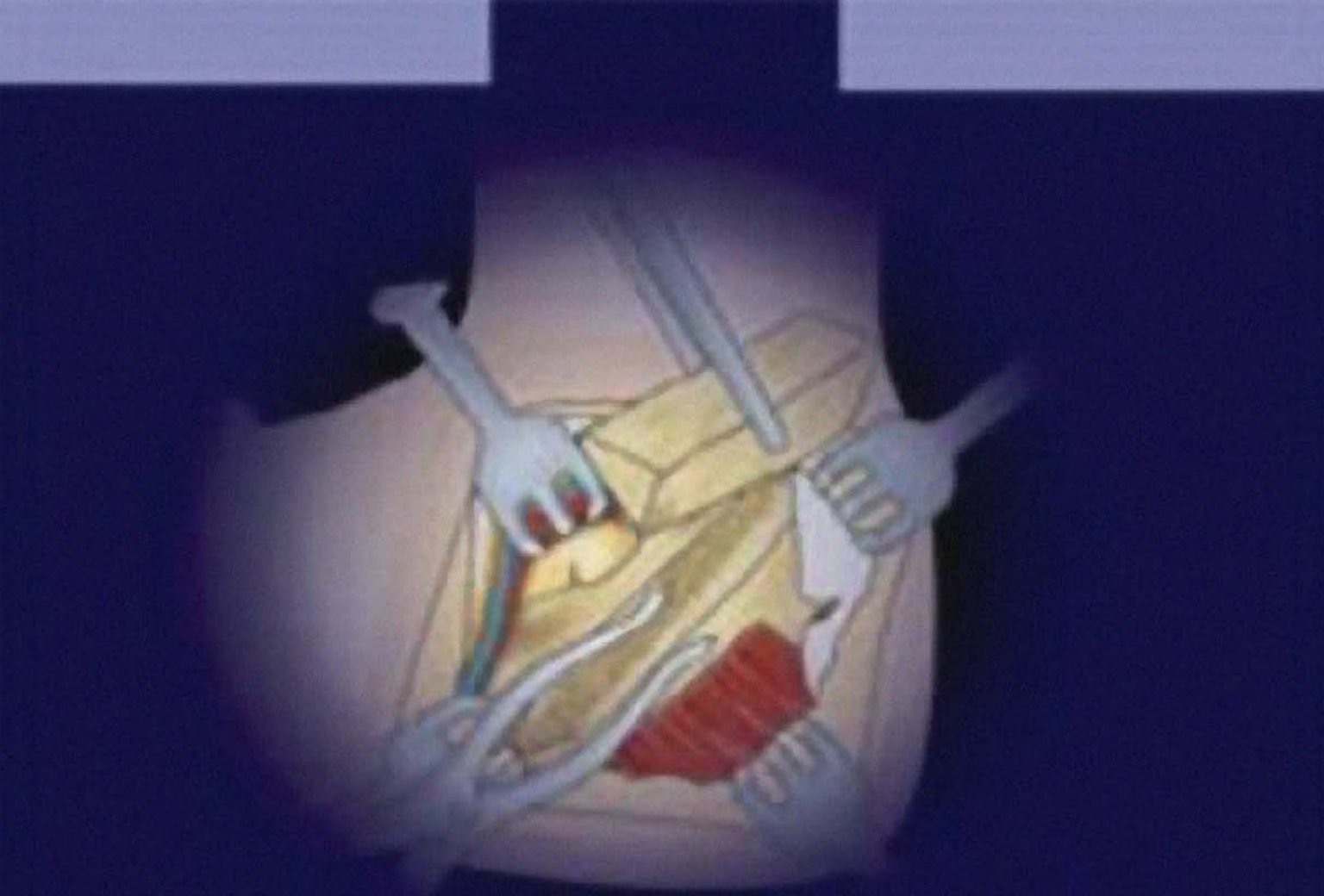

Essentials of ORTHOPEDICS

RM Shenoy

Foreword
M Shastharam Shetty

JAYPEE

Essentials of
ORTHOPEDICS

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With Eternal Blessings of
Shree Guru Nithyanandam

This book is dedicated to my parents
Dear Father Late Mr. Mohan R Shenoy,
who encouraged me constantly during my endeavor but
unfortunately did not live to see the release of this book
and
To my mother Mrs Padma Shenoy
who always inspires me

Foreword

It was a pleasure going through the pages of this highly informative well-presented book entitled "Essentials of Orthopedics" written by Dr Ravindra M Shenoy whom I had the pleasure of knowing him since his childhood and later as a student and now a revered colleague.

He has a inborn dominant gene of a writer and a leader. His mother an award winning writer of short stories and poems and father a grass root social worker of repute. Rightly he has dedicated this work to his parents.

I am sure this book will be a beacon of light and guide for undergraduates to understand orthopedics and the postgraduates to have their basics well forwarded and to the practicing surgeons as a ready reckoner.

I am sure this book will find a place in the shelf of every Orthopedic Surgeon.

I wish Dr Ravi all the best in his endeavors and may the good God bless him in all his endeavors.

Prof M Shantharam Shetty
President –IOA & AO Trustee from India
Vice Chancellor Nitte University
Chairman, Tejasvini Hospital
Mangalore, Karnataka, India

Good Wishes

Professor RM Shenoy is a teacher par excellence and committed to his profession. His dedication to academic activities is worth appreciating. He is an innovative Orthopedic Surgeon who is highly disciplined and meticulous in his work. He likes to improve the current status of medicine, always aiming at perfection. He expects the same from his pupils too. His original research of developing a single incision for exposure of forearm fracture speaks of his capabilities. This was published in the Journal of Bone and Joint Surgery 1995 British: 77B. No 4-568-570. I find it to be an extremely good and a cosmetic exposure. The book "Essentials of Orthopedics" written by him is his latest venture. It is going to be a boon for both undergraduates and postgraduates as well as practicing Orthopedic Consultants. As a teacher, I am thrilled to see one of my pupils Prof RM Shenoy, reaching great heights and carving out a niche for himself among the Orthopedic Surgeons. I wish him good luck and expect many more innovative things from him in future.

Prof PK Usman
Former Professor
Department of Orthopedics
Kasturba Medical College
Mangalore, Karnataka, India
Emeritus Professor and HOD
AJ Institute of Medical Sciences
Mangalore, Karnataka, India

Preface

Medicine today is a highly specialized science. Innovations are too many and what was held good yesterday becomes obsolete today. Newer and newer concepts continue to evolve and to keep abreast with these one should have a sound basic knowledge of the subject. The subject orthopedics has very few books which impart this basic knowledge to a medical student. The book ‘Essentials of Orthopedics’ is written keeping this fact in mind. It aims at imparting basic orthopedic knowledge to a student of medicine who has acquired basic knowledge of pre- and para-clinical subjects. The original concepts have been highlighted and the persons who put forth these concepts have been duly recognized. Every attempt has been made to narrate the concepts in a simplified manner keeping the originality. Wherever possible illustrations have been placed to help the reader to understand the subject. Radiographs have been used wherever required so that the learning process becomes easy because of the visual effect they impart. The essential topics have been covered methodically and adequately. On the whole this book is a complete book which imparts essential basic orthopedic knowledge to a medical student. Questions have been provided at the end of each chapter for revision as well as preparation for the examination. Suggestions are most welcome.

RM Shenoy

Acknowledgments

The author being honored by the revered Chancellor of Manipal University Dr Ramdas M Pai for completing 25 years of meritorious service.

At the outset, I would like to acknowledge my alma mater Kasturba Medical College which nurtured me for 39 long years first as an undergraduate student, then as a postgraduate and finally now as its faculty. I am thankful to the illustrious Chancellor of Manipal University, Dr Ramdas M Pai who guided me and gave me an opportunity to buildup my career as a young Orthopedic surgeon. The guidance and encouragement which I had received then has put me on a sound footing today.

I am indebted to my teachers Professor M Shantharam Shetty, the Vice Chancellor of Nitte University and President of Indian Orthopedic Association and Professor PK Usman, the two living legends who shaped my career as a teacher and an Orthopedic surgeon.

I am grateful to my innumerable colleagues, interaction with whom considerably improved my knowledge and to the innumerable undergraduate and postgraduate students whom I have taught during the past 27 years and in the process learnt a lot.

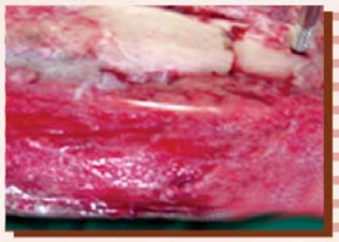
I am extremely grateful to my present colleagues Dr Deepak Pinto, Dr Vivek Mahajan, Dr Saurabh Bansal and Dr Harshvardhan who helped me in many ways during the preparation of this book.

I acknowledge the inspiration given by my mother Smt Padma Shenoy, a State Award winning novelist, whose literary talents perhaps I have inherited, my wife Renu for kindling the spark within me to write a book for the benefit of the students thereby enabling me to transmit this scientific, pure and unsullied knowledge to the future generation and my daughter Archana for helping me in the proofreading and correction, as well as giving all possible help and support during completion of this task.

I also acknowledge my younger brother Dr Surendra Shenoy and sister-in-law Dr Shalini Shenoy for the help they have renderd in the preparation of this book.

And last but not the least I acknowledge the efforts of M/s Jaypee Brothers Medical Publishers (P) Ltd., and its enterprising Chairman and Managing Director Shri Jitendar P Vij, the dedicated team of staff at Delhi office and Mr Venu Gopal the Branch Manager of Bengaluru office who have worked with me promptly and efficiently in bringing out this book in a grand manner.

Contents



1

Introduction to Fractures and Dislocations

- *Definition of a Fracture, Types, Healing, and Treatment of Fractures*
- *Complications and their Treatment*
- *Recent Concept of Fracture Healing*
- *Definition of Dislocation, Types, Management in General and Complications*

Fractures and dislocations are among the most common injuries seen in day to day practice. It is important to remember the following basic facts to understand these injuries.

A. A force of considerable magnitude is necessary to cause these injuries (Unless the bone is already weak or the structure of a joint is already disturbed due to disease).

B. The resultant failure pattern (deformation) is directly proportional to the nature, magnitude and direction of the force.

C. It is possible to classify these failure patterns with certain limitations.

D. The treatment protocol is based on the nature of the failure pattern and follows a definite path which is consistent, with minor variations.

E. Healing is indirectly proportional to the severity of the injury.

F. Complications that develop with these injuries are related to the severity of the deforming force, the resultant failure pattern and the site and multiplicity of the injury.

G.. Though the terms ‘ Fracture and Dislocation’ refer to the bone and joint pathology, one should always remember that in a skeletal injury, there is considerable damage to the soft tissue envelope that surrounds the bones and the

joints. Namely the periosteum and the muscles. This soft tissue injury has an inverse relation to the normal healing process.

Fractures

Definition

Fracture is a break in continuity of a bone or loss of normal anatomical continuity of a bone.

Types of Fracture

I. Depending on basic nature
   a. Closed or simple.
   b. Open or compound (Figs 1.1A to F).

II. Depending on the displacements
   a. Displaced fracture
   b. Undisplaced fracture.
      – Incomplete fracture
      – Complete fracture.

III. Depending on the nature of the fracture line (Figs 1.2 to 1.8)
   a. Transverse fracture.
   b. Oblique fracture.
      – Long oblique
      – Short oblique
   c. Spiral fracture.
      – Long spiral
      – Short spiral
   d. Comminuted fracture: In this type, there are more than two fragments at one fracture site.
   e. Segmental fracture: In a single bone, fracture occurs at two different levels.
   f. Intra-articular fracture: A fracture that involves (extends into) the articular surface of a joint.
   g. Avulsion fracture: This is a fracture occurring due to a pull by a muscle, tendon or a ligament at its insertion to the bone, e.g. mallet finger, fracture of the olecrenon process of the ulna.

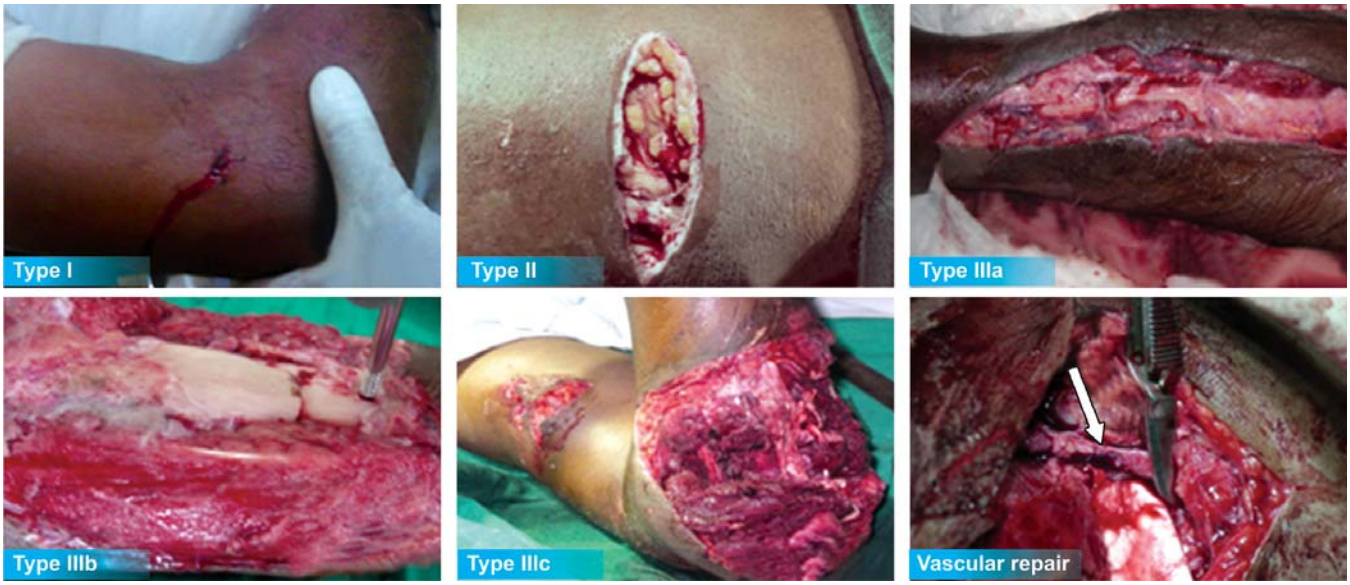


Figure 1.1

Classification of open fractures—based on the classification by Gustilo and Anderson
Criteria for classification: Extent of injury to skin, soft tissue, bone, vessels and the degree of contamination.
Type I: Wound smaller than 1 cm in diameter, no skin crushing with no or little contamination. Fracture pattern is not complex
Type II: A lacerated wound larger than 1 cm but without significant soft tissue crushing, no degloving, or contusion with moderate contamination. Fracture pattern may or may not be complex.
Type III: An open injury with extensive soft tissue crushing and contamination, fracture pattern is single or complex. Injury is further subdivided into three types:
a. Adequate soft tissue coverage of the fracture can be acquired at closure.
b. Periosteal stripping is seen.Inadequate soft tissue coverage at closure. Hence, soft tissue reconstruction is necessary.
c. Open fracture that is associated with vascular injury (or nerve injury or both) and that needs repair.

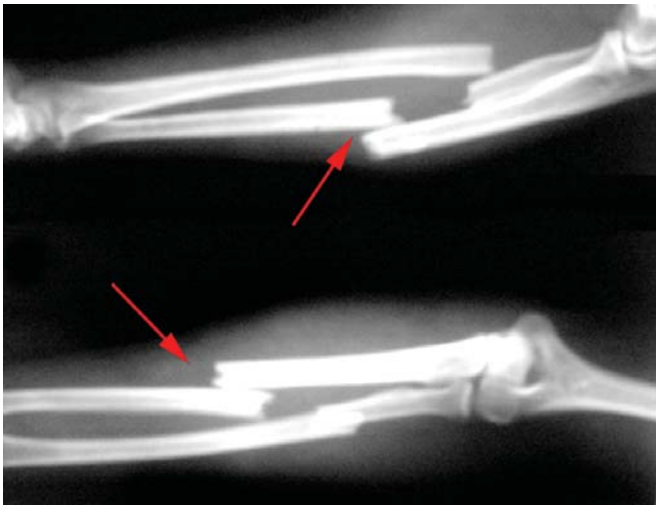


Figure 1.2

Radiograph showing fracture of radius and ulna. Note the transverse nature of the fracture line.

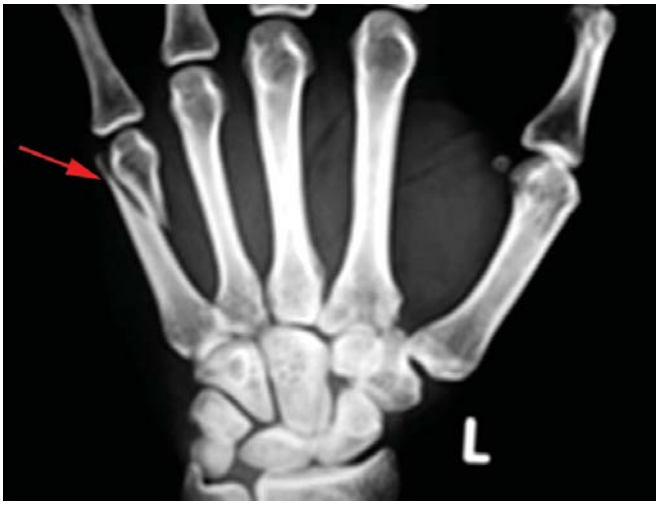


Figure 1.3

Radiograph showing a short oblique fracture at the neck of the 5th metacarpal bone of the left hand.

h. Multiple fractures: Many bones are fractured in the same individual.

Deforming Forces

The deforming forces causing fractures can be classified as:

a. Direct
b. Indirect

Direct impact causes severe injuries, e.g. open fractures, comminuted fractures, etc.

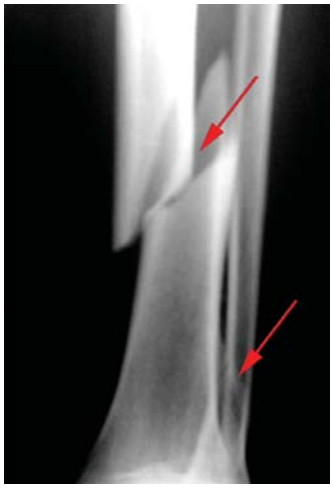
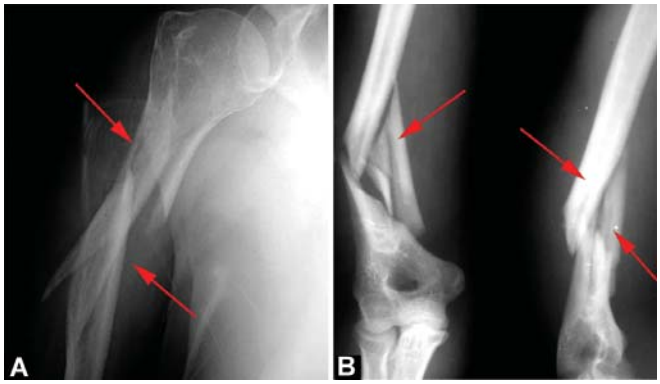


Figure 1.4

Radiograph showing a short spiral fracture of the tibia and the fibula, in the lower third.



Figures 1.5A and B

Radiograph showing fracture of upper and lower humerus respectively and comminution at the fracture site with many fragments of bone which are displaced.

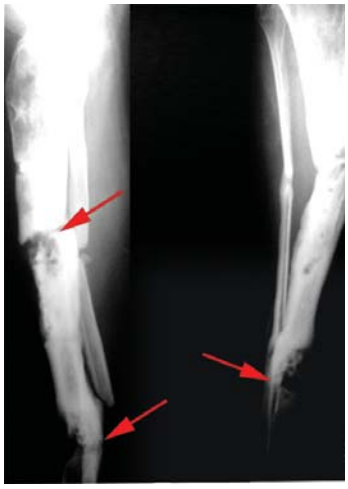


Figure 1.6

Radiograph of an open segmental fracture of tibia and fabula with secondary infection and changes of chronic osteomyelitis. Bone resorption is seen at the fracture site. Both the fracture sites are showing features of nonunion.

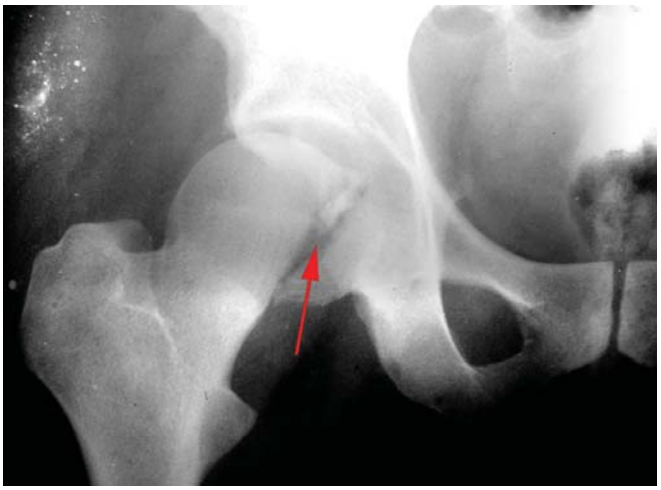


Figure 1.7

Radiograph showing fracture of the head of the femur. This is a very rare injury and generally associated with a dislocation of the hip. In this case, there was posterior dislocation which was reduced. The reduction achieved is obviously not congruous.

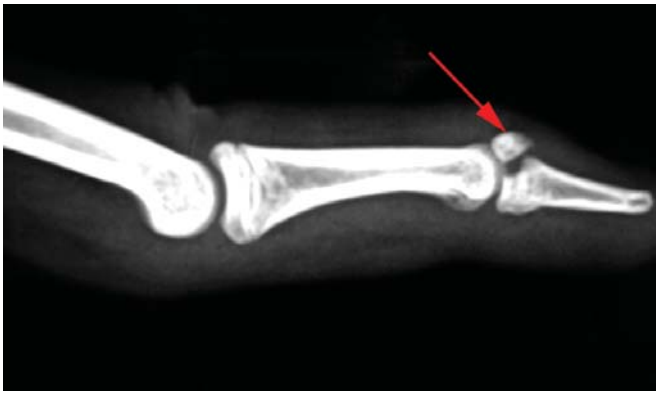


Figure 1.8

Radiograph of an avulsion fracture of the base of distal phalanx. The resultant deformity is a 'Mallet finger'. The patient is a goal keeper and the injury happened during a soccer match when he blocked a goal kick. Avulsion force is exerted by the lateral slips of the extensor expansion.

Indirect impact causes less severe injuries and is classified as follows:

i. A bending force—produces a transverse fracture.
ii. A torsional force—produces a spiral fracture.
iii. A combination of both bending and torsional force—produces a comminuted fracture with a butterfly fragment/fragments.

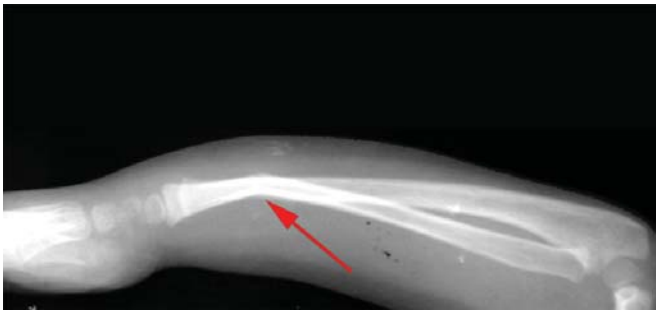


Figure 1.9

Radiograph showing greenstick fracture of both, the radius and the ulna. Note the gross deformity.

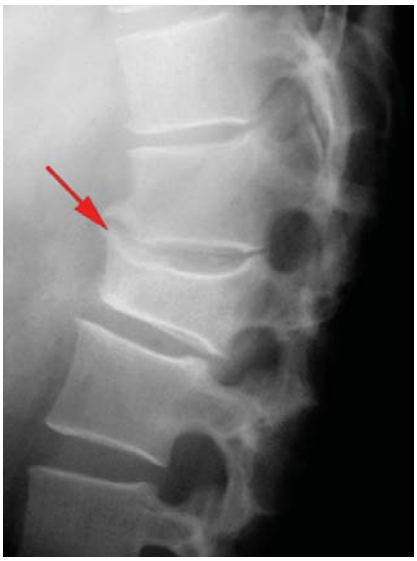


Figure 1.10

Radiograph showing a compression fracture of L2 vertebra. Note the decrease in anterior height of the vertebral body.

Specific Types of Fractures

Greenstick Fracture (Fig. 1.9)

It is called so because the bone breaks like a greenstick branch of a tree. Only a part (one side) of the bone breaks and rest of the bone bends. It is an incomplete break occurring in the bone and seen in children whose bones are more elastic, soft and pliable. There is no abnormal mobility.

Compression Fracture (Fig. 1.10)

It is seen in the vertebral column wherein the height of the vertebral body decreases following fracture.

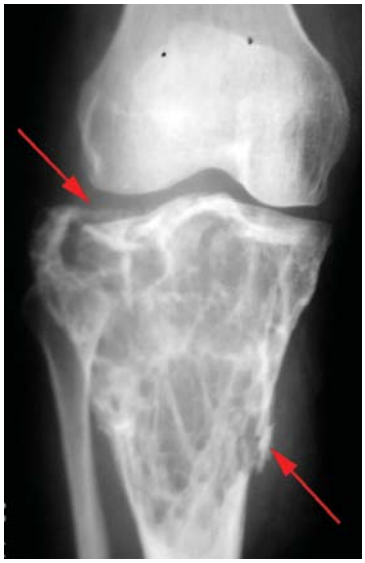


Figure 1.11

Radiograph showing a Giant cell tumor arising from the upper end of tibia with multiple pathological fractures due to thinning of the cortex. Pathological fracture is a late feature of a Giant cell tumor

Pathological Fracture (Fig. 1.11)

It is a fracture occurring as a result of pre-existing pathology. The pathology softens the bone considerably and this soft bone yields to a very trivial trauma and fractures, e.g. malignancy, osteomyelitis, etc.

Stress Fracture (Fig. 1.12)

It is a type of pathological fracture due to unaccustomed stress getting concentrated on one part of the bone, e.g. March fracture seen in soldiers after a long route march.

Signs and Symptoms of a Fracture

1. Pain and Swelling
2. Deformity.
3. Loss of continuity.
4. Irregularity.
5. Crepitus.
6. Bony tenderness.
7. Loss of function.
8. Abnormal mobility.

(Abnormal mobility is a sign to be observed and not a sign to be elicited in a fresh fracture. It is very painful because the movement is taking place at the fracture site. It is the sure sign of a fracture and further examination to ascertain the fracture is unnecessary.)

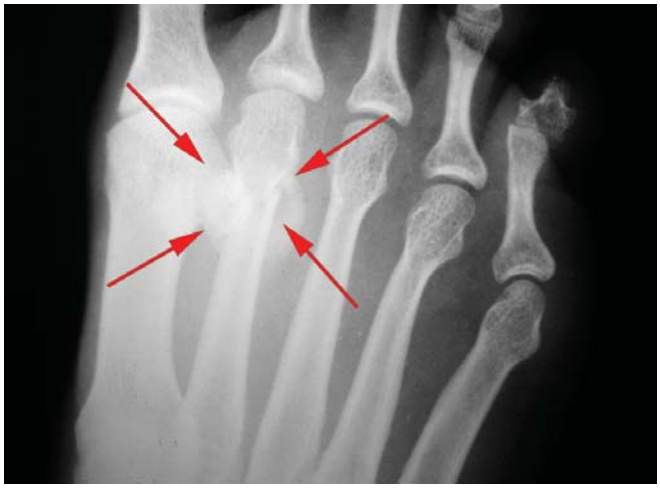


Figure 1.12

Radiograph showing 'March fracture' at the neck of 2nd metatarsal. Note the healing response by formation of abundant callus.

Healing of a Fracture

Healing takes place in stages and over a period of time (approximately 4 weeks minimum). Four distinct stages are recognized (Fig. 1.13).

1st Stage: Stage of Hematoma Formation

This is an important stage of fracture healing. During the process of fracture the blood vessels are torn and hence bleeding occurs almost immediately. Hematoma acts as a vehicle delivering required material for union and clearing the unwanted material by a process of chemotaxis of cells. If this stage of hematoma is deficient as seen in cases of open fractures, healing is interfered with and fracture fails to unite.

(Hence open fractures are to be converted into closed fractures as early as possible to promote healing. Many a times this is not possible and *nonunion is accepted and treated accordingly at a later date.)*

2nd Stage: Stage of Cellular Proliferation

Within 8 hours of the fracture there is inflammation resulting in subperiosteal and endosteal cellular proliferation. These cells surround the broken ends of the bone. At the same time the clotted hematoma progressively gets absorbed and new capillaries start infiltrating these cellular masses.

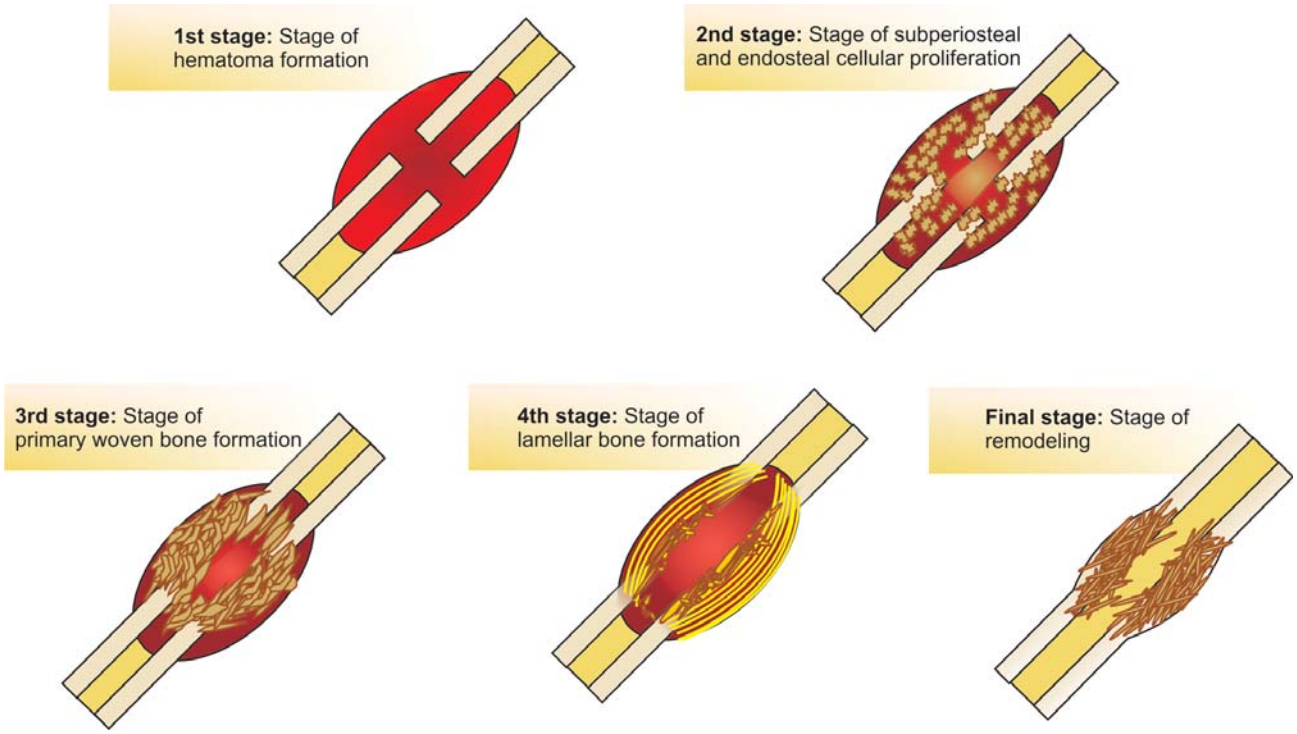


Figure 1.13
Stages of healing of fracture

3rd Stage: Stage of Primary Woven Bone Formation (Soft Callus)

The proliferating cells which are mainly osteogenic and chondrogenic start to get incorporated into the fibrogenic matrix under the influence of Bone Morphogenic Proteins (BMP), Transforming Growth Factor Beta (TGF-β) and Fibroblast Growth Factor (FGF), thus forming primary woven bone. This bone is soft as it is not fully mineralized. This is supposed to occur during 2nd and 3rd week.

4th Stage: Stage of Lamellar Bone Formation (Hard Callus)

Mineralization occurs and this primary woven bone is transformed into lamellar bone. This is supposed to occur between 3 and 6 weeks

This is hard bone and is seen as a bridge or a cuff across the fracture site. It indicates early stage of fracture union.

Stage of Remodeling

This stage is better not considered as one of the stages of fracture healing because remodeling takes place only after the fracture unites (heals) and takes months and years. Here the body attempts to give the normal shape and strength to the fractured bone or in other words to restore its preinjured status. **Remodeling is rapid in children and in growing bones and slow in adult bones and almost nil in osteoporotic bones.**

Note: ***Healing of a fracture in a cancellous bone does not follow these stages. Cancellous bone heals by direct formation of osteoblastic new bone.***

Factors Influencing Healing

a. Factors not (at all) in control of the treating doctor
   - Nature of the trauma
     - High velocity trauma.
     - Low velocity trauma.
   - Nature of the fracture.
   - Vascularity of the bone.
   - Age of the patient.

b. Factors in control (some control) of the treating doctor.
   - Proper reduction.
   - Adequate fixation.
   - Adequate immobilization.
   - Prevention of distraction.
   - Prevention of infection.
   - Maintaining adequate nutrition.
   - Adequate management of other comorbid conditions.

Management of a Fracture

The treatment begins at the site of injury. The first step is to apply a splint to the injured part and look for other associated injuries. Namely, the vascular, the visceral and the neurological, by doing a thorough general and systemic examination. Visceral injuries when present may be life-threatening, e.g. liver laceration, splenic rupture, hemopneumothorax, etc. Early repair of these structures is of paramount importance. In polytrauma maintaining the airway, the breathing and the blood pressure and treatment of shock and hemorrhage is essential. A skilled paramedical team should be available to transport a severely injured patient to the specialized center where definitive treatment is instituted. Delay is detrimental.

Basic Methods of Treating a Fracture

1. Immobilization in a cast.
2. Closed reduction and immobilization in a cast.
3. Open reduction and internal fixation.
4. Closed reduction and internal fixation (with the help of C-Arm imaging).
5. External fixation.
6. Tractions.

All the above methods are practised and can be employed to treat a fracture depending on the indication. All these methods help to enhance the biological process of healing by maintaining the anatomical alignment and providing the necessary stability at the fracture site. Every method has its own merits and demerits and to be chosen carefully. Aim of fracture treatment is to minimize the confinement to bed, achieve union at the earliest and make the patient to regain his activity as early as possible.

Complications

Following are the complications that can occur after a fracture.

A. *Specific and local complications*
   1. Nonunion.
   2. Delayed union.
   3. Malunion.
   4. Sudecks atrophy.
   5. Associated nerve injuries.
   6. Associated vascular injuries (Fig. 1.14).
   7. Associated visceral injuries.
   8. Infection leading to osteomyelitis and pyogenic arthritis.
      (as a consequence of open fracture or surgical sepsis.)

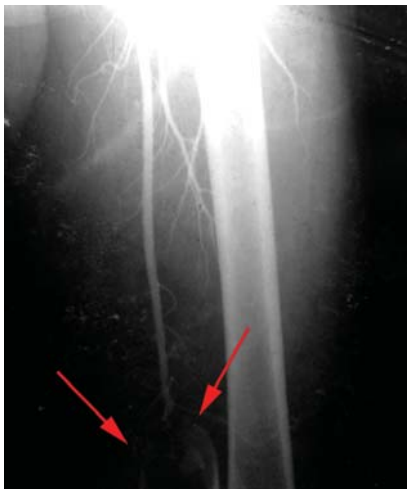


Figure 1.14
A femoral artery arteriogram showing injury to the femoral artery secondary to a badly displaced fracture of the lower third of the shaft of the femur (Vascular injury in a fracture is an emergency. Hemorrhage, shock and compartment syndrome is common. It has to be treated immediately to save the limb and the life of an individual).

B. *Systemic and general complications.*
   1. Shock and hemorrhage.
   2. Fat embolism.
   3. Crush syndrome.
   4. Pulmonary embolism.

A. *Specific Complications*

Nonunion

When a fracture fails to show progressive signs of union at review both clinically and radiologically for a consecutive period of three months after the specified time expected for union it is known as nonunion.

Clinically, it is diagnosed by *painless abnormal mobility.* Radiologically, it shows rounding of the ends of the bones, sclerosis of the margins and poor callus. The fracture site is not viable and the *biological response at union has ceased.*

Nonunion is seen commonly in following fractures because of damage to the vessels resulting in loss of / poor blood supply.

a. Fracture of the waist of the scaphoid
b. Fracture of the neck of the femur
c. Fracture of the neck of the talus
d. Fracture of the lower 1/3 of the tibia fibula

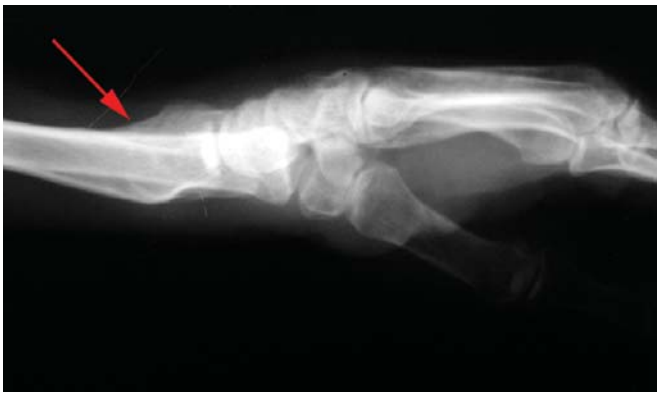


Figure 1.15

Radiograph showing lateral view of wrist and hand showing malunited Colles' fracture with a classical 'Dinner fork' deformity.

Delayed Union

A fracture is said to have gone in for delayed union when there is undue delay at union.

This is so because the attempt at union for some reason does not proceed towards complete union or the attempt at union is not strong enough or adequate enough to progress towards complete union.

Clinically, it is diagnosed by *painful abnormal mobility*. Radiologically, it shows callus but callus formation is not adequate enough to bridge the fracture site and cause complete union. The fracture site is viable and *there exists a biological response at the fracture site (but inadequate).*

Abnormal mobility is a sign to be elicited in nonunions and delayed unions.

Malunion (Figs 1.15 and 1.16)

When a fracture unites in anatomical malalignment it is known as malunion. These malalignments are *angulation, rotation and over-riding.* Single or malalignment in one plane is rare. Usually, there is combination of two or all the three malalignments. Angulation causes angular deformity, rotation causes rotational malalignment and over-riding causes shortening.

Clinically, it is diagnosed by the presence of deformity with no abnormal mobility. Radiologically, it shows deformity and malalignment with adequate bridging callus.

The normal 'Neck shaft angle' between the head, neck and shaft of the femur varies from 117-137°, average being 127°. If it is less than 117°, it is known as COXA VARA and more than 137°, it is known as COXA VALGA. An intertrochanteric fracture malunites in Coxa Vara.

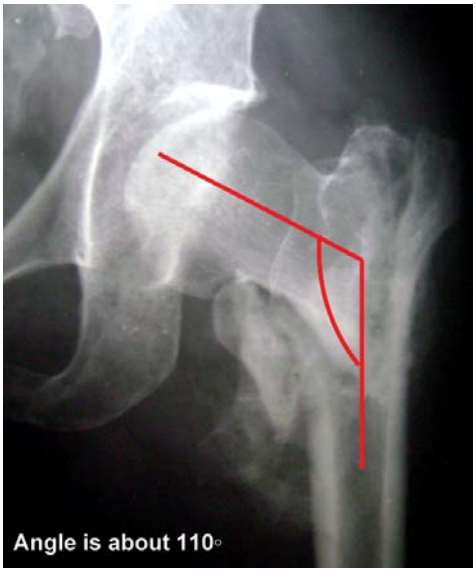


Figure 1.16

Radiograph showing malunion of a comminuted intertrochanteric fracture.

Note the neck shaft angle. It is reduced. Hence, there is Coxa vara deformity at the hip.

(Left alone most of the intertrochanteric fractures unite in coxa vara.This happens because of the cancellous nature of the bone that is present at the fracture site).

Classification of Delayed and Nonunions

Basically, fractures which do not unite and need a secondary procedure for achieving union are considered as the ones that have gone in for delayed or nonunion. They show both clinical and radiological features of the same.

Depending on Strontium 85 uptake at the ends of the fracture site the vascularity (viability) is assessed and classified as hypervascular (hypertrophic) and avascular (atrophic). *(Based on Description by Weber BG and Cech, O Pseudarthrosis, Berne Switzerland 1976, Hans Huber Medical Publisher)*

Hypervascular nonunions (true delayed unions) (Fig. 1.17A)

a. *Elephant foot type:* Presents with exuberant expansile callus and the picture resembles the foot of an elephant. It is the result of movement occurring at the fracture site before union has occurred, e.g. premature weight-bearing.
b. *Horse hoof type:* Presents with little callus and picture resembles a horse hoof. Perhaps, this is the result of instability at the fracture site following inadequate reduction or fixation.
c. *Oligotrophic type:* These are hypervascular but are not hypertrophic and do not show callus.

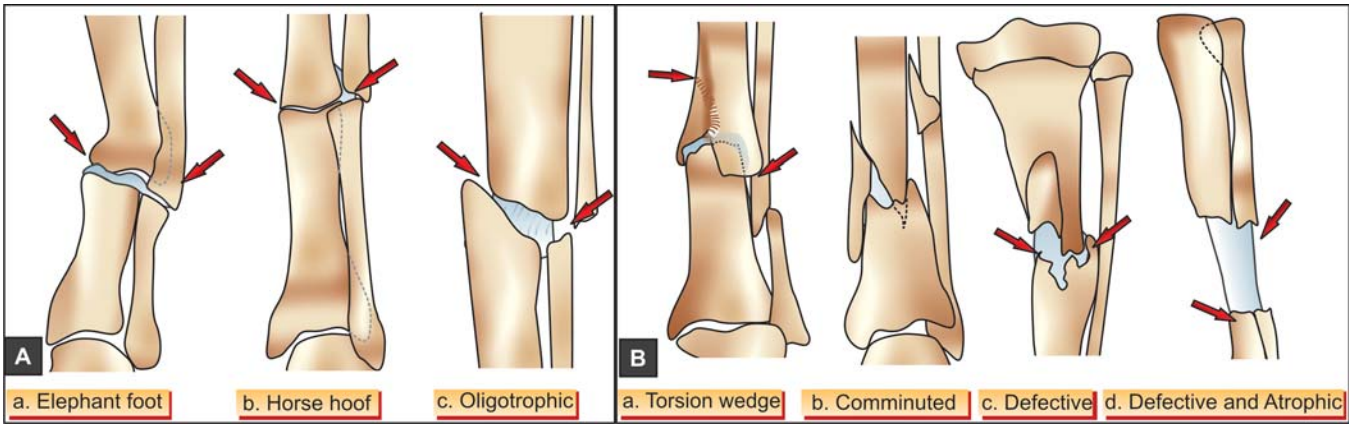


Figure 1.17

(A) Hypervascular type (B) Avascular type

(Ref: Based on description by Weber BG and Cech O; Pseudarthrosis, Berne Switzerland 1976, Hans Huber Medical Publisher).

They are considered to be the result of major displacement/distraction persisting after treatment.

Avascular nonunions (true nonunions) (Fig. 1.17B)

a. *Torsion wedge type:* Seen when there is an intermediate fragment with poor blood supply. It unites on one side but does not unite on the other.
b. *Comminuted type:* Is the result of many intermediate fragments with poor blood supply.
d. *Defect type:* Seen when there is bone loss.
e. *Atrophic type:* Seen when intermediate fragments are small and are missing. The defect is replaced by scar tissue.

(See the X-ray pictures in the Figs 1.18A to G in the next page).

Treatment of Delayed and Nonunions

Standard methods: Cancellous bone grafting is the procedure of choice to achieve union. Cancellous bone grafting is done only after confirming good apposition of the ends of bone without any soft tissue interposition. Freshening the edges of the fractured bone is a must. Cortical grafting is done in cases of defect non unions to bridge the defect, prior to the placement of cancellous graft.

(In delayed union with exuberant callus and when the cause is certain that it is the movement taking place at the fracture site which is preventing union, rigid fixation and immobilization alone may result in union).

To conclude the role of cortical graft is to bridge the defect. The role of cancellous graft is to induce osteogenesis. Hence, when the need is osteogenesis only, cancellous bone grafting is the procedure of choice. When the need is to bridge the defect as well as to induce osteogenesis both cortical and cancellous bone grafting (corticocancellous grafting) procedure is to be chosen.

The procedure needs immobility at the site for the incorporation of the graft and the union to take place. Thus internal fixation/external immobilization is necessary.

Sources of bone graft

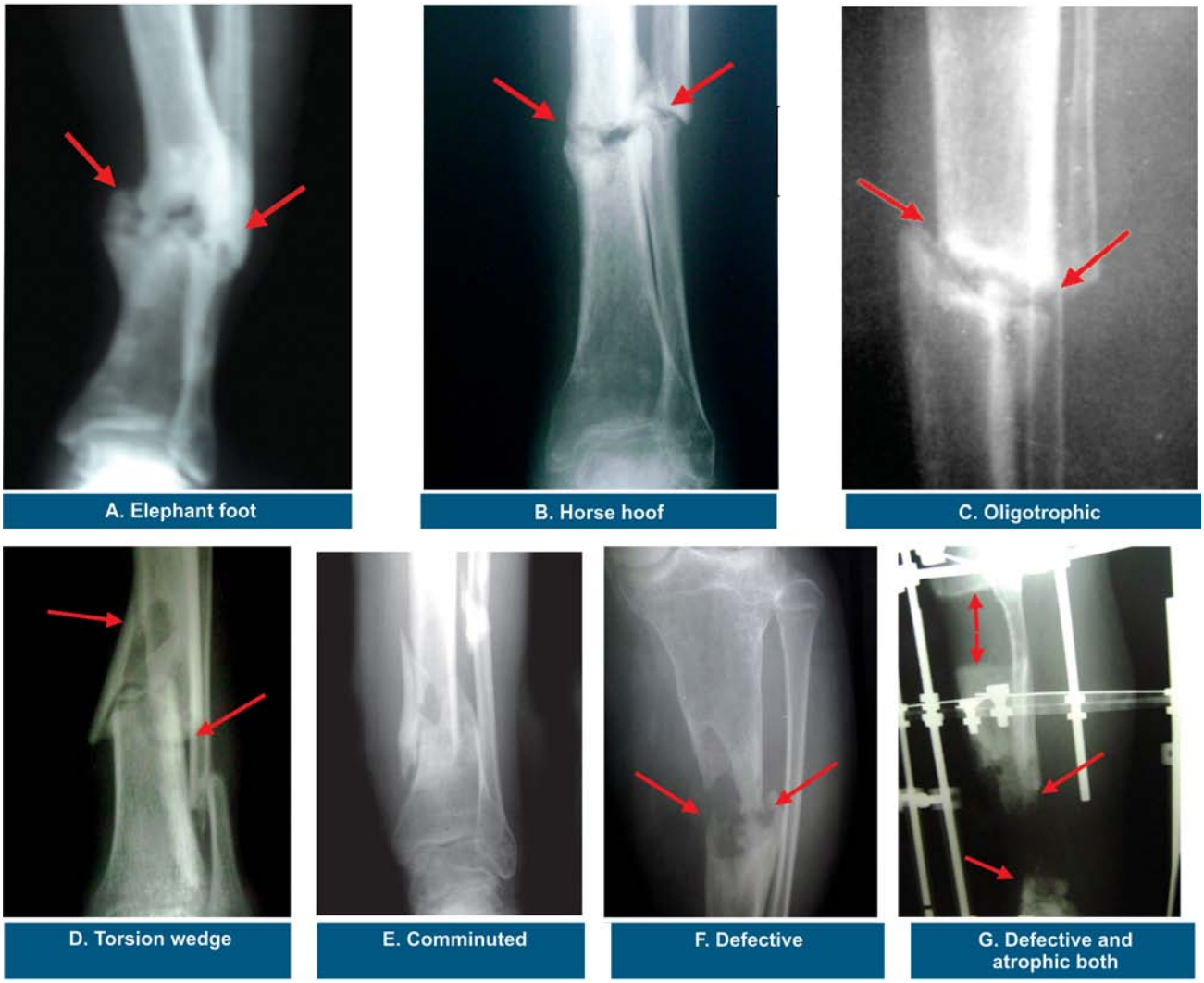
a. Allo/Homograft from the same species, e.g. bank bone, maternal fibula.
b. Auto/Isograft from the same individual.

Source of cancellous graft: Iliac crest, excised ribs, excised head and neck of femur.

Source of cortical graft: Upper 2/3 of the fibula, anteromedial tibia.

Specialized Methods for the Treatment of Delayed Unions and Nonunions

a. Distraction/compression osteogenesis based on the principle of Illizarov; It is proved by Illizarov that controlled progressive distraction and/or compression leads to tissue regeneration. This principle is of immense use when the skin condition does not permit open bone grafting procedures (Figs 1.19A and B).



Figures 1.18A to G

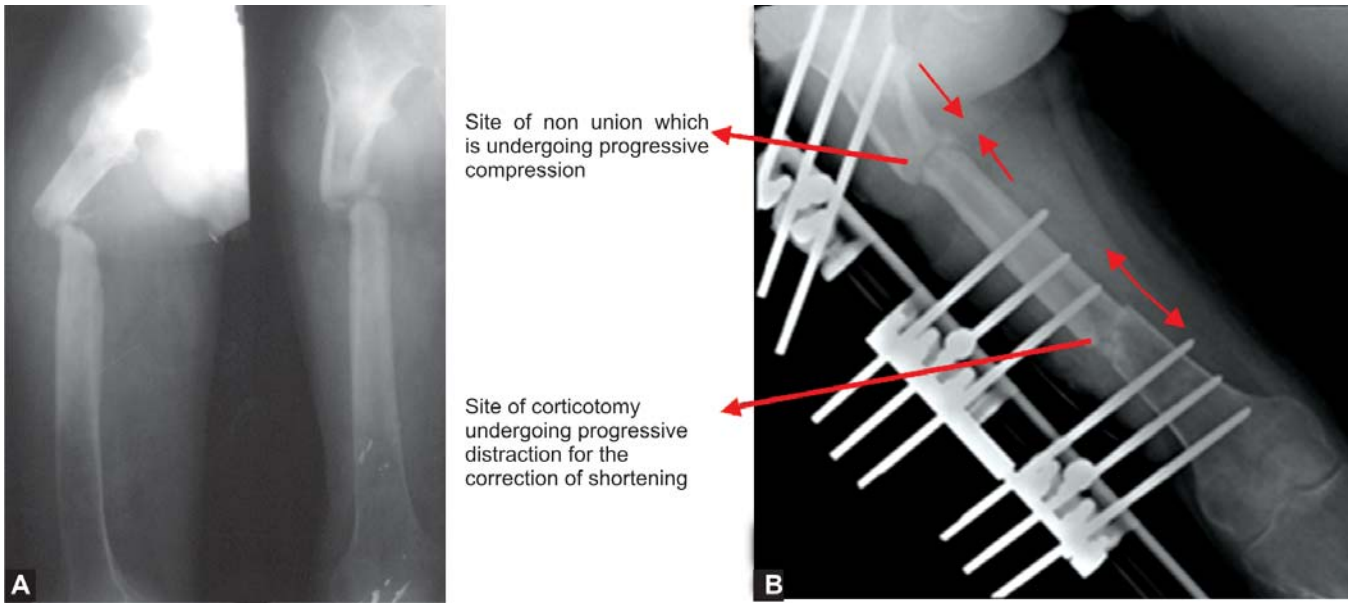
Radiographs showing the different types of pseudarthrosis based on the description by Weber BG and Cech O. Figure 1.18G shows a bone distraction (transportation) procedure in an attempt to salvage the limb, being carried out in a combined, both defective and atrophic nonunion, employing Illizarov methodology.

b. Bone marrow grafting procedure; in this the bone marrow is aspirated and injected into the site of nonunion/delayed union. The sample may be injected *de novo* before clotting or may be injected in a concentrated form with anticoagulants after centrifuging and obtaining the concentrate under aseptic precautions (Figs 1.20A and B).

Treatment of Malunions

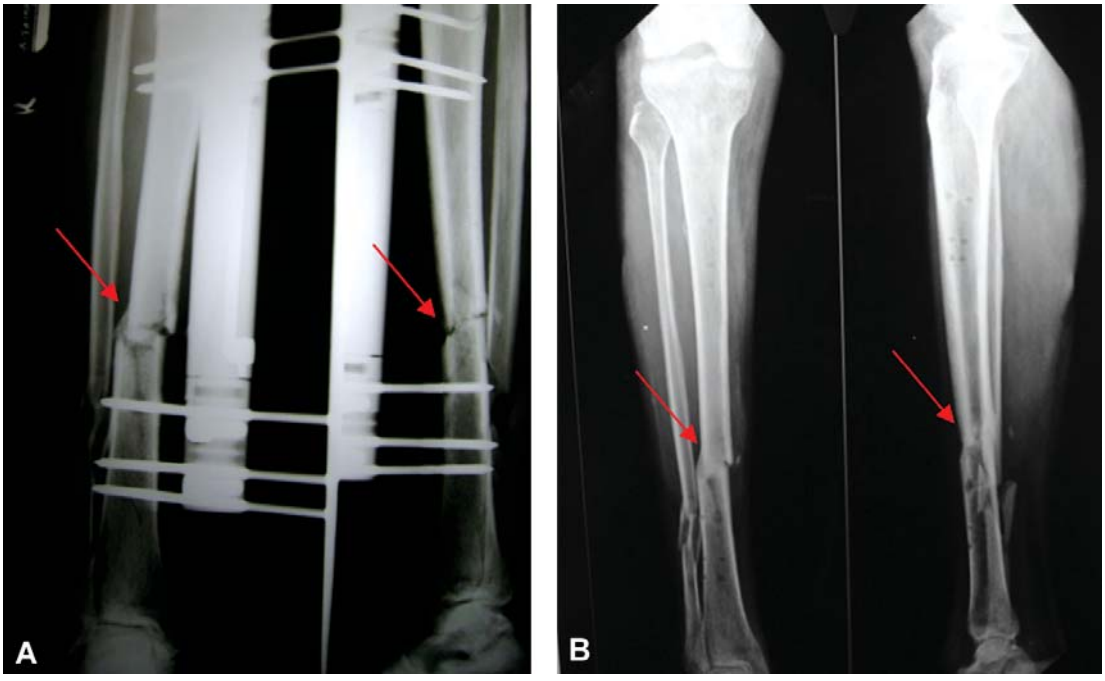
Malunions are treated by the help of a surgical procedure known as corrective osteotomy. In this procedure, the site of malunion is osteotomized surgically and the deformity is corrected accordingly. The alignment thus obtained is maintained by internal fixation devices and appropriate external immobilization (very rarely with only external cast). Immobilization is continued till healing takes place.

Osteoclasis: This is a closed procedure wherein the malunited bone is broken manually, reduced and aligned and immobilized in appropriate cast. This procedure is indicated in early cases of malunion and in children when the bridging callus has just formed and the callus formed is soft. Osteoclasis should never be attempted after consolidation of callus.



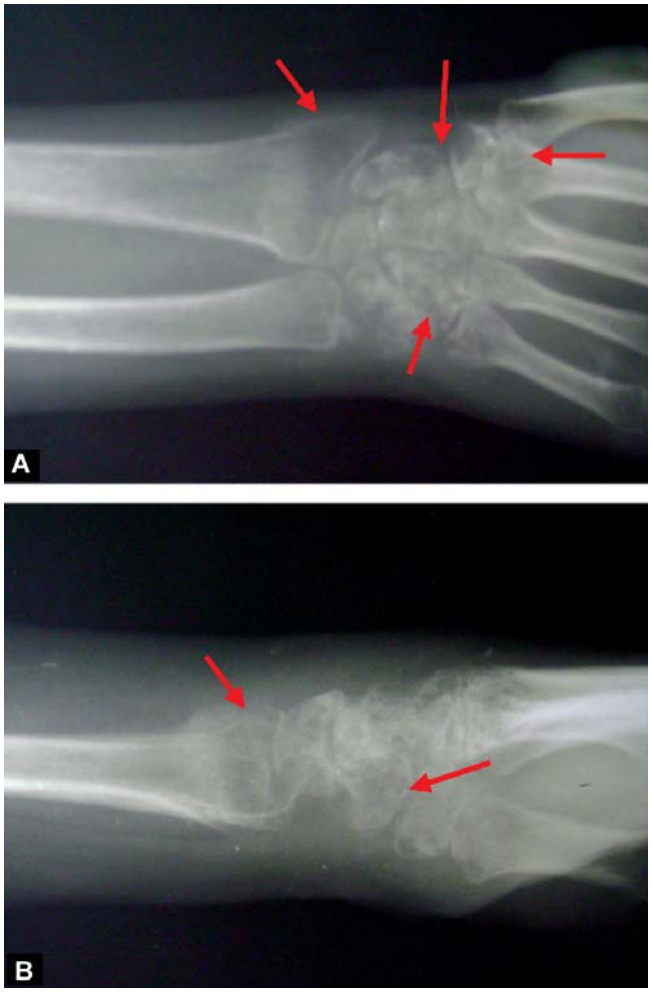
Figures 1.19A and B

(A) Nonunion of fracture shaft femur associated with shortening of the limb, (B) Procedure of compression/ distraction osteogenesis



Figures 1.20A and B

Radiographs showing union in a 12 weeks old ununited type III open fracture tibia fibula after 2 injections of *de novo* bone marrow grafting at 6 weekly intervals



Figures 1.21A and B

Radiograph showing a normally united Colles' fracture with Sudeck's Osteodystrophy. Note the severe nature of demineralization and rarefaction in the cancellous areas of the bones of the forearm wrist and the hand.

Sudeck's Atrophy (Figs 1.21A and B)

Synonyms are Reflex Sympathetic Dystrophy, Complex Regional Pain Syndrome, Causalgia, Shoulder hand Syndrome.

It is a chronic condition characterized by pain and stiffness in the extremity as a result of dysfunction of central or peripheral nervous system.

Pathogenesis is thought to be due to

a. Activation of pain pathways by the release of catecholamines, e.g. norepinephrine following injury.
b. Exaggerated inflammation and immune response following injury.

Signs and symptoms

a. Burning pain.
b. Skin temperature changes (warm initially, cold later).
c. Skin color and texture changes.
d. Alteration in nail and hair growth.
e. Swelling and stiffness of joints.
f. Impairment of function of extremity.

Diagnosis

Diagnosis is made by clinical signs, X-ray (shows demineralization) and nuclear bone scan (shows increased uptake).

Treatment

Treatment aims at reducing sympathetic overactivity.

Drug therapy is given using following drugs as per need:

- Opiates.
- Anti-inflammatory analgesics.
- Antidepressants.
- Antiepileptics.
- Calcitonin.
- Corticosteroids.

Interventional therapy as follows

- Physical and exercise therapy.
- Psychotherapy to relieve anxiety and depression.
- Sympathetic nerve block.
- Spinal cord stimulation.
- Intrathecal drug pump.

B. *Systemic and General Complications*

Shock and Hemorrhage

Hemorrhagic shock: This is the most common complication of a severe injury. Inadequate oxygenation of tissues leads to a chain of events resulting in hypotension, multi organ failure and death.

Measures to be adopted include

a. Adequate oxygenation of tissues by maintaining the airway and administration of oxygen (important and to be employed in all types of shock).
b. Immediate stoppage of bleeding.
c. Treatment of lost volume by infusing Saline/Ringer lactate and Blood.

Diagnosis of hemorrhagic shock is made by cold and clammy feeling of the body, hypotension (initially diastolic and later both systolic as well as diastolic). Tachycardia, tachypnea and poor urine output. Loss of more than 40% of the blood volume will pose grave danger to life.

Neurogenic shock: This type of shock is commonly seen in spinal cord injury. The disturbance in sympathetic innervation

causes decrease in the heart rate, dilatation of peripheral vessels and as a result fall in blood pressure. Monitoring the central venous pressure and infusion of plasma expanders help in the management of this difficult problem.

Cardiogenic shock: This type of shock is the result of chest injuries, e.g. tension pneumothorax impeding venous return, Myocardial contusion, etc. should be managed promptly depending on the underlying cause.

Septic shock: This type of shock is seen as a result of septicemia and usually occurs several days after the open injury.

It is characterized by increased warmth of the body, tachycardia, hypotension (little fall in systolic but marked fall in diastolic pressure) tachypnoea. Multiorgan failure is common. Septicemia with gram-negative organism may not increase the body warmth.

Aggressive antibiotic therapy along with all other supportive measures is essential for the recovery.

Fat Embolism

In a long bone fractures always, there is dissemination of fat globules from the marrow into the bloodstream. This can also happen from a spongy bone. At times these fat globules block the capillaries of the pulmonary and the cerebral vessels causing the fat embolism syndrome. The syndrome is more common in a young patient with multiple fractures.

Diagnosis of the fat embolism is difficult. But the following features developing after a fracture should arouse the suspicion.

a. Confusion and restlessness.
b. Increased body temperature and tachycardia in a patient who is otherwise normal and not in a state of shock.
c. Breathlessness.
d. Petechiae over the chest, back, axilla, the conjunctival folds and the retina.

In severe cases of pulmonary embolism, there can be blood tinged frothy secretion which is coughed out by the patient or in case of cerebral embolism patient may become comatose. There is no definite treatment for fat embolism except supportive measures of giving high concentration of oxygen and maintaining capillary perfusion. Low molecular weight dextran may help in maintaining capillary perfusion. If the oxygen saturation falls severely, intubation and ventilation is the treatment of choice. If there is blood loss and hemoglobin is low, blood tansfusion is to be given. Good quality blood is essential for maintaining the oxygen saturation.

Crush Syndrome

First described by a British physician Eric Bywaters in the year 1914. It is a traumatic rhabdomyolysis occurring because of crushing. Also known as reperfusion injury it occurs in those whose limbs are compressed for a long-time, e.g. limb trapped in a vehicular collision or buried in a land slide, etc. Such a limb is deprived of blood flow for quite a long-time and the tissues release toxic metabolites because of cell death. When the compression is released and reperfusion occurs myoglobin from the dead muscle and the toxic metabolites are released into the bloodstream.

The flow of oxygenated blood through the damaged area causes formation reactive oxygen metabolites resulting in further damage to the tissues. The resultant hyperkalemia, hypocalcemia and metabolic acidosis may cause cardiac arrest. Renal failure may occur when myoglobinuria is severe and blocks the renal tubules.

Management: Large amount of fluid infusion to dissolve the metabolites and simultaneous forced diuresis with help of diuretics is the treatment of choice till myoglobinuria becomes negligible. Crushed tissues should be radically debrided and a tight compartment when present should be adequately decompressed. Dialysis may be necessary when renal failure is observed.

Pulmonary Embolism

Pulmonary embolism commonly occurs in those patients who are confined to bed for a prolonged period after the fracture and in those elderly high-risk patients who present with major fractures. Heparinization of blood with the help of low molecular weight heparin and prevention is the treatment of choice.

Recent Concept of Fracture Healing

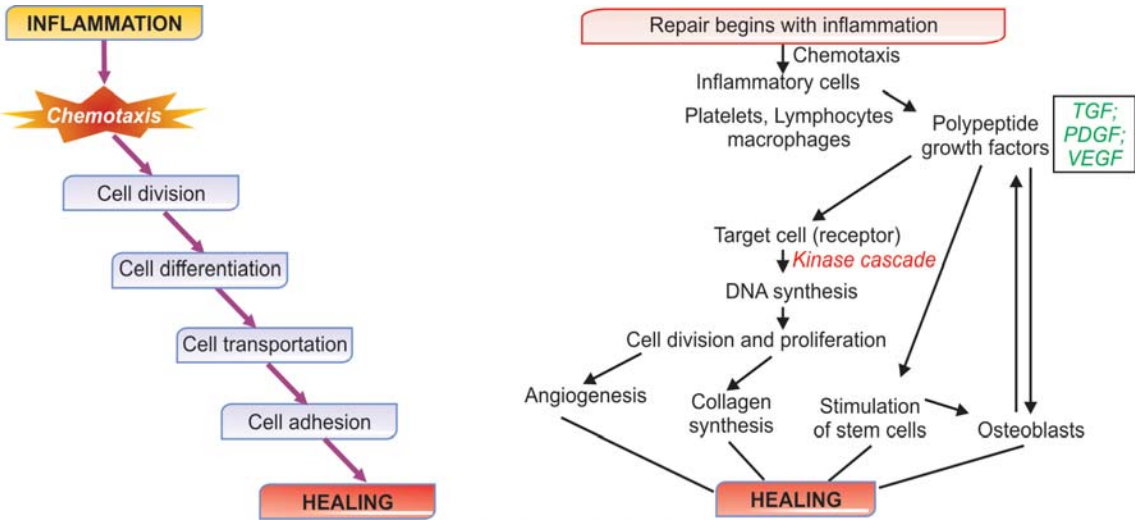
Regenerative Medicine

Regenerative medicine is an evolving branch which tries to restore the normalcy from a diseased state, by going deep into the cellular and molecular response. Thus if fracture healing is analysed, it is found that the healing process begins with inflammation. As a result of this a series of events follow, bringing to the site of fracture an outflow of chemical mediators, cells and growth factors (TGF, etc). This in turn initiates a chain of reactions resulting in fracture healing (Flow chart 1.1).

Thus, after understanding the exact process of healing, these days, methods which are different from conventional

Flow chart 1.1

Schematic representation of a chain of events occurring at the cellular level which is responsible for the healing of the fracture.



ones have evolved. Bone marrow grafting, Stem cell injection alone or insertion of a Scaffold mixed with Stem cells are some of the methods currently practiced. In the years to come this concept is going to revolutionize the treatment of fractures and nonunions.

Dislocations

Definition of Dislocation and Subluxation

When the two articular surfaces are totally out of contact it is known as dislocation.

(Total loss of contact between the two articular surfaces)

When the two articular surfaces are partly in contact and partly out of contact it is known as subluxation. Subluxation is also known as partial dislocation.

(Partial loss of contact between the two aritcular surfaces).

Types of Dislocation (Figs 1.22A to D)

a. Traumatic.
b. Pathological, e.g. pyogenic arthritis
c. Paralytic, e.g. poliomyelitis
d. Congenital, e.g. DDH

In a dislocation, the part which loses contact with the rest of the body is considered as minor segment. The position which this part, i.e. minor segment occupies in relation to the major segment gives the name for the dislocation as anterior, posterior, medial, lateral, central, etc.

When there is a fracture involving the articular surface of a joint along with dislocation or a fracture of the adjoining part of the bone along with dislocation the term fracture dislocation is used to describe the injury (Figs 1.23A and B).

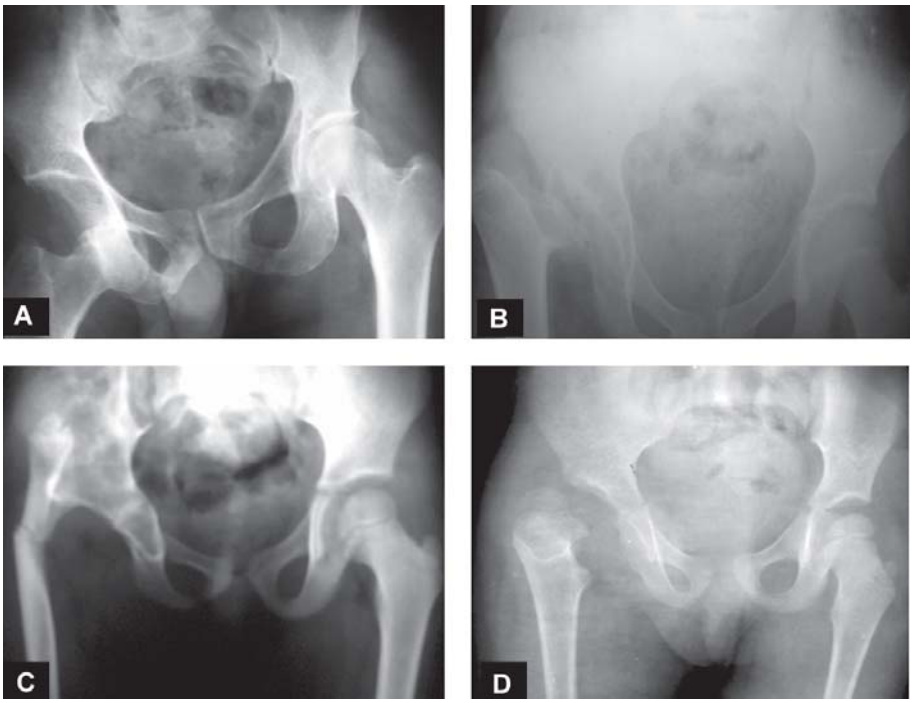
Diagnosis

Clinical diagnosis is simple because of the classical attitude and the signs. Total loss of joint movement is characteristic and any attempted movement results in severe pain.

Treatment

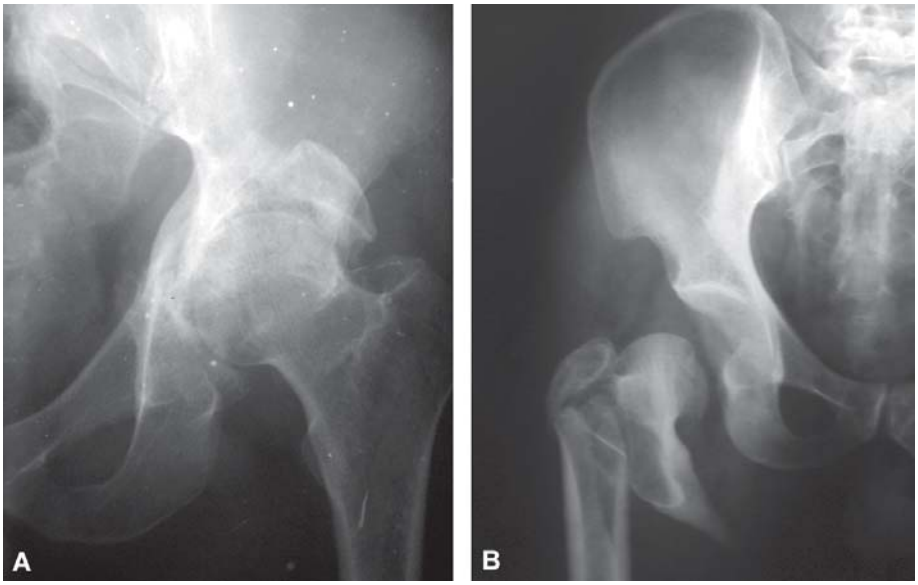
Any dislocated joint has to be reduced under anesthesia as early as possible. Delay poses difficulty in reduction. This is because of re organization of muscles and soft tissue structures around the joint. Irreducibility under anesthesia with adequate muscle relaxation indicates entanglement of the dislocated part in the surrounding tissues, e.g. joint capsule, muscles and tendons and rarely vessels and nerves.

Following successful reduction, immobilization for a minimum period of three weeks is essential. This is necessary for healing of the damaged soft tissues. Poor healing of



Figures 1.22A to D

Radiographs showing different types of dislocations. (A) Traumatic Anterior dislocation of the Hip. (B) Pathological dislocation secondary to Tuberculosis of the Hip joint. (C) Paralytic dislocation secondary to Postpolio Residual Paralysis. Note a recent fracture in a thin femur. (D) Neglected Developmental Dysplasia of the Hip.



Figures 1.23A and B

Radiograph, AP view of the hip joint showing (A) Fracture of the Acetabulum with posterior subluxation of the hip joint. (B) Comminuted Intertrochanteric fracture with posterior dislocation of the hip joint. Both are fracture dislocations.

damaged soft tissues increases the chance of recurrent dislocation and subluxation.

Complications of Dislocation

i. Injury to the neighboring nerves and vessels, e.g. Sciatic nerve injury in posterior dislocation of the hip, Popliteal artery in posterior dislocation of the knee, etc.
ii. Recurrent dislocation.
iii. Habitual dislocation.
iv. Avascular necrosis.

Revision Questions

Q. Define a fracture. How do you classify fractures? Add a note on fracture healing.
Q. Define and classify non unions. Discuss the management.
Q. Enumerate the complications of a fracture. Discuss their management.
Note: For questions on dislocations refer Chapter 4.

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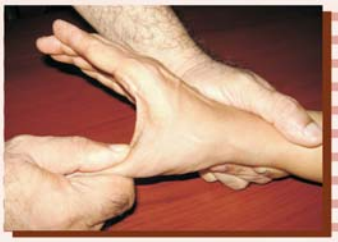


2

Fractures in the Upper Limb

General Information, Diagnosis, Treatment and Complications are dealt with in the following order:

- *Fractures Around the Wrist*
- *Fractures in the Forearm*
- *Fractures Around the Elbow Joint*
- *Fractures in the Arm and the Shoulder*
- *Fractures of the Scapula*

Fractures Around the Wrist

The common fractures that occur around the wrist joint are:

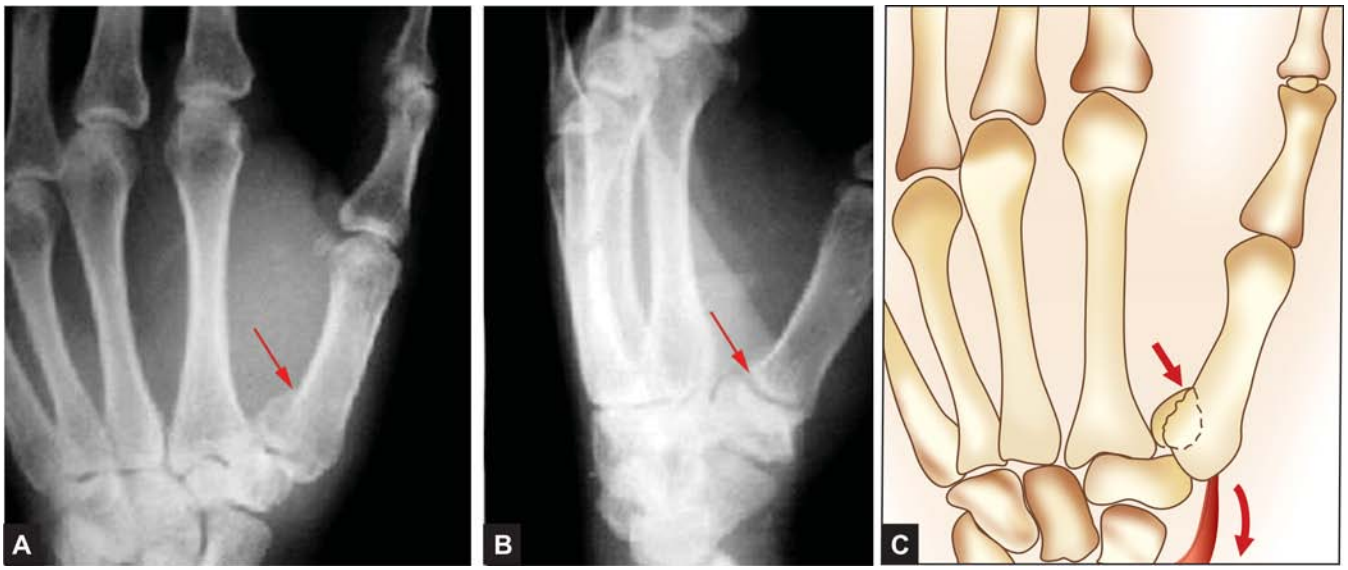
1. Bennett's Fracture.
2. Rolando's Fracture.
3. Scaphoid Fracture.
4. Colles' Fracture.
5. Smith's Fracture.
6. Barton's Fracture.
7. Essex-Lopresti Fracture.
8. Chauffeur's Fracture.

These are Eponyms (except Scaphoid fracture) and are named after the persons who described the fracture.

Bennett's and Rolando's Fracture

General Information

Bennett's and Rolando's fracture are the intra-articular fractures involving the base of the thumb metacarpal. Restoration of articular surface congruity is of utmost importance while treating these fractures. Persistent articular surface incongruity results in painful limitation of movements and later carpometacarpal degenerative arthritis which may require treatment in the form of arthrodesis or arthroplasty for restoration of useful function of the thumb.



Figures 2.1A to C

(A and B) Radiograph of Bennett's fracture showing classical displacements, (C) Line diagram of the same showing the fracture and the displacement occurring due to the pull of Abductor pollicis longus tendon.

Bennett's Fracture

Definition

It is an oblique fracture occurring at the base of the 1st metacarpal bone (thumb metacarpal) with subluxation of the carpometacarpal joint.

Who Described this Fracture?

It was described by Edward Hallaran Bennett, MD, an Irish surgeon in the year 1882 and hence known as Bennett's fracture.

Note: He suggested that early diagnosis and treatment of this fracture is absolutely necessary to prevent complications of highly mobile carpometacarpal joint of the thumb.

Mechanism of Injury

The mechanism of injury is an axial loading of partially flexed thumb metacarpal, e.g. Delivery of a punch with a clenched fist (Figs 2.1A to C).

Displacements

The two fragments are placed as follows.

The ulnar fragment remains in place and the radial fragment is displaced.

The volar ulnar triangular fragment- is held firmly in place by the volar oblique ligament.

The radial distal fragment- is displaced radially, dorsally and proximally by the pull of the abductor pollicis longus tendon.

Treatment

A. *Nonoperative:* Fractures with < 1-2 mm disruption of articular surface and with minimal displacement are treated by closed manipulation, reduction and maintaining the reduction in thumb spica cast for a period of 6-8 weeks.
B. *Operative:* Closed reduction / Open reduction and K-wire fixation and immobilization in thumb spica for a period of 6-8 weeks.

Gradual mobilization is to be followed after removal of spica cast.

Complications

1. Osteoarthritis of carpometacarpal joint (due to joint incongruity and articular surface injury).
2. Stiffness and loss of mobility of carpometacarpal joint of the thumb (result of prolonged immobilization).
3. Loss of reduction and recurrent subluxation and instability.
4. Surgical complications of infection and injury to sensory branch of the radial nerve.

Revision Questions

Q. Define Bennett's fracture.
Q. Who described Bennett's fracture?
Q. What is the mechanism of injury?
Q. What are the displacements?
Q. Describe the management of Bennett's fracture.
Q. What are the Complications of Bennett's fracture?

Essay Question

Q. Define Bennett's fracture. Discus the mechanism of injury, diagnosis and management of Bennett's fracture. Enumerate its complications.

Rolando's Fracture

Definition

It is a three way intra- articular fracture involving the base of the thumb metacarpal in the shape of T or Y with subluxation of carpometacarpal joint.

Who described this fracture?

It was described by Silvio Rolando in the year 1910 and hence known as Rolando's fracture.

Mechanism of Injury

It is a combination of axial loading and abduction injury of the thumb, e.g. a sporting event, impaction of a steering wheel or a handle bar (Figs 2.2A and B).

Treatment

A. Badly comminuted fractures are treated in thumb spica immobilization followed by gradual mobilization.
B. Three way fractures are treated by open reduction and internal fixation using K-wires or mini T plates and screws and immobilization in thumb spica. Later followed by gradual mobilization depending on the stability of fixation.

Prognosis

Prognosis is generally poor. Carpometacarpal degenerative arthrirtis is common which needs arthroplasty or arthrodesis.

Revision Questions

Q. Define Rolando's Fracture.
Q. Why it is known as "Rolando's Fracture"?
Q. What is the mechanism of injury?
Q. How is this fracture treated?
Q. What is the prognosis in Rolando's fracture?

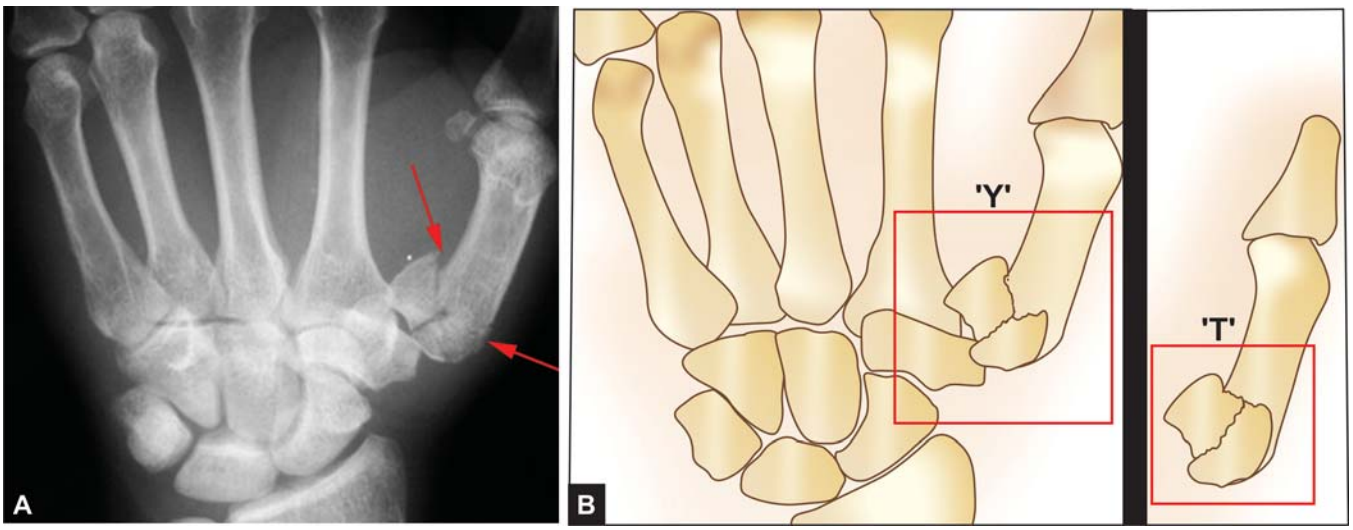
Scaphoid Fracture

General Information

The word Scaphoid is derived from Greek language. "Skaphos" in Greek means boat.

Scaphoid is a boat shaped bone. Scaphoid bone is also known as navicular. The articular cartilage covers 80% of the bone surface. It is placed at 45° to the longitudinal axis of the wrist. It articulates with capitate, trapezium, and trapezoid distally, distal radius proximally and lunate medially.

Fracture Scaphoid accounts for 60% of carpal injuries. Commonly seen in young adults. Rare in children and elderly because of relative weakness of lower radius and a fall on an outstretched hand results in fracture of the lower radius and not the scaphoid.



Figures 2.2A and B

(A) Radiograph showing 'Y' type of Rolando's fracture. (B) Line diagram showing 'Y'and 'T' type of Rolando's fracture.

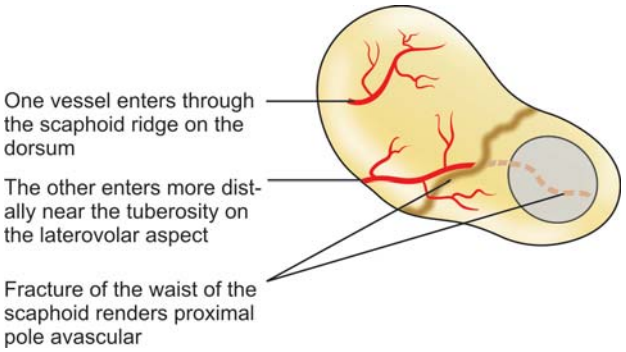


Figure 2.3

The blood supply to the scaphoid (Right : PA view). Note that the vessel enters the bone distally and runs proximally. These are branches arising from superficial branch of radial artery.

Unlike other fractures it does not produce severe symptoms. Hence, there is a chance of delay in diagnosis. This delay when significant can result in a variety of adverse outcomes such as delayed union, nonunion, avascular necrosis, decrease in grip strength, limitation of joint movements and radiocarpal degenerative arthritis. So timely diagnosis and appropriate treatment is absolutely essential to prevent these complications.

Blood Supply of Scaphoid

See Figure 2.3.

Mechanism of Injury (Figs 2.4A and B)

Two mechanisms are described.

1. Radial compression and dorsiflexion occurring at the wrist during a fall on an outstretched hand: generally results in an undisplaced fracture.
2. Hyperextension occurring at the wrist during a fall on an outstretched hand: generally results in displaced fracture scaphoid.

Classification

A. Based on Mayo's Classification (Fig. 2.5):
   1. Fracture of the tuberosity.
   2. Fracture of the distal body.
   3. Fracture of the waist.
   4. Fracture of the proximal pole.
   5. Osteochondral fracture.

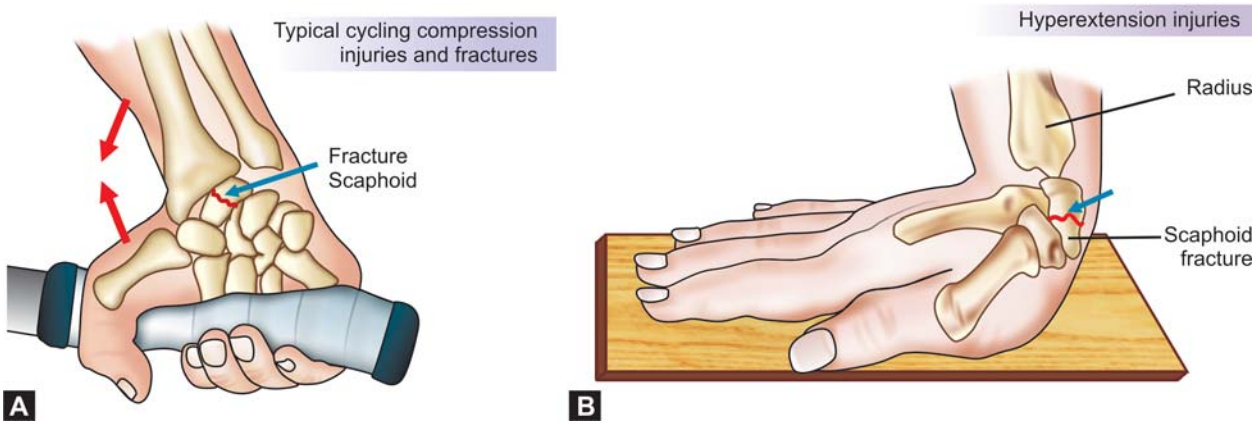
B. Based on Russe classification, the fracture scaphoid is classified as
   i. Stable
   ii. Unstable

All displaced scaphoid fractures irrespective of the nature of the fracture line are considered as unstable scaphoid fractures.

Horizontal oblique and transverse fractures are considered as stable fractures when undisplaced (Figs 2.6A to C).

Clinical signs that help in diagnosing the fracture (Figs 2.7A to D)

1. Tenderness in the anatomical snuff box.
2. Tenderness over the scaphoid tubercle (Freeland 1989).
3. Scaphoid compression test (Chen 1989).
4. Painful limitation of movements of wrist and thumb.



Figures 2.4A and B

Pictures show typical (A) Radial compression and (B) Hyperextension injury.

1. Fracture of the tuberosity.
2. Fracture of the distal body.
3. Fracture of the waist.
4. Fracture of the proximal pole.
5. Osteochondral fracture.

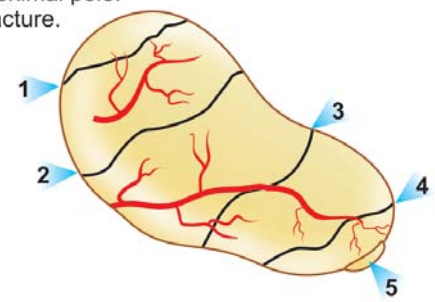


Figure 2.5

Showing different sites of fracture based on Mayo's classification. Osteochondral fracture is difficult to identify radiologically and may need an MRI.

Investigations

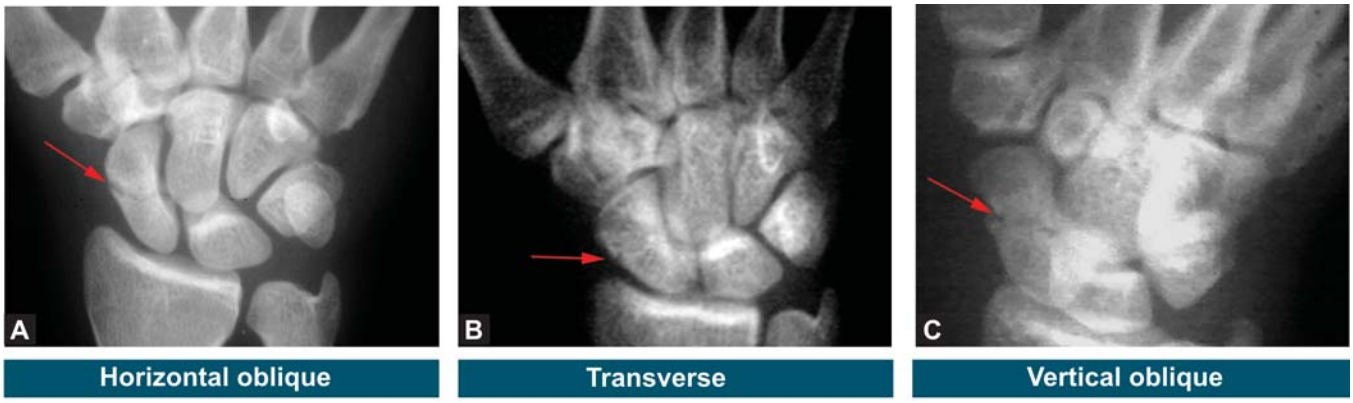
Ninety percent of the fractures are diagnosed by the standard views for the scaphoid. Those which are not seen radiologically are either incomplete or undisplaced fractures and are diagnosed by repeat X-ray after 15 days.

MRI is indicated when in doubt after the repeat X-ray or to assess the extent of avascularity and carpal malalignment as a consequence of nonunion and avascular necrosis.

Radiological views taken to diagnose fracture scaphoid

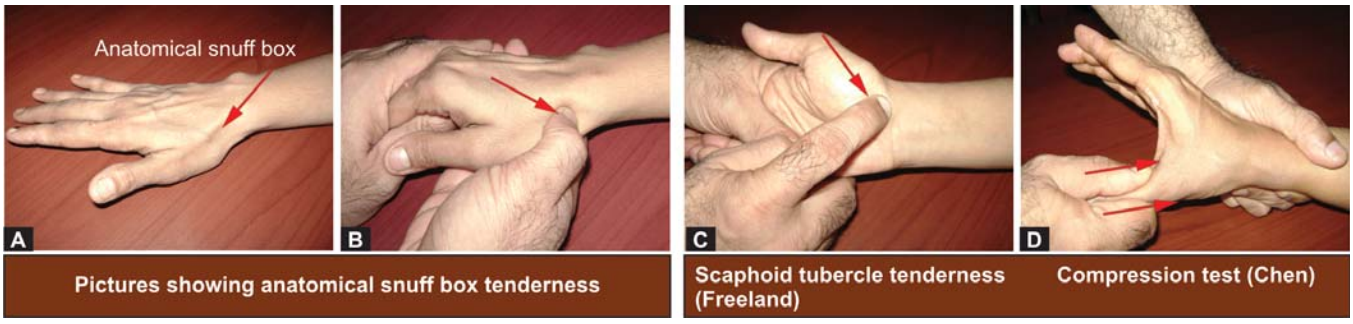
1. Standard PA view of the wrist (with wrist in dorsiflexion and ulnar deviation after making a fist)
2. Standard lateral view.
3. Radioulnar oblique view in midprone position.
4. Stress views only if needed (Stress view opens up the fracture site).

Note: Normally Scaphoid is placed at an angle of 45° volar to the longitudinal axis of the radius. Thus making a



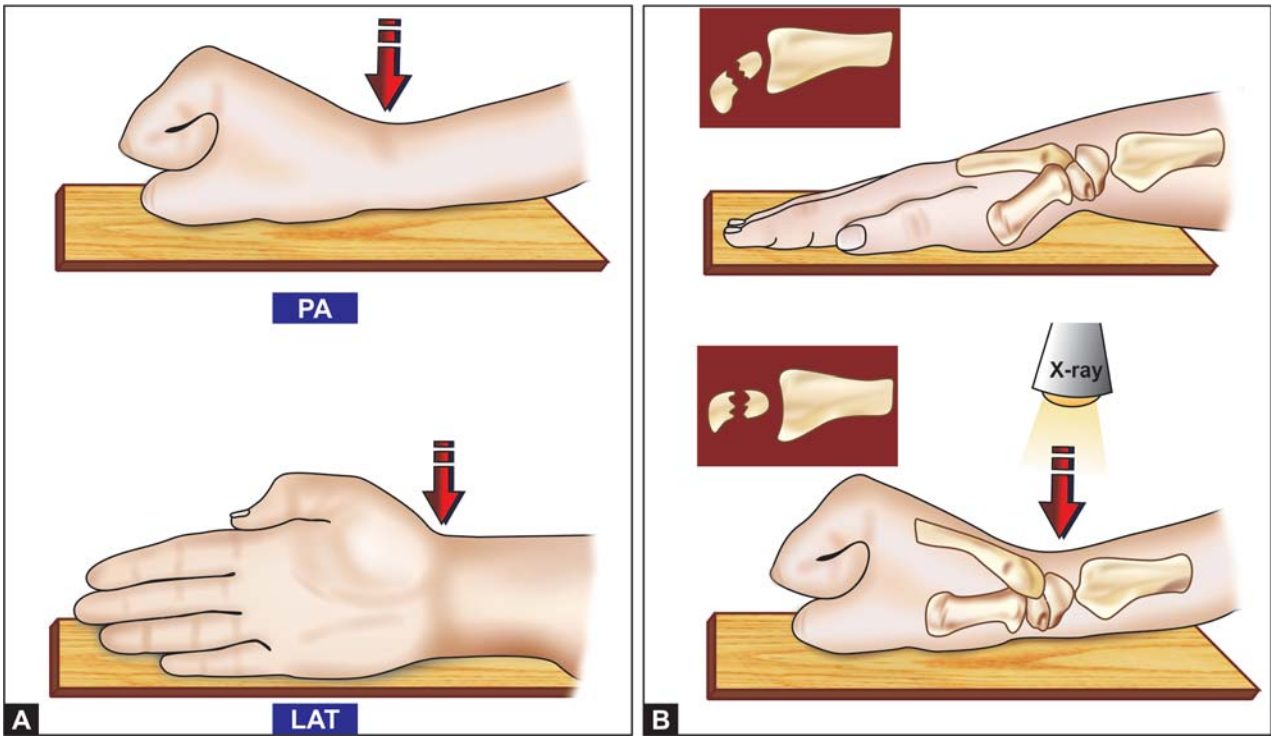
Figures 2.6A to C

Radiographs of scaphoid fracture showing nature of fracture lines. Horizontal oblique and transverse fractures are considered as stable fractures when undisplaced. Whereas vertical fractures whether displaced or undisplaced are unstable.



Figures 2.7A to D

(A and B) Pictures showing anatomical snuff box tenderness, (C) Scaphoid tubercle tenderness (Freeland), (D) Compression test (Chen). Note: Tenderness varies depending on the site and type of the fracture. Hence, it is advisable to make use of all the above signs in combination for a proper diagnosis.



Figures 2.8A and B

(A) PA (Posteroanterior) and Lateral view, (B) Normal volar tilt of the scaphoid is negated by dorsiflexion of the wrist.

Figures 2.9A to C

(A) A thin lucent line, (B) Distinct break in a cortical continuity (C) A step

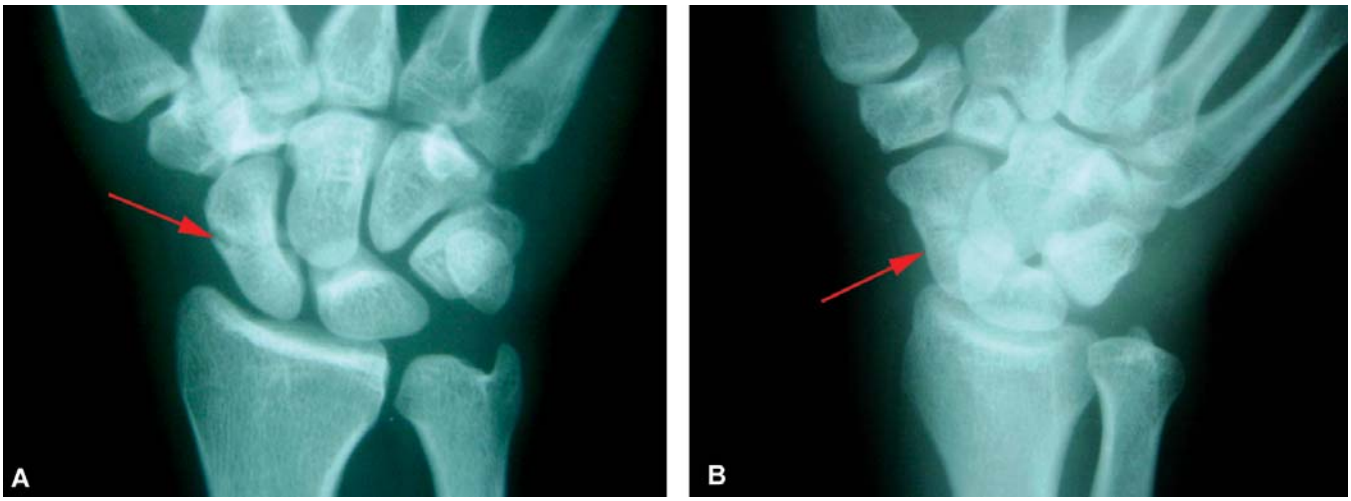
fist in PA view brings it in alignment with the radius and is parallel to the X-ray plate (Figs 2.8A and B).

Interpretation of the radiograph: Scaphoid fracture is generally identified as (Figs 2.9A to C and 2.10A and B)

a. A clear lucent line across the bone.
b. A distinct break in continuity.
c. A distinct sharp step.

Methods of Treating Fracture Scaphoid

A. *Nonoperative:* It is employed in undisplaced and incomplete fractures. By immobilizing in a cast, a special cast known as ***Scaphoid Cast*** with wrist in 15° of dorsiflexion and 5° of radial deviation. The position of immobilization is sometimes described as glass holding position (Some studies show that a standard below elbow cast is sufficient



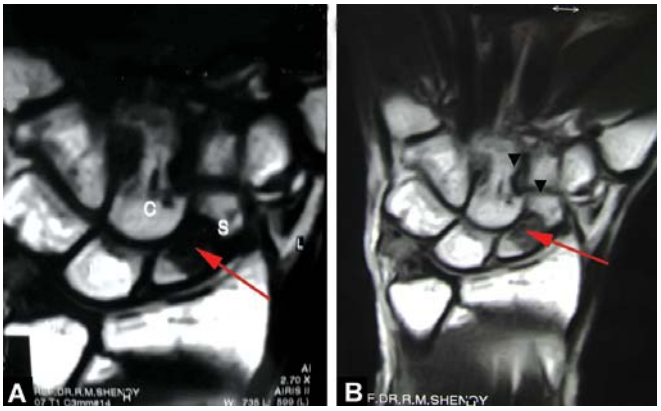
Figures 2.10A and B

(A) Standard PA view. (B) Standard radioulnar oblique view. Fracture of the Scaphoid is seen here as (A) a distinct break in cortical continuity, (B) A sharp radiolucent line.

to immobilize these fractures and scaphoid cast is unnecessary).

B. *Operative:* Is employed in displaced fractures. Closed/Open reduction and internal fixation done using special screws, e.g. Herbert screws, Acutrak, AO scaphoid screws, etc. This helps in early mobilization and functional recovery.

Need for immobilization in 15° of dorsiflexion and 5° radial deviation: This is because of the mechanism of injury which is radial compression and dorsiflexion. Hence, immobilization in a position of dorsiflexion and radial deviation relaxes the fracture site (Avoids tension stress leading to opening up of the fracture site).



Figures 2.11A and B

MRI showing nonunion of fracture Scaphoid and Avascular necrosis. Note the lack of density at the avascular portion of the scaphoid observed in both T1 and T2-weighted images.

Complications of Fracture Scaphoid

1. Nonunion.
2. Avascular necrosis (Figs 2.11A and B).
3. Radiocarpal degenerative arthritis (Fig. 2.12).

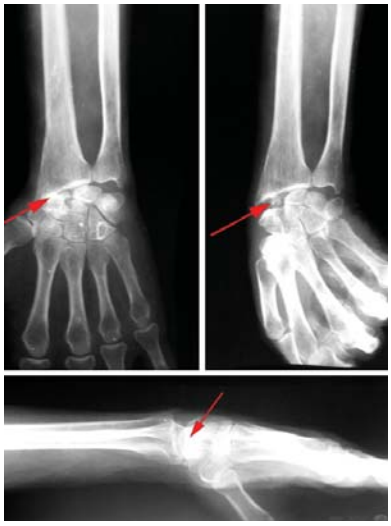


Figure 2.12

Radiographs showing Scaphoid nonunion with Avascular necrosis of proximal pole which has shrunk and almost on the verge of disappearing. This happens because of repetitive stress on the avascular bone over long period of time. Also note the evidence of radiocarpal arthritis which is shown by narrowing of the joint space and osteophyte formation.

Cause for Avascular Necrosis

Avascular necrosis occurs because of peculiarity in blood supply which is from distal to proximal (Fig. 2.3). It depends on the type of fracture and the degree of displacement which is directly proportional to the nature and severity of the injury.

Treatment of the Complications of Fracture Scaphoid

Nonunion with avascular necrosis is treated by means of internal fixation and bone grafting. Several types of bone grafting techniques are described (including vascularized bone graft).

Radiocarpal degenerative arthritis is treated by wrist arthrodesis in 15° of dorsiflexion and 5°-10° of ulnar deviation.

Other procedures

1. Excision of avascular proximal pole.
2. Excision of the whole of scaphoid.
3. Excision of proximal row of carpus.
4. Replacement of the scaphoid.
5. Wrist arthroplasty.

Reason as to why these fractures are commonly missed

1. Mistaken for minor injury, sprain, by the patient because they do not cause severe symptoms.
2. Improper X-rays.
3. Incomplete and undisplaced fractures are difficult to diagnose by X-rays.

Treatment of Suspected Cases of Fracture Scaphoid

When suspected the injury should always be immobilized in scaphoid cast for a period of two weeks. Cast is removed at the end of two weeks and patient is re- examined. If signs are persistent a repeat X- ray / MRI is done for confirmation and treated accordingly.

Revision Questions

Q. What is the other name for Scaphoid?
Q. What are the two mechanisms of injury?
Q. How do you classify fracture scaphoid?
Q. What are the clinical signs that help in diagnosing this fracture?
Q. What are the radiological views taken to diagnose fracture scaphoid?
Q. What are the methods of treating fracture scaphoid?
Q. Why immobilization in 15° of dorsiflexion and 5° radial deviation preferred?
Q. What are the complications of fracture scaphoid?
Q. What is the cause for avascular necrosis?
Q. How do you treat complications of fracture scaphoid?
Q. Why these fractures are often missed?
Q. What is the course of action when fracture scaphoid is suspected clinically but there are no signs radiologically?

Essay Questions

Q. Describe the mechanism of injury of fracture scaphoid. Discuss the diagnosis and management of fracture scaphoid. Enumerate its complications.
Q. Discuss the complications of fracture Scaphoid, their diagnosis and management.

Fall on an Outstretched Hand

Fall on an outstretched hand has a definite mechanism of injury. Many injuries can occur with a fall on an outstretched hand. It depends on the magnitude and direction of the force, the age and quality of the bone as well as the position of the wrist, elbow and shoulder at the time of fall.

In the order from distal to proximal direction they are as follows.

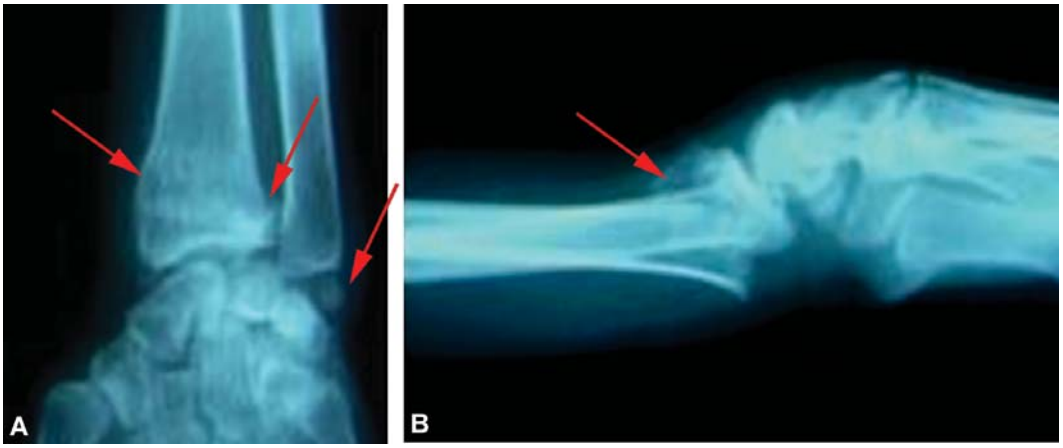
1. Scaphoid fracture.
2. Colles' fracture.
3. Fracture of the radius and ulna.
4. Posterior dislocation of the elbow joint.
5. Supracondylar fracture.
6. Fracture shaft of the humerus.
7. Fracture neck of the humerus.
8. Fracture of the clavicle.

If there are associated forces such as valgus and varus stress, tortional stress, hyperpronation, hyperextension, etc. along with the fall on an outstretched hand other injuries occur (other than already mentioned).

Colles' Fracture

Definition
(Based on original description by Sir Abraham Colles)

It is a fracture occurring approximately within an inch and half of the inferior articular surface of the radius, with or without fracture of the ulnar styloid process, with or without subluxation/dislocation of the inferior radioulnar joint (Figs 2.13 and 2.14).



Figures 2.13A and B

Radiograph (A) PA (postero anterior) and (B) Lateral view showing Colles' fracture. Note the classical site, displacements, dislocation of inferior radioulnar joint, fracture of the ulnar styloid process and the soft tissue shadow showing the typical 'Dinner fork deformity'.

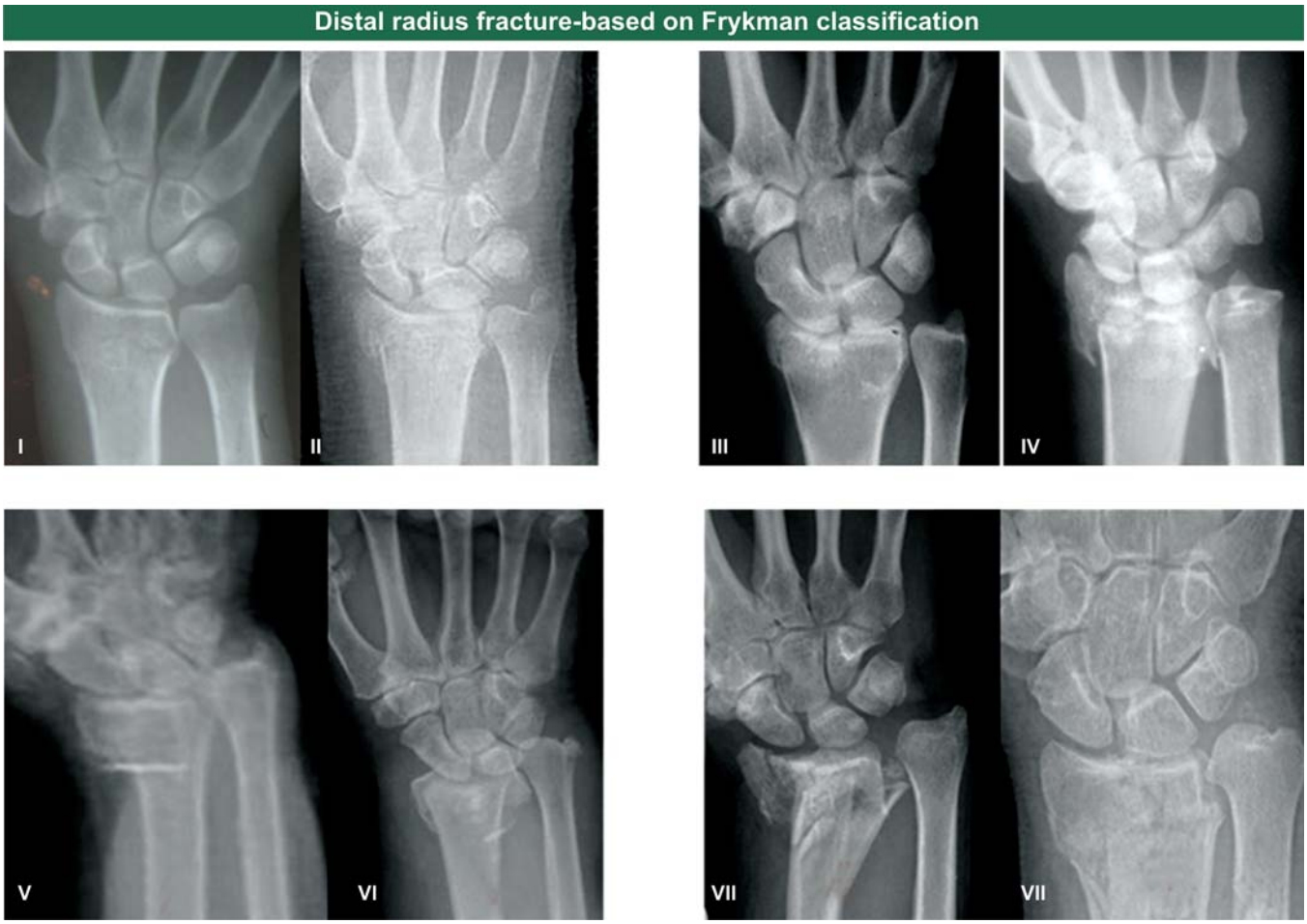


Figure 2.14

Radiographs showing different types of distal radius fracture based on Frykman classification. Type I and II are extra articular fractures. Type III and IV are fractures that involve radiocarpal joint. Type V and VI are fractures that involve radioulnar joint. Type VII and VIII are those involving both radiocarpal and radioulnar joint. Type I, III, V and VII are not associated with ulnar styloid process fracture. Type II,IV,VI and VIII are associated with ulnar styloid process fracture.

Figure 2.15
Colles' fracture is usually due to a slip and fall on the outstretched hands in elderly females.

Who Described Colles' Fracture?

It was described by Sir Abraham Colles' in the year 1814. Hence, it is known as Colles' fracture.

Mechanism of Injury

Mechanism of injury is by means of a fall on an outstretched hand (Fig. 2.15).

Common Age Group

This injury is commonly seen in elderly, especially in women with osteoporosis.

Note: In the young a fall on an outstretched hand causes an Epiphyseal injury and not a Colles' fracture.

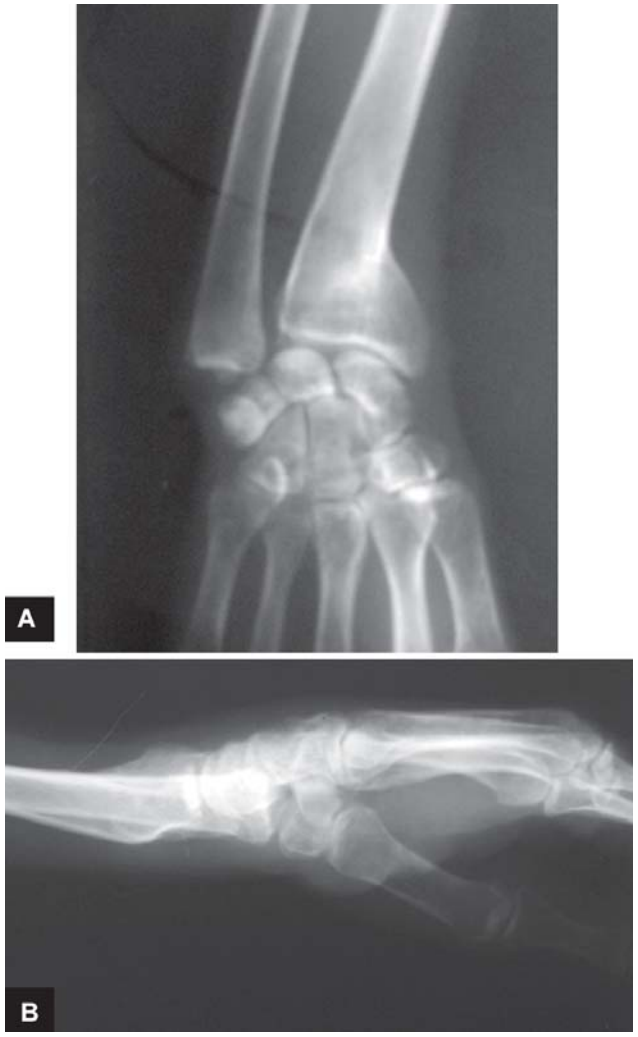
Classical Deformity

The classical deformity is known as 'Dinner Fork' deformity (Figs 2.16A and B).

Displacements Seen in Colles' Fracture

The classical displacements seen in Colles' Fracture are dorsal, lateral, and proximal. In addition this displaced fragment can rotate, angulate or tilt either dorsally (dorsal rotation, angulation or tilt) or laterally (lateral rotation, angulation or tilt). Impaction and Supination are the other displacements seen in some cases (Figs 2.17A and B).

Note: Supination is an extreme form of displacement and is seen in Compound Colles' Fracture, when proximal radius projects out through the wound and distal radius rotates into supination.



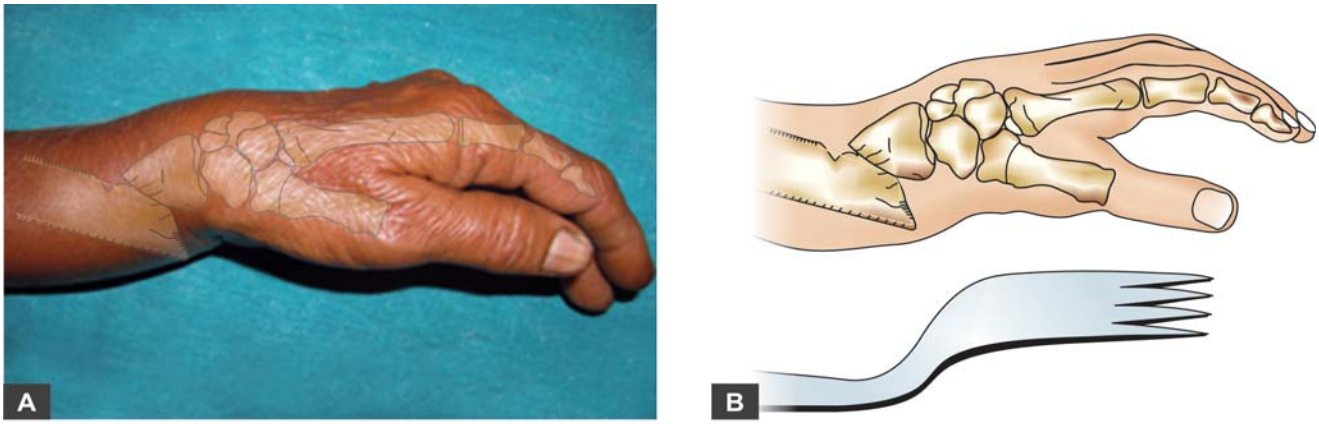
Figures 2.16A and B
Radiograph AP and Lat views showing malunited Colles' fracture. Note the subluxation of the inferior radioulnar joint as seen by the proximal migration of the radial styloid process and the classical 'Dinner fork deformity'.

Treatment

A. *Nonoperative:* It is treated by closed manipulation and reduction under general anesthesia and immobilization in plaster of Paris or fiber below elbow cast moulded in 5° flexion and 5° ulnar deviation (Figs 2.18A and B).

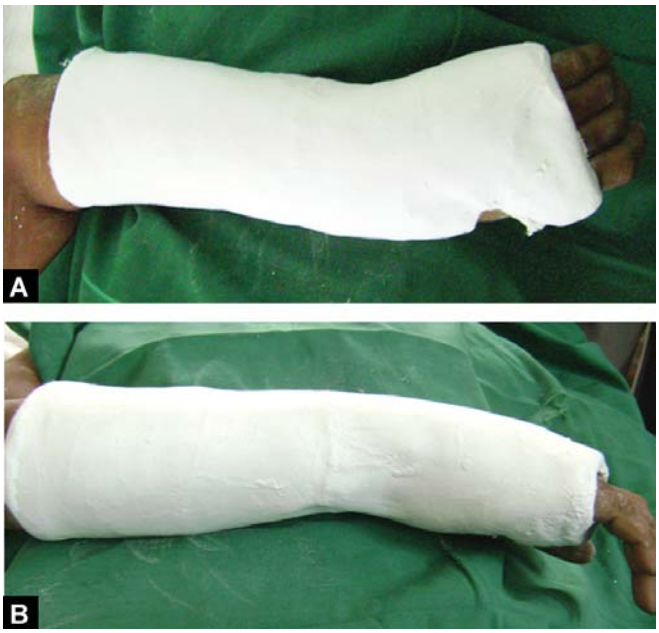
Why below elbow cast is preferred and not above elbow cast?

1. Fracture remains stable once the cast is moulded in 5 degrees of flexion and 5 degrees of ulnar deviation. So there is no need to immobilize the joint above.



Figures 2.17A and B

Clinical picture and line drawing showing the classical 'Dinner fork' deformity of a Colles' fracture. *Normally the radial styloid process is at a lower level than the ulnar styloid process by about 1/2 an inch or 1.25 cm. In a Colles' fracture both are at the same level or the radial styloid is at a higher level than the ulnar styloid process.*



Figures 2.18A and B

Pictures showing the position of moulding of below elbow cast /Colles' cast in 5° flexion and 5° ulnar deviation.

2. To avoid stiffness of the elbow as a consequence of immobilization.

Indication for Above Elbow Cast

Above elbow cast is indicated when comminution is observed and loss of reduction is suspected.

Note: When Sir Abraham Colles' described the fracture in the year 1814 the injury was of low velocity in nature . Therefore, comminuted fractures were unlikely to occur. Further there was no X-ray facility available to know the exact nature of the injury. Hence, these days it is recommended that comminuted articular fractures of the lower radius are not to be considered as Colles' fractures.

B. *Operative:* Surgery is done in the form of closed Kirschner wire fixation under 'C' arm image intensifier control when there is certain degree of comminution and articular surface involvement is seen.

Note: Strictly speaking fractures with articular surface involvement are not to be considered as Colles' Fractures. These are 'Intra-articular fractures of the lower radius'. Mechanism of injury is somewhat different in these fractures. A separate classification system exists for fractures of the lower radius (for clarification refer 'Fernandez and Jupiter' mechanistic classification).

Complications

1. Malunion.
2. Delayed union.
3. Nonunion (rare).
4. Sudeck's atrophy.
5. Shoulder hand syndrome.
6. Carpal tunnel compression of median nerve.
7. Rupture of extensor pollicis longus tendon.

Treatment of Complications

Malunion: Is treated by:

1. *Corrective osteotomy:* By an instrument known as Osteotome the bone is cut at the site of deformity, *the deformity is corrected*, stabilized internally by means of an implant (plates and screws or K-wire) or externally by means of a plaster cast. Thus cosmesis is achieved.
2. *Darrach's procedure (for improving movement):* Six cm skin incision is made over the lower third ulna , 4 cm incision in the periosteum and 2 cm of lower ulna is excised. *The procedure does not correct the deformity but masks the deformity.* A false joint develops which allows full range of pronation and supination.

Delayed Union and Non Union: Managed by means of 'Bone Grafting' with or without internal fixation.

Sudeck's Atrophy and Shoulder Hand Syndrome: Managed by physiotherapy and adequate drugs for 'Sympatholysis'. Prevention of this complication is always better (refer Chapter 1).

Carpal Tunnel Compression of Median Nerve: Carpal Tunnel Decompression is the treatment advocated.

Extensor Pollicis Longus Tendon Rupture: Reconstruction. Using Extensor Indicis as a donor tendon for Tendon transfer.

Revision Questions

Q. Define Colles' fracture.
Q. Why it is called "Colles' fracture"?
Q. What is the mechanism of injury?
Q. In which age group this injury is commonly seen?
Q. What is the injury occurring in the young by a similar fall on an outstretched hand?
Q. What is the classical deformity seen in Colles' fracture?
Q. What are the classical displacements seen in Colles' fracture ?
Q. When do you see Supination?
Q. How do you treat Colles' fracture?
Q. Why is below elbow cast preferred and not above elbow cast?
Q. When do you do surgery in Colles' fracture?
Q. When an above elbow cast is preferred in Colles' fracture?
Q. What are the complications of Colles' fracture?
Q. How will you manage fracture complications of Colles' fracture?

Essay Questions

Q. Define Colles' fracture. Discuss the mechanism of injury, signs and symptoms, diagnosis and management of Colles' fracture.
Q. What are the complications of Colles' fracture? Discuss their management.

Smith's Fracture

Definition

Smith's fracture is defined as transverse fracture of the lower radius within an inch of the articular surface with volar displacement of the fractured fragment (Also known as reverse "Colles'Fracture") (Figs 2.19A to C).

Who Described Smith's Fracture?

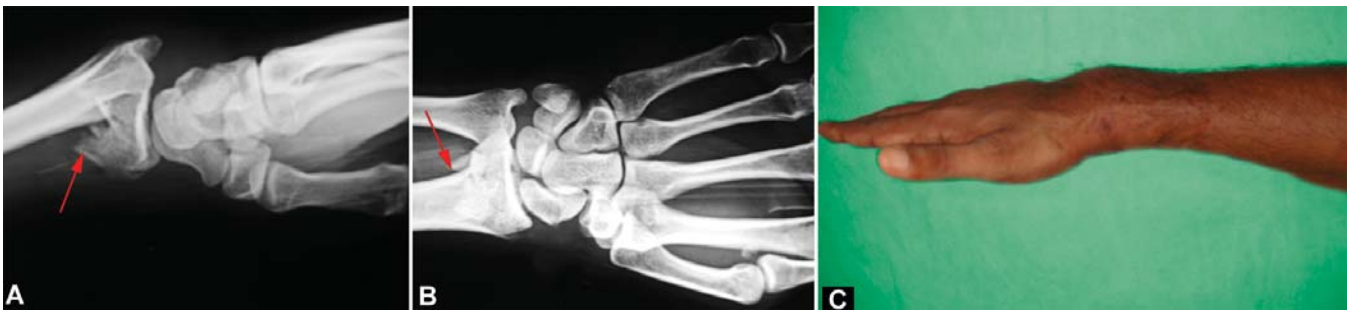
It was described by Robert William Smith (1807-1873) an Irish surgeon in the year1841.

Mechanism of Injury

The mechanism of injury is by means of a fall on the flexed wrist.

Classical Deformity

It is the "Garden Spade" deformity (reverse of "Dinner Fork" deformity).



Figures 2.19A to C

Radiographs showing (A and B) classical volar displacement of the lower radius carrying along with it the carpus, metacarpals and phalanges. This gives rise to a classical 'Garden spade' deformity. Also see (C) the clinical photograph of the deformity.

Treatment

A. *Operative:* This is an unstable fracture. Hence treated by open reduction and internal fixation using a specific type of plate known as "Buttress Plate" (Ellis Buttress Plate).
B. *Nonoperative:* Employed only in minimally displaced fractures. Following manipulation and reduction, an above elbow cast is given for immobilization which is continued for a period of 6 weeks with a periodic check for loss of reduction. If loss of reduction is observed an open reduction and internal fixation is done (at earliest, within 2 weeks).

Complications

1. Malunion.
2. Subluxation of inferior radioulnar joint.
3. Sudeck's atrophy.

Treatment of Complications

The complications are treated in a similar manner as described in Colles' fracture.

Revision Questions

Q. What is the definition of Smith's fracture?
Q. Who described "Smith's fracture"?
Q. What is the mechanism of injury?
Q. What is the classical deformity seen?
Q. How do you treat "Smith's fracture"?
Q. Is there a role for closed manipulation, reduction and a plaster of Paris or fiber cast immobilization?
Q. What are the common complications?

Essay Question

Q. Define Smith's fracture. Discuss the mechanism of injury, clinical signs, diagnosis and management of Smith's fracture.

Barton's Fracture

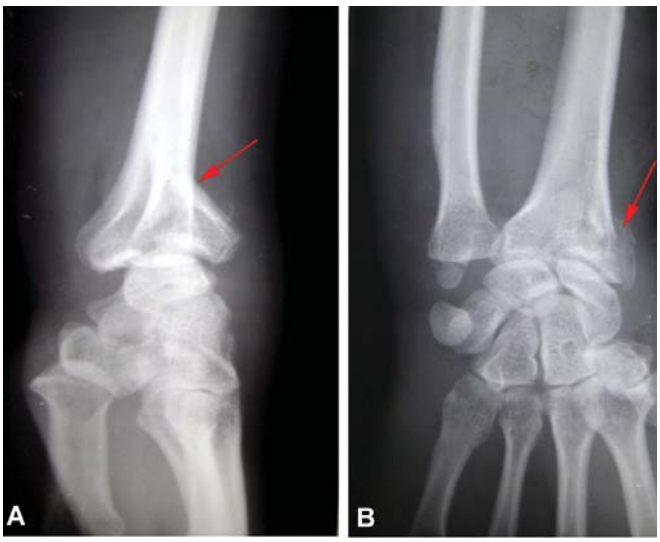
Definition

Barton's fracture is defined as an articular marginal fracture involving the dorsal 1/3 or volar 1/3 articular surface of the lower radius with subluxation or dislocation of the carpus.

Who Described "Barton's Fracture"?

It was described by John Rhea Barton an American surgeon in the year 1814.

Note: Original article describes only dorsal Barton's fracture. Volar Barton's fracture was recognized later.



Figures 2.20A and B
Radiographs PA and Lat views showing a dorsal Barton's fracture.

Mechanism of Injury

Dorsal Barton's fracture: The mechanism of injury is an extreme dorsiflexion occurring at the wrist accompanied by a pronating force resulting in shear and dorsal articular marginal fracture (Figs 2.20A and B).

Volar Barton's fracture: The mechanism of injury is an extreme palmar flexion occurring at the wrist accompanied by supinating force resulting in shear and volar articular marginal fracture (Figs 2.21A to D).

Note: Clinical diagnosis is difficult. It can be easily mistaken for Colles' fracture (Dorsal Barton's) or Smith's fracture (Volar Barton's). X- ray is confirmatory.

Treatment

A. *Operative:* This fracture is treated by open reduction and internal fixation using special plate known as Buttress plate (Ellis Buttress plate, Jupiter plate).
B. *Nonoperative:* No role for nonoperative method except in very selected cases of incomplete fracture. Complete fractures invariably get displaced sooner or later because of the pull of carpal ligament.

Complications

Following are the complications seen:
1. Malunion with angulation and radial shortening.
2. DRUJ subluxation (Distal radioulnar joint subluxation).
3. Sudeck's atrophy.

Figures 2.21A to D

Radiographs showing volar Barton's fracture pre- and postoperative. Note classical displacements. A T-buttress plate has been used to fix the fracture.

4. Rupture of Extensor Pollicis Longus tendon (Dorsal Barton's).
5. Radiocarpal degenerative arthritis.

Revision Questions

Q. Can Barton's fracture be clinically diagnosed?
Q. What is the mechanism of injury for a volar Barton's fracture?
Q. What is the mechanism of injury for a dorsal Barton's fracture?
Q. Why it is called "Barton's fracture"?
Q. Define Barton's fracture.
Q. How will you treat Barton's fracture?
Q. Is there any role for nonoperative method with casts?

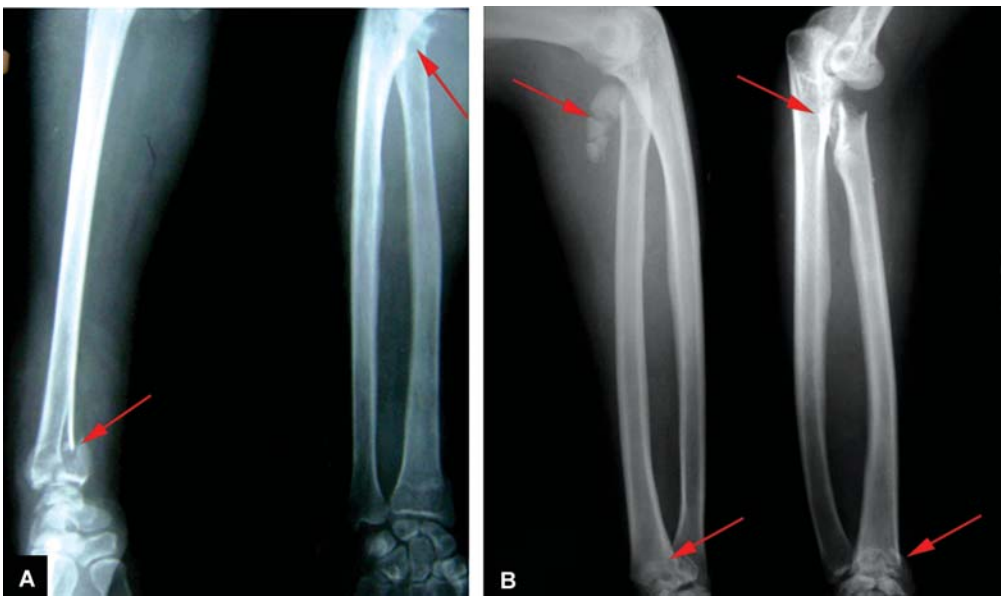
Essay Question

Q. Define Barton's fracture. Discuss the diagnosis and management of Barton's fracture. Enumerate its complications.

Essex-Lopresti Fracture

Definition

It is a comminuted fracture of the radial head with dislocation of the inferior radioulnar joint due to longitudinal compression force (DRUJ subluxation). Described by Essex- Lopresti in 1951 (Figs 2.22A and B).



Figures 2.22A and B

Radiographs showing Essex-Lopresti injury as a result of severe form of axial loading resulting in inferior radioulnar subluxation, comminuted fracture of the head of the radius as well as comminuted articular fracture of the lower radius.

Mechanism of Injury

Mechanism of injury is by means of a fall from a height on an acutely dorsiflexed wrist resulting in axial loading of the radius. This disrupts the interosseous membrane.

Clinical Importance

1. Always suspect this injury when a radial head fracture is seen and examine the inferior radioulnar joint both clinically and radiologically.
2. Never excise the head in these injuries immediately without stabilization of the inferior radioulnar joint as it will aggravate the subluxation of the inferior radioulnar joint.

Treatment

1 Inferior radioulnar joint reduction is achieved primarily by supinating the forearm and if needed stabilized with K- wire (Interosseous membrane takes about 6 weeks for healing).
2. Fractured head of the radius is managed next by ORIF or excision depending on the nature of injury.

Essay Question

Q. Define Essex–Lopresti fracture. Discuss the mechanism of injury and management.

Chauffeur's Fracture

It is a fracture of the radial styloid process, which used to occur among Chauffeur's in olden days, when the crank used to start the car kicked back and hit the thenar side of the wrist thereby causing the fracture. Shearing force by the carpus causes this fracture (Fig. 2.23).

Figure 2.23
Picture showing the mechanism of injury of a Chauffeur's fracture.

Treatment

This fracture is usually unstable because of the pull of the brachioradialis and strong radiocarpal ligament. Hence, internally fixed using "Kirschner Wire".

Note: "Kirschner Wire" also known as K-wire in short is a thin stainless steel wire with a sharp-pointed tip designed by Martin Kirschner 1909.

Fractures in the Forearm

General Information

A fracture of the single bone of the forearm rarely occurs without subluxation or dislocation of a joint above or joint below. So it is a must to examine the superior and inferior radioulnar joint in a patient with a forearm fracture and take X-rays including elbow and the wrist in accordance with the findings.

Fracture of both the bones, that is fracture of radius and ulna more commonly occurs than a single bone fracture.

The common fractures occurring in the forearm are
1. Fracture of the radius and the ulna.
2. Monteggia Fracture.
3. Galeazzi Fracture.

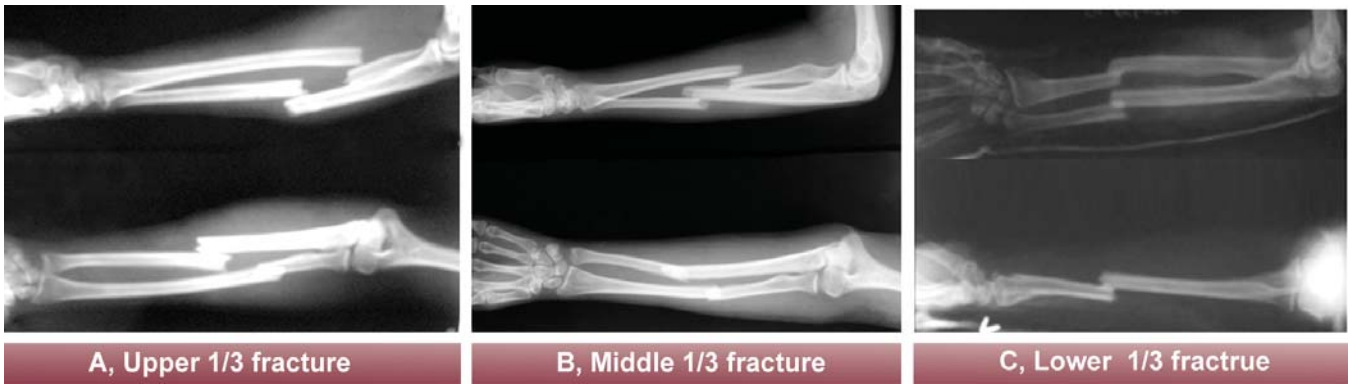
Fracture of the Radius and the Ulna

Mechanism of Injury

Is by means of a
1. Direct blow onto the forearm.
2. Fall on an outstretched hand with forearm pronated.

Deforming Forces (Figs 2.24A to C)

a. Proximal 1/3 fractures (Above the insertion of pronator teres). The proximal fragment is flexed and supinated because of the unopposed action of biceps brachii and supinator and distal fragment is pronated because of the action of pronator teres and pronator quadratus.
b. Middle 1/3 fractures and lower 1/3 fractures (below the insertion of pronator teres). The proximal fragment is in midprone position because the action of supinator and pronator teres balance and neutralize each other. The distal



Figures 2.24A to C

Radiograph showing classical deformity at different levels.

fragment is in pronation because of the action of pronator quadratus.

An understanding of the deforming forces is important in planning the treatment of these fractures. It dictates the alignment of distal fragment in relation to proximal fragment. In proximal 1/3 fractures the distal fragment is aligned in supination. In middle 1/3 fracture and lower 1/3 fractures, the distal fragment is aligned in midprone position.

Treatment

These fractures are highly unstable. Except in children and in undisplaced fractures (in adults with a check on displacement during closed treatment) there is no role for closed reduction and plaster cast immobilization. Open reduction and internal fixation using plates and screws or intramedullary nails in selected cases is the treatment of choice. *Special attention must be paid to maintain the radial bow and the interosseous space so as to get back the movement of pronation and supination (Fig. 2.25).*

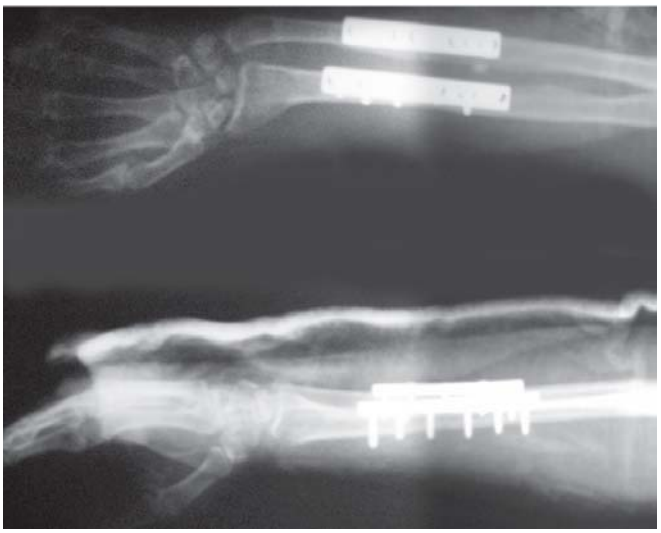


Figure 2.25

Radiograph showing internal fixation of fracture of both the bones of the forearm using DCP. Note the presence of old malunited Colles' fracture with subluxation of the distal radio-ulnar joint.

Complications

1. Malunion
2. Nonunion
3. Cross union (synostosis) because of single hematoma formation.

Revision Questions

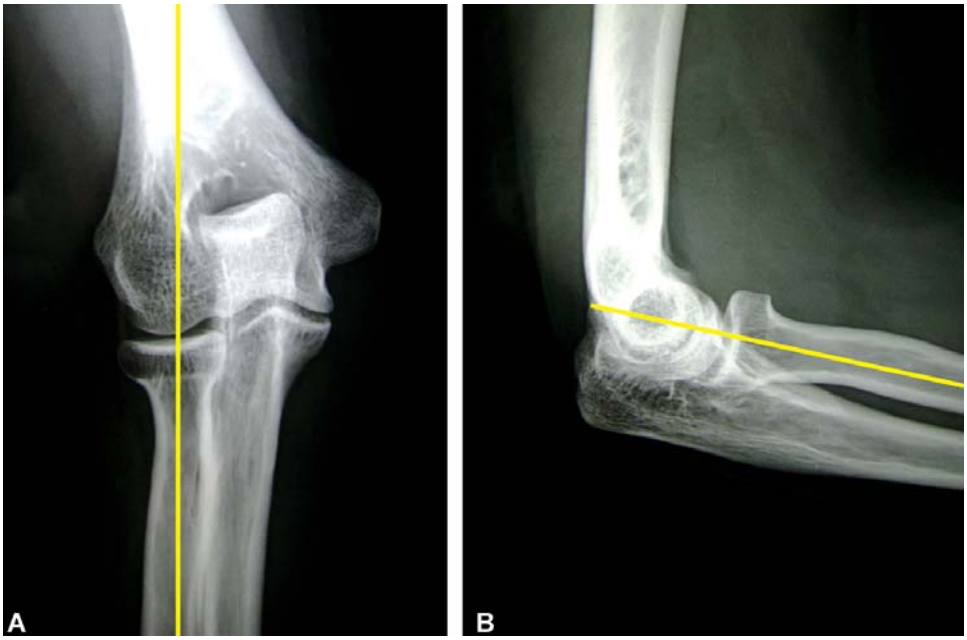
Q. What is the mechanism of injury for fracture of both bones of the forearm?
Q. What are the deforming forces responsible for displacement and what is their importance?
Q. What is the treatment of this fracture?
Q What are the complications of the fracture of both the bones of the forearm?

Monteggia Fracture

Monteggia fracture was described first by an Italian surgeon Giovanni Batista Monteggia in Milan in the year 1814, i.e in the pre Roentgen era without the help of X-rays.

Definition

Monteggia fracture is defined as the fracture of the proximal ulna with subluxation or dislocation of the superior radioulnar joint. Not only the superior radioulnar joint gets dislocated but also the radiohumeral component of the elbow joint, i.e the radio capitular articulation also gets dislocated.



