmosis with a fracture at the proximal fibula. Hence, it is imperative that in ankle fractures one must look for tenderness at the upper end of fibula and a fracture there in the radiograph.

Vertical Compression Fractures

Vertical compression fractures are caused by a fall from a height on the heels. Fractures of the intra-articular distal tibia are called pilon fractures.

Plafond or Pilon Fracture

Plafond or pilon fracture is the comminuted fracture of the distal tibial articular surface resulting from a vertical compression and injury (Fig. 20.6). It is almost always associated with syndesmotic disruption.

It is treated by open reduction and internal fixation with the following:

- Cancellous screws
- Plates and screws
- External fixator in cases of open fractures

Clinical Features

There is a history of forcible twist of the foot. The patient presents with a swelling around the ankle. There is obvious deformity if there is lateral or medial displacement of the foot. In first-degree injuries, there is tenderness over the involved malleolus only. In second-degree injuries, there is tenderness over the medial and lateral aspects of the ankle. In third-degree injuries, the foot is found displaced posteriorly when the leg is seen from the side.

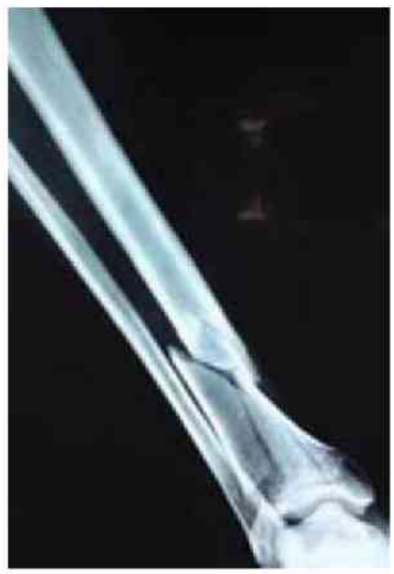


FIGURE 20.6 X-ray of leg with ankle anteroposterior view showing tibial plafond fracture. Note the discontinuity of the tibial plafond (distal articular surface) due to a compression injury at the ankle.

Radiological Features

Anteroposterior and lateral views of the ankle joint show the following:

- An avulsion, transverse oblique fracture of the medial malleolus
- An avulsion, transverse, oblique or comminuted fracture of the lateral malleolus
- Disruption of tibiofibular syndesmosis shown by the lateral talar shift, medial joint space widening and posterior tilt of talus

Treatment

The goal of the treatment for ankle injuries is achievement of the anatomical reduction of the ankle mortise and minimising the chances of stiffness and osteoarthritis of ankle in future. While some of the fractures are treated conservatively, most of the fractures and fracture dislocations around the ankle joint are treated by surgical methods.

Conservative Treatment

In isolated undisplaced fracture of the lateral or medial malleolus, the foot is first immobilised in a below-knee padded posterior plaster slab. When the oedema subsides, a complete plaster cast is applied for 6 weeks.

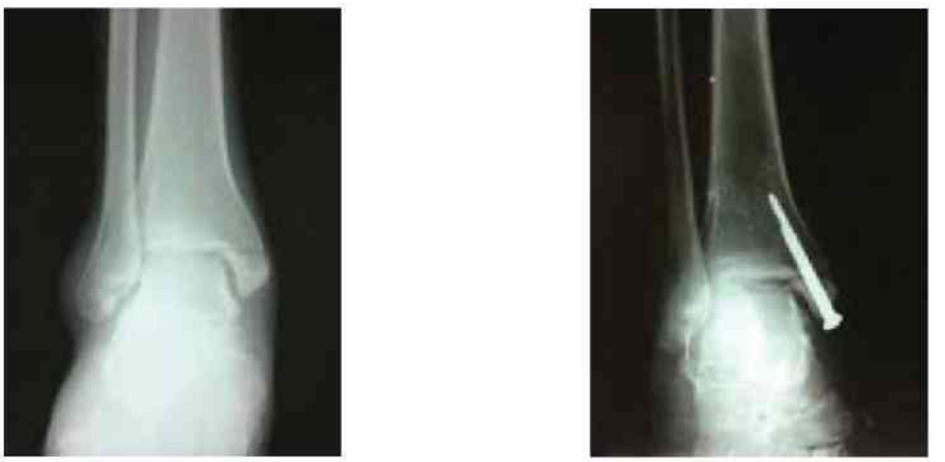
Treatment of Fracture Dislocation

It is important to obtain accurate anatomical reduction of all displaced fractures and those involving the articular surface of the ankle joint.

A single attempt of closed manipulative reduction under anaesthesia is done and a below-knee plaster is applied. If the position is acceptable, by X-rays, this is maintained for 6–10 weeks. For satisfactory reduction, the ankle mortise should be aligned between the medial and lateral malleoli. After 10 weeks, the plaster is removed and physiotherapy started.

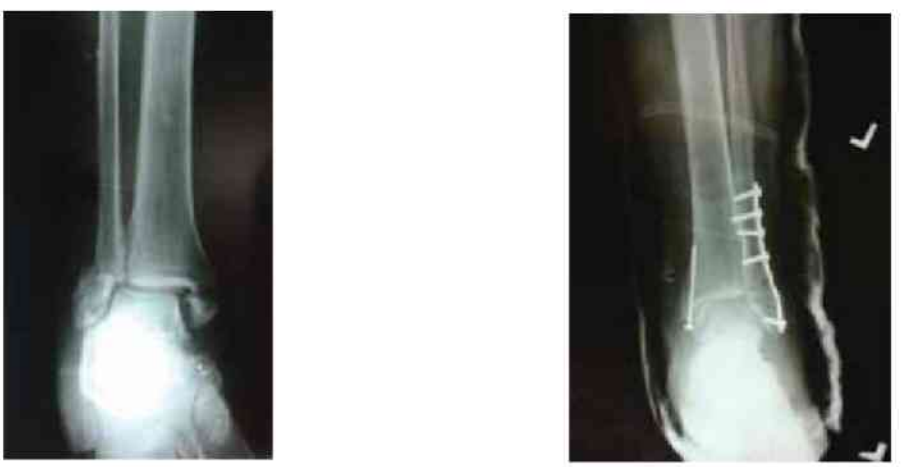
Surgical Treatment

If reduction is not achieved by closed methods, open reduction and internal fixation is done to achieve perfect reduction and stable fixation (Figs 20.7 and 20.8).



(a) (b)

FIGURE 20.7 X-ray of leg with ankle anteroposterior view showing (a) medial malleolus fracture and (b) medial malleolus fracture which is fixed with cancellous screw.



(a) (b)

FIGURE 20.8 X-ray of leg with ankle anteroposterior view showing (a) bimalleolar fracture and (b) bimalleolar fracture fixed with cancellous screws for medial malleolus and plates and screws for lateral malleolus.

TABLE 20.1 Operative Method Used in the Management of Ankle Fracture

Operative Method	Indications
Compression screws (Fig. 20.7)	Done for transverse or oblique fractures of medial malleolus and short oblique fractures of lateral malleolus Posterior malleolar fracture involving more than one-third of the articulating surface of tibia
Tension band wiring	Done for transverse fractures of medial or lateral malleoli
Plate and screw fixations (Fig. 20.8)	Distal fibular fracture
External fixation (Fig. 20.9)	Compound fractures and fracture dislocations Fracture with comminution in the distal articular tibia As a step one procedure in closed ankle fracture for the soft tissue to heel

- Bimalleolar fractures with dislocation which are unstable after manipulative reduction need open reduction and screw fixation.
- Fractures with tibiofibular diastasis are often unstable after reduction. They are best fixed by an additional transverse screw, fixing the lower ends of the tibia and fibula.

The internal fixation may be of different types (Table 20.1).

Complications

Complications include malunion, non-union, joint stiffness and osteoarthrosis. Non-union is commoner in medial malleolus when it has to be treated by surgery. Malunion will later result in osteoarthrosis.

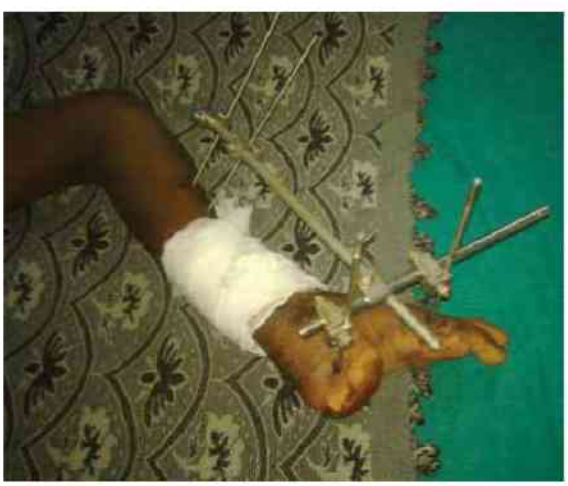


FIGURE 20.9 Clinical photo showing ankle spanning external fixator used for compound severely comminuted tibial plafond fractures as a temporary measure until soft tissue heals.

FOOT

The Indian foot is exposed to injury as well as infections, since most people walk barefoot indoors and even while at work. The working population in loading and unloading operations in the harbours, building work and packaging industries show the highest incidence of injuries to the foot due to lack of protective footwear. Bad treatment for foot injuries leading to stiff and painful feet is a serious disability in an Indian worker.

Fracture of the Talus

Fracture of the talus is the second commonest tarsal bone to be fractured (Fig. 20.10).

Anatomy

Talus is supplied by a critical anastomosis of vessels entering into the body of talus through the neck. Hence, fracture of the neck may jeopardise the blood supply of the body. Talus is a bone that does not give attachment to any muscles.

Mechanism

Fracture talus is caused by a fall on the foot with forced hyperdorsiflexion of the ankle. The talar

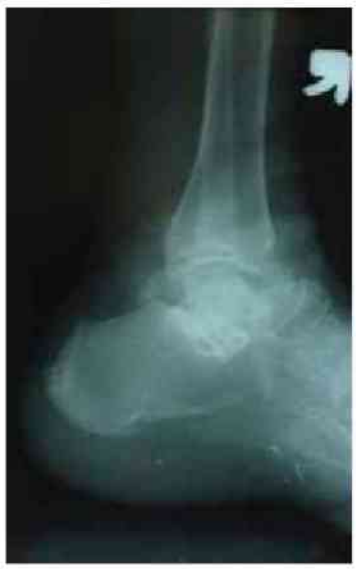


FIGURE 20.10 X-ray of lateral view of the ankle showing talus fracture after careful evaluation. Chances of avascular necrosis are high in fractures of neck and in displaced fractures.

neck fractures, as it impinges on the anterior margin of the tibia. Fracture of talar neck is called *aviator's fracture.* There may also be associated posterior tilt of the talus with the dislocation of the joint.

Hawkin's Classification of Talar Neck Fractures

- *Group I:* Non-displaced vertical fracture of the talar neck
- *Group II:* Displaced fracture of the talar neck with subluxation or dislocation of the subtalar joint (the ankle joint remains aligned)
- *Group III:* Displaced fracture of the talar neck with dislocation of the body of the talus from both the subtalar and the ankle joints.

Clinical Features

There will be marked swelling around the ankle. In fractures with displacement, the body of the talus may be felt subcutaneously in the posteromedial aspect of the ankle.

Treatment

Undisplaced fractures of the neck of the talus are treated by immobilisation, in a below-knee plaster with the foot in plantar flexion position for 6–10 weeks.

When the body is displaced and in fracture dislocations, open surgical reduction and internal fixation with screws are required.

Complications

Avascular Necrosis

Fracture through the neck of the talus compromises the blood supply of the body resulting in avascular necrosis of the fragment (body) and leading to non-union of the fracture. It is a late complication and can lead to early osteoarthritis of the ankle joint.

Osteoarthritis

Intra-articular fractures may lead to late osteoarthritis of the ankle. The treatment includes arthrodesis of the ankle joint.

In cases where the body is badly displaced posteromedially, the superficial skin may necrose making it compound.

Fractures of the Calcaneus

Anatomy

The calcaneus forms the heel. It articulates with the talus superiorly and cuboid anteriorly. The posterior tuberosity of the calcaneus is elongated backwards, and this constitutes the major weight-bearing surface.

Incidence and Mechanism

Calcaneus is the commonest tarsal bone to be fractured. Injuries to the calcaneus are caused by falling from a height and landing on the foot. It is common in young adults engaged in building construction work and in those climbing coconut or palmyra trees.

Classification (Essex Lopresti)

- *Extra-articular:* These fractures constitute 30% of the calcaneal fractures. Fracture of the tuberosity, sustentaculum talus and the body which do not involve the articular surface of the subtaloid joints are extra-articular fractures.

- *Intra-articular:* Compression fractures of the body which involve the subtaloid joint with varying degrees of comminution. This constitutes about 70% of the calcaneal fractures. These fractures involve the articular surface of the calcaneum and reduce the Bohler angle (see below).

Clinical Features

There is a history of a fall from a height and the patient complains of pain in the heel. On inspection, there is swelling in the region of the heel. Seen from behind, the heel is broader when compared with the normal side. There is tenderness on holding the heel below the malleoli between the thumb and fingers. This also confirms the broadening of the injured heel. The movements of inversion and eversion are markedly limited and painful. The vertical distance between the tip of the malleoli and the ground is diminished as the calcaneum is vertically compressed in the fracture.

In examining a patient with injury to the calcaneum, one should ***always examine the other foot*** to detect any fracture in the other calcaneum, as the fall from a height often produces bilateral fracture calcaneum. It is also very important to ***examine the spine*** for the presence of any associated fracture of the spine, which is also caused by fall from a height.

Radiological Features

The compression fracture in a solid irregular bone like the calcaneum may not be obvious as a fracture line in the radiograph (Fig. 20.11). Due to the vertical compression, the trabecular pattern in the bone will be distorted and an area of increased density may appear due to interlocking of the trabeculae. Minimal compression will be detected by the diminution of the tuber ***joint angle*** (Bohler's) in the lateral radiograph (Fig. 20.12).

Bohler's Angle

Bohler tuber angle is the angle between the superior articular surface of calcaneus and superior surface of the tuber. It is 20–40° normally and in calcaneal fractures this angle is reduced.

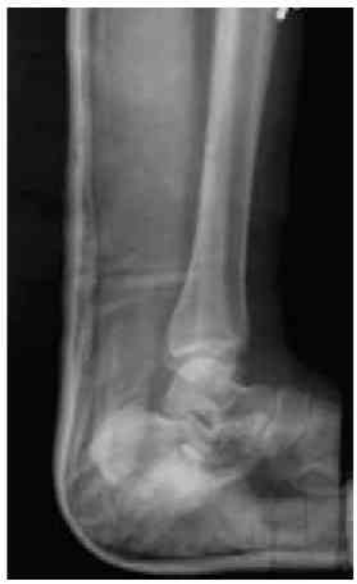


FIGURE 20.11 X-ray of lateral view of the ankle showing calcaneal fracture.

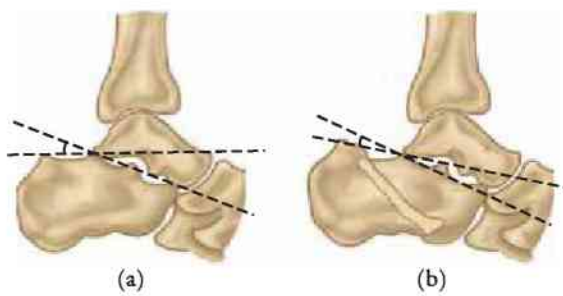


FIGURE 20.12 Diagrammatic representation of Bohler's angle. (a) Normal tuber joint (Bohler's) angle and (b) reduced angle in fracture calcaneum.

Crucial Angle of Gissane

Gissane's angle is the angle between the line along the posterior facet and another line from the anterior facet to the beak of the calcaneus. These lines form the cortical struts of the calcaneus. They form an obtuse angle of 95–105°.

Axial view radiographs should be taken to show involvement of the subtaloid joint and comminution.

Treatment

Simple fractures which involve the peripheral processes of the bone are treated by rest in a below-knee

plaster slab for 4–6 weeks. In cases where there is minimal involvement of the joint surface, the patient is kept non-weight bearing with a plaster slab. In cases with severe comminution and distortion, surgical intervention is needed. The height of the calcaneus is reduced in these cases, which is identified by reduced Bohler's angle. Then the fragment is elevated surgically and internally fixed with plates and screws. Bone grafting may be needed.

Complications

In cases with the fracture involving the articular surfaces, the patient will develop subtaloid osteoarthrosis causing pain.

Stiffness

Stiffness of the subtalar and midtarsal joints will occur, when the articular surface is involved.

Osteoarthritis

If the articular surface is not perfectly reduced, osteoarthritis of the subtalar joint will occur. In severe cases, it may need arthrodesis of the joint.

Fracture of the Other Tarsal Bones

Fracture of the other tarsal bones (Fig. 20.13) and fracture dislocation of the midtarsal and tarsometatarsal joints are uncommon. They need closed reduction and below-knee plaster cast in most cases. Some rare cases may need open reduction and 'K' wire fixation.

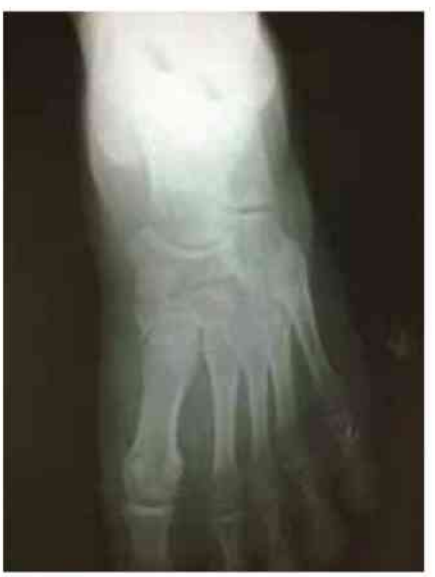


FIGURE 20.13 X-ray of foot anteroposterior view showing comminuted fracture of medial cuneiform.

Fractures of Metatarsals and Phalanges

Fractures of the metatarsals are due to direct violence, the foot being crushed by weights falling on it. Crack fractures without displacement are best treated by strapping the foot and weight bearing after a few days. In cases where two or more metatarsals are fractured (Fig. 20.14) with gross displacement, open reduction and K wire fixation are indicated.

Stress Fracture Metatarsal Bone

Stress fracture metatarsal bone is one of the common forms of stress fracture. The fracture occurs in new recruits to the army and police due to the unaccustomed strain on the foot during training. It is also called *march fracture* or *fatigue fracture*. Treatment is mainly symptomatic by strapping.

Fracture Base of the Fifth Metatarsal Bone (Jones Fracture)

Jones fracture is caused by severe inversion of the forefoot, when the foot gets doubled up with the body weight falling on it. This is often associated with sprain of the lateral ligament of the ankle.

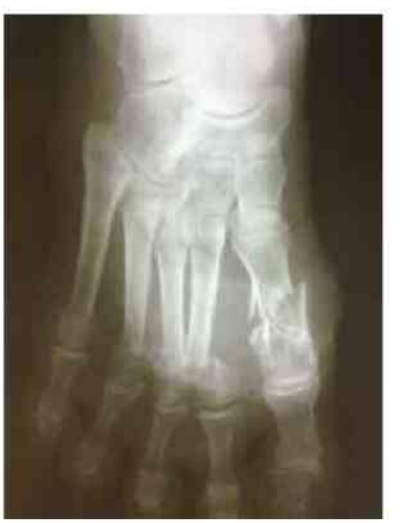


FIGURE 20.14 X-ray of foot oblique view showing fracture of the metatarsals.

The base of the bone can be avulsed by the peroneus brevis insertion (pseudo-Jones fracture). Treatment is strapping the foot and early weight bearing if the oedema is minimal. If the oedema is severe, the foot is immobilised in a below-knee slab. It can be internally fixed using screws.

Fractures of the Phalanges of Toes

Fractures of the phalanges are common injuries that result from direct trauma. They may be closed or compound fractures. Only strapping and immobilisation are needed. In some compound fractures, 'K' wire fixation is done.

KEY POINTS

- The ankle joint is a complex hinge joint formed by the distal articular surface of the tibia and fibula with dome of talus.
- The medial aspect of the ankle joint is supported by deltoid ligament and laterally by lateral fibular collateral ligament.
- The term 'Pott's fracture' is loosely applied to most of the severe fractures around the ankle.
- Dupytren's fracture is due to abduction external rotation violence with fracture of medial malleolus, diastasis of inferior tibiofibular syndesmosis and fracture of distal fibula.
- Maisonneuve fracture is partial diastasis of the inferior tibiofibular syndesmosis with fracture of the proximal fibula.
- Tibial plafond or pilon fracture is comminuted fracture of the distal fibula articular surface resulting from a vertical compression injury.
- Undisplaced vertical fracture of the talar neck is called aviator fracture.
- Fractures through the neck of the talus compromise the blood supply of the body resulting in avascular necrosis of the body and leading to non-union of the fracture.
- Bohler tuber angle is the angle between the superior articular surface of calcaneus and the superior surface of the tuber. It is 20–40° and is reduced in calcaneal fracture.
- Stress fracture of metatarsal bone is called march fracture or fatigue fractures which occurs in new recruits to the army and police.
- Jones fractures is fracture base of the fifth metatarsal caused by severe inversion of the forefoot. The base of the bone can be avulsed by peroneus brevis.

MULTIPLE CHOICE QUESTIONS

1. March fracture affects
   a. Neck of the second metatarsal bone
   b. Body of the second metatarsal bone
   c. Neck of the first metatarsal bone
   d. Fracture of the lower end of tibia
2. Transverse fracture of medial malleolus is caused by
   a. Abduction
   b. Adduction
   c. Rotation of foot
   d. Dorsiflexion of foot
3. The stability of ankle joint is maintained by all of the following except
   a. Plantar calcaneonavicular (spring) ligament
   b. Deltoid ligament
   c. Lateral ligament
   d. Shape of superior articular surface
4. Bohler's angle is decreased in fracture of
   a. Calcaneum
   b. Talus
   c. Navicular
   d. Cuboid
5. Fracture of talus may give rise to
   a. Avascular necrosis of the body
   b. Avascular necrosis of the neck
   c. Avascular necrosis of the head
   d. Osteoarthritis ankle

CHAPTER 21

Injuries of the Spine

The commonest mode of injury in India is fall from a height. It has recently been seen that road traffic accidents and sport injuries have contributed to spinal injury. From the viewpoint of stability, the spine can be divided into the following three columns (Dennis classification):

1. *Anterior column:* It consists of the anterior half of the vertebral body, anterior part of the disc and anterior longitudinal ligament.
2. *Middle column:* It consists of the posterior half of the body, posterior part of the disc and posterior longitudinal ligament.
3. *Posterior column:* It consists of the posterior bony arch and the posterior ligament complex.

Based on the involvements of column, the stability is determined as follows:

- One column—stable injuries
- Two or more columns—unstable injuries

CLASSIFICATION

The type of injury to the spine depends on the nature of violence (Table 21.1). Injuries of the spine will be considered under the following heads:

1. Injuries of the thoracic and lumbar spine
   - Stable injuries without paraplegia
   - Unstable injuries (fracture dislocation) with paraplegia
2. Injuries of the cervical spine
   - Stable injuries without quadriplegia
   - Unstable injuries (fracture dislocation) with quadriplegia
   - Whiplash injuries

FRACTURES OF THE THORACOLUMBAR SPINE

The spine may be subjected to four types of violence (Fig. 21.1):

1. Flexion violence
2. Flexion rotation violence
3. Vertical compression
4. Extension violence

The type of lesion depends on whether the posterior ligament complex is ruptured or intact. The rupture is usually caused by flexion rotation violence resulting in instability. Hence, clinically, the spine injuries can be divided into stable and unstable types.

Stable Fractures Without Paraplegia

Clinical Features

Stable fractures without paraplegia is the common type of fracture spine. There is usually a history of a fall from a height or a weight falling on the back. On examination, there may be local bruises in the back and a localised point of tenderness in

TABLE 21.1 Nature of Violence and Types of Spinal Injuries

Nature of Violence	Type of Injury	Site	Stability	Complication
Pure flexion violence	Wedge compression Fracture Posterior ligament complex intact	Thoracic and upper lumbar	Stable	Nil
Flexion rotation violence	Fracture dislocation (fracture of the articular processes, ligament complex ruptured)	Thoracolumbar D11–L2	Unstable	Paralysis common
	Pure dislocation	Cervical spine	–	–
Vertical compression	Burst fracture of the vertebral body (posterior ligaments intact)	Cervical spine	Stable	–
Extension violence	Rupture of the anterior ligament Posterior ligaments intact	Cervical spine	Stable	Common in whiplash injuries

the spine. An angular gibbus is sometimes seen or felt at the site of fracture.

A general examination must be made for evidence of shock and injuries to the head, chest and abdomen. The legs must be examined for preliminary assessment of motor paralysis and sensory loss, and their extent is estimated. One must also look for distension of the bladder, due to paralysis of the bladder function. In cases of falls from a height, examine the heels for fracture calcaneum.

Anteroposterior and lateral radiographs of the spine must be taken to confirm the presence and type of fracture.

Treatment

- *First aid:* Any patient with an injured spine must be carefully shifted in the face-down position so that further flexion of the spine and injury to the spinal cord are avoided.

Wedge Compression Fracture

In these cases, the posterior ligaments are intact (Fig. 21.la). Prolonged immobilisation is not necessary. These patients are kept in bed for 2 or 3 weeks. As soon as there is healing of the soft tissue injury and the pain subsides, the patient is made to do active spinal extension exercises. It is not necessary to attempt restoration of the shape of the vertebrae.

Comminuted Fracture (Burst Fracture)

Comminuted fractures are more severe injuries. The fracture in the vertebral body is comminuted (Fig. 21.1b). These are treated

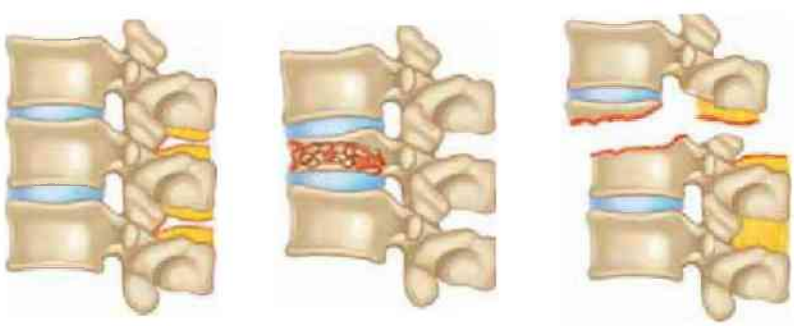


FIGURE 21.1 Diagrammatic representation of (a) flexion violence, (b) vertical compression violence and (c) flexion rotation violence.

by immobilisation in a plaster jacket in slight extension. The plaster jacket extends from the suprasternal notch to the symphysis pubis anteriorly and C7 spinous process to the top of the gluteal fold posteriorly. The patient is allowed to move in about 6 weeks. The patient may return to work with a spinal brace.

Fracture Dislocation with Paraplegia

Fracture dislocation with paraplegia is caused by a very severe flexion rotation violence (Fig. 21.1c) and is complicated in most cases by injuries to the cord or cauda equina resulting in paraplegia.

The treatment and rehabilitation of these cripples need a team of specialists including the orthopaedic surgeon, neurosurgeon, plastic surgeon, urologist and good physiotherapy and rehabilitation facilities.

Pathology

In fracture dislocation, the cord or the cauda equina gets damaged by being compressed by the lamina of the upper vertebra against the posterosuperior margin of the vertebral body below (Fig. 21.2). It may suffer from the following types of injuries:

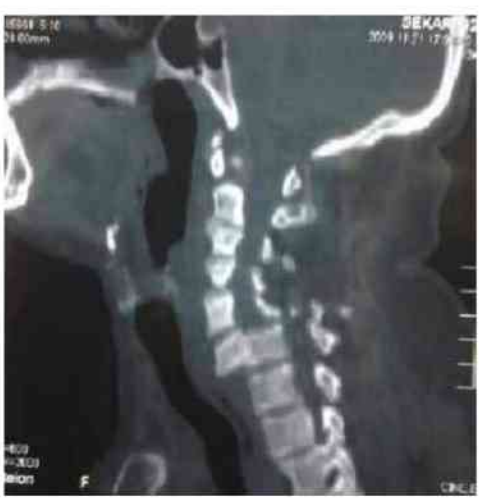


FIGURE 21.2 CT of the cervical spine showing fracture dislocation C6 vertebra over C7.

1. Cord injuries
   - Contusion and oedema
   - Laceration and transection of the tract fibres, incomplete or complete. This is followed by degeneration and liquefaction of the damaged cord substance
   - Haemorrhage into or around the cord substance
2. Cauda equina injuries
   - Partial injury: Crushing injury to the nerves of the cauda equina
   - Complete transection
3. Combined injury to cord and the cauda equina at the D12–L1 level

Clinical Features

The patient is brought with a history of fall, severe pain in the back and inability to move the legs. On examination, there is a gibbus with tenderness at the level of vertebral damage. A general examination must be done to exclude injuries to abdomen, chest and head. One should then determine the level of the cord injury. There will be motor paralysis of both lower limbs.

The level of sensory loss will depend on the level of vertebral damage (Fig. 21.3). There will be a level of hyperaesthesia at the upper margin of sensory loss due to nerve irritation.

In fracture dislocations at the L2 level and below, the spinal cord escapes injury and there is only a cauda equina lesion. Between D12 and L1 levels, it will be a mixed cord and cauda equina lesion.

The clinical picture will depend on the stage (spinal shock and stage of reflex activity) at which the patient is brought. On first examination, it will be difficult to diagnose the exact nature of the cord injury. Concussion, partial and complete transection of the cord will all present a similar picture of spinal shock with flaccid paraplegia. The nature of the lesion may partly be inferred by the level and displacement of the fracture.

- *Stage of spinal shock:* This stage shows complete flaccid paralysis and loss of all functions, below the level of the injury. In the case of contusions, this stage is only a physiological transection of the cord and in the course of

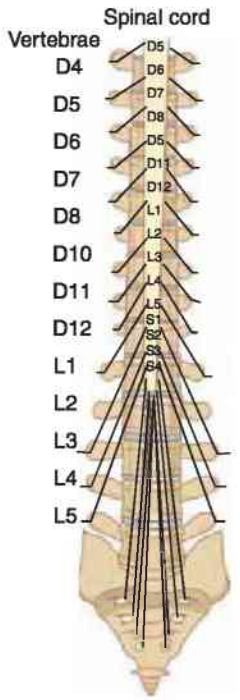


FIGURE 21.3 Diagrammatic representation showing spinal cord injury in relation to vertebral damage.

a few days or weeks shows complete recovery of function.

- *Stage of reflex activity:* In the case of partial disruption of the cord fibres, the stage of spinal shock is followed by the stage of paraplegia in extension and the return of reflex activity of the bladder. Depending on the degree of damage, there will be partial recovery of motor and sensory functions.

In the case of total anatomical disruption of the cord fibres, the stage of spinal shock is followed by a spastic paraplegia in flexion, with mass reflex below the level of injury. No recovery could be expected in such cases.

- *Stage of failure of reflex functions:* This occurs in cases where urinary sepsis and septicaemia become overwhelming and there is loss of even whatever functional recovery that had occurred earlier. This is really a terminal stage and the paraplegia becomes flaccid again. In injuries at the T12–L1 vertebral level, the injuries to the cord and cauda equina are often combined and the clinical picture is a mixed one. Injuries to cauda equina recover to a greater or lesser extent.

Radiological Features

Anteroposterior and lateral views will show the level and type of lesion. CT scan will be useful to accurately locate displaced fragments into the spinal cord.

Management of a Fracture

Shifting of the patient from the reception (Casualty) to the Radiology department or the ward should be done with extreme care to prevent jerking or flexion which will cause cord damage after arrival in the hospital. This is called the 'Second Accident'.

Conservative Treatment

The patient is positioned to lie supine in a split bed with a pillow under the gibbus to produce postural reduction. The fracture dislocation may get reduced. The patient is turned every 2 hours to prevent pressure sores. When the paraplegia begins to recover, a complete plaster jacket is applied.

Surgical Treatment

Open reduction and internal fixation to stabilise the spine can be done. The internal fixation is done by posterior stabilisation using pedicle screws, by anterior stabilisation using cages and pedicle screws or by combined global stabilisation. Surgery is indicated in the following situations:

- In the case of partial cord injury, when the recovery of paraplegia stops or deteriorates, decompression of the cord and internal fixation of the spine can also be done.
- If an obvious large fragment of the vertebral body is seen displaced posteriorly into the spinal canal, laminectomy to decompress the cord is done and the spine is stabilised.
- In gross degrees of fracture dislocation with total damage to the cord, early surgery to stabilise the spine is done to facilitate rehabilitation.

- Compound fracture, for example in gunshot or missile injuries, and wound debridement have to be done.
- Irreducible dislocation: Open reduction and internal fixation are done.

Management of Paraplegia

The exact diagnosis of the traumatic pathology will become obvious any time between 3 days and 3 weeks. A concussion will recover completely. A patient with incomplete cord laceration will develop paraplegia in extension and complete lesion will produce paraplegia in flexion.

The aim of the early treatment is to prevent bedsores and urinary sepsis as they are the commonest causes of death. The next step is the rehabilitation of the paraplegic to enable him to lead an independent life.

The management of an acute traumatic paraplegia should include the following:

- General care
- Psychological care
- Skin care
- Bedsores and their management
- Bladder care
- Bowel care
- Rehabilitation

General Care

The general treatment is important as these patients develop negative nitrogen balance and anaemia. A high protein diet and antianaemic treatment will increase the patient's resistance. Corticosteroids are given initially till the oedema of cord subsides.

Psychological Care

The emotional shock and depression following the realisation that he will not walk again is the most important factor in the total clinical evaluation of the patient. It is extremely important that the patient be assured and given all the psychological support that he will be restored to active social life in spite of his locomotor disability.

Skin Care

Skin care consists mainly in the prevention and treatment of bed sores.

Bedsores (Pressure Sores)

Bedsores (pressure sores) or decubitus ulcers are localised areas of cellular necrosis resulting from prolonged excessive stresses on soft tissues. Areas over bony prominences with compromised sensation are more susceptible to breakdown into decubitus ulcers. They are common over pressure areas such as sacrum, back of the heels and greater trochanters.

They are graded into Grades I, II and III depending upon the area and depth of the ulcer.

Pathogenesis

The bedsore starts as a hyperaemic area over pressure points (Grade I). If neglected, it leads to breakdown of the skin and ends up as deep ulcer due to sloughing of necrotic muscle exposing the underlying bone (Grade III).

This must be prevented by careful attention to pressure points such as sacral, trochanteric and heel areas. The use of spinal turning frames, air beds and water beds for the paraplegic patients will certainly minimise the incidence of bed sores. In places where such equipments are not available, the Madras method (Shanmugasundaram) of frequent turning of the patient by a trained team of nursing assistants, using an adequate number of pillows, is very successful in completely preventing bedsores.

Treatment

Treatment of bedsores consists of daily dressing, excision of necrotic tissue and slough and control of infection. When the sore area shows clean granulation tissue, skin grafting is done. Grade III sores with exposed bone will need plastic surgical reconstruction with fasciocutaneous or myocutaneous skin flaps.

Bladder Care (Stage of Spinal Shock)

During the stage of spinal shock, there is retention of urine and as the paralysed bladder distends, there is overflow of urine from the distended bladder. The bladder acts as a denervated organ and is unable to empty.

The further behaviour of the bladder depends on the type of disruption of the reflex pathways which are controlling the bladder functions. The

FIGURE 21.4 Diagrammatic representation showing innervation of the bladder from S2 and S3 levels (B—bladder, P—prostate).

reflex centre for the bladder is at the S2–S3 level in the cord, and this is controlled by the inhibiting influence of the higher centres in the cerebral cortex (Fig. 21.4).

Automatic Neurogenic Bladder (Upper Motor Neuron Bladder)

Automatic neurogenic bladder is that type of neurogenic bladder that occurs after complete transection of the cord above the S2 level. The reflex centres take over the control of the bladder.

The bladder becomes an organ controlled by a simple reflex. When the bladder is distended with urine, the patient involuntarily empties the bladder by the reflex activity. Urination occurs without warning as soon as the reflex is closed by summation of afferent stimuli. There will be minimal residual urine. This is called cord or automatic bladder. This may take 2–6 weeks to develop.

Autonomous Neurogenic Bladder (Lower Motor Neuron Bladder)

Autonomous neurogenic bladder occurs in injuries at the S2 level or below, that is, at the conus and cauda equina. This destroys the reflex centre with the afferents and efferents in the nerve roots. This isolates the bladder, which then depends on the intrinsic plexus in the musculature of the bladder wall, that is detrusor ganglia. This bladder which resumes some function is called autonomous bladder or atonic bladder. The emptying can be assisted by manual pressure or by trained contraction of the abdominal musculature. Due to spasm of the internal sphincter, the residual urine is large (300–400 cc).

Treatment

The aims of bladder management include the following:

- Avoidance of bladder overdistension
- Prevention of urinary infection
- Restoration of continence, by bladder training depending on the type of bladder

Retraining of the Bladder

The recovery of bladder function may take some 2–6 weeks. When there are signs of motor and sensory recovery, retraining of the bladder must be undertaken. This is done by clamping the catheter and encouraging reflex emptying of the bladder. When automatic bladder with reflex emptying is established, the catheter is removed.

In cases of autonomous bladder, the patient is taught methods such as suprapubic compression and increasing abdominal pressure. (If there is evidence of bladder neck contracture, surgical measures such as bladder neck resection may be necessary.)

Bowel Care

Bowel paralysis leads to foecal retention. Care includes foecal softening, digital evacuation, use of suppositories and enema.

Rehabilitation

The rehabilitation of paraplegic patients has been one of the achievements of medical science, during the past two decades. These patients need physical, psychological and economic independence and social integration. Medical rehabilitation should start right from the earliest stage of treatment of paraplegia.

Examination and Evaluation of Spinal Injuries

Fractures and dislocations of the spine are devastating injuries, which commonly occur in young individuals. When these injuries are associated with neurological deficit, the morbidity and mortality are very high. Spinal injuries occur in about 6% of all trauma cases. More than 50% of patients with spinal column injury have neurological deficit.

1. History of the mechanism of injury should be sought.
   The common causes of spine injuries are as follows:
   a. Road traffic accidents
   b. Fall from height
   c. Diving injuries
2. Physical examination
   a. The spinous processes of cervical, thoracic and lumbar regions should be palpated for tenderness/defect.
   b. Sensation to light touch and pinprick should be tested. Perianal sensation evaluation is important.
   c. Motor examination should be done and the muscle power should be documented. The key muscles to be examined are as follows:
      - C5—biceps brachii
      - C6—extensor carpi radialis longus and brevis
      - C7—triceps
      - C8—flexor digitorum profundus: middle finger flexion
      - T1—abductor digiti minimi: little finger abduction
      - L2—iliopsoas: hip flexion
      - L3—quadriceps: knee flexion
      - L4—tibialis anterior: ankle dorsiflexion
      - L5—extensor hallucis longus: long toe extension
      - S1—gastrocnemius-soleus: ankle plantar flexion
   d. Deep tendon reflexes and pathological reflexes should be tested.
   e. A positive bulbocavernosus reflex or return of anal wink reflex signals the end of spinal shock.
   f. Bladder and bowel functions should be noted. Any disturbance indicates a significant spinal injury.
   g. Radiographic evaluation includes the following:
      - Anteroposterior and lateral views of the involved spine
      - CT scans are done to assess the osseous integrity
      - Magnetic resonance imaging (MRI) shows finer details about spinal cord involvement and ligamentous disruption.
   h. SCIWORA stands for Spinal Cord Injury Without Radiographic Abnormalities. It may be due to oedema in the spinal canal, cord contusion and cord transection, which cannot be detected by routine radiographs. MRI detects it.

INJURIES OF THE CERVICAL SPINE

The common type of cervical spine injury is the hyperflexion injury caused by a heavy weight falling on the head or by landing on the head while diving in shallow swimming pools. It is usually a dislocation at the lower cervical level, the commonest being at C5, C6 and C7. Dislocations at this level cause severe injuries to the spinal cord, resulting in quadriplegia (Figs 21.2 and 21.5).

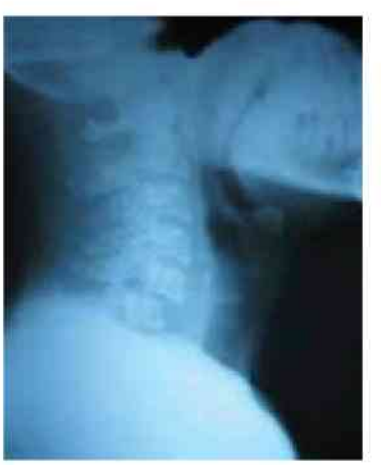


FIGURE 21.5 X-ray of the cervical spine lateral view showing C5 fracture with C5 over C6 subluxation.

Clinical Features

The patient presents with a history of trauma. There will be extreme pain and muscle spasm in the neck and tenderness at the site of the dislocation. There will be restriction of neck movements. A neurological examination is done to assess the extent of paralysis. This will show evidence of nerve irritation at the segment just above the level of cord damage, causing muscle spasm and hyperaesthesia and typical postures of the arms.

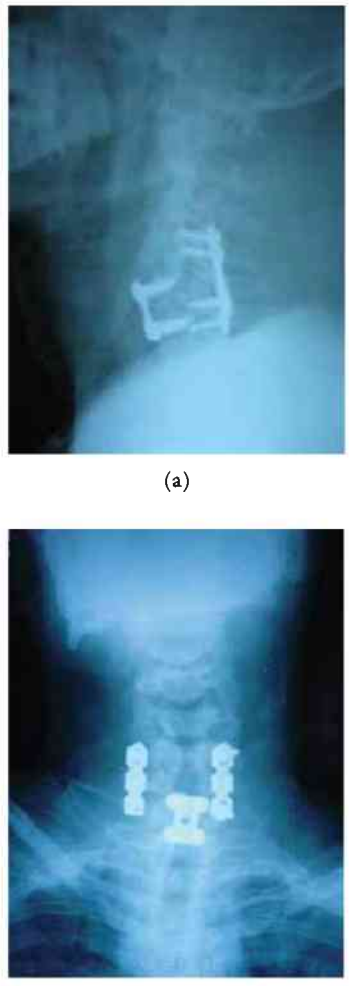
If the cord will get damaged at the C6 level, there will be evidence of irritation at the C5 segment. There will be spasm of deltoid, flexors of the elbow and the supinators. The patient will lie with the arms abducted, externally rotated and the forearm supinated. If the injury is at the C7 level, the C6 segment shows irritation with spasm of adductors of the shoulder and pronators of the forearm. The patient lies with the arms adducted and the forearm lying pronated over the body. Both lower limbs will be paralysed. In injuries at C4 and C5, there will be respiratory distress due to diaphragmatic paralyses.

Radiological Features

Radiological examination will show the level of damage and the type of injury and displacement; lateral and anteroposterior views (from C1 up to the upper border of T1) are taken (Fig. 21.6). In cases of upper cervical injuries, always take radiographs of the skull to exclude fractures at the base of the skull. CT scan of the cervical spines is done to check for bony integrity. The use of MRI can demonstrate the structural damage to the spinal cord and ligaments at the level of injury.

Treatment

- *First aid:* These patients must be handled with extreme care. All patients with head injury and unconsciousness should be assumed to have cervical spine injury and immobilised in a hard cervical collar unless proven otherwise. The head and neck must be steadied by sand bags, pillows or cervical spine boards, and no flexion movement must be allowed even momentarily during transport or radiography.



(a)

(b)

FIGURE 21.6 (a, b) X-ray of cervical spine anteroposterior and lateral views showing global anterior and posterior stabilisation.

The aim of treatment is to reduce the dislocation and maintain it till stability is achieved. The best method of treatment is to apply skull traction with Gardner Wells skull tongs. The patient is treated in a spinal turning frame.

Glisson Sling Halter Traction

Where facilities for a skull traction are not available, traction to the neck is applied with a sling (Fig. 21.7). The sling exerts traction through the chin and the occiput and hence is

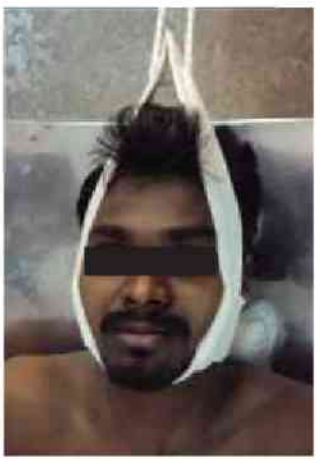


FIGURE 21.7 Clinical photograph showing Glisson's sling traction.

not comfortable to the patient and cannot be kept too long.

Lateral view radiographs of the cervical spine are taken to check for reduction of the dislocation. If the dislocation persists, the weight is increased and radiographs repeated. When the dislocation is found reduced in the radiograph, the neck is slowly extended over a pad support and the weight reduced to a minimum to maintain the reduction.

The patient is turned periodically to prevent bedsores. After about 2 weeks when the local pain has subsided, the traction is removed and an extended plaster collar is applied. The ambulation of the patient is timed according to the recovery of the paralysis. The plaster is kept for 2 months.

Important points that should be taken care of during initial management of the patients with cervical trauma are as follows:

- Immobilisation of the cervical spine
- Safe transportation to a trauma care centre
- Steroid infusion if the patient presents within 8 hours after injury
- Injection methyl prednisolone—30 mg/kg bolus in 1/2 hour followed by a pause for 1/2 hour and 5.4 mg/kg/h for the next 23 hours
- Application of skull tongs—traction for appropriate fractures and dislocation

Surgical Treatment

Indications

- Unstable injuries
- Unreduced dislocations
- Fractures and dislocation with neurological deficit

Sometimes, even heavy prolonged traction fails to reduce the dislocation due to locking of the articular processes. In such cases, operative reduction is carried out.

After open reduction, the spinous processes of the vertebra are held together by wiring. The skull traction is dispensed with. The cord lesion with its attendant quadriplegia will be managed as in the case of paraplegia. Surgical techniques include the following:

1. Anterior decompression by removing the fractured vertebral body compressing the cord, and stabilisation with bone grafting and instrumentation using cage pedicle screws and rods
2. Posterior stabilisation with instrumentation with pedicle screws and rods

Lower Cervical Spine Injuries

Lower cervical spine injuries are classified by Allen and Ferguson into the following types:

- Compression flexion injuries
- Vertical compression injuries
- Distractive flexion injuries
- Compressive extension injuries
- Distractive extension injuries
- Lateral flexion injuries

Clay-Shoveler's Fracture

Clay-shoveler's fracture is an avulsion of the spinous process of lower cervical vertebra or upper thoracic vertebra following violent muscular contraction (Fig. 21.8).

Chalk-Stick Fracture

In ankylosing spondylitis, calcification of ligamentous structure occurs. A fracture through these calcifications is termed 'chalk-stick fracture'.

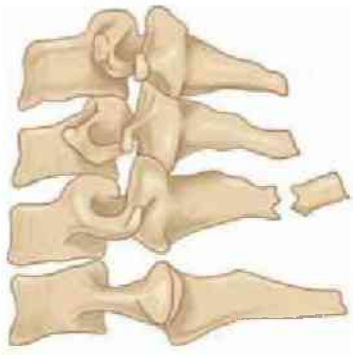


FIGURE 21.8 Clay-shoveler's fracture.

Upper Cervical Spine Injuries

Injuries of the C1, C2 level are very serious as any displacement can cause fatal neurological complication. These fractures may be associated with fractures of the occiput and the base of skull.

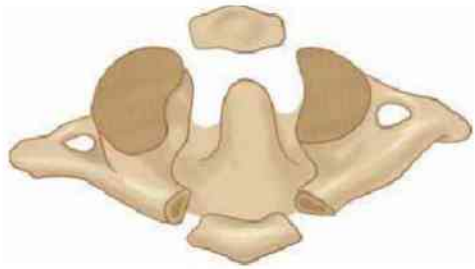
Fracture Atlas (C1) (Jefferson's fracture)

Fractures of the atlas are due to vertical compression violence. The fractures occur at the anterior or posterior arch causing separation of the lateral masses. The cord escapes injury, and usually there is no neurological deficit. The management is conservative (Fig. 21.9).

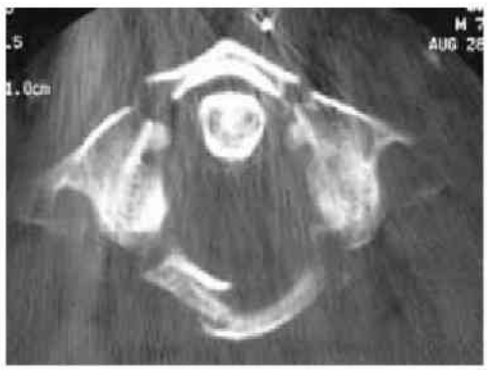
Fracture Axis (C2)

The important fractures are as follows:

- *Fracture odontoid:* This can occur at the tip or the base. The fracture can result in atlantoaxial dislocation (Fig. 21.10). Pain occurring at the base of skull is the usual complaint. An open mouth odontoid view X-ray film suggests the diagnosis. This is treated by skull traction followed by plaster.
- *Hangman's fracture dislocation:* This is a traumatic spondylolisthesis at the C2, C3 level (Fig. 21.11). It may be associated with the cranial nerve, vertebral artery and facial injuries. It is treated by reduction by skull traction and immobilisation in plaster.



(a)



(b)

FIGURE 21.9 (a) Illustration and (b) CT scan showing Jefferson's fracture.

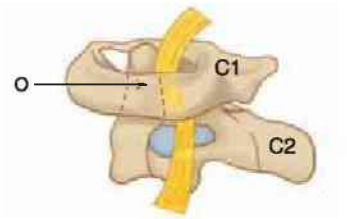
Atlantoaxial Dislocation

Pure atlantoaxial dislocation without fracture of the odontoid process causes sudden cord compression and is fatal. The dislocation may be rotatory subluxation, translation in sagittal plane or multiplanar instability. A pure dislocation of C1, C2 is dangerous as it results in compression of the spinal cord (Fig. 21.10a). These injuries are associated with damage to alar and transverse ligament. If there is a fracture at the base of odontoid also, it moves along with the atlas and the cord escapes injury (Fig. 21.10b).

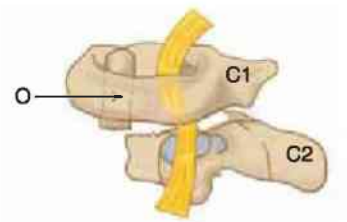
Incomplete Spinal Cord Syndromes

The paralysis is considered complete when the bulbocavernous reflex is positive with no sacral sensation or motor function.

Incomplete spinal cord syndromes are characterised by the presence of some motor or sensory function distal to the cord injury.



(a)



(b)

FIGURE 21.10 Diagrammatic representation of (a) atlantoaxial dislocation and (b) atlantoaxial dislocation with fracture odontoid (O—odontoid process).

Central Cord Syndrome

The central portion of the spinal cord is destroyed with quadriparesis involving upper extremity more than lower extremities.

Brown–Sequard Syndrome

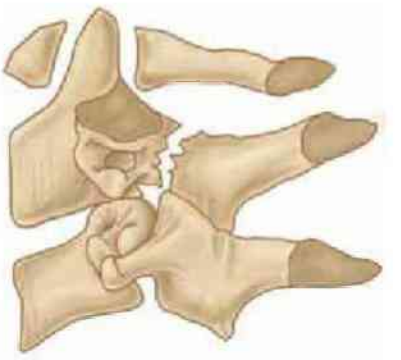
One-half of the spinal cord is damaged with motor weakness on the side of lesion with contralateral loss of pain and temperature sensation and ipsilateral loss of vibration sense.

Anterior Cord Syndrome

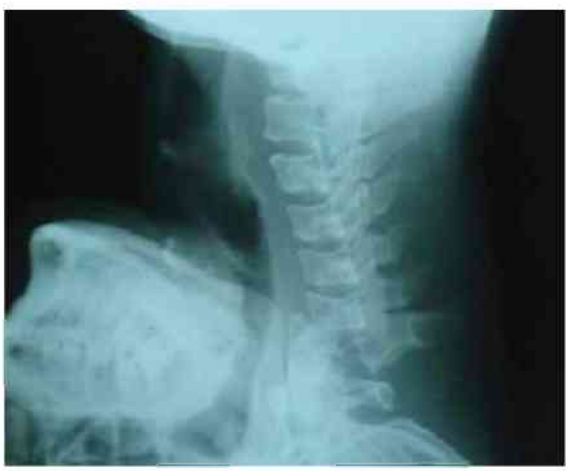
The anterior portion of the cord is injured with complete motor loss and loss of pain and temperature below the lesion.

Posterior Cord Syndrome

The posterior portion of the cord is injured with loss of proprioception and vibration sense.



(a)



(b)

FIGURE 21.11 (a) Illustration and (b) radiograph showing Hangman's fracture.

Conus Medullaris Syndrome

Injury to the sacral cord occurs with areflexic bladder, bowel and lower extremities.

Cauda Equina Syndrome

Injury to the part between conus and lumbosacral nerve roots occurs resulting in areflexia and bladder and bowel involvement.

KEY POINTS

- Spine can be divided into three columns (Dennis classification).
- Fractures of the thoracolumbar spine may be caused by flexion, flexion rotation, vertical compression and extension violence.
- In fracture dislocation of vertebral column, any of the following injury may occur: Spinal cord injury, injury to cauda equina and combined injury to cord and cauda equina.
- In fracture dislocations at the L2 level and below, the spinal cord escapes injury and there is only a cauda equina lesion.
- Early management in paraplegia aims to prevent bedsores and urinary sepsis.
- Bedsore or decubitus ulcers are localised areas of cellular necrosis resulting from prolonged excessive stress on soft tissues.
- A positive bullocavernosus reflex signals end of spinal shock. Bladder and bowel function should be noted.
- In C4 and C5 injuries, there will be respiratory distress due to diaphragmatic paralysis.
- All head injury and unconscious patients should be assumed to have cervical spine injury and immobilised with cervical collar unless proven otherwise.
- Steroid infusion is given in patients with cervical spine injury presenting within 8 hours after injury. Inject methylprednisolone 300 mg/kg bolus in 1/2 hours followed by 5.4 mg/kg/h for next 23 hours.
- Anterior decompression and stabilization, and posterior stabilisation are surgical optional for cervical spine injury.
- Avulsion of spines process of lower cervical and upper thoracic vertebra due to muscular contraction is called clay-shoveler fracture.
- In ankylosing spondylitis, calcification of ligaments occurs. Fractures in these calcifications are termed 'chalk stick fractures'.
- Fracture of atlas is due to vertical compression violence called Jefferson fracture.
- Traumatic spondylolisthesis of C2 and C3 levels is called Hangman's fracture dislocation.
- Atlantoaxial dislocation without fracture of the odontoid process causes sudden cord compression which is fatal.
- Brown–Sequard syndrome is defined as ipsilateral motor weakness with contralateral loss of pain and temperature due to damage to one-half of the spinal cord.
- Cauda equina syndrome: Injures to part between conus and lumbosacral nerve roots results in areflexia; bladder and bowel involvement is also seen.

MULTIPLE CHOICE QUESTIONS

1. The commonest cause of paraplegia is
   a. Tuberculosis
   b. Trauma
   c. Secondaries
   d. Transverse myelitis
2. When a person reports with vertebral fracture hemiplegia and urinary retention, the acute measure to be taken is
   a. Supra pubic cystostomy
   b. Catheterisation
   c. Hot fomentation
   d. Condom drainage
3. Jefferson fracture occurs at
   a. C1
   b. C2
   c. C1, C2
   d. C2, C3
4. The most common force involved in fracture of the spine is
   a. Flexion
   b. Extension
   c. Rotation
   d. Compression
5. The most unstable spinal injury is
   a. Flexion injury
   b. Flexion rotation injury
   c. Extension injury
   d. Flexion distraction injury

CHAPTER 22

Fractures of the Pelvis

ANATOMY OF PELVIS

Pelvis is formed by

- two innominate bones,
- sacrum.

Innominate is formed by three parts: ilium, ischium and pubis. The two innominate bones are joined anteriorly by pubic symphysis and posteriorly with the sacrum by sacroiliac joints.

Pelvic Ring

Pelvic ring is formed by two innominate bones and the sacrum (Fig. 22.1). From anterior to posterior, it consists of the following:

- Pubic symphysis
- Pubic crest
- Pectineal line of pubis
- Arcuate line of the ilium
- Sacral ala
- Sacral promontory

Pelvic ring is supported by ligaments that are amongst the strongest in the body. Anterior and posterior sacroiliac ligaments prevent displacement of the sacroiliac joint. Sacrotuberous and sacrospinous ligaments prevent rotational deformation at the hemipelvis.

The pelvic ring can be divided functionally into a posterior weight transmitting segment and an anterior segment serving only for muscular attachment. Injuries to the posterior segment

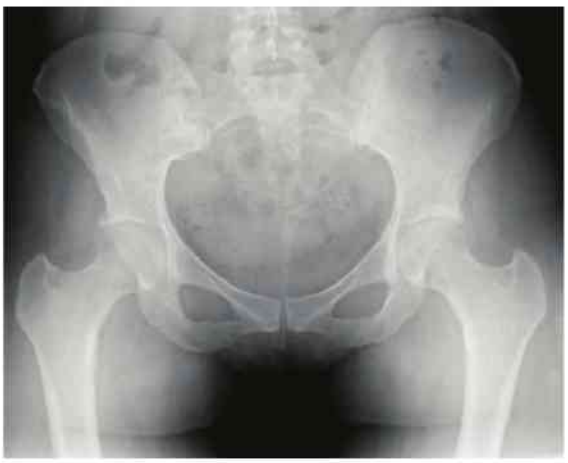


FIGURE 22.1 X-ray of pelvis with both hips' anteroposterior view showing a normal pelvis.

are important from the locomotion point of view and are more disabling as they involve the weight transmitting part. Injuries to the pelvis can also result in serious injuries to the pelvic viscera such as the bladder, urethra, rectum and the veins.

FRACTURES OF PELVIS: MECHANISM AND INCIDENCE

Fractures of the pelvis are caused by crush injuries and are becoming common due to automobile accidents. The direction of the violence can be anteroposterior compression, lateral (side-to-side) compression and vertical shearing.

When the force is applied in the anteroposterior direction, the initial injury is to the anterior segment of the pelvis, causing fractures of the pubic rami or separation of the symphysis pubis. With further violence, the posterior half of the ring also gives way resulting in a disruption of the sacroiliac joint or fracture of the ilium near the sacroiliac joint. When the violence is lateral compression, the disruption involves both segments. Vertical shearing violence causes an upward displacement of one-half of the pelvis. Other types of injuries include the following:

- Open book injuries
- Avulsion injuries
- Injuries caused by falls
- Crush injuries

Classification

Pelvis fractures are classified depending upon the anteroposterior and vertical stability of the pelvic ring:

- Single segment fracture of the pelvic ring (stable fracture)
- Double segment fracture (unstable fracture)
- Avulsion fracture
- Fracture of the acetabulum
- Fractures of sacrum and coccyx

1. Tile's classification
   - *Type A:* Stable
     A1 = Fractures of the pelvis not involving the ring
     A2 = Stable, minimally displaced fractures of the ring
   - *Type B:* Rotationally unstable and vertically stable
     B1 = Open book
     B2 = Lateral compression; ipsilateral
     B3 = Lateral compression; contralateral (bucket handle type)
   - *Type C:* Rotationally and vertically unstable
     C1 = Rotationally and vertically unstable = Unilateral
     C2 = Rotationally and vertically unstable = Bilateral
     C3 = Associated with an acetabular fracture
2. Young and Burger system: It classifies pelvic injuries based on radiography:
   - Lateral compression injury
   - Anteroposterior compression injury
   - Vertical shear
   - Combined mechanical injury

FRACTURES OF THE PELVIC RING

1. *Single segment fracture of the pelvic ring:* Here the fracture is either in the anterior segment or in the posterior segment. These are stable fractures.
   - Fracture of the pubic rami (Figs 22.2a and b)
   - Separation of symphysis pubis
   - Single fracture of the ilium
2. *Double segment fracture of the pelvic ring:* Here both the segments of the ring are injured. These are unstable fractures.
   - Separation of symphysis pubis with fracture of the ilium parallel to the sacroiliac joint (Fig. 22.2c)
   - Fracture of the pubic rami with separation of the sacroiliac joint (Fig. 22.2d)
   - Separation of symphysis pubis and sacroiliac joint (Fig. 22.3)

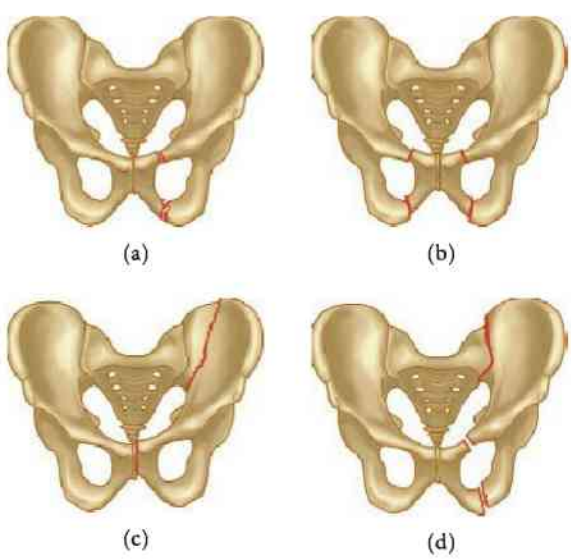


FIGURE 22.2 Illustrations showing types of fracture pelvis. (a, b) Single segment fractures and (c, d) double segment fractures.

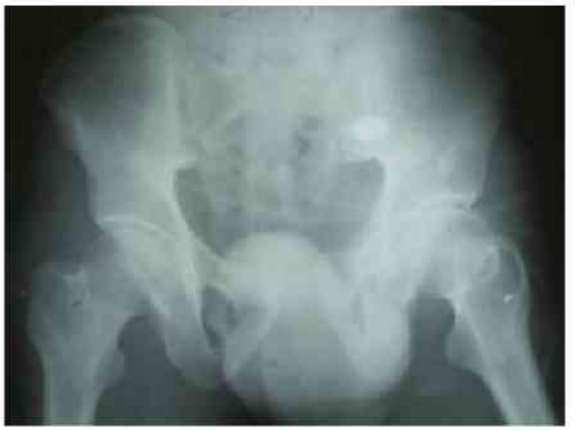


FIGURE 22.3 X-ray of pelvis anteroposterior view showing separation of pubic symphysis and disruption of the right sacroiliac joint.

FIGURE 22.4 Clinical photograph of a severe high velocity trauma in which pelvic fracture becomes a compound injury due to a perineal laceration.

3. *Avulsion fracture*
   - Avulsion of the anterior superior iliac spine by sartorius
   - Avulsion of the anterior inferior iliac spine by rectus femoris
   - Avulsion of ischium by hamstring muscle

Avulsion fractures are due to muscular violence avulsing the muscle origin of anterior superior or anterior inferior iliac spines. These occur commonly as sports injuries. Usually, these need symptomatic treatment only with no active intervention.

Clinical Features

The patients are usually young adults with a history of a runover injury or crush injury caused by falling weight. The patient will be in shock and there will be evidence of local bruises. Shock in unstable fractures is often due to intrapelvic haemorrhage from the veins in the pelvic wall. There will be localised bony tenderness at the symphysis pubis or sacroiliac joint. Pelvic compression and distraction tests will be positive and painful. One should always look for any evidence of rupture of the bladder or urethra and injury to the rectum.

Bladder and Urethral Injury

In injuries to the bladder complicating these fractures, extraperitoneal rupture is the commonest, resulting in extravasation of the urine into the extraperitoneal space (Fig. 22.4). Rupture of the urethra may be intrapelvic, where the extravasation is similar to that in the extraperitoneal rupture of the bladder; it may be extrapelvic where the extravasation occurs into the perineum extending into the groin and anterior abdominal wall. The patient will be unable to pass urine.

Injury to Urethral Meatus

Urethral meatus should be examined for blood. Similarly, the colour of the urine must be noted. Haematuria is a sign of urinary tract injury. Digital rectal and vaginal examination should also be done to rule out bony fragments tearing into rectum and vagina, respectively.

Diagnosis

- Pelvic fracture is one among the few bony injuries that can lead to a patient's death.
- Patients with major pelvic injures (Types B and C) may present in a state of shock and require emergency resuscitation.
- In haemodynamically stable patients (Type A injuries), the following tests can be done.

Pelvic Compression Test

- Pelvic compression test exposes instability in internal rotation.
- The test is carried out by compressing in the two iliac wings towards each other. In a positive test, the patient complains pain and the examiner can feel the instability.

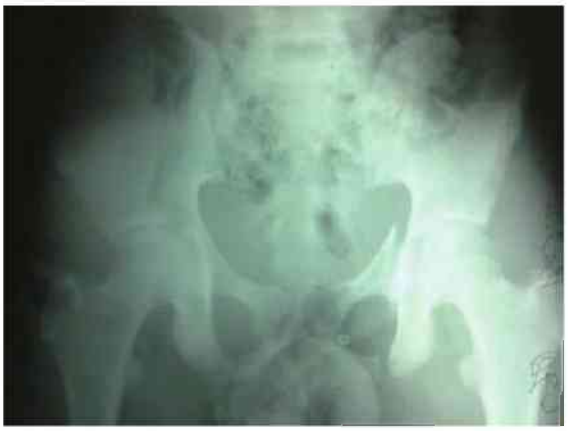


FIGURE 22.5 X-ray of pelvis anteroposterior view showing bilateral superior and inferior pubic rami fracture with ileum fracture on the left side.

Pelvic Distraction Test

- Pelvic distraction test exposes instability in external rotation.
- Two iliac wings are distracted out away from each other to elicit pelvis or instability.
- Perineum should be inspected for lacerations and blood.

Radiograph

Radiograph of the pelvis anteroposterior view will show the type of fracture (Fig. 22.5). Pelvic inlet, outlet and oblique views will add more information about the fracture. A cystogram may be needed to confirm the diagnosis of injury to the urinary tract.

Computed Tomography

Computed tomography reveals more information about the extent of fractures seen in an X-ray film and some subtle fractures not seen in X-ray (Fig. 22.6).

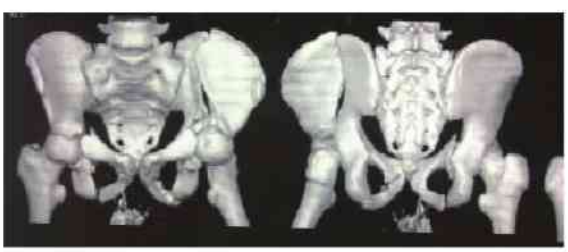


FIGURE 22.6 CT of pelvis 3D reconstruction of the same patient, whose X-ray is given in Figure 22.5, clearly showing ileum fracture on the left side.

Management of Stable Pelvic Fracture (Conservative Management)

Conservative management is followed in cases of *single segment fractures* of the *ilium* or *pubis*. The management plan is also conservative in patients with an intact pelvic ring or with minimally displaced fractures (isolated fractures), for example

- Pubic ramus fracture
- Iliac wing fracture

Strict bed rest is advised for 3–4 weeks and then gradual mobilisation is begun.

Management of Unstable Pelvis Fracture

Transportation From the Scene of Trauma

In major pelvic injuries, pre-hospital transport of the patient is so important because any handling of pelvis during transport may risk the life of the patient.

Transportation is facilitated by application of either of the following:

- Pelvic binder
- Military Anti Shock Trousers (MAST)

Emergency Management in the Trauma Ward

- Assessment of airway, breathing, circulation (ABC)
- Immediate resuscitation with crystalloids initially, followed by colloids and compatible blood (if not available use O –ve)
- Pelvic ring stability assessment and application of an external fixation in the ward, before shifting the patient anywhere.

As this injury is due to severe violence, shock must be looked for and prevented. Complications of visceral injuries must be looked for and treated. The bony injuries are managed as follows.

Double Segment Fracture with Displacement

Many of these show an outward rotation of the hemipelvis as well as an upward displacement. The pelvis opens out like a book as the patient lies supine.

Lateral recumbency reduces the rotation and displacement. Upward displacement is corrected by applying tibial traction to the leg. The patient

TABLE 22.1 Definitive Treatment for Specific Pelvic Fractures

Type of Fracture	Treatment
Pubic fracture	It refers to pubic symphysis disruption and separation. It is treated by open reduction and internal fixation by plating.
Crescent fracture	It is a fracture involving the posterior iliac wing. It is caused by lateral compression injury and is treated by closed reduction and screw fixation.
Jumper's fracture	It is a fracture dissociation of the central portion of the sacrum from the lateral portions. Usually, it occurs after fall from height. It is treated by screw fixation.

is supported in a pelvis sling or hammock, which helps in nursing and the traction is continued. After 6 weeks, a sacroiliac support is prescribed and the patient made ambulant in stages. The definitive treatment of specific pelvic fractures is given in Table 22.1

Surgical Treatment

Surgical treatment is indicated in cases of unstable injuries of the pelvis. These are stabilised by

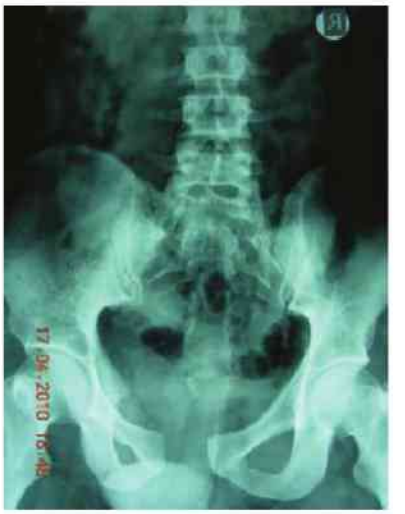
- external fixation,
- internal fixation.

External Fixation

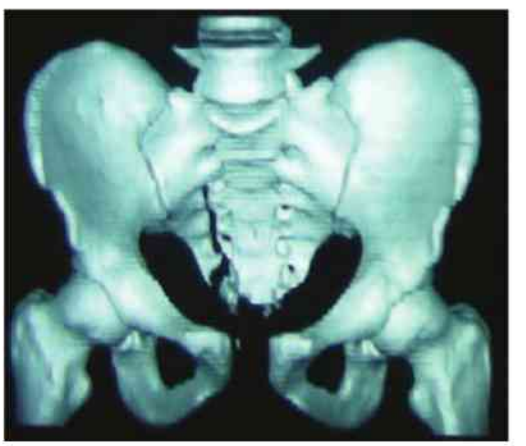
External fixation is done using pelvic frames and fixation pins passed through the iliac bones. This helps in stabilising the pelvic ring. In the acute stage, it also helps in reducing the internal haemorrhage from pelvic wall veins. In the later stage, it is also a definite treatment which lasts for 6–8 weeks.

Internal Fixation

In selected cases of gross displacement of symphysis pubis (Fig. 22.7) and the sacroiliac joint, open reduction and fixation by special plates and screws are done (Fig. 22.8). This makes the mobilisation of the patient easier and possible earlier.



(a)



(b)

FIGURE 22.7 (a) X-ray and (b) CT 3D reconstruction of the pelvis showing pubic diastasis (separation of symphysis pubis).

Complications

Early complications include injury to bladder or urethra, injuries to rectum or colon or injuries to the intrapelvic veins. A rupture of the bladder or urethra will need suprapubic cystostomy and urgent surgical repair. Fat embolism is another serious complication.

Late complications include (1) malunion resulting in sacroiliac pain, leg shortening and altered gait and (2) non-union resulting in instability. The complications of pelvic fracture and its effects are summarised in Table 22.2.

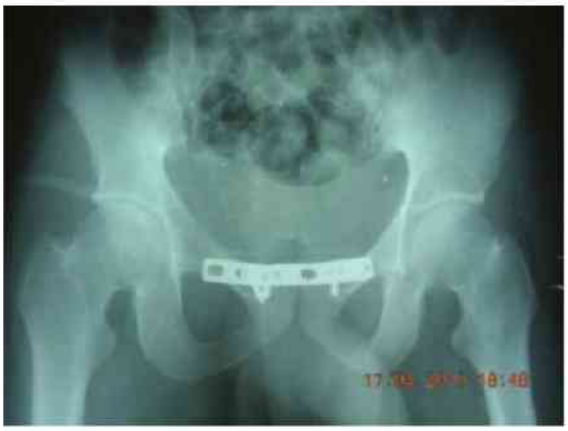


FIGURE 22.8 X-ray of the pelvis anteroposterior view showing pubic diastasis—ORIF done with a dynamic compression plate.

TABLE 22.2 Complications of Pelvic Fracture

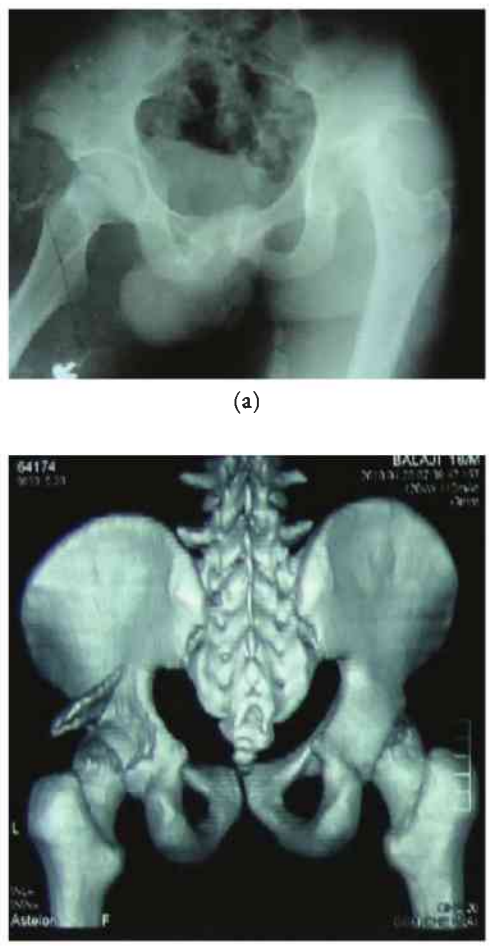
Type of Complication	Effect of Complication
Haemodynamic instability in unstable pelvic fracture	Puts the life into risk
Genitourinary tract injuries	Urethral injury Bladder injury—may be either intraperitoneal or extraperitoneal rupture Vaginal injury
Gastrointestinal tract injuries	Rectal laceration may occur with a spike of bone tearing the wall
Injury to pelvic nerves (nerves of the lumbosacral region)	This may result later into • Impotence • Lower extremity motor and sensory dysfunction • Bladder and bowel dysfunction
Deep vein thrombosis (due to prolonged immobilisation)	This may cause thromboembolic disease

FRACTURES OF THE ACETABULUM

Classification

The following is the recent classification (Tile).

- *Type I (Posterior type):* This includes fractures of the posterior wall or the whole column. These may be associated with posterior dislocation of the hip (Fig. 22.9).
- *Type II (Anterior type):* This includes fractures of the anterior wall or column of the acetabulum. These may be associated with anterior dislocation of the hip joint.
- *Type III (Transverse type):* This includes fractures of the floor of the acetabulum. This is associated with central dislocation of the hip.



(a)

(b)

FIGURE 22.9 (a) X-ray of pelvis anteroposterior view showing fracture dislocation of the left hip. (b) CT 3D reconstruction clearly showing the posterior wall acetabular fracture taken after reduction of the hip dislocation.

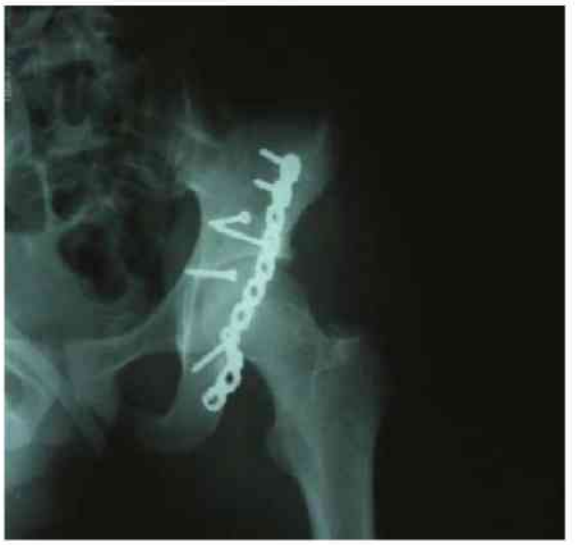


FIGURE 22.10 X-ray of left hip anteroposterior view showing anterior wall fracture acetabulum fixed by plate and screws.

Treatment

In recent years, acetabular fractures are actively treated, as malunion leads to disabling osteoarthrosis of the hip joint. The assessment requires additional special oblique view radiographs of the pelvis. The treatment aims to produce a congruous articular surface of the acetabulum. This is done conservatively by femoral pin traction. Unstable fractures require open reduction and internal fixation by special plates (Fig. 22.10).

FRACTURES OF THE SACRUM

Major fractures of the pelvis are often associated with fractures of the sacrum. The fracture usually involves the ala of the sacrum through the sacral foramen. There may be associated injuries with the sacral nerves. Direct trauma to the back of the pelvis produces fractures of the sacrum with or without displacement. The sacral fractures do not need any special treatment apart from the management of the major pelvic injury.

FRACTURE COCCYX

Injury to the coccyx is commonly due to slipping and falling on the sitting posture while coming down the staircase. The patient complains of pain in the tail end of the spine and there is tenderness in the coccyx. The pain often becomes chronic and causes marked discomfort on sitting. This is called coccydynia. Treatment is symptomatic with analgesics and soft cushioned seats. Exercises to the gluteus maximus muscles help by toning up the muscles which protect the coccyx from direct pressure on sitting.

KEY POINTS

- Pelvis is formed by two innominate bones and a sacrum. Innominate bone consist of three parts: ilium, ischium and pubis.
- Pelvic ring is supported by strong interosseous ligaments. Anterior and posterior ligaments prevent displacement at sacroiliac joint.
- Sacrotuberous and sacrospinous ligaments prevent rotational deformation at the hemipelvis.
- Fracture of pelvis may involve anterior segment or the posterior segment (single segment fractures).
- In some cases, fracture pelvis involves both the segments (double segment fracture).
- Injuries to the pelvis can also result in serious injuries to the pelvic viscera such as the bladder, urethra and rectum.
- Jumper's fracture is a fracture dissociation of the central portion of the sacrum (from the lateral portions). The fracture is managed by screw fixation.
- Pelvic fractures may be stable or unstable. Stable pelvic fractures are treated conservatively while the unstable injuries are treated surgically (internal or external fixation).

MULTIPLE CHOICE QUESTIONS

1. Complication of acetabular fracture is
   a. Avascular necrosis of head of femur
   b. Sciatic nerve injury
   c. Secondary osteoarthritis of hip joint
   d. All of the above
2. All of the following areas are commonly involved sites in pelvic fracture except
   a. Pubic rami
   b. Alae of ileum
   c. Acetabula
   d. Ischial tuberosities
3. Which is the most common complication of fracture of the ischiopubic ramus pelvic bone?
   a. Rupture of urinary bladder
   b. Rupture of prostatic urethra
   c. Rupture of membranous urethra
   d. Injury to penile urethra
4. Late complication of acetabular fracture is
   a. Avascular necrosis of the head of femur
   b. Avascular necrosis of the iliac crest
   c. Fixed deformity of the hip joint
   d. Secondary osteoarthritis of the hip joint

CHAPTER 23

Polytrauma

Polytrauma is either multiple fractures in limbs and spine or multiple system injuries, involving head, thorax abdomen and pelvis in victims of accidents. In general, it is defined as injury to at least two organ systems with a potential life-threatening condition of the patient. Polytrauma follows

- road traffic accidents,
- train traffic accident,
- warfare blast injury,
- fall from height.

Road transportation is massively increasing in our cities and highways due to industrialisation and urbanisation. Road traffic accidents have become a daily occurrence, thus taking an increasing toll on human lives and limbs.

Any doctor could be confronted with a situation resulting from a two-wheeler accident injuring one or two persons or even a major accident involving trucks or buses. It is essential that one is prepared to provide the minimum possible emergency care to accident victims. Injuries in a victim of polytrauma can be described by injury severity score.

INCIDENCE

Polytrauma is mostly caused by high-velocity impact of automobiles and other vehicles on the roads. The increasing use of two wheelers by the younger generation has increased the incidence of multiple trauma. It can also result from building collapse or a major railway accident.

MANAGEMENT OF A POLYTRAUMA PATIENT

The primary care of such patients may have to be done in the casualty department of even taluk and district hospitals. A sudden arrival of a large number of accident victims to the casualty department of even big hospitals can be upsetting to the meagre staff on duty. In a major accident, the arrival of a large number of polytrauma cases in groups needs quick responses by the junior doctors on duty while alerting and mobilising help from senior staff. Guidelines for management of a polytrauma patient are set under the advanced trauma life support system.

It includes the following:

- Pre-hospital care
- Rapid primary evaluation
- Secondary evaluation
- Definitive care

Every medical man should be reasonably competent in the primary survey, triage and emergency care of the injured patient in the reception room.

Primary Survey

Primary survey is a quick survey of the patient to assess him regarding the seriousness of his injury by looking for the presence of the following:

- Airway obstruction
- Breathing distress due to flail chest and pneumothorax

- Circulatory failure and haemorrhage
- Disability—neurological, such as coma and paralysis
- Fractures of limbs and spine

This survey is meant to quickly assess the threat to life and limb by the injuries. The pulse is counted and respiration is observed and resuscitation initiated.

Airway

Airway clearance is the first life-saving step in resuscitation. This is done by

- extension of the neck,
- pulling forward of the jaw,
- removing any foreign body in the mouth,
- pressing the tongue down.

In addition to manual clearance and positioning of the head, obstruction in the throat by blood, mucus or vomitus may also need clearance using mechanical suction apparatus. In some cases, endotracheal intubation will have to be done to clear the vomitus blocking the tracheobronchial passages. Endotracheal intubation is a life-saving procedure which must be learnt by every intern. With this, the stertorous breathing will become normal and oxygenation will improve.

The next step in life saving is the restoration of ventilation and oxygenation which are compromised in cases of injuries to chest resulting in a flail chest or a pneumothorax. Stabilising the chest wall, by strapping over a pad and letting out the air or blood in the pleural cavity by a needle, will restore respiratory efficiency and oxygenation. The injured extremities are stabilised by putting the splint.

Circulation

One should simultaneously attend to the circulatory failure due to open haemorrhage and hypovolaemic shock as evidenced by pallor sweat in the forehead, weak pulse and low BP. Arrest external haemorrhage by direct pressure over a clean pad. Quickly start IV fluids through an open vein if necessary and arrange for blood matching and transfusion.

Neurological Status

The next step is the quick survey of the neurological status noting the following:

- Disturbance of consciousness
- Pupillary changes
- Blood or cerebrospinal fluid oozing from nose and ears
- Paralysis and spontaneous or reflex movement of the limbs

One should record the level of consciousness at the first examination using the Glasgow Coma Scale (Fig. 23.1).

In all cases with diminished consciousness, look for other signs of intracranial damage like dilated pupils. Intracranial haemorrhage, if present, may necessitate emergency decompression by a neurosurgeon.

Secondary Evaluation

A thorough and complete examination of the patient should be done.

1. Fractures involving extremities of bones, pelvis and spine are evaluated with radiographs and proper splinting should be done.
2. Distal pulses are evaluated manually or by a hand-held Doppler system.
3. Vascular injuries are dealt with emergency surgery.
4. Broad spectrum antibiotics and tetanus/gas gangrene prophylaxis are instituted in open wounds.
5. A pelvic external fixator should be applied in haemodynamically unstable injuries.
6. Detailed neurological examination is carried out and radiological evaluation with CT and MRI are done. Craniotomy for intracranial haemorrhage should be done
7. Detail abdominal examination should be done. Blunt injury abdomen causing damage to the viscera should be evaluated initially by ultrasonography. Emergency laparotomy and exploration if required should be carried out
8. Detailed chest examination should be done to rule out major cardiothoracic injuries such as
   - cardiac tamponade,
   - tension pneumothorax,

- haemothorax,
- flail chest.

9. Appropriate emergency management including pericardiocentesis, pleurocentesis, ICD tube insertion and emergency thoracotomy should be carried out.
10. Facial injuries and eye injuries should be managed according to the severity of the injury.

Glasgow Coma Scale: Level of Consciousness

Points to look for

Consciousness Score

I. Eye Opening
Watch him open the eyes
a. Does not open at all 1
b. Opens in response to pain 2
c. Opens to verbal order 3
d. Opens spontaneously (Normal) 4

II. Verbal Response on talking: Talk to him. Response may be
a. No response 1
b. Incomprehensible blabbering 2
c. Inappropriate words 3
d. Disoriented conversation 4
e. Oriented conversation (Normal) 5

III. Motor Response: Tell him to move the limb. Response may be
a. No response at all 1
b. Limb goes into extension (Decerebrate rigidity) 2
c. Limb goes into flexion (Decorticate rigidity) 3
d. Flexion withdrawal 4
e. Able to localise pain 5
f. Able to obey and move (Normal) 6

Normal consciousness: Total score I + II + III, i.e. 4 + 5 + 6 = 15

Lesser score means worse condition, i.e. deeper coma

a. Mild coma has a score of 13–15
b. Moderate coma is when the score is between 9 and 12
c. Scores 3–8 denote deep coma and means that the patient is critical.

FIGURE 23.1. Glasgow Coma Scale.

Definitive Care

Definitive treatment of the fractures can be done after the general condition of the patient is stabilised as early as possible.

The priorities in skeletal trauma are as follows:

- External fixator application for haemodynamically unstable pelvic fractures
- Reduction of dislocations of joints with neurovascular compromise
- Repair of vascular injuries
- Open fracture, debridement and stabilisation
- Stabilisation of unstable spine

TRIAGE

Triage is the process of clinically sorting out a group of trauma victims at the reception room according to the severity of the injuries and the urgency of threat to their lives or limbs. The priorities for emergency care are decided and patients shifted to the emergency operation theatre or resuscitation and treatment rooms. It also means avoiding the ineffective use of time and scarce resources on hopelessly sinking victims with terminal jaw breathing and concentrating attention on those who have a better chance to survive.

The criteria for triage include the following:

- BP <90 mm Hg
- Pulse >120/min
- Respiratory rate >35 or <12/min
- Penetrating injuries
- Unconscious patient
- Traumatic amputation of extremities
- Flail chest
- More than or equal to two long bone injuries

Ethical Dilemma

Allowing a terminally sinking patient to die may appear unethical, but neglecting some patients, who have a greater chance of survival, thus resulting in avoidable deaths is more unethical.

RESUSCITATION

Team work is very essential to minimise waste of time. By now, vigorous efforts must have been

taken to resuscitate patients in respiratory distress and shock. The aim is to quickly restore oxygenation to the vital tissues of brain, heart and kidneys by IV fluid and blood, before irreversible damage occurs to vital organs.

When the condition is stabilised, the patient needing surgery is shifted to the operation theatre. Others are shifted to the Trauma ward.

Head injury is assessed according to the level of consciousness. In all cases of head injury, associated injury to cervical spine must be looked for. If present, extra care must be taken in handling and turning the patient to prevent cord injury as a 'second accident'. An emergency intervention is decided by the neurosurgeon in cases of depressed fracture skull as well as middle meningeal haemorrhage.

Faciomaxillary injuries often cause obstruction to breathing, needing immediate life-saving attention. Faciomaxillary dental and ophthalmic surgeons have to team up for stabilising the jaw fractures and repairing the wounds of the face, tongue and eyes. In chest injuries, flail chest due to multiple fractures in the ribs is treated by strapping to stabilise the chest wall. Open sucking wounds causing respiratory distress are covered by sterile pads and strapping. Acute pneumothorax must be dealt with by aspiration by needle.

Closed abdominal injuries with internal haemorrhage will be given priority for emergency surgery. Open wounds such as stab wound must be dealt with by immediate exploration and repair.

Major limb fractures should be splinted immediately. Fractures with vascular embarrassment should be dealt with, urgently.

Essential haematological and biochemical tests for Hb, PCV and blood sugar are carried out first. If facilities are available, arterial blood gas analysis and serum electrolytes are also estimated. Radiological examinations should include cervical spine, chest, abdomen, pelvis, skull and limbs.

Following is the list of procedures that need to be followed in an emergency room:

- Resuscitation—securing airway, breathing, circulation
- Nasal O_2
- Intravenous fluids
- Fracture splinting
- Haemodynamically stable
- Reduce major joint dislocations with neurovascular compromise
- Emergency vascular repair if present
- Long bone fracture stabilisation
- External/internal fixation
- Open wound debridement
- Neurosurgical evaluation and surgery if needed
- Definitive fixation of fractures of spine, joint articular surface and early wound cover

KEY POINTS

- Polytrauma is either multiple fractures in limbs and spine or multiple system injuries, involving head, thorax abdomen and pelvis in victims of accidents.
- Injuries in a victim of a polytrauma can be described by Injury Severity Score (ISS).
- Guidelines for management of a polytrauma patient are set under Advanced Trauma Life Support system.
- Airway clearance is the first life-saving step in resuscitation
- The injured extremities are stabilised by putting the splint.
- Broad spectrum antibiotics and tetanus/gas gangrene prophylaxis are instituted in open wounds.
- Triage is the process of clinically sorting out a group of trauma victims at the reception room according to the severity of the injuries and the urgency of threat to their lives or limbs.
- Extra care must be taken in handling and turning the patient to prevent cord injury as a 'second accident'.

MULTIPLE CHOICE QUESTIONS

1. Physiologic response to trauma is mediated by
   a. Sympathoadrenal axis
   b. Antidiuretic hormone
   c. Renin and angiotensin system
   d. All of the above
2. One of the following is not included in trauma index
   a. Output of urine
   b. Injured part or region
   c. Status of cardiovascular system
   d. Status of the central nervous system
3. One of the following is not seen in crush syndrome
   a. Rhabdomyolysis
   b. Myoglobinuria
   c. Alkalosis
   d. DIC
4. False regarding the 'opening of airways'
   a. Modified jaw thrust
   b. Chin lift
   c. Jaw lift
   d. Endotracheal intubation

CHAPTER 24

Compound Fractures

A fracture is called a compound or open fracture when the skin overlying it is broken, resulting in a communication between the fracture and the external environment. The break in the covering immediately exposes the fracture to the most serious complication of fracture, namely infection. Almost all fractures are contaminated and therefore should be treated aggressively.

Open fractures are usually caused by a high energy trauma. In many occasions, the patients are associated with multiple injuries and haemodynamic instability. Compound fractures commonly occur in tibia and phalanges.

CLASSIFICATION

- *Compound from within:* In this, the broken bone end pierces the skin and causes an open wound. This is less likely to be infected.
- *Compound from without:* Here, the open wound is caused by external violence, causing laceration of the skin with varying degrees of skin and soft tissue loss (Fig. 14.5). This type is more open for contamination and infection. Foreign matter also gets lodged inside the open wound both in road traffic and in factory accidents.

Compound fracture can be classified based on the size of skin wound and soft tissue damage (modified Gustillo and Anderson's classification)

Gustillo–Anderson's Classification

Type I	Clean wound of less than or equal to 1 cm in length
Type II	Wound larger than 1 cm in length without extensive soft tissue damage
Type III	Wound associated with extensive soft tissue damage; usually longer than 5 cm Open segmental fracture Traumatic amputation Gunshot injuries Farmyard injuries Fractures associated with vascular repair Fractures more than 8 hours old
Subtype IIIA	Adequate periosteal cover
IIIB	Significant periosteal stripping is seen
IIIC	Arterial injury; vascular repair required to revascularise leg

MANAGEMENT

An open fracture is a surgical emergency. Management of such patients begins at the scene of trauma.

1. At the scene of trauma
   - Splint the fracture
   - Arrest bleeding, if any, by tight compressive bandage

- Safely transport to the nearby trauma care centre

2. At the trauma care centre

Surgery is undertaken as soon as the patient's general condition permits. The aim of the treatment is to convert the contaminated or infected wound into a clean wound to promote early wound healing.

Emergency Surgical Treatment of Open Fracture

Contamination of wound is inevitable in every open fracture due to traffic or industrial accidents. In such cases, the infecting organisms lie only on the surface of the wound. In 6–8 hours, the organisms succeed in penetrating into the deeper tissues and get a firm foothold in the wound. Any fracture which comes into the hospital 6–9 hours after injury must be assumed to be infected.

The aim of emergency treatment is to carry out a thorough surgical debridement to eliminate every trace of contamination from the skin edge down to the bone, thus rendering the wound clean and preventing infection. The local treatment of open fracture depends on the surgical facilities available, the time since injury and the extent of soft tissue damage. The principal of treatment will be discussed under the following headings:

1. Prophylaxis against infection
2. Management of the wound/surgical debridement
3. Management of the fracture/skeletal stabilisation
4. Closure of the wound

1. Prophylaxis against infections
   a. *General infection:* Broad spectrum antibiotics, preferably a cephalosporin (III/IV generation) combined with aminoglycoside, are started.
   b. *Tetanus:* In actively immunised patients, tetanus toxoid booster dose is given. In doubtful or non-immunised patients, human tetanus immuno globulin (HTIG) is given.
   c. *Gas gangrene:* Antigas gangrene serum must be given when there is extensive muscle damage with devitalisation and in cases with contamination by faecal matter in injuries in the gluteal region.
2. Management of wound/surgical debridement
   a. *Skin:* Debridement of the wound is carried out starting from the skin edge. Excise a very thin (about 1 or 2 mm) margin of the skin edges. Excise also the contaminated deep fascia.
   b. *Muscles:* Remove all devitalised and dead muscle tissue till fresh bleeding muscle tissue is seen. This is very important as bacterial infection, particularly anaerobic infection, thrives in dead muscle tissue. Development of gas gangrene is a serious danger, if dead or dying muscle tissue is left behind. Look for and remove all foreign bodies found in the depth.

      The viability of the muscle is tested by
      - capacity to bleed,
      - colour,
      - contractility to a stimulus,
      - consistency.

   c. *Tendon:* Cut tendons should be correctly identified and sutured.
   d. *Nerve:* Severed nerves should not be sutured but hitched together by a single black silk stitch. This helps identification of the cut ends during later elective surgery.
   e. *Bone:* Only small fragments of the bone, which are lying absolutely free without any muscular or periosteal attachment, should be removed. Do not remove fragments with some periosteal or soft tissue attachments. Reduce the fracture and hitch the fragments into good position.
   f. *Joint:* If a joint has been opened into, irrigate the joint with sterile saline and close the capsule of the joint and instil antibiotic solution into the joint.
3. Management of the fracture/skeletal stabilisation: The soft tissue healing and reduction of infection are aided by stabilising the fracture. This can be achieved by one of

FIGURE 24.1 Clinical photograph of a patient with ankle spanning external fixator.

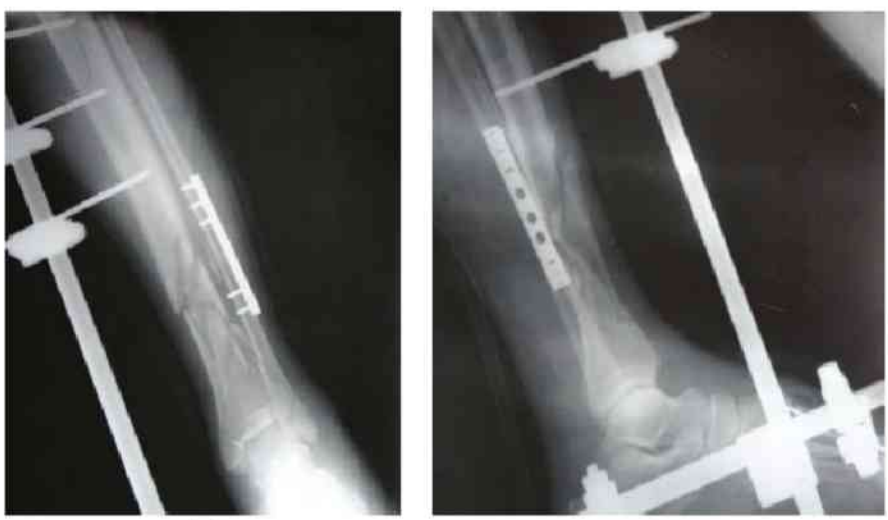
the following methods:

a. *External fixation:* In cases of open fracture, better stabilisation of the fracture is possible with external fixation which consists of the use of two or three Schanz pins passed across the proximal and distal fragments and held rigidly by a frame external to the limb (Fig. 24.1). Use of external fixation stabilises the fracture and allows the open wound to be treated by dressings, without disturbing the fracture alignment and promotes union of the fracture (Fig. 24.2). It is not safe to do internal fixation as a primary treatment in open fractures.

b. *Internal fixation:* Compound fractures up to Grade III A can be treated with internal fixation devices, after thorough wound wash and debridement.

c. *Cast splintage*: Open fractures with small lacerations can be managed in a plaster cast with a window made at the site of laceration, for wound care. As soon as the wound heals, the fracture can be treated with definitive internal fixation methods.

4. Closure of the wound: The decision regarding closure of the wound must be made with great caution. Types of closure include the following:
   a. Primary closure
   b. Delayed primary closure
   c. Secondary closure
   d. Skin grafting
      a. *Primary closure:* If the open fracture has been properly debrided within 6–9 hours and the wound is surgically



(a) (b)

FIGURE 24.2 X-ray showing (a) anteroposterior and (b) lateral views of compound fracture of both bones of the leg stabilised by an external fixator.

clean, the ideal procedure is primary wound closure.

b. *Delayed primary closure:* The primary wound debridement, as described above, is done, the wound is left open, loosely packed with vaseline gauze, covered with sterile dressing and bandaged. The limb is supported in a plaster slab. The wound is re-examined after 3–5 days in the operation theatre. If the wound is surgically clean, it is sutured. This method of closure before the wound granulates is called delayed primary suture.
c. *Secondary suture:* Under unfavourable surgical conditions, the primary closure will not be feasible. The wound will granulate in about 8–10 days. If at this stage, the sepsis in the wound is well controlled, secondary closure may be done to convert the fracture into a closed one.
d. *Skin grafting:* An open fracture with loss of skin does not unite till the skin cover is restored. If a patient is seen with established infection, skin graft should not be done. When the infection is controlled and healthy granulation tissue forms, skin grafting is done. In places where bone is covered by muscle and fascia, split skin graft is sufficient. The options include the following:
   - Split skin graft
   - Full thickness skin graft
   - Local/Regional flap cover
   - Free flap cover

In places such as the tibia where the bone is superficial, a full thickness-free skin graft or fasciocutaneous flap is done to provide healthy skin over the fracture site.

COMPLICATIONS OF OPEN FRACTURES

The complications of an open fracture could be early or late. Early complications occur within the first few days of the injury and the incidence depends on the site of occurrence of the accident. The early complications are as follows:

- Gas gangrene
- Tetanus
- Crush syndrome

The patient may present late with the following late complications:

- Chronic osteomyelitis with discharging sinuses
- Skin loss with thin and adherent skin
- Non-union with bone segment loss

Crush Syndrome

In cases of massive crush injuries to limbs with muscle damage, one serious threat to life is crush syndrome. This occurs when the ischaemia of several hours is relieved suddenly and the toxic products of muscle breakdown (myohaemoglobin) enter the general circulation. The same phenomenon occurs when a tourniquet in the leg is left forgotten for some hours and then released. Clinically, the patient develops shock and acute renal failure. The patient's life could be saved by amputation of the limb.

MANAGEMENT OF LATE COMPLICATIONS OF OPEN FRACTURE

The following surgical procedures will be required for the special problems:

- *Sequestrectomy:* Infection may persist due to osteomyelitis and the presence of a sequestrum (dead bone; Fig 4.3). This will delay fracture union. Surgical removal of the sequestrum will control infection and hasten the healing of the wound.
- *Bone grafting:* In severe crush injuries or gunshot injuries, there is often loss of bone segment and a gap is left. Such cases will end in non-union of the fracture unless bone grafting is done. After the infection is controlled and good skin cover restored, bone grafting is done to restore continuity

of the bone and to promote union of the fracture.

- *Ilizarov fixation:* Non-union with bone loss can be treated with the Ilizarov method.
- *Physiotherapy:* In cases of gross injury where the limb has been immobilised for months, efficient physiotherapy is required to restore mobility of the joints and muscle power and obtain full function of the injured limb.

CRUSH INJURIES

Crush injuries of the limb are severe types of compound fractures. All attempts must be made to save the limb and avoid an amputation.

A primary amputation should be done only when all the blood vessels have been totally severed and there is total ischaemia. When the general condition of the patient is poor, an emergency guillotine amputation is done. Elective revision amputation is done at a later stage. Some cases might be brought after a delay of some days, when infection is well established with purulent discharge. In the lower extremity, the amputation is best done at the site of election.

In the case of the upper extremity, every attempt should be made to save as much of the limb as possible. Even a part of the hand is useful as a good sensory organ. Hence, in the upper limb injuries, primary amputation should never be done at the site of election.

Crushed hand injuries should not be treated by amputation at the wrist, as a routine. One must remember that the best artificial hand can never be a substitute for even a deformed hand. When a deformed hand gives an opportunity for reconstructive surgery, primary amputation is an irrecoverable step. The only indication for proximal amputation in the upper extremity should be gas gangrene and a very badly mangled non-viable hand. All other injuries in the forearm and hand should be treated with conservative surgery till a final decision can be made at a later date.

GUNSHOT AND BOMB BLAST INJURIES

Gunshot and bomb blast injuries are the most serious types of open fractures. The injuries to the muscles, vessels, tendons and nerves are more extensive and the fracture itself is badly comminuted.

The trauma in such injury is of a high energy type or high velocity type and hence often carry an uncertain prognosis due to the more extensive tissue necrosis. The wound of entry and the wound of exit should be appropriately treated.

Most wounds are best treated by debridement, packing with vaseline gauze, stabilisation of the fracture by external fixation and delayed primary or secondary closure.

KEY POINTS

- A fracture is called a compound or open fracture when the skin overlying it is broken, resulting in a communication between the fracture and the external environment
- Compound fracture can be classified based on the size of skin wound and soft tissue damage: modified Gustillo and Anderson's classification.
- The aim of emergency treatment is to carry out a thorough surgical debridement to eliminate every trace of contamination from the skin edge down to the bone.
- Broad spectrum antibiotics and tetanus/gas gangrene prophylaxis are instituted.
- The soft tissue healing and reduction of infection are aided by stabilising the fracture.
- In cases of massive crush injuries to limbs with muscle damage, one serious threat to life is crush syndrome.
- Non-union with bone loss can be treated with the Ilizarov method.

MULTIPLE CHOICE QUESTIONS

1. One of the following statements is not true regarding the compound fractures
   a. Compound fractures communicate with the external environment
   b. Patients with compound fractures have haemodynamic instability
   c. Tibia and phalanges are commonly involved
   d. Infection is rarely associated with these fractures
2. The principle(s) of management of compound fractures is/are
   a. Prophylaxis against infection
   b. Management of wound and fracture
   c. Closure of wound
   d. All of the above
3. One of the following is not an early complication of compound fracture
   a. Gas gangrene
   b. Tetanus
   c. Crush syndrome
   d. Non-union
4. According to Gustillo–Anderson's classification, a compound fracture with periosteal loss is included in the subtype
   a. I
   b. II
   c. III
   d. None

CHAPTER 25

Soft Tissue Injuries Including Sports Injuries

Among the total cases of injuries attended by hospitals, soft tissue injuries far outnumber the cases of fractures and dislocations. Efficient treatment of common soft tissue injuries such as sprains to ligaments will result in a great reduction of morbidity and disability among the sportsmen as well as factory and farm workers.

The following common soft tissue injuries will be considered:

- Injuries to ligaments
- Injuries to muscles
- Tendon ruptures

INJURIES TO LIGAMENTS

The function of the ligament is to limit excessive movement of a joint in one direction. When a joint is moved suddenly in one direction, the ligament on the opposite aspect is overstretched and damaged. Domestic and sports accidents often result in various degrees of acute injuries to ligaments. Chronic injury to ligaments is due to occupational strains and bad postures. The sites where ligaments are commonly injured are the ankle, knee and wrist.

Grades of Injury

Sprain is defined as an injury to ligaments. It is graded according to its severity. A first-degree sprain is a tear of a minimum number of fibres of the ligament.

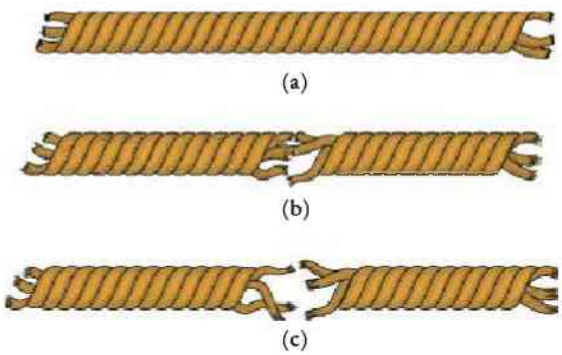


FIGURE 25.1 (a) Normal rope, (b) partial tear and (c) complete tear.

Partial rupture is a disruption of more fibres but with no instability. This is also referred to as second-degree sprain.

Total rupture is a complete disruption of the ligament resulting in joint instability. It is a very severe injury and is also referred to as third-degree sprain.

The varying grades of sprain can be compared with the tear of fibres where a thick cord of jute (rope) is put on a sustained or increasing stretch. Starting with the tear of a few individual fibres, more and more fibres give way till the rope tears across totally (Fig. 25.1).

ANKLE JOINT

The commonest lesion is injury to the lateral ligament.

Lateral Ligament Injuries

The lateral ligament of the ankle consists of three segments—anterior talofibular, posterior talofibular and middle calcaneofibular.

Mechanism

A sudden inversion strain of the ankle during walking on irregular ground or falling during the game results in injury to the lateral ligament. This usually damages the anterior or middle segment of the ligament. Walking with a shoe badly worn out on the outer side of the heel is a predisposing factor and the foot gets easily tripped into inversion.

Clinical Features

The patient presents with a history of a sudden fall with foot inverted and complains of pain and swelling in the lateral aspect of the ankle.

If the tear is complete, there is ecchymosis and oedema on the lateral aspect of the ankle. On palpation, there is tenderness at the distal or proximal attachment of the ligament as the tear usually occurs at these sites. Inversion of the joint will cause acute pain but eversion will be painless. Gross abnormal mobility denotes total tear.

Radiograph

Anteroposterior and lateral views of the ankle must be taken to exclude crack or avulsion fracture of the lateral malleolus. Stress radiography can be taken to visualise widening of the lateral half of the joint space in complete ruptures. MRI of the ankle can confirm the diagnosis.

Treatment

First Aid

Immediate first aid should follow the I.C.E. system:

- Application of ice
- Compression bandage
- Elevation of the part

This prevents and minimises swelling and relieves pain.

First-Degree Sprain

In first-degree sprain, only some fibres of the ligament are torn. The ankle should be strapped with the foot held in maximum eversion. This keeps the injured lateral ligament in a relaxed position and permits healing in about 2–3 weeks.

Partial Tear

In cases of partial tear, there will be more swelling and a slight increase in inversion movement. In such cases, the ankle is immobilised in a padded below-knee plaster slab with the joint held in eversion.

Total Tear

When there is total tear or avulsion of the ligament at one end, the swelling and pain are very severe. There is marked ecchymosis on the outer aspect of the ankle. There is also obvious abnormal mobility of the joint, permitting excessive inversion. This is better demonstrated by examination under anaesthesia.

When the total tear of the ligament is diagnosed, the best treatment is early surgery to repair the ligament and plaster protection for 3–4 weeks.

Complication

Inadequate treatment of injuries to lateral ligament results in chronic pain and instability at the ankle.

Medial Ligament Injury

The medial or deltoid ligament extends between the medial malleolus and the calcaneus. Injuries to the medial ligament are much less common. A sudden eversion violence causes injury to the medial ligament. The tenderness is at the upper attachment of the medial ligament to the medial malleolus. In treating this, strapping is done with the ankle held in an inverted position.

INTERNAL DERANGEMENT OF THE KNEE

Internal derangement of the knee is common in sports injuries. The following conditions are included under this heading:

1. Injuries to collateral ligaments
   - Medial collateral ligament
   - Lateral collateral ligament
2. Injuries to cruciate ligaments
   - Anterior cruciate ligament (ACL)
   - Posterior cruciate ligament (PCL)
3. Injuries to semilunar cartilage
   - Medial semilunar cartilage
   - Lateral semilunar cartilage
4. Loose body in the knee
   - Osteochondritis dissecans

The collateral and cruciate ligaments are commonly injured in athletes, football and kabaddi players.

Collateral Ligament Injuries of the Knee

Medial Collateral Ligament Injuries

The medial collateral ligament is more commonly injured than the lateral. A sudden valgus or abduction strain at the knee associated with external rotation of tibia causes stretching of the medial ligament producing

1. first-degree sprain,
2. partial rupture,
3. complete rupture.

1. *First-degree sprain of the ligament:* In cases of first-degree sprain of the ligament, there is pain in the medial aspect of the knee and tenderness over the upper or lower attachment of the ligament. Pain increases on applying abduction strain at the knee. There is no laxity or abnormal mobility. The knee is given rest with a posterior splint and compression bandage for a week. If the effusion is marked, it is aspirated. Quadriceps exercises are started straight away.

   In haemarthrosis, the swelling occurs within an hour or two of the injury. In traumatic synovial effusion, the swelling occurs in 12–24 hours.
2. *Partial rupture of the medial ligament:* In this condition, the ligament is partially torn resulting in haemarthrosis. On applying abduction force at the knee, there will be severe pain and abnormal mobility. There will be tenderness at the medial ligament attachments. Radiograph will not show any abnormality. The knee is aspirated under strict aseptic precautions. A compression bandage and posterior plaster slab are applied with the knee in slight flexion and kept for 3 weeks. Quadriceps exercises are started.
3. *Complete rupture of the medial ligament:* This is caused by a very severe valgus strain at the knee. This may be associated with the fracture of the lateral condyle of the tibia. In addition to a marked swelling of the knee due to haemarthrosis, there will be abnormal abduction mobility possible at the knee, when the knee is held at 10° flexion. Complete tear takes longer time to heal. In this condition, there is often an associated rupture of the ACL. In some cases, the medial meniscus is also torn causing the triad of injuries termed *Unhappy Triad of O'Donoughue.*

   The usual radiography may not show any bony injury. The anteroposterior view taken with an abduction strain (stress radiograph) shows widening of the medial joint space.

Surgical Treatment

In cases seen early, exploration to assess the full extent of injury and repair of the torn structures are indicated. Early surgical repair of the ligament prevents later joint instability.

Cases presenting late will need surgical reconstruction of the ligament.

Injuries of the Lateral Collateral Ligament

Injuries of the lateral collateral ligament are much less common than those of the medial ligament. The same types of injuries, sprain, partial rupture and complete rupture of the ligament occur due to a hit on the inner aspect of the weight-bearing knee, which forces the knee into a sudden adduction strain. The signs and symptoms will be similar except that tenderness will be on the outer aspect of the knee. The treatment is also based on the same principles as discussed earlier.

Cruciate Ligament Injuries

The cruciate ligaments play an important role in maintaining the stability of the knee. They may be injured alone or in combination with collateral

ligaments. The ACL is more commonly injured and its injury is more disabling.

Anterior Cruciate Ligament Injuries

The common mechanism of injury is a forced full flexion of the knee with the tibia in internal rotation. It occurs in injuries in gymnastic exercises, basketball and football. Clinically, the patient presents with acute pain, swelling and haemarthrosis of the knee. There is abnormal anterior gliding of the tibia on the femur, with the knee held in 15° flexion. This is called Lachman's test. Abnormal anterior movement of the tibia on the femur with the knee held in 90° flexion is called ***anterior drawer sign***. Associated injuries with the lateral collateral ligament by varus strain can also be demonstrated. Examination under anaesthesia will be necessary to fully assess the extent of injuries.

Radiology may demonstrate a small chip avulsion of the bone from the intercondylar area of the tibia.

Arthroscopic examination will reveal the site of the rupture, and it will also show whether it is a partial or total tear.

Treatment

Partial tears of the ligament can be treated conservatively by aspiration of the haemarthrosis and plaster immobilisation with knee in 45° flexion. Rupture near the lower or upper end is better treated by surgical reattachment. Patients presenting late with disability need surgical reconstruction of the ligament. More recently, prosthetic replacement of the ligament by synthetic materials is being done with success. Materials used for ACL reconstruction include the following:

- Bone patellar bone graft
- Hamstring graft

Posterior Cruciate Injury

Posterior cruciate ligament tears are less common and occur due to sudden posterior displacement of the upper end of tibia. Clinically, the posterior drawer sign is positive and treatment is mostly conservative.

Injuries of the Semilunar Cartilages

The semilunar cartilages are commonly called menisci and form an important shock-absorbing mechanism, which helps in the smooth functioning of the knee. They help in the gliding movement of the tibia on the femur.

Incidence

Injuries to the meniscus are common in young adults and are often sustained by the football and kabaddi players.

Mechanism

An abduction external rotation violence, on a flexed weight-bearing knee, causes a tear in the medial meniscus. In football, it occurs when the player standing on one leg, which is slightly flexed at the knee, turns to tackle the ball with the other leg.

The lateral meniscus is damaged by the opposite violence, that is, internal rotation and abduction violence of the tibia or a semiflexed weight-bearing knee.

Clinical Features

At the time of the first injury, the patient gets an acute pain in the knee and is unable to continue in the game. The knee gets locked in flexion. The knee gets swollen soon after, due to effusion. This may subside in about a week.

Subsequently, he gets recurrent attacks of 'locking' of the knee with effusion. He feels insecure in the knee while walking and the knee 'gives way'. In the typical case, the patient gives a history of recurrent episodes of locking, pain and swelling associated with 'clicking' at the knee.

On examination, there is some wasting of the quadriceps. The knee is kept in about 5° flexion. In tears of the medial cartilage, the point of maximum tenderness is between the medial ligament and ligamentum patellae at the joint line. In tears of the lateral meniscus, the tender point is lateral to the ligamentum patellae on the joint line.

Special Clinical Tests

McMurray's Test

McMurray's test elicits a click of the cartilage. A click demonstrates the presence of a tear.

FIGURE 25.2 Types of meniscal tears. (a) Bucket handle tear, (b) posterior horn tear and (c) anterior horn tear.

Apley's Grinding Test

When the meniscus is torn, pain and click localised to the joint line are elicited.

Radiological Features

In recent cases of tear in the semilunar cartilage, radiological appearance of the knee is normal. In long-standing cases, there will be some evidence of osteoarthritic changes in the knee. MRI of the knee joint is very sensitive in picking up tears of menisci.

Arthroscopy

Arthroscopic examination of the knee is very useful in confirming the presence of the tear and its position.

Types of Meniscal Tear *(Fig. 25.2)*

1. Longitudinal tears
   - Bucket handle tear
   - Posterior horn tear
   - Anterior horn tear
2. Horizontal tears
3. Oblique tears

Treatment

When the patient presents with a history of ***repeated attacks*** of locking, the best treatment is surgical excision. A ***partial meniscectomy*** of torn segment of the damaged meniscus is done.

More recently, arthroscopic meniscectomies are being done. This is less traumatising to the joint and rehabilitation is quicker.

INJURIES TO MUSCLES

Closed injuries to muscles are due to a sudden violent contraction of the muscle. They are more common in young active adults.

Pathology

The process of repair in muscle injuries passes through three stages: (1) acute inflammatory phase for 1–3 days, (2) repair phase up to 3–4 weeks with scarring and (c) remodelling phase up to 3–6 months with restoration of normal shape and length.

The common closed injuries to muscles are as follows:

1. Contusion
2. Rupture of muscle
   - Partial rupture
   - Complete rupture

Contusion

Contusion is caused by a blunt injury and results in a haematoma due to the tear of scattered muscle fibres and small blood vessels. Immediate first aid for all muscle injuries is application of ***ice cold pad***, and anti-inflammatory drugs for a week are sufficient to give relief. Early mobilisation prevents scarring and contracture and helps functional restoration.

Rupture of Muscle

Violent contraction of muscle to stop it from overstretching results in various degrees of tear of a muscle. In the lower limb, when a person is thrown off balance and tries to stop himself from falling, the quadriceps mechanism could be torn across. The common site for rupture is near the musculotendinous junction. The muscles commonly ruptured are biceps brachii, supraspinatus, quadriceps and gastrocnemius.

Rupture of the Biceps Brachii

The rupture in this muscle can occur

- at the tendon of the long head at the proximal end,
- as tear through the musculotendinous junction,
- as an avulsion through the distal end.

Rupture of the tendon of the long head occurs when the muscle suddenly contracts against an unexpected resistance. The patient feels something

giving way inside the shoulder as the long head retracts downwards. When the patient attempts to flex the elbow, a bulge is seen anteriorly in the middle of the arm and there is a depression proximal to the middle of the muscle.

Treatment

In the young individual, the treatment is essentially a surgical repair in fresh cases. When the tendon is avulsed from the proximal end, the repair consists of fixing it to the lower end of the bicipital groove. In cases of tear at the musculotendinous junction, the best treatment is to expose the muscle and repair the tear. Avulsion of the distal end is treated by fixing the tendon to the branchialis.

Rupture of the Supraspinatus

Rupture of the supraspinatus tendon can occur

- across the tendon as part of the tear of rotator cuff,
- at the insertion with avulsion of a chip of greater tuberosity.

Rupture of supraspinatus occurs in the young due to violent muscular contraction. In the elderly, the tear occurs with minor violence in a degenerated rotator cuff.

Partial tear of the supraspinatus is characterised by acute pain and tenderness in the subacromial point and a 'painful arc' of abduction movement as in the case of supraspinatus tendinitis. In most cases, conservative treatment—rest, analgesics, cuff and collar and short-wave diathermy—will relieve the symptoms. Persistent pain may be treated by local infiltration of hydrocortisone. If the pain is severe and not relieved by the earlier treatment, excision of the acromion process relieves the condition.

In complete tears, there is total inability to initiate abduction movement. The 'arm drop' sign is the classical clinical sign of such a lesion. The patient is unable to maintain the shoulder in abduction and the arm drops to the side. Passive abduction is full. Total tears are best treated by surgical exploration and repair of the tear as early as possible.

Rupture of Quadriceps Femoris

Refer to Chapter 19.

Rupture of the Achilles Tendon

Incidence and Mechanism

The rupture of the strong achilles tendon can occur in two age groups. The first is the *young adult patient*. The achilles tendon ruptures partially or completely due to a sudden stretching violence when the muscle is in active contraction. This is a *traumatic rupture*. The second is the *older age group* of patients in the fourth or fifth decade. Here, it is a *pathological rupture* due to violence acting on a degenerated tendon.

Clinical Features

The patient gives a history of feeling something giving way with a snapping noise in the back of the heel while engaged in some unaccustomed exertion during work or play. He complains of pain and difficulty in walking. On examination, the prominence of the achilles tendon in the back of the ankle is lost when compared with the normal side. There is a soft swelling at the site of injury and a gap may be palpable in the course of the tendon. The site is tender and the pain increases on passive dorsiflexion of the ankle. The power of active plantar flexion of the foot is diminished or lost according to whether the tear is partial or complete.

Stair (Climbing) Test

Ask the patient to put the forefoot of the normal leg on the edge of a step and lift himself up on that leg as in stair climbing. Repeat the test with the injured leg. The patient will be unable to lift himself up on that leg due to discontinuity of the achilles tendon.

Thompson's Grip Test

In the normal leg with the knee in flexion, when the calf muscle is compressed by gripping, the foot moves into plantar flexion. When the continuity of the achilles tendon is lost by a rupture, the grip compression does not cause plantar flexion of the foot.

Treatment

Partial tears are treated by the application of a below-knee plaster cast with the foot in *plantar flexion*.

The best treatment for complete rupture, whether traumatic or pathological, is surgical exploration and repair of the torn tendon. In cases with degenerated tendons, the suture is reinforced by darning the gap with strips of fascia lata.

KEY POINTS

- Soft tissue injuries broadly include injury to ligaments, injury to muscles and tendon ruptures.
- Ligament injuries commonly involve ankle and knee joints.
- The lateral ligament of the ankle consists of three segments—anterior talofibular, posterior talofibular and the middle calcaneo fibular.
- Soft tissue injury at knee joint may involve collateral ligaments, cruciate ligaments and semilunar cartilage.
- Medial collateral ligament tear may occur in association with the anterior collateral ligament and medial meniscus tear. This is called *Unhappy triad of O'Donoghue.*
- The cruciate ligaments play an important role in maintaining the stability of the knee
- In anterior cruciate ligament tear, the abnormal anterior movement of the tibia on the femur with the knee held in 90° flexion is called *anterior drawer sign.*
- The semilunar cartilages are commonly called menisci and form an important shock-absorbing mechanism, which helps in the smooth functioning of the knee.
- Arthroscopic examination of the knee is very useful in confirming the presence of the tear and its position.

MULTIPLE CHOICE QUESTIONS

1. Which of the following is true in medial meniscus injury?
   a. Rotation of femur on tibia
   b. Menisci do not heal and should be excised
   c. Locking and unlocking episode
   d. All of the above
2. Medial meniscus is more vulnerable to injury because of
   a. Its fixity to the tibial collateral ligament
   b. Its semicircular shape
   c. Action of adductor magnus
   d. Its attachment to fibrous capsule
3. The best diagnostic procedure for anterior cruciate ligament injury is
   a. Lachman's test
   b. Pivot shift test
   c. Anterior drawer test
   d. McMurray's test
4. Anterior drawer's sign is positive in
   a. Medial meniscus tear
   b. Anterior cruciate ligament injury
   c. Lateral collateral ligament injury
   d. Lateral meniscus injury
5. McMurray's test is positive for
   a. Medial meniscus
   b. Lateral meniscus
   c. Anterior cruciate ligament
   d. Posterior cruciate ligament
6. The most common cause of sprained ankle is injury to
   a. Deltoid ligament
   b. Lateral ligament
   c. Inferior tibiofibular ligament
   d. Anterior talofibular ligament

CHAPTER 26

Nerve Injuries

INJURIES OF THE PERIPHERAL NERVES

Injuries to peripheral nerves can be classified into two categories: open and closed injuries.

- *Open injuries:* Open injuries to peripheral nerves are common complications of open or cut wounds with or without injuries to bones. They can also occur in gunshot injuries in the thigh, arm, etc.
- *Closed injuries:* Closed injuries to the nerve often present as complications of fractures and dislocations.

Seddon's Classification

Seddon classified the injuries into the following pathological types, on the basis of structural changes in the nerves:

- *Neurotmesis:* Complete anatomic division of nerve fibres with obvious discontinuity of the nerve sheath
- *Axonotmesis:* Microscopic division of nerve fibres (axons) without obvious discontinuity of the nerve sheath
- *Neuropraxia:* Injury without any anatomical discontinuity but resulting in functional disruption (nerve concussion)

Sunderland's Classification

In this classification, peripheral nerve injuries are arranged in ascending order of severity:

- *First-degree injury:* In first-degree injury, conduction along the axon is physiologically interrupted at the site of injury, but the axon is not actually disrupted (neuropraxia).
- *Second-degree injury:* In second-degree injury, axonal disruption is present but the integrity of the endoneural tube is maintained (axonotmesis).

Further degrees of injuries (third, fourth and fifth) are based on the increasing degrees of anatomic disruption of the fibres with or without rupture of the ensheathing membrane. In the final fifth degree, there is total anatomical rupture of the whole nerve (neurotmesis).

Mechanism of Closed Nerve Injuries

Nerves could be injured by any one of the following types of violence:

1. In a closed fracture, nerves could be damaged by direct injury by the fracture ends (e.g. radial nerve palsy in fracture midshaft humerus)
2. In dislocations, the nerve could be damaged by stretching of the nerve by the displaced bone end (e.g. circumflex nerve palsy in the dislocated shoulder and sciatic nerve palsy in the dislocated hip)
3. Traction of the nerve as one falls from a height or when thrown off a two-wheeler in road traffic accidents (e.g. brachial plexus paralysis)

4. Compression violence due to external pressure on the nerve
   - Wrist drop as in crutch palsy due to compression in the axilla
   - Foot drop due to lateral popliteal nerve palsy as a complication of B.K. Plaster cast or pressure by the outer road of Thomas splint
   - Tourniquet palsy due to pressure on median, ulnar and radial nerves by tight tourniquet during surgery
5. Chemical injury by injection of drugs (e.g. radial nerve palsy following intramuscular injection in the arm)

Diagnosis of Nerve Lesion

A complete diagnosis of a traumatic nerve lesion should include the identification of the following:

- The nerve or nerves injured
- The anatomical level of injury to the nerve
- The pathological type of injury: Neurotmesis, axonotmesis, neuropraxia
- Associated bone, vascular and tendon injuries
- Secondary effects such as deformities and contractures
- Any evidence of recovery of the nerve palsy

Clinical Assessment of a Nerve Lesion

In the case of gunshot or other major injuries, a detailed examination should be done only when the patient's general condition permits. All findings, including the nature of violence, etc., should be recorded so that the subsequent findings could be correlated. The detailed examination regarding the particular nerve injured should be done, keeping in mind the presence of the other injuries such as fractures, vascular and tendon injuries. Clinical findings must be recorded under the following headings:

1. *Motor signs:* Note the muscles paralysed distal to the lesion and the wasting of muscles. Motor power is to be assessed in terms of the Medical Research Council Gradings 0, 1, 2, 3, 4, 5. On a command to move a joint by a muscle under test, the response could be:
   - 0—Nil. No power at all.
   - 1—Muscle flicker only present. No power to move the joint.
   - 2—Power to move a joint but only when gravity is eliminated.
   - 3—Power to move a joint against gravity.
   - 4—Power to move a joint against gravity and resistance.
   - 5—Normal power.
2. *Sensory signs*
   - Subjective: Note the distribution of pain, tingling or burning sensation
   - Objective: Blunting or loss of sensation to pinprick, touch and temperature
3. *Sudomotor signs:* Anhidrosis: Note the area of dry skin due to absence of sweating
4. *Vasomotor signs*
   - Warm phase in the first 2 weeks
   - Cold phase later
5. *Trophic changes:* Note the area of skin smoothness and shiny areas, ulceration and subcutaneous tissue atrophy.
6. *Reflexes:* Loss of tendon reflexes
7. *Recovery sign:* Tinel's sign

Special Tests

Special tests are done to accurately assess the degree of nerve damage and also to note the evidence of recovery.

1. *Electrodiagnostic tests:* These are based on the alteration in the electrophysiological functions of the muscle and nerve.
   a. Muscle
      - Electromyography (EMG)
      - Strength–duration curve (S–D curve)
   b. Nerve
      - Nerve conduction study
      - Conduction velocity
2. *Sudomotor function:* This is tested by the *sweating test* using Anizarine powder. The iodised starch powder is dusted over the area. On inducing sweating, the powder remains grey in the denervated area but turns purple in the normal areas which sweat.
3. *Thermography:* This is based on skin temperature changes through the post-ganglionic sympathetic (sudomotor) fibres.

MANAGEMENT OF NERVE INJURIES

Management of nerve injuries depends on the type of nerve injury—open or closed.

Open Injuries

The treatment of such injuries is part of the treatment of the open wounds as described elsewhere. As a rule, cut injuries are not treated by primary suture. If cut, the cut ends are fixed by black silk stay sutures to prevent their retraction. This helps in identification when the wounds are opened later for a regular nerve repair operation.

Closed Injuries

Conservative Treatment

Periodic examinations must be done to detect any recovery of the paralysed muscles. The paralysed muscles should be splinted in a position that prevents their overstretching. The activity of the opposing muscles should be permitted without stretching the paralysed muscles by the use of *dynamic splints*. The joint should be kept supple by passive movements, to prevent contracture of the capsule. The wasting of the muscles should be prevented by graded exercise therapy when signs of recovery occur.

Signs of Recovery

The paralysed muscles when recovering show a gradual increase in motor power from Grade I (Flicker) upwards.

Tinel's Sign

Tinel's sign is elicited as follows. Tap the injured nerve along its course starting peripherally. The point at which the patient feels tingling sensation along the course of the nerve indicates that recovery has started and has reached that point.

If the injury was neuropraxia (Grade I) a gradual recovery of motor power and sensation occurs in a few weeks, and when the power is satisfactory splinting could be done away with.

Surgical Treatment

Normally, nerve regeneration occurs roughly at the rate of 1 mm a day or 25 mm a month. If there is no sign of motor or sensory recovery in the expected time of recovery, surgery is indicated. The appearance of a palpable neuroma at the site of the nerve injury is another indication for surgery.

The following procedures may be required:

- *Neurolysis (nerve release):* If the nerve is in continuity and there is no neuroma at the site of injury, the mere release of the injured segment from the surrounding scar tissue is sufficient.
- *Neurorrhaphy (nerve repair):* If a neuroma is found in the course of the nerve, it is excised and the cut ends are sutured. Recently, microsurgical techniques are used in suturing nerve ends.
- *Nerve grafting:* If the gap between the cut ends is small, the ends could be approximated by proper positioning and sutured in a relaxed position. If the gap is too large, continuity can be restored by use of nerve grafting procedures.

Reconstruction Operations

In some cases, it may not be possible to restore continuity of the nerve. In such a situation, the function of the limb could be improved by *reconstruction operations* such as tendon transfer procedures or arthrodesis of joints to stabilise the unstable joint.

RADIAL NERVE INJURIES

The injury can be at the axilla, humeral or elbow level:

- *Axilla:* The nerve is compressed in the axilla in those using the old type of crutch with a T-type support at the top.
- *Humerus:* At the humeral level, it occurs as follows. The radial nerve is most commonly injured as it winds round the spiral groove in cases of fracture of humerus at this level. Pressure on the nerve when a person sleeps with his arm resting over the edge of the cot causes wrist drop. Such a pressure can also occur in a drunken man sleeping off in a sofa with the arm on the top edge of the seat. This is called '*Saturday night palsy*'. The nerve is accidentally injured when intramuscular injections

are carelessly given in the lower part of the arm. The wrist drop develops soon after the injection due to chemical neuritis.

It also occurs as a part of the tourniquet palsy, as a badly applied rubber tourniquet in the arm could cause paralysis of the radial, median and ulnar nerves.

- *Elbow:* The posterior interosseous branch of the radial nerve is injured in cases of dislocation of the head of radius or accidentally injured during surgical excision of the head of the radius.

Clinical Features

In the case of injury to the nerve at the level of humeral shaft, the patient presents with a wrist drop.

- *Motor signs:* Ask the patient to actively dorsiflex the wrist. He will be unable to do this, due to paralysis of the extensor carpi radialis longus and brevis muscles.

  Hold the patient's wrist steady. Ask the patient to actively extend the fingers at the metacarpophalangeal (MP) joints. He will be unable to extend them (finger drop). This is due to paralysis of the extensor digitorum.

  Ask the patient to extend the whole thumb. He will be unable to extend (thumb drop) it due to paralysis of the extensor pollicis longus and brevis.

  The patient is asked to hold his forearm in 90° flexion and mid-prone position. Ask him to flex the elbow against resistance applied at the wrist. The brachioradialis does not stand out prominently as it is paralysed.
- *Sensory signs:* Sensory loss is minimal and is confined to a small area in the dorsum of the hand over the metacarpal bones of the thumb and index fingers.

  In case of injury just *below the elbow*, the posterior interosseous nerve is paralysed. The wrist extension is preserved. There is loss of extension power only at the MP joints of the four fingers and thumb.

  In case of paralysis of the radial nerve in the axilla, in addition to paralysis in the finger, wrist and forearm, there is also paralysis of active *extension at the elbow* due to paralysis of the triceps.

Conservative Treatment

Initially, the wrist and fingers are *splinted* in a position of extension at the wrist and MP joints by a 'cock up' splint. This is to prevent overstretching of the paralysed muscles. The modern splint for radial nerve palsy is a *dynamic* or *lively* splint, applied to the dorsal aspect which keeps the wrist and fingers extended by elastic bands or springs attached to it but allows active flexion of the fingers and wrist (Fig. 26.1).

Passive movements are given to the wrist and finger joints. Electrical stimulation is given to the paralysed muscles to prevent wasting of muscles. Progressive active exercises are given to the muscles showing recovery. As most of the lesions are neuropraxia or axonotmesis, recovery occurs in about 4–6 weeks.

Surgical Treatment

When there is evidence of neurotmesis, exploration and repair of the nerve give good results as it is mostly a motor nerve. When the radial nerve is irreparably damaged, tendon transfer operations are done to restore the extensor functions at the wrist, fingers and thumb.

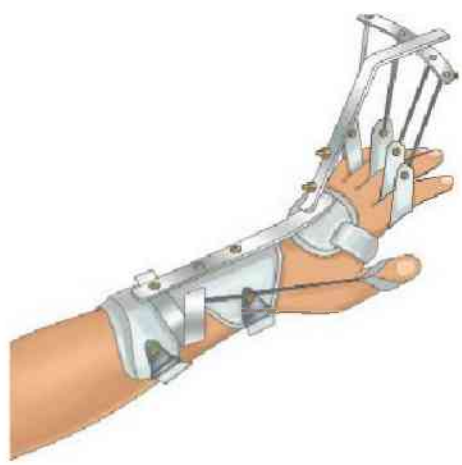


FIGURE 26.1 Diagrammatic representation of dynamic cock up splint used for radial nerve palsy.

Tendon Transfer Operation

In the modified Robert Jones operation which is commonly performed, the following are carried out:

- The pronator teres tendon is transferred to the extensor carpi radialis longus and brevis to restore wrist extension.
- Flexor carpi ulnaris tendon is transferred to the extensor digitorum tendons of the four fingers to restore extension of the fingers.
- The palmaris longus tendon is transferred to the extensors of the thumb to restore thumb extension.

MEDIAN NERVE INJURIES

The median nerve can be injured at the following levels:

1. Elbow level
2. At the level of the wrist
3. In the carpal tunnel

1. *At the elbow level:* This is called the high median nerve lesion. This occurs commonly as a complication of supracondylar fracture or a dislocation of the elbow joint.
2. *At the level of the wrist:* The median nerve is injured by knife cuts or by glass cut when the fist is pushed through a window glass. In open injuries, it is associated with cut injuries to flexor tendons or even the radial artery.
3. *At the carpal tunnel:* The injury here is often due to pressure by dislocated lunate bone and chronic compression by swellings in the tunnel (e.g. compound palmar ganglion).

Clinical Features

1. *Motor signs:* In an injury to the median nerve at the elbow level (high median nerve lesion) the muscles paralysed are flexor carpi radialis, flexor digitorum sublimis, flexor digitorum profundus to the middle and index fingers, flexor pollicis longus, the pronator teres and quadrates and the thenar muscles.

   The following clinical tests are elicited:

   - The '*pointing index*' finger elicited by the clasping test is as follows. Ask the patient to clasp the two hands by interlocking the fingers and thumb. The index finger of the affected hand will stick out due to paralysis of the long flexor tendons of the index finger.
   - Inability to flex the interphalangeal (IP) joint of the thumb due to paralysis of the flexor pollicis longus.
   - Ape thumb deformity (simian hand): Ask the patient to place the hand on the table with the palm facing up. The thumb lies in the plane of the hand due to the paralysis of the opponens and short flexor muscles.
   - Pencil test for abductor pollicis brevis: With the hand flat on the table and palm facing up, a pencil is held a few inches above the thumb across the palm. The patient will be unable to abduct the thumb and touch the pencil due to paralysis of the abductor pollicis brevis.
   - Opponens palsy is demonstrated by the patient's inability to oppose the thumb and touch the tips of the other fingers.
2. *Sensory signs:* There is loss of sensation in the thumb, index, middle and the radial half of the ring finger.

   In the lesions at the wrist level, signs will be confined distal to the level. The diagnosis could be easily missed as the clinical signs are mainly sensory loss in the thumb, index and middle fingers. The only motor loss is the paralysis of thenar muscles of the hand. Trophic and other changes which occur late are also confined mostly to the thumb and index fingers.

Treatment

In case of open injuries, nerve exploration and suture operation are done. In case of irreparable lesions of the nerve at the wrist level, restoration of opponens power of the thumb is done by tendon transfer operation. The flexor digitorum sublimis tendon of the ring finger is transferred to the radial side of the thumb. The flexor power of thumb and index can also be restored by appropriate tendon transfer operation.

ULNAR NERVE INJURIES

Injuries to the ulnar nerve commonly occur at the level of the elbow region and at the level of the wrist.

Traction injuries result from violent valgus injuries to the elbow as in avulsion fracture of the medial epicondyle and lateral dislocation of the elbow. It can also be directly damaged in supracondylar fracture of humerus. Tardy or *late ulnar neuritis* with palsy is caused by increasing valgus deformity due to non-union of the fracture of the lateral condyle humerus. Recurrent subluxation of the ulnar nerve also causes ulnar neuritis and paralysis.

At the level of the wrist, cuts and lacerations are the common causes of injury.

Clinical Features

Injury at the elbow: The patient presents with a typical ulnar claw hand deformity of the ring and little fingers, wasting of the hypothenar muscles and depression in the interosseous spaces in the dorsal aspect of the hand.

There is paralysis of flexor carpi ulnaris and medial half of flexor digitorum profundus in the forearm and the intrinsic muscles in the hand.

The ulnar claw hand deformity is characterised by extension deformity at the joints and flexion deformity at the IP joints of the little and ring fingers (Fig. 26.2). The deformity is due to the paralysis of the lumbricals which flex the MP joint and extend the IP joints allowing the unopposed action of the long extensors at the MP joint and the long flexors at the IP joint.

The interosseous function of abduction and adduction of the fingers is tested. Adduction is tested by asking the patient to hold a card or paper tightly between two fingers. The patient will be unable to grip the card. Abduction of the index finger is tested by asking him to abduct the index finger against resistance. The contractions of the first dorsal interossei are palpated and found missing.

FIGURE 26.2 Diagrammatic representation of hand showing ulnar claw hand.

The abduction of the little finger against resistance tests the integrity of the abductor digiti minimi.

Test for adductor pollicis (*Froment's test*), with both palms held together, keep a card or book between the thumb and the index finger in both hands. Ask the patient to grip the card tightly. The thumb on the affected side goes into flexion at the IP joint due to the action of flexor pollicis longus which compensates the action of the paralysed adductor. The power of flexion with adduction (ulnar deviation) at the wrist is tested against resistance. This power is weak or absent.

Sensory loss is confined to the little finger and medial half of the ring finger and the ulnar border of the hand.

Treatment

The treatment of cut injuries to the ulnar nerve follows the general principles of treatment of cut nerves. The cut nerve is explored and managed by neurolysis or nerve repair as required.

The anterior transposition of the ulnar nerve is a very useful operation to remove local irritation to the nerve following elbow injuries and to prevent tension on the nerve suture line in all cases of repair of the injured nerve. The management of irreparable damage to ulnar nerve is by tendon transfer operation. In Bunnell's operation, the flexor digitorum sublimis to the ring finger is disinserted, split into two, rerouted and attached to the extensor expansion of the ring and little fingers.

BRACHIAL PLEXUS INJURIES

Injuries to the brachial plexus could be due to the following:

- Cut, stab or gunshot injuries in the neck
- Traction injuries by any violence which stretches the plexus by pushing the shoulder down and the head to the opposite side during fall from height. They also occur in automobile or two-wheeler accidents where the clavicle may also be fractured
- It also occurs as a birth injury in breech presentation, when the child's shoulder is

pulled down due to difficulty in the delivery of the aftercoming head

Classification

- Total brachial plexus paralysis
- Proximal or Erb's paralysis
- Distal or Klumpke's paralysis

Total Brachial Plexus Paralysis

Total brachial plexus paralysis is often caused by high speed motorcycle accidents. This involves all the roots, trunks or cords of the plexus. The paralysis is total and the whole arm is flail with total loss of sensation. The patient may also suffer from intolerable causalgic pain.

Traction injury resulting in the avulsion of the *pre-ganglionic level* of all the roots C5 to T1 is a very serious one.

The presence of Horner's syndrome in which there is ptosis and constricted pupil on the affected side means that the damage includes the T1 root at the pre-ganglionic level and the prognosis is bad. This is paralysis of serratus anterior and rhomboid muscles. The lesion is mostly irrecoverable and the limb is functionless. In these, rehabilitation is by above-elbow amputation. Some function could then be restored by arthrodesis of the shoulder joint and fitting an above-elbow prosthesis.

In post-ganglionic injuries, sensation is partly present. The serratus anterior and rhomboid muscles escape paralysis. When the lesion is an axonotmesis, some recovery is possible. In cases of neurotmesis, surgical exploration and repair may be attempted.

Erb's Palsy (Proximal Brachial Plexus Palsy)

Erb's palsy is the common form of birth palsy. The lesion is usually at the junction of the C5 and C6 roots (Erb's point). It causes paralysis of the deltoid, supraspinatus, infraspinatus, biceps and brachialis muscles.

The arm hangs by the side with the shoulder in internal rotation, the elbow in extension and the forearm pronated with the palm facing backwards, the so-called tips position. The hand and finger functions are preserved. The treatment is usually conservative.

In the newborn, the baby's arm is positioned with the shoulder in abduction and external rotation and the wrist behind the neck and held with a bandage. This position prevents contracture in the subscapularis muscle. Most cases recover in a few months with good splinting and physiotherapy. The common complication is internal rotation contracture of the shoulder. This contracture is disabling even after the recovery of the deltoid muscle. The initial treatment is by passive stretching by physiotherapy. In resistant cases, surgical correction is done by dividing the contracted structures in front of the shoulder joint.

In traumatic Erb's palsy, when the root level injury is excluded, surgical exploration and repair may be possible.

Klumpke's Palsy (Distal Brachial Plexus palsy)

Klumpke's palsy involves the lowest trunk, and there is paralysis of the muscles supplied by C8 and T1 roots. This is caused by a violent upward pull of the arm in accidental falls from a tree when the patient catches a branch and gets suspended.

The small muscles of the hand are paralysed in addition to the paralysis of wrist and finger flexors, and there is sensory loss in the C8 and T1 dermatome areas in the ulnar border of the hand. The sympathetic trunk may also be involved resulting in Horner's syndrome. Conservative treatment is done by physiotherapy. In irrecoverable lesions, hand function could be improved by tendon transfer operation.

SCIATIC NERVE INJURIES

The sciatic nerve is injured in the gluteal region as a complication of the posterior dislocation of the hip joint. It can also be injured in the thigh by cuts or gunshot injuries.

In high lesions, the hamstring muscles and all the muscles below the knee will be paralysed. The

hamstring muscles will escape paralysis in lower thigh level lesions. There will be sensory loss in the leg and foot corresponding to the dermatome areas L4, L5, S1, S2 and S3 roots.

Management

In compression injuries due to the dislocation of the hip, recovery occurs in about 6–9 months by conservative measures. Suturing the nerve may succeed in clean-cut injuries in the thigh, but prognosis is not very good.

LATERAL POPLITEAL NERVE INJURIES

Aetiology

- Cuts and lacerations over the neck of fibula
- Fracture neck of fibula associated with fracture lateral tibial condyle in abduction injuries to the knee
- Traction injury due to adduction violence at the knee associated with medial tibial condyle fracture

Clinical Features

Clinically, the patient presents with a foot drop. On examination, the patient will be unable to dorsiflex the ankle and extend the toes due to paralysis of the extensors. He will also be unable to evert the foot due to paralysis of the peroneal muscles. There will be loss of sensation in the outer aspect of the leg and dorsum of the foot.

Treatment

The nerve is explored and repaired in cases of cut injuries and prognosis is good. In closed injuries, the patient is given a below-knee orthotic appliance for foot drop, while waiting for recovery. In irrecoverable cases, the treatment is by tendon transfer operations.

This is done by transferring the tibialis posterior tendon to the dorsum of the foot and inserting into a tarsal bone. In adults, the foot is stabilised by triple arthrodesis.

ENTRAPMENT NEUROPATHY

Entrapment neuropathy is a clinical condition of nerve paresis or paralysis due to chronic compression of some peripheral nerves. This is often a result of old injury to a nearby bone.

The nerve is compressed in a subcutaneous situation where it is caught (entrapped) in an osteofascial or musculofascial canal. The following are common examples:

- *Carpal tunnel syndrome:* Median nerve at wrist
- *Tarsal tunnel syndrome:* Posterior tibial nerve at ankle
- *Epicondylar tunnel syndrome:* Ulnar nerve at elbow
- *Meralgia paraesthetica:* Lateral cutaneous nerve of thigh about an inch inferior to the anterior superior iliac spine

Tarsal Tunnel Syndrome

Here, the posterior tibial nerve gets compressed in the fibro-osseous tunnel deep to the flexor retinaculum behind the medial malleolus. In the irritable phase, there is burning and tingling in the medial border and sole of the foot and later there is severe pain which is worse at night and is relieved by hanging the leg.

In the early stage, local infiltration of hydrocortisone will relieve the pain. If the pain is severe and persistent, surgical decompression is done by dividing the flexor retinaculum.

In some situations such as carpal tunnel syndrome, entrapment of the nerve is due to closed space of the tunnel in conditions such as myxoedema and pregnancy. Appropriate general management gives relief to the entrapment symptoms.

KEY POINTS

- Nerve injuries can be classified (Seddon's classification) as neurotmesis, axonotmesis and neuropraxia.
- Clinical examination and special diagnostic tests help in assessment of nerve lesions.
- Special diagnostic tests include electrodiagnostic tests, sudomotor functions and thermography.
- Electrodiagnostic tests include electrophysiological studies of muscle and nerve.
- Tinel's sign indicates recovery of nerve injury. Beginning from periphery, the injured nerve is tapped and the point at which the patient feels tingling sensation indicates that recovery has started.
- Normally, nerve regeneration occurs roughly at the rate of 1 mm a day or 25 mm a month.
- The following surgical procedures can be used in cases of nerve injury: Neurolysis (nerve release), neurorrhaphy (nerve repair) and nerve grafting.
- Radial nerve may be injured (leading to wrist drop) in axilla: crutch palsy, arm: Saturday night palsy and elbow: due to dislocation of head of radius.
- Brachial plexus injury can be total paralyses, proximal or Erb's paralysis, and distal or Klumpke's paralysis.
- Erb's palsy occurs at the junction of the C5 and C6 roots (Erb's point). It causes paralysis of the deltoid, supraspinatus, infraspinatus, biceps and brachialis muscles.
- Klumpke's paralysis involves the lowest trunk, and there is paralysis of the muscles supplied by C8 and T1 roots.
- Entrapment neuropathy is a clinical condition of nerve paresis or paralysis due to chronic compression of some peripheral nerves.

MULTIPLE CHOICE QUESTIONS

1. Pointing index sign is seen in … nerve palsy
   a. Ulnar
   b. Radial
   c. Median
   d. Axillary
2. The commonest cause of wrist drop is
   a. Intramuscular injection
   b. Fracture humerus
   c. Dislocation of elbow
   d. Dislocation of shoulder
3. Radial nerve injury above elbow leads to
   a. Ape thumb
   b. Wrist drop
   c. Trigger finger
   d. Claw hand
4. The 'card test' tests the function of
   a. Median nerve
   b. Ulnar nerve
   c. Axillary nerve
   d. Radial nerve
5. Injury of median nerve at wrist is best detected by
   a. Action of abductor pollicis brevis
   b. Action of flexor pollicis brevis
   c. Loss of sensation on the radial half of the palm
   d. Loss of sensation on the tip of the finger

CHAPTER 27

Vascular Injuries

INJURIES OF MAJOR BLOOD VESSELS

In severe injuries to the lower and upper limbs, particularly those near the knee and the elbow, fractures are often complicated by injuries to the major blood vessels. Open injuries often lead to severe blood loss, shock and even death. Closed fractures with injury to blood vessels result in distal ischaemia or gangrene, in addition to shock. This often ends up in amputation of the limbs. With the increasing incidence of road traffic accidents resulting in multiple major fractures, it is essential for every medical professional to be able to recognise the presence of severe vascular trauma in limb injuries so that urgent steps could be taken to shift the casualty to a proper hospital and save the limb.

In the case of patients with multiple major limb injuries, next to the airway obstructions, haemorrhage from major vessels and acute ischaemia in the limbs are the major complications that need emergency attention.

Types of Injuries Affecting Blood Vessels

The following are the types of injuries to arteries:

Open Injuries

- Division or laceration of the artery
- Traumatic false aneurysm (pulsating haematoma)
- Arteriovenous fistula

Closed Injuries

- External compression
- Arterial spasm
- Thrombosis following intimal tears

Vascular Injuries: Aetiopathogenesis

Aetiology

Closed injuries are the commonest cause of acute traumatic ischaemia in a limb. The cause of vascular obstruction could be (1) outside the wall of the vessel, (2) in the wall and (3) inside the lumen of the vessel.

External compression of the vessels can be caused by (1) tight plaster, tight bandages, splints, etc.; (2) subfascial haematoma in places such as cubital fossa, popliteal fossa; (3) increasing traumatic oedema of the muscles in the forearm or in calf and (4) direct pressure by the fractured bone end.

Internal obstruction may follow injury to the arterial wall, arterial spasm, thrombosis or embolism.

Pathology

The pathological changes in arterial ischaemia are reversible in early stages and recover if treated vigorously. If they are allowed to progress, it leads to ischaemic necrosis and contracture. The pathological changes in the various tissues of the limbs following ischaemia are as follows:

- *Muscle:* The muscle undergoes infarction with the formation of muscle 'sequestrum'. This is followed by a central area of necrosis, surrounded by an area of cellular response, leading to fibrosis. The contracture of this fibrous tissue leads to shortening of the muscle and deformity in joints.

  In the forearm, the long flexor muscles are involved in an ellipsoid area of necrosis and their contracture causes Volkmann's ischaemic contracture. In the leg, the muscles of the deep posterior compartment undergo ischaemic necrosis and the resulting contracture causes an equinus deformity.
- *Nerves:* The nerves in the area of vascular ischaemia also undergo ischaemic paralysis which becomes irreversible if the occlusion is prolonged.
- *Skin:* When the occlusion is total and is not relieved for more than 12 hours, the skin and all other soft tissues also undergo necrosis and result in sloughing or gangrene.

Symptomatology

Clinically, vascular injuries may present as acute Volkmann's ischaemia, subfascial compartment syndrome and gangrene.

In the acute stage, the patient presents with a history of injury to the elbow or knee. He presents with severe pain and oedema of the fingers or toes with flexion deformity. There will be delayed capillary filling in the nail bed. There will be pallor, pulselessness, paraesthesia and paralysis of muscles. The fingers or toes are held in flexion and attempts to passively extend them cause severe pain.

Management of Vascular Injuries

It is necessary to tackle a case of acute traumatic ischaemia as a surgical emergency. If local ischaemia is not relived within 6 hours of injury, the muscles suffer irreversible ischaemic changes and if the total occlusion lasts for more than 12 hours the danger of gangrene increases rapidly making amputation inevitable. The threat to the viability of the limbs by arterial injury and threatened ischaemia entitles the patient a higher priority during triage.

The general condition of the patient including shock is treated and blood pressure improved which itself increases the oxygenation of the ischaemic tissues. Any open bleeding is arrested. Open major vessel injuries are treated by exploration and arterial repair.

Closed injuries may manifest as subfascial compartment syndromes.

Subfascial Compartment Syndrome

Subfascial compartment syndrome is commonly seen after injuries to both bones in the forearm and leg. Haemorrhage into the musculofascial compartments in these sites causes compression of the arteries resulting in acute ischaemia of the limb. Such a compression can also occur when the muscles inside these fascial compartments undergo a reactionary oedema after the release of the vascular obstruction causing further obstruction to the circulation (referred to as Volkmann's ischaemia).

In both these conditions, the ischaemia should be urgently relieved by surgical decompression by incisions in the deep fascia (fasciotomy).

A few common arterial injuries—brachial, popliteal and tibial artery—are described below.

Brachial Artery Injury

Brachial artery injury occurs as a primary complication of supracondylar fracture in children, comminuted fractures of the lower end of the humerus and dislocation of the elbow in adults. It can also occur due to tight encircling bandages applied by traditional bone setters or even by injudicious bandaging while applying plaster splints, or application of complete plaster cast as a primary procedure after fracture.

Clinical Features

A child with a supracondylar fracture with severe displacement may be brought with gross oedema of the elbow. In all such cases of elbow injuries, one must examine the radial pulse as well as the capillary pulsation in the nail bed. If the pulse is weak or missing, acute ischaemia should be suspected and energetic steps taken to relieve the compression and vascular insufficiency.

Severe cramp such as pain is the earliest symptom of vascular insufficiency; the fingers are in flexion and there is pain on attempted extension. There is pallor of the skin in the fingers and later it can become cyanotic. The limb is cold on palpation and there is numbness in the fingers. Paralysis of the muscles develops within 4 hours of the occlusion. Thus, pain, pallor and paralysis are the three characteristic features which confirm the presence of ischaemia and call for immediate measures to save the limb by relieving the compression of the artery.

Popliteal Artery Injury

Popliteal artery is injured often by a supracondylar fracture of the femur of comminuted T or Y intercondylar fractures of the lower end of the femur. In the supracondylar fracture, the distal fragment gets flexed by the origin of the gastrocnemius muscle. The popliteal artery gets damaged by the sharp edge of the flexed lower fragment.

Clinical Features

The patient presents with a history of injury in the lower thigh and a tense swelling around the knee, particularly in the popliteal fossa. The limb is cold and the dorsalis pedis and posterior tibial pulsations may be absent. The limb may rapidly develop gangrene, unless energetic steps are taken to relieve the compression in the popliteal fossa.

Emergency arteriography of the femoral artery may be helpful in identifying the level of arterial obstruction and the extent of the collateral circulation.

Treatment

The first step is the removal of all constricting bandages and splints.

Urgent manipulation must be done to reduce the fracture and it may need skeletal traction through the tibial tuberosity to relieve the pressure on the vessel. If the condition of pulse and the colour do not improve within 2 hours, urgent surgical intervention is mandatory. Decompression of the popliteal artery and bathing the wound with warm saline will often relieve the arterial spasm and restore the circulation. Obvious division or other injury to the artery should be repaired.

In the leg, it is also necessary to do a fascial decompression to prevent compression of the vessels by the reactionary oedema which follows the release of proximal pressure.

Injury to Tibial Arteries

In fractures of the upper end of the tibial shaft, the displaced fragments as well as deep haematoma often seriously threaten the vascularity of the leg by pressure on the origin of the anterior or posterior tibial arteries. Such acute ischaemia must be urgently relieved by longitudinal incisions in the deep fascia over the anterior tibial compartment as well as the superficial and deep posterior tibial compartments.

Thus, the presence of traumatic vascular ischaemia or threatened ischaemia needs emergency treatment, both general and local to restore circulation and save the limb from ischaemia and amputation.

KEY POINTS

- Severe open injuries of limbs often lead to significant blood loss, which may further lead to shock and death.
- Closed fracture injuries, in addition to causing shock, may also cause distal ischaemia or gangrene.
- Acute traumatic ischaemia is commonly caused by closed injuries.
- Progressive ischaemia in the forearm may cause contracture of long flexor muscles due to necrosis (Volkmann's ischaemic contracture).
- Progressive ischaemia in leg may result in equinus deformity.
- Compartment syndrome should be urgently relieved by surgical decompression by incisions in the deep fascia (fasciotomy).
- Pain, pallor and paralysis are the three characteristic features which confirm the presence of ischaemia and call for immediate measures to save the limb.

MULTIPLE CHOICE QUESTIONS

1. Injury to popliteal artery in fracture lower end of femur is often due to
   a. Distal fragment pressing artery
   b. Proximal fragment pressing the artery
   c. Tight plaster
   d. Haematoma
2. The single most important sign in suspecting early Volkmann's contracture is
   a. Pallor of fingers
   b. Pain
   c. Obliteration of the pulse
   d. Paralysis of the involved muscles
3. Volkmann's ischaemic contracture mostly involves
   a. Flexor digitorum superficialis
   b. Pronator teres
   c. Flexor digitorum profundus
   d. Flexor carpi radialis longus

CHAPTER 28

Amputations

Amputation is defined as the surgical removal of a part or whole of a limb. This term should not be confused with disarticulation which means removal of the limb through a joint.

INDICATIONS OF LIMB AMPUTATION

The ablation of a limb is an extreme step and an irreversible operation, and every care should be taken to see that an amputation is done only when absolutely indicated. Recent advances have shown a decline in limb amputation surgeries due to an increase in limb salvage (saving) procedures.

Amputation should be considered only if the limb is

- dead (gangrenous),
- dying (grossly ischaemic),
- dangerous (due to malignancy),
- dud (useless limb, e.g. leprosy).

The common indications that require amputation are given in Table 28.1

The common causes of amputation in different age groups are as follows:

- *Children:* Congenital anomalies
- *Young adults:* Crush injuries
- *Elderly:* Vascular conditions (diabetes)

TYPES OF AMPUTATIONS

1. *Guillotine amputation:* This is an emergency amputation done as a life-saving measure.

TABLE 28.1 Indications for Amputation

Condition	Indication
Traumatic conditions: Crush injuries of limb	Road traffic accidents Occupational crush injuries Train traffic accidents
Vascular conditions: Ischaemic conditions	Arteriosclerosis (e.g. diabetic) Gangrene Thromboangiitis obliterans
Neoplastic conditions	Malignant tumours of bone and soft tissue (e.g. osteosarcoma, synovial sarcoma)
Infective conditions	Leprosy—useless grossly destroyed limb Actinomycosis Filarial elephantiasis Gas gangrene
Congenital conditions	If the limb is grossly deformed and useless

This is done in cases of gross crush injuries of the limb. It is also indicated in cases of gas gangrene, when a rapid removal of the dangerously infected part is a life-saving procedure. In guillotine amputation, the incision is circular around the limb at the site of bone section and all the tissues are cut at the same level and the wound is left open to provide free drainage.

2. *Classical amputation:* These are planned amputations where regular skin flaps are raised

and the wound is closed after ablation of the limb.

3. *Revision amputation:* Revision amputations are done
   - as a second stage, in guillotine amputation;
   - in those with very unsatisfactory stumps following a previous amputation.

SELECTION OF LEVELS OF AMPUTATION

The classical sites of amputation of limbs were determined on the basis of the following considerations:

- The disease process for which the amputation was done
- The vascular supply to the skin flaps
- The requirements and availability of limb-fitting procedures and techniques

Radiography of the part is done to see the extent of the malignant disease. In some cases, arteriography may be used to assess the vascularity of the limb and level of viability.

Modern advances in the technology of limb fitting have made it possible to achieve satisfactory limb fitting to stumps of any length. Hence, one should not stick to the classical levels too rigidly and sacrifice parts of limbs which could be saved.

Levels of Amputation

- *Upper limbs (Fig. 28.1)*
  1. Forequarter amputation
  2. Shoulder disarticulation
  3. Above-elbow amputation
     a. Elbow disarticulation
  4. Below-elbow amputation
     a. Krukenberg's amputation (Fork-like forearm bones)
  5. Wrist disarticulation
     a. Ray amputation (for fingers)
- *Lower limbs (Fig. 28.2; Table 28.2)*
  1. Hindquarter amputation
  2. Hip disarticulation
  3. Above-knee amputation
  4. Knee joint disarticulation
  5. Below-knee amputation
  6. Ankle and foot amputation

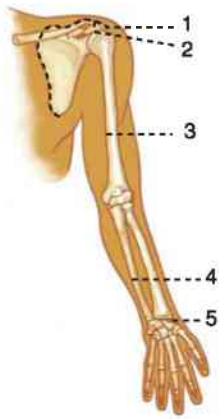


FIGURE 28.1 Levels of amputation in the upper limb.

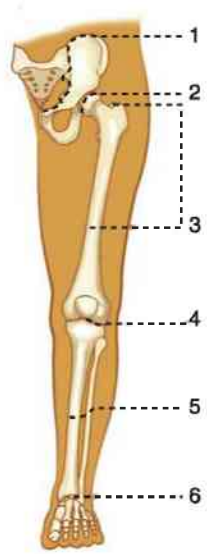


FIGURE 28.2 Levels of amputation in the lower limb.

TABLE 28.2 Named Amputations in Lower Limb Ankle and Foot

Name of Amputation	Level of Amputation
Syme's amputation	Through ankle joint
Lisfranc's amputation	Through intertarsal joint
Chopart's amputation	Through talonavicular joint

PRINCIPLES OF AMPUTATION

1. *Remove diseased or damaged part*
2. *Bones:* Leave enough for a good prosthesis and rehabilitation
3. *Muscles:* Should be cut distal to the bone so as to enable
   - myoplasty—opposing two groups of antagonising muscles
   - myodesis— suturing of muscles to bone
4. *Nerves:* Should be cut short so that they retract into the muscle tissue
5. *Blood vessels:* Isolated and ligated doubly
6. *Skin flaps:* Tension-free flaps
   - Avoid anaesthetic skin on the stump
   - Delay primary closure if required

STUMP AND ITS MANAGEMENT

The stump is the residual part of the limb left after the amputation, and a surgeon's work does not finish with amputation. It should not be just an anatomical residue but should be an active motor organ to move the prosthesis and also give some sensory feedback. Hence, the care of the stump is very important to provide good function in the limb. The stump should be mobilised with muscle-strengthening exercises for early rehabilitation. In lower limb amputations, the functions to be restored are weight bearing and locomotion and sensory feedback. Weight bearing could be end bearing, side bearing or proximal bearing. The modern method of total contact socket has made weight bearing more comfortable to patients. The physiological method of weight transmission is through the terminal ends of bones. Hence, artificial limbs with weight bearing through the calcaneus, lower end of tibia or lower end of femur are more physiological. Syme's amputation and knee disarticulation are becoming increasingly popular, from the point of view of limb fitting.

A good stump should

- neither be too long nor too short,
- have good muscle power with full movement in the proximal joint,
- have a healthy non-adherent scar,
- have a fleshy end with no bony spurs.

The care of the stump consists of the following:

- Stump bandaging with crepe bandage to improve its shape for limb fitting
- Stump exercises to improve its motor power and movements in the proximal joint
- Stump hygiene to maintain the skin and scar in good condition

The amputation may be the end of the management of the pathology, but it is the beginning of the phase of retraining of the stump for prosthetic fitting and functional restoration.

Complications

1. Immediate
   - Infection
   - Ischaemia
   - Secondary haemorrhage
   - Skin sloughing

   These are preventable by control of infection and proper technique in suturing.
2. Late
   - Stump painful neuroma
   - Phantom limb
   - Contractures
   - Oedema of stump
   - Shrinkage of stump
   - Osteomyelitis of bones

Infection

Infection must be vigorously controlled as it may lead to sloughing of the edges of the flaps or even osteomyelitis with sequestrum formation. Severe infection can lead to separation of a *ring sequestrum* from the tip of the sectioned bone. This also results in a puckered and adherent scar which is painful, and it interferes with proper limb fitting.

Neuroma

Neuroma is the development of a bulbous swelling at the cut nerve end. It is tender and causes pain on weight bearing. Pain may be relieved by local hydrocortisone injection or in some cases by ultrasonic therapy.

Phantom Limb

In this condition, the patient feels that the limb is still present even though it is not and he may feel pain in some areas in the non-existent foot or toes. It usually clears up with assurance, analgesics, stump exercises and regularity in the use of prosthesis.

AMPUTATION IN CHILDREN

The special feature of amputations in children is the growth potential of the bone in the stump. In cases of below-knee or above-knee amputation in children, the bone in the stump contains the *growing epiphyseal plate* of the upper end of tibia and the upper end of humerus, respectively. In the years following the amputation, the growth of the bone continues. If the stump is covered snugly with the skin flap, the subsequent growth of the bone will stretch the skin in the tip of the stump and the bone may even tend to protrude. This will need a *revision amputation* to excise the excess bone. One must remember to leave the skin flaps a bit flabby so that it will fit snugly when the bone growth occurs. In the lower limb, disarticulation through knee is preferable to the above-knee amputation through the femur, as there will be growth of the stump proportionate to the growth of the child.

PROSTHETIC FITTING AND REHABILITATION

The responsibility of the surgeon does not end with the healing of the wound but must include the restoration of locomotion by correct limb fitting and re-education. The limb must fit the amputee not only physically but also physiologically. It must be integrated emotionally with the patient and be socially acceptable.

Prosthesis is a replacement or substitution of a missing or a diseased part. Prosthetic instruments that are used for the limbs are given in Tables 28.3 and 28.4.

TABLE 28.3 Prosthesis for Lower Limbs

Hemipelvectomy	Wrap around flexible plastic socket prosthesis
Above knee	Suction socketed limb Non-suction socketed limb
Below knee	SACH (solid ankle cushioned heel) foot Patellar tendon bearing Jaipur foot
Syme's amputation	Syme's prosthesis

TABLE 28.4 Prosthesis for Upper Limbs

Fore quarter	Cosmetic sleeve fitter prosthesis
Above elbow	Suction sockets, 'figure-of-eight' harness
Below elbow	Split hook prosthesis with harness
Digits	Silicone finger prosthesis Pollicisation of index finger

Uses of the Prosthetic Limb

- Cosmetic—to provide an improved appearance
- Functional—to provide function of the missing part

ORTHOSIS

Orthosis is a device that aids the body to make better use of that part to which it is fitted. Prosthesis replaces a missing part of the body but orthosis augments a weak part of the body.

Some Varieties of Orthosis

- Spinal orthosis
- Cervical orthosis
- Knee-ankle foot orthosis
- Knee orthosis
- Ankle-foot orthosis

KEY POINTS

- Amputation is the surgical removal of a part or whole of a limb.
- Amputation can be of following types: Guillotine amputation, classical amputation and revision amputation.
- The level of amputation depends on the disease process, vascular supply of skin flaps, and requirements and availability of limb-fitting prosthesis and techniques.
- The care of the stump is very important to provide good function in the limb.
- The stump should be mobilised with muscle strengthening exercises for early rehabilitation.
- *Phantom limb:* In this condition, the patient feels that the limb is still present and he may even feel pain in some areas in the non-existent foot or toes.
- The special feature of amputations in children is the growth potential of the bone in the stump.
- A prosthesis is a replacement or substitution of a missing or a diseased part.
- Orthosis is a device that aids the body part to enable better use of that part to which it is fitted.

MULTIPLE CHOICE QUESTIONS

1. The distance from the knee joint in below-knee amputation is … inches
   a. 4
   b. 5.5
   c. 6
   d. 7
2. Complication of an amputation may be
   a. Phantom limb
   b. Stump neuroma
   c. Ring sequestrum
   d. All of the above
3. Syme's amputation involves joint
   a. Ankle
   b. Midtarsal
   c. Tarsometatarsal
   d. All of the above
4. Distance from the lip of greater trochanter in thigh amputation is
   a. 5
   b. 7
   c. 8
   d. 9

CHAPTER 29

Disaster Management

A disaster is a natural or man-made event that negatively affects life, property, livelihood or industry often resulting in permanent changes to human societies, ecosystems and environment.

Natural calamities may be broadly grouped into major and minor types depending upon their potential to cause damage to human life and property. While natural calamities such as earthquakes, droughts, floods and cyclones could be regarded as major, hailstorms, avalanches, landslides, fire accidents, etc., whose impact is localised and intensity of the damage is much less, can be categorised as minor calamities.

Disaster management is a multipronged strategy where the health system and health care professionals are kept prepared and ready along with the paramedical team and other infrastructure, for any emergency or major hazard that causes disruption and damage to people and their environment on a large-scale basis.

The relative number of people injured or killed differs depending on the type of disaster, density and distribution of population where the disaster occurs, degree of preparedness and opportunity for a warning.

DISASTER MANAGEMENT

Disaster management is a three-pronged strategy:

1. Disaster preparedness
2. Disaster response
3. Disaster mitigation

Disaster Preparedness

Disaster preparedness is a long-term programme where the idea is to improve the overall capability of our system to manage all types of emergencies efficiently. It requires appropriate systems, procedures and resources to be ready to provide prompt and effective assistance to victims in a disaster.

Disaster Response

Disaster response is the phase when the medical treatment is required for a large number of casualities. Most injuries are sustained at the time of impact, so maximum need is required during the first few hours.

At the Site of Disaster

There is an important role for training paramedical staff on life-saving procedures as these are the people who are actually going to be at the site of disaster well within the 'Golden Hour'.

A massive search operation is started after a major disaster, and first aid and relief services may be available only for a fraction of the rescued.

Triage

When the number of injured floods the medical capacity, the routine practice of treating all does not serve the purpose. Triage is the process by which the injured are classified on the

basis of the severity of the injuries and the likelihood of their survival with prompt medical intervention.

High priority is given to victims whose immediate or long-term prognosis can be dramatically improved by simple invasive care. Moribund patients who require a lot of attention but with questionable benefit are given lower priority.

The internationally accepted four-code system is most commonly used:

1. RED: High priority treatment or transfer
2. YELLOW: Medium priority
3. GREEN: Ambulatory patients
4. BLACK: Dead or moribound patients

Triage is carried out at the site of disaster by the paramedical team in order not to overwhelm the medical system. The victims are tagged accordingly along with the basic information such as name, age, place of origin, diagnosis and treatment plan.

Relief Phase

Relief phase is the time when assistance of various forms comes from outside. This includes food and clothing and some critical health supplies.

Disease control and surveillance for diseases form an important part of relief after natural calamities as they may produce more damage than the disaster itself. Outbreak of gastroenteritis is the most common disease reported in the post-disaster period.

Rehabilitation

Rehabilitation is the final phase where the restoration of normalcy occurs as the pre-disaster conditions. This is usually a long-drawn, tedious process and hurried measures of relief hinder this process by providing an unsustainable resource inflow for a very brief period. Water, food, sanitation and shelter are basic requirements of good rehabilitation.

Disaster Mitigation

Disaster mitigation measures include the preparedness of a country to prevent hazards from causing an emergency or to lessen the likely effects of emergencies. This requires a strong moral and political will by policy makers and effective implementation of the policy framed.

Disaster mitigation is done by flood mitigation works, appropriate land use planning, improved codes and quality of buildings, protection for vulnerable population and buildings.

A National Crisis Management Committee (NCMC) has been constituted in the Cabinet Secretariat. The Government of India has adopted mitigation and prevention as essential components of its development strategy. Currently, the Eleventh Five Year Plan document has a detailed chapter on Disaster Management. The plan emphasises the fact that development cannot be sustainable without mitigation being built into a developmental process. Each state is supposed to prepare a plan scheme for disaster mitigation in accordance with the approach outlined in the plan.

The Twelfth Five Year Plan that will come into effect from 2012 emphasises on disaster mitigation rather than relief and rehabilitation. The states have been advised to constitute Hazard Safety Cells (HSC) headed by the Chief Engineer (Designs), State Public Works Department, with necessary engineering staff to establish a mechanism for proper implementation of the building codes in all future constructions and to ensure the safety of buildings and structures from various hazards.

KEY POINTS

- Disaster management is a multipronged strategy where the health system and health care professionals are kept prepared and ready along with the paramedical team and other infrastructure, for any emergency or major hazard.
- Triage is the process by which the injured are classified on the basis of severity of the injuries and the likelihood of their survival with prompt medical intervention.
- Disease control and surveillance for diseases form an important part of relief after natural calamities as they may produce more damage than the disasters themselves.
- Water, food, sanitation and shelter are basic requirements of good rehabilitation.

CHAPTER 30

Physiotherapy and Rehabilitation

Rehabilitation signifies the whole process of restoring a disabled person to a condition in which he is able to resume a normal or near-normal life. With the better understanding of various modalities used by a physiotherapist, a doctor can make this process of rehabilitation faster and smoother for a patient.

An appropriate rehabilitation protocol is a useful adjunct to a well-performed surgery and can help to obtain very good functional results. At the same time, ill-advised and poorly directed therapy can lessen and probably even reverse the outcome of a good surgery.

TYPES OF PHYSIOTHERAPY

There are two basic types of physiotherapy depending on what outcome is desired:

1. Passive physiotherapy
2. Active physiotherapy

Passive Physiotherapy

Passive physiotherapy is used to alleviate symptoms such as pain or discomfort in any part of the body.

The modalities used are as follows:

- Heat or cold
- Electrical stimulation
- Techniques of counter-irritation
- Massage
- Mobilisation and manipulation

These modalities of physiotherapy may have unproven therapeutic effects but they afford an important placebo effect which forms an important part of the therapy (Figs 30.1 and 30.2). Passive physiotherapy may be used as an adjunct to active physiotherapy, enhancing its effects.

Active Physiotherapy

Active physiotherapy is used to restore the function of a limb of a body part by repeated activity. It is the advanced modification and application of certain basic exercises to the muscle (Fig. 30.3). These may be given to a particular muscle, muscle groups, affected portions of the body or the body as a whole.

Proprioceptive rehabilitation and functional training are two most important areas of research

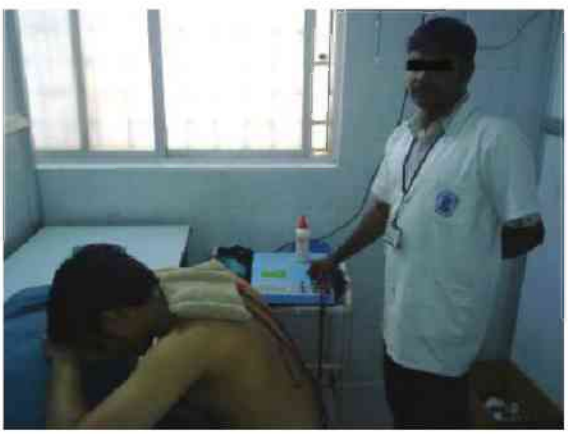


FIGURE 30.1 Clinical photograph showing how an interferential therapy is given.

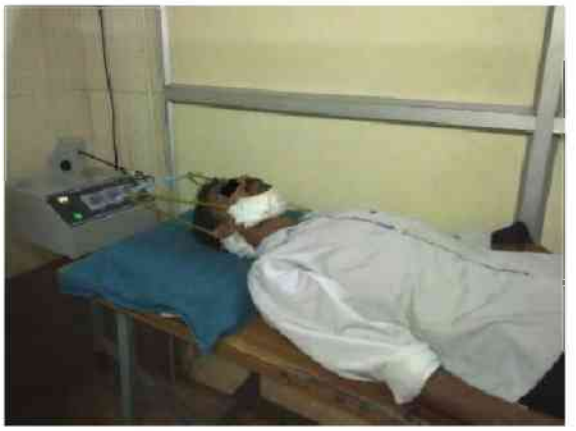


FIGURE 30.2 Clinical photograph showing how an intermittent cervical traction is given.

FIGURE 30.3 Clinical photograph showing how an intermittent pelvic traction is given.

and scope for improvement in active physiotherapy. A further important aspect of rehabilitation in a chronically ill and debilitated patient is the shift from hospital-based therapy to a much more cost-effective, self-administered home programme.

PHYSICAL MODALITIES

Heat

Heat is transmitted through conduction, convection and radiation.

1. Conduction and convection heating modalities: Movement of heat within a substance, flowing from warmer to cooler parts, is called conduction. Convection is transmission of heat by flow of heated liquid or gas
2. Hot water in the form of hot water packs
3. Wax baths (Fig. 30.4): Wax baths contain molten wax and other smoothening materials such as glycerine, heated electrically and applied directly over the skin. They are very useful in rheumatoid arthritis and other degenerative bone diseases
4. Radiation modalities
   - Radiation heat is the passage of heat via a medium such as air, ultimately to be absorbed by another body. Radiant heat is applied using
   - radiant heat lamps,
   - electric pads,
   - infrared lamps.

High-Frequency Currents

1. *Short wave diathermy:* It is the most effective and efficient way of heating large areas of subcutaneous tissue and muscle (Fig. 30.5). It uses high-frequency alternating current to produce its effect. It is administered via pads kept in close approximation to the body.
2. *Microwave diathermy:* It has a higher frequency and shorter wavelength as compared

FIGURE 30.4 Clinical photograph showing wax bath preparation.

FIGURE 30.5 Clinical photograph showing how a short wave diathermy is given.

to short wave diathermy. It occasionally causes burn wounds to the part they are applied on.

3. *Ultrasonic therapy:* Therapeutic ultrasound uses a similar principle to diagnostic ultrasound by using piezoelectric crystal vibration effect in the range from 750 kHz to 3 MHz (Fig. 30.6). It is used in tennis elbow, haematoma resorption, tissue regeneration and inflammation reduction.
4. *Laser therapy:* Lasers are light amplifiers that emit infrared light. They help in setting up resonance in tissues and this effect gives the beneficial effect. They are used in inflammations, reduction of oedema and stimulate fibroblast proliferation. Infrared gallium arsenide (GaAs) and helium–neon (HeNe) lasers are used for this purpose.
5. *Cryotherapy:* Ice packs are well-known agents that help to soothen pain and reduce oedema and inflammation by the principle of vasoconstriction. They can also reduce spasm of muscles. Cold therapy can be applied in the form of
   - ice massages,
   - ice baths,
   - ice towels,
   - ice packs.
6. *Electrical muscle stimulation:* Electricity to stimulate muscles can be used to enhance contraction of a muscle group to supplement contraction in post-injury states after limb immobilisation, to regain tone and contraction in a paralysed viable muscle (Fig. 30.7). They may be used intradurally/extradurally to control urinary continence in spinal injury patients. The most common current used is faradic current (alternating current).
7. *Transcutaneous electrical neural stimulation*: Transcutaneous electrical neural stimulation (TENS) is electrical stimulation of superficial nerve fibres through which deep pain is masked using the principle of 'Gate' theory. Electrical stimulation can also cause endorphin and enkephalin release that are natural analgesic agents, and these chemical mediators released by our body help in prolonging the effect of TENS.

FIGURE 30.6 Clinical photograph showing how an ultrasonic therapy is given.

FIGURE 30.7 Photograph of an electrical muscle stimulator.

Therapeutic Exercise

Wasting of muscles is a universal effect that occurs after a disease process, injury or immobilisation. To improve the tone, power and strength of a muscle, we have to initiate a targeted therapeutic exercise for each group of wasted muscle.

1. *Passive movements:* Movement of joints and muscles by a physiotherapist unaided by voluntary contraction of muscle is called passive movement. This is used in excessively wasted or paralysed muscle (Fig. 30.8).
2. *Assisted movements:* Here the patient provides voluntary activity while the therapist assists the movement. This is useful during early recovery after paralysis.
3. *Free movements:* These are movements achieved without assistance from the therapist or the patient himself. This is useful in recovery before minimal antigravity lifting is possible, after which resisted exercises are started.
4. *Resisted exercises:* They are exercises introduced to improve the power, strength and endurance of a muscle. They may be
   - isometric: increasing muscle tension without altering the muscle length,
   - isotonic: exercises performed against resistance with joint movement.

FIGURE 30.8 Photograph showing continuous passive mobilisation equipment.

OCCUPATIONAL THERAPY

Assessment of the patient's capabilities, exploitation of residual skills and knowing his/her disabilities will help the patient rehabilitate himself for a particular occupation or vocational activity which may be different from one that he has already been doing.

KEY POINTS

- Rehabilitation signifies the whole process of restoring a disabled person to a condition in which he is able to resume a normal or near-normal life.
- Passive physiotherapy is used to relieve symptoms such as pain and discomfort.
- Active physiotherapy is used to restore function of a limb of a body part by repeated activity.
- Physical modalities used in physiotherapy include heat, high-frequency currents and therapeutic exercise.
- Wax bath contains molten wax and is useful in rheumatoid arthritis and degenerative bone diseases.
- Short wave diathermy uses high-frequency alternating current to produce its effect.
- Therapeutic ultrasound uses the piezoelectric crystal vibration effect in the range from 750 kHz to 3 MHz.
- Cryotherapy (cold therapy) helps in reducing the signs of inflammation
- TENS is electrical stimulation of superficial nerve fibres through which deep pain is masked using the principle of 'Gate' theory.

Appendixes

APPENDIX A: ABNORMAL GAITS

- *Scissoring gait:* It is seen in upper motor neuron lesion, commonly in cerebral palsy, in which the child walks with legs crossed in front of each other.
- *Ataxic gait:* It is due to lack of co-ordination of muscle movement in cerebellar lesions.
- *Antalgic gait:* The stance phase of the gait is shortened relative to swing phase due to painful conditions of leg.
- *High stepping gait:* The leg is lifted high to get ground clearance and lands on the forefoot. It is common in peroneal nerve palsy.
- *Festinant gait:* It is characterised by small shuffling steps with initial difficulty. Stopping after starting is also difficult. It occurs in parkinsonism.
- *Trendelenburg gait:* The weakened abductor muscles allow the pelvis to tilt down to opposite side (gluteus medius lurch). It occurs in abductor weakness, superior gluteal nerve palsy, rarely in polio and L5 radiculopathy.
- *Waddling gait:* It occurs due to weakness of proximal muscles of the pelvic girdle. The patient uses circumduction to compensate for gluteal weakness. This gait is seen in pregnancy, muscular dystrophy, SMA and hip dysplasia.
- *Magnetic gait:* Each step is initiated in a wresting motion carrying feet upwards and forwards. It occurs in normal pressure hydrocephalus.
- *Stiff hip gait:* In ankylosed hip, the patient swings the pelvis along with leg as one piece to bring it forwards. Examples are tuberculosis hip, rheumatoid hip and ankylosing spondylitis.
- *Stiff knee gait:* In ankylosed knee, the patients will circumduct the leg to bring it forwards. Examples are tuberculosis of knee and painful stiff knee.
- *Short limb gait:* The body of the affected side moves up and down every time the weight is borne on the affected leg. The gait is prominent when the limb length discrepancy is more than 2 inches.
- *Gluteus maximus lurch:* In gluteus maximus paralysis, the body swings backwards every time the weight is borne.
- *Quadriceps lurch:* In quadriceps paralysis, the patient walks with hyperextending the knee and thereby locking it.
- *Hand knee gait:* The patient keeps the hand on the knee to support and prevent the knee from buckling as in polio.

APPENDIX B: PLASTER TECHNIQUES

1. A plaster slab covers only a part of the circumference of a limb used to splint a fracture or soft tissue injuries.
2. A cast covers the full circumference of a limb.
3. A plaster can be applied to hold a closely reduced fracture or to splint the fracture.

4. Slab is made by unrolling a PoP and making layers to and fro for several layers.
5. Cast is done by wetting the PoP roll and encircled around the limb after adequate padding.
6. The sizes of plaster bandage recommended for normal application are as follows
   - *Upper arm and forearm:* 15 cm (6 inches)
   - *Wrist:* 10 cm (4 inches)
   - *Thumb and fingers:* 7.5 cm (3 inches)
   - *Trunk and hip:* 20 cm (8 inches)
   - *Thigh and leg:* 20 cm (8 inches)
   - *Ankle and foot:* 15 cm (6 inches)
7. A slab is applied initially to accommodate the tissue oedema and to avoid the disadvantages of cast. Cast conversion may be done after 2–3 days.

Above-Knee Plaster

- A plaster extending from the upper thigh to the metatarsal head posteriorly is above-knee slab.
- It needs reinforcement laterally in the thigh.
- It is used for fracture of both bones of the leg, supracondylar fracture femur, tibial plateau fractures and isolated fractures of fibula or tibia.

Below-Knee Plaster

- A plaster extending from 3–4 cm distal to appoint behind tibial tubercle to the metatarsal head is below-knee plaster.
- It is used in fractures around ankle, ankle subluxation and foot injuries.

Tube Plaster

- This plaster is applied from the upper thigh to the level just above the ankle.
- This is applied in knee injuries.

Above-Elbow Plaster

- A plaster applied from the upper arm to the metacarpal head with a cut-out at the thumb is above-elbow plaster.
- It is used in the fracture of distal humerus, fractures around the elbow and fractures of both bones of the forearm.

Below-Elbow Plaster

- A plaster extending from the olecranon to the metacarpal head dorsally is below-elbow plaster.
- It is used in fracture of distal radius and wrist injuries.

Volar Below-Elbow Plaster

- It is an anterior slab used in scaphoid fracture (extending to proximal palmar crease) and fracture of metacarpals and phalanges (extending up to finger tips).

U Slab

- This starts from the root of the neck and extends over the lateral aspect of the arm and takes a U around the elbow to end in axilla.
- It is used in fractures of humerus and shoulder injuries.
- Hanging arm cast is used in fracture of humerus in which reduction is achieved by gravity.

Complications of Plaster

1. Tissue oedema leads to compartment syndrome and gangrene. DANGER signs are unrelenting pain, swelling in fingers, hypoaesthesia and bluish discoloration of digits. This complication can be prevented by taking the following measures:
   - Elevate leg and arm whenever possible
   - All joints not included in plaster must be mobilised, especially fingers and toes
2. Plaster sores
3. Nerve palsies (if not adequately padded)

Clavicle Strapping

- An elastoplaster is applied over the fracture site from back to chest immobilising the clavicle.
- It is used in clavicle fractures.

Figure of Eight

- A wool roll bandage is applied in a figure of eight fashion around the shoulders in braced back position.
- It is used in clavicle fractures.

Jones Strapping

- It is used in acromioclavicular joint disruption.
- An elastoplaster is applied starting from back over the AC joint, extending over the anterior aspect of arm and takes a U to end posteriorly in the arm.

Buddy Strapping

- It is done in phalanges fractures of toe and fingers. The adjacent finger is used as a splint.

APPENDIX C: WALKING AIDS AND MOBILITY AIDS

Walking Aids

1. Restoration of normal walking is one of the goals of management of most of the orthopaedic disorders of leg and spine.
2. The common appliances used are walkers, crutches and sticks which help the patient to put apart the weight through the upper limb while walking.
   - *Walking frame:* It consists of four tubular uprights joined on three sides by horizontal tubes. It opens on one side for the patient to enter (Fig. C.1). It is mainly used indoors by elderly patients who are unsteady in gait and move with post-operative pain in the hip and knee. Reciprocating walking frame is one with two side horizontal bars connected to front horizontal bars by swivel joint.

FIGURE C.1 Walking frame.

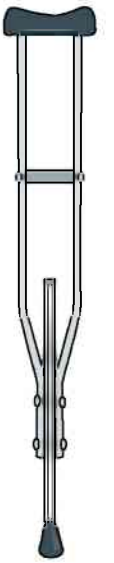


FIGURE C.2 Axillary crutch.

   - *Axillary crutches:* The crutches help in non-weight–bearing walking when one leg cannot be rested on ground due to pain or presence of plaster cast (Fig. C.2). It has two wooden uprights connected at the top by a padded transverse bar to fit into the lateral chest wall.
   - *Walking sticks:* It is prescribed to the patients who need partial weight bearing on the hip or knee (Fig. C.3). The length should be from the top of the greater trochanter to the ground.

Mobility Aids

- Wheel chair: When ambulatory capacity is permanently lost, wheel chair is prescribed. Battery-driven wheel chairs and even stairs climbing wheel chairs are also available.
- Hand-propelled three wheelers
- Hand-operated automobiles

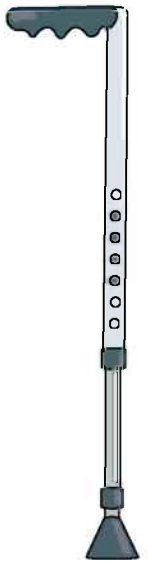
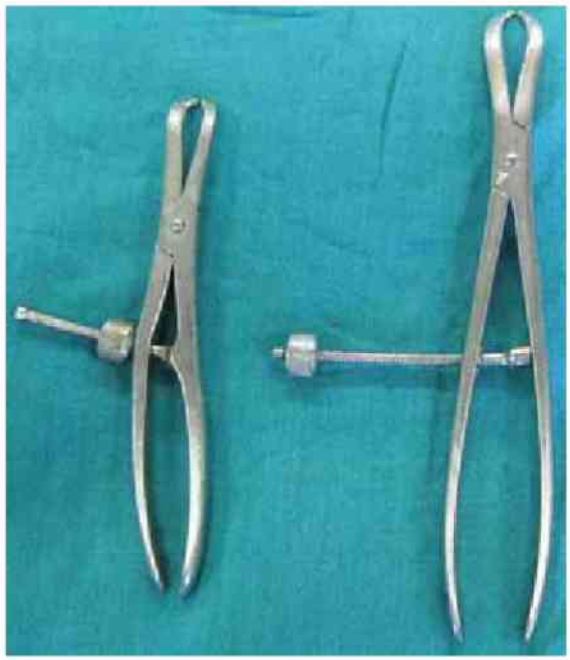


FIGURE C.3 Walking stick.

APPENDIX D: EPONYMOUS FRACTURES

- *Aviator's fracture:* Fracture neck of talus
- *Bankart's fracture:* Fracture of anterior glenoid associated with anterior shoulder dislocation
- *Barton's fracture:* Distal radius fracture involving the articular surface with dislocation of the radiocarpal joint
- *Bennett's fracture:* Intra-articular fracture of the base of first metacarpal
- *Boxer's fracture:* Fracture of distal fifth metacarpal
- *Bumper fracture:* Compression fracture of lateral tibial plateau
- *Chance fracture:* Horizontal fracture of the vertebral body
- *Chauffeur's fracture:* Intra-articular fracture of radial styloid
- *Chopart's fracture-dislocation:* Foot dislocation through talonavicular and calcaneocuboid joints with associated fractures, usually after ankle twisting
- *Clay-shoveler's fracture:* Fracture of spinous process of C7
- *Colles' fracture:* Distal radius fracture with dorsal angulation, impaction and radial drift
- *Cotton's fracture:* Trimalleolar ankle fracture
- *Galeazzi fracture:* Radius shaft fracture with dislocation of distal radioulnar joint
- *Hangman's fracture:* Fracture of both pedicles of C2
- *Hill Sachs fracture:* Impacted posterior humeral head fracture occurring during anterior shoulder dislocation
- *Hume fracture:* Olecranon fracture with anterior dislocation of radial head
- *Jefferson fracture:* Fracture of first cervical vertebra
- *Jones fracture:* Fracture of the base of fifth metatarsal extending into intermetatarsal joint
- *Lisfranc fracture:* Fracture dislocation through intertarsal joints
- *Maisonneuve fracture:* Spiral fracture of proximal fibula
- *Malgaigne's fracture:* Vertical pelvic fracture through both pubic rami and the ilium or sacroiliac joint with vertical displacement
- *March fracture:* Stress fracture of metatarsal shaft
- *Monteggia fracture:* Proximal ulna fracture with dislocation of radial head
- *Pipkin fracture-dislocation:* Posterior dislocation of hip with avulsion fracture of fragment of femoral head by the ligamentum teres
- *Pott's fracture:* Bimalleolar fracture of the ankle
- *Rolando fracture:* Comminuted fracture of the base of first metacarpal
- *Salter–Harris fractures:* Fractures involving a growth plate
- *Smith's fracture:* Distal radius fracture with volar displacement

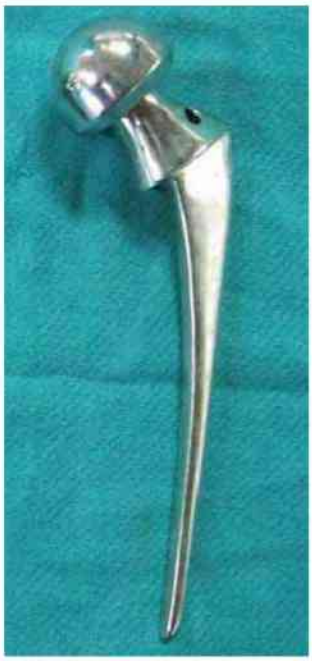
APPENDIX E: INSTRUMENTS



AO clamp

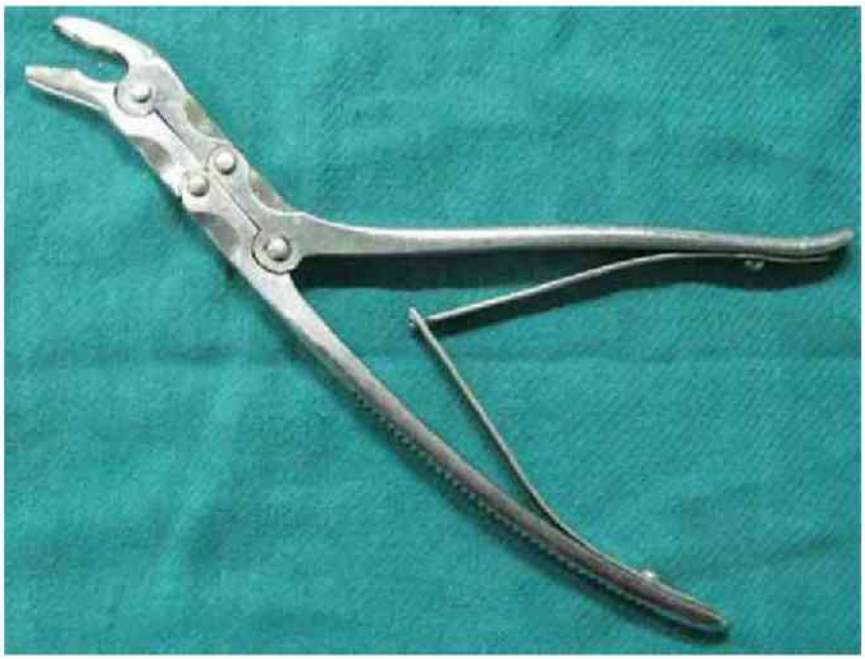
Asian dynamic compression plate

Austin Moore prosthesis

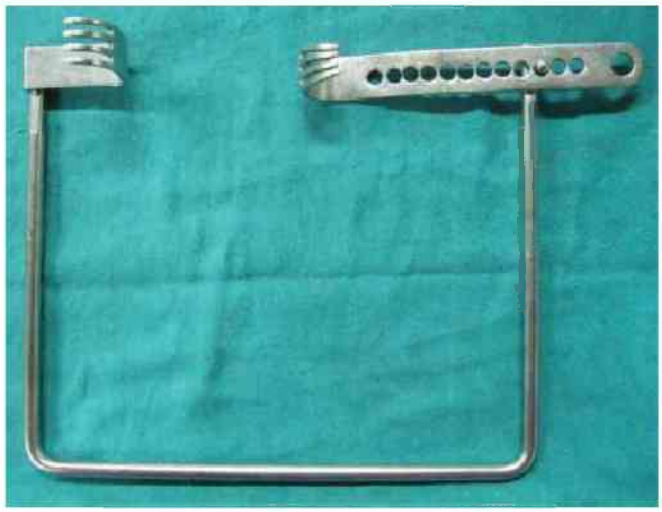


Bipolar prosthesis

Broad dynamic compression plate



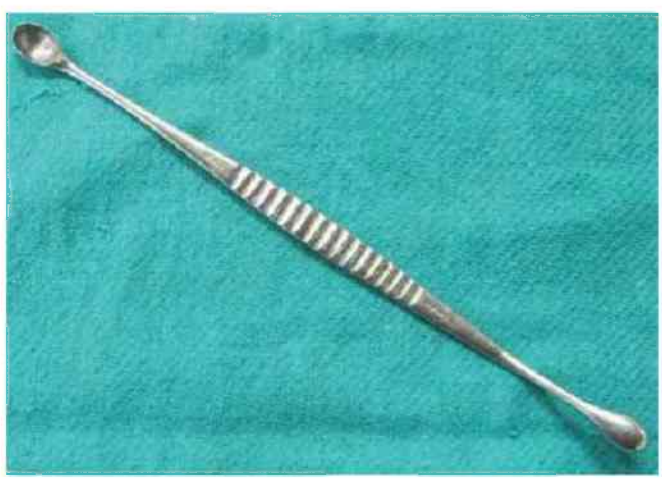
Bone nibbler



Charnley's retractor

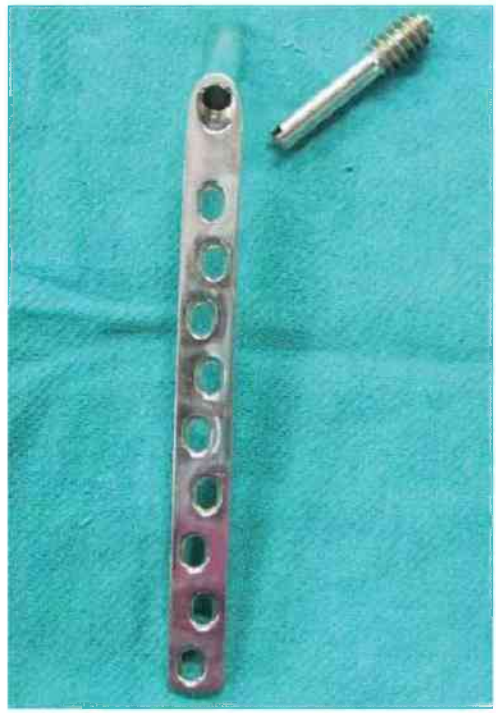
Cancellous screws

Cortical screws



Curette

Depth gauge

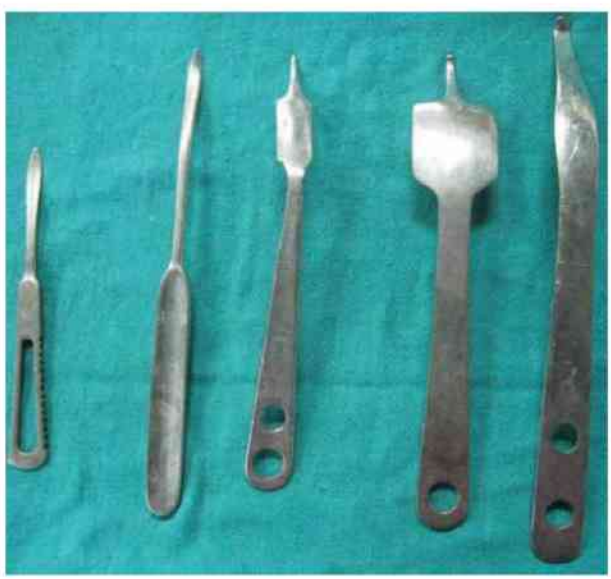


DHS screw and plate

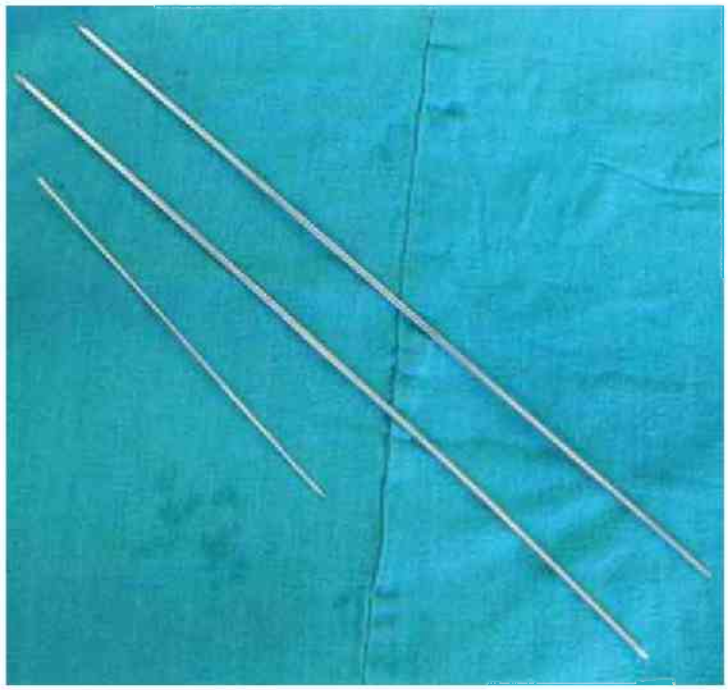
Drill bit

Drill sleeve

Drill



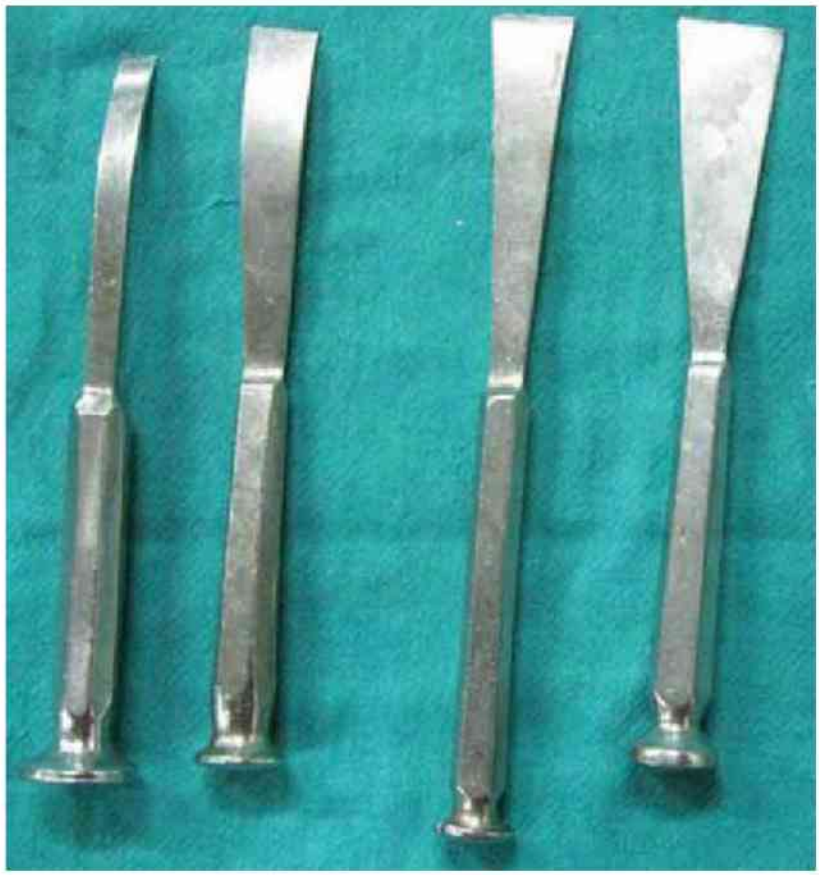
Hohmann Muller spike



Kirschner wires

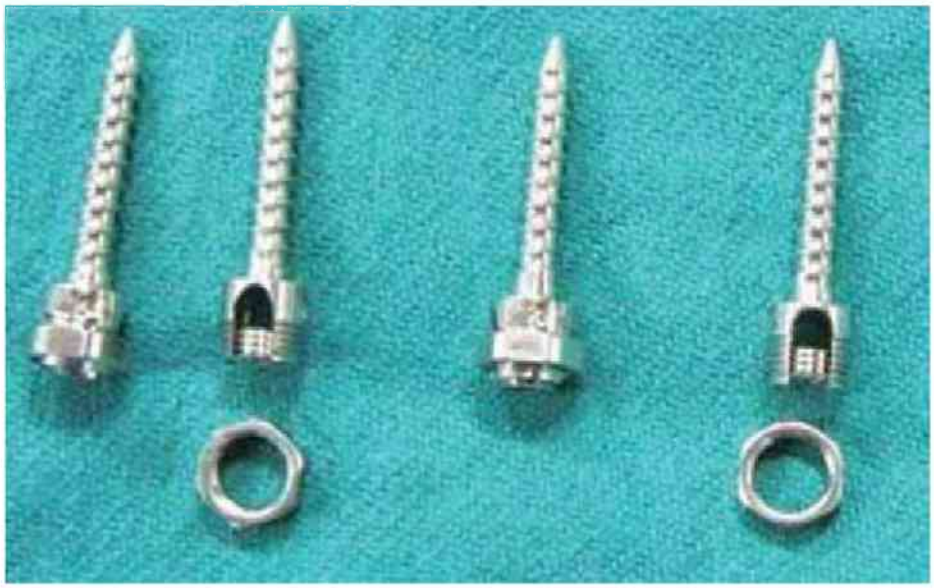
L buttress plate

Mallet

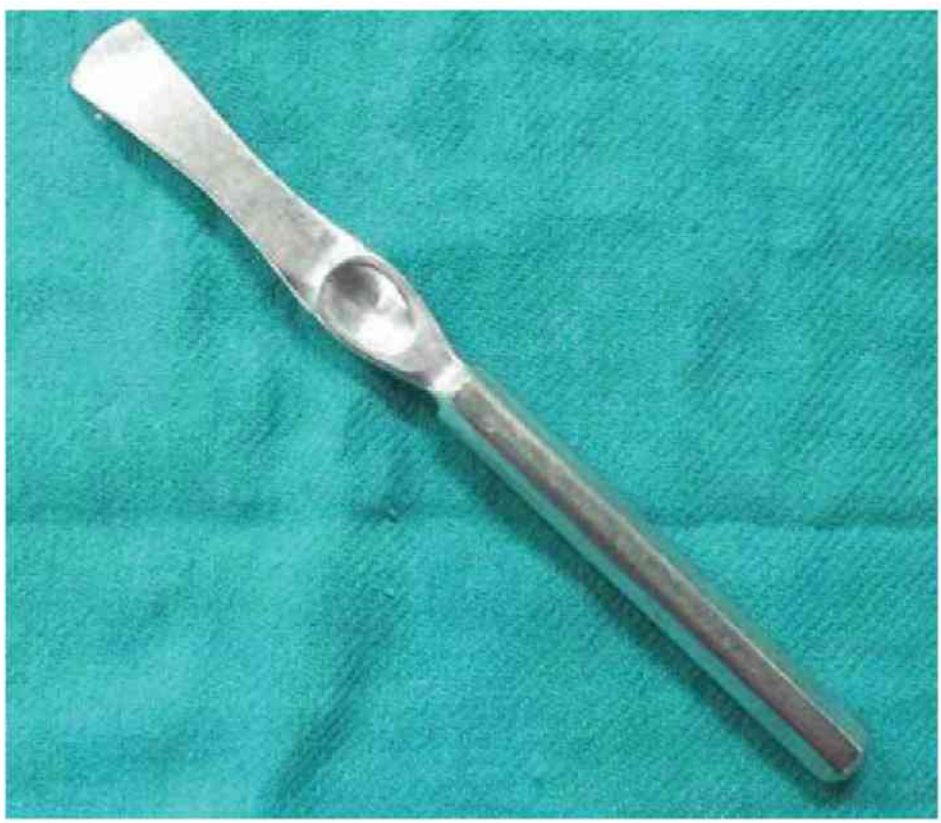


Osteotome

Narrow dynamic compression plate



Pedicular screws



Periosteal elevator

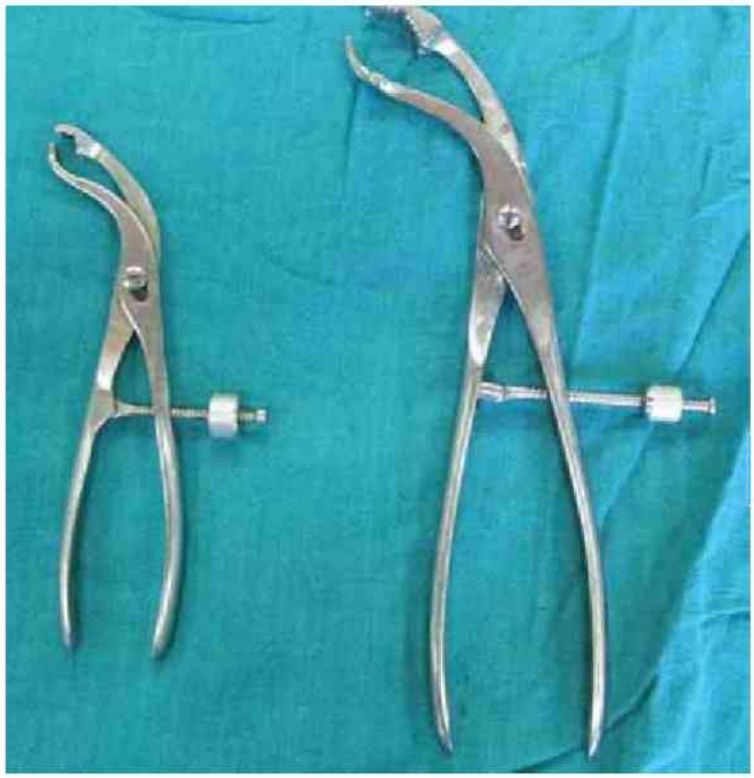
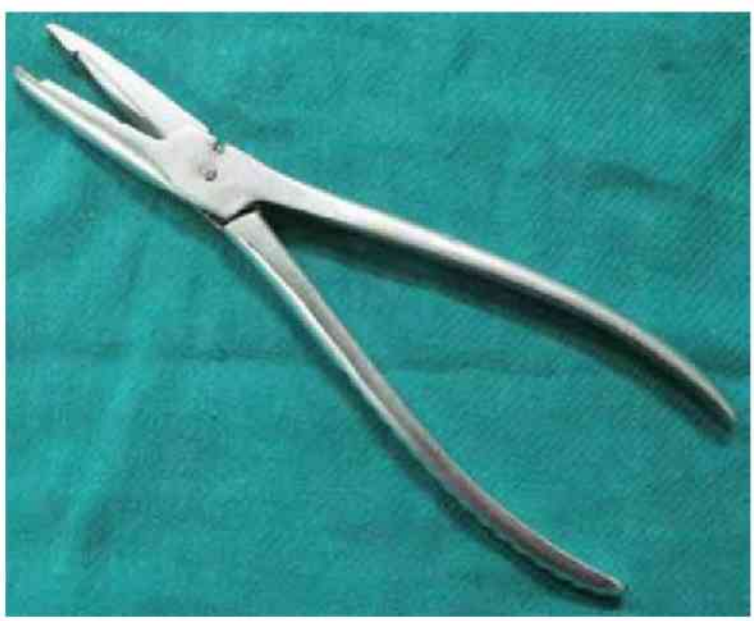
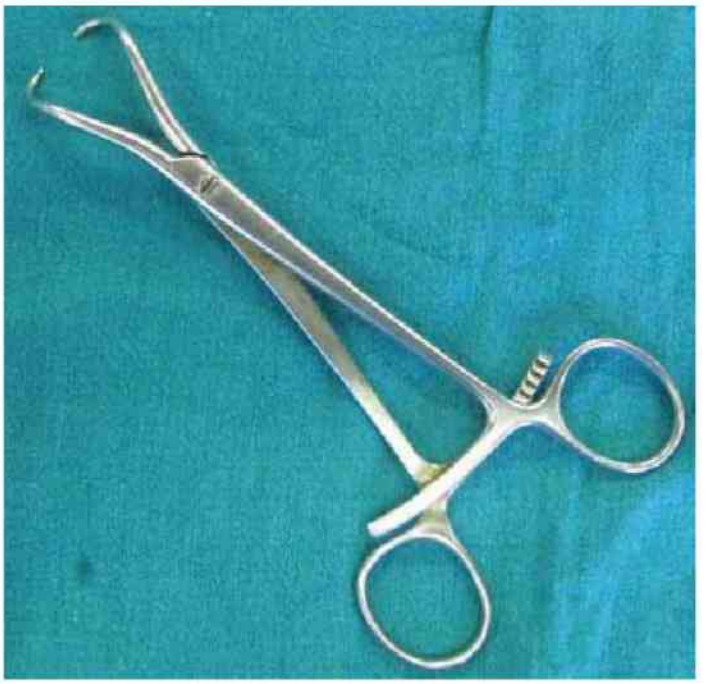


Plate holder



Plier

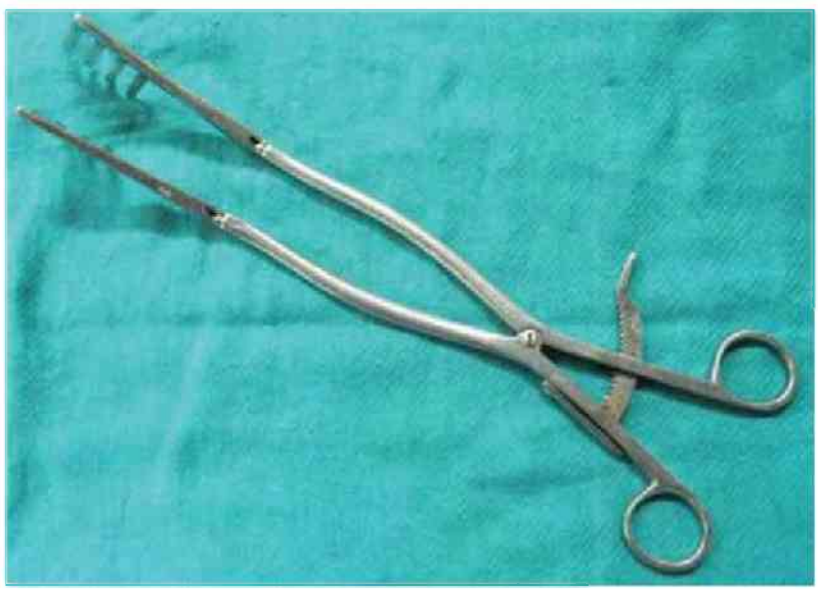


Point reduction clamp

Recon plate

Screwdriver

Tap



Self-retaining retractor

Stainless steel wire

T buttress plate

Thompson prosthesis

Index

B

C

D

E

F

G

H

I

J

K

L

M

N

O

P

Q

R

S

T

U

V

W

Y

Z