# C J Mieny, U Mennen: Principles of Surgical Patient Care - Volume II

## **Chapter 7: Orthopaedic Surgery**

## **Chapter 7.1: Fractures and Major Joint Injuries**

# K S Naidoo

#### **Introduction and Definitions**

A fracture is a break in the continuity of bone. The forces causing normal bone to fracture also damage the surrounding soft tissue. It is important to assess the extent of this soft tissue injury in planning treatment of fractures.

#### **Etiology**

Fractures are caused by direct or indirect violence. In direct violence the injuring force (a severe blow or a crushing force) is applied directly to the bone at the site of the fracture. In indirect violence the force is applied some distance from the fracture, ie, a fall on the outstretched hand may cause a fracture anywhere in the upper limb.

#### **Types of Fractures**

#### **Closed** (simple)

This is a fracture where the overlying skin is intact.

#### **Open** (compound)

This is a fracture where the overlying skin is breached so that the fracture communicates with the exterior. All open fractures should be regarded as potentially infected.

### **Complicated Fracture**

A complicated fracture is one where the fracture is accompanied by important soft tissue damage to, ie, nerve, vessel or internal organ.

#### Greenstick

This is an incomplete fracture which occurs in the bones of children. This occurs because of the springiness of the children's bones.

## Comminuted

A comminuted fracture is one where the bone is broken into more than two fragments. This indicates that severe trauma must be responsible, usually from direct violence.

#### Segmental

A segmental fracture is one where there are two complete fractures at different sites in a long bone with an intact segment between them.

### Line of Fracture

Transverse, oblique and spiral are terms used to describe the line of a fracture. Transverse fractures are caused either by direct violence or a pathological fracture. Oblique and spiral fractures are caused by indirect violence.

#### **Pathological Fracture**

This is a fracture occurring in abnormal bone. Also, the force producing a pathological fracture would be insufficient to break normal bone. A wide variety of congenital, inflammatory, neoplastic and metabolic causes may weaken the bone.

#### **Stress Fracture**

A stress or fatigue fracture is one which occurs in normal bone in a healthy patient. It is not caused by a single specific traumatic event, but by repeated stresses in the same direction, ie, marching or long distance running, etc.

#### **Clinical Features of Fractures**

A fracture should be suspected if after an injury the patient complains of pain in the region of a bone. In taking the history one must try and assess both the mechanism and severity of the trauma. This knowledge enables one to make a better analysis of the fracture which then helps in the planning of treatment. Also, one must search for a less obvious injury.

#### **Symptoms**

- Pain is the most important symptom of a fracture.

- Excessive pain, (out of proportion to the fracture) should alert one to the possibility of an associated vascular injury.

- Loss of function, whether partial or total, is usually a feature of a fracture.

Loss of sensation or motor power will suggest either a nerve or vascular injury.

## Signs

Local tenderness, swelling with deformity, abnormal mobility and the posture of the limb are the chief signs of a fracture. While crepitus is pathognomonic of a fracture, one must avoid over-exuberant attempts to elicit it in clinical practice.

Clinical examination must include an assessment of the state of the soft tissue as well as the presence of absence of neurovascular complications.

#### **Radiological Examination**

X-ray examination of the injured limb including the joints at each end of the bone is an essential part of fracture assessment. In all cases at least two views, taken at right angles to each other, are mandatory. Sometimes oblique views or other special views (ie, scaphoid views) must be taken. Tomography is useful in assessing details of a fracture. In some instances the fracture line may not be immediately visible (ie, certain scaphoid fractures or stress fractures), but repeat X-rays a few weeks later may reveal a fracture line. Bone scans are sometimes helpful in doubtful cases.

During the healing phase X-rays are used to check the maintenance of the reduction of fractures as well as the progress of union.

Clinically, a fracture is united when on testing the injured part, there is no discomfort or tenderness and the bone feels rigid. X-rays show adequate callus as well as trabeculations crossing the fracture line.

# **The Treatment of Fractures**

#### **General Measures**

The first important principle in treating fractures is to be aware of the systemic effects of the trauma that causes fractures, ie, shock, fat embolism, etc. These effects may not be obvious initially, but may appear only during the course of treatment. Secondly, the violence that produces a fracture may well cause injury to other parts of the body. So one must do a thorough examination of the patient looking for other possible injuries, especially the hidden injury, ie, dislocation and nerve injuries.

# Local Treatment

Broadly speaking, one of three situations may apply:

- The fracture is unimportant

Here the attention is directed to the soft-tissue injury, the bony injury being ignored. Examples are fractures of the flat bones such as the scapula, and avulsion of certain muscle attachments like the rectus femoris. Early mobilization is the preferred treatment.

- The fracture has the potential to unite

In this case the blood supply to the bone is adequate, and the fracture will unite irrespective of the method of treatment. However, if care is not taken to maintain the reduction of the fracture, then malunion will occur resulting in cosmetic and functional disabilities.

- Rigid immobilization of the fracture is required for union

In this case the blood supply to the area of fracture is precarious and therefore accurate reduction and internal fixation of the fracture is necessary. A subcapital fracture of the femur is an example.

## **Local Treatment of Fractures**

In considering the local treatment of fractures, one must always ensure that the general measures such as first-aid, proper transport and the treatment of shock, haemorrhage and associated injuries is not neglected. There are three specific aspects of fracture treatment:

- Reduction: by closed or open methods

- Maintenance of reduction until fracture union: by external splintage, internal fixation or external fixation

- Rehabilitation: restoring the injured limb (and the patient as a whole) to the preinjury state, or if irreparable damage has been done, to retrain the patient.

# Reduction

The general rule is that all displaced fractures must be reduced. This could be achieved by closed or open methods.

#### **Closed Reduction**

This could be achieved immediately by manipulation or gradually by traction.

### Manipulation

Adequate anaesthesia (intravenous, regional or general) is necessary. The technique of manipulation includes a series of calculated manoeuvres in which the fracture segments are first disengaged and the repositioned so that all displacements are corrected. This is an orderly procedure which must always be performed systematically. Basically, one reverses the forces that caused the fracture. If performed properly, excessive force is hardly necessary! A check X-ray must be performed to confirm reduction.

# Traction

This is a technique of gradual reduction of a fracture using skin or skeletal traction. An example is fractures of the femur in children or adults.

### **Open Reduction**

This method is indicated in those cases where closed reduction fails or closed reduction is not possible, ie, intra-articular fractures, fractures of the lateral mass of the humerus in children, etc. The risk of open reduction is the fear of introducing infection and

therefore strict aseptic measures must be instituted. Once bone is infected, it tends to be a life-long problem for the patient. During open reduction one must avoid damage to the nerves and vessels and also aggravating the initial bone injury. A tourniquet is always helpful.

## **Maintenance of Reduction**

External splintage: The general principle is to immobilize the joints proximal and distal to the fracture. The usual method is to use a plaster of Paris cast. Care must be taken in applying the cast to avoid the complications of swelling and pressure sores. Recently a wide variety of substitutes for plaster of Paris have been introduced.

Traction: Many forms of skin and skeletal traction may be used to control the deforming forces of a fracture. These include the use of a Thomas splint and fixed or balanced traction, Hamilton Russel traction, Dunlip traction (for elbow injuries), etc.

## **Internal Fixation**

Advances in technology and biomechanics have made it possible for a wide range of implants to be available for the internal fixation of fractures. The chief features of an implant include strength, durability and inertness. Previously, corrosion and breakage of the implant were major problems. Another feature of progress in internal fixation is the availability of appropriate equipment to insert the implants with greater ease and less damage to soft tissue and bone.

Certain basic forms of implants are available, ie, wires, screws and plates which can be introduced from one cortex to engage the opposite cortex, and rods which can be inserted into the medullary cavity of bone. Progress in biomechanics has greatly influenced the techniques of inserting these implants to give the best possible fixation of fractures as well as promoting union of the fractures.

# Indications

Indications for open reduction and internal fixation include:

- Irreducible fractures, ie, fractures of the lateral condyle of the humerus in children, displaced intra-articular fractures in adults, etc.

- Those cases where the blood supply to the bone may be precarious, ie, those cases where the risk of non-union is high, ie, subcapital fracture of femur.

- Where reduction may be difficult to maintain by closed methods - ie, forearm fractures in adults, ankle fractures, etc.

- In polytrauma - ie, ipsilateral fractures of femur and tibia.

- Pathological fractures.
- To facilitate nursing care ie, patients with head injuries.

In fixing fractures one must remember the risk of infection and take every precaution to prevent it.

### **External Fixation**

This is a technique in which a fracture is stabilized by inserting special screws at proximal and distal sites remote from the fracture and linking these screws with longitudinal bars. One therefore avoids using metal directly at the fracture site. It is a method which was developed as a result of problems encountered in treating open fractures. Since the reintroduction of this technique a wide variety of modifications of external fixation systems have been introduced. These modifications resulted from the experience gained in previous systems as well as due to a better understanding of the biomechanics of fractures. The main indication for the use of the external fixator is in severe open fractures. However, its use in other fractures is being explored, ie, pelvic fractures.

#### Rehabilitation

Irrespective of the special method of treatment of fractures, it is important to remember that one of the goals of treatment is to restore the injured limb to full function. This process must be started straightaway. The patient must be kept as mobile as possible. Physiotherapists and occupational therapists play a big part in this phase of fracture treatment. Previously, misconceptions existed with regard to how much activity was allowed. Now with better understanding of the effects of trauma, early movement is encouraged - of the patient as a whole as well as the injured part. While appreciating that immobilization may be necessary to maintain the reduction of a fracture, one must also keep in mind that inactivity will accentuate the stiffness caused by the associated soft-tissue injury that occurs with fractures. Therefore isometric exercises must be instituted immediately.

Another factor to consider is the psychological effects of trauma on individual patients. This may prove as much of a challenge to treatment as the fracture. In some cases of the injury on duty, the compensation aspects may influence the quality of result of treatment.

#### Retraining

In those cases where permanent disability results from injury to such a degree that the patient cannot return to his previous occupation, it may be necessary to train him for another suitable form of employment. As a result special retraining and resettlement centres are being developed. Medical social workers are an important link in this field.

#### **Complications of Fractures**

A wide variety of local and general complications may occur with fractures. These complications may occur at the time of the injury, or during the early treatment phase or long after the fracture occurred. The immediate and early complications may be regarded as associated injuries.

## **Immediate Complications**

- Severe haemorrhage external or internal.
- Injury to internal organs.
- Injury to local soft tissue skin, muscle, tendon, nerve or vessel.

# **Early Complications**

#### Local

- Skin and soft-tissue necrosis.

- Gangrene or Volkmann's ischaemia from vascular damage.
- Pressure sores and nerve palsies from splintage or traction.
- Infection and wound breakdown.
- Loss of fracture position.

### General

#### - Tetanus

- Gas gangrene
- Crush syndrome
- Venous thrombosis and pulmonary embolism
- Fat embolism
- Pneumonia
- Urinary tract problems
- Delirium tremens.

# Late Complications

- Delayed and non-union
- Malunion
- Late wound sepsis
- Failure of internal fixation
- Joint stiffness and contracture
- Avascular necrosis
- Sudek's osteodystrophy
- Psychological problems including compensation neurosis.

# **Local Complicationsw of Fractures**

### **Skin Complications**

- Open fractures will be discussed later.
- Fracture blisters are due to elevation of the superficial layers of the skin by oedema.
- Skin necrosis may be caused by displaced bone or an ill-fitting plaster.
- Bed sores may occur in the elderly or in paralysed patients.

#### **Muscle Complications**

In most fractures some damage occurs to muscle fibres. This may heal by fibrosis and contribute towards the post-traumatic stiffness. Very occasionally ossification of damaged muscle may occur (Myositis ossificans). The cause is unknown. The elbow region is the commonest site.

## **Tendon Complications**

- Immediate tendon rupture is uncommon with fractures except for transverse fractures of the patella and olecranon. These fractures are essentially disruption of the extensor mechanism of the respective joints.

- Avulsion fractures: This fracture is produced by forcible muscle contraction which pulls off a fragment of bone at its site of insertion, ie, avulsion of the greater tuberosity of the humerus by the *supraspinatus* tendon or extensor tendon insertion from the distal phalanx (mallet finger); the rectus, psoas and patellar tendons can also cause similar lesions.

- Late rupture: In some cases late rupture of a tendon may occur six to twelve weeks after the fracture, ie, rupture of extensor pollicis longus with fractrues of the distal radius.

#### **Nerve Complications**

- Early: Major nerves may be injured by a fracture especially at sites where the nerves are closely related to bone, ie, axillary nerve and fractures of the surgical neck of humerus, the radial nerve with fractures of the humerus, the ulnar nerve with fractures of the medial epicondyle, the median nerve with supracondylar or wrist fractures, the sciatic nerve with posterior dislocations of the hip, etc. Many of the nerve lesions are caused by sudden stretching of the nerve during injury. There is no permanent damge and rapid recovery occurs. This lesion is called neuropraxia.

In some cases the nerve damage is more severe and involves the axons. This is called axonotmesis. These lesions may take three to six months to recover. Very occasionally a nerve is completely severed in closed fractures. This is a neurotmesis. Spontaneous recovery cannot occur.

In general terms, nerve injuries associated with closed fractures have a good prognosis and should recover. If recovery has not occurred by the time the fracture has united, there may be an indication for surgical exploration. In selected cases there may be a place for earlier surgical intervention, ie, significantly displaced medial epicondyle fractures with ulnar palsy.

- Delayed: If emaciated or elderly patients lie in bed with their legs in external rotation, a lateral popliteal palsy may result. Faulty use of crutches may cause a radial palsy.

Late ulnar neuritis may be caused by a progressive cubitus valgus deformity.

### **Vascular Injuries**

Injury to major vessels may occur at any site but the elbow and knee regions are common sites because at these level the vessels bifurcate and are also anchored by muscle attachments. The vascular injury includes complete division, contusion with intimal damage and subsequent thrombosis, and compression by swelling within a tight compartment. One must always be on the lookout for vascular injuries. Warning signs include excessive pain out of proportion to the fracture, and the limitation of passive extension of the affected digits. Action must be taken promptly if a disaster is to be avoided.

## **Bone Complications**

- Malunion: This refers to a situation where the fracture hasunited in an unsatisfactory position. This occurs either because the fracture was not adequately reduced, or it slipped during the after care. Both cosmetic and mechanical problems result from malunion. A corrective osteotomy is necessary in significant malunion.

- Delayed and non-union: The rate of union of a fracture is influenced by the age of the patient, thesite of the fracture and the blood supply to the area concerned. In adults fractures in the upper limb take roughly six to twelve weeks to unite while lower limb fractures may take twelve to 24 weeks. Fractures in cancellous ends and short bones take from six to eight weeks. In children the times are halved and even less.

- Delayed union: This means that the fracture is taking longer than usual to unite. This state may progress to complete union or end up with non-union. Clinical examination and X-rays help to distinguish the two types. In the first type (likely to unite) there will be decreasing pain and tenderness at the fracture site - increasing stability of the fracture and radiological evidence of callus formation. In the second type (non-union type) there is instability of the fracture and radiologically no callus formation with the fractured ends becoming sclerotic.