Figures 2.26A and B

The maintained relation of superior radioulnar and radiocapitular joint as proved by a line drawn along the long axis of the radius which is continued through the elbow.

Note: Because of this Monteggia fracture is considered to be more unstable than Galleazzi fracture.

Diagnosis of superior radioulnar joint dislocation/subluxation: It is diagnosed by the help of a line drawn along the long axis of the radius and continued through the elbow joint. It should cut the lateral condyle in any view. If it does not, it indicates subluxation or dislocation of superior radioulnar (Figs 2.26A and B).

Mechanism of Injury (Fig. 2.27)

By means of a:

1. Fall on an out stretched hand with forced pronation.
2. Direct forceful blow to the forearm.

Classification

It is classified by the help of "Bado's" classification as follows.

Type I Fracture of the upper or middle third of the ulna with anterior displacement of the head of the radius (Figs 2.29A and B).

Type II Fracture of the upper or middle third of the ulna with posterior displacement of the head of the radius.

Type III Fracture of the ulnar metaphysis with lateral displacement of the head of the radius.

Type IV Fracture of upper or middle third of radius and ulna with anterior displacement of the head of the radius.

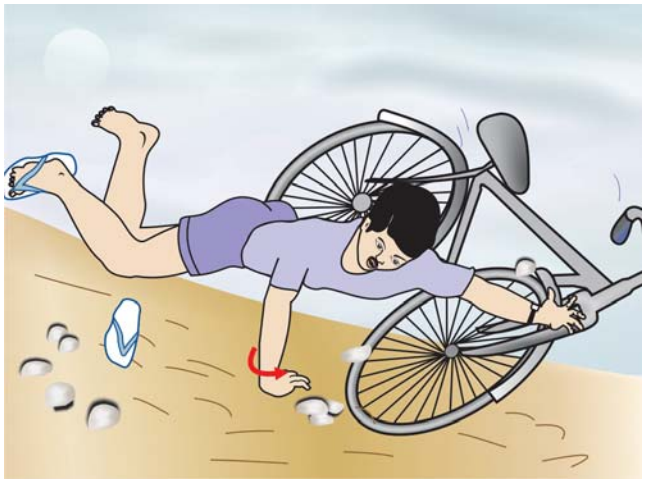


Figure 2.27

Diagrammatic representation of a hyperpronation injury which is responsible for Moteggia fracture.

Hume's Fracture

It is nothing but a *high Monteggia fracture* commonly occurring in children and one of the Monteggia variants.

Hume's fracture is a high Monteggia fracture (Fig. 2.28). On the left (A) it is a recent injury. On the right (B) old negelected injury with malunion and with superior radioulnar joint dislocation. It needs osteotomy of the malunion of the ulna, reduction of the superior radioulnar joint dislocation and stabilization along with internal fixation of the osteotomy.

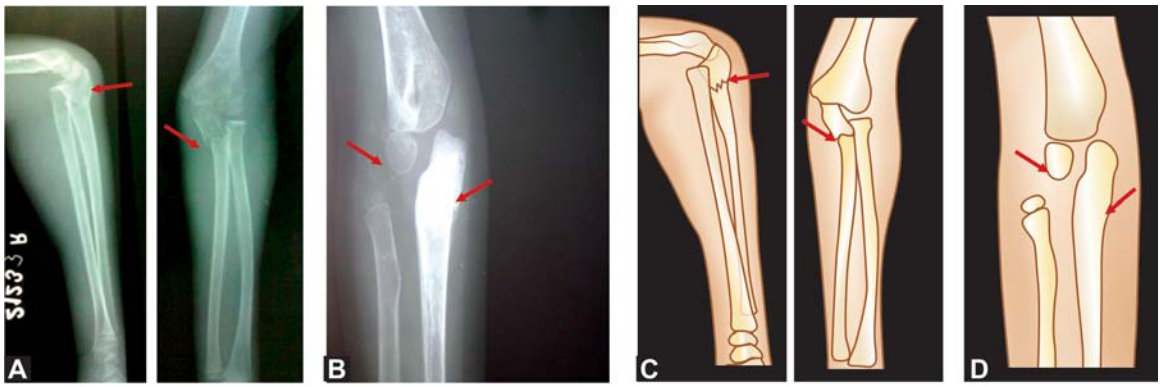
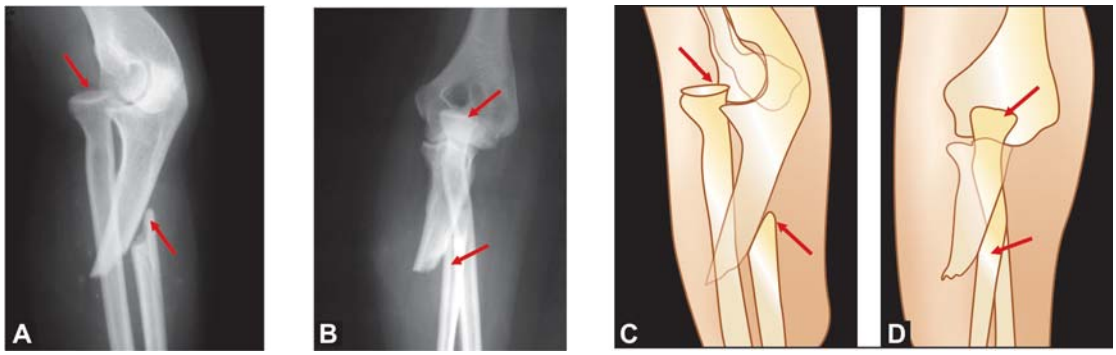


Figure 2.28

Radiograph showing (A and B) Hume's fracture (pediatric). On the right hand side (C and D) diagrammatic representation of the same injury.



Figures 2.29A and B

Radiograph showing an extension type of Monteggia fracture in an adult and the diagrammatic representation (C and D) of the same. Note that the direction of angulation of the ulna shows the direction of the displacement of the head of the radius. In this case the radius displaced anteriorly and medially (Bado's Type I)

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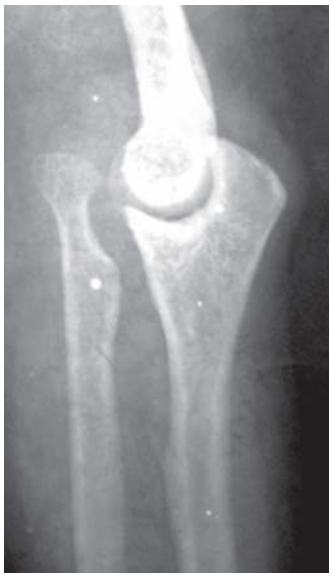


Figure 2.30

Radiograph showing an old unreduced Hume's fracture dislocation. Note the persistent dislocation of the superior radioulnar joint, atrophy of the head of the radius and the malunion at the upper 1/4th of the ulna.The patient has adapted so well for the changed structure at the elbow and but for cosmesis does not complain of any other problem.

Treatment

Operative: The fracture is invariably treated by open reduction and internal fixation. The fracture of the ulna is always fixed in a stable manner. Due attention is given to the stable anatomical reduction of superior radioulnar joint. If needed a ligament repair/reconstruction is done primarily (Fig. 2.29).

Complications

1. Malunion (Fig. 2.30).
2. Nonunion.
3. Redislocation.
4. Radioulnar synostosis.
5. Chronic pain.
6. Nerve injuries.

Revision Questions

Q. Who described Monteggia Fracture?

Q. What is Monteggia fracture?
Q. In a Monteggia fracture, which other joint gets dislocated along with superior radioulnar joint dislocation ?
Q. How will you diagnose superior radioulnar joint subluxation or dislocation radiologically?
Q. What is the mechanism of injury of a Monteggia fracture?
Q. How do you classify Monteggia fracture?
Q. What is "Hume's fracture" ?
Q. What is the treatment of Monteggia fracture ?
Q. What are the complications of Monteggia fracture?

Essay Question

Q. Define Monteggia fracture. Discuss the mechanisms of injury, diagnosis and treatment of Monteggia fracture. Enumerate the complications.

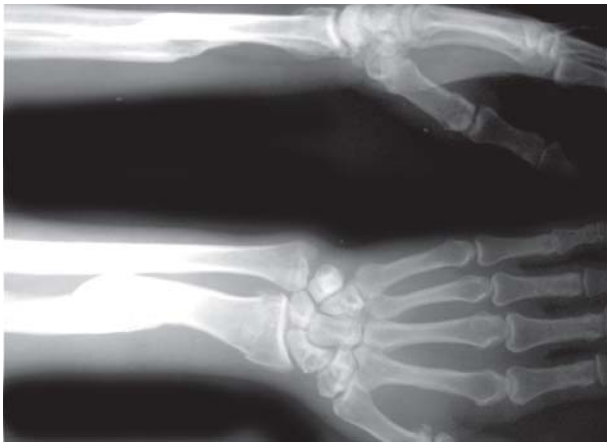
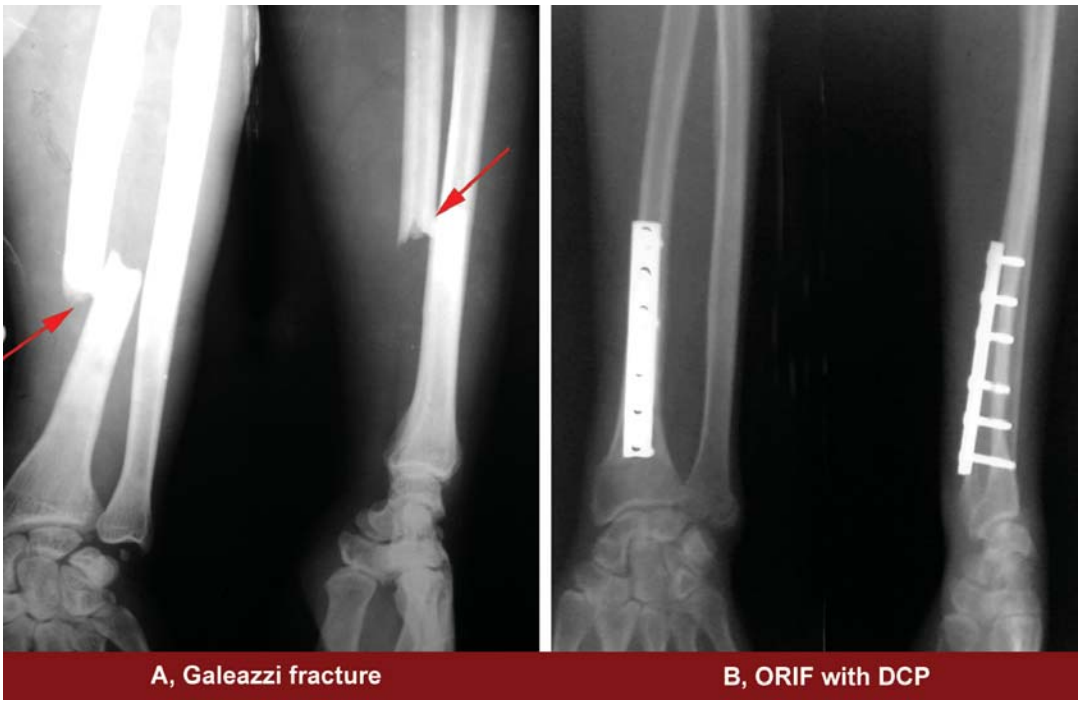


Figure 2.31

Radiograph showing malunited Galeazzi fracture with distal radioulnar joint (DRUJ) subluxation with radial deviation of the carpus. For the very same reason William Campbell called it as a fracture of necessity and advocated surgery.

Galeazzi Fracture

The Galeazzi injury pattern was first described by Sir Astley Cooper in the year 1842 exactly 92 years before Ricardo Galeazzi(1934) an Italian Surgeon at the Instituto de Rachitici Milan described the results of the treatment in 18 cases. In 1941, William Campbell called this *as a fracture of necessity* because surgery is necessary for the treatment of this fracture and also named it as Galeazzi fracture This fracture is also called as reverse Monteggia fracture (Fig. 2.31).



Figures 2.32A and B

Radiographs of Galeazzi Fracture pre and postoperatively. Note the anatomic reduction of the fracture which in turn reduces the inferior radioulnar joint subluxation automatically.

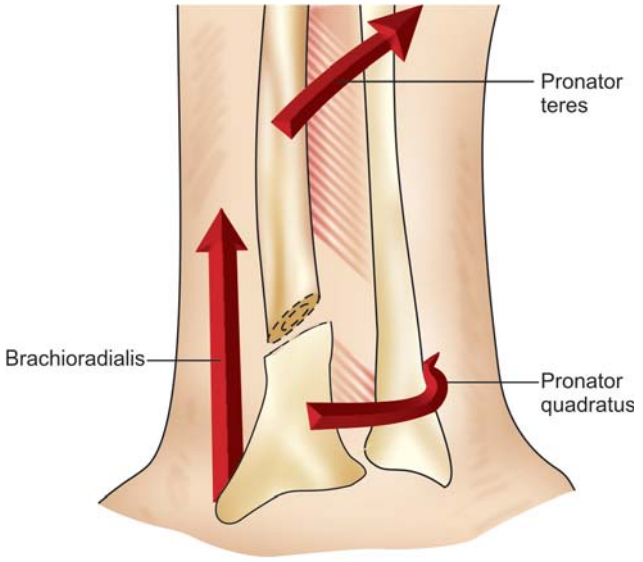


Figure 2.33
Picture showing the muscular forces responsible for the displacement in a Galeazzi fracture.

Definition

It is a fracture of the lower third or lower fourth of radius with subluxation or dislocation of inferior radioulnar joint (DRUJ-distal radioulnar joint) (Figs 2.32A and B).

Note: This fracture can occur anywhere in the radial shaft at a site between the bicipital tuberosity and approximately 5 cms from the distal articular surface of the radius. More proximal the fracture, lesser is the DRUJ instability. More distal the fracture, greater is the DRUJ instability.

Mechanism of Injury

The mechanism of injury is an axial loading of radius in a hyperpronated forearm (Fig. 2.33).

Treatment

In children: Closed manipulation, reduction and immobilisation in POP cast is the treatment.

Because the periosteum is thick and bones are elastic, the reduction is maintained.

In adults: Open reduction and internal fixation using plates and screws, e.g. LCDCP, DCP. Distal radioulnar joint (DRUJ) has to be reduced always during the procedure of open reduction and internal fixation by closed methods and rarely by open methods.

Complications

1. Malunion.
2. Nonunion.
3. DRUJ instability.

Revision Questions

Q. What is Galeazzi fracture?
Q. What is the mechanism of injury of a Galeazzi fracture?
Q. What is the treatment of a Galeazzi fracture?
Q. What are the complications?

Essay Question

Q. Define Galeazzi fracture. Discuss the mechanism of injury, diagnosis and treatment of Galeazzi fracture. Enumerate the complications.

Fractures Around the Elbow Joint

General Information

Elbow is a hinge joint with a radiohumeral and radioulnar articular component. Before skeletal maturity, trauma causes epiphyseal injuries and after skeletal maturity, ossified components get fractured and derive their names accordingly.

Among the medial epicondylar and lateral epicondylar fractures, it is the medial epicondyle which is commonly fractured because medial epicondyle fuses with the shaft as a single epiphysis, separately at skeletal maturity, whereas other components that is trochlea, capitulum and lateral epicondyle form a single centre at puberty and fuse with the shaft collectively. For the same reason lateral condylar fractures are common and not the fractures of the medial condyle. A fracture which occurs above the condyles is known as supracondylar fracture. A fracture which splits the condyles into two is known as intercondylar fracture.

The common fractures around the elbow joint are:

1. Radial head fracture.
2. Olecrenon fracture.
3. Capitulum and trochlear fracture.
4. Medial epicondyle fracture.
5. Lateral epicondyle fracture.
6. Medial condyle fracture.
7. Lateral condyle fracture.
8. Supracondylar fracture.
9. Intercondylar fracture.

Radial Head Fracture

Classification (Figs 2.34 to 2.36)

Based on Mason's classification classified as:

Type I Undisplaced.

Type II Displaced, single fragment fracture less than 1/3rd of the articular surface.

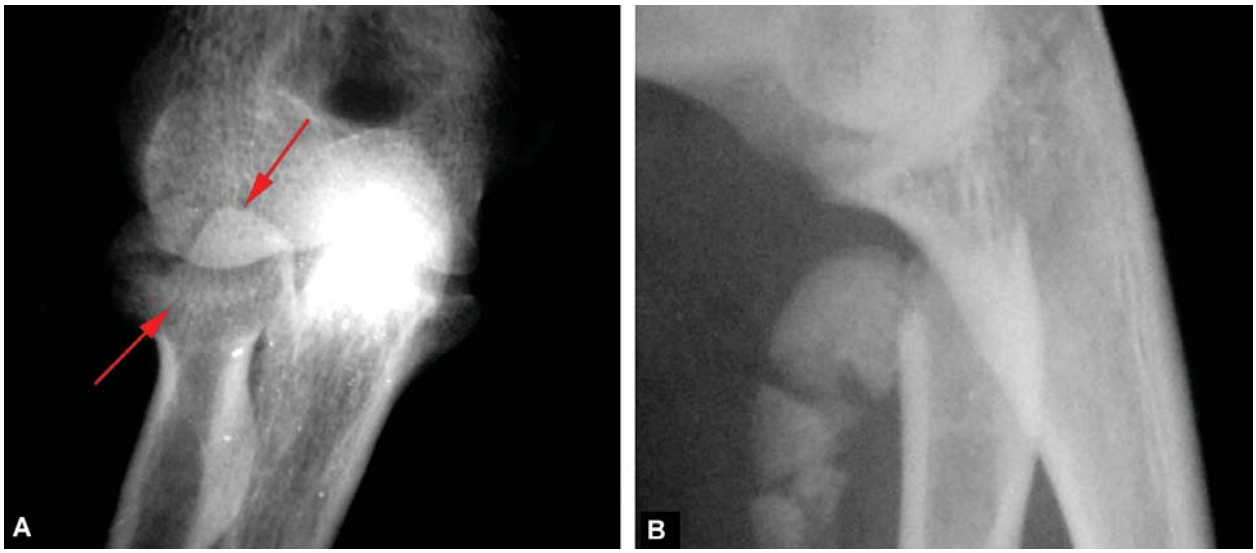
Type III Displaced, involving more than 1/3rd of the articular surface or comminuted fracture.

Mechanism of injury

Is by a fall on an outstretched arm with elbow in extension. The compression stress is transmitted along the axis of the radius and the radial head hits the capitulum of the humerus thereby causing the fracture.

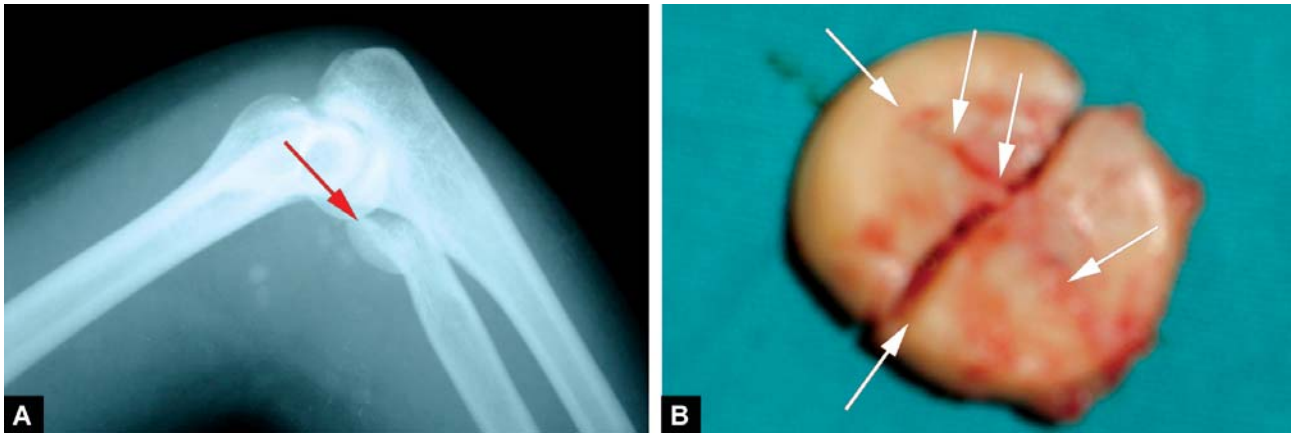
Treatment

Undisplaced and incomplete fractures are managed by immobilization in a plaster cast or a slab for a period of three weeks followed by removal and mobilization.



Figures 2.34A and B

Radiograph showing Type II and Type III fracture of the head of the radius based on Mason's classification.



Figures 2.35A and B

Radiograph of a three weeks old fracture Mason's type III involving the head of the radius. Patient underwent excision of the head of the radius. Note the two major fragments with split in the fragments. Also note the edges of the fracture which have become rounded.

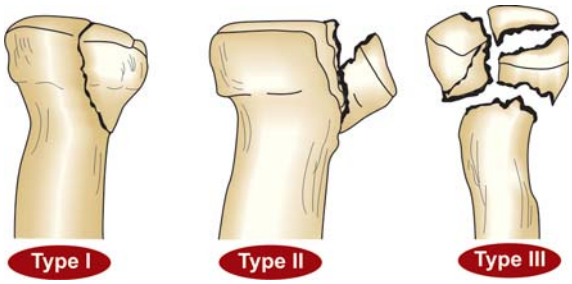


Figure 2.36
Classification of radial head fractures as classified by Mason.

Comminuted, displaced fractures are managed by excision of the head of the radius. Displaced noncomminuted fractures are managed by open reduction and internal fixation depending on the size of the fragment or by excision of the head of the radius.

Rule of Three: Less than 1/3 involvement, Less than 30° angulation, Less than 3 mm displacement is to be treated non-operatively.

Complications

1. Avascular necrosis of the head of the radius.
2. Malunion with restriction of pronation and supination.
3. Painful limitation of pronation and supination.

Revision Questions

Q. How do you classify radial head fracture?
Q. What is the mechanism of injury of fracture head of the radius?
Q. How is this fracture managed?
Q. Name the complications of Radial head fracture.

Olecrenon Fracture

General Information

Olecrenon fractures are seen in a diverse group of injuries ranging from a simple undisplaced fracture to a complex fracture dislocation of elbow joint.

Mechanism of Injury

Three mechanisms are identified

1. Avulsion fracture. That occurs when triceps contracts against the resistance of a fall on a semiflexed elbow and supinated forearm (Fig. 2.37).
2. Direct impact onto the olecrenon by a fall on the point of the elbow or by a direct blow.
3. A hyperextension injury.

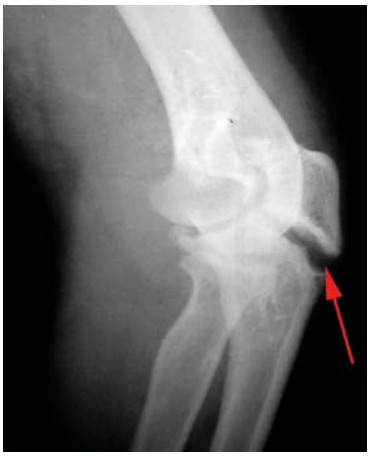


Figure 2.37
Radiograph showing an oblique fracture of the olecrenon process of the ulna.

Classification

Based on Schatzker classification:

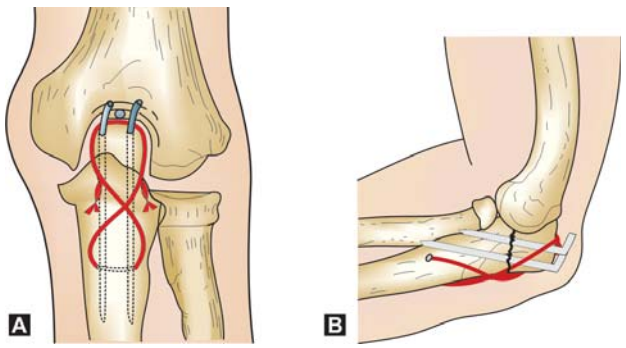
Type A	Simple transverse fracture.
Type B	Transverse impacted fracture.
Type C	Oblique fracture.
Type D	Comminuted fracture.
Type E	More distal fracture, extra-articular.
Type F	Fracture dislocation.

Treatment

Operative:

Noncomminuted and transverse fractures (Figs 2.38A and B)

Treatment of choice is open reduction and internal fixation using the technique of tension bandwiring, which converts the distraction force of triceps pull into a compression force and promotes healing.



Figures 2.38A and B
Diagrammatic representation of Tension band wiring of fracture olecrenon.

Alternately long AO cancellous bone screw can also be used.

Comminuted fractures:
Fixation using plates and screws, e.g. Hookplate.

Revision Questions

Q. What are the mechanisms of injury of Olecrenon fracture?
Q. Classify Olecrenon fractures.
Q. What is the treatment of Olecrenon fracture ?

Capitulum and Trochlear Fractures

These are rare fractures involving the articular surface of the lower humerus. Hence need open reduction and internal fixation to restore articular surface congruity. Mini screws, Herbert screws and sometimes Kirschner wires are used to fix these fractures.

Medial and Lateral Epicondylar Fractures

These are avulsion fractures which occur due to the pull of common flexor and extensor muscle origin respectively. They have to be fixed firmly and immobilized in a cast or a slab till union occurs. In adults sometimes the medial epicondylar fragment is excised and ulnar nerve is transposed anteriorly. In children medial epicondylar fracture is considered as a type of epiphysial injury and is always fixed anatomically (Fig. 2.39).

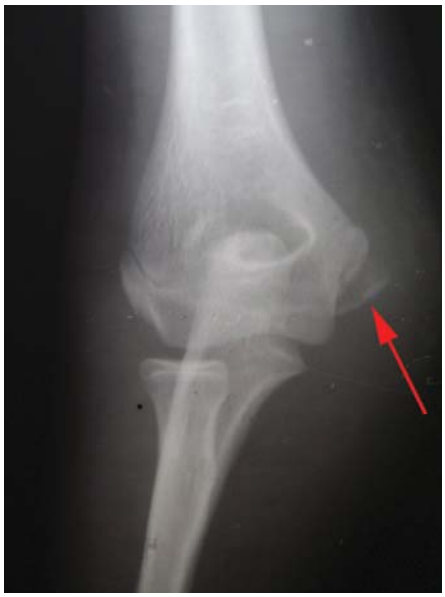


Figure 2.39
Radiograph showing medial epicondylar fracture. It is an avulsion fracture occurring because of the pull of common flexor origin.

Medial and Lateral Condylar Fractures

General Information

Lateral half of trochlea with capitulum and lateral epicondyle is known as *lateral condyle*.

Medial half of trochlea with medial epicondyle is known as *medial condyle*.

Stimson in 1883 was the first to describe the lateral condyle fracture in his book *Treatise on fractures.* Milch in 1955 recognized the significance of these fracture patterns to elbow stability and classified them.

Mechanism of Injury

The lateral and medial condylar fractures are avulsion fractures (pull off by common extensor and common flexor origin respectively) occurring as result of a severe varus or a valgus stress respectively.

The other mechanism for lateral condyle fracture is by means of a compressive impact of the radial head onto the lateral condyle (push off theory) as a result of a fall on an extended hand.

The other mechanism for a medial condyle fracture is by means of a direct blow onto the posteromedial aspect of the epicondyle by a fall on the point of the elbow.

Since they are commonly seen in children, involve the articular surface of the lower humerus and are considered as epiphysial injuries they need open/closed reduction and internal fixation in order to restore articular surface congruity and prevent growth disturbance.

Classifications (Figs 2.40A to C)

Lateral Condyle fracture: Based on Milch anatomical classification (1964)

Type I — Extends through the ossification center, exits at the radiocapitular groove with intact lateral crista of trochlea (Salter and Harris Type IV Epiphyseal Injury). The elbow is relatively stable.

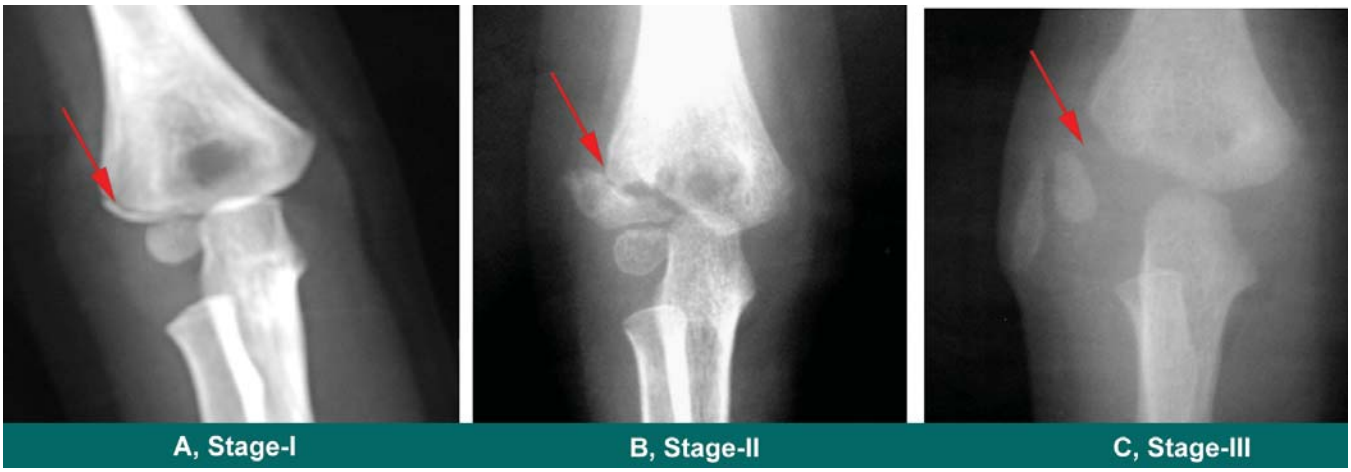
Type II — Extends across the physis, exits through the trochlea fracturing the lateral crista of trochlea (Commonly Salter and Harris Type II. Can sometimes be Type IV Epiphyseal Injury). Results in unstable elbow.

Based on Jakob's classification (1975) (Figs 2.40A to C)

Stage I — Nondisplaced with intact articular surface.

Stage II — Moderate rotational displacement with fractured articular surface.

Stage III — Complete displacement with rotation and elbow instability.



Figures 2.40A to C

Radiographs showing three stages of lateral condylar fracture according to Jakob's classification.

Medial Condyle Fracture
Milch anatomical classification (1964)

Type I	Splits the trochlear groove, lateral trochlear ridge intact.
Type II	Spilts the capitotrochlear sulcus; trochlear ridge is part of the fracture.

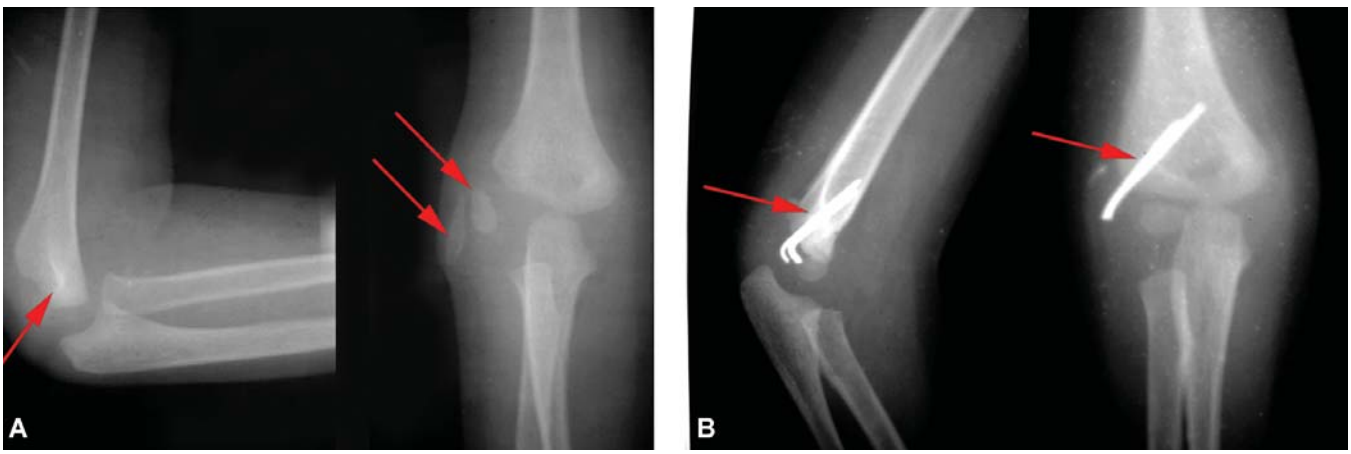
Treatment

Operative: Both medial and lateral condyle fractures are treated by open reduction and internal fixation using K-wires, plates and screws, etc. as per indication (Figs 2.41A and B).

Complications of Lateral Condyle Fracture

1. Nonunion.
2. Malunion with deformity.
3. Limitation of movement of the elbow.
4. Tardy Ulnar Nerve Palsy.
   "Tardy Ulnar Nerve Palsy" means, delayed ulnar nerve paralysis. Nonunion of the lateral condyle causes a progressive valgus deformity at the elbow. This stretches the ulnar nerve gradually, resulting in a progressive ulnar nerve palsy.

Treatment: Treatment for "Tardy Ulnar Nerve Palsy" is anterior transposition of the ulnar nerve. At the same time lateral condyle is fixed in the best possible manner.



Figures 2.41A and B

Radiograph showing (A) grossly displaced Lateral condylar fracture. Note that the fractured surface is rotated and is facing outwards. Accurate reduction is necessary to restore function. (B) After open reduction and internal fixation with the help of two 'Kirschner wires'.

Revision Questions

Q. What is a Lateral and a Medial Condyle ?
Q. In which age group these fractures are commonly seen ?
Q. Who described Lateral Condyle fracture first ?
Q. What are the mechanisms of injury of a Lateral Condyle fracture ?
Q. What are the mechanisms of injury for Medial Condyle fracture ?
Q. What is the treatment for Lateral and Medial Condyle fracture ?
Q. Name the complications of Lateral Condyle fracture.
Q. What is "Tardy Ulnar Nerve Palsy" ?
Q. What is the treatment for "Tardy Ulnar Nerve Palsy" ?

Supracondylar Fracture of the Humerus

This fracture occurs just above the two condyles of the lower humerus. Hence, derives the name supracondylar fracture. The fracture is commonly seen in children between the age of 5-10 yrs.

Two types are described (Figs 2.42A to C).

1. Extension type.
2. Flexion type.

It is the extension type which is commonly seen. Flexion type is not very common.

Mechanism of Injury

a. *Extension type:* By means of a fall on an out stretched hand wherein the elbow is in extension or minimal flexion. The force is transmitted through the supracondylar portion of the humerus (Fig. 2.43).
b. *Flexion type:* By means of a fall on the point of the fully flexed elbow.

Displacements: Displacements seen are as follows

Extension type: Posterior, medial/ lateral and rotation.
Flexion type: Anterior, medial/ lateral and rotation.

Nature of the fracture line and its importance

- In extension type, it is directed obliquely upwards and backwards.
- In flexion type, it is directed obliquely downwards and forwards. The nature of the fracture line is of importance in the treatment of supracondylar fracture.
- An extension type of supracondylar fracture is stable in flexion and hence after reduction, it is immobilized in flexion. It is unstable in extended position of the elbow.
- A flexion type of supracondylar fracture is stable in extension and hence after reduction, it is immobilized in extension. It is unstable in flexed position of the elbow.

The Role of Triceps and Brachialis Muscle

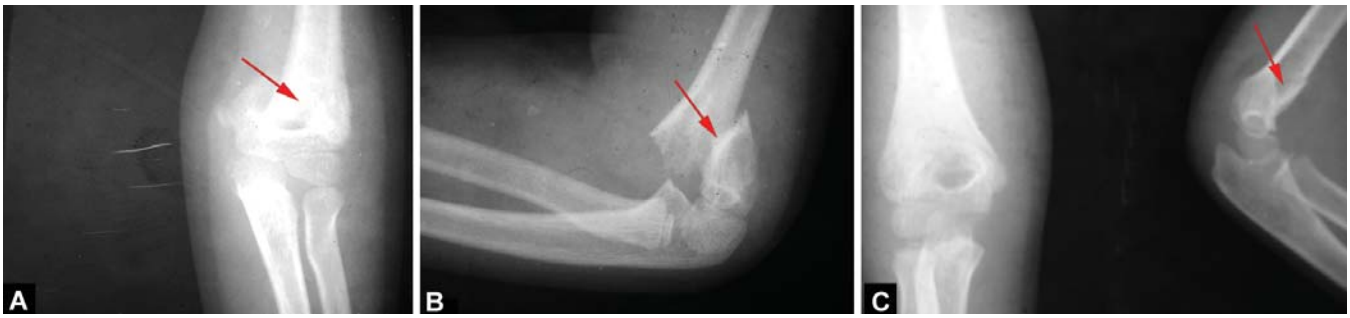
These muscles play a role in maintaining the reduction of this fracture.

The triceps muscle acts as an internal splint and helps in maintaining the reduction in extension type of supracondylar fracture when the elbow is immobilized in flexion. The brachialis muscle acts as an internal splint and helps in maintaining the reduction in flexion type of supracondylar fracture when the elbow is immobilized in extension.

Classification

Based on **Gartland** classification, supracondylar fractures is classified as follows:

Type I	Undispaced.
Type II	Displaced with intact posterior cortex.
Type III	Displaced with no cortical contact.



Figures 2.42A to C

(A and B) Radiographs showing extension type of supracondylar fracture. (C) Radiograph showing flexion type of supracondylar fracture. *Note the site of the fracture and direction of the fracture line. The site of the fracture is just above the condyles. The direction of the fracture line is opposite to each other. In* ***extension*** *type it is* ***obliquely upwards and backwards****. In* ***flexion*** *type it is* ***obliquely upwards and forwards.***

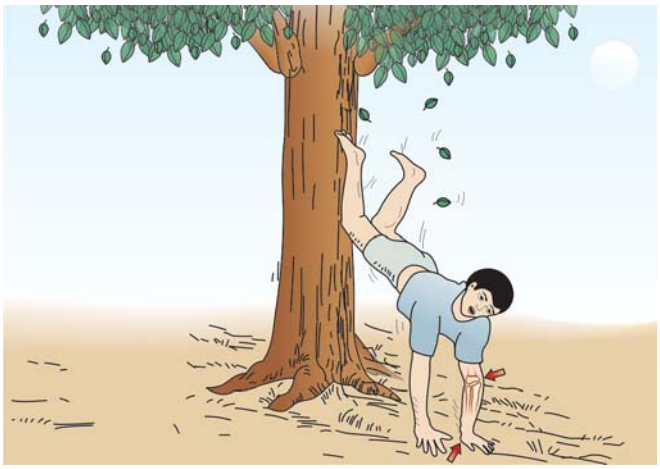


Figure 2.43
The mechanism of injury of an extension type of supracondylar fracture.

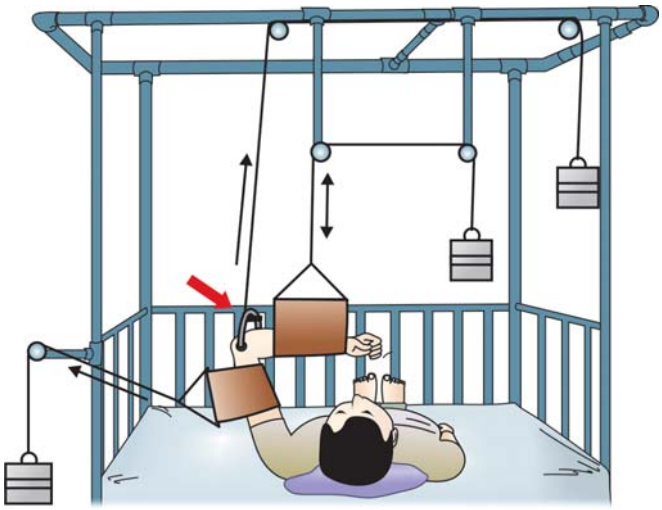


Figure 2.44
Showing diagrammatic representation of treatment of supracondylar fracture by traction with help of a Balcon beam.

Treatment

A. *Nonoperative:*
   i. Closed manipulation and reduction and maintenance of reduction in a plaster splint.
   ii. Skeletal traction: A K-wire is passed through the olecrenon process and an overhead traction is applied. It is indicated when there is gross swelling at the elbow and manipulation and surgical methods of fixation are contraindicated due to poor skin condition or ensuing vascular insufficiency (Generally employed when the patient comes late after undergoing oil massage) (Fig. 2.44).

B. *Operative:* Closed / Open reduction and internal fixation using 'Kirschner Wires'.

Note: A 'K-Wire' fixation is done for a badly displaced and unstable closed supracondylar fracture as well as for an open and complicated supracondylar fracture with vascular

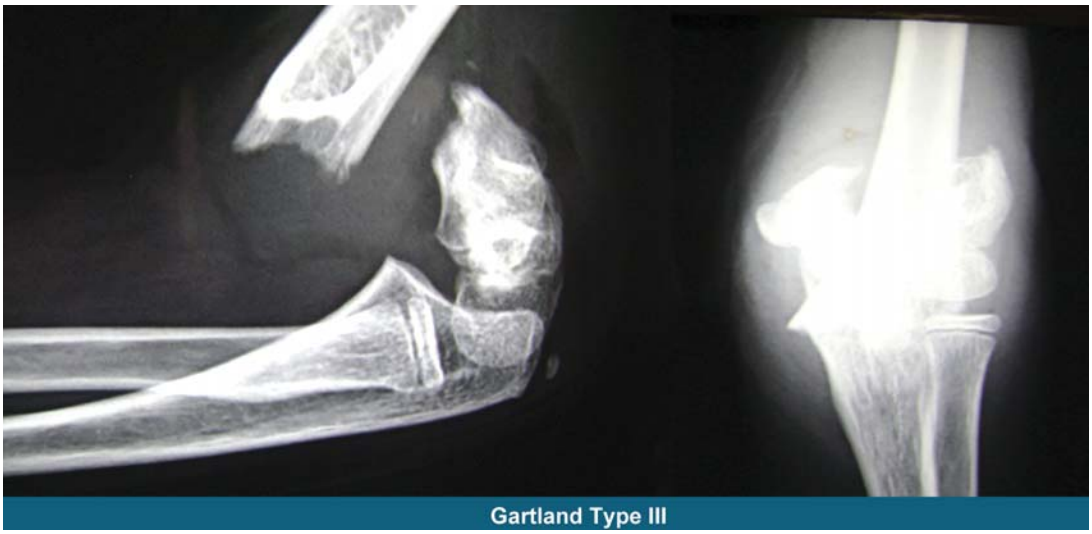


Figure 2.45
Radiographs AP and Lat view showing badly displaced extension type of supracondylar fracture of the humerus. The chances of having neurovascular complication is very high.

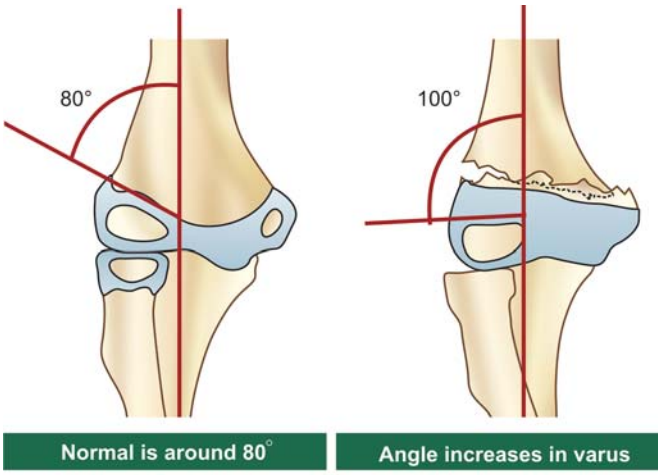


Figure 2.46
The Baumann's angle normal and abnormal.

deficit which needs both wound care and vascular repair (Fig. 2.45).

In closed fractures this fixation can be done by a closed method using image intensifier or by minimally opening the fracture site medially and laterally. It is not advisable to split the triceps muscle for exposing the fracture.

Baumann's Angle (Fig. 2.46)

In children, it is difficult to assess the accuracy of reduction of varus. The Baumann's angle helps to determine the accuracy. It is an angle formed by a line along the long axis of the humerus and the line along the coronal axis of the capitular physis. Normal angle is around 80°. If the angle increases it indicates the varus position of the fragment.

Complications

The complications seen in supracondylar fractures are classified as

a. Immediate
b. Late

Immediate

- Injury to brachial artery resulting in "Volkmann's ischemia".
- Injury to median, ulnar and radial nerves.

Late

- Volkmann's ischemic contracture.
- Malunion. Results in cubitus varus and "Gun Stock Deformity".
- Myositis ossificans.
- Limitation of movements.

Complications of Supracondylar Fracture and their Management

1. ***Volkmann's Ischemia***

It is a compartmental syndrome occurring in the compartment of the extremities due to sudden loss of blood supply. It was first described by Dr Richard von Volkmann (1830-1889), a German doctor in a paper on "noninfective ischemic conditions of various fascial compartments in the extremities".

If the ischemia is left untreated leads to classical contracture known as Volkmann's ischemic contracture.

This ischemic phenomenon passes of in three stages or phases.

The three stages or phases of "Volkmann's ischemic phenomenon are:

i. Stage of impending ischemia (also known as stage of threatened ishemic contracture) lasts from 24-48 hrs.
ii. Stage of established ischemia, lasts from 48 hrs—3 weeks.
iii. Stage of established ischemic contracture, seen after 3 weeks.

The treatment of these three stages is different. Hence it is important to identify the stages of "Volkmann's ischemic phenomenon" by examining the patient and identifying the signs.

Signs of impending ischemia: The five cardinal signs of impending ischemia are the "Five P's".

i. Pain.
ii. Pallor.
iii. Pulselessness.
iv. Parasthesia.
v. Paralysis.

(There is a 6th 'P' that is increased pressure in the compartment which is an investigation and needs a manometer for measurement).

'Stretch pain' and 'Stretch sign: Seen in the stage of impending ischemia. With advancing ischemia, the flexor muscles become edematous and go into contraction in order to reduce their muscle mass and accommodate themselves in a tight compartment.This results in progressive flexion of fingers.

This ischemic episode is painful and results in 'Stretch pain', when the flexed fingers are stretched and extended passively by the examiner (thereby stretching the contracted muscles). The sign is called the 'stretch sign'.

Sign of established ischemia: The hand remains paralyzed with no or presence of a flicker of movement. Sensory impairment also develops.

Note: Paralysis occurring in Volkmann's ischemia is mostly due to the ischemia of the muscles involved than the ischemia of the nerves.

Sign of established contracture
'Volkmann's Sign': It is a sign seen in 'Volkmann's Ischemic Contracture', and is described as follows, i.e. flexion of the wrist allows passive extension of the fingers and dorsiflexion does not.

Pathomechanics of 'Volkmann's Sign': When there is ischemic contracture, the muscles are fibrosed and shortened. Hence, dorsiflexion of the wrist does not allow passive extension of the fingers. Whereas, when the wrist is flexed these contracted and fibrosed muscles are relaxed, there is relative lengthening of the fibrosed muscles. This allows passive extension of the fingers.

Treatment

The principle of treatment in the 1st stage of impending ischemia: Is to restore the circulation back to normal so that the damage is reversed and no further progression takes place. It is an emergency.

Methods employed to restore the circulation back to normal
The methods employed are as follows in a sequential order.
1. Split the tight plaster or cut the tight bandage, if any.
2. Emergency closed reduction of fracture with or without 'K wire' fixation.
3. Open reduction and internal fixation of fracture using 'K wires', along with exploration and repair of the damaged vessel.

Note: At every step of treatment look for the return of pulse and capillary filling. At any point of time, if the circulation comes back the next step in the sequential order to restore the circulation is not carried out.

Vascular procedures employed to restore the circulation: The vascular procedures selected to restore the circulation depend on the nature of the vascular injury which may vary from a spasm, a laceration, a cut vessel, to a crushed vessel (e.g. Open fracture) and are as follows.
1. Injection of 1% Lignocaine or Papaverine into the vessel wall if only a spasm of the vessel is detected and there is no damage. This will relieve the spasm and restore the circulation.
2. Repair of the laceration, when a laceration is detected on exploration.
3. End-to-end anastomosis, in a cut vessel.
4. Resection and a vein graft, in a crushed vessel.

Indicators for return of circulation: The indicators for return of circulation are:
Clinical
   a. Return of pulse.
   b. Return of capillary filling.

Confirmatory (employing gadgets)
   a. Pulse oxymeter shows good saturation of oxygen above 90%.
   b. Doppler probe picks up a good signal of blood flow.

Principle of treatment in the 2nd stage: The principle of treatment in the 2nd stage is to improve the circulation by decreasing the compartmental pressure.

Surgical procedure employed in the 2nd stage: The surgical procedure employed is a wide fasciotomy of dividing the tight deep fascia, dividing the aponeurosis and the septae, excising the dead and devitalized tissue and leaving the wound open either for secondary closure or for closure by skin grafting procedure. This procedure of decompression is supposed to open up the collaterals, perfuse the remaining tissues and minimize the contracture, when the limb is protected in an appropriate splint.

Note: The paralysis occurring in Volkmann's ischemia is mainly because of ischemic muscles and not due to nerve paralysis.

If in transition phase between the 1st and the 2nd stage, both vascular repair and fasciotomy may have to be done (i.e. vascular repair is followed by fasciotomy).

Principle of treament in the 3rd stage: The principle of treatment in the 3rd stage is to restore some useful function in the contracted hand.

Restoration of useful function: This is accomplished by means of plastic and reconstructive procedures in the form of release of contractures, muscle-sliding procedures, lengthening of tendons, tendon transfers, shortening of the bones, fusion of joints, etc. as per the merit of the case.

2. ***Nerve Injuries***

In the order of frequency as observed in this country it is as follows:

In extension type of supracondylar fracture, median nerve followed by the ulnar nerve and then the radial nerve.

In flexion type of supracondylar fracture, ulnar nerve followed by the median nerve and then the radial nerve.

Note: There is difference of opinion regarding the order of frequency of nerve involvement in a supracondylar fracture. Some of the books mention that the radial nerve is the one that is commonly involved followed by median nerve and the ulnar nerve.

Type of nerve injury: In closed fractures, it is either neuropraxia or axonotmesis. Hence proper splinting and later supervised physiotherapy, results in normal functional recovery.

In open fractures however, there is a possibility of a Neurotmesis. In such a case a nerve repair is needed, i.e Neurorraphy. Recovery depends on severity of neurotmesis and quality of repair.

At times, there is a third possibility of a nerve getting adhered in the callus or myositis mass. This needs release of a nerve and neurolysis. Recovery is slow and results are unpredictable.

3. ***Malunion***

Malunion results in cubitus varus and 'Gun Stock' deformity. Rarely Cubitus valgus.

Note: Malunion in cubitus varus with internal rotation results in a 'Gun Stock' deformity (Fig. 2.47).

Definition of Carrying Angle, Cubitus Varus and Cubitus Valgus: The angle made by the long axis of the arm with the long axis of the forearm in extended position of the elbow is known as 'Carrying Angle'. Normal angle is 5-10° in males and 10-15° in females.

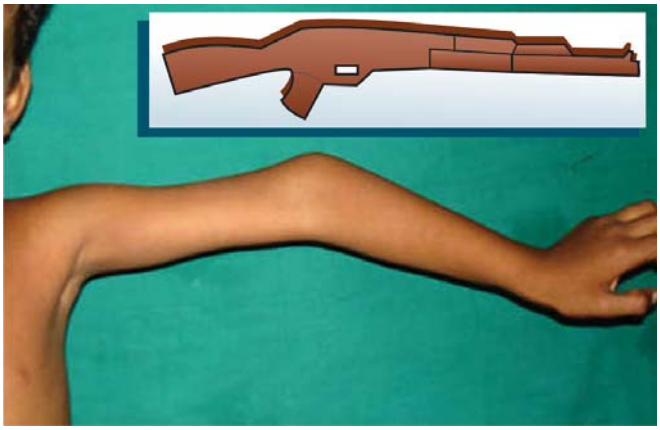


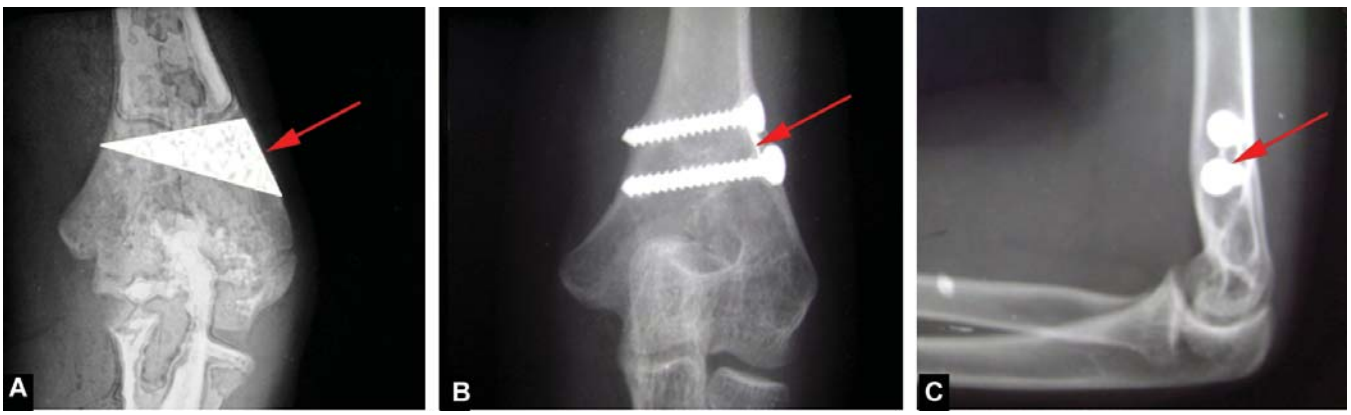
Figure 2.47

A photograph showing a classical 'Gun stock deformity' of a malunited supracondylar fracture.

When this 'Carrying Angle' reverses it is said that the elbow is in Cubitus varus and when this 'Carrying Angle' increases it is said that the elbow is in Cubitus valgus.

Note: If there is a flexion deformity at the elbow carrying angle cannot be measured. This angle always is measured with the elbow in extension.

Surgical procedure for correction of deformity: The deformity is corrected by means of a corrective osteotomy. French osteotomy corrects both varus and internal rotation malalignment of a 'Gun Stock'deformity. It is a lateral closed wedge derotational type of osteotomy, fixed internally with 'fig of 8' SS wires wound round the screws which are passed proximal and distal to the site of osteotomy prior to osteotomy in a specific manner (Figs 2.48A to C).



Figures 2.48A to C

Radiographs showing classical procedure of French osteotomy done for a gun stock deformity of malunited supracondylar fracture. Note the appropriately marked wedge of bone to be removed, the placement of screws and binding them with SS wires. The lower screw is placed anterior to the upper screw. So when the wedge is closed and the screws are aligned, along with correction of varus deformity internal rotation deformity also gets corrected.

Rare deformity of 'Cubitus Valgus' is corrected by medial closed wedge osteotomy.

Myositis Ossificans

It is an ossification or calcification occurring within a muscle following injury. Brachialis muscle is the commonly affected muscle.

Treatment: When in the acute and active phase no intervention should be attempted. Intervention in any form will aggravate the condition. When the acute phase subsides and the mass lies dormant surgical removal followed by adequate gradual and active physiotherapy to regain the movement is necessary.

Revision Questions

General

Q. What is the age group for Supracondylar fractures?
Q. What are the types of Supracondylar fractures of the humerus?
Q. Which type of Supracondylar fracture is common?
Q. What is the mechanism of injury?
Q. What is the nature of the fracture line?
Q. What is the importance of knowing the nature of the fracture line in a Supracondylar fracture?
Q. What is role of the muscle triceps and brachialis in the treatment of Supracondular fracture?
Q. When do you do 'K-Wire' fixation of a Supracondyar fracture?

Complications

Q. What are the complications seen in supracondylar fracture?
Q. What is "Volkmann's Ischemia" and Ischemic Contracture?
Q. What are the 3 stages of " Volkmann's Ischemic Phenomenon" ?
Q. What is importance of understanding the 3 stages?
Q. What are the cardinal signs of impending ischemia?
Q. What is 'Stretch pain' and Stretch sign'?
Q. What is 'Volkmann's sign'?
Q. What is the principle of treatment in the 1st stage of ischemia?
Q. What are the methods employed to restore the circulation back to normal at earliest possible moment?
Q. What are the vascular procedures employed to restore the circulation?
Q. What is the principle of treatment in the 2nd stage of ischemia?
Q. What is the surgical procedure indicated in the second stage of ischemia?
Q. What is the principle of treatment in the 3rd stage of ischemia?
Q. What are procedures indicated in the 3rd stage of ischemia for restoration of useful function?
Q. Name the nerves that may get injured in a Supracondylar fracture.
Q. What is the nature of nerve involvement in a Supracondylar fracture?
Q. What is the prognosis of nerve injury in a Supracondylar fracture?
Q. What are the deformities seen in a malunited Supracondylar fracture?
Q. What is 'Carrying Angle' at the Elbow?
Q. What is Cubitus varus and Cubitus valgus?
Q. How do you correct the deformity in malunited supracondylar fracture?
Q. What is Myositis Ossificans?
Q. Which muscle is commonly affected in Myositis Ossificans?
Q. What is the treatment of Myositis Ossificans?

Essay Questions

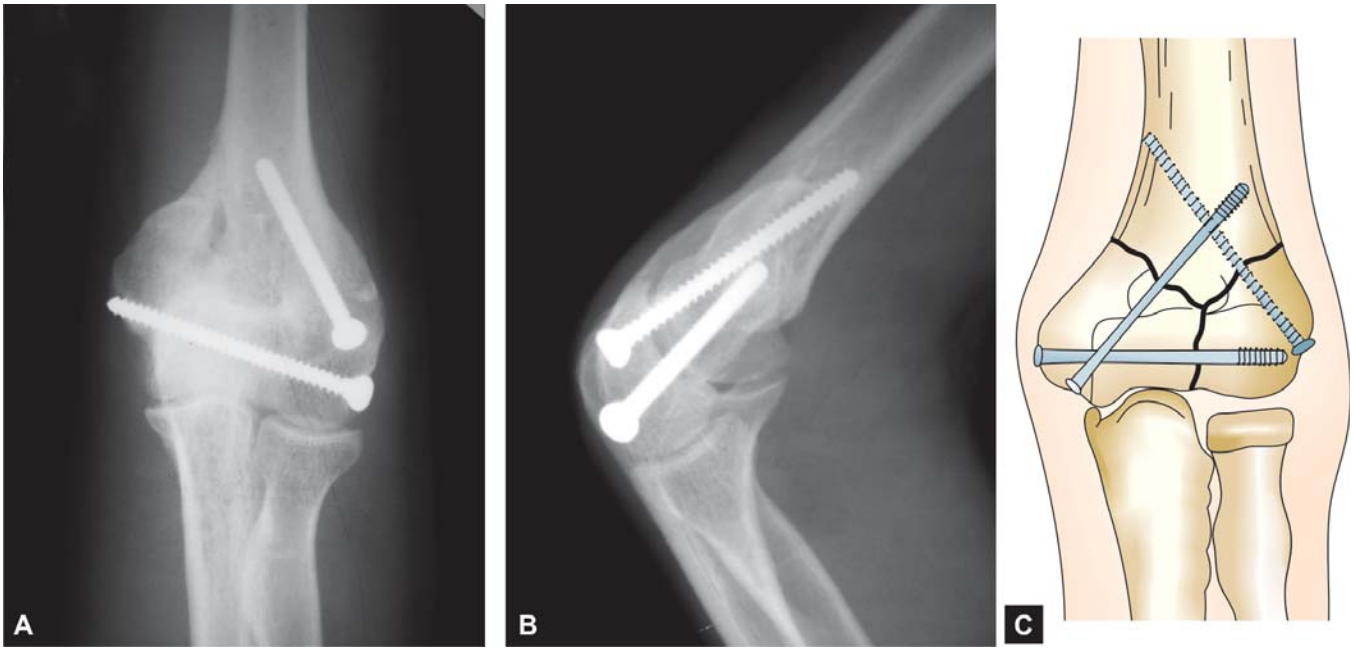
Q. What are the types of Supracondylar fractures of the humerus? Discuss the mechanism of injury, diagnosis and treatment of Supracondylar fracture. Enumerate the complications.
Q. What are the complications of Supracondylar fracture? Discuss the management.
Q. What is Volkmann's Ischemic phenomenon? Discuss the management of Volkmann's Ischemia and contracture.
Q. What is Gun Stock deformity? Discuss the management of Gun stock deformity.

Intercondylar Fracture

These are the fractures seen at the lower end of the humerus as a result of severe trauma. The fracture line runs between the condyles and hence the name intercondylar fracture is derived.

Mechanism of Injury

As a result of severe impact on the point of the elbow the olecrenon process is firmly driven upwards splitting the condyles into two. Comminution is seen when the impact is severe.



Figures 2.49A to C

Radiograph showing (A) Intercondylar fracture AP view, (B) Intercondylar fracture lateral view, (C) Line diagram showing fixation of a 'Y' type of non comminuted intercondylar fracture using cancellous and cortical screws.

Types

A. 'T' type—In this the humeral fracture line is transverse and the intercondylar fracture line is vertical resembling the letter T.
B. 'Y' type—In this both humeral as well as the intercondylar fracture line are obliquely placed resembling the letter Y.

Diagnosis

Proper X-rays are essential for clear definition of the injury.

Treatment

Non-operative: There is limited role for nonoperative treatment. It is indicated only in patients who are not fit for surgery or the injury is so severe and the elbow is reduced to a bag of bones. In such cases the elbow is initially splinted and later, after the initial swelling subsides a hinge brace is given and movements of the elbow is encouraged as much as feasible.

Skeletal traction for a limited period is the other option available.

Operative: Surgical treatment definitely gives better results with respect to union and function.

Seniority and skill of the surgeon has direct bearing on the functional out come. Surgery aims at restoring the medial and the lateral column. Though congruity of the articular surface is restored limitation of movements of flexion and extension in terminal degrees is common. This is attributed to the severity of surrounding soft tissue injury. 1/3 tubular plates, Recon plates and screws (both cortical and cancellous) are the implants used to stabilise these fractures (Figs 2.49A to C).

Total elbow replacement is an option which may be considered in elderly when the elbow is reduced to a bag of bones.

Complications

Early

1. Injury to the brachial artery.
2. Injury to the median/ulnar/radial nerve.

Late

1. Limtation of joint movements.
2. Myositis ossificans.

Fractures in the Arm and the Shoulder

Fracture Shaft of the Humerus

General Information

This fracture is also known as a diaphyseal fracture of the humerus. ***The shaft of the humerus bone is an area extending from the upper border of pectoralis major muscle in the region of the shoulder to supracondylar ridge at the elbow.*** The shape of the bone is cylindrical in its proximal half but changes to triangular or prismatic in its distal half. The two intermuscular septae, namely the medial and the lateral divide the area into two compartments, i.e anterior and posterior. The anterior compartment contains the muscle biceps brachii, coracobrachialis, brachialis, brachial artery and vein, the median, musculocutaneous and ulnar nerves. The posterior compartment contains the triceps and the radial nerve. Although these fractures are inherently unstable, nonoperative method is the treatment of choice except when definite indications for surgery exist.

Mechanisms of Injury of the Fracture Shaft Humerus (Figs 2.50A to C)

Indirect mechanism

Is by means of a fall on an out stretched hand.

a. A bending force produces a transverse fracture.
b. A tortional force produces a spiral fracture.
c. A combination of both bending and tortional force produces a comminuted fracture with a butterfly fragment/fragments.

Direct mechanism

Is by means of a blow on to the arm which results in a shattered displaced fracture of the shaft of the humerus, e.g. assault by a stick, a high velocity injury etc.

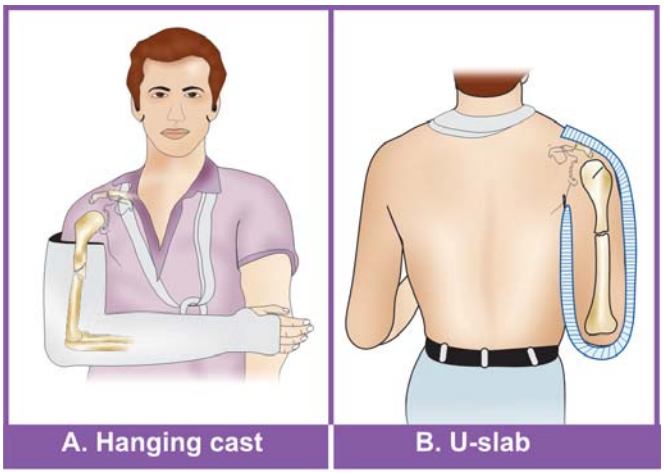
Treatment

A. *Nonoperative:* The nonoperative methods of treatment of this fracture are:
   a. By closed reduction and maintenance in a 'U' slab or a cast (Fig. 2.51B).
   b. By maintaining the fracture reduction in a 'Hanging Cast' (Fig. 2.51A).

B. *Operative (Figs 2.51C and D):*
   Indications
   1. Noncompliance.
   2. Unacceptable reduction (failure of closed reduction).
   3. Displaced, comminuted or segmental fracture.
   4. Multiple fractures (Polytrauma, ipsilateral ulna or radius fracture).
   5. Open fractures.
   6. Fractures associated with neurovascular injury.
   7. Fractures with intra-articular extension.

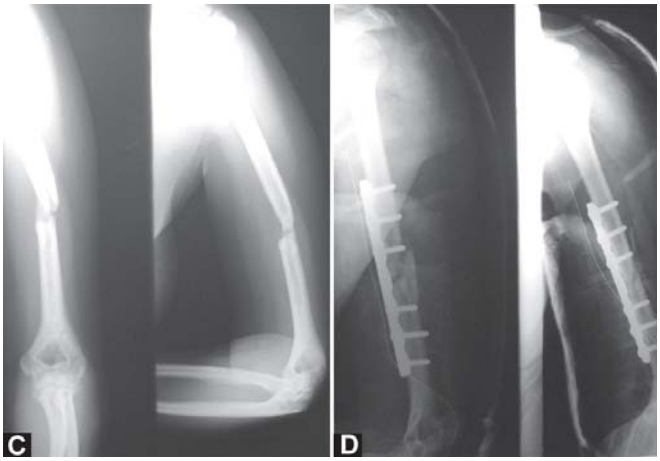
Figures 2.50A to C

Radiographs showing transverse, spiral and comminuted fracture of the humerus at different levels.



Figures 2.51A and B

Showing different non-operative treatment methods of fracture shaft of humerus



Figures 2.51C and D

Radiographs showing (C) A delayed union of fracture shaft of humerus. (D) Internal fixation using LCDCP and cancellous bone grafting from the iliac crest.

Implants used for surgery: Various plates and screws, DCP, LCDCP, LCP. Intramedullary nails including interlocking nails. Other nails include flexible nails like Enders, AO, etc. External fixators are used in open fractures.

Complications

1. Injury to the Radial nerve (Incomplete or Complete)
2. Delayed union.
3. Nonunion.

Treatment of Complications

Radial nerve injury: The radial nerve gets injured because of its close relation to the bone in the radial groove. The type of nerve injury is generally neuroparaxia/axonotmesis. Patient develops wrist drop which generally recovers on its own over a period of time. Drug therapy splints and physiotherapeutic measures help in the recovery. Surgical exploration is rarely needed (Fig. 2.52).

Delayed union and nonunion: The delayed union and nonunion of fracture shaft of humerus is treated by means of internal fixation and cancellous bone grafting.

Questions

Q. What are the mechanisms of injury of fracture shaft humerus?
Q. Discuss the treatment of fracture shaft humerus.
Q. Enumerate the complications of fracture shaft humerus and discuss their management.

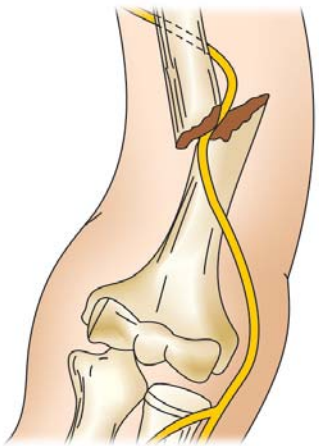


Figure 2.52

Diagrammatic representation of radial nerve entrapment in a fracture shaft humerus as a result of its close relation to the shaft of the humerus in the radial groove.

Fractures of the Proximal Humerus

General Information

In the year 460BC, it was Hippocrates who documented a case of fracture neck humerus first and treated it by using traction. Codman, in 1934 divided the proximal humerus into four parts based on epiphyseal lines. The head (the articulating surface with anatomical neck), the greater tuberosity, the lesser tuberosity and the shaft. The surgical neck is distal to both the tuberosities and it is that portion of bone between the tuberosities and the shaft. According

to Codman, fractures of the proximal humerus produce a combination of the four segments.

The blood supply is from the branches of the axillary artery running in a distal to proximal direction. Hence, fracture of the anatomical neck may result in loss of blood supply to the head of the humerus and avascular necrosis of the head of the humerus.

Shoulder is an important ball and socket type of a joint linking the upper extremity to the thorax. Because of an arc of movement taking place at the shoulder joint an individual is able to perform explosive activities involving power, e.g. throwing an object, as well as activities that are refined such as playing a violin. So, fracture of the proximal humerus can cause severe disability in the upper extremity by limiting the function of the shoulder.

Mechanisms of Injury

1. Common mechanism—by a fall on an outstretched hand from a standing height.
2. Other mechanism—a direct blow on to the proximal humerus.

Classification

The commonly accepted classification is ' Neer's' classification (Fig. 2.53).

This system of classification includes four segments.

- The head of the humerus.
- The greater tuberosity.
- The lesser tuberosity.
- The shaft of the humerus.

According to Neer, a fracture is displaced when there is more than 1 cm of displacement and 45° of angulation of any one fragment with respect to the others.

Displacements occur because of the muscle pull.

a. The supraspinatus and the Infraspinatus pull the greater tuberosity superiorly.
b. The *Subscapularis* pulls the *lesser tuberosity medially.*
c. The *Pectoralis Major adducts the shaft medially.*

The 'Two-part fractures' involve any of the 4 parts and include 1 fragment that is displaced.

The 'Three-part fractures' include a displaced fracture of the surgical neck in addition to either a displaced greater tuberosity or lesser tuberosity fracture.

The 'Four-part fractures' include displaced fracture of the surgical neck and both tuberosities.

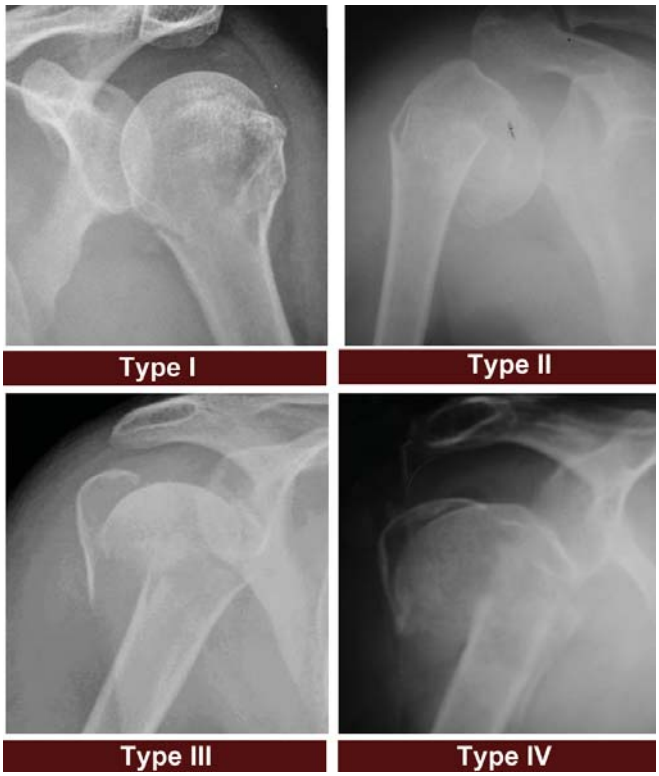


Figure 2.53

Showing radiographs of different types of surgical neck fractures based on Neer's classification. Note that type one is undisplaced or displacement is less than a mm and type II ,III and IV are displaced and refer to 2 part, 3 part and 4 part fracture respectively with regards to number of fragments.

Treatment

A. *Nonoperative:* Undisplaced or minimally displaced fractures are managed nonoperatively and immobilized in plaster slab or by *Velpeau* bandage and strapping techniques.
B. *Operative:* Displaced fractures and fractures that pose difficulty for closed manipulation are managed by surgical intervention.

Methods employed: The surgical methods employed are

1. Closed reduction and percutaneous fixation.
2. Open reduction and internal fixation using plates and screws, e.g. 'T' plates, proximal humerus locking plates (Fig. 2.54).
3. Minimally invasive percutaneous plate osteosynthesis.
4. Primary arthroplasty (3 part and 4 part fractures and fractures involving head especially in middle aged and elderly).

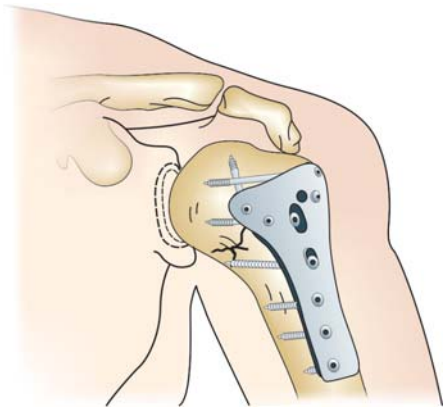


Figure 2.54
Method of fixation of proximal humeral fracture with T-plate and screws

Complications

1. Malunion.
2. Non union.
3. Avascular necrosis of the head of the humerus.

Treatment of Complications

Malunion: The scapulothoracic movement compensates to a certain instant, the limitation of movement that occurs at the glenohumeral joint secondary to malunion, corrective osteotomy is rarely indicated.

Non union and AVN are managed by shoulder arthroplasty using prosthesis, e.g. Neer's prothesis.

Resotoration of useful shoulder movement is the principle involved in these surgeries.

Revision Questions

Q. What are the mechanisms of injury of fracture surgical neck of humerus?
Q. Which is the commonly accepted classification of fracture neck humerus?
Q. How do you manage this fracture?
Q. What are the surgical methods employed to treat this fracture surgical neck of the humerus?
Q. What are the complications seen?
Q. How are these complications managed?

Fracture of the Clavicle

General Information

Also known as collar bone, clavicle is the only bone which connects shoulder girdle to the trunk (connects the breast bone i.e sternum to shoulder blade, i.e scapula.)

It is a curved subcutaneous bone which changes its shape from round and somewhat cylindrical medially to flat and quadrilateral laterally. It protects the underlying vessels, lungs and brachial plexus.

Though this fracture is seen in all age groups it is most commonly seen in neonates (following a difficult delivery) and in children.

Mechanisms of Injury (Fig. 2.56)

1. By means of fall on an outstretched hand
2. By means of a fall on the point of a shoulder.
3. By a direct blow on to the clavicle.

Common Site and Classification (Fig. 2.55)

- 80% occur in the middle 1/3 (Class A)
- 15% occur in the lateral or distal 1/3 (Class B)
- 5% occur in the medial or proximal 1/3 (Class C)

Note: Class B is further classified as

I. Non displaced with intact supporting ligaments.
II. Displaced because of coracoclavicular ligament rupture and pull of the sternocliedomastoid muscle
III. Articular fracture involving the aromioclavicular joint.

Figure 2.55
Radiographs showing different types of clavicular fracture Class A fracture, i.e. in the middle 1/3. Class B fracture, i.e. in the lateral end or distal 1/3 and Class C fracture, i.e. in the medial 1/3.

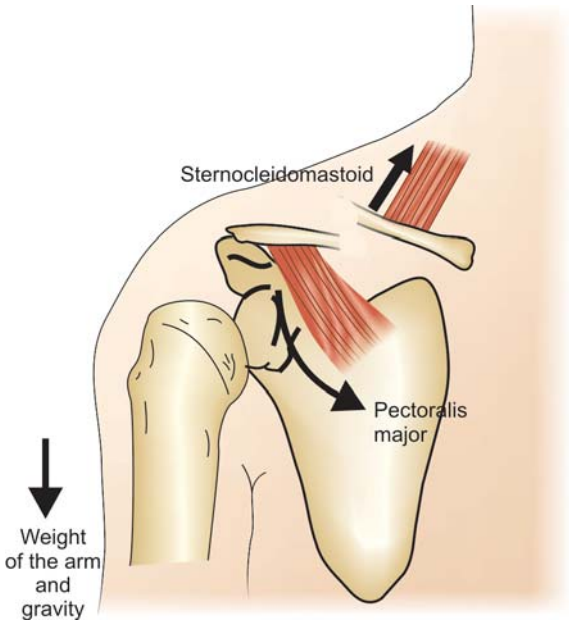


Figure 2.56
Showing various displacing forces in fracture clavicle

Treatment

In infants and children fracture invariably heals with simple strapping or even with an arm sling immobilization.

In adults treatment depends on the Class of the fracture (Fig. 2.57).

Class A fractures—May need immobilization in a Fig of 8 bandage and a sling, with or without reduction as per the displacement of the fragments.

Class B fractures—Type I needs only supportive strapping and a sling for the arm and fracture heals without any problem.

Type II and III need surgical methods for reduction and stabilization.

Class C displaced fractures—May need surgical reduction.

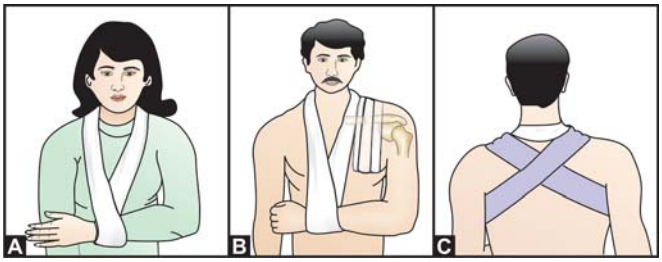


Figure 2.57
Methods of conservative treatment of fractures clavicle: (A) Collar and cuff sling, (B) Strapping and sling suspension, (C) Figure of 8 bandaging

Complications and Treatment (In Brief)

1. *Life threatening and limb threatening* complications may be associated with fracture clavicle, e.g. hemothorax, pneumothorax, hemopneumothorax, injury to subclavian vessels, injury to brachial plexus, etc. These are surgical emergencies and need specialized care.
2. Delayed union and non union. Only if symptomatic the surgical procedures for achieving union in the form of internal fixation and bone grafting are done. If asymptomatic and no disability is observed they are left alone.
3. Malunion. Generally left alone.

Revision Questions

Q. What are the mechanisms of injury of fracture Clavicle?
Q. What is the acceptable classification of fracture Clavicle?
Q. What is the treatment?
Q. What are complications associated with fracture Clavicle? How do you manage them?

Essay Question

Q. Discuss the mechanism of injury, diagnosis and management of fracture clavicle. Enumerate the complications.

Fractures of the Scapula

Mechanism of Injury

1. A direct blow on to the scapula, e.g. assault (Fig. 2.58).
2. Axial loading through the glenohumeral joint.

Sites of the Fracture (Figs 2.59A and B)

In the order of frequency the fracture sites are as follows:

a. Fracture of the Body.
b. Fracture of the Neck.
c. Fracture of the Glenoid.
d. Fracture of the Acromian.
e. Fracture of the Coracoid.

Incidence

Other Injuries Associated with Fracture Scapula and their Incidence

The incidence of associated injuries is around 80-90%. The following injuries are commonly seen:

1. Pulmonary contusion and Pneumothorax (23%)
2. Clavicle fracture resulting in 'Floating Shoulder' (23%)
3. Anterior or posterior dislocation of the shoulder.

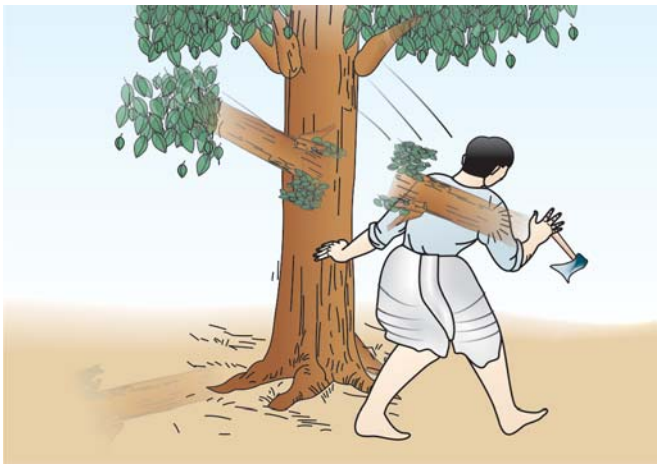


Figure 2.58

Most common mode of injury causing scapular fracture is a direct impact on the scapula. The diagram shows a direct impact by the branch of a tree falling on to the shoulder blade.

4. Brachial plexus and axillary artery injury.
5. Rib fracture.

Note: Scapular body fractures heal without any problem in about 6 weeks. A simple immobilization in a sling is sufficient. Similarly fracture of the acromion process of the scapula unless it causes compression of the rotator cuff is treated nonoperatively.

Classification of the Scapular Neck Fractures

The scapular neck fractures are classified as follows:

Type I—Nondisplaced and non angulated
Type IIa—Shortened/displaced >1 cm.
Type IIb—Angulated > 45°.

Treatment of Scapular Neck Fracture

Type I—are treated nonoperatively and yield good functional results.

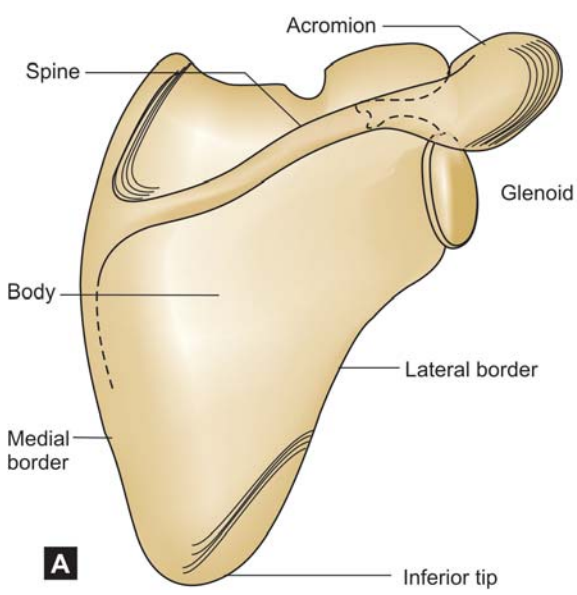
Type IIa and IIb fractures need surgical treatment as they are displaced and angulated.

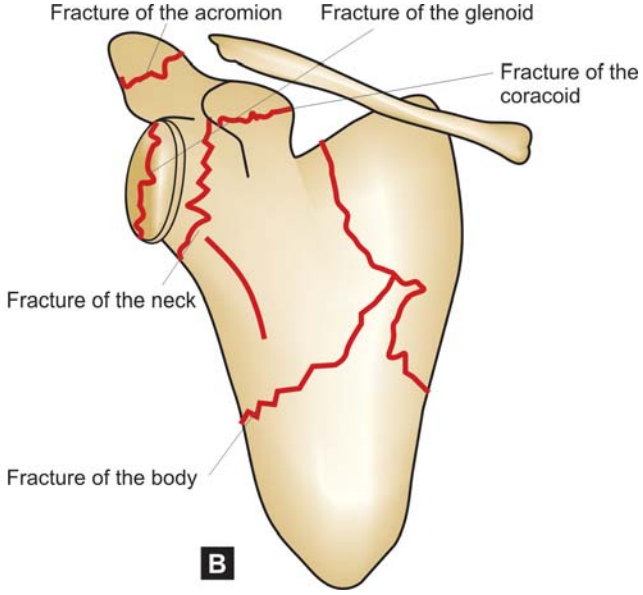
Classification of the Fractures of the Glenoid

Based on Ideberg classification classified as:

Type I—Anterior avulsion fractures.
Type II—Transverse, inferior glenoid fractures.
Type III—Transverse superior glenoid fractures.
Type IV—Transverse fractures through the body
Type V—A combination of Type II and Type IV.

Indications for surgery in glenoid fractures: Those fractures which are likely to cause instability are definite indicators of surgery. These are





Figures 2.59A and B

Showing (A) bony anatomical features of scapula (B) different types of fracture scapula

1. Rim fractures with > 1cm displacement / involving > 25% of the auricular surface.
2. Intra-articular fractures with subluxation of the head of the humerus.
3. Glenoid fossa fractures which are displaced > 5 mm.

S*urgical approaches used*: Anterior fractures are exposed by means of anterior exposure to the shoulder and posterior fractures are exposed through the posterior exposure.

Implants that are used to fix these fractures: The various implants used are 3.5 mm cortical and 4.0 mm Cancellous screws (for lag effect) and 1/3 tubular plate (for buttress effect).

Questions

Q. What are the sites of the fracture of the scapula bone?
Q. What are the injuries associated with fracture Scapula and their incidence?
Q. How do you classify scapular neck fractures?
Q. What is the treatment of scapular neck fracture?
Q. How do you classify the fractures of the glenoid?
Q. What are the indications for surgery in glenoid fractures?
Q. What are the surgical approaches used?
Q. What are the implants that are used to fix these fractures?

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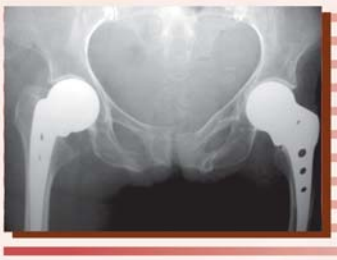


3

Fractures in the Lower Limb

Introduction, General Information, Diagnosis, Treatment and Complications are dealt with as follows:

- *Fractures of the Foot*
- *Injuries Around the Ankle*
- *Tibial Diaphyseal Fractures*
- *Injuries Around the Knee*
- *Fracture Shaft of Femur*
- *Fractures Around the Hip*

Fractures of the Foot

Introduction

The foot has a very important role in locomotion. It is subjected to constant stress of weight bearing. Loss or impairment of anatomy of the foot following injury may thus lead to serious disability and malfunction. Hence, a super speciality of ***Foot and ankle surgery*** has evolved and meticulous foot and ankle surgery is being performed these days.

The foot is generally divided into (Fig. 3.1):

A. Hind foot consisting of calcaneus and talus.
B. Mid foot consisting of navicular, cuboid and the three cuneiforms.
C. Fore foot consisting of metatarsals and phalanges.

The Hind Foot

The articulation between the talus and the calcaneus is known as subtalar joint. The movement of inversion and eversion takes place at this joint.

Figure 3.1

Lateral radiograph of a normal foot showing (A) The fore foot: portion (B) The mid foot: portion (C) The hind foot.

The talus articulates with the ankle mortise formed by the lower end of tibia and fibula. The movement of dorsiflexion and plantar flexion takes place at the ankle joint.

The Mid Foot

The tarsal bones are connected together with multiple articulations and movement in these articulations occurs in a very unique way as a unit. When inversion/eversion takes place at the subtalar joint the tarsal bones lock themselves and move together only to get unlocked when the movement occurs in the opposite direction (eversion/inversion).

It is this effective, constant and multidirectional adjustment of the tarsus, makes the foot to adapt itself to any terrain for a smooth propulsion. Thus, loss or impairment of this mechanism following injury to the tarsal bones/articulation results in severe disability.

The articulation between the hind foot and the mid foot is known as mid tarsal joint also known as ***Chopart's Joint*** named after ***Francois Chopart*** (1743–1795) who performed amputations at the level of calcaneocuboid and talonavicular joint. This procedure removes almost all the insertions of tendons around the ankle and renders the ankle joint unstable.

The articulation between the mid foot and fore foot is known as ***Lisfranc's joint*** named after ***Jacques Lisfranc*** a French surgeon in Napolean's army. An amputation at this level is known as ***Lisfranc's amputation*** as he used to perform amputations at this level for frost bite on soldiers at the Russian front.

The Fore Foot

The articulations between the metatarsals and phalanges are more firm and regular and do not allow multidirectional adjustments. These joints are more involved in transmission of body weight during push off phase of gait cycle. Hence, any disturbance in anatomy following injury will affect the push off phase of gait cycle.

The important injuries occurring in the foot are:

1. Chopart's injury.
2. Lisfranc's injury.
3. Jones' fracture.
4. Pseudo-Jones' fracture.
5. Talus fracture.
6. Calcaneus fracture.

Chopart's Injury

Definition

It is a fracture dislocation involving the talonavicular and the calcaneocuboid joint (Mid Foot).

Mechanism

Common mechanism of injury (80%): Is by means of a severe twisting injury on a plantar flexed and inverted foot causing medial and upward displacement of distal fragments.

Rare mechanism of injury: Is an 'Eversion injury' which causes lateral displacement of distal fragments. (In both the talus always remains in the ankle mortise.)

Fractures associated with Chopart's injury

1. Calcaneus fractures.
2. Talus fractures.
3. Navicular fractures.

Management

Prompt and early accurate reduction and stabilization is the treatment of choice. Hence, nonoperative methods have only a limited role to play. They are useful only in minor grades of injury.

Prognosis

Poor prognosis is seen in those cases which are associated with severe soft tissue injury, malalignment, other bone fractures and present late.

Lisfranc's Injury

Definition

By definition this is a fracture or a dislocation or a fracture dislocation occurring at the junction between the tarsal bones of the mid foot and the metatarsal bones of the fore foot

named after ***Jaques Lisfranc,*** a French surgeon in Napolean's army.

Note: It is also said that apart from performing amputation at this level for frost bite Lisfranc described this injury in soldiers when they fell down from their horse, when their foot did not get released from the stirrup.

Mechanism

Mechanism involves severe plantar flexion of the foot, e.g. stepping into a small hole, sports-related injuries, motor vehicle accidents, falling from a height, down stairs or off a curb.

Note: Instability on weight bearing can occur with ligamentous injuries alone, even without fracture or dislocation.

Lisfranc ligament diagonally connects the 1st (medial) cuneiform with the base of the 2nd metatarsal. If it remains intact, either an avulsion of the lateral border of the 1st cuneiform or an avulsion of the base or medial border of the 2nd metatarsal occurs. If it tears, these fractures may not occur.

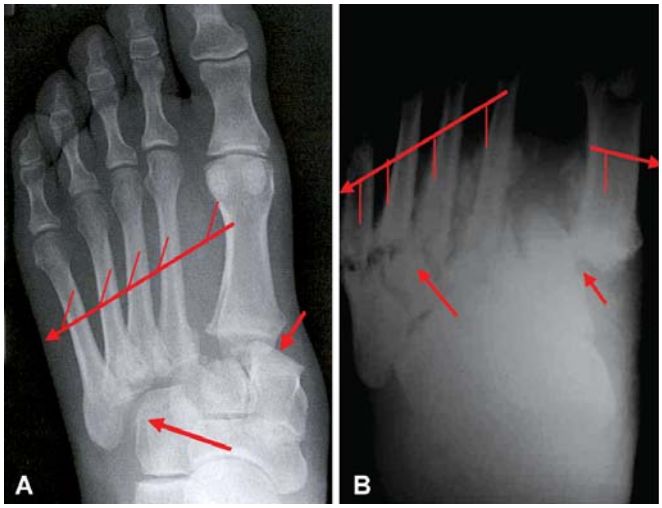
Types

Two basic types are described.

Homolateral (Fig. 3.2A)
- All of the metatarsals are dislocated to the same side.
- More common than divergent.
- Usually involves the 2nd through 5th metatarsal with dislocate laterally.
- May involve all 5 metatarsals.

Divergent (Fig. 3.2B)
- Usually more severe than homolateral.
- May be associated with a fracture of the 1st cuneiform.
- Usually involves medial displacement of the 1st metatarsal and lateral displacement of 2nd-5th metatarsals.
- Occasionally medial displacement of only the 1st metatarsal may be seen.

Fractures associated with Lisfranc dislocations
- Base of 2nd metatarsal fracture.
- Cuboid fracture.
- Fractures of shafts of metatarsals.
- Dislocations of the 1st (medial) and 2nd (middle) and cuneonavicular joints.
- Fractures of the navicular bone.



Figures 3.2A and B

Radiographs of the foot showing (A) Homolateral and (B) Divergent types of Lisfranc's injury. Note the direction of displacements of the 1st metatarsal and the 2nd, 3rd, 4th and 5th metatarsals.

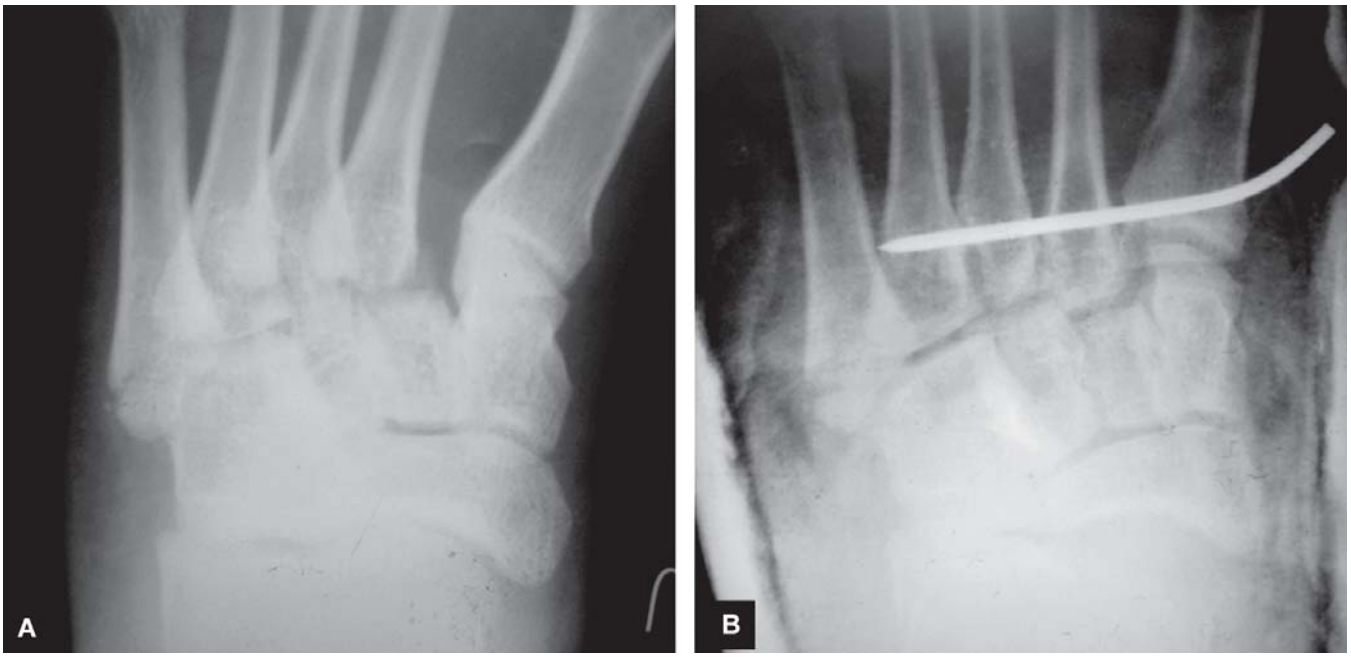
Imaging

Conventional radiographs are usually sufficient to demonstrate the injury. Normal alignment of the cuneiforms and the bases of the metatarsals are as follows:
- Lateral border of 1st metatarsal is aligned with lateral border of 1st (medial) cuneiform in AP view.
- Medial border of 2nd metatarsal is aligned with medial border of 2nd (intermediate or middle) cuneiform in AP view.
- Medial and lateral borders of the 3rd (lateral) cuneiform should align with medial and lateral borders of 3rd metatarsal in oblique view.
- Medial border of 4th metatarsal is aligned with medial border of cuboid in oblique view.
- Lateral margin of the 5th metatarsal may project lateral to cuboid by as many as 3mm in oblique view.
- In lateral view, a line drawn along long axis of talus should intersect long axis of the 5th metatarsal.

Any loss of this pattern of alignment is an indicator of this injury.

Management

Restoration of anatomical alignment at the earliest is the key for successful result. Hence, closed methods of reduction/fixation and immoblization have a role to play only in less



Figures 3.3A and B

Radiographs showing successful reduction and stabilization of a Lisfranc's injury (Homolateral type) by closed reduction and pinning.

severe forms of injury. Severe forms are always treated by open reduction and fixation (Figs 3.3A and B).

Complications

a. Compartmental syndrome involving the foot compartment (immediate).
b. Malalignment with foot deformities resulting in chronic pain and degenerative arthritis involving tarsal joints (late).

Prognosis

Severe soft tissue injury, persistent malalignment, associated fractures, late presentations results in poor prognosis.

Revision Questions

1. What is Chopart's and Lisfranc's joint? Why it is named so?
2. What is Chopart's and Lisfranc's amputation?
3. What is Chopart's and Lisfranc's injury?
4. What is the mechanism of Chopart's and Lisfranc's injury?
5. What is Lisfranc's ligament? What are the types of Lisfranc's injury?
6. What are the fractures associated with Chopart's and Lisfranc's injury?
7. How do you manage Lisfranc's and Chopart's injury?
8. What are the complications of Chopart's and Lisfranc's injury?
9. What is the prognosis of Chopart's and Lisfranc's injury?

Fracture of the Fifth Metatarsal Bone

The proximal fifth metatarsal fracture is the most common injury among the mid foot fractures. The distal and the mid shaft fractures are not common.

Types:

A. Jones Fracture.
B. Pseudo–Jones Fracture (Tennis Fracture).

Jones' Fracture

This injury was first described in 1902 by Sir Robert Jones in an article titled "Fractures of the Base of the Fifth Metatarsal Bone by Indirect Violence." He himself is said to have suffered from this fracture after dancing.

Definition

It is a transverse fracture occurring at base of the fifth metatarsal 1.5-3.0 cm distal to the tuberosity (Fig. 3.4).

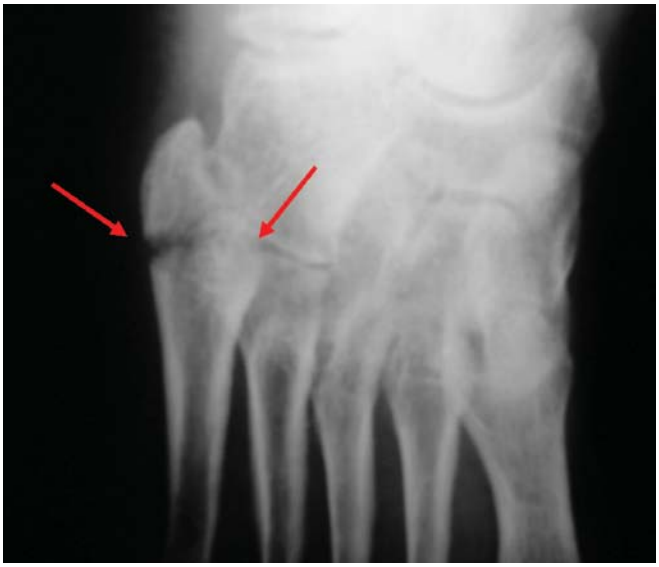


Figure 3.4
Radiograph of a Jones fracture.

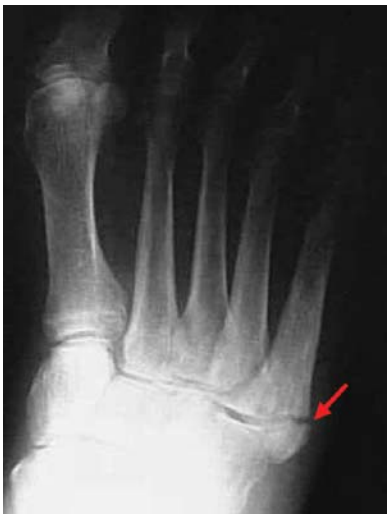


Figure 3.5
Radiograph showing pseudo-Jones fracture.

Diagnosis and Treatment

1. Pain in the region of the fifth metatarsal after an indirect violence.
2. Difficulty in walking.
3. X-ray confirming the diagnosis.

Always treated by a non-weight bearing cast for the fear of becoming displaced and causing nonunion at a later period. Nonunion may need bone grafting.

In athletes and in displaced fractures surgical stabilization is considered.

Pseudo-Jones Fracture

Definition

It is an avulsion fracture of the base of the the fifth metatarsal with a lateral ankle strain occurring as a result of the pull of Peroneus brevis tendon (Fig. 3.5).

Treatment

Heals well with a compression bandage or a walking cast in about 3 weeks without complications.

Fracture of the Talus

General Information

Talus also known as Astragalus is *a unique bone which has no muscle or tendon attachments* and held in place mainly by bony and ligamentous support. *Superiorly* it articulates with the inferior articular surface of the *tibia, Inferiorly* with the superior articular surface of the *calcaneus, medially* with the *medial malleolus, laterally* with the *lateral malleolus* and *anteriorly* with the *navicular.*

Hence plays an important role in the function of the ankle and the foot being *responsible for 90% of the movement of the ankle and foot.*

History

It was Sir Astley Cooper 1832 who gave a first hand account of a case of dislocated Talus which he treated. (Mr. Downes 20th July 1820,who fell down from his horse…...)

Anderson 1919 gave the name **'Aviators Astragalus'** because he found these injuries occurring in aviators when they crashed with their machine. The foot resting on the rudder bar took the impact of extreme dorsiflexion, plantar flexion or inversion thereby causing injuries of the talus. Namely fractures, dislocations and fracture-dislocations.

Blood Supply (Figs 3.6 and 3.7)

G.L Mulfinger and J Trueta have described in detail the blood supply of the talus (J Bone Joint Surgery 1970;52B.).

Three main arteries of the leg supply the Talus through *periosteal network* and through *two discrete vessels*. They are:

1. Posterior Tibial artery—through calcaneal branches.
2. Anterior Tibial artery—through anterolateral/anteromedial malleolar branches or through medial tarsal artery.

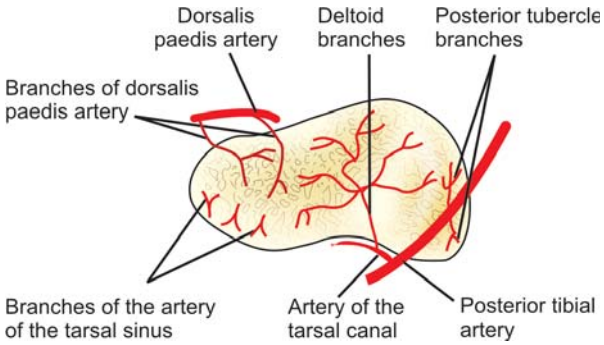


Figure 3.6

Showing blood supply to the talus. Note that the important supply is through the artery of the tarsal canal which anastomoses with the artery of the sinus tarsi.

3. Peroneal artery—through perforating branches.
4. Artery of the Tarsal Canal, a branch of Posterior Tibial artery.
5. Artery of the Sinus Tarsi, a branch of dorsalis pedis artery.

Two plexus are formed by anastomosis.

i. In the region of the posterior tubercle.
ii. In the region of the sinus tarsi.

Thus 'vascular slings' are formed all round the talus.

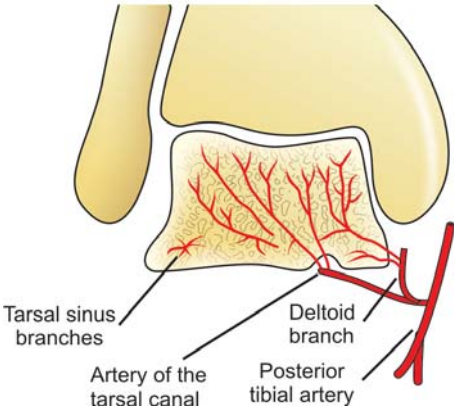
(The calcaneal branches of posterior tibial artery form plexus in the region of posterior tubercle and the anterolateral malleolar branches of anterior tibial artery along with branches from dorsalis pedis artery form plexus in the region of sinus tarsi. Thus, there is formation of 'vascular slings' all around the talus in the form of anastomosis between different vessels).

An undisplaced fracture of the neck disturbs the blood supply the least. Hence, the incidence of *AVN is low.* (Intact 'vascular slings').

A displaced fracture neck of the Talus is likely to disturb the blood supply to the body and *causes avacular necrosis.* Similarly a *fracture of the body of the talus* impairs the blood supply to the body and *causes avascular necrosis.* (Damage to 'vascular slings') (Figs 3.8A and B).

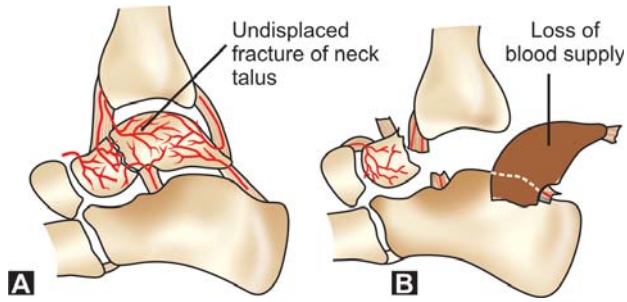
Mechanism of Injury

Talar neck fractures: Extreme dorsi flexion of the ankle or direct axial loading causes rupture of the posterior capsule and impaction of the talar neck against the distal tibia resulting in *a vertical fracture of the neck.* Subsequently one of the two things can happen. Either *the foot subluxates forwards* with talar body adapting a position of equinus *or the body of the talus is pushed backwards out of the ankle mortise* and lies between the medial malleololus and Achilles tendon.



Figures 3.7

Showing blood supply of the talus in coronal plane.



Figures 3.8A and B

Showing that (A) fracture of the neck of the talus alone does not disturb the blood supply without displacement of the body. (B) When the body is displaced the blood supply is lost.

Classification of the Fractures/Dislocations and Fracture—Dislocations of the Talus

Following injuries are seen in the talus

A. Talar neck fracture.
B. Talar body fracture.
C. Talar head fracture (rare).
D. Subtalar dislocation.
E. Total talar dislocation.

Classification of talar neck fracture

Based on Hawkins' classification (1970) it is classified as follows (Figs 3.9 and 3.10)

I. Undisplaced vertical fracture.
II. Displaced fracture with subtalar joint subluxation/ dislocation.
III. Displaced fracture with both subtalar and ankle dislocation.

(Canale and Kelly, proposed a IV Group)

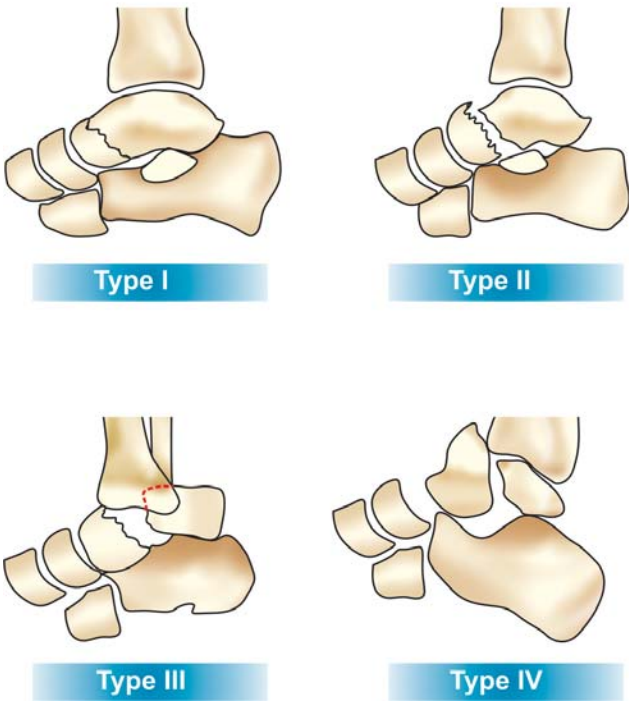


Figure 3.9

Diagrammatic representation of different types of fracture of talus base on Hawkin's classification modified by Canale and Kelly.

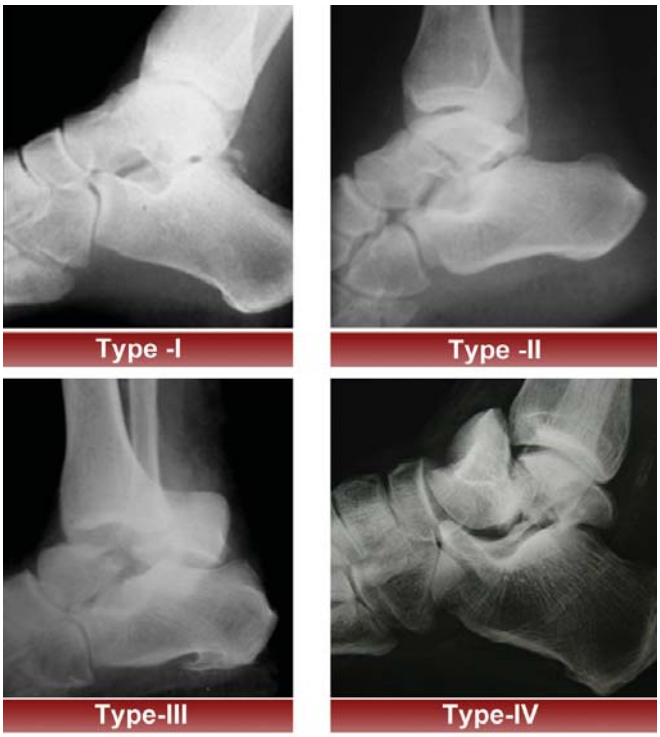


Figure 3.10

Radiographs showing the different fractures based on Hawkins, classification and modification by Canale and Kelly. Type-I: Undisplaced fracture. Type-II: Displaced fracture with subtalar subluxation/dislocation. Type-III: Displaced fracture with both subtalar and ankle subluxation/dislocation. Type- IV: Displaced fracture with subtalar, ankle and talonavicular dislocation.

IV. Displaced fracture with subtalar, ankle and talonavicular dislocation.

Classification of talar body fracture

Based on Sneppen classification (1977) it is classified as follows:

I. Osteochondral dome fracture.
II. Coronal, sagittal, horizontal shear fractures.
III. Posterior process fractures.
IV. Lateral process fractures (Snowboarders fracture)
V. Crush fractures.

Talar head fractures are rare and generally associated with talo-navicular dislocation/subluxation.

Complications

a. Osteochondral fracture may cause loose body inside the joint with persistent pain and limitation of movement.
b. Non-union (Neck of the talus)
c. Avascular necrosis of the body of the talus.
d. Degenerative arthritis of the ankle.

Treatment

A. *Nonoperative:* Undisplaced (displacement less than 1mm) fractures are immobilized in plaster cast.
B. *Operative:*

Internal fixation: Displaced fractures (Type II, Type III, Type IV Neck fractures; Coronal, sagittal and horizontal shear fractures of the body) are openly reduced and internally fixed.

Arthroscopy: Arthroscopic procedures are done for removal of loose body, management of osteochondral lesions and in early degenerative arthritis of ankle.

Arthrodesis: Indicated in secondary complication of non-union, avascular necrosis and degenerative arthritis.

Total joint arthroplasty: Indicated in secondary degenerative arthritis (without AVN).

Talectomy or Astragalectomy: Rarely done as it renders the ankle highly unstable.

Essay Questions

Q. Describe the blood supply of Talus. Discuss the mechanism of injury, diagnosis and management of fracture neck of Talus and its complications.
Q. Classify fractures of the Talus. Discuss the management of the fractures of the Talus. Enumerate their complications.

Calcaneus Fracture

Also known as " Lover's Fracture"and" Don Juan Fracture".

Functional Importance of the Calcaneus

a. It acts as a lever arm for Gastrosoleus Complex.
b. Transmits the body weight.
c. Supports and maintains the lateral column of the foot.

Hence, when its anatomy is not restored after a fracture not only these functions are impaired but also the gait is affected.

Surgical Anatomy

There are three articular facets on the superior surface of the calcaneus

1. The convex posterior facet.
2. The concave middle facet.
3. The concave anterior facet.

The middle facet lies over sustentaculum tali. Between the middle and the posterior facet is the calcaneal groove. The anterior articular surface is saddle shaped and articulates with the cuboid bone.

Hence, those fractures which involve the articular surfaces produce complex articular surface irregularities and need to be managed accurately in order to reduce the functional disability after recovery.

Mechanisms of injury

a. *Axial loading and shear:* Occurs when a person falls from a height and lands on the heel. The Talus is driven down onto the calcaneus causing articular surface fracture there by disturbing the subtalar joint anatomy.
b. *Direct impact by blunt force:* Results in extra articular fractures of the plantar tuberosity and the body.
c. *Twisting injuries:* Results in avulsion fractures of the calcaneus because of the pull of tendons and ligaments. E.g. posterior tuberosity fracture due to the pull of tendo-achillis.

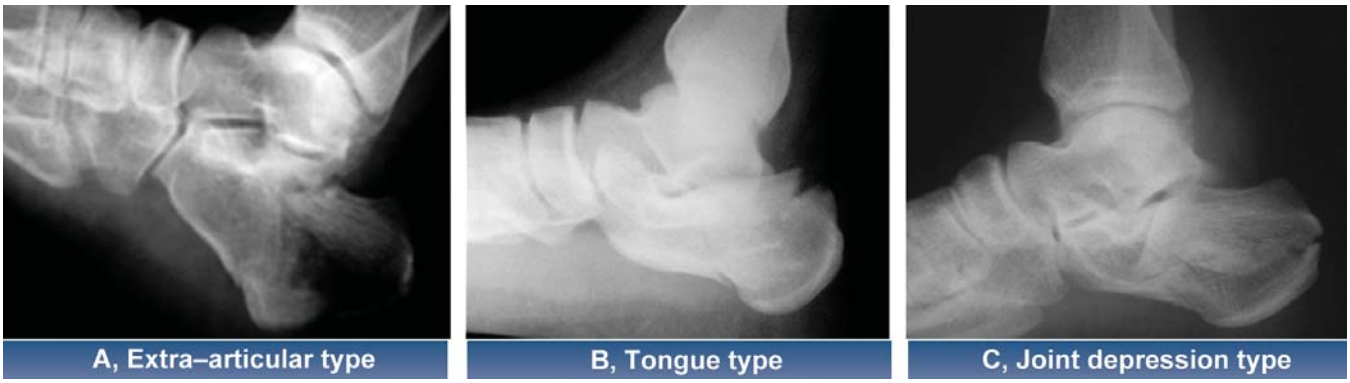
***Note:* When a person presents with calcaneus fracture, always there is a need to examine the pelvis, spine and the other limb especially when there is a history of fall from a height or high velocity injury, to rule out other more serious fractures such as *Fracture of the pelvis and the spine*. Also it is very important to note whether there is developing *Compartmental Syndrome* involving the central compartment of the foot.**

Classification of Fracture Calcaneum

Essex Lopresti Classification (Figs 3.11A to C and 3.12):

a. Extra-articular
b. Intra-articular
   Tongue type.
   Joint depression type.

The calcaneum is subjected to two deforming forces which are transmitted through the talus.



Figures 3.11A to C
Radiographs showing different types of calcaneal fractures based on Essex Lopresti classification.

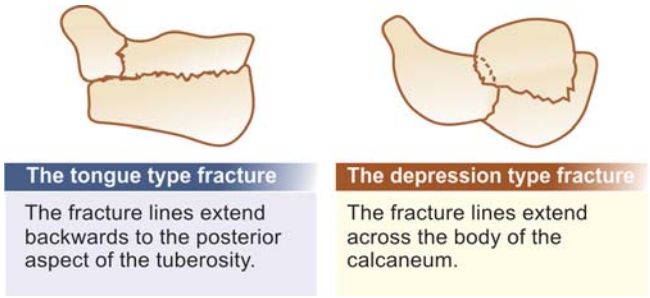


Figure 3.12
Diagrammatic representation of Essex Lopresti classification of fracture involving the joint.

a. Shearing force.
b. Compression force.

As the sharp edge of the lateral process of the talus is driven into the angle of the calcaneus a primary fracture develops extending from the lateral cortex to sustentaculum tali which is a medial structure. This gets sheared from the body of the calcaneus along with the medial 1/3 or 1/2 of the posterior facet of the subtalar joint.

The secondary fracture line that develops as a result of continuation of the deforming force may either be a toungue type or a joint depression type.

Sander's classification: Developed in 1992, based on coronal and axial CT scans of the calcaneus. This classification is useful in the treatment for decision making as well as in the reporting of results. This system defines 4 types of fractures (Fig. 3.13).

Type 1—Undisplaced fractures.
Type 2—2 part split fractures.

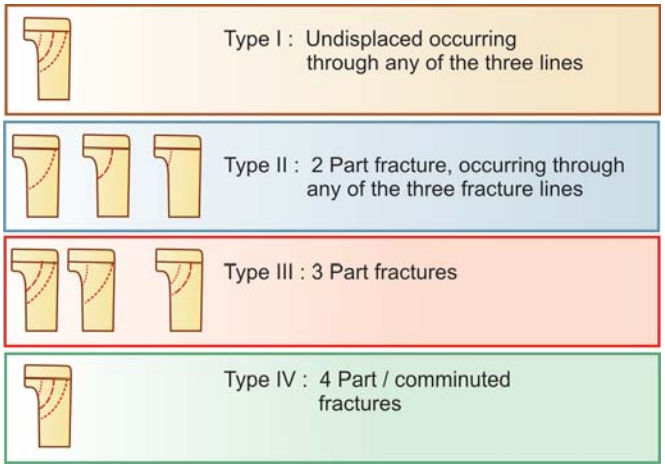


Figure 3.13
Diagrammatic representation of calcaneal fractures based on Sander's classification.

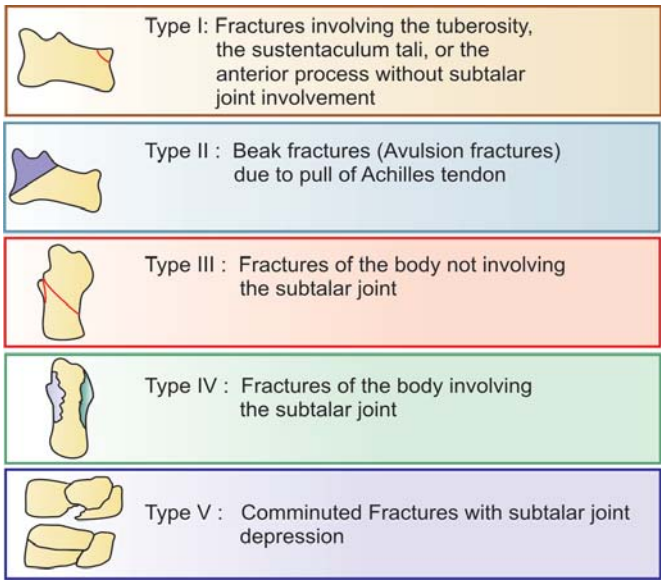


Figure 3.14
Diagrammatic representation of calcaneal fractures based on Rowe classification.

Type 3—Depressed fractures and/or 3 part split fractures.
Type 4—4 part/Comminuted fractures.

Accordingly patients with type 1 injuries do well with non-operative treatment; patients with injuries of types 2 and 3 may be treated effectively with open reduction and internal fixation; and type 4 injuries defy operative reduction.

Rowe Classification

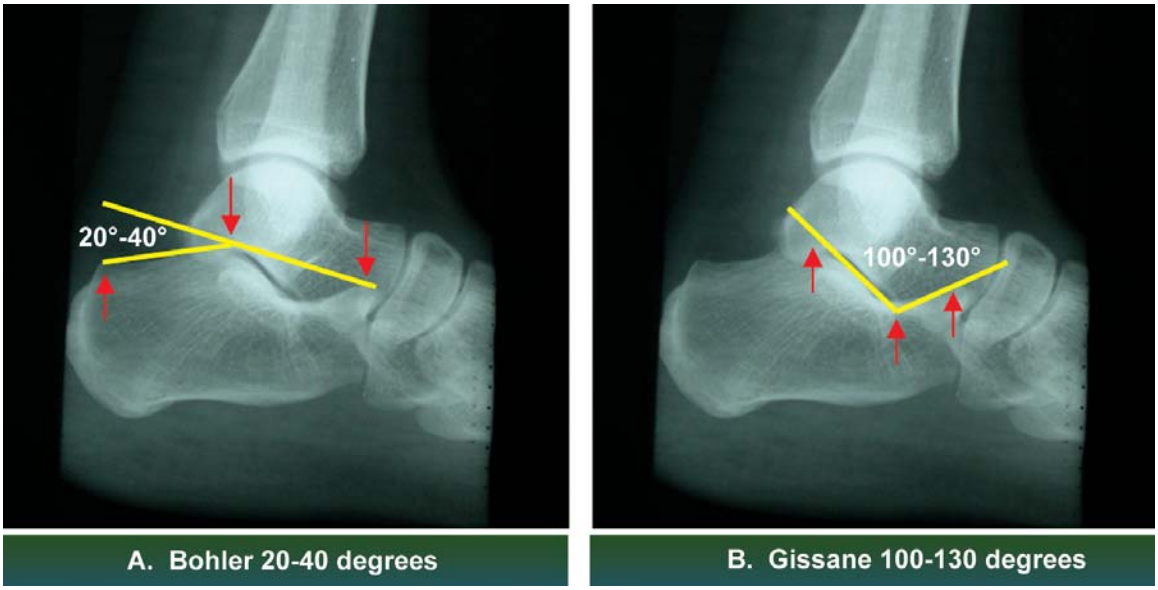
Based on this classification the fracture is classified into 5 types as described (Fig. 3.14).

Type 1a— Tuberosity fracture medial or lateral
Type 1b— Fracture of the sustentaculum tali
Type 1c— Fracture of the anterior process of the calcaneus
Type 2a— Beak fracture of the posterior calcaneus
Type 2b— Avulsion fracture involving the insertion of the tendo-Achillles
Type 3— Oblique fracture of the body not involving the subtalar joint
Type 4— Body fracture involving the subtalar joint
Type 5— Body fracture with subtalar joint depression and comminution.

Radiology in Fracture Calcaneum

Following views are necessary for a detailed evaluation.

- Lateral view—for assessment of 'Bohler tuber angle' and 'Crucial angle of Gissane' (Fig. 3.15).



Figures 3.15A and B

Radiographs showing (A) Normal 'Bohler joint tuber angle' which is 20°-40° It is formed by the lines drawn between the superior margin of the posterior facet to the superior margin of the anterior process and a line drawn from the same point on the superior margin of the posterior facet to the superior margin of the tuberosity. The angle decreases or at times may get reversed in posterior facet collapse. (B) Normal 'Crucial angle of Gissane' which is 100°-130°. It is formed by the lines drawn between the lateral margin of the posterior facet and the anterior process. It increases in posterior facet depression.

- Axial view (Harris view)—for assessment of subtalar joint involvement.
- Anteroposterior view—for assessment of calcaneocuboid joint and avulsion fractures at the anterior and lateral part of the calcaneus.
- Oblique view (Anthonsen view and Borden view)—for assessment of articular extension of fractures.
- CT scan in complex and comminuted fractures for detailed and 3D evaluation.

Treatment

Aim of treatment of fracture calcaneum is to maintain the 'Crucial angle of Gissane' and 'Bohler joint tuber angle'. Maintain the anatomy of calcaneo-cuboid articulation when indicated.

Undisplaced fractures and fractures which do not involve the articular surface are treated nonoperatively by application of well padded bandages, plasters slabs, casts and splints depending on severity of injury and immobilized for a period of 2-4 weeks. This is followed by gradual mobilization.

Displaced fractures and fractures involving articular surface and subtalar joint are treated by anatomical reduction, Closed / Open and internal fixation using ,K- wires or special plates, e.g. H-plates.

Avulsion fractures are treated by closed/ open reduction and internal fixation (Figs 3.16A and B).

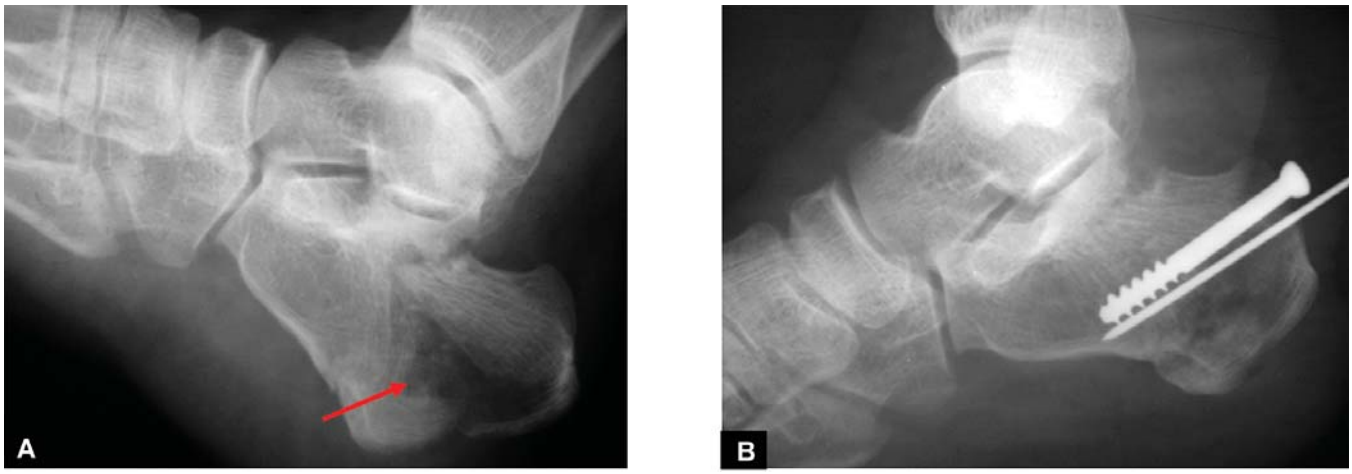
Severely comminuted and complex fractures are generally treated by arthrodesis of subtalar joint (secondary procedure rarely primary).

Revision Questions

1. Discuss briefly the surgical anatomy and the functions of the calcaneum.
2. What is the mechanism of injury of fracture calcaneum?
3. Classify fracture calcaneum.
4. Draw the 'Bohler joint tuber angle' and 'Cruial angle of Gissane' and discuss their clinical importance.
5. How will you proceed with the investigations in a case of fracture calcaneum?

Essay Question

Classify fracture calcaneum. Discuss the mechanism of injury, diagnosis and management of fracture calcaneum. Briefly discuss the prognosis.



Figures 3.16A and B

Radiographs of an extra-articular fracture of the calcaneum being fixed by closed reduction and internal fixation. Note the technique of fixation. A pin is driven through the fractured bone. With the pin as the lever the fracture is manipulated and reduced with the foot in plantar flexion. The wire is driven further through the fracture site and the fracture is firmly transfixed. Next the cancellous screw is passed to achieve firm fixation.

Injuries Around the Ankle

A. Ligament injuries.
B. Bony injuries.

Ligament Injuries

The common injuries are the lateral ligament injury, the medial or the deltoid ligament injury and the syndesmotic ligament injury (the ligament binding the inferior tibiofibular syndesmosis).

- Grade 1—Stretching of the ligaments.
- Grade 2—Partial tear of the ligaments.
- Grade 3—Complete tear of the ligaments.

Management

Complete and severe tears require surgical repair. Less severe, strains and sprains heal well with adequate immobilization. Physiotherapy and gradual mobilization is always essential for a good recovery.

Bony Injuries (Pott's Fracture)

Bony injuries of the ankle are grouped under one heading, i.e **'Pott's Fracture'** because it was Sir Percival Pott (1714- 1788) who first made an attempt to describe the various injury patterns occurring at the ankle.

It was also thought that Sir Percival Pott had this fracture, after he fell down from his horse in the year 1756. The fact is that Sir Percival Pott had a more serious compound fracture of the femur.

Hence, the term is to be considered as a misnomer. Sir Percival Pott, neither described these injuries completely nor suffered from this fracture.

Definition

Pott's fracture is defined as a uni, bi or trimalleolar fracture with or without subluxation or dislocation of the ankle joint.

Points to Remember

Ankle joint is a beautiful hinge joint which *allows* only the movement of ***dorsi-flexion and plantar flexion.*** This movement takes place in a mortise known as ankle mortise formed by medial, lateral and posterior malleolus (posterior 1/3 of the inferior articular surface of the lower tibia is known as posterior malleolus). The ligaments that support the mortise are the deltoid ligament medially, the lateral ligament laterally and the syndesmotic ligament superiorly. (which binds the lower tibia and fibula, i.e. the tibiofibular syndesmosis).

Any other movement occurring in the ankle joint* other than dorsiflexion and plantar flexion *is abnormal* and *tends to injure* the supporting bony and ligamentous structures *leading to deformation of the joint. Failure patterns thus seen are several because the ankle joint is amenable for a variety of abnormal stresses. These *failure patterns are similar* and are reproducible *for a specific type of stress.* This has been

accurately described by *Lauge and Hansen* in their *'Genetic Classification.'* which they derived after experiments on cadavers. They used a wrench and subjected the cadaver ankle to a variety of stresses and documented the results. It was worth noting that a particular type of stress produced a particular failure pattern.

Lauge-Hansen's Genetic Classification

Based on this classification the injuries are classified as follows:

1. Abduction Injuries (pronation abduction)
2. Adduction Injuries (supination-adduction)
3. Pronation External Rotation Injuries.
4. Supination External Rotation Injuries.
5. Vertical Compression Injuries.
6. Unclassifiable.

Note: All the terms used in the classification refer to the (abnormal) movement of the talus in the ankle mortise. If the ligament is stronger than the bone, the bone fractures and if the bone is stronger than the ligament, the ligament ruptures. It is also essential to remember certain 'Keywords' in order to understand the mechanism of ankle injuries.

Keywords
Traction force—Pulling
Compression force—Direct impact
Partial failure—Action of the force is incomplete
Complete failure—Action of the force is complete
Failure pattern—End result
Diastasis—Separation

Pathomechanics

Abduction Injuries: In these, the Talus abducts in the ankle mortise. Abduction occurs in the long axis of the Talus. The foot is in pronation when this occurs. Hence, the term pronation-abduction injuries is used. It exerts traction force on the medial structures and compression force on the lateral structures.

Complete Failure Pattern

Traction on the medial structures causes either a transverse pull-off fracture of the medial malleolus or a rupture of the deltoid ligament (depending on the strength of the ligament and the bone) thus resulting in failure of the medial structures. (medial support)—Stage 1

The talus which is now free of its medial tether abducts further and hits the lateral malleolus thereby exerting compression force on the lateral structures causing oblique or comminuted fracture of the lateral malleolus—Stage 2 (Fig. 3.17).

Adduction injuries: (Mechanism is exactly opposite to that of the abduction injuries). In these, the talus adducts in the ankle mortise. Adduction occurs in the long axis of the Talus. The foot is in supination when this occurs. Hence, the term supination- adduction injuries is used. It exerts traction force on the lateral structures and compression force on the medial structures.

Complete Failure Pattern

Traction on the lateral structures causes either a transverse pull-off fracture of the lateral malleolus or a lateral ligament

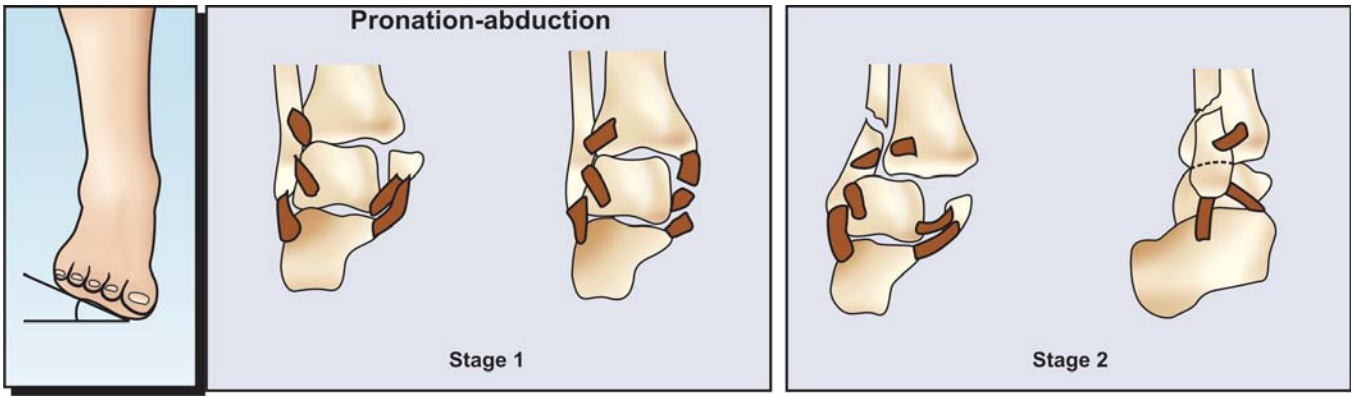


Figure 3.17
Diagrammatic representation of the sequence of events in a pronation-abduction injury.

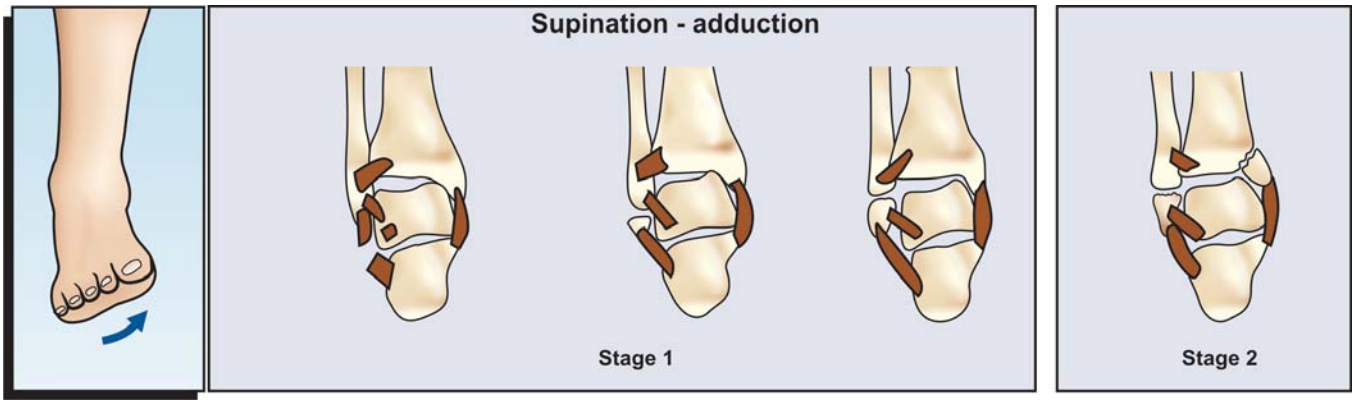


Figure 3.18
Diagrammatic representation of sequence of events occurring in a supination-adduction injury.

rupture (depending on the strength of the ligament and the bone) thus resulting in failure of the lateral structures. (lateral support)—Stage 1.

The Talus which is now free of its lateral tether adducts further and hits the medial malleolus thereby exerting compression force on the medial structures causing oblique fracture of the medial malleolus—Stage 2 (Fig. 3.18).

Complete failure pattern of both Abduction and Adduction injuries is a Bi-malleolar fracture dislocation/subluxation of the ankle joint (except when ligament rupture occurs instead of malleolar fracture), the subluxation being lateral in Abduction injuries and medial in adduction injuries.

Incomplete failure pattern presents either with isolated ligament rupture or uni-malleolar fracture with or without subluxation of the ankle joint.

Pronation–external rotation injuries: In these the talus pronates and rotates externally (Fig. 3.19).

a. *The medial failure* pattern is the same as in abduction injuries, i.e. either there is a medial malleolar fracture or a deltoid ligament rupture. (The medial malleolar fragment is generally smaller when compared to that occurring in abduction injury—Stage 1).
b. *The talus* which is free of its medial tether *springs forwards and out* of the ankle mortise. This exerts *stress on the syndesmotic ligaments* which bind the inferior tibio fibular syndesmosis. The first one to fail is the anterior tibio-fibular ligament followed by interosseous tibiofibular ligament. This failure causes *partial diastasis of inferior tibiofibular syndesmosis*. Thus, the fibula starts rotating on the intact posterior tibiofbular ligament using it as a pivot. The resultant stress on the fibula may be as low as lower 1/3 to as high as fibular neck and failure results in an oblique/spiral fracture which can occur in a similar manner as low as lower 1/3 of the fibula to as high as fibular neck. This is known as *Maisonneuve Injury–Stages 2 and 3.* (Named after *Jules Germain Francois Maisonneuve* who described the mechanism.)
c. At this juncture when partial diastasis has occurred if the *subject is running,* e.g. in an athletic event , the talus is vertically pushed upwards between the tibia and the fibula, rupturing the intact posterior tibiofibular ligament and causing *complete diastasis* of inferior tibiofibular syndesmosis thereby causing a comminuted fracture of the the lower 1/3 of the fibula. This is known as *Dupuytren's fracture* (Fig. 3.20)—Stage 4.

(Named after *Guillaume Dupuytren* who described the mechanism in 1816.)

Tillaux fracture (Named after *Paul Jules Tillaux* who described the fracture in 1848). It is a fracture occurring during the diastasis of inferior tibiofibular syndesmosis. If the strength of the tibiofibular ligament is more than that of the bone there develops an avulsion fracture at the anterior/posterior lip of the inferior fibular facet of the tibia (Anterior *Tillaux Fracture, see diagram marked (Fig. 3.19)—Stage 2 B* and Posterior *Chaput- Tillaux Fracture* described by *Chaput. See diagram marked (Fig. 3.19)—Stage 4 D.*

Complete failure pattern in pronation externl rotation injury is either Maisonneuve or Dupuytren's fracture dislocation.

Incomplete failure pattern presents with only a portion of the sequence of the failure pattern. (Deltoid ligament rupture, Medial malleolar fracture, diastasis of syndesmosis etc).

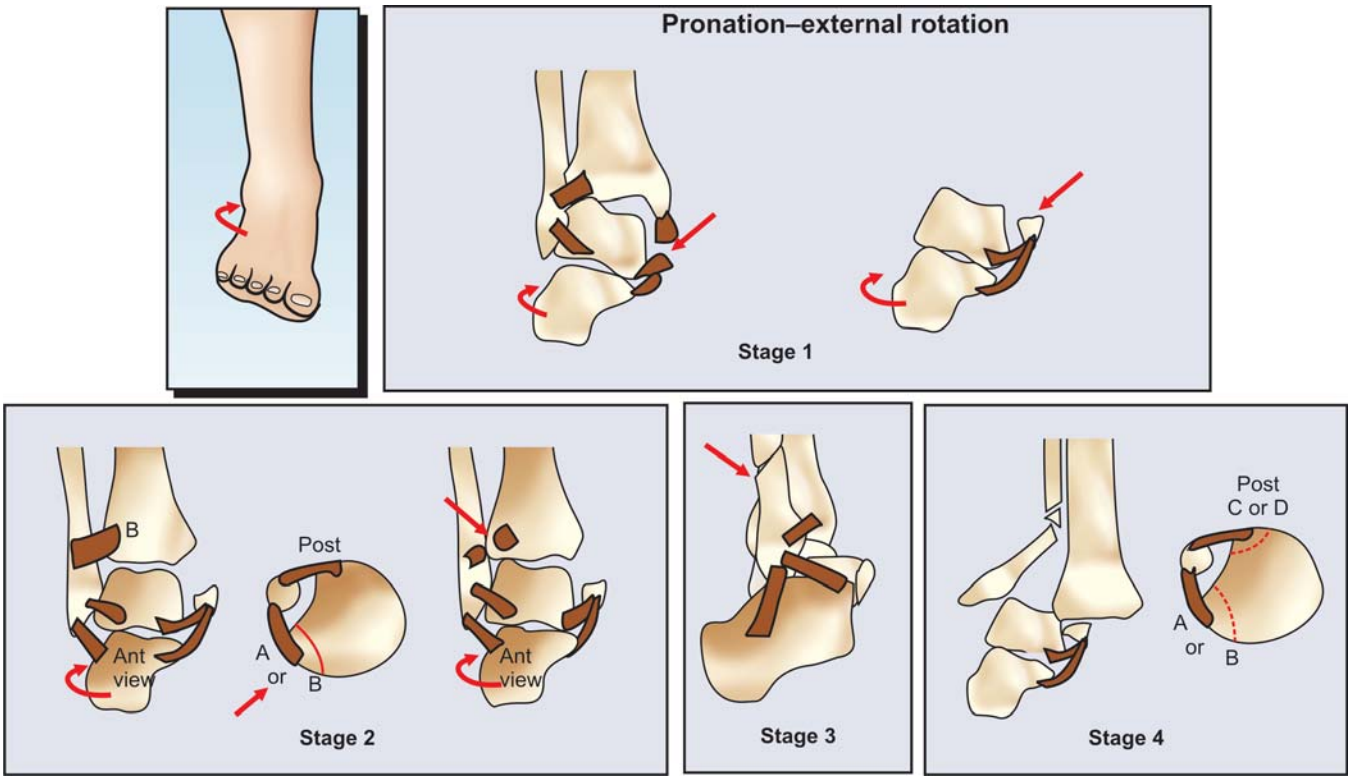


Figure 3.19

Diagrammatic representation of the sequence of events occurring in a pronation-external rotation injury. Stage 2 mark A indicates rupture of anterior tibio fibular ligament and B indicates Anterior Tillaux fracture. Similarly In Stage 4 mark C indicates Posterior tibio fibular ligament rupture and mark D indicates Posterior Tillaux fracture.

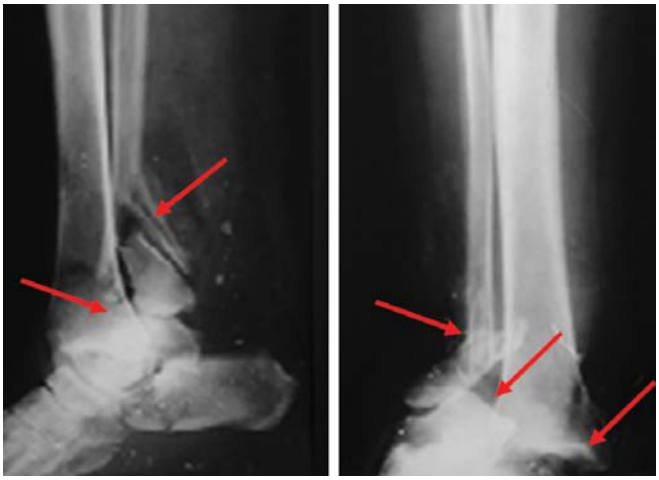


Figure 3.20

Radiographs AP and Lat views showing a Dupuytren's fracture dislocation. Presence of foreign bodies in the X-rays indicate the compound nature of the injury.

Uncommon and variable pattern can present with only rupture of ligaments or a combination of rupture of ligaments and fractures.

Supination–external rotation injuries: In these the Talus supinates and rotates externally. When the *Talus supinates* and *rotates externally,* it *springs backwards and out of the ankle mortise.* Hence, the failure occurs as follows (Fig. 3.21):

a. First structure to fail is the lateral malleolus—Stage 1
b. Next is the posterior malleolus—Stage 2
c. Last is the medial malleolus—Stage 3

The fracture is known as a Tri malleolar fracture or a 'Cotton's fracture'. The direction of the fracture line in these fractures is obliquely upwards and backwards.

Complete failure pattern is a Tri malleolar fracture or a 'Cotton's fracture' described by Cotton F.J. in the year 1915.Incomplete failure pattern may manifest with any of the isolated fractures or any two of them together.

Direction of the fracture line gives the clue.

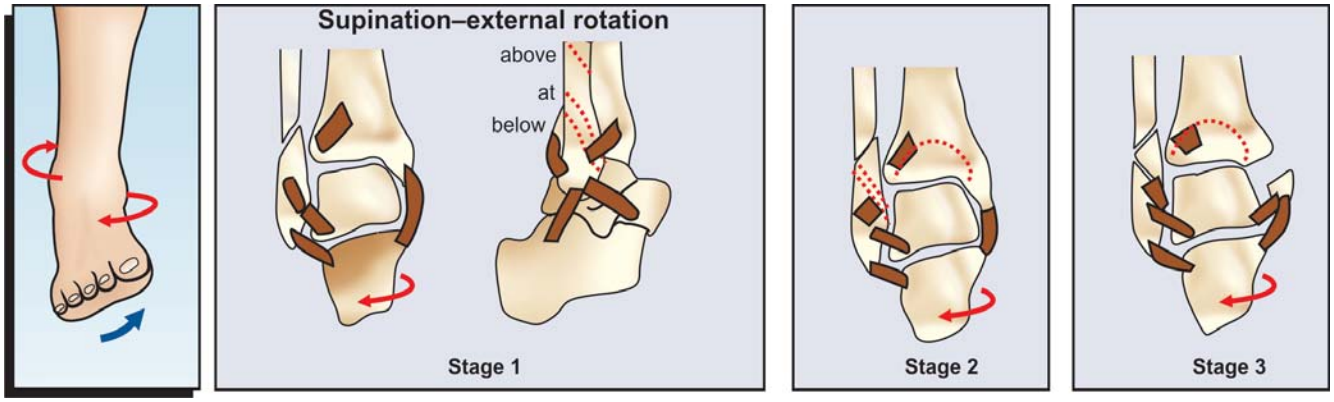


Figure 3.21
Diagrammatic representation of sequence of events occurring in supination-external rotation injury.

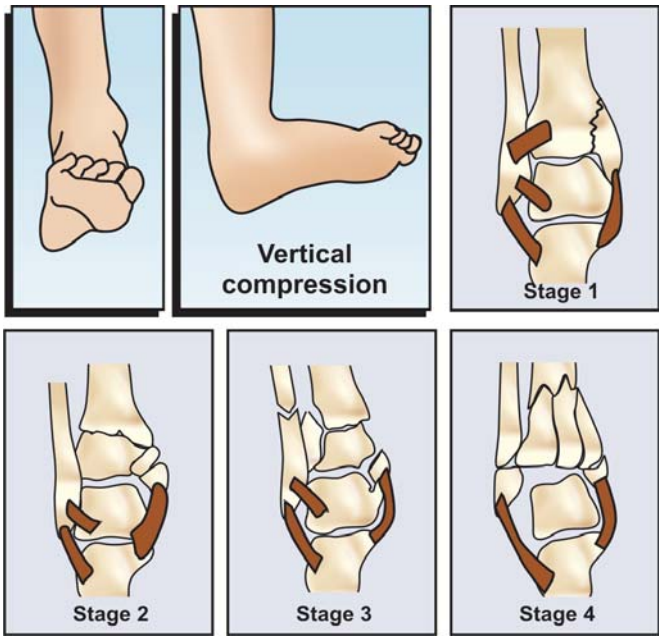


Figure 3.22
Diagrammatic representation of the stages of a vertical compression injury.

Vertical compression injuries: Occurs when a person falls from a height. The talus is violently pushed upwards into the inferior articular surface of the tibia, shattering the articular surface. Extent of disruption is directly proportional to the severity of the impact. The term Pilon fracture is given for these injuries (Fig. 3.22).

Unclassifiable injuries: Nature of injury is unclassifiable because a variety of forces acting simultaneously or in succession have caused the failure (Fig. 3.23).

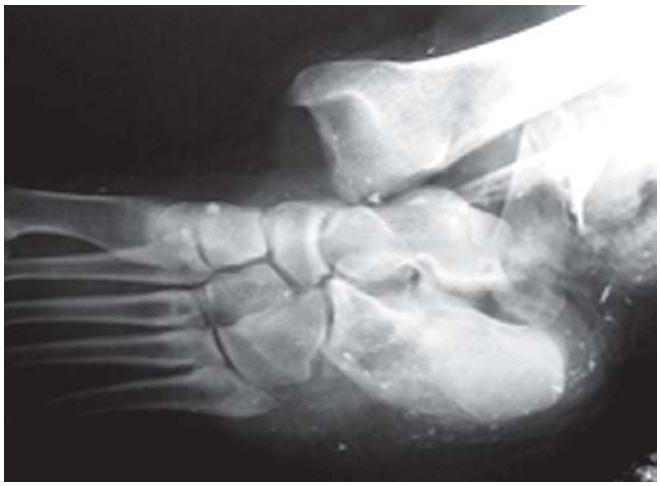


Figure 3.23
Radiograph of an ankle joint involved in a high velocity injury of a road traffic accident. The injury is a compound fracture dislocation. Mechanism is simply unclassifiable.

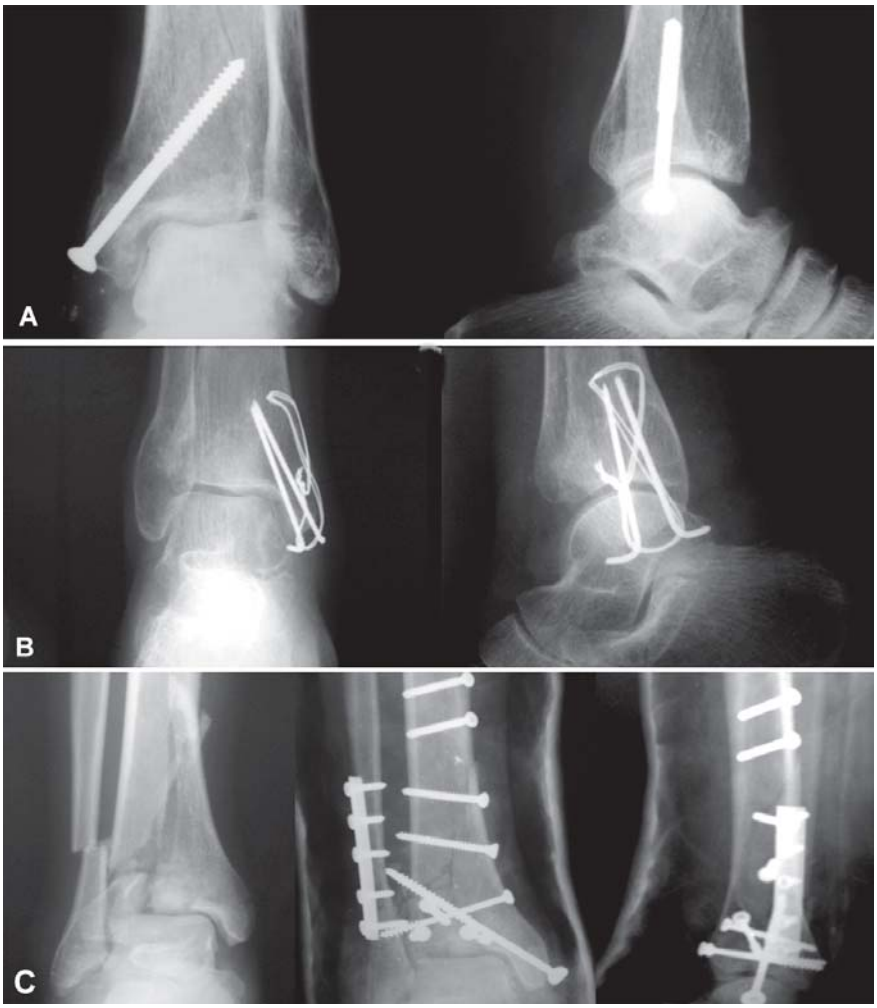
Always these injuries are compound/open injuries resulting in severe morbidity.

Management (Figs 3.24A to C)

Undisplaced and incomplete fractures: They are managed with immobilization in plaster/fibre below knee or above knee cast as per indication.

Displaced fractures: They are managed by open reduction and internal fixation with emphasis on restoration of articular surface congruity.

Mobilization of the joint is to be started at earliest after wound healing.



Figures 3.24A to C

Radiographs showing different methods of fixation of ankle fractures. (A) An isolated fracture of the medial malleolus fixed internally using a malleolar screw. (B) Another malleolar fracture where the malleolar fragment was small. This was fixed using tension band wiring. (C) A vertical compression injury with Pilon fracture internally fixed using malleolar screws, cancellous screws and cortical screws. The fibular fracture is fixed using 1/3 tubular plate and screws.

Implants used: Malleolar screws, cannulated cancellous screws, 1/3 tubular plates, 3.5 mm DCP, rush nails, K-wires and SS wires. (for tension band wiring.)

Biodegradable screw is the latest among the implants.

Complications:

- Nonunion – seen when the injury is neglected.
- Improper restoration of articular congruity by neglect or otherwise (e.g. Pilon fracture) results in painful limitation of movement and degenerative arthritis at a later date.

Treatment:

- Nonunion is treated open reduction internal fixation and Bone grafting.
- Degenerative arthritis is treated by arthrodesis of the ankle joint or total joint replacement.

Revision Questions

1. Define Pott's fracture.
2. What is Maisonneuve injury?
3. What is Dupuytren's fracture?
4. What is Cotton's fracture?
5. What is Tillaux fracture?
6. What are various implants used to fix the fracture dislocation of the ankle?

Essay Question

Discuss the classification, mechanism of injury, diagnosis and management of fracture dislocation of ankle otherwise called as Pott's fracture. Enumerate its complications.

Tibial Diaphyseal Fractures

Almost 1/3 of the tibia is subcutaneous, i.e its anteromedial border (known as shin) and the flat medial surface. Hence, this bone is predisposed for open fractures. The nutrient artery which arises from posterior tibial artery enters the bone just distal to the origin of soleus muscle and runs distally (from the knee I flee.) Hence, chances of delayed union and non union increases especially in more distal fractures because of poor blood supply.

Mechanism of Injury

Direct: This mechanism operates in a high energy trauma, e.g. Road traffic accidents, and penetrating injuries. Open, comminuted, displaced fractures and crush injuries (injury to the bone and soft tissue) are commonly seen.

Indirect: This mechanism operates in a low energy trauma, e.g. slip and fall. The resultant torsional stress causes oblique, spiral and minimally displaced fractures, with minimal soft tissue damage.

The injury is considered as more severe when the fracture pattern is comminuted or spiral than when it is transverse.

Management

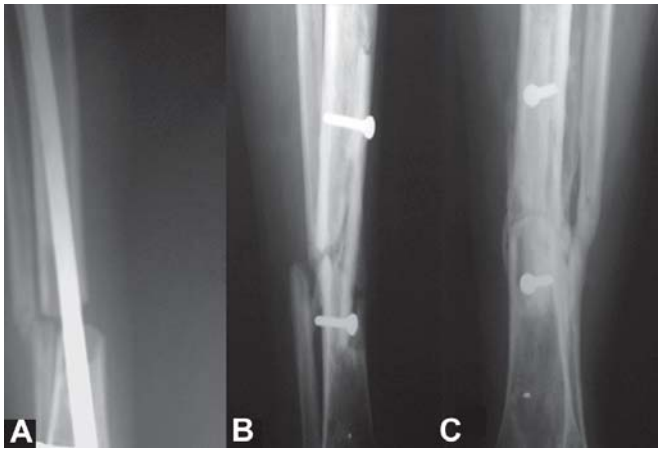
Methods employed are *Nonoperative and Operative.*

Nonoperative Methods

Nonoperative method are used in less severe form of fractures. These are in the form of closed reduction and maintaining the reduction in an above knee plaster of Paris or fibre cast. PTB cast and the Sarmiento technique of cast bracing (Refer) is employed to enhance healing when delayed union is observed at follow up.

Operative Methods

Closed/ Open reduction and internal fixation is the procedure of choice in more severe, displaced, comminuted and segmental fractures. Generally Ender nails, Interlocking nails are the implants that are used. Plates are also used employing the technique of MIPPO (minimally invasive percutaneous plate osteosynthesis). External fixator application after thorough wound debridement is indicated in open fractures to save the limb and facilitate good postoperative wound care and undertake secondary flap coverage procedure.



Figures 3.25A to C

Radiographs showing (A) Type III open fracture with bone loss. Initially stabilized by an intramedullary interlocking nail to save the limb. Remained ununited at the end of three months by which time soft tissue cover had been obtained and infection controlled. (B and C) showing successful treatment by cortico-cancellous grafting using sliding cortical graft from the same tibia to bridge the defect and cancellous grafts from the iliac crest to induce osteogenesis. Note the sound union with adequate new bone formation.

Complications and their Management

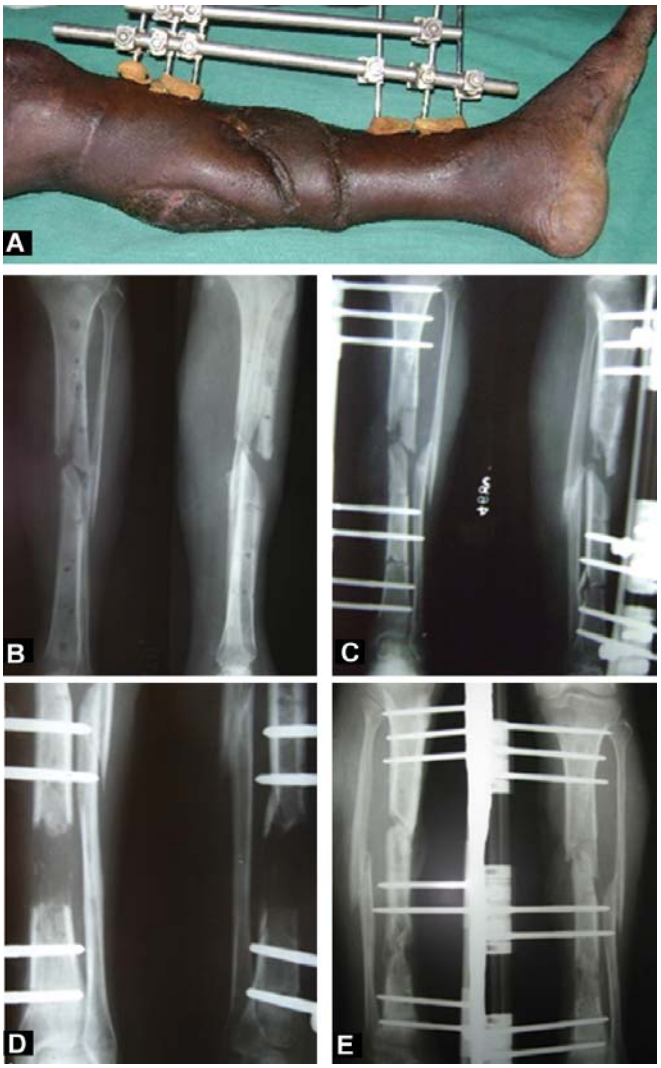
a. Compartmental syndrome.
b. Delayed union.
c. Nonunion.

Compartmental syndrome is managed by immediate adequate decompression of leg compartment along with appropriate skeletal stabilization as per indication. (External/ Internal).

Delayed unions and nonunions are treated by bone grafting techniques (cancellous/cortico-cancellous grafting) along with stabilization by means of internal fixation or immobilization in plaster/ fibre cast (Figs 3.25A to C).

In difficult cases with bone loss and bad scarring, the method of corticotomy and bone transportation is employed using Illizarov or Rail fixator or any other appropriate fixator (Figs 3.26A to E).

Open tibial fractures should always to be converted into closed fractures as early as possible in order to prevent (a) leak of fracture hematoma, (b) devitalization of exposed bone, and (c) secondary infection; thereby facilitating normal healing process and preventing the complication of delayed union, nonunion and osteomyelitis. Many a times, it is not possible to achieve an early soft tissue cover and nonunion and delayed unions are common. This



Figures 3.26A to E

(A) Picture showing the result after sural flap cover for a grade III open fracture. (B) Radiograph showing persistent defect secondary to bone loss. (C to E) Radiographs showing distraction and compression osteogenesis by bone transportation technique using uniplanar fixator. Because of bad scarring over the anteromedial and lateral aspect of the tibia it was decided to employ this technique. Note the successful result (E).

complication is expected in open tibial diaphyseal fractures and is treated accordingly.

Essay Question

Classify open fractures. Discuss the management of an open diaphyseal fracture of the tibia and its complications.

Injuries Around the Knee Joint

Injuries around the the knee joint include:

a. Ligament injuries.
b. Meniscal injuries.
c. Fractures.

Ligament Injuries

The principal ligaments which stabilize the knee joint are the Medial and Lateral collateral ligaments and the Anterior and Posterior cruciate ligaments.

Types of Ligament Disruptions

a. *Medial collateral ligament:* Extends from medial femoral condyle to medial tibial condyle. Has two components: A superficial and a deep. The deep component is attached to the medial meniscus and divided further into the menisco-femoral and menisco-tibial ligament. It is the main stabilizer of the knee in 30° of flexion. It resists valgus stress. Hence, an excessive valgus stress tears or ruptures the ligament (Fig. 3.27).

   Thus, the test employed to diagnose the ruptured medial collateral ligament is known as "Valgus stress test" (Fig. 3.28).

b. *Lateral collateral ligament:* Extends from lateral femoral condyle to the head of the fibula.It is not attached to the lateral meniscus and is separated from it by the tendon of popliteus muscle.

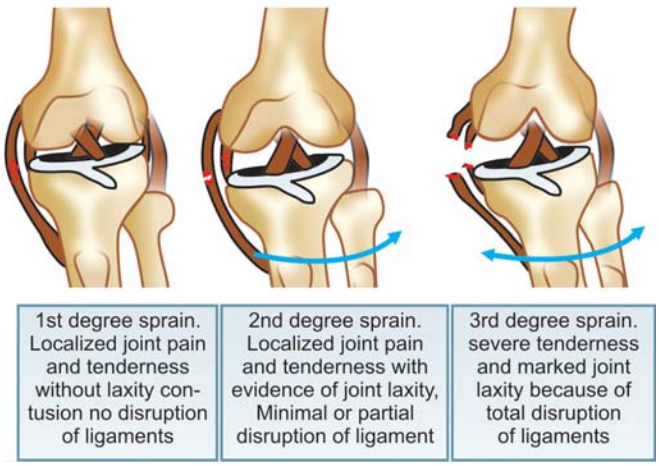


Figure 3.27

Showing the different types of ligament disruptions that can occur following valgus stress.

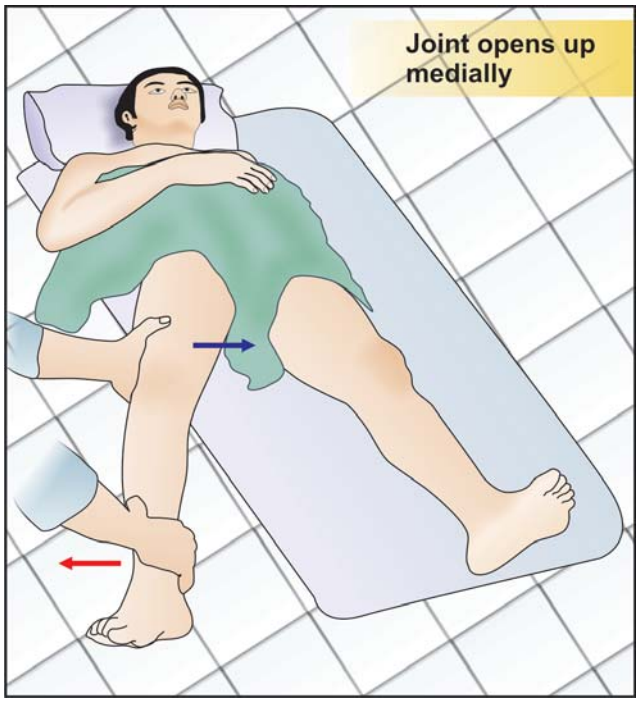


Figure 3.28
Showing the performance of valgus stress test.

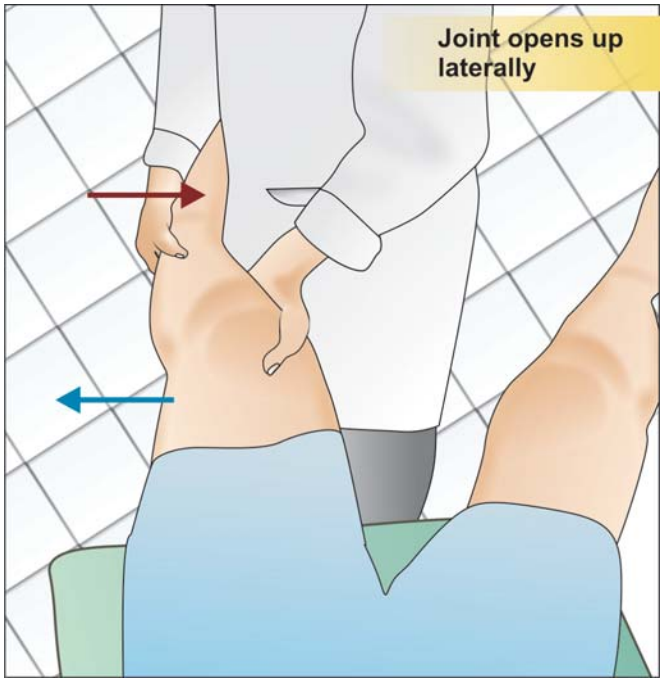


Figure 3.29
Showing performance of a varus stress test.

It resists varus stress. Hence an excessive varus stress tears or ruptures the ligament.

Thus the test employed to diagnose the ruptured lateral collateral ligament is known as " Varus Stress Test" (Fig. 3.29).

c. *Anterior cruciate ligament:* Extends from the anterior intercondylar eminence of the tibial plateu to the inner aspect of the lateral femoral condyle. Because the ligament twists on itself before getting attached to the femur, its anerior fibres are posterior at insertion and posterior fibers are anterior. It is thus divided into two components. The anteromedial and the bulk. The anteromedial portion becomes taut in flexion and the bulk becomes taut in extension thus imparting anterior translational stability during flexion as well as during extension of the knee joint. It also prevents the femur from sliding posteriorly on the tibia, prevents hyperextension of the knee and limits medial rotation when the foot is on the ground. It gets injured when subjected to a twisting force on a hyper-extended knee (Figs 3.30A and B).

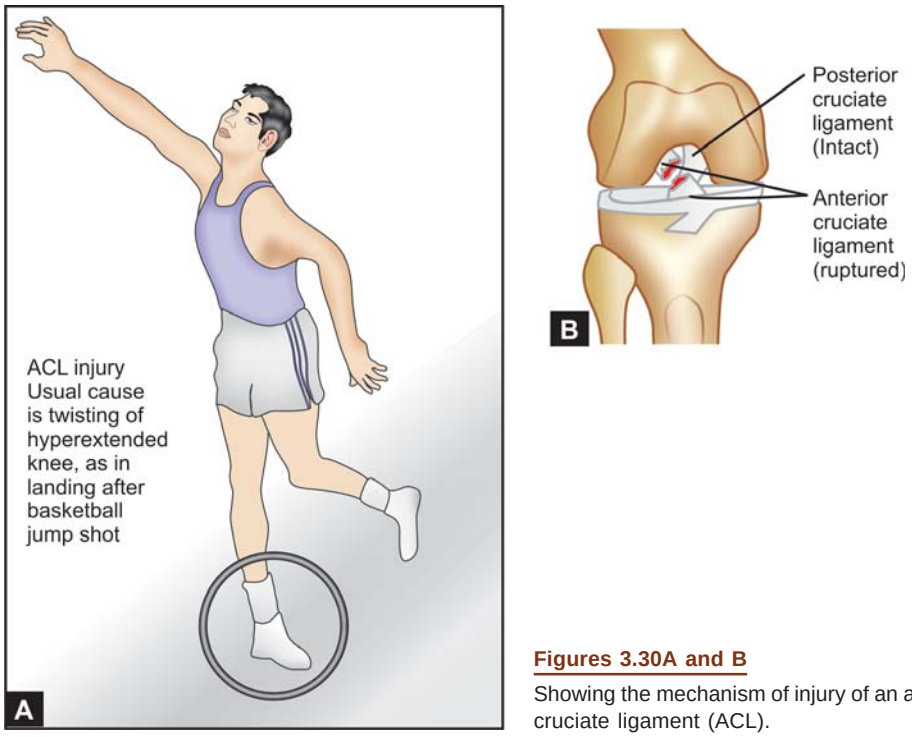
The tests employed to diagnose anterior cruciate ligament injury are the "Anterior Drawer Test" and the "Lachmann Test." They demonstrate anterior translation of the tibia on the femur. Lachmann test is more sensitive. Meniscus may sometimes block the anterior translation of the tibia in Anterior Drawer Test (Figs 3.31 and 3.32).

d. *Posterior cruciate ligament:* Extends from the posterior intercondylar eminence to the inner aspect of the medial femoral condyle. Imparts posterior translational stability during flexion and extension of the knee. It also prevents femur from sliding anteriorly on the tibia and limits lateral rotation when the foot is on the ground. It gets injured when there is a posterior thrust on a hyperextended knee, e.g. stepping into a pot hole or a direct blow on to the upper tibia (Fig 3.33).

The test employed to diagnose the posterior cruciate ligament injury is the "Posterior Drawer Test." It demonstrates posterior translation of the tibia on the femur (Fig. 3.34).

Terrible Triad of O'Donoghue (Unhappy Triad)

It is a combination of injuries of Medial Meniscus, Medial Collateral and Anterior Cruciate Ligaments. Results in a highly unstable knee. Needs highly skilled, professional management (Fig. 3.35).



Figures 3.30A and B

Showing the mechanism of injury of an anterior cruciate ligament (ACL).

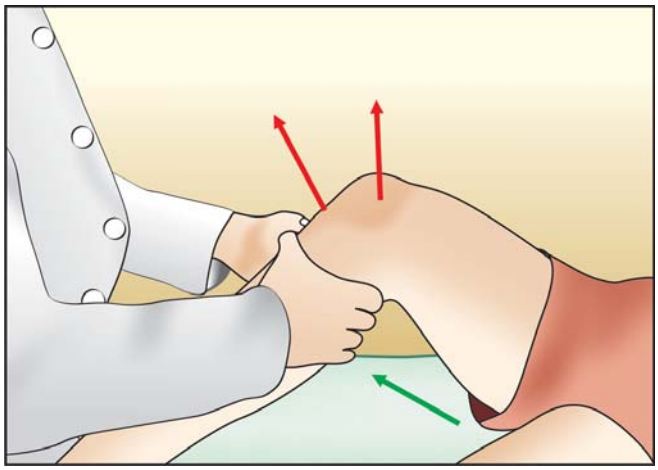


Figure 3.31

Showing the Anterior Drawer Test being performed.

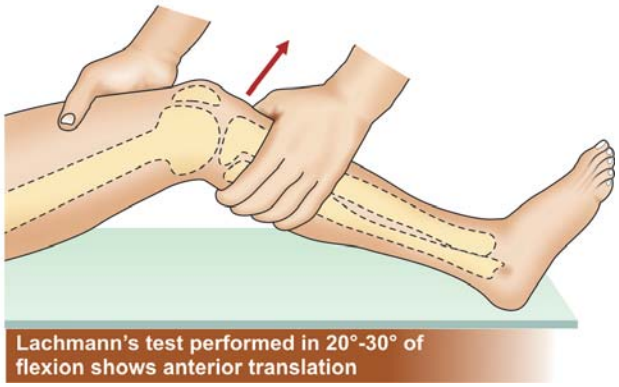


Figure 3.32

Showing the performance of Lachmann's test.

Management of Ligament Injuries

a. *Primary repair:* Repair is done as an immediate procedure within 24-48 hrs after the injury. The limb is immobilized in cast for a period of 3-6 weeks followed by gradual and active mobilization and muscle strengthening exercises.
b. *Secondary reconstruction:* It is done as a secondary procedure. Tendon and fascial grafts are used to substitute the torn ligaments e.g semitendinosus, bone patella bone graft, fascia lata. Some times even artificial ligaments are used. The limb is immobilized in a cast or a knee brace and mobilized gradually.

Both the above procedures can be done by means of an open and arthroscopic assisted techniques depending on the indication and proficiency of the operating surgeon.

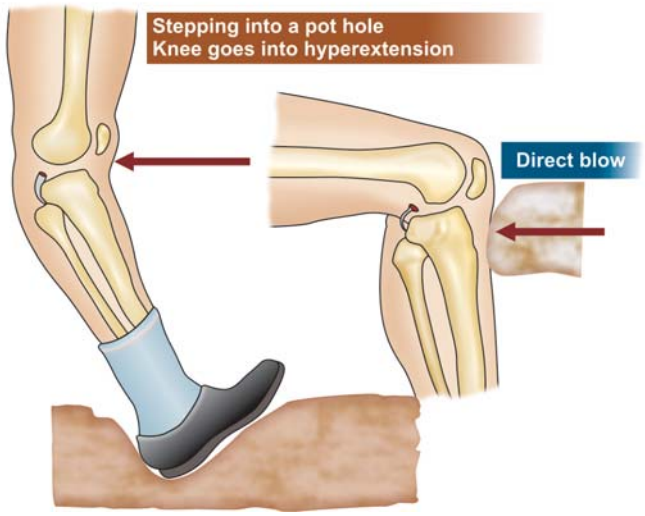


Figure 3.33
Showing mechanism of injury of a posterior cruciate ligament (PCL).

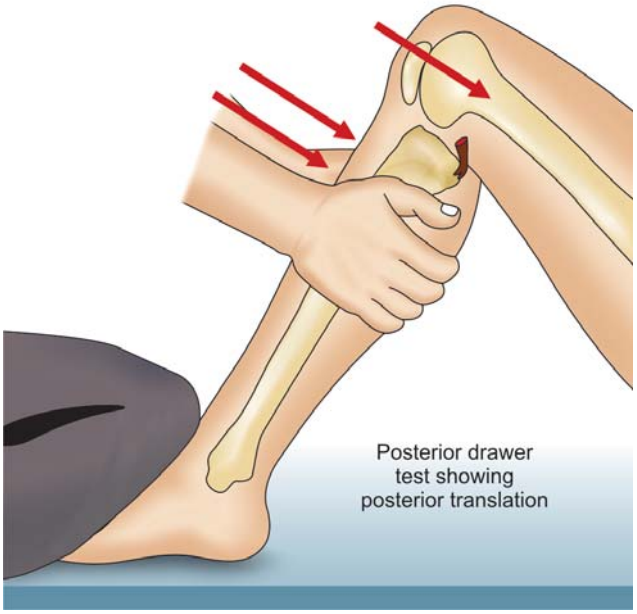


Figure 3.34
Showing the performance of Posterior Drawer Test.

Revision Questions

1. Valgus stress test.
2. Varus stress test.
3. Anterior drawer test.
4. Posterior drawer test.
5. Lachmann test.

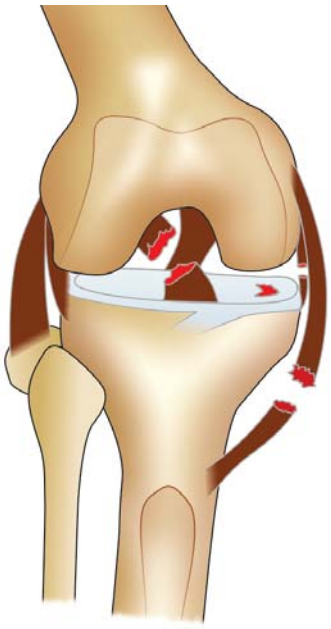


Figure 3.35
Showing the 'terrible/unhappy triad' of O'Donoghue.

Essay Question

Discuss ligament injuries of the knee joint and their management.

Meniscal Injuries

'Meniskos' in Greek means Crescent.

The meniscus is a fibrocartilagenous semilunar (C-shaped) structure situated within the knee joint and is moulded according to the shape of the tibial and femoral condyle, being convex on its undersurface and concave on its upper surface, thicker at periphery and thinner at the center. It is attached to the tibia along its anterior (anterior horn) lateral and posterior (posterior horn) margins but is free in its medial margin. The blood supply to the meniscus is more towards the periphery than at the center and as it approaches the center it thins out. (Hence, peripheral tears heal better when compared to central tears).

Functions of the Meniscus

During the movements of the knee and weight bearing the meniscus:

1. Prevents friction by cushioning effect.
2. Helps in the distribution of synovial fluid.
3. Helps in the distribution body weight.

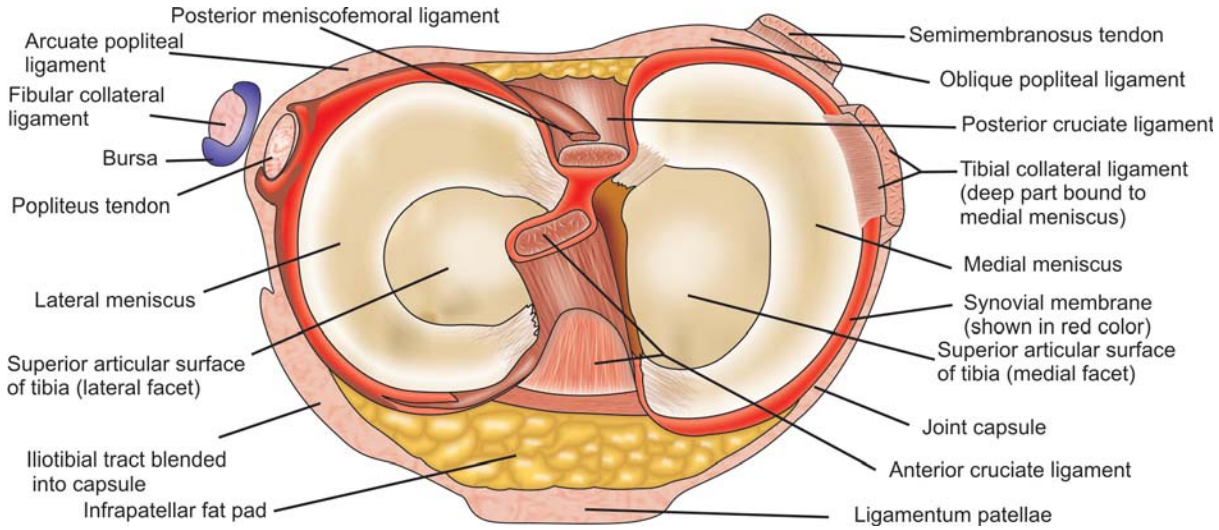


Figure 3.36

Diagrammatic representation of the structures on the tibial plateau.

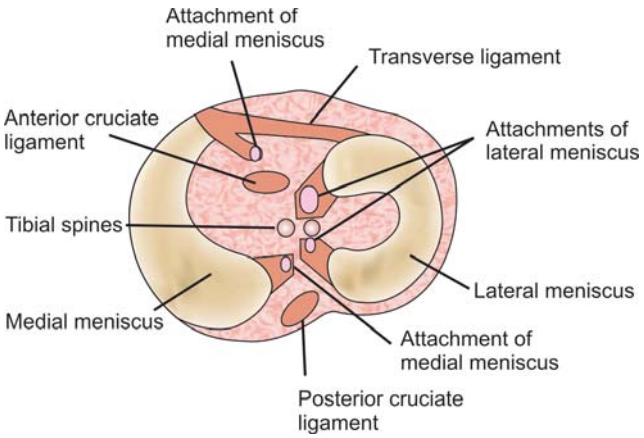


Figure 3.37

Line diagram showing the structures attached to the upper surface of the tibia.

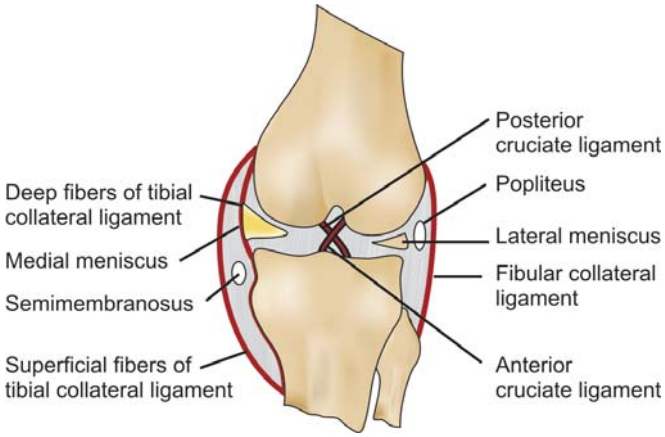


Figure 3.38

Showing the attachment of the deep layer of the medial collateral ligament to the medial meniscus and how the tendon of popliteus is inbetween and separates the lateral meniscus from the lateral ligament.

Thus, loss/removal of the meniscus results in uneven distribution of body weight and predisposes to degenerative arthritis.

An injured meniscus too leads to derangement in the congruity of the joint, interferes with the normal movement and promotes uneven distribution of stress thus promoting degenerative changes even faster.

Medial meniscus* is *larger in diameter, narrower in body and less mobile because it is attached to the deep portion of the medial collateral ligament through the menisco-femoral and menisco-tibial ligaments.

Lateral Meniscus* is *smaller in diameter, wider in body and more mobile because it is not attached to the lateral collateral ligament but separated from it by the tendon of popliteus.

For these reasons, medial meniscus is injured more commonly than the lateral meniscus (Figs 3.36 to 3.38).

Mechanism of Injury

During normal flexion and extension of the knee joint the menisci follow the femoral and the tibial condyle and allow a

smooth gliding. But when there is an abnormal stress of rotation instead of following the condyles they start moving on the condyles. Thus, they are drawn into the center of the joint where they get trapped, pinched and torn. Flexion, Abduction, External rotation stress at the knee causes internal rotation of femur on the tibia and draws the medial meniscus into the center and injures it. The reverse, i.e. Flexion, Adduction, Internal rotation stress causes external rotation of femur on the tibia and draws the lateral meniscus into the center of the joint and injures it. The site of tear is related to the degree of flexion. In more flexion, more posterior part of the meniscus (posterior horn) is involved. In more extension (lesser degrees of flexion) more anterior part is involved. Thus, the tear begins posteriorly and extends from posterior to anterior direction as the knee extends from a flexed position.

Types of tear (Fig. 3.39)

A. Longitudinal tear – May proceed to become a 'Bucket handle tear'.
B. Radial tear – May proceed to become a 'Parrot beak tear'.
C. Horizontal tear – May proceed to become a 'Flap tear'.
D. Combined tear – A combination of different tears, generally seen in a degenerative meniscus (in elderly).

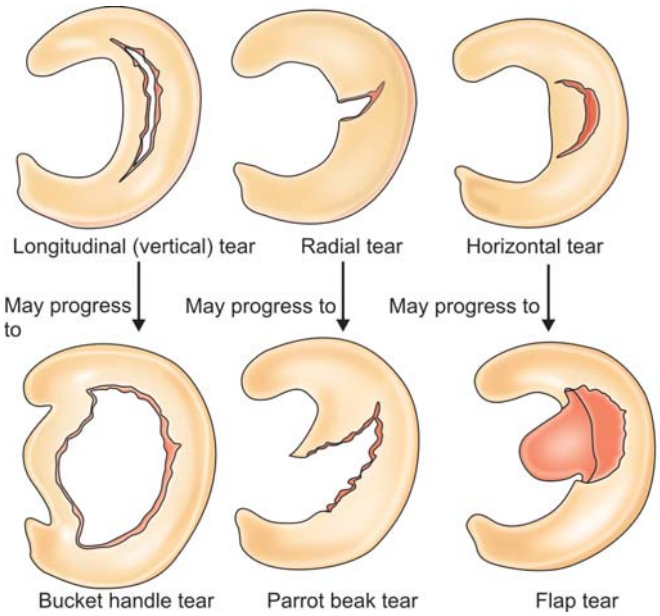


Figure 3.39
Showing different types of meniscal tears.

Diagnosis

Signs and symptoms

a. Constant pain in the knee aggravated by movement.
b. Tenderness in the joint line. (Medial/Lateral)
c. Swelling when effusion is present.
d. Sensation of giving way.
(Other causes for Sensation of giving way are Loose bodies, Chondromalacia patella, Weakness of quadriceps, Instability due to ligament injury.)
e. Pathological locking. This is seen only in 'Bucket handle tear'. Pseudo locking is seen in acute injuries of the knee with hemarthrosis.

Note: Sensation of giving way and pathological locking occurs when the torn portion of the meniscus comes in contact with the articular surface of the femur during movement.

Clinical Diagnostic Tests

1. *McMurray's test*
   a. For medial meniscus: With the patient lying supine on the examination table the *knee is completely flexed.* To check the medial meniscus the examiner *palpates the posteromedial joint with one hand and holds the foot with the other. The leg is then externally rotated and the knee is gradually extended. A click is appreciated* when the torn portion of the medial meniscus comes in contact with the femur.
   b. For lateral meniscus: With the patient lying supine on the examination table the *knee is completely flexed.* To check the lateral meniscus the examiner *palpates the posterolateral joint with one hand and holds the foot with the other. The leg is then internally rotated and the knee is gradually extended. A click is appreciated* when the torn portion of the lateral meniscus comes in contact with the femur.

   The Medial meniscus is more commonly injured than the lateral meniscus. The click appreciated is not only felt but can be audible at times. The tear of the posterior portion of the meniscus produces a click in 0°-90°of movement as the knee is extended from a completely flexed position. The tear involving the middle and anterior portion of the meniscus produces a click beyond 90° of movement.

2. *Apley's grinding test*: With the patient prone the knee is flexed to 90° and the thigh is fixed against the examination table. The leg is then held, pulled upwards and a rotational strain is given. When ligaments are torn this part of the test is painful.

Next the leg is pushed downwards and rotated while the joint is slowly flexed and extended. When the meniscus is injured, pain is observed in the joint.

3. *Squat test*: This test is performed by asking the patient to take full squats with the leg and the feet together, alternately in internal and external rotation. Pain in the joint in internally rotated position (external rotation of femur on the tibia) suggests injury to the lateral meniscus and pain in externally rotated position (internal rotation of femur on the tibia) suggests injury to the medial meniscus.

Note: Presence of pain and click are diagnostic of meniscal injury. But absence doesn't rule out tears of the meniscus.

Investigations

a. *Plain X-ray:* It is done mainly to identify associated fractures. It is useful in long standing injuries to look for degenerative changes in the knee joint.
b. *Diagnostic arthroscopy:* It is an ideal and confirmatory investigation. Other associated injuries, e.g. osteochondral fractures are also identified. (If necessary to be followed immediately with arthroscopic surgery in the same sitting as this is an invasive investigation and is done under anesthesia.)
c. *MRI:* It is a noninvasive procedure. It needs a good sensitive machine as well as accurate interpretation to avoid false-negative and false positive reports.
d. *Arthrography:* After the advent of arthroscopy, arthrography has a very limited role and not done routinely.

Management

Absence of meniscus does not compromise the function of the knee markedly. It only leads to late degenerative changes. But an injured/ torn meniscus severely compromises the function of the knee and as this is directly proportional to the severity of injury needs immediate attention.

When possible the torn meniscus is repaired, e.g. smaller tears. The procedure is known as *meniscorraphy.*

When repair is not possible the torn meniscus is excised either partially, e.g. in larger tears involving posterior horn, bucket handle tear, etc. or completely, e.g. in tears with a degenerate meniscus, complete tear, etc. The procedure is known as *meniscectomy. (partial/complete.).* It is usually done by *arthroscopic surgery.* It can also be done by *open surgical methods.*

Current research is towards meniscal replacement using meniscus obtained either from a brain dead cadaver or using an artificial meniscus.

Revision Questions

1. McMurrays test.
2. Apley's grinding test.
3. Discuss the mechanism of meniscal injury.

Essay Question

1. Discuss the anatomy of the knee joint. Classify the meniscal injury and discuss its management .
2. Discuss meniscal injury with reference to the mechanism, classification, diagnosis and management.

Fracture of the Patella

General Information

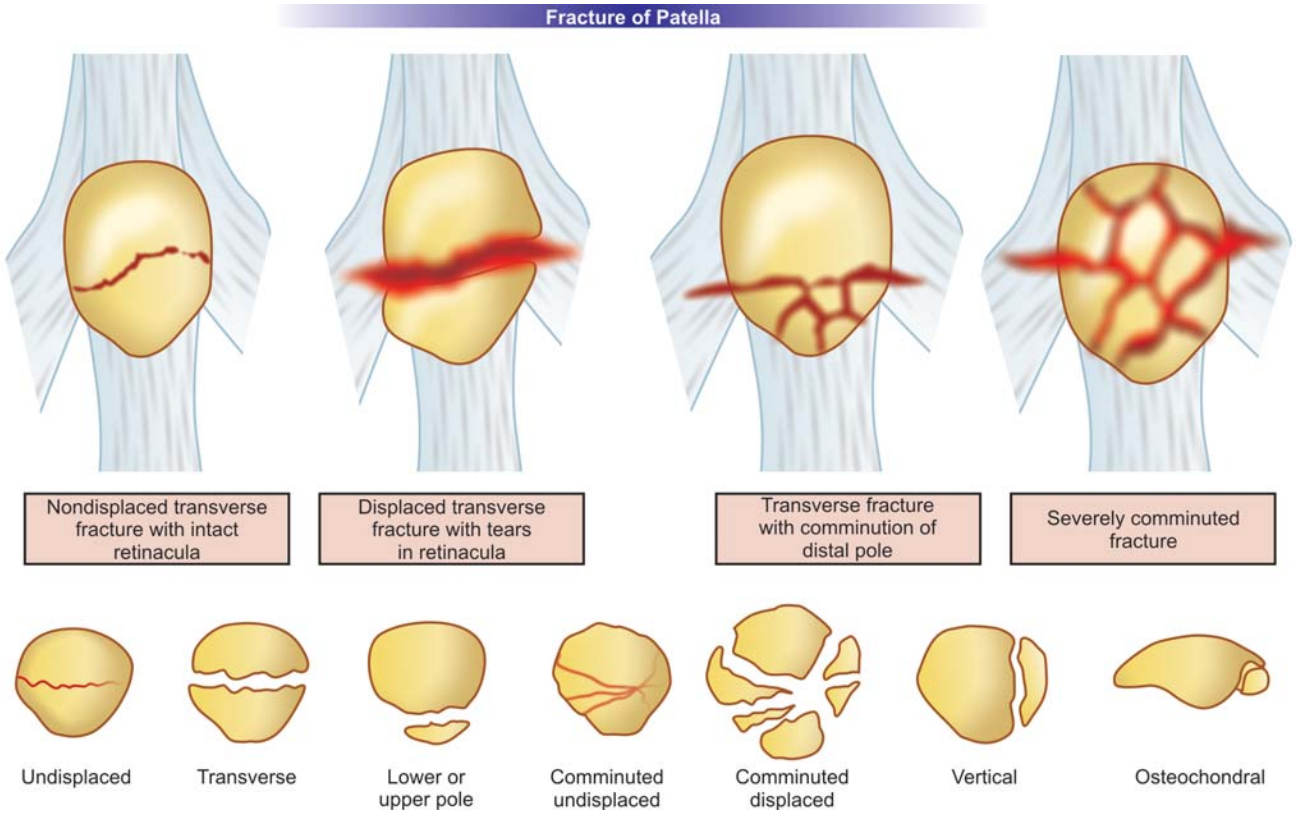
Patella is a sesamoid bone within the quadriceps muscle. It was thought to be a *phylogenetically inherited bone without any function. (Brooke-1937).* This theory made the people to remove the fractured patella and patellectomy became the treatment of choice until *Haxton (1945) proved beyond doubt that patella certainly has a definite function.* He quoted the *Law of von Schwann* which says that the *tension of contraction decreases as the muscle fiber shortens.* This is true at the elbow and for triceps. Thus a boxer delivers a punch in flexion to generate maximum power. *But this is not true at the knee and for quadriceps.* A football player kicks the ball with the knee in full extension (and not in flexion) to generate maximum power. *Thus quadriceps behaves against the Law of Von Schwann* and the muscle power (tension) in the quadriceps increases as the muscle fibre shortens. *The patella acts as the fulcrum and augments the power of the quadriceps and increases it. This is the function of the patella.* Much of this power is lost after *patellectomy* and quadriceps behaves like triceps in accordance with the Law of Von Schwann. This *results in quadriceps lag* (Quadriceps stops short of terminal 5° of extension.)

Mechanism of Injury (Fig. 3.40)

a. *Direct:* In this there is *direct impact on the patella,* e.g. dash board injury, direct blow in assault.

The type of fracture is a comminuted (shattered into pieces) fracture . The classical variety is known as a '*Stellate fracture.*'

b. *Indirect:* In this mechanism the force acts indirectly and causes the fracture, e.g. slipping while walking. When a



Figures 3.40A and B

Showing different types of fracture patella.

person *slips on a slippery surface* one limb is off the ground and the limb which is on the ground goes into progressive flexion at the knee. If this flexion continues it results in a fall. Thus it is the *reflex mechanism that operates to avoid a fall* wherein the flexed *limb tries to straighten out* and maintain balance. This *straightening occurs against gravity and the body weight*. Thus patella breaks. *The type of fracture* that occurs *is transverse* in nature.

c. *Combined mechanism:* The major fragment remains non-comminuted and the fracture is transverse and the other fragment is comminuted (minor fragment).
d. *Avulsion:* It occurs due to the pull of quadriceps muscle or patellar tendon and results in an isolated fracture of the superior or inferior pole.

Note: Whenever a comminution is observed it suggests that a direct mechanism has operated in causing a fracture and vice versa.

Clinical Signs and Symptoms

1. All the classical signs of a fracture are present.
2. Presence of hemarthrosis, resulting in acute swelling.
3. Broken patellar fragments can be felt by the palpating hand.
4. Hand can be insinuated between the fragments and palpation of femoral condyle is possible.
5. Loss of active extension at the knee (suggesting injury to the quadriceps mechanism).

Investigation

X-ray of the Knee joint, standard AP and Lat views give accurate diagnosis .

Management

Patellectomy: This procedure is to be considered as the treatment of yesterday. *Rarely done these days.* Only indication—perhaps is in elderly patients with osteoporotic bone, severe

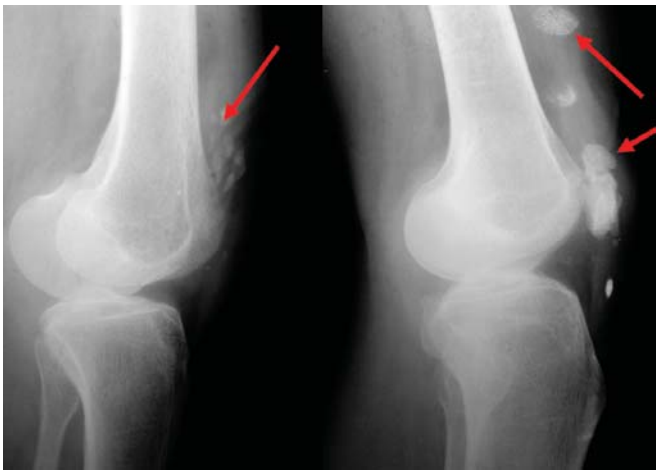
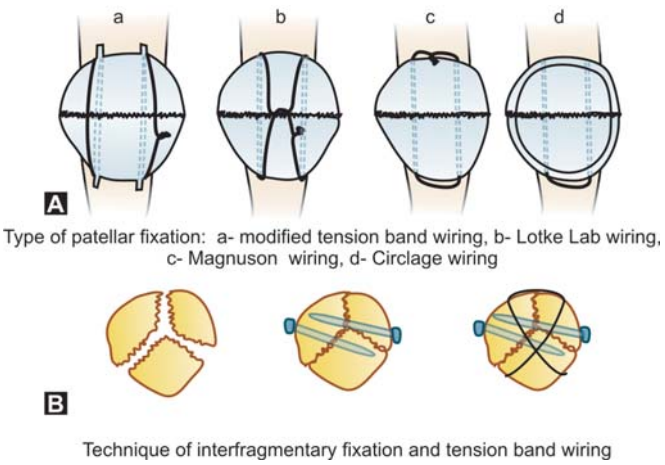


Figure 3.41

Radiographs showing pathologic ossification, a complication of patellectomy.



Figures 3.42A and B

Showing some of the techniques employed in the fixation of fracture patella.

degenerative changes in the joint and the fracture is badly comminuted. *Never done in the young.*

Disadvantages

a. Normal protection to the knee is lost.
b. Quadriceps lag develops.
c. Permanent atrophy of the quadriceps.
d. Prolonged immobilization.
e. Pathologic ossification (Fig. 3.41).

Open reduction and internal fixation:
This procedure is the treatment of choice. Methods used consist of (Figs 3.42A and B)—

1. Circlage wiring of Martin.
2. Figure of 8 tension band wiring.
3. Interfragmentary screw fixation combined with one of the above techniques (Figs 3.43 and 3.44).

Advantages

a. Normal protection to the knee is preserved.
b. No quadriceps lag.
c. No permanent atrophy of quadriceps.
d. No prolonged immobilization.
e. No pathologic ossification is possible.

Disadvantage

a. Possibility of a late patellofemoral osteoarthritis (rare).

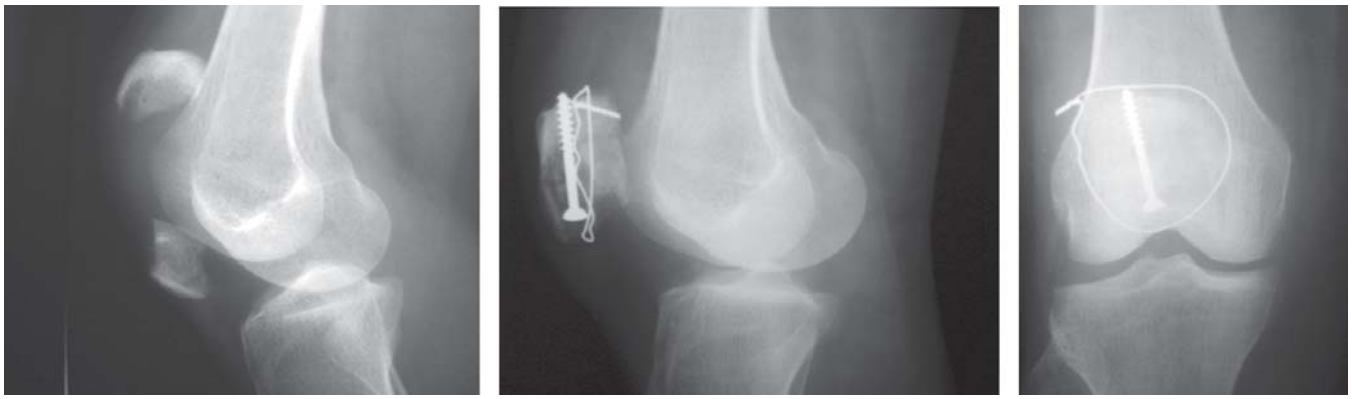


Figure 3.43

Radiographs of a Transverse Fracture Patella fixed by a single Cancellous screw and SS Circlage Wiring. Note the displacement of the fractured proximal fragment because of the pull of the Quadriceps muscle.

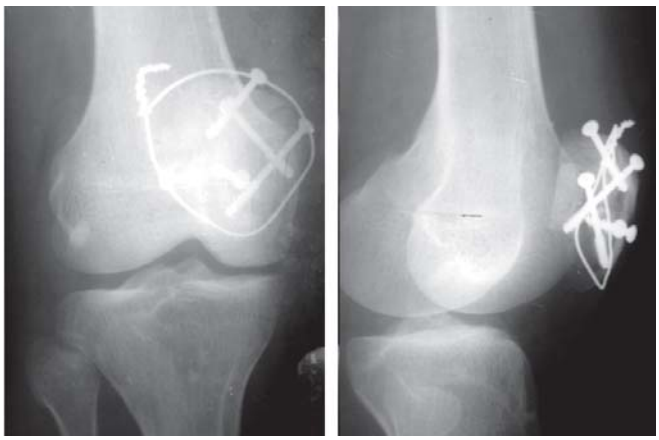


Figure 3.44

Radiographs AP and Lat view showing a Stellate Fracture Patella internally fixed with the help of multiple Inter-fragmentary screws and Circlage Wiring.

Revision Questions

1. Stellate fracture.
2. Patellectomy.

Essay Question

Discuss the function of the patella. Describe the mechanism of injury of Fracture Patella and discuss its management.

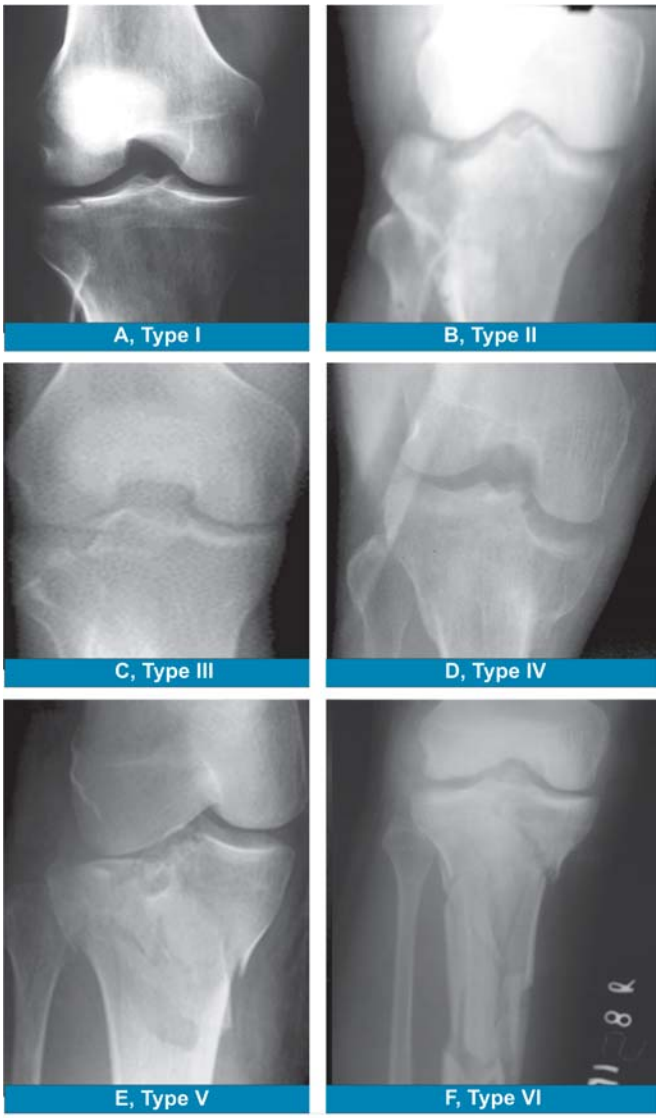
Fractures of the Tibial Condyle (Tibial Plateau)

General Information

Tibial plateau fractures commonly occur as a result of axial loading. They are known as bumper or fender fractures because they occur as a result of an impact of the bumper on the knee in a road traffic accident. They are often associated with ligamentous disruption of the knee. Also injury to the nerves and vessels and compartment syndrome is quite common with these injuries. Hence they demand a careful evaluation and efficient management. Being articular fracture secondary degenerative changes are common and osteoarthritis may develop at a later period however well the fracture is treated.

Mechanism of Injury

These fractures occur as a result of a direct impact onto the condyles of the tibia from the side, e.g. a vehicle striking the pedestrian. As a result of this the knee is subjected to a severe varus or a valgus stress when there is axial loading. The tibial condyle is split or crushed by the opposing medial / lateral condyle of the femur.



Figures 3.45A to F

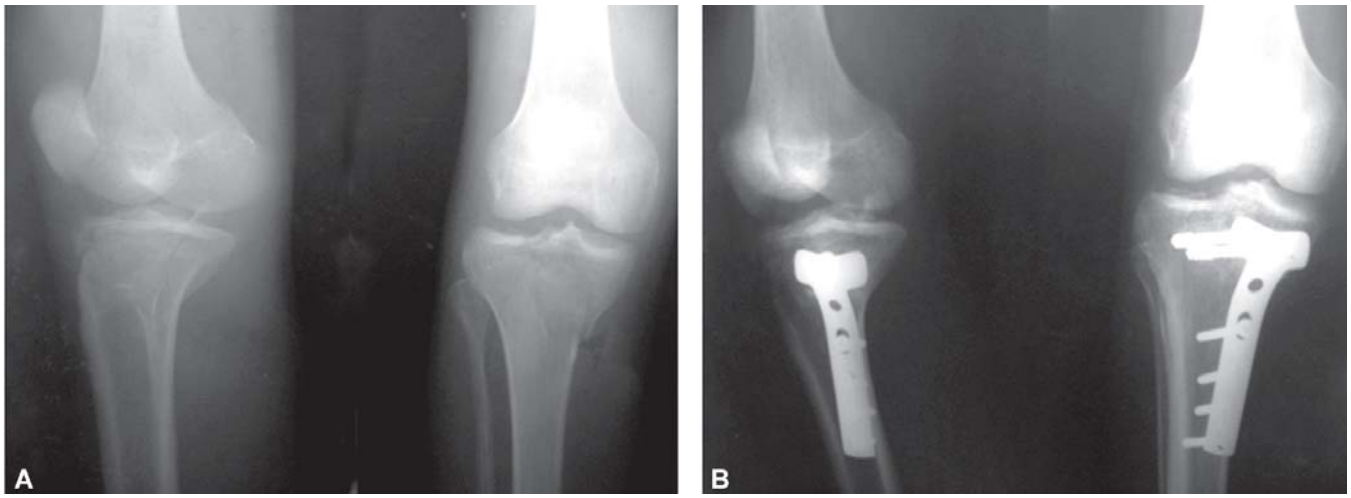
Radiographs showing classification of tibial plateau fractures based on Schatzker. (A) Type I–A cleavage or split. (B) Type II–Clevage or split with depression (C) Type III–Pure central depression. (D) Type IV–Medial condyle fracture. (E) Type V–Bicondylar fracture (F) Type VI–Associated with metphyseal and diaphyseal fractures.

Classification

Based on Schatzker classification—Tibial plateau fractures are classified into VI types (Figs 3.45A to F).

Clinical Signs and Symptoms

a. All the signs of a fracture are present.
b. In addition knee may be swollen due to hemarthrosis.
c. Instability at the knee may be present.



Figures 3.46A and B

Radiographs showing a Schatzker Type IV fracture of the medial condyle fixed internally using a 'T' buttress plate.

Diagnosis

An X-ray gives the diagnosis of these fractures. Complex fractures demand detailed evaluation by CT with 3D reconstruction and MRI which reveals the exact nature of bony and soft tissue injury, respectively.

Treatment

Aim of the treatment: The treatment aims at maintaining the articular surface alignment and achieving good union at the earliest to restore the joint function. Hence demands accurate reduction and fixation. Surgery is the treatment of choice to achieve this goal.

When defect is observed at reduction bone grafts are used to bridge the same, especially in those fractures with depression and extensive comminution.

Nonoperative treatment:

a. Immobilization in plaster casts: This is instituted only in minor degrees of fractures which are undisplaced and the articular surface involvement is not extensive.
b. Low tibial traction. This is an excellent alternative method. Is indicated when the skin condition or the general condition of the patient does not permit surgical intervention, e.g. blistering and threatened compartmental syndrome, associated head injury, etc.

Operative:

a. Closed manipulation, reduction and fixation using cannulated cancellous screws only for less severe fractures; cannulated cancellous screws along with plates and screws for more severe fractures. Plates fixation may be done under C-arm image intensifier guidance, e.g. MIPPO technique.
b. Open reduction and reconstruction of articular surface, internal fixation and repair of soft tissue injury. At times bone grafting may be necessary (Figs 3.46A and B).

Complications

a. Compartment syndrome.
b. Injury to the Popliteal vessels.
c. Injury to the lateral Popliteal nerve.
d. Skin necrosis.
e. Malunion with deformity and restricted joint movement.
f. Secondary degenerative arthritis.

Essay Question

Classify Tibial plateau fractures. Discus the mechanism of injury, diagnosis, management and the complications of tibial plateau fractures.

Fractures of the Femoral Condyles

Fracture involving the femoral condyles consists of–

a. Those extra-articular fractures which occur just above the condyles and hence are known as Supracondylar.
b. Those intra-articulr fractures which involve the medial or the lateral condyle and hence are known as Unicondylar fractures.
c. Those fractures which split the condyles and hence are known as Intercondylar fractures. The split may occur in the form of 'T'or 'Y'.

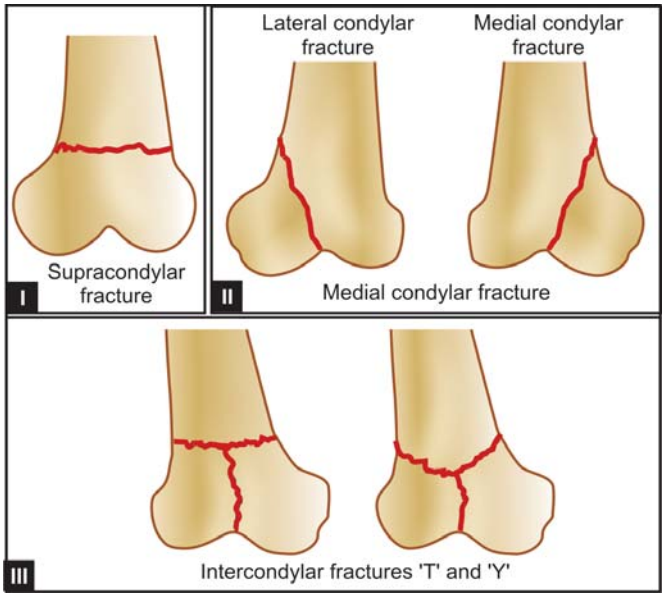


Figure 3.47

Showing the three groups of condylar fractures based on Muller's Classification.

Mechanism of Injury

These fractures can occur as a result of a direct injury or a fall from a height by means of tibia being driven firmly into the condyles of the femur.

Classification (Fig. 3.47)

Based on Muller's classification supracondylar fractures are divided into three groups:

Group A–Extra-articular fractures, i.e. Supracondylar fractures.

Group B–Intra-articular fractures involving one condyle, either lateral (common) or medial.

Group C–Bicondylar intra-articular fractures. These are basically intercondylar fractures with supracondylar extension. 'T'and 'Y' fractures.

Clinical Signs and Symptoms

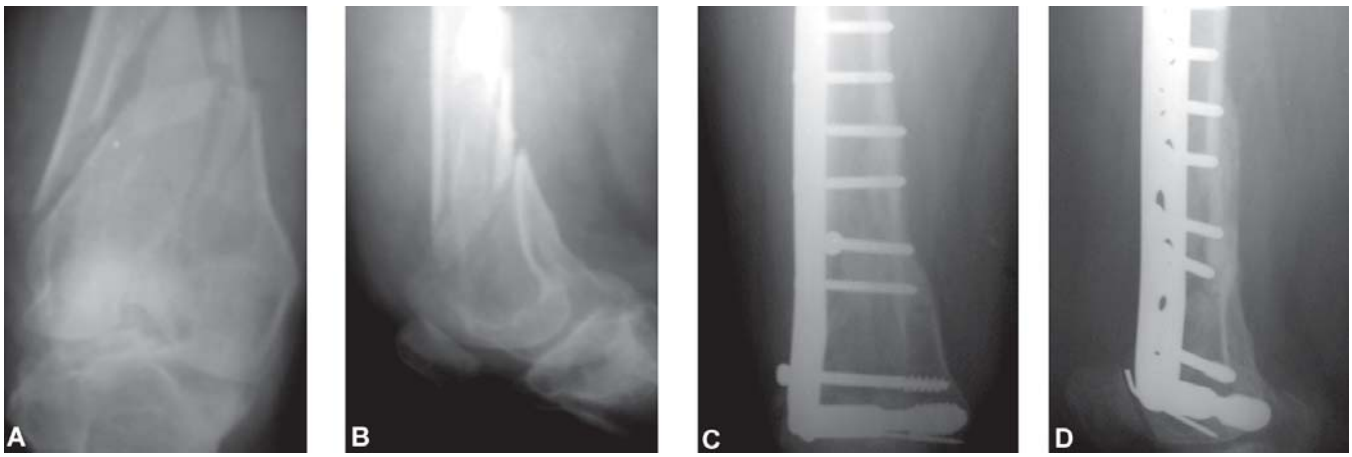
a. All the signs of a fracture.
b. Swelling around the knee due to hemarthrosis. (If there is break in the capsule the hemarthrosis spreads into the soft tissues).
c. All the movements at the knee are lost.

Diagnosis

X-ray–AP and Lat views give the diagnosis. Always keep in mind a coronal plane fracture, i.e. the Hoffa fracture.

Treatment

Though nonoperative treatment in the form of reduction and skeletal traction may result in union in less severe forms of fracture, open reduction and internal fixation is the treatment of choice. Surgery aims at achieving a sound stabilization so that early mobilization is begun to avoid disabling stiffness of the knee joint. The principle of treatment in Group C fractures is to convert it first into a supracondylar fracture and then fix it to the main fragment. When there is bad



Figures 3.48A to D

Radiographs showing fixation of a group C condylar fracture of the femur showing supracondylar comminution, internally fixed by means of a Dynamic Condylar Screw. Note that the insertion of a K-wire has converted it into a Supracondylar fracture. Interfragmentary screw fixation has been done for comminuted fragments in the supracondylar region. Then the Dynamic Condylar Screw and a Barrel plate is inserted. At times interfragmantary screw fixation may be necessary to hold the split condyles in position prior to the Dynamic Condylar Screw.

metaphyseal comminution wherein restoration of skeletal anatomy is not possible, a primary bone grafting procedure is undertaken (Figs 3.48A to D).

Essay Question

Classify condylar fractures of the femur. Discus the mechanism of injury diagnosis management and the complications of condylar fractures.

Fracture of the Shaft of the Femur

General Information

The fracture of the shaft of the femur is one of the major long bone fractures. The portion of the bone extending from about 7.5 cm below the lesser trochanter to about 5 cm proximal to the supracondylar portion of the femur is known as the shaft of the femur. The narrowest portion of the shaft is known as isthmus. The bone is highly vascular and surrounded all round by strong muscles of the thigh. Heavy blood loss up to 1.5 liters can occur and if not promptly replaced patient may go in for hypovolemic shock. The accompanying soft tissue injury too is considerable and adds on to the insult.

Mechanism of Injury

A force of considerable magnitude either bending or direct impact is necessary to break the femur. Hence the fracture shaft femur occurs as a result of severe trauma.

Types of Fracture Femur

a. Transverse fracture.
b. Oblique fracture.
c. Spiral fracture.
d. Comminuted fracture.
e. Segmental fracture.

Clinical Signs and Symptoms

The clinical diagnosis is easy as the fracture invariably gets displaced and abnormal mobility develops immediately. The thigh is swollen considerably because of heavy bleeding and associated muscle injury. Deformity and shortening of the limb is always present.

Investigations

a. Hb%, PCV and blood grouping and cross-matching is a must.
b. X-rays help in identifying the type and level of fracture as well as the diameter of the medullary canal (Proper assessment of the bone is absolutely essential before the treatment is planned).

Treatment

a. Immediate immobilization of the fracture in a splint and traction to prevent complications.
b. Administration of IV fluids and blood.
c. Treatment of other associated injuries if any.
c. Definitive treatment of the fracture is considered only after the patient is stabilized.

Definitive Treatment of Fracture Femur

Fracture femur in adults is always treated by reduction and internal fixation. Several methods are described. Closed methods are preferred to open methods. The methods are:

i. Open reduction and internal fixation using plates and screws or open nailing (Figs 3.49A and B).
ii. Closed reduction and internal fixation, e.g intramedullary interlocking nail, MIPPO technique, etc.
iii. Reduction and external fixation (Only in case of open fractures).

Advantages and Disadvantages of Closed and Open Methods

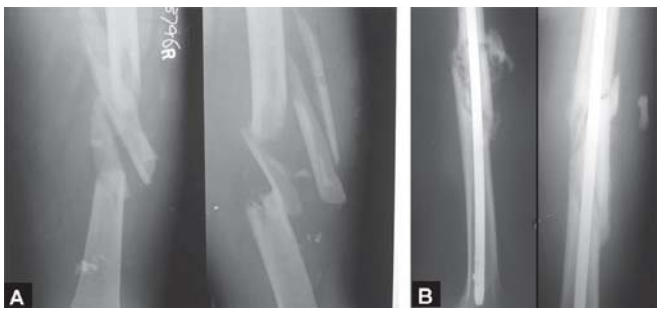
Closed method

Advantages:

a. Retains the fracture hematoma and facilitates the process of union.
b. No further soft tissue damage.

Disadvantages:

a. Anatomical reduction is not possible (Acceptable alignment is possible).



Figures 3.49A and B

Radiographs showing (A) A badly comminuted fracture of the shaft of the femur. It is not possible to achieve good apposition of fractured fragments by closed methods. (B) Showing fracture union after open reduction and internal fixation by Interlocking nailing procedure.

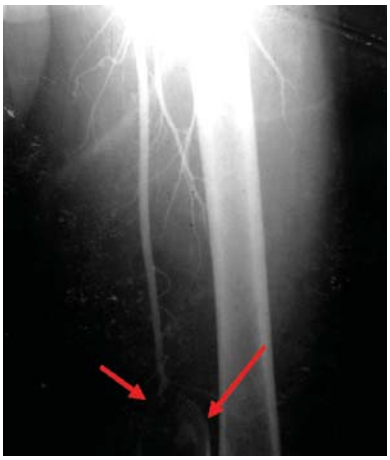


Figure 3.50

Radiograph of a fracture shaft femur lower 1/3 with an arteriogram showing transection of the femoral artery. Junction of the upper 2/3 and lower 1/3 is the site where the femoral artery enters the popliteal fossa through the opening in the Adductor magnus muscle. This is the vulnerable area for injury.

Open method

Advantage:

a. Anatomical reduction is possible.

Disadvantages:

a. Fracture hematoma is lost and union depends on secondary fracture hematoma.
b. Dissection of soft tissues causes a second insult (One has to be meticulous in dissection).

Good apposition of the fragments with at least more than 75% contact is essential for union. When this is not achieved by closed methods do not hesitate to do an open reduction and internal fixation.

Complications

Apart from routine complications like Delayed union, Non-union and Malunion, fracture shaft femur poses some serious life threatening and limb threatening complications. They are:

a. Shock and Hemorrhage.
b. Fat Embolism.
c. Injury to Femoral artery (Fig. 3.50).
d. Injury to Sciatic Nerve.

These complications have to be identified and dealt with efficiently to avoid disastrous outcome.

Essay Question

Discuss the mechanism of injury diagnosis management and complications of femoral shaft fractures.

Fractures Around the Hip

The fractures seen around the hip are:

a. Fracture of the head of the femur. (Very very rare. Usually associated with hip dislocation).
b. Fracture of the neck of the femur (True intracapsular fracture).
c. Intertrochanteric fracture (True extracapsular fracture)
d. Subtrochanteric fracture.

Fracture of the Neck of the Femur

General Information

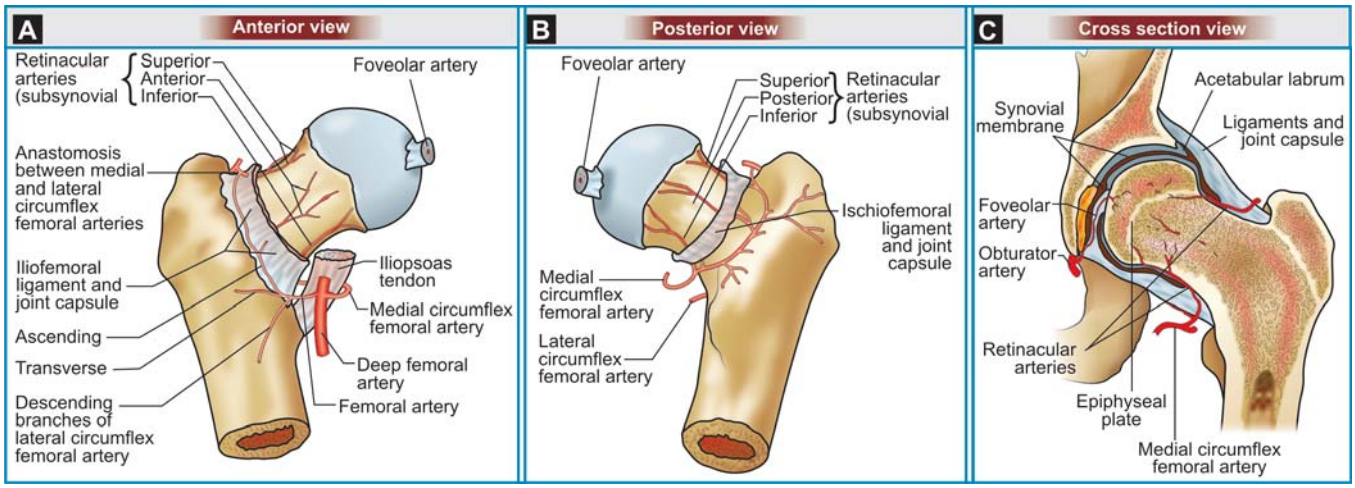
This fracture commonly occurs in elderly in the 7th decade and in women with osteoporosis (after menopause), following a very trivial torsional trauma (slipping). *Sir Astley Cooper was convinced about this fact way back in 1800's and said "A patient sustains a fracture and then falls and not that he falls and then sustains this fracture."* Though this is true in elderly patients, in the young more severe trauma is necessary to produce a fracture neck of the femur. Thus it is natural to conclude that more than one mechanism operates to cause a fracture neck of the femur.

This fracture even today poses several problems to the treating surgeon. Hence is known as a problem fracture, unsolved fracture and some call this (out of frustration) as an unsolvable fracture.

Problems Associated with the Fracture

1. There is no role for non-operative treatment. Surgery is the treatment of choice.

   Whitman spica given in the yester years was a failure and also associated with all the problems of prolonged confinement to bed, e.g. bed sore, hypostatic pneumonia, deep vein thrombosis, etc.
2. Difficulty at reduction because of spherical nature of the head which when broken is free to rotate within the acetabulum (To achieve reduction, a fracture table is necessary and different methods of reduction are to be employed).
3. No design of the implant, currently available satisfies all the criteria necessary for a secure fixation of the proximal fragment (This is true because of the small size and spherical shape of the proximal fragment. The entire variety of implants designed so far from Smith Peterson Nail, Moores pins, Knowels pins, Deyreles pins, Asnis screws, Garden's screws to cannulated cancellous screws justifies this fact).
4. Precarious blood supply may cause avascular necrosis of the head and nonunion of the fracture. (Retinacular vessels pass along the neck of the femur. These retinacular



Figures 3.51A to C

Showing the blood supply to the head and neck of the femur. From the Anastomotic Vascular Ring, formed at the base of the neck by the medial and the lateral circumflex femoral vessels, arise the anterior and posterior retinacular vessels which run along the neck of the femur towards the head and supply it. Also note the the area supplied by the foveolar artery which is very negligible when compared to the area supplied by the retinacular vessels. When the neck is fractured the retinacular vessels are invariably damaged. More so if the fracture is displaced.

vessels break when the neck is fractured. Artery through the ligament of the head of the femur supplies approximately 10% of the head) (Figs 3.51A to C).

Problems of Old Age Associated with the Fracture

During old age a person is susceptible for multiorgan failure and perhaps lives with compromised vital organs. Thus the diseases of old age like diabetes, hypertension, CNS compromise and stroke, renal compromise, increased incidence of DVT, poor lung function, etc. all complicate the ensuing surgical procedure.

Because of the above reasons, the name problem, unsolved and unsolvable fracture is fully justified even today.

Mechanisms of Injury

a. *Indirect mechanism (torsional stress, low energy trauma):* This is the most common mechanism operating in elderly patients when they slip. It is postulated that if the hip is in extension, the head of the femur remains fixed in the acetabulum so that the external rotation stress of the slip falls on the neck of the femur and breaks it. More distal fractures occur if the hip is in flexion during the external rotation stress.

b. *Direct mechanism:* This is the result of a fall on the trochanter. The force is transmitted to the neck directly and the neck breaks.

c. *Axial loading (high energy trauma):* The force is transmitted along the long axis of the femur with or without rotational component. This is the commonest mechanism operating in the young.

Classification

Depending on the site (Figs 3.52A to C)

A. *Subcapital* (constitute about 70% of the fractures. Fractures occur just below the caput or the head of the femur/at the site where the epiphyseal plate existed.)

B. *Transcervical* (constitute about 20% of the fractures. Fractures occur through the neck of the femur.)

C. *Basal* (Constitute about 10% of the fractures. Fractures occur at the base/ origin of the neck.)

Depending on the displacements

IV stages are identified based on Garden's classification depending on the displacements.

Garden's classification (Figs 3.53 and 3.54A to D)

Stage-I–Incomplete fracture (or valgus impaction).

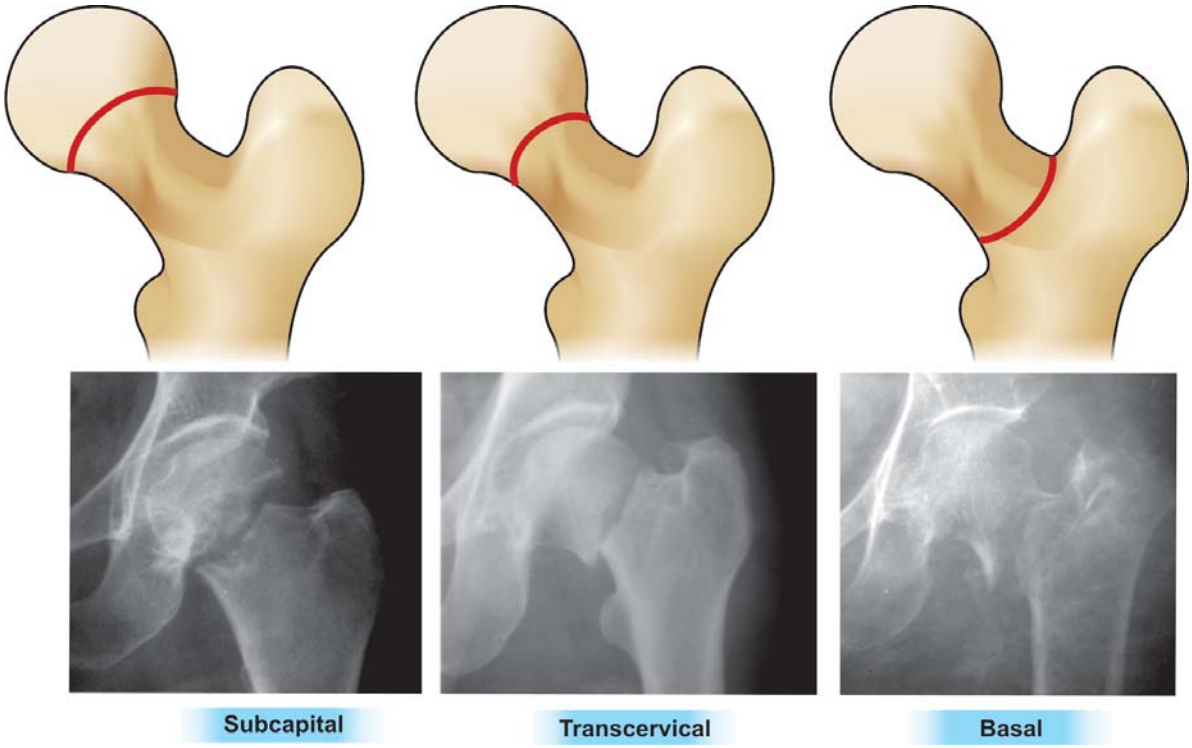
Stage-II–Complete fracture without displacement.

Stage III–Complete fracture with minimal displacement.

Stage IV–Complete fracture with marked displacement.

Clinical Signs and Symptoms

1. Following trivial/severe trauma a patient develops severe pain in the hip and unable to stand and walk.



Figures 3.52A to F

Diagrams and radiographs showing different types of fractures of the neck of femur.

2. The involved limb is in flexion, abduction external rotation. *(Helpless attitude of the* limb)
3. Minimal shortening is observed.
4. There is tenderness over the mid inguinal point.
5. The greater trochanter is normal and non-tender.

Diagnosis

Radiography: Views suggested are X-ray pelvis with both hips AP with the limb in maximum internal rotation (to visualize the neck and to assess the quality of the bone) and cross-table lateral view (to visualize the anterior and posterior portion of the neck and also to look for posterior comminution).

Bone scan and MRI in late presentations are useful to diagnose AVN.

Bigelow's Dictate:* In 1864, Bigelow remarked that *"While the impacted fracture of the base of the femoral neck unites by bone, if at all, there seems to be a decreasing tendency to osseous union as we approach the smaller portion of the neck near its head."

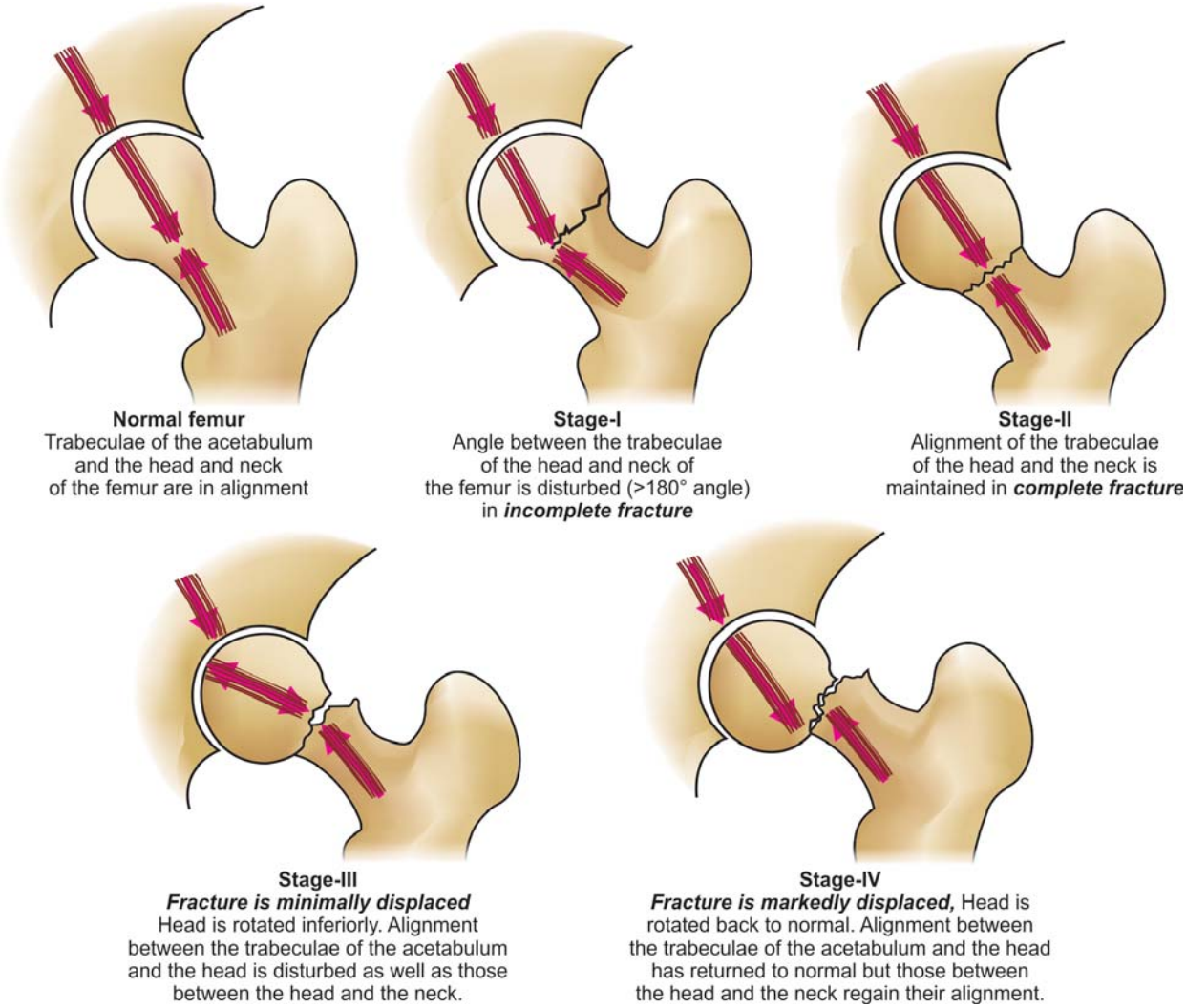
Management

All fractures are managed by surgery except impacted fractures. Procedures employed are:

a. Internal fixation.
b. Arthroplasty (Fig. 3.55).
c. Reconstructive procedures.

Factors to be considered before internal fixation:

a. *Age of the patient*: In younger age, attempt should always be made to preserve the head and neck of the femur. Hence the fractures are to be anatomically reduced and internally fixed. (Closed reduction and internal fixation. The fracture site is not opened).
b. *Type and nature of the fracture:* Displaced and comminuted fracture indicates severe trauma and they are more prone for complications of AVN and nonunion even if internal fixation is done. Hence to be treated at earliest as an emergency procedure to achieve secure bony contact and minimize the risk of AVN and nonunion.
c. *Duration of the fracture:* Longer the duration of the fracture the incidence of AVN and nonunion is more.



Figures 3.53A to E

Showing the trabecular pattern along the head and neck of the femur in different types of fractures. The red arrrows represent the direction of compression trabeculae. The fracture displacement is evaluated by looking at the relation of the trabecular pattern of the neck with the shaft for Stage I and Stage II fractures and trabecular pattern of the neck with the acetabulum for Stage III and Stage IV fractures. Break in continuity and loss of relation is observed.

Hence the fixation is to be augmented with vascularised bone grafting techniques, e.g. Meyer's muscle pedicle graft.

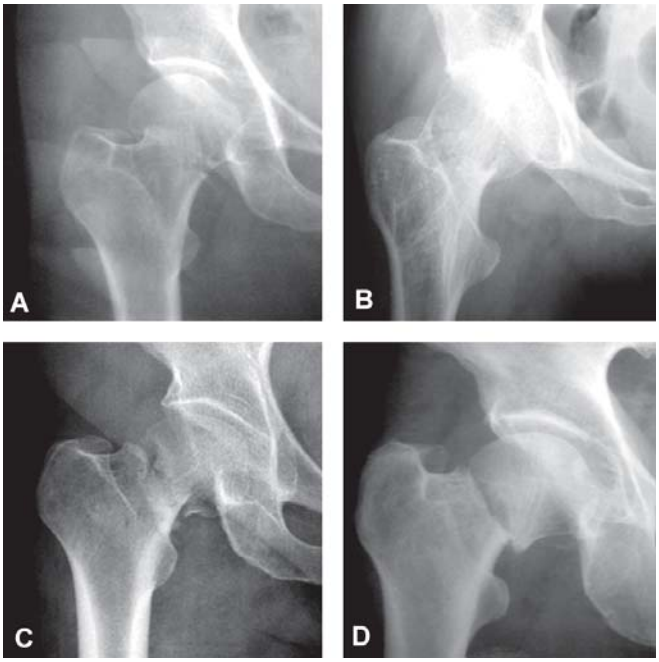
d. *Quality of bone* (Singh index). The quality of the bone has a direct relation to quality of healing. Osteoporotic bone of an old person heals poorly when compared to a nonosteoporotic bone of the young.

Implants used for internal fixation: Canulated cancellous screws, Garden's screws, Moore's pins and Knowel's pins.

Factors to be considered before replacement hemi-arthroplasty.

a. *Age of the patient:* Elderly whose life expectancy is limited and who cannot tolerate prolonged confinement to bed and limited activity till the fracture unites are considered for arthroplasty.

b. *Type of the fracture*: All fractures other than incomplete fractures and impacted fractures are suitable for arthroplasty.



Figures 3.54A to D

Radiographs showing all the four stages of fracture neck of femur based on Garden's classification. Note the trabecular disruption between the head and neck of the femur and the acetabulum which helps in classifying these fractures.

c. *Duration of the fracture:* Duration has no relation to arthroplastic procedures. It can be done at any period of presentation with good preoperative evaluation and choosing the appropriate prosthesis.
When Calcar femorale is sufficient Austin Moore's prosthesis is used. When calcar femorale is insufficient Thomson's prosthesis is used with bone cement.
(Cemented bipolar prosthesis and primary Total hip arthroplasty are also the treatment modalities to be considered in selected cases).

d. ***Treatment modalities depending on status and vascularity of the neck and head of the femur:***

Management of AVN with or without nonunion:

a. In the older age group, cemented total joint arthroplasty is the procedure of choice.
b. In the young,uncemented total joint arthroplasty is the procedure advocated.

Management of nonunion in young without AVN:

a. Without resorption of the neck of the femur—reconstructive procedure in the form of vascularized muscle pedicle graft (Figs 3.56A and 3.57A, B).

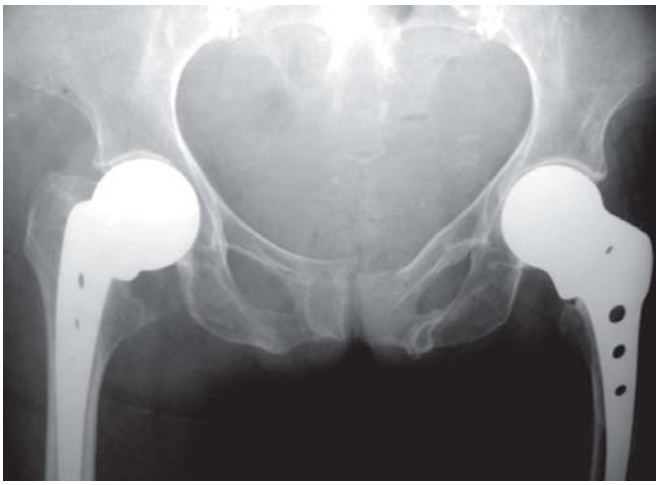


Figure 3.55

Radiograph showing a bilateral Hemiarthroplasty using Austin Moore prosthesis done for a case of bilateral fracture neck femur. First the patient sustained the fracture in the right hip and an year later in the left hip.

b. With resorption of the neck of the femur—uncemented Total hip arthroplasty (Fig. 3.56B).

Relationship between Senile Osteoporosis and Fracture Neck Femur

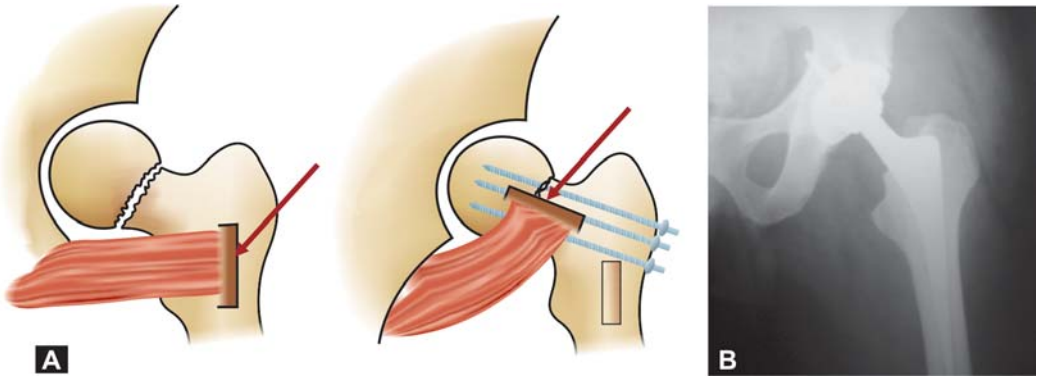
1. It acts as a predisposing factor for a fracture to occur with a trivial trauma (Not among black Americans and Bantu race in whom osteoporosis is uncommon)
2. It is responsible for comminution of the neck.
3. It is responsible for poor healing.
4. It does not allow firm purchase of the implant in the bone, causes loosening of the implant and failure of fracture fixation.

'Singh Index of Osteoporosis' and its Importance in Fracture Neck of Femur

Singh Index ((For more information Ref: Singh. et al JBJS 1970;52-A:457-67).

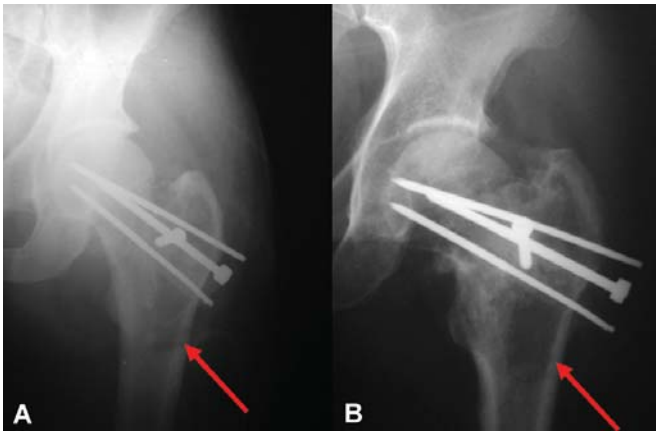
This index is based on the nature of the bony trabeculae of the Proximal Femur. They are grouped as follows:

a. Ward's Triangle.
b. Greater Trochanteric Group.
c. Secondary Compressive Group.
d. Secondary Tensile Group.
e. Principal Tensile Group.
f. Principal Compressive Group.



Figures 3.56A and B

Showing diagrams and a radiograph of (A) Quadratus femoris vascularized muscle pedicle grafting procedure and (B) an uncemented Total hip replacement.



Figures 3.57A and B

(A) Showing radiographs of—Quadratus femoris muscle pedicle bone grafting procedure done for a 3-month old ununited fracture neck of femur. (A) Immediate postoperative X-ray (B) X-ray at 3 months follow up. Note that the fracture has soundly united and there is no evidence of AVN. The arrow points out at the resultant defect at the donor site.

Depending on the texture they were assessed and graded into VI Grades, Grade VI being normal and Grade I suggests severe osteoporosis.

Grade VI
- All normal trabecular groups are visible.
- Upper end of femur seems to be completely occupied by cancellous bone.

Grade V
- Principal tensile and principal compressive trabeculae is accentuated *(thinned out).*
- Ward's triangle appears prominent.

Grade IV
- Principal tensile trabeculae are markedly reduced but can still be traced from lateral cortex to upper part of the femoral neck. *(thinned out and reduced but no break is observed).*

Grade III
- There is a break in the continuity of the principal tensile trabeculae opposite the greater trochanter *(break in continuity observed).*
- This grade indicates definite osteoporosis.

Grade II
- Only principal compressive trabeculae stand out prominently.
- Remaining trabeculae have been essentially absorbed. *(absence of tensile trabeculae and thinning of compression trabeculae)*

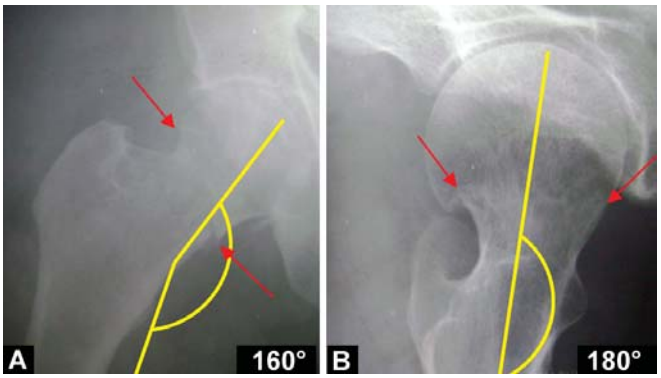
Grade I
- Principal compressive trabeculae are markedly reduced in number and are no longer prominent. *(few compressive trabeculae are left).*

Singh index helps in the fixation of fracture neck of femur in the following way:

1. It gives the physiological age of the bone. (physiological age is different from chronological age.)
2. Helps in taking a decision whether to fix or not to fix. Grade VI to Grade IV are acceptable indices for fixation. Grade III to Grade I are poor indices for fixation.
3. Helps in placement of the implant in non-osteoporotic inferior part of the neck (low insertion to prevent cut through).

Relationship of Garden's Alignment Index to Fracture Neck of Femur

Garden's alignment index: It is an index derived from the angle formed by the primary compression trabeculae on the AP view with the longitudinal axis of the femoral shaft and



Figures 3.58A and B

Radiographs AP and Lateral views of hip joint showing satisfactory index after reduction as per Garden's Alignment index. (Refer text for details).

primary compression trabeculae on the lateral view with the longitudinal axis of the femoral shaft. The ideal angle is 160° in the AP view and 180° in the lateral view. This varies from individual to individual. The range is between 155°-180°. This index helps in assessing the reduction of the fracture as acceptable within this range. < 155° and > 180° increases the risk of AVN by almost 50%. Nonunion is also commonly seen when the angle of reduction is beyond this acceptable range.

Lowell's Alignment Theory

Radiographically the outline of the femoral head is convex and the outline of the femoral neck is concave. At the point where they meet it produces an image of S (right hip) or reversed S (left hip) curve. Instead of S if an unbroken C curve is seen after reduction, the reduction is considered as unacceptable. (See Fig. 3.58A at the place where the arrow is pointing. The curve is 'S' shaped and smooth suggesting acceptable reduction. On the left side the curve formed would take the shape of a reverse 'S').

Revision Questions

1. Classify fracture Neck of femur.
2. Discuss complications of fracture neck of femur.
3. Garden's Alignment index.
4. Singh index.

Essay Questions

1. Discuss the mechanism of injury, classification, diagnosis and management of fracture neck of femur.
2. Fracture neck of femur an unsolved fracture. Discuss.
3. Enumerate the complications of fracture neck of femur. Discuss their management.

Intertrochanteric Fracture

Intertrochanteric fractures are those fractures *occurring in the extracapsular portion* of the proximal femur, i.e. in the portion between the greater and the lesser trochanter of the femur. These fractures, like fracture of the neck of the femur, occur in elderly people *in the 7th decade* perhaps at a little later age, *3-5 yr later than fracture neck of the femur.* The capsule of the hip joint is attached anteriorly at the intertrochanteric line and posteriorly stops short about a centimeter before the intertrochanteric crest. These fractures occur beyond the attachment of the capsule of the hip joint. The *bone in this region is cancellous* in nature and hence the *union rate in these fractures is higher* when compared to the fracture neck of femur. Also these fractures are encountered with *less complications.*

Mechanisms of Injury

a. *Indirect mechanism (torsional stress, low energy trauma):* It is postulated that a torsional stress occurring in an elderly individual with osteoporotic femur can cause this fracture. The hip should be in some degree of flexion for the torsional force to act on the intertrochanteric portion of the femur.
b. *Direct mechanism:* A direct impact on the trochanter resulting in intertrochanteric fracture.
c. *Axial loading (High energy trauma):* Commonly seen in young patients, e.g. vehicular accidents, fall from a height.

Classifications

Based on Evans' classification (1949) classified as follows (Fig. 3.59)

Type I–Undisplaced 2-fragment fracture.

Type II–Displaced 2-fragment fracture.

Type III–3-fragment fracture without posterolateral support.

Type IV–3-fragment fracture without medial support.

Type V–4-fragment fracture without posterolateral and medial support.

Type VI—Reversed obliquity fracture.

(Type I is a stable fracture. Type II, III,IV and V when remain displaced after attempted reduction are considered unstable. When reduced satisfactorily they are considered stable. Type VI- Always unstable).

Based on Boyd and Griffin's classification (1949) classified as follows (Figs 3.60 and 3.61)

Type I–Linear intertrochanteric fractures (without comminution)

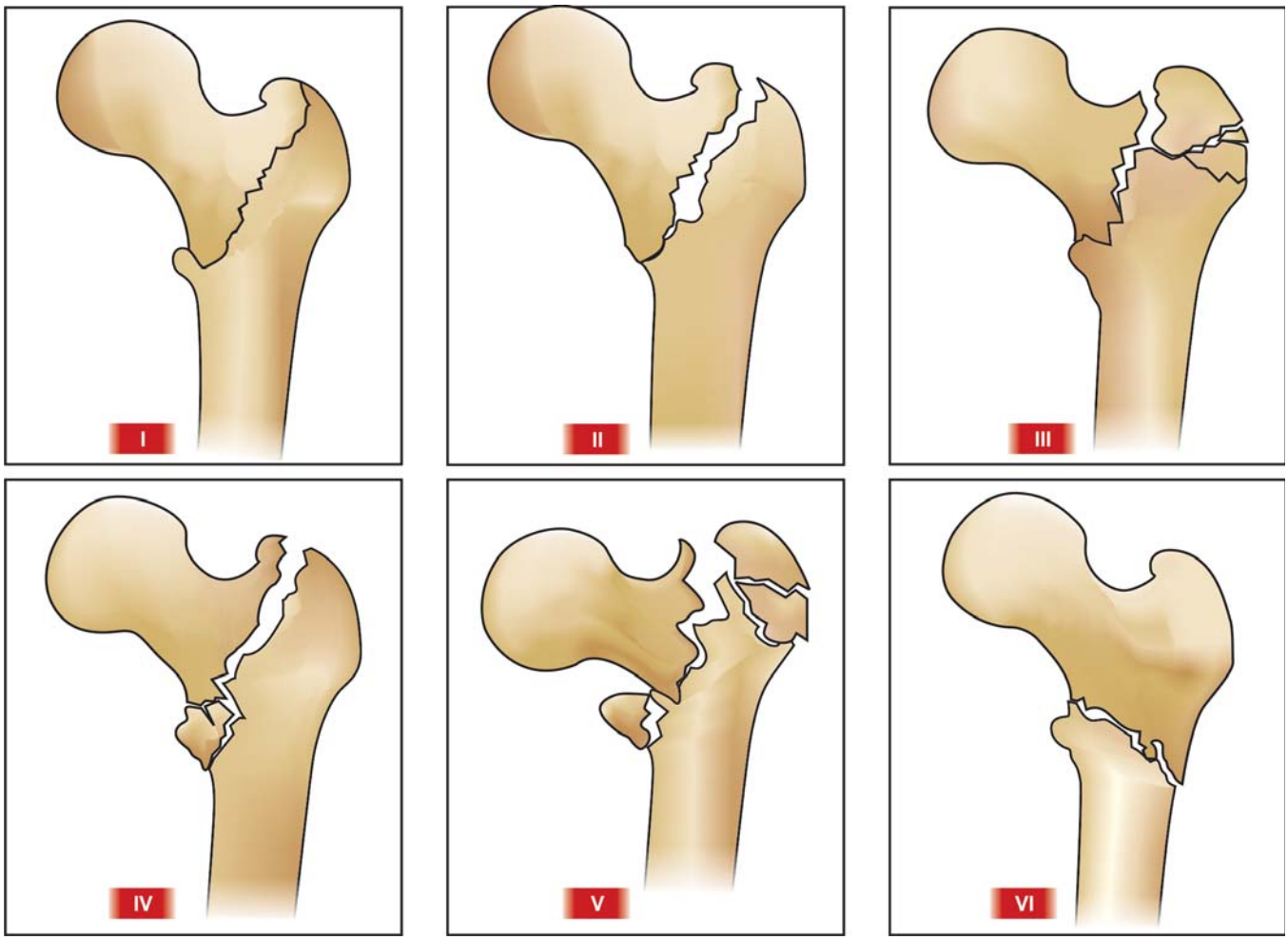


Figure 3.59
Showing Evans classification of intertrochanteric fracture diagrammatically represented.

Type II–Intertrochanteric fractures with comminution.

Type III–Intertrochanteric fracture with Subtrochanteric extension.

Type IV–Fractures of the trochanteric region and the proximal shaft (fracture in two planes).

Clinical Signs and Symptoms

1. History of a trivial trauma (in young severe trauma).
2. Gross shortening of limb.
3. Flexion abduction external rotation deformity.
4. Tenderness and irregularity over the greater trochanter.

Diagnosis

By means of standard AP view of the pelvis with both hips and cross table lateral view (Fig. 3.62).

Management

a. *Operative:* Results are far superior to nonoperative methods and fractures invariably unite when fixed by proper techniques using appropriate implants. Risks of surgery in elderly patients and poor quality of bone do pose problem for operative management.

Stable Intertrochanteric fractures: The fracture is reduced on a fracture table and internal fixation is done using a Sliding hip screw and a Barrel plate. Failure to reduce by closed reduction on a fracture table necessitates open reduction of the fracture before internal fixation.

Unstable Intertrochanteric fractures: Medial arch involvement with fracture of the calcar and posterior comminution are the causes for instability. These have to be well reduced and internally fixed by providing additional support. Buttress plate, trochanteric hook plate,

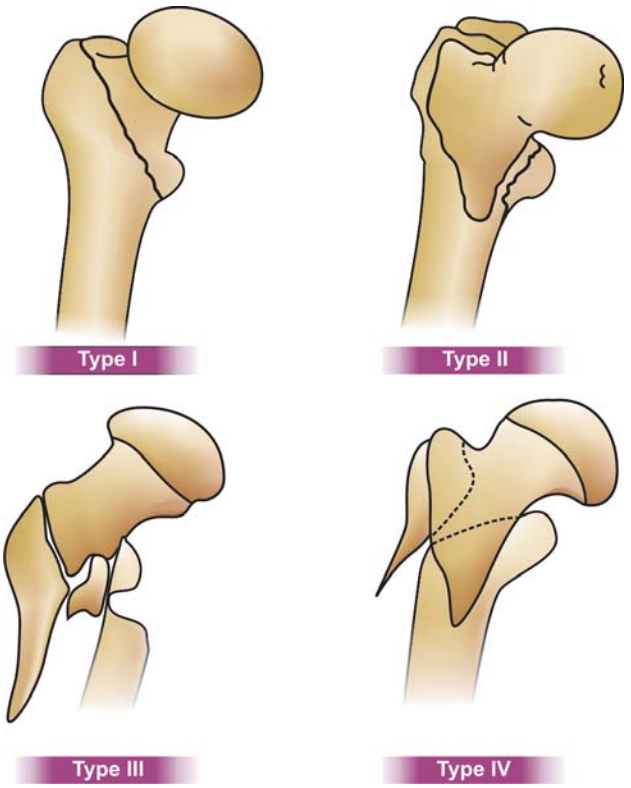


Figure 3.60
Showing Boyd and Griffin classification of Intertrochanteric fracture.

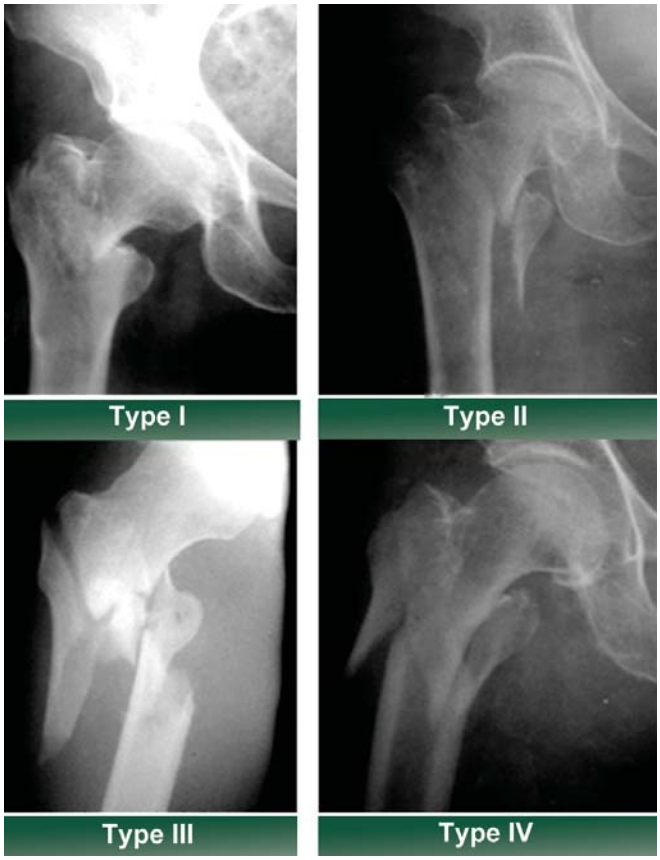


Figure 3.61
Intertrochanteric fractures. Radiographs showing IV types of fractures based on Boyd and Griffin classification.

interfragmentary screws are some of the additional implants that are useful. Modified internal fixation techniques too are employed in certain fractures, e.g. Dimon- Houston technique: Sarmiento technique, etc.

b. *Nonoperative:* Not desirable in elderly as chances of succumbing to problems of prolonged confinement to bed are very high.

Nonoperatively managed by upper tibial skeletal traction on a supportive splint for 6-8 weeks with appropriate weights.

Factors affecting union:

1. Poor quality of bone.
2. Poor quality of muscles.
3. Unstable fractures.
4. Inappropriate technique of fixation.

Essay Question

Discuss the mechanism of injury, classification , diagnosis and management of intertrochanteric fracture of femur.

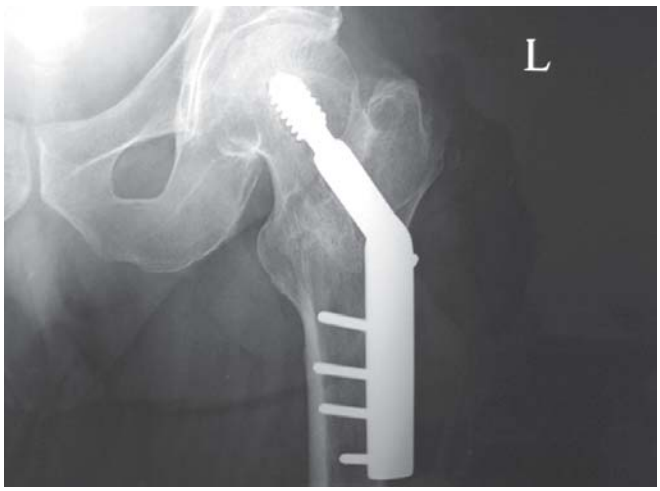


Figure 3.62
Radiograph showing union of an intertrochanteric fracture after Dynamic Hip Screw fixation. Note the central placement of the screw in the head and neck of the femur in the AP view. The same is desirable in lateral view too for a stable fixation.

Subtrochanteric Fracture

The part of the bone from the lower end of the lesser trochanter to about 5-7.5 cms below the lesser trochanter (depending on the height of the individual) is considered as the subtrochanteric portion of the femur. A fracture occurring in this portion of the femur is known as Subtrochanteric fracture.

These fractures are considered to be one among the most difficult fractures to treat for following reasons.

i. The bone involved is cortical. Hence takes a longer time to heal when compared to a cancellous bone.
ii. This area is subjected to greater stress when compared to rest of the femur. Hence failure of treatment is common.

Mechanism of Injury

a. Direct—a direct impact onto the lateral thigh by a fall. This mechanism is common in elderly.
b. High velocity injury, e.g. Road traffic accident. This mechanism is common in the young wherein a force of considerable magnitude breaks this portion of the bone.

(Because this area is the one subjected to maximum stress during activities of daily living, the bone too is quite strong and does not break easily).

Following fracture the muscles attached to the proximal femur displace the fractured fragments (Fig. 3.63).

a. The gluteus medius and minimus abduct the greater trochanteric (proximal)segment.
b. The iliopsoas flexus the lesser trochanteric (proximal) segment.
c. The adductors ,adduct the shaft (distal) segment.

As this area is highly vascular heavy bleeding also is common.

Classification

Based on Seinsheimer classification (Ref: J Bone and Joint Surgery Am 1978;60:300-6) (Figs 3.64A to H).

Type I Non displaced fractures/fractures < 2 mm displacement.

Type II Two-part fracture.
- A. Transverse fracture.
- B. Spiral fracture with lesser trochanter in the proximal fragment.

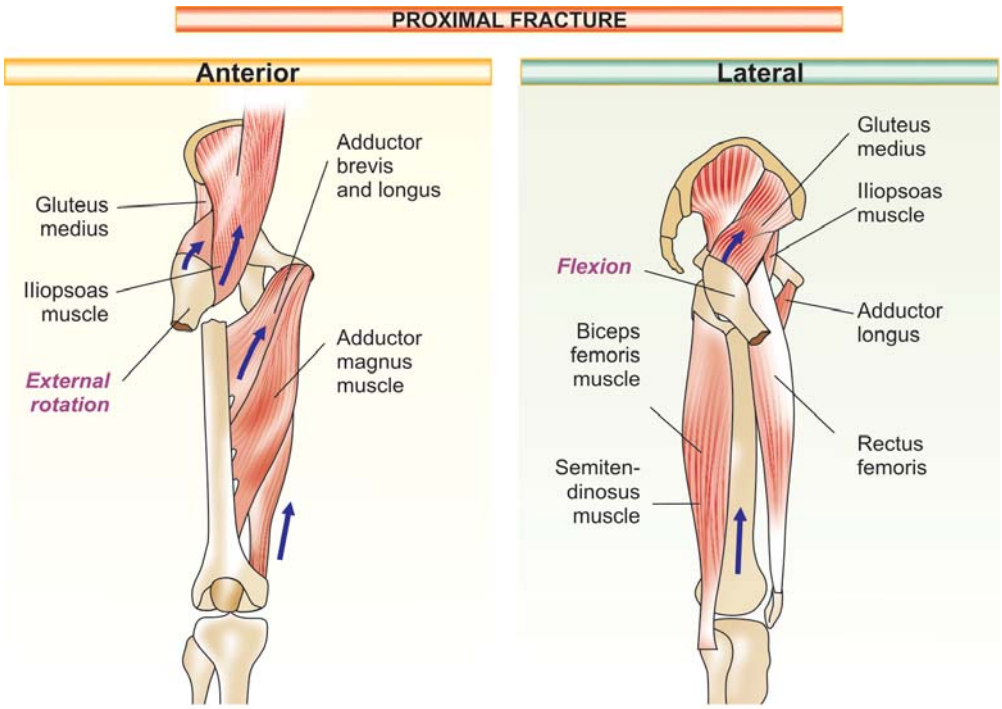
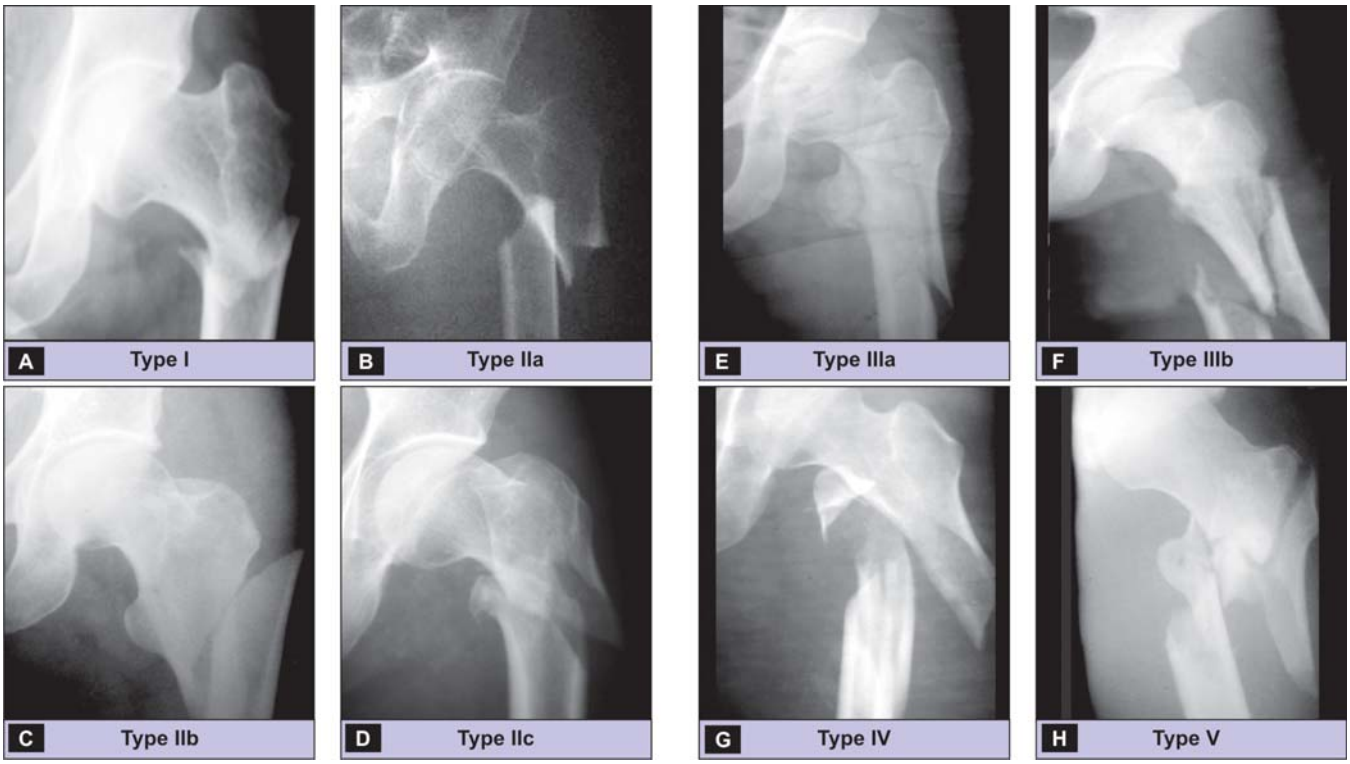


Figure 3.63
Picture showing displacing forces in a subtrochanteric fracture.



Figures 3.64A to H

Radiograph of subtrochanteric fractures—based on Seinsheimer classification. Refer text for details.

C. Spiral fracture with lesser trochanter in the distal fragment.

Type III Three-part fracture.

A. With lesser trochanter a separate fragment.

B. The third part is a butterfly fragment other than lesser trochanter.

Type IV Four-part (or more) comminuted fractures.

Type V Subtrochanteric with intertrochanteric fracture. (fracture through the greater trochanter.)

Treatment

Non-operative: There is no role for nonoperative treatment unless the patient is not fit for surgical procedure.

The treatment consists of skeletal traction on a supportive splint which is maintained till the fracture unites.

Operative: Surgical stabilization is the treatment of choice.

Implants used currently are cephalomedullary and centromedullary interlocking nails.

Surface implants such as locking compression plates, dynamic hip screw, dynamic condylar screw, etc. are also used in selected cases. As far as the decision with respect to choice of the implant required for the fixation of subtrochanteric fracture two major variables are to be considered.

- Whether the fracture is extending into the greater trochanter posteriorly and involving the Piriformis fossa

Differences	*Inter- trochanteric fracture*	*Fracture neck of the femur*
Attitude of the limb	Marked external rotation	Minimal external rotation
Shortening	Marked	Minimal
Tenderness	Over the trochanter	Over the hip joint
Irregularity	Over the trochanter	Trochanter, normal
Swelling	Considerable,in the trochanteric region and in the upper1//3 thigh	Minimal, around the hip only

because it is one of the most commonly used nail entry portal.

- Whether there is continuity of the lesser trochanter.

(Refer Russell–Taylor Classification for guidance in selecting the appropriate implant).

Further Reading

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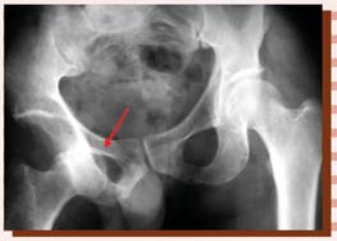


4 Dislocations

- *Specific Dislocations in the Upper Limb, their Clinical Features, Diagnosis and Management*
- *Specific Dislocations in the Lower Limb, their Clinical Features, Diagnosis and Management*

Dislocation of the Acromioclavicular Joint

General Information

The acromioclavicular joint is a diarthrodial joint between the lateral end of clavicle and the acromian. A fibrocartilaginous disc is interposed between the two articular surfaces preventing them from coming into direct contact. The stability of the joint depends mainly on the capsule and the ligaments binding the joint.

The acromioclavicular joint dislocates only when the coracoclavicular and the acromioclavicular ligaments are completely torn.

Mechanism of Injury (Fig. 4.1)

Fall on the point of an adducted shoulder causes strain on the acromioclavicular ligaments first which results in subluxation of the acromioclavicular joint. If the force continues to act further it ruptures the conoid and trapezoid part of the

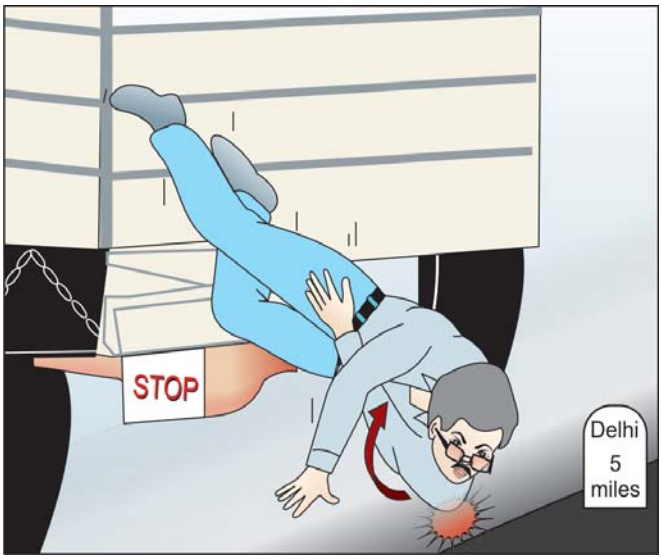


Figure 4.1

Showing the most common mechanism of injury of Acromioclavicular joint.

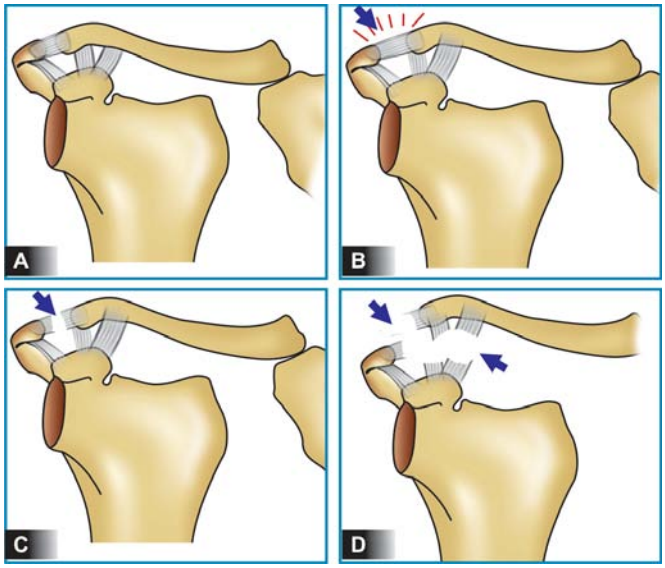


Figure 4.2

Showing (A) A normal joint with intact acromioclavicular and coracoclavicular ligaments. (B, C and D) Varying grades of injury classified as Type-I, Type-II, Type-III respectively.

coracoclavicular ligament and a complete dislocation of the acromioclavicular joint results.

Classification (Fig. 4.2)

The injury is classified into Type I, Type II and Type III.

Type-I: It is an acute sprain (contusion) of the acromioclavicular ligaments.

Type-II: Rupture of the acromioclavicular ligament occurs.

Type-III: Rupture of both acromioclavicular and the coraco clavicular ligament occurs.

In Type I there is no joint subluxation or dislocation. In Type-II there is subluxation of the acromioclavicular joint. In Type III there is dislocation of the acromioclavicular joint.

Clinical Features

a. Pain in the region is the only feature in Type I and Type II injuries.
b. Dislocation of the acromioclavicular joint with prominence of the lateral end of the clavicle is seen in Type III injuries.

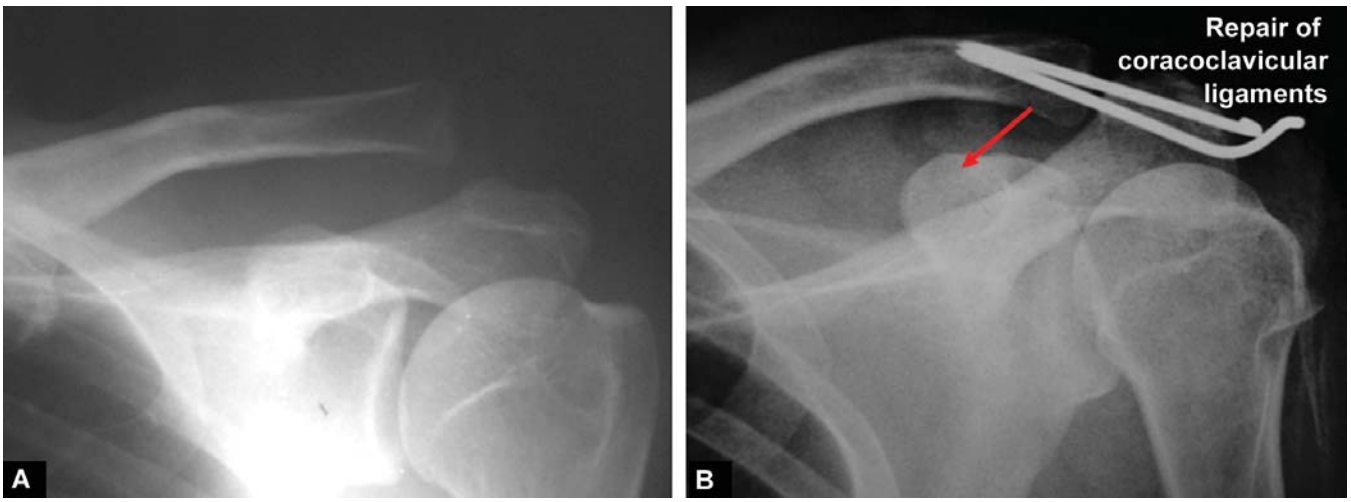
Diagnosis

X-ray–A normal AP view of the shoulder may be deceptive at times. For better diagnosis, a stress view with the patient standing and holding a 5 kg weight in the hand with the hand by the side of the body, is useful. This demonstrates the subluxation/dislocation of the acromioclavicular joint.

Management

Type I and Type II are treated nonoperatively in a shoulder arm sling. Type III is treated surgically by the repair of the conoid and trapezoid part of the coracoclavicular ligament (Figs 4.3A and B). At the same time the joint is transfixed with 'K'wires.

In old unreduced acromioclavicular dislocation which is untreated the projecting lateral end of the clavicle may be excised.



Figures 4.3A and B

Radiograph showing (A) Type III acromioclavicular dislocation with disruption of both coracoclavicular and acromioclavicular ligaments. (B) After reduction, transfixation of acromioclavicular joint with two Kirschner wires and repair of the coracoclavicular ligaments as shown by the red arrow.

Revision Questions

Q. Discuss the anatomy of the acromioclavicular joint.
Q. What are the stabilizing structures of the acromioclavicular joint?
Q. Classify the acromioclavicular dislocation.
Q. Discuss the treatment of acromioclavicular dislocation.
Q. Discuss the complication of acromioclavicular joint dislocation.

Essay Question

Discuss the pathoanatomy of the acromioclavicular joint. How do you classify the acromioclavicular joint dislocation? Outline the diagnosis and management.

Dislocation of the Shoulder

When the head of the humerus loses its articulation with the glenoid cavity of the scapula, a dislocation of the shoulder occurs. (Dislocation is defined as total loss of contact between the two articular surfaces).

Types

a. Anterior 98%
b. Posterior 2%

Anterior Dislocation of the Shoulder

Mechanism: Occurs as a result of a hyperabduction and external rotation strain.

Types

a. Subcoracoid
b. Subglenoid
c. Subclavicular
d. Intrathoracic (rare) } associated with
e. Superior (very rare) } complications

Diagnosis

Signs and symptoms: Patient always presents with the injured limb held and supported by the help of other upper limb.

a. Severe pain in the shoulder.
b. Loss of all the movements of the shoulder.
c. Loss of (prominence) contour of deltoid muscle bulge.
d. A hollow is appreciated at the shoulder.
e. Tenderness.
f. Head of the humerus is palpable in a place other than the glenoid.

Tests

a. **Duga's test:** Patient is asked to touch the opposite shoulder.

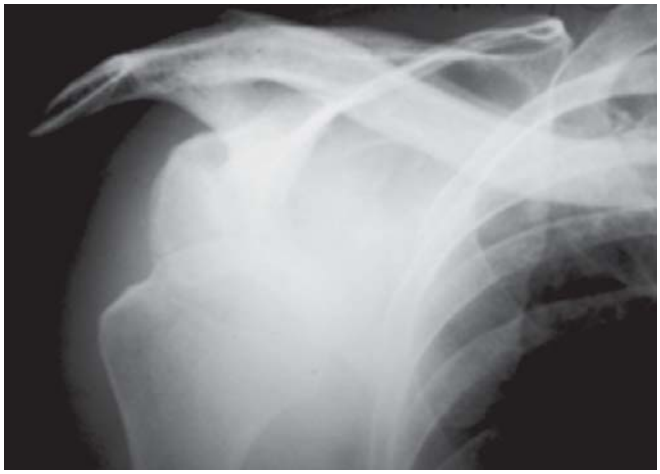


Figure 4.4
Radiograph showing a subglenoid type of anterior dislocation of shoulder.

If the patient is unable to touch the opposite shoulder the test is positive.

If the patient is able to touch the opposite shoulder the test is negative.

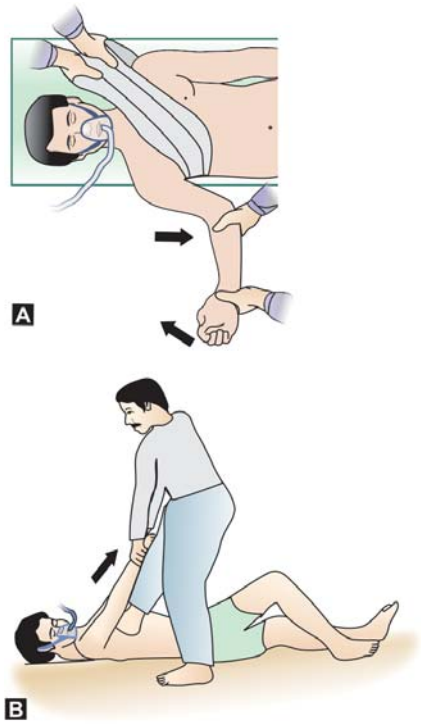
A negative test in a case of anterior dislocation of the shoulder suggests the presence of a fracture of the surgical neck of the humerus.

b. **Hamilton ruler test:** When the head of the humerus is in the glenoid cavity, the bulging contour of the deltoid muscle prevents one from placing a ruler touching both acromion and lateral condyle of the humerus. In dislocation this contour is lost and it is possible to place a ruler straight across the shoulder touching both, the acromion and the lateral condyle of the humerus. The test is said to be positive.
c. **Bryant's sign:** Positive sign is shown by lowering and prominence of the anterior axillary fold.
d. **Callaway's test/sign:** Increase in girth of the shoulder.

Investigation: X-ray of the shoulder to know the type of dislocation and to ascertain the presence or absence of fracture. Commonest fracture associated with the anterior dislocation is fracture of the greater tuberosity of the humerus (Fig. 4.4).

Management: Always by closed reduction and immobilization in arm-chest bandage. Open reduction is indicated in late presentation (after three weeks) and when the dislocation is irreducible by closed methods.

Kocher's method: Patient under anesthesia, lies supine on the examination table. Traction is given with the elbow flexed and



Figures 4.5A and B

Pictures showing (A) Kocher's method and (B) Hippocrates method of reduction of anterior dislocation of the shoulder. Note that in Hippocrates method the patient is on the floor.The foot of the surgeon is used as a lever and not to apply counter force.

counter traction is given in the axilla with the help of folded towel. Both traction and counter traction is given along the axis of the humerus. The limb is slowly abducted and externally rotated. When the position is attained which the head of the humerus had occupied just after the dislocation, the dislocation gets reduced almost spontaneously because of the counter force. Once reduced then the shoulder is internally rotated, adducted and flexed and immobilized in arm chest position.

Hippocrates method (Figs 4.5A and B): Patient under anesthesia, lies supine on the floor. The dislocated limb is held firmly by the surgeon at the wrist and traction is given. With the foot in the axilla the head of the humerus is gently maneuvered into the glenoid cavity.

Hippocrates method is a safer method when compared to Kocher's method. If not carefully executed, in Kocher's method there is a possibility of a fracture of the surgical neck of the humerus occurring during the attempt. Especially if the patient is elderly and the bone is osteoporotic or when there is an occult fracture.

Recurrent anterior dislocation: If recurrent episodes of dislocation occurs following the first episode it is known as recurrent dislocation.

Pathology: Triple deformity (lesion) is responsible for these episodes.

a. *Bankart's lesion:* It is tear of the anterior inferior glenoid labrum (2 o' clock to 6 o' clock right shoulder: 6 o' clock to 10 o' clock left shoulder).
   Diagnosis is established by MRI with contrast.
b. *Hill- Sach's lesion:* A defect in the posterolateral part of the head of the humerus.
   Diagnosis is established by special view known as **Stryker notch view** (Refer for details) if in doubt MRI to be done for confirmation.
c. *A defect in the anterior inferior capsule.*

Management: By surgical stabilization in which the defects are attended to and the shoulder is stabilized, e.g. Putti- Platt procedure, Bankart procedure, Helfet–Bristow procedure. These days stabilization procedures are done through shoulder arthroscopy too. (Refer for more details regarding arthroscopy).

Posterior Dislocation of the Shoulder

Mechanism: A powerful adduction internal rotation strain, e.g. in seizures, electric shock or sometimes a direct posterior impact on the shoulder.

Types

a. Subacromial
b. Subglenoid
c. Supraspinous

Diagnosis

Symptoms and signs

a. History of sustaining electric shock or an episode of seizures followed by severe pain and inability to move the shoulder.
b. Prominence of the coracoid process.
c. Limb is in a position of adduction and internal rotation.
d. Attempted abduction and external rotation is painful.

Investigation: X-rays–A standard AP view is taken. This gives oblique profile of the glenoid. Following signs are seen:

a. *Loss of elliptical overlap:* Normally there is an overlap of at least 1/3 of the head over the posterior glenoid (obliquely profiled glenoid). This overlap becomes less or absent.
b. *Empty (vacant) glenoid sign:* The glenoid cavity is void of articulating head of the humerus (Fig. 4.6).

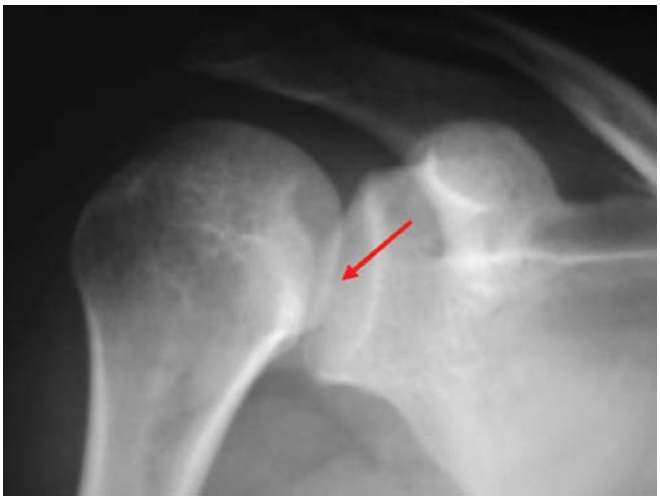


Figure 4.6

Radiograph showing the loss of elliptical overlap as well as 'Empty glenoid sign' suggestive of posterior dislocation of the shoulder. In fact, this patient is a known epileptic and sustained the injury in an episode of seizure.

Management: Reduction is achieved under anesthesia by giving gentle longitudinal as well as lateral traction followed by external rotation and abduction. Limb is immobilized in external rotation and extension for a period of 3-4 weeks.

Recurrent posterior dislocation: Occurs as a result of posterior lesions.

a. Reverse Bankart's lesion.
b. Reverse Hill- Sach's lesion.
c. Posterior glenoid deficiency.
d. Increased retroversion of the head and the glenoid.

Management: By surgical stabilization procedures, e.g. Mc Laughlin procedure. (Refer for details)

Luxatio erecta–It is an exaggerated form of infraglenoid type of dislocation of the shoulder. Patient presents with raised upper limb with an inability to bring it down.

Revision Questions

Q. What are the different types of shoulder dislocation?
Q. Discuss the mechanism of shoulder dislocations.
Q. What are the clinical signs of anterior dislocation of the shoulder?
Q. Duga's test.
Q. Empty glenoid sign.
Q. What are the lesions responsible for recurrent anterior dislocation of the shoulder?
Q. Bankart's lesion.
Q. Hill-Sach's lesion.

Essay Questions

Q. Discuss the pathoanatomy , mechanism of injury diagnosis and management of anterior dislocation of the shoulder.
Q. Define recurrent dislocation of the shoulder joint. Discuss the pathomechanics and management of recurrent anterior dislocation of the shoulder.
Q. Discuss the clinical features diagnosis and management of posterior dislocation of the shoulder. Enumerate the causes of recurrence.

Dislocation of the Elbow

The elbow joint is a hinge joint which is inherently stable because of the design and does not depend much on ligaments and other soft tissue structures surrounding it for its stability. Hence considerable force is necessary to dislocate an elbow joint. Thus in about 1/3 of the cases of elbow dislocation fractures are commonly seen. Without fractures the elbow dislocation is termed as simple and with associated fractures it is termed as a complex dislocation.

Types

a. Posterior – 90% (Figs 4.8A and B)
b. Anterior – 10% (Fig. 4.7B)

Note–Figure 4.7A showing old unreduced posterior dislocation.

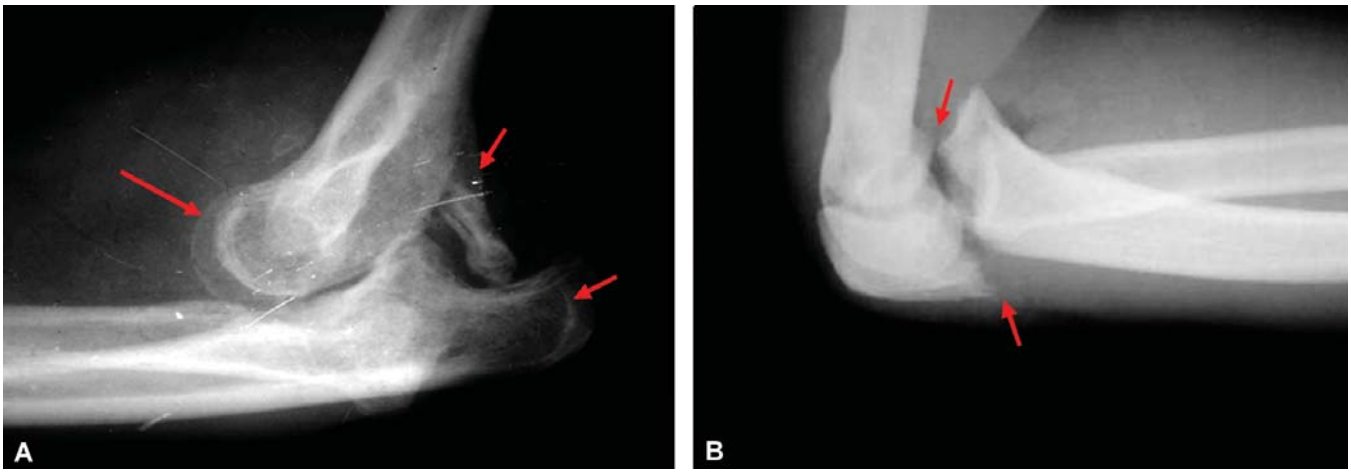
Mechanism of Injury

Posterior–Fall on an out-stretched hand with arm in abduction and extension, e.g. falling backwards after slipping. (Commonest fracture associated is the fracture of the medial epicondyle).

Anterior–A powerful blow to the posterior aspect of the elbow. (Complication of median nerve and brachial artery injury is common in anterior dislocation of the elbow. Commonest fracture associated is the fracture of the olecrenon process.)

Diagnosis

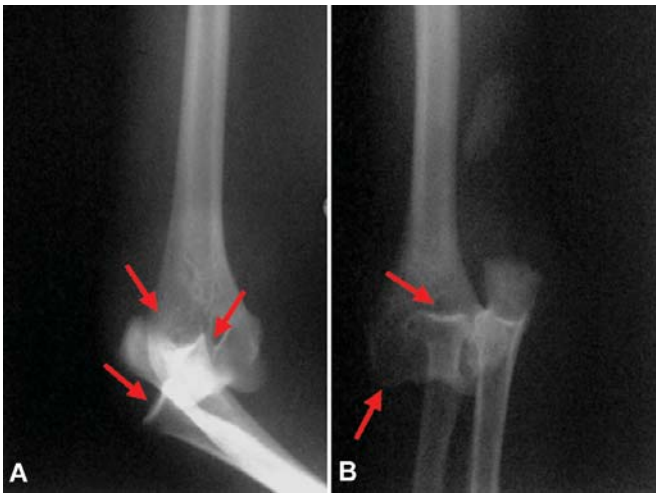
The posterior dislocation is diagnosed by clinical examination which shows loss of triangular relationship of three bony points at the elbow and severe restriction of movements. Also



Figures 4.7A and B

(A) Radiograph showing an old unreduced posterior dislocation of the elbow with medial epicondylar fragment in the joint. When this fragment is in the elbow, the elbow becomes irreducible unless the fragment is dislodged. (B) Radiograph showing fracture of the olecrenon process of the ulna with anterior dislocation of the elbow. Carefully note the loss of articulation between the head of the radius and the capitulum, the coronoid process of the ulna and the coronoid fossa of the humerus as well as *in situ* position of the olecrenon, which is articulating with the humerus in the flexed position of the elbow.

Also there is no superior radio ulnar joint disruption. These features suggest that there has been a direct blow to the elbow from behind causing anterior dislocation. The fracture of the olecranon process is not an avulsion fracture which generally occurs because of the pull of triceps.



Figures 4.8A and B

Radiograph LAT and AP view of the elbow showing traumatic posterior dislocation of the elbow joint.

there is shortening of the forearm component of the limb. The arm component is normal.

X-ray proves conclusively and also determines the presence or absence of fracture.

Management

a. Simple dislocations are managed by closed manipulation under anesthesia, reduction and immobilization for a period of three weeks. This is followed by gradual mobilization.
   Posterior dislocations are immobilized in flexion.
   Anterior dislocations are immobilized in extension.
b. Complex dislocations are managed by open reduction and stabilization.
   Associated fractures are anatomically reduced and fixed.

Revision Questions

Q. Discuss the clinical diagnosis of posterior dislocation of the elbow joint.
Q. Classify dislocations of the elbow.
Q. Discuss the mechanisms of injury of different elbow dislocations.

Essay Question

Q. Discuss the mechanism of injury, clinical features diagnosis and management of different elbow dislocations.

Dislocation of the Hip Joint

Hip joint is a ball and socket joint in which the spherical head of the femur articulates with the cup, i.e. the acetabulum. Inherent stability is imparted through this articulation. The strong ligaments and the tough capsule which surround the joint and the powerful muscles impart additional stability. Among the ligaments the Y ligament of Bigelow (the iliofemoral ligament) and the Ligamentum Capitis Femoris namely the ligament of the head of the femur are the important stabilizers. Among the muscles it is the Gluteus medius and Iliopsoas which are the important stabilizers. Considerable force is necessary for a hip dislocation to occur (Trauma of severe magnitude).

Bigelow's Ligament—other names are Ligamentum Iliofemorale, 'Y' ligament, Bertin's ligament and Hipsiloid ligament. It is a Y shaped ligament with the apex attached to the anterior inferior iliac spine and the base diverges into a medial and lateral band and gets inserted to the intertrochanteric line, thus imparting a Y shape to the ligament. The medial part is attached to the lower part of the intertrochanteric line and the lateral part is attached to the tubercle at the upper part (Fig. 4.9).

Types of Dislocation

a. Anterior.
   - Obturator type (Fig. 4.10)
   - Pubic type
   - Perineal type

b. Posterior.
   - Gluteal type
   - Sciatic type
   - Ilial type

c. Central (Fracture dislocation).

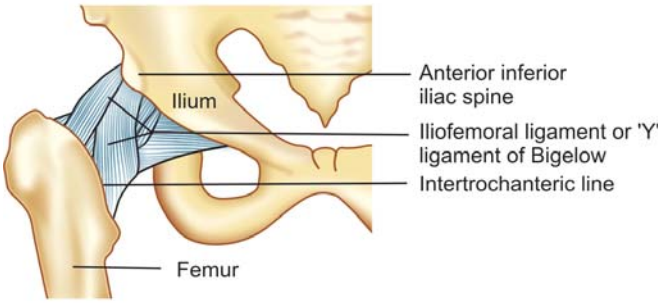


Figure 4.9
Showing ligaments supporting the hip joint

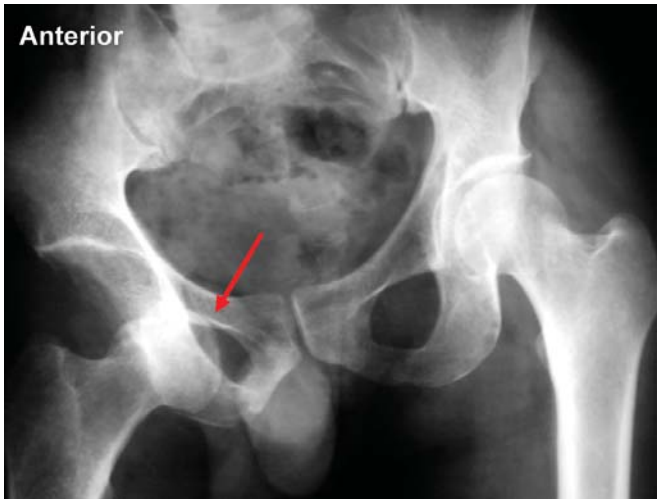


Figure 4.10
Radiograph showing obturator type of anterior dislocation of the hip. It is known as obturator type because the femoral head is in the region of obturator foramen.

Mechanism of Injury

Anterior Dislocation

It is the hyperabduction and the external rotation strain which causes the the anterior dislocation of the hip joint. In olden days when sea voyage was common the classical mechanism was that of having one leg on the boat and the other leg on the shore and the boat moves. The leg on the shore (the one which is more fixed) used to dislocate. These days skidding of a speeding two wheeler imparts a similar stress. Also in a landslide occurring in a mine where the miner is stooping forwards and working and on whose back a heavy mass of mud falls. In these circumstances the hip goes into hyper abduction and external rotation and dislocates (Fig. 4.11).

Posterior Dislocation

Classical mechanism is a 'Dash board injury'. In a road traffic accident, when there is head on collision the dash board hits the knee. The position of the lower limb at the time of impact is that of flexion, adduction and internal rotation. Thus an anterior impact of the crashing dash board on to the knee pushes the head of the femur out of the acetabulum in a posterior direction (Figs 4.12A and 4.13).

Central Dislocation

The classical mechanism is a side impact over the trochanter of the femur in an adducted limb (Fig. 4.12B).

Figure 4.11

Diagram showing mechanism of injury for anterior dislocation of hip joint.

Classification

Thompson-Epstein Classification

This classification system is based on radiographic findings . Based on this classification, the posterior dislocation of the hip is classified into 5 types.

- Type 1–Dislocation with or without minor fracture.
- Type 2–Dislocation with a single, large, fracture of posterior acetabular rim.
- Type 3–Dislocation with comminuted fracture of rim of acetabulum, with or without major fragments
- Type 4–Dislocation with fracture of the floor of the acetabulum.
- Type 5–Dislocation with femoral head fracture.

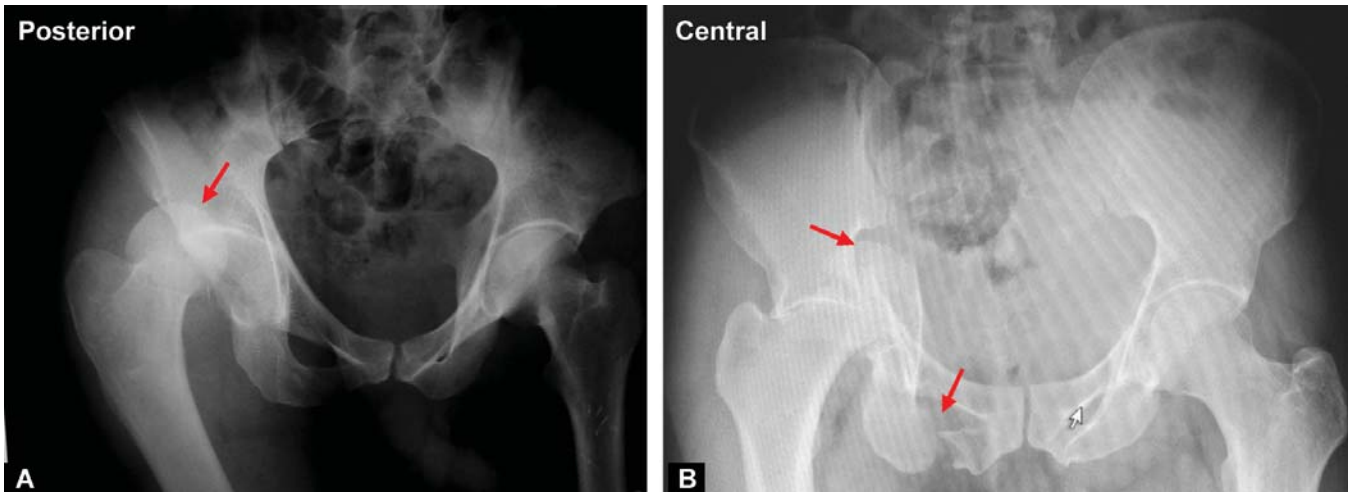
Steward and Milford classification:

This classification is based on functional hip stability.

- Type 1–Dislocation with no fracture or an insignificant fracture.
- Type 2–Dislocation with a single or comminuted posterior wall fracture, but a functionally stable hip.
- Type 3–Associated with gross instability of the hip joint secondary to loss of structural support.
- Type 4–Associated with femoral head fracture.

Diagnosis

- Diagnosis is made by clinical signs and X-ray.
- Classical clinical attitude of the lower limb in various types of dislocation is as follows:
  - — Anterior dislocation: Flexion, abduction and external rotation with apparent lengthening (Fig. 4.14A).
  - — Posterior dislocation: Flexion, adduction and internal rotation with apparent shortening (Fig. 4.14B).
  - — Central dislocation: Adduction and neutral/ external rotation with signs of fracture pelvis. Shortening is mimimum.

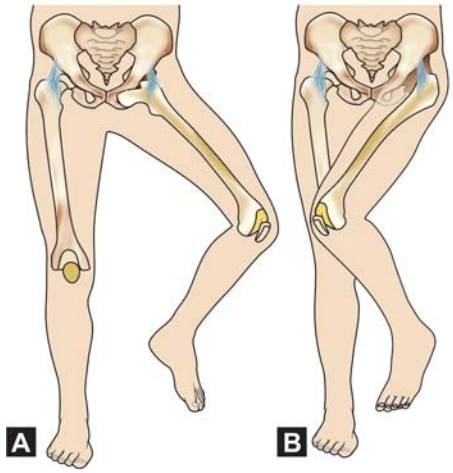


Figures 4.12A and B

(A) Radiograph of AP view showing traumatic posterior dislocation in a young patient. The femoral head is in the region of the sciatic notch. Hence it is known as Sciatic type (B) Radiograph of pelvis with both hips showing fracture of the pelvis with central dislocation of the hip. Note that the distance between the symphysis pubis and the greater trochanter is decreased when compared to the opposite side.

Figure 4.13

Mechanism of injury for posterior dislocation of hip joint. The picture shows the classical impact of a dashboard in a head on collision.



Figures 4.14A and B

Diagram showing the classical attitudes in anterior and posterior dislocation of the hip joint. (A) Anterior dislocation (B) Posterior dislocation (refer text).

- In addition to these classical attitudes, there will be severe painful restriction of all the movements of the hip joint.
- Radiograph confirms the diagnosis. The classical attitude of the limb is also made out in the AP view. Hence there is no need to take a lateral view unless an irregular dislocation is suspected.

CT and MRI are the other investigations that are recommended when there is fracture of the acetabulum or the femoral head for detailed evaluation and planning of appropriate treatment. They give valuable information about the shape and vascularity of the head of the femur.

Management

Most of the simple dislocations without fracture are reducible by closed methods except when the head is entrapped in the torn capsule or muscle mass. All the closed maneuvers are done under general anesthesia. The basic principle is to provide a counter force after achieving that position of the limb immediately after the dislocation. *If the force in one direction in a particular position of the limb has driven the head of the femur out of the socket (acetabulum) the force in opposite direction, when the limb is in the same position as it was immediately after the dislocation should bring it back into the sockect.* Following reduction the limb is immobilized in a Thomas' splint for a period of three weeks in order to achieve good soft tissue healing.

Anterior Dislocation

Allis' method: The position of the patient is supine. The hip and the knee is flexed, as it is, in the same abducted externally rotated position and traction is given along the long axis of the femur with assistant giving counter traction at the pelvis. Next a second assistant gives lateral traction to the thigh (femur) and the surgeon slowly internally rotates and adducts the limb. Finally when the dislocation is reduced he extends the limb (Fig. 4.15).

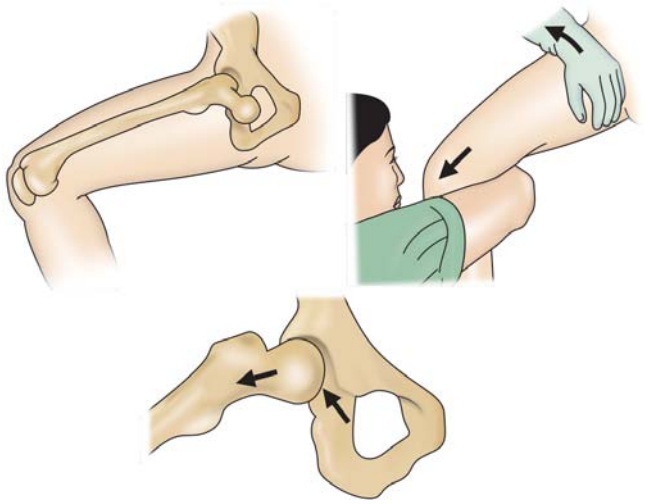


Figure 4.15

Showing the Allis' method of reducing the anterior dislocation of the hip. The arrows show the direction of the pull.

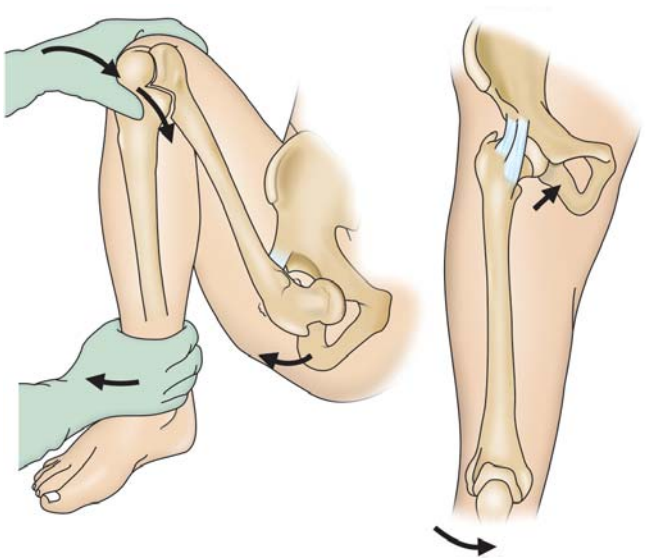


Figure 4.16
Showing the reverse Bigelow's method for reduction of anterior dislocation of the hip.

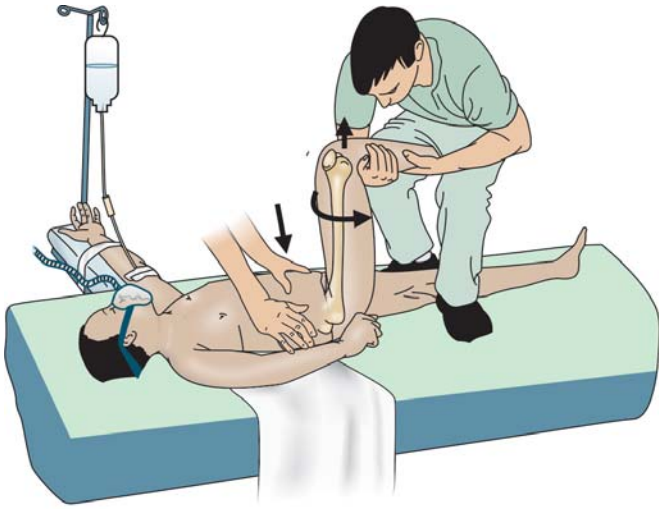


Figure 4.17
Showing Alli's method of reduction for posterior dislocation of the hip joint.

Reverse Bigelow's method
In this circumductory movement is given at the hip in an exactly opposite manner to that of reducing a posterior dislocation of the hip. That is the limb is circumducted in an inward direction.

The assistant stabilizes the pelvis and the surgeon does the maneuver beginning from the same flexed abducted and externally rotated position of the limb (Fig. 4.16).

Posterior Dislocation

Allis' method: Position of the patient is supine. Longitudinal traction is given along the axis of the limb in the same dislocated position of the limb with assistant stabilizing the pelvis(counter traction) and next with the traction force acting the limb is gently abducted, externally rotated and extended (Fig. 4.17).

Bigelow's method: The pelvis is stabilized by the assistant. The surgeon does the maneuver. The hip is gently flexed to 90°, rotated externally in a circumductory manner in an outward direction till reduction is achieved (Fig. 4.18).

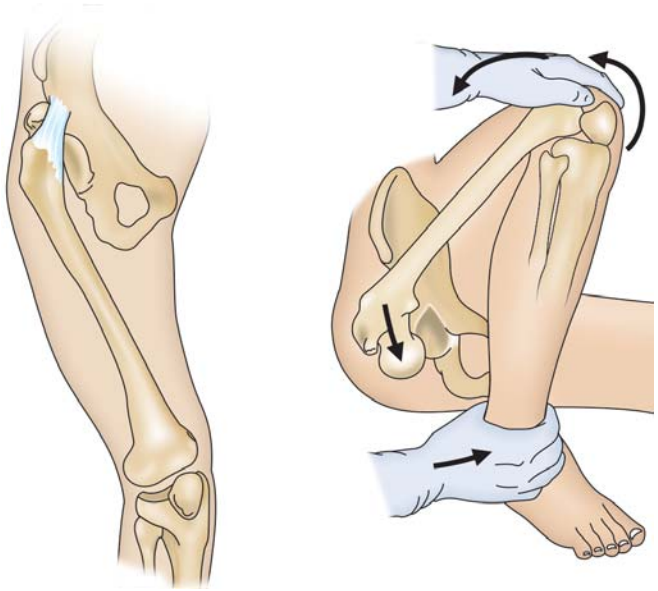


Figure 4.18
Showing Bigelow's method of reduction for posterior dislocation of the hip joint.

Stimson's method: Considered to be the least traumatic method as gravity is made use of in this method. With patient prone and the limb hanging out of the table with hip and the knee flexed to 90° traction force is applied just distal to the knee (over the upper third of the leg) by the assistant. The surgeon gives counter traction by stabilizing the pelvis by applying pressure over the sacrum with the hand. Then with the other hand the head of the femur is gently maneuvered into the acetabulum with the help of rotary movement of the limb by the assistant (Fig. 4.19).

Note – 'Y' ligament of Bigelow acts as an anchor and helps in the reduction of dislocation. When the Y ligament of Bigelow is torn the hip dislocation is known as irregular. The dislocation becomes highly unstable.

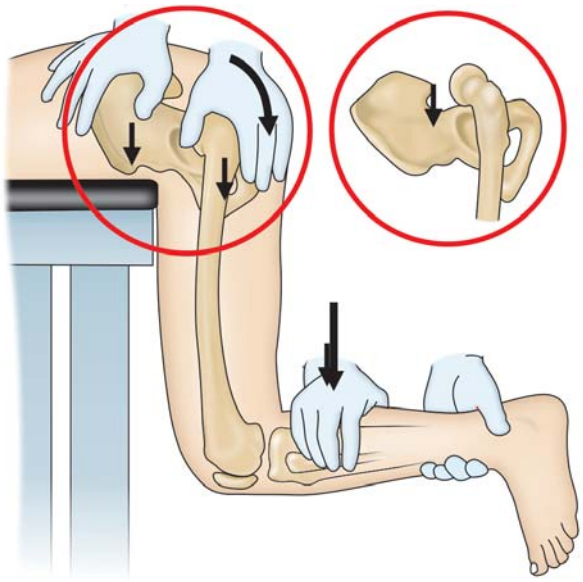


Figure 4.19

Showing Stimson's method of reduction for posterior dislocation of the hip joint.

Complications

1. Irreducible dislocation (Figs 4.20A and B).
2. Avascular necrosis of the head of the femur and late degenerative arthritis (Figs 4.20A and B).
3. Sciatic nerve palsy (about 10%)
4. Associated fractures, e.g. fracture of the head, acetabulum, neck of the femur, etc.
5. Fractures around the knee e.g. patella
6. Myositis ossificans (common after open reduction)
7. Recurrent dislocation (rare).

Revision Questions

Q. What are the basic types of dislocation?

Q. Write notes on–
(a) Pathological dislocation (b) Paralytic dislocation (c) Traumatic dislocation.

Q. What is Bigelow's ligament ? Discuss its role in hip dislocation.

Q. Discuss the pathoanatomy of the hip joint and classify hip dislocations. Explain in detail the different mechanisms responsible for hip dislocations.

Q. Discuss the different methods of reduction of posterior dislocation of the hip joint.

Q. Discuss the attitudes of the lower limb in different dislocations of the hip joint.

Essay Questions

Q. Discuss the mechanisms of injury, diagnosis and management of posterior dislocation of the hip joint. Enumerate the complications.

Q. Discuss the mechanisms of injury, diagnosis and management of anterior dislocation of the hip joint.

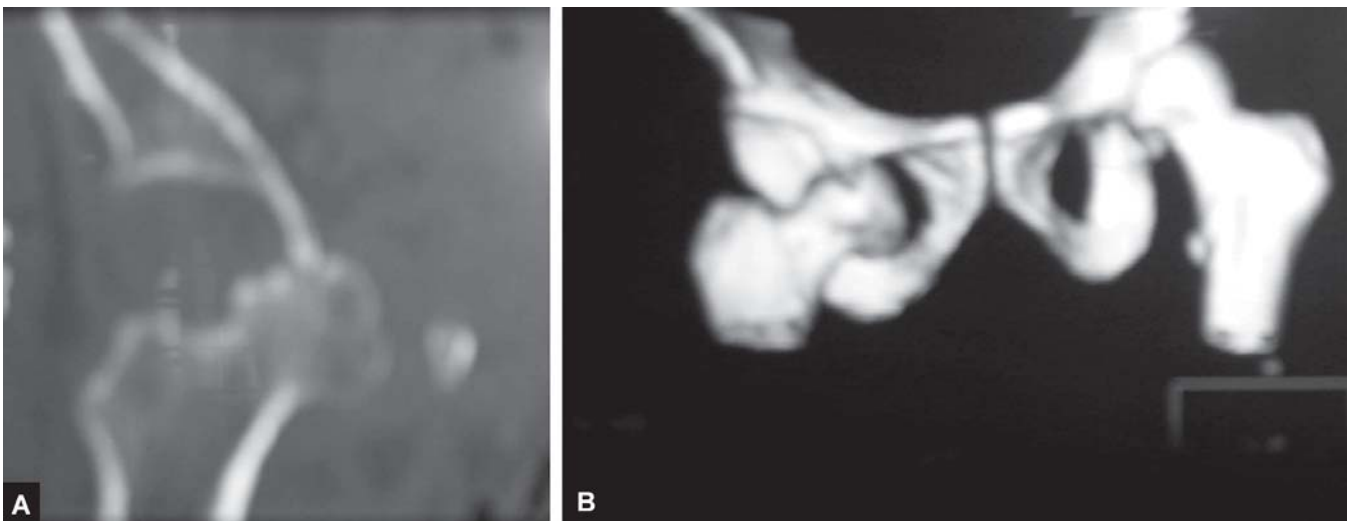


Figure 4.20A and B

Showing the CT image of an old unreduced anterior dislocation (4 months old) of the hip. Note the articular degeneration and the incongruity which is not in favor of open reduction.

Q. Discuss the role of CT scan in the evaluation of a hip dislocation. Discuss the management of a fracture dislocation of hip joint. Enumerate the complications.
Q. Discuss the mechanism of injury, the clinical features diagnosis and management of a central fracture dislocation of hip joint.

Dislocation of the Knee

One of the very serious injuries which is the result of a direct impact onto the front of the knee by a force of considerable magnitude. The cruciate ligaments are torn and so are one or both of the collateral ligaments. As a result the femur is pushed in a posterior direction over the tibia and a dislocation of the knee occurs.

Clinical Features

a. Bruising and contusion around the knee.
b. Presence of neurological and vascular deficit in the lower limb.
c. Signs of acute compartmental syndrome may be present.

Diagnosis

X-ray–gives a clear cut diagnosis. Always look for associated fractures, e.g tibial spine.

Treatment

It is an emergency. The dislocation has to be reduced as early as possible. Always keep a watch on vascular status of the limb. Insufficiency can develop any time within a week.

Plaster immobilization is to be avoided. Also the knee should never be immobilized in extension. If stability is essential, external fixator application is a better option. Ligament repair is not indicated in closed injuries. However, in open injuries repair of cruciates ligaments may be considered.

Essay Question

Q. Discuss the mechanism of injury diagnosis and management of a traumatic knee dislocation.
Q. What are the complications related to acute traumatic dislocation of the knee joint? Discuss their management.

Note: Management of vascular injury and compartmental syndrome is discussed in Chapter 2 under Volkmann's ischemia.

Further Reading

Shoulder Dislocation

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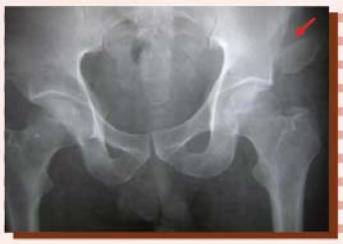


5

Fractures of the Pelvis

- *Structures Imparting Stability to the Pelvis*
- *Deforming Forces Responsible for Failure*
- *Classification*
- *Clinical Features and Investigations*
- *Treatment*

Formation of Pelvis

The pelvis is formed by the sacrum articulating with a pair of ilium ischium and pubis (united together to form hemipelvis) *on either sides.* Stability to the pelvis is imparted anteriorly by the pubic symphysis, with its strong fibrocartilagenous cuff and ligament. Posteriorly by the sacral articulation, with its strong ligaments, i.e. the anterior, interosseous and posterior sacroiliac ligament and the iliolumbar ligament. Additional stability is imparted by the muscles and soft tissues that are attached to the pelvis, (e.g. sacrospinous and sacrotuberous ligament postero-inferiorly, gluteal muscles posteriorly, iliopsoas anteriorly) (Figs 5.1A and B).

Thus it is an established fact that though the pelvis is the only link that connects the axial and the appendicular skeleton, *it has poor inherent stability and its stability entirely depends on integrity of soft tissues surrounding it and the strong ligament complex.*

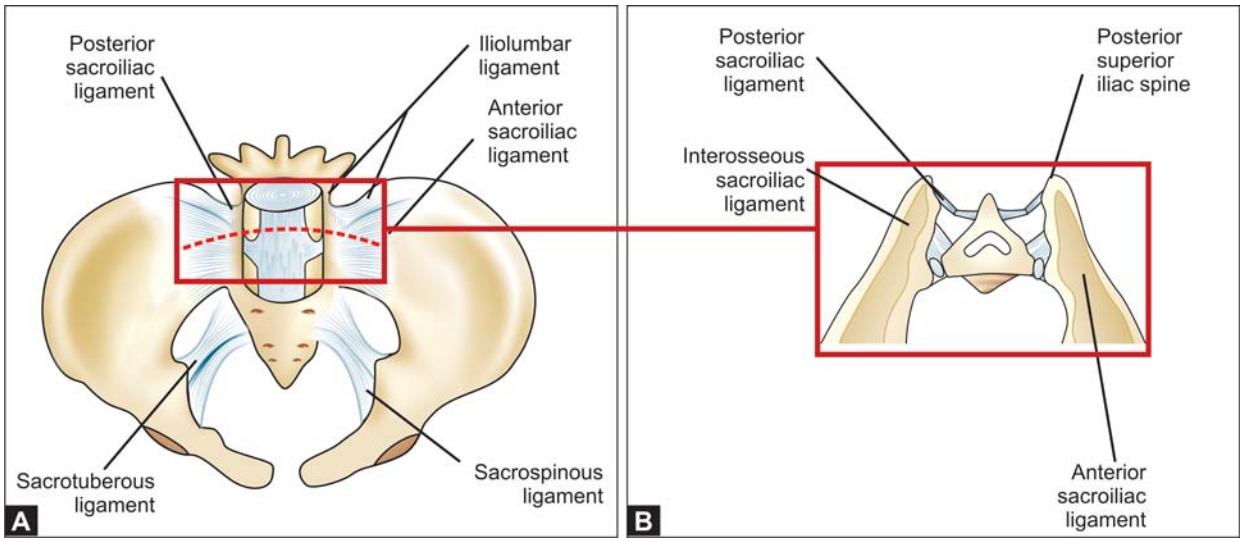
The pelvis has an inlet and an outlet and thus allows entry and exit of structures at the same time protecting them. It has room for accommodating viscera too.

For these reasons the injuries of the pelvis have to be considered very seriously as there can be associated vascular, visceral and neurological compromise leading to serious complications.

Further the acetabulum may also get involved in pelvic fractures. Failure to evaluate and treat these injuries adequately may result in serious impairment of the functions of the hip joint.

The pelvic girdle is formed by *5th lumbar vertebra, the coxal bones, the sacrum and coccyx* bound firmly by *capsule-ligamentous complex.*

The pelvic ring is formed by *the anterior arch* and *the posterior arch.*



Figures 5.1A and B

Showing (A) the supporting ligaments that impart stability to the pelvis. (B) The cross section of the inset showing the posterior stabilizers of the sacroiliac articulation.

The anterior arch comprises of *the pubic symphysis, the ilioischiopubic rami* and *the obturator foraminae*.

The posterior arch comprises of *the sacrum, the two iliac wings* and *the acetabulae.*

The anterior and the posterior arch together form 'The radiographic U'.

Mechanisms of Injury (Figs 5.2A to C)

The injurious forces responsible are four in number.

a. External rotation force.
b. Compression force.
   - Anteroposterior
   - Lateral
c. Vertical shear.
d. Combined.

External Rotation Force

Forced external rotation stress on the lower limb causes disruption to occur in an orderly manner from anterior to posterior described as follows.

Separation of symphysis pubis → opening of sacroiliac joint → stretching and disruption of sacrospinous ligament. (***Open book injury***): ***Open book injuries are unstable in external rotation.***

Compression Force

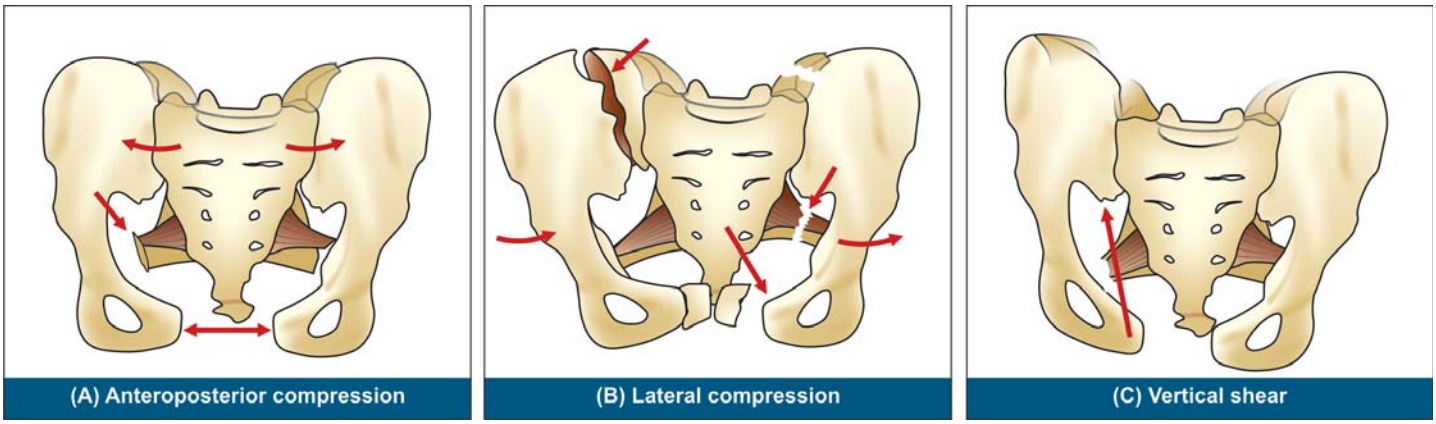
Anteroposterior

(Analyzed taking into consideration posterior failure, i.e. sacroiliac joint, as the final event.)

i. Without resistance from the opposite side, e.g. direct vehicular impact.
   The failure starts from anterior to posterior.
   Anterior failure pattern: Anteroposterior compression occurring in the midline central plane results in *bilateral open book injury* (rare).
   The posterior failure pattern: Is similar to open book injury. As a first step of failure it results in disruption of the sacrospinous ligament. Next it leads to opening of the sacroiliac joint/sacral crushing which is the final event in the ensuing failure.
ii. With resistance from the opposite side, e.g. caught between the wall and the vehicle.
   Results in very severe injuries. Visceral and vascular injuries are common. When there is resistance, anteroposterior compression occurring in midline central plane results in Butterfly like fragment comprising of all the four rami anteriorly and bilateral involvement of sacroiliac joint in the form of crushing/disruption, posteriorly.

Lateral

i. Without resistance from the opposite side, e.g. direct vehicular impact.



Figures 5.2A to C

Diagrams showing the nature of the fracture as per the direction of the force.

Anteriorly, results in closed book type of injury which manifests either as overlapping of symphysis pubis or ipsilateral/contralateral bucket handle bi-ramal fracture of the ilioischiopubic rami. Ipsilateral fracture of the ilium may also occur.

Posteriorly, results in either crushing of the sacrum or disruption of interosseous and posterior sacroiliac ligament occurs.

These closed book injuries are unstable in internal rotation.

ii. With resistance from the opposite side, e.g. caught between wall and the vehicle, fall of a tree or a heavy object over the pelvis in lying down posture.

Very severe crushing occurs involving opposite hemipelvis. Visceral, vascular and neurological injuries are common.

Compression impact with resistance results in very severe crushing of the pelvis which is many times not compatible with life because of associated vascular and visceral injuries. Hemorrhage is severe resulting in death even if timely medical aid is given. Morbidity rate is also quite high.

Vertical Shear

Results in vertical displacement of ipsilateral pelvic bone. ***These are unstable in both vertical and horizontal planes.*** Patterns of injury may be as follows:

i. Displacement occurring through disrupted symphysis pubis anteriorly and disrupted sacroiliac joint posteriorly (ligamentous disruption).
ii. Displacement occurring through disrupted symphysis pubis anteriorly and fractured ilium posteriorly.
iii. Displacement occurring through fractured ilioischiopubic ramus anteriorly and disrupted sacroiliac joint posteriorly.
iv. Displacement occurring through fractured ilioischiopubic ramus anteriorly and fractured ilium posteriorly.

Any of these four patterns may be seen depending on the direction of the force.

Combined

It is the result of mutiplanar stress causing ***high degree of instability*** (total instability). Shear, rotation, compression, translation, etc. more than one mechanism act together or in succession.

Classifications

Based on Tile's Classification (1995) as Follows

A. Rotationally and vertically stable fractures. These are subdivided into—

A1. Avulsion fractures that do not interrupt the pelvic ring (Fig. 5.3).

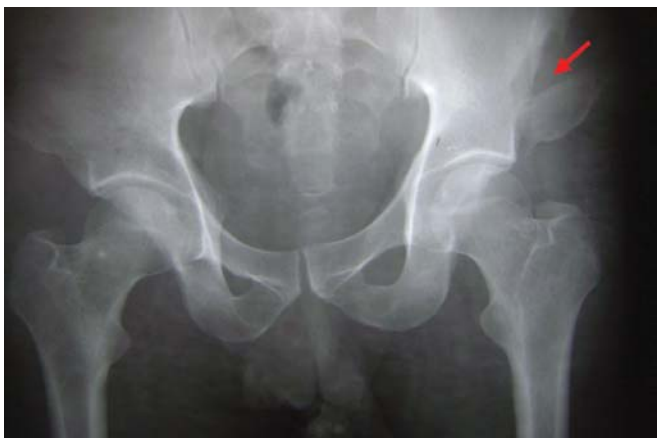
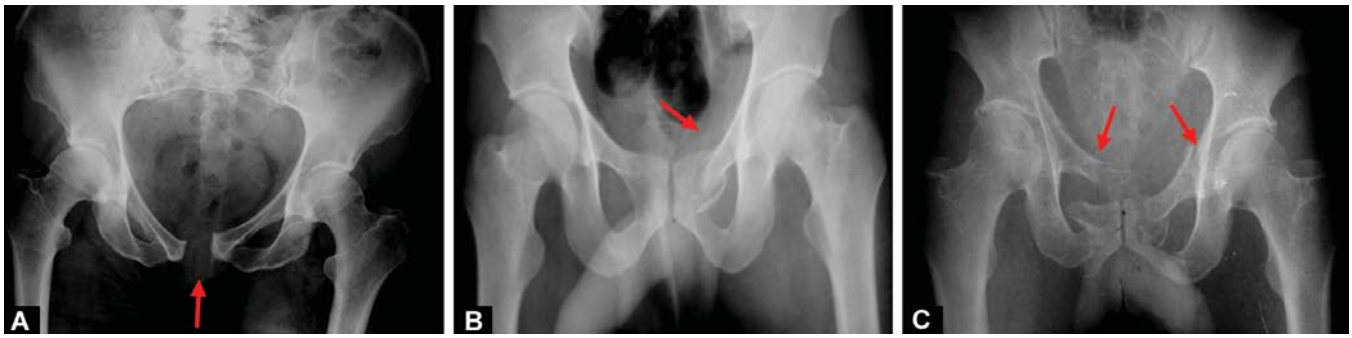


Figure 5.3

Radiograph showing Tile's A1 fracture.



Figures 5.4A to C

Radiographs showing Tile's B1, B2, B3 fracture.

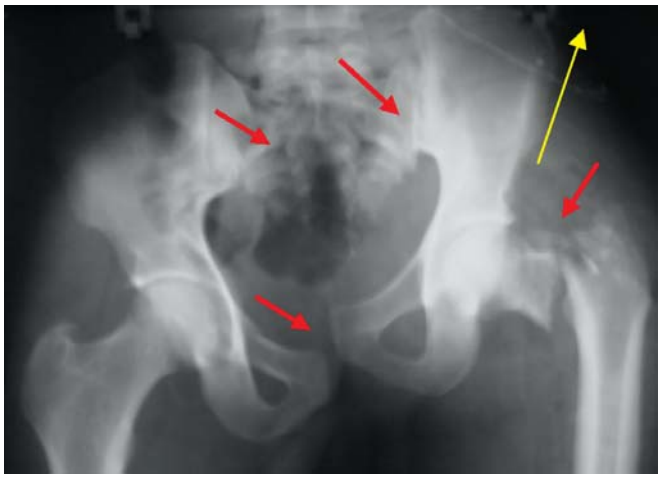


Figure 5.5

Radiograph showing Tile's C3 fracture. Note the presence of sacral fracture on the opposite side which classifies this as Type C3. Associated comminuted intertrochanteric fracture was an open fracture.

A2. Stable iliac wing fractures or minimally displaced pelvic ring fractures with minimal instability.
A3. Transverse fractures involving sacrum and coccyx.

B. Rotationally unstable, vertically stable fractures. Subdivided into (Figs 5.4A to C)—
B1. Open book (due to anteroposterior compression).
B2. Closed book (due to lateral compression).
B3. Bilateral type B injuries.

C. Rotationally and vertically unstable. Subdivided into—
C1. Unilateral.
C2. Bilateral with one side type B and contralateral side type C.
C3. Bilateral with both sides type C (Fig. 5.5).

Sacral Fracture Classification

Based on Denis' classification, these fractures are classified according to their zone of injury.

Zone I — Involvement of alar region of sacrum. (Neurologic complication least 6% approx).
Zone II — Involvement of sacral foramina. (Neurologic complication moderate 30% approx).
Zone III — Involvement of central canal. (Neurologic complication rate 60% approx. Also known for bowel and bladder involvement and sexual dysfunction).

Clinical Signs of Importance

1. Destot's sign: A large hematoma seen in the inguinal region or the scrotum.
2. Roux sign: Distance between the greater trochanter and the pubis is decreased.
3. Earle sign: A tender bony prominence or a large hematoma felt on per rectal examination.
4. Bleeding through urethra vagina and rectum indicate visceral complications.

Investigations

It is very important to assess a pelvic fracture both qualitatively and quantitatively. Hence proper radiological views are absolutely essential with special attention to know the disturbance in 'Radiographic U'.

a. Standard AP view
   - Identifies fractures.

b. Pelvic inlet view, discloses
   - AP displacement of pelvis
   - Inward/outward rotation of iliac wing

- Sacral impaction or alar fractures
- Avulsion fracture of the ischial spine.

c. Pelvic outlet view, discloses
- Superior displacement of pelvis
- Sagittal plane rotation
- True AP view of the sacrum
- Avulsion fracture of L5 transverse process.

d. Oblique views
- Evaluates hemipelvis.

In complex fractures CT scan and 3D reconstruction is necessary to plan accurate treatment.

Principles of Management

a. Carefully evaluate the mechanism of injury to know whether they are stable or unstable.
b. Immediate assessment of general condition of the patient and administration of resuscitative, life saving measures when necessary.
c. Emergency management of shock and hemorrhage and stabilization of other associated fractures when present.
d. Multidisciplinary approach when vascular, visceral and neurological injuries are associated.
e. Early reduction and stabilization of pelvic fractures to prevent further complications.

(Wrapping firmly in bed sheet: Pneumatic Antishock Garments PASG: Military antishock trousers MAST: Therapeutic embolization are some of the measures undertaken to stop hemorrhage).

Uncomplicated stable injuries (not involving the pelvic ring) are managed nonoperatively. Skin and skeletal traction are employed to reduce and immobilize the fracture or after reduction of the fracture under anesthesia.

Complicated and unstable injuries (involving and disrupting the pelvic ring) are stabilized surgically either by open reduction and internal fixation using reconstruction plates and screws or by using external fixator frames after reduction.

Some of the indications for the surgical procedure:
a. Diastases of pubic symphysis greater than 2.5 cm.
b. Sacroiliac joint dislocations.
c. Displaced sacral fractures.
d. Posterior or vertical displacement of the hemipelvis (>1 cm).
e. Rotationally unstable pelvic ring injuries.
f. Sacral fractures in patients with unstable pelvic ring injuries.
g. Displaced sacral fractures with associated neurologic injury.

Some of the contraindications for the surgical procedure:
a. Patients who are unstable and critically ill with multiple associated injuries.
b. Badly contaminated open fractures with inadequate wound debridement.
c. Crush injuries.
d. Morel-Lavalle lesion (closed degloving).

The lesion is diagnosed by fluctuation (fat necrosis) occurring under the skin of the involved area as a result of shearing of the subcutaneous tissue from the underlying fascia. It is associated with high rates of bacterial contamination.

Hence debridement and drainage before operative intervention is a must.

Complications of Fracture Pelvis

Early
a. Visceral, e.g. injury to bladder, bowel and urethra, other abdominal organs and chest.
b. Vascular, e.g. internal iliac artery, superior gluteal artery.
c. Neurological, e.g. sacral fractures.
d. Shock and hemorrhage.

Late
Malunion with disrupted pubic and sacroiliac articulation resulting in severe morbidity and impairment of physical function.

Malgaigne Fracture (Fig. 5.6)

It is a vertical shear fracture described by Malgaigne in the year 1885. It is highly unstable and characterized by anterior failure occurring through both the rami or through the symphysis pubis and posterior failure (with a massive disruption) occurring through the sacrum or the sacroiliac joint or the ilium consequent to severe trauma. Inadequate treatment results in significant late complications which are as follows:
a. Paresthesias in the ipsilateral lower extremity.
b. Gait disturbances with significant limp.
c. Severe low back pain.
d. Groin pain.
e. Neurologic abnormality.
f. Leg length discrepancy.
g. Pelvic obliquity.

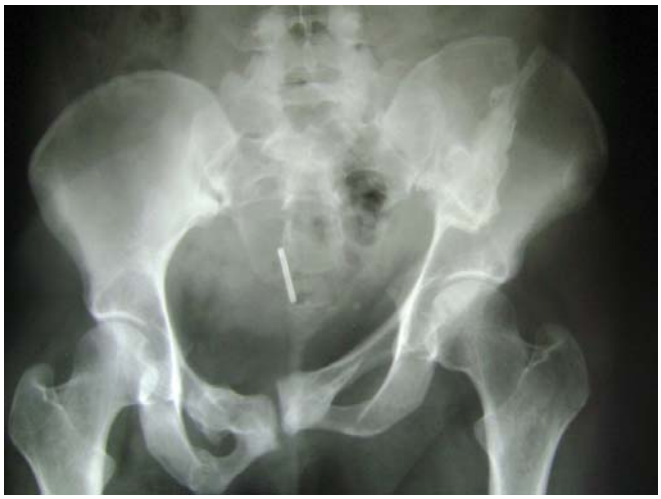


Figure 5.6

Radiograph showing a malunited Malgaigne fracture. Note the displacement as a result of vertical shear and malunion in the displaced position.

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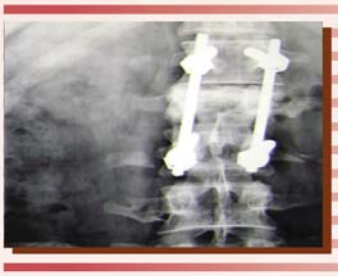


6 Spinal Injuries

- *Structure and Functions of the Spinal Column*
- *Mechanisms of Injury and Concepts*
- *Specific Types of Fractures and Incomplete Cord Injury Syndromes*
- *Management*
- *Signs of Grave Prognosis and a Ray of Hope*

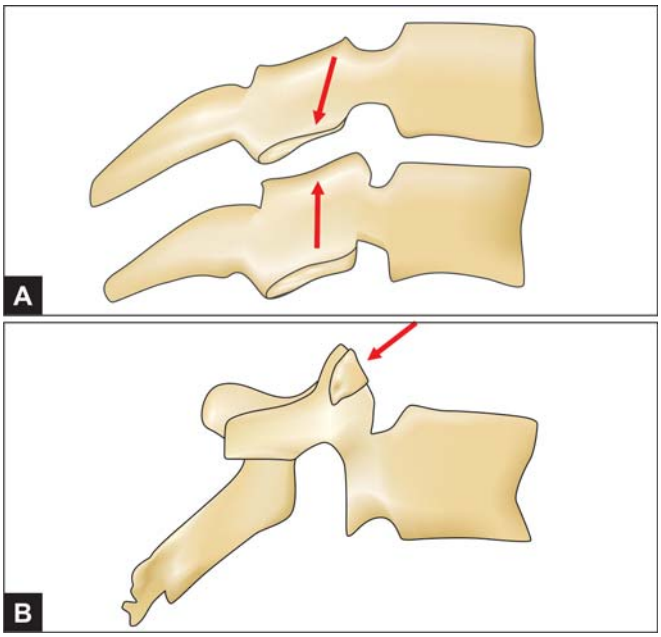
Spine is a marvel of creation. It has many functions, yet remains stable during the process and protects the vital structures throughout.

Structure of the Spinal Column

The basic unit of this structure is a vertebra. At birth there are 33 vertebrae, 7 cervical, 12 thoracic, 5 lumbar, 5 sacral and 4 coccygeal. In adults there are only 24 vertebrae because of the fusion occurring in the sacral and coccygeal region resulting in the formation of the bone sacrum and coccyx. The curvature of the spinal column too changes from straight at birth to S-shaped one in adulthood.

Each vertebra consists of a body and a posterior arch. The body is made up of a block of spongy bone and between the bodies of the two vertebrae is the fibrocartilagenous intervertebral disc which binds the bodies of the adjacent vertebrae. The posterior arch consists of a spinous process, two transverse processes and a lamina. The arch is attached to the body by two pedicles. Between the body and the posterior arch is the central canal which harbors the spinal cord and the nerve roots. The articular facets on the posterior arch help in the articulation of the adjacent vertebrae. Thus, the spinal column is formed.

Additional support and stability is given to the spinal column by the ligaments. They are from before backwards, the anterior longitudinal ligament binding the vertebral body anteriorly, the posterior longitudinal ligament binding the vertebral body posteriorly, ligamentum flavum binding the adjacent laminae, interspinous ligament extending between the spinous processes and binding them and the supraspinous ligament extending over the spinous processes and binding



Figures 6.1A and B

Showing the direction of facetal joint in (A) cervical and (B) thoracic vertebra.

them. (The names themselves describe their location) (Fig. 6.2).

Structures that Impart Stability to the Spinal Column

Anterior Interbody Synarthrosis

Comprises of the intervertebral disc binding the adjacent bodies of the vertebra, and the anterior and posterior longitudinal ligaments.

Posterior Ligament Complex

The posterior ligament complex, binds the posterior elements (posterior arch) and consists of the supraspinous ligament, the interspinous ligament and the ligamentum flavum.

The Facetal Joints (Figs 6.1A and B)

The direction of the facet joint is almost transverse in the cervical spine, anteroposterior in the thoracic spine and in the lumbar spine, it is facing mediolateral. Thus, the facetal articulation in the cervical spine is less stable when compared to thoracic and lumbar articulation.

Functions of the Spinal Column

1. Provides protection of the spinal cord and associated nerves.

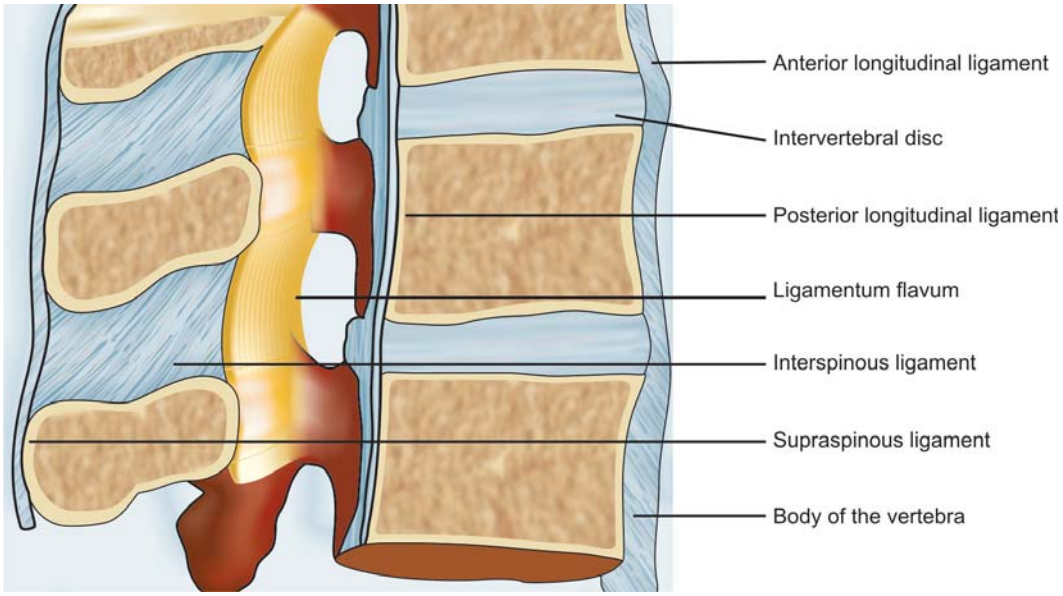


Figure 6.2

The picture shows the ligamentous structures which impart stability to the the spinal column.

2. Supports the body frame in an upright position.
3. Supports vital organs.
4. Provides a structural foundation for the shoulder girdle and the pelvic girdle.
5. Acts as shock absorber on load-bearing.
6. Provides attachment to the thoracic cage.
7. Provides attachment to the muscles.
8. Allows movement.

Hence, spinal injuries result in serious disability, as it affects several functions.

Spinal Injury

The spinal injury has two components.

a. Injury to the spinal column. Bony injury.
b. Injury to the spinal cord and nerve roots. Neural injury.

It is possible to have spinal column injury without spinal cord injury and vice versa, i.e. spinal cord injury without column injury or both spinal column and spinal cord injury.

Dennis' Three Column Concept (Fig. 6.3)

The spine is divided into three columns namely anterior, middle and posterior by Dennis. Failure of one or more column occurs in spinal injury when deforming forces act by causing axial compression, axial distraction or translation.

Anterior column: Consists of anterior longitudinal ligament, anterior half of the vertebral body and the anterior portion of annulus fibrosus.

Middle column: Consists of posterior longitudinal ligament, posterior half of the vertebral body and the posterior portion of annulus fibrosus.

Posterior column: Consists of the posterior elements (neural arch) with ligamentum flavum, interspinous and supraspinous ligament, facetal joints and the capsule.

Mechanisms of Spinal Injuries

Several mechanisms can disrupt the spinal column causing fracture, dislocation or fracture dislocation. Depending on the severity of injury and the compromise of stabilizing structures the injuries are diagnosed as *stable* or *unstable (Nicoll's classification).*

Basically these injury mechanisms are classified based on the deforming effect of an indirect impact, direct impact or a traction force on the spine.

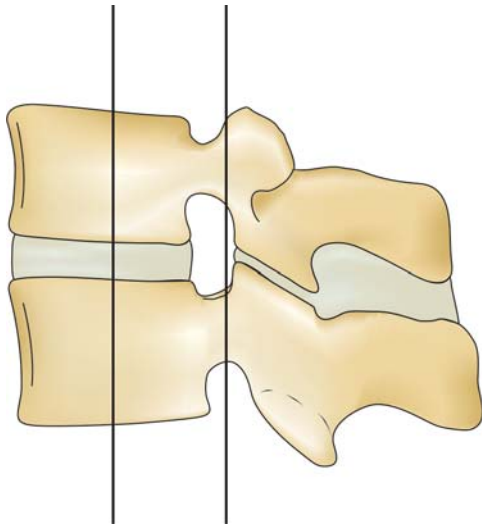


Figure 6.3
Showing the Dennis' three column concept.

Indirect Mechanism

Injuries as a result of this are classified as:

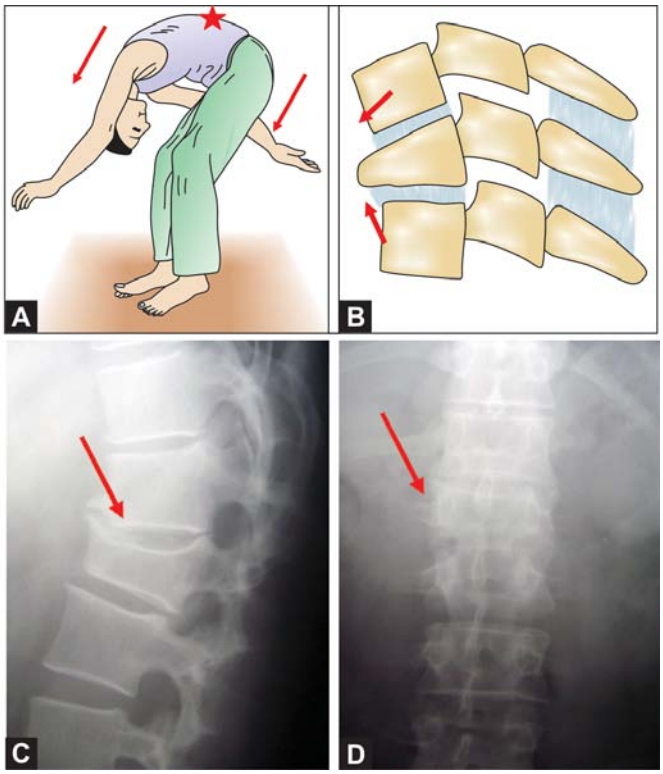
a. Flexion injury.
b. Flexion rotation injury.
c. Hyperextension injury.
d. Vertical compression injury.
e. Shearing.
f. Flexion distraction.

Flexion Injury

In this is the deforming force is unidirectional. It exerts compression stress (closing stress) on the anterior structures and results in a wedge compression fracture of the body of the vertebra. The posterior elements do not fail. The injury is diagnosed as unstable when there is evidence of compression fracture which is more than 50% of height of the body. Otherwise the injury is considered as stable (Figs 6.4A to D).

Flexion Rotation Injury

In this the force is multidirectional. Thus in addition to fracture of the body of the vertebra and injury to the posterior elements, (posterior ligament complex) the torsional stress in this mechanism disrupts the facetal articulation and causes dislocation. This results in fracture dislocation. Injury is highly unstable and incidence of spinal cord injury is high (Figs 6.5A to D).



Figures 6.4A to D

Pictures and radiographs showing pure flexion injury of the spine resulting in wedge compression of the vertebra. If the compression is less than 50% of the original height of the vertebra the injury is considered as stable.

Hyperextension Injury

In this too the force is unidirectional. It exerts distraction stress on the anterior structures. Thus, the failure is mainly limited to anterior interbody synarthrosis. Sometimes a small triangular fragment of bone is avulsed from the inferior vertebra due to the pull of the anterior longitudinal ligament. This feature is considered as a tell tale evidence of a hyperextension injury (Figs 6.6A and B).

Vertical Compression Injury

In this the force is axially transmitted and between the two vertebrae the involved vertebra gets compressed axially and bursts into fragments. The disc material and fragments of bone are retropulsed into the spinal canal. Burst fractures are unstable fractures and even if immediate neurological deficit is not observed, they tend to cause chronic instability pain. The risk of ensuing neurological deficit always exists (Figs 6.7A and B).

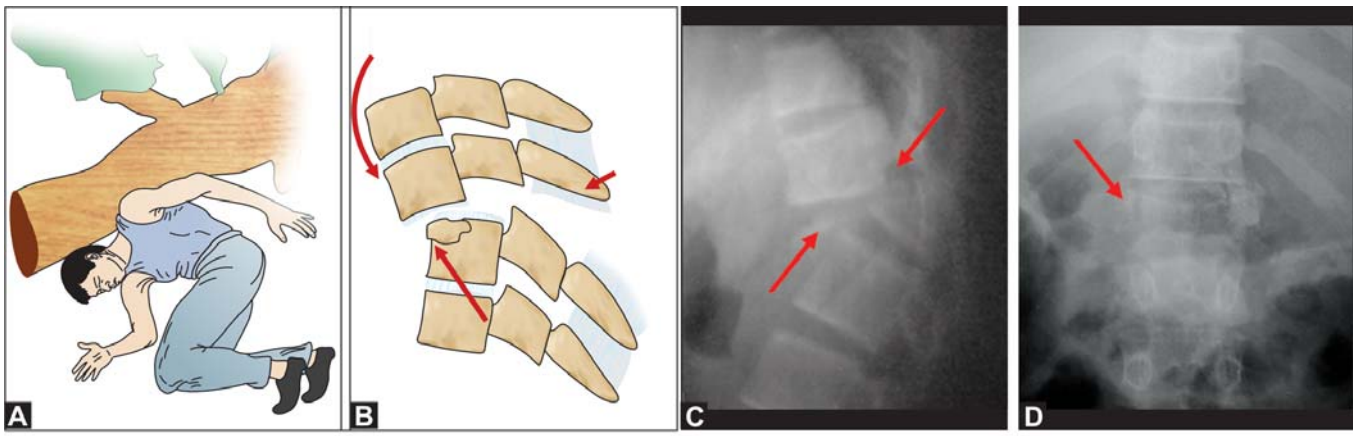
Shearing Injury

In this the force is transmitted transversely through the anterior interbody synarthrosis and posterior ligament complex causing dislocation at the level of injury. Fracture of the pedicles and facetal joint also is observed. Highly unstable and spinal cord injury is common.

Flexion Distraction Injury

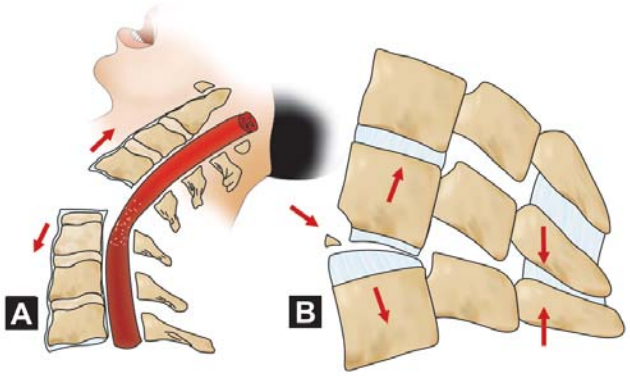
In this the fulcrum of flexion is anterior to the body of the vertebra. The force may pass entirely through the bone (Chance fracture) or through the posterior ligament complex.

Chance fracture (injury entirely through the bone) is stable after reduction. Injury through the ligament complex need surgical stabilization (Figs 6.8A and B).



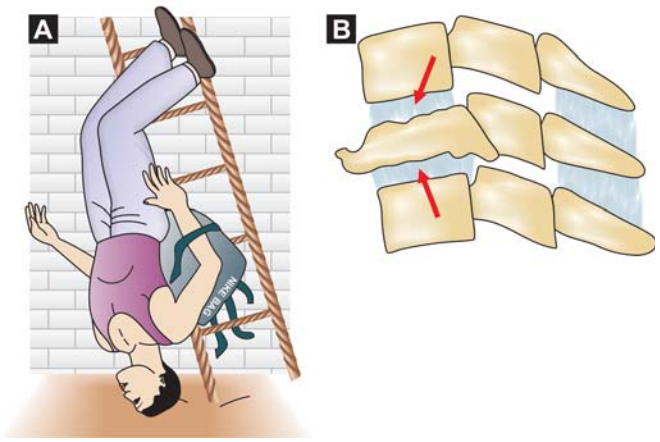
Figures 6.5A to D

Showing pictures and radiographs of flexion rotation injury resulting in a fracture dislocation. The rotational element in the mechanism causes disruption of the interbody synarthrosis, the facetal articulation and the posterior ligament complex.



Figures 6.6A and B

Showing the hyperextension injury. Note the direction of forces and site of distraction and compression. The small triangular fragment avulsed from the upper vertebral body when seen in the radiograph, indicates hyperextension injury.



Figures 6.7A and B

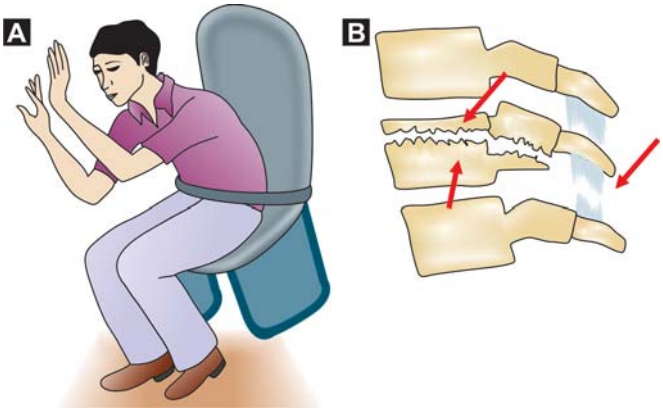
Showing the burst fracture due to a vertical compression injury. The classical example is a vertical fall from a ladder or a heavy object falling on the vertex.

Direct Mechanism

Direct penetrating injury, e.g. fire arms and knives.

Traction Injuries

In this the muscle and ligaments pull the bone and result in avulsion fracture, e.g. transverse process fracture, spinous process fracture.



Figures 6.8A and B

Showing 'Chance fracture' (jack knife fracture) secondary to 'Lap Seat Belt' injury.

Specific Types of Spinal Column Injury

Jefferson's Fracture

Named after Goeffrey Jefferson, a British neurosurgeon who reported four cases in the year 1920.

It is a four part burst fracture of the C1 (the Atlas) vertebra involving the anterior and the posterior arch as a result of axial compression. The variant can occur as two or three part fracture, e.g. diving into a shallow pool, impact of the head against the roof of a vehicle, fall on the vertex, etc.

The patient presents with pain in the neck.

Neurological deficit is rare except when vertebral artery is involved which results in Wallenberg's syndrome characterized by ipsilateral involvement of cranial nerves, Horner's syndrome, ataxia and loss of contralateral pain and temperature sensation.

Treatment depends on whether the injury is stable or unstable. This is evaluated by the amount of injury to the anterior arch and the intactness of the transverse ligament. Stable injury needs prolonged immobilization in collar or halo traction (at least for 3 months). Unstable injury is treated by fusion of the first three cervical vertebrae (Fig. 6.9).

Hangman's Fracture

This fracture traditionally occurs after judicial hanging with the noose being placed below the chin. Hence, this name is given.

It is fracture of both the pedicles or the pars interarticularis of C2 (the Axis) vertebra as a result of distraction that occurs

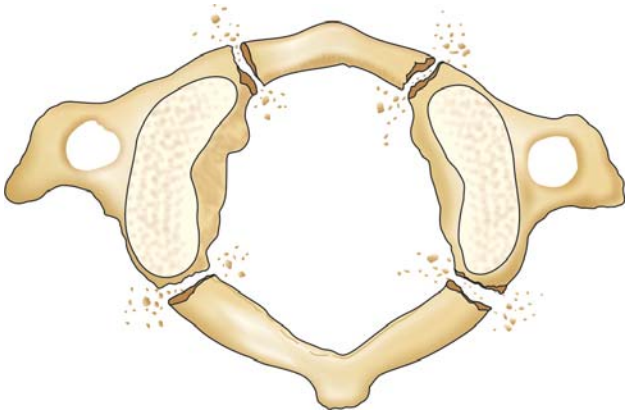


Figure 6.9
Picture showing the Jefferson's fracture.

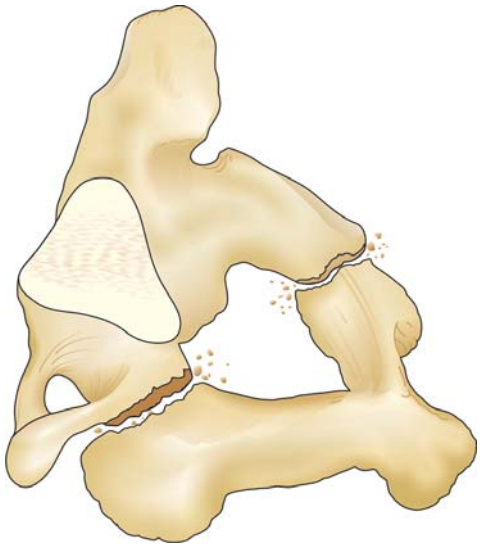


Figure 6.10
Picture showing the Hangman's fracture.

after the drop when the neck is forcibly hyperextended by the weight of the body. The common sign is constricted pupil on the ipsilateral side due to injury to the sympathetic trunk (Horner's syndrome) (Fig. 6.10).

Clay Shoveller's Fracture

First seen among Australian Clay Shoveller's who used to lift heavy loads of Clay. The mechanism is avulsion injury. As a result avulsion fracture of the spinous processes occurs from C6 to T1. Treatment is to immobilize the area with collar till good union is seen. Detailed evaluation for stability by taking flexion/ extension radiographs, CT, MRI is necessary when the fracture involves multiple vertebrae, the laminae and the facetal joints and should not be loosely branded as Clay Shoveller's fracture on the basis of spinous process fracture.

Chance Fracture (Lap Seat Belt Injury)

It was described by GQ Chance in 1948. It was common in motor vehicle accidents, when the vehicle was involved in a head on collision and the passenger was wearing a lap seat belt. The incidence has sharply fallen since the implementation of shoulder restraint system. The injury occurs as a result of violent forward flexion. The posterior elements namely the spinous process, lamina, the pedicle and the posterior part of the vertebral body fail as a result of distraction force resulting in characteristic transverse fracture whereas the anterior portion of the vertebral body fractures as a result of compressive force. This injury, when the entire component is bony and stable, heals uneventfully with immobilization.

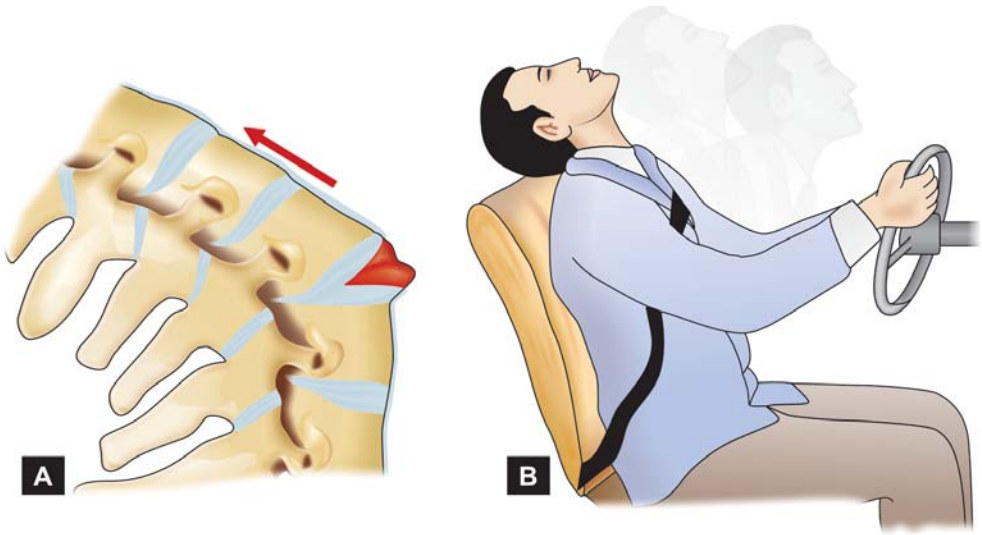
But if the posterior injury is that of failure of posterior ligament complex even though the anterior injury is bony, the injury is unstable. So also is pure ligamentous disruption. Incidence of cord involvement is high in these injuries which occurs as a variant of classical Chance fracture. These unstable injuries need surgical stabilization (Figs 6.8A and B).

Whiplash Injury

Common in road traffic accidents when there is rear impact (rear- end collision). The neck is violently hyperextended and flexed. If the impact is less severe it results in only soft tissue injury. If the impact is severe it results in injury to intervertebral disc, the facetal joints, ligaments, the muscles and the nerve roots. Concussion injury to the brain is also common. Depending on the severity, the patient may present with following symptoms.

i. Pain and stiffness in the neck.
ii. Limitation of movements.
iii. Headache, dizziness, nausea, (result of concussion).
iv. Hoarseness of voice, (involvement of recurrent laryngeal nerve).
v. Difficulty in swallowing, (stretching and contusion of esophagus).
vi. Pain in the back and the shoulder, (radiating pain).
vii. Pain and paresthesia (due to involvement of nerves).

Symptoms may develop as early as within 2 hours to as late as 8 days.



Figures 6.11A and B
Pictures showing the mechanism of an hyperextension/ whiplash injury.

Radiographic evaluation is a must and in patients having symptoms of severe injury CT; MRI is indicated.

Soft tissue whiplash injury invariably heals in 2-3 weeks with immobilization in collar. Severe forms may cause residual, mild degree of pain to remain (Figs 6.11A and B).

Spinal Cord Injury

Spinal cord injury can be *complete or incomplete*. *Complete transection* of the cord results in *Quadriplegia in cases of cervical spine injury* and *Paraplegia in cases of thoracic spine injury* and is characterized by *complete sensory loss, motor paralysis and paralysis of urinary bladder and bowels.*

Because the spinal cord ends at the lower border of L1 vertebra any *injury at or below the level of L1 will result in Conus medullaris and/ or 'Cauda Equina' lesion.*

For the same reason the level of the column injury does not correspond with the level of cord injury. Hence, a formula is derived to determine the level of cord injury.

In the cervical spine add one to the level of the column injury, to determine the level of cord injury. In the upper thoracic spine add two and in the lower thoracic add three to determine the level of cord injury.

Incomplete transection of the cord *results in various types of syndromes.*

Anterior Cord Syndrome

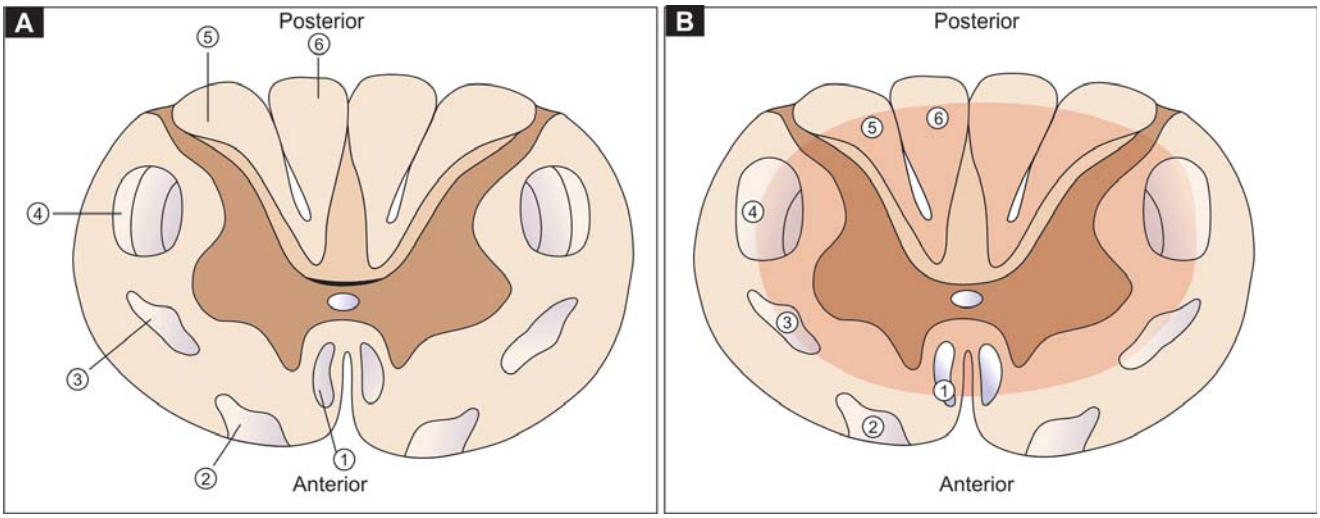
In this the *anterior portion of the spinal cord is involved* due to direct injury by a bony fragment of a fractured vertebra, intervertebral disc or at times injury to the anterior spinal artery (loss of blood supply). Neurological involvement *manifests with complete motor loss, loss of pain and temperature discrimination below the level of injury preserving (intact) deep touch, position and vibration sense. Prognosis is poor.* Some sensory recovery can occur but motor paralysis is generally permanent.

Central Cord Syndrome

In this the *central portion of the spinal cord is involved due to pinching* of the cord between the vertebral body anteriorly and the ligamentum flavum posteriorly. *Hematomyelia* occurs followed by *destruction of the grey matter of the central spinothalamic and pyramidal tract.* Upper extremities are more involved than the lower. Common in elderly with degenerative changes in the cervical spine (spondylotic spine). *Mechanism of injury is hyperextension.* Around 50% of the patients show functional recovery (Figs 6.12A and B).

Brown-Séquard's Syndrome

In this *one-half of the cord is involved*. Manifests with *complete paralysis of the ipsilateral side and hypoesthesia*



Figures 6.12A and B

Showing cross-section of the spinal cord and the position of the different tracts in the spinal cord; and their involvement in different types of incomplete cord syndromes (B) (refer text); 1- Anterior corticospinal tract; 2- Anterior spinothalamic tract; 3- Lateral spinothalamic tract; 4- Lateral corticospinal tract; 5- Fasciculus cuneatus; 6- Fasciculus gracilis. (Upper limb portion is the most medial part nearer to the midline followed by the trunk and then the lower limbs).

of the contralateral side. Functional recovery generally occurs and patients become ambulant.

Management of Spinal Injuries

Emergency Management

At the Site of the Incident

Attempt should immediately be made to immobilize the spinal column. If collars, braces and jackets are available to be made use of immediately. If not the patient may be strapped to a wooden plank, e.g. a detached door or a board and carried or transported on a stretcher. Sand bags may be kept on either side of the neck during transportation if cervical spine injury is suspected, to prevent rotation of the neck.

Medical Management

To be instituted as early as possible. Patient is advised not to take any thing orally.

Measures

i. Treatment of spinal shock with adequate intravenous fluids and vasopressors.
ii. Treatment of pain by giving analgesic injections.
iii. Catheterization of the bladder and recording of urine output.
iv. Prophylactic antibiotic therapy to prevent infection.
v. Methylprednisolone injection as a bolus dose of 1.5-2.0 gm is given at earliest if spinal cord injury is suspected to reduce the cord edema and further compromise.
vi. Assistance of breathing when required.
vii. Transportation to a center having facilities for the treatment of spinal injuries, at earliest.

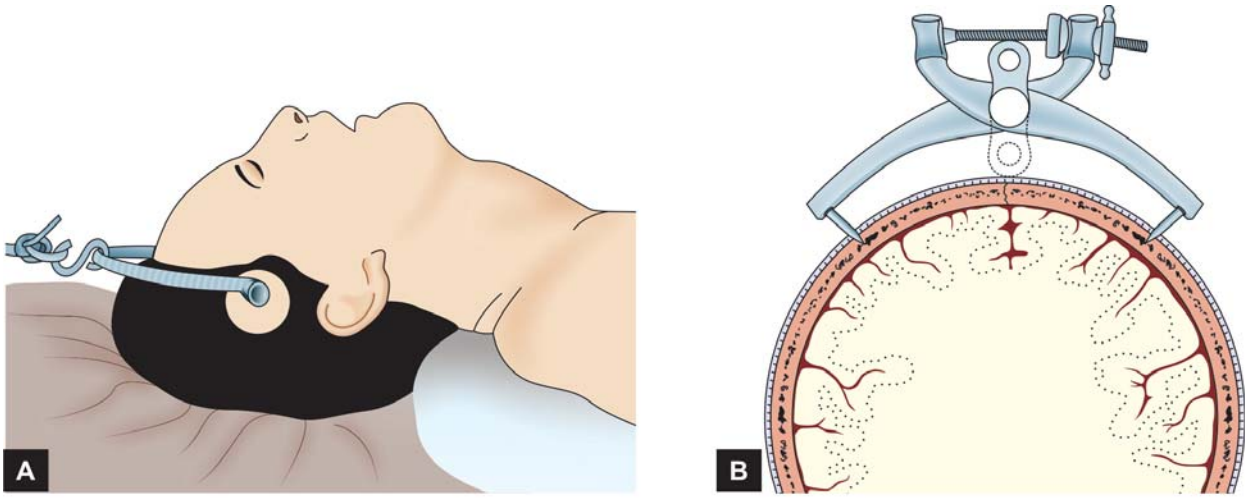
Definitive Management

The definitive management starts only after initial stabilization of the general condition of the patient and after taking appropriate X-rays to assess the spinal column injury and MRI to assess in detail the nature of both column and the cord injury. Dynamic flexion extension X-rays may be necessary when the static X-rays are normal but there is high degree of suspicion of ligamentous disruption and presence of instability.

Definitive management depends on two factors:

a. Whether the injury is stable or unstable.
b. Whether there is cord injury or no cord injury.

Loss of height of the vertebral body which is more than 50%, injury to posterior ligament complex and facetal joints and presence of malalignment are the indicators of instability.



Figures 6.13A and B

Showing the picture of skull traction. Note that the sharp point of the tongs engage in the diploe and do not penetrate the inner table of the skull.

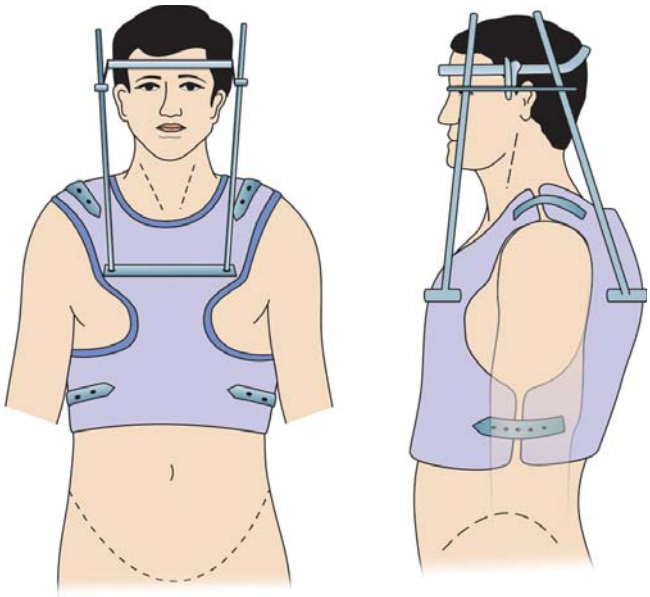


Figure 6.14

Showing the picture of halo body orthosis.

Non-operative management: Indicated only in stable spinal column injury with irrecoverable cord injury or without cord injury. Measures include immobilization in various forms of plaster jackets, spinal braces and in cervical spine injuries skull traction (Figs 6.13A and B), halo-pelvic traction (Fig. 6.14), etc.

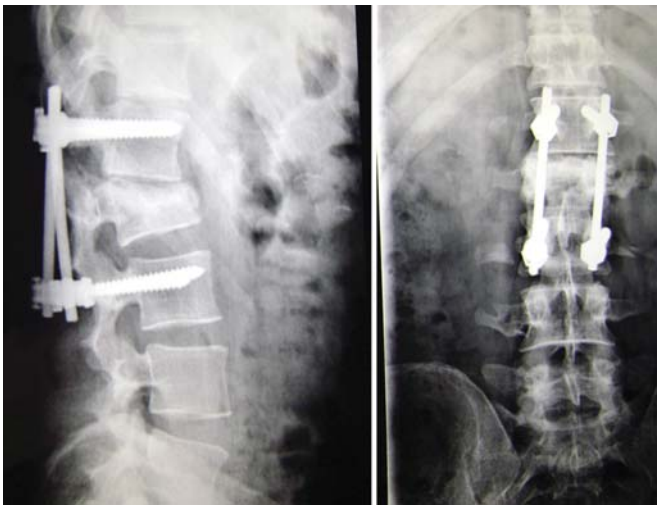


Figure 6.15

Radiograph after fixation of a fracture dislocation at L1-L2 with pedicular screws.

Operative Management (Fig. 6.15)

Indicated in all unstable spinal injury with or without cord injury. Aim is not only to stabilize the spine immediately but also to take away the compressive pathology on the cord and help in its recovery. Early mobilization is possible after surgical stabilization which prevents all the complications of prolonged confinement to the bed, e.g. bed sores, hypostatic pneumonia, thromboembolism, etc.

Procedures

- Posterior decompression and instrumentation and fusion.
- Anterior decompression and fusion.
- Combined posterior and anterior instrumentation and fusion.

Management of Established Paralysis (Quadriplegia and Paraplegia)

Basic principles involved are to take care of:

a. The paralyzed bladder and the paralyzed bowel.

In the stage of spinal shock there is flaccid paralysis of the bladder. Hence needs catheterization for emptying of urine. Once, the spinal shock passes off a neurogenic bladder develops.

Types of neurogenic bladder (Fig. 6.16)

i. *Automatic bladder:* This type of bladder is seen in complete transection of the spinal cord. In this the bladder is devoid of inhibitory or facilitatory control from the higher centers in the brain. Hence, behaves independent of the higher centers. When the bladder is filled with urine it sends afferent sensory signals to lower centers of micturition situated in the S2, S3, S4 segments of the spinal cord which responds by sending efferent motor signals to the detrusor muscle in the bladder wall. The reflex arc becomes complete and the detrusor contracts powerfully resulting in complete emptying of the bladder. No residual urine is left in the bladder. Hence, there is less chance of developing complications and sequale of urinary tract infection.

ii. *Automatic bladder:* This type of bladder is seen when the lesion is affecting the nerve roots after the spinal cord ends at lower border of L1, e.g. conus medullaris and cauda equine lesion.

The bladder is cut off from the lower centers of micturition situated in the S2, S3, S4 segments of spinal cord. No pathway is available for transmission of afferent sensory signals to lower centers when the bladder is filled with urine. Hence, emptying has to occur only with the help of local myoneural stretch reflexes situated in the detrusor muscle. Emptying is less forceful and is incomplete. There is always residual urine in the bladder. Dribbling may occur. High chance of developing all the complications and sequel of urinary retention and recurrent urinary tract infection.

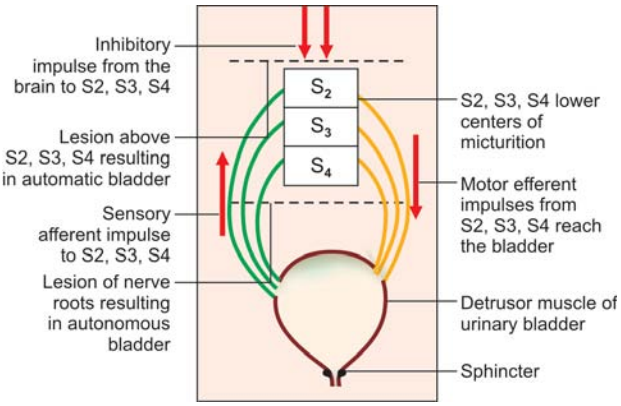


Figure 6.16

Diagram showing the normal mechanism of voiding of urine and the level of lesions responsible for the development of automatic and autonomous bladder.

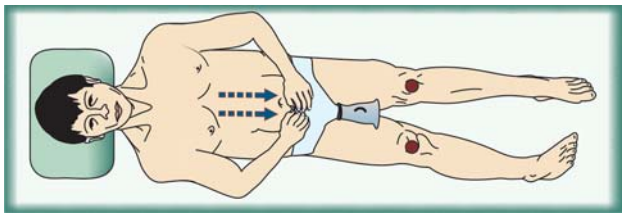


Figure 6.17

Showing the picture of Crede's maneuver. Firm suprapubic pressure is applied to express the urine from the bladder.

Patient has to be taught to empty the bladder by "Crede's Maneuver" i.e. by applying gentle suprapubic pressure or by intermittent catheterization (Fig. 6.17).

Bowels remain paralyzed in the stage of spinal shock and later start functioning autonomously. Frequent spontaneous small quantity emptying is seen. Should be taken care of by good nursing care.

b. To prevent other complications of prolonged confinement to bed, e.g. pressure sore hypostatic pneumonia, thrombo-embolism, etc.

Back care is a must to prevent pressure sore. Good nursing care, use of water bed or different types of pressure relieving mattresses will help in preventing pressure sores. These complications can also be prevented by early mobilization (Wheel chair) and exercise therapy by a good trained physiotherapist.

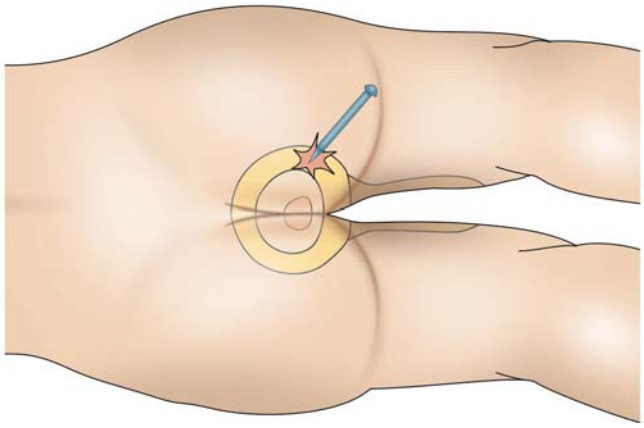


Figure 6.18

Diagrammatic representation of testing perianal anesthesia. Persistence of anesthesia is an indicator of complete cord injury. Descrimination between sharpness and dullness when present indicates incomplete injury.

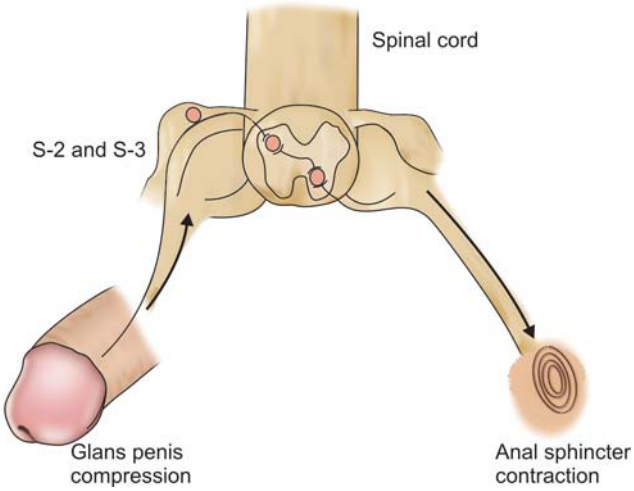


Figure 6.19

Diagrammatic representation of bulbocavernous reflex. Compression of glans penis will cause contraction of anal sphincter. Return of this cord mediated reflex indicates the termination of spinal shock and grave prognosis.

Signs of Grave Prognosis

If the cord transection is complete the prognosis is poor. During the stage of spinal shock there is flaccid paralysis with absence of perianal sensation and complete lumbar and sacral areflexia which may last for 24 hours to 7 days. Return of bulbocavernous reflex and anal wink which are normal cord mediated reflexes signifies the termination of the stage of spinal shock and indicates grave prognosis.

Bulbocavernous Reflex (Fig. 6.18)

It is a polysynaptic reflex mediated through S1, S2, S3 segments of the spinal cord. The reflex is elicited by applying compression over the glans penis by squeezing or tugging on indwelling catheter at the same time monitoring reflex contraction of anal sphincter (Fig. 6.19).

Anal Wink

Otherwise known as anal reflex, perineal reflex, anocutaneous reflex is mediated through S1, S2, S3, segments of the spinal cord. The pudendal nerve carries these signals to the spinal centers.

The reflex is elicited by using a sharp pin and inducing a noxious tactile stimulus on the skin around the anal sphincter. This results in reflex contraction of the sphincter indicating the intactness of the reflex arc (Fig. 6.20).

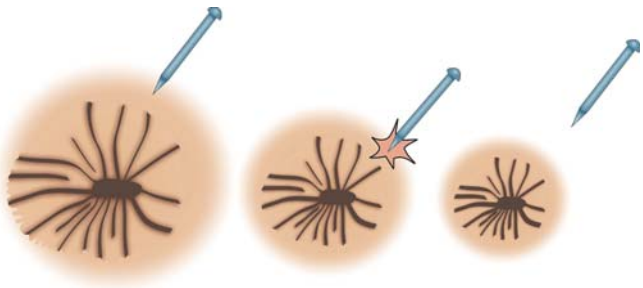


Figure 6.20

Showing diagrammatic representation of anal reflex. Return of this reflex after termination of spinal shock in a person who remains as a quadriplegic indicates grave prognosis.

Interpretation

i. Absence of the reflex–stage of spinal shock.
ii. Return of the reflex–spinal shock has passed of and prognosis is grave.
iii. If spinal shock does not exist/ not suspected but the reflexes are absent, these tests indicate that there can be conus medullaris or cauda equina lesion.

Rehabilitation

Centers for rehabilitation play a great role in helping paraplegics to lead an independent life and assistance should

be sought at earliest. The magnitude of injury is such that a breadwinner for the family becomes a dependent for rest of his life!. Psychological trauma is tremendous. Economic status is shattered. Hence, a paraplegic needs in addition to medical treatment a good emotional support not only from family members but also from the medical team treating the patient. Rehabilitation of a quadriplegic is more challenging than a paraplegic. There is no greater satisfaction than to see these paralyzed people become vocationally rehabilitated, economically independent and emotionally stable.

A Ray of Hope for Paraplegics and Quadriplegics

On Friday the 23rd January 2009 FDA of United States of America, is said to have given permission for the first ever clinical trial on Embryonic Stem cell therapy for irrecoverable spinal cord injury.

Although animal experiments on rats had promising results as early as 2005, Stem cell therapy had acted as a double edged sword. On one hand it had improved motor function below the level of the lesion. On the other hand the rats developed greater pain sensitivity superior to it. Also there existed a predictable possibility of stem cells growing into a tumor.

After much research, experimental study (multicentric) is currently being done on human beings by ***injecting Stem cells*** *14 days after the injury because of the possibility of stem cells getting destroyed in the process of inflammation occurring after injury if injected early. Too late an injection may not be effective because of degeneration occurring in the spinal cord and formation of scar tissue. The role of growth factors and other chemical mediators are also found to be crucial.*

Approximately 2 millions of stem cells are to be injected in an attempt to generate oligodendrocyte progenitor cell that could ultimately form new myelin and develop capability to transmit signals from the brain down the spinal cord.

Experiments of transfusing human embryonic cord blood in spinal cord transected rats has also shown promising results.

However, concrete concepts are yet to be established and every one hopes that ***this dream turns out to be a reality in future.***

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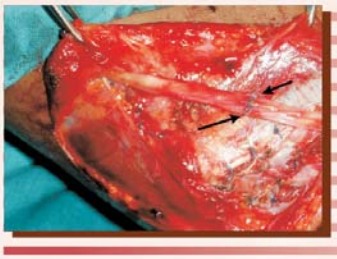


7

Peripheral Nerve and Brachial Plexus Injuries

- *Introduction*
- *Structure of a Peripheral Nerve*
- *Response of a Nerve to Injury*
- *Classification of Nerve Injury*
- *Clinical Symptoms, Signs and Tests Useful in Diagnosis*
- *Electrodiagnosis*
- *Management*
- *Pathomechanics of Paralytic Deformities and Clinical Signs*
- *Formation of Brachial Plexus*
- *Different Injuries of Brachial Plexus*
- *Signs, Symptoms, Diagnosis and Management*

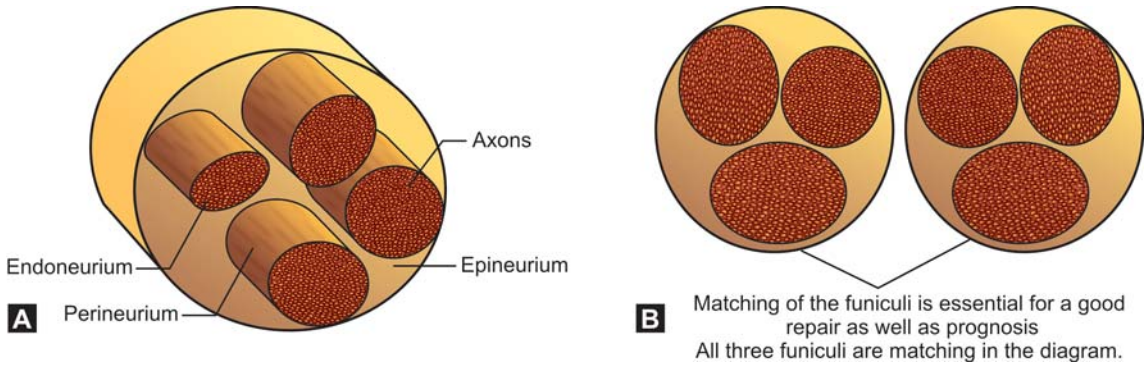
PERIPHERAL NERVE INJURY

Introduction

Every peripheral nerve is a mixed nerve (Unlike cranial nerves which are purely sensory, motor or mixed). Hence injury of a peripheral nerve results in disturbance of sensory, motor and autonomic function. As the spinal nerves exit through the spinal foramina they form plexus, i.e. the cervical, the brachial, the lumbar and the sacral plexus from which these peripheral nerves arise. Hence peripheral nerve consists of components from several nerve roots.

Structure of a Peripheral Nerve

The basic unit of a peripheral nerve is an axon. Axon is a direct extension of a nerve cell either from anterior horn cell, a dorsal root ganglion or a regional sympathetic ganglion. Each axon is covered by myelin and Schwann cell sheath. These axons with their sheaths are enclosed further in a



Figures 7.1A and B
Showing the structure of a peripheral nerve.

delicate fibrous sheath known as endoneurium. A group or cluster of axons with their endoneurial tubes is known as a fasciculus or funiculus. This funiculus is enclosed in another sheath known as perineurium. Thus a bundle is formed. These bundles of groups of axons are surrounded by a fairly thick fibrous sheath known as epineurium. Thus each axon is covered and protected by three sheaths, namely the endoneurium (the innermost), perineurium (the intermediate) and epineurium (the outermost).

The number and disposition of the axons as well as the fascicles vary from nerve to nerve. So is the thickness and the amount of neural sheath. Also, in the same nerve the structure varies at different levels. This is because the nerve branches out and the number of fibers become progressively less and less towards the terminal end of the nerve (Figs 7.1A and B).

Response of a Neuron to Injury

The neuron disintegrates and degenerates following an injury. The time required is different in sensory and motor fibers and depends also on the size and myelination of the nerve. The change that occurs *distal* to the injury is known as secondary or *antegrade, (Wallerian degeneration)* and the change that occurs *proximal* to the injury is known as *primary or retrograde degeneration.* Distal degeneration manifests in the form of clearing the dead tissue and emptying the tube and preparing it to receive sprouting axonal buds. Where as the changes taking place proximally is to recoup and prepare the neuron to regenerate and grow. These changes take place over a period of 15 to 30 days (Fig. 7.2).

Lesser the disruption, greater is the recovery and return of function. Greater the disruption, lesser is the recovery and return of function.

Classification of Nerve Injury

Based on Seddon's Classification (1943)

Neuropraxia

In this the injury is either contusion or compression of the nerve. There is a physiological block in conduction of the nerve impulse but the anatomical continuity is preserved. It is the result of transient ischemia. No Wallerian degeneration is observed. Recovery is complete, e.g. Saturday night palsy, Crutch palsy.

Axonotmesis

In this the axons break but the endoneurial tube (the sheath) is preserved. Occurs as a result of traction and stretching of the nerve. Though wallerian degeneration occurs, good functional recovery is possible because of the intact nerve sheath, e.g. Birth injury (except avulsion injury) Tardy Ulnar nerve palsy.

Neurotmesis

It is complete division of the nerve. Both nerve fibers and sheath are disrupted. May be partial or complete. Recovery is impossible without repair. Failure to recover and poor recovery is not uncommon. At times needs secondary reconstructive procedures to gain useful function.

Based on Sunderland Classification (1951): (In brief)

I Degree: Conduction of the nerve is interfered.] Neuropraxia of Seddon's

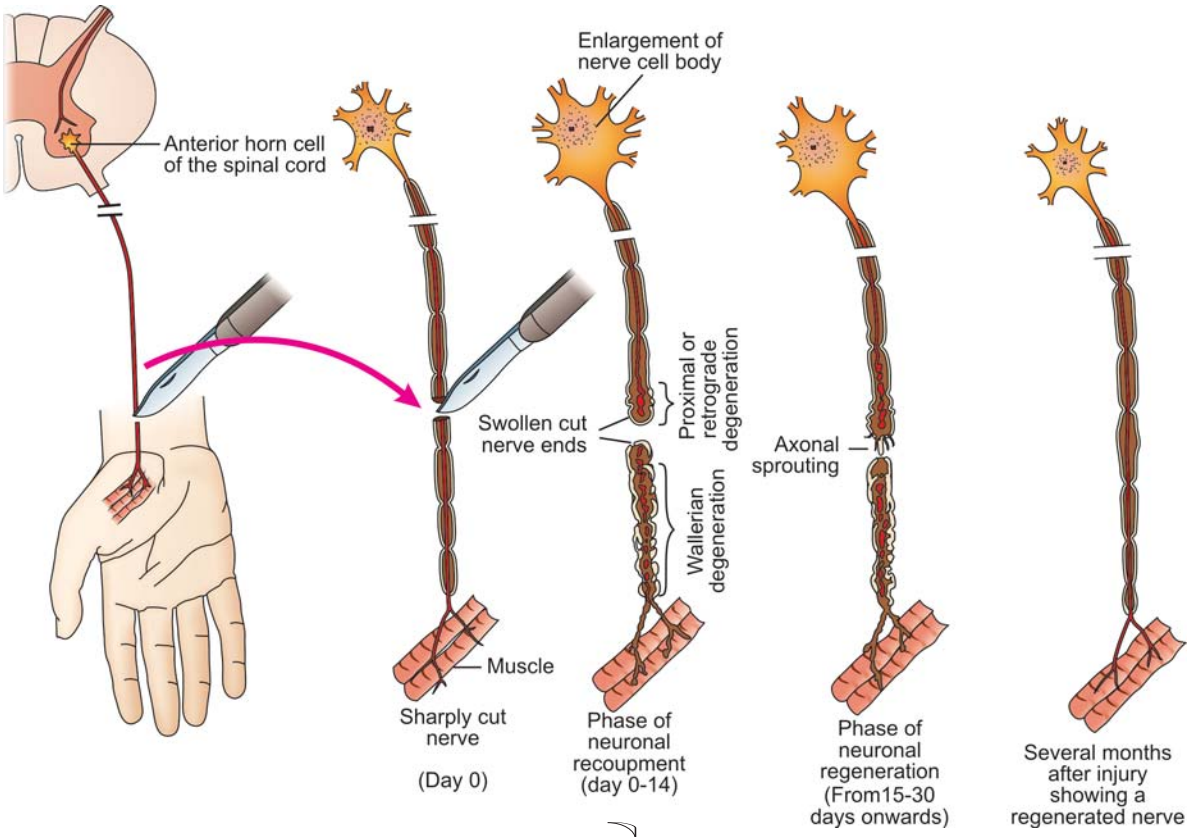


Figure 7.2

Showing response of a nerve to injury. Note the Wallerian degeneration occurring distal to injury, proximal or retrograde degeneration occurring proximal to the injury. The process of regeneration begins only after a period of 14-21 days following injury.

II Degree: Continuity of the axon is interfered.

III Degree: Continuity of the endoneurial tube and its contents is interfered.

IV Degree: Continuity of the funiculus and its contents is interfered.

V Degree: Continuity of the entire nerve trunk is interfered (Disruption of the entire nerve).

These represent varying grades of Axonotmesis of Seddon's

Neurotmesis of Seddon's

VI Degree of MacKinnon Dellon(1988): A combination of varying grades of Sunderland which co-exist in the same nerve. It means a part of the nerve may have neuropraxia. Other part may have axonotmesis.

Further a sharp object can cause partial cut injury to the nerve, i.e. partial neurotmesis considered as IV Degree of Sunderland's classification. Thus IV degree of Sunderland's could be either axonotmesis or partial neurotmesis.

Diagnosis

Diagnosis is done on the basis of clinical evaluation of autonomic disturbance, sensory changes, motor changes and by performing relevant special tests. Sensory and motor loss develops immediately after injury. Autonomic disturbance though present after injury, takes some time to establish. Certain changes develop progressively over a period of time and are seen after a few weeks, e.g muscle wasting.

A Scar/ penetrating wound along the course of the nerve especially at right angles indicates the possibility of neurotmesis.

Autonomic and Sensory Changes

a. Smoothness and dryness of the skin.
b. Atrophy of the pulp and conical tapering of the digits.

c. Brittle nails.
d. Loss of callosities and creases.
e. Absence of sweating.
f. Trophic ulcers.
g. Loss of sensation over the sensory area supplied by the peripheral nerve especially in the autonomous zone (Figs 7.3A to E).

Motor Changes

a. Paralysis of the muscles supplied by the peripheral nerve resulting in loss of function.
b. Pathognomonic deformities as a result of muscle imbalance.

Median nerve: Injury at a higher level, results in both ape thumb and pointing index deformity.

Injury at a lower level, results in only ape thumb deformity

Ulnar nerve: Ulnar claw hand.

Combined Ulnar and Median nerve: Simian hand (hand of an Ape)

Radial nerve: Wrist drop

Posterior interosseous nerve: Finger drop and thumb drop.

Sciatic nerve: Flail foot.

Posterior tibial nerve: Claw toes.

Lateral popliteal nerve: Foot drop.

These pathognomonic deformities when present are diagnostic of a peripheral nerve injury.

Special Tests

a. Oschner's clasp test.
b. Pen test.
c. Card test.
d. Book test.
e. Igawa's sign.
f. Tinel's sign.

Sensory Zones of a Peripheral Nerve

Three zones are recognized. They are the maximal zone, the intermediate zone, and the autonomous zone.

Maximal zone: Maximal area supplied by a peripheral nerve.

Intermediate zone: Is the area of overlap of the maximal zone of the different peripheral nerves.

Autonomous zone: Area exclusively supplied by a particular peripheral nerve.

Sensory loss over the autonomous zone is a sure sign of a particular peripheral nerve injury. Of immense help in a quick diagnosis especially in a case of polytrauma.

Diagnostic Tests

Electromyography (EMG)

The action potential generated in the muscle is recorded graphically both at rest and during activity (voluntary motor action potentials) by the insertion of a needle electrode. It is done at early and late intervals, as well as before and after repair and interpreted.

Denervation potentials: Every muscle is inherently vibrant with an electrical potential. This electrical potential remains masked as long as the muscle has an intact nerve supply. This is because the impulse generated by a peripheral nerve is much greater than the resting inherent electrical potential of the muscle. When there is denervation no impulse is transmitted through the peripheral nerve. Thus it is possible to record this inherent electrical potential of the muscle. This is recorded initially as sharp positive waves and later as fibrillatory waves and are known as denervation potentials (Figs 7.4A to C).

Thus following conclusions are drawn.

a. Presence of fibrillation potentials indicate denervation.
b. Absence of denervation potentials as late as 3 weeks indicate intactness of nerve.
c. Absence of voluntary motor unit potentials indicate paralysis of the nerve.
d. Presence of voluntary motor unit potentials indicate intact functional nerve.
e. Polyphasic motor unit potential developing after denervation indicate reinnervation.

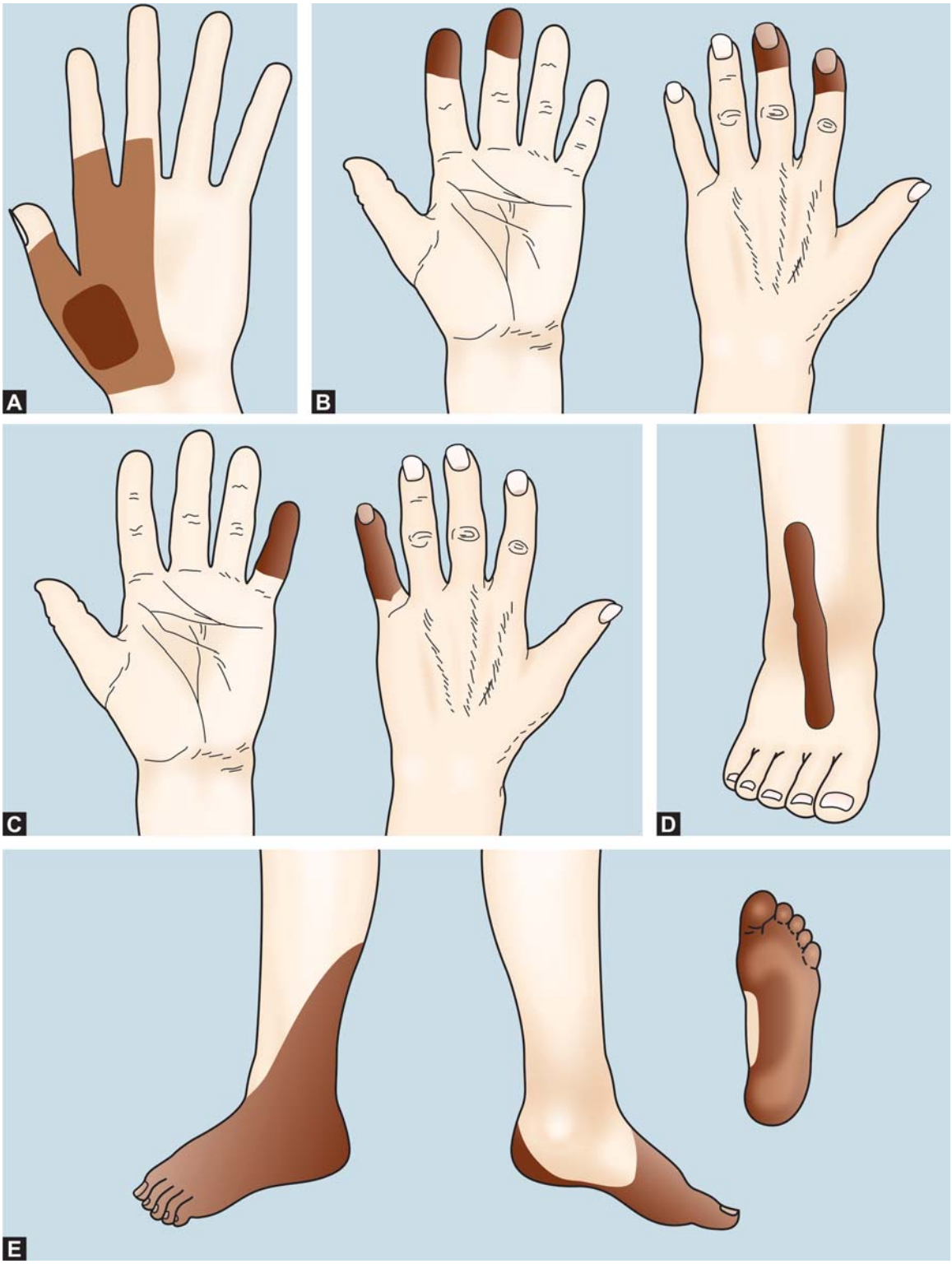
Strength Duration Curve

Uses the motor conduction property to assess the status of the injured nerve. As the name indicates this is a curve plotted on a graph for different strengths and different durations of electrical stimulation within a physiological limit (Galvanic/Faradic current). The surface electrodes are used for stimulation.

The physiological limit of stimulation is expressed in terms of rheobase and chronaxie.

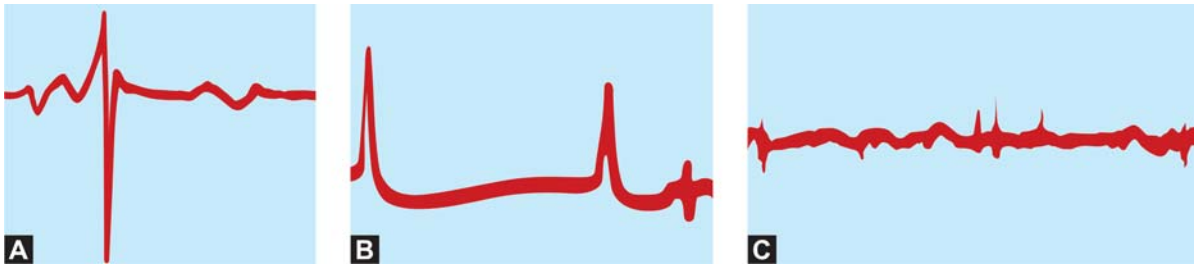
Rheobase

It is the minimal strength of the current of infinite duration required to stimulate a muscle (e.g. 300 milliseconds/long duration).



Figures 7.3A to E

Showing the autonomous zones of different peripheral nerves (A) Radial nerve (B) Median nerve (C) Ulnar nerve (D) Common peroneal nerve (E) Sciatic nerve.



Figures 7.4A to C

Showing action potential during EMG study. (A) Electromyogram showing normal insertional activity (B) Electromyogram showing positive sharp wave of denervation potential (C) Electromyogram showing spontaneous fibrillation potentials of denervation.

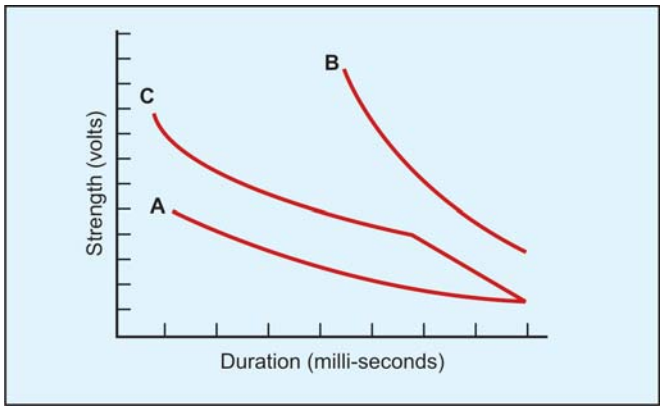


Figure 7.5

Showing changes in the SD curve during denervation as well as at regeneration. Strength duration curve (A) A normal nerve curve (re inervation) (B) A curve seen in denervation (shift to the right) (C) Partial denervation (regeneration.) Showing a kink in the curve.

Chronaxie

It is the duration required to stimulate a muscle with the current strength of twice the rheobase. Chronaxie in a human skeletal muscle varies from 0.08 to 0.32 milliseconds.

A muscle with intact nerve responds to any strength and any duration of current within this physiological limit. The contraction is directly proportional to the strength and duration of the stimulus. Where as a paralyzed muscle does not respond to a stimulus of low strength of short duration (faradic). But responds only to a stimulus of high strength and long duration (galvanic). So the response is absent for a faradic stimulation but is present for a galvanic stimulation. Hence the curve plotted, smoothly ascends to the right of the normal curve. As regeneration takes place or in a partially injured nerve, the curve starts shifting to the left and a kink develops in the curve. And finally when the regeneration is complete the curve descends, becomes flatter and near normal (Fig. 7.5).

Starch Iodine Test (Tests Autonomous Function)

In starch iodine test the area in question is first painted with iodine and kept dry. Then starch powder is sprinkled and sweating is induced (covering with cloth or making the patient drink a cup of hot coffee). If sweating is present the color changes to purple indicating intactness of autonomous innervation. If absent, no color change is observed indicating loss of autonomous function.

Management

Neuropraxia and Axonotmesis

These injuries invariably recover and hence need only supportive measures. For example, Saturday night palsy, Traction injury (not avulsion) of brachial plexus or any other peripheral nerve.

The management is always nonoperative.

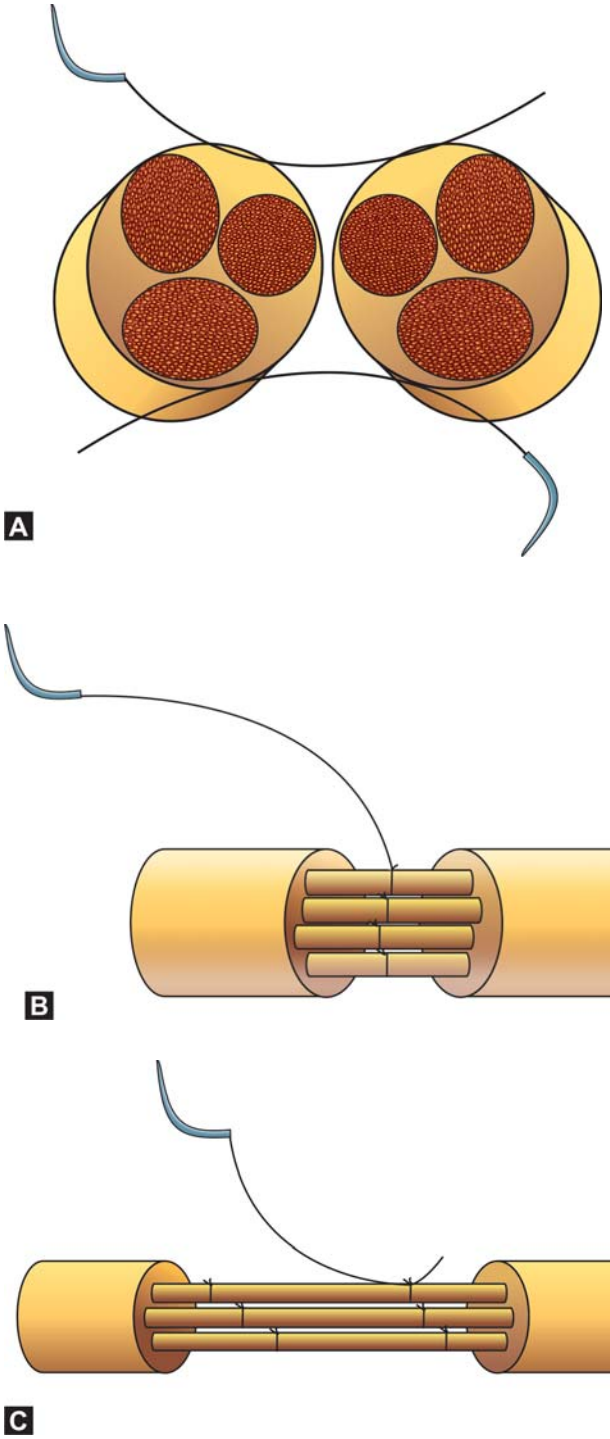
Measures undertaken are:

a. Drug therapy to relieve the edema and improve vascularity, e.g. anti-inflammatory drugs, steroids, etc.
b. Rest to the part by means of static splinting (prevents stretching) to facilitate healing.
c. Later, preventing contractures by means of dynamic splinting and exercise therapy.

Neurotmesis

Is always treated by nerve repair, i.e. neurorrhaphy. Repair is done either immediately after the injury which is known as *primary repair.* Or at a later period after the initial wound heals which is known as *secondary repair.*

Nerve grafting is done only when there is loss of nerve tissue and when end to end repair by any means is not possible.



Accurate matching of the funiculi and suturing of the corresponding funiculus is absolutely essential for a successful repair. Also the sutured site should be free of tension.

Figures 7.6A to C

Showing the different techniques of nerve repair diagrammatically.

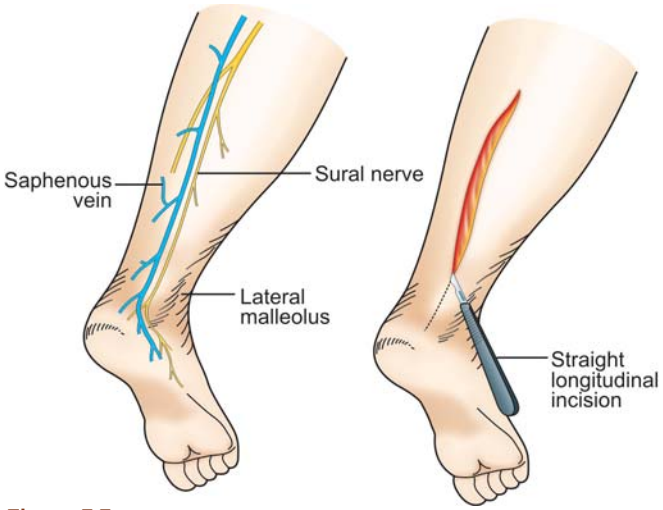


Figure 7.7

Showing the method of obtaining sural nerve for nerve grafting procedure.

Sural nerve is used as the donor nerve. Cadaveric nerve transplants are also in practice to bridge large defects (Fig. 7.7).

Types of Nerve Repair (Neurorrhaphy)

a. Epineurial repair—epineurium is used for placement of sutures (Figs 7.6A and 7.8).
b. Perineurial repair—perineurium is used for placement of sutures (Figs 7.6B and C)

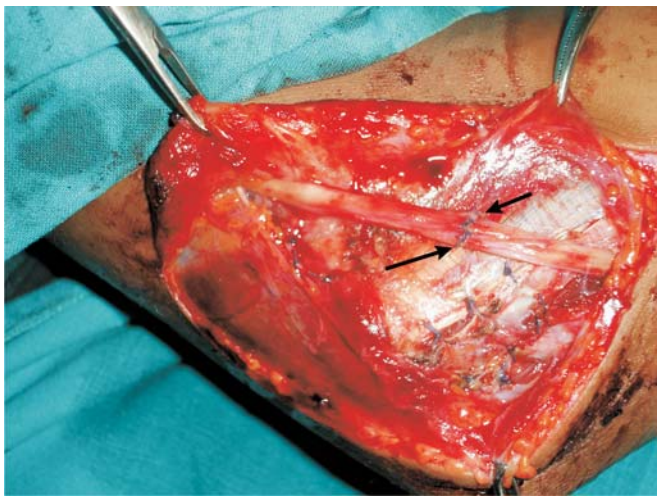


Figure 7.8

Showing primary repair of an ulnar nerve injury at the elbow. The nerve has been transposed anteriorly. Approximation of the fasciculi and epineurial nerve repair Using 6-0 poly propylene sutures has been done. Good recovery was seen and patient regained useful grade of motor power with sensory recovery almost up to S3.

c. Combined epi-perineurial repair—both are used for placement of sutures.

Factors Influencing Nerve Repair

a. Age of the patient—younger the age better is the result and vice versa.
b. Duration of the injury—longer the duration poorer is the result and vice versa.
c. Level of the injury—more proximal the injury poorer is the chance of recovery and vice versa.
d. Loss of the nerve tissue with defect—larger the defect poorer is the result and vice versa.
e. Nature of the cut ends—cleaner the cut ends better is the result when compared to ragged and lacerated cut ends.
f. Experience and skill of the surgeon.
g. Technique employed—epineurial repair is always better than perineurial except when perineurial repair is better than epineurial. Direct end to end repair is better than nerve grafting except when nerve grafting is more desirable.

Methods of Closing the Gaps While Repair

a. Mobilization of the nerve.
b. Positioning of the joints.
c. Transposition or translocation.
d. Shortening of the bone (e.g. during reimplantation).

Critical Limit of Delay

Is that period within which if a nerve is repaired some useful functional recovery can be expected. Beyond this period no functional recovery is possible. This is because of irreversible fibrotic changes taking place in the tissues distal to the injury and no functional tissue is available either for recovery or for action.

The critical limit of delay varies from nerve to nerve and ranges from 6 to 15 months.

Motor and sensory grading in nerve injuries

Motor Grading:

Grade 0	No movement.
Grade 1	A flicker of movement.
Grade 2	Movement possible when gravity is eliminated.
Grade 3	Movement possible against gravity
Grade 4	Movement possible against gravity and resistance.
Grade 5	Normal power (Complete recovery).

Sensory Grading:

S 0	No Sensation.
S 1	Presence of pain sensation.
S 2	Presence of pain and some touch.
S 3	Presence of pain and touch with no over reaction.
S 3+	Presence of pain touch with two point discrimination.
S 4	Complete recovery (Normal).

Reconstructive Procedures in Irrecoverable Nerve Injuries

Tendon Transfers (Dynamic Procedure)

Tendon transfer is one among the reconstructive procedures. When origin of a tendon is left intact and the insertion is detached and shifted to a new location the procedure is known as tendon transfer. Tendon transfers are done in peripheral nerve injuries when there is no motor recovery. Transferred tendon substitutes the action of paralysed musculotendinous unit and provides some useful function.

Static Procedures

Basic principle in these procedures is to achieve a fixity at the site of deformity in a functional position (desirable position to facilitate the function). This fixity is achieved by shortening the soft tissue and causing contracture or by fusing the joints. The procedures are as follows:

a. Dermodesis
b. Tenodesis
c. Capsulodesis
d. Arthrodesis

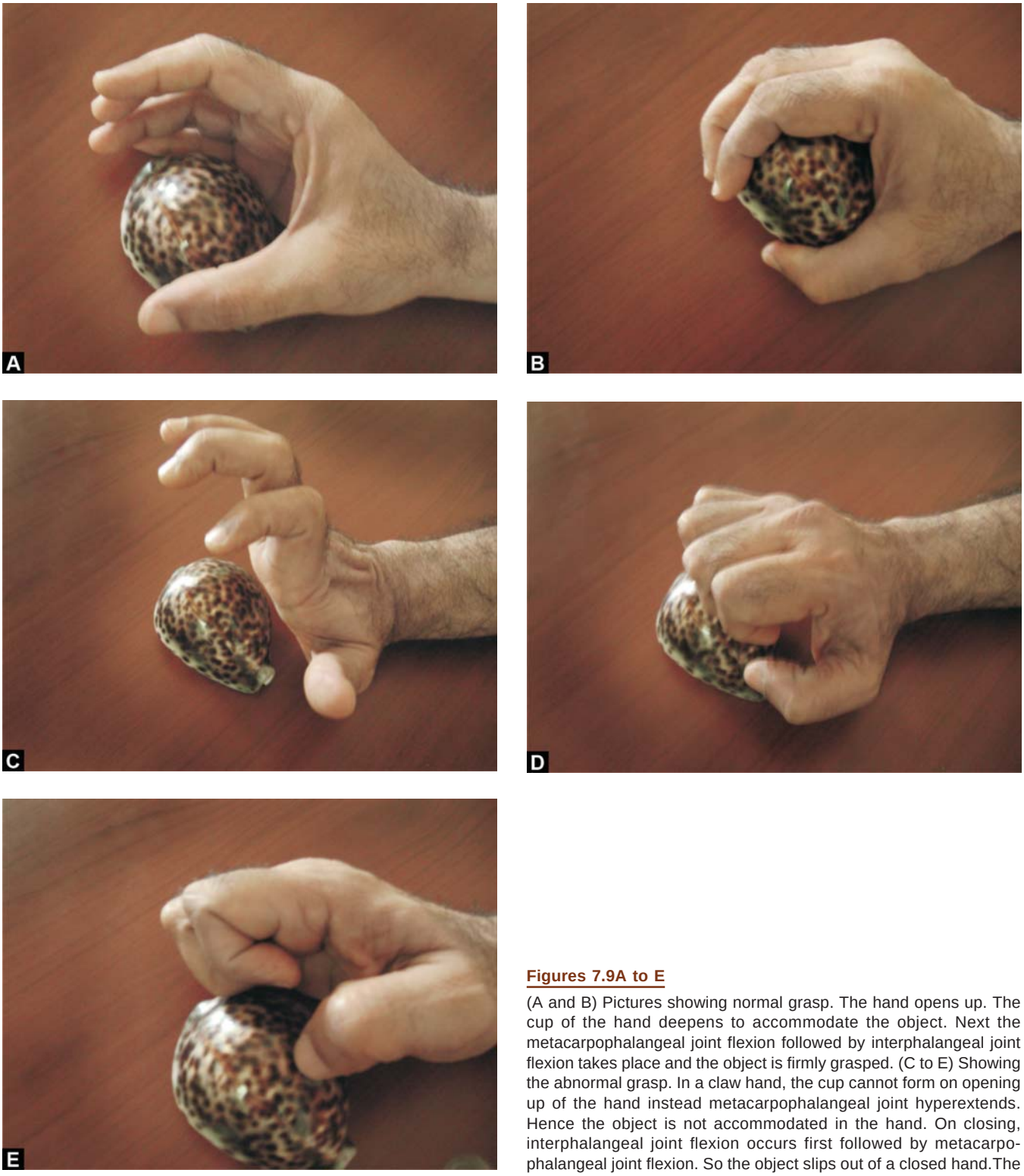
Only capsulodesis and arthrodesis are successful and are done when indicated. Dermodesis and tenodesis stretch and fail over a period of time.

Correction of Claw Hand

Disability in a claw hand and the need for claw correction:

Among the three basic hand functions, namely the grasp, pinch and the hook the grasp is severely affected in claw hand. Pinch is affected to certain extent. The hook function is preserved.

In a normal grasp, the hand first opens up fully thereby deepening the cup in order to accommodate an object which is to be grasped in the palm of the hand. Then the fingers flex and close over the object. The flexion occurs first at the metacarpophalangeal joint and then at the interphalangeal



Figures 7.9A to E

(A and B) Pictures showing normal grasp. The hand opens up. The cup of the hand deepens to accommodate the object. Next the metacarpophalangeal joint flexion followed by interphalangeal joint flexion takes place and the object is firmly grasped. (C to E) Showing the abnormal grasp. In a claw hand, the cup cannot form on opening up of the hand instead metacarpophalangeal joint hyperextends. Hence the object is not accommodated in the hand. On closing, interphalangeal joint flexion occurs first followed by metacarpophalangeal joint flexion. So the object slips out of a closed hand.The grasp function is affected.

joints. Thus the fingers close over the object and the object is firmly grasped in the palm of the hand.

In a claw hand when a person opens up the hand for grasping an object the metacarpophalangeal joints go into hyper-extension and the interphalangeal joints go into flexion. Thus in the hand instead of a concavity developing at MCP joint in order to deepen the cup, a convexity develops. Because of this the object does not come into the palm of the hand. Further when the person tries to close the hand over the object in order to grasp the object, the interphalangeal joints flex first followed by the metacarpophalangeal joint and the object slips out of an already closed hand (Figs 7.9A to E).

Principle of claw correction: The basic principle of claw correction is to prevent hyperextension at the metcarpo-phalangeal joint. This makes the long extensor i.e. extensor digitorum to act on the interphalangeal joints through its attachment to the extensor hood and extend them.

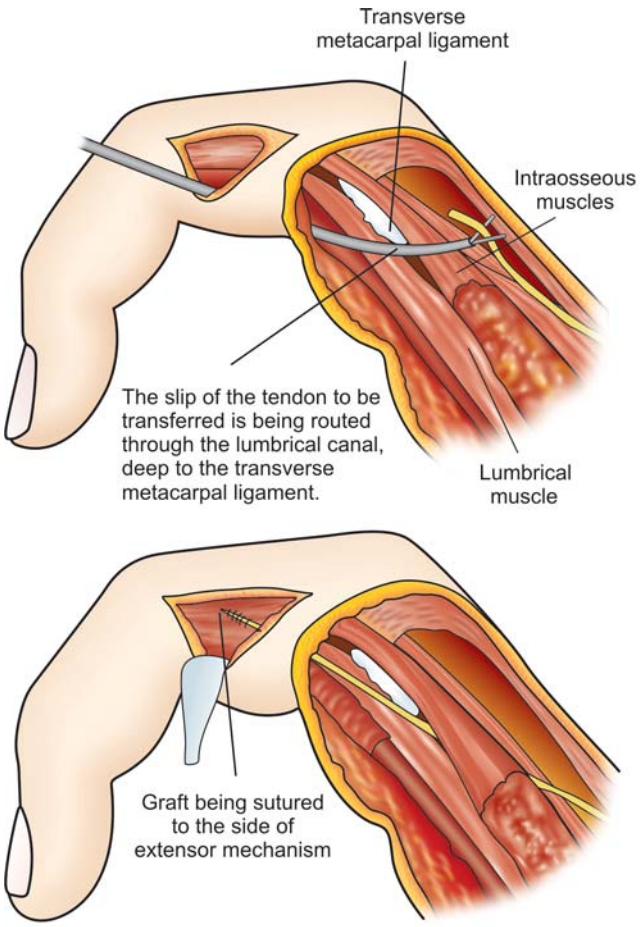


Figure 7.10

Showing the re-routing of the tendon for correction of claw deformity.

Tendon transfers for claw correction: Following tendons may be taken for claw correction

- Extensor carpi radialis brevis/longus; **Paul Brand's procedure.**
- Extensor indicis and Extensor digiti minimi; **Fowler's procedure.**
- Flexor digitorum superficialis of middle or ring finger: **Sterling Bunnel's procedure.**

Re-routing of the tendon (Fig. 7.10): Always re-routed volar to the transverse metacarpal ligament, through the lumbrical canal and attached to the extensor hood.

Static procedure for claw correction: Capsulodesis or Capsuloplasty of Zancolli. In this a flap of volar capsule of the metacarpophalangeal joints of the involved finger is raised with a base distally. Then with the finger in about 30° of flexion the capsular flap is pulled and sutured more proximally on itself. This produces a flexion contracture at the metacarpo-phalangeal joint of about 30° and prevents hyperextension. Alternatively, a small elliptical portion of the capsule is excised and the capsule shortened and sutured to produce a 30° flexion contracture (Fig. 7.11). Thus the extensor digitorum is now free to act on interphalangeal joints and bring about extension.

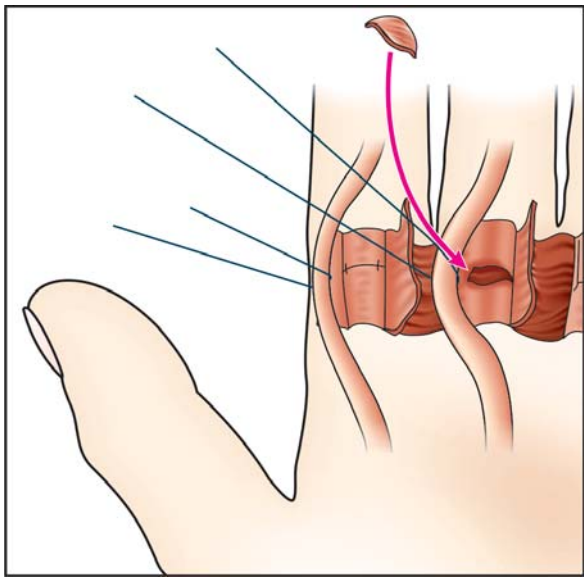


Figure 7.11

Diagrammatic representation of Zancolli's capsulodesis. A portion of the capsule of the MCP joint has been excised as shown in the diagram.When the remaining capsule is sutured it produces a flexion contracture at the metacarpophalangeal joint and prevents extension.

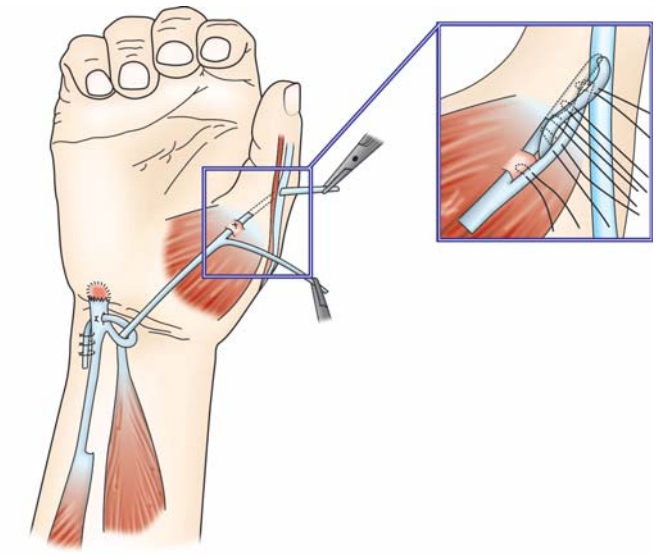


Figure 7.12

Showing the opponens plasty based on Riordan's technique. Note a pulley is made at the flexor carpi ulnaris and the method of attachment at the thumb to APB,EPL and hood of the thumb MCP joint.

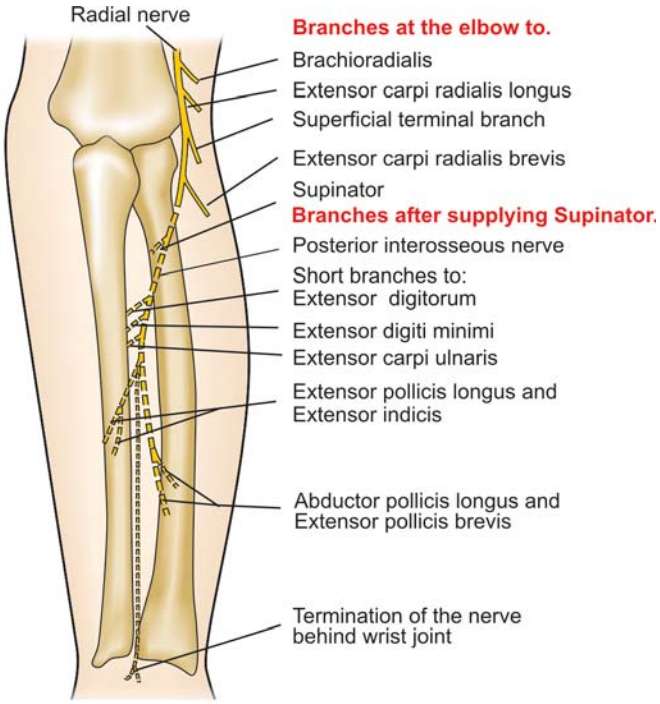


Figure 7.13

Showing the muscles supplied by the radial nerve and the posterior interosseous nerve (dotted line).
Note that the Brachioradialis, Extensor carpi radialis longus and brevis receive their nerve supply from radial nerve and so are spared from being paralyzed when posterior interosseous nerve is injured.

Opponens Plasty (Fig. 7.12)

This is a reconstructive procedure to restore opposition in irrecoverable median nerve injury. Following tendons may be taken.

- Flexor digitorum superficialis of middle or ring finger; Royle-Thompson procedure and Sterling-Bunnel procedure.
- Extensor indicis proprious.

Re-routing of the tendon: This is done by withdrawing the donor tendon proximally to the level of the wrist crease and then making use of the distal part of palmar fascia or carpal ligament or flexor carpi ulnaris as a pulley, it is passed subcutaneously to the thumb. The attachment is to the neck of the metacarpal and the extensor pollicis longus or to the abductor pollicis brevis, hood of the thumb MCP joint and extensor pollicis longus.

Tendon Transfer for Wrist Drop (High Radial Nerve Injury)

Functional deficit and substitution options (Fig. 7.13):

a. Wrist extension: Pronator teres to Extensor carpi radialis longus and brevis.
b. Finger extension: Flexor digitor superficialis (iii)/Flexor carpi ulnaris/Flexor carpi radialis to extensor digitorum.
c. Thumb extension: Flexor digitorum superficialis (iv)/ Palmaris longus to Extensor pollicis longus.

Tendon Transfer for Finger and Thumb Drop (Low Radial Nerve Injury)

a. Finger extension: Brachioradialis/Flexor digitorum superficialis (iii)
b. Thumb extension: Palmaris longus/flexor digitorum superficialis (iv).

Tendon Transfer for Foot Drop

a. *Ober's procedure:* Tibialis posterior is tranferred and inserted to base of the third metatarsal/ III cuneiform (subcutaneous tunnel)
b. *Barr's procedure:* Tibialis posterior is transferred and inserted to III cuneiform (Interosseous route).

Tendon transfers for foot drop are non phasic transfers and need accurate post op management for phasic conversion.

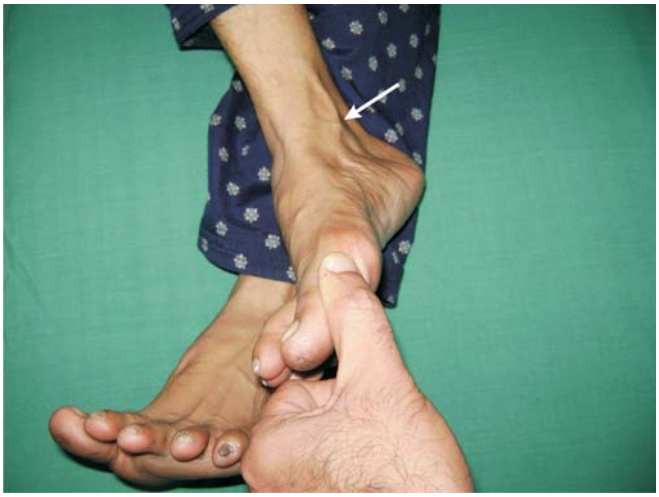


Figure 7.14

Picture demonstrating the method of assessing the power of tibialis posterior tendon both against gravity and resistance. Note the subject is made to cross the limb over the other and asked to invert the foot against resistance which is applied by the examiner at the fore foot. The tibialis posterior stands out prominently just behind and little above the medial malleolus.

Prerequisites for a Tendon Transfer

a. The tendon chosen for transfer should have power of grade IV or more (Fig. 7.14).
b. Agonists are preferred to antagonists.
c. Joints on which the tendon acts should be supple.
d. Range of excursion of the transferred tendon should be almost similar to that of paralyzed tendon.
e. Line of action should be as straight as possible.

Injuries of the Other Peripheral Nerves

Spinal Accessory Nerve

Gets injured as a result of penetrating injury and accidental injury during surgical procedures.

It is a motor nerve supplying trapezius muscle. Hence paralysis results in rotation of the scapula in a distal and lateral direction drawing the inferior angle closer to the midline. This results in not only in the weakness of the shoulder girdle but also in the inability to lift the shoulder beyond 90°. The winging of the inferior angle seen in this disappears on raising the arm anteriorly unlike the winging seen in serratus anterior paralysis which remains.

Suprascapular Nerve

Commonly involved in penetrating injuries.

As it supplies the supraspinatus, infraspinatus and the teres minor and gives an articular branch to the shoulder joint. Injury results in paralysis of these muscles manifested by wasting and limitation of the movement of the shoulder.

Long Thoracic Nerve C5 C6 C7

Commonly injured in a penetrating injury or by means of a traction force in the angle of the shoulder and the neck.

As it supplies the serratus anterior muscle the classical winging of the scapula is seen. This is demonstrated by asking the patient to push the wall with both upper limbs raised forwards.

Axillary Nerve C5 C6

It is injured by direct penetrating injury, as a result of fractures, dislocations and fracture dislocation around the shoulder.

Results in weakness of abduction because of the paralysis of the deltoid muscle. Sensory loss when present is over the deltoid region.

Musculocutaneous Nerve C5 C6

Is injured by direct penetrating injuries and in anterior dislocation shoulder. Often missed though supplies the biceps brachii, coracobrachialis and brachioradialis. Only biceps is amenable for testing.

Femoral Nerve L2 L3 L4

Is commonly injured by means of a penetrating injury.

Often missed because of

a. Alarming hemorrhage due to associated vascular injury.
b. Presence of extension because of active functioning tensor fascia lata, gracilis, gluteus maximus and gastrocnemius.
c. Small autonomous zone which is situated superior and medial to patella.

Sciatic Nerve

Injured by penetrating injury, fracture dislocations around the hip and fracture shaft of femur. Autonomous zone includes almost the entire foot except a small area on the medial aspect in the mid foot region. Functional loss is severe as there is paralysis of all the muscles of the posterior compartment of the thigh and muscles of the leg and the foot. The results of repair is poor. This is especially true when repaired late beyond 12 to 15 months, i.e. beyond critical limit of delay.

Figure 7.15

Picture showing a classical 'Ulnar claw' hand with hyperextension at the metacarpophalangeal joints and flexion at the interphalangeal joints of ring and little finger only. Hypothenar muscle atrophy is pronounced. Note the scar at the elbow along the course of the nerve (marked by an arrow) which indicates possibility of neurotmesis of the nerve.

Management

Principle of management of these nerve injuries remain the same.

As the injury involving the above nerves is not very common and the functional loss is not very severe (except sciatic nerve) and patients generally accept the resulting deficit easily, no much information is available regarding the repair of these nerves. However reconstructive procedures are described when deficits warrant intervention.

Pathomechanics of Various Deformities and Signs

Claw Hand (Intrinsic Minus Deformity) (Fig. 7.15)

Claw hand is defined as a dynamic deformity characterized by *hyperextension at the metacarpophalangeal joints and flexion at the interphalangeal joints*. If only ulnar nerve is involved the deformity of *ulnar claw hand* or partial claw hand develops. If both ulnar and median nerves are involved (low ulnar and low median; high ulnar and low median) the deformity of *total or combined claw hand*, otherwise called the 'Simian hand' develops.

At every joint in the hand there is a balance, maintained by the action of flexors on one side and the extensors on the other. If one is paralyzed, the joint moves in the other direction. At the metacarpophalangeal joint the flexor is the lumbrical and extensor is the extensor digitorum. At the proximal interphalangeal joint the flexor is the flexor digitorum superficialis and extensor is the extensor expansion (the dorsal digital expansion, extensor hood, extensor apparatus, extensor aponeurosis). At the distal interphalageal joint the flexor is the flexor digitorum proundus and the extensor is the extensor hood (Figs 7.16 and 7.17A, B).

In ulnar nerve injury there is paralysis of all the interossei and the medial two lumbricals. Therefore at the interphalangeal joint, extension is lost and the joint is subjected to unopposed action of flexor digitorum superficialis/profundus. Whereas at the metacarpophalangeal joint flexion is lost and the metacarpophalangeal joint is subjected to unopposed action of extensor digitorum resulting in hyperextension of the joint. Hence deformity of ulnar claw develops (Fig. 7.15).

Ulnar Paradox

Higher the lesion lesser is the deformity. Lower the lesion greater is the deformity. This because in high ulnar nerve injury both flexors namely flexor digitorum superficialis and flexor digitorum profundus are paralyzed. Hence, deformity is less. Whereas in low ulnar nerve lesion the flexor digitorum profundus is spared. Hence the deformity is more.

In combined low ulnar and low median nerve injury, there is paralysis of lateral two lumbricals in addition. Hence, the deformity of combined claw hand develops. The only extensor muscle acting is the extensor digitorum which hyperextends the metacarpophalangeal joints. The flexors which are not paralyzed are the flexor digitorum superficialis and flexor digitorum profundus. These cause flexion of the interphalangeal joints. The thumb remains by the side of other fingers. Hence, the hand resembles the hand of an ape and is known as 'simian hand'.

In combined high ulnar and median nerve injury, all the flexors are paralyzed. Hence classical claw deformity is not seen except for hyperextesion at the metacarpophalangeal joints. The flexion deformity at the interphalangeal joints is minimum.

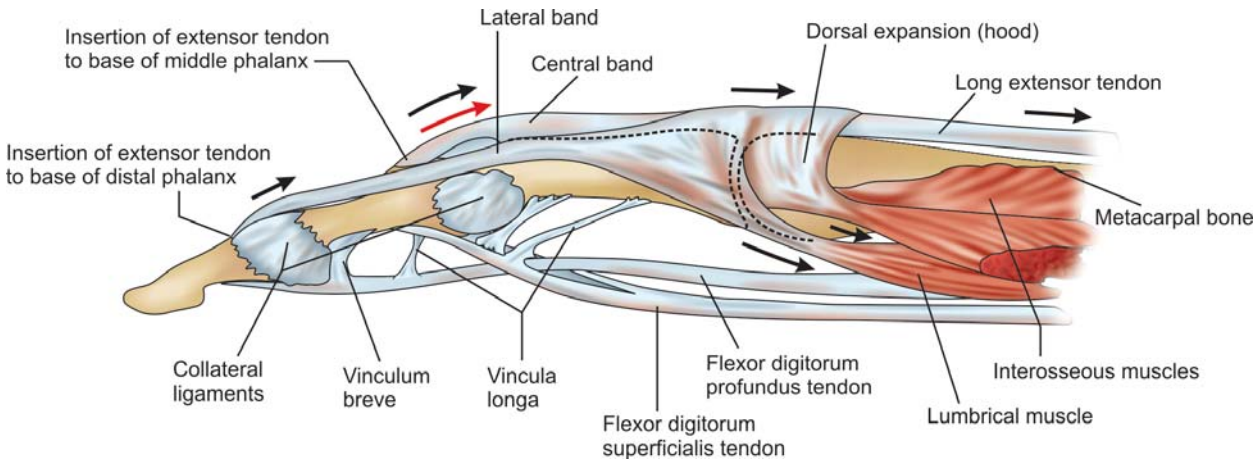
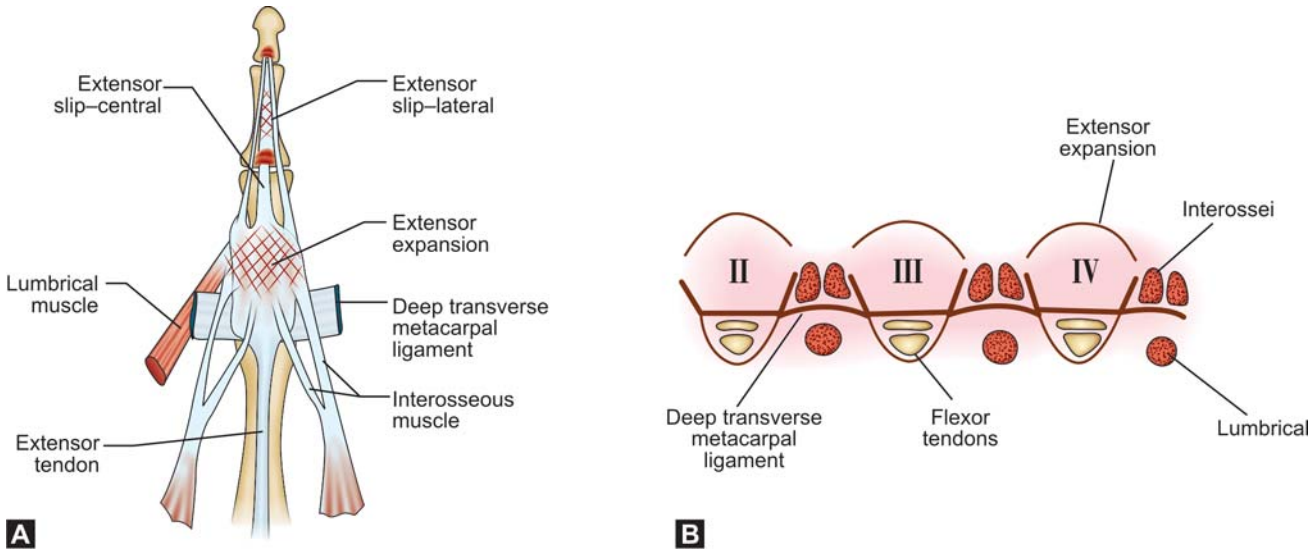


Figure 7.16

Picture showing how the balance at the joints of the hand is maintained by the flexor muscles on one side and the extensor muscle on the other.



Figures 7.17A and B

Diagrammatic representation of (A) extensor expansion and (B) the relation of interossei lumbricals, extensor expansion and flexor tendons at the level of the MCP joint.

This classical pathomechanics is true only in low ulnar as well as in combined low ulnar and low median and and not true when there is a combination of high median along with high ulnar nerve injury.

Ape Thumb Deformity

Normally the thumb is placed at right angles to other fingers. When there is median nerve injury the thumb remains in line with other fingers, resembling the thumb of an ape. This is because of the unopposed action of adductor pollicis and

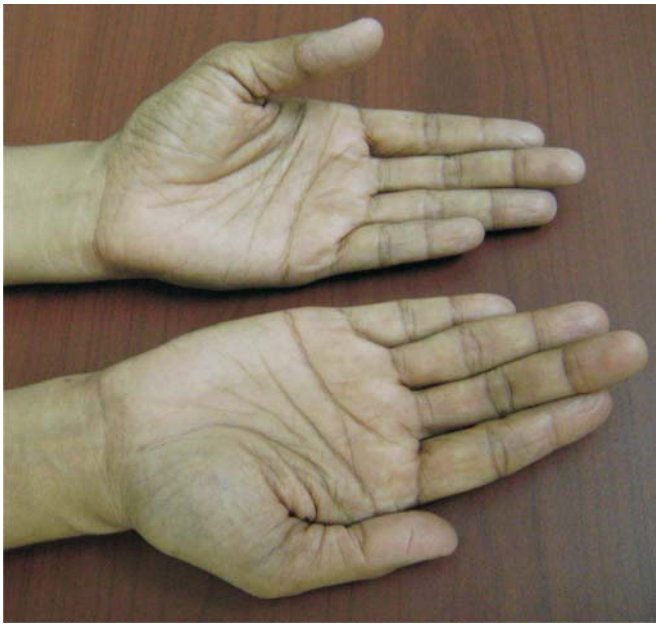


Figure 7.18

Picture showing normal hand on the left side where the thumb is placed at right angles to the other fingers and the hand with thumb deformity, on the right side where the thumb is remaining by the side of the other fingers.

extensor pollicis brevis which are supplied by ulnar and radial nerve respectively (Fig. 7.18).

Wrist Drop (Fig. 7.19A)

It is the inability to dorsiflex the wrist, fingers and the thumb.

Results because of paralysis of all the extensor muscles of the forearm as a result of radial nerve injury. The muscles paralyzed are:

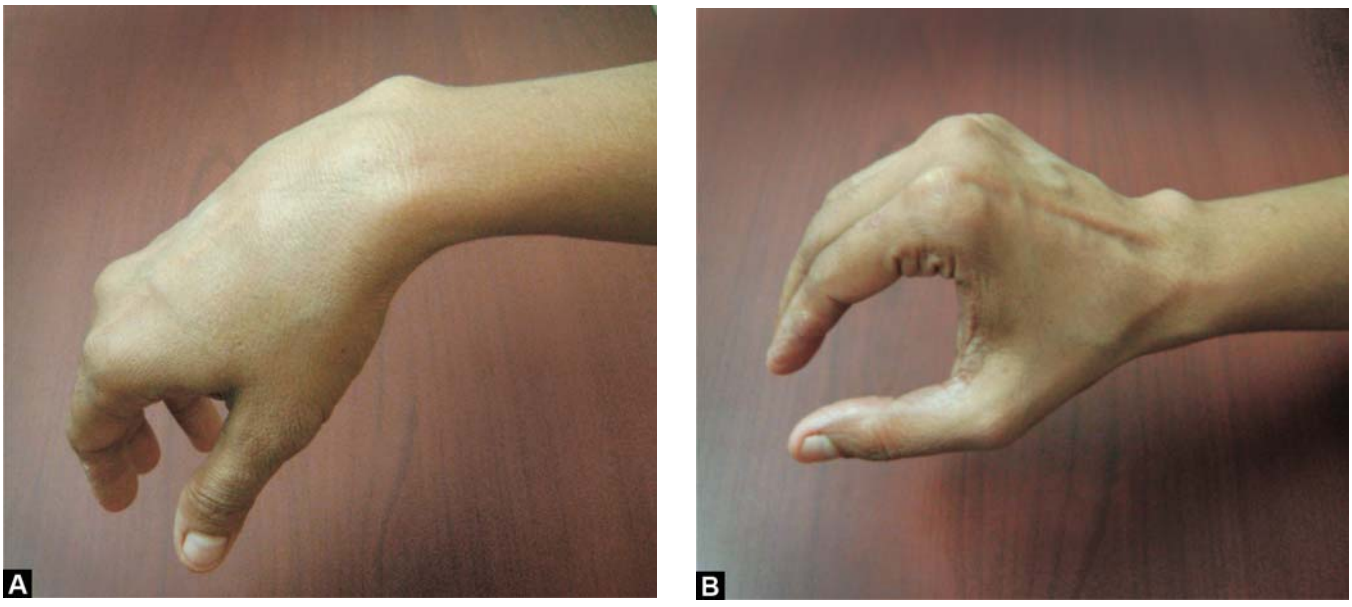
a. Abductor pollicis longus and extensor pollicis brevis
b. Extensor carpi radialis longus and brevis
c. Extensor pollicis longus
d. Extensor digitorum and extensor indicis
e. Extensor digiti minimi
f. Extensor carpi ulnaris
g. Brachioradialis
h. Supinator.

Finger Drop and Thumb Drop

Seen in posterior interosseous nerve injury. Except brachioradialis, supinator, extensor carpi radialis longus and brevis all the other muscles of the extensor compartment of the fore arm are paralyzed. Hence wrist extension is possible but not the finger and the thumb extension (Fig. 7.19B).

Flail Foot

Seen in sciatic nerve injury because of paralysis of both dorsiflexors and plantar flexors of the foot. (All the muscles in the leg and in the foot are paralyzed).



Figures 7.19A and B

Picture showing the classical attitude of (A) Wrist drop and (B) Finger drop.

Claw Toes

Is seen in posterior tibial nerve injury in the region of the ankle. Deformity occurs because of paralysis of the intrinsic muscles of the foot.

Foot Drop

Seen in lateral popliteal nerve injury because of paralysis of dorsiflexors of the foot. The muscles paralyzed are:

a. Tibialis anterior
b. Flexor digitorum longus
c. Flexor hallucis longus
d. Peroneus longus and brevis
e. Peroneus tertius
f. Extensor digitorum brevis.

The classical gait seen in foot drop is the "High stepping gait". Normal gait is heel to toe gait whereas the high stepping gait is a toe to heel gait. This is because of the paralysis of the dorsiflexors. In this gait the heel strike of stance phase of gait cycle is lacking. Hence to avoid injury patient lifts the leg up and brings it down. Thus, toes touch the ground before the heel resulting in a high stepping gait.

Oschner's Clasp Test

This is a test employed to diagnose high median nerve injury. It is performed by asking the patient to clasp the hands. When there is high median nerve injury the index finger remains straight and points forwards (pointing index) indicating paralysis of flexor digitorum superficialis and flexor digitorum profundus. The test is positive when there is pointing index.

Though median nerve supplies the lateral two profundi the middle finger does not point. This is because the middle finger profundus takes origin from a common aponeurosis for medial two profoundi which are supplied by the ulnar nerve. Hence, when the medial two profundi contract to some extent the middle finger profundus also contracts (Fig. 7.20).

Pen Test

This is a test employed to test the abductor pollicis brevis muscle. The test is done by asking the patient to place the hand flat on the table with the palm facing upwards and keeping a pen at some distance over the thumb. Next he is asked to touch the pen with his thumb. If the patient is able to touch the pen with his thumb abducted the test is negative. If the patient is not able to touch the pen the test is positive.

The test is positive both in high and low median nerve injury (Fig. 7.21).

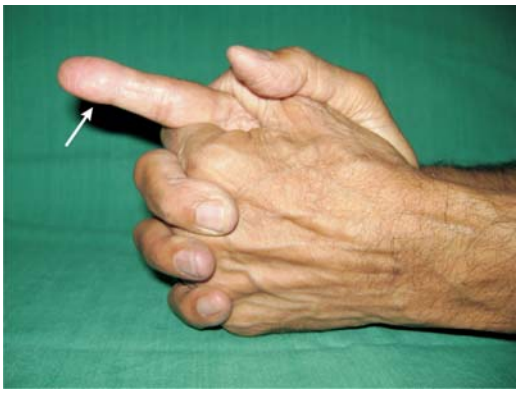


Figure 7.20

Showing a positive Oschner's clasp test and the pointing index *sign.*

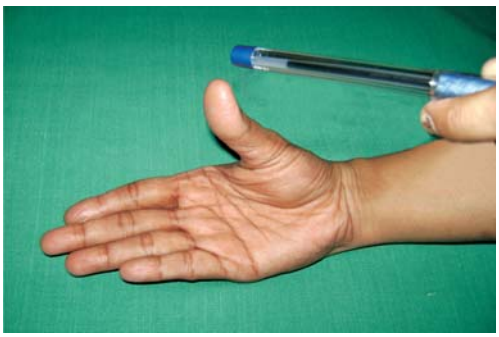


Figure 7.21

Picture showing a negative pen test. The person is able to touch the pen using his abductor pollicis brevis.

Card Test

This is a test employed to test the interossei muscle. The function of the palmar interossei is adduction and the dorsal interossei is abduction. The test is performed by asking the patient to hold a card inserted between the two fingers. During the process, first the finger abducts and then while gripping the card it adducts. The test is said to be positive when the patient is not able to grip the card between the fingers and card is easily pulled out by the examiner who uses a similar grip to pull out the card (Fig. 7.22).

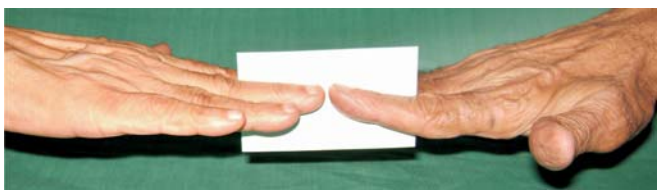


Figure 7.22

Showing card test being performed. Note that both the examiner and the subject to be examined should use the same grip, i.e. inter digital clasp.

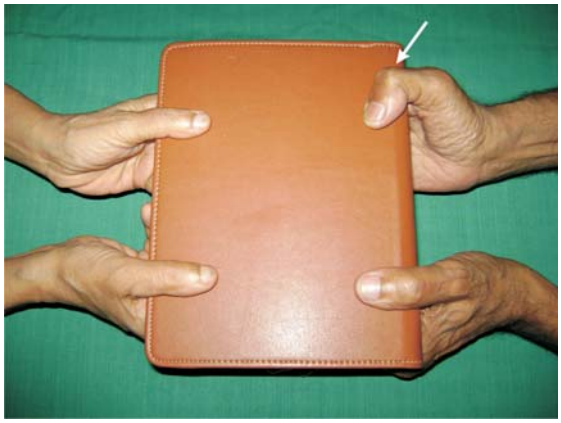


Figure 7.23

Showing the book test being performed and the Froment's sign. Note the flexion of the IP joint of the thumb which is brought about by the Flexor pollicis longus.

Book Test and Froment's Sign

It is a test employed to test the adductor pollicis muscle. The patient is asked to hold a book in the 1st web space. The examiner holds the same book in a similar manner and tries to pull out the book. When adductor pollicis is paralyzed the patient uses flexor pollicis longus to hold the book and prevent it from slipping out of the hand. This results in flexion of the thumb and a positive test (Fig. 7.23).

Igawa's Sign

This is a quick test to assess the interossei. The patient is made to place the hand on the table and asked to raise the middle finger and move it side to side. If the interossei are paralyzed he is not able to do so (Fig. 7.24).

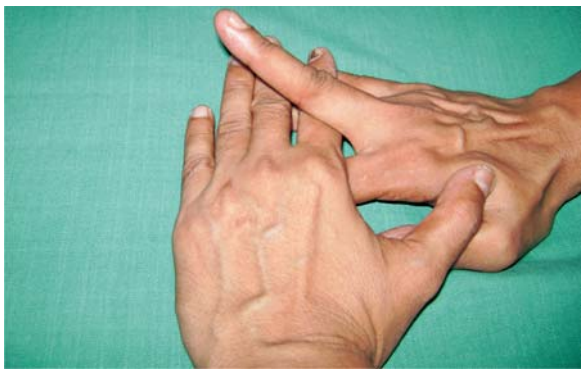


Figure 7.24

Picture showing the Igawa's test being performed. Note how the other fingers are stabilized and the middle finger is made to move sideways both medially and laterally, testing both palmar and dorsal interossei.

Tinel's Sign

This sign was described by Jules Tinel a French physician (1879-1952). The test is performed by percussion of the nerve along its course from distal to proximal there by causing stimulation. At the site where the nerve is irritable patient experiences pins and needles/ tingling down the course of the nerve. It is a useful sign after nerve repair when done at intervals and the findings are recorded.

Three types of responses are observed.

a. Strong response at the site of injury not progressing distally – indicates poor prognosis (recovery).
b. Fading response at the site of injury progressing distally – indicates good prognosis (recovery).
c. Persistent response at the site of injury as well as progressing distally–unpredictable prognosis (recovery).

Note: The Tinel's sign does not predict the quality or quantity of regeneration. So also it does not predict the return of function. It only suggests the type of nerve regeneration, i.e whether the regeneration is proceeding in an orderly manner or not. Functional recovery is dependent on several other factors such as status of the muscle, status of the joint, presence of associated tendon injury, age of the patient, type of the nerve, etc.

Revision Questions

Q. Describe the structure of a peripheral nerve.
Q. What are the clinical deficits that arise after a peripheral nerve injury?
Q. Classify nerve injuries.
Q. Discuss the following
   a. Neuropraxia.
   b. Axonotmesis
   c. Neurotmesis.
Q. What is Wallerian degeneration?
Q. What is electrodiagnosis?
Q. Write notes on
   a. Rheobase
   b. Chronaxie
   c. Nerve conduction velocity
   d. Strength duration curve
Q. Write notes on
   a. Claw hand
   b. Ulnar paradox
   c. Simian hand
   d. Ape thumb deformity

e. Wrist drop and finger drop
f. Foot drop
g. Flail foot

Q. Write notes on
a. Card test
b. Book test
c. Pen test
d. Igawa's sign.
e. High stepping gait.

Q. Discuss the methods of repair of a peripheral nerve.

Q. Discuss nerve grafting.

Essay Questions

Q. Discuss the pathoanatomy, clinical features, diagnosis and management of a peripheral nerve injury.

Q. Discuss the response of a nerve to injury. Describe in detail the clinical features, diagnosis and management of a case of ulnar nerve injury.
(The same question can be applied for other peripheral nerves.)

Q. What is tendon transfer? Discuss the tendon transfers for
a. Claw hand
b. Wrist drop
c. Median nerve injury
d. Foot drop.

Q. Classify the reconstructive procedures designed for claw hand correction. Discuss the merits and demerits of the same.

BRACHIAL PLEXUS

Formation of Brachial Plexus

Brachial plexus is formed by the union of anterior rami of C5, C6, C7, C8 and T1. When C5 receives contribution from C4 it is known as pre fixed and when T1 receives contribution from T2 it is known as post fixed. The plexus comprises of trunks, divisions, cords and branches (Figs 7.25 and 7.26).

C5 and C6 unite to form the upper trunk, C7 alone continues as middle trunk, C8 and T1 unite to form the lower trunk. Each trunk divides into an anterior division and a

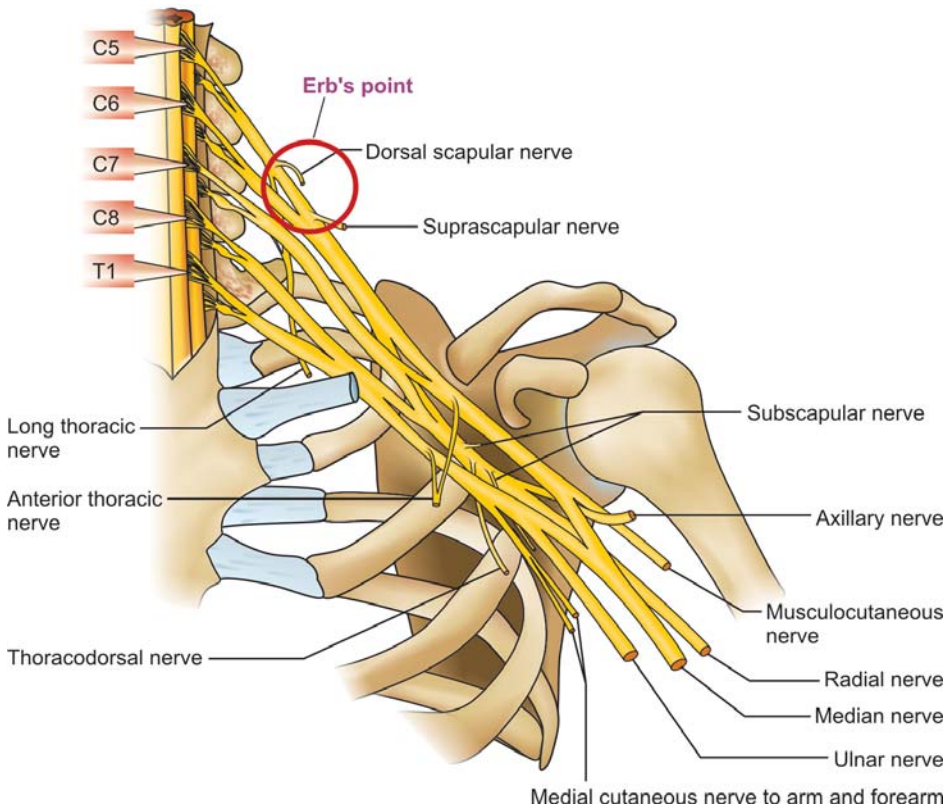


Figure 7.25

Showing the drawing of the brachial plexus.C5,C6 forms the upper trunk, C7 root continues as middle trunk and C8, T1 forms the lower trunk. The circled area in red shows the Erb's point, a point of origin of five nerves.

posterior division. The anterior division of the upper and the middle trunk unite to form the lateral cord, the anterior division of the lower trunk continues as medial cord and posterior divisions of all the trunks unite to form the posterior cord. The branches of the cord are as follows.

Lateral cord

a. Lateral pectoral.
b. Musculocutaneous.
c. Lateral root of the median nerve.

Medial cord

a. Medial cutaneous nerve of the forearm.
b. Medial cutaneous nerve of the arm.
c. Medial pectoral.
d. Medial root of the median nerve.
e. Ulnar nerve.

Posterior cord

a. Upper subscapular.
b. Lower subscapular.
c. Thoracodorsal.
d. Radial.
e. Axillary.

Nerves arising from the roots

a. Long thoracic nerve –form C5, C6, C7 roots
b. Dorsal scapular nerve – from C5

Nerve arising from the upper trunk

a. Suprascapular nerve.

Injuries of Brachial Plexus

Brachial plexus can get injured either by direct penetrating wounds, e.g. assault, bullet and missile injuries or by traction force, e.g. vehicular accidents, birth injuries in which the angle between the shoulder and the neck widens (increases).

A thorough knowledge of anatomy is essential for an accurate diagnosis with reference to the level of lesion. The lesion can be at the level of the roots, trunk, divisions or cord. The functional loss is directly related to the paralysis of the corresponding nerves which take origin from these. Examination of various muscles helps in the conclusion.

Further, it is necessary to distinguish between pre and postganglionic type of injury. The prognosis in preganglionic type is poor as repair is difficult. Prognosis in postganglionic type is better as repair is possible. Absence of 'Horner's syndrome' and absence of cutaneous axon reflex indicates that the lesion is postganglionic.

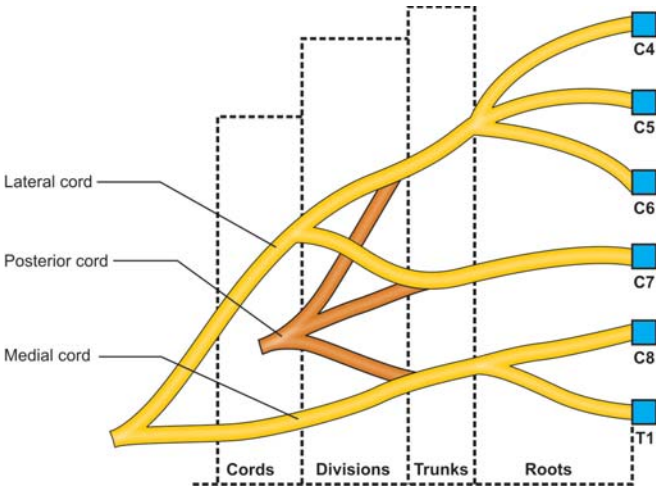


Figure 7.26
Showing the diagrammatic representation of brachial plexus with roots trunks, divisions and cords. The plexus shown here is prefixed with contribution from C4.

Signs of Preganglionic Lesions

a. Intractable burning pain in a sensationless, paralyzed extremity.
b. Presence of spinal fractures.
c. *Horner's syndrome:* It is characterized by ptosis, anophthalmos, myosis and anhydrosis.
d. *Cutaneous axon reflex:* This reflex is elicited by inducing irritation by placing a drop of histamine over the skin along the distribution of the nerve to be examined and scratching the site (may also be done by injecting histamine intradermally). Normal reaction is vasodilation followed by wheel and a flare (triple response). This reflex is absent in postganglionic lesions where the response is only a wheel. No flare response is seen.
e. Posterior cervical electromyography may show denervation potentials in posterior cervical paraspinal muscles indicating the lesion is preganglionic.
f. Presence of long tract signs in the lower limbs indicating the involvement of the spinal cord and the proximal nature of the injury.
g. CT myelography/ MRI may show pseudomeningoceles indicating root avulsion and the lesion is preganglionic. Unreliable during the early phase as dural tears can also give a false picture of pseudomeningocele.

Upper Plexus Injury Erb's Paralysis (Fig. 7.27)

It is characterized by involvement of C5, C6 nerve roots. (C7 involvement may or may not be present).

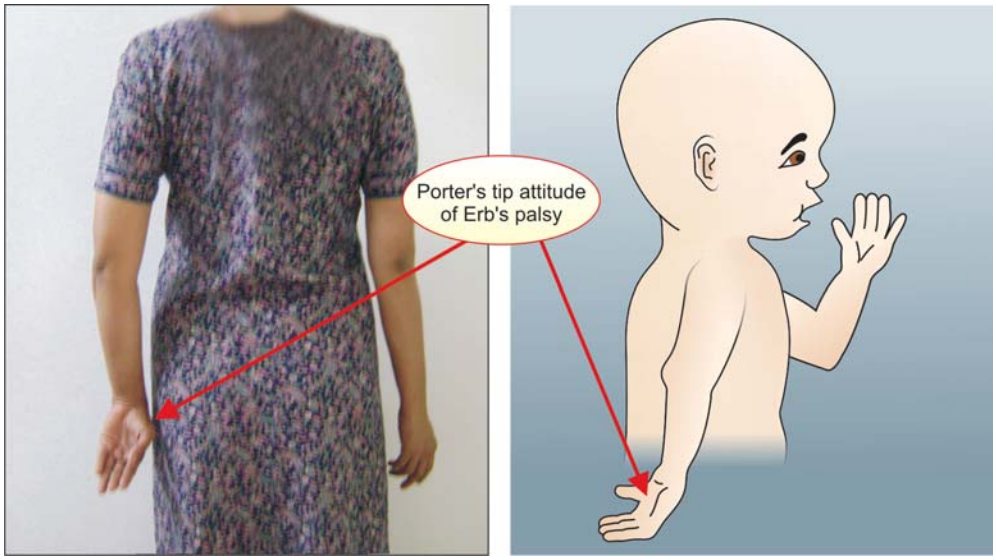


Figure 7.27

Picture showing the characteristic attitude of 'the Porter's tip hand' of Erb's palsy.

Functional loss results in a typical attitude of the upper limb. The limb remains by the side of the trunk with shoulder in adduction internal rotation, elbow in extension, forearm in pronation and wrist in flexion and ulnar deviation. This is because of the paralysis of abductors namely the deltoid and supraspinatus and external rotators namely infraspinatus and teres minor at the shoulder; paralysis of flexors namely the biceps, brachialis and the brachoiradialis at the elbow; paralysis of the supinator at the forearm.

As the limb hangs by the side of the body and *resembles* the attitude of *a porter waiting for the tip*, it is known as *"the porter's tip hand"*.

Sensory loss over the deltoid, lateral aspect of the forearm and hand is seen.

Paralysis of the serratus anterior supplied by long thoracic nerve, rhomboids and levator sapulae muscle supplied by dorsal scapular nerve indicates that the lesion is proximal to the origin of these nerves (these nerves take origin from the roots, before the plexus is formed) and may be preganglionic.

Lower Plexus Injury, Klumpke's Paralysis

It is characterized by involvement C8 T1 nerve roots. C7 involvement may or may not be present.

Functional loss is sensory and motor deficits involving C8 T1. Hence, there is paralysis of intrinsic muscles of the hand along with wrist and finger flexors. The sensory deficit is on the medial aspect of the hand, forearm and arm.

Whole Plexus Injury

It is characterized by complete flaccid paralysis of the entire upper limb. Considered as one of the most serious injuries as chance of recovery is very poor.

Injuries of the trunks: Injuries of the upper trunk manifests with similar deficits as their rami. But the long thoracic and dorsal scapular nerve escape paralysis.

Injuries of the lower trunk manifests with similar deficits as their rami. But there is no sympathetic involvement and Horner's syndrome is not seen.

Injuries of the divisions: Injuries of the divisions of the plexus is extremely rare and it is difficult to distinguish from trunk and cord injuries.

Injuries of the cords: Injuries of the cords manifest with paralysis of the nerves which take origin from the respective cords. Paralysis of the muscles supplied by these nerves and sensory loss in the corresponding autonomous zones are the diagnostic features.

Management

Methods available for the management are:

Nonoperative

It is to be followed only after confirming that the injury is I or II degree according to Sunderland classification. It should never be followed in more severe disruptions.

Appropriate splints are given as per the deficit to give rest to the paralyzed part and prevent contractures, e.g. shoulder abduction splint, spica cast, etc.

Drug therapy is given to reduce edema and increase vascularity. Electrical stimulation and exercise therapy is given during the phase of recovery.

Operative

i. Repair—It is done when there is partial or complete neurotmesis.
ii. Interfasciculus nerve grafting—It is done when there is loss of nerve tissue.
iii. Neurolysis—This is done when there is scarring around the plexus.
iv. Neurotization and nerve transfer—This is done in root avulsion where repair is not possible. It is a highly specialized procedure and to be done by trained personnel.

Some of the recommendations are as follows:

Avulsion of C5 C6 roots:

Spinal accessory nerve to suprascapular nerve; two or three intercostals nerves to musculocutaneous nerve.

If C5 root is available this is grafted to lateral cord to provide elbow flexion, finger flexion and sensation along the radial side of the hand.

If C5 C6 roots are available they are grafted to posterior and lateral cords.

Results of neurotization will be evident only after a period of 2 to 3 years. Good postoperative care and regular follow up is essential for successful outcome.

Reconstructive Procedures

Procedures such as tendon transfers with available tendons, can restore some useful function. Corrective osteotomies to correct rotational deformities, if any are indicated in cases where recovery is poor.

Note: The treatment of brachial plexus injury is challenging. A highly specialized care is necessary for useful functional recovery. Detailed description is beyond the purview of this book and hence not described. The description limits itself to basic principles to be followed in the treatment.

Revision Questions

Q. Erb's paralysis
Q. Klumpke's paralysis
Q. Whole arm paralysis
Q. Erb's point

Essay Question

Q. Discuss the pathoanatomy of the brachial plexus. Describe in detail the mechanism of injury, clinical features, diagnosis and management of brachial plexus injury.

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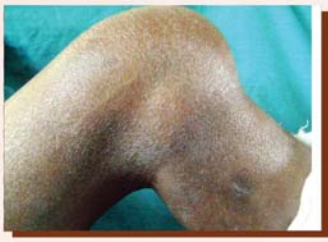


8

Bone and Joint Infections

- *Introduction*
- *Etiopathogenesis of Nonspecific Bone and Joint Infections (Osteomyelitis and Pyogenic Arthritis)*
- *Clinical Features and Diagnosis*
- *Treatment and Outcome of Treatment*
- *Complications and Treatment of Complications*
- *Etiopathogenesis of Specific Bone and Joint Infections: Bone and Joint Tuberculosis, Mycotic Infection, Syphilitic Infection, etc*
- *Clinical Features and Diagnosis of Specific Bone and Joint Infections*
- *Treatment and its Outcome*

Introduction

When the medical fraternity got convinced that microbes were responsible for bone and joint infections, a new era began. This changed the scenario in mid-19th century and the 20th century witnessed the invention of several antibiotics staring from penicillin and sulfa to the more recent, the generations of cephalosporins, imepenam, etc. Inspite of this even today the bone and joint infection poses a challenge to an orthopaedic surgeon. Effective control of infection is many a times not possible especially when bacterial resistance is encountered or if the patient is immunocompromised.

Nonspecific Infections

Osteomyelitis

The word osteomyelitis is derived from Osteon (bone) myelo (marrow) itis (inflammation). It is said to be coined by Nelaton in the year 1834. ***Hence osteomyelitis is defined as inflammation of the bone and the bone marrow.*** This word osteomyelitis has become synonymous with infection of the bone and the bone marrow because most of the time it is the infection that causes this inflammation (Other causes for inflammation such as thermal and chemical burns, mechanical

trauma and radiation exist in less than 1% of cases) and secondary bacterial infection invariably follows.

Route of Entry of Microorganism

Endogenous: In this type, the organism enters the bone from an infective focus existing elsewhere in the body. Two modes of spread have been identified.

i. When the infective focus is present in the vicinity of the bone, the infection spreads to the bone by direct extension, e.g. abscess in the soft tissue.
ii. When the infective focus is a distant one, e.g. a septic tooth (caries tooth), septic tonsil, chronic otitis media, etc the infection spreads through blood stream. This is known as hematogenous spread (most common).

Exogenous: In this type, a break in the soft tissue should occur. This allows direct entry of the organisms from the environment, e.g. penetrating wound, open fracture, etc.

Specific Osteomyelitis

When the bone is infected with those organisms which cause specific features of a disease, e.g. tuberculosis, syphilis, mycotic infection, etc. it is known as specific osteomyelitis. The terminologies such as tuberculosis of the bone, mycotic infection of the bone, etc. are also used to describe the pathology.

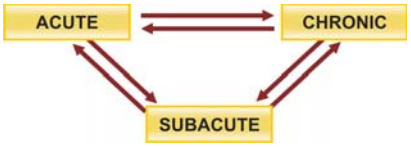
Nonspecific Osteomyelitis

When the bone is infected with those microorganisms which produce general features of infection in the bone (without specific features) it is known as nonspecific osteomyelitis.

Hematogenous Osteomyelitis (Nonspecific Osteomyelitis)

This is a progressive infection of the bone and the bone marrow resulting in destruction.

Simultaneously the body reacts by extruding the dead and devitalized tissue and forming new bone. This reaction is considered as a part of natural process of healing. Many a times it is not possible to eradicate the focus of infection completely and a persistent discharging sinus remains suggesting chronicity. Thus *the infection starts as an acute process and progresses to remain as subacute or chronic when total eradication of infection is not achieved.* Reactivation of this persistent subacute or chronic focus of infection into acute form is also possible.



Etiopathogenesis

Predisposing factors

Age: Common in children and in young. Less common in adolescents and adults (adult risk factors are immunocompromised status, diabetics, drug addiction).

Sex: M:F ratio is 5:1.

Trauma: Trauma may cause a hematoma which may get secondarily infected and result in osteomyelitis.

Location: Metaphysis of a long bone.

Presence of septic focus: A septic tonsil, septic tooth, chronic otitis media, enteric fever septicemia, etc.

Poor nutritional status: General debility and malnutrition.

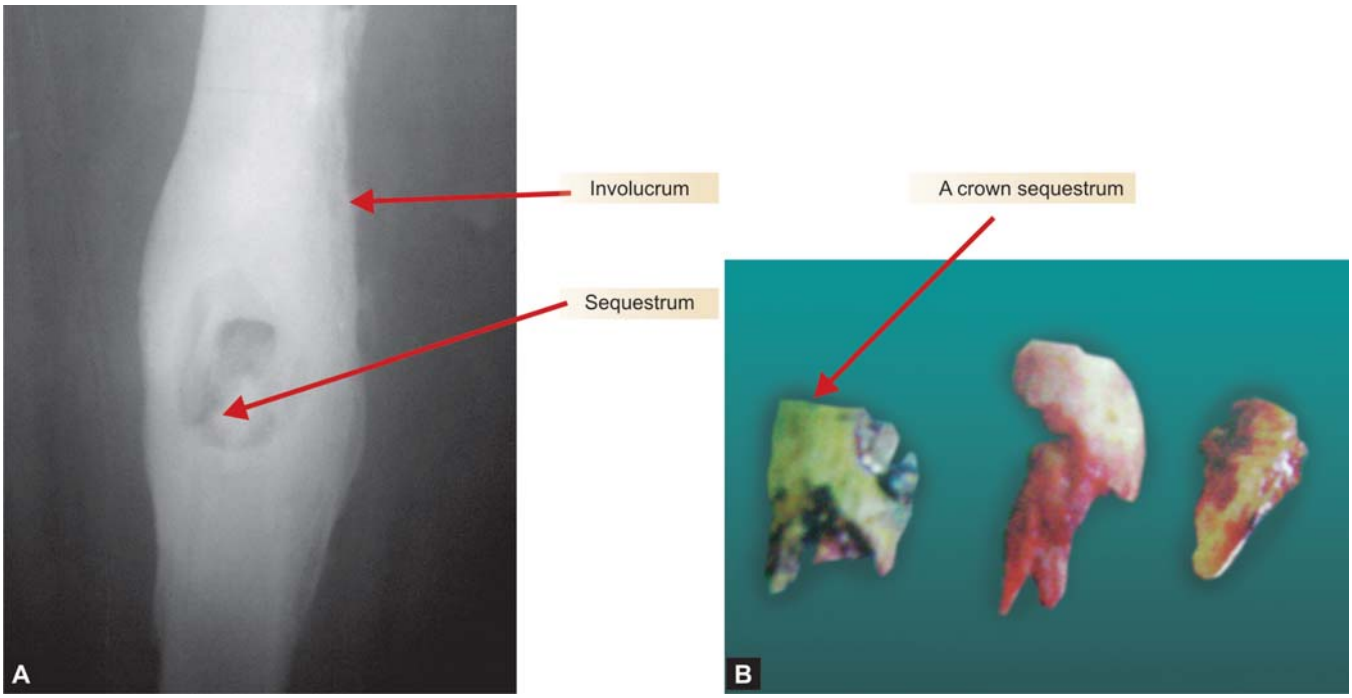
Organisms responsible: Staphylococcus aureus, being the most virulent human pathogen, is the most common organism that causes acute hematogenous osteomyelitis (>95% of cases). Other organisms include *H. influenzae, Streptococcus, E. coli, Proteus*, etc. (< 5% of cases). *Salmonella typhi* infection is more common among patients with sickle cell anemia.

Pathology

The pathology begins with *entrapment of an infective embolus* in the metaphysis of a long bone. It is said to get entrapped in this region because of the following reasons:

- Metaphysis is more vascular.
- Most of the arteries are end arteries.
- Vessels show a hairpin bend.
- Lumen is of a narrower caliber.
- Relative lack of phagocytosis.

Avascular necrosis occurs distal to the block . The dead tissue acts as a good media for the bacteria to multiply. At the same time the body defense mechanisms try to overcome the infection. Hence the ensuing antigen antibody reaction results in the *formation of an exudate.* At this stage, if the body defense mechanisms overcome the infection a small exudate forms which subsequently gets absorbed. The clinical symptoms subside and no further destruction takes place. But if the organism is of high virulence and body defense mechanisms fail to overcome the infection, the process of destruction continues and a large exudate, a bone abscess forms. This large



Figures 8.1A and B

(A) Showing the sequestrum, (B) Types of sequestrum.

exudate exerts pressure on the surrounding vessels and causes compression, *further ischemia and necrosis*. Thus, a vicious cycle sets in. This causes all the systemic signs of acute infection to develop and persist.

The bone abscess thus formed has to find its way out. Its path is blocked proximally by the epiphysis and the growth plate. Its spread into the joint is prevented by the attachment of capsule at the epiphysiometaphyseal region. So the *only way it can flow is down the medullary canal*. It can *also come out and lie in the subperiosteal region* through the normal openings in the bone, the Haversian and the Volkmann's canal as well as the openings of the nutrient vessels. Thus there develops a *subperiosteal and intramedullary abscess.* This subperiosteal abscess ultimately finds its wayout by *breaking the fascia subcutaneous tissue and skin and a sinus of chronic osteomyelitis develops*. The whole process takes about 14-21 days. And upon development of this draining sinus, all the acute signs of infection subside.

That *part of the bone between the intramedullary and the subperiosteal abscesses loses the blood supply* (the subperiosteal abscess cuts off the periosteal blood supply and the intramedullary abscess cuts off the endosteal blood supply). This *results in the formation of a sequestrum.* Smaller sequestra are absorbed by the body and the larger sequestra which cannot be absorbed are thrown out by the body through the sinus (Hence the classical history of a discharging sinus with bony spicules is obtained in chronic osteomyelitis). Still larger sequestra which can neither be absorbed nor be thrown out remain and contribute for a persistent discharging sinus with sprouting granulation tissue at its mouth (Fig. 8.2).

At the same time *the periosteum forms the* new bone from its deeper cambial layer. This is visible radiologically as a layer of new bone formation and is known as *involucrum.*

Sequestrum is defined as 'a dead bone in situ' or a dead bone within a living bone or a dead bone surrounded by granlulation tissue within a living bone (Figs 8.1A and B).

Involucrum is defined as a reactive periosteal new bone formation in osteomyelitis (Fig. 8.1A).

A sequestrum is identified by its dusky naked eye appearance. The margins of the sequestrum are always irregular. It is always found in a cavity and is surrounded by unhealthy granulation tissue.

Sequesrum is always identified radiologically by:

i. Its increased density than the surrounding normal bone.
ii. A parasequestral clear zone of unhealthy granulation tissue which casts a radiolucent shadow.
iii. Its irregular margins.
iv. It is being surrounded by involucrum (at times not always).

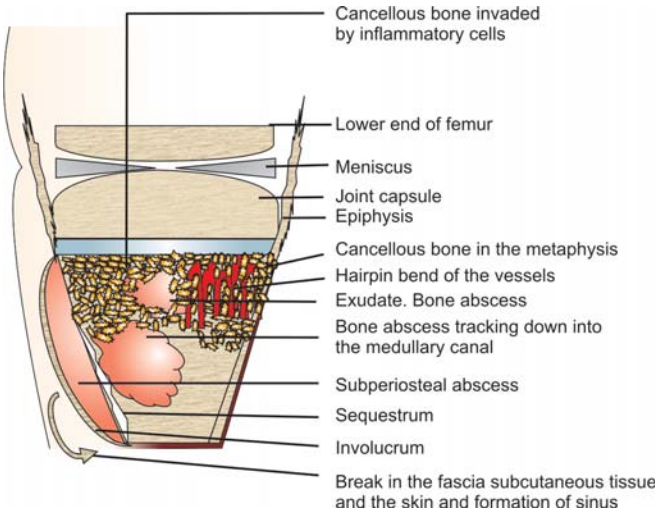


Figure 8.2

Diagrammatic representation of the pathology and progress of acute hematogenous osteomyelitis.

Types of Sequestrum

Ivory—seen in syphilitic osteomyelitis
Black—in fungal osteomyelitis
Sandy or rice grain—tuberculosis
Diaphyseal—in children
Crown—in amputation
Ring—infected pin track
Feathery—tuberculosis involving the ribs.

Clinical Features

May present with following features:

- Presence of focus of infection elsewhere in the body.
- Presence of local trauma.
- Abrupt onset of high grade fever.
- Signs of systemic toxicity.
- Swollen, tender, edematous, limb with refusal to move (Pseudoparalysis).
- Effusion may be present in neighboring joints.

Investigations

Blood investigations

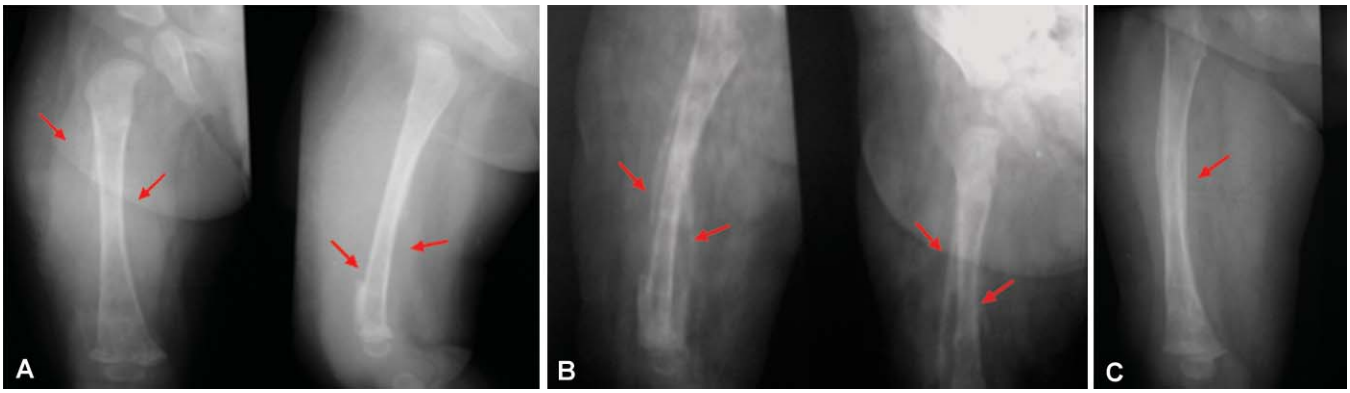
Hb%—may be low.
TC—leukocytosis, DC- increase neutrophils, ESR-elevated
CRP—elevated levels.
Blood culture—positive for the organism.

Other investigations X-ray does not show any signs of infection early in the disease. The first positive radiological sign is periosteal elevation which takes 2-3 weeks (Figs 8.3A to C). Hence not of use in early diagnosis (Fig. 8.4A). Sequestrum and involucrum are seen around 4-6 weeks only when the infection establishes and becomes chronic.

Bone scan, MRI (Fig. 8.4B), CT are of use in the order of preference. These help to confirm the clinical diagnosis and delineate the extent of inflammation.

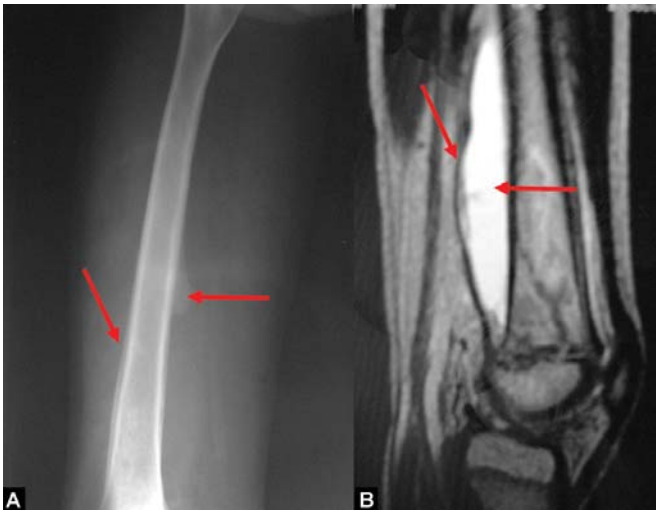
Treatment

Acute osteomyelitis: Aim of the treatment is to *eradicate the infection* and prevent the development of chronic osteomye-



Figures 8.3A to C

Serial radiographs of different stages of osteomyelitis in children showing the outcome following drainage of pus, protection with splint and adequate antibiotic therapy: (A) The first positive radiological sign is the periosteal reaction which takes about 14 days to 21 days to develop. Since in children the periosteum is loosely attached, the spread is extensive along the diaphysis. So is the periosteal reaction. (B) Radiograph at 6 weeks showing a large diaphyseal sequestrum, pronounced periosteal reaction and formation of involucrum following drainage of pus. (C) Showing complete resolution at 3 months following drainage of pus and adequate antibiotic therapy. Note that no sequestrectomy has been done.



Figures 8.4A and B

(A) Radiograph of the femur at the end of 3 weeks after the onset of the disease showing periosteal new bone formation. (B) MRI done at the same time showing that there is collection of fluid around the femur. This collection of fluid lifts the periosteum. Hence, the new bone formation occurring in the cambial layer of periosteum becomes visible (Involucrum).

litis. So, once the diagnosis is made the treatment should be carried out *on an emergency basis.* More the time lapses, more is the spread of infection and more is the difficulty in achieving total eradication. The systematic approach to be followed is as follows:

a. *Antibiotics: Broad spectrum antibiotics* are to be administered immediately after drawing the sample for blood culture.
b. *Drainage of pus:* The pus inside the bone (bone abscess) has to be drained. The *drainage* procedure is known as *decompression.* This can be accomplished by making multiple drill holes or cutting a large window in the involved bone for the drainage of pus. The pus which is under tension is drained out by this procedure. It is done under broad spectrum antibiotic cover and later followed up with specific and adequate antibiotic therapy depending on culture and sensitivity report.
c. *Control of infection:* Locally the *method of continuous irrigation and suction* (i.e. infusing fluid containing appropriate antibiotics can be employed to achieve the eradication of infection.
d. *Immobilization:* The limb is always immobilized and protected with the help of external splint. Long-term antibiotic therapy is necessary for total eradication. The potential hazards of long-term therapy have to be considered and observed during the period.

(Since *Staphylococcus aureus* is the common pathogen for acute hematogenous osteomyelitis, Penicillin group of drugs are the best ones to begin with. Later on according to the blood culture or pus culture reports, appropriate drugs can be started).

e. *Eradication of infection:* Administration of prolonged, adequate and appropriate antibiotic therapy eradicates the focus of infection (Fig. 8.5).

Complications

General

i. Septicemia, pyemia, multiple pyemic abscesses, Death.
ii. Amyloidosis.

Local

i. Chronic/subacute osteomyelitis/pyogenic arthritis.
ii. Pathological fracture.
iii. Limb length discrepancy.
iv. Squamous cell carcinoma of the sinus tract.
v. Deformity.

Chronic Osteomyelitis

When the infection is established in the bone it results in chronic osteomyelitis.

Clinical features

- Persistent discharging sinus fixed to the bone.
- Bony thickening.
- Bony irregularity.
- Bony tenderness may be present.
- Deformity may be present.
- Limb length discrepancy may be present.

Radiological features

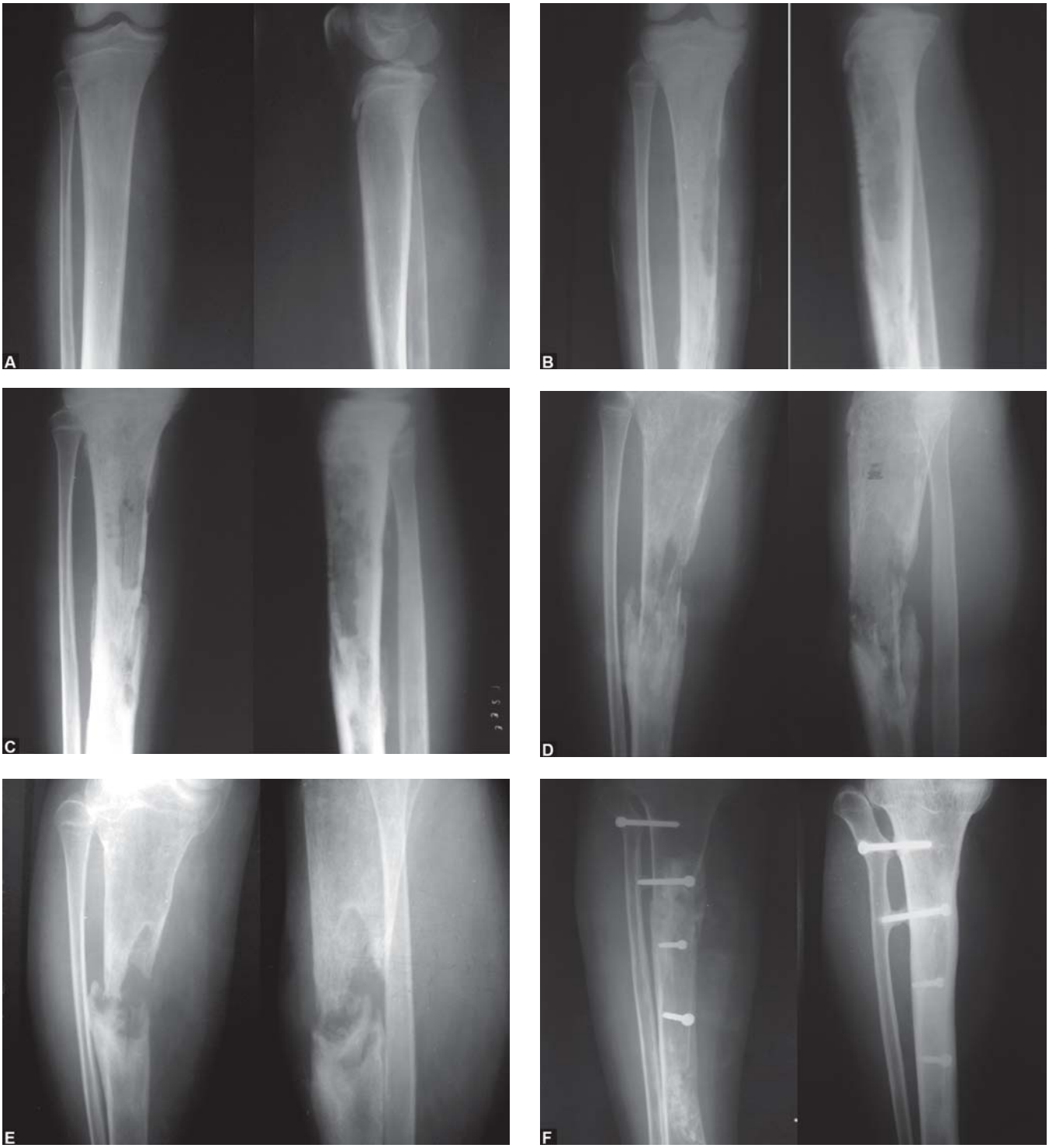
- Bony irregularity.
- Increased density.
- Presence of cavities.
- Presence of sequestrum.
- Presence of involucrum.

Investigations

a. Routine blood investigations. Hb%, TC, DC, ESR, CRP, etc.
b. Pus for culture sensitivity.
c. X-ray of the involved bone shows typical features. (Ref: X-ray- in acute osteomyelitis).

Treatment

- Adequate and specific long-term antibiotic therapy.



Figures 8.5A to F

Radiographs showing the natural course of acute osteomyelitis, presented late for treatment: (A) Though there was pus inside the bone X-ray did not show any abnormality. (B) Three weeks after decompression and adequate drainage of pus. (C and D) Sequestrum formation without any involucrum, at 12 and 18 weeks respectively. (E) Defective nonunion after sequestrectomy at 24 weeks. Note the intact fibula. (F) Successful results after sliding bone graft and cancellous grafting along with attempted tibialization of fibula. Procedure was done after complete *eradication of infection.*

- Sequestrectomy and saucerization:
  - Removal of the sequestrum is done only when the sequestrum is fully separated as confirmed by a parasequestral clear zone and presence of a healthy involucrum. The deep cavity which contained the sequestrum is made shallower in the form of a saucer to facilitate the drainage by lymphatics and the venules.
- Continuous irrigation and suction system may be employed (Figs 8.6A and B).

Subacute Osteomyelitis

It is a distinct form of bone infection wherein the organisms though they are present in the bone, do not cause symptoms of either acute or chronic osteomyelitis. This can occur either primarily, when the virulence of the organism is low or the host resistance is high or secondarily when acute osteomyelitis following treatment turns to subacute.

Brodie's Abscess

It was described by Sir Benjamin Brodie a surgeon in St George's Hospital, London, in 1832.

He came to a conclusion of a subacute infection from a specimen of an amputated limb which he did for intractable pain. He observed a cavity filled with dark colored thick pus and the wall of the cavity consisted of hard white bone.

Age—5-15 yr

Sex—M:F ratio 2:1

Site—Diaphysis common. Metaphysis next common site. Rare in the epiphysis.

Clinical features: Mild to moderate pain. A hard swelling may be appreciated.

Radiological features: The disease presents with variable features. Classically, a cystic lesion with surrounding sclerotic bone is the feature. But in some instances onion skin periosteal bone resembling Ewing's, cortical hyperostosis resembling osteoid osteoma, etc. are observed (Figs 8.7A and B).

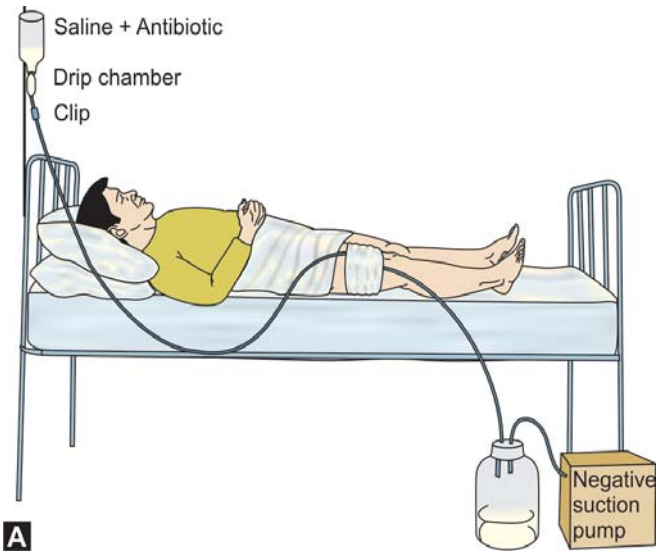
Treatment Once the diagnosis is established long-term antibiotic therapy is started taking into consideration the bacteriological culture sensitivity report from the sample obtained from the cavity.

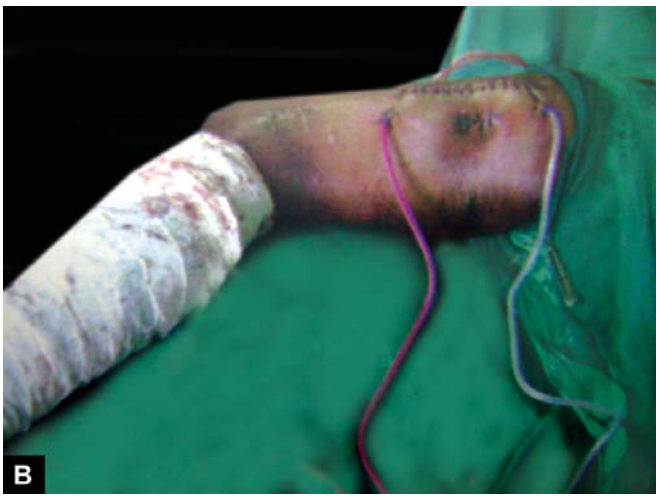
Surgery of draining the cavity under antibiotic cover is considered only when nonoperative treatment fails.

Sclerosing Osteomyelitis of Garre

It is a nonsuppurative ossifying periostitis which occurs as a response to low grade infection and ensuing irritation. Radiograph shows sclerosis all round the bone.

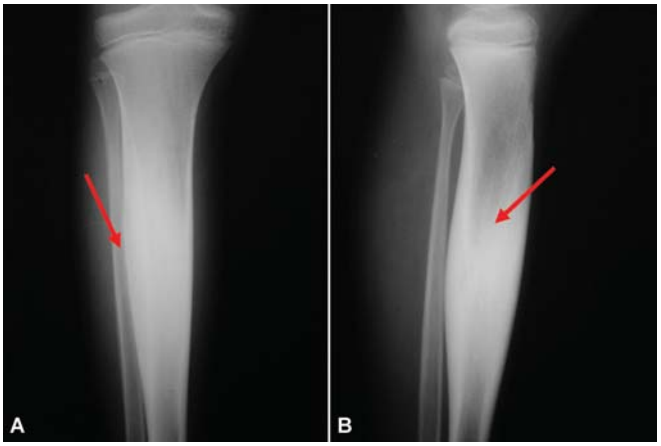
Bone scan and PET scan may be useful in the diagnosis. Treatment consists of adequate antibiotic therapy and when feasible and indicated excision of the focus.





Figures 8.6A and B

Showing the application of continuous irrigation and suction in an attempt to achieve control of infection.



Figures 8.7A and B

Radiograph showing Brodie's abscess in the diaphysis of tibia. The only complaint the child had is low grade pain. Limp local signs of acute inflammation and constitutional symptoms were absent.

Revision Questions

Q. Define osteomyelitis.
Q. What are the organisms responsible for acute hematogenous osteomyelitis?
Q. Discuss the mode of spread of infection to the bone in osteomyelitis.
Q. Discuss the pathogenesis of chronic osteomyelitis.
Q. Define sequestrum.
Q. Define involucrum.
Q. Write notes on
   a. Decompression.
   b. Sequestrectomy and saucerization.
   c. Brodie's abscess.
   d. Sclerosing osteomyelitis of Garre.
Q. What are the complications of acute hematogenous osteomyelitis?

Essay Question

Q. Discuss the etiopathogenesis, diagnosis and management of acute hematogenous osteomyelitis. Enumerate the complications and discuss in detail the management.
Q. Discuss the pathogenesis of chronic osteomyelitis, its clinical features, diagnosis, complications and management.

Acute Suppurative Arthritis

This is known by various names as acute pyogenic arthritis, acute septic arthritis, acute pyoarthrosis and acute infective arthritis. It refers to presence of pus in the joint secondary to infection of the joint.

Age of onset—common in infants and children can occur at any age.

Sex—M:F ratio 5:1

Site—Any joint. Common in hip and knee.

Mode of Spread

- Exogenous, e.g. penetrating injury.
- Endogenous, e.g. hematogenous form a distant foci ; local extension form the vicinity.

Organism

Staphylococcus aureus 90%, *Streptococcus, H. influenzae, E. coli,* Proteus, etc. are responsible in remaining 10% of the patients

Focus of infection—spreads from the synovium or from the bone.

Pathology

Once the infection gets into the joint there is seropurulent exudate formation. The articular cartilage gets destroyed very rapidly due to the enzymatic degradation resulting from the enzymes released from the bacteria as well as from the inflamed synovium and inflammatory cells. The subchondral bone gets exposed and later gets eroded too. If the pus is not drained surgically, a sinus develops.

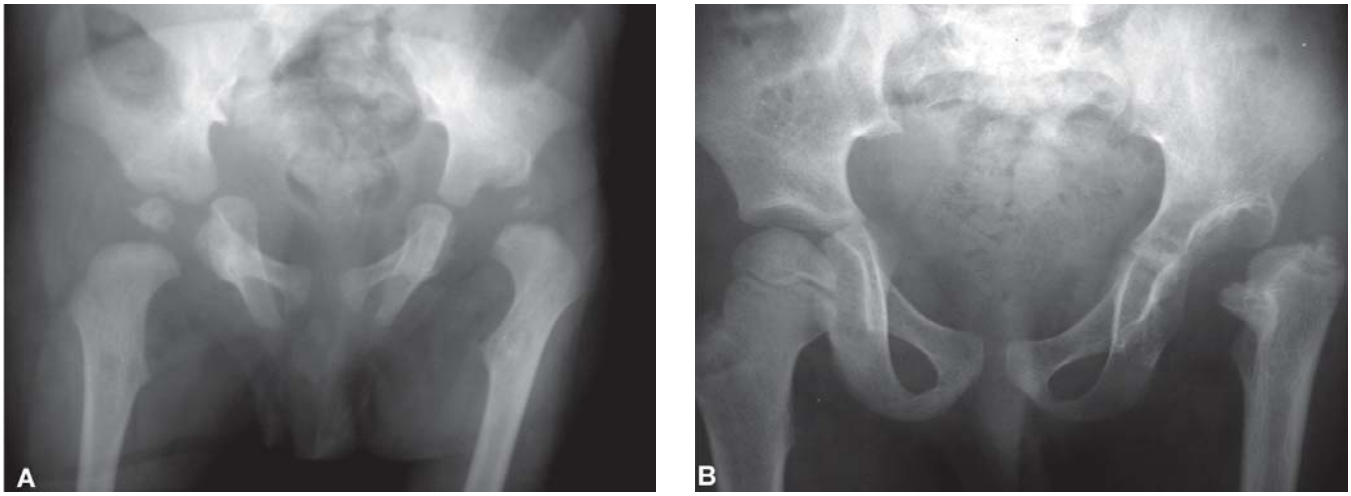
Sequel depends on the speed at which the infection is controlled. If the infection is controlled before the destruction of the cartilage, the joint becomes normal. No residual deformity is seen. After some destruction of cartilage, the joint goes in for fibrous ankylosis. After considerable destruction of cartilage, the joint goes in for bony ankylosis. If the infection takes natural course then bony ankylosis with deformity is the result.

Diagnosis

Diagnosis is mainly on clinical grounds. It is not difficult because the patient presents with all the signs of acute infection, i.e. calor, rubor, dolor, tumor with painful immobile joint. Signs of systemic toxicity such as high grade fever with delirium may also be present. Investigations such as TC, DC, ESR, CRP, ultrasound, X-ray, MRI are useful and should be done as per indication.

Treatment

Emergency surgical drainage of the joint of pus and debris under cover of systemic antibiotics is the treatment of choice.



Figures 8.8A and B

(A) Radiograph showing the sequel of a Tom Smith's arthritis at 2 years. At the age of three months the infant had presented with the disease within 24 hrs of onset of symptoms. The pus was drained under adequate antibiotic cover within 24 hrs of presentation. Though treated aggressively and control of infection was achieved, note the hypoplastic change in the left femur and pathologic dislocation of the left hip joint. (B) Radiograph of an adolescent with Tom smith's arthritis in childhood showing complete destruction of the femoral epiphysis and dislocation. This results in gross shortening and exaggerated movements in the hip. The hip is painless.

Continuous irrigation and suction can be employed for achieving good control of infection. Immobilization is essential till control of infection is achieved. This is followed by gradual mobilization as early as possible to avoid stiffness of the joint. Systemic antibiotics are used for considerable period to get rid of any residual focus of infection depending on the organism isolated in culture and its antibiotic sensitivity.

Arthrodesis is the treatment of choice for unsound ankylosis. In selected cases of hip and knee ankylosis with no reactivation of the disease, total joint arthroplasty can be considered. This procedure considerably reduces the morbidity.

Tom Smith's Arthritis of Infancy and Childhood

This is a type of pyogenic arthritis occurring due to osteomyelitis of the upper femoral metaphysis. The focus of infection can also start from the synovium. Since the capsular attachment is beyond the physis the infection directly spreads into the joint causing pyogenic arthritis. The epiphysis invariably gets destroyed and the hip dislocates. Prompt and early attention with an accurate diagnosis followed by drainage of pus under antibiotic cover may reduce the morbidity. Delay causes severe destruction of the joint due to lysosomal enzymes and increase in pressure within the joint, which further disturbs the vascularity (Figs 8.8A and B).

Revision Questions

Q. Define septic arthritis.
Q. Discuss the etiopathogenesis of acute septic arthritis.
Q Discuss the treatment and sequel of acute pyogenic arthritis.

Essay Questions

Q. Discuss the etiopathogenesis, clinical features, diagnosis, management and complications of acute pyogenic arthritis.
Q. What is Tom Smith's arthritis? Discuss the diagnosis and management of Tom Smith's arthritis and its sequel.

Specific Infections

Tuberculosis of bones and joints

The disease tuberculosis is as ancient as mankind. Hippocrates during his period recorded this disease as phthisis or consumption. In Charaka and Sushrutha Samhitha in ancient India it is identified as Yakshma. Studies on Egyptian mummies (2050 BC) have shown positive results for *M. tuberculosis, M. africanum* and *M. bovis*. Hippocrates was able to differentiate pyogenic abscesses from tubercular cold abscesses and to point out the fact that paraplegia in tuberculosis of the spine improved once the cold abscess pointed over the back (natural decompression).

The tuberculosis of bones and joints is always secondary to a primary focus elsewhere in the body. Many a times this primary focus is not detectable. The bacilli enter the bone (dissemination occurs) through the bloodstream and get lodged in different areas. Thus, the lesion may be found in the synovium or in the bone. It is said that it takes almost 18-24 months after the development of primary lesion to cause bone and joint tuberculosis. Poor nutrition, malnutrition, poor sanitation and poor resistance are the predisposing factors for the infection. Age is not a bar for bone and joint tuberculosis. It can occur at any age. But when it occurs at a young age, the destruction of the growing bone results in severe deformities. This is specially true in the spine.

The 3 stages of the disease were identified in the pre-antitubercular drug therapy era as follows:

I Early or stage of onset.
II Florid or stage of active destruction.
III Stage of repair or stage of healing or stage of ankylosis. (Was seen only in those who survived. More than 70% died of the disease).

The typical lesion of tuberculosis histopathologically, is known as a tubercle. It is a granuloma. It consists of a central area of caseation necrosis surrounded by epitheloid cells, macrophages, lymphocytes and Langhans' type of giant cells. It indicates that granuloma is a defensive response to bacterial invasion. It not only cordons off the bacteria but also allows the immune system to kill the bacteria. The T lymphocyte (CD4+) secrete cytokines (interferon gamma) which activates macrophages to destroy the engulfed bacteria, T lymphocyte (CD8+) also directly kills the infected cell.

Investigations

- Hb% - low; TC- moderate increase; DC- predominantly lymphocytes; ESR-elevated.
- X-ray—always shows narrowing of the joint space as earliest feature followed by destruction of the adjacent bones. Surrounding area of rarefaction is a striking feature. Soft tissue shadow of cold abscess may also be seen.
- CT, MRI—dilineate extent of soft tissue and bone involvement better than X-rays.
- Mantoux test—when positive tells that the patient has or has had tuberculosis.
  The test is negative in milliary tuberculosis.
- IgA and IgG—immunoglobulin assay may be useful as an additional evidence.
- Tissue samples showing positive PCR (polymerase chain reaction), presence of bacilli in the smear, and classical histopathological picture is confirmatory.

Treatment

Standard medical therapy in an adult comprises of using a combination of minimum three of the following drugs.

- Streptomycin (0.75-1.0 G).
- INH (200-300 mg).
- Rifampicin (300-450 mg), INH (300 mg).
- Ethambutol (800-1200 mg).
- Pyrazinamide (750-1250 mg).

Drugs are used as per body weight and continuation of therapy for a minimum period of 18 months is absolutely necessary for cure. Daily regimen ensuring regular intake of drugs gives the best results. DOTS trial is yet to prove its efficacy and so are short-term therapies. It is authors experience that reactivation of the lesion and manifestation as some other form of tuberculosis is common with DOTS and short-term antitubercular therapy in spite of taking drugs as per the schedule.

Good sanitation and good nutrition plays a great role in the recovery.

Role of surgery in active tuberculosis is to debride the tuberculous debris and repair the bone loss by adopting a stabilization procedure. It aims at rendering a subject totally disease free at the same time attempting to restore the functional capacity to the best possible extent. Also aims at preventing future complications.

Surgery is always done under cover of antitubercular drug therapy (ATT). The drug therapy is continued even after surgery as per schedule. Role of surgery in a burnt-out case is reconstruction, thereby attempting to restore maximum function.

Injection streptomycin has a disadvantage of administration by intramuscular route and over a period of time patient may become uncooperative. But till such time it is guaranteed that he cannot become a defaulter. Autotoxicity is a common complication in elderly patients; hence, to be used with caution.

Essay Question

Discuss the clinical features, diagnosis and drug therapy of bone and joint tuberculosis in general.

Tuberculosis of the Spine

It was popularly known as Pott's disease in the 18th century named after Sir Percival Pott a successful British surgeon. The name still continues.

Development of Vertebral Column

By about 4th week in an embryo, paired cubical masses called somites start developing in the primitive mesenchyme. These somites differentiate into dermatome, myotome and sclerotome. Sclerotome surrounds the notochord. Each sclerotome starts segmentation and condenses into a cephalic mass and a caudal mass and a posterior element. The formation of the vertebra thus begins. Blood supply also follows this embryonic pattern. Thus each segmental artery in the vertebral column supplies the lower half of the one, the upper half of the other caudal and cephalic mass respectively. The notochord ultimately disappears in the region of the sclerotomes except in the region of the intervertebral disc, where it remains as nucleus pulposus. Nucleus pulposus along with fibrous annulus fibrosus, makes an intervetebral disc.

Clinical importance of this embryonic blood supply is shown by the classical lesion of tuberculosis of the spine. It involves the lower half or the upper half of the vertebra. Manifests radiologically as narrowing of the disc space, (one of the earliest radiological signs). This also proves the fact that dissemination of focus occurs through the blood-stream.

Batson's Plexus of Veins

These are veins that connect pelvic veins with internal vertebral venous plexus. They do not have valves.

Hence dissemination of focus from the genitourinary tract and abdomen to the vertebra can occur easily.

Spinal tuberculosis is the most common type among skeletal tuberculosis and accounts for about 50% of bone and joint tuberculosis. It affects males and females equally and does not show any predilection to age. Though it is common in the first three decades of life it can occur at any age. Lesion is common in lower thoracic spine followed by upper thoracic cervical, lumbar and sacral spine.

Probable reasons for lower thoracic spine as common site:

- Batson's plexus of veins
- Cysterna chyli begins at lower border of T12.
- Close relation of thoracic duct.
- Continuous movement with respiration which helps in dissemination.
- More spongy bone.

Vertebral Lesion

a. Paradiscal—Involving adjacent vertebra, intervertebral disc involvement is a late feature. Presents with narrowing of the disc space radiologically (>90%). Vertebral body (5-7%) (Figs 8.9A to C and 8.10).
b. Central—Involvement of the body. Presents with cavity formation, no collapse of the body is seen radiologically but a cystic lesion is seen.
c. Lateral—Lateral segments of the body are involved. Shows lateral collapse of the body radiologically.
d. Anterior— Anterior segments are involved. Shows anterior collapse of the body radiologically.
e. Posterior elements—Articular facets, posterior spinal disease and spinal tumor syndrome (<5%).
f. Lesions involving multiple vertebrae and skipped lesions.

Clinical Features

a. Pain which is dull and vague initially becomes progressively severe and constant. Aggravated by movement of the spine particularly jarring.
b. Loss of weight. Loss of appetite.
c. Fever and malaise with evening rise of temperature and night sweats.
d. Paraspinal spasm.
e. Presence of gibbus (angular, knuckle and round kyphosis).
f. Tenderness over the spine.
g. Presence of cold abscess.
h. Aldermanic gait—small steps with straight back to avoid jarring.
i. In cervical spine patient supports the chin over the palm of the hand.
j. Neurologic symptoms may be present.

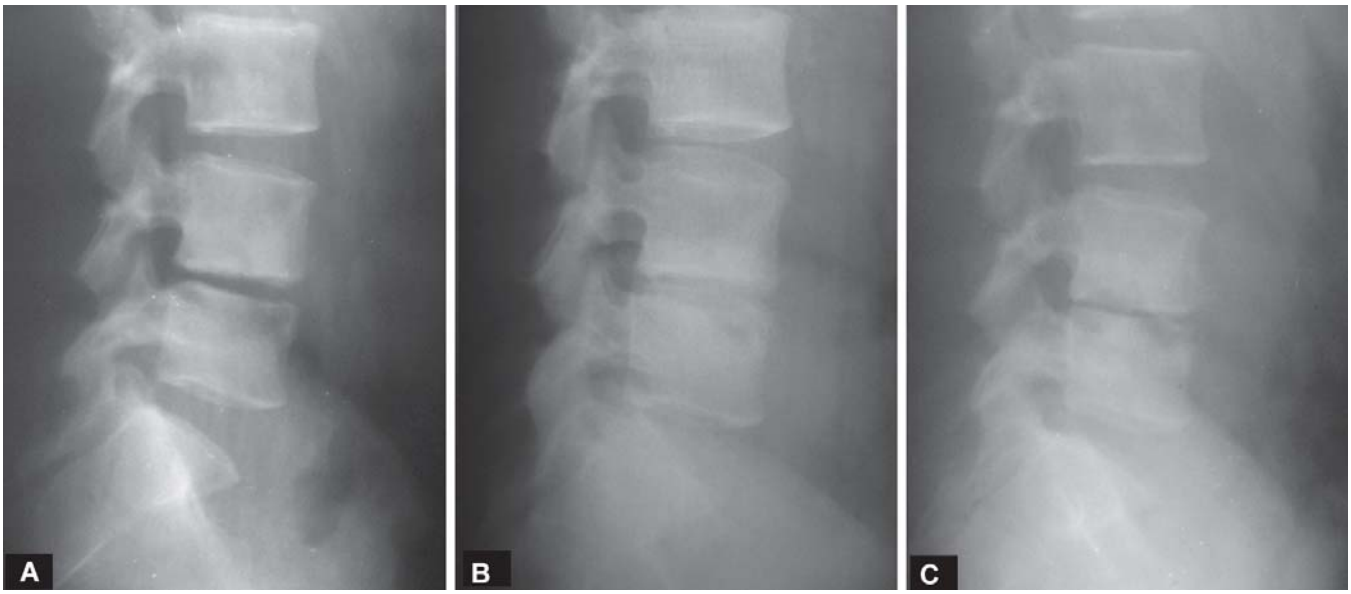
Involvement of adjacent portions of the vertebra will result in angular kyphosis (Figs 8.11A and B).

Involvement of more than two but less than three will result in knuckle kyphosis (Figs 8.12A and B).

Involvement of more than three vertebrae will result in round kyphosis.

Cold Abscess

It is collection of fluid and debris in tuberculosis. It is called cold because it is devoid of signs of acute inflammation such as heat, redness, and pain. In the cervical spine, radiologically, the soft tissue shadow of a cold abscess appears like a bird's nest and in the mediastinum it is heart-shaped or fusiform.



Figures 8.9A to C

Radiographs taken at monthly intervals at 1st, 2nd and 3rd months of presentation respectively. (A) Radiograph shows narrowing of the joint space which is the earliest classical sign of spinal tuberculosis. (B and C) Radiographs show progressive narrowing of the joint space as well as development of bony lesions which are visible . Healing occurs in the form of interbody fusion.

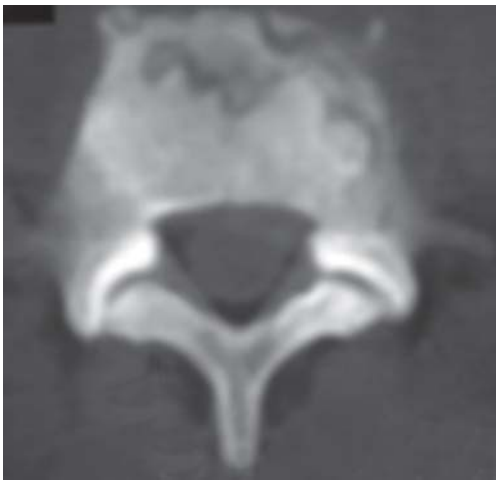


Figure 8.10

Showing the CT scan of the L5 vertebra taken in the 1st month itself at presentation. Note that the lesions are spotted much before the X-ray evidence.

Position and Course of Cold Abscess in Spinal Tuberculosis (Figs 8.13 to 8.15)

In any region

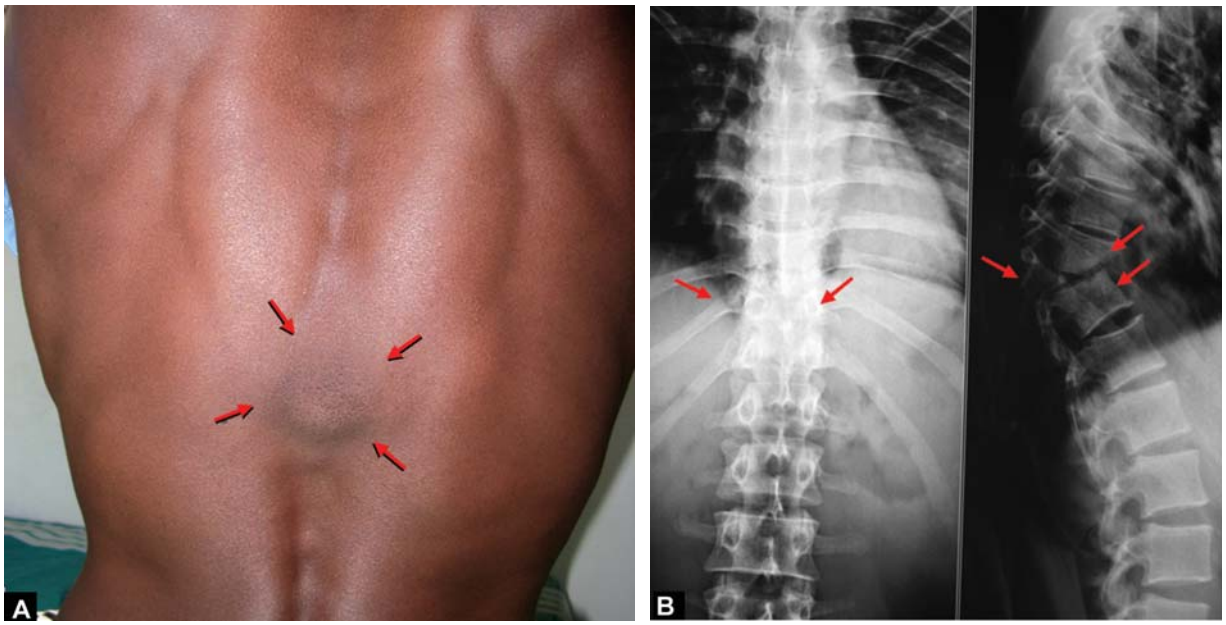
a. Within the prevertebral fascia as prevertebral abscess in front of the vertebral body
b. Within the spinal canal compressing the spinal cord.

In the cervical region

a. Retropharyngeal abscess (cervical prevertebral abscess). Acute pyogenic abscess is in front of the prevertebral fascia and one side of the mid line.
b. In the mediastinum, tracking down from the prevertebral region.
c. At the back of the neck, tracking along the posterior division of the spinal nerves.
d. At the posterior border of the sternomastoid muscle tracking laterally.
e. In the axilla, through the open mouth of axillary sheath.

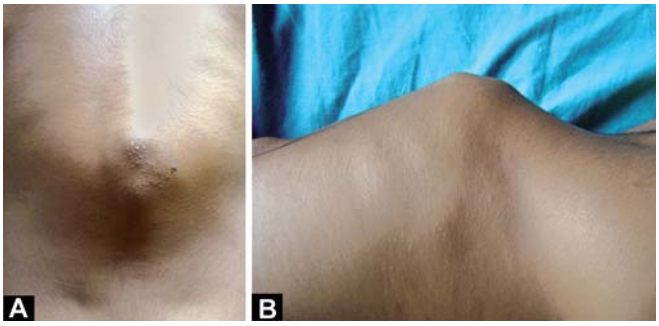
In the thoracic region

a. May remain as prevertebral abscess in the posterior mediastinum.
b. Through the openings in the diaphragm, namely the lateral lumbocostal arch, the medial lumbocostal arch and median arcuate ligament tracks distally out of the thorax.
    - Lateral lumbocostal arch:
        i. Traversing between the lumbodorsal fascia and quadratus lumborum muscle may remain behind the kidney.
        ii. Traversing along the 12th thoracic, iliohypogastric and ilio inguinal nerves to the anterior abdominal wall.



Figures 8.11A and B

(A) Clinical picture and (B) Radiograph showing an angular kyphosis. Note near total destruction of T11 and posterior subluxation. T10 is partially destroyed. T12 has subluxated posteriorly following T11. Altogether two vertebrae are involved resulting in an angular kyphosis. In the AP view note that the 11th rib is free of its attachment to the vertebral body because the vertebral body is destroyed almost completely.



Figures 8.12A and B

Showing a knuckle kyphosis.

- Medial lumbocostal arch:
  It is the open mouth of psoas sheath. It can track down to the insertion of psoas muscle into the thigh.
- Median arcuate ligament:
  The median arcuate ligament crosses over the aorta. So the abscess may spread along the aorta and its branches.

c. Along the intercostal nerves in the anterior middle or posterior axillary lines in the chest wall.
d. In the rectus sheath over the anterior abdominal wall.

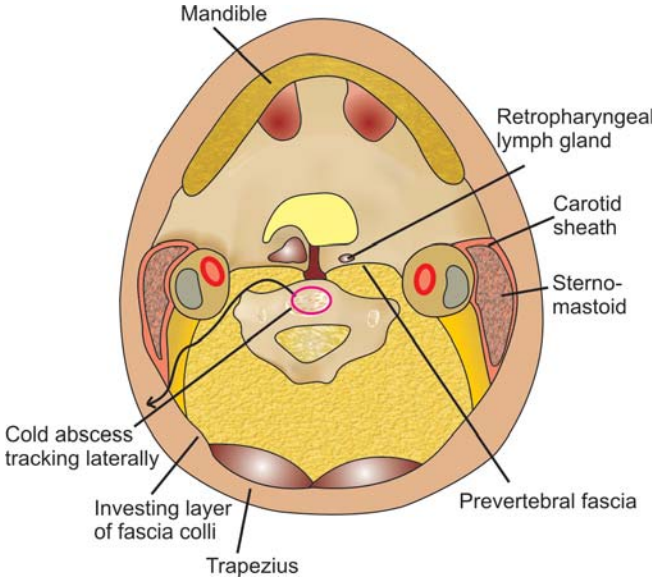


Figure 8.13

Showing the paths of spread of cold abscess in the cervical spine.

In the lumbar region

a. Along the aorta and its branches, internal pudendal, superior gluteal, etc.

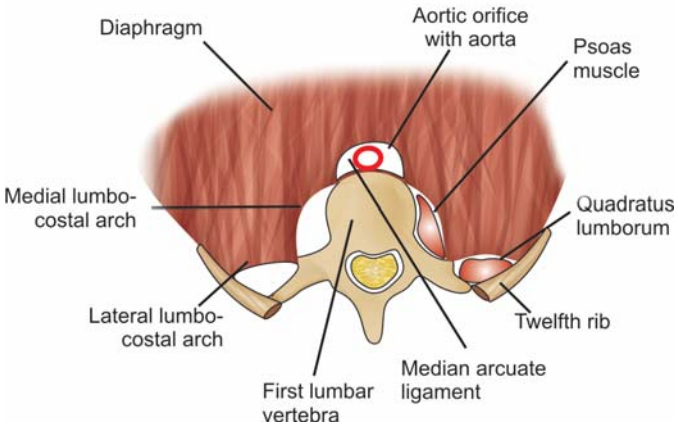


Figure 8.14
Showing the paths of spread of the cold abscess in the thoracic region.

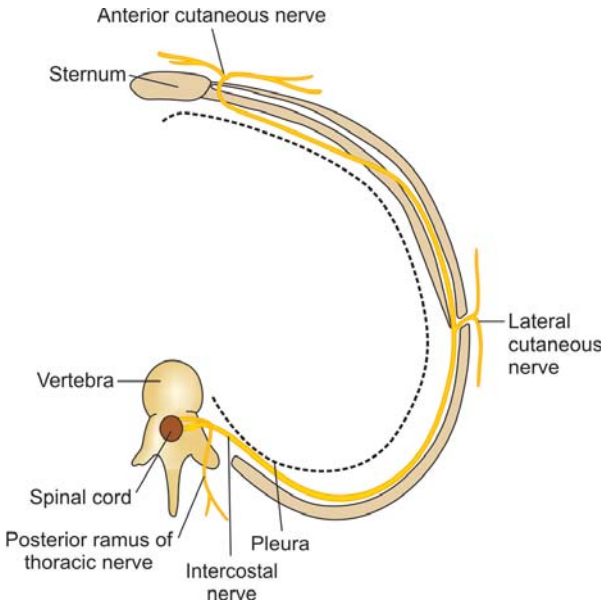


Figure 8.15
Showing the lateral path for the spread of the thoracic cold abscess along the intercostal nerve.

b. Along the sheath of psoas or quadratus lumborum.
c. Along the lumbar, femoral and obturator nerves into the thigh.
d. Into the lumbar triangle of Petit, through the flat muscles of the abdominal wall.

Investigations

- Hb% low; TC—moderate increase; DC—predominantly lymphocytes; ESR—raised
- X-ray—shows picture of destruction of the vertebra as per involvement. Narrowing of the intervertebral space, anterior collapse, lateral collapse, cavity formation, soft tissue shadow of cold abscess, etc. may be seen.
- CT, MRI—done for better evaluation of bone and soft tissue involvement.

Confirmatory tests

- Identifying presence of bacteria in the pus. Smears are made and stained for acid-fast bacilli.
- Classical tubercle seen histopathologically under microscope.
- PCR (Polymerase chain reaction), positive for tuberculosis.
- Pus culture and guinea pig inoculation. Takes long time and not done regularly.

Pott's Paraplegia

When the thoracic spinal cord is involved in tuberculosis it results in paralysis of the lower limbs. In severe and late cases bladder and bowels also are involved. This is known as Pott's paraplegia named after Sir Percival Pott.

Sequential events of paralysis:
a. Muscle weakness.
b. Awkward gait.
c. Incoordination.
d. Spasticity.
e. Paraplegia in extension.
f. Paraplegia in flexion.

Types of Pott's Paraplegia

As proposed by Sorrel-Dejerine and later by Seddon.
A. Early onset paraplegia: This is the paralysis occurring during the florid phase of the disease, i.e. within 2 years.
B. Late onset paraplegia: This is the paralysis occurring many years after the disease when the disease has become quiescent and sometimes even without reactivation.

Causes for Paraplegia as Put forth by Seddon (Based on)

Early onset
i. Inflammatory
   a. Abscess, inflammatory tissue or a caseating mass.
   b. Spinal tumor syndrome.

c. Posterior spinal disease.
d. Infective thrombosis of the cord.

ii. Mechanical
a. Pathologic dislocation.
b. Compression by sequestra, loose fragments of bone or disc, pressure of the granulation tissue and debris.

Late onset

i. Inflammatory: Persistent activity or reactivation of the disease.
ii. Mechanical: Progressive stretching of the cord ultimately jeopardizing its circulation.

In brief the cause for paralysis is either the active disease process itself which damages the cord or the sequestrated material and structural abnormality which mechanically interferes with the function of the cord.

Treatment

Medical line of treatment Always indicated when there is no neurological involvement. Drainage of the cold abscess is the only surgical procedure that may be necessary. It is chosen when investigations and initial assessment do not lead to the absolute indications for surgery (indications for surgery as proposed by Griffths and Seddon are described in surgical line of treatment)

1. Antitubercular drug therapy has to be started at earliest. It is author's firm belief that one should not hesitate to use injection streptomycin 0.75-1.0 gm daily at least for 60-90 days whenever indicated. A minimum of three if needed four drugs have to be used to achieve an early effective control of the disease. The site of injection has to be rotated daily (on four limbs) for maximum comfort. The toxicity of the drugs should always be borne in mind during therapy and the therapy should be modified accordingly. Patient is to be evaluated on a daily basis and if necessary liver enzyme study (SGOT/ALT, SGPT/AST, Gamma GT) and renal function tests may have to be done.

   Secondary infection if any should be taken care of by appropriate antibiotics.
2. Improve the nutritional status by giving good attention to the food intake.
3. Immobilize the spine by appropriate brace or even a plaster of Paris jacket when rigid immobilization is indicated. This helps not only in giving comfort to the patient but also prevents development of severe deformities. Also helps in early consolidation of the diseased bone.
4. Watch on signs of improvement (if no improvement is observed surgical treatment is indicated).

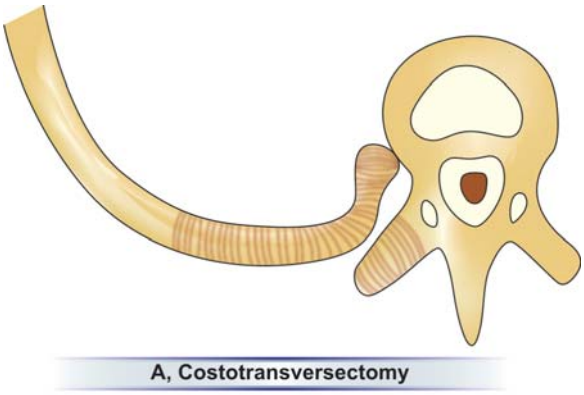
Surgical line of treatment: Single most important indication for surgery in tuberculosis is presence of paralysis. This is further analyzed and indications are formulated.

Indications for surgery (based on indications as put forth by Griffiths and Seddon):

- *Absolute indications:*
  1. Paraplegia developing when the patient is on adequate medical line of treatment.
  2. Paraplegia not showing improvement with adequate medical line of treatment.
  3. Paraplegia progressing even with adequate medical line of treatment.
  4. Complete paraplegia with no improvement with adequate medical line of treatment.
  5. Severe spastic paraplegia.
  6. Paraplegia of rapid onset.
  7. Long standing paraplegia >6 months, paraplegia in flexion, flaccid paraplegia, etc.
- *Relative indications:*
  1. Recurrent paraplegia.
  2. Paraplegia in old age.
  3. Painful paraplegia.
  4. Paraplegia with complications such as urinary infection and stones.
- *Rare indications:*
  1. Posterior spinal disease.
  2. Spinal tumor syndrome.
  3. Cervical spinal tuberculosis.
  4. Severe cauda equina paralysis.

Surgical Procedures

1. *Drainage of abscess:* An abscess large enough for drainage is drained under cover of antitubercular drugs and antibiotics. Smaller abscesses resolve with adequate ATT.
2. *Costotransversectomy:* It is a surgical procedure for drainage of a large paravertebral abscess. The drainage is accomplished by excising the ribs , the transverse process and corresponding pedicle. A minimum of one and maximum of two ribs and transverse processes are excised (Fig. 18.16A).
3. *Anterolateral decompression:* It is a surgical procedure which decompresses the anterolateral portion of the spinal cord by excising posterolateral part of the vertebral body, corresponding ribs, transverse processes and pedicles. This is indicated when costotransversectomy does not yield pus



Figures 8.16A and B

Showing the portion of the bone removed (the shaded portion) in costotransversectomy and anterolateral decompression.

under tension. The tubercular debris and granulation tissue is cleared by this procedure (Fig. 18.16B).

Minimum of three or maximum of four ribs and corresponding transverse processes are excised.

4. *Anterior decompression and fusion:* It is a procedure employed when the lesion is anterior and more anterior portion of the vertebral body is destroyed. Spinal fusion always follows decompression.
5. *Laminectomy:* Done only in cases of posterior spinal disease and spinal tumor syndrome.

Order of recovery of paralysis The recovery of paralysis always takes place in the following order irrespective of modalities of treatment.

1. Vibration and joint sensation.
2. Temperature, touch, pain.
3. Voluntary motor activity.
4. Sphincter functions.
5. Wasting of muscles.

Factors governing prognosis:

1. Age: Younger the age better is the prognosis and vice versa.
2. General condition: Good general condition better is the prognosis and vice versa.
3. Duration: Shorter the duration better is the prognosis and vice versa.
4. Severity: Incomplete paralysis without bladder and bowel involvement and sensory loss better is the recovery and vice versa.
5. Type: Early onset better is the prognosis and vice versa.
6. Speed of onset: Slow onset better is the prognosis than rapid onset.
7. Kyphotic deformity: More than 60° poorer is the prognosis and vice versa.

Revision Questions

Q. Discuss the development of the vertebral column.
Q. Which is the most common site for spinal tuberculosis and why?
Q. What are the types of lesions found in the tuberculosis of the spine?
Q. What is a cold abscess? Discuss the natural mode of spread of cold abscess in different regions of the spine.
Q. Discuss the management of cold abscess.
Q. Write short notes on
   a. Bird's nest abscess.
   b. Vertebral lesions in tuberculosis.
   c. Battson's plexus of veins.
   d. Pott's paraplegia.
   e. Posterior spinal disease.
   f. Spinal tumor syndrome.
Q. Write notes on
   a. Anterolateral decompression.
   b. Costotransversectomy.
   c. Anterior decompression.

Essay Questions

Q. Discuss the pathogenesis, clinical features diagnosis and management of tuberculosis of the spine.
Q. Classify Pott's paraplegia. Discuss the management of Pott's paraplegia in detail and its prognosis.

Tuberculosis of the Hip

Tuberculosis of the hip is next common site after tuberculosis of the spine. The disease is common during 1st 3 decades. The foci of infection may get lodged as a solitary or as multiple deposits during the phase of dissemination. They get lodged in the synovium or in the bones of the hip. In the bone the order of frequency starts from the roof of the acetabulum

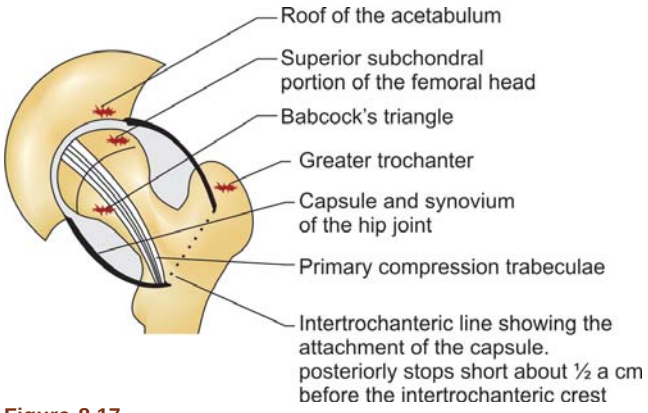


Figure 8.17

Diagram showing common sites of origin of foci of tuberculosis in the hip joint.

followed by superior subchondral portion of the femoral head, Babcock's triangle and finally the greater trochanter. In the bone it begins as tuberculous osteomyelitis (Fig. 8.17). From both the foci the infection subsequently spreads to the joint and the articular cartilage gets destroyed. Thus, osteoarticular tuberculosis developes.

Babcock's triangle is a triangular area at the inferomedial portion of the neck of the femur identified radiologically. Medially it is bounded by the epiphyseal plate or by stress trabeculae, laterally by primary compression trabeculae and inferiorly by the neck of the femur (Fig. 8.18).

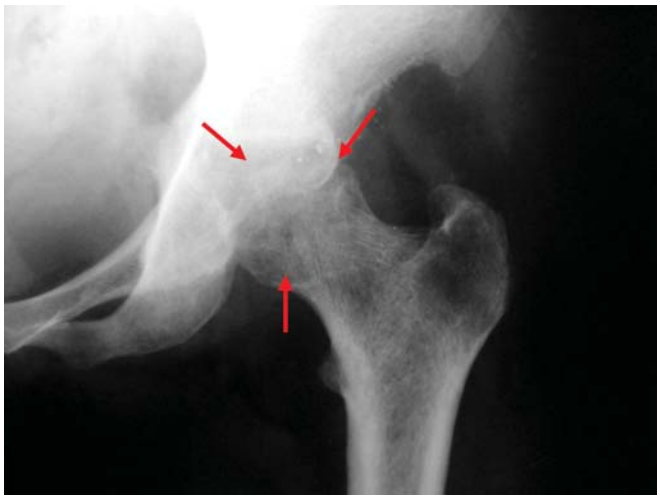


Figure 8.18

Radiograph showing narrowing of the joint space with a lesion in the 'Babcock's triangle. Patient was a known case of pulmonary tuberculosis and was not taking regular treatment. Also note the soft tissue swelling around the joint. Skeletal tuberculosis is almost always secondary to a primary lesion elsewhere in the body.

Clinical Features

- Low grade fever with evening rise of temperature and night sweats.
- Night pains.
- Loss of weight and appetite.

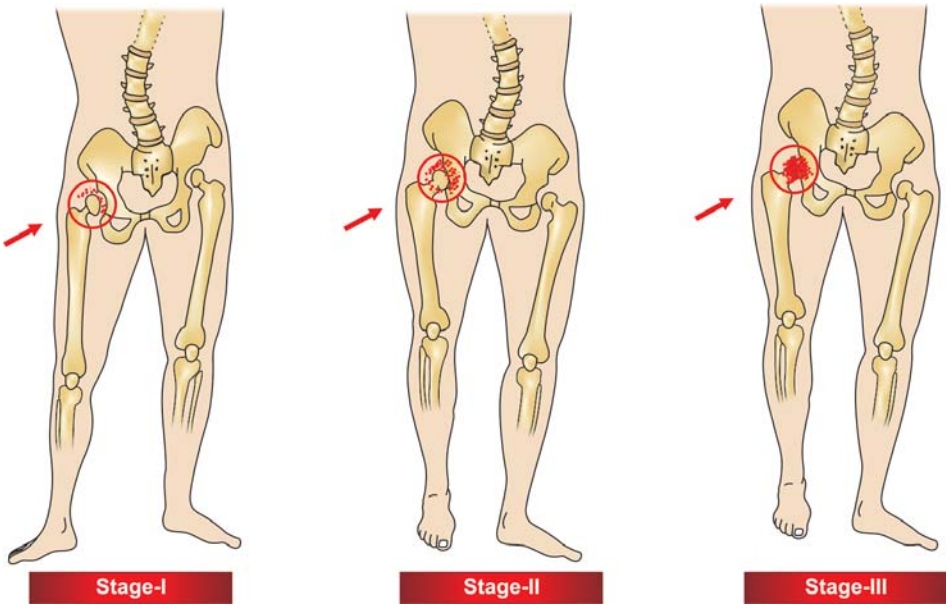


Figure 8.19

Showing the deformities in three stages of tuberculosis of the hip joint. Stage I: There is apparent lengthening because of abduction deformity; Stage II: There is apparent shortening because of adduction deformity; Stage III: True shortening develops because of destruction of the joint.

- Progressing painful limp and antalgic gait.
- Development of deformities.

Stages of the Disease (Fig. 8.19)

Stage I – Stage of synovitis (Apparent lengthening) In this stage the focus of infection (whether in the synovium or in the bone) irritates the joint. Hence, there is collection of fluid in the joint. In order to accommodate the fluid the joint adopts a position which increases its capacity. Hence, the limb is in flexion, abduction and external rotation.

Stage II – Stage of arthritis (Apparent shortening) In this stage the osteoarticular spread of infection has occurred. The joint destruction has begun and progressing. Thus, intensity of pain is severe and patient sleeps in a lateral position opposite to that of the involved hip. Hence, the deformity of flexion, adduction internal rotation develops. Night pain is common in this stage.

Night pain/Night cries: Patient gets up from sleep at night with severe and excruciating pain. Once he is awake the pain gradually decreases. This is known as night pain. It occurs because of the denuded articular sufaces (denuded of its articular cartilage) coming into contact with one another and the exposed subchondral bone rubbing against its counterpart. During daytime the protective muscle spasm prevents this contact. But at night in sleep when the muscles are relaxed the contact develops. Again when the patient gets up from sleep the protective muscle spasm keeps the denuded articular surfaces away and the pain decreases. This phenomenon is known as night pain.

Stage III – Stage of deformity (True shortening) In this the deformity of second stage becomes fixed. Destruction is pronounced and the joint goes into a state of fibrous ankylosis. The classical deformity is that of fixed flexion, adduction and internal rotation. But if the Y ligament of Bigelow is destroyed in the process of infection it is possible to get a deformity of flexion, abduction and external rotation.

Radiological Classification

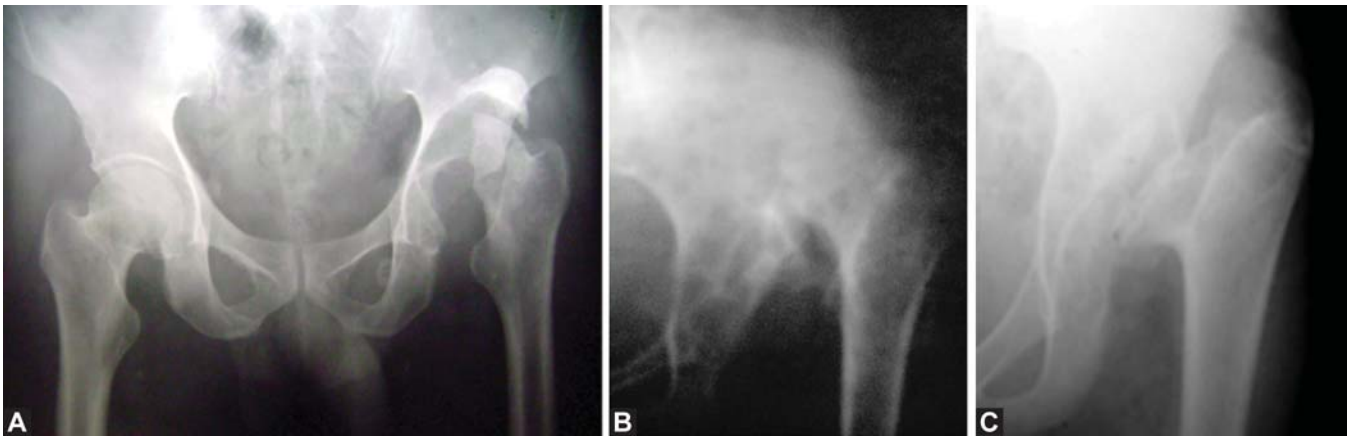
An excellent classification is suggested by Prof Shanmugasundaram, Chennai (1983), India (Figs 8.20A to C).

Normal hip type	Seen in children
Perthes' hip type	
Dislocated hip type	
Atrophic hip type	Seen in adults
Traveling acetabulum type	
Mortar and pestle type	Seen both in children
Protrusio acetabuli type	and adults

Functional outcome is different in different types.

Treatment

General Adequate ATT till the disease is cured is absolutely necessary. Immobilization in a splint or a plaster cast only for the required period is a must. This will help to restore the functional position of the joint and prevent unacceptable deformities. As soon as possible depending on the recovery patient has to be mobilized. This will avoid all the complications of unnecessary prolonged immobilization.



Figures 8.20A to C

Radiographs of different types of presentation based on Prof. Shanmugasundaram's description of tuberculosis of the hip joint: (A) Mortar and pestle type, (B) Traveling acetabulum type, (C) Atrophic type.

Surgical Procedures

Clearence procedure This procedure is indicated early in the disease when the response to medical line of treatment is poor because of the presence of large amount of debris. It aims at salvaging the joint by extensive debridement of the infected material and dead tissue thereby facilitating the action of drugs of ATT. At the same time the joint is immobilized in desirable functional position. So healing takes place in a position which restores maximum function.

Arthrodesis It is indicated in a case of unsound ankylosis of the hip, where stability is of prime concern. Functional position of arthrodesis is about 5-10° of flexion, about 10° of abduction and 0-5° of external rotation. Disadvantage is the inability to squat in future. Hence, all those activities which involve squatting have to be modified. The procedure gives a painless stable hip. Procedure should never be done in children.

Osteotomy This procedure is done in a case of bony ankylosis which has occurred in a functionally poor position. The osteotomy is done as close to the deformity as possible in the femur (just distal to the deformity) and the deformity is corrected.

Excision arthroplasty The procedure involves excising all the diseased components including the head of the femur in an attempt to provide a painless mobile hip. Postoperatively skeletal traction is employed for a period of 6-12 weeks for organization of a false joint. Exaggerated painless movement, instability and gross shortening are the disadvantages. This procedure is known as Girdlestone procedure. It was being done in the past in patient who preferred a painless mobile hip and did not mind shortening. This procedure is rarely done these days.

Joint replacement surgery Total joint replacement is the preferred option these days. At times, the acetabulum may have to be reconstructed. The procedure is well accepted by the patient and it gives a painless stable hip. Patient should be totally free of infection which is the prerequisite before surgery.

Prognosis Early presentation, diagnosis and adequate control of the disease give better prognosis than late presentation and delay in the treatment, i.e. Stage of synovitis has better prognosis than stage of arthritis and fixed deformities. Once the destruction occurs in the joint the end-result is fibrous ankylosis.

Tuberculosis of the Knee

Tuberculosis of the knee is the third common site after spine and hip.

Similar to any other bone and joint tuberculosis the dissemination occurs through the blood stream. The foci of infection get enlodged in the synovium or in the subchondral bone of the distal femur, proximal tibia or patella.

Unlike other joints since the knee joint has the largest joint space and has large amount of synovial covering, the disease remains as synovial for a considerable time before it becomes osteoarticular. Synovium studded with nodules are visible for the naked eye during surgery. Hence, it is thickened because of invasion by tuberculous granulation tissue. The synovial fluid which is serous initially becomes seropurulent and turbid later and may contain fibrinous flakes.

The synovial disease becomes osteoarticular by the formation of pannus which starts eroding the bone at the junction of the synovial membrane and the articular cartilage, the capsule and the ligaments. The pannus can extend over the articular cartilage as well as under the cartilage thereby separating it from the subchondral bone. This results in flakes of cartilaginous debris being present within the joint.

When the disease starts as osteoarticular the pathology is that of tubercular osteomyelitis.

Generally, the location of the lesion is epiphyseal in adults and metphyseal in children. The cold abscess thus formed destroys the bone and the articular cartilage, and finally, enters the joint.

Pathology

Early lesions in synovium and bone (Figs 8.21A to 8.22)

Tuberculous arthritis (Fig. 8.23)

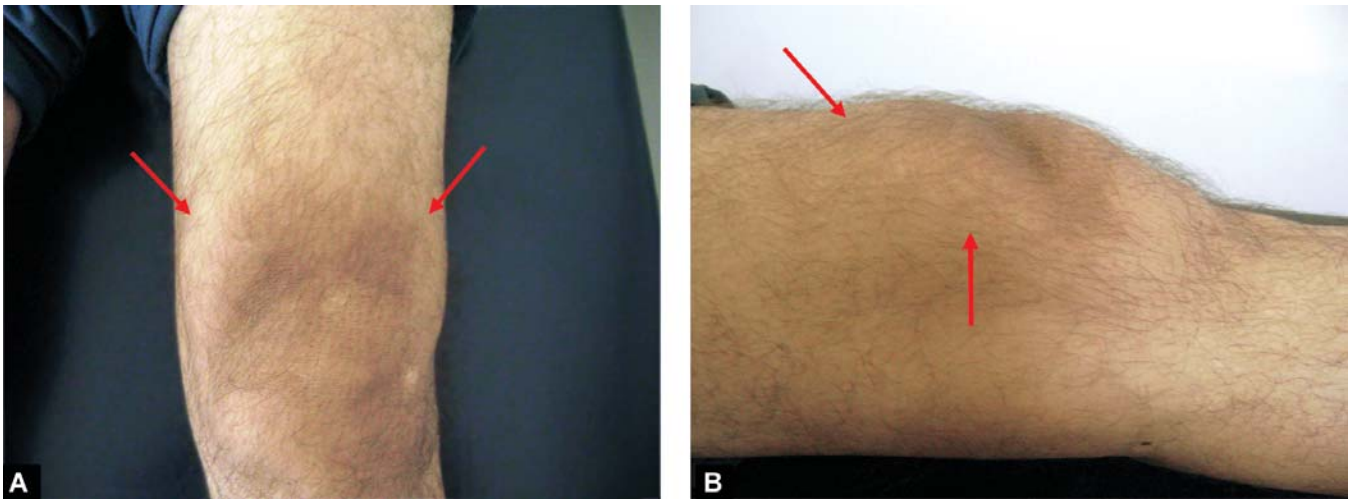
Fibrous Ankylosis (Fig. 8.24)

Triple deformity Whether synovial or osteoarticular total destruction of the joint along with its capsule and ligaments (the cruciates and the collaterals, which is a late feature) leads to classical triple deformity, i.e. flexion, posterior subluxation, lateral subluxation, rotation and abduction.

Initially flexion deformity develops because the knee adopts a position of flexion to accommodate the debris and the fluid. If it remains in flexion for a long time posterior subluxation develops. The powerful pull of the iliotibial band and the hamstrings causes rest of the displacements, i.e. lateral subluxation, rotation and abduction.

Clinical Signs and Symptoms

- Low grade fever with evening rise of temperature and night sweats.



Figures 8.21A and B

Showing synovitis with effusion. Note the horse-shoe shaped swelling in the suprapatellar pouch obliterating the normal hollows around the knee joint. This is one of the earliest signs seen in TB knee (arrows).

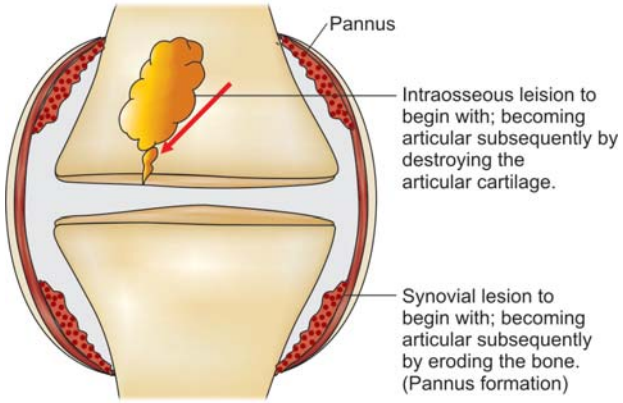


Figure 8.22

Diagram showing how an osseous/synovial lesion subsequently destroys the joint and causes tubercular arthritis. The end result is fibrous ankylosis. Only if secondary infection sets in the joint goes in for bony ankylosis.

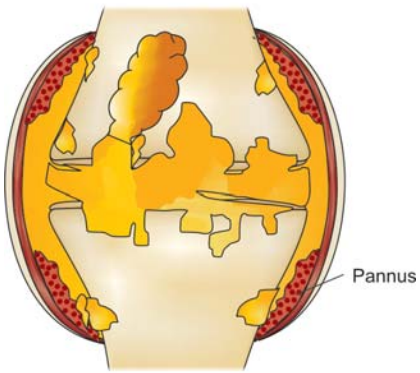


Figure 8.23

Diagram showing the destruction of articular cartilage and the involved bone. This results in loss of joint congruity.

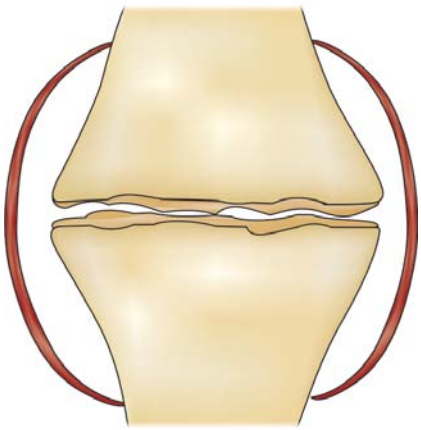


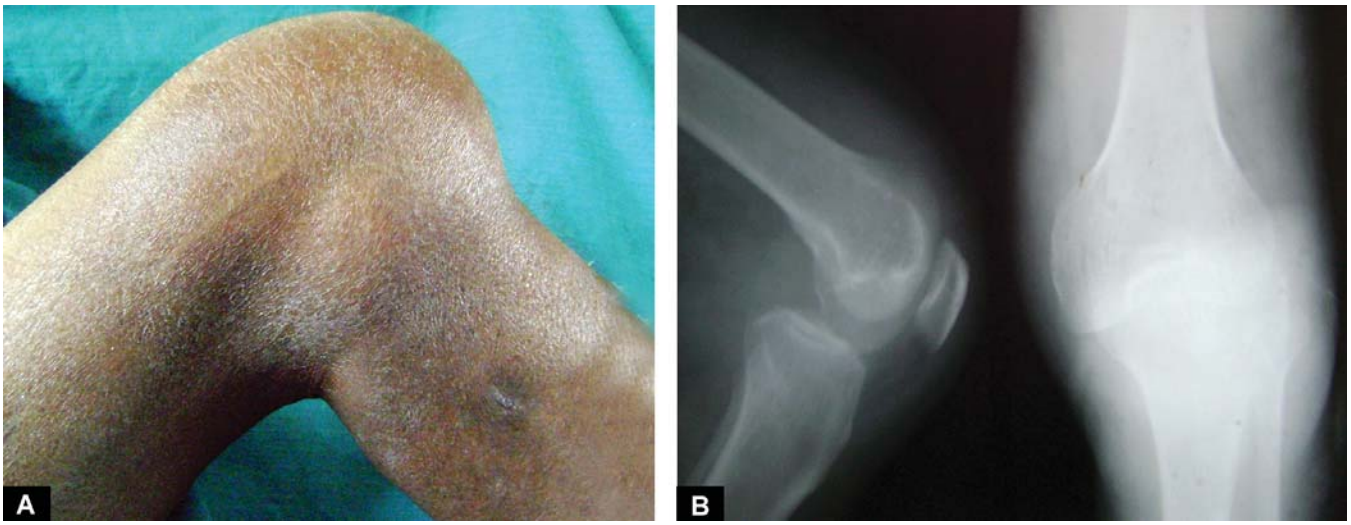
Figure 8.24

Diagram showing the end result. The joint space is narrowed. The area of denuded articular surfaces are opposed with a bridge of fibrous tissue.

- Loss of weight and appetite.
- Progressing painful limp and antalgic gait.
- Effusion and synovial thickening.
- Wasting of the muscles around the knee mainly the quadriceps.
- Night pains.
- Development of deformities (late feature).

Investigations

Blood and other investigations are similar to any other bone and joint tuberculosis and give positive indications of the disease.



Figures 8.25A and B

(A) Clinical picture and (B) Radiograph showing the classical triple displacement which developed secondary to pyoarthrosis. Note the classical displacements of flexion, lateral rotation and posterior subluxation.

X-ray—depending on the stage of the disease it shows from soft tissue prominence to narrowing joint space, destruction of the articular surface, osteolytic cavities, sequestrum and triple deformity (Figs 8.25A and B).

CT and MRI gives more useful information for treatment and planning surgery.

Treatment

Medical management is indicated only in very early cases which are diagnosed and confirmed by biopsy. (Arthroscopy and biopsy/trochar biopsy). In these cases the synovial involvement is not extensive. ATT and adequate immobilization result in cure. In children whose growth and remodeling capacity is very high always medical line of management is to be kept in mind before planning surgery.

Surgical Procedures

Synovectomy Done in cases with grossly thickened synovium. It aims at removing the diseased synovium and clearing the joint of tubercular debris. Though total synovectomy is aimed at, practically it always results in subtotal synovectomy. This is because some areas are inaccessible surgically.

Arthrodesis Done for a destroyed, deranged and deformed joint which is painful. The position of arthrodesis is 180° of extension. Charnley's compression arthrodesis using Charnley's compression clamps is one of the best methods for arthrodesis.

Total joint arthroplasty Is the best accepted procedure and to be considered in adults and elderly. It gives a painless mobile joint and gives considerable function when muscle atrophy is not very severe. Patient's disease free status should be confirmed before surgery to avoid flaring-up of the disease.

Tuberculosis of the Ankle and the Foot

Uncommon. Focus of infection may be synovial or bony, i.e. lower tibia and talus calcaneum are the other commonly involved bones. Infection can spread to the ankle joint from calcaneal tuberculosis after involving subtalar joint. Diagnosis and treatment follow the general principles as discussed before.

Tuberculosis of the Shoulder and Elbow

Synovial type of tuberculosis of the shoulder is very rare. So is the formation of cold abscess and draining sinus. The variety of tuberculosis of the shoulder is a dry one and is known as 'caries sicca'. It is commonly seen in adults and is considered as an atrophic variety of tuberculosis. Pain, limitation of movement and marked wasting of the muscles will draw attention. Radiologically destruction of the articular surfaces

and cavities are seen. Management follows the the general principles as discussed before.

The focus of infection in tuberculosis of the elbow is mostly bony and rarely synovial.

The disease is commonly seen in older children and adults. The signs and symptoms are similar to any other bone and joint tuberculosis. Diagnosis and treatment follow the general principles.

Tuberculosis of the Wrist

It is rare and commonly seen in adults. May start as a synovial or a bony focus. The common site is capitate and lower end of radius. Whether originates as synovial or bony, the dissemination to neighboring structures is rapid. The flexor and extensor tendon sheaths too get involved. The signs and symptoms are similar to any other bone and joint tuberculosis. Diagnosis and treatment follow the general principles.

Spina ventosa

Spina, means short bones, Ventosa means air. Thus the meaning is short bone filled with air. Also known as tuberculous dactylitis. Commonly seen in children and in short tubular bones, the metacarpals, metatarsals and phalanges. The blood supply is very rich and point of entry of the nutrient artery is in the center. Hence, the focus of infection is lodged in the center. Central destruction occurs initially. This gives the picture of bone filled with air. Later layers of subperiosteal new bone formation occurs. This cause a spindle shaped expasile lesion. Sinus formation can lead to secondary infection and the picture changes. Adequate ATT with antibiotics in case of secondary infection leads to complete resolution.

Revision Questions

Q. Discuss the onset and progress of lesions in tuberculosis of the hip.
Q. Write notes on Babcock's triangle.
Q. Discuss the types of deformities in tuberculosis of the hip joint.
Q. Discuss the different radiological types of tuberculosis of the hip joint.
Q. Discuss the management of the tuberculosis of the hip joint.
Q. What is the sequel of the tuberculosis of the hip joint?
Q. Discuss the pathology of tuberculosis of the knee joint.
Q. Write notes on:
   a. Spina ventosa
   b. Triple displacement
   c. Fibrous ankylosis
   d. Clinical features of tuberculosis of the knee joint.

Essay Questions

Q. Discuss the etiopathogenesis, clinical features, diagnosis, and management of tuberculosis of the hip including the sequel.
Q. What is triple displacement? Discuss the etiopathogenesis, diagnosis and mangement of tuberculosis of the knee.

Mycotic infections of the Bone

The mycotic infections of the bone are classified as superficial (common) and deep (very rare).

Superficial Mycoses

Involve skin and mucous membrane as a primary infection and then spread to adjacent soft tissue and bone, e.g. Maduramycosis, *Candida*, etc.

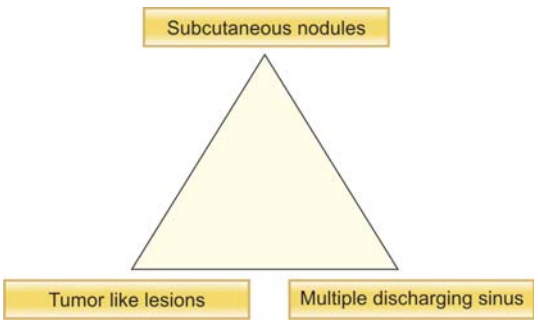
Deep Mycoses

Do not generally affect the bone except in immunocompromised patients, e.g. *Aspergillus, Cryptococcus, Blastomyces*, etc.

Maduramycosis

First identified in Madurai, India in the year 1842. Thus named as Madura foot.

Caused by *Madurella mycetomi*, and *Madurella grisea*. Agricultural workers and those who walk on bare foot are more susceptible. Causes nodular lesions once infection gets established in the tissues. Hence derives the name mycetoma.



Route of entry is through skin following a cut wound. Spreads through subcutaneous tissue the fascia and the tendon sheath and later gets established in the bone and joint. It evokes a granulomatous reaction in the tissue involved which breaks up causing multiple ulcerations and formation of multiple sinuses. The surrounding area is indurated and gives a woody feel.

Diagnosis is established by identification of the organism in the biopsy specimen and discharge.

Drug therapy with trimethoprim-sulphamethoxozole, ketoconozole, dapsone, etc. are effective only in early cases. Severe forms of involvement necessitate amputation.

Syphilitic Infection of Bone

Seen in infants as well as in adults as tertiary lesion. In infants it manifests after several weeks of birth though infestation has occurred during pregnancy in the womb. *Trepenoma pallidum* crosses the placental barrier in the later months of pregnancy and infects the fetus. An irritable infant with symmetrical skeletal swelling in the long bones, painful and refuses to move the limb should raise the suspicion. The pathology is either syphilitic periostitis or metaphysitis. Periostitis manifests radiologically in formation of new bone either diffuse or in layers. Metaphysitis shows trabeculer erosion and frank destruction.

In older children and adults, gummatous lesions develop in the bone. Radiologically seen as lytic punched out areas in the medulla surrounded by thick sclerotic bone. In tibia the syphilitic osteitis causes diffuse thickening of the cortex and bending/bowing of the tibia which is classical. The resultant deformity is known as 'Sabre tibia'.

Antibiotic therapy in tertiary syphilis is not very effective.

Parasitic Infestation of Bone

Hydatid Cysts in the Bone

The dog tape worm *Echinococcus granulosus* (5 mm long with 3 segments) is responsible for hydatid cysts in the bone. Dog is the definitive host and the tape worm harbors in the bowel. The scolices are excreted in the feces. Man, sheep and cattle are intermediate hosts and get infested by ingesting food contaminated with dog feces. Infested meet when consumed by dog continues the life cycle and new generation of tape worms evolve.

The scolices when ingested, hatch into larvae and through the portal system enter the blood stream. And through the blood stream may get enlodged in the bone leading to hydatid cysts. The common bones affected are the pelvis, vertebrae, ribs and femur. The cyst goes on destroying the bone. The growth plate does not form a barrier for destruction and the destruction may extend even to the epiphysis.

Radiologically solitary or multiloculated cystic lesions are seen with extensive destruction of cortex and medulla. In case of vertebral involvement patient may present with neurological signs. CT and MRI help in diagnosing by identifying para-skeletal cysts. Needle biopsy and Casoni's complement fixation test are confirmatory.

Albendazole is the drug of choice and is to be given at four weekly intervals at least for 6-8 weeks. Protection may be necessary to avoid pathological fracture. Once the control is established the bone cysts may need excision and bone grafting. Irrigating the cavity with hypertonic saline helps in preventing recurrence.

Revision Questions

Write notes on

a. Mycotic infections of the bone
b. Hydatid bone disease
c. Sabre tibia.

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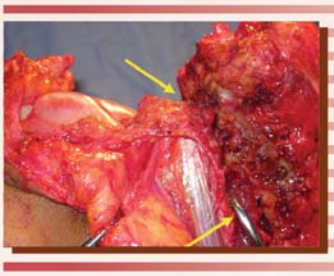


9 Bone Tumors

- *Evaluation of Bone Tumors in General with Relevant Investigations*
- *Classification*
- *Common Benign Bone Tumors, their Clinical Features, Diagnosis and Treatment*
- *Common Malignant Bone Tumors, their Clinical Features, Diagnosis and Treatment*

Bone tumors basically belong either to benign or to malignant nature and can arise from any tissue present in the bone. [Namely bone and cartilage progenitor cells (most common), periosteal cells, hemopoietic cells, lipocytes, nerve and schwann cells, fibroblasts, endothelial cells, perithelial cells, epithelial cells, smooth muscle cells, notocordal cells, histiocytic cells, etc.]. They have a specific predilection to age, specific bones and in the bones to specific sites. The nature of the lesion is either to form neoplastic new bone or to destroy the normal bone, i.e. either tumor osteogenesis or tumor osteolysis (Figs 9.1A to C). Tumor osteolysis is generally seen when the bone tumor arises from elements other than bone and cartilage progenitor cells. Predilection to sex is also seen and generally males are more susceptible when compared to females. All these points are to be kept in mind during evaluation of a bone tumor.

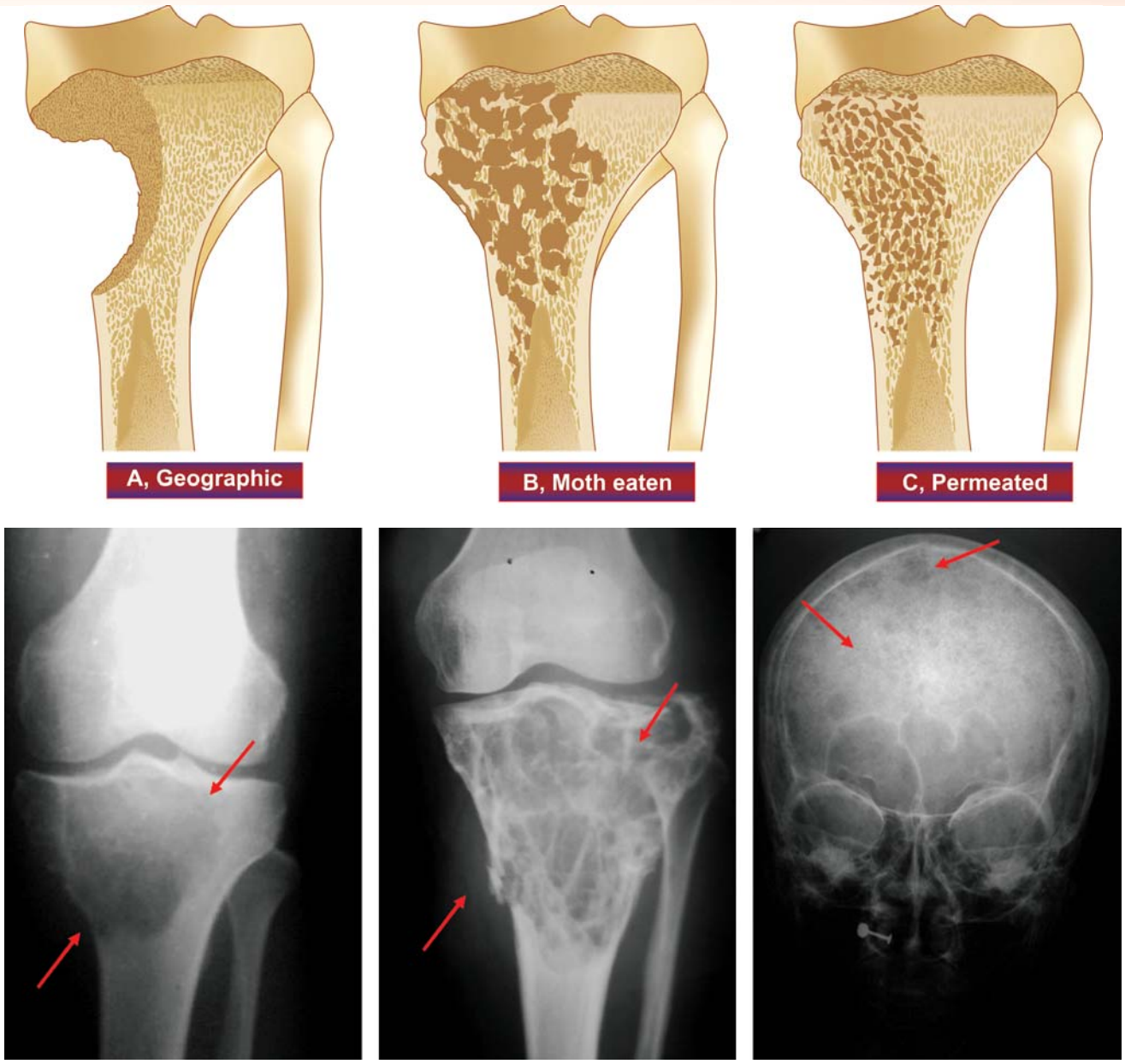
Evaluation of a Bone Tumor

Clinical

Apart from age, sex, location and the bone from which the tumor arises following are to be considered for primary evaluation.

Onset: Benign tumors onset is insidious in benign tumors. The first sign is either development of a well demarcated swelling with or without accompanying tenderness, i.e. neoplastic new bone forming variety. It can also be an accidental discovery with no symptoms. At times presentation is a pathological fracture with pain, i.e. osteolytic variety. Further constitutional symptoms are uncommon in benign tumors unless secondary complications develop.

Malignant tumors: In malignant tumors pain always precedes other symptoms like swelling and most of the time associated



Figures 9.1A to C

Showing diagrammatic representation and radiographs of the types of destructive lesions based on description by Lodwick. (A) Geographic (B) Moth-eaten (C) Permeative.

with constitutional symptoms like malaise, low grade fever, loss of appetite, weight, etc. Tumor related pain is constant and not relieved by rest.

Plane and consistency of the swelling: Plane of the swelling is always deep to the muscle and arising from the bone. The swelling is smooth and uniform in benign lesions, whereas in malignant lesions it is irregular and consistency is variegated. Consistency varies from soft, firm and hard depending on the predominance of the tissue involved as well as the amount of tissue destruction/formation which has resulted on account of the tumor.

Rate of growth: Rapid in malignant bone tumor. Slow in benign bone tumor.

A General Classification of Bone Tumors (Depending on Tissue of Origin)

Nature	*Benign*	*Malignant*
A Angiod tumors	Angioma, ABC Glomus tumor	Angiosarcoma
B Bone forming tumors	Osteoma Osteoid osteoma Osteoblastoma Chondroma	Osteosarcoma
C Cartilage forming tumor	Osteochondroma Chondroblastoma	Chondrosarcoma
D Dental and allied structures	Odontogenic cyst Amelloblastoma	Malignant odontoma
E Embryogenic vestigeal tissue		Chordoma
F Fibroblastic tissue	Fibroma	Fibrosarcoma
H Heterotropic tissue	Dermoid	Adamantioma of long bones
N Non osseous connective tissue	Lipoma Neurofibroma Neurilemmoma	Liposarcoma Myeloma Leukemia, Ewings
S Synovial tissue	Synovioma	Synovial sarcoma
U Undifferentiated	Giant cell tumor	Malignant Osteoclastoma

Extension to surrounding soft tissue: Present in malignant bone tumor. Absent in benign bone tumor.

Regional lymph node: Lymph nodes enlarged and firm/hard in consistency in malignant tumors suggesting lymphatic spread. Lymph nodes are not enlarged in benign tumors.

Radiological

A radiograph picture is viewed as follows:

a. Age of the patient: Certain tumors are common in certain age groups, e.g. Ewings tumor 5-15 years, Osteosarcoma 15-25 yrs, Giant cell tumor 20-30 yrs, etc. Hence attempt should be made to ascertain the age of the patient radiologically.
b. Bone involved: Certain tumors are common in long bones and in appendicular skeleton, while others are common in flat bones and axial skeleton, e.g. osteosarcoma is commonly seen in long bones, multiple myeloma is commonly seen in flat bones and the vertebra.
c. Site in the bone

Long Bone
- Epiphysis—Giant cell tumor.
- Metaphysis—Osteosarcoma.
- Diaphysis—Ewing's tumor.

Vertebra
- Body—Primary tumor.
- Appendages—Secondary deposits.

d. Nature of the lesion
Osteogenic—Osteoid Osteoma, Osteosarcoma
Osteolytic—Osteoblastoma, Multiple Myeloma
e. Specific characteristics
Soap bubble appearance—Giant cell tumor.
Onion skin appearance—Ewing's sarcoma.
Speckled calcification—Chondrosarcoma.
Punched out appearance—Multiple Myeloma.

Bone scan: Bone scan using Technetium 99 m diphosphonate (99 m Tc- HDP) is of immense help as it clearly shows the hot spots in the skeleton. Even the smallest of the tumors can be diagnosed and so are the skipped metastasis.

CT scan: CT scan is not a substitute for radiographs or bone scan but it is an adjuvant to these investigations, when there is a need for clear delineation of tissues. It is useful to assess the intra and extra osseous extension. Also helps in early detection of tumors before radiological changes are seen..

MRI: MRI delineates the soft tissues better than CT and useful in visualizing the soft tissue extension and invasion of the tumor into the neighboring structures, e.g. muscles, vessels and nerves. This helps in staging of the tumor and planning of the surgery.

Laboratory Investigations

Hb%, TC, DC, ESR and serum alkaline phosphatase, serum calcium, serum phosphorus are nonspecific investigations. Mainly help to differentiate tumors from other bone pathologies.

Serum electrophoresis, Bence Jones proteins, help to diagnose Multiple Myeloma.

Serum acid phosphatase help to diagnose Prostatic Carcinoma. These days PSA essay is more widely used.

Biopsy: Most useful investigation to establish the diagnosis and grade the tumor. Biopsy is done by the help of a bone biopsy needle or sometimes an open biopsy.

Management

Total eradication of the lesion is the treatment for bone tumors. In benign tumors this is possible by surgical excision and reconstruction (when necessary). In malignant tumors chemotherapy and radiotherapy is necessary in addition to surgical excision and reconstruction. This is to eradicate the tumor from the system and prevent metastasis. Prognosis in those malignant tumors which are radioresistant and chemoresistant is grave. And so is the prognosis when the patient presents late in the course of the disease.

Different applications of chemotherapy

a. Adjuvant chemotherapy: Is the therapy which follows after primary treatment of malignant tumor. It aims at taking care of the risk of recurrence and spread of the tumor.
b. Neo-adjuvant chemotherapy: Is the chemotherapy which is administered prior to ablative procedure. It aims at shrinking the size of the tumor and preventing the spread of tumor during the procedure as well as in the immediate postoperative period.
c. Radiosensitizing chemotherapy: It is the chemotherapy given in conjunction with radiotherapy. It aims at sensitizing the malignant cells to the effect of radiation and facilitate destruction.
d. Curative chemotherapy: It is the therapy which aims at curing the malignancy.
e. Palliative chemotherapy: It aims at providing some comfort to the patient by lessening the symptoms of malignancy.

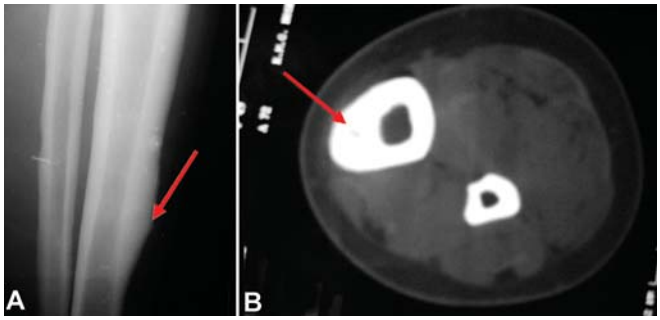
Radiotherapy: It is generally given by using linear accelerator (Ref: Intensity Modulated Radiation Therapy). It aims at killing the cancer cells using the safe limit of radiation. The damage to the normal tissue is kept to a minimum.

Common Benign Bone Tumors

Osteoid Osteoma (Figs 9.2A and B)

This is a tumor consisting of osteoid and woven bone characterized by a central nidus of osteoid surrounded by an exaggerated zone of sclerosis.

- Age—Can occur at any age. Common between 10-30 yrs. Rare below 5 yrs and above 45 yrs.
- Sex—M:F = 2:1.
- Site—It is common in long bones and in the appendicular skeleton. Rare in flat bones and axial skeleton. Ratio is about 2/3 to 1/3.
- Size—Approximately 1.5 cm (0.5-2.0).



Figures 9.2A and B

Radiograph (A) and CT scan (B) showing the nidus of osteoid surrounded by an exaggerated zone of sclerosis.

- Types
  - — Cortical variety is most common.
  - — Cancellous variety less common.
  - — Subperiosteal variety rare.

Clinical Symptoms

Classical focal bone pain which is worse at night and relieved by small dose of aspirin (this forms the basis of a clinical test). Focal tenderness may be present. Absence of constitutional symptoms helps to rule out malignancy.

Investigations

a. Radiograph: Shows a central round or ovoid nidus surrounded by an exaggerated zone of sclerosis (Fig. 9.2A).
b. Bone scan using Technetium 99 m diphosphonate. Most sensitive and may show positive results even before radiograph shows signs of the tumor. Also helps in complete removal of the tumor with the nidus with the help of a hand held and operated radioactive detector.
c. CT scan is useful to localize the lesion precisely. Also useful in guiding the ablation probe when percutaneous ablation technique is chosen (Fig. 9.2B).
d. SPECT: Single Photon Emission Computed Tomography is useful in detecting lesions involving spinal arch and spinous process.

Treatment

Total excision of the tumor with nidus gives permanent relief of symptoms.

Cause for pain in Osteoid Osteoma is said to be due to presence of axons in the lesion as demonstrated by neural staining techniques as well as marked elevation of levels of Prostaglandin E2.

Benign Osteoblastoma

This tumor shows features almost similar to that of Osteoid Osteoma with formation of osteoid and primitive woven bone in a fibrovascular stroma but in a much bigger manner. Hence the term Giant Osteoid Osteoma sometimes is used to describe this tumor.

The neural elements namely the axons are absent. Hence pain is not as disabling as in Osteoid Osteoma.

Unlike Osteoid Osteoma which have limited growth potential, these tumors have a huge growth potential and attain a big size.

- Age: Can occur at any age. It is common in younger age group, between 20-30 yrs.
- Sex: M:F ratio is 3:1.
- Site: Spine. Commonest site being sacrum followed by diaphysis of long bones. Common in lower extremities. Rare in other flat bones.

Clinical Features

Pain is constant. Not as intense as in Osteoid Osteoma but long standing and not relieved by salicylates. Painful Scoliosis and neurological deficits may develop because of mechanical interference with spinal cord and nerve roots.

Investigations

Investigations are similar as in Osteoid Osteoma but the diagnosis is best confirmed by biopsy and histopathological study.

Treatment

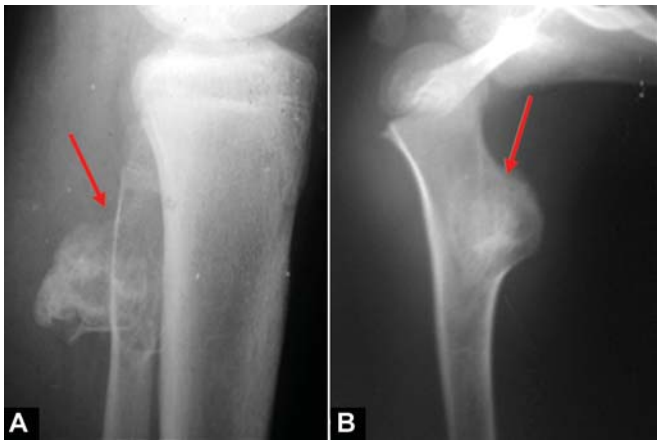
Total excision and bone grafting is the treatment of choice. Incomplete excision will result in recurrence and the rate is as high 20%.

There is a variant of Osteoblastoma known as aggressive Osteoblastoma which behaves like an Osteosarcoma. Metastasis and recurrence after excision is very common.

Osteochondroma

This tumor has both osseous and cartilaginous component and hence derives the name Osteochondroma. The cartilaginous portion is generally radioluscent unless it is calcified.

- Age: Common in young adolescent age between 10-20 yrs. May be present at birth and develop in early childhood.
- Sex: M:F ratio 3:1.



Figures 9.3A and B

Radiograph showing (A) Pedunculated and (B) Sessile osteochondroma respectively. Note the age. The presence of growth plate indicates that the patients are adolescents.

- Site: Commonly seen at the epiphysiometaphyseal region (physis), growing away from the physis. Around the knee is the commonest site. Next common site is around the shoulder.

Types

a. Pedunculated—One with a stalk. Commonly seen in long bones and in appendicular skeleton. These are less prone for malignant transformation (Figs 9.3A and 9.4).
b. Sessile—One without stalk. Commonly seen in flat bones and in axial skeleton. These are more prone for malignant transformation (Fig. 9.3B).

Pathogenesis

a. Theory of herniation of the growth plate: Trauma or a deficiency of the perichondrial ring may cause herniation of the physis. This herniated physis starts growing like a physis, producing bone in a manner similar to physis and showing zones of different stages of bone formation, starting from resting cartilage cell layer to proliferating, hypertrophic and maturing layer.
b. Theory of genetic abnormality: This theory is yet to be conclusively proved.

Clinical Features

The tumor is accidentally detected as an abnormal swelling, which draws attention of an adolescent who is more conscious about cosmesis.

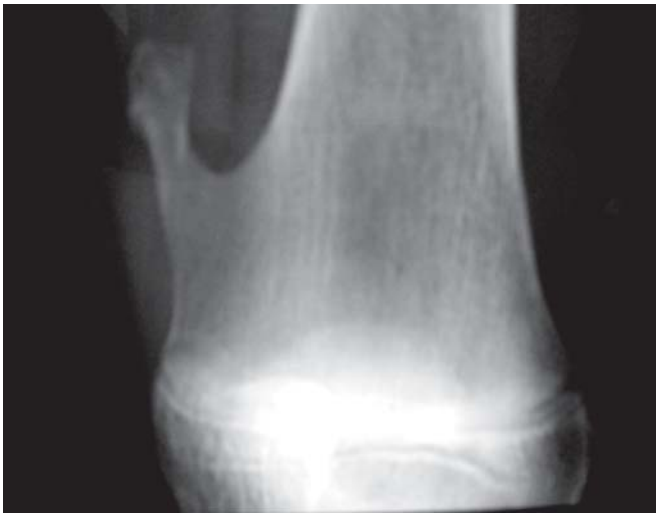


Figure 9.4

Radiograph showing classical features of pedunculated Osteochondroma. Note the osseous stalk with a bulbous expansionat the end, growing away from the epiphysis.

When neglected can cause pressure symptoms on neighboring structures namely the muscles, vessels and nerves. It can also cause mechanical obstruction of joint movements. An adventitious bursa can develop with recurrent attacks of bursitis. Trauma can cause pathological fracture of the stalk with profuse bleeding. Long standing osteochondromas can transform to chondrosarcomas and present with pain and increase in size of the tumor.

Investigations

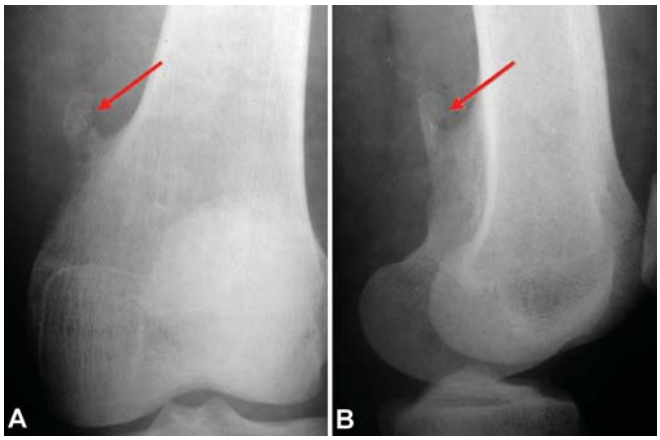
Radiograph shows the classical features of osteochondroma. It shows a pedunculated or sessile tumor arising from the epiphysiometaphyseal region and growing away. The cartilaginous cap is not seen unless it is calcified.

Treatment

Total excision of the tumor along with a good amount of normal bone around the osseous stalk. Recurrence is uncommon after total excision. Ideal period for excision is after skeletal maturity and not before for the fear of recurrence.

Indication for excision

a. Cosmetic reasons.
b. Recurrent attacks of bursitis.
c. Pressure symptoms.
d. Mechanical obstruction.
e. Fracture of the stalk (Figs 9.5A and B).
f. Malignant transformation.



Figures 9.5A and B

Showing a **rare complication of fracture of the stalk** of the Osteochondroma. The incidence occurred during a soccer match when the ball hit the tumor. Till then patient did not have any symptoms before the injury.

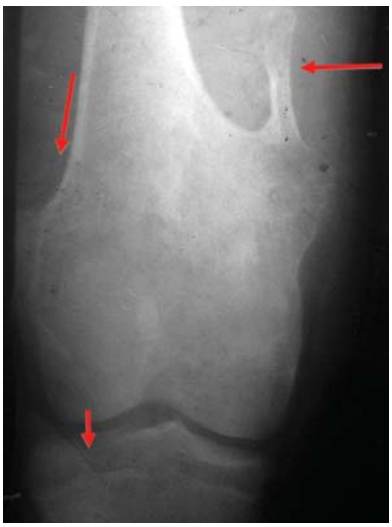


Figure 9.6

Radiograph showing Metaphyseal Aclasis. Note the presence of multiple Osteochondromas arising from the metaphysis of femur and tibia.

Osteochondromatosis is also known as Heriditary Multiple Exostosis, Metaphyseal or Diaphyseal Aclasis (meaning of the word Aclasis is, a pathological outgrowth). First identified by John Hunter (1786) is an autosomal dominant disorder running in families. M:F ratio 1:1 and detected as early as 3-4 yrs of age. Multiple bones are affected and results in growth abnormalities and deformities. Malignant transformation is also common when compared to Solitary Osteochondroma (Fig. 9.6).

Dysplasia Epihysialis Hemimelica, also known as Trevor's disease is characterized by osteochondromas occurring in one limb and on one side of the epiphysis. Medial side is twice commonly affected than the lateral side.

Chondroblastoma

This tumor was first identified by Codman in the year 1931 who termed it as Chondroblastic variant of Giant cell tumor. Henry Jaffe and Louis Lichtenstein a decade later gave the name 'Benign Chondroblastoma' signifying the chondroblastic origin of this tumor. It accounts for about 1% of all bone tumors. Though considered as a benign local erosion, distant metastasis to lungs is seen at times.

- Age: 10-30 yrs.
- Sex: M:F ratio 3:1.
- Site: Epiphysis of a long bone. Common site is around the knee. Next common site is proximal femur and proximal humerus. Rare in pelvis, talus, calcaneum and patella.

Clinical Features

Slow growing tumor. It may remain asymptomatic for quite a long period. Presents with swelling and pain. Joint effusion may be present. It may also present with a pathological fracture.

Histopathology

The tumor consists of primitive chondroblasts in a dense eosinophyllic matrix. Coarse calcification may be seen (Chicken-wire calcification). Atypism and mitotic figures are not seen unless malignant transformation has occurred.

Investigations

- Radiograph: Shows a lytic area in the epiphyseal region extending into the metaphysis. Speckled calcification may be seen.
- CT: Delineates the tumor better and shows calcified areas.
- MRI: Helps in identifying transepiphyseal and transcortical extension of the tumor.

Treatment

Total excision of the tumor with bone grafting to fill the defect. Incomplete excision may lead to recurrence of the tumor. Recurrence rate is high 10-35%.

Chondromyxoid Fibroma

This is a tumor comprising of chondroid, myxoid and fibrous tissue and hence derives the name. It was first described by Jaffe and Lichtenstein in 1948. It accounts for about 1% of the tumors.

- Age: 15-30 yrs.
- Sex: M:F ratio 1:1 or 2:1 (a little more in men).
- Site: In the metaphysis of a long bone extending on to epiphysis and diaphysis. Common site is upper end of tibia followed by lower end of femur upper end of humerus.

Clinical Features

Many a times it is discovered accidentally. Not diagnosed till tumor grows to a considerable size or causes pain. Limitation of joint movement is not seen.

Histopathology

The tumor is supposed to arise from cartilage forming connective tissue of the marrow. Comprises of chondroid, myxoid and fibrous tissue. No atypism is seen. Malignant transformation is rare and when occurs it is difficult to distinguish from Primary Chondrosarcoma.

Investigations

Radiograph: Shows an expansile, eccentric, lytic, metaphyseal lesion with a regular or at times irregular sclerotic margin.

CT and MRI help in better evaluation and planning surgery.

Treatment

Total excision and bone grafting is the treatment of choice.

Enchondroma

It is a benign, solitary, intramedullary, cartilaginous neoplasm.

- Age: 20-40 yrs.
- Sex: M:F ratio 1:1.
- Site: Common in small bones of the hand and feet. Next common site is long bones and pelvis. The location is diaphyseal in short tubular bones and metaphyseal in long tubular bones.

Clinical Features

Early: Swelling is the only feature which draws attention.

Late: Swelling associated with pain due to
a. Pathological fracture.
b. Malignant transformation. This tumor is prone for pathological fracture.

Histopathology

The cartilaginous cell rests, get displaced during growth and rest elsewhere, and start multiplying. Thus, the normal bone is replaced by mineralized or nonmineralized hyaline cartilage. Presents under microscope as lobules of cartilage containing chondrocytes surrounded by a narrow rim of bone. Calcification may or may not be present.

Investigations

- Radiograph: Shows a solitary lytic lesion in the bone involved.
- CT scan: Helps to diagnose subtle calcification.
- MRI: Helps to differentiate bone infarct from enchondroma.

Treatment

Excision of the tumor and bone grafting (care should be taken to prevent spilling of tumor at the donor site of bone graft because the tumor can develop by seedling).

Chondrosarcoma is a likely complication of Enchondroma. Hence, it is important to have periodic follow up when surgery is deferred.

Syndromes associated with Enchondromas are
a. Ollier disease is nonhereditary and presents with multiple Enchondromas.
b. Mafucci's Syndrome is nonhereditary and presents with multiple Enchondromas and multiple Hemangiomas.
c Metachondromatosis is a hereditary disorder, autosomal dominant and presents with multiple Enchondromas and Osteochondromas.

Aneurysmal Bone Cyst (ABC)

This is an expansile destructive (osteolytic) lesion consisting of blood filled spaces separated by trabeculae of osteoid or septae with interspersed osteoclasts. It was first described by Jaffe and Lichtenstein in the year 1942. It accounts for about 6% of bone tumors.

- Age: 10-20 yrs.
- Sex: M:F ratio 1:1.5. Incidence is slightly more in females.
- Site: In the metaphysis of long bones. Sometimes occurs subperiosteally. Rare in the spine and flat bones.

Etiology

The etiology is unknown and thought to be multifactorial.
a. De novo presentation because of AV malformation and function.
b. Degeneration and destruction occurring secondary to trauma and formation of hematoma.
c. Developing in already existing tumor (secondary aneurismal bone cyst), e.g. chondroblastoma, osteosarcoma, etc.

Clinical Features

Pain, swelling and at times a pathological fracture. Considerable time might have lapsed before the development of symptoms developed. Involvement of the spine may cause neurologic symptoms.

Histopathology

Thin walled blood filled cavities along with trabeculae of Osteoid or septae with interspersed osteoclasts.

Four distinct phases of the behavior are recognized.
a. Osteolytic phase.
b. Active phase of rapid destruction.
c. Maturing phase/phase of stabilization.
d. Healing phase.

The osteolytic phase presents as a focal area of osteolysis. The active phase presents with occurrence of rapid destruction, thinning of the cortex, bulging of the periosteum (periosteal blow out).

The maturing phase presents with formation of distinct peripheral bony shell and internal septae. This gives rise to soap bubble appearance.

The healing phase presents with irregular new bone formation as a result of progressive calcification and ossification.

Investigations

Radiograph, CT and MRI. The pictures vary depending upon the phase of the cyst.

Classical cases may show 'Finger in the balloon' sign radiologically.

Treatment

a. Intralesional injections like Phenol, liquid nitrogen, etc. have not gained popularity. Their results are uncertain and there is a great risk of injury to neighboring structures like vessels and nerves.

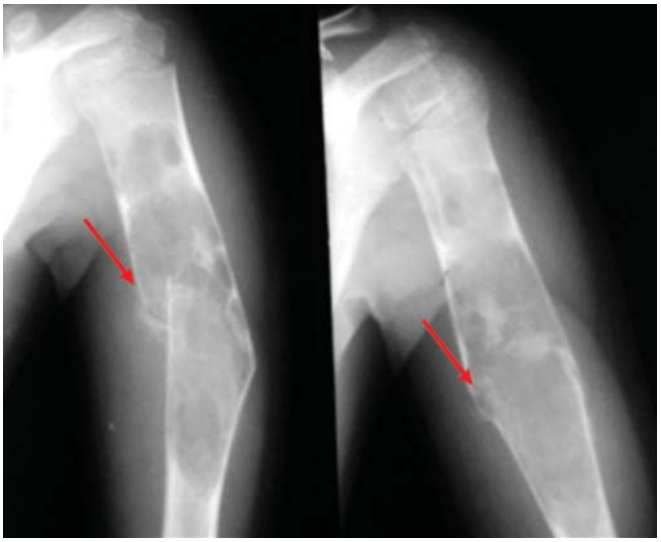


Figure 9.7

Radiograph showing a huge unicameral bone cyst with classical features of pathological fracture, localations and extension into the diaphysis.

b. Arterial embolization has shown successful results in selected cases (when there is extensive subchondral involvement which may compromise joint function after extensive surgical excision or in areas which are surgically inaccessible for a complete excision).
c. Surgical excision and bone grafting is the treatment of choice whenever feasible.

Unicameral Bone Cyst (UBC)

It is a unique benign cystic lesion filled with fluid seen in children. Virchow recognized this lesion as early as 1870. In 1942 Henry Jaffe and Louis Lichtenstein published their article about unicameral bone cyst (Fig. 9.7).

- Age: 5-15 yrs.
- Sex: M:F ratio 2:1.
- Site: Proximal humerus followed by proximal femur (account for about 90%).

Etiology

Several theories have been put forth.

a. Blockage of drainage of interstitial fluid in rapidly growing and rapidly remodeling cancellous bone—Cohen 1960.
b. Congenital cell rest of synovial tissue as proved by presence of macrophage like Synovial type A and fibroblast like type B cell in the lining of the cyst—suggested by Joseph M. Mira.

Clinical Features

Always detected accidentally except in cases with pathological fracture when patient presents with pain. When adjacent to the growth plates it is considered as an active cyst and when at a distance it is considered as a latent cyst.

Investigation

Radiograph: Shows an osteolytic lesion in the metaphysis extending to the diaphysis. At times may show loculations. Evidence of pathological fracture may be present.

Treatment

As spontaneous resolution is seen, asymptomatic cysts when detected accidentally are left alone with periodic follow up till adulthood. Treatment is necessary only if no resorption is observed. Pathological fractures are treated nonoperatively by immobilization. Healing invariably occurs.

Percutaneous techniques

a. Steroid in the form of methyl prednisolone is injected into the cyst. Results have been satisfactory.
b. Bone marrow injection with about 25 ml of autologous marrow aspirated form the iliac crest and injected immediately into the site. Resolution has been observed. This procedure is yet to be accepted as a universal procedure.

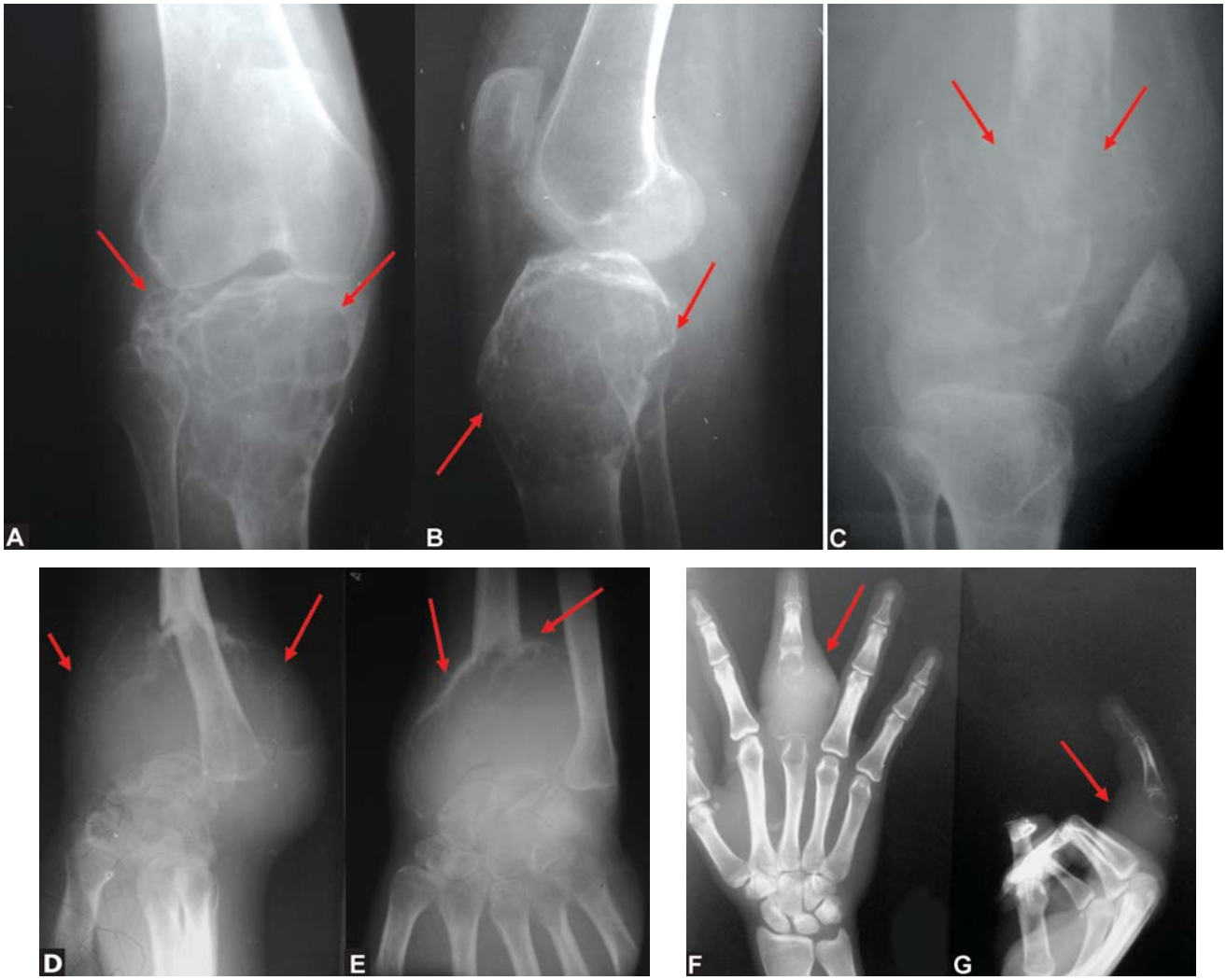
Operative procedures: Total excision and bone grafting done only selectively. It is a highly morbid procedure for a young individual. Hence to be viewed critically.

Common Malignant Bone Tumors

Giant Cell Tumor (GCT) (Figs 9.8A to G)

It is considered as a locally malignant tumor of the bone because local recurrence is more common (around 50%) when compared to distant metastasis (occurs only in 5-10% of the cases). It is said to have been first reported by Cooper in 18th century. It accounts for about 5% of bone tumors.

- Age: 20-40 yrs.
- Site: Epiphyseal end of long bones. Upper end of tibia, lower end of humerus, lower end of radius, proximal humerus. Rare in the vertebra and other spongy bones like calcaneus and pelvis.
- Sex: A little higher predilection to female sex is seen.



Figures 9.8A to G

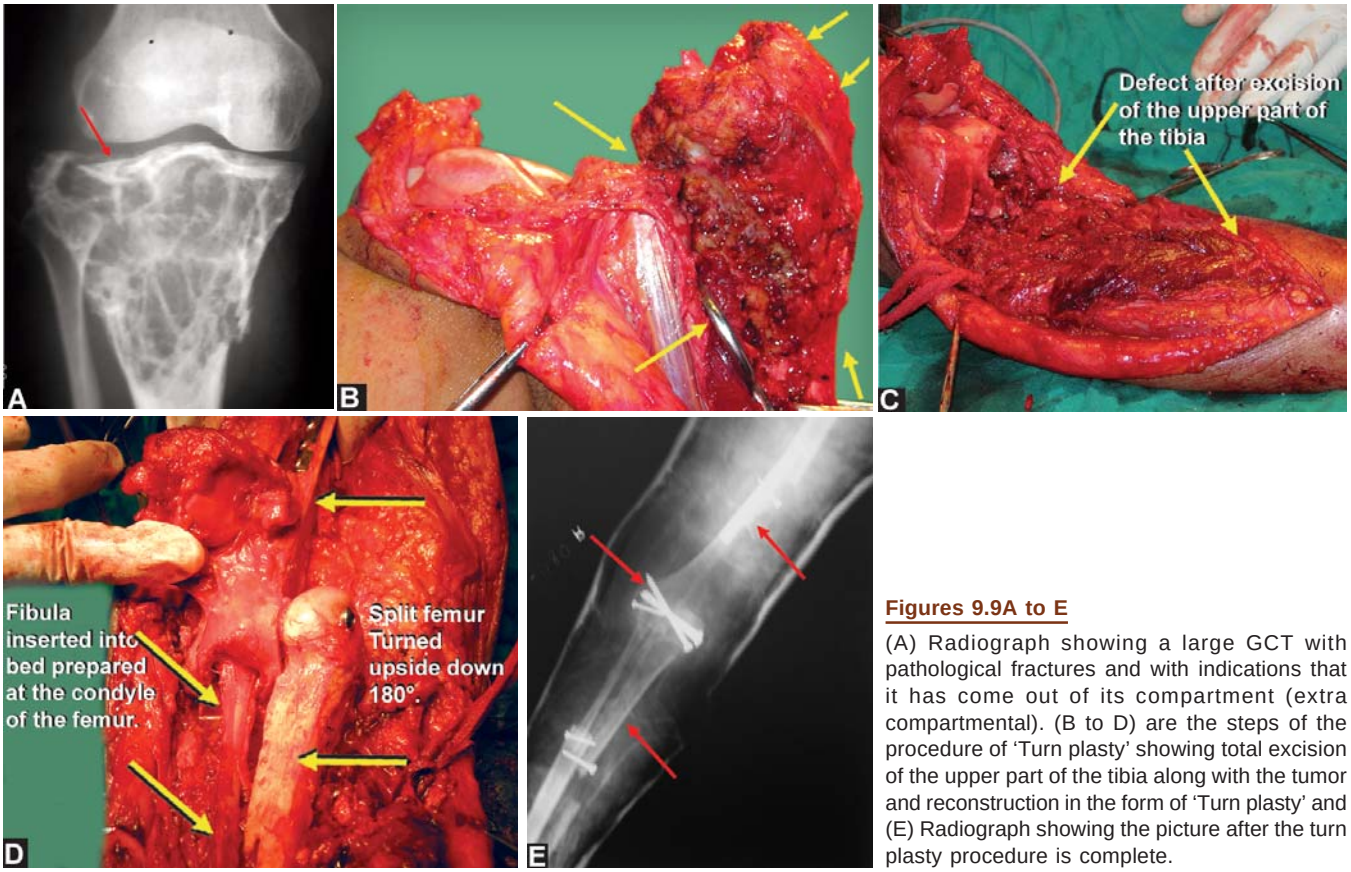
Showing GCT arising from various bones. Note the Epiphyseal, Expansile, Eccentirc nature of the lesion which is lytic. Soap bubble appearance is present in tibial GCT. All the cases seen here are advanced and late presentation and are associated with pathological fractures.

Clinical Features

Presents as a slow growing, painful swelling arising from the epiphyseal end of a long bone. Egg shell crackling may be present on palpation when thinning of the cortex and pathological fracture has occurred. Limitation of joint movement is a late feature.

Histopathology

A picture consisting of spindle shaped and rounded mononuclear cells with osteoclastic type multinuclear giant cells and blood vessels is seen under microscope. The grading in relation to aggressiveness is done by Jaffe (1940) into 3 grades and by Netherlands committee on bone tumors into 4 grades. While grading, atypism shown by the stromal spindle shaped cells alone is taken into consideration and not features shown by multinucleated giant cells. The more the atypism, i.e. anisocytosis, anisonucleosis, hyperchromatinism, etc. the more aggressive (malignant) is the tumor. The number and size of the multinucleated giant cells proportionately gets reduced as the atypism of stromal cell increases.



Figures 9.9A to E

(A) Radiograph showing a large GCT with pathological fractures and with indications that it has come out of its compartment (extra compartmental). (B to D) are the steps of the procedure of 'Turn plasty' showing total excision of the upper part of the tibia along with the tumor and reconstruction in the form of 'Turn plasty' and (E) Radiograph showing the picture after the turn plasty procedure is complete.

Based on Jaffe's Grading (1940)

Grade I—No minimal atypism of stromal cells. Giant cells are many and big sized. Grade II—Moderate atypism of stromal cells. Giant cells are a few and big sized.	Benign
Grade III—Severe atypism of stromal cells. Giant cells are a few small sized. Netherlands committee on bone tumors has added one more grade.	Borderline malignant
Grade IV—Sarcomatous dedifferentiation. Very few small sized giant cells (resembling malignant fibrous histiocytoma).	Malignant

Investigations

Radiograph: Shows classical epiphyseal, eccentric, expansile, lytic lesion which is loculated (soap bubble appearance) or non-loculated in a mature skeleton (growth plate closed).

Cortex is thinned out. Pathological fracture and invasion into a joint is a late feature and when seen early in the course of the disease, points out to the aggressiveness of the tumor.

CT and MRI helps in better delineation of the extent of involvement.

Treatment

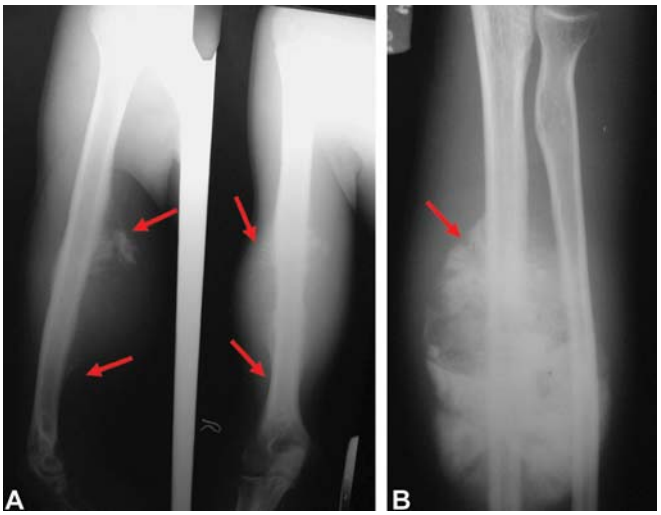
Total excision and reconstruction (Figs 9.9A to E) either by filling with bone grafts, polymethyl methacrylate or both. When reconstruction and salvage of joint is not possible arthrodeisis/total joint replacement with custom made prosthesis is to be considered.

Pulmonary metastasis if any when detected is excised locally.

Radiotherapy is reserved as a palliative procedure for tumors that arise in surgically inaccessible area.

Osteosarcoma

This is a highly malignant intramedullary bone tumor with high rate of mortality. Accounts for about 35% of bone malignancies.



Figures 9.10A and B

Radiograph showing radiological features of osteosarcoma. Note the Codman's triangle indicating reactive new bone formation.

The term 'Osteosarcoma' was coined in the year 1805 by Alexis Boyer, personal surgeon to Napoleon.

Types

Primary

a. Osteoblastic.
b. Chondroblastic.
c. Fibroblastic

} Are the conventional types and the name is given depending on the predominant tissue.

d. Sclerosing.
e. Osteolytic.
f. Telangiectatic (Figs 9.11A to C).
g. Multifocal.
h. Parosteal.
i. Periosteal.

Secondary

e.g. Osteosarcoma developing in Paget's disease, Fibrous Dysplasia, etc.

- Age: 10-20 yrs (except in secondary osteosarcoma which is seen in older age group).
- Sex: Incidence is slightly more in males.
- Site: Metaphysis of long bones. Common in lower end of femur, upper end of tibia and upper end of humerus. It is rare in jaw and skull. Very rare in extra skeletal sites. Multifocal origin is also seen rarely (either synchronously or metachronously).

Clinical Features

Pain is the earliest presenting feature which becomes the cause for concern. The pain gets aggravated with activity but not relieved by rest. Gradually limp/limitation of joint movement develops as swelling appears. It may be associated with constitutional symptoms.

Histopathology

This tumor is thought to arise from primitive mesenchymal cells and is diagnosed by the presence of malignant stromal cells laying down tumor osteoid (osteoblastic) alongwith areas of hemorrhage and necrosis and spicules of destroyed bone. Depending on the predominant tissue can be chondroblastic, fibroblastic, telangiectatic, etc.

Investigations

Radiograph: Shows in typical cases, 'Sun ray' or 'Sun burst' appearance with Codman's triangle. Cumulus cloud pattern in Sclerosing variety and expansile, eccentric lysis in Telangiectatic variety are the other radiographic features.

Sun burst or Sun ray appearance is seen because of bone formation along the Sharpey's fibers which firmly bind the periosteum to the bone (Figs 9.10A and B).

Bone scan, CT and MRI are useful in detail evaluation and planning the required treatment.

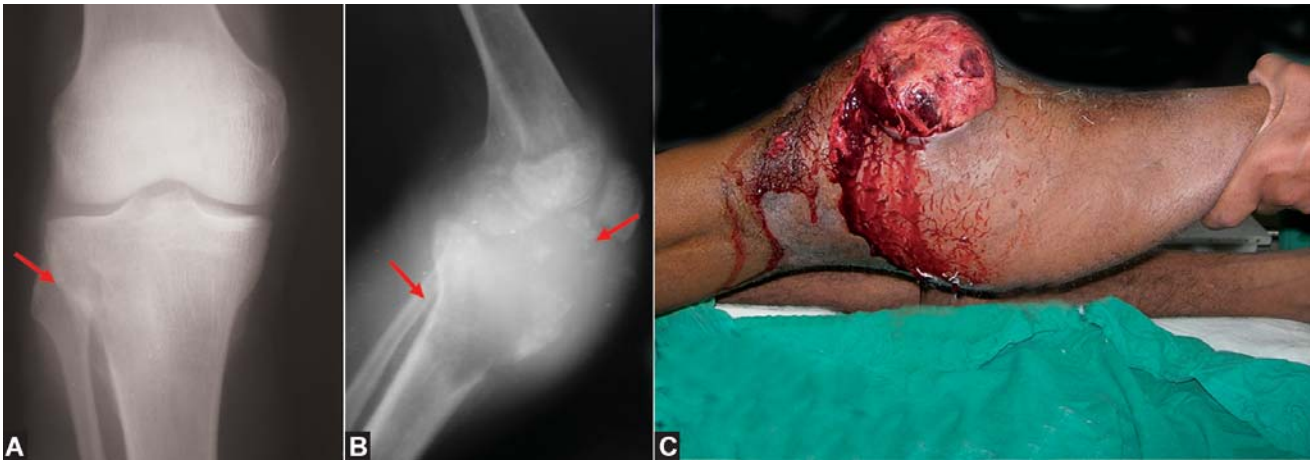
Treatment

Primary objective of treatment is to have a long term disease free survival. Limb salvaging is of secondary importance. Thus the treatment aims at increasing the survival rate with restoration of maximum function as feasible. **Limb salvage should always be attempted when possible but one should not hesitate to recommend amputation when needed.**

The approach is multidisciplinary. It varies depending on the site, size, type, local extension and distant spread. Pre-operative chemotherapy has increased the 5 years survival rate from 12% to 65-75%. Preoperative radiotherapy is given only when metastasis is detected.

Enneking system of staging (1980):

I—Low-grade tumor
  A—Intracompartmental
  B—Extracompartmental
II—High grade tumor
  A—Intracompartmental
  B—Extracompartmental
III—Either grade with distant metastasis.



Figures 9.11A to C

Showing a case of telangiectatic osteosarcoma. (A) At early presentation when grave nature of the tumor was explained and surgery was advised. (B and C) after 6 months of refusal of treatment and opting to take native medicine. Note the rapid increase in size, extensive destruction and no new bone formation as seen radiographically with fungation and active bleeding seen clinically.

Preoperative chemotherapy: It is useful and effective in shrinking or circumscribing the tumor mass, helping in planning of the surgical procedure, i.e. feasibility of a limb salvaging procedure. Also prevents the spread of the disease.

Drugs used are methotrexate, adriamycin and cisplatin (MAP regimen) in a cyclical manner. Expert evaluation is necessary to assess the physiological limit of tolerance of these cytotoxic drugs for each individual. Careful monitoring of heart, lungs, kidney, liver and hemopoietic system is a must.

Poor response: If the response is poor, other drugs like ifosfamide or etoposide are added.

Good response: If the response is good, interferon is given to get a cure.

5 years survival rate decreases in those who show poor response and vice versa.

Angiography

a. Helps to localize the displaced vessels.
b. Tumor blush: Osteosarcoma is a tumor which promotes neovascularization. Hence, there is enhancement of the contrast medium. This is referred to as 'Tumor blush'. After giving chemotherapy if this neovascularization is not seen (disappearance of vascularity) it infers that the chemotherapy had been effective.

Surgical Procedures

Surgery should always be done with adequate planning and after giving preoperative chemotherapy.

Limb salvaging procedures when feasible should always be done especially in early stage of the disease and when the tumor is localized. The procedure involves radical excision of the tumor and reconstruction with auto/ allograft (fibula). At times custom made prosthesis is implanted to substitute lost bone and joint.

Amputation is to be reserved only for late presentations. The procedure of rotation plasty is possible in lower limbs where after resection of considerable length of distal femur and proximal tibia, the limb is shortened and turned to 180° so that the ankle becomes the knee. A suitable prosthesis gives considerable function.

Lung metastasis are treated by local resection.

Postoperative chemotherapy is to be continued. Radiotherapy if needed to be given.

Chondrosarcoma

Chondrosarcomas are next common to osteosarcomas and account for about 25% of the primary malignant skeletal noeplasms. Present with a highly varied behavior from slow growing non-metastasizing tumor to fast growing metastasizing tumor. Histopathological features too vary accordingly.

- Types Primary and secondary
  Central (intramedullary) and peripheral.
- Age Primary occur above 40 yr
  Secondary occur 20-40 yr
- Sex M:F ratio 2:1
- Site—Flat bones like pelvis and scapula. From the metaphysis of long bones like femur, tibia and humerus.

Clinical Features

Presents with dull aching pain in the vicinity of a joint, aggravated by movement, worse at night. Joint effusion and limitation of movements develop later when swelling starts appearing.

Histopathology

Presents a varied picture of a well differentiated tumor producing normal hyaline cartilage to poorly differentiated tumor producing atypical hyaline cartilage. Hence grading is done. Grade I—low grade malignant; Grade II—moderately malignant; Grade III—high grade malignant. Calcification may be seen in areas. In addition, three specific types are recognized histopathologically:

a. Mesenchymal chondrosarcoma: Presents with bimorphic picture of both low and high grade malignancy.Common in spine, ribs and jaw.
b. Clear cell chondrosarcoma: Is a low grade chondrosarcoma consisting of clear cells with vacuolated cytoplasm. Matrix is significantly calcified. It is common in the epiphysis of the femur and the humerus.
c. Dedifferentiated chondrosarcoma: Is a highly malignant tumor presenting with features of other sarcomas like osteosarcoma, malignant fibrous histiocytoma, etc.

The 5-year survival in Grade I lesions is around 90% and in Grade III lesions is around 30%

Investigations

X-ray shows an expansile tumor with speckled calcification (Fig. 9.12). CT, MRI help in further delineation of the tumor.

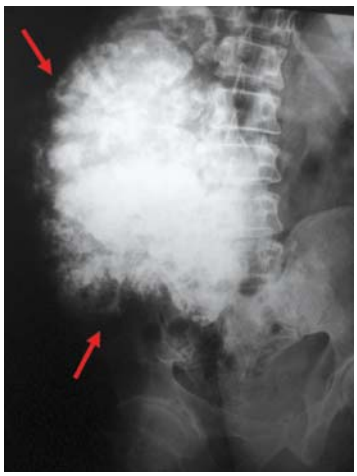


Figure 9.12

Radiograph showing a huge secondary chondrosarcoma arising from an aclatic lesion in the ilium in a patient with metaphyseal aclasis. Being radioresistant and chemoresistant and in this instance inoperable, it proved fatal.

Treatment

Wide surgical excision is the treatment of choice. Local recurrence is common by seedlings than distant metastasis except in aggressive type where pulmonary spread occurs via bloodstream. These are resistant to radiotherapy and chemotherapy. Hence, there is no role for radiotherapy and chemotherapy.

Ewing's Tumor

Described by James Ewing in the year 1921. Hence derives the name. It is a highly malignant primary bone tumor derived from the red marrow. Accounts for about 33% of primary bone tumors and stands second among the common malignant bone tumors in the young.

- Types
  a. Sclerotic
  b. Lytic.
- Age: 5-15 yr. Rare before 5 yr and after 30 yr.
- Sex: M:F ratio 1.5:1
- Site: Diaphysis of a long bone extending into metaphysis. It is rare in flat bones.

Clinical Features

Presenting feature is insidious onset of pain which is intermittent initially and then progresses to become continuous and more severe. Next, a rapidly growing swelling develops. If the axial skeleton is involved neurological deficits may be observed. Constitutional symptoms such as fever, malaise, etc may be present.

Histopathology

Highly cellular tumor comprising of sheets of small round neoplastic cells with large oval nuclei. Exhibits hyperchromatinism (stippled chromatin) and mitotic figures. Cytoplasm is sparse with ill defined cytoplasmic membrane. Pseudo-rosette formation may be present.

The tumor is considered to be arising from neural elements. Electron microscopy reveals neural elements such as neurites and dense core granules. The cells exhibit following characters. They are PAS positive, reticulum stain negative, stains for S-protein positive and stains against neuron specific enolase expression.

Investigations

X-ray shows a lytic, permeative lesion not well demarcated. The pattern of bone destruction varies from permeative pin

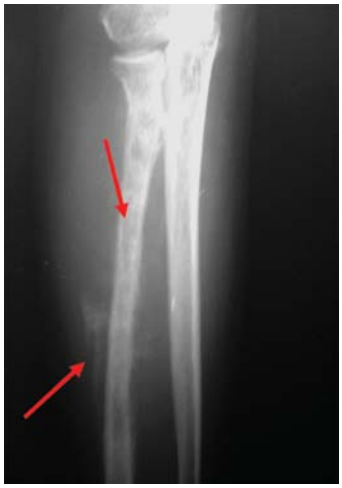


Figure 9.13

Showing layers of new bone formation giving rise to 'onion skin' appearance. Note that the tumor is primarily intramedullary, diaphyseal and extending into the metaphysis.

head sized holes, moth eaten, rotten wood to pure lytic. Hence, it can mimic a variety of bone tumors both benign and malignant. Periosteal reaction manifests with formation of a typical 'onion skin' appearance (Fig. 9.13) and is identified as a late and advanced feature of the tumor. Codman's triangle may be present.

CT and MRI help in better delineation of the tumor.

Treatment

As occult metastasis is very common in these tumors, multi-drug chemotherapy and local control by means of radiotherapy or surgery (only if local lesion is resectable) is the treatment of choice. This improves the chances of survival. Thus the approach is a multidisciplinary one.

The widely followed regimen is VAdriaC regimen/(VAC) i.e. vincristin, adriamycin and cyclophosphamide. Ifosfamide and etoposide are used when indicated to improve the outcome. After this multidisciplinary approach the 5-year survival rate has risen from 10% to almost 70%.

Myeloma

It is a tumor arising from neoplastic proliferation of plasma cells or myeloma cells. Depending on the site and nature of proliferation several presentations are recognized.

Types

Multiple myeloma Presents with multiple skeletal foci of proliferation (About 50%).

Solitary myeloma Also knowm as plasmacytoma, presents with a solitary skeletal foci of proliferation (about 25%).

Myelomatosis Also known as generalized myeloma, presents with a generalized and diffuse involvement of the skeleton. Results in thinning of the skeleton, resembling osteoporosis (About15%).

Plasma cell leukemia Malignant plasma cells in abundant in the bloodstream (About 1%) rare.

Extraskeletal myeloma Very rare. It is a variant and presents with a solitary tumor in the nasopharyngeal or oral cavity.

- Age: Multiple myeloma 60-70 yr.
- Solitary myeloma 50-60 yr.
- Sex: M:F ratio is 3:1
- Site: Originates from bones containing red marrow. Hence multiple myeloma is common in axial skeleton and flat bones than long bones.

  Solitary myeloma is common in the diaphysis of long bones and in the vertebral body.

Clinical Features

It presents with aching type of pain which is intermittent to begin with. It is either generalized or localized and relieved by bed rest. Later pain becomes more severe and constant. Constitutional symptoms develop. Easy fatigue is seen due to ensuing anemia, bleeding tendency and recurrent infections may develop because of the replacement of marrow by tumor cells. Renal failure (Myeloma kidney) and pathological fracture can develop late in the disease.

Histopathology

Nodular or diffuse infiltrate of plasma cells with loss of marrow fat and dissolution of bone is observed. These cells present with a single large 'clock face' or 'cart wheel' nucleus with abundant basophilic cytoplasm with rich endoplasmic reticulum. Perinuclear halo is characteristic. Atypism with presence of two or more nuclei, loss of perinuclear halo, loss of clock face and presence of mitotic figures indicate malignancy.

Investigations

a. Hb%, TC, DC, ESR
   Low Hb%, Low TC, granulocytopenia, raised ESR
b. Platelet count
   Thrombocytopenia
c. Serum Ca, PO_4, ALP
   Raised serum Ca, ALP normal except in patients pathological fracture.

d. Electrophoresis for increased gamma globulin fraction.
Shows increase in gamma globulin fraction.

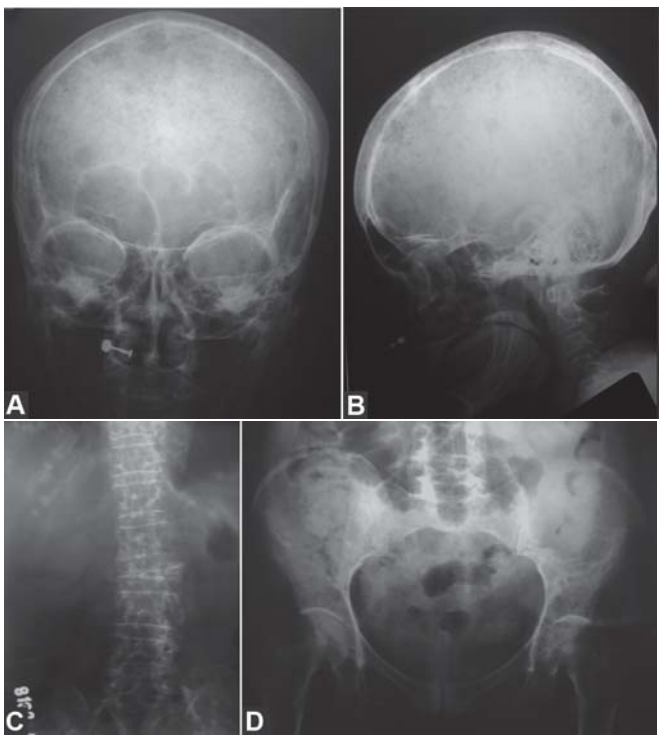
e. Urine for 'Bence Jones' proteins.
Described by English physician Henry Bence Jones in the year 1847.
These are immunoglobulins produced by plasma cells. These light chain molecules precipitate at 60°C and disappear on further heating at 90°C.
Reappear on cooling to 60°C

f. Bone marrow aspiration biopsy.

g. X-ray, CT, MRI
Radiograph shows solitary or multiple punched out lesions or generalized osteopenia with or without pathological fracture (depending on presentation) (Figs 9.14A to D).
CT and MRI help in detailed evaluation before instituting the therapy.

Treatment

- Myeloma cannot be cured. But can be controlled with treatment.
- Stable otherwise known as 'smoldering myeloma' does not require treatment.



Figures 9.14A to D

Radiographs showing generalized punched out lesions in the skull, vertebrae, ribs and pelvic bones which are classical of multiple myeloma.

- Unstable myeloma is treated by chemotherapy, radiotherapy and bone marrow transplantation. Treatment aims at replacing the abnormal marrow with normal marrow by means of peripheral stem cell transplantation.
- Some of the chemotherapeutic regimens are as follows:
  i. Thalidomide/dexamethasone, vincristine
  ii. Vincristine, adriamycin, dexamethasone
  iii. Melphalan, prednisone
  iv. Melphalan, prednisone, thalidomide
  v. Cyclophosphamide, prednisone.

Metastatic Bone Disease

These are generally metastasis from adenocarcinomas. Following adenocarcinomas are known to cause secondaries in the bone (Fig. 9.15).

i. Thyroid
ii. Breast
iii. Lungs
iv. Liver
v. Kidneys
vi. Prostate

Of these all are lytic in nature except from the prostate which is sclerotic. At times the primary tumor is not detectable with routine investigations and bone scan is useful for identification.

The treatment of metastatic bone disease is always palliative and aims at providing pain relief and increasing the comfort of a terminally ill patient.

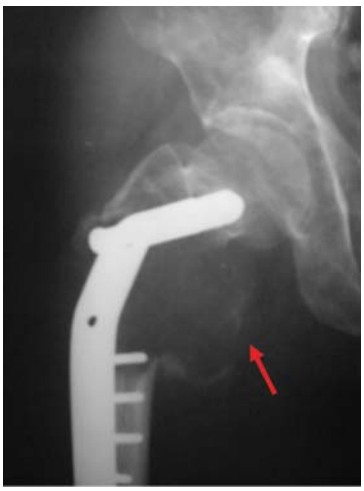


Figure 9.15

Radiograph of a missed diagnosis. A pathological fracture which was routinely fixed as a traumatic fracture. Patient was not relieved of pain even after surgery. An X-ray taken after 2 months confirms the diagnosis of a secondary.

A summary of different radiological features seen in bone lesions and their probable diagnosis	
Radiological features	*Probable diagnosis*
"Kissing" bones (lytic lesions in adjacent epiphysis)	GCT, Angiosarcoma, Pigmented villonodular synovitis, Infections
Codman's triangle	Osteosarcoma, Osteomyelitis, ABC
Complete sclerotic rim, no break	Benign lesion (95% accuracy)
Cumulus cloud appearence	Osteosarcoma, Stress fracture
Epiphyseal, solitary, eccentric lytic lesion with sclerotic margin	Chondroblastoma, Enchondroma, GCT
Epiphyseal, solitary, lytic lesion without sclerotic margin	GCT
Expansile lesion, poorly demarcated with windblown calcifications	Chondrosarcoma
Expansile lesion nontrabeculated lesion.	Benign tumor (majority of cases), Grade I sarcoma, Solitary Myeloma, Metastasis (a small percent of cases)
Expansile, trabeculated lesion	Grade I sarcoma, GCT, Myeloma
Fallen fragment sign	Simple bone cyst
Finger-in-the-balloon appearance	ABC
Ground glass appearance	Fibrous dysplasia, Osteoblastoma, Grade I osteosarcoma
Onion-skinning	Ewing's sarcoma, Subacute Osteomyelitis, Eosinophilic granuloma
Ring-like to popcorn density	Enchondroma and secondary Chondrosarcoma

Revision Questions

Q. Write notes on
   a. Osteoid osteoma
   b. Enchondroma
   c. Benign chondroblastoma
   d. Aneurysmal bone cyst
   e. Unicameral bone cyst
   f. Giant cell tumor
   g. Osteochondroma
   h. Chondromyxoid fibroma
   i. Osteosarcoma
   j. Chodrosarcoma

Q. Write briefly on
   a. Multiple myeloma
   b. Myelomatosis
   c. Bence Jones proteins
   d. Electrophoresis
   e. Plasma cells

Q. Write notes on
   a. Codman's triangle
   b. Sun ray appearance
   c. Onion skin appearance.
   d. Metastatic bone disease
   e. Pathological fracture.

Essay Questions

Q. Discuss the clinical features, radiological features, diagnosis and management of osteosarcoma (The same question can be put for any other bone tumor).

Q. How will you evaluate a case of bone tumor? Discuss the management of Giant cell tumor of the bone arising from the upper end of tibia.

Q. What are the types of Chondrosarcoma? Discuss the diagnosis and management of chondrosarcoma.

Further Reading

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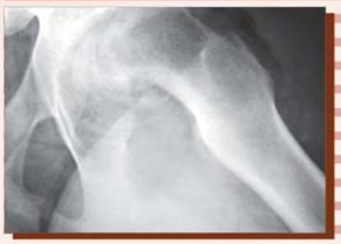


10

SCFE, Perthes' Disease and Other Osteochondritis

Slipped Capital Femoral Epiphysis (SCFE)

This condition is known by various names as adolescent coxa vara, epiphyseal coxa vara and epiphyseolisthesis. It refers to an idiopathic slipping of the capital femoral epiphysis which occurs commonly during the period of rapid growth, i.e. between 12 and 18 years. The slip may occur gradually, acutely or acute on a gradual slip.

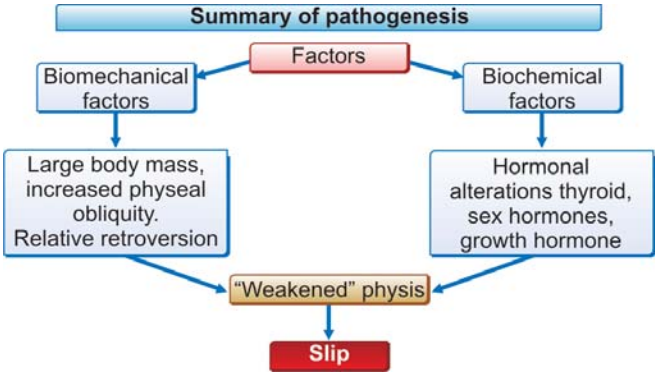


Figure 10.1
Showing the flow chart of the possible factors in pathogenesis.

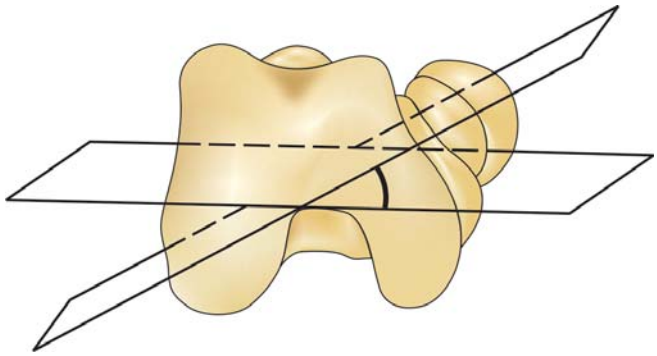


Figure 10.2
Showing the axis of femoral neck and axis of the transcondylar plane by which the ante/retroversion is measured.

Predisposing Factors (Fig. 10.1)

a. Male sex. M:F ratio 5:1 left hip commonly affected than the right hip.
b. Obesity.
c. Weakening and thinning of the physis.
   Exact reason is not known. Thought to be due to malnutrition and in some hormonal imbalance as characterized by relative preponderance of growth hormone, resulting in either Frohlich's type (sex hormone decrease) or Tall type (growth hormone increase) of individuals with respective growth abnormality.
d. Altered inclination of the upper femoral physis.
   During adolescence the inclination of the physis changes from horizontal to oblique.
e. Relative or absolute retroversion of the neck.

Anteversion and retroversion: Angle of inclination of the axis femoral neck in relation to transcondylar plane is known as version. If the inclination is in anterior direction it is known as, anteversion, antetorsion or anterior twist. If this angle of inclination is in posterior direction and the neck points in posterior direction it is known as retroversion, retrotorsion or posterior twist. In a newborn anteversion is as high as 30°. It progressively decreases in childhood and in an adult it is around 10-15° (Fig. 10.2).

Thus, it is postulated that even a normal stress can cause gradual displacement to occur at this weakened physis when there is excessive anteversion. In fact, the femoral epiphysis remains in contact with the acetabulum and it is the neck of the femur which moves anteriorly and superiorly. But for some reason the slip is described, taking into account the relationship of the epiphysis (minor fragment) to the femoral neck. And it is described that the slipping of the epiphysis occurs posteriorly and inferiorly.

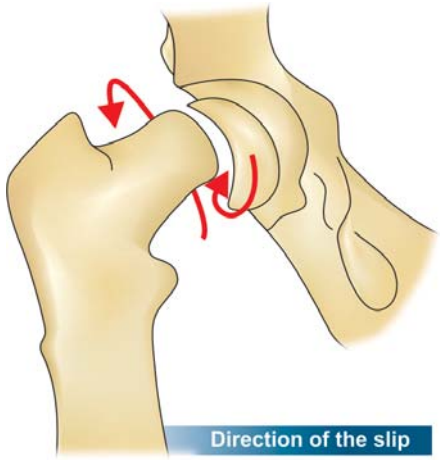


Figure 10.3
Diagrammatic representation of the slip which always occurs in posterior and medial direction.

Pathology of the Slip (Figs 10.3 and 10.4)

The slip occurs in the zone of hypertrophic cartilage, or in some (rearely), in the zone of provisional calcification. In these zones, histopathologically, very few normal chondrocytes are seen along with some degenerated and dead chondrocytes. The supportive collagenous matrix is also found to be abnormal. Whether these changes are the cause of the slip or the result of the slip is a debatable issue.

In other words whether biochemical/endocrinal factors have caused this change or the mechanical trauma of the slip has resulted in the change is being debated.

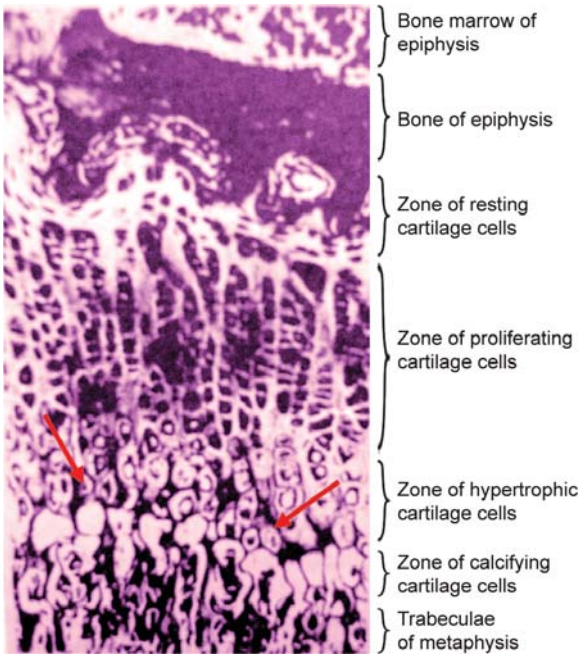


Figure 10.4

Showing histopathologic section of the physis. Slip always occurs at the zone of hypertrophic and maturing cartilage as shown by red arrow.

Clinical Features

- Adolescent. Obesity should increase the suspicion.
- Pain in the hip of gradual onset (at times may present with knee pain).
- Progressive limp.
- Progressive limitation of movement.
- Progressive deformity of adduction and external rotation.
- Axis deviation sign. (Flexion of the hip causes the abduction and external rotation of the limb and the limb falls away from the shoulder.)
- Antalgic gait (indicates that the slip is unstable).

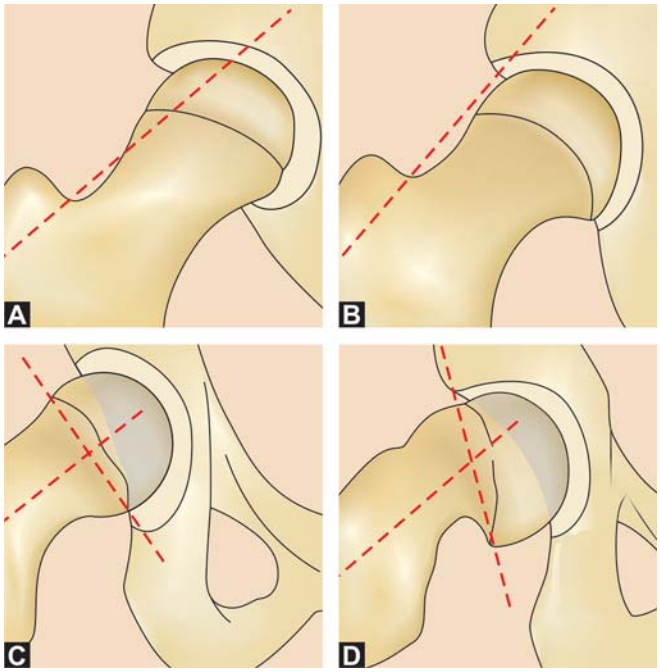
Investigations

X-rays of both hips to be taken as the slip may be bilateral and if unilateral opposite hip is used for comparison. Standard AP, LAT and Frog leg views of pelvis with both hips are taken (Figs 10.5A to D).

AP View

Early sign of slip is indicated by Trethoven's line.

A line drawn along the superior border of the neck of the femur cuts the superior part of the epiphysis in normal hips. In SCFE it passes over the superior border of the epiphysis.



Figures 10.5A to D

Showing A and C the normal hip wherein the Trethoven's line cuts the upper part of the femoral epiphysis in AP view and in LAT view, the line at the base of the epiphysis and the line bisecting the neck of the femur are at right angles. B and D showing the line passing above the epiphysis in AP view and the angle is less than 90° in the LAT view. These changes are the early indicators of the slip.

Lateral View

It is more reliable and identifies even the minor degrees of the slip. Two lines are drawn .One bisecting the neck and the other at the base of the epiphysis. In a normal hip these lines are at right angles. An angle less than 90° indicates that the slip has occurred.

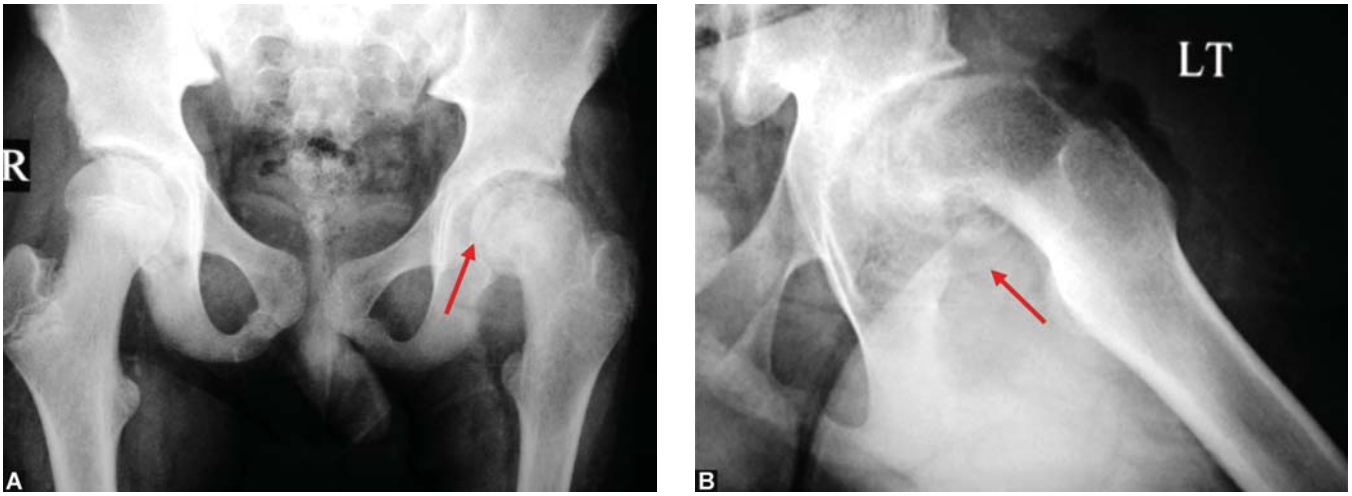
Frog Leg View

A straight line drawn along the center of the femoral neck when continued proximally passes through the center of the epiphysis in Frog leg view. If it passes anteriorly it indicates SCFE.

Radiological Classification of the Slip

Type I slip—less than 33% displacement.
Type II slip—between 33% and 50% displacement.
Type III slip—greater than 50% displacement (Figs 10.6A and B).

In the long standing cases of SCFE secondary changes are seen in the head and neck such as avascularіy of the epiphysis and malunion of the slipped epiphysis.



Figures 10.6A and B

Radiograph showing Type III slip. Note the posteroinferior direction of the slip and anterosuperior projection of the neck of the femur.

Bone scanning, magnetic resonance imaging (MRI), and computed tomography (CT) scanning help in more accurate assessment of the slip and the presence of any complications. These also help in the accurate planning of the surgical procedure.

Treatment

Aim

The aim of the treatment is to:

a. Prevent further slip.
b. Achieve premature closure.
c. Reduce the risk of AVN and chondrolysis.

A preliminary traction helps significantly in decreasing the degree of the slip.

The Surgical Procedures

The procedures are based on the type and the nature of the slip (i.e. the percentage of slip) and whether the slip is stable or unstable. Those slips which present with pain and antalgic gait are considered unstable and need immediate stabilization; thus, preventing further progression.

Cannulated Screw Fixation and Osteotomies

In unstable Types I, II and III, by means of gentle manipulation on a fracture table the slip is reduced and then fixed using a single cannulated screw. The screw fixation helps in premature closure of the physis and prevents further slipping.

Further slipping means that the stable slip has become unstable.

In stable type I, the fixation is done in situ. It aims at preventing further slip.

In stable type II, choice is between fixation in situ only or fixation with osteotomy.

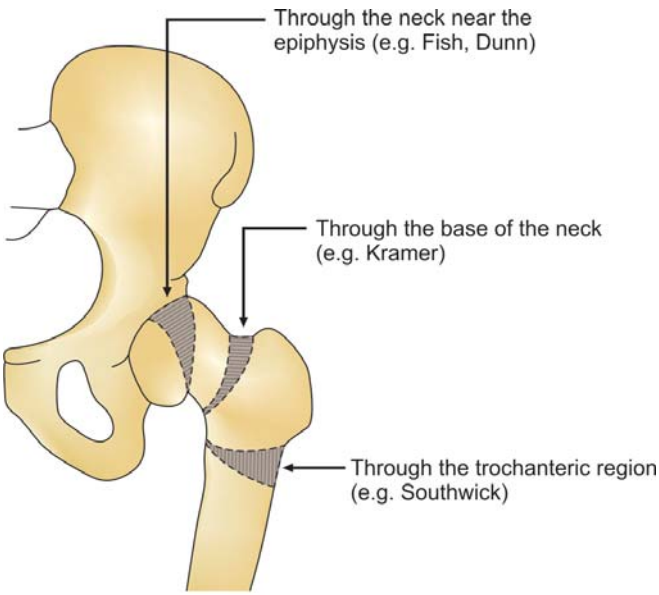
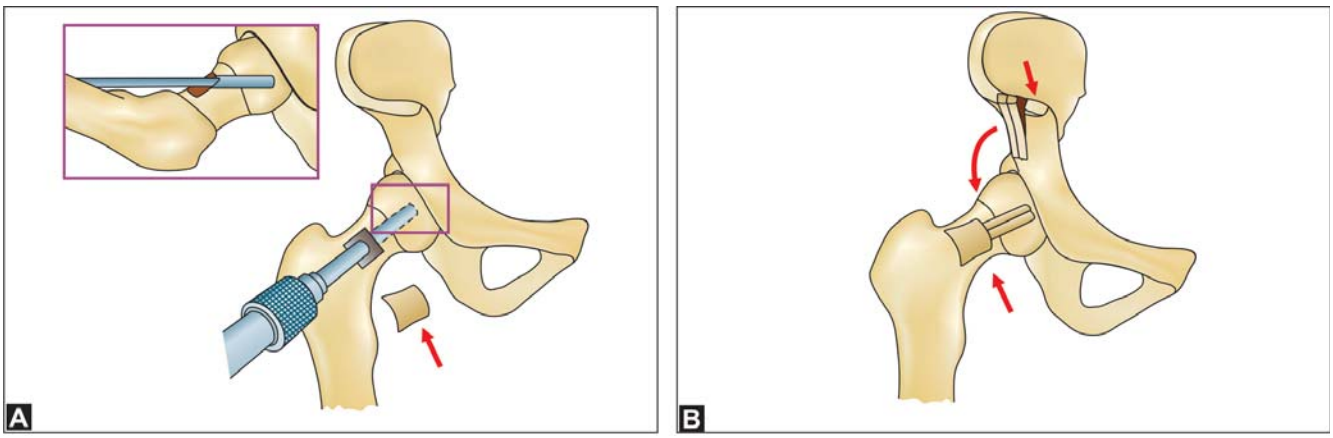


Figure 10.7

Diagrammatic representation of the sites of different osteotomies done for SCFE.



Figures 10.8A and B

Diagram showing (A) the method of preparation of the neck and epiphysis of the femur by lifting an osteomised rectangular block of bone and then (B) insertion of sandwiched iliac crest graft for achieving epiphysiodesis followed by placing the bone block back in its place.

In stable type III, osteotomy/epiphysiodesis may be considered.

Osteotomy aim: An osteotomy aims at restoring the relationship of the head and the neck of the femur. ***The more proximal is the osteotomy better is the correction but greater is the risk of AVN and vice versa.***

Several osteotomies are designed through the apex of the femoral neck, base of the femoral neck and intertrochanteric area, along with or without fixation of the epiphysis to the neck of the femur (Fig. 10.7).

Bone Peg Epiphysiodesis

Aim: The procedure aims at achieving a premature closure of the epiphysis.

Procedure: In this procedure, corticocancellous graft obtained from the iliac crest is sandwiched and driven through a window created in the metaphysis of the neck of the femur into a tunnel created in the physis. Lower incidence of AVN and absence of implant related complications are distinct advantages of this procedure (Figs 10.8A and B).

Prognosis: Stable slips have better prognosis following treatment.

- Unstable slips have a higher risk of poor prognosis following treatment. This is because of the damage that occurs to the blood supply of the epiphysis at the time of acute slip and not because of the surgical procedure.
- Similarly early presentation and minor degree of slip have better prognosis than late presentation and more severe degree of slip.

Prophylactic Pinning of the Opposite Hip

Prophylactic pinning is indicated based on Oxford bone score. This score is based on the stages of development of ilium, triradiate cartilage, head of the femur, greater trochanter and lesser trochanter. A score around 16 shows 85% chance of slipping occurring in contralateral epiphysis.

Correction of residual deformity after closure of the physis is not recommended. Because there is not enough proof that it helps in preventing the late complications.

Complications

Chondrolysis and avascular necrosis are the late complications of SCFE. When the pain is severe and functional limitation is troublesome the answer is either total joint arthroplasty or arthrodesis.

Revision Questions

Q. What are the other names for slipped capital femoral epiphysis?
Q. How will you classify the SCFE?
Q. Discuss the etiopathogenesis of SCFE
Q. Draw Trethoven' s line.What is its importance?
Q. Discuss the management of SCFE.
Q. What are the complications of SCFE? How will you manage them?

Essay Question

Q. Discuss the etiopathogenesis of slipped capital femoral epiphysis. How will you investigate, diagnose and treat a case of slipped capital femoral epiphysis. Enumerate its complications.

Legg-Calve-Perthes' Disease

It was Arthur Legg in 1909 who described the disease in the annual meeting of American Orthopaedic Association in Hartford, Connecticut, USA. At the time he published his work in 1910, Jacques Calve of France and George Clemens Perthes of Germany had published their cases too. Hence the name is derived.

Though Henning Waldenström had first described the radiological changes of this disease he thought this to be a type of benign tuberculosis. The disease is also known by different names as osteochondritis deformans juvenilis, pseudocoxalgia and coxa plana.

It is a disease occurring in the young children between the age of 5-15 years characterized by varying degrees of idiopathic avascular necrosis of the upper femoral epiphysis followed by spontaneous revascularization occurring over a period of time, i.e 1½-3 yr during which period the head is exposed to the risk of deformation. Hence, needs protection from deforming forces (Figs 10.16A and B).

Predisposing Factors

The origin of the disease is considered to be multifactorial following are the factors observed.

1. *Genetic:* The disease shows a male predominance with a M:F ratio 5:1. Also incidence is higher among later siblings. The Asians, Eskimos and Whites have a higher incidence when compared to Australian aborigines, Polynesians, American Indians and Blacks.
2. *Abnormal growth and development:* The bone age in patients with Perthes' disease is found to be lower than their chronologic age by 1-3 years.
3. *Environmental factors:* Higher percentage of involvement is seen among the lower socioeconomic group.
4. *Trauma*
5. *Transient synovitis*
6. *Passive smoking*
7. *Abnormal venous drainage:* Normally venous blood flows through medial circumflex vein. Because of the increased pressure in Perthe's disease there is congestion in the metaphysis and the blood flows through the diaphyseal veins.
8. *Arterial block and infarction.*
9. *Increased viscosity of the blood.*

Pathology

The pathology of idiopathic avascular necrosis followed by spontaneous revascularization passes of in 4 stages:

1st stage—stage of synovitis: In this stage the synovium is swollen, edematous and thickened. There is effusion into the joint. But no inflammatory cells are found in the fluid.

2nd stage—stage of avascular necrosis: In this stage the trabeculae are crushed and compressed into a compact mass. Debris filled spaces as well as vacant lacunae are seen. Metaphyseal hypervascularity is evident. Articular cartilage remains intact and shape of the head is maintained.

3rd stage—stage of (fragmentation) revascularization: In this stage highly vascular connective tissue invades the area and the necrotic tissue is replaced by osteogenic tissue. Occurs over a period of 1½-3 yr.

4th stage—residual/healed stage: All the necrotic bone is replaced by mature bone.

The metaphysis also shows certain changes such as lytic areas with sclerotic margins, abundant fatty marrow, disarrayed ossification process, etc.

Radiological Features

The pathological changes that occur produce certain characteristic radiological features.

Thus the disease has been staged by Elizabethtown staging into 4 stages and grouped by Catterall into 4 groups.

Based on Elizabethtown Staging (Figs 10.9A to D)

Stage I (Initial): Shows condensation, compression and increased density of the epiphysis with widening of the medial joint space (Waldenstrom's sign).

Stage II (Fragmentation): Further shrinkage and fragmentation of the epiphysis is seen. It indicates an intermediate reparative phase.

Stage III (Regeneration): Shows more pronounced fragmentation along with new bone formation. It indicates healing phase of the disease.

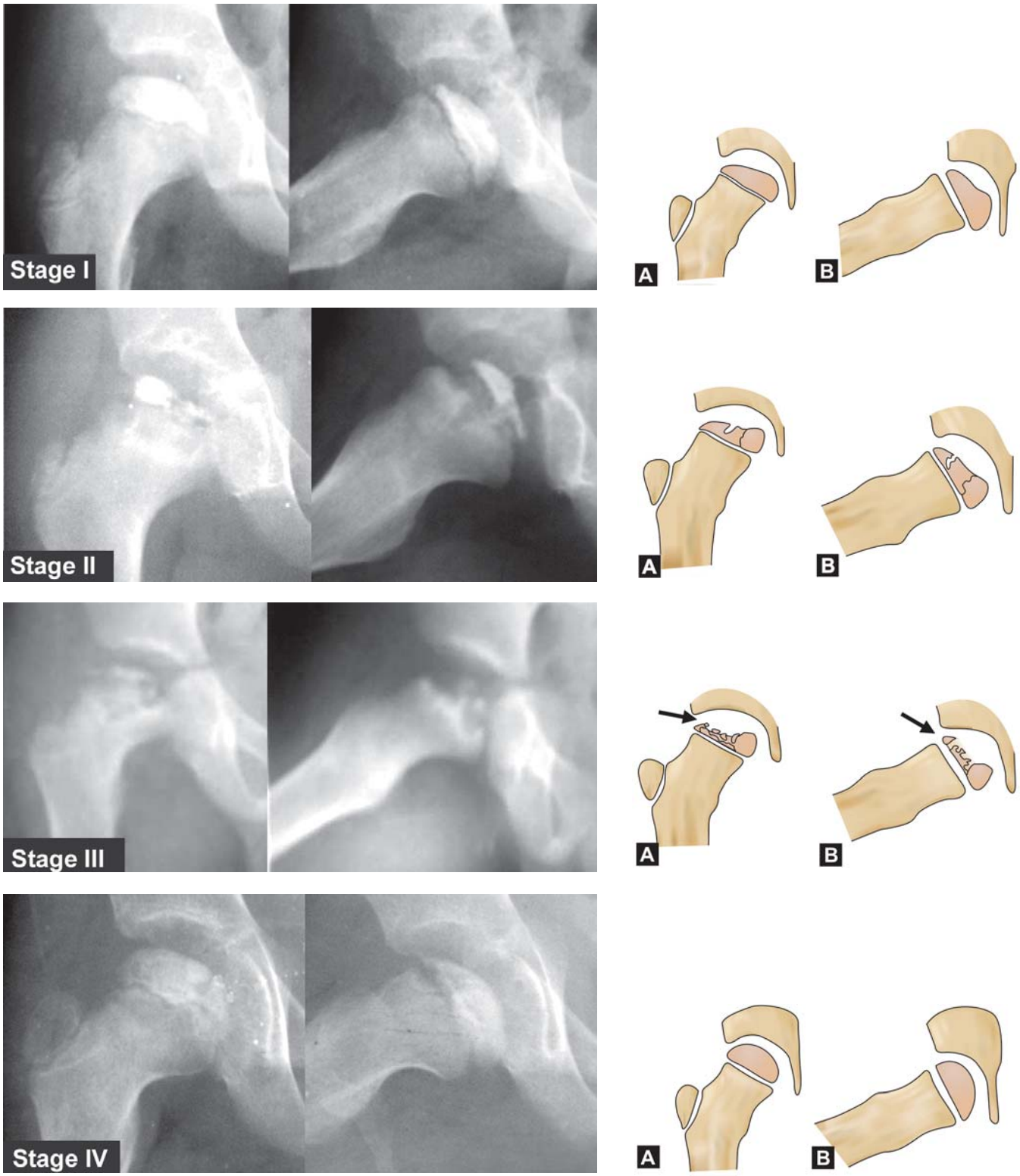
Stage IV(Healed): Complete re-ossification. No fragmentation or increased density observed.

Based on Catterall Grouping (Figs 10.10A to D)

The disease is grouped into 4 groups depending on

- Extent of epiphyseal involvement
- Presence of sequestrum.
- Presence of collapse.
- Metaphyseal abnormality.

It is summarized in Table 10.1.



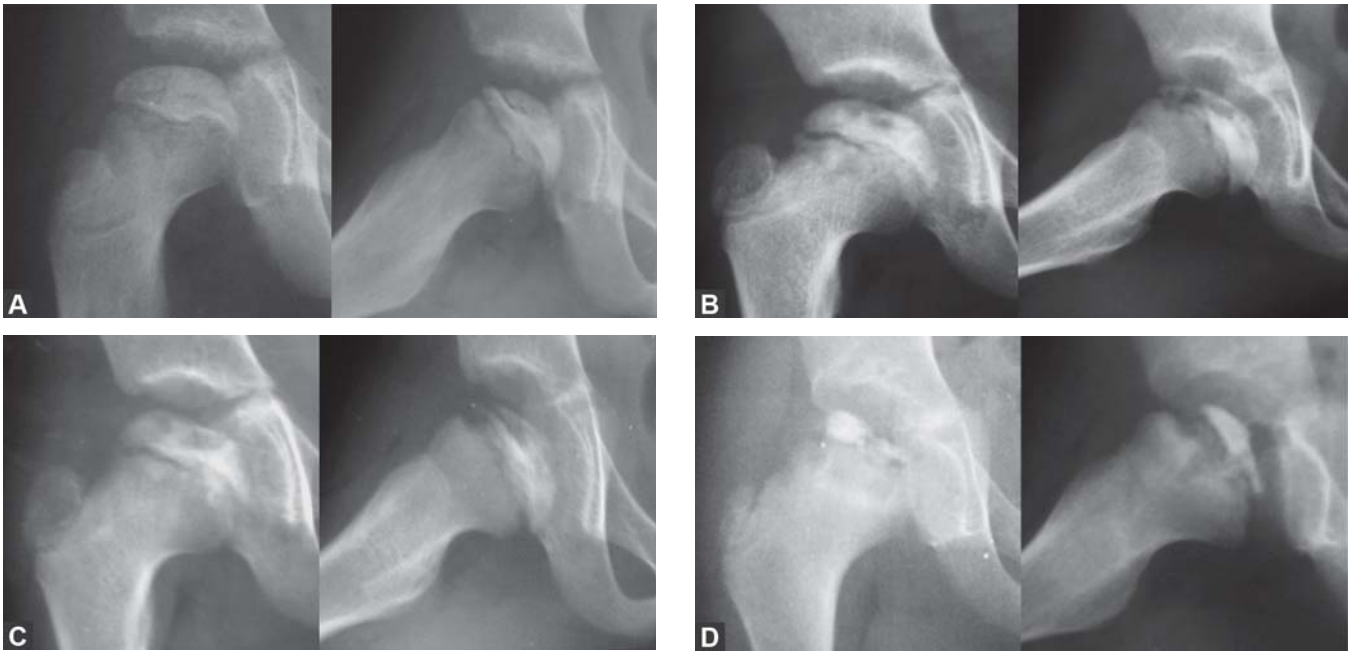
Figures 10.9A to D

Radiographs of different clinical stages of Perthes' disease as per Elizabethtown staging and diagrammatic representation of Elizabethtown staging.

Table 10.1

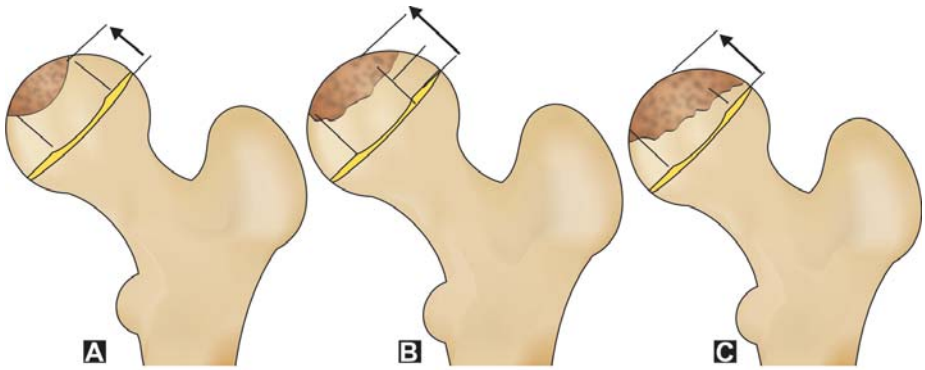
Showing the portion and nature of involvement of the epiphysis and metaphysis in different groups based on Caterall's classification

	Group I	Group II	Group III	Group IV
Epiphyseal involvement	Anterior and lateral part	More anterior part	Entire epiphysis except a small posterior part	Entire epiphysis
Sequestrum	No	Yes	Yes	Yes
Collapse	No	Yes	Yes	Yes
Metaphyseal abnormality	No	Localised	Diffuse	Diffuse



Figures 10.10A to D

X-ray picture AP and LAT view showing different groups according to extent of head involvement based on classification by Catterall. A-Group I, B-Group II, C-Group III, D-Group IV. Note the head within the head sign in Group III.



Figures 10.11A to C

Showing the fragmentation based on lateral pillar concept of Herring: (A) Only central pillar fregmentation. The lateral pillar protects the fragmented part, (B) Lateral pillar fragmentation < 50% of the height of the epiphysis and (C) Lateral pillar fragmentation > 50% of the height of the epiphysis.

Classifications Useful to Assess Prognosis

Based on Herring's lateral pillar classification: In this the epiphysis is divided into three pillars; the medial, central and lateral respectively (Figs 10.11A to C).

The classification is based on the changes that occur in the lateral portion of the femoral head (lateral pillar) when early fragmentation begins. When the lateral pillar is intact it acts as a weight bearing support to protect the central avascular fragment. When lateral pillar is involved and collapsed the weight bearing stresses damage the epiphysis rapidly.

Salter and Thomson classification: This is based on the presence, location and extent of subchondral fracture. 4 groups are identified. Prognosis is directly proportional to the chronological order.

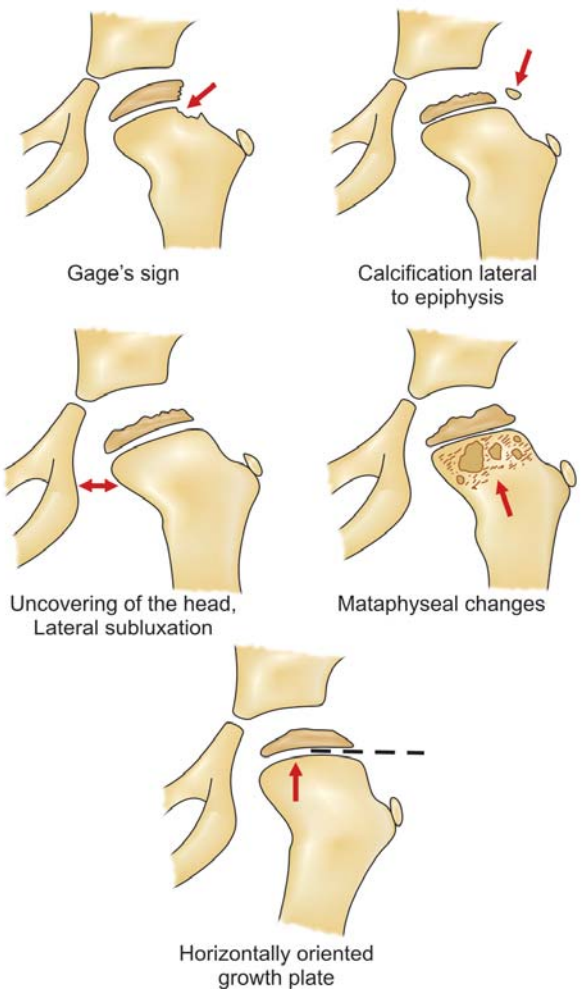


Figure 10.12
Diagrammatic representation of head at risk signs.

Clinical Features

Onset is insidious. Presenting features are as follows:

1. Limp associated with or without dull pain.
2. Pain in the hip region. Medially in the groin, laterally in the region of the greater trochanter and anteriorly in the inguinal region. At times radiates to the knee.
3. Pain is exaggerated by activity, relieved by rest.
4. Classical limitation of abduction internal rotation.
5. Trendelenburg gait.

Head at Risk Signs

Clinical "Head at Risk" Signs

1. Older child.
2. Heavy child.
3. Progressive loss of movement.
4. Adduction contracture.
5. Flexion with adduction deformity.

Radiological "Head at Risk" Signs (Fig. 10.12)

1. Gage sign (1933)
   It is a 'V' shaped radiolucent defect on the lateral side of the epiphysis.
2. Calcification lateral to the epiphysis.
   Occurs due to thickened and extruded epiphysis. Indicates epiphyseal enlargement.
3. Lateral subluxation.
4. Horizontal growth plate.
5. Diffuse metaphyseal lesion.

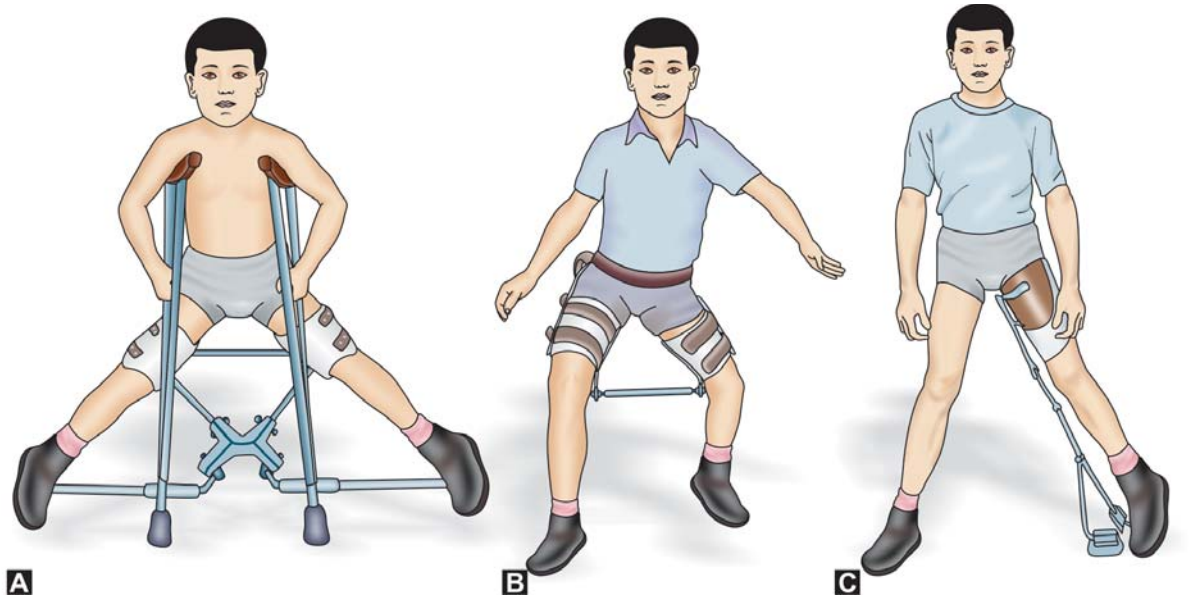
Among these the only pathology that is amenable for treatment is the lateral subluxation (Fig. 10.12).

Investigations

1. X-ray: Shows avascular changes as per stage of the disease.
2. Bone scan: Can diagnose the avascularity much before the radiological changes develop. Also useful in differentiating septic from aseptic inflammation.
3. MRI: Delineates the changes in the cartilaginous epiphysis and the joint much better than X-ray. Also useful to assess vascularity of the epiphysis.
4. Arthrography: Useful in the assessment of joint congruity and helps in planning the containment procedure.

Treatment

Aim: The aim of the treatment is to prevent the damage to the vulnerable, avascular epiphysis by protecting it till it gets fully vascularized.



Figures 10.13A to C

Showing nonsurgical method of containment with the help of braces which aim at keeping the hip in about 40-45° of abduction. Some of the popular designs are: (A) Toronto brace, (B) Atlanta Scottish Rite Children's Hospital brace, and (C) Tachdjian abduction brace. Diagramatic representation of the same is shown.

Such environment is created either non-operatively or operatively, whichever is found appropriate at the time of presentation. The procedure is known as containment. It contains the epiphysis within the confines of the acetabulum and takes off the stresses to which it is subjected, till complete revascularization takes place.

An effective treatment prevents complications of growth disturbance and secondary degenerative arthritis as a sequel of avascular necrosis.

Factors to be Considered in Planning the Treatment

1. Age of the patient
   <5 yr—consider nonsurgical containment only if subluxation is observed.
   5-7 yr—consider nonsurgical/surgical containment only if subluxation is observed.
   7-12 yr—consider nonsurgical/surgical containment before lateral subluxation occurs (when other head at risk signs are present).
   >12 yr—do not consider containment.
2. Extent of epiphyseal involvement:
   Consider containment if half or more than half of the epiphysis is involved (Catterall group II, III and IV).
   If less than half of the epiphysis is involved, do not consider containment.
3. Stage of the disease:
   In Elizabethtown stages I and II consider containment. In stages III and IV do not consider containment.
4. Range of hip movement
   If hip movements are normal consider containment. If hip movements are restricted do not consider containment.

To summarize surgical containment is considered in a vulnerable epiphysis, in a child of 7-12 years of age, with no restriction of hip movements, the stage of the disease according to Elizabethtown staging is Stage I or II and the extent of involvement as per Catterall clasification is group II III or IV. (Ref: reference 41-45 for more information).

Nonsurgical Containment Methods

A. Nonambulatory
   - Bed rest and traction in abduction.
   - Broom stick plaster.
   - Hip spica cast.

B. Ambulatory (Figs 10.13A to C)
   - Newington abduction frame orthosis.
   - Toronto brace.
   - Scottish- Rite orthosis.
   - Tachdjian brace (Trilateral socket hip containment orthosis).

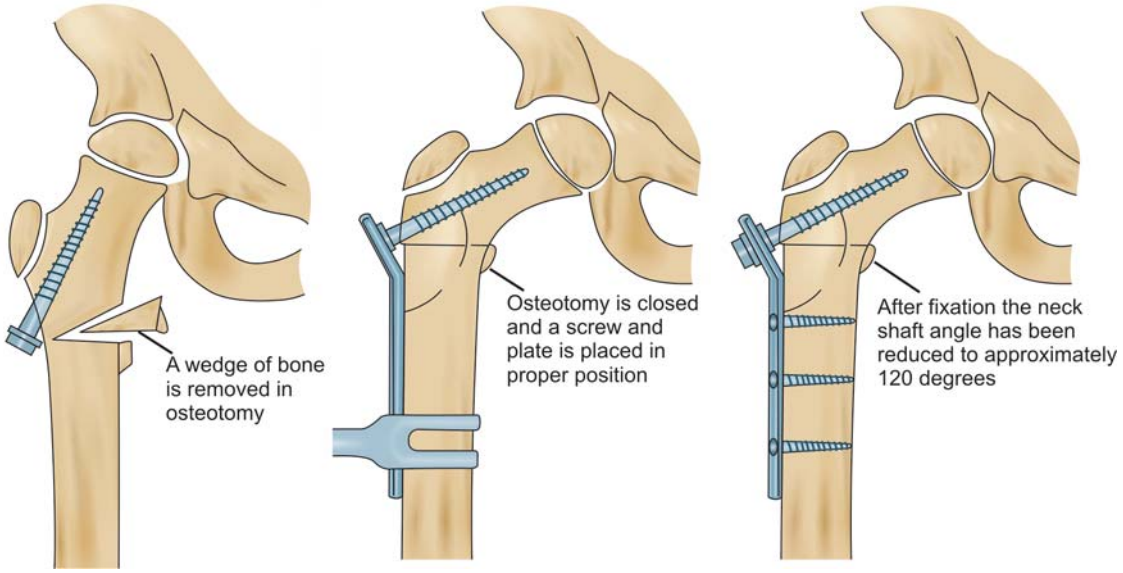


Figure 10.14

Diagrammatic representation of medial closed wedge osteotomy for containment. (The same may be performed by opening wedge laterally).

Surgical Methods of Containment

A. Femoral varus derotation osteotomy (Fig. 10.14).
B. Salter's innominate osteotomy (Fig. 10.15).

Prognostic Factors

- Age of onset.
  Early onset has better prognosis than late onset.
- Female gender.
  Said to have poor prognosis when compared to male.
- Remodeling potential.
  Varies from person to person. Greater the remodeling better is the prognosis.
- Protracted course.
  If the course of the disease is protracted, the prognosis is poor.
- Growth disturbance (Figs 10.16A and B).
  Premature closure of the physis leads to growth disturbance and may affect the outcome.
- Stage at which the patient presents for treatment.
  If the patient presents for treatment late the epiphysis would already have been deformed. This results in poor prognosis.

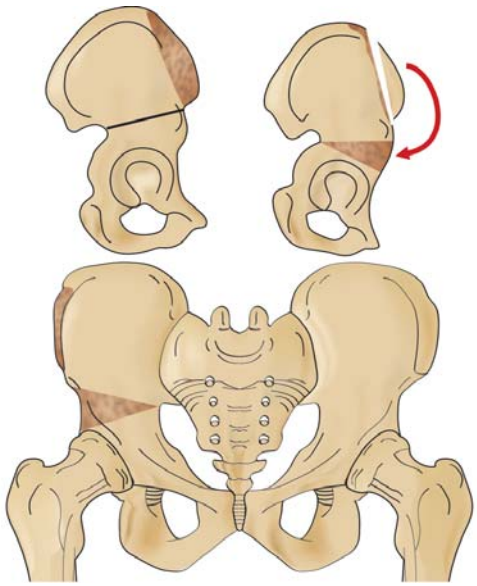
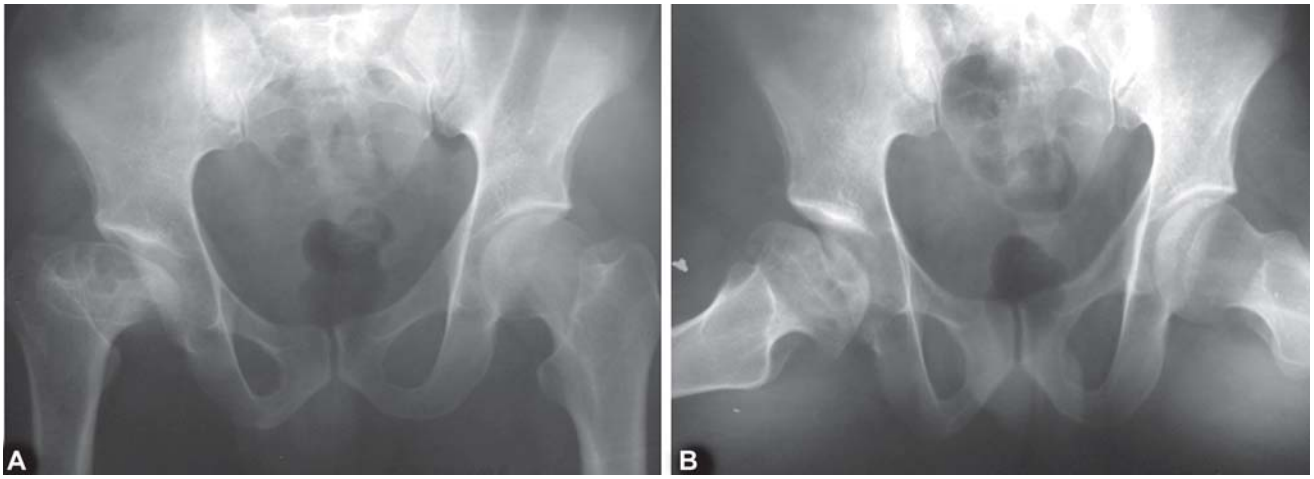


Figure 10.15

Showing Salter's innominate osteotomy procedure for containment of the epiphysis of the femur. Note that the ostetomy is done just above the roof of the acetabulum and opened .A triangular wedge of bone from the iliac crest is taken and inserted at the opened site and fixed.



Figures 10.16A and B

Radiographs AP and Frog leg view of pelvis with both hips showing late sequel of Perthes' disease. Note the irregularity and mushrooming of the head of the femur and the adaptive changes in the acetabulum in relation to the shape and size of the head. Both are incongruous when compared to the normal head but between them have maintained the congruity (incongruous congruity). Patient is walking with a pain free limp with shortening, i.e with a short limb gait. He is a candidate for secondary degenerative arthritis developing at a later period.

Revision Questions

Q. Define Legg-Calve-Perthes' disease.
Q. Discuss the stages of Perthes' disease
Q. Discuss the etiopathogenesis of Perthes' disease.
Q. What are the head at risk signs? Enumerate them.
Q. What is Waldenström's sign?
Q. Head within the head sign.
Q. Write briefly on classification of Perthes' disease.

Essay Question

Discuss the etiopathogenesis diagnosis and management of a case of Perthes' disease. Enumerate the complications.

Idiopathic Avascular Necrosis/ Osteochondritis

The exact etiology of these diseases is not known. Hence they are grouped under Idiopathic Avascular Necrosis. The various factors observed in different areas definitely indicate the role of mechanical factor in the origin and progress of the disease. Abnormal blood supply, increased viscocity of the blood, infarction and increased intraosseous pressure are also some of the factors which are identified. Thus it is wise to conclude that the origin and the progress of these diseases is multifactorial and so is the treatment. It varies from simple stress relieving measures such as restricted weight bearing, abstaining from strenuous activities, protective splints, etc. to surgical methods. Few of the common disease are discussed in this chapter.

Bones involved commonly in Idiopathic avascular necrosis/osteochondritis

Bone involved	*Name of the disease*
Scaphoid	Preiser's disease
Capitulum	Panner's disease
Lunate	Kienbock's disease
2nd metatarsal head	Freiberg's disease (infarction)
Navicular	Kohler's disease
Calcaneal apophysis	Sever's disease
Tibial tuberosity	Osgood Schlatter's disease
Upper femoral epiphysis	Legg-Calve-Perthes disease
Ring epiphysis of the vertebra	Scheurmann's disease
Central epiphysis of the vertebra	Calve's disease

Kienbock's Disease

It is avascular necrosis of the carpal lunate. It was first described by Robert Kienbock in the year 1910. He called it as lunatomalacia.

Etiology

Exact etiology is yet to be ascertained. It is considered as multifactorial. Certain predisposing factors have been identified. These factors perhaps predispose lunate to excessive/abnormal stresses, resulting in damage to the blood supply and avascular necrosis.

a. Ulnar variant (Hulten 1928)
   Ulnar-neutral–the distal radius and ulna are at the same level.
   Ulnar-plus–the distal ulna is farther than the radius.
   Ulnar-minus–the distal ulna is shorter than radius.
   In Kienbock's disease ulnar-minus variant has been commonly observed.
b. Shape of the lunate (Antuna Zepico, 1966)
   Type I—lunate with a proximal apex or crest.
   Type II—lunate is more squarish.
   Type III—lunate is more rectangular or squarish.
   In Kienbocks disease with Ulnar-minus variant, Type I lunate is observed. Whereas
   Type II and Type III lunates are seen in ulnar-neutral and ulnar-plus variants.
c. Slope of the distal articular surface of the radius (Mirabello, Werner, Palmer)
   In Kienbock's disease the normal inclination of the articular surface of the lower radius is found to be decreased.
d. Primary circulatory disturbances and increased intra-osseous pressure are also considered as predisposing factors.

Investigations

X-ray: Shows increased density of the lunate suggesting avascularity. Fractures and fissuring, carpal collapse and degenerative changes are seen as the disease progresses.

MRI: Delineates the abnormality more clearly.

Treatment

Treatment depends upon the age of the patient and the state of the lunate and the carpus.

Early: Early in the disease if mechanical variants are present they are corrected by osteotomy and radial shortening procedures.

Late: In advanced cases where degenerative changes are present Scaphotrapezio-triquetral/scaphocapitate arthrodesis or wrist arthrodeisis is indicated.

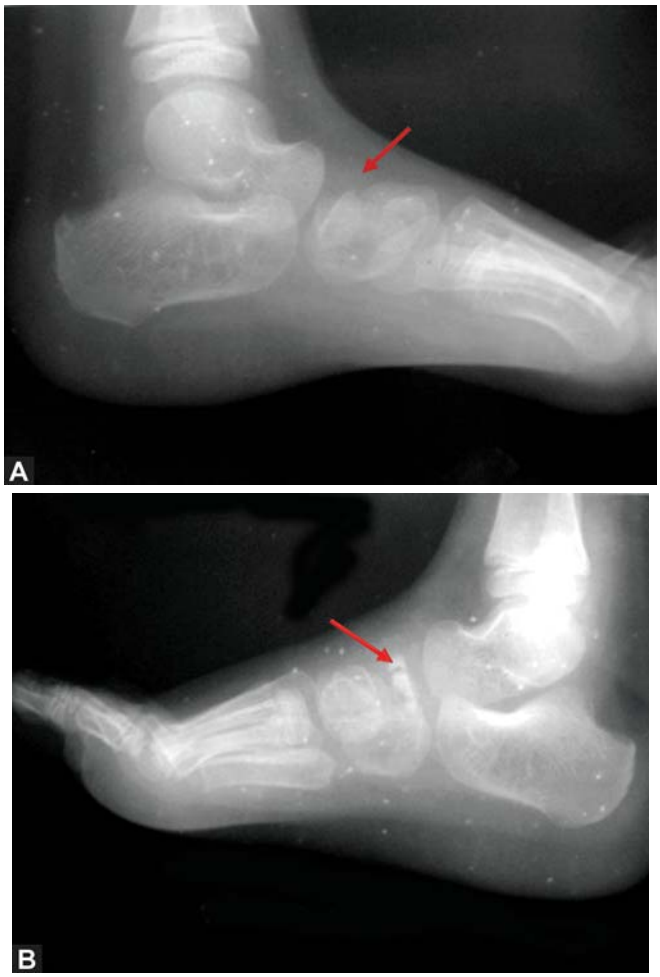
(Nonoperative treatment in the form of prolonged immobilization is known to cause progression of the disease).

Kohler's Disease

It is avasculr necrosis (osteochondritis) of the tarsal navicular. First described by Kohler in 1908, the disease is self-limiting and invariably resolves over a period of time. The disease is more common in boys than girls. It is common between 2 and 9 years (Figs 10.17A and B).

Etiology

a. Repetitive mechanical stress is thought to produce microfractures and avascularity.
b. Abnormal vascularity of the navicular.



Figures 10.17A and B

Radiograph B showing Kohler's disease. Note the increased density and fragmentation and the small size of the navicular bone. A is the radiograph of normal foot placed for comparison.

Investigations

X-ray and MRI if needed. These investigations confirm the avascularity.

Treatment

The disease should be treated by restricted weight bearing and protection in cast and splint. Healing invariably occurs over a period of time.

Revision Question

Q. Write briefly on osteochondritis. Enumerate different osteochondritis.

Q. How will you manage a case of osteochondritis?

Further Reading

Slipped Capital Femoral Epiphysis

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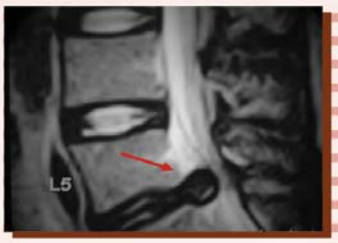


11

Intervertebral Disc Prolapse/Herniation

- *Structure and Function of the Disc*
- *Pathomechanics of Disc Prolapse*
- *Terminologies and Types*
- *Clinical Presentation: Symptoms, Signs and Diagnosis*
- *Investigations*
- *Treatment*
- *Disc Replacement, its Current Status*

Introduction

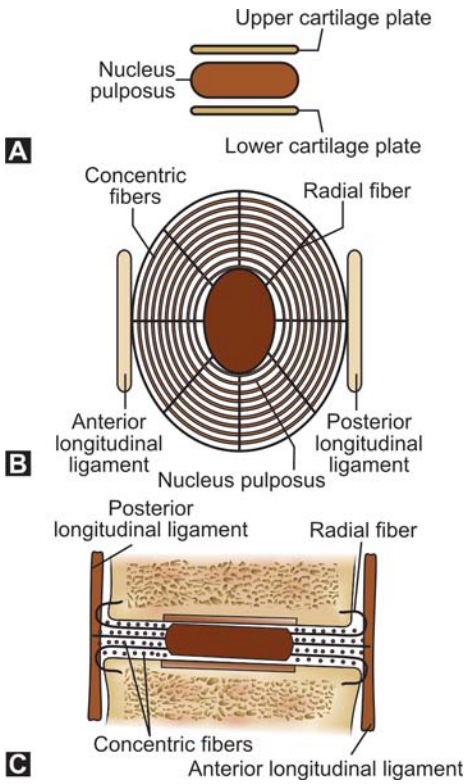
Intervertebral discs constitute approximately 1/4th of the vertebral column. They are designed to absorb the shocks when the spinal column is subjected to mechanical loading during the activities of daily living. It has a central nucleus pulposus made up of collagen, proteoglycans and water and its consistency is gel-like. It is placed somewhat like a cushion between the cartilaginous end-plate of the adjacent vertebra. It is surrounded by annulus fibrosus made up of concentric sheets of collagen fibers which are connected to vertebral end-plates. These concentric sheets are held firmly by radial fibers which are placed at intervals (Figs 11.1A to C).

Pathomechanics of Intervertebral Disc Prolapse

When the nucleus pulposus invaginates through the annulus fibrosus the condition is known as prolapsed intervertebral disc. In milder forms this results only in a protrusion. But in severe forms it extrudes into the spinal canal and later gets sequestrated. ***For a prolapse to occur there should be a breach in the annulus.***

A breach in the annulus can develop as a result of:

a. *Severe and sudden trauma, e.g. lifting very heavy loads:* Bending and torsional forces of lifting heavy loads causes sudden increase pressure within the disc and ruptures the annulus (Figs 11.2A and B).



Figures 11.1A to C

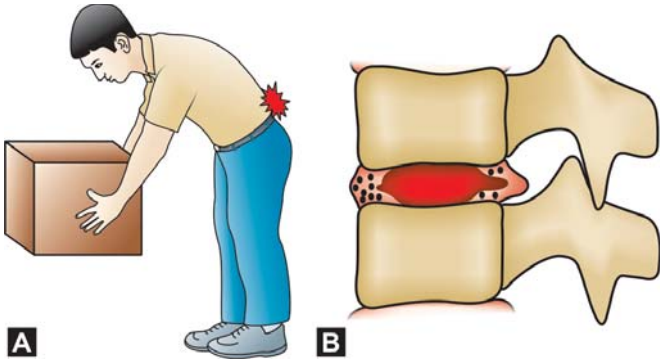
Showing diagrammatic representation of the basic structure of an intervertebral disc.

b. *Trivial trauma like coughing and sneezing:* Trivial trauma causes prolapse in a disc which is already weakened by degenerative changes both in the annulus fibrosus and nucleus pulposus as a result of repetitive stress (wear and tear fatigue) and advancing age.

The different ways in which a prolapse takes place is as follows (Figs 11.3A to C):

a. Protrusion
b. Extrusion
c. Sequestration

} Prolapse



Figures 11.2A and B

Showing the diagrammatic representation of the pathomechanics of disc prolapse: (A) Disc subjected to sudden trauma of lifting heavy load, (B) Degenerative disc is more prone for prolapse when subjected to such stress.

In lumbar region L4–L5 is the most common site for disc prolapse followed by L5–S1. These are the areas prone for maximum concentration of stresses during daily activities related to loading of the spine.

In the cervical spine most of the disc prolapses are degenerative. Purely traumatic disc prolapse is rare without spinal column injury. C5, C6 and C6, C7 are the common sites.

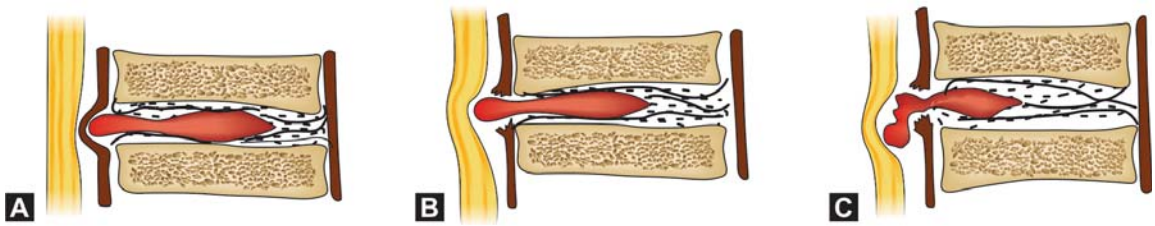
A prolapsed disc occupies various positions in relation to the nerve root and compresses the root.

a. It may remain central.
b. It may remain medial to the root.
c. It may remain lateral to the root.
d. It may remain intraforaminal (rare).

Depending on the position the symptoms vary. Sometimes disc prolapse can occur at multiple levels.

Diagnosis

Clinical diagnosis of intervertebral disc prolapse is based on the symptoms and deficits caused by the compression of the nerve roots.



Figures 11.3A to C

Diagrams showing the stages of disc herniation, namely protrusion, extrusion and sequestration. Note that the compression on the spinal cord is more with extrusion and sequestration. So also the indication for surgery.

1. **When there is no neurological involvement:** It manifests with acute onset of severe low backache and paraspinal spasm. Movements of the spine also may be restricted and associated with pain. Patient may have difficulty in walking too.
2. **When there is neurological involvement:** Along with pain and limitation of movement, there is neurological deficit. Derivation is almost mathematical and corresponds to the particular nerve root affected. Hence a thorough knowledge of the dermatomes and root values of the nerve supply to the muscles is necessary not only to make a clinical diagnosis but also to ascertain the of the level of the lesion.

In cauda equina lesions due to central herniation of the disc, presentation is classic. Presents with leg pain, perianal anesthesia,weakness of the anal sphincter and urinary retention.

Tables 11.1 and 11.2 depict methodically the clinical manifestations of a disc prolapse. Figure 11.4 gives the pictorial representation of different examination findings in a lumbar disc prolapse.

Presence of the List or Tilt of the Spinal Column

When the extruded disc is medial to the nerve root and compresses the nerve root from its medial side causing irritation and pain, in order to relieve this compression, the spine tilts laterally on the same side. This posture prevents the impingement and relieves the pain. If the extruded disc is lateral to the nerve root and compresses the nerve root from its lateral side the spinal column tilts to the opposite side in order to prevent the impingement. So the tilt of the spinal column indicates the position of the disc in relation to its nerve root. The presence of a tilt to the same side as that of symptoms indicates that the compression is occurring medial to the nerve root and the presence of tilt to the opposite side as that of the symptoms indicates that the compression is occurring lateral to the nerve root (Fig. 11.5).

Straight Leg Raising Test and Well Leg Raising Test

The straight leg raising test (SLRT) is also known as the Lasegue's test (1864).

The lower limb on the affected side is raised with the hip in flexion and knee in extension (i.e. with the leg in straight position). This results in pain in the hip region, radiating down to the leg along the course of the sciatic nerve.

The opposite lower limb when raised in a similar manner, produces pain on the involved side. This is known as well leg raising test or the Fajersztajn's test (1901). The phenomenon is known as crossed sciatic phenomenon or sciatic paradox phenomenon.

The symptoms can be exaggerated by dorsiflexing the ankle or by asking the patient to flex the neck. These maneuvers stretch the nerve root further.

The above tests are based on the principle of stretching the lumbar and sacral nerve roots. Cadaveric experiments have demonstrated that the nerve roots start moving towards the foramina with as low as 30° flexion movement of the hip. In a normal person the range of excursion of the nerve roots is about 2-5 mm. In disc prolapse when a leg raising test is

Table 11.1

Clinical features of cervical disc lesions

Levels of cervical disc lesion and features	*C-4, C-5 disc with C-5 root compression*	*C-5, C-6 disc with C-6 root compression*	*C-6, C-7 disc with C-7 root compression*	*C-7, T-1 disc with C-8 root compression*	*T-1, T-2 disc with T-1 root compression*
Sensory changes (Deficit)	Lateral aspect of upper arm and elbow	Lateral aspect of the forearm thumb and the index finger	Usually middle finger but variable due to cross over	Ring and little finger and ulnar border of the hand	Medial aspect of the elbow
Motor changes (Weakness)	Deltoid and biceps brachii	Biceps brachii, extensor carpi radialis longus and brevis	Triceps, flexor carpi radialis, flexor digitorum superficialis and profundus	Interossei and the finger flexors, flexor carpi ulnaris	Interossei
Reflexes	Absence of or sluggish biceps reflex	Absence or sluggish biceps and brachioradialis reflex	Absence or sluggish triceps reflex	No change	No change

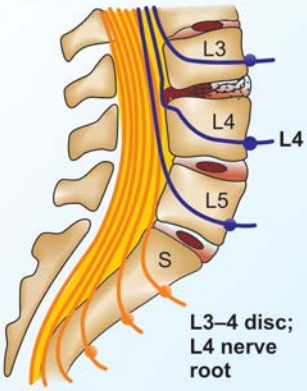
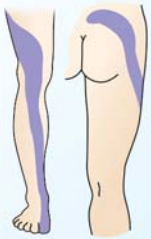
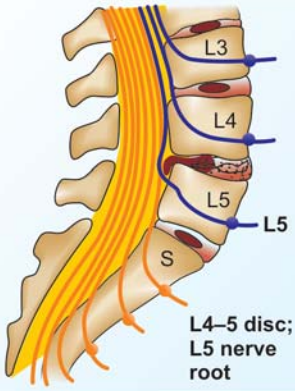
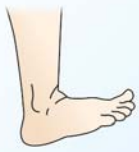
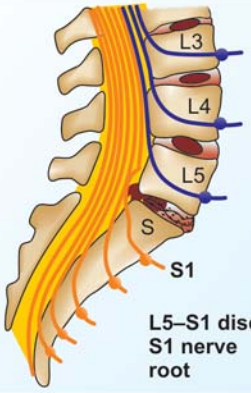
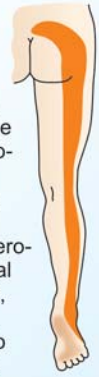
Clinical features					
Level of herniation	**Pain**	**Numbness**	**Weakness**	**Atrophy**	**Reflexes**
 **L3–4 disc; L4 nerve root**	 Lower back, hip posterolateral thigh, anterior leg	 Anteromedial thigh, knee and leg	 Quadriceps and knee extension	 Quadriceps	 Knee jerk diminished
 **L4–5 disc; L5 nerve root**	 Just above sacro-iliac joint, hip, lateral thigh, and leg	 Along lateral leg and first 3 toes	 Dorsiflexion of great toe and foot; difficulty in walking on heels; foot drop may occur	Not prominent	No change in knee and ankle jerk, Diminished or absent Tibialis posterior reflex
 **L5–S1 disc; S1 nerve root**	 Just above sacro-iliac joint, hip, postero-lateral thigh, and leg to heel	 Along back of the calf, lateral heel, foot and toe	Plantar flexion of foot and great toe and difficulty in walking on toes	 Gastrocnemius and soleus	 Ankle jerk diminished or absent

Figure 11.4

A pictorial diagram showing the deficits observed at examination in intervertebral disc prolapse at various levels.

performed, this excursion takes place over the protruded disc thereby causing irritation of the root and pain (Fig. 11.6). In a positive well leg raising test which is positive in about one-third of the cases of intervertebral disc prolapse, it has been found that the protruded disc is in the axilla or medial to the opposite root, and never lateral.

Table 11.2

Clinical features of lumbar disc lesions

Levels of the lumbar disc lesions and features	*Ruptured disc between L-3 and L-4 with L4 root compression (L-4 component of femoral and sciatic nerve)*	*Ruptured disc between L-4 and L-5 with L-5 root compression (L-5 component of the sciatic nerve)*	*Ruptured disc between L-5 and S-1 with S-1 root compression (S-1 component of the sciatic nerve)*
Pain	SI joint and hip region. Posterolateral aspect of the thigh. Anterior aspect of the leg	SI joint and hip region. Posterolateral aspect of the thigh and leg.	SI joint and hip region. Posterolateral aspect of the thigh, leg, and heel.
Numbness	Anteromedial aspect of the leg.	Lateral aspect of the leg or dorsum of the foot even over great toe.	Lateral aspect of the leg foot and lateral three toes.
Weakness	Extension of the knee.	Dorsiflexion of great toe and occasionally foot.	Not common. Occasionally plantar flexion of foot and great toe.
Sensory changes (Deficit)	Posterolateral aspect of the thigh, anterior aspect of knee and medial aspect of the leg.	Anterolateral aspect of the leg, dorsum of the foot and great toe.	Lateral malleolus, lateral aspect of the foot and heel and dorsum of lateral one and a half toes including web.
Motor changes (Weakness)	In quadriceps and hip adductors	In extensor hallucis longus, extensor digitorum longus and brevis and at the hip in gluteus medius.	In Gastrocnemius and soleus, peroneus longus and brevis and at the hip gluteus maximus.
Reflexes	Sluggish or absent knee jerk	No change both in knee and ankle jerk (or sometimes sluggish reflex). Never absent. Tibialis posetrior reflex may be sluggish/ absent.	Sluggish or absent ankle jerk.

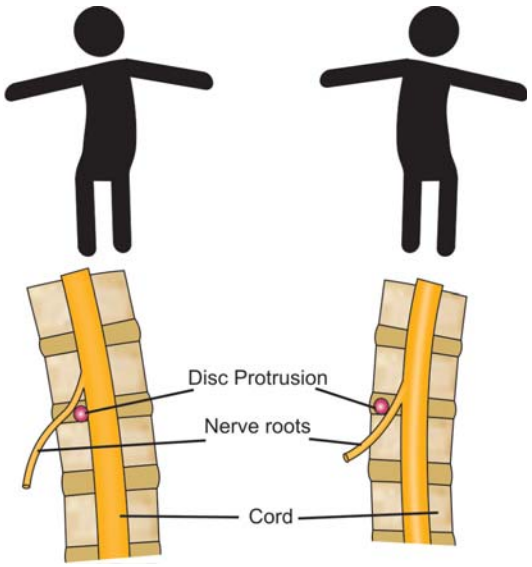
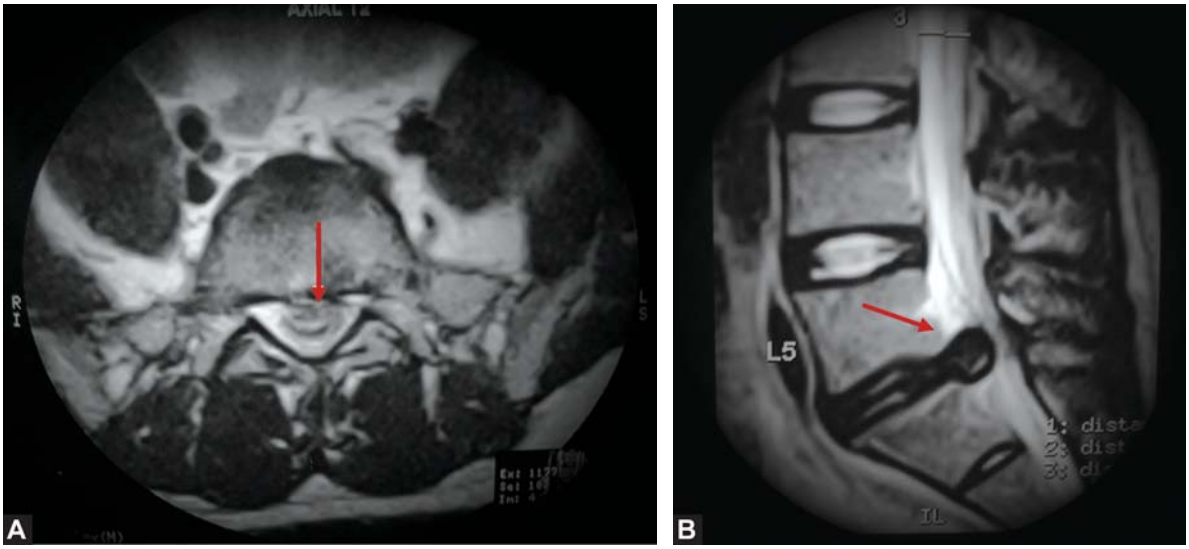


Figure 11.5

Diagram showing the medial and lateral position of the extruded disc in relation to the nerve root.To avoid irritation of the nerve root the spine tilts accordingly. To the same side when the extrusion is medial to the root and to the opposite side when the extrusion is lateral to the root. The patient develops a list.

Figure 11.6

Photograph showing the straight leg raising test being performed. In this it is negative and the leg can be raised up to 80°.



Figures 11.7A and B

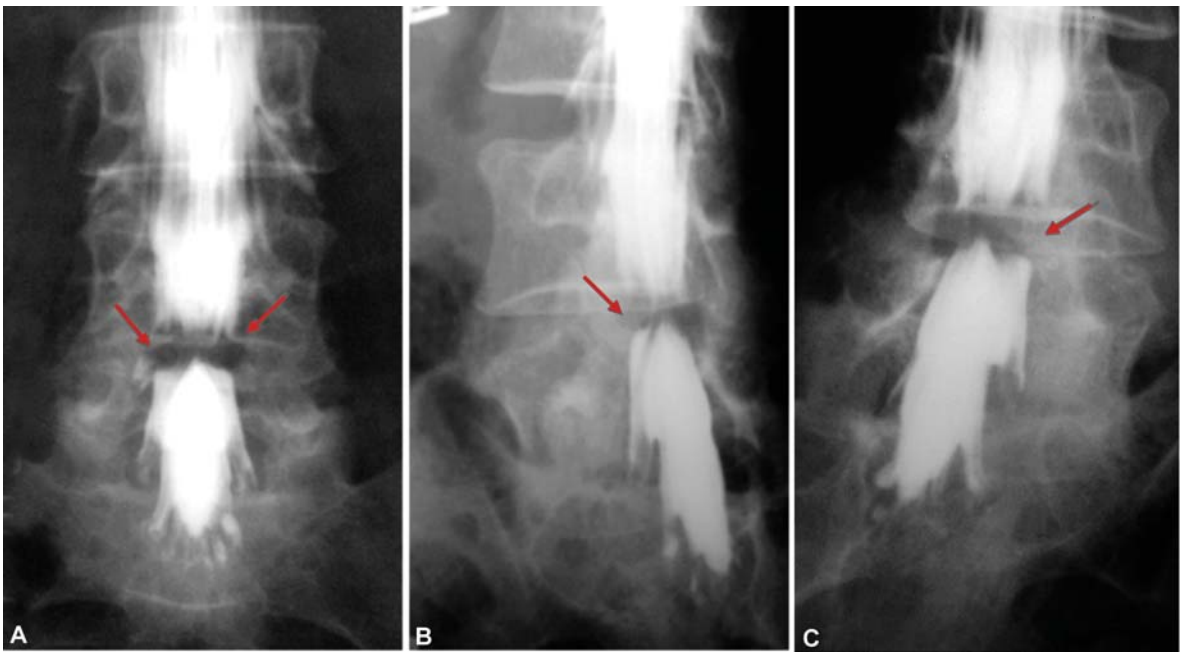
MRI showing prolapsed and extruded intervertebral disc at L5 S1 compressing the S1 root.

Investigations

X-ray may show loss of normal lordosis, presence of scoliosis and in cases of degenerative disc shows changes of degeneration with osteophyte formation in the spinal column.

MRI is the investigation of choice. It clearly shows the nature and extent of prolapse and the site of nerve root compression (Figs 11.7A and B).

Myelogram was the standard investigation for intervertebral disc prolapse before MRI was invented. Because of its invasiveness, the risks of spinal puncture and the side effects and complications of the dye used it is given up these days. The picture of myelogram is used here (instead of diagrammatic representation) to show the exact nature of nerve root involvement in IVDP (Figs 11.8A to C).



Figures 11.8A to C

Myelogram showing a midline disc herniation L4- L5 with bilateral L5 root cut off.

Treatment

The intervertebral disc prolapse can be treated nonoperatively or operatively. The decision is always taken in consideration with clinical features and MRI findings.

Nonoperative Management

Is indicated in those cases with no or minimal neurological symptoms and MRI shows only protrusion and not extrusion. Following are the measures taken.

a. Bed rest and immobilization in traction.
b. Anti-inflammatory drugs.
c. Lumbosacral corsets for support during the period of gradual mobilization.
d. Spinal strengthening exercises to follow.
e. Advise to abstain from such activities which impart loading stress on the spine, e.g. lifting heavy weights, uncomfortable travel, poor posture at work, etc.

Operative Management

It is indicated when the symptoms and neurological deficits are severe and MRI shows extrusion or sequestration of the disc. The more earlier is the decompression of the root, the faster is the relief and better is the recovery.

Procedures

a. Laminectomy and discectomy.
b. Hemilaminectomy and discectomy.
c. Fenestration/laminotomy and microdiscectomy.
d. Endoscopic discectomy.

Of the above procedures indications for (a) and (b) procedures are few. More commonly (c) and (d) procedures are preferred and done routinely because they are less invasive.

Chemonucleolysis: In this procedure the protruded *disc* which is symptomatic is *dissolved by injecting the enzyme chymopapain* derived from the papaya into the disc space. The technique needs expertise and skill. Also in USA specific training and board certification is mandatory. *On table discogram* is a must. If the *dye leaks out into the epidural space the procedure is abandoned.* The procedure is *also abandoned* if there is *dural puncture and spinal fluid leak.*

Anaphylaxis due to allergy for the enzyme and *late development of transverse myelitis* and its neurologic sequel are the risks involved. The procedure is rarely done these days.

Artificial disc replacement: Artificial disc replacement is being done in those patients with degenerated discs who have disabling back pain, who have not responded to nonoperative line of management and in whom arthrodesis is the option. The artificial disc is designed with ultrahigh molecular polyethylene core, sandwiched between cobalt chromium endplates and aims at restoring pain free movement at that level.. Centers where these surgeries are being done have claimed successful results in the range of 60-90% at 7 yr follow-up.

Problem of degeneration of posterior elements and facetal joints, the polyethylene wear and the effect of its debris on the spine and the cord and the salvage procedures adopted when the implant fails, are some of the problems which are being closely studied at this point of time.

Revision Questions

Q. Write notes on:
   a. SLRT
   b. Well leg raising test.
   c. List
   d. Sciatic scoliosis
   e. Disc prolapse.

Q. Write briefly on
   a. Laminectomy
   b. Hemilaminectomy
   c. Fenestration
   d. Microlumbar discectomy.

Essay Question

Discuss the etiopathogenesis, clinical features, diagnosis and management of a case of acute Intervertebral disc prolapse. Enumerate its complications.

Further Reading

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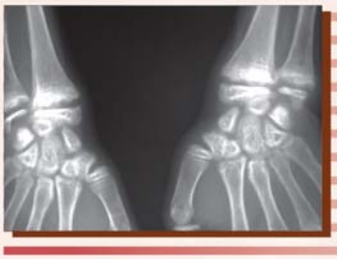


12

Nutritional, Endocrine, Degenerative and Autoimmune Disorders

- *Rickets and Osteomalacia*
- *Hyperparathyroidism*
- *Osteoporosis*
- *Osteoarthritis*
- *Rheumatoid Arthritis*

Rickets and Osteomalacia

Rickets and osteomalacia is an abnormality resulting in lack of mineralization of the bone due to deficiency of vitamin D. Vitamin D is the name given for a group of sterol molecules. The two basic forms of vitamin D are vitamin *D2 otherwise known as ergocalciferol* derived from plants and *D3 otherwise known as cholecalciferol* derived from animals, e.g. egg yolk, cod liver oil, etc. Cholecalciferol is also synthesized in the skin of animals from 7 dehydrocholesterol by exposure to sunlight. Ergocalciferol is synthesized exogenously from ergosterol by exposure to ultraviolet rays of sunlight. These *two forms of vitamin D, namely D2 and D3 are converted into biologically active forms of vitamin D by a process of hydroxylation which occurs first in the liver and then in the kidneys, respectively.*

In the liver the enzyme 25-hydoxylase converts the cholecalciferol into 25 hydroxycholecalciferol.

In the kidneys the enzyme 1-alpha hydroxylase converts the 25-hydroxycholecalciferol into 1,25-dihydroxy cholecalciferol (Fig. 12.1).

These are active forms of vitamin D which are important for the regulation of calcium and phosphororus levels in the blood and for mineralization of bone.

Biologically active vitamin D is fat-soluble and is transported through the blood stream by the help of a binding

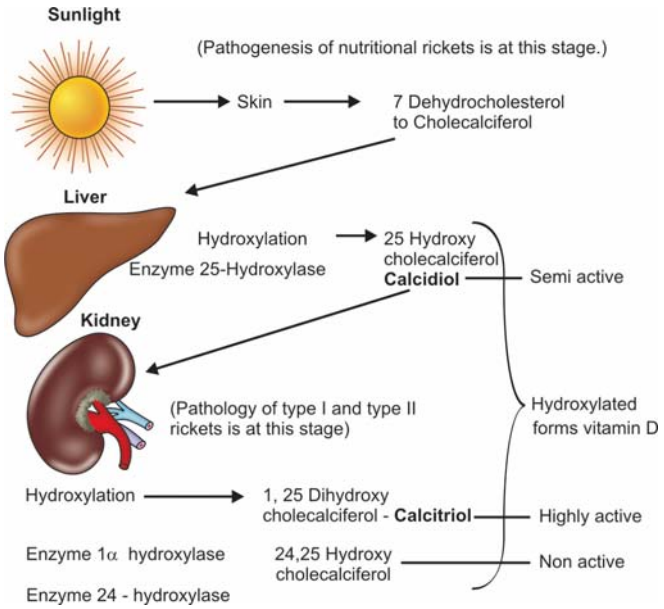


Figure 12.1
Picture showing the pathogenesis of various types of rickets.

protein.1,25 dihydroxy vitamin D has a half-life of a few hours. Whereas 25, hydroxy vitamin D has a half-life of several weeks. Thus the *liver acts as a store house of vitamin D. It stores the vitamin D synthesized in the skin and absorbed from the intestine, in 25,hydroxylated form and releases it into the bloodstream.* This form of vitamin D is semiactive with a long half-life. It is provided to the kidneys for 2nd hydroxylation process as per need. In the kidney it gets converted into the highly active form, i.e. 1,25 dihydroxy vitamin D (Calcitriol) which is responsible for the regulation of calcium and phosphorous level. *Once the calcium and phosphorus level is regulated the formation of 1,25 dihydroxy cholecalciferol stops and formation of nonactive 24,25, hydroxyl cholecalciferol begins. Thus calcitriol has an autoregulatory influence on the kidneys.*

(Cod liver oil, Shark liver oil, Halibut liver oil are some of the richest sources of vitamin D. The polar bear liver is the richest source of vitamin D among animals. It is so toxic that ingestion results in death in humans. Hence Eskimos do not eat the liver of the polar bear. The toxic property of vitamin D is made use of in preparing rodenticide. Baits smeared with vitamin D are available as rodenticide.)

Thus in short for the vitamin D to become active, two hydroxylation processes are necessary. The first one taking place in the liver and the second one in the kidney.

Etiology and Nature of Rickets

A. *Nutritional:* Dietary deficiency of Vitamin D, calcium and phosphorus or intestinal malabsorption of calcium results in this form of rickets. The condition is reversible when attention is given to the diet. Not sex-linked and no inheritance is observed.

B. *Lack of exposure to sunlight.*

C. *Genetic causes*: Vitamin D-dependant rickets

   a. Type I—In this type there is *deficiency of the enzyme 1-alpha hydroxylase* in the kidneys.
   Thus *deficiency of 1,25 dihyroxy vitamin D* (Calcitriol) *develops.* Autosomal recessive inheritance is observed. It is also known as pseudovitamin D deficiency rickets. Supplementation with active form of vitamin D, i.e. Calcitriol results in cure.

   b. Type II—In this type the *production of 1,25 dihydroxy vitamin D is normal.* But the *receptor is abnormal.* Hence there is defective interaction between the vitamin D and the receptor *resulting in resistance.* Increased level of Calcitriol in the blood helps in the diagnosis. Autosomal recessive inheritance is seen. Large doses of vitamin D is necessary for control. It cannot be cured.
   Vitamin D resistant rickets: (Hypophosphatemic in nature, also was known as phosphate diabetes)

      i. Familial—In this variety there is renal tubular abnormality which leads to decreased re-absorption of phosphates. This results in *phosphaturia and hypophosphatemia.* X-linked dominant inheritance is observed. *Calcitriol levels are low.* Hence, need supplementation of both, Calcitriol and phosphates.

      ii. Hereditary—In this variety there is *hypercalciuria, phosphaturia and hypophosphatemia* because of decreased renal tubular re absorption of phosphates. But the serum levels of *Calcitriol is normal.* Hence needs supplementation of phosphorous only. Autosomal dominant/ recessive inheritance is seen.

D. Chronic renal and liver diseases.
Renal osteodystrophy, cirrhosis, etc.

E. Others
Oncogenic, drug induced (phenytoin, antacids containing aluminum), etc.

(It is perhaps ideal to consider type II rickets as true vitamin D resistant rickets and vitamin D resistant rickets as mentioned above as hypophosphatemic rickets).

Differences between nutritional rickets and vitamin D resistant rickets

Nutritional	*Vitamin D resistant*
Acquired	Inherited
Responds to adequate doses of vitamin D	Responds only to large doses of vitamin D. Supplementation of phosphorous may be necessary
Spontaneous resolution of deformities is possible with vitamin D	Spontaneous resolution of deformities is not possible even with large doses of vitamin D
Growth becomes normal on healing	Growth never becomes normal Always remains stunted.
Serum chemistry becomes normal following vitamin D administration.	Serum chemistry remains abnormal even after administration of large amounts of vitamin D

Pathology

Pathology is defective mineralization. *In children both,* the growth plate and bone get affected. Hence skeletal changes of both *rickets and osteomalacia develop.* Whereas *in adults* vitamin D deficiency causes changes of *osteomalacia only because the growth plate is already fused.*

In the growth plate the stages of bone formation proceeds normally up to the stage of appearence of mature chondroblasts leading to chondrocyte formation and laying down of cartilaginous matrix. At this stage, the process of calcification is deficient. Hence, poor mineralized osteoid formation occurs in the physis which results in accumulation of nonossified cartilage. This leads to the classical radiological picture of widening of the physis and cupping of the metaphysis.

In the diaphyseal bone in children as well as in adults the mineralization of the osteoid does not take place. Hence osteomalacic changes develop.

Thus the bones become soft because of lack of mineralization and bend. Pathological fractures too can develop. This results in classical deformities, i.e. bow legs, knock knees, wind swipe deformity, etc.

Clinical Features

Rickets

Child may present with following features (Fig. 12.2):

General

- Irritable child.
- Lethargy and hypotonia.
- Pot belly.
- Delayed mile stones.
- Stunted growth.

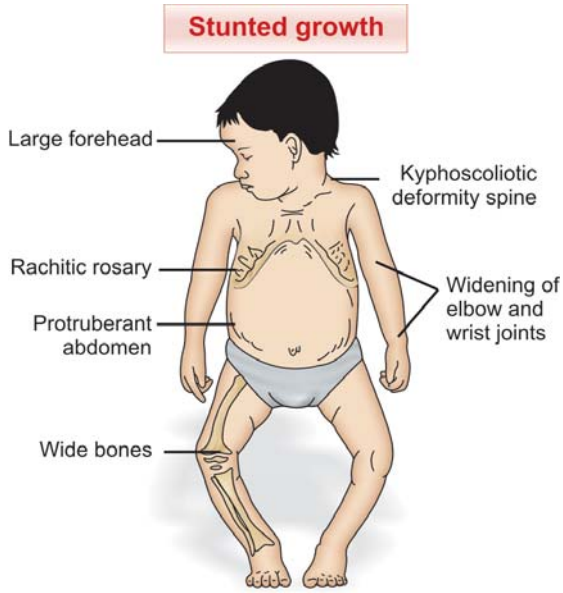


Figure 12.2
Picture of a child showing features of ricket.

Specific

- Craniotabes (soft skull)
- Bossing of the skull
- Pigeon chest.
- Rachitic rosary/rickety rosary (Costochondral beading)
- Harrisons' groove—is a line observed at the place where the diaphragm attaches to the ribs.
- Bowing or widening of the physis.
- Deformities in the lower limbs, e.g. genu valgum, genu varum, wind swipe deformity (Figs 12.3B and C).
- Deformities in the spine—kyphoscoliosis.
- Radiologically, osteopenia with widening of the epiphysis and cupping of the metaphysis (Figs 12.4A to F).
- Pathological fractures due to softening of the bones.

Osteomalacia

- Nonspecific features in early stages.
- Bone pain may be the presenting feature.
- Muscular weakness and easy fatigue.
- Carpopedal spasm and facial twitching.
- Pathological fractures (commonly seen in the spine).

Investigations

Investigations are done to confirm the type of rickets so that adequate treatment is instituted.

Nutritional Rickets

a. Serum calcium, phosphorous and alkaline phosphatase. Serum calcium and phosphorous is low alkaline phosphatase is elevated.
b. Urinary calcium is low.
c. Serum calcidiol and calcitriol is low.
d. PTH is elevated.
e. Urinary phosphorous is elevated.

Vitamin D Resistant Rickets (Type-II)

a. Urinary calcium may increase.
b. Serum calcitriol levels may show an increase.

X-rays Changes in Rickets

Classical changes of *widening of the physis and cupping of the metaphysis* is seen. These changes are more pronounced in the region of the knee and the wrist. Further, rarefaction of the bone is observed confirming *osteomalacic changes.*

Skull may appear square, known as *Caput Quadratum.*

X-rays Changes in Osteomalcia

- The skeleton looks thinned out and casts a poor radiological image.
- Looser's zones may be seen. These are areas of pseudo-fractures occurring at the sites of stress. Pubic rami, medial cortex of neck of femur, axillary border of the scapula are some of the sites.These are the result of increased rate of bone resorption and poor new bone deposition.
- Protusio acetabuli may be seen in the pelvis.

Treatment

Nutritional Rickets

a. Adequate exposure to sunlight (not excessive exposure)
b. Supplementation of vitamin D in the diet. (Recommended even for breast-fed babies after two months of age)

} Preventive and curative measure

c. Treatment with therapeutic doses of vitamin D 200-600 units/day or as a single intramuscular inj of 600,000 IU. May have to be repeated at 4-6 weekly intervals.

Radiological sign of healing: A sclerotic line developing in the metaphyseal region (Figs 12.5A to C).

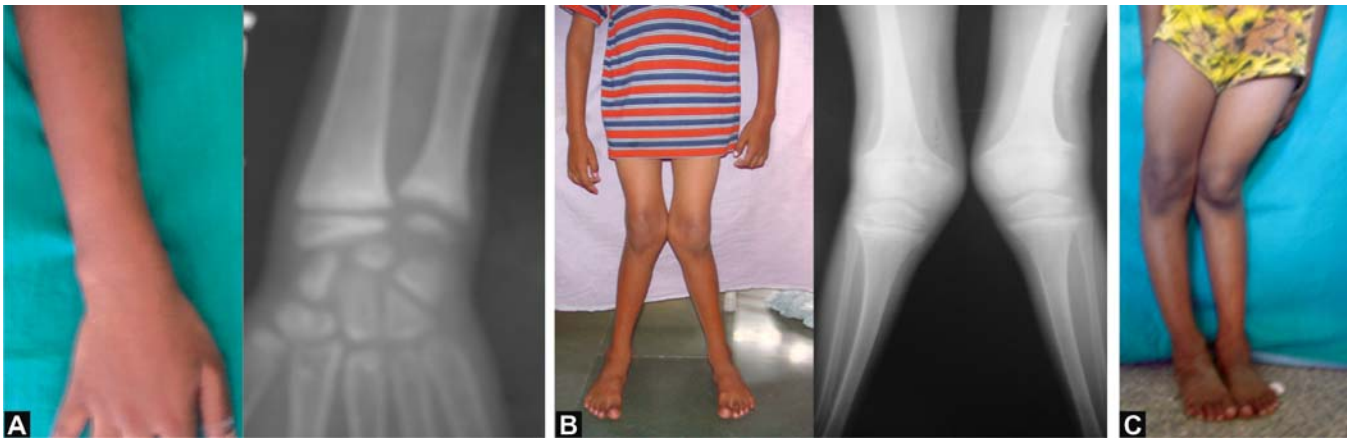
In refractory rickets this does not occur with normal doses of vitamin D.

Familial and Hereditary Rickets

These are refractory to usual treatment because of genetic abnormality. Depending on the cause as discussed in the etiology, the treatment varies. Large doses of vitamin D and supplementation of phosphorous may be necessary and the treatment may last life long.

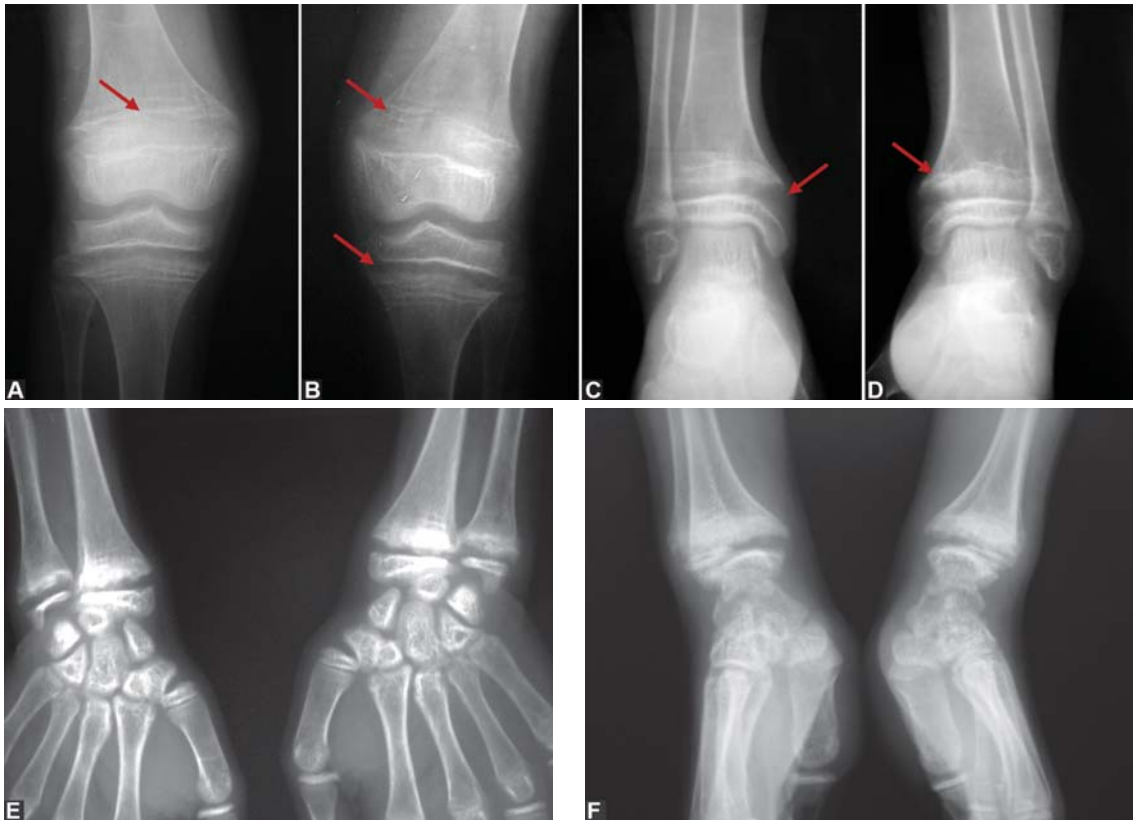
Correction of Deformities in Rickets (Figs 12.6A and B)

In young the deformities get corrected with adequate supplementation of vitamin D and splinting. Deformities which are



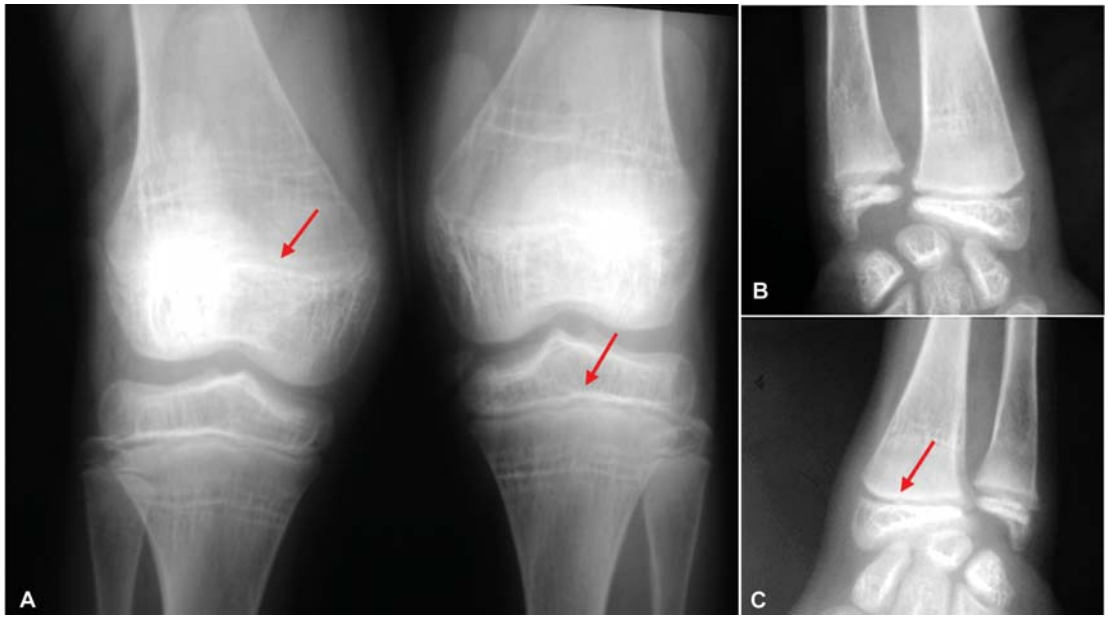
Figures 12.3A to C

Showing cupping of the metaphysis, widening of the physis and Genu valgum and Wind swipe deformities. (A) Trumpetting of the metaphysis; (B) Knock knee (C) Wind swipe deformity: Bow leg on one side and knock knee on the other.



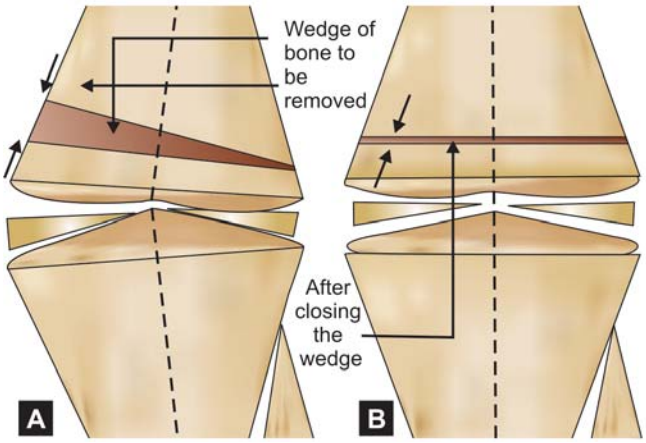
Figures 12.4A to F

Radiographs showing classical changes of rickets at the ends of long bones. Note the presence of Harris growth lines in the metaphyseal portion of the bone which indicate intermittent growth retardation. These lines are named after Henry Harris a Welsh anatomist. They are seen in children when temporary growth retardation takes place.



Figures 12.5A to C

Radiographs of the same patient shown in Figures 12.4A to F after adequate vitamin D administration. Complete healing is seen as evidenced by the presence of sclerosis and physeal width becoming normal. Note the presence of Harris growth lines.



Figures 12.6A and B

Diagram showing Mac Evans medial closed wedge osteotomy for the correction of genu valgum. Note the angle of valgus deformity. This angle decreases after taking an appropriate wedge of bone with the base directed medially. The defect is closed after removing the wedge of bone. The size of the wedge depends on the extent of correction required.

of severe nature and are established need correction. Correction is always done when the serum parameters become normal after treatment with vitamin D.

The procedure of corrective osteotomy is undertaken to correct the deformities, e.g. Mac Evan's osteotomy (medial close wedge osteotomy) for genu valgum.

Revision Questions

Q. Write notes on:
- a. Active forms of vitamin D
- b. Actions of vitamin D
- c. Rickets
- d. Osteomalacia
- e. Radiological picture of rickets
- f. Vitamin D resistant rickets.

Essay Questions

Q. Classify rickets. Discuss the clinical features, diagnosis and management of different type of rickets.

Osteoporosis

Osteoporosis may be defined as a generalized disorder involving the skeleton in which the bone density decreases, the fragility of bone increases and, as a result, the bone becomes more susceptible for fractures.

Types

a. Postmenopausal (Estrogen lack)
b Senile (Natural process)

Risk Factors

a. Advancing age.
   Women >45 yr (after menopause); Men > 60 yr.
b. Low calcium and vitamin D in the diet.
c. Lack of exercise.
d. Smoking.
e. Drug-induced, e.g. long-term steroids.

Pathology

The strength of the bone depends on proteins, collagen and calcium. This inherent strength is acquired genetically *in utero* itself influenced by nutritional, hormonal and environmental factors. The influence of these factors on bone mass continues through out life.

Bone is a dynamic structure. Throughout life there is a continuous process of new bone deposition and bone resorption and remodeling. In the normal course the deposition and resorption of bone balance each other and is responsible for maintaining the standard quality of the bone. When the resorption of bone exceeds that of deposition thinning of bone occurs and osteoporosis develops, i.e. osteoclastic activity > osteoblastic ativity. The osteoclastic activity is regulated by RANK ligand and the osteoblastic activity is regulated by osteoprotegerin (OPG). RANK and OPG have opposite effects on the bone.

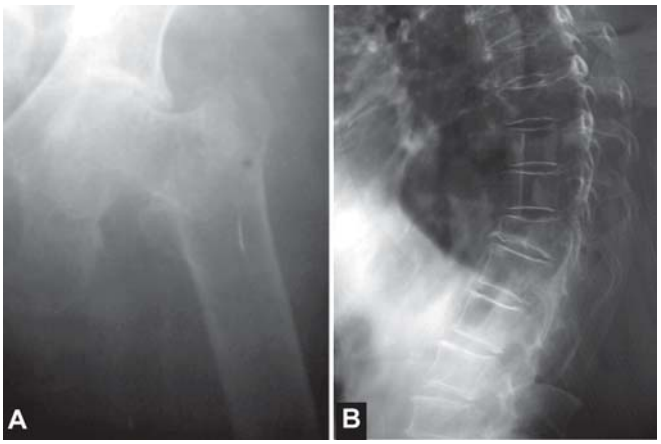
Clinical Features

Osteoporosis does not give rise to any clinical symptoms unless a fracture develops (Fig. 12.7A) when a patient starts complaining of pain and inability to move the part. Generally the injury is very trivial. The fracture can be incomplete or complete. In the spine compression fracture is common and patient presents with backache and radiating pain. If the fracture involves the pelvic bone, the patient complains of pain in the groin and difficulty in walking.

Investigations

X-ray shows thinning of the the bony trabeculae and generalized rarefaction.

In the spinal column, osteoporotic compression fractures may be seen in the vertebra (Fig. 12.7).



Figures 12.7A and B

Showing a fracture in the intertrochanteric region and a wedge compression of T-12 vertebra due to osteoporosis. The radiographs show marked thinning of both cortical and cancellous bones.

Bone densitometry by DEXA (Dual energy X-ray absorptiometry) is more precise and can detect osteoporosis early.

Treatment

Drug Therapy

It is to be instituted when risk factors exist, especially after 45 yr of age, after confirming the diagnosis. *Aim is to prevent osteoporotic fractures.*

Two basic types of drugs are used to treat osteoporosis.

a. Drugs that inhibit the osteoclast-induced bone resorption, e.g. Residronate.
b. Drugs that promote osteoblastic induction of bone, e.g. Teriparatide.

Along with these drugs calcium and vitamin D are also given in adequate doses.

Recent drug is a stable strontium compound. Strontium renolate which has dual action of increasing bone synthesis and decreasing bone resorption. Monoclonal antibodies that regulate osteoclasts at cytokine level are also being developed by recombinant technique. Hence in future there may be two more additional drugs—dual acting bone agents (DABA) and cytokines.

Treatment of Fractures

- Vertebral fractures involving *single vertebra* can be managed by *vertebroplasty* using injection of bone cement. This is followed by a protective brace at least for three months.

  During this period it is advisable that the patient does not lift any heavy object.
- *Long bone fractures* are really problematic to treat. They have to be treated by reduction and *internal fixation* followed by protective immobilization. In severely *comminuted* osteoporotic fractures *bone grafting may be necessary.*
- Appropriate drug therapy is a must.

Essay Question

Discuss the etiopathogenesis, clinical features, diagnosis and management of senile osteoporosis. What are the complications of senile osteoporosis.

Osteoarthritis

Degenerative arthritis, hypertrophic arthritis, chondromalacic arthrosis and osteoarthrosis are the other names for osteoarthritis. The condition is the result of degenerative changes taking place in the joint. The articular cartilage and the subchondral bone progressively degenerates. This results in inflammation and mechanical derangement which promotes further degeneration. Ultimately results in badly deranged joint, associated with deformity and ligamentous laxity.

Types

a. Primary osteoarthritis is the type where there is no preexisting abnormality or damage (incongruity) as a cause for progressive degeneration.
b. Secondary osteoarthritis is the type where there is a preexisting pathology or damage (incongruity) which is responsible for the progressive degeneration.

The basic difference in joint destruction occurring in Rheumatoid arthritis and Osteoarthritis is that, in rheumatoid arthritis, inflammation occurs first. The joint destruction that follows is the result of inflammation. Whereas in Osteoarthritis, the joint destruction occurs first and the inflammation that follows is secondary to joint destruction.

Etiology and Pathogenesis (Fig. 12.8)

Exact etiology is not yet diagnosed. But certain factors predispose the development of osteoarthritis.

a. *Trauma:* It is the minor degrees of trauma of repetitive nature occurring during activities of daily living which causes osteoarthritis. There is protective mechanism operating in the human body when it is subjected to an

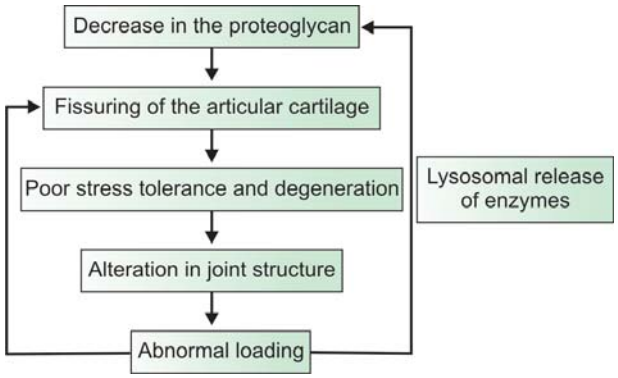


Figure 12.8
Vicious cycle of idiopathic degeneration in primary osteoarthritis.

unprepared stress. For this reflex protective mechanism to operate it takes a few milliseconds, e.g. when there is a slip. In this the injury is avoided if the reflex protective mechanism operates by contraction of different muscles which in turn averts the fall. But it is those degrees of repetitive minor stress (trauma) occurring within those few milliseconds before the reflex protective mechanism could operate, that causes injury to the joint structures which (perhaps a microfracture) is thought to be responsible for most of the degenerative arthritis, e.g. osteoarthrtis of the finger joints occurring in musicians and typists, cervical spondylosis occurring in head load workers and sky divers of Acupulco in Mexico. (La Quebrada cliff where from ages sky divers dive into the sea from a height of about 45 meters). Osteoarthrosis of the knee is common among sailors and people who stand for long hours at work.

b. *Genetic factors*: Osteoarthritis is known to occur among many family members. It is especially common in identical twins. These factors support genetic predisposition.

Pathology

Primary

With advancing age the proteoglycan content of the articular cartilage decreases thereby reducing the water content of the cartilage. Such a cartilage becomes less resilient and cracks during activities of daily living. This is responsible for the onset and progression of degenerative process. It starts innocuously in the beginning, progresses slowly over a period of time and then becomes severe.

Secondary

There is a joint incongruity which has developed due to some disease in the past. Thus degeneration occurs secondary to a pre-existing mechanical abnormality. This is secondary osteoarthrosis. (See Chapter 10 Figs 10.17A and B).

Clinical Features

a. *Pain* is the primary feature of osteoarthritis. It is gradual in onset and slowly progressing in severity. There can be pain free intervals too.

b. *Swelling and joint effusion.* Swelling in osteoarthritis is due to effusion. This is commonly seen early in the course disease and late in the course of the disease both swelling and deformity is seen.

c. *Limitation of movement.* Painful limitation of movement is a late feature of osteoarthritis. In the early phase the joint movements are associated with pain but not limted.

d. *Heberden's nodes and Bouchard's nodes.* These are nodular formation occurring in the DIP and PIP joints respectively in osteoarthrtis.

Diagnosis

It is not difficult. Clinical features and X-rays confirm the diagnosis.

Radiological Findings (Figs 12.9A to D)

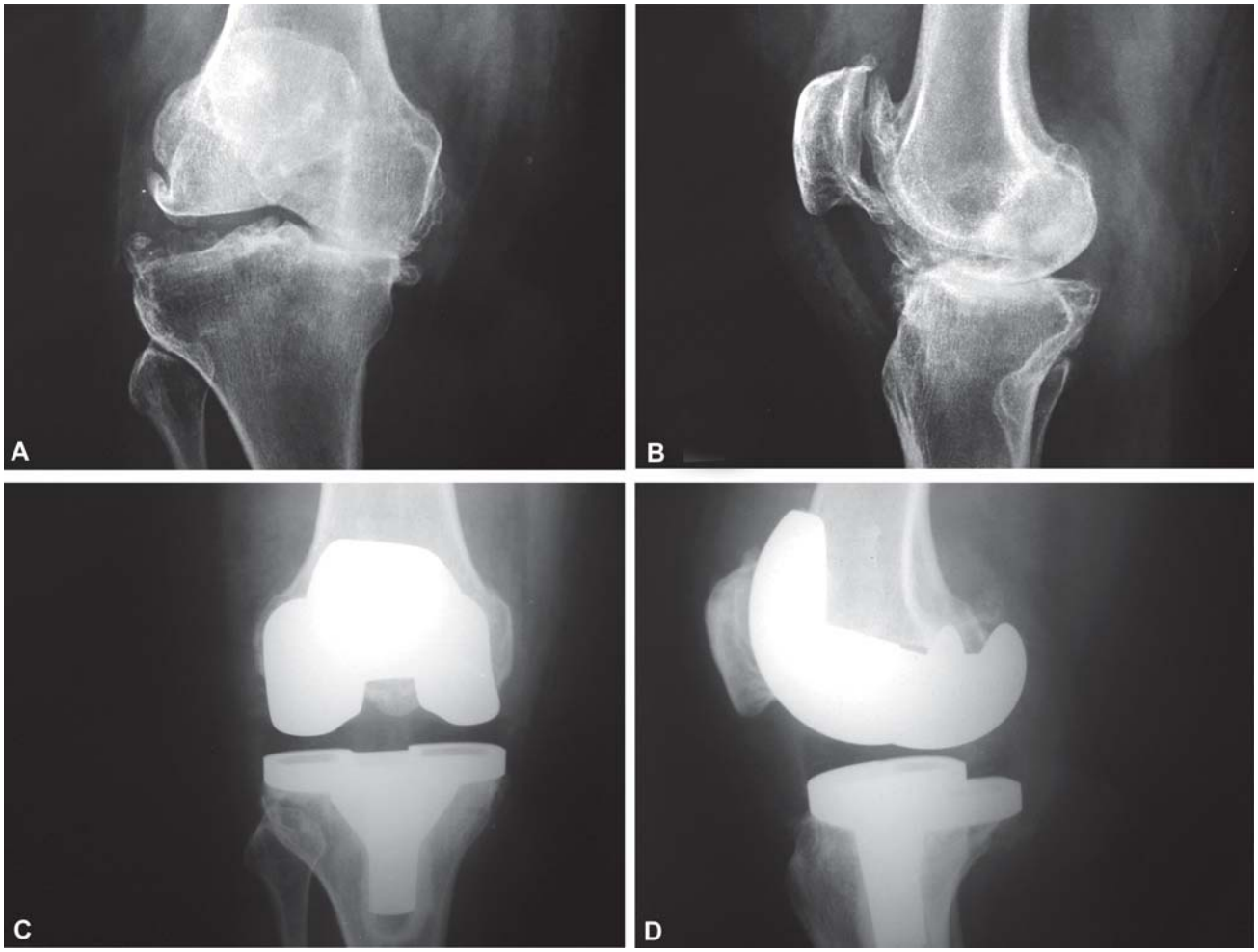
- Narrowing of the joint space (due to loss of articular cartilage.)
- Sclerosis of the subchondral bone.
- Subchondral cysts.
- Osteophytes (the bony excrescence or outgrowth, usually branched in shape)
- Deformities.

Management

Nonoperative Management

Early in the stage of the disease treatment aims at providing pain relief and slowing down the pace of progression of the disease by taking care of the offending factors if any, e.g. overweight and giving exercise therapy to strengthen the muscles. A good functioning muscle acts as a shock absorber and takes care of the stress which otherwise would have fallen maximally on the joint.

Nonsteroidal anti-inflammatory drugs take care of the pain and inflammation. Along with good physiotherapy gives considerable amount of pain relief.



Figures 12.9A to D

Radiographs of a 60 years old patient with severe osteoarthritis. Note the extensive degenerative changes suggestive of eburnation of the articular surface. Osteophytes are seen in plenty. Joint space is narrowed and there is evidence of instability as seen by opening up of the lateral joint space. This patient underwent total knee replacement and is totally symptom free.

Infiltration of hyaluronidase into the joint is known to give sustained temporary relief.

Surgical (Operative) Management

It is done late in the course of the disease when the joint is degenerated and clinically associated with pain and deformity. Total joint arthroplasty is the treatment of choice (Figs 12.9A to D).

In selective cases osteotomies can be done. Osteotomy alters the line of weight bearing and distribution of stress and is known to give relief, e.g. high tibial osteotomy.

Arthroscopic lavage and excision of loose bodies is done in high risk patients who are not fit for joint replacement surgery. This procedure gives temporary relief.

Essay Question

Q. Discuss the etiopathogenesis, clinical features, radiological features, diagnosis and management of osteoarthritis.

Rheumatoid Arthritis

Definition

It is an autoimmune systemic disorder wherein the body defence mechanism identifies the normal tissue as antigen and evokes a chain of inflammatory reaction resulting in destruction.

Etiology

Exact cause is not known. Following factors are considered as triggering factors.

a. Chronic infection—bacterial,fungal or viral.
b. Environmental factors—polluted atmosphere, smoking, etc.
c. Genetic factors—HLA with DR4 specificity is observed.
d. Endocrinal factors.
e. Poor quality food with constant assault on the gut.

Joints, Organs and Tissues Involved

Mainly involves the joints. Joints of the wrist, hand, knee, ankle and foot are commonly affected. Elbow, shoulder, hip and spine are not commonly affected. Monoarticular involvement is rare.

Involvement of soft tissue structures surrounding the joint too occurs, e.g the synovium, capsule, tendons, muscles, etc.

Lungs, pericardium, eyes, vessels and nerves are the other structures that can get involved.

- Age 20-40 yr.
- M:F ratio is 3:1

Pathology

The disease process starts in the synovium as synovitis with infiltration by inflammatory cells; to begin with monocytes infiltrate the synovium. This is later followed by lymphocytes. As a result the synovium gets thickened. An effusion into the joint develops and as the disease progresses, the cytokines released in the process, e.g. TNF α, interleukin-1 and interleukin-6, histamine, etc destroy the articular cartilage, the bone, the capsule, the ligaments, the tendons and the muscles. The destruction occurs by the formation of a pannus of tissue at the junction of the synovium and the articular cartilage. This pannus erodes the cartilage and the bone and destroys it. Thus arthritis develops.

Clinical Features

- Systemic features start insidiously with low grade fever, malaise, fatigue and joint pains.
- The joints are swollen edematous, warm, tender with effusion and the movements are painful and limited.
- Early morning stiffness is characteristic.
- Synovial thickening is appreciable.
- Subcutaneous nodules may be palpable.

Investigations

Blood Investigations

Hb%—may be low.
TC—leukocytosis.
DC—neutrophil increase in the early acute phase and in chronic phase may show increase in lymphocytes.
ESR—elevated.
CRP—elevated. It is nonspecific and only suggests that there is active inflammation.
ANA—when positive suggests sensitization has occurred and autoimmunity has developed.
(Normal titer is 1:40. Higher titer indicates presence of autoimmunity).
RA factor—positive in 80% of the patients. > 20 IU/ml is considered as positive.
CCP (Citruline antibody)—elevated. It is more sensitive than RA factor. Can be positive even when RA factor is negative.

What is ANA (Antinuclear Antibodies)?

Antinuclear antibodies are not the usual antibodies. They are autoantibodies. These have a capability of binding to the nucleus of a normal cell, which it recognizes as an antigen and makes it vulnerable for destruction.

What is RA Factor?

RA factor is an autoantibody against the Fc portion of IgG. It is seen in about 80% of the patients with rheumatoid arthritis. It is not 100% specific. False-positive results are possible.

Hence the result is to be correlated with clinical symptoms.

What is CCP Antibody?

CCP stands for Cyclic Citrulinated Peptide Antibody. Citruline antibody titre is a better diagnostic test than RA factor. When elevated, it not only helps to diagnose rheumatoid arthritis but also indicates the possibility of the enhanced risk of joint destruction.

X-rays (Figs 12.10B and C)

In the early stages diffuse rarefaction is the only finding. Later other changes develop such as

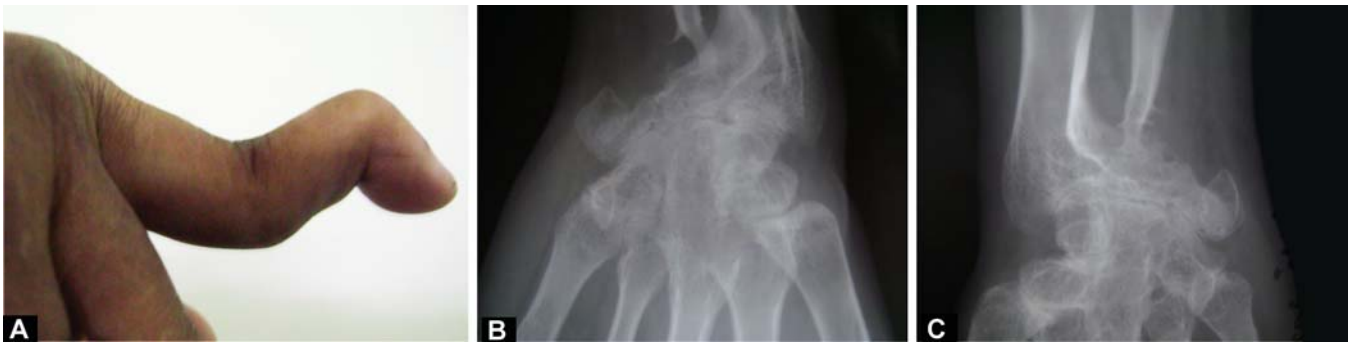
- Narrowing of the joint space.
- Destruction of the articular surfaces.
- Subchondral sclerosis.
- Subchondral cysts.

Treatment

Medical

Early in the disease medical line of treatment is followed.

a. Cold packs to reduce inflammation.



Figures 12.10A to C

Showing (A) classical swan neck deformity due to tightness of the intrinsic muscles as a result of inflammation and fibrosis. (B and C) Radiographs showing ankylosis of the wrist joint in a burnt out case of rheumatoid arthritis. Note the extensive destruction that has occurred.

b. Rest to facilitate recovery.
c. Drug therapy.
   In the stage of active inflammation, anti-inflammatory drugs and steroids local as well as systemic is indicated. These are the first line of drugs.
   DMARD (Disease Modifying Anti-rheumatic Drugs) are the second line of drugs which are given within 3 months of control of acute phase of the disease. Hydroxy chloroquine, methotrexate, leflunamide, etanercept, infliximab are some of the common drugs that are used. These drugs act specifically by inhibiting the chemical maediators of inflammation, e.g. TNFα, interleukin-1, etc.

Surgical

Total joint arthroplasty When considerable joint damage and fibrous/unsound ankylosis has occurred resulting in a painful deranged joint, total joint replacement is the treatment of choice. It should always be done in the remission phase of the disease and never in the active phase of the disease.

Arthrodesis It is the last option to give a painless, stable joint when all other options have failed. The joint involved is fused in functional position. Indications are more in wrist and hand.

Reconstructive procedures These are mainly done for deformities of the hand. Deformities like mallet finger, boutonniere and swan neck are correctable by surgical release and suturing of the slips of the extensor expansion. Once the joints are damaged such reconstructive procedures are not possible (Ref: For details of the procedures).

Revision Questions

Q. Write notes on:
   a. DMARD
   b. Swan neck deformity
   c. CCP
   d. Pathology of rheumatoid arthritis.

Essay Question

Q. Discuss the etiopathogenesis, clinical features, radiological features, diagnosis and management of rheumatoid arthritis. Enumerate its complications.
Q. Discuss the drug therapy of rheumatoid arthritis.

Hyperparathyroidism

Parathyroid glands are responsible for maintaining a fine balance of levels of calcium in the human body (Fig. 12.11). Normally they are 4 in number 2 on each side of the midline in the vicinity of the thyroid gland. But the number may vary between 2 and 6. The superior parathyroid glands lie on the posterior surface of the middle 1/3rd of the thyroid and the inferior parathyroid glands lie on the posterior surface of the lower pole of the thyroid.

Classification

The hyperparathyroidism may be classified as:
A. Primary—due to adenoma of the parathyroid.
B. Secondary—due to persistant hypocalcemia e.g renal rickets
C. Tertiary—the secondary hyperplasia remains and becomes autonomous.
   All types result in excessive secretion of parathormone.

Pathology

Excess of parathormone enhances conservation of calcium directly through renal tubular absorption, and bone

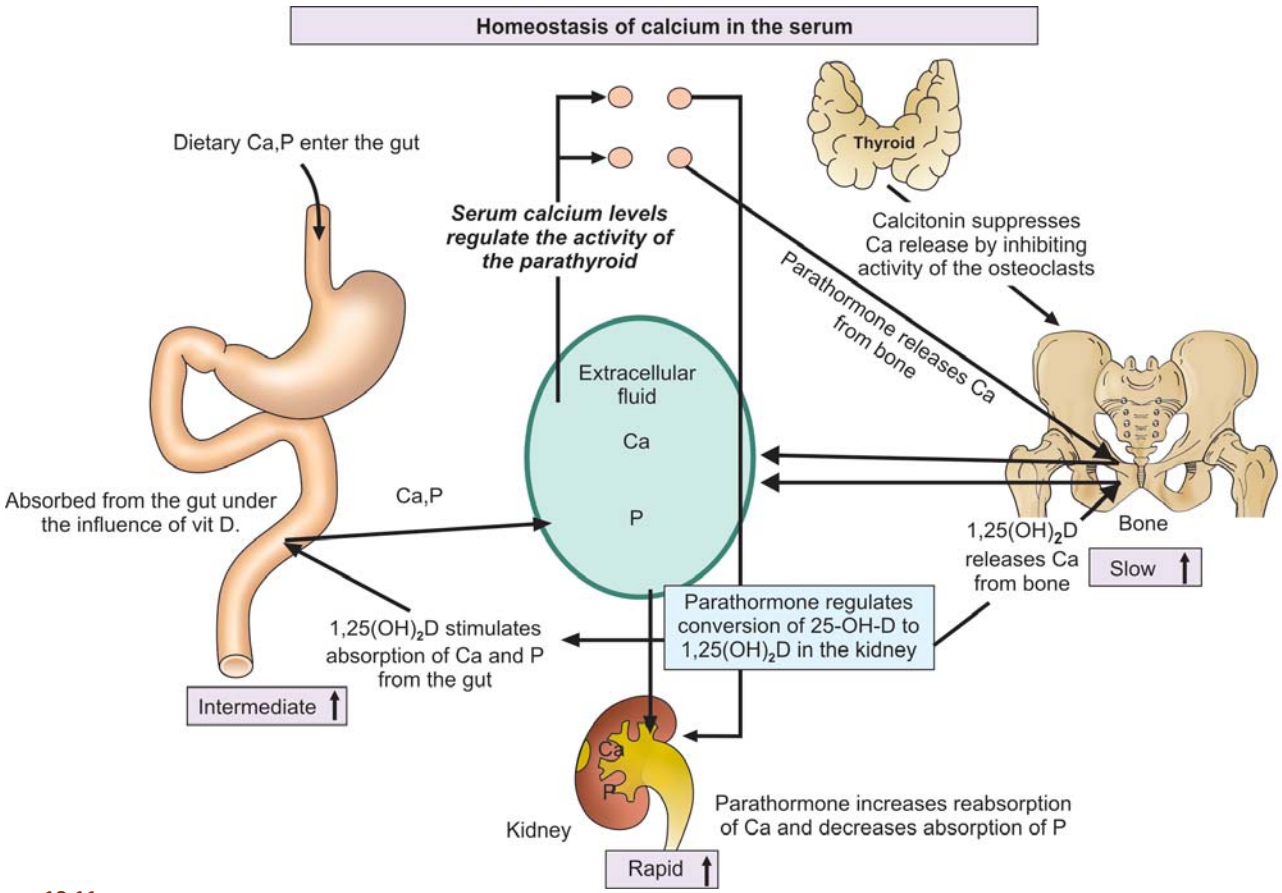


Figure 12.11

Showing the regulation of calcium in the human body. Note that the parathormone has a direct action on the bone and the kidneys and an indirect action on the intestines through the active metabolite of vitamin D. Decrease in serum calcium stimulates the release of parathormone.

resorption and indirectly through intestinal absorption of calcium by its regulatory action on vitamin D synthesis. Thus, there is hypercalcemia. Despite of augmented renal tubular absorption of calcium some amount of calcium is excreted in the urine which results in calciuria.

The renal tubular absorption of phosphate is suppressed. Hence there is hypophosphatemia and phophaturia.

This abnormality has generalized effect on the body and the organs.

Effect on Kidneys

It results in calcinosis, nephrolithiasis, recurrent infection and impaired renal function.

Effect on the Bone

It results in loss of bone substance due to direct (resorption) dissolution of bone. Classical subperiosteal erosion,endosteal cavitation and replacement of the marrow by vascular granulation tissue as well as fibrous tissue leads to a classical change known as 'osteitis fibrosa cystica'. Hemorrhage occurring in this fibrous tissue stroma leads to the formation of cysts with fluid with a brownish look. These are known as 'Brown tumors'.

These changes of softening can cause pathological fracture.

Clinical Features

Age: 3rd to 5th decade

Male: Female Ratio 1:3

- Primary hyperparathyroidism may remain asymptomatic. Classical features are depicted as bones, stones, abdominal groans and psychic moans and are due to hypercalcemia.
- Anorexia, fatigue,weakness, abdominal pain and depression.
- Nephrocalinosis and lithiasis,
- Pathological fractures.

Radiological Features

In the Hand

Classical subperiosteal cortical resorption of the middle phalanges.

In the Spine

Biconcave vertebral body due to the bulging of the disc, 'Cod fish spine'.

In the Long Bones

Generalised rarefaction with cystic lesions,osteitis fibrosa cystica and brown tumors.

In the Skull

Stippling calcification. 'Pepper pot skull' (also known as salt pepper appearance).

Laboratory Investigations

- Elevated serum calcium, low serum phosphorous
- Elevated serum alkaline phosphotase.
- Elevated serum parathormone levels.

Treatment

A. Primary hyperparathyroidism is treated nonoperatively with adequate hydration and reduced intake of calcium.

   Indications for surgical removal

   i. Marked and sustained hypercalcemia.
   ii. Progressive nephrocalcinosis and renal calculi.
   iii. Severe bone loss and rarefaction of bone.

   Hungry bone syndrome: This is because of brisk new bone formation that begins to occur after parathyroidectomy. Sudden drop in the levels of serum calcium is observed. This may cause severe tetany. Should be treated by active forms of Vitamin D.

B. Secondary and tertiary hyperparathyroidism. Treatment should always be directed towards treating the primary pathology.

C. Treatment of pathological/stress fractures. These are always treated by protection and immobilization. Once the primary cause is treated, recalcification begins to occur and the lesions heal.

D. Treatment of deformities. Deformities are always treated by corrective osteotomies after the serum levels of calcium and alkaline phosphotase become normal.

Essay Question

What is the role of parathyroid gland in calcium metabolism? Discuss hyperparathyroidism.

Further Reading

Rickets Osteomalacia and Hyperparathyroidism

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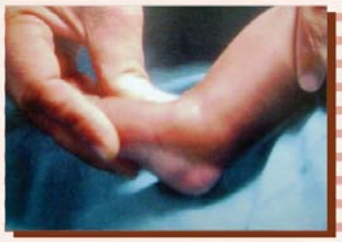


13

Congenital and Developmental Anomalies

- *Congenital Talipes Equinovarus*
- *Congenital Vertical Talus*
- *Developmental Dysplasia of the Hip (DDH)*
- *Skeletal Dysplasia and Other Conditions*

Congenital Talipes Equinovarus

Popularly known abbreviation is CTEV. It is also known as Congenital Clubfoot. Hippocrates was the first to describe the condition and mention the importance of gentle manipulation and bandaging for successful correction. In ancient Mexico the Aztecs treated clubfeet with splints prepared out of cactus and applied casts using flour, lime, tar and cloth. Egyptian tombs have depictions of clubfeet.

Lorenz was the first one who advocated the principles of gradual correction. He emphasized the need for correcting adduction deformity first before correcting varus and equinus. He was also the first one to perform Achilles tendon tenotomy.

Since then several physicians have treated clubfoot successfully using the method of gradual manipulation and splinting. Kites and Ponsetti's technique of manipulation and correction by the application of serial plaster of Paris casts and later the use of Dennis-Brown splint are some of the well established nonoperative methods.

Etiology and Pathogenesis

Etiology of clubfoot still remains unsolved and is thought to be multifactorial. Following factors are considered.

a. Hereditary.
b. Increased intrauterine pressure and malposition (Dennis-Brown 1933).
c. Arrest in development.
d. Bony abnormalities.
e. Tendon and ligament abnormalities.

(The clubfoot secondary to neuromuscular disorders are not included in the discussion as they are considered as teratologic).

None of the factors have been conclusively proved. But it is understood that the deformity occurs in the territory supplied by the posterior tibial nerve. Excessive collagen synthesis leading to fibrosis of the muscles and the ligaments seems to be the cause for the development of deformity.

Deformities in Clubfoot

The deformities present are:

a. Equinus,
b. Adduction.
c. Inversion.

These deformities are present both in the forefoot and in the hind foot. The combination of adduction and inversion results in varus deformity. The deformities are the result of tight tendons and ligaments. All the ligaments of the ankle (except on the lateral side) and tendons of the posteromedial compartment of the ankle are contracted (Figs 13.1A and B).

Ligaments Involved

All the ligaments around the talus with the exception of lateral side are contracted.

Medially

i. The deltoid ligament.
ii. The talonavicular ligament.
iii. The plantar calcaneonavicular ligament (Spring ligament).

Posteriorly

i. Posterior talofibular ligament.
ii. Posterior calcaneofibular ligament.

Inferiorly

i. The interosseus talocalcaneal ligament.

In addition to this the posterior capsule of the ankle and the subtalar joint and the plantar fascia are also contracted.

Tendons and Muscles Involved

i. Tibialis posterior.
ii. Flexor digitorum longus.
iii. Flexor hallucis longus.
iv. Tendoachilles.
v. Abductor hallucis brevis (in the foot).

Diagnosis

It is a straight forward diagnosis. In severe cases the foot is placed upside down. The size of the heel and the foot is small. Always rule out other associated congenital anomalies and teratological causes, e.g. spina bifida, menigomyelocele, arthrogryposis multiplex congenita, etc.

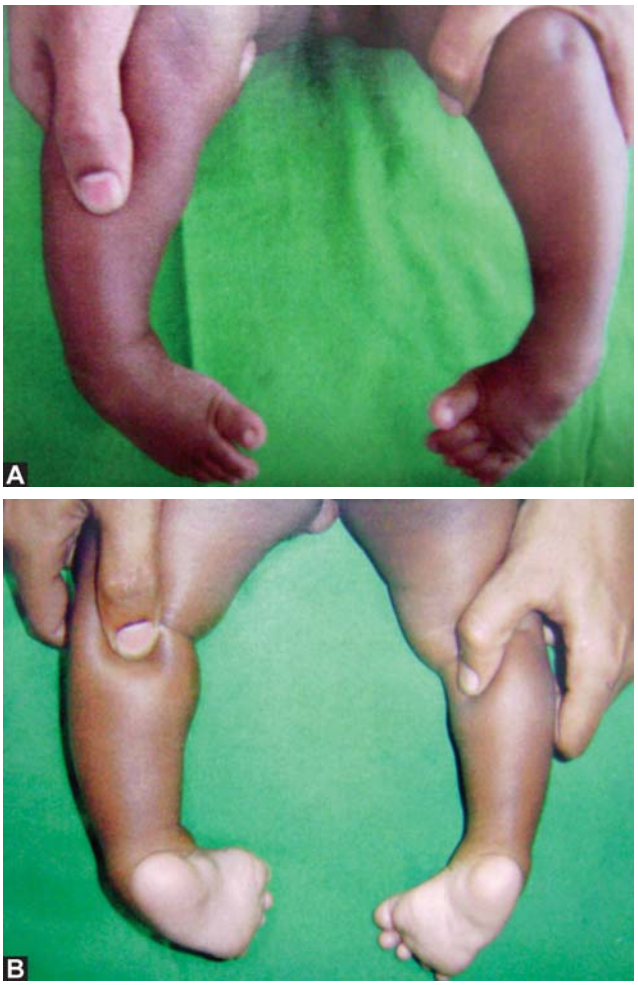
Radiological assessment of clubfoot (Figs 13.2A and B)

Angles in radiological views	Normal	CTEV
AP view		
Talocalcaneal angle-A	20-50°	15°
Talo-first metatarsal angle-B	0 to –15°	< –20°
Lateral view		
Talocalcaneal angle	35-50°	< 20° to –10°
Tibiocalcaneal angle (Maximum dorsiflexion angle)	40° ± 15°	> 70°

Talocalcaneal Index

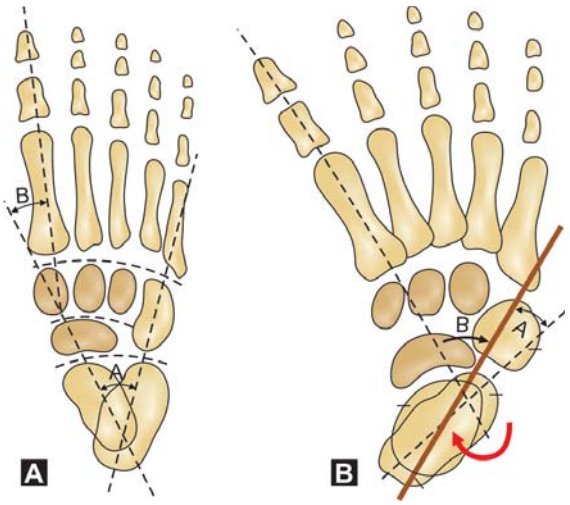
The sum of talocalcaneal angle in AP and lateral views gives the index

40° and more	Excellent
20°-40°	Good
< 20°	Poor



Figures 13.1A and B

Clinical photograph of bilateral clubfoot seen from (A) the front and (B) the back showing all the classical deformities. Note that the deformities are present both in the forefoot and the hind foot. Also note the size of the heel which is small.



Figures 13.2A and B

Picture showing the angle (A) the talocalcaneal angle in AP view, and (B) the talo 1st metatarsal angle in AP view in normal foot **(A)** and **(B)** in clubfoot.

Treatment

Modalities of treatment are classified as nonoperative and operative. The surgical procedures are further classified as:

a. Procedures for early correction.
b. Procedures for correction of residual deformities.
c. Procedures for recurrent clubfoot and for neglected clubfoot.

Age of presentation has an important bearing on the choice of the procedure. However, it is an established fact that an attempted manipulative correction prior to planned surgery definitely lessens the deformity and decreases the extensiveness of the surgical procedure. Hence, treatment should always start with manipulation as the first step.

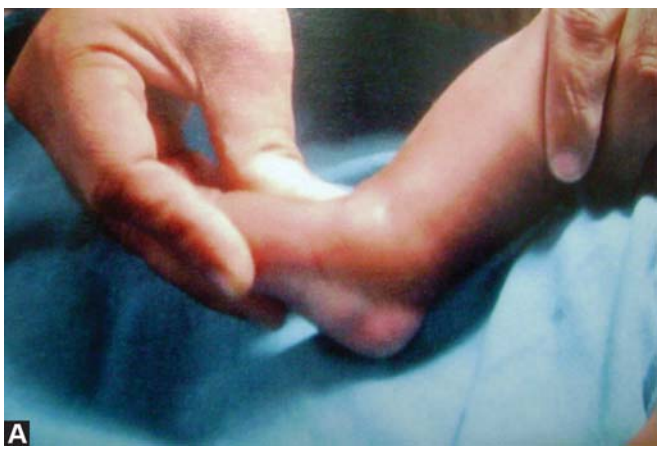
It is author's opinion that the manipulation done under anesthesia with relaxed muscles always gives a better correction, especially when the foot is rigid and less flexible.

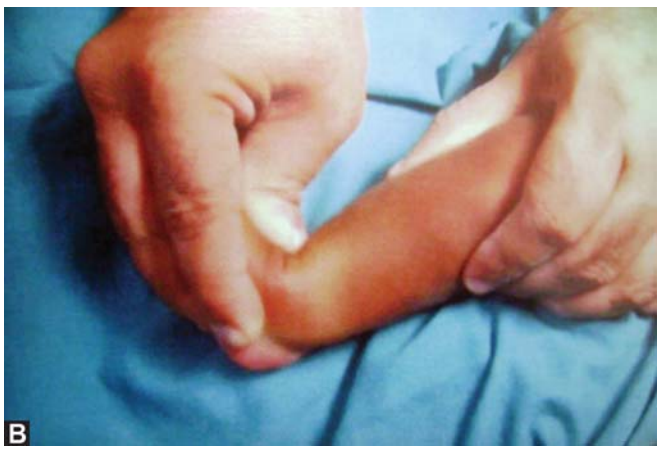
Nonoperative

Treatment starts at birth. Mother is taught how to manipulate the deformity gently without inflicting injury, till such time the child is fit for serial cast technique. Milder deformities are managed by corrective strapping and corrective splinting. More severe deformities are managed by serial casts after manipulation based on Ponsetti's method (Figs 13.3 and 13.4).

Ponsetti's method of manipulation (Ignatio Ponsetti, 1940) In this method, the manipulation is done in the following order to correct the deformity. First cavus, next adductus, then varus and last equinus. Non-adherence to these principles may result in poor correction or development of some other deformity, e.g rocker bottom foot. Manipulation is done for 1-3 minutes and then the baby is given a feeding bottle while the plaster cast is applied. The cast is changed after 5-7 days and the procedure repeated. Full correction can be achieved with 5-7 casts.

1. *1st, correction of cavus:* This is achieved by supinating and abducting the fore foot which is in contradistinction to other methods which recommend pronation of the first metatarsal for correction of the cavus. The deformity always gets corrected with one manipulation.
2. *2nd and 3rd, corrections of adductus and hind foot varus:* This is achieved simultaneously by abducting the foot





Figures 13.3A and B

Showing the manipulation being done to achieve gradual correction. (A) Shows the correction of fore foot varus. (B) Shows correction of hind foot equinus.

Note: Without correcting the forefoot varus and achieving some amount of valgus, correction of the hind foot equinus should never be attempted as it may result in development of rocker bottom deformity.

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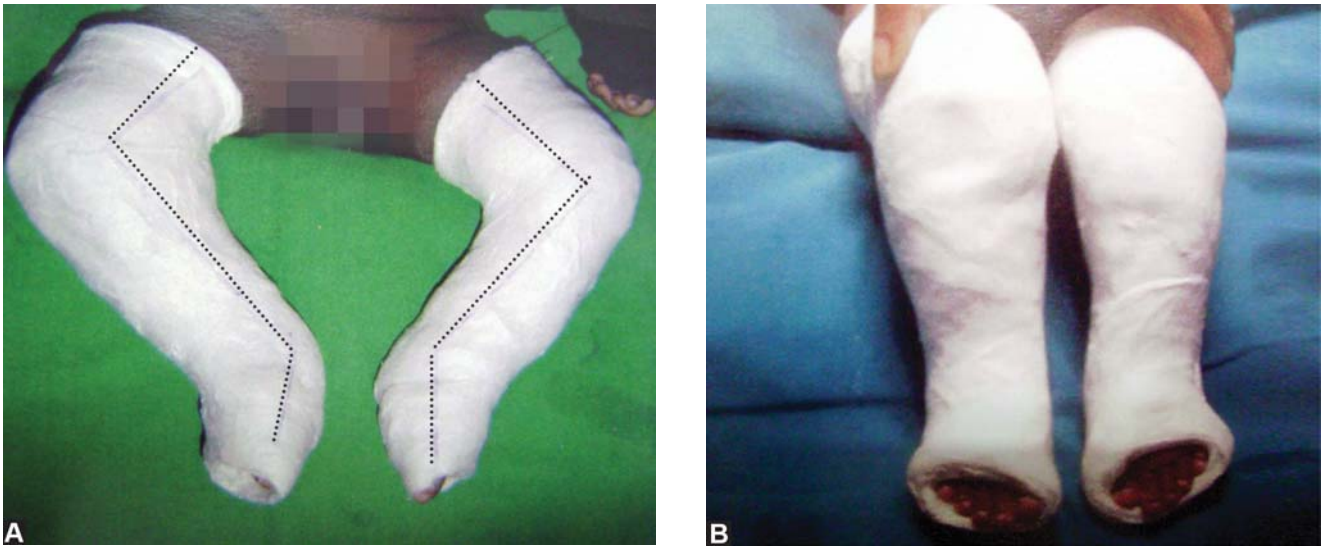


Figures 13.4A and B

Showing the position of immobilization after (A) full correction of forefoot varus and (B**)** overcorrected position of the foot after total correction. The cast given should always extend above knee with knee in 90° of flexion to relax the gastrosoleus.

while pressure is applied over talar head. This corrects the major portion of the deformity.

3. *4th, correction of equinus:* Correction should be attempted only when the hind foot is neutral and the forefoot is in 70° valgus. Foot is always dorsiflexed by applying pressure over the sole of the foot and never under the metatarsal (heel cord tenotomy facilitates the correction of equinus).

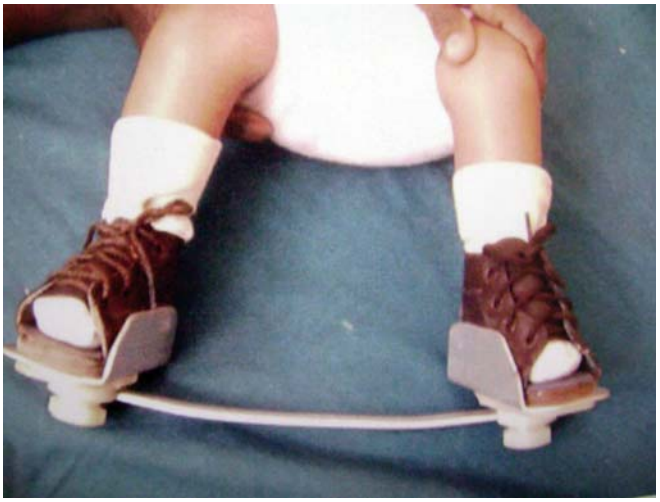


Figure 13.5

Dennis Brown splint for maintaining the correction achieved. Note how the heel rests on the footplate and is held firmly by the flap shoe with laces thereby maintaining the position of the foot in valgus.

Once the correction is achieved the foot is protected in a splint to prevent recurrence of the deformity. Folllow up is always necessary till 5 years of age (Fig. 13.5).

Operative Methods

Surgical procedure should be individualized and should limit itself to the release of tight structures only. This is achieved either by dividing them or by lengthening them. Bony correction is indicated in fixed and residual deformities.

For early correction before 12 months The accepted methods are:

a. Posteromedial soft tissue release, in which the ligaments which are contracted and tight are released and the tendons lengthened by the technique of Z-plasty. (Ref: Structures that are tight as mentioned above). The alignment obtained after the surgery is maintained in a plaster of paris cast for a period of 6 weeks. Then protected with a splint.
b. Peritalar release and stabilization of talonavicular and calcaneocuboid articulation with K-wire. A Cincinnati approach is used for the same. The K-wire is removed at 6 weeks. Postoperative care is same as above (Fig. 13.6).

(The Cincinnati approach is a transverse approach centered at the level of the tibiotalar joint. It extends from the anteromedial aspect at the naviculocuneiform joint and then over the back of the ankle to the anterolateral aspect of the ankle just distal to sinus tarsi) (Fig. 13.7A).

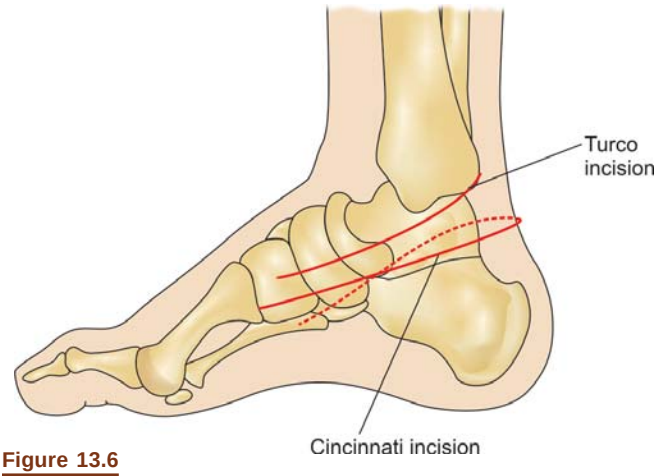
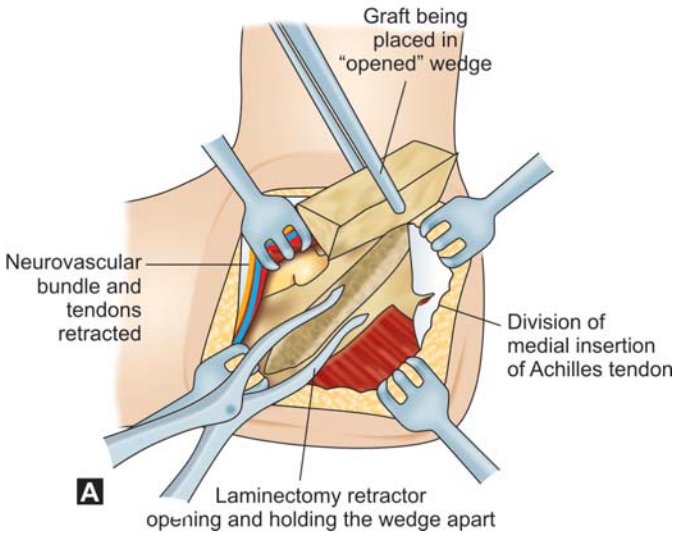


Figure 13.6

Picture showing surgical exposures for correction of CTEV.



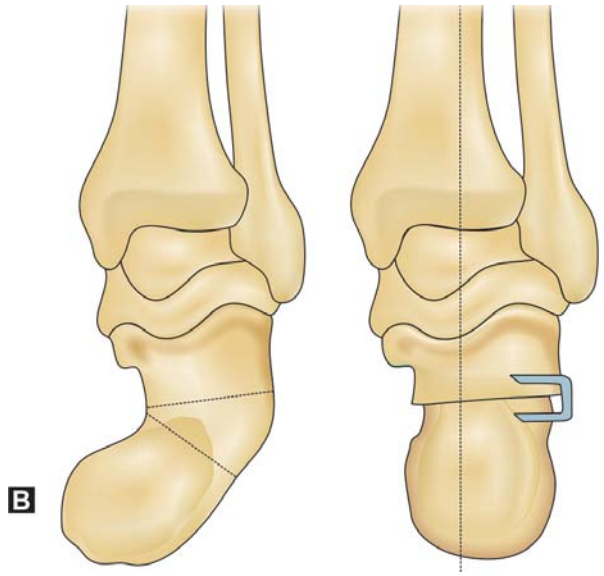
Figures 13.7A and B

Dwyer's osteotomy for the correction of residual heel varus.

For residual deformity after 4 yr

a. Hind foot varus—a calcaneal lateral closed wedge or medial open wedge osteotomy as suggested by Dwyer (Fig. 13.7B).
b. Fore foot varus—a cuboidal close wedge and medial cuneiform open wedge into which the wedge of bone taken from the cuboid is placed (Fig. 13.8).

For neglected clubfoot and recurrent clubfoot between 4-12 yrs **Illizarov** method or in the younger age group JESS fixator may be used. These methods not only correct the deformity but also have distinct advantage of lengthening the foot.



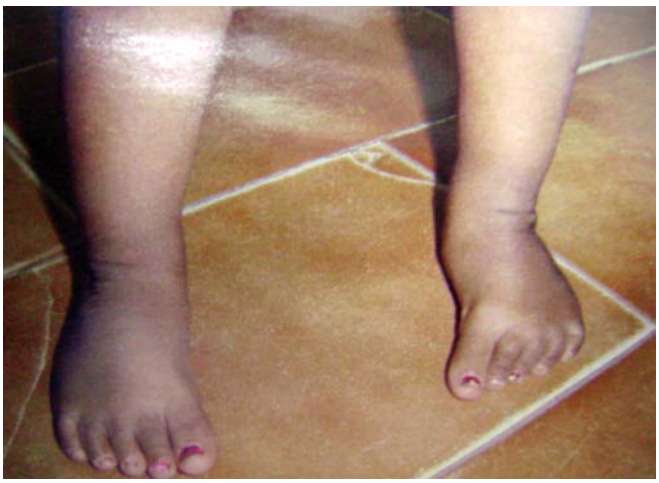
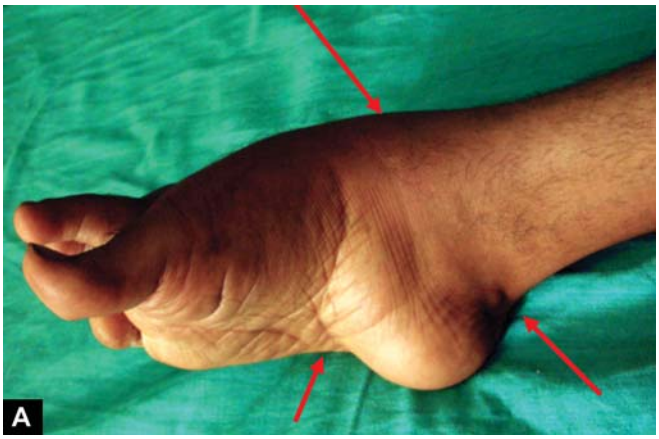
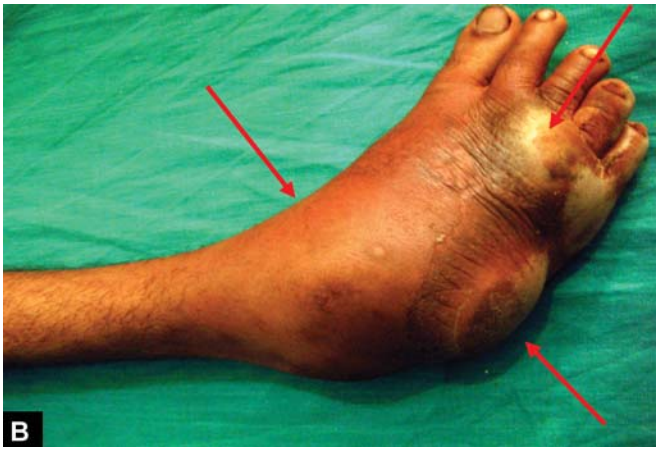
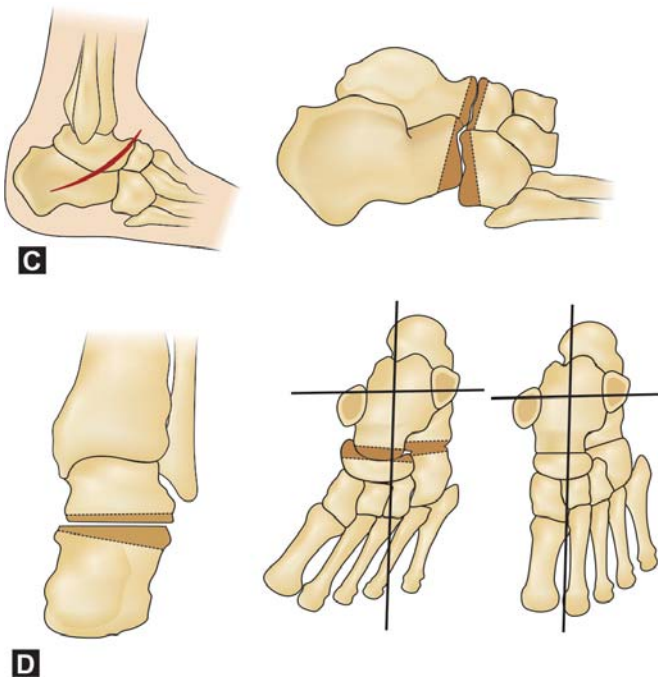


Figure 13.8

Clinical photograph showing residual deformity of forefoot adduction and supination. The child had undergone manipulation and cast application for correction. This is perhaps due to the faulty manipulation technique. It is likely that the first step of correction of cavus was not achieved and the manipulation was carried further.







Figures 13.9A to D

A neglected clubfoot showing a fixed deformity of talipes equinovarus. Note the callosities developed on the pressure points on the dorsolateral aspect of the foot due to weight-bearing stress. One of the callosities is infected. Triple arthrodesis is indicated in such feet for the correction of the deformities. (D) The three joints fused are the Subtalar,the Talonavicular and the Calcaneocuboid. Hence the name triple arthrodesis.

After 14 yrs In skeletally mature foot wedge osteotomy and triple arthrodesis may be considered. These procedures fuse the talonavicular, calcaneocuboid and subtalar joint (Figs 13.9A to D).

Essay Question

Discuss the etiopathogenesis, diagnosis and management of a case of congenital talipes equiovarus. Enumerate the complications and discuss the management.

Congenital Vertical Talus

Synonyms

- Rocker bottom flat foot.
- Congenital convex pes valgus.
- Teratological dislocation of talonavicular joint.
- Heneken was the first one to describe this in 1914.

Definition

It is a convex deformity of the plantar surface of the foot occurring due to the vertical disposition of the talus and dorsal dislplacement of the navicular secondary to dislocation of talonavicular joint.

Etiology

Exact etiology is not known. Following factors may predispose the occurrence.

a. Muscular imbalance resulting in over action of tibialis anterior.
b. Intrauterine compression.
c. Autosomal dominant transmission.
d. As a part of other associated congenital anomalies, e.g arthrogryposis DDH, etc.

Pathological Anatomy

Bone

The talus is fixed in a vertical position with associated hypoplasia of the talar head and the neck. The talonavicular joint is dislocated and the navicular bone is displaced dorsally articulating with the dorsal aspect of the neck of the talus. There is varying degree of subluxation of the calcaneocuboid joint. Thus there is elongation of the medial column and shortening of the lateral column of the foot.

Ligaments

The tibionavicular and the dorsal talonavicular ligaments are contracted. This prevents the reduction of the talonavicular joint. Also the posterior capsule of the ankle is contracted.

Muscles and Tendons

The tibialis anterior, extensor hallucis longus, extensor digitorum longus, peroneus brevis and tendoachilles are contracted. The tibialis posterior and the peroneal tendons are displaced anteriorly and may act as dorsiflexors instead of plantar flexors.

Diagnosis

The condition is easily diagnosed at birth. The convex plantar surface of the foot, severe dorsiflexion and abduction of the foot, the valgus and equinus position of the heel are the striking features.

Radiological Features (Figs 13.10A and B)

AP view (Fig. 13.11A)

- Increased talocalcaneal angle
- Fore foot is in abduction.

Lateral view

- Calcaneus is in equinus.
- Talus is vertically placed. (normal is horizontal).
- Navicular is displaced dorsally.

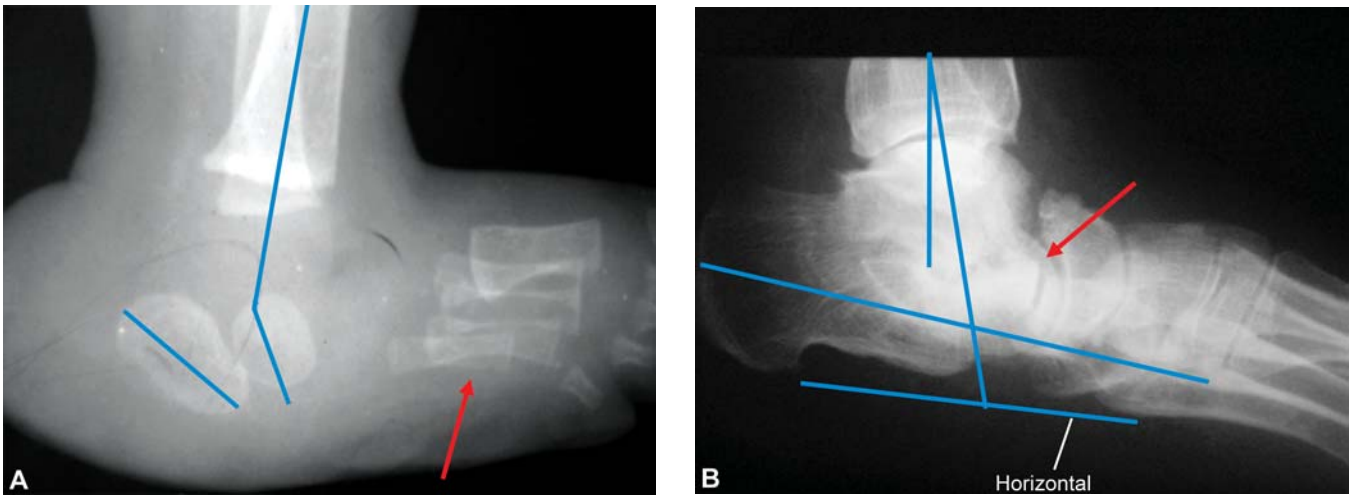
Lateral plantar flexion view

- To assess the talar metatarsal and calcaneal metatarsal axis. Both are increased because of equinus position of the bones (Normal values are 3° and 10° respectively).

Lateral dorsiflexion view

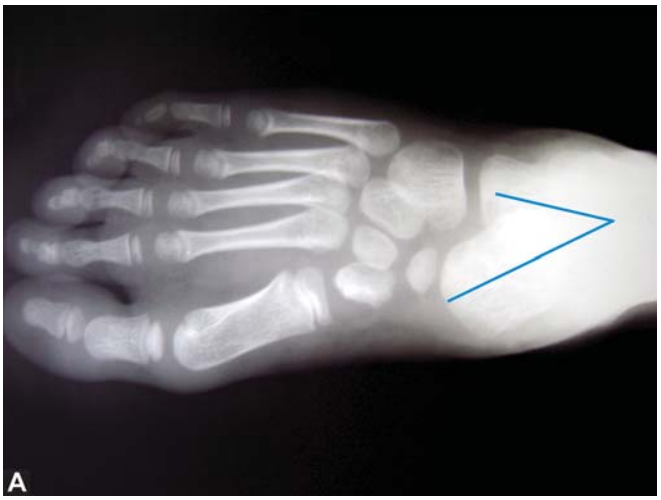
- To assess the heel equinus.

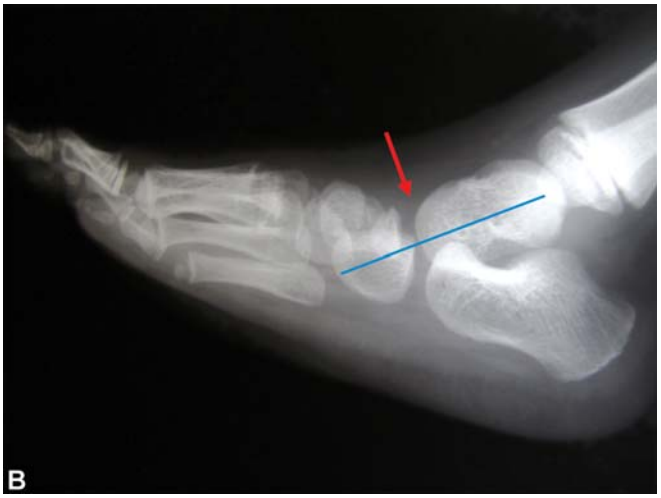
Note: The lines drawn for assessment of the position of the talus and the calcaneum in relation to the rest of the foot are all drawn along the long axis of the bones. In the AP view they are divergent and in the lateral views they are almost vertical and nearly at right angles to the horizontal (except the line of the calcaneum). Hence they derive the names tibiocalcaneal, tibiotalar, taloclcaneal, talohorizontal, talometatarsal, calcaneometatarsal, etc (Figs 13.10A and B).

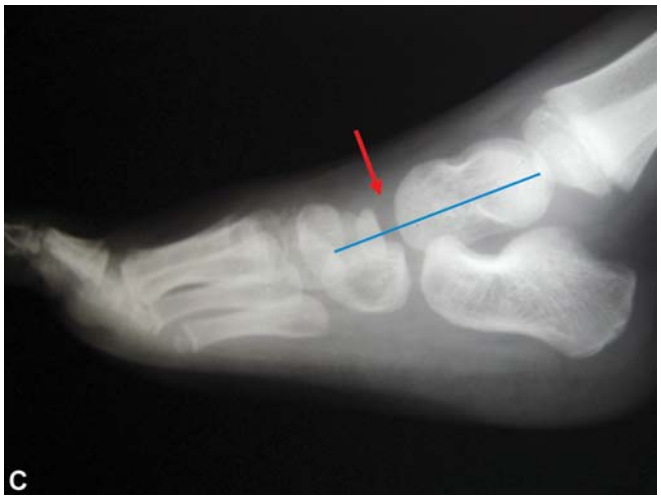


Figures 13.10A and B

Radiographs (A) of an infant with Congenital Vertical Talus and and (B) an adult with neglected Congenital Vertical Talus with secondary degenerative arthritis. (A) The soft tissue shadow shows the convexity of the pantar surface of the foot. The talus is near vertical and the calcaneum is in planar flexion. The navicular bone has not appeared but note the position of the metatarsals. They are dorsally displaced indicating the dorsal displacement of the navicular. (B) An adult with rocker bottom foot. Note that the tibio talar and the talo horizontal angle are near 180° and 90° respectively.







Figures 13.11A to C

Radiographs of (A) congenital oblique talus. Note that it has all the features of a vertical talus except that (B) the talonavicular joint subluxates in neutral position of the foot and (C) gets reduced in equinus. The red arrow shows the same.

Differential Diagnosis

Congenital Oblique Talus

This resembles congenital vertical talus but with certain differences as shown in Figures 13.11A to C.

Treatment

a. Nonoperative treatment starts at birth. Manipulation of the foot into plantar flexion and inversion in an attempt to reduce the dislocation is carried out. Rarely successful and if successful the reduction is maintained with closed pinning and subsequently removal of pin and application of splints. Even if it is not successful it helps in future surgery by stretching the soft tissues.
b. Surgical treatment consists of open reduction of dislocation and maintaining the correction achieved. Done in single stage or in stages. Ideal time for surgery is at 1 year. More severe the deformity more extensive is the surgery.

One Stage Release

1st step—Reduction of talonavicular joint by release of tibialis anterior, tibionavicular and talonavicular ligaments and the capsule. Reduction is stabilized with pin.

2nd step—Lengthening of peroneals and toe extensors and reduction of calcaneo cuboid joint.

3rd step—Correction of equinus contracture, release of Achilles tendon and capsule of the subtalar and the ankle joint.

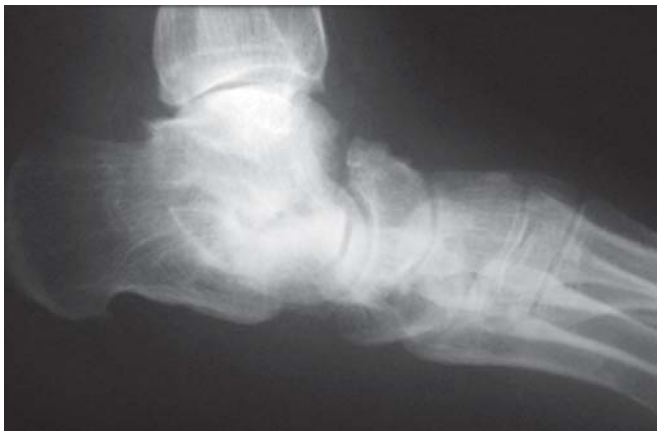


Figure 13.12

Radiograph showing the neglected Rocker bottom foot in a middle aged person. Note the degenerative changes in the subtalar, talonavicular, and the calcaneocuboid joint. Patient was treated by triple arthrodesis.

Appropriate cast is given postoperatively which is changed periodically and maintained for at least 8-12 weeks.

Excision of the navicular bone and Grice Green arthrodesis is indicated in older children and in resistant deformities.

Always keep a watch on recurrence during the follow-up upto 8-10 yr of age.

Complication of a neglected rocker bottom foot is tarsal degeneration resulting in degenerative arthritis. This occurs because of altered biomechanics during stress loading (Fig. 13.12).

Essay Question

Discuss the pathomechanics, diagnosis and management of Rocker bottom flat foot.

Developmental Dysplasia of the Hip (DDH)

The abbreviation CDH (congenital dislocation of the hip) is now replaced by DDH (developmental dysplasia of the hip). DDH is more appropriate because there is no congenital embryonic anomaly as such. The development starts normally in an embryo and due to some extraneous factors the head of the femur is not contained in the acetabulum. The most common factor is the laxity of the capsule. Other factors like malposition of the femoral head and the effect of abnormal stresses on the developing femoral head also play a part in the pathology. The child may be born with dislocated/subluxated hip or born with dislocatable and relocatable/reducible hip. In either case the hip is reducible with manipulation. According to Klisic (1989) " developmental displacement of the hip is a dynamic disorder potentially capable of becoming better or worse as the child develops". This statement is absolutely true. If the hip remains dislocated for longer periods structural changes develop and reduction becomes progressively difficult. But if the hip is maintained in a position of reduction right from the age of infancy it becomes normal with growth. So the statement supports the fact that the posture has a great role to play in the origin, progress and the regress of the pathology (Fig. 13.13).

Thus DDH is not only multifactorial in origin but also may present differently in different age groups.

Predisposing Factors

a. *Racial dominance:* Commonly seen in Native Americans and Caucasians. Not common in Asians and Africans.
b. *Ligamentous laxity:* Studies by Wynne-Davis suggests that there is a genetic predisposition for the ligamentous laxity. This is the most common pathology that causes the hip to dislocate. However this does not explain unilateral hip dislocation. Also suggests the postural influence on the development of DDH.
c. *Prenatal positioning:* A frank breech presentation has a higher incidence when compared to single footling breech and double breech presentation.

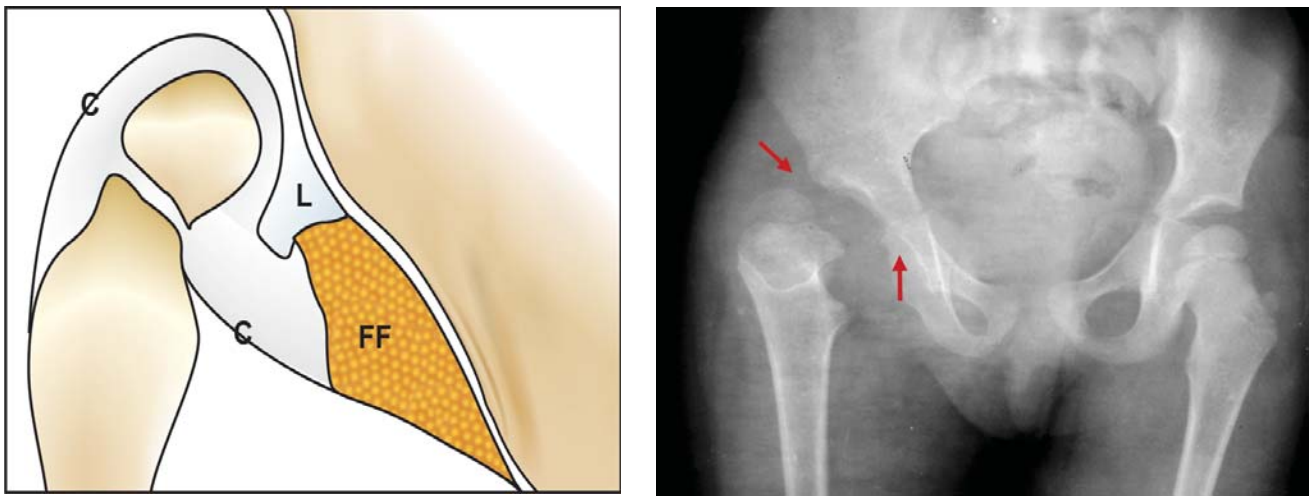
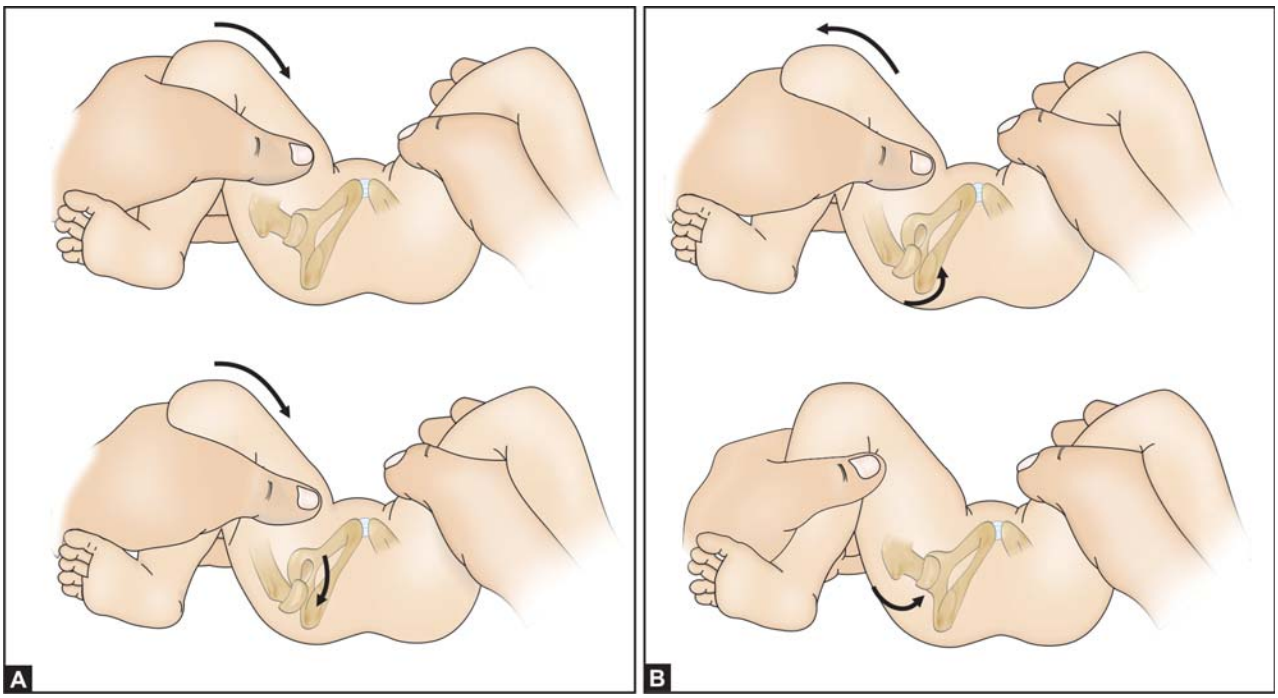


Figure 13.13

Showing the diagrammatic representation and the radiograph of a neglected DDH. FF—shows the acetabulum filled with fibro-fatty tissue, L—the limbus is inverted and C—the capsule is stretched.



Figures 13.14A and B

Picture showing tests of (A) Barlow and (B) Ortolani being performed to dislocate and relocate/reduce the hip.

d. *Postnatal positioning:* Incidence has been found high in Native Americans who carry their babies wrapped up with the hip in full extension in cradle board.

Considering these facts, it can be concluded that relatively gentle factors applied persistently cause deformation and dislocation/subluxation.

e. Primary acetabular dysplasias.
f. *Other associated anomalies:* DDH may be a part of other associated congenital anomalies, e.g. torticollis, congenital dislocation of the knee, etc.
g. *Sex:* The incidence of DDH is more in females when compared to males.

Clinical Features

Up to 2-3 Months (the neonate)

The hip is always dislocatable and reducible. The classical test of Barlow's and Ortolani's is always positive.

Barlow's Test (Fig. 13.14A)
With the baby in supine position (on the couch or mother's lap) the examiner holds baby's both the knees and gives a gentle adduction push to one of the knees. If the hip is dislocatable the feel of femoral head jumping out of the acetabulum is made out by the fingers placed in the region of the greater trochanter.

The release of pressure slips the head back into the acetabulum.

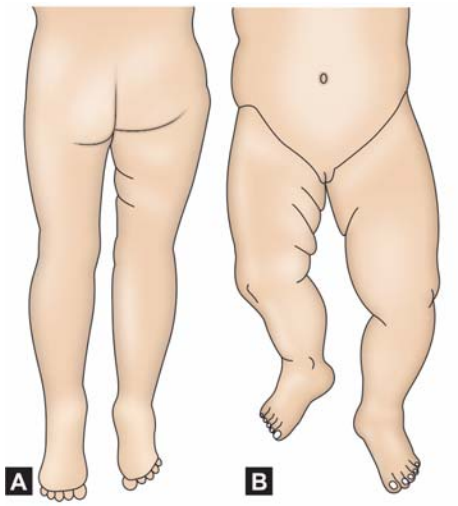
Ortolani's Test (Fig. 13.14B)
This is the reverse of Barlow's test. In this test the examiner tries to reduce the dislocated hip. The test is performed with the baby in supine position as in Barlow's test. The babies thigh is grasped between the thumb and the index finger. With the other fingers the greater trochanter is gently lifted up, abducting the hip at the same time. The clunk of reduction is felt. When the hip is adducted the head gently slips out (Barlow's test).

These tests have to be repeated a couple of times to make certain that there is DDH. Also should be performed on the other hip to assess the status of the other hip. .

After 3-6 Months (the infant)

During this period the irreducibility becomes gradually established. As the hip remains in dislocated position other physical findings appear.

a. Asymmetry of the thigh and gluteal folds (Figs 13.15A and B).



Figures 13.15A and B

Picture showing asymmetry of skin creases on the involved side in a unilateral DDH.

b. Shortening of the thigh.
c. Superior location of the greater trochanter.
d. Positive telescopy.
e. Limitation of abduction (Fig. 13.16B).
f. Discrepancy in the levels of the knee(Allis',Perkin's or Galeazzi sign) (Fig. 13.16A).
g. Klisic test (Figs 13.17A and B).

With the child supine the index finger is kept over the anterior superior iliac spine and the middle finger over the greater trochanter. Next a imaginary line is drawn between the two. This line when extended upwards cuts through the umbilicus in a normal hip.

In DDH because the trochanter is elevated this line passes half way between the umbilicus and pubis.

After the Child Begins to Walk

a. The shortening becomes pronounced in unilateral cases.
b. Trendelenburg gait develops in unilateral cases and waddling gait in bilateral cases.
c. Exaggerated lumbar lordosis secondary to flexion contracture at the hip becomes noticeable (Fig. 13.18).

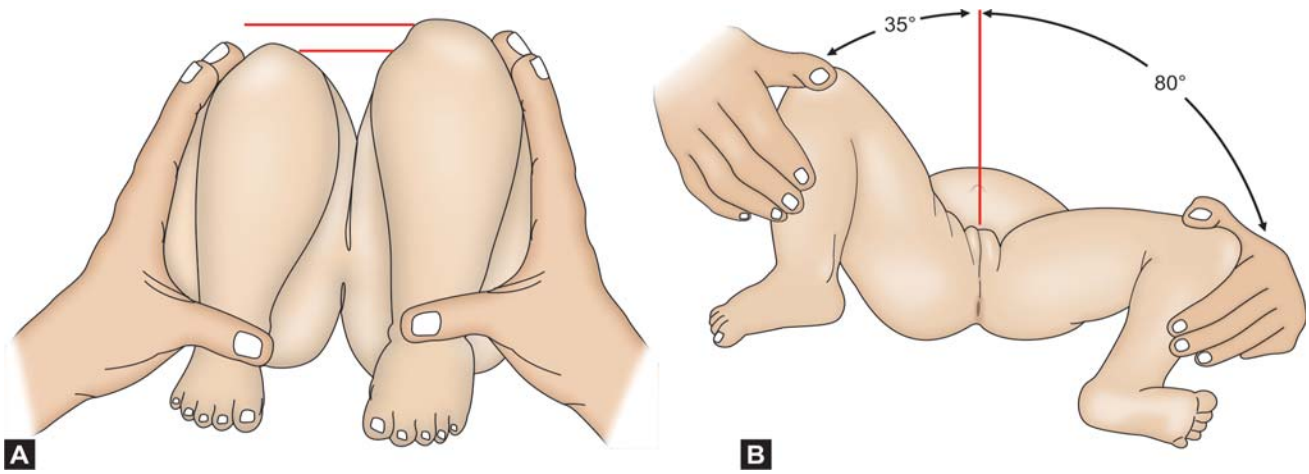
Note: Delay in milestones may or may not be observed. However, if there is a delay in walking, the possibility of DDH should be kept in mind.

Investigations

a. Ultrasound is a useful investigation in the newborn in whom X- ray imaging is of no use because of cartilaginous nature of the bones. It should be used judiciously and findings should be always be corelated with clinical findings to prevent overdiagnosis. (for more information refer α and β angle of Graf).
b. MRI is a better investigation than ultrasound. But, it is expensive and is accompanied by the disadvantage of sedating the newborn.
c. X-ray gives useful information in an infant, only after 3 months.

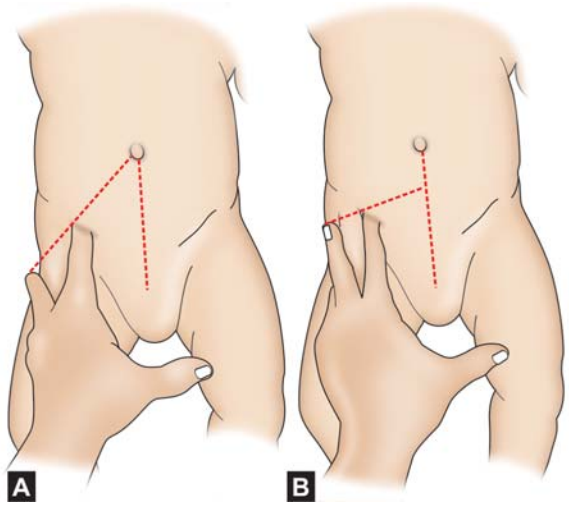
With the help of Hilgenreiner's and Perkin's line and the Shenton's line the location of the femoral head can be assessed. Also the center edge angle of Wilberg and the Acetabular index can be measured (Figs 13.19 to 13.20B).

Hilgenreiner's and the Perkin's line divides the hip into four quadrants. A normal hip lies usually in the lower



Figures 13.16A and B

(A) Showing asymmetry in the level of the knees which is known as Allis', Perkin's or Galeazzi Sign. On the involved side in a unilateral DDH the knee will be at a lower level. (B) Showing limitation of abduction of the involved right hip.



Figures 13.17A and B

Picture showing the Klisic test. Note the imaginary line cutting through the umbilicus in a normal hip and cutting midway between the umbilicus and the pubis in a DDH.

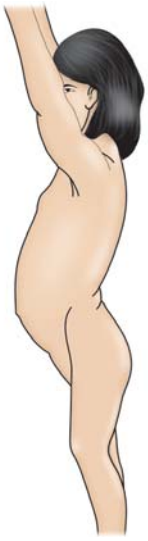


Figure 13.18

Picture showing exaggerated lumbar lordosis indicating the presence of flexion deformity in a neglected bilateral DDH. The child will have a waddling gait.

inner quadrant. A subluxated or dislocated hip moves towards the upper outer quadrant.

d. Arthrography gives useful information regarding the concentric nature of reduction.

Treatment

Nonoperative

Treatment starts soon after birth. It aims at maintaining the femoral head in the acetabulum till the relation gets organized and there is no risk of redislocation.

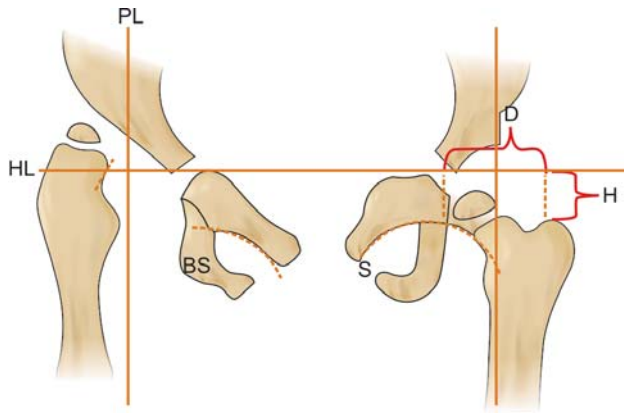


Figure 13.19

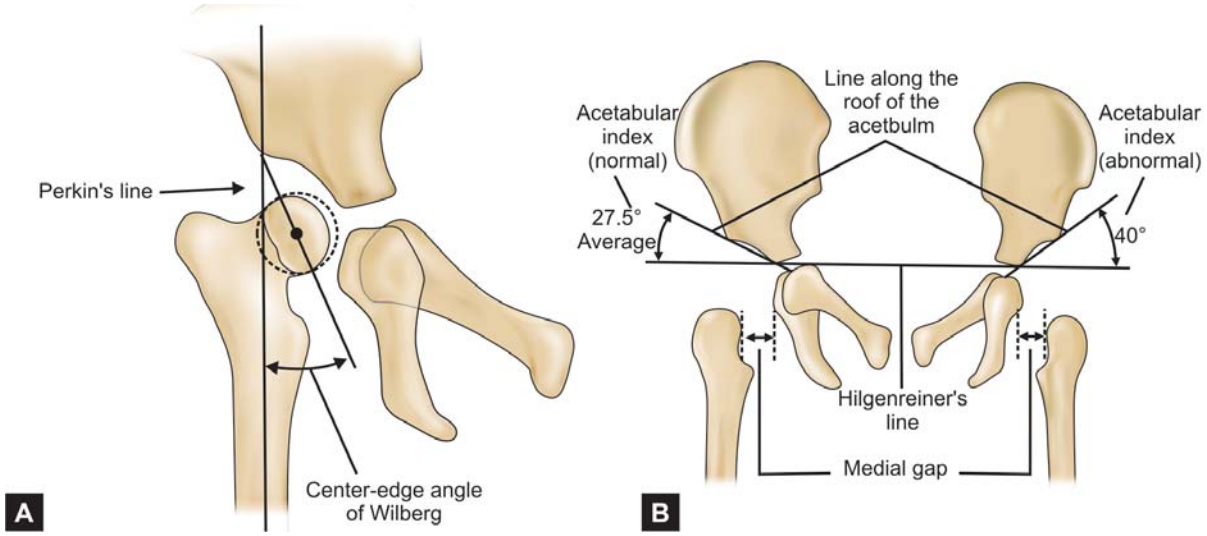
Picutre showing the following:
HL—Hilgenreiner's line. It is a horizontal line drawn passing through the center of the triradiate cartilage.
PL—Perkin's line. It is a line drawn perpendicularly down from the bony edge of the acetabulum.
S—Shenton's line. It is a line along the neck of the femur to the inner margin of the pubic bone. It forms a smooth 'C' shaped curve.
BS—Broken Shenton's line. This is seen in subluxation/dislocation
H—Dimension H. This is the height measured from the top of the ossified femur to the Hilgenreiner's line.
D—Dimension D. This is measured from the inner border of the tear drop to the center of the tip of the ossified femur.
Dimension H and D quantify proximal and lateral displacement of the hip respectively. They are useful even at an age where the epiphysis of the head of the femur is not ossified.

In neonates, infants and young children reducible hips are treated by *immobilization in reduced position* with the help of splints and plaster of Paris casts. *Watched* carefully *for* the possibility of *redislocation, avascular necrosis and nerve palsies,* e.g. femoral nerve in Pavlik harness (Figs 13.21 and 13.22).

Open Reduction

Surgery of open reduction is done in older children (6 months-6 yr). In the young (<6 months) open reduction is done only when closed reduction fails. Those hips which remain in dislocated position for a considerable time are not amenable for closed methods of reduction. The adaptive changes which take place in the soft tissues and later in the bone prevent reduction. Preoperative traction in the desirable position has proved its usefulness beyond doubt. It should always be given before open reduction. This is known to reduce the risk of avascular necrosis AVN (Fig. 13.23).

When structural skeletal changes have not taken place release of constricted/contracted soft tissues such as muscles and tendons, caspluse and ligaments, excision of the limbus etc. will help in reducing the head into the acetabulum.



Figures 13.20A and B

(A) *Center-edge angle of Wilberg:* It is an angle formed between the Perkin's line and the line drawn from the lateral lip of the acetabulum passing through the center of the femoral head. In older children (10-13 yr) the angle should always be more than 10°. (B) *Acetabular index:* This is an angle formed between a line drawn along the margin of the roof of the acetabulum and Hilgenreiner's line average angle in newborn is about 27.5°. It decreases with age. *Medial gap:* This is the distance between the inner margin of the tear drop and the inner margin of the neck of the femur. The gap increases in dislocation. Always compared with the the opposite hip. Not useful in bilateral cases.

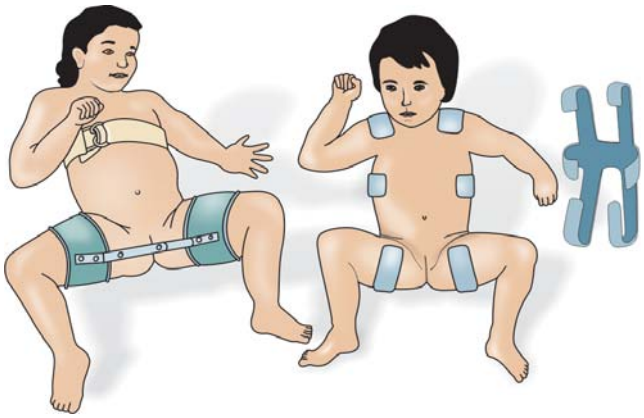


Figure 13.21

Picture showing Craig splint and von Rosen splint.

Figure 13.22

Picture showing the position of immobilization in spica cast after closed manipulation and reduction.

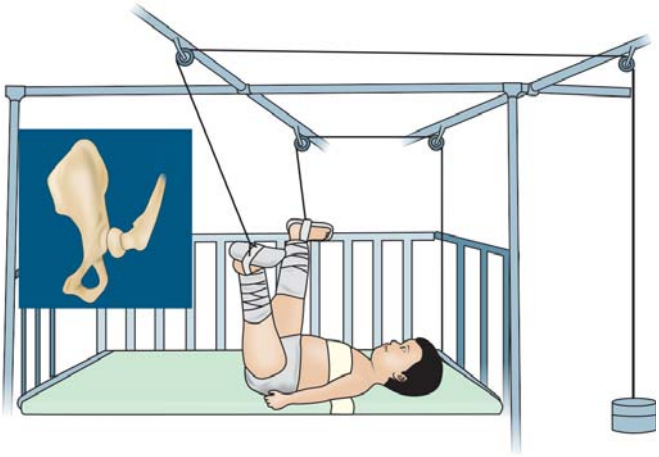


Figure 13.23

Picture showing Bryant's traction/Gallows traction.

When structural skeletal changes have taken place, femoral osteotomy and femoral shortening procedures for containment or shelf operations and osteotomies of the ilium for deepening the shallow acetabulum, are indicated. These procedures help in containing the head of the femur in the acetabulum (Fig. 13.24).

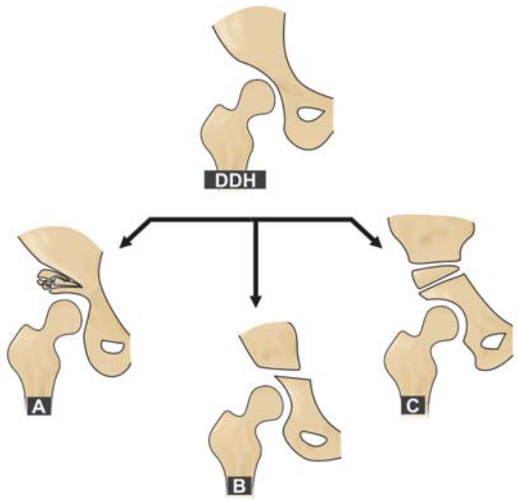


Figure 13.24

Showing different types of containment procedures for DDH. A—Shelf procedure; B—Chiari's osteotomy; C—Salter's osteotomy.

Reconstructive Procedures

In adults with unilateral dislocated hip, *limb lengthening procedures* are considered. These procedures take care of the shortening and the limp. Those hips which become painful (AVN) *total hip replacement* may be considered.

Revision Questions

Q. Write notes on
- a. DDH
- b. Barlow's test
- c. Ortolani's test.
- d. Hilgenreiner's and Perkin's line.
- e. CE angle of Wilberg.
- f. Salter's innominate osteotomy.

Essay Question

Q. Discuss the etiopathogenesis, diagnosis and management of a case of DDH. Enumerate the complications and discuss the management of complications.

Skeletal Dysplasia and Other Conditions

Skeletal Dysplasias

Skeletal dysplasias are a group of disorders that result in disturbances in the normal growth of the skeleton. They are genetically determined. Most of them belong to autosomal dominant type of inheritance. Few of them are autosomal recessive and X-linked.

The terminologies and the pattern of dysplasias have been clearly explained by Rubin P through his dynamic classification.

Dynamic Classification of Bone Dysplasias

(Based on Rubin P Dynamic Classification of Bone Dysplasias. Chicago year book medical publishers, 1964, p 82). According to this dynamic classification, the dysplasia is the result of either hypoplasia or hyperplasia of the specific cells in different regions of the bone, i.e Epiphysis, Physis, Metaphysis and Diaphysis. Hypoplasia is deficient or failure and hyperplasia is stimulation and excess.

1. **Epiphyseal dysplasias**
   - *A. Epiphyseal hypoplasias*
     - i. Articular cartilage, e.g. spondyloepiphyseal dysplasia congenita and tarda.
     - ii. Ossification center, e.g. multiple epiphyseal dysplasia congenita and tarda.
   - *B. Epiphyseal hyperplasias*
     - i. Articular cartilage, e.g. dysplasia epiphysalis hemimelica
2. **Physeal dysplasias**
   - *A. Cartilage hypoplasias*
     - i. Proliferating cartilage, e.g. achondroplasia congenita and tarda.
     - ii. Hypertrophic cartilage, e.g. metaphyseal dysostosis congenita and tarda.
   - *B. Cartilage hyperplasias*
     - i. Proliferating cartilage, e.g. hyperchondroplasia.
     - ii. Hypertrophic cartilage, e.g. enchondromatosis.
3. **Metaphyseal dysplasias**
   - *A. Metaphyseal hypoplasias*
     - i. In formation of primary spongiosa, e.g. hypophosphatasia.
     - ii. In absorbtion primary spongiosa, e.g. osteopetrosis congenital and tarda.
     - iii. In absorbtion of secondary spongiosa, e.g. craniometaphyseal dysplasia congenita and tarda.
   - *B. Metaphyseal hyperplasias*
     - i. Spongiosa, e.g. multiple exostosis.
4. **Diaphyseal dysplasias**
   - *A. Diaphyseal hypoplasias*
     - i. Periosteal bone formation, e.g. osteogenesis imperfecta congenital and tarda.
     - ii. Endosteal bone formation, e.g. idiopathic osteoporosis congenita and tarda.
   - *B. Diaphyseal hyperplasias*
     - i. Periosteal bone formation, e.g. progressive diaphyseal dysplasia.

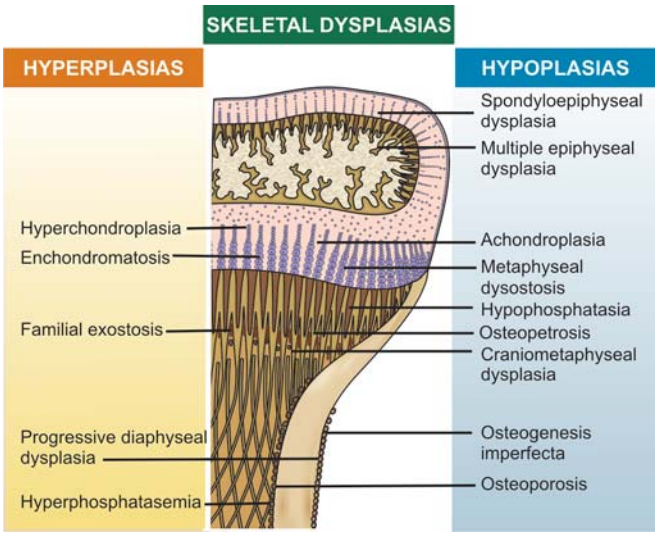


Figure 13.25
Showing areas of bone responsible for skeletal abnormality and different dysplasias.

ii. Endosteal bone formation, e.g. hyperphosphatasemia.

Important Skeletal Dysplasias (Fig. 13.25)

Spondyloepiphyseal Dysplasia

This is characterized by generalized involvement of the epiphysis of the spine and long bones resulting in disproportionate dwarfism.

Types

a. Congenita—severe form presents at birth. Disproportionate dwarfism is pronounced.
b. Tarda—less severe form presents in early childhood with less disturbance in growth. Loss of height is minimal.

Transmission: Autosomal dominant.

Clinical features

- Wide set eyes
- Short neck
- Barrel chest
- Exaggerated lumbar lordosis with protuberant abdomen
- Coxa vara with waddling gait
- Angular deformity in the lower limb, e.g genu valgum.

Radiological features

- Delay in the appearance of the epiphysis (is responsible for the short stature)
- Coxa vara in the hip
- Genu valgum in the knee
- Platyspondyly in the spine
- Hypoplasia of the odontoid process.

Orthopedic care

The care is directed towards *correction of angular limb deformities*. This possibly prevents the development of degenerative arthritis at a later period.

Odontoid hypoplasia may result in *atlantoaxial instability.* When present it needs to be *stabilized.*

Multiple Epiphyseal Dysplasia

The condition was first described by Fairbank as Dysplasia Epiphysealis Multiplex.

In this unlike spondyloepiphyseal dysplasia, the spine, skull and vertebra are not involved. The epiphysis of the long bones and short tubular bones are involved. Transmission is autosomal dominant; rarely recessive. Delay in appearance of epiphyseal ossification centers is observed radiologically mainly in long bones and short tubular bones. When appears the epiphysis is small, flat and may have mottled appearance. Early diagnosis and correction of the deformity is indicated to prevent early degenerative arthritis.

Achondroplasia

It is the most common form of dwarfism. The incidence is around 1.5-2.0 per 10,000 live births (Fig. 13.26).

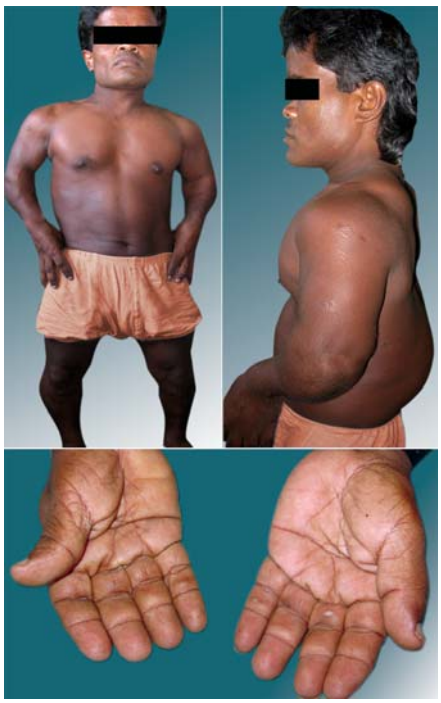


Figure 13.26
Pictures showing clinical features of an achondroplastic dwarf. Note the 'Starfish hand' on the right side hand and trident hand on the left side hand. Also note the presence of a single transverse crease in the palm.

Types

- *Spontaneous mutation occurring in paternal gene* (more common than maternal) of normal parents. The *offspring* is an *Achondroplastic dwarf*. Can expect *normal life expectancy.*
- *Homozygous Achondroplasia* occurring in the *offspring of achondroplastic parents*. Generally the *outcome is fatal* in the neonatal period.

Transmission: Autosomal dominant with complete penetrance.

Clinical features (Fig. 13.26)

- Normal intelligence.
- Bossing of the skull.
- Small maxillae with prominent mandibles.
- Deformities in the spine.
- Short stubby hand with shortening of the middle finger resulting in equal length of all the digits *(Starfish hand).* The middle and the ring finger is separated by a greater space which is referred as *trident hand.*
- Short upper and lower limbs.
- Protuberant abdomen.

Radiological features

- Shortening of the tubular bones with increased diameter.
- V or U shaped growth plate of the distal femur.
- Widening of the metaphysis of long bone.
- Squared ilium with horizontal acetabulum.
- Posterior scalloping of the vertebral body.
- Narrow spinal canal with decreased interpedicular distance.
- Shortness of the base of the skull with small foramen magnum.

Orthopedic care

Care is necessary for cervical spinal cord compression in young infants and lumbar cord compression due to canal stenosis at a later age group.The treatment is decompression of the foramen magnum/spinal canal.

Shortening of the limbs may need limb lengthening procedures.

Hypochondroplasia

This is a less severe form of dwarfism resembling achondroplasia. Transmission is autosomal dominant. The condition cannot be diagnosed at birth but presents itself at a later period of growth. Limb lengthening procedures are indicated to correct short stature.

Osteopetrosis

This also also known as Albers-Schonberg disease (after Albers-Schonberg who described the disease in the year 1904), Marble bone disease and Chalk bone disease. Failure of osteoclastic resorption leads to the formation of calcified cartilage and primitive osteoid. Thus there is increased sclerosis and brittleness of bone with a tendency for easy fracture.

Histopathology identifies abundant osteoclasts. But these osteoclasts do not respond to PTH because of inherent abnormality. No activation of macrophages and monocytes is observed. Thus calcified cartilage and primary woven bone is not replaced by lamellar bone and is found in abundance. This immature bone is tightly embedded into the coarse fibrous matrix. This increases the fragility of the bone and makes it susceptible for pathological fracture.

Types

- *Malignant form* in which there is *obliteration of the marrow* resulting in *pancytopenia, anemia,* hepatosplenomegaly, easy bruising, bleeding abnormalities and delayed dentition and caries teeth. Bony overgrowth leads to *obliteration of the cranial foramina* leading to *nerve palsies.* Blindness and deafness may develop. *Pathological fractures* are common. The condition is *lethal.* Bone marrow transplantation is the only hope.
- *Benign or tarda form* in which the diagnosis is incidental. No clinical signs are present. Patients have a normal life span. Pathological fracture and premature osteoarthritis can occur.
- *Associated with renal tubular acidosis.* In this form the lack of carbonic anhydrase interferes with the acidification of the bony surface and interferes with the resorption by osteoclasts. Except anemia other features are same as that of malignant form.

Transmission

- Malignant form is transmitted as autosomal recessive.
- Benign or tarda form is transmitted as autosomal dominant.
- Tubular acidosis form is transmitted as autosomal recessive.

Clinical features

- Features of malignant form (see above).
- Pathological fractures and deformities
- Bone pain and back pain.
- Premature osteoarthritis.
- Osteomyelitis (mandible).

Radiological features
- Increased density of bone.
- Filling of inrtamedullary canal with bone.
- Endobone formation (bone within the bone).
- 'Rugger jersy' spine (as a result of sclerosis near the end plates of the vertebra)
- Dense skull bone with shallow fossae.

Orthopedic care
- Malignant form is treated by early bone marrow transplantation.
- Benign or tarda form does not require any active treatment unless pathological fracture occurs.
- Associated acidosis is treated by alkaline therapy.
- Pathological fractures in the pediatric age group are treated by closed reduction and cast application. Healing occurs eventually.
- Adult fractures need reduction and internal fixation. The procedure is technically highly demanding because drilling and reaming is extremely difficult in these bones.
- Joint replacement is done for premature degenerative arthritis.
- Complication of cranial nerve impingement is treated by foraminotomy and decompression
- Anemia and thrombocytopenia is treated by administration of erythropoietin.
- Interferon-gamma is used to allow bone resorption by enhancing superoxide production.
- Calcitriol in high doses can be given in an attempt to promote osteoclastic resorption.
- Medical treatment improves the condition but does not cure it.

Other Conditions

Paget's Disease

Described by Sir James Paget a British surgeon in the year 1877. Hence, the name is an Eponym.

It is considered as a disease, causing a disturbance in normal osteoclastic and osteoblastic activity of a bone there by causing weakening, deformities and pathological fractures. Thus it is also known as 'osteitis deformans'.

Etiology

a. *Viral*: Viral cause is attributed to certain viruses. Some of the viruses are paramyxovirus, canine distemper virus, etc.

b. *Genetic:* Paget's disease is seen among siblings which proves the presence of a hereditary factor.
Some genes have been identified, e.g. PDB2- 18q22.1 RANK.

Pathology

Bones affected are the bones of the spine, pelvis skull, thigh, and legs.

Generally, involvement is polyostotic. Very rarely monoostotic. The basic pathology is that of a massive turnover of bone with increased osteoclastic activity to begin with followed by increased osteoblastic activity.

Three distinct stages are recognized.

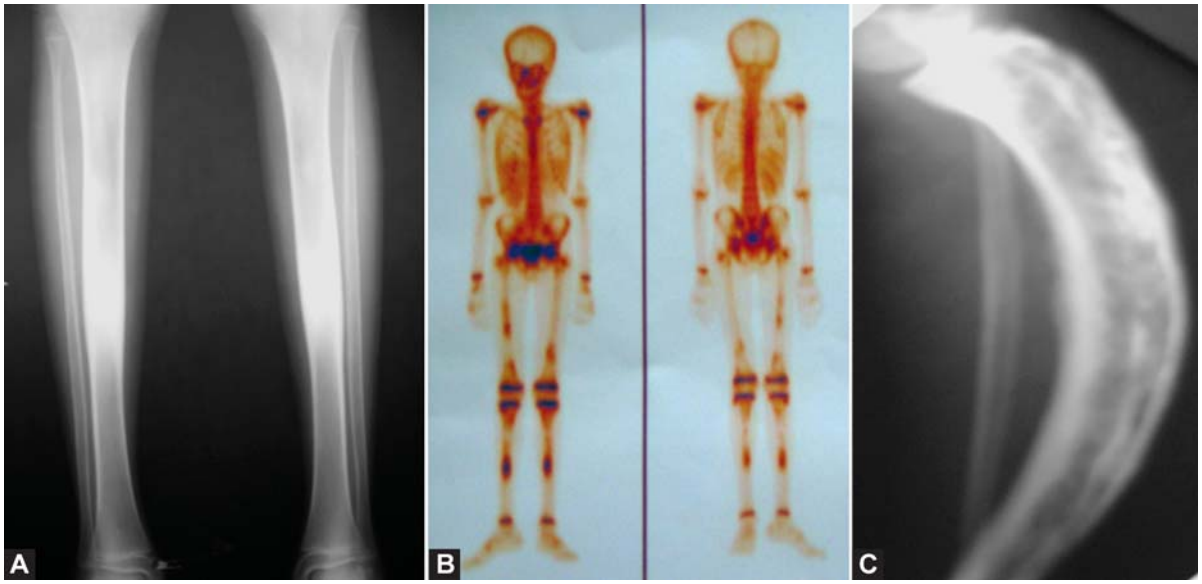
i. *Osteolytic or hypervascular phase*. In this stage there is increased osteoclastic activty followed by an intense osteoblastic activity which ultimately results in disorganised mass of primary woven bone formation in the areas affected. The blood flow increases to such an extent as to form AV shunts in the bone there by causing significant increase in cardiac out put and strain on the heart.

ii. *Intermediate phase*. In this phase there is mixed activity the predominant being the osteoblastic activity. Deformities begin to develop in this phase of the disease.

iii. *Burnt-out or quiescent phase*. The exaggerated activity stops. The vascular fibrous tissue replaces the marrow..
Absence of haversian system and development of sclerotic bone is characteristic.
Remodeling is poor and the quality of bone changes.

Clinical signs and symptoms
- Bone pain is the common feature.
- Constitutional symptoms like headache and drowsiness
- Neuritic pains along the nerves, e.g. in skull here the nerves get compressed in the foramina.
- Arthritis
- Deformities in long bones.
- Pathological fractures.

Investigations
- *Laboratory*
  i. Increased serum alkaline phosphotase with normal serum calcium and phosphorous.
  ii. Increase serum and urinary hydroxyproline which is an indicator of bone lysis.
- *Radiology (Figs 13.27A and C)*
  i. The involved bones show areas of lysis and sclerotic new bone formation. In the skull focal areas of lysis known a osteoporosis circumscripta may be present
  ii. Bowing and bending deformities are common.



Figures 13.27A to C

(A) Radiograph showing symmetrical hyperdense areas in the tibia suggestive of Paget's disease. (B) The bone scan shows increased uptake of the neucleotide. (C) The characteristic bowing of both anterior and posterior cortices of the tibia seen in a mono-ostotic Paget's disease.

- *Bone scan (Fig. 13.27B)*
  Shows increase uptake of the nucleotide in affected areas much before the radiological changes appear.
- *Histopathology*
  Shows excessive osteoclasts, irregular lamellar bone formation and replacement of the marrow by fibrous tissue.

Treatment Consists of two parts:

a. *Treatment of the pathology:* This is achieved by giving drugs to decrease the increased osteoclastic activity, e.g. Residronate at the same time by administering adequate amounts of calcium and vitamin D. Exposure to sunlight is advised. Cacitonin is the drug of choice in extensive disease as well as a preoperative adjuvant.
b. *Treatment of complications*
   i. Pathological fractures are fixed. Deformities are corrected by osteotomies.
   ii. Arthritis treated by total joint replacement when non-operative methods fail to give relief.
   iii. Secondary osteosarcoma is treated with amputation and necessary chemotherapy and radiotherapy.

Fibrous Dysplasia

Fibrous dysplasia is a nonhereditary condition. It occurs due to a spontaneous mutation in the gene that encodes the subunit of a stimulatory G protein (Gs-α) located on chromosome 20. Due to this there occurs a substitution of amino acid arginine for cysteine or histidine. Thus the osteoblasts lay down fibrous tissue instead of osteoid. This results in a gradual replacement of bone by fibrous tissue (during physiologic remodeling) and development of fibrous dysplasia.

Types of fibrous dysplasia

a. Mono-ostotic; single bone is involved.
b. Poly-ostotic; many bones are involved.
c. McCune–Albright syndrome
   Poly-ostotic fibrous dysplasia, precocious sexual puberty, café au lait skin lesions.
d. Mazabraud syndrome (Mazabraud and Girard 1957)
   Polyostotic fibrous dysplasia with skeletal muscle myxomas.

Clinical signs and symptoms

i. Polyostotic variety presents early in childhood. Mono-ostotic variety presents as late as at 30 yrs of age.
ii. Medical attention is sought for either bone pain, pathological fracture or the deformity.

Diagnosis

a. X-ray—Typical ground glass appearence is seen radiologically (Fig. 13.28).

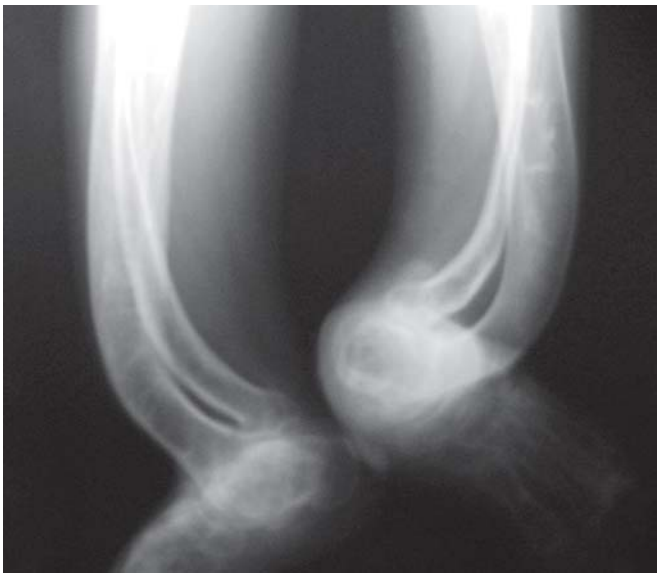


Figure 13.28

Radiograph of tibia and fibula of both the legs showing the ground glass appearence and bending deformity of fibrous dysplasia.

b. Histopathology—shows the trabeculae of woven bone embedded with fluid filled cysts. The surrounding matrix is largely fibrous.

Treatment

- *Drug therapy of fibrous dysplasia*
  Bisposphonates by their inhibitory action on osteoclasts are found to be useful. Intravenous Pamidronate is being used in pediatric age group. Supplementation of calcium and vitamin D is a must.
- *Operative treatment*
  Those present with pathological fractures and deformities are treated surgically with internal fixation and corrective osteotomies. Intramedullay devices should be preferred and used for internal fixation.

Osteogenesis Imperfecta

This condition is also known by several names some are synonyms and others are eponyms.

- Periosteal dysplasia.
- Fragilitas osseum.
- Idiopathic ostopsathyrosis.
- Brittle bone disease.
- Vrolik's disease.
- Lobsteins disease (1835).

The condition is a hereditary one characterized by fragile bones, blue sclerae, deafness, laxity of joints and a tendency towards improvement with age.

Pathology

The disease affects both endochondral and intramembranous ossification.

The initiation of osteogenesis proceeds normally up to the stage of infiltration by osteoblasts .

At this stage the invasion of the matrix takes place by abnormal osteoblasts which are incapable of laying down normal type-I collagen to form the osteoid suitable for adequate mineralization. Very few osteocytes appear and the bone that is laid down is thin and weak.

The trabeculae formed are disorganized, sparse, thin and delicate. Thus the stress tolerance is poor and there is a tendency for easy fracture.

Classification

Based on Shapiro's classification following types are recognized depending on the age of presentation.

I. Osteogenesis imperfecta congenita
   A. Fractures occur in utero or at birth. The short broad crumpled femora and ribs.
   B. Fractures occur in utero or at birth. The long bone contours are normal. No chest deformities

II. Osteogenesis impefecta tarda
   A. Fractures occur after birth but before walking.
   B. Fracture occur after the child starts walking.

Based on genetic abnormality clinical features or both, I-IV types have been described.

Type I—Dominant with blue sclera (Autosomal dominant)

Type II—Lethal perinatal (Autosomal recessive)

Type III—Progressive deformity. Sclerae become less blue with age (Autosomal recessive.)

Type IV—Dominant with normal sclerae (Autosomal dominant).

Clinical features

The features vary depending on the severity of the condition. Fracture is the sign with which a child presents.

1. *Fetal and prenatal form:* This the severe form of the disease. Stillbirth and death in the early newborn period is common. Life expectancy is poor.
2. *Infantile form:* This form is less severe when compared to foetal form. If the child survives the initial few weeks may survive for long.

3. *Adolescent form:* This is identified late in childhood and adolescence when the child sustains a fracture with a relatively trivial trauma.

   On examination following findings are detected:
   - Blue sclera.
   - Deafness.
   - Laxity of joints.
   - Short stature.
   - Broad skull.
   - Dentigerous imperfecta (poor calcification of teeth).

Investigations

- *X-ray- shows*
  - Thin bone with bulbous ends.
  - Pencil line cortex.
  - Thin sparse trabeculae.
  - Plastic bowing may be seen.
  - Fractures in various stages of healing may be seen.
  - Kyphoscoliotic deformity in the spine may be present.
  - Popcorn calcification at the distal ends of long bones.
- *Laboratory investigations*
  - Calcium and phosphorous are normal. Alkaline phosphotase is elevated.

Treatment

Following are the aims of treatment.

A. To strengthen the bone by giving Bisphosphonates.
B. To prevent the pathological fractures.
C. To correct the deformities.

Prognosis

In lethal variety there are no survivals. Stillbirth and perinatal death are common.

In other less severe varieties, repeated fractures are common and the child will remain stunted in growth . Deformities are commonly seen.

Note: Any of the above conditions can be asked as a short notes or a long question.

Short notes

Write notes on:

a. Skeletal dysplasias.
b. Achondroplasia.
c. Hypochondroplasia.
d. Pagets diseae.
e. Osteopetrosis.
f. Fibrous dysplasia.
g. Osteogenesis Imperfecta.

Further Reading

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14

Miscellaneous Conditions

- *Mallet Finger*
- *Boutonniere Deformity*
- *Trigger Finger/Thumb*
- *de Quervain's Disease*
- *Dupuytren's Contracture*
- *Carpal Tunnel Syndrome*
- *Tennis Elbow*
- *Golfer's Elbow*
- *Supraspinatus Tendinitis*
- *Periarthritis Shoulder*
- *Calcaneal Spur*
- *Neuropathic Joint*
- *Congenital Torticollis or Wry Neck*
- *Idiopathic Avascular Necrosis of the Head of the Femur*
- *Spondylolisthesis*
- *Bursa and Bursitis*

Mallet Finger

Loss of continuity of the extensor mechanism at the distal interphalangeal joint leads to a flexion deformity. This is known as mallet finger.

Etiology

A direct blow to a finger tip which is rigidly held in extension, causes a sudden flexion at the DIP joint of the finger causing:

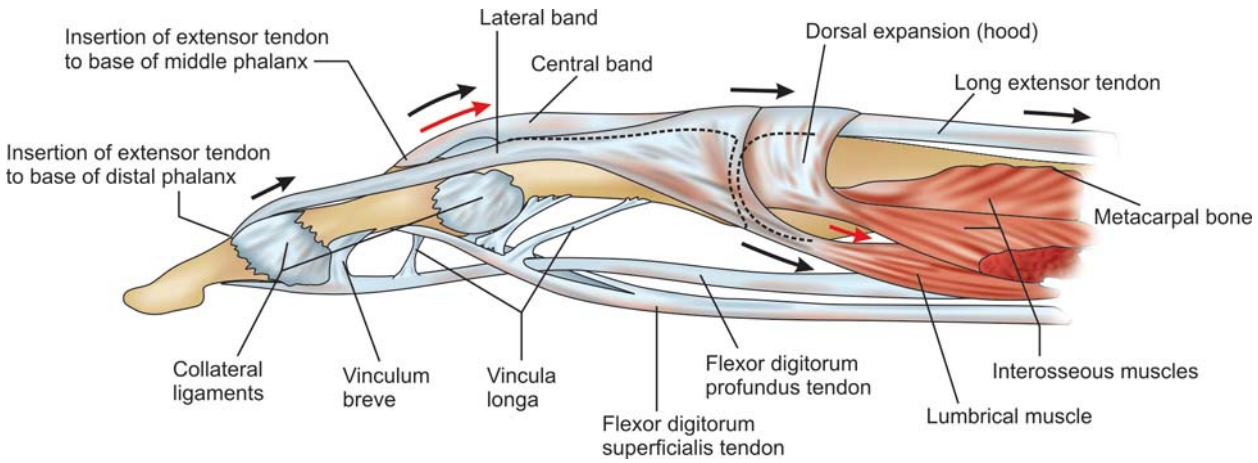


Figure 14.1A

Showing the insertion of the central slip and the lateral slips to the middle and the distal phalanx respectively.

a. The rupture of the lateral slips of the extensor mechanism.
b. An avulsion fracture of the base of the distal phalanx, e.g a base ball or a volley ball injury (Fig. 14.1A and ref. Fig. 1.8).

Treatment

Soft tissue injuries are treated by mallet finger splints and immobilized for a period of three weeks followed by gradual mobilization. Different splits are available.

Avulsion fractures are fixed by using K-wires/pull-out sutures by an expert. Preferably by a surgeon who is trained in hand surgery.

Boutonniere Deformity

Loss of continuity of extensor mechanism at the PIP joint results in flexion deformity at the PIP joint and hyperextension at the DIP joint. This is known as Boutonniere deformity.

Etiology

It occurs due to the rupture of the central slip of the extensor mechanism as a result of trauma or inflammation.

a. Trauma

In trauma it can occur as a result of a direct penetrating injury or a forcible flexion at the PIP joint in a finger held firmly in extension or in a volar dislocation of the PIP joint.

b. Rheumatoid Arthritis

Rupture occurs because of the synovitis occurring in the PIP joint. This slowly flexes the joint and stretches the central slip which ultimately becomes thinner as a result of inflammation and stretching and ultimately gets ruptured.

Treatment

Splinting is the first option in closed injuries as well as in early rheumatoid pathology.

Surgical repair and reconstruction is the option in total rupture.

Trigger Finger/Thumb

It is known as trigger finger because a bent and locked finger opens with a snap, i.e. like a trigger, on straightening.

Etiology

More common in women than in men and in diabetics. Those who are involved in manual labor which involves grippng of a hard object are at a greater risk of developing trigger finger. This is the result of inflammation of the tendon sheath of the flexor tendon of the finger. As the sheath gets thickened the canal for the gliding of the tendon narrows and triggering develops.

Treatment

Begins with nonoperative measures in mild and early cases. If symptoms are not relieved surgical release is done.

a. Nonoperatively treated by anti-inflammatory drugs and steroid infiltration into the flexor tendon sheath.
b. Surgically treated by the release of the flexor tendon sheath. This relieves the symptoms permanently.

de Quervain's Disease

Named after Fritz de Quervain a Swiss surgeon who described it in 1895.

Also known as de Quervain's syndrome, Washerwoman's sprain, Mother's wrist, it is an inflammation of the tendon sheath or the tunnel that surrounds the tendons of abductor pollicis longus and extensor pollicis brevis. Commonly seen in women and is thought to be due to greater radial styloid process angle.

Symptoms

Pain swelling and tenderness on the radial side of the wrist with difficulty in griping the object.

Finkelstein's Test

The patient is asked to make a fist. The test is performed by passively ulnar deviating the fist. This results in exaggeration of pain at the inflammed site.

Treatment

a. Nonoperatively treated by giving rest to the involved hand using splints, administration of anti-inflammatory drugs and infiltration with steroids.
b. Surgically treated by release of the sheath. This gives permanent relief. Superficial branch of the radial nerve is to be taken care of during the procedure.

Dupuytren's Contracture

It is the contracture of the palmar aponeurosis which results in the flexion deformity of the fingers. Ring and the little fingers are commonly involved.

Named after Baron Dupuytren who described the condition in 1831. He attributed it to holding the reins of the horse for many years. This was found not to be true.

Structure of the Palmar Aponeurosis

The palmar aponeurosis (fascia) is nothing but thick condensation of the deep fascia. It is triangular in shape and attached

Proximally—to the Flexor retinaculum and Palmaris longus tendon.

Laterally—covers the thenar muscles of the hand.

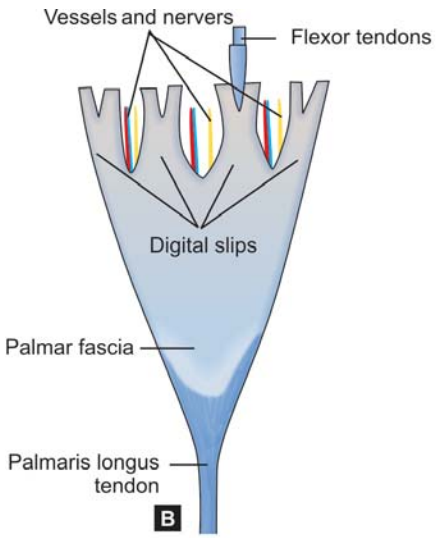
Medially—covers the hypothenar muscles.

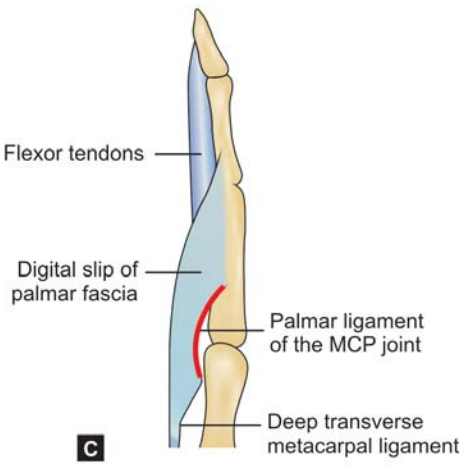
Distally—divides into four major slips. Each of the major slip bifurcates into two and get attached to the Transverse metacarpal ligament , the Fibrous flexor sheath and to the sides of the whole of the proximal phalanx and proximal part of the middle phalanx of each digit. It is not attached to the distal phalanx at all (Figs 14.1B and C).

(Hence in Dupuytren's contracture the distal interphalangeal joint does not develop flexion contracture but remains extended).

Etiology

It shows a genetic predisposition in some. Diabetes, epilepsy and alcohol dependency are the other predisposing factors.





Figures 14.1B and C

Showing the palmar aponeurosis, its division and attachment to the deep transverse metacarpal ligament. The digital vessels and nerves, the lumbricals and the flexor tendons emerge from underneath the slips and enter the digits.

Treatment

Minor deformities are treated nonoperatively and patients are advised to take care of the predisposing factors, e.g. alcohol intake to be reduced.

In those cases where the contracture is severe and painful surgical excision of the contracted tissue is advised.

Carpal Tunnel Syndrome

It is the compression of the median nerve in the carpal tunnel causing numbness in the hand. If left untreated paralysis of the thenar muscles takes place over a period of time. Rarely severe sensory disturbance and trophic ulcers can develop (Fig. 14.2).

Attachment of Flexor retinaculum:

Medially

i. Pisiform
ii. Hook of hamate

Laterally

i. Tubercle of scaphoid
ii. Crest of trapezium.

Etiology

For a compression to occur either the volume of the contents in the canal should increase or the amount of space in the canal should decrease.

a. Space occupying lesions in the canal such as ganglion, tenosynovitis, lipoma, etc. increase the volume of the contents in the canal and cause compression.

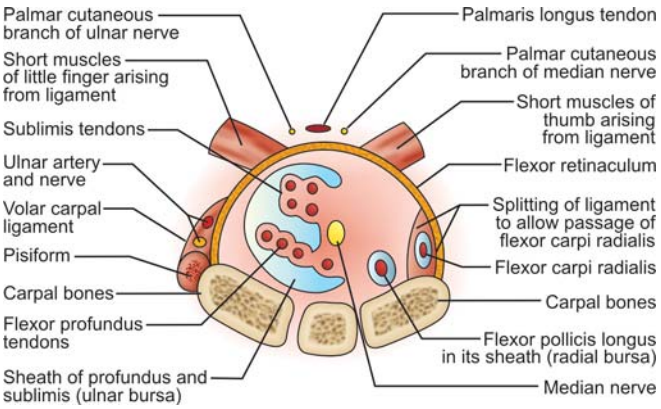


Figure 14.2

Showing the structures under the Flexor retinaculum and the boundaries and the contents of the Carpal tunnel.

b. Malunited Colles' fracture, degenerative and inflammatory arthritis of the wrist decrease the amount of space in the canal and cause the compression.
c. Physiological cause is pregnancy. The compression is transitory. It becomes normal after delivery.

Diagnosis

a. Patient presents with diminished sensation and pain along the distribution of the median nerve in the hand. Starts initially at night and then becomes more constant. Interferes with daily activities.
b. Phalen's test positive (Figs 14.3A and B).
c. Tinel's sign positive.
d. Nerve conduction velocity shows delay in conduction.

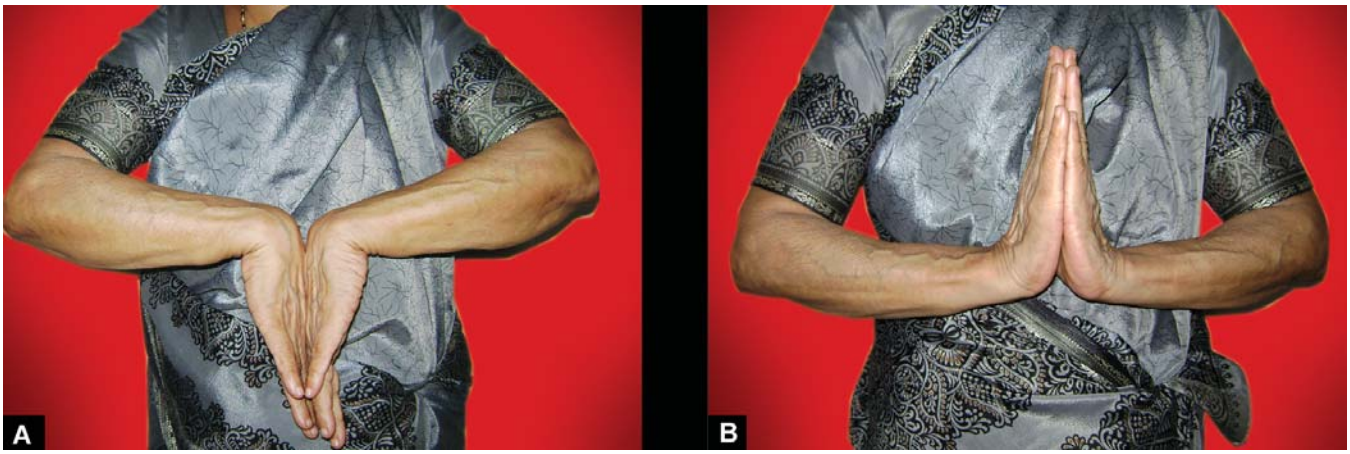
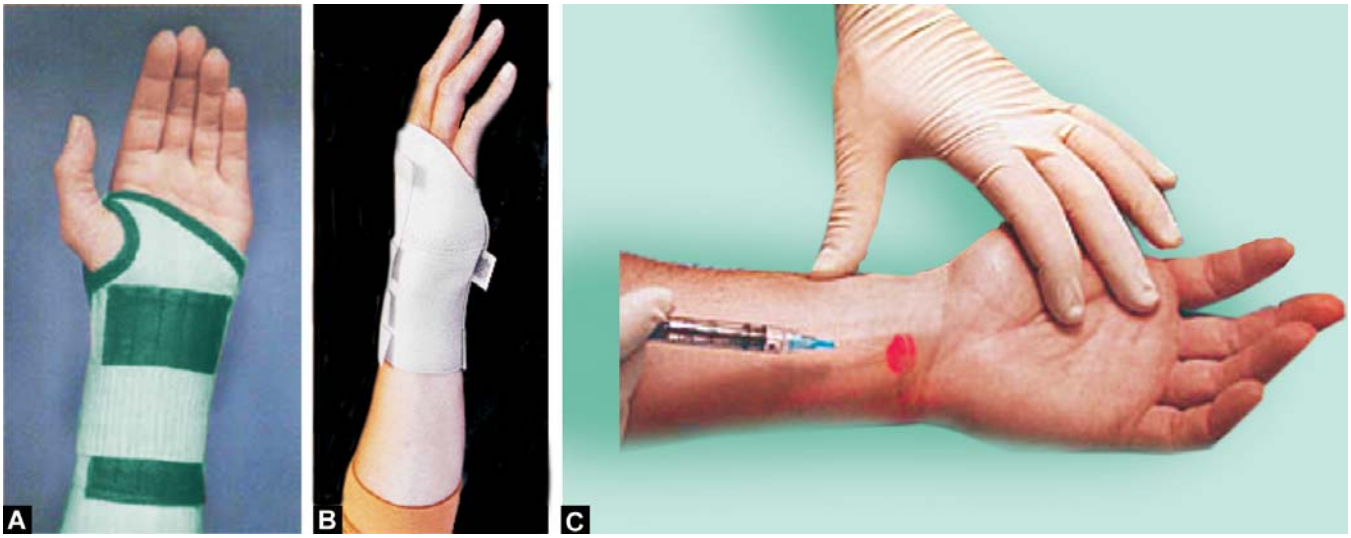


Figure 14.3

Picture showing (A) Phalen's test (B) Reverse Phalen's test



Figures 14.4A to C

(A and B) Pictures showing supportive splints for carpal tunnel syndrome. (C) Picture showing steroid injection into the carpal tunnel.

Treatment

Nonoperative treatment is indicated if the compression is not due to space occupying lesions (Figs 14.4A to C).

Measures undertaken are rest to the part, supportive splints and anti-inflammatory drug therapy. Infiltration of the tunnel with steroids can also be tried. If no relief is obtained surgical decompression is undertaken by dividing the flexor retinaculum at the wrist.

When space occupying lesions are detected the tunnel has to be explored. The lesion has to be excised and the tunnel is deroofed by not suturing the divided flexor retinaculum.

Tennis Elbow

It is also known as lateral epicondylitis. It occurs due to repetitive stretch of the common extensor origin at the lateral epicondyle. Common in tennis players especially during the back hand stroke. Hence the name is derived. It is also common in manual laborers, e.g. plumbers, gardeners, etc.

Pathology

Repetitive stretch causes microtears resulting in inflammation, degeneration and fibrosis of the common extensor origin there by making it less elastic. Extensor carpi radialis brevis is commonly affected (Fig. 14.5).

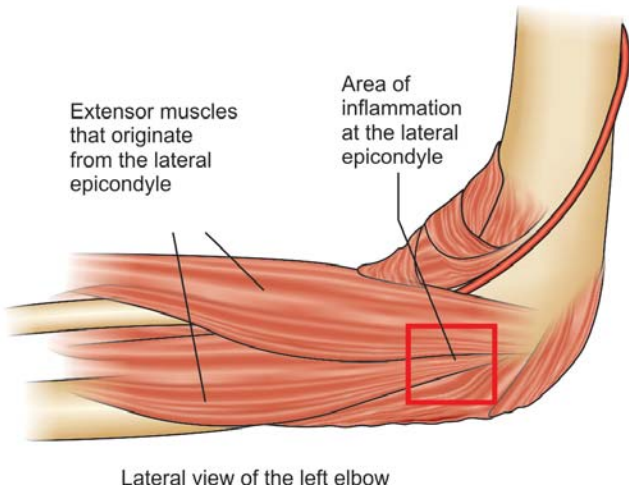


Figure 14.5

Showing the common extensor origin of muscles at the lateral condyle.

Diagnosis

Tennis elbow is diagnosed by a specific test known as Cozen's test (Fig. 14.6). The patient is asked to hold the limb with elbow in flexion and the forearm in full pronation. Then asked to dorsiflex and radially deviate the wrist while resistance is applied. Severe pain is observed at the common extensor origin.

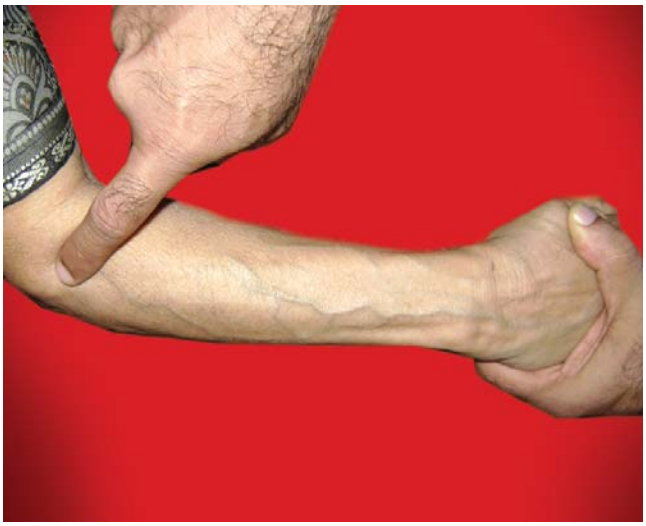


Figure 14.6
Picture showing Cozen test.

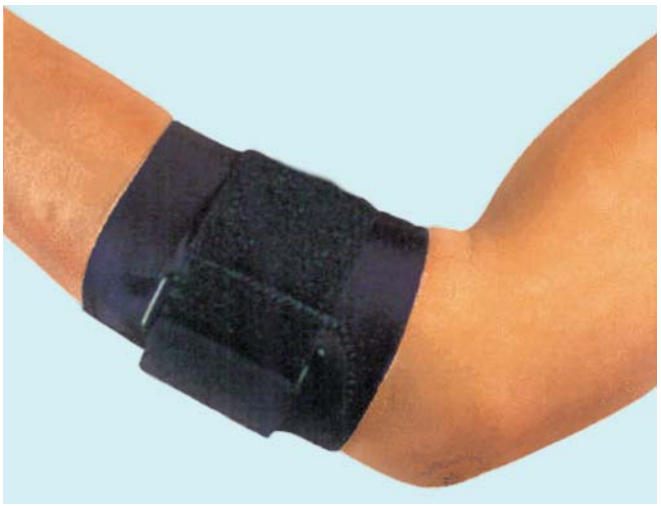


Figure 14.7
Showing the tennis elbow strap.

Treatment

a. Avoid repetitive stretch by not doing such activities which cause pain and discomfort till healing occurs. This is followed by gradual resumption of activities.
b. Wearing supportive splints and straps for tennis elbow (Fig. 14.7).
c. Ice packs for acute episode and heat therapy for chronic.
d. Anti-inflammatory drugs.
e. Corticosteroid injections.
f. Exercise therapy and occupation therapy.

Golfer's Elbow

It is known as medial epicondylitis and commonly seen in golf players. Hence the name. It results due to chronic repetitive stretch of the common flexor origin at the medial epicondyle. Pathology is similar to that of tennis elbow.

Diagnosis

By asking the patient to hold the limb straight with the elbow in extension. Then he is asked to make a fist and flex the wrist while resistance is applied. Patient complains of severe pain.

Treatment

Same manner as in tennis elbow.

Supraspinatus Tendinitis

It is the inflammation of the supraspinatus tendon due to minor degrees of trauma. Results in difficulty to initiate abduction. Diagnosed by the presence of a tender spot over the tendon and difficulty in initiation of abduction.

Treatment

It is treated by rest to the part anti-inflammatory drugs and local cortisone injections.

Current belief is that the condition is a part of the shoulder impingement syndrome and it is the impingement of the supraspinatus tendon leads to inflammation.

Calcification

It was Codman who first identified calcification in the tendons of the rotator cuff. The exact cause is not known. It is thought that poor blood supply and attrition causes a conducive atmosphere for calcification (Figs 14.8 and 14.9).

Symptoms

Patient presents with acute onset of pain, worse at night along with limitation of abduction of the shoulder. X-ray confirms the diagnosis.

Treatment

a. Ice packs to the shoulder.
b. Anti-inflammatory drugs.
c. Corticosteroid injections.
d. Large deposits which do not respond to nonoperative methods are excised by open/arthroscopic surgery.

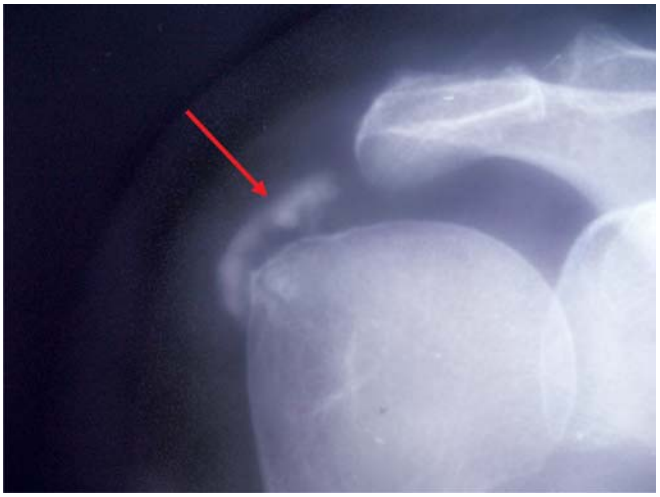


Figure 14.8

Radiograph showing calcific supraspinatus tendinitis. The amorphous nature shows resolution phase of the lesion.

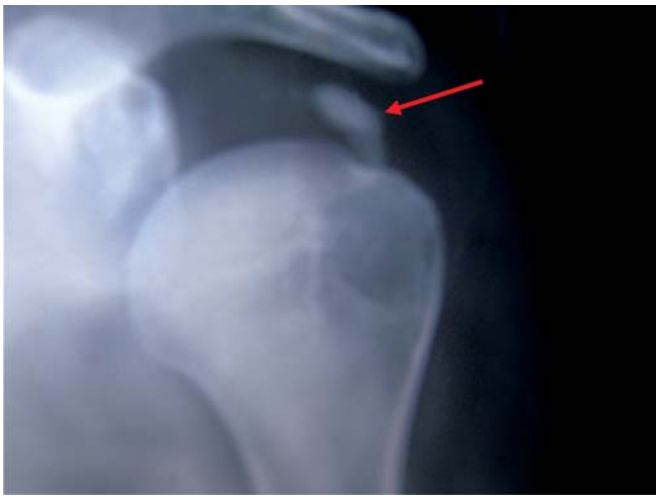


Figure 14.9

Radiograph showing uniformly dense deposit. This indicates the acute phase of calcific supraspinatus tendinitis.

Periarthritis Shoulder

It is also known as Adhesive Capsulitis and Frozen Shoulder. Also referred to as the shoulder of fifties because the condition is very common around this age. It presents with gradual painful restriction of all the movements of the shoulder. Thought to be due to a retrograde and inflammatory degeneration of the shoulder joint capsule and the soft tissues surrounding it.

Clinical Features

a. Pain of insidious onset. Worse at night. Increases on movement.
b. Gradual onset of painful limitation of all the movements. Abduction and external rotation in particular.
c. Tenderness all round the shoulder with more than one tender spot.

X-ray—shows rarefaction of the head of the humerus. Degenerative changes may be seen in acromioclavicular joint.

Treatment

a. Anti-inflammatory drugs.
b. Gradual and active mobilization of the shoulder.
c. Physiotherapeutic application of Moist heat therapy, Ultrasound therapy etc.
d. Manipulation under anesthesia followed by exercise therapy.
e. Infiltration with corticosteroids.

Calcaneal Spur

It is an ossification/ calcification occurring at the insertion of the plantar fascia to the periosteum on the undersurface of the calcaneus (Fig. 14.10).

Etiology

Repetitive stress especially unprepared for, results in stretching of the plantar fascia and microtears. Ultimately, calcium

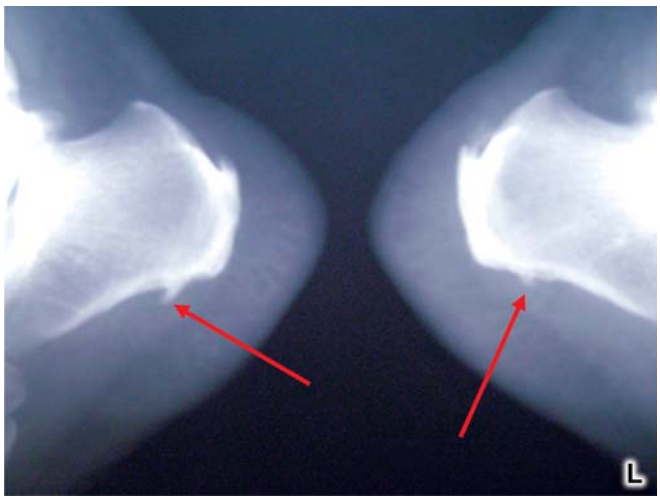


Figure 14.10

Radiograph showing bilateral calcaneal spur. See the sharp pointed nature of the spur on the right side and amorphous appearance on the left side. The spur on the right side was asymptomatic. It was the spur on the left side, which is just developing, that was painful.

gets deposited and a spur develops. Thus spur is the result of the inflammation. The symptoms are due to the inflammation and are present during the early process of spur formation and not after the spur formation is complete. This is proved beyond doubt because many spurs are found as an incidental finding. They are totally asymptomatic. But heel pain can occur without the presence of a spur.

Symptoms

a. Pain on the undersurface of the heel which is relieved by rest and starts again at the initiation of activity.
b. Classically worst when a person gets up in the morning.
c. Aggravated by walking on a hard surface.

Treatment

a. Anti-inflammatory drugs.
b. Soft foot wear/ cushioned heel.
c. Physiotherapeutic measures.
d. Reducing unaccustomed stress.

Neuropathic Joint

Also known as Charcot's joint is named after Charcot who first described this condition in a case of Tabes Dorsalis in the year 1868. Steindler was the first person to observe the Destructive Atrophic form and Hypertrophic Proliferative form of this disease (Fig. 14.11).

Etiology

The cause is neuropathia occurring secondary to:

i. Tabes dorsalis.
ii. Hansen's disease.
iii. Diabetic neuropathy.
iv. Syringomyelia.
v. Peripheral nerve lesions.
vi. Hereditary sensory neuropathy.

Pathology

The pathological process begins with an injury. A single injury heals uneventfully even in a neuropathic joint when diagnosed and treated adequately. Animal experiments have proved this fact beyond doubt. It is the repetitive trauma which is known to initiate the degenerative process. Though the joint is senseless, it responds to injury by causing inflammation. The joint shows all the signs of inflammation such as calor, rubor and tumor except dolor which is absent. This is the initial

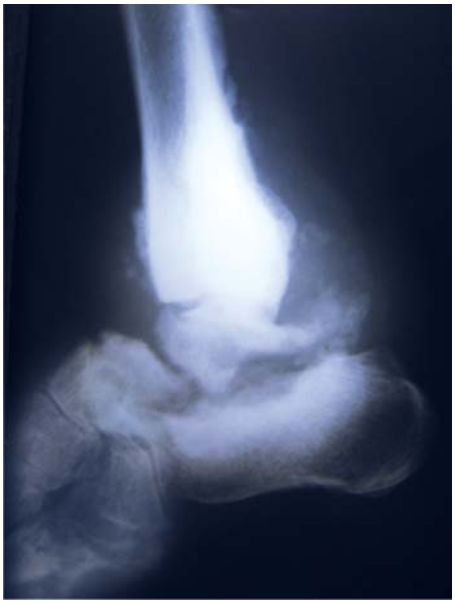


Figure 14.11

Radiograph showing classical changes of Charcot's joint. Note extensive degeneration and disorganization of ankle, subtalar, tanonavicular and calcaneocuboid joints. Shape of the talus is totally altered.
New bone formation seen is an attempt at healing of fractures. Despite of such severe alterations the joint is totally painless.

Destructive Atrophic phase of the pathology where there is hyperemia. Hyperemia is necessary to clear the debris. Since patient does not experience pain there is a likely hood that the initial injury is neglected and this injured hyperaemic joint is exposed to repetitive stress and further injury. Thus a vicious cycle sets in. Thus the initial inflammatory reaction that had set in becomes persistent and prolonged. This causes progressive degeneration and formation of debris. The joint in this stage is badly swollen, shows increased warmth and redness. If the joint is not protected at this stage it leads to total destruction and disorganization.

If the joint is protected the process of repair begins. Repair results in the formation of dense fibrous tissue in the surrounding soft tissue and dense sclerotic bone on either side of the joint. Massive callus may form in the soft tissue. Lipping may develop at the margins of the joint. This is the Hypertrophic Proliferative form of the disease which is the phase of repair. The signs of inflammation decrease and no calor, rubor and tumor is observed during this phase.

Clinical Features

There is definite history of having neuropathic problem.
a. A badly swollen joint due to hemarthrosis/effusion.

b. Signs of inflammation present in the acute phase.
c. Lack of pain is the classical feature.
d. Exaggerated movements and laxity is the feature in later stages.
e. Deformity and total disorganization is the feature in the final stage.

Diagnosis

X-ray—shows classical features of a neuropathic joint with destruction and disorganization out of proportion to clinical symptoms (Fig. 14.11).

Treatment

The proverb *Prevention is better than cure* is very apt for neuropathic joint. Inadequately protected injuries of the joint are the ones which are responsible for destruction.

a. Protection of the joint is of great importance when neuropathia is suspected in order to prevent the onset of the degenerative process.
b. Once the changes have taken place the aim of treatment is to prevent further degeneration and damage. This is achieved through occupational guidance and advocating protective splints and calipers. Joint aspiration may be indicated to empty the collected fluid. Protection should be continued till warmth and redness subsides. This is followed by measures which prevent further injury during activities of daily living.
c. Arthrodesis is done for a totally destroyed and disorganized joint.

Arthrodesis should never be done during the acute inflammatory phase. It should always be attempted after healing has occurred. X-rays help to confirm healing. The dense sclerotic bone has to be excised till the raw bleeding bone is reached, in order to achieve sound fusion.

When the joint is badly infected amputation may have to be considered.

Congenital Torticollis or Wry Neck

It is a condition seen at birth due to fibromatosis of the sternomastoid muscle. The incidence of associated DDH is reported to be from 7-20%.

Natural Course of the Fibromatosis

It manifests as a palpable tumor at birth which attains a maximum size within 1-2 months and then starts regressing. If does not disappear within an year, fibrosis occurs which results in a fixed deformity.

Clinical Features

i. Inclination of the head to the same side and turning of the face to the opposite side.
ii. Elevation of the ipsilateral shoulder.
iii. Decrease in fronto-occipital diameter of the skull.

Diagnosis

It is not difficult to diagnose Torticollis. Diagnosis is simple and is made by inspection, confirmed by palpation (sternomastoid tumor) and elicitating limitation of movements of the neck.

Treatment

Treatment should start at birth. Up to 1 year of age the child is managed nonoperatively by advocating passive stretching and maintaining the position in sleep.

Role of Surgery

Surgery is done only when the nonoperative treatment fails. Ideal age of surgery is at 1 year. Surgery should never be done in infancy as there is good chance of regression with growth and nonoperative treatment.

Type of Surgeries

i. Release of the clavicular end of the muscle.
ii. Release at both the clavicular and the mastoid end.
iii. 'Z' lengthening.

Prognosis

Prognosis is good. The deformity is fully correctable when the surgery is done early (at 1 year) before the deformity gets established.

Idiopathic Avascular Necrosis of the Head of the Femur

Other Names

- Aseptic necrosis.
- Osteonecrosis.
- Ischemic bone necrosis.

It is a condition of unknown etiology occurring between the age of 20 and 40 years. Men are more commonly affected than the women.

Clinical Features

i. Dull aching pain in the hip to begin with.
ii. Pain progressively becomes severe and painful limitation of movement develops.
iii. Fixed deformities in later stages.

Staging: Ficat and Arlet (1980)

(Based on this classification 4 stages were identified)

Stage I: Showed no X-ray changes and diagnosis was made by measurement of intraosseous pressure and bone biopsy.

Stage II: The femoral head contour was still normal but there were early signs of reactive changes in the subchondral area. Crescent sign (crescent-shaped area of increased density in the subchondral region.)

Stage III: It was defined by clear cut X-ray signs of osteonecrosis with evidence of structural damage and distortion of the bone outline.

Stage IV: There was collapse of the articular surface and signs of secondary OA.

These days MRI is used in the early diagnosis (a band like low intensity signal on the T1- weighted image). Also the disease is staged based on MRI findings.

Radiological Features of AVN

- Increased density of the subchondral bone.
- Maintenance of joint space until late period of the disease which is in contrast to osteoarthritis and rheumatoid arthritis.
- Secondary osteoarthritic changes in advanced stage.

Treatment

Stages I and II: Femoral core decompression with or without fibular strut grafting.

Stages III and IV: Total hip replacement.

Known causes of AVN of femoral head.

Traumatic causes
- Fracture neck of femur/ femoral head
- Dislocation around hip
- Slipped capital femoral epiphysis

Nontraumatic causes
- Massive arterial occlusion
- Perthes' disease
- Venous thrombosis
- Alcoholism
- Hypercortisonism/ steroid intake
- Caison's disease
- Sickle cell disease
- Gaucher's disease
- Infections
- Familial hyperlipidemias

Spondylolisthesis

Spondy—means Spine. Olisthesis – means slipping in Greek language .Thus the word Spondylolisthesis is derived which refers to slipping of one vertebra over the other. This condition was first described by a Belgian obstetrician Dr. Herbinaux in the year 1782, when he found a bone anterior to the sacrum obstructing the vagina.

For a slip to occur a defect should develop in the stabilizing structures namely the aritcular facets, the neural arch or the normal bony structure. The lumbosacral area is the common site where this slip occurs . The slip in majority of the cases is in a forward direction because of the lumbar lordosis. When the slip is backwards it is known as Retrolisthesis.

Types: Based on Wiltse Classification (Fig. 14.12)

i. Dysplastic (congenital).
ii. Isthmic (defect/spodylolysis in Pars interarticularis).
iii. Degenerative.
iv. Traumatic.
v. Pathologic.

Etiopathogenesis

Defect in any of the stabilizing structures can lead to listhesis. Some times there can be more than one pathology.

In dysplastic variety there is agenesis or hypoplasia of the facets . This leads to the slip . It is one of the severe forms of listhesis.

In the isthmic variety there is a defect in pars interarticularis.

Pars—means Part. Inter—means between and articularis—means articular components (Facet.)

It refers to the part between the superior and the inferior articular facets. When there is defect (which is also known as spondylolysis) in this area two segments develop in the neural arch.

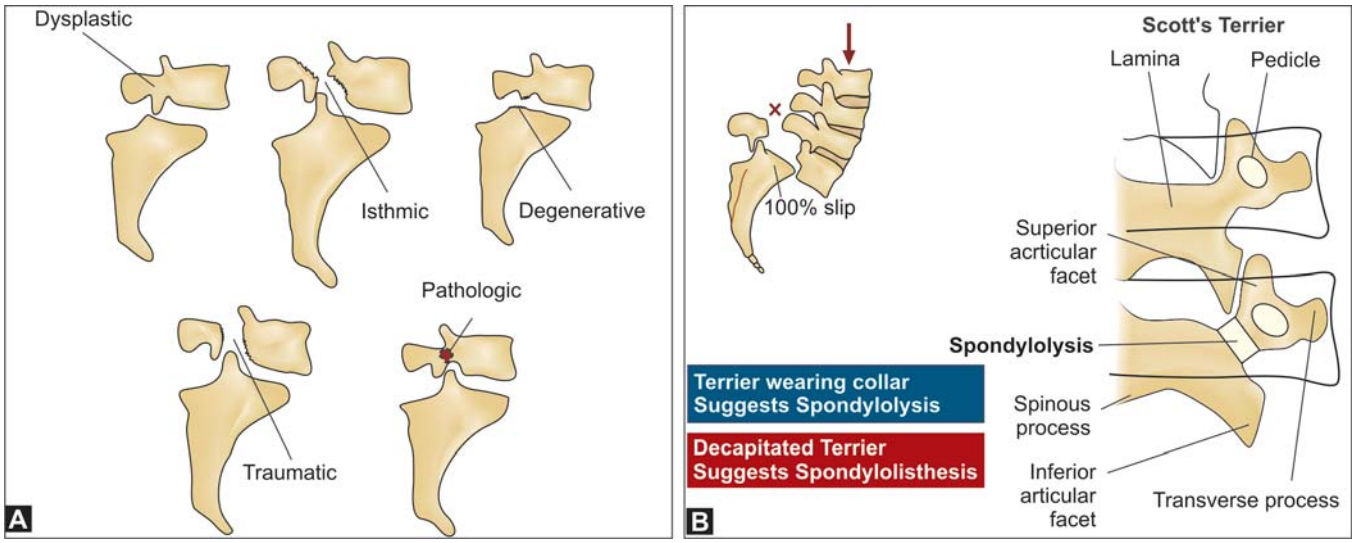


Figure 14.12

Picture showing different types of Spondylolisthesis and radiographic picture of Scott's Terrier

a. A posterior segment consisting of the spinous process, lamina and the inferior articular facet.
b. An anterior segment consisting of the pedicle transverse process and the superior articular facet.

With stress the anterior segment along with the vertebral body and the column slips forwards whereas the posterior segment remains connected to the sacrum.

In degenerative variety the degeneration involves the facetal articulation and results in instability and slip.

Traumatic type of slip results when there is trauma to the facets or the fracture of pars interarticularis.

Pathologic type is seen when the facets and the pars interarticularis are involved in a neoplasia or infection like tuberculosis and is destoyred.

Traumatic and pathologic types are very rare.

Grading of the severity of the slip:
Based on Meyerding grading system 4 grades are recognized

Grade 1	0-25% slip
Grade 2	25-50% slip
Grade 3	50-75% slip
Grade 4	75-100% slip.

Beyond 100% the word spondyloptosis is used to denote total dissociation from the lower vertebra.

Clinical Symptoms and Signs

Backache and varying grades of neurologic deficits are the main symptoms and signs. They are directly proportional to the severity of the slip. Flattening of the buttocks and prominence of the loin crease are the local findings. The deformity becomes more pronounced as the severity progresses and a step may be palpated at the site of listhesis.

Diagnosis

X-ray—confirms the diagnosis.
CT and MRI—Delineate the pathology better especially when there are neurological signs.

Treatment

Aim of treatment is to prevent further slip

Nonoperative

It is indicated only in minor grades of slip with little or no neurological signs.

Measures include modification of activity, refraining from strenuous activity, back exercises, spinal corset for support, drug therapy and regular follow-up to monitor progression.

Operative

This treatment is always indicated in severe forms with neurological deficit. Spinal fusion is achieved and further slip is prevented. In degenerative type decompression may be necessary.

Bursa and Bursitis

Bursa is a sac of fibrous tisue lined by synovial membrane, containing synovial fluid. Generally they are situated around the joint between the bone and the insertion of a tendon. Some times a bursa develops at a place which is subjected to constant stress. This is known as adventitious bursa. e.g dorsum of the foot in neglected CTEV. When the bursa gets inflammed the condition is known as Bursitis.

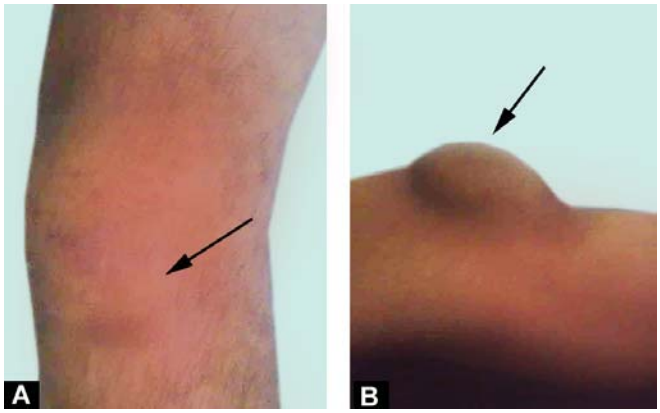
Common sites of bursae in the human body:

A. Around the shoulder
   i. Subdeltoid.
   ii. Subacromial.
   iii. Subscapular.
   iv. Under the Infraspinatus.
   v. In the synovial sheath of the long head of biceps.

B. Around the elbow
   i. Two in realtion to triceps at its insertion. One at the upper part of the olecrenon and the other at the lower part .
   ii. Two in relation to the biceps at its insertion to the radial tuberosity.

C. Around the hip
   i. Posterior – Four under the Gluteus maximus, one under the Gluteus medius and one under the Gluteues minimus.
   ii. Anterior – One under the Psoas tendon. (This might communicate with the hip joint)

D. Around the knee
   i. Anterior: There are four bursae anteriorly namely, a Suprapatellar, a Prepatellar and two Infra-patellar. One between the patellar tendon and the tibia and the other between the tibial tuberosity and the skin.
   ii. Posterior: There are two bursae posteriorly one each between the heads of the Gastronemius and the capsule of the knee joint.
   iii. Medially: Two bursae are present medially one between the Tibial collateral ligament and the Sartorius,Gracilis and Semi tendinosus and the other between the Tibial collateral ligament and the Semimembranosus.
   iv. Lateral: There are three bursae laterally. One between the Fibular collateral ligament and the Biceps, the second between the Fibular collateral ligament and the Popliteus and the third is between the Popliteus and the Lateral femoral condyle.



Figures 14.13A and B

Showing a infected infrapatellar bursitis (Clergy man's knee) and B prepatellar bursitis (House maids knee).

Some of the names of commonly seen bursitis:

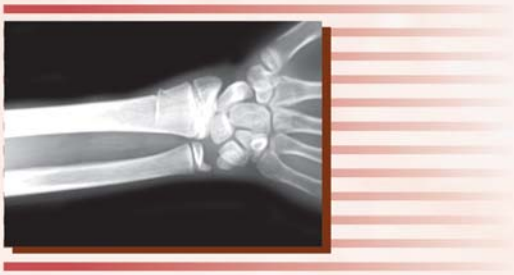
- Housemaids knee—Prepatellar bursitis (Fig. 14.13A).
- Clergyman's knee—Infrapatellar bursitis (Fig. 14.13B).
- Tailor's ankle—Advetitious bursitis in the region of lateral malleolus.
- Porter's shoulder—Advetitious bursitis in the in the region of lateral end of Clavicle and the skin.
- Weaver's Bottom—Advetitious bursitis between Gluteus maximus and Ischial tuberosity.

Treatment of Bursitis

Treated with rest and supportive bandage along with anti-inflammatory drugs, if the cause is aseptic inflammation.

If the cause is sepsis along with anti-inflammatory drugs, Adequte antibiotic therapy is necessary. This is followed by excision of the sac.

Note: Any of the above can be asked as short notes.



15

Fractures in Children

- *Introduction*
- *Types of Injuries*
- *Physeal Injuries*
- *Structure of a Physis*
- *Diagnosis and Management*
- *Other Injuries*
- *Common Fractures in the Lower Limb: Diagnosis and Management*

Introduction

Pediatric skeleton is a growing skeleton. It is different from an adult mature skeleton in many ways. Hence the response to injury is different from an adult bone with respect to nature of deformation and healing. Healing occurs faster which emphasizes the need for an early management of these injuries. Delay in management, increases the difficulty in reduction and promotes the possibility of development of deformities and abnormalities in growth. So,it is absolutely necessary to understand these facts before a pediatric fracture is managed.

Differences seen	Pediatric skeleton	Adult skeleton
Growth	Growing, with the presence of epiphysis, physis and metaphysis	Growth is complete. Mature and fully ossified.
Plasticity	Present	Absent
Periosteum	Thick and loosely attached	Thin and firmly attached
Healing	Rapid and fast	Steady and slow
Remodeling capacity and speed	High and fast	Low and slow. Capacity decreases with advancing age
Plastic deformation	Possible	Not possible

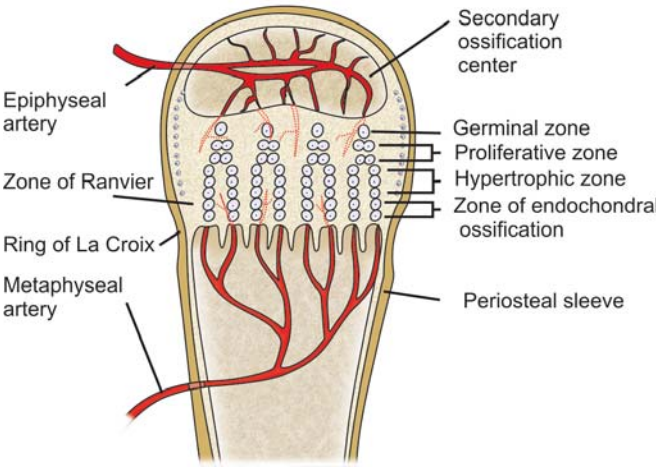


Figure 15.1
Diagrammatic representation of the structure of the physis

Types of Injuries

i. Physeal injuries.
ii. Plastic deformation.
iii. Buckle fractures.
iv. Green stick fractures.
v. Complete fractures.

Physeal Injuries

These injuries are also known as Growth plate injuries, Epiphyseal plate injuries and Epiphyseal cartilage injuries. It is incorrect to identify these injuries as Epiphyseal injuries because Physis is the growth plate and Epiphysis is the secondary center of ossification. The two are different.

Structure of a Physis (Fig. 15.1)

The physis consists of chondrocytes placed longitudinally and extracellular matrix. Four zones are identified. From the epiphysis to the metaphysis they are arranged as follows:

- Germinal zone.
- Proliferative zone.
- Hypertrophic zone.
- Zone of endochondral ossification.

There is abundant extracellular matrix in the germinal zone and the proliferative zone. Thus they are inherently strong to resist shear. The hypertrophic zone has mature uncalcified hypertophic cells with scanty matrix. Hence the inherent strength is poor against the deforming force especially shearing. The zone of endochondral ossification has calicified

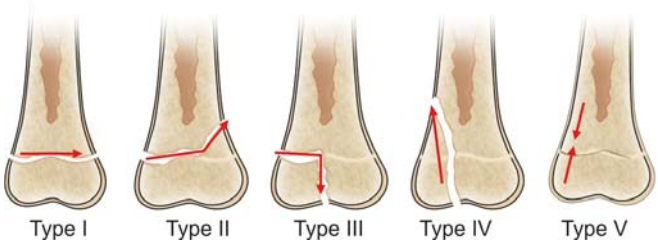


Figure 15.2
Pictorial representation of the physeal injuries based on Salter and Harris classification.

cells and it blends smoothly with the metaphysis. This increases the inherent stability.

Thus, the weakest area of the physis is the zone of hypertrophic cells through which most of the injuries occur.

The inner core of physis is surrounded by an outer perichondrial fibro-chondro-osseous ring which is sometimes referred to as 'Periphysis'. This ring is responsible for the stability of the physis. The portion of the peiphysis in the region of or adjacent to the physis is known as the 'Zone or Groove of Ranvier'. That portion which is adjacent to the metaphysis is known as the 'Ring of La Croix'.

The Zone of Ranvier is a wedge shaped structure consisting of cells contiguous with the epiphysis and is responsible for the latitudinal (widening)growth of the physis.

Epiphyseal arteries enter the physis and the terminal branches end at the proliferative zone.

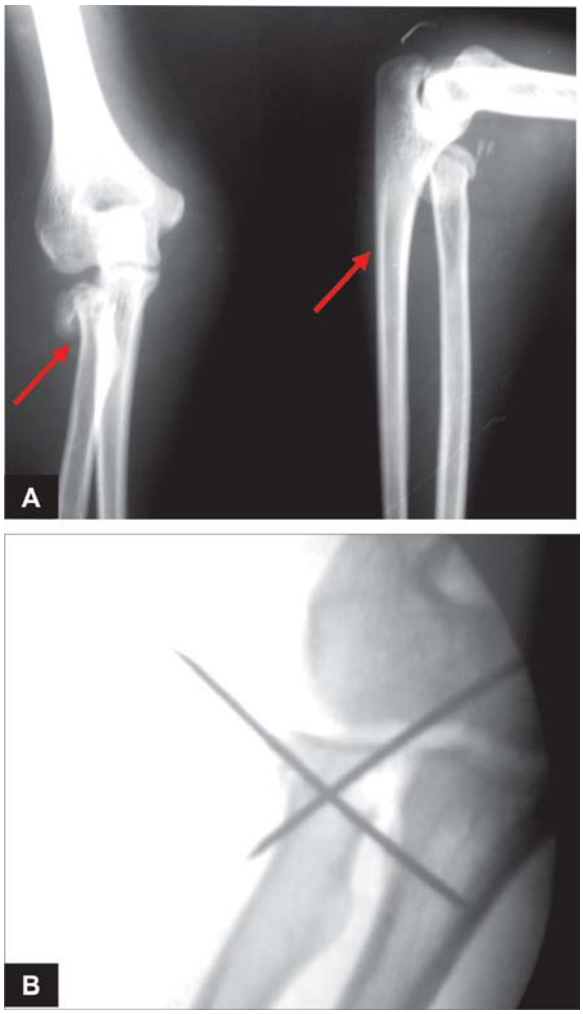
The diaphyseal nutrient artery with its loop like capillary network supplies the metaphyseal end of the physis (growth plate).

Classification of Physeal Injuries based on Salter and Harris (Fig. 15.2)

Type I: Shearing injury extending through the entire physis (Figs 15.3A and B).
Type II: Shearing injury extending through the portion of the physis and exiting through the metaphysis.
Type III: Shearing injury extending through the portion of the physis and exiting through the epiphysis.
Type IV: Injuries extending from the epiphysis through the physis into the metaphysis.
Type V: Crushing injury to the physis.

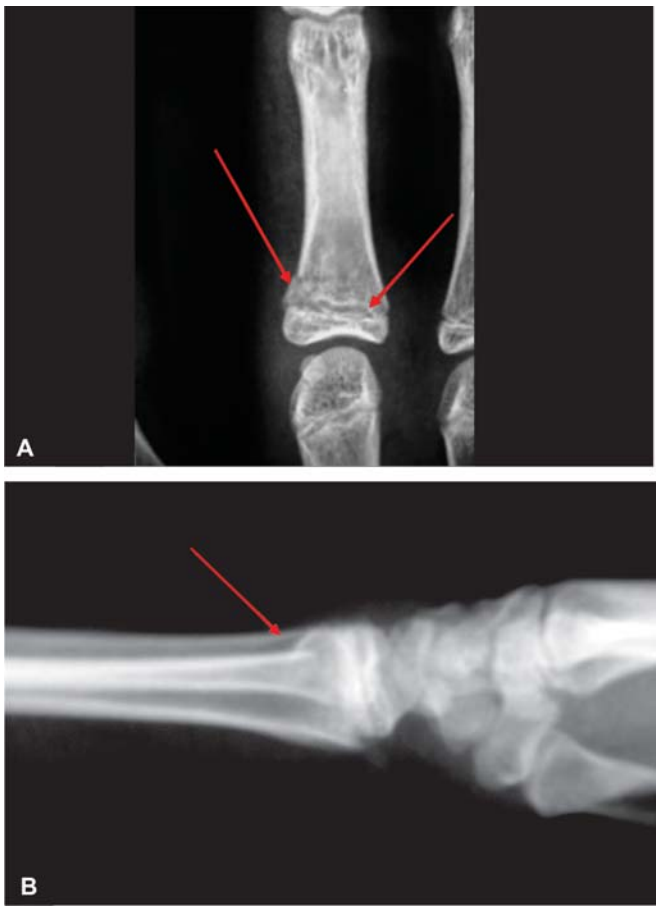
Thurston Holland Sign (Shiny Corner Sign)

This is a sign seen in type II physeal injuries. The triangular metaphyseal fragment is the only indicator of this injury (Figs



Figures 15.3A and B

Radiographs showing Type I Salter and Harris injury involving radial epiphysis. (A) Pre- and (B) Intraoperative.



Figures 15.4A and B

Radiograph showing the Salter and Harris type II physeal injury involving the base of the proximal phalanx of the index finger. Note the triangular metaphyseal fragment the 'Thurston Holland sign', which is the only indicator of type II physeal injury. When missed may lead to malunion.

15.4A and B). The force would have passed through the physis and caused shearing of the rest of the physis. Hence, these injuries need attention like any other displaced typeII physeal injuries.

Diagnosis of Physeal Injuries

X-ray A good radiograph gives the diagnosis in majority of the cases when ossific nucleus of the epiphysis has appeared. If not when the injury is suspected one has to resort to MRI.

Management

Accurate management of these injuries is absolutely essential to avoid deformities and growth abnormalities.

Aims of management

- To anatomically reduce the physis.
- To stabilize the reduction obtained either externally by cast or internally by closed pinning/open reduction and internal fixation.
- To protect and avoid damage to the germinal layer at any cost during the procedures.

In those cases which present late always consider secondary corrective procedures.

When the reduction is nonanatomic, following factors are helpful in assessing the outcome and making decisions.

Age of the patient: Younger the age greater is the remodeling capacity.

Type of the injury: Types I and II have better chance of remodeling than III and IV.

Location of the injury: An injury in the vicinity of a weight bearing joint is subjected to greater stress than an injury near a nonweight bearing joint when a nonanatomic reduction is accepted.

Duration of injury: More the lapse of time poorer is the outcome of primary treatment procedures.

Complications

Inherent complications

- Malunion
- Osteonecrosis
- Growth arrest.

Associated complications

- Infections
- Neurovascular injury.

Management of inherent complications Complications of malunion and osteonecrosis are managed by taking into consideration the resultant deformity and disability. Carefully planned corrective osteotomies considerably improve the function.

Growth disturbance Disturbance develops because of the formation of a bony bridge or a bar across the physis or retardation of growth because of injury. After careful assessment of growth retardation and adequate planning, procedures like resection of the bony bar, epiphyseodesis, etc. will help in the correction of the discrepancy.

Management of associated complications These are managed accordingly like any other cases of infection and neurovascular injury.

Harris growth arrest lines:

These when present/ absent help in determining nature of the arrest.

***Absence** indicates no growth has occurred after total physeal injury.*

***Transverse and parallel** lines indicate normal growth pattern.*

***Asymmetric** growth line indicates partial injury to the physis.*

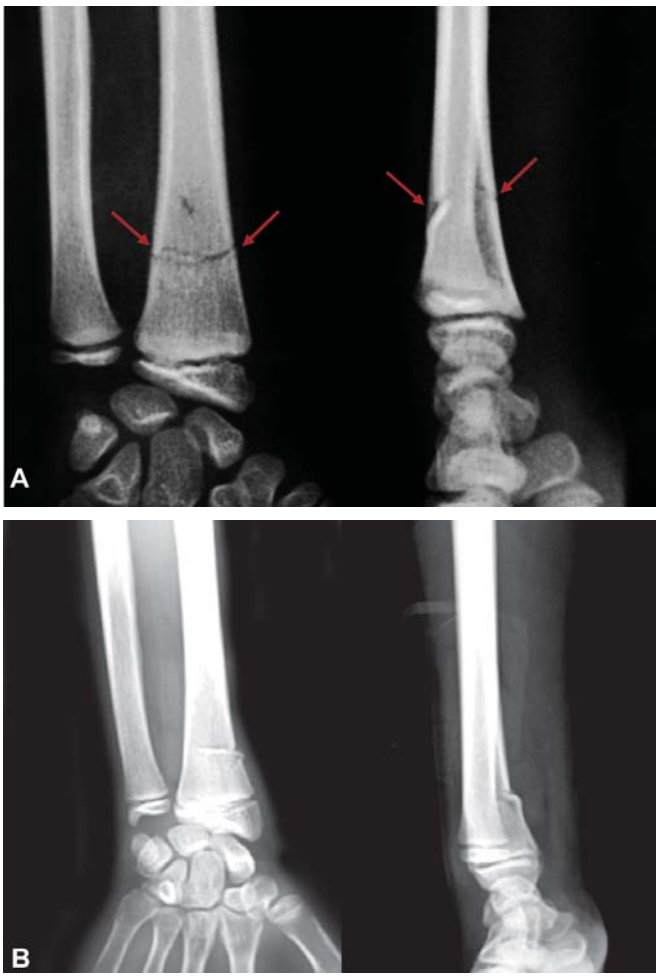
Plastic Deformation

Borden first described the plastic deformation of bones in children. Immature bone is weaker in bending strength. Hence, absorbs more energy and bends before the fracture occurs. This results in plastic deformation. Forearm is the common site for plastic deformation and ulna is the common bone. Some people have found the plastic deformation occurring in the femur too.

Lesser degrees of deformation get remodeled with growth. But deformities in the forearm more than 20° with limitation of pronation and supination in a child older than 4 yrs needs correction. Immobilization for a full period of 4-6 weeks is essential for a good healing (Refer Fig. 1.9).

Buckle Fractures (Figs 15.5A and B)

Also known as torus fractures because radiologically a ridge of bone (torus) is observed. These injuries are common at the



Figures 15.5A and B

Showing the radiographs of (A) a complete fracture with a buckle (B) incomplete fracture with a buckle. The presence of thick periosteum prevents displacements

transitional area of woven bone of the metaphysis and the lamellar bone of the diaphysis. The fracture may present as:

a. A simple cortical break with a ridge (Fig. 15.5B).
b. A ridge with plastic deformation of the rest of the bone.
c. A ridge with complete break of the rest of the bone (Fig. 15.5A).

A simple break with formation of a ridge can be managed with a well padded splint and it heals uneventfully. More severe forms of break get displaced over a period of time and result in deformities. To prevent this they need correction and immobilization in a well moulded plaster cast.

Greenstick Fractures

This is the most common fracture seen in children. In these the fracture is not seen as a complete break though the entire bone is involved. A part of the bone breaks and the rest of the bone bends like a Greenstick. Hence the name. The bone on the tension side breaks completely. The bone on the compression side bends and undergoes plastic deformation because of thick periosteum. It is necessary to break the intact cortex on the compression side to achieve good reduction. Reduction should be maintained for a full period of approximately 6 weeks to avoid the risk of refracture due to premature removal.

Common Fractures in the Lower Limb: Diagnosis and Management

Fracture Shaft of Femur

Mechanism of Injury

The trauma should be of a severe nature for this fracture to occur. Fall from a height, motor vehicular accidents are some of the examples of severe trauma.

In the younger age group child abuse must be suspected especially when there is delay in seeking the medical treatment and there are other injuries.

Diagnosis

It is a straight forward diagnosis. X-rays help in assessing the nature of the fracture and planning the treatment.

Treatment

Following factors serve as a guide to decide the type of treatment to be instituted.

i. Age of the child.

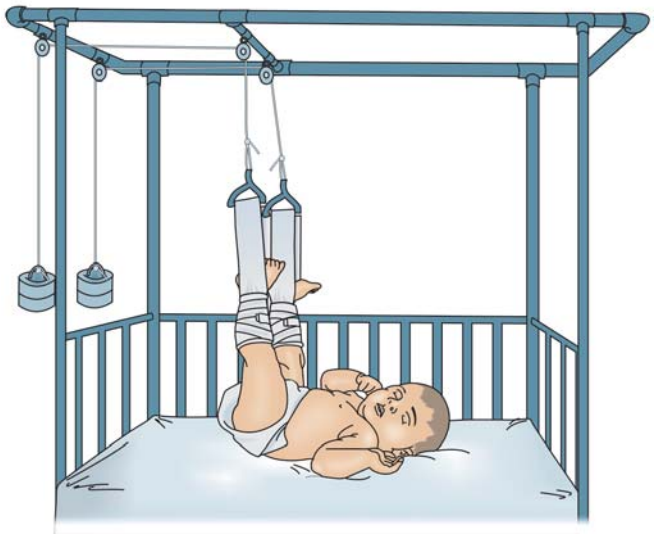


Figure 15.6

Showing the Bryant's otherwise known as Gallows traction. Note how the buttocks are lifted off the bed. This is essential for effective traction.

ii. The degree of comminution.
iii. The degree of displacement, amount of over-riding and shortening.
iv. Open wounds with disturbed soft tissue envelop.
v. Presence of vascular injury.
vi. Presence of other associated injuries.

0-6 months of age Excellent results are obtained with non-operative treatment. Strapping the thigh to the abdomen in a newborn, using the Pavlik harness in an infant are some of the methods employed. Compression of the femoral nerve during the treatment is a possibility because of flexed position of the limb and should always be kept in mind.

6 months-2 years

- Bryant's traction.
- Closed reduction and spica cast application.

2-5 years Skin or skeletal traction (Fig. 15.6) on a splint followed by protection in a spica cast or a brace. Spica cast gives better protection in children for the simple reason that a child does not understand the importance of non-weight bearing and tries to walk when the brace is given. Also, children tolerate this extensive plaster very well.

Closed reduction and flexible nailing, open reduction and flexible nailing may be necessary in selected cases, e.g. when shortening is more than 3 cm and not amenable for manipulative correction.

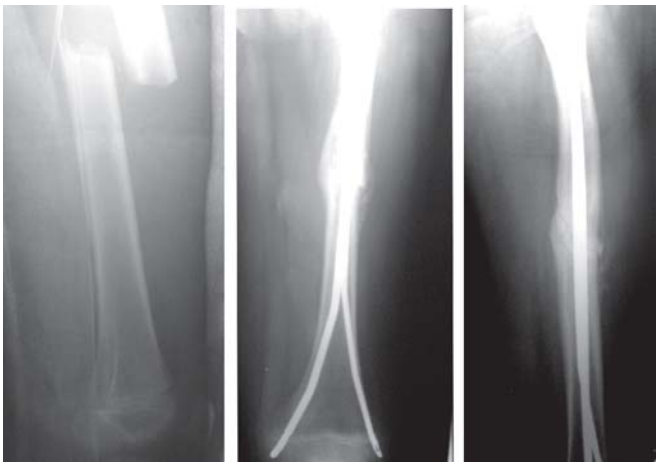


Figure 15.7
Radiographs showing closed reduction and fixation of fracture shaft femur using elastic nails.

5-12 years Closed/ open reduction and flexible nailing is the treatment of choice (Fig. 15.7) except in those minimally displaced and long oblique fractures which are amenable for closed treatment with traction.

Minimally invasive plate osteosynthesis should be reserved for those cases with segmental comminution. These fractures are best stabilised by bridge plate technique.

Open fractures External fixators are used in the treatment of open fractures.

Vascular injuries need immediate repair and other associated injuries are dealt with accordingly, e.g. visceral injury, chest injury, head injury, etc.

Fracture Neck of Femur in Children

Mechanism of Injury

Always as a result of high energy trauma or fall from a height. Associated injuries are common.

Classification

Based on Delbet's classification 4 types are identified (Fig. 15.8).

- Type I: Transepiphyseal fractures
- Type II: Transcervical fractures
- Type III: Cervicotrochanteric fractures
- Type IV: Intertrochanteric fractures.

Diagnosis

X-rays give adequate information of the injury. Diagnosis is straight forward.

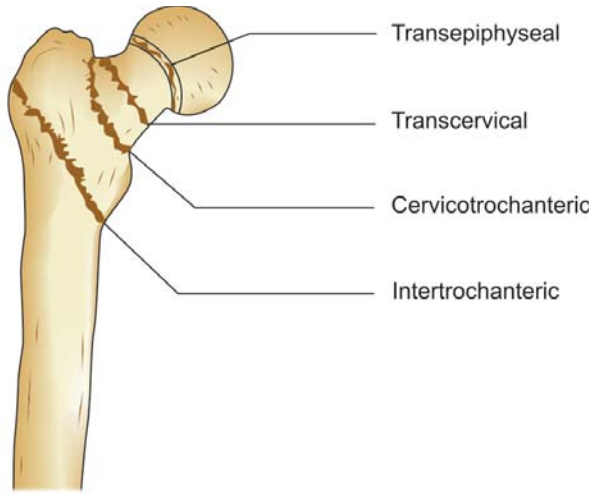


Figure 15.8
Diagram showing different types of fractures of the neck of the femur in children according to Delbet's classification.

MRI may be necessary to evaluate the vascularity of the femoral epiphysis at a later period after treatment. Use of titanium implants allows MRI to be done with implants in situ.

Treatment

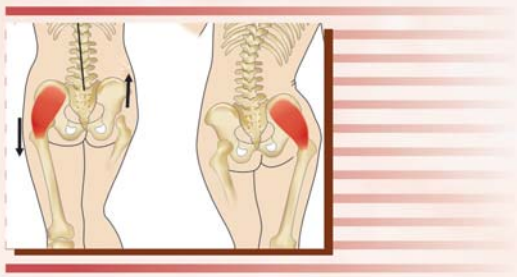
Reduction closed/open and internal fixation is the treatment of choice

Bigelow's Dictate In 1864, Bigelow remarked that *While the impacted fracture of the base of the femoral neck unites by bone, if at all, there seems to be a decreasing tendency to osseous union as we approach the smaller portion of the neck near its head.*

The above statement is found to be true even in pediatrics femoral neck fractures. Worst prognosis is seen in transepiphyseal fractures and best prognosis is seen in intertrochanteric fractures and the prognosis is found to become better from type II to type III fractures.

Further Reading

1. Alan E Oestreich, Borhaan S Ahmad. The periphysis and its effects on the metaphysis: I Definition and normal radiographic pattern. Skeletal Radiol 1992;21:283-6.
2. John A Herring (Ed). Tachdjian's Pediatric Orthopedics. 4th edition, Saunders: Elsevier, Philadelphia 2008;3.
3. Raymond T Morrissy, Stuart L (Eds). Weinstein Lovell and Winter's Paediatric Orthopaedics. 6th edition, Lippincott, Williams and Wilkins 2005.



16 Gait

- *Definition*
- *Phases*
- *Gait Cycle*
- *Abnormal Gaits*
- *Trendelenburg Gait*

Definition

A normal gait is defined as a forward propulsion of the human body occurring as a result of rhythmic, sequential movements taking place in the lower limbs.

Phases (Fig. 16.1)

A. Stance phase
   Which is further divided into:
   - Heel strike
   - Foot flat
   - Mid stance
   - Heel off
   - Toes off (push off)

B. Swing phase
   - Acceleration
   - Mid swing
   - Deceleration

It is the alternating pattern of movements taking place between the stance phase of one limb and the swing phase of the other that makes propulsion (walking) possible.

Gait Cycle

Gait cycle is defined as the cyclical movement taking place from heel strike of one limb to its next heel strike.

Thus to conclude for a normal gait a healthy support, i.e. the bone; a healthy power, i.e. the muscle and a healthy stimulus to initiate the action, i.e. the intact nervous system is necessary. If any of these are affected it leads to abnormal gait.

Abnormal Gaits

Due to Muscle Weakness, e.g. Poliomyelitis

Gluteus Maximus Lurch

Gluteus maximus muscle extends and laterally rotates the hip. To maintain the center of gravity the pelvis remains backwards during the mid stance phase because of this action.

If the gluteus maximus is paralysed this maintenance of hip in extension is not possible.

Instead the body swings backwards everytime when the weight is borne. Thus it results in a gluteus maximus lurch.

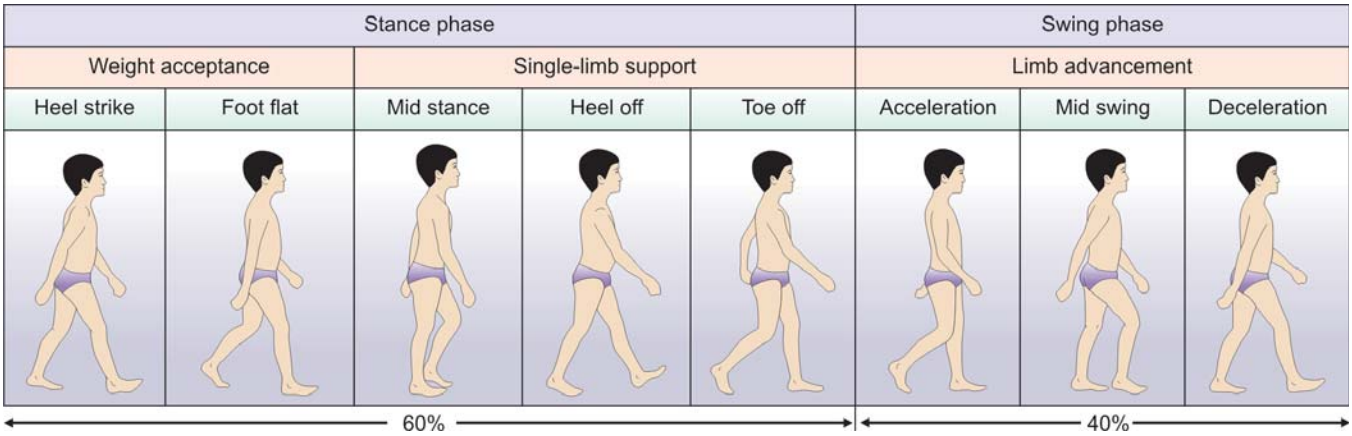


Figure 16.1

Pictures showing various phases of gait and weight distribution during Stance and Swing phase.

Quadriceps Lurch

In quadriceps paralysis, there is loss of active extension of the knee. During the stance phase which begins with Heel strike and ends with Heel off and Toes off, the knee has to remain in extension. This is not possible because of paralysis of Quadriceps. So, a patient locks the knee in extension and bends forwards to support the front of the thigh with the hand. This results in Hand knee gait or Quadriceps lurch.

High Stepping Gait of Foot Drop

A normal gait begins with Heel strike of Stance phase. In foot drop because of the paralysis of the dorsiflexors, the Heel strike is not possible and in order to clear the ground the patient lifts the leg high and when the foot is brought down, the toes touch the ground first before the heel. This results in a toe to heel gait, which is exactly the opposite of the normal heel to toe gait. This is known as High stepping gait.

Calcaneus Gait

This occurs due to paralysis of the gastrosoleus. All the steps of Stance phase except Heel strike are affected. During the later part of stance the weight is borne by the calcaneum and the foot rotates outwards in order to complete the Stance phase. This results in a Calcaneus gait.

Due to Changes in the Bones and Joints

Stiff Hip Gait

This type of gait is seen in ankylosis of the hip. The patient is not able to flex the hip during the Swing phase. To complete the Swing phase of gait cycle he lifts the pelvis on that side and circumducts the involved leg. This results in a circumductory Stiff hip gait.

Stiff Knee Gait

It is difficult to appreciate Stiff knee gait because the changes that take place are subtle and needs careful observation. The toe off phase is affected. To clear the ground for initiation of swing and for heel strike after completion a lesser degree of circumductory movement is required. This can be made out only by careful observation.

Short Leg Gait

When shortening of the limb is more than an inch, an appreciable limp develops. The shoulder on the opposite side dips down and lifts up because of upward and downward movement of the pelvis during weight bearing.

Due to Abnormalities in the Nervous System

Scissors Gait

Due to the spasm of the adductors the legs get crossed in front of each other during walking resulting in classical Scissoring. This is known as Scissors gait and is seen in spastic paralysis of cerebral palsy.

Festinant Gait

Seen in Parkinsonism and is characterized by short strides. The feet barely clear the ground.

Stamping Gait

Seen in conditions which result in sensory loss, e.g. Peripheral neuritis, Tabes dorsalis, etc.

In this type of gait because of sensory ataxia patient stamps the feet to the ground.

Drunkard's Gait

Lesions of the cerebellum result in motor ataxia. Patient is not able to maintain motor balance and walks in an irregular path. This results in a Drunkard's gait.

Hemiplegic Gait

This is a classical gait seen in hemiplegia. The shoulder is adducted, the elbow and the wrist are flexed and the involved lower limb is circmducted during the swing phase.

Antalgic Gait

Any painful condition in the limb which causes increase in pain during weight bearing results in this type of gait. The condition may vary from a small thorn prick in the foot to tuberculosis of the hip. The stance phase is affected and on weight bearing the patient immediately shifts the weight to the opposite normal limb because of pain. This results in a short and incomplete stance and an antalgic gait.

Trendelenburg Gait (Fig. 16.2)

This is a type of gait that occurs when the Abductor Mechanism which is responsible for abduction at the hip is affected. The mechanism has three components:

a. The *fulcrum* which is the Head of the femur
b. The *lever* which is the Neck of the femur
c. The *power* which is the Gluteus medius.

Any abnormality of these, occurring alone or in combination disturbs the function of Abductor Mechanism and results in this type of gait. The fulcrum is disturbed in dislocation of the hip. The lever is disturbed in Fracture neck of the femur. The power is disturbed in paralysis of Gluteus medius.

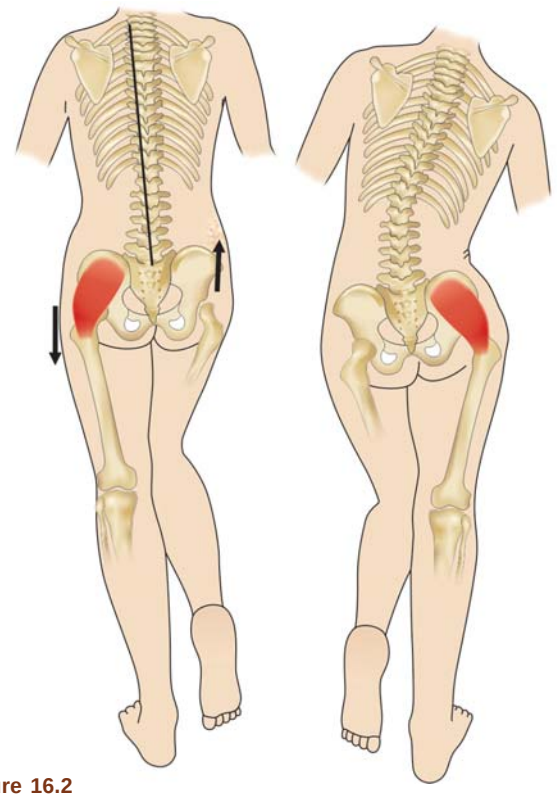


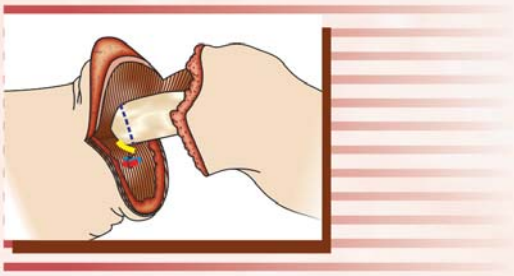
Figure 16.2
Showing the changes occurring in the hip in Trendelenburg gait. In this illustration the Right hip is the involved hip.

Pathomechanics of Trendelenburg Gait

The abductor mechanism has two-fold function. Acting from above it abducts the limb. On weight bearing, the limb is fixed and it acts from below and this causes lifting of the pelvis. So when the pelvis is lifted up the anterior superior iliac spine on the opposite side moves up. When the Abductor Mechanism is affected this lifting of pelvis does not take place. Instead the pelvis sags on the opposite side when weight is borne on the affected limb.

This causes the anterior superior iliac spine on the opposite side and the shoulder on the same side to dip down for maintaining the balance. A characteristic lurch develops on the affected side resulting in Tredelenburg gait.

When there is bilateral affection of the Abductor Mechanism a bilateral Trendelenburg gait or a Waddling gait results.



17 Amputations

- *Definition*
- *Indications*
- *Types of Amputations*
- *An Ideal Stump of Amputation*

Definition

Amputation is one of the meanest yet one of the greatest operations in surgery. Meanest when resorted to while better things could have been done .Greatest when it is done to save the life of an individual.

Indications

i. A **d**ead limb, e.g. vascular gangrene.
ii. A **d**ying limb, e.g. TAO, frost bite, etc.
iii. A **d**estroyed limb, e.g. crush injury.
iv. A **d**enervated limb,e.g. hereditary sensory neuropathy, whole arm type of brachial plexus injury.
v. A **d**angerous limb, e.g. malignant bone tumor, gas gangrene.
vi. A **d**eformed limb.

In all the above situations either the function of the limb is lost or the limb poses a threat to the life of an individual. Hence there is a need for amputation.

Types of Amputations (Fig. 17.1)

General Amputations

- A ray amputation.
- Below knee amputation
- Above knee amputation
- Below elbow amputation
- Above elbow amputation.
- Disarticulation of hip, knee, etc.
- Hind quarter and Fore-quarter amputation

Specific Amputation

- Gille's amputation
- Lisfrank's amputation
- Chopart's amputation
- Syme's amputation
- Pirogoff's amputation
- Kruckenberg's amputation.

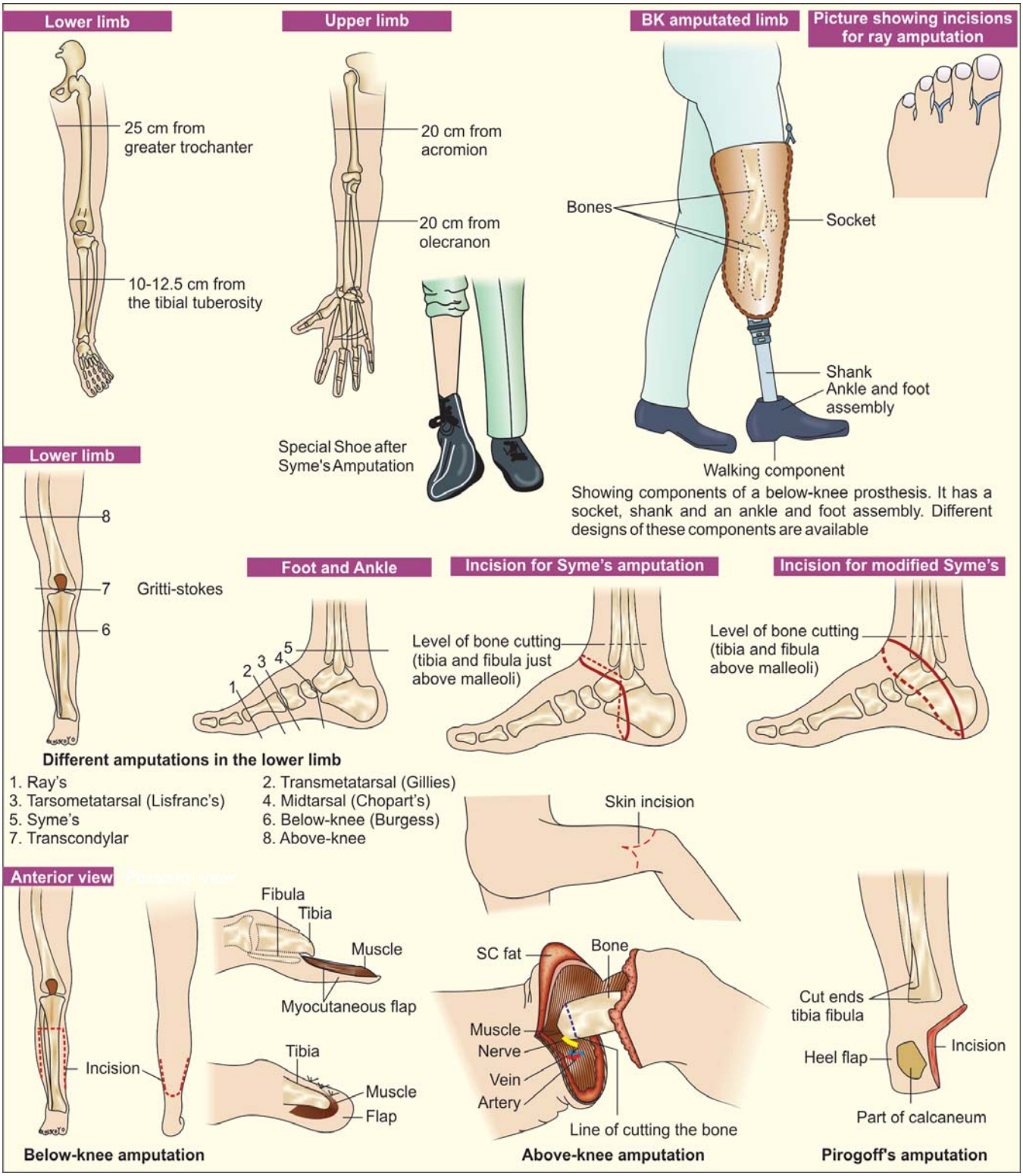


Figure 17.1

Different types of amputations, their levels, the ideal length of the stump and some of the prosthesis

Gille's Amputation

It is a transmetatarsal amputation done in the foot. Preserves the function of weight bearing. Toes off phase of stance is lost. Needs special shoes with toe inset filled for a good cosmesis and to overcome the deficit of push off.

Lisfrank's and Chopart's amputation
(Ref: Foot injuries in the text)

Deficit develops from the foot flat stage of stance phase of gait. ***In fact Chopart's amputation can result in foot drop because of lack of muscle and tendon attachments.*** Weight bearing function is preserved. Special shoes with anterior fill is used for cosmesis. Gait is affected.

Syme's Amputation

Named after James Syme , Professor of surgery in Edinburg, who described this amputation in the year 1843 in London and Edinburgh Monthly Journal of Medical Science. It is an excellent amputation through the ankle which retains the function of weight bearing because of intact heel pad. It results in a bulbous stump. To reduce the mediolateral diameter and the bulbous nature of the stump trimming of the malleoli (Mazet 1968; Sarmiento 1972) and supramalleoar resection (Elmslie 1924) has been reported.

A classical Syme's amputation is performed by:

a. Subperiosteal dissection and excision of the calcaneus.
b. Ligating the posterior tibial artery distally then by preserving the blood supply to the heel pad.
c. Resection of the proximal tibia at the level of the dome.

(Some do not recommend this step of classical Syme's)

A special prosthetic shoe is necessary after Syme's amputation. Weight bearing function is preserved. But all the stages of stance phase of gait are affected.

Pirogoff's Amputation

In this modification the calcaneum is resected partly and turned 90° upwards towards the tibia. This increases the length of the stump.

Kruckenberg's Amputation

This amputation was described by Kruckenberg and Putti .The amputation involves forcepisation of the amputated stump of the forearm into radial and ulnar pincers (Bifid forceps) resulting in two antebrachial fingers. These two pincers open and close in pronation and supination movements of the forearm. The length of the pincers can be varied from 7-12 cm. Longer the pincer length greater is the interdigital defect and may necessitate a skin coverage procedure. Also the strength of the grip decreases. The amputation is unsightly but highly efficacious functionally with retained sensation. Can be fitted with a cosmetic hand prosthesis.

An Ideal Stump of Amputation

It should fulfill the following criteria

- Long enough to fit a prosthesis
- Good power
- Good sensation
- Good blood supply
- Good soft tissue cover
- Conical shape
- Proximal joint should be normal
- No neuroma
- No bad scarring
- No infection.

Recommended Ideal Length of the Stump

- In below-knee amputations 10.0-12.5 cm from the Tibial tuberosity
- In above-knee amputations 22.5-25.0 cm from the greater trochanter.
- In above and below elbow amputations 20.0 cm from the Acromion process and the Olecrenon process respectively.

These stump lengths recommended are not constant. The length varies depending on the length of the limb. Basically it gives a rough idea as to how much length of the stump is desirable for fitting a prosthesis.

Procedure of amputation and general principles to be observed

i. Always use a tourniquet **except in a case of vascular disease.**
ii. **Mark the flaps properly** and then make the skin incision**.**
iii. **Equal antero-posterior flaps** are desirable.
iv. **Aim at an ideal stump** towards the end of the procedure
v. Skin division is the farthest, followed by fascia, muscle and the bone. Each of them is divided, a little more proximal, to the preceding tissue in the above mentioned order.

vi. Proximal part of the flap should contain full thickness of the tissue, from skin to bone whereas the distal part of the flap should contain only skin and the fascia to allow good closure of the wound.
vii. **Do not strip the periosteum more than the required extent** as this will result in formation of sequestrum at the end of the stump (crown sequestrum).
viii. Take care of sharp **spikes of bone** and **make them rounded.**
ix. **The nerves to be pulled a little and then divided** to allow them to retract.
x. **The major vessels are to be perfectly ligated**.
xi. **Always release tourniquet before closure** and achieve hemostasis.
xii. **Close over a drain.**
xiii. **Good stump bandage** for support to be given immobilizing the joint above.

18

Implants and Instruments

- *Introduction*
- *Surface Implants*
- *Intramedullary Implants*
- *Biodegradable Implants*
- *General Instruments Used in Orthopedics*
- *William Arbuthnot Lane*
- *William Ferguson*
- *Designs of Limb Lengthening Apparatus*

Introduction

Instruments are the devices which are used to perform the surgical procedures. Implants are the devices which are placed (planted) into the human body. When an implant is used to replace a diseased/damaged part totally or partially the term Prosthesis is used.

Prosthesis is designed for permanent use. They are removed only when they fail.

Whereas implants other than prosthesis serve only as an internal splint for a specified period within which the healing occurs and have no role to play afterwards. They may have to be removed. The implants once removed should not be reused.

Materials Used for Orthopedic Implants

Alloys stainless steel 316 L and titanium and more recently biodegradable materials like polyglycolic acid, polylactic acid and polydioxanone.

Desirable Properties of an Implant

1. The modulus of elasticity of the implant should be close to that of the bone. Titanium alloy is the closest among the alloys used. Biodegradable materials are still closer (Fig. 18.1).
2. Should be biocompatible.
3. Should be chemically stable.
4. Should have good ductility.

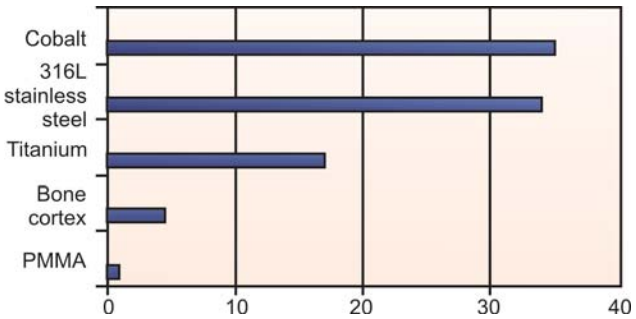


Figure 18.1

Showing the modulus of elasticity of different materials. Titanium is the closest when compared to other materials.

5. High fatigue resistance.
6. High mechanical strength (Lacking in biodegradable materials).
7. Nontoxic.
8. Noncarcinogenic.

Types of Implants

Basically there are two types:

a. Surface implants, e.g. plates and screws.
b. Intramedullary implants, e.g. nails.

Types of Prosthesis

Two Types

a. Those which replace components of the bone, e.g. prosthesis used for joint replacement.
b. Those which are used to replace the whole structure, e.g. intervertebral disc, scaphoid, lunate, etc.

Points to be observed while selecting the implant:

It should always be remembered that the implants which are used to stabilize the procedure, have a great role to maintain the correction achieved by surgery. They have no role to play once the healing occurs.

Healing is a natural process and is dependant on several factors. Immobility at the site is one of the factors essential for healing and implants take care of this factor by providing immobility at the site. None of the implants allow full weight bearing or full activity immediately following surgery.

There is a postoperative protocol to follow. Failure to observe this results in implant failure.

Following factors are to be taken into consideration while selecting an implant.

a. Age of the patient.
b. Weight of the patient.
c. Mental status and intelligence.
d. Demand of the situation.
e. Quality of the bone.
f. Associated comorbid factors.
g. Foreign body sensitivity.

In pediatric age group unlike adults implants are used only with definite indications. Less traumatic and easily removable implants are preferred, e.g. 'K'-wires, titanium elastic nails, etc.

More stronger implants are preferred in heavily built individuals. Senility, alcoholism, mental illness may lead to implant failure because of failure to adhere to postoperative care. Associated comorbid factors may contribute to implant failure.

Demand of the situation dictates the type of the implant. A transverse fracture is stable to axial compression but unstable to torsion stress. An oblique fracture is stable to torsion stress but unstable to axial compression and bending. A spiral fracture is unstable to both axial compression and torsion. A comminuted fracture entirely depends on the strength of the implant to maintain stability. An open fracture in addition to the nature of bony injury poses a challenge because of associated injury to soft tissue envelope.

Further presence of other fractures and injury to the vital organs also have a bearing on selection of the implant. All these factors are to be taken into consideration when an implant is chosen.

When the individual is sensitive to stainless steel, titanium implants are preferred. Also when MRI is indicated during the postoperative and follow-up period, e.g. in spinal stabilization, titanium implants are preferred.

Correct Handling of the Implant

Insertion of an implant is an art. Correct handling of the implant is an absolute must while inserting. This skill has to be acquired from the very beginning itself. Meticulous attention is to be given to the use of proper instruments and the techniques of insertion. Techniques vary depending on the design of the implant and hence specialized instruments are provided. Improper handling increases the failure rate and complications.

Asepsis in the Operating Room

This is of utmost importance. There goes a popular saying which says that ***"You can spit into the abdomen but you cannot breathe over the bone"***. Meticulous measures for asepsis is to be observed in the orthopedic operation theater.

Infection following an insertion of an implant is a disaster. The implant gets rejected and the whole procedure is a failure. A center where the infection rate is less than 1% is an ideal set up for orthopedic surgery. 1-3% is acceptable. But above 3% is to be considered as not suitable for orthopedic surgery.

A separate dedicated theater for orthopedic surgery is desirable.

Surface Implants

These are placed on the surface and hence share the load of axial stress.

Screw (Used to Fix the Surface Implant to the Bone)

A screw is an implement which converts a rotational force into linear motion (Fig. 18.2). The design of the screw enables it to bring about this.

The Screw Design (Figs 18.3 to 18.7)

i. *Head of the screw:* It gives attachment to the device that applies a rotational force (driver) and also it gives resistance when the insertion is complete and prevents further motion.

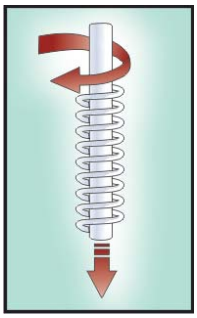
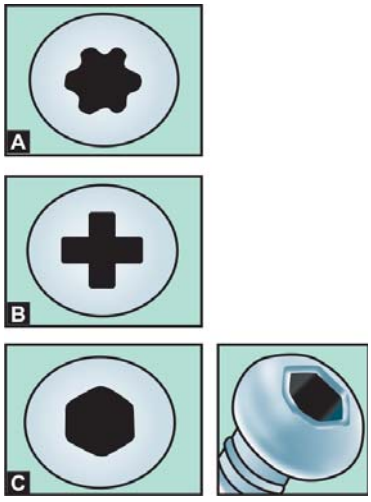


Figure 18.2

Showing the rotational force being converted into a linear movement



Figures 18.3A to C

Showing different types of screw heads: (A) Torx head, (B) Philips head, and (C) Hexagonal head.

ii. *Shank with threads:* This helps to gain purchase in the object during gliding and helps in the firm fixation of the screw after insertion.

iii. The tip directs the screw.

The distance between the threads is called the pitch of the screw and the distance which the screw moves during one complete turn is the lead of the screw. The pitch and the lead are equal, i.e. if the pitch is 1 mm the lead also is 1 mm.

The variation in the pitch of a cortical and cancellous screw is for the purpose of gaining good purchase in the cortical and cancellous bone respectively. The cortical bone is a compact bone and the cancellous bone is a spongy bone. Hence, the difference in the pitch design (Figs 18.5 and 18.6).

The screws are available in different sizes and different diameters.

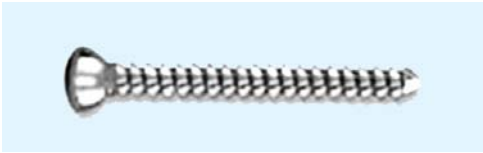


Figure 18.4

Showing picture of a cortical screw which has threads along the length of its shank and has a smaller pitch.

Figure 18.5

Showing pictures of cancellous screws. The bigger one is a 6.5 mm and a smaller one is 4.0 mm. They have threads only at the tip and a larger pitch. They are available in 16 mm and 32 mm thread length as well as in a fully threaded design.

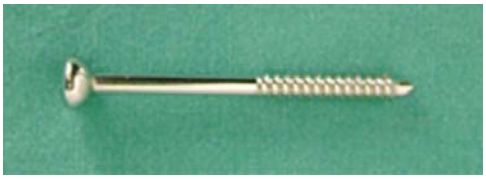


Figure 18.6

Showing the picture of a malleolar screw. This screw is used for fixation of the malleolus and has a conical sharp cutting tip with threads of a different pitch as well as width from that of a cortical or a cancellous screw. The extent of thread on the shank is more than that of cancellous screw, extending almost to 1/2 the length of the shank.

Figure 18.7

Picture of Herbert screw used in the fixation of the fracture scaphoid. These are headless screws having threads of different pitch at the proximal and the distal ends.

Plates (Figs 18.8 to 18.11)

Plate is a surface implant with holes. They are placed on the surface of a bone and are fixed to the bone by means of screws (Figs 18.14 and 18.19). The outer surface is convex and the inner surface is concave to accommodate for the cylindrical structure of the bone. Different designs of plates are available.

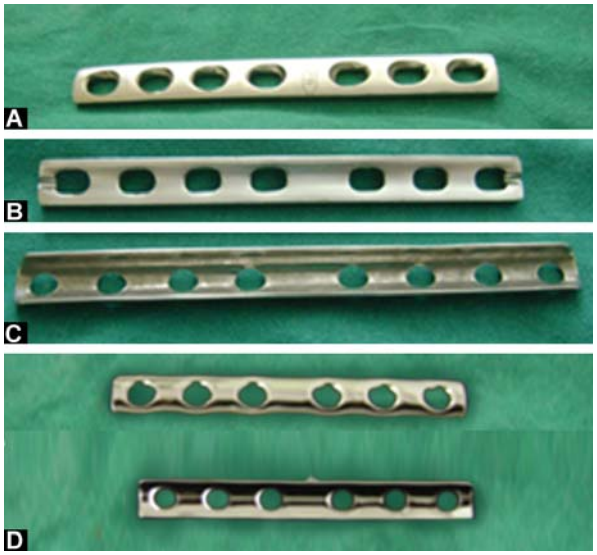
Dynamic Compression Plate (DCP)

The curvature of this plate is equal to 1/8 of a circle. The holes are distributed equidistant from a solid non-fenestrated center portion of the plate. The holes are oval in shape and the distal edges of the oval hole have a slope towards the center to bring about compression effect when the screws are tightened.

Very popular design of 1980's. ***Now not used***. The fractures were thought to unite early because of compression force generated by the inherent design of the plate as well as the method of insertion of the screws. Patients were mobilized early and fracture disease was prevented. Now it is not used because the principle of compression fixation of fractures using DCP is known to cause secondary changes which are detrimental to the bone. Also union without callus is far inferior when compared to union with callus and remodeling with respect to strength of the bone.

Some of the changes observed which proved to be detrimental are as follows:

i. Union without callus formation in the form of direct substitution by the creeping trabeculae caused refracture after removal of the plate.
ii. Stress fractures occurred at the ends of the plate.
iii. On removal of the plate the bone under the plate was found to be avascular.



Figures 18.8A to D

Showing the dynamic compression plate the semitubular plate and a 1/3 tubular plate. Note the placement and the sloping nature of the holes and the curvature which amounts to 1/8 of a circle in a DCP (A and B). The curvature of the semitubular plate amounts to 1/2 the circumference of a circle (C). The curvature of a 1/3 tubular plate amounts to 1/3 the circumference of a circle (D).

1/3 Tubular and Semitubular Plates

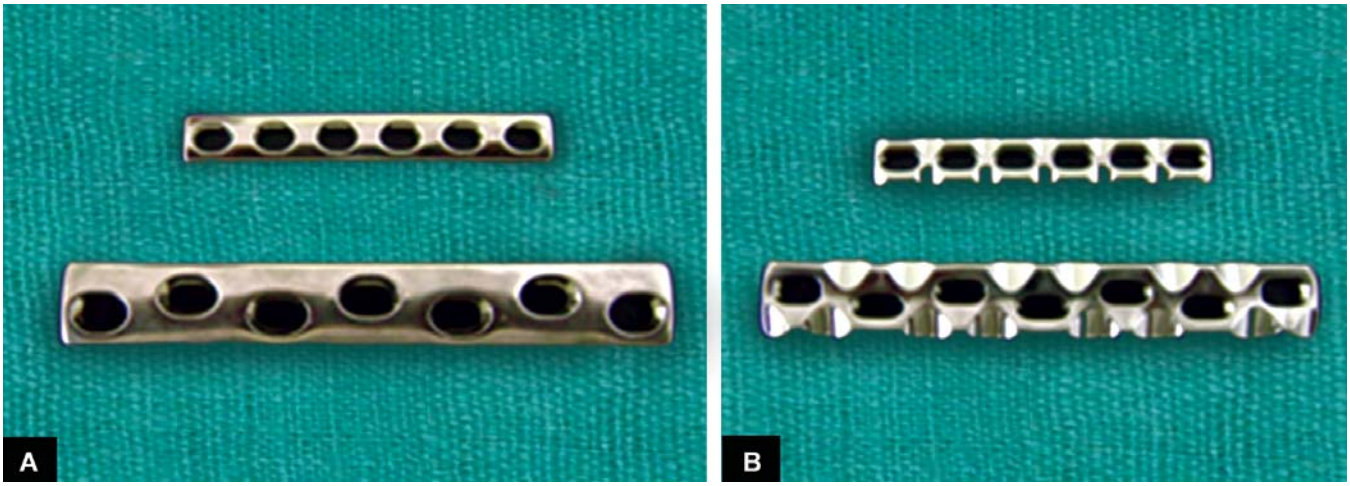
These are used mainly to fix the fractures in the supportive bones like ulna and fibula.

Limited Contact, Dynamic Compression Plate: LC, DCP (Figs 18.9A and B)

To overcome the disadvantages of the DCP, the design was changed. Grooves were designed on the concave under surface of the plate for the blood vessels to grow. The holes were distributed evenly all along the plate to prevent concentration of stress, without taking into consideration a central reference point. Currently these plates are being used in place of DCP.

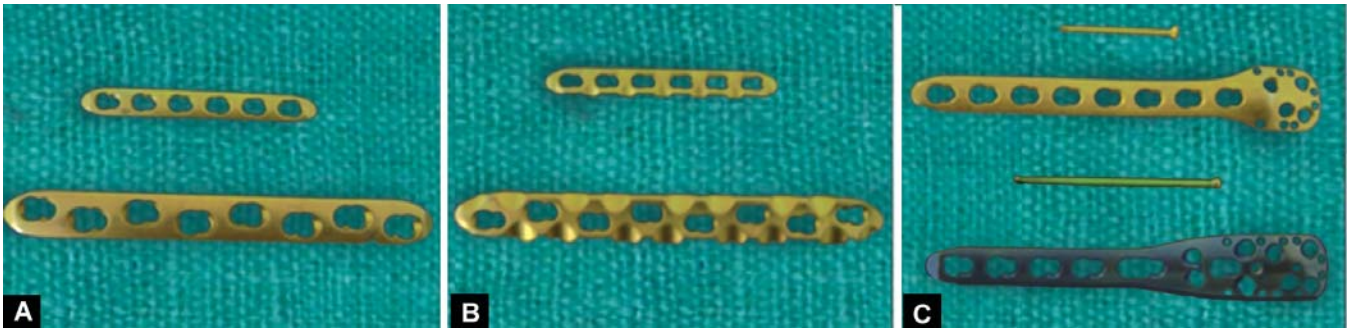
Locking Compression Plate (LCP) (Figs 18.10A to C)

These are specially designed to fix fractures in osteoporotic skeleton. The problem faced in an osteoporotic skeleton is one of having firm purchase in the bone. Without firm purchase the screws will become loose and failure of fixation occurs. These plates have holes designed in such a way that it gives freedom to exercise locking or nonlocking option. At the same time options are given for placing the screws at right angle to the surface of the bone or at an oblique angle. These options improves the firmness of fixation. In practice this plate serves as an internal fixator.



Figures 18.9A and B

Showing LC DCP. Note the changed design with grooves on the under surface of the plate and uniform distribution of holes for insertion of screws. Also note that the changed design of the under surface of the plate allows limited contact with the bone.



Figures 18.10A to C

Showing (A and B) the different sizes of LCP. Note the option for locking as well as nonlocking for the head of the screw. (C) Proximal humerus LCP with a variety of options for the insertion of screw with respect to size, type and direction of the screw. Note that the screw in green color has a head with threads for locking.

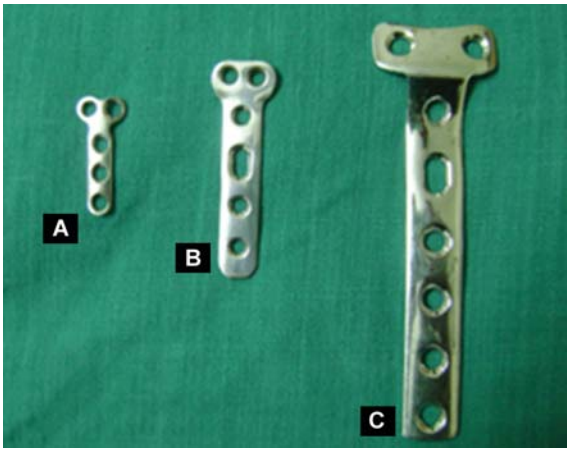


Figure 18.11

Showing 'T' type Buttress plates for different injuries. (A) Thumb metacarpal (Rolando's fracture), (B) Lower end of radius (Smith's fracture) (C) Tibial condyles (Fender fracture)

SS wires

These are stainless steel wires used in the fixation of comminuted fractures and in tension band wiring, circlage wiring, etc. (Fig. 18.17).

Intramedullary Implants

These implants are placed in the medullary canal of a bone and hence bear the stress of axial loading.

Basically classified as rigid nails, hollow nails and elastic nails (Figs 18.12 and 18.13).

Rigid Nails

These nails are solid rods and do not have a canal. They have a good tolerance for axial loading but poor bending property and easily break when subjected to bending stress, e.g. unreamed interlocking nails.

Indicated in open fractures and badly comminuted fractures.

Hollow Nails

These nails have a central canal. They are not as strong as rigid nails. But have a good tolerance for bending stress and do not break easily when subjected to such a stress.

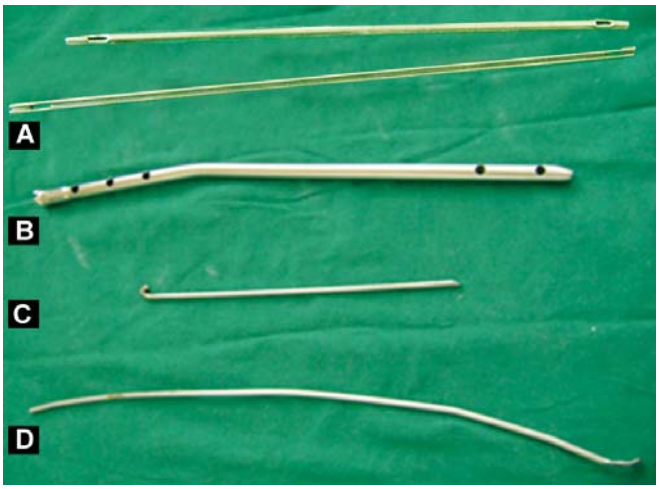
Indicated in all closed noncomminuted fractures.

Elastic Nails

These are thin solid rods and on their own do not have any strength to resist axial stress but show excellent adaptiveness for bending stress. Multiple nails have to be used to get sufficiently rigid fixation and resist the stress of axial loading, e.g. ender nails, titanium elastic nails, etc. Indicated in pediatric long bone fractures and in elderly osteoporotic long bone fractures.

Kuntscher Nail or K-nail

This is an intramedullary nail designed by Gerhardt Kuntscher a German surgeon during II world war. It is a hollow slotted nail which is clover leaf shaped in cross-section. It has extraction slots at either ends. It was used to fix the fracture shaft femur based on the principle of three point fixation (Fig. 18.13). The three points being the cancellous portion of the intertrochanteric bone, the isthmus and the cancellous portion of the intercondylar bone. It was in use till mid 80's for almost 40 years until interlocking nails became popular. Even now they are selectively used.



Figures 18.12A to D

Showing (A) Kuntscher nail, (B) Tibial interlocking nail, (C) A rush nail (D) An ender nail.

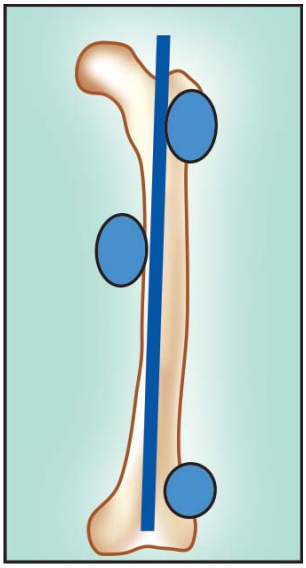


Figure 18.13

Showing the three point fixation of the Kuntscher intramedullary nail. *Gerhardt Kuntscher knew that the nail did not give much rotational stability and came out with Detensor nails with perforated ends for locking bolts. This became the precursor of all the interlocking nails of today.*

Rush Nail

This is a rigid nail designed by Lesley Rush and Lowry Rush, the American orthopedic surgeons in 1930's. These are rigid nails having a beveled tip and a curved hook like head with a bevel on it.

The direction of bevel on the head corresponds to the direction of the bevel at the tip so that the surgeon knows the direction of the bevel at the time of insertion of the nail after its entry into the bone.

They were used to fix the fracture of the forearm bones and supposed to work on the principle of trifocal buttressing, i.e. according to Rush 'A vase of flower'.

Biodegradable Implants

These implants have a modulus of elasticity very close to the bone . Even lesser than titanium. But they exhibit a very poor strength to axial and bending stress. Hence currently they are used only in fractures of minor nature, e.g. malleolar fracture, metacarpal fracture lateral condyle fracture. Osteochondral fracture, etc. The greatest advantage is that they get degraded and absorbed in 6 months to 1 year. Disadvantage observed is that some patients react showing signs of inflammation at the time of biodegradation process.

Dynamic Hip Screw and a Barrel Plate (Fig. 18.15A)

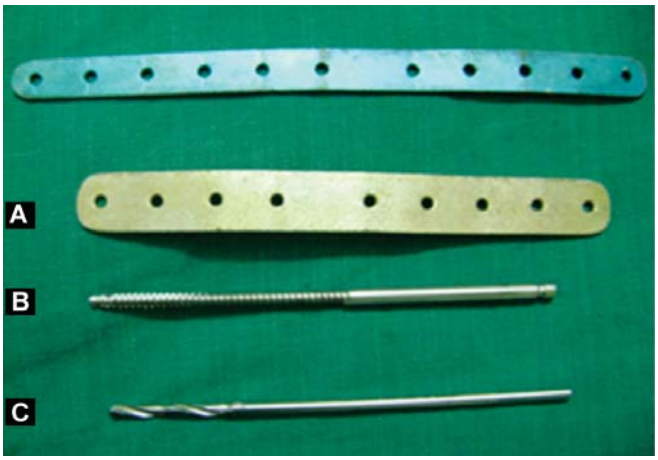
This is an implant which is used to fix the intertrochantric and some types of subtrochanteric fracture of the femur. The implant consists of a cannulated hip screw, a barrel plate and a locking screw. It works on the principle of rigid internal fixation. Firm fixation is brought about when the locking screw locks the hip screw and the barrel plate and is tightened. Compression is also brought about when the patient starts bearing weight and screw begins to slide within the barrel plate.

Jewet Nail (Fig. 18.15B)

This is a fixed angle nail plate device used to fix the intertrochaneric fracture. It has a cannulated triflanged nail fixed to a plate at a specified angle. These are available from 120-145° angles and chosen according to the angle desirable at fixation.

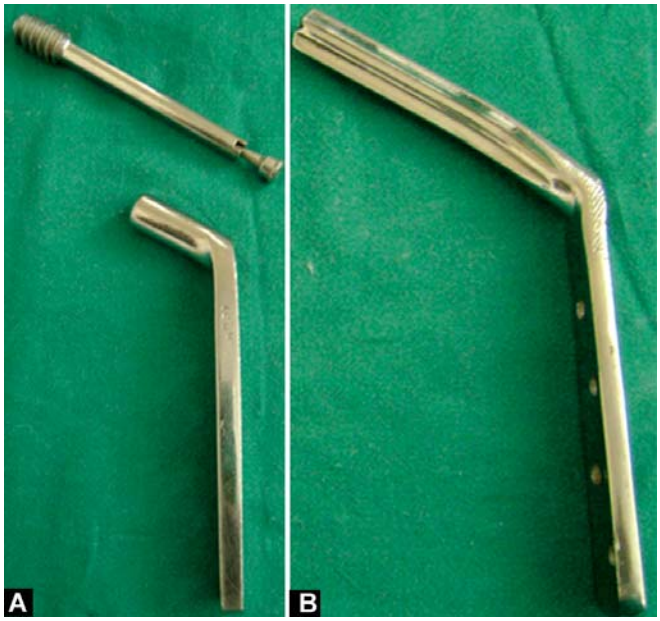
Cannulated Cancellous Hip Screw

Fixation of fracture neck of the femur started with the use of Smith Peterson Nail. To begin with this was a solid implant with 4 flanges. Later modified to a triflanged nail. Johanssen modified the nail by introducing a central canal into the nail. And in current practice the canulated cancellous screw is the standard implant that is used to fix the fracture neck femur. Minimum two and a maximum of three screws are used (Fig. 18.16).



Figures 18.14A to C

Some of the instruments used for plating: (A) Bending template to assess the curvature of the bone and bend the plate accordingly. (B) Tap for cutting threads after drilling hole in the bone. (C) Drill bit to drill a hole.



Figures 18.15A and B

(A) Dynamic hip screw and barrel plate (B) Jewet nail.

Figure 18.16

Cannulated cancellous hip screw for the fixation of fracture neck of the femur.

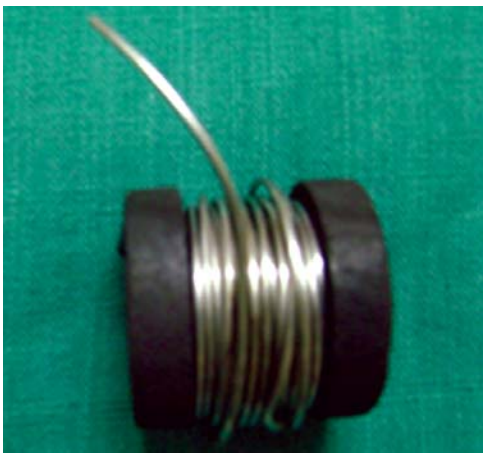


Figure 18.17

Stainless steel—SS wire used for Tension band and Circlage wiring in fixation of fractures. Available in different gauges.

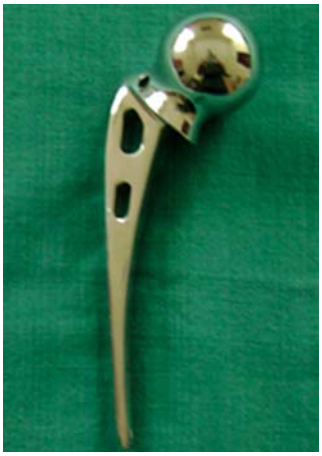


Figure 18.18

An Austin Moore's prosthesis, which is used in fracture neck of femur for replacement hemiarthroplasty. Note the fenestrations in the stem for the bone growth and to initiate a self-locking process. This is to be used when calcar femorale is sufficient. When calcar femorale is deficient Thompson's prosthesis with bone cement is to be used.

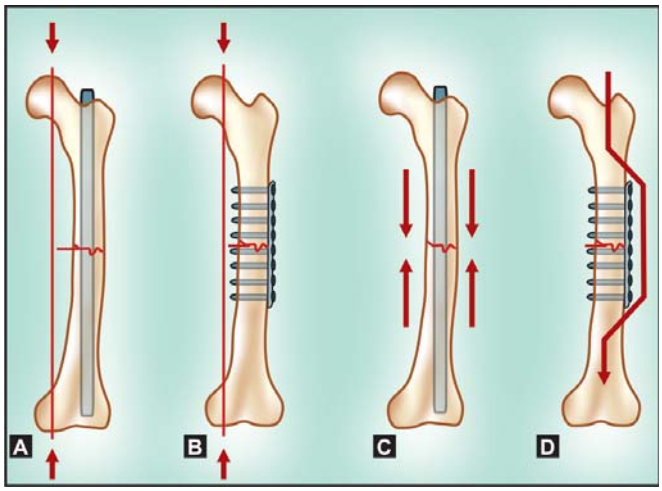


Figure 18.19

The bending moment arm in a nail and a plate fixation. The moment arm of a plate is longer when compared to a nail. Hence the plate is subjected to greater stress when compared to a nail. Thus the plate acts more as a load bearing device and the nail acts more as a load sharing device.

General Instruments Used in Orthopedics (Figs 18.20 to 18.29)

Mallet

A mallet is used to deliver a blow either to drive an implant or to drive an instrument, e.g. in the insertion of a nail or a prosthesis with the help of an impactor, in osteotomies to drive an osteotome, in bone grafting to drive a chisel, etc.

Chisel

It is a sharp instrument having one edge beveled used to chip the bone.

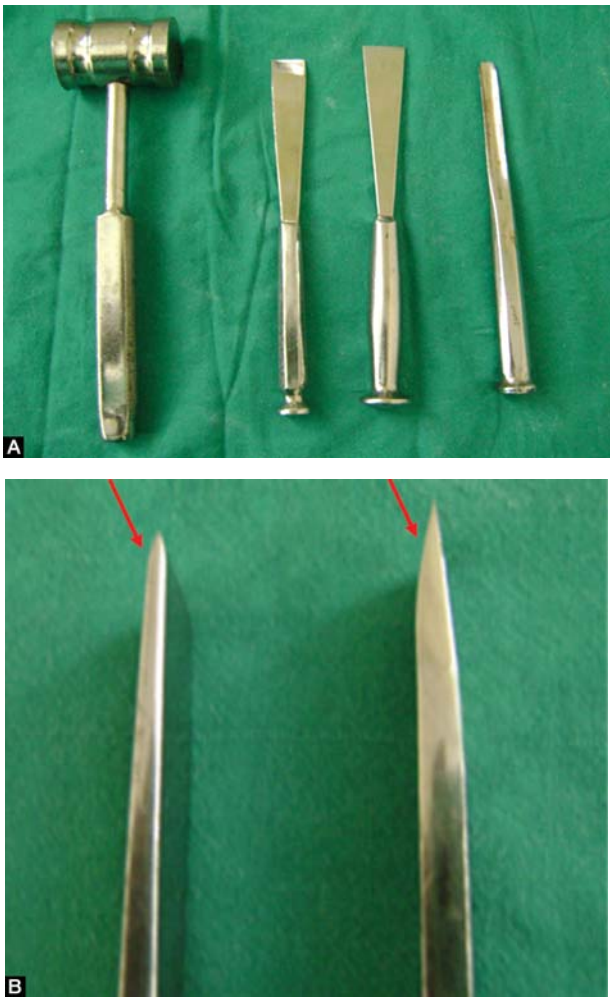
Osteotome

It is a sharp instrument having both the edges beveled. It is used to cut the bone.

Bone Gouge

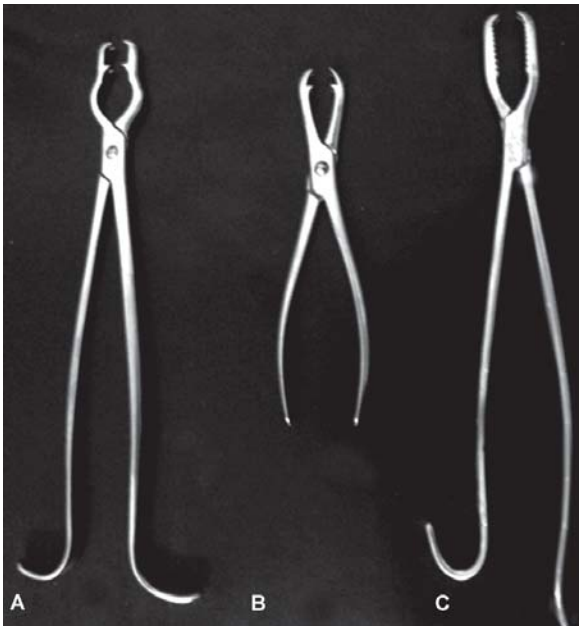
It is a sharp instrument with a trough used to gouge out the soft bone.

A mallet is necessary to drive these sharp instruments.



Figures 18.20A and B

(A) a Mallet, a Chisel, an osteotome and a bone gouge from left to right respectively. (B) The difference between the cutting edge of an osteotome and a chisel. An osteotome has beveling on both sides (left) where as a chisel has a beveling on one side (right).

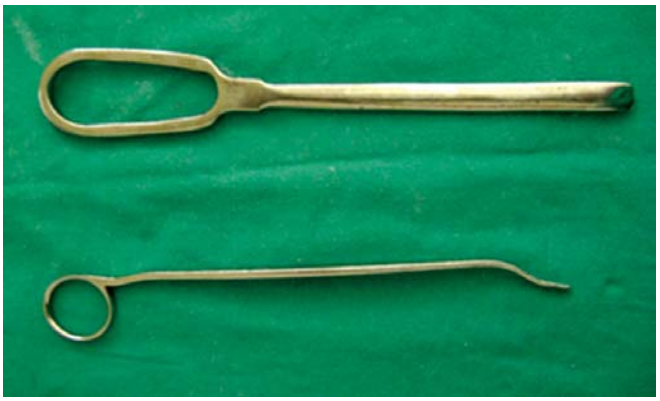


Figures 18.21A to C

The three types of bone holding forceps. (A) Lane's bone holding forceps. (B) Ferguson's Lion toothed (or Jawed) bone holding forceps. (C) Hey Groves bone holding forceps.

William Arbuthnot Lane

A brilliant surgeon who was working in Guy's hospital, London in the beginning of 20th century. He popularized what he called 'No touch technique' as a safety measure against infection by operating from a distance using instruments with long handles. All his instruments have long handles.



Figures 18.22A and B

The two types of bone levers. These are used to lever the bone free of its soft tissue. (A) Bristow's type. Sir Walter Rowley Bristow devised this lever. Hence the name. (B) Trethowen's type. Sir William H Trethowen devised this lever. Hence the name.

William Ferguson

He was a famous surgeon of 19th century. He designed this forceps to hold the head of the femur during disarticulation of the hip or excision of the head in tuberculosis of the hip. This forceps was also used to hold the jaw bones the mandible and maxilla.

Figure 18.23

The picture of a periosteal elevator used to strip the periosteum from the bone note the serrated portion in the handle which is used to for placing the thumb in order to have a firm grip.

Figure 18.24

A bone hook which is used to hook the bone and draw it up from within the soft tissue.

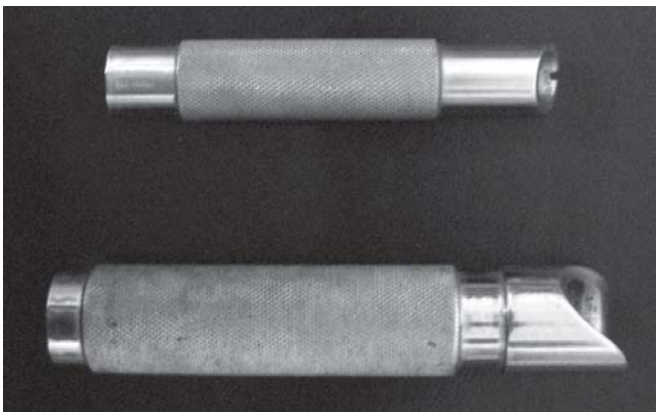
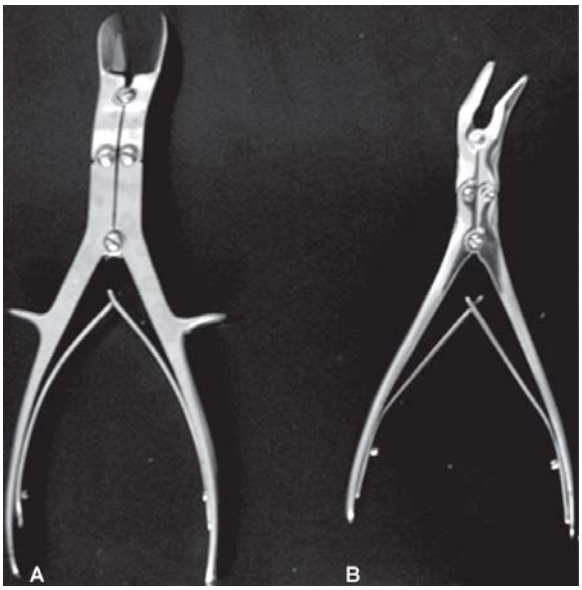


Figure 18.25

Two types of nail impactors. Note the slotted and the hollow design at one end and the knob at the other for delivering the blow with the mallet.

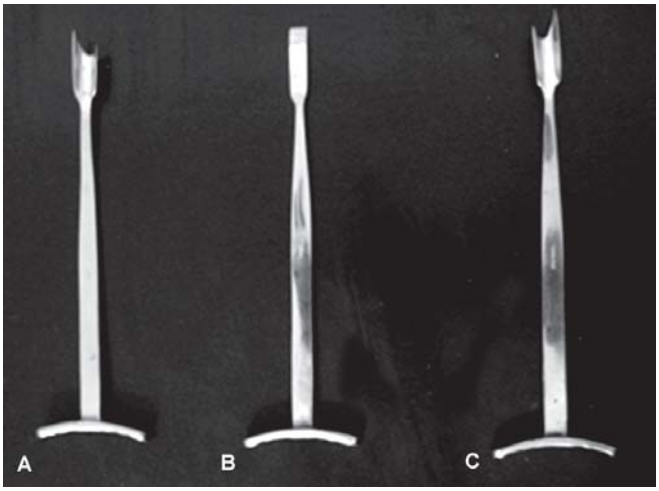
Figure 18.26

Lowmann's Bone clamp which is used to hold the fracture reduction along with the implant during plate fixation. Note the jaws and the knob with threads for obtaining a firm grip and adjustment.



Figures 18.27A and B

(A) Bone cutter and (B) a bone nibbler respectively. Note at the tip the sharp cutting edge of a bone cutter and trough with a sharp edge of a nibbler. The bone cutter is used for cutting small bone such as the phalanges, ribs, etc. The bone nibbler is used to nibble off sharp pieces of bones. The nibbled pieces get collected in the trough.



Figures 18.28A to C

(B) Smillie's meniscectomy knife. (A and C) Meiscectomy spike for the medial meniscus of let and right side respectively. The same is interchanged (A and C are interchanged) and used for the lateral meniscus of left and right side respectively.

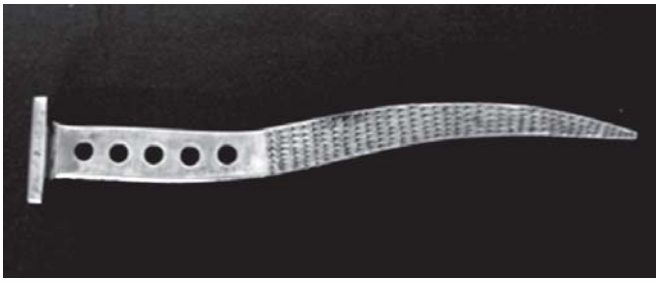


Figure 18.29

Medullary rasp which is used to rasp the medullary canal of upper 1/3 femur for proper seating of a prosthesis in hip arthroplasty. Note the fenestration at the upper part for insertion of a tommy bar for extraction and a flat knob for delivering a blow for driving the rasp in.

Designs of Limb Lengthening Apparatus

Illizarov (Fig. 18.30)

It is a ring fixator offering three-dimentional stability during correction.

Assembly is constructed by joining two 1/2 rings of equal diameter chosen according to the size of the limb using ring fixation bolts and nuts (E). A minimum of four rings are to be assembled.

These rings are next connected by threaded rods (C) and the distance between the rings is adjusted as per requirement.

Next the Illizarov wires are passed through the bone, two at the level of each ring preferably at right angles and fixed to the ring using wire fixation bolts and nuts (D) and tensionized by an instrument known as tensionizer. Extra-wire is cut and the cut end is bent flush with the ring.

The Illizarov principle is that ***a controlled progressive distraction and/or compression leads to tissue regeneration.*** This principle is made use of and accordingly a progressive distraction or compression stress (1 mm a day) is given at the desirable site by loosening and tightening the nuts of the assembly (refer text Fig, 1.18G).

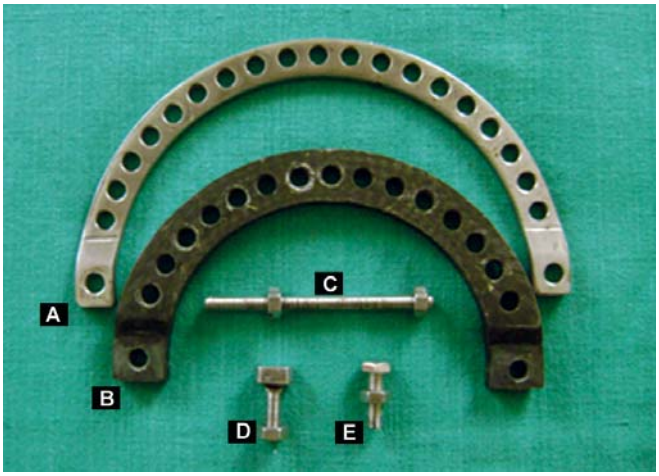


Figure 18.30

The basic components of Illizarov ring fixator. (A) Stainless steel 1/2 ring, (B) Carbon 1/2 ring, (C) Threaded rod, (D) Wire fixation bolt and nut, (E) Ring fixation bolt and nut. Carbon in rings are lighter and radiolucent when compared to stainless steel and being radiolucent have distinct advantage while taking radiographs.

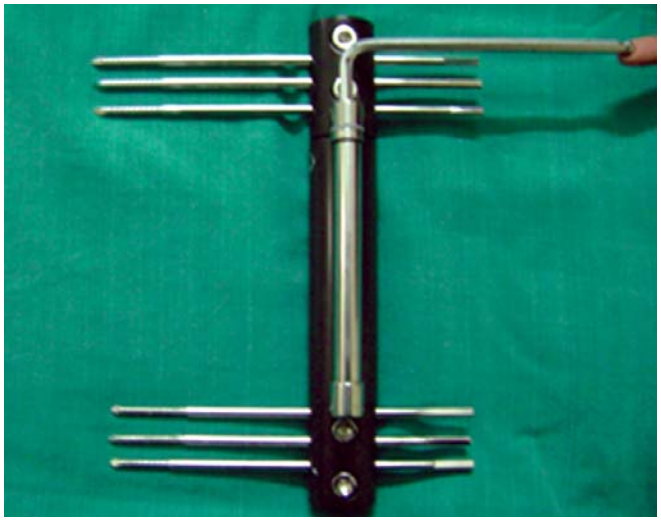


Figure 18.31

Orthofix type fixator, Uniplanar with Schanz pins *in situ* and CD devise pointed at by the insertion of an Allen key. One full turn of the key causes either distraction or compression by 1 mm.

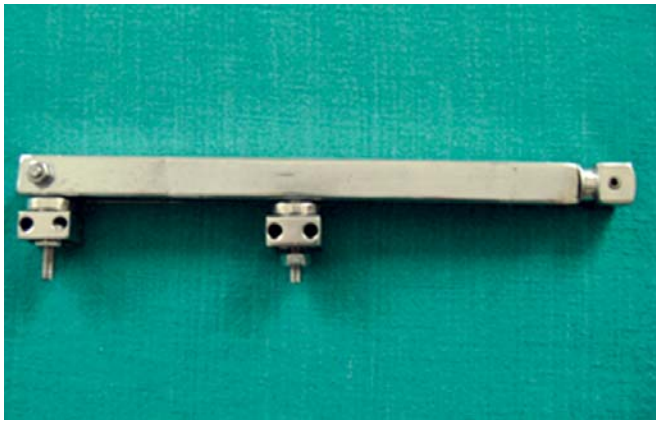


Figure 18.32

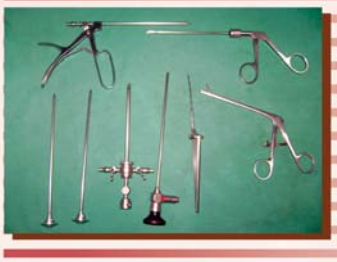
A Wagner type of Lengthening apparatus. It has a fixed clamp on one side, a turnbuckle device on the other and a gliding clamp in between. This clamp glides either way according to the rotation of the turnbuckle at the other end. 1 mm translation occurs for a full turn.

Orthofix Type (Fig. 18.31)

This is a uniplanar fixator device with an external compression-distraction (CD) unit. Has distinct advantage of ease of application and manipulation. But gives less rigid fixation (Refer text Figs 1.19A and B).

Wagner Type (Fig. 18.32)

This devise has a built in Turnbuckle mechanism for distraction or compression. One turn offers 1 mm movement.



19

Arthroscopy and Total Joint Arthroplasty

- *Arthroscopy*
- *Total Joint Replacement*

Arthroscopy

Arthroscope is an instrument which allows viewing of the interior of a joint. The procedure is known as diagnostic arthroscopy. When surgical procedures are performed under the guidance of viewing through the scope the procedure is called arthroscopic surgery.

Evolution of Arthroscopes

It was Professor Kenji Takagi who first viewed the interior of a cadaveric knee in the year 1918 using a cystoscope and later developed a scope known as Takagi scope in the year 1931. Dr Philip Kreuscher recommended the use of arthroscope for recognition and treatment of meniscal lesions in the year 1925. Credit goes to Dr Masaki Watanabe for not only developing the first modern arthroscope in the year 1958 but also performing the first ever arthroscopic surgery of excising a xanthomatous giant cell tumor as well as performing meniscectomy in the year 1962. In mid 80's the videoscope and motorized arthroscopic instruments were developed and advanced techniques started evolving. These days many surgeries are being performed using arthroscope . Arthroscopes of different sizes and designs are available for all the joints. Techniques are also highly advanced.

Components of an Arthroscope

The basic component of an Arthroscope is a Telescope with a metal casing. The scope has varying degrees of viewing angle of 0°, 30°, 70°, etc. at the distal end and an eye piece (to which a video camera can be connected) at the proximal end. It has a portal for the attachment of a light source. The inside of a scope consists of a series of lenses and optical fibers which transmit light from the light source to the interior of the joint (Fig. 19.1).

Technique

The procedure is carried out under general anesthesia with full muscle relaxation. Muscle relaxation is necessary as it allows good maneuverability of joints during the procedure. For proper visualization, the joint is distended with infusion of fluid. For the purpose of diagnosis a single portal generally is sufficient. But for doing arthroscopic surgery additional portals for insertion of instruments are necessary. Insertion portals for the scope as well as for the instruments are planned

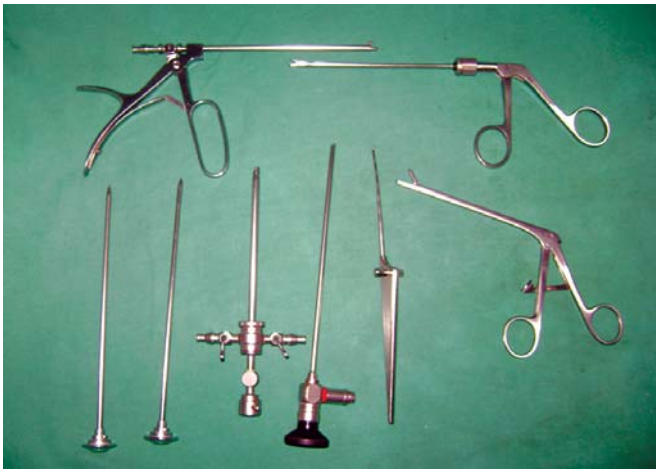


Figure 19.1

Picture showing the arthroscope along with some of the accessory instruments used for surgical procedures.

depending on the nature of the lesion (Fig. 19.2). The joint is viewed in a systematic manner. At the end of the procedure the joint is thoroughly irrigated the skin wounds are sutured and a pressure bandage is given.

Advantages of Arthroscopic Procedures

1. Less invasive, less morbid and less painful.
2. Day care procedure with no hospital stay.

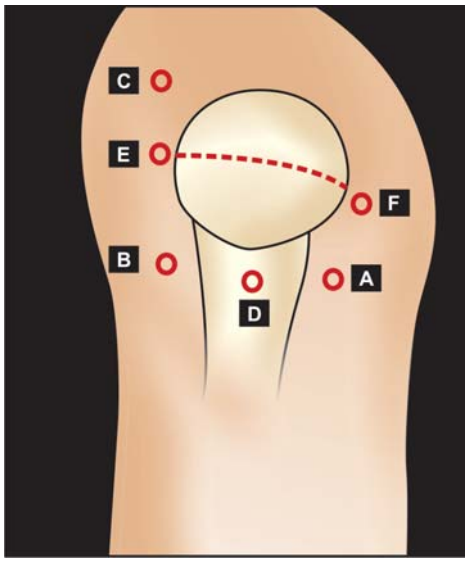


Figure 19.2

Showing different portals of entry for knee arthroscopy. A, B, C are standard portals D, E, F are additional portals. All are marked thus O. (A) Anteromedial (B) Anterolateral (C) Superolateral (D) Transpatellar (E) Midpatellar lateral (F) Mid patellar medial.

3. Early mobilization possible.
4. Direct visualization of the abnormality is possible.
5. Dynamic picture of the joint is recordable.
6. Good cosmesis.

Limitation of the Arthroscopic Procedures

Not indicated in advanced involvement of a joint, e.g. osteoarthritis, infective conditions, major joint injuries, etc.

Complications

1. Infection.
2. Hemarthrosis.
3. Reflex sympathetic dystrophy.
4. Thrombophlebitis.

Common Arthroscopic Procedures

Synovium

1. Synovial biopsy.
2. Synovectomy.

Articular cartilage

1. Excision of loose bodies, e.g. osteochondritis desicans.
2. Shaving of the degenerated cartilage, e.g. osteoarthritis.

Bone

1. Correction of patellar tracking.
2. Biopsy of an intra-articular lesion.

Ligaments

1. Primary repair, e.g. MCL.
2. Secondary reconstruction. ACL.

Meniscus

1. Partial or complete menisectomy.
2. Meniscal repair.

Lavage

Thorough irrigation and drainage, e.g. infective arthritis, osteoarthritis.

Total Joint Replacement (Arthroplasty)

When both the articular surfaces of a joint are replaced by an artificial joint (Prosthesis), the procedure is known as total joint arthroplasty. A total joint arthroplasty converts a destroyed, disorganized, painful joint which has lost function into a near normal, well organized, painless joint with good function. This is achieved by clearing the damaged portion of the bone and inserting an appropriate artificial prosthesis. Adequate preoperative planning is necessary for this surgery.

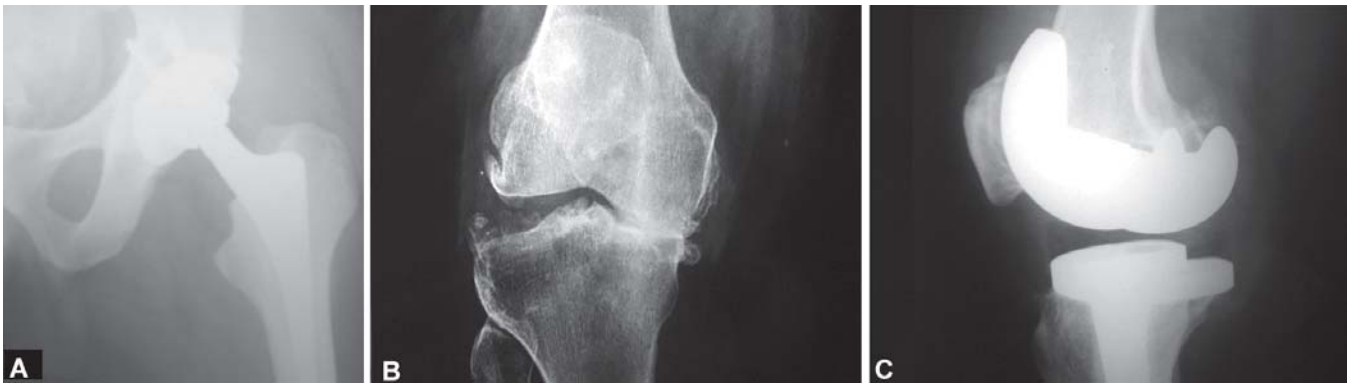


Figure 19.3

(A) Showing uncemented total hip replacement (B and C) Showing total knee replacement for osteoarthrosis.

Evolution

Sir John Charnley is considered as the pioneer of total joint replacement, especially hip. He popularized the concept of low friction arthroplasty in 1960's by designing a metal femoral stem component and a polyethylene cup component for the acetabulum. He did this work in the center for hip surgery, Wrightington' England. He used polymethyl-methacrylate, the bone cement which he initially borrowed from his dental friends. Cement acted as a filler and helped in the firm fixation of the components. The procedure proved to be very successful and subsequently many proshetic designs were developed. Because of the complications faced at the time of insertion of the cement and problems faced at the time of removal of a failed prosthesis, uncemented prosthetic designs developed. These designs were later modified into hydroxyapetite coated prosthesis aiming at firm fixation at bone metal interface. Currently, the use of cemented prosthesis is restricted to elderly people and uncemented prosthesis is always preferred in a young individual.

The year 1968 was the year of development of a successful knee prosthesis. A Canadian Orthopedist Frank Gunston from John Charnley's center replaced the knee with a metal and polyethylene component which were fixed to the bone by bone cement. In 1972, John Insall, MD, developed a design of total knee which became the prototype of all the total knees available these days.

Total ankle, total shoulder and total elbow are the other prosthesis which are available these days, for other joint replacements.

Success of joint replacement depends on preoperative planning and accurate intraoperative execution of the preoperative plan. The alignment of both the components of a total joint prosthesis should be perfect.

Computer assisted joint replacement surgery is also practiced in some centers for more accuracy.

Complications

Some of the complications encountered during joint replacement are as follows:

1. Infection.
2. Hemorrhage.
3. DVT and pulmonary embolism.
4. Loosening of the implant.
5. Dislocation.
6. Nerve palsies.

Postoperative Rehabilitaton after Joint Replacement Surgery

1. The patient is mobilized on the bed (joint movements) as early as possible after the initial pain subsides.
2. Weight bearing in hip replacement surgeries is usually delayed when compared to knee replacement surgeries and is related to the type of prosthesis used (cemented and uncemented).
3. Always a walking aid is given during the initial period of weight bearing.
4. Advise is given to avoid strenuous activities to increase the longevity of the prosthesis.

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