- Non-union: This is the state where the fracture remains ununited with a gap at the fracture site and sclerosis of the fractured ends. The fractured ends may be bridged by fibrous tissue (fibrous union) or a false joint may form (pseudoarthrosis). The essential diagnostic difference between delayed and non-union is the presence of sclerosis at the fracture fragments.

The causes of non-union could be classified as follows:

- Initial injury - significant soft tissue loss, bone loss and soft tissue interposition.

- Local factors - this includes poor blood supply to the area, infection and a pathological lesion.

- Faulty treatment - poor treatment may be the cause. This includes distraction of the fracture, inadequate immobilization and premature removal of splintage.

### **Treatment of Non-Union**

One must assess each case on its merits and rectify the cause. In general two principles must be applied. The first is to provide better stability of the fracture usually by internal fixation methods. Secondly, bone induction must be encouraged by the use of bone grafts. Recently various forms of electromagnetic stimulation of bone formation has come into vogue. Infected non-unions pose a special challenge and complex treatment may be needed to control sepsis and promote bone union.

- Avascular necrosis: This is the condition where death of bone occurs because of poor blood supply. Certain bones are more at risk than others, the best examples being the head of femur, the scaphoid and talus.

- Sudek's osteodystrophy: This is a complication that occurs after certain wrist and hand or foot injuries. The injuries are usually trivial. There is an exaggerated stiffness, swelling, warmth and sweating of the part which is also discoloured. Severe pain is present. X-rays show marked osteoporosis. The exact cause is not known, but it is thought to be due to an overreaction of the sympathetic nervous system.

#### **Open Fractures**

These are fractures in which the skin is breached so that there is direct communication between the fracture and the exterior. Bacteria can therefore enter and contaminate the fracture haematoma. The break in the skin can occur from within (caused by the fracture fragment) or from without (by the injuring force). The extent of the wound can vary from a puncture to major skin and soft-tissue loss. The damage as well can vary from simple lacerations to severe crushing injuries. The bony injury can also vary from minor fractures to severely comminuted fractures or even bone loss. Survival of the affected part may well be threatened by the injury.

The chief risk in open fractures is infection. Treatment must therefore be started promptly. These injuries are surgical emergencies. One must prevent further contamination during transport.

The treatment of open fractures is discussed elsewhere, but it is useful to remember that the treatment of the soft tissue and bony injury must be a co-ordinated affair. One can easily aggravate the already severe injury by bad judgement and improper treatment. The external fixator is a useful aid in managing these injuries.

### **Fractures in Children**

Bones in children differ from adult bones in being softer, more pliable and more porous. They can also tolerate a greater degree of deformation. As a result, greenstick fractures are common in children. Two types occur. The first is one where the force causes the bone to buckle (torus fracture). The second is the type where there is a break in one cortex while the opposite cortex bends. Complete fractures may also occur in children and the fracture patterns may be similar to those of adults. Relative to their size, a greater amount of force is required to cause severe fractures in children. A feature of children's fractures is that the strong periosteum tends to remain intact although it may be stripped off a wide area. The intact periosteum helps one to both reduce and maintain reduction of the fracture. A good example is displaced fractures of the forearm bones. In adults open reduction and internal fixation is the usual method of treatment, while in children closed reduction and immobilization in plaster of Paris is invariably successful.

The presence of the growth plate in children's bones give rise to a special group of fractures. The epiphysis may separate from the growth plate, a fracture across the growth plate or a longitudinal force may crush part or whole of the growth plate. Another feature is that the growth plate is weaker than the ligaments and capsule of joints therefore, unlike in adults, complete ligament ruptures and dislocations are less common in children. There will be a fracture of the growth plate instead.

Salter and Harris (1963) classified growth plate injuries into five types:

I The epiphysis separates completely (along the layer of hypertrophic cartilage cells) from the epiphyseal plate without a metaphyseal fracture.

II Is similar to type I with the epiphysis taking a triangular fragment of metaphysis with it.

III Here a shearing force causes a vertical split extending from the joint surface to the epiphyseal plate, and then along it to the periphery.

IV The vertical fracture extends through the epiphysis, epiphyseal plate and metaphysis.

V There is crushinhg of the epiphyseal plate.

### **Treatment and Prognosis**

Types I and II epiphyseal injuries have a good prognosis. They can easily be treated by closed methods. In types III and IV injuries accurate reduction and maintenance of reduction is necessary to prevent fracture morbidity (growth arrest and deformity). These types therefore tend to require open reduction and internal fixation with Kirschner wires. Type V injuries are not easy to diagnose initially because of the lack of ossification of the injured area. They therefore tend to be diagnosed retrospectively. Type V injuries cause growth arrest.

# **Fractures of the Pelvis**

Injuries of the pelvis may give cause for considerable morbidity and even mortality. This is because the bony pelvis contains major nerves and vessels, the terminal part of the intestinal tract and the urogenital structures. The force required to fracture the pelvis could easily damage the local visceral structures and also cause damage to other parts of the body. Haemorrhage and shock is common and is the first priority in management. Unfortunately, the unwary miss important visceral damage because their attention is diverted by the bony injury.

#### Causes

The usual causes include road traffic accidents, falls from heights, direct blows and sporting injuries.

#### **Assessment and Diagnosis**

General and local examination is conducted along conventional lines. Blood at the external urethral meatus, perineal haematoma or the inability to pass urine suggest urological injury. Rectal examination helps in assessing the state of the rectum (and prostate in males) as well as providing information about the sacrum and coccyx. It is worth remembering that in some cases the clinical features of shock may not be present at the time of examination, but one should always anticipate this possibility on the basis of the injury. Resuscitation must always be started before X-ray examination. It should be mandatory for any patient with polytrauma to have X-rays of the pelvis even if fractures are not obvious.

#### **Classification of Pelvic Fractures**

These may be closed or open. An open fracture carries great risks of infection irrespective of the extent of the wound.

Pelvic fractures may be classified in many ways but essentially one needs to assess:

- stability of the fracture and

- whether it involves the hip joint.

## **Stable Fractures**

These include isolated fractures of the pelvic ring, ie, fracture of the pubic ramus, fracture of the iliac wing or avulsions of muscle attachments (sartorius, rectus or hamstrings).

## **Unstable Fractures**

This implies that the pelvic ring has been broken at two or more places. Several combinations may occur but the main types are:

- The anterior portion of the ring may be crushed and all four pubic rami fractured with the loose fragment being driven backwards.

- *Hinge type* - the pelvis opens like a book due to an injury at the symphysis or on one side of the pubis.

- *Vertical type* - the fracture involves the pubis as well as the sacro-iliac area causing both an outward and an upward displacement.

- Fractures of the pelvis may also involve the hip joint or sacrum and coccyx.

## **Principles of Treatment**

The first priority is resuscitation and thorough assessment of, in the first place, obvious, and secondly, hidden injury.

### **Treatment of the Bony Injury**

As far as bony injuries are concerned, all unstable displaced fractures of the pelvic ring must be reduced and treated by external splintage, internal fixation or external fixation. These fractures pose a challenge and should not be undertaken lightly. Complicated fractures are particularly difficult to manage. Stabilization of the fracture by internal fixation or the use of the external fixator may facilitate the care of visceral or soft-tissue injuries - especially if more than one operation at intervals of days or weeks becomes necessary.

#### **Joint Injuries**

# **Types**

Joint injuries include:

- sprains
- subluxations
- dislocations
- fracture dislocations.

### Sprain

This injury can vary from a minor tear of the joint capsule to one where major ligaments are completely ruptured alone or in combination with others, ie, collateral ligament rupture with cruciate and meniscal damage in the knee. Careful assessment and appropriate treatment must be provided to avoid future morbidity.

#### Subluxation

In this case there is partial displacement of the articular surfaces of a joint with the constituent bones still remaining in some contact. Treatment depends on the joint concerned.

#### **Dislocations**

This occurs when there is complete separation of the articular surfaces of the constituent bones. The cause may be traumatic or pathological. In traumatic cases the soft tissue damage may be so severe as to render the joint prone to further dislocations or subluxations. Associated neurovascular injuries may be present, ie, brachial plexus or axillary nerve palsy with shoulder dislocations, sciatic palsy with hip dislocations and arterial damage with knee dislocations.

Principles of treatment are:

- reduction of the dislocation
- immobilization (until the soft-tissue damage heals)
- treatment of associated neurovascular complications on their merits
- rehabilitation.

If closed reduction fails, open reduction must be performed. In selected patients surgical treatment may be preferred in the first instance, ie, knee dislocations with complete collateral ligament rupture.

### **Fracture Dislocations**

In this case the dislocation occurs with one or more fractures, minor or significant. The lateral fractures may affect the congruity of the joint surface or the stability of the joint. Neurovascular complications may also occur.

In major joints open reduction and internal fixation is the preferred method of treatment. The after care would include the principles outlined in the case of simple dislocations.

#### Comment

#### **Fractures and Major Joint Injuries**

### E V D Neluheni

In broad outlines I fully agree with Dr Naidoo's view of the principles of diagnosis and management of fractures and major joint injuries. Much of my commentary was directed at improving minor changes in the text.

However, I would like to add a few points:

1. It is better to speak of stable immobilization, rather than rigid immobilization. The latter is a concept popularized by the Association Osteosynthesis (AO) or ASIF Group of Switzerland. Rigid immobilization implies absolute absence of motion. This concept is not correct, because motion does occur at a microscopic level at the fracture site. Furthermore, a small biological degree of movement is necessary for normal bone healing and this is not allowed by the so-called rigid internal fixation advocated by the AO group.

2. A method of local anaesthesia which may be used in a controlled theatre situation is infiltration of the local anaesthetic into the fracture gap. This may be considered in the elderly where general anaesthesia is not indicated.

With insurance policies around, one must remember than the so-called compensation neurosis may affect the result of any treatment. This is particularly true when it comes to musculo-skeletal injuries. 4. When one refers to blood supply to a fracture site, this automatically should include the venous drainage system as well, which usually runs outwards via the periosteum into the soft tissues. It is extremely important to protect this soft tissue draining system, since any backlog or obstruction of the venous system is detrimental to the circulation of the blood in the bone. This of course results in ischaemia of the bone.

5. As far as the epiphyseal injuries are concerned, I would like to point out that apart from the well-known Salter Harris classification, another group, namely the type VI has been added by Mercer Rang, which describes the type of injury where the perichondral ring around the physis is avulsed. The resultant haematoma undergoes ossification. This acts as a tether on the one side of the physis. It results in growth disturbance.

6. Stabilization of unstable pelvic fractures has been found to be very helpful in preventing further haemorrhage from the fracture site and pelvic veins. One should therefore not delay an operative stabilization with an external fixator of the injured pelvic ring.

7. It is important to understand the concept of non-union. There is no unanimity among surgeons regarding the definition of non-union. Definite non-union exists when there is no potential for fracture healing. A true non-union (pseudoarthrosis) is present when the bony ends are radiologically tapered and sclerotic and the medullary cavity is closed off. The fracture is still mobile but with little or no tenderness. Sepsis at the fracture site does not lead to this situation per se. Bony healing does occur in the presence of sepsis. Infection thus causes delayed union and not non-union.

## **Chapter 7.2: Hand Injuries Including Hand Infections**

### U Mennen

### Introduction

Injuries to the hand come about in many ways. The effect of the various insults on the tissues of the hand is by and large similar. The body always responds by an inflammatory reaction. The result is, among others, swelling and cell proliferation. If these reactions are not dealt with urgently, irreversible changes will occur leaving a hand which may lack balance, power, suppleness, excursion, tactility and aesthetic appearance.

## Definition

Since swelling is the result of a noxious stimulus, it could be stated that all swollen hands are injured hands and should be managed as emergencies.

# Pathophysiology

Inflammation is the cause of all hand swellings. The swelling is due to oedema fluid, hyperaemia, vascular dilatation, or extravasation of fluid. The oedema fluid is a protein-rich transudate which is "irritative" to normal tissue. Granulation tissue and eventually scar tissue forms if the oedema fluid is not eliminated in a few days, resulting in irreversible stiffness.

The oedema fluid develops within a short time and by virtue of its space-occupying nature, will cause immobility of joints and gliding tissues due to the mechanical blocking effect.

If this fluid is not removed, adhesions will form between the delicate gliding planes which will further handicap movement.

The hand assumes a position of rest when injured. The ligaments and webspaces are relaxed. If this position is maintained without correct functional splinting, reduction of swelling or mobilization of the hand, the ligaments will adapt and become permanently shortened, and the webspaces will narrow irreversibly. It is virtually impossible to "stretch out" or "pull out" shortened ligaments and narrowed webspaces. The result is therefore a permanent non-functional stiff hand, which is often aggravated by algodystrophy (autonomic or Sudeck's dystrophy).

From the above it must be clear that all swollen hands, regardless of the type of injury, should be regarded as severe and potentially crippling.

### Etiology

Any agent or factor which causes swelling of the hand may be regarded as traumatic and harmful to the tissues. These factors can be divided as follows:

- Physical injury, ie, direct blow, cuts, crush injuries, gunshot wounds.

- Infections: acute or chronic infections caused by various organisms, ie, staphylococci, streptococci, fungi and leprosy.

- Burns caused by thermal, electrical or chemical insults.

- Metabolic diseases, ie, gout.

- Auto-immune diseases, ie, rheumatoid arthritis.

- Iatrogenic factors such as extravasation of intravenous fluids, incorrect dressings, suspensory slings, inadequate mobilization of an injured or post-operated part and accidental intra-arterial injections.

# **Clinical Symptoms and Signs**

When dealing with an injured hand, it is of paramount importance to get the following information which will influence the primary management and planning of later stages of such an injured hand:

- Sex
- Age
- Occupation
- Dominance
- Hobbies
- General diseases
- Previous injuries
- Previous and present treatment
- Mental and physical health.

With this information as background, one should proceed to examine the injured hand.

It should be noted that the hand starts at the cerebral cortex and ends at the fingertips. The hand is also part of the body and it is therefore important for the examiner to know the functional status of the patient, ie, contralateral upper limb, ipsi-lateral shoulder and arm, and both lower limbs.

A full neurovascular examination of the injured hand should be done. The easiest and best way to record any deformities or lesions of a hand is to draw a schematic outline of the volar and dorsal aspects of a hand and indicate directly on the schematic drawing the extent and shape of the abnormalities. This type of recording is more comprehensive and precise than using many words.

It is always helpful to photograph the injured hand, which not only serves as a precise record of the exact status of such a hand, but also serves to compare progress in the healing of such an injured part.

It is difficult to judge the amount of swelling of a hand exactly. One way is to measure the circumference of a hand using a measuring tape. Another helpful device is the volumeter. This apparatus is based on the amount of displacement of water by the injured hand. The left and right hands can be compared and the progress of the injured hand could be followed reasonably accurately.

After the general and specific clinical examination, the patient should be sent for an X-ray of both hands. It is important to have a comparison of the normal side to exclude any normal variations.

Should the patient be in a state of shock due to blood loss and/or pain, this should obviously be dealt with in the appropriate fashion, ie, intravenous fluid and pain killers. The injured hand and arm should be temporarily splinted. Open wounds should be dressed with a sterile dressing, and inspected only in theatre under sterile conditions.

# **Decision About Procedure**

In the case of trauma by physical injury, burns or infection as well as some iatrogenic injuries, the final decision about the procedure and further management can be done only in theatre after the wound has been debrided and cleansed thoroughly. It is at this stage that the information regarding the total status of the patient and the needs of the particular patient will

influence the final decision. If we deal with an unskilled labourer who has injured the ring finger of his non-dominant hand, the approach would be more radical and definite in order to get the patient back to work as soon as possible. In this case precision grip and cosmetic result might not be of prime importance. The patient may only need a good strong hand with scars placed in such a position as not to cause unnecessary tenderness. The approach in a skilled artist would be very different. Mobility and digital balance as well as normal two-point discrimination would rate much higher. The patient would also be prepared to undergo a longer period of treatment and rehabilitation to enable him to continue with his specific kind of skilled work.

The healing capabilities of children are much better than in adults and some parts or tissues that are vascularly compromised may stand a much better chance of survival if sutured back.

A very important factor to remember is that the initial management of a physically injured hand is the most important stage. Primary correct handling of such an injured hand gives much better results than later reconstruction. It should also be remembered that in the inexperienced hand a better result would be achieved if the wound is thoroughly cleansed and debrided initially, covered by a sterile dressing and left for an elective list the following day. If a clean wound is present, the surgeon is inexperienced and some delay to reach a specialized centre is anticipated, the initial management should also be primary wound toilet and suturing of the skin only. The patient should then be transferred as soon as possible to a referral centre.

### Treatment

### **General Management**

### Splinting

All injured hands should be splinted in the functional position unless a specific good reason exists such as an injured flexor or extensor tendon. The functional position is based on a number of determinants, among others the lateral ligaments, volar plate, joint capsule, muscle, tendons, webspace, sub-cutaneous tissue and skin.

The functional (physiological) position is as follows:

- Wrist joint: lateral 20-30° dorsiflexion.
- Anteroposterior: neutral.
- Metacarpo-phalangeal joint: 90° flexion.
- Interphalangeal joints: extension.

- First webspace, ie, between thumb and index metacarpals: wide abduction and opposition.

The function of the physiological splint is not only to prevent shortening of the various tissues, but also to alleviate pain and promote drainage of oedema fluid via the venous and lymphatic systems.

The splint should be used only for as long as is absolutely necessary, ie, until mobilization can take place. The splint should be either permanently fixed for a certain period or be removable to allow the patient mobilization exercises.

## Mobilization

It is the aim of rehabilitation of an injured hand to eventually have a supple and fully functional hand. Mobilization should be started as soon as possible after the injury. The advantages of early mobilization are not only to promote a better function, but also to reduce swelling by the pumping action. Reduced swelling will result in less pain. Another advantage is that early mobilization will prevent adhesions of the delicate gliding systems, and of articular spaces. A further function of early mobilization is to "indicate" during the healing phase to the various tissues such as ligaments and tendons in what way collaged has to be laid down in order to achieve maximal strength of these tissues.

Mobilization could be implemented in two various ways:

- active mobilization
- passive mobilization.

Passive mobilization is achieved through physiotherapy which is assisted by ultrasound, local heat, interferential therapy, etc. and occupational therapy which includes specific movements and actions to help the patient regain those functions which are necessary for his daily activities. It should be pointed out that the patient's own effort is by far the most important contribution to the whole rehabiliation program.

Mobilization cannot be substituted by hand therapy or gadgets. The function of the hand therapist is mainly to guide and educate the patient in his passive and especially active exercise program.

In general, slings should not be used at all on the patient with a hand injury. This emphatic statement is based on many complications suffered by the dependent and incorrect position of an injured arm. The hanging arm prevents drainage of the swollen hand. Furthermore, the wrist is in flexion while the metacarpo-phalangeal joints are in extension and the inter-phalangeal joints in flexion. The first webspace is adducted and the patient keeps his hand fixed to the body. This position is totally unphysiological and speells grave consequences for a normal functioning hand. Apart from arm and shoulder girdle pathology, there is no indication whatsoever for slings to be used in the injured hand.

## Elevation

Since the patient is reluctant to move his swollen and painful injured hand, the only alternative means to reduce swelling is by elevation. This elevation should ideally be just above the level of the heart, regardless of the position of the patient. In the injured hand, a dependent position for 5-10 minutes will increase the oedema to such extent that many hours of elevation will be needed to regain the preswelling stage. The patient should therefore be taught to keep the hand elevated at all times, regardless of his activity during the day. Mobilization exercises should also be done in the elevated position.

### Skin Cover

In general, wounds in the hand heal very well. Where a large area of skin loss has occurred in the hand, it is of great importance to get early skin cover. Fibroblast tends to produce collagen within 48 to 72 hours and this could be prevented by skin cover. Furthermore, skin cover acts as a biological dressing, prevents infection and protects the underlying tendons, nerves, blood vessels, muscles and bone. It further prevents damage to deeper structures from repeated dressings and exposure to the atmosphere and drying out of tissues.

Various methods of skin cover could be employed:

- Split skin: Split skin is a very helpful method of covering large areas of skin loss. In those wounds where the surgeon is uncertain how to cover the defect, a split skin graft would serve as a good temporary measure. In most cases the split skin graft will suffice anyway. Split skin could be placed on any surface as long as fibrin is present.

- Full-thickness grafts: These may be used in children on large surface defects. The skin is harvested from the groin. No fat should be included in this graft. The donor area is closed per primam.

For adults a full-thickness graft should not be larger than a 50 cent piece.

A major disadvantage of a full-thickness graft in the adult is the total absence of sensation, unlike the deep protective sensation present with the split skin graft.

- Pedicle flaps: It is worth while to acquaint oneself with the technique of covering extensive full-thickness skin losses by pedicle grafts such as groin flap. The groin flap is a safe and reliable method especially to cover areas as the first webspace and the dorsal aspect of the metacarpo-phalangeal joints.

- Free vascularized flaps: These flaps are very useful in special cases and should be attempted by experienced microsurgeons.

# **Basic Surgical Principles**

#### Anaesthesia

A thorough examination, exploration, debridement and repair can be done only with adequate anaesthesia.

The anaesthesia can be achieved by:

- Local anaesthesia: This method is not advocated because of additional damage to the local nerves by the needle point or compartment syndrome of the nerve itself with resultant intraneural fibrosis.

- Regional anaesthesia: Various methods can be employed such as brachial plexus block, axillary block and intravenous "Bier" block. These methods are advantageous in emergency cases and give adequate anaesthesia for all major hand surgery. If long-acting local anaesthesia is used plus general sedation of the patient, operative procedures can easily continue for a number of hours.

- General anaesthesia: This method is used in the patients who will not tolerate regional blocks, such as children. It is also used when distant flaps such as groin flaps are employed for skin coverage.

## **Bloodless Field**

A bloodless field is necessary to enable the surgeon to explore the wound and allow for accurate diagnosis and repair. It is achieved by applying a broad pneumatic tourniquet around the upper arm and either using a lightly applied Esmarch bandage or by simply elevating and squeezing the arm. It should be noted that in the presence of sepsis an Esmarch bandage could be dangerous because of proximal spread of infection by the squeezing effect.

The tourniquet pressure should be 30-50 mm Hg above the patient's systolic pressurew. The time allowed for the tourniquet is 90-120 minutes maximum.

### Debridement

By debridement is meant a thorough mechanical cleansing of the wound using adequate irrigation fluid and meticulous surgical removal of devitalized tissues.

This "wound toilet" is done in the following way:

- The wound is initially covered with a sterile dressing while the surrounding skin of the hand and arm is washed with antiseptic soap.

- The surgeon scrubs as for a major operation. The wound is uncovered and thoroughly washed with antiseptic soap using the gloved hand. A brush or any abrasive instrument should not be used. The wound is thoroughly irrigated by using a balanced electrolyte solution (not Saline) or sterile water.

- A bloodless field is secured.

- A meticulous surgical debridement is done by removing all the devitalized tissues as well as foreign bodies and dirt which may be present. Extreme care must be taken to protect vital structures and to constantly plan ahead in preserving any tissue that may be used for reconstruction.

This surgical debridement can best be done with magnification such as operating loupes (two to three times magnification).

This procedure takes time and should be executed with great care and skill.

- The wound is rinsed out once again with a balanced electrolyte solution.

- Haemostasis should be achieved using a bi-polar coagulation. Ligatures are avoided where possible because of the danger of creating septic foci.

- The various structures should now be repaired (see below "Specific Conditions").

- Dressings are applied (see below).

- Antibiotics (see below).

- Analgesics (see below).

#### Dressings

The main function of dressings is to protect the wound from further contamination while allowing free drainage of serous and sanguineous fluids. It should also be applied in such a way as to allow early mobilization. The dressings should not obstruct any venous drainage and should not act as plugs to prevent free flow from a drained abscess.

A method to ensure free flow from the wound, is by applying one layer of paraffin gauze, covered with cotton swabs which will absorb the draining fluids.

Part of the dressing is to apply a volar splint which will not only support the painful hand, but will alleviate the pain and promote drainage. This splint will also add to the co-operation of the patient in the rehabilitation phase.

#### Antibiotics

A distinction should be made between prophylactic and therapeutic use of antibiotics.

Prophylactic antibiotics are given to patients with poor resistance, ie, suppressed immuno responses. These patients lack normal defence mechanisms to resist micro-organismal contamination of wounds.

Therapeutic antibiotics are given to patients with suspected pathogenic contamination, with soiled wounds and with wounds due to human bites.

Antibiotics should be used with care and discretion to prevent the formation of resistant strains.

In general, the clean or unsoiled wound will not need an antibiotic. The debridement and irrigation procedure should be sufficient to prevent infection. In the case where the wound has been sustained for some eight hours or more before the debridement procedure or some contamination is suspected, the wound should only be dressed, and sutured at a second-look operation 24 to 48 hours later. In the case where contamination is suspected by special organisms such as in human bites or farm-related injuries, the appropriate antibiotics should be given. In the case of human bites it is necessary to combine various antibiotics because of the mixed organisms found in the mouth. The antibiotics of choice will be penicillin (ampicillin), aminoglycoside (Gentamycin) and metronidazole (Flagyl).

### Analgesics

It is totally unnecessary in this moder day and age to let patients suffer from unnecessary pain. Good analgesics should be given in sufficient amounts post-operatively. Analgesics such as intravenous diclophenac (Voltaren) are helpful because of their pain-killing effect as well as their anti-inflammatory actions. Another advantage is that these drugs do not lead to dependence.

### **Specific Conditions**

Since this chapter deals with the principles in the management of hand injuries, a detailed in-depth discussion of each injury is inappropriate. The reader is referred to more comprehensive descriptions on the management of the various individual injuries. A broad outline will, however, be given on the management of specific conditions.

#### **Injury by Physical Trauma**

For simplicity's sake the various structures that can be injured by physical trauma will be dealt with separately, ie, skin, tendons, nerves, vessels, joints and bones.

## Skin

The principles have been discussed above. For burn injuries please see below under "Injury by burns".

### Tendons

Tendons are classified into extensor tendons and flexor tendons.

Extensor tendons give far better results than flexor tendons due to a less intricate gliding mechanism. Extensor tendons should be sutured immediately using the modified Kessler-Tajima suture technique. The hand is splinted with a volar plaster of Paris splint with the wrist in 45° dorsiflexion, and the MP joints in near full extension, ie, the splint is extended to support the proximal phalanges. This splint is kept on for five weeks after which active flexion exercises should take place. The splint is worn as a night splint for another three weeks. Should the patient be unable to flex the fingers fully, a tenolysis could be done three to six months post primary suture.

Extensor and tendon injuries which need special mention are mallet, swan-neck and boutonnière deformities.

- Mallet deformity: The pathology is a cut of the conjoint tendon as it inserts into the base of the distal phalanx. The conjoint tendon may avulse the dorsal part of the joint surface causing the distal phalanx to subluxate volarly due to the pull by flexor digitorum profundus. The basic treatment consists of a splint which keeps the distal interphalangeal joint in extension for eight to twelve weeks.

- Swan-neck deformity: The most common causes of traumatic swan-neck deformity is a lesion of the flexor superficialis or secondary to an untreated mallet deformity. Recognition of these lesions and their early treatment will make difficult reconstruction unnecessary.

- Boutonnière deformity: The middle slip of the extensor tendon is either cut or ruptured. This will result in unopposed flexion of the PIP joint and hyperextension of the DIP joint. The treatment consists of immediate splinting of the PIP joint in extension leaving the DIP joint free to flex, for eight weeks.

Flexor tendons are much more difficult to treat because of the high risk of adhesions and lack of necessary excursion to enable the patient to fully flex the fingers. These tendons should also be sutured as an emergency by experienced surgeon using the Kessler-Tajima type suture. The sutures should be placed in the volar third of the tendons because of the relative avascular nature of this area. It is of paramount importance that the tendon should not be handled by any instrument since damage to the delicate paratenon will inevitably result in scar formation and adhesions. The two ends of the flexor tendons are pulled together to allow easy contact. It should be noted that a tight suture has many disadvantages such as strangulation and bulging of the sutured area which will prevent easy gliding in the sheath. Tendon has the intrinsic potential to produce new tendon through the tenoblasts which most probably originate from the intrinsic blood vessels of the tendon. It is therefore possible for a tendon to heal and close a gap of 1 or 2 mm at a suture site. Should the suture site be untidy, a 6/0 or 7/0 running suture could be used to tidy the area. It must be stressed once again that the handling of the tendon should be extremely atraumatic since the slightest damage to the delicate paratenon will certainly mean a bad result. It is acceptable that the inecperience surgeon who with tendon injuries in the finger and arm only to suture the *flexor digitorum profundus*. However, should the injury be in the wrist area, and especially if the ulnar nerve is also involved, it is important to attempt suturing the *flexor digitorum profundus* as well as the flexor digitorum superficialis. The latter tendons may be important for later intrinsic muscle reconstruction.

The flexor tendon sheath should at all costs be closed and therefore the incision made in the flexor sheath to approach the cut tendon, should be fashioned in such a way as to allow easy closure, ie, a Z or square flap. The function of the flexor tendon sheath is important to prevent adhesions and to promote healing through the sinovial fluid. It also helps to smooth down the sutured area during the healing phase.

Postoperatively the hand is splinted as follows:

- Wrist in  $30^{\circ}$  of volar flexion.
- MP joints in 90° of flexion.
- IP joints in 30° of flexion each.

The dorsal splint which is applied has to reach the tip of the fingers to prevent extension of these joints. The volar aspect of the fingers up to the mid palmar crease should be left free to allow the patient to passively and actively flex the fingers during the healing stage. This splint stays on for four weeks after which the patient is fitted with a second splint for another three weeks. This splint should hold the joints in the following position:

- Wrist neutral.MP joints 90°.
- IP joints 30° of flexion.

The patient still continues with active and passive flexion exercises. After this period of seven weeks the patient is allowed to remove the splint and to do active well-controlled flexion and extension exercises taking care not to force the fingers in extension. The splint, however, should be worn at night for at least another three weeks.

The hand therapist who is experienced in this kind of injury is invaluable during these stages in advising the patient.

After the ten weeks of splinting and controlled mobilization, the patient may use the hand for normal activities taking care not to force any residual stiffness. The stiffness should be treated by the hand therapist using various modalities to release or stretch adhesions. Should some residual stiffness be present after six months, a tenolysis may be considered. After such a tenolysis the patient must immediately start flexing and extending his fingers and continue rigorous mobilization activities for not less than six weeks.

Late reconstructions such as tendon grafts, tendon trasnfers, Hunter rods, etc, will never reproduce the good results which could be achieved by primary tendon suture and wellcontrolled post-surgery hand therapy.

The suture material for the tendons suggested is a 3/0 atraumatic suture. We prefer a braided suture material because of greater strength and pliability than a monofilamentous type.

#### Nerves

Nerve sutures, just as tendon sutures, should be done with utmost care and a scrupulous atraumatic technique. Tension on the suture line should be avoided at all times, since this tension will lead to ischaemia with resultant intraneural fibrosis for which very little can be done.

The suture material used will depend on the experience of the surgeon and the facilities available. The minimum magnification required to do a nerve suture is at least a two and a half to three times using an operating loupe. The suture material is an atraumatic 8/0 to 9/0 stitch. The number of sutures inserted should be just enough to approximate the ends of the nerve. This approximation is done very carefully to allow light contact only between the two ends. Under no circumstances should the nerve ends be pulled tightly together. This will cause the delicate fasciculi to bend sideways and prevent the axons from crossing the joints. As with the tendons, the nerve is capable of bridging a few millimetres gap, and therefore a slight gap would be more advantageous than a tight suture.

The sutures are placed in such a way as to prevent rotation of the nerve. The guide to use is the fascicular pattern on both sides of the nerve ends, as well as the longitudinal artery which should be aligned. Just enough sutures are used to approximate the ends. Any unnecessary sutures will add unnecessary trauma and foreign material to the sutured area. The body is compelled to react to this trauma and foreign material by way of an inflammatory reaction and granulation tissue formation. This type of nerve suture is the epineural suture which to the average surgeon should give reasonable results. It is still contentious whether the fascicular suture is superior to a well executed epineural stitch.

The splint must be applied in such a way that the nerve will be free of any tension during the healing phase. The splint stays on for three to four weeks after which the patient receives well-controlled hand therapy to mobilize the joints and to re-educate the patient for sensation. The patient must be warned against trauma such as burns from cigarettes, hot food and drinks, and abrasions from repetitive insults.

Nerves should be repaired as a primary procedure. The sooner a major nerve is repaired, the sooner the function will be restored. In the grossly contaminated wound an exception to this rule would be to tack the nerve ends down to the surrounding tissue by one monofilamentous suture. This will prevent the nerves from retracting. Nerves are not marked. A surgeon who feels capable and confident to do an operation should also be knowledgeable about the anatomy. It is a basic principle that an injured structure should be explored by approaching if from the normal area towards the area of injury. It is a perpetual and serious error to try and "mark" the nerves.

After a peripheral nerve suture enough time is allowed for the wound to heal, say three to five weeks, while maintaining the functional position of joints affected by the motor loss of the nerve. It is a basic principle not to delay tendon transfers for too long. Ligamentous changes take place which cannot be restored by reconstructions. For example, intrinsic muyscle reconstruction is done three to four weeks after the initial *Nervus ulnaris* injury and tendon transfers for *Nervus radialis* paralysis is also done one month after the initial injury.

#### Vessels

Fortunately the hand is very well supplied by vessels (arteries, veins and lymphatics). The normal function of the hand and especially the sensory modalities are directly linked to the blood supply.

In case of vessel damage and compromise of blood supply to some part of the hand, one should not hesitate to suture these vessels. In general, two veins are sutured for each artery to accomodate adequate drainage.

Once again magnification should be used to suture the vessels. Suture material will depend on the size of the vessel, but an 8/0-9/0 suture is ideal.

#### Joints

The anatomical construction of the joints in the hand are complex. Various tissues may be involved during an injury to a joint, such as collateral ligaments, volar plate, capsule, cartilage, sinovial membrane, bone and tendons.

Intra-articular fractures are reduced to achieve a smooth gliding surface. The intraarticular space should be irrigated to remove all loose fragments.

Dislocated joints are reduced as soon as possible, and checked by a control X-ray for joint congruity. If the joint space is not absolutely parallel, soft-tissue interposition may be implicated. The treatment would then be open reduction. It should be pointed out that the metacarpo-phalangeal joint of the thumb and index finger have the reputation of being notoriously difficult to reduce without operative intervention. The reason is the special anatomical configuration which creates a buttonhole deformity. The harder one's attempts at reducing the dislocation, the tighter the grip around the metacarpal necks become, making reduction extremely difficult.

Hyper-extension injuries of the MP, PIP and DIP joints involve injury to the volar plates. Mostly this is a painful condition which does not need more than conservative treatment by "buddy splinting". However, should the patient be unable fully to flex and extend the joint, a control X-ray will indicate an incongruous joint which needs operative clearance of the joints.

Avulsion fractures by the volar plates and the tendons should be treated as intraarticular fractures, ie, open reduction and fixation.

## **Collateral Ligaments**

It is important to repair avulsed or torn collateral ligaments of especially the following immediately:

- Ulnar aspect of the thumb MP and IP joints.
- Radial aspect of the index MP, PIP and DIP joints.
- Ulnar aspect of the small finger MP, PIP and DIP joints.

If these ligaments are not repaired, instability during normal use of the hand will result. The other ligaments can be treated conservatively by "buddy strapping" since they are protected and supported by the neighbouring fingers.

#### Bones

### **Phalanx and Metacarpal Fractures**

These fractures can be classified into stable and unstable fractures.

Stable fractures include fractures such as the impacted fractures of the neck of the fifth metacarpal. These fractures are mobilized as soon as possible to prevent adhesions and to regain full function.

Unstable fractures. In general terms these fractures are stabilized by some means of internal fixation such as Kirschner wires before mobilization is started the following day. However, some fractures have a reputation for a bad functional result after operative reduction and internal fixation such as the fractures of the proximal phalanx of the fingers. These could be treated by a dorsal POP splint with the wrist in  $30^{\circ}$  of dorsiflexion, the MP joints in  $90^{\circ}$  of flexion and the PIP and DIP joints in full extension. The palmar aspect of the fingers should be left free to be fully flexible. The injured fingers could be strapped to their neighbours to prevent rotation.

Intra-articular fractures are also classified as stable and unstable. If a major part of the joint surface is involved and displacement is present, an open reduction and internal fixation may be indicated.

Some intra-articular fractures such as the avulsion intra-articular fracture of the first metacarpal (Bennett's fracture) can be reduced, closed and pinned percutaneously with Kirschner wires. The position of the thumb is held in abduction and opposition. A POP splint is applied for six weeks to protect the K-wires.

## **Carpal Fractures**

By far the most common carpal fracture is the fracture of the scaphoid bone. The following régime is suggested:

- All wirst injuries should be X-rayed for possible fracture.

- If on the initial X-ray no obvious fracture line of the scaphoid is visible, the wrist is nevertheless protected by a POP circular cast including the thumb to the proximal phalanx for three weeks, after which a control X-ray is taken. If no fracture line is visible on the second set of X-rays and the patient is without pain, a retrospective diagnosis may be made of "sprained wrist".

However, if a fracture line is seen, this would represent a crack fracture with the cartilage covering still intact. This would make the scaphoid fracture reasonably stable and it may therefore be treated conservatively by an above-elbow POP cast including the proximal phalanx of the thumb leaving the fingers including the MP joints, free to move. This POP cast is removed after six weeks and the wrist re-X-rayed. Should there be any signs of non-union such as a sclerotic line, cyst formation, widening of the fracture line, etc, an open reduction with bone grafting and with or without internal fixation is done. The POP cast is re-applied until union is evident.

- On the other hand, if the fracture shows signs of union, a POP cast should be reapplied until union is complete. The total healing time for a simple undisplaced scaphoid fracture is about 12 weeks.

- If on the original X-rays a scaphoid fracture is visible, a posterior-anterior X-ray should be taken of the wrist in ulnar and radial deviation. This procedure will distinguish between the stable and unstable fracture.

- A stable fracture should be treated along the lines as described above.

- An unstable fracture, ie, one that displaces on ulnar or radial deviation, should be openly reduced and internally fixed including bone grafting if necessary.

## Wrist Fracture Dislocations

One of the worst injuries to the hand is a dislocation or fracture dislocation of the wrist. This diagnosis is usually made from the history and clinical examination. On X-rays various signs are helpful. Often the only sign that might be present is an avulsion of the ulnar styloid and/or radial styloid process.

A very high index of suspicion should be exercised when viewing the X-rays. Incongruity of the joint spaces will indicate ligamentous injury and therefore wrist instability. By simply counting the number of carpal bones one may discover more than the normal eight which will indicate a fracture, and it might again be part of a major fracture dislocation of the wrist. For further information the reader is referred to more comprehensive works on the subject.

If a diagnosis of a fracture dislocation of the wrist is made, a simple volar splint is applied and the patient referred to a specialized centre for further management.

A lunate dislocation is usually part of a major wrist dislocation, ie, peri-lunate injury to ligaments with a dorsal displacement of the wrist and subsequently volar dislocation by the capitate of the lunate. "Lunate dislocation" should therefore not be seen as an isolated bone injury but as part of a more extended injury. Closed reduction of the lunate will not only cause damage to the median nerve but is insufficient treatment for a major wrist problem. The treatment for wrist instability due to injury to the ligaments will be an open reduction and internal fixation. Ligaments reconstruction may also be needed for stability.

# **Fingertip Injuries**

Traumatic amputations of the fingertips are very common. If the bone is not exposed, conservative treatment by twice daily dressings will produce a cosmetically and functionally excellent result, which is difficult to match with flaps or skin graft.

In cases where bone is exposed, a little trimming will enable primary closure of the wound. If only a small part of the nailbed remains, breaking of the nail will result. It may then be wise to remove the entire nailbed and germinal layer.

## **Injury by an Infection**

One of the most serious injuries to the hand is by micro-organismal invasion. All hand infections should be regarded as emergency cases and be treated aggressively along the following lines:

- Elevation of the hand.

- Drainage of all collections of pus and walled-off abscesses. The drainage procedure should be done over the area of maximal fluctuation. Note that the dorsum of the hand will in most instances of infection be swollen due to the lymphatic drainage to the dorsum. This swelling should not be confused with an abscess. The incision should be an excision of a diamond-shaped piece of skin. Any other type of incision such as a straight line or an ellipse of skin will not suffice. A diamond shape will, after evacuation of the abscess, get smaller, but will remain open right up to the end for drainage in contrast to the other types of incision. The wound will heal without much scar tissue and will not need a secondary suture. The abscess should be approached using a blunt forceps to spread the tissues apart and to prevent damage to nerves, vessels, tendons, etc. After the abscess is found and the pus drained, the abscess wall should be cleaned out using a finger covered with gauze.

- Dressings. Under no circumstances should a plug be used. This illogical method prevents drainage of the abscess and defeats the whole operative exercise. A layer of paraffin gauze covers the diamond-shaped excision of skin. The wound is further packed using cotton gauze swabs. This dressing should be removed after 24 hours to allow for wound baths and mobilization exercises.

- Splinting. A POP volar slab is applied to provide rest and alleviate pain. The splint should be removed the following day to allow for mobilization exercises.

- Mobilization. This should commence within 12-24 hours after drainage. If the hand is still painful, a lukewarm handbath will be most comfortable and soothing.

- Antibiotics. Antibiotics are given for mixed infections such as human bites. Another indication for administration of antibiotics would be in the very early stages of infection. This may be enough to either abort the infection or localize the abscess. A third indication for the use of antibiotics would be in streptococcal infection which will be recognized by a rapidly spreading erysipelas. Other indications for the use of antibiotics include mycobacterium marinum, tuberculosis and fungal infections. For more information on the treatment of infections in the hand, the reader is referred to the relevant references.

## **Burn Injuries**

This is a collective term for three completely separate type of injuries, ie, thernmal, chemical and electrical burns.

## **Thermal (Heat Burns)**

The general management of thermal burns is covered by another chapter in this book.

The damage done to the tissues of the hand by the heat depends on:

- the exposure time
- the temperature of the heat
- the latent energy of the burning material.

First aid would initially consist of immediate removal of the burning material and/or application of cold compresses. This will arrest further penetration of the heat and damage to deeper structures.

In hospital a surgical debridement as an emergency procedure should be carried out. All the devitalized and dead tissues is removed regardless of the depth of the injury. The skin defect should immediately be covered with a split skin graft or a local flap. The split skin graft will act as a biological dressing, prevent the invasion of organisms and suppress the activity of fibroblasts from producing scar tissue. Even if the split skin graft does not take at the first setting, much will have been gained. A helpful hint is to take double the amount of skin if possible and store the skin in a balanced electrolyte solution at 4-10 °C. This skin can be used by applying it to the area where the initial skin graft has not "taken". This application can be done painlessly in the clean-air side-ward without any anaesthetics.

The skin graft areas are covered by one layer of paraffin gauze which in turn should be covered by cotton gauze swabs impregnated in acriflavin.

The hands should be splinted as follows:

For burns on the dorsum of the hand the wrist is flexed to  $30^\circ$ , the MP joints in  $90^\circ$  of flexion and the PIP and DIP joints each in  $45^\circ$  of flexion. This position is held with a POP splint for five to seven days. The reverse holds true for injuries on the volar aspect where the wrist must be splinted in  $45^\circ$  of dorsiflexion, and the MP joints, PIP and DIP joints in full extension. These also are kept on for no longer than five to seven days.

Under special circumstances internal fixation with K-wires for no longer than one to two weeks may be advantageous to allow for wound inspection and re-dressing.

## **Chemical Burns**

The extent and type of injury depend entirely on the type and concentration of the chemical. The severity of the injury may be apparent only after a few days and repeated debridements may be necessary.

However, although one needs to compromise, the basic principles must always be kept in mind, ie, the joints must be splinted in the correct position, the skin defects should be closed as soon as possible, even if only temporary, the hand must be elevated, etc.

## **Electrical Burns**

The term "electrical burn" is a misnomer. The wound on the skin reflects only a very small part of the actual injury deeper inside. The electrical current travels along the pathway of least resistance, ie, blood vessels, nerves, muscle, medullary canal and spinal cord. The damage caused to the deeper structures may be extensive. The major complication from the so-called electrical burn is usually a compartment syndrome which may be of limb-threatening proportions. Another complication due to the large volume of tissue damage is crush syndrome and kidney failure.

The treatment of electrical burns follows the following principles:

- A high index of suspicion for compartment syndrome, crush syndrome and renal failure must be exercised.

- All the affected compartments must be released.

- Large volumes of intravenous fluid must be administered to prevent clogging of the kidneys and excretion of the absorbed, damaged tissues.

- Repeated debridement of devitalized tissues must be undertaken regularly.

- One should not hesitate to amputate digits or even limbs in cases of severe electrical burns. The rehabilitation of burned tissue is extremely difficult apart from the life-threatening crush syndrome which the patient might develop.

- Splinting of joints and mobilization during the healing phase must be executed as discussed above regardless of the presence of open wounds.

- It is an interesting phenomenon that the tissues injured by an electrical burn are "paralysed" for 14 to 21 days. The inflammatory reaction by the body is severely delayed and may complicate reconstructive surgery and rehabilitation. Skin grafting usually does not take and should be either used only as a biological dressing, or applied when the first phase of granulation tissue appears.

## **Injury by Metabolic Disorders**

These disorders include, ie, gout, diabetes mellitus, etc. The discussion of the specific treatment of these conditions falls outside the scope of this chapter.

Since the injury caused by these metabolic conditions is met by an inflammatory response by the body, the basic principles in the handling of this type of swollen hand are similar to those discussed above.

Medical treatment and the hand swelling therefore go hand in hand.

## **Injury Through Auto-Immune Diseases**

The major auto-immune disease is rheumatoid arthritis in its many various forms.

Again the medical treatment of these conditions should go hand in hand with the treatment of the swollen, painful hand as discussed under general principles.

### **Injury Through Iatrogenic Causes**

Injury through iatrogenic causes is not an uncommon occurrence. These causes may include skin damage by application of local drugs or extravasation of fluids. Damage to nerves may be caused by surgical exploration or injections. Vascular injuries may be caused by arterial canalization and blood sampling. Simple causes such as incorrect bandaging may have severe consequences.

These lesions are for the most part avoidable.

Should such an unfortunate incidence occur, the most important step would be early diagnosis and immediate attention to the problem. Apart from the specific treatment the general guidelines must also be observed.

### Conclusion

Hand injuries, if treated early, aggressively and correctly, may be rewarded with a satisfactory result.

If the basic guidelines of management are followed, many of the major disabilities could be avoided. These basic principles include elevation, splinting in the functional position for as long as necessary, mobilization of the injured parts as soon as possible which should be supervised by hand therapists, skin coverage of the denuded area to prevent scar formation and antibiotic treatment when indicated.

#### Comment

## Hand Injuries and Infection

#### S L Biddulph

Eventually all injurious agents will result in inflammation. However, immediately after the accident, the mechanical effect of the injury may cause major damage, ie, haematomata and high-pressure injection injuries. During the ensuing oedema, protein molecules may be irritative causing inflammation. These molecules may also be too large to be drained by the lymphatic system. Their organization leads to further loss of pliability of all soft tissues.

The importance of prevention is self-evident.

Mobilization plays a major role in prevention and should be started immediately unless contraindicated. Splintage is usually in the position of function and should also incorporate an element of compression to discourage oedema formation. Specific injuries, ie, tendons, may command immobilization in non-functional positions. All splints and dressings should be discarded as soon as possible. In the interim, elevation must be maintained at all times.

With inept or un-cooperative patients, it may be safer to place the hand in a collar and cuff to ensure elevation, particularly during the immediate post-trauma period.

### **Wound Debridement**

Almost all tissues in the hand are vital and these cannot be resected until healthy tissues are left, as with wounds elsewhere. Fortunately, a thorough mechanical cleansing has proven to be equally effective.

## **Skin Cover**

A simple split skin graft will take on most tissue except bared tendon, bone and joint.

# **Tourniquet Time**

I believe that anoxia causes tissue damage from the ouset and that the term "tourniquet time" is a fallacy. Fifteen minutes is better than thirty minutes and thirty minutes is better than sixty minutes. The best tourniquet time is the absolute minimum and should not be squandered under the false impression that 120 minutes is "safe".

### **Contaminated Wounds**

In wounds with a high risk of infection, a further anaesthetic and secondary suture can be avoided by using loose suturing and irrigation of the wound with or without an antibiotic fluid.

### **Extensor Tendon Injuries**

These injuries are often managed with a disturbing degree of disconcern. The poor results of mallet and boutonnière deformities are vivid evidence of this.

#### **Flexor Tendon Injuries**

All structures should be repaired. Desperate attempts toclose the sheath may have disastrous effects on the underlying tendons. Vein grafts further complicate the injury with no advantage. Where there has been loss of sheath tissue, it is better to leave the sheath open. I believe that four weeks of immobilization is quite sufficient for all flexor tendon repairs.

### **Suture Material**

A coated and braided material is preferred. Non-coated material is too rough and may cause unacceptable damage.

#### Nerves

The nerve ends should be gently approximated. Too much tension will cause doubling up of the fascicles and too little tension will result in a gap and haematoma formation.

Secondary procedures such as intrinsic muscle replacements should be delayed until it becomes evident what type of recovery has been achieved, particularly in the young patient. There are many patients with near full intrinsic recovery.

# Joints

The use of invasive techniques to ensure immobilization in the correct position is unjustified.

### Fractures

Many techniques for internal fixation exist. The most appropriate and simplest technique is usually the best. K-wires and tension bands require minimal stripping of soft tissue while a plate and screws are difficult to perform and require extensive soft-tissue stripping.

## **Treatment of Burns**

After the initial debridement the hand is covered with an ointment like Flamazine, placed in a plastic bag and exercised immediately. Allowing the joints to go through a full range of movement not only prevents stiffness but also stimulates circulation. Immediately after a split skin graft a period of one week is allowed in the position of function for the graft to take.

### **Chapter 7.3: Spinal Injuries**

## J A Louw

#### Introduction

The first recorded examination and treatment of injuries of the spinal column was in 2800 BC by Imhotep, the Chief Minister of Zoser, the first king of the Third Dynasty. In the Edwin Smith *Surgical Papyrus*, written during approximately the same period, the unknown author also describes the symptoms of spinal cord injuries during that period led him to classify this as "an ailment not to be treated".

During the First World War the mortality rate of battle casualties with spinal cord injuries was still 80% within the first two weeks following injury. Sir Ludwig Guttman initiatated his pioneering work on the management of spinal cord injured patients in 1943. This gave a new meaning to the life of these unfortunate individuals. This newly found art in medicine flourished to such an extent that the mortality rate decreased to as little as 8% within the first year. This decrease in the mortality rate is a direct consequence of the early recognition and treatment of the complications of spinal injuries.

This chapter deals with the principles in the management of spine injuries. It is by no means an attempt to a detailed discussion of this subject, because it then fails its primary goal which is instruction to surgical disciplines outside orthopaedic surgery. The first part will dealwith specific injuries, the second part with complications, and the third part with the management of spinal injuries.

# **Evaluation of the Spinal Injured Patient**

The management and rehabilitation of the spinal injured patient starts at the accident scene. The patient should be lifted and transferred from the accident scene to the ambulance stretcher with the greatest care to avoid further damage to the spinal cord. The key to an atraumatic transfer is to keep the spine aligned at all times, and to avoid any rotation or angulation of the vertebral column. In the emergency unit, the patient should immediately be transferred to a trolley with tilting facilities and with radiolucent boards to allow radiographic examination without lifting or turning the patient.

A meticulous history should be obtained. In addition to routine questioning, attention should be directed at the following:

- First of all, the type of injury. High velocity injuries (ie, high speed motor vehicle accidents, fall from great heights, etc) carry a high incidence of single or double level spinal injuries. Other obvious life-threatening injuries may obscure the lesser ones, with the result that the spinal injury may pass unnoticed.

- Secondly, the patient should localize the injury as accurately as possible.

- Thirdly, any neurological lesion should be identified, and whether such a lesion occurred immediately with the injury, or whether it developed insidiously following the injury. The latter situtation signifies an incomplete neurological lesion with a better prognosis and where a more aggressive surgical approach is justified.

The physical examination of the patient should be conducted in the supine position. A meticulous general examination is essential. When examining the spine, the patient should not be turned unless an accurate radiological diagnosis has been established. Abrasions, lacerations and ecchymosis convey important information on the direction and magnitude of the force that caused the injury. With the patient supine, the examiner's hand is placed carefully underneath the patient's spine, and the spinous processes, interspinous spaces and paravertebral areas are palpated.

An enlarged gap between the spinous processes, or a lateral or antero-posterior step in the alignment, signifies instability. Pressure tenderness is accurately localized. The cervical spine is also palpated from anteriorly and laterally. Gentle palpation between the trachea and carotid sheath anteriorly, and on the lateral masses of the cervical vertebrae laterally, add valuable information. Anterior palpation of the lumbar spine is both possible and informative, and is done by deep palpation through the abdomen.

Range of spinal movement is never carried out unless a specific diagnosis is established and spinal stability confirmed.

Certain injuries are often associated with spinal injuries. Head and facial injuries are associated with injuries of the cervical spine. Sternum and rib fractures are associated with thoracic spine fractures. Any blunt trauma to the thoracic cage should raise the suspicion of a thoracic spine fracture. Patients with an injury at one spinal level have a 15% change of injury at another level. Pulmonary contusion may follow severe blung chest trauma, and further decrease the already compromised respiratory reserve. Traumatic chylothorax may occur after injury to the thoracic duct, which has a close relationship to the mid-thoracic vertebrae. In patients with penetrating injuries to the spinal cord (ie, stab or bullet wounds), the oesophagus may be injured directly. A severe fracture-dislocation of the cervical and thoracic vertebrae may also indirectly injure the oesophagus, which may even rupture. Oesophageal injury may lead to early unexplained fatality in patients with spinal cord injury. Endoscopy and barium swallow are therefore essential procedures in the high-risk patient.

Radiological evaluation of the spine is imperative in the patient with a suspected spinal injury. This should be done with the patient in the supine position, and without turning or lifting the patient. High quality antero-posterior and lateral radiographs should be obtained from the skull to the sacrum. Chest and pelvic radiographs form part of the routine to rule out rib and pelvic fractures.

### **Injuries to the Cervical Spine**

# **Soft-Tissue Injury**

The danger of these injuries lies in the lulling effect of normal radiographs obtained at the initial examination, where muscle spasm and pain prevent any displacement. However, when muscle spasm and pain have subsided after three to seven days, catastrophic displacement of vertebrae may occur. Therefore, *all* patients with a suspected cervical spine injury must have flexion and extension radiographs after all pain and muscle spasm have subsided.

#### Fractures of the Atlas (C1)

- Anterior or posterior arch fractures

The transverse ligament and lateral masses of C1 are undisturbed. Stability is therefore not jeopardized. The management is by halter traction until painfree and free of muscle spasm, followed by flexion and extension radiographs to confirm stability. Thereafter, a soft collar will suffice.

#### - Jefferson fracture

With an axial force on the head, the occipital condyles are driven downwards. The facet joints of the occipital condyles and axis (C2) are directed laterally, with the result that an axial force tends to displace the lateral masses of C1 outwards. Fractures of the ring of C1 occur both anteriorly and posteriorly to each of the lateral masses. When lateral displacement of the lateral masses of C1 is more than 7 mm, the transverse ligament ruptures with additional atlanto-axial instability. When lateral displacement is less than 7 mm, fracture is

usually stable, and SOMI brace is applied. With more than 7 mm displacement, the atlantoaxial instability necessitates more rigid immobilization in the form of a halo-thoracic brace.

# **Rupture of the Transverse Ligament**

The transverse ligament of the atlas (C1) extends betweent the lateral masses of C1. It is the only primary stabilizer of the atlanto-axial complex. The other ligaments in this complex are unable to withstand translational forces when the transverse ligament has ruptured. This injury is caused by a postero-anterior sheering force. The diagnosis is confirmed on a lateral radiograph of the cervical spine. If the distance between the anterior arch of C1 and the odontoid process exceeds 3 mm, rupture of the transverse ligament has occurred in the trauma situation. Management is by a posterior atlanto-axial fusion with wiring of the arch of C1 to the spinous process of C2.

### Fracture of the Odontoid Process of C2

This fracture is caused in 80% of the cases by a forward flexion or flexion-rotation injury to the upper cervical spine. Extension and lateral flexion injuries account for the rest. The most common associated injury is fractures of the mandible. Furthermore, 40% of the patients with odontoid fractures have associated face and head injuries. An open mouth antero-posterior radiograph of the C1-2 complex forms the cornerstone in the diagnosis. Undisplaced fractures are best managed by a halo-thoracic brace; displaced fractures by alignment in traction and posterior C1-2 fusion.

## Traumatic Spondylolisthesis of the Axis (Hangman's fracture)

This fracture is caused by hyperextension and compression of the upper cervical spine. Fractures occur bilaterally through the neural arch of C2 in such a way that the vertebral body is separate from the facet joints and laminae. If the intervertebral disc between C2 and C3 is uninvolved, the spine is stable and a cervical collar suffices. However, when the C2-3 disc is damaged the lesion is unstable and should be treated with a halo-thoracic orthosis. The C2-3 instability is characterized by anterior displacement of the C2 vertebral body on C3 for more than 3 mm, and abnormal widening of the disc on a plain lateral radiograph. Careful flexion and extension of the cervical spine under image intensifier control serves to confirm instability.

# C3 to T1 Levels

Injuries to these levels are best discussed under the hradings of the mechanisms that cause the injury. The forces that act on the spine are either distraction or compression; flexion or extensions; and lateral flexion to left or right. These forces usually act as coupled movements, ie, flexion coupled with either distraction or compression. Axial compression and lateral flexion forces may act alone on the cervical spine.

The principles in the management of all these injuries may be tabulated as follows:

1. Alignment of the spine (reduction of fracture or dislocation) with:

a. Traction - skull or halter

b. Braces - halo-thoracic

c. Surgery - open reduction and fusion (with or without instrumentation)

2. Maintain alignment to allow healing with the modalities metnioned in 1a, b and c.

3. Incomplete neurological lesions:

a. Align spine/reduce fracture

b. CT scan, magnetic resonance imaging or myelogram to define pathological anatomy

c. Surgical decompression of the spinal cord if 3(a) did not succeed.

4. Rehabilitation

a. Of the spinal cord injury (see later)

b. Of the vertebral column injury

- Regain muscle tone and strength
  - Mobilize spine to regain full mobility
  - Correct posture.

### **Compression-Flexion Injuries**

With the cervical spine in a flexed position, an axial compression force is added. A fracture line develops from the superior corner of the vertebral body to the inferior endplate. If the force continues, the involved vertebral body dislocates posteriorly, leaving the anteroinferior fragment attached to the caudal vertebral body. In undisplaced fractures, skeletal traction is applied until the patient is free of pain and muscle spasm. Careful flexion and extension of the cervical spine is monitored under image intensifier control. If the injury is stable, the patient is immobilized in a SOMI brace until radiological consolidation of the fracture. Displaced fractures are aligned with skull traction in extention, after which a myelogram or CT scan is performed in incomplete neurological lesions to prove adequate decompression of the spinal cord. If this is not accomplished, anterior surgical decompression of the spinal cord and fusion of the vertebrae are performed.

# **Distraction Flexion**

The interspinous ligament complex is first to fail; then rupture of the capsules of the facet joints (cause bilateral facet joint subluxation); followed by dislocation of one or both facet joints. If the force continues, the vertebral body moves anteriorly until it dislocates completely. A bilateral facet joint subluxation is reduced by traction in extension. Reduction can be maintained by a halo-thoracic orthosis, or by interspinous wiring and fusion. A unilateral or bilateral facet joint dislocation may be reduced by skull traction, and the reduction maintained in a halo-thoracic orthosis. Both surgical and non-surgical management are accepted methods of treatment.

### **Axial Compression**

Axial compression alone produces a burst fracture. Initially a fracture of one or both vertebral endplates (the superior and inferior cortex of the vertebral body in direct contact

with the intervertebral discs) occurs, and eventually a fracture of the centre of the vertebral body. This is an explosive injury, with fragments propelled sideways in all directions.

This injury is best managed by skull traction in extension. The amount of weight necessary for reduction of the fracture is maintained while CT or myelographic evaluation is conducted to prove patency of the spinal canal in patients with an incomplete neurological lesion. If this is not achieved, anterior surgical decompression and fusion are indicated.

### **Extension Distraction**

The first structures to fail are the anterior longitudinal ligament and the anterior part of the annulus fibrosus of the intervertebral disc. The force continues and ruptures the intervertebral disc from anterior to posterior. With a severe injury, the posterior longitudinal ligament and posterior part of the annulus fibrosus may also rupture. This injury is often overlooked on plain radiographs, but can be clearly demonstrated on flexion and extension radiographs taken after all pain and muscle spasm have subsided. Immobilization in flexion with traction or a halo-thoracic orthosis for six weeks is followed by immobilization in a SOMI until stable.

#### **Extension Compression**

If extension and compression proceed from the neutral position, the facet joints lock. After this, no further physiological movement is possible, and further extension-compression produces a fracture line through the lamina or pedicles. This may be a stable injury, when a SOMI brace allows enough immobilization for the fracture to consolidate. If instability is present, a halo-thoracic brace should be applied.

## **Lateral Flexion**

There is a distraction force on the lateral masses of one side, and compression of the contralateral side. These lateral flexion injuries cause avulsion fractures of the lateral masses, the uncus (uncovertebral joint), and widening of the intervertebral disc on the ipsilateral side, while compression of the vertebral body and facet joints are produced on the contralateral side. Unstable lesions (diagnosis on flexion and extension radiographs) are managed in a halo-thoracic brace, and stable lesions in a SOMI brace.

### Fractures of the Thoracic and Lumbar Vertebrae

The thoracic and lumbar spine are divided into three columns. The anterior column consists of the anterior longitudinal ligament, the anterior half of the annulus fibrosus and intervertebral disc, and the anterior half of the vertebral body. The middle column is made up of the posterior longitudinal ligament, posterior half of the annulus fibrosus and intervertebral disc, and the posterior half of the vertebral body. The posterior column consists of the neural arch and its ligaments. This three-column concept is used to discuss thoraco-lumbar fracture dislocations.

Four types of fracture patterns are identified, namely:

- compression
- burst
- flexion distraction
- fracture dislocation or dislocation.

# **Compression Fracture**

The anterior column fails in compression, while the middle and posterior columns remain intact. This fracture does not jeopardize spinal stability, therefore the patient is kept in bed until painfree, when flexion and extension radiographs are performed to confirm stability. The patient is then rehabilitated and mobilized without any spinal support.

### **Burst Fracture**

Both the anterior and middle columns fail in compression. The middle column displaces posteriorly into the spinal canal and may cause neurological deficit.

A CT scan is a valuable diagnostic aid to quantify the displacement of fragments accurately. The management of thoraco-lumbar burst fractures is one of the most controversial subjects in medicine today. A well accepted policy would be to manage the following patients non-surgically:

- neurologically intact
- angle of kyphosis less than 15°
- angle of scoliosis less than  $15^{\circ}$
- displacemnent in any direction of less than 5 mm.

Bed rest for four to six weeks, followed by spinal brace immobilization should yield a satisfactory outcome. With an angle of either scoliosis or kyphosis of more than 15°, the incidence of increased deformity rises sharply. These patients are best treated by operative reduction, spinal instrumentation and a posterior spinal fusion. Furthermore, an incomplete neurological lesion justifies surgical decompression of the spinal cord to allow the best prospects of neurological recovery.

#### **Flexion Distraction**

The posterior and middle columns fail in distraction, while the anterior column either remains uninvolved or displays a slight compression element. Posterior spinal instrumentation with a compression of the posterior column reinforce the deficit, and a posterior spinal fusion is performed.

### **Fracture Dislocation**

With dislocations or fracture dislocations, all three columns fail and are malaligned. This may be the result of flexion rotation, axial compression, sheering force, flexion distraction, or any other mechanism that operates on the spine. The spine should be aligned, and proper alignment maintained until healing has occurred. This can be accomplished by non-surgical measures in minor translations (postural reduction, bed rest, followed by spinal braces), but is best managed by surgical alignment, spinal instrumentation and fusion in more severe cases. In cases with an incomplete neurological lesion, adequate spinal decompression is advised.

# **Sacrum Fractures**

Sacrum fractures are easily overlooked on plain radiographs of the spine. A high index of suspicion should be maintained in suicidal jumps and high velocity motore-vehicle accidents. Almost all these cases have important perineal neurological deficit. Fracture of the transverse process of L5 is an important tell-tale. The diagnosis is confirmed by high quality sacrum radiographs. The fracture is treated by reduction and decompression of the neurological tissue. This can be accomplished by skeletal traction through the distal femora, or by surgical intervention.

# **Complications of Spinal Injuries**

Complications of spinal injuries are related to the vertebral column and the spinal cord.

# **Vertebral Column Complications**

The most frequent complications are malunion, nonunion, instability and pain.

Malunion usually occurs when the traumatic spinal deformity was not properly reduced. A common cause is to wait too long before reduction of the deformity is attempted. The vertebral column is a higly vascular structure. Early callus may prevent reduction as early as two weeks after injury.

Nonunion (or pseudoarthrosis) follows inadequate immobilization, or immobilization for too short a period. A bony nonunion of the vertebral column does not necessarily produce symptoms. An adequate fibrous union may produce spinal stability. It may, however, also produce pain and instability, which should be managed surgically by a spinal fusion.

Spinal instability may follow as a consequence of spinal injury. A stable spine is defined as one that can withstand physiological forces without displacement of any segment or progressive deformity, thus functioning to hold the body erect and protect the spinal contents from further injury. Instability is therefore marked by abnormal movement or displacement of any given segment, progressive deformity (kyphosis or scoliosis), or further neurological injury.

Pain is another complication of spinal injury. This may be caused by injury to the spinal cord or nerve roots (which will be dealt with later), or injury to the vertebral column. Instability may cause pain as a result of undue tension on ligaments, capsules and other surrounding soft tissue. Likewise, a non-union may cause pain, with accurate localization at the area of pathology.

The cornerstone of successful management of spinal injuries is to provide the patient with a stable, pain free spine. This is accomplished by prompt and successful reduction and alignment of fractures and dislocations, and adequate immobilization for a sufficient period to allow healing.

# **Complications Related to Spinal Cord Injury**

A complete lesion of the spinal cord at a given level divides the body into two parts: a cephalad part with normal neurological function, and a caudal part without any stimulating or inhibitory impulses from higher centres, and consequently with disturbed physiology. This causes numerous complications in almost all the systems. The most important complications are listed and briefly discussed.

## **Respiratory Complications**

In the tetraplegic patients, respiratory complications are the most common cause of death in the immediate and early post-injury period. If the injury to the spinal cord occurred above the fourth cervical segment, all the primary respiratory muscles are paralysed, including the diaphragm (innervation from C2, 3 and 4 nerve roots). These patients either die before they reach the hospital, or they receive immediate resuscitation at the accident scene and during transportation, and are kept alive by permanent mechanical ventilation.

With a spinal cord injury between the fourth cervical and first thoracic segments, total paralysis of the intercostal muscles occurs. The action of the intercostal muscles is responsible for 63% of the tidal volume during respiration in the normal individual, while the diaphragm contributes the remaining 37%. In the tetraplegic patient, however, the diaphragm is the only major functioning respiratory muscle. The effort required to push down the abdominal viscera in the tetraplegic patient has been calculated as being nine times greater than in normal subjects. This causes a marked decrease in the vital capacity from the normal 4.5 litres in a male to as low as 0.35 litres in acute tetraplegics. Paralytic ileus and gastric dilation occur very frequently in acute tetraplegia. If left untreated, the free movement of the diaphragm is further hampered with a detrimental effect on respiratory function.

The following specific respiratory complications are identified:

- Pulmonary collapse and infection

Tetraplegics cannot cough effectively, due to paralysis of respiratory muscles. This causes sputum retention and inspissation which leads to the formation of mucus plugs, lobar and pulmonary collapse and infection. Furthermore, secretions drain to the most dependent parts of the lung and cannot be expelled due to extensive respiratory paralysis, with hypostatic pneumonia as the result. Due to shallow breathing, alveolar collapse may occur which may spread to form atelectases in parts of the lung. Paralytic ileus which had not been adequately decompressed by a nasogastric tube may lead to aspiration of vomitus and aspiration pneumonia.

### - Pulmonary embolism

The incidence of dep-vein thrombosis in the spinal cord injured patient depends to a great extent on the management protocol. In a series where admission of patients to a spinal unit was delayed for an average of 19 days after injury, 60% of patients developed deep-vein thrombosis, compared to an incidence of 7% in patients transferred to a spinal unit within two days of injury.

Pulmonary embolism as a complication of venous thrombosis has been reported in 7-20% of spinal cord injured cases, and is the cause of death in 3% of these cases during the first three months after injury.

## - Pulmonary oedema

Most commonly this occurs as a result of overhydration of the spinal cords injured patient with haemodynamic instability. Rarely, however, it may occur as a direct complication of acute spinal cord injury.

#### **Gastro-Intestinal Complications**

The gastrointestinal tract has inherent automaticity. However, when external nervous input is withdrawn, profound and often pathological changes result. Injury to the spinal cord releases the external anal sphincter from central control, and disrupts autonomic innervation of gastrointestinal tract.

The parasympathetic innervation of the gut is supplied by vagus (serving the stomach, duodenum, small bowel and proximal two thirds of the colon); and by the pelvic nerves (innervating the descending colon and rectosigmoid).

Sympathetic innervation of the gastrointestinal tract is through the iliac, superior mesenteric and inferior mesenteric ganglion which receive preganglionic neurons from the thoraco-lumbar sympathetic outflow (T4 to L2). Therefore spinal cord injuries above the fourth thoracic segment will interrupt sympathetic input to the gastrointestinal tract, and leave the parasympathetic activity (via vagus) uninhibited. This enhances glandular secretion (ie, pancreatic and gastric) and relaxes junctional sphincters such as cardiac, pylorus, and ileocecal valve.

The following are specific gastrointestinal complications:

#### **Paralytic Ileus**

This complication is the result of spinal shock, depression of all reflexes (including peristalsis) and bilateral flaccid paralysis below the level of the lesion.

Spinal shock will affect the gastro-intestinal tract when cord transection occurs at or above the level of visceral innervation (T4 to L3). Therefore ileus most commonly accompanies upper thoracic and cervical cord injuries, and in cases with complete neurological deficit.

# **Peptic Ulcer**

This occurs in up to 22% of spinal cord injured patients. The routine use of antacids and cimethidine may lower this incidence to 2.5%. Although by no means certain, the following is suggested as possible etiological factors: Spinal cord injury loses the moderating sympathetic input which leaves vagal (parasympathetic) action unopposed. This results in increased gastrin and hydrochloric acid production as well as gastric mucosal vasodilation. Corticosteroids endogenously released (part of posttraumatic reaction and emotional stress) and therapeutically administered to reduce spinal cord oedema, further predisposes to ulcer formation.

The ulcers are usually small, superficial and predominantly gastric in location. Endoscopy is considered the most accurate diagnostic procedure because of the small size of the ulcers.

#### Pancreatitis

This complication may be explained by the overstimulation of the sphincter of Oddi by uninhibited parasympathetic activity; direct activation of trypsinogen within the pancreas by the hypercalcaemia of immobilization; and increased viscosity of pancreatic secretions due to endogenously released or therapeutically administered steroids.

Pancreatitis may be difficult to diagnose in the spinal cord injured patients as a result of the change in the appreciation of epigastric pain and the nonspecific signs of pancreatitis (ileus, distension, nausea, vomiting, etc) which could well be explained as direct complications of the injured spinal cord. Body computed tomography, sonographic evaluation, and serum and urine amylase analysis assist in making the diagnosis.

### **Faecal Impaction**

This is the most common gastrointestinal complication in patients with spinal cord injury. There are two types of faecal impaction. First, in patients with spinal cord lesions from the cervical segments to the S1 segment in the conus medullaris. In these patients the vagus nerve gives parasympathetic input up to the transverse colon while the pelvic nerves (S2, 3 and 4) fail to give any input to the descending colon and rectosigmoid. Therefore a proximal faecal impaction at the level of the transverse colon is found in these cases. The second type of faecal impaction is found in patients with lesions of the S2, 3 and 4 segments in the conus medullaris, or with cauda equina lesions interrupting the supply of these segments. Faecal impaction follows due to loss of the defaecation reflex.

### **Superior Mesenteric Artery Syndrome**

This entity is related to the small vascular angle created between the superior mesenteric artery and the aorta. The mid-transverse duodenum passes between the two limbs of the angle thus created. Thoraco-lumbar hyperlordosis, produced by a body spica or positioning of the patient for reduction of fractures, may acutely reduce this vascular angle and result in duodenal obstruction. Transient duodenal obstruction may also occur during the chronic stages of spinal cord injury as a consequence of decreased periduodenal areolar tissue, the result of severe mass loss and wasting.

#### Amyloidosis

Chronic infections occur frequently in patients with spinal cord injury. Of these, recurrent decubitus ulcers, chronic osteomyelitis and chronic pyelonephritis are the most common. The chronic reticuloendothelial stimulation produced by these inflammatory processes leads to amyloid deposition in all portions of the alimentary tract. This may produce malabsorption or diarrhoea.

### **Premature Gastro-Intestinal Senescence**

Hiatus hernia, gastro-oesophageal reflux and diverticulosis are frequent complications in middle-aged spinal cord injured patients (third and fourth decades) with disability of longer than five years' duration. It is suggested that chronic spinal cord injury accelerates the ageing process of the gastrointestinal tract, because these conditions are usually seen in older patients without spinal cord injury.

Hiatus hernia and reflux are possibly caused by limitations of motion of the diaphragm, atrophy of the muscles, chronically raised intra-abdominal pressure and the sphincterolytic effect of unopposed vagal action. Chronic faecal sludging with raised intraluminal pressure proximally is a suggested reason for the precocious appearance of diverticula.

# **Complications of the Urinary System**

During the phase of spinal shock after an acute spinal cord injury, there is total paralysis of the bladder. As a result, all volitional and reflex functions of the bladder are abolished. There is no desire to urinate and the bladder no longer responds to an appropriate degree of distention by efficient reflex contraction. There is flaccid paralysis of the detrusor muscle as well as the striated bladder muscles. The bladder wall, however, is not completely atonic, thanks to its autonomous intramural innervation and its inherent elasticity. The total bladder paralysis leads to retention of urine. Voiding through overflow will take place only when the maximum expansion of the bladder wall is reached and the passive blockage of the bladder exit and paralysed external urethral sphincter are overcome by excessive intravesicular pressure. After the phase of spinal shock, any one of the different types of neurogenic bladders may develop. The following urological complications may occur in the spinal cord injured patient:

### Damage to the Bladder Wall

If overdistension of the bladder is allowed to continue in the spinal shock phase, irreversible damage to the detrusor muscle may occur, resulting in adverse effects on bladder function once the period of spinal shock has subsided.

## Hydro-Ureter and Hydronephrosis

Chronic overdistension of the bladder may lead to dilatation of the ureters and kidney pelvices, resulting in hydro-ureter and hydro-nephrosis. Dilation of the upper urinary tract may also occur in cases with a spastic external sphincter. During bladder contraction, the spastic external sphincter prevents urine from flowing into the urethra. Instead, the urine is forced retrograde into the ureters and kidney pelvices, with hydro-ureter and hydro-nephrosis as the long-term complications.

# **Urinary Tract Infection**

Incomplete emptying of the bladder and unsterile manipulation of the lower urinary tract count as the most important aetiological factors of urinary tract infection in spinal cord injured patients. Chronic pyelonephritis is an important cause of death in these patients.

# **Urinary Calculi**

Urinary status and infection create an ideal situation for the formation of some types of calculi. Massive calculi may form in the kidney, ureter or bladder, and may pose a threat to the kidneys and the patient's life expectancy.

# **Kidney Failure**

All the foregoing urogenital complications may cause kidney failure and death.

# **Epididymo-Orchitis**

This is mainly a complication of an indwelling catheter. The patient has no sensation in his scrotum, therefore epididymo-orchitis is usually discovered by the patient as a testicular swelling. Fever and general malaise may be accompanying symptoms.

# **Peri-Urethral Abscess and Urethral Diverticulum**

These are also complications of an indwelling catheter. It usually occurs in the bulbous urethra and can be palpated at the inferior surface of the penis at the penoscrotal junction.

### Complications of the Skin and Subcutaneous Tissue

Pressure sores are one of the most fearful complications of spinal cord injury. Skin damage may be irreversible after as little as two hours after injury. It usually occurs under a bony prominence, where continuous pressure renders the skin and subcutaneous tissue ischaemic. This occurs particularly over the scapulae, sacrum, ischial tuberosities, trochanters and heels. Thrombosis occurs in the microcirculation, followed by necrosis of the tissue involved. Removal of the necrotic tissue may leave the underlying bone bare. Secondary infection accelerates the tissue destruction, and may cause septicaemia and death.

### **Emotional and Psychological Disturbances**

There is an image in the mind of each person of what he or she looks like when the person could look at himself from outside. This image, laid down in the ego of mankind, is strengthened daily by central and perpipheral stimulation - the nervous system gives continuous feedback to the ego that the different parts of the body still function. Then comes the spinal cord injury, and part of the body is amputated from the image in the ego. In the high tetraplegic patient, for example, the body image in the ego consists only of a head and neck. The patient now has to accept the new body image, and reprogram his ego to a new bodily form.

Different stages of emotional disturbances are recognized. The stage of shock comes first, when the patient appears isolated, confused and numb. He fails to comprehend what has happened to him. This may last for several days. Then comes the stage of partial recognition, when he becomes more responsive to outside stimuli. He tries to understand the horror of the trauma that has occurred to him, while concomitantly defending himself against the recognition of the finality of his injury. This is followed by the stage of initial stabilization, which may last months or years. This stage lasts as long as the patient is undisturbed.

As soon as rehabilitation and adjustment to a new way of life starts and stress of reality becomes apparent, the next stage of regression is entered. He begins to realize the implications for his future. The rehabilitation team then observes a rapid return to a state of preoccupied anxiety and helplessness. He expresses his feelings through selfpity and egocentric demands. This stage of regression may last many months, and leads to a series of potential outcome pathways, the first of which is the stage of denial. The patient may insist, that he will walk again and that complete cure will follow soon. Through this denial, he becomes pleasant and comfortable once again. This tranquility vanishes when renewed pressure is put on him to participate in rehabilitation activities. He often accuses personnel of erroneous treatment, and that they prolong his illness. He may become openly hostile and paranoid. The patient should once again be assisted out of this condition. The state of denial may gradually give way to depression, where the moody and pessimistic stage of mind may lead to attempts at suicide. With help the patient enters the final stage of social recovery. The patient's major concern is not to increase his capacity for independent living.

All the members of the rehabilitation team form an integral part to manage the emotional complications of the spinal cord injured patient.

## **Musculoskeletal Complications**

#### **Osteoporosis with Spontaneous Fractures**

Physical activity is the most important stimulus for bone formation, through stimulation of the osteoblasts. The lack of normal physical activity in the spinal cord injured patient causes disuse osteoporosis, with spontaneous fractures occurring sometimes.

### **Myositis Ossificans**

Myositis ossificans is a heterotopic formation of bone, usually in the collagenous supportive tissue of the skeletal muscle, in tendons, ligaments, fascias and aponeuroses. New bone is laid down around a joint. This matures until a thin cortex surrounds normal cancellous bone. If extensive, myositis ossificans may limit or even completely block joint motion. Although the aetiology is still unknown, vigorous passive stretching of peri-articular soft tissue in the spinal cord injured patient seem to play an important role. Since the development of careful physiotherapy techniques, the incidence of myositis ossificans in the spinal cord injured patients has decreased from 40% before the Second World War to 4% in this decade.

#### Spasticity

During the spinal shock phase of acute spinal cord injury, a flaccid paralysis is present of all the muscles below the level of injury. In complete spinal cord lesions, the spinal cord distal to the level of injury is without inhibitory and controlling input from higher centres. As a result of this, spasma, a heightened reflex activity and rigidity of the muscles below the injured level occur after the phase of spinal shock has subsided. Excessive spasticity hampers nursing and rehabilitation, and may cause pressure sores. A number of factors lower the threshold of reflex activity of the damaged spinal cord, and increase spasticity. These include distention of any internal organ in the paralysed area (particularly the bladder and rectum), infection, anaemia, and irritation of sensory organs in contracted tendons and joints. In the treatment of spasticity, the causative agent should be removed. Passive movements and exercises have a depressant effect on spasticity. Drugs of great value in control of spasms are diazepam (Valium) and baclofen (Lioresal). In the resistant cases, injection of alcohol in the subarachnoid space converts the spastic upper neurone lesion. Ventral root neurectomy and selectvie rhizotomy are surgical alternatives.

### Contractures

Spinal cord injured patients should be positioned correctly to prevent contractures. The correct position of the joints is maintained by splints and the correct positioning of the patient after each turning procedure. All the patient's joints are daily mobilized through the full range of movement, either by active or passive motion. Failure to adhere strictly to the foregoing precautions may lead to contractures, which in turn cause pressure sores, and which hamper rehabilitation.

### **Haemodynamic Complications**

The critical spinal cord level for haemodynamic dysequilibrium is the fourth thoracic level, because the sympathetic innervation for intra-abdominal viscera and the lower extremities occurs below this level. A lesion above this level causes loss of vascular tone in the intra-abdominal viscera and skin of the lower extremities. This creates pooling of blood in these vessels and a decreased peripheral resistance, with a resultant fall in the blood pressure. The acute traumatic tetraplegic patient frequently presents with a systolic blood pressure of 60-80 mm Hg. This hypotension is accompanied by a bradycardia (vagus stimulation). If this "normal" hypotension of the tetraplegic patient is treated as hypovolaemic shock, overtransfusion and pulmonary oedema may follow. Important points of differentiation

are that the patient with spinal shock and hypotension has bradycardia, and that the urinary output is well maintained.

The foregoing mechanism is also responsible for orthostatic hypotension. When these patients (lesion above T4) are brought upright from the supine position, a rapid fall in blood pressure may occur.

# Autonomic Dysreflexia

This complication occurs frequently in patients with spinal cord injuries above the fourth thoracic segment. Autonomic dysreflexia is characterized by exaggerated autonomic responses to stimuli that are usually modulated by higher centres in non-paralysed individuals. Stimuli below the level of the injury (bladder or colorectal distention, cutaneous stimuli, etc) cause an uncontrolled sympathetic reaction below the injury level (no central inhibitory impulses) resulting in pilomotor erection, abnormal sweating, and vasoconstriction. This vasoconstriction causes paroxysmal hypertension, which in turn stimulates the baroreceptors in the aortic arch and carotid sinus. This results in parasympathetic reaction above the level of injury (an attempt to control the hypertension) with bradycardia and vasodilatation of the cerebral arterioles (headache); nasal arterioles (nasal congestion); and skin arterioles (flushing above injury level). The paroxysmal hypertension may be severe enough to cause blindness, convulsions, aphasia, coma, retinal and subarachnoid haemorrhage, and even fatal cerebral haemorrhage.

The treatment and prophylaxis of autonomic dysreflexia are aimed at decreasing the stimuli below the level of injury. In high-risk patients, phenoxybensamine (an adrenergic antagonist) can be used to block the reflex vasoconstriction, and therefore minimize the paroxysmal hypertension.

#### Pain

True anaesthesia is virtually non-existent in complete spinal cord injuries. Most of these patients experience at some time during their disease unpleasant (phantom) sensations, or pain. Fortunately, only 5-10% will have chronic pain severe enough to give attention to. The following types of pain are differentiated:

- Acute or chronic musculoskeletal pain related to the vertebral column injury. Early adequate immobilization of the vertebral column and a solid bony fusion of the injured level will prevent this form of pain.

- Nerve root pain which follows the segmental dermatone innervation. Proper alignment of the vertebral column and adequate decompression of the neural structure will help to prevent this.

- Pain from spinal cord origin. This gives rise to the most common pain syndrome, ie, a poorly localized, continuous burning discomfort. The intensity varies from an acute burning sensation to a chronic dull ache. The modification of the central response to pain forms the foundation of the management. The patient must be encouraged to partake in recreational and sporting activities. Medication in the form of a combination of phenothiazine

and tricyclics may help to change the central response. Epidural and deep brain stimulation has been used with success.

# Post-Traumatic Syringomyelia (Ascending Post-Traumatic Hydromyelic Syndrome)

From four months to several years after a complete or incomplete spinal cord lesion, cystic cavities may appear within the spinal cord in 2% of patients. These cavities mainly extend in a cephalad direction above the level of the original injury, but may also progress caudally. Pain is usually the presenting symptom. It is intensified by sneezing, coughing or straining. Eventually the pain disappears and shifts to a more cephalad location. The original painful area then displays ascending sensory loss, which becomes permanent. Eventually motor changes follow in the same dermatomal distribution. Although less common, there may be signs of damage to the autonomic nervous system. The diagnosis is confirmed by watersoluble myelography, with a contrast CT scan done two to four hours thereafter. Surgery is the only means of achieving cure or at least halting the progression. The objective is to promote drainage of the intramedullary cyst into the subarachnoid space.

# **Treatment of Acute Spinal Cord Injuries**

The treatment of acute spinal cord injuries involves mainly the prevention of complications, the alignment of the vertebral column, and the decompression of the spinal cord in incomplete neurological lesions.

## **Preventing Complications**

The basic principles of resuscitation and clinical examination are respected. An intravenous line is installed through which maintenance fluid is given. Overhydration is a serious complication. If the blood pressure remains low but is associated with bradycardia and an adequate urinary output, only maintenance fluid is given. If tachycardia and oliguria are present, the patient is hypovolaemic and appropriate measures should be taken.

Urinary output must be monitored from the beginning. Intermittent catheterization twohourly, done by aseptic non-touch technique, is continued until the patient is haemodynamically stable. Thereafter four-hourly intermittent catheterization is done. Weekly urine MCS is mandatory to diagnose and treat urinary tract infection early and effectively.

Paralytic ileus and acute gastric dilatation are frequent early complications. If left untreated, they cause decreased movement of the diaphragm, and consequently hypoventilation and vomiting with aspiration pneumonia. This complication is treated by immediate nasogastric tube insertion to decompress the abdominal content. Anti-acid therapy is routinely administered to combat acute stress ulceration. As soon as intestinal sounds are present, the nasogastric tube is removed and clear fluids given orally. This proceeds to soft and later ward diet. The patient is usually constipated as a result of the sympathetic paralysis and the antiacid therapy. Therefore, routine fleet enema and purgatives are given to start bowl rehabilitation. A high index of suspicion should be maintained for the other gastrointestinal complications, as listed previously. Tetraplegic patients are highly susceptible to respiratory complication. An immediate baseline astrup should be done. A chest radiograph rules out associated rib fractures. Lung physiotherapy is instituted as soon as possible. Respiratory failure is managed appropriately.

Pressure sores may develop as soon as two to four hours after injury. An immediate two-hourly turning programme is instituted. During turning, pressure points are massaged. Thighs and calves are also massaged, and the calves are pumped to prevent deep vein thrombosis. The circumference of the thighs and calves are monitored two-hourly to diagnose deep vein thrombosis early.

Contractures are prevented by correct positioning of the patient, splints, and passive and active movements of the joints. The correct positions for the upper extremities are:

- $90^{\circ}$  abduction of the shoulders
- $90^{\circ}$  flexion of the elbows
- dorsiflexion of the wrists
- 90° flexion of the metacarpo-phalangeal joints
- interphalangeal extension.

The lower extremities are positioned with the hips in extension and slight abduction, the knees in extension and the ankles in dorsiflexion. Twice daily a physiotherapist should carefully move each joint several times through the full range of movement. Wherever possible, active movement is encouraged.

# **Reduction of Dislocations and Decompression of the Spinal Cord**

This part of the treatment depends entirely on the type of injury, and was outlined previously.

## Conclusion

The foregoing basic measures form the baseline of the management of spinal cord injured patients. It should be appreciated that the treatment of these patients needs the care of specialists in various fields of medicine:

- the nursing staff need a nursing sister with spintal training
- the physiotherapist and occupational therapist play their specialized role
- the social worker faces some of the most complicated social work problems

- the clinical psychologist fulfils an invaluable role in the patient's psychological dysfunctio

- the urologist makes the greatest impact on the life expectancy of the patient
- the plastic surgeon cares for the most fearful complication of all, the pressure sore

- the spinal surgeon should be well trained in the basic biomechanical requirements and instrumentation of the vertebral column, as well as the techniques of surgical decompression of the spinal cord (from both anteriorly and posteriorly). Devoted members of this multidisciplinary team should join forces in the management of the spinal cord injured patient. This then often produces gratifying and highly satisfactory results.

## **Chapter 7.4: Osteomyelitis**

# M M Malan

#### Introduction

Appropriate antibiotic therapy has reduced the mortality of acute osteomyelitis from 20-30% to virtually zero. Despite optimal treatment permanent bone and joint destruction cannot be completely eliminated in cases with unusually virulent infections, in immuno-compromised patients and in patients presenting late.

#### **Classification and Definitions**

- Acute haematogenous osteomyelitis is a bacterial infection of bone occurring most commonly in children.

- Post-traumatic osteomyelitis may follow the exposure of traumatised bone to an infected environment.

- Chronic osteomyelitis may follow any form of osteomyelitis and is characterized by the presence of necrotic bone (sequestrum).

## **Surgical Anatomy and Physiology**

The blood supply of long bones comes from vessels passing through foramina in the epiphysis, metaphysis and the nutrient foramen in the diaphysis. They connect with the vessels in the medullary spaces which supplies cortical and medullary bone, either directly or by the haversian system which runs longitudinally in cortical bone. The growth plate, when present, acts as an avascular barrier between the epiphyseal and metaphyseal circulation. Before 12 months however the growth plate may be corssed by a few vascular canals. This may explain the spread of metaphyseal osteomyelitis towards the epiphysis which only occurs in this age group.

The growth plate depends for its nutritional needs on the epiphyseal circulation and may sustain permanent damage after epiphyseal osteomyelitis. This results in growth disturbance. Permanent destruction of the epiphysis can also occur, especially in the hip joint because it is intra-capsular.

Periosteum is firmly attached to the epiphysis and the growth plate, but more loosely attached to the metaphysis and diaphysis, where it can be easily displaced from bone by any space occupying mass such as pus, tumour or blood. Whenever this happens new bone, dependent on periosteal circulation, is rapidly laid down between the periosteum and the bone. The periosteum contributes little to the blood supply of bone but is more important in venous drainage of bone. The firm attachment of the periosteum to the growth plate and the avascularity of the growth plate usually prevents pus spreading from metaphysis to joint space and epiphysis. The proximal femur and radius and the metaphyses are intra-capsular and pus escaping through the metaphyseal foramina is separated from the joint space by synovium only. Septic arthritis, epiphyseal osteomyelitis and growthplate damage are frequent sequelae of proximal femoral osteomyelitis.

# Etiology

Non traumatic osteomyelitis occurs after inoculation of the bone during a phase of bacteraemia. The tendency for bacterial accumulation and multiplication in bone is controlled by local tissue factors and the general resistance of a patient on the one hand, and the dosage and virulence of the infective organism on the other.

Staphylococcus aureus remains the most common organism causing acute osteomyelitis in all ages. Infective skin lesions, ie, scabies and insect bites are common causes for the primary bacteraemia.

The ability of S. aureus to adhere to cartilage explains why the metaphyseal side of the growth plate is the primary site of infection in long bones. Infections in other bone regions in growing children and adult bone are rare in the absence of trauma or foreign bodies. The discontinuous endothelium of the vessels growing rapidly towards the growth plate, as well as the poorly developed reticulo-endothelial and phagocytic systems in the metaphysis adjacent to the growthplate are additional factors contributing to metaphyseal osteitis. An experimental model for studying acute staphylococcal osteomyelitis has only recently been developed. The disease could be produced by the intravenous injection of S. aureus in chickens. The strain used came from a naturally occurring osteomyelitis in another avian species.

Pyogenic osteitis caused by other organisms is rare and never as destructive as S. aureus infections. Others organisms, ie, salmonella typhi, brucella and fungi prefer other regions in bone, ie, diaphyseal and small bone osteitis in the case of salmonella, spinal osteitis in the case of tuberculosis and brucella, and unusual sites with sinus formation in the case of fungi.

Mycobacterium tuberculosis can remain dormant for long periods after inoculation in bone. Multiplication and bone destruction occurs slowly and cause few symptoms. Vertebral bodies and the epiphysis of long bones are common sites for this infection.

### Pathology

Multiplication of pyogenic micro-organisms in the metaphysis evokes a severe inflammatory response. Bone cannot expand and the accumulation of tissue fluids, which is part of the normal inflammatory response, results in spreading the infections through available pathways. The foraminae in the metaphysis, the marrow spaces and the canaliculli in the bone are the routes available. The formation of sub-periosteal and intramedullary pus collection results. Blood vessels supplying the infective are are occluded by septic thrombi. In most cases normal tissue reactions seal off the infected area. Most infective organisms, ie, S. aureus, are dependent on oxygen to continue growth, multiplication and toxin production. Onced the abscess is sealed off, the organism becomes dormant and toxin production ceases.

In virulent infections, severe septicaemia results. Distant abscess formation occurs in other organs, ie, bones, lungs, brain, myocardium etc. Death may follow.

The result of the primary abscess in the bone is the death of osteocytes as a result of a vascular occlusion. The size of this sequestrum varies from a small area of trabecular bone to the complete metaphysis and diaphyses of the involved bone.

The osteocytes in the epiphysis and the chondrocytes in the growthplate are usually not involved. The barrier, protecting the epiphysis (see section 3) may be incomplete in patients under 1 year and epiphyseal involvement with secondary growthplate destruction is common in this young patient group.

The extent of bone death (sequestrum formation) is an important prognostic factor. The virulence of the infection and the quality of the natural defence mechanisms are the determinants of the extent of bone death that will occur.

Once the abscess is sealed off, and multiplication of the infective organism ceases, reparative processes proceed.

New vessels, from the regional blood supply and periosteum, grow into the subperiosteal space and new bone formation rapidly takes place where pus had elevated the periosteum from bone. The source of the new osteocytes can either be from the endothelium of ingrowing capillaries or the cambium layer of the periosteum which survives due to the periosteal blood supply which is undisturbed. The new bone formation in the sub-periosteal space is called the involucrum.

Blood vessels also grow from the Haversian system of the dead bone into the Haversian system of the sequestrated bone. New bone is laid down on dead trabecula bone and the dead bone is slowly resorbed by osteoclastic activity. In compact bone osteoclastic resorption is followed by new bone deposition.

While this process of creeping substitution is under way, the mechanical stability of the bone is mostly dependent on the sub-periosteal new bone. Under ideal circumstances this process of resorption of dead bone and replacement by viable bone can be complete. The involucrum can then remodel to a shape virtually similar to the original bone.

Sequestrae not covered by involucrum or disturbed by surgical intervention, ie, drilling or chiseling seldom completely resorbs and usually requires surgical removal.

During the revascularization phase the dormant S. aureus may find itself again in a favourable environment for new growth. If the normal defence mechanisms cannot halt this process, a new infection may result.

### **Clinical Symptoms and Signs**

Clinical presentation and the signs and symptoms follow the stages of the developing pathological process closely. The bacteraemia phase causes no symptoms or only a slight malaise. Once the inflammatory response is evoked, severe local pain, made worse by percussion of the involved area or percussion transmitted along the axis of the involved limb, develops.

Symptoms and signs of septicaemia and toxaemia present as soon as local resistance has been overcomed. The toxicity can be very severe and patients often present dehydrated and anaemic.

In deeply positioned bones, local signs of inflammation cannot be detected early in the disease. Localization only becomes obvious after extensive subperiosteal pus collection. Rarely pus may track to subcutaneous tissue planes. Sinus formation is uncommon in cases where no surgery has been performed.

### Diagnosis

A high index of suspicion is required. The medical treatment should be commenced in every child with pain in a limb and with signs of toxicity. Bone involvement can be proved early in the disease by 99 Tc phosphate scintigraphy. This investigation is non specific and takes up to four hours to perform and should not delay initiation of therapy. Normally it is used only for special cases, ie, suspected spinal osteitis in a child with severe spinal pain in which no other cause can be found.

In the early phase of the disease plain X-rays show only soft tissue changes namely oedema and the loss of planes between tissues which normally have different X-ray density. The inflammatory oedema is responsible for the change in tissue density.

New subperiosteal bone formation and destruction of metaphyseal cortex becomes visible after ten days.

The presence of organisms and/or pus cells from an aspirate of the inflamed area can confirm the diagnosis early. The investigation is indicated where unusual organisms are suspected. In typical cases it can be accepted that S. aureus is the causative organism.

Positive blood cultures proves the associated septicaemia.

A positive response to treatment by appropriate antibiotic usually prove the diagnosis. Surgical exploration for diagnostic purposes is contraindicated, the exception being the proximal metaphysis of the femur where exploration and drainage of the hip joint, which may be inoculated, is indicated.

The differential diagnosis of acute osteitis includes primary and secondary bone tumours, histiocytosis X and infarctions of sickle cell anaemia. Some of these conditions, ie, Ewing sarcoma can also give rise to pyrexia.

In immune compromised patients and patients with overwhelming infections, ie, bacterial endocarditis, multi-focal osteitis often results. In these cases relatively minor local signs are present because the inflammatory response is subdued.

## Treatment

Appropriate antibiotic therapy is the key in the prevention of systemic complications as well as the control of the extension of the local disease. Supportive care should include rehydration, blood transfusion (when indicated), analgesics and protective splinting. S. aureus remains the most common organism and is the only organism responsible for osteitis with destruction of large areas of bone. Optimal antibiotic therapy against Staphylococcus aureus should be initiated in all cases where osteitis is suspected. Cloxacillin, in an initial dose of 100 mg/kg is given intravenously, followed by 200 mg/kg/24 h in 4 divided dosages.

Intravenous therapy is continued until the local signs of inflammation and systemic signs and symptoms of infection disappear, usually after 2-4 days. Oral cloxacillin or flucloxacillin 50-100 mg/kg is then given.

After 10 days, if little or no X-ray changes are visible antibiotic therapy is discontinued. When sequestration of bone is radiographically shown antibiotic treatment can be continued for another week althout it is unlikely to be of benefit because cloxacillin is only effective while S. aureus is actively growing.

Broad spectrum antibiotics are not sufficiently potent against Staphylococcus aureus and should not be used. The disturbance of the gram negative flora caused by them often leads to superinfection by overgrowing resistant organisms.

Other specific anti-staphylococcal drugs, ie, sodium fusidate are not recommended because better results have been obtained by cloxacillin therapy.

Methicillin resistant S. aureus (that includes Cloxacillin resistance) has not yet been diagnosed in primary pyogenic acute osteitis. Where suboptimal therapy with cloxacillin has been used, secondary culture often indicates resistance to Cloxacillin. These cases however usually respond to high doses of cloxacillin.

The role of surgery in the treatment of osteomyelitis was until recently empirical and unproved in clinical trials.

Before the availability of antibiotics, it was established that early surgery increases both the mortality of the disease and the tendency for recurrences in survivors.

The majority of cases of osteitis can be managed without any surgical intervention. Surgical intervention disturbs the periosteum and is often followed by a sinus, inadequate involucrum formation in the area where the periosteum was disturbed, and a sequestrum not covered by involucrum requiring surgical removal at a later stage.

Experimental evidence supports the clinical data that indicate the inability of surgical drainage or decompression to eradicate the infection or to limit the size of the sequestrum.

The adverse effects of early drainage were proved in experimental models. The process of resorption and revascularization is always incomplete in an area where bone has been disturbed by surgery.

Remaining indications for surgery are:

1. Failure of response after 48-72 hours of optimal antibiotic therapy.

2. A severely swollen limb with subcutaneous pus formation.

3. Cases where the diagnosis is uncertain.

When surgery is performed, the periosteum should be disturbed as little as possible and the only bony surgery should be a biopsy which is always indicated in "atypical" osteitis.

Catastrophic results are often blamed on the delay of surgical drainage. It is however due to the virulence of the infection that large parts of bone and joints are sometimes destroyed. It must still be proved that surgery can prevent these bad results.

### Prognosis

Antibiotics have decreased the mortality of acute pyogenic osteitis to virtually zero, provided that supportive treatment is adequate.

Neither antibiotics, nor surgery, or the combination of the two modalities of treatment have been able to prevent the occasional destruction of large parts of long bones. Fortunately, if the formation of an adequate sequestrum has not been prevented by badly timed or extensive drainage procedures, the final outcome is favourable. Involvement of the epiphysis, however, often leads to permanent destruction.

Chronic osteitis with sinus formation can in most cases be controlled by short courses of antibiotics during episodes of pain, or increased or renewed drainage through sinuses. Adequate provision for the increased nutritional needs is of equal importance. Sequestrae, lying free and without evidence of resorption, should be removed by surgery with the least possible disturbance of viable bone.

### Complications

Pathological fractures are serious complications and should be prevented by splinting in the phase before a sufficient involucrum has formed.

Pathiological fractures following acute osteitis should be treated as any other infective pseudoarthrosis namely by:

- Immobilization - by plaster or external fixators.

- Optimal control of infection. Flora cultured from sinuses are not necessarily the same as the more important deep infection which is almost always S. aureus.

- Removal of sequestrae once and adequate involucrum has formed.

- Bone grafting by cancellous autografts.

Growth disturbances. This should be expected after infantile osteitis. As soon as it is detected carefully planned epiphysiolysis should be performed.

Loss of bone.

The proximal femoral epiphysis is not infrequently completely destroyed after osteitis in infancy.

In patients under 4 years of age an exploration with the aim of relocating a remnant of the epiphysis in the acetabular or alternatively the positioning the greater trochanter in the acetabular may be indicated.

Above 4 years the pseudoarthrosis is left unchanged and the shortening is treated by a built up shoe.

If a severe flexion or adduction contracture of the pseudoarthrosis develops, a corrective subtrochanteric femoral osteotomy may be indicated.

Complete loss of a large segment of diaphyseal bone with inadequate involucrum formation results from inappropriate surgery done at an early stage.

In the case of the humerus or femur cancellous bone graft can be successfully done if the periosteum is reasonably intact.

In the forearm or leg the uninvolved bone can be centralized in order to provide stability.

Free vascularized bone grafts or composite tissue grafts are alternative possibilities for reconstruction.

## Comment

# Osteomyelitis

# **H** Pretorius

To miss a diagnosis of acute haematogenous osteomyelitis in a child is a catastrophe it will lead to a lifetime disability that can never be corrected.

It must be kept in mind that acute haematogenous osteomyelitis may involve more than one bone and one should keep on searching for it. This may be the reason for persistence of an elevated temperature, general feeling of malaise, etc. Acute haematogenous osteomyelitis in the typical case is usually found in:

- (a) Males; more common than females.
- (b) Usually between the ages of 2 to 8.
- (c) In about 80% of cases the bone around the knee is involved.
- (d) The possible association of trauma; and
- (e) A possible source of infection (ENT, boil, tooth abscess, etc).

In children under 2 years of age the infection is often due to Haemophilus influenzae or streptococcus.

In my experience the role of continuous intramedullary suction-irrigation, as propagated by Anderson and Horn, quite often leads to a super-imposed infection and should only be used in a hospital geared for this type of treatment or only in cases of chronic osteomyelitis.

Any child presenting with acute pain, swelling, redness and warmth over a metaphysis and loss of function of a limb, is to be regarded as a case of acute haematogenous osteomyelitis until proved otherwise.

The differential diagnosis of acute osteomyelitis is:

- An acute rheumatic fever.
- Early juvenile rheumatoid arthritis.
- Ewing's sarcoma.
- Osteogenic sarcoma.
- Cellulitis.

### **Chapter 7.5: Infective Arthritis**

### M M Malan

### Introduction

The mode of presentation of this disease varies widely.

In acute bacterial arthritis in superficial joints of children the diagnosis is usually made early. In joints well covered with soft tissues, ie, hip, shoulder and spine, and more so where the reaction to infection is subdued, ie, in neonates and elderly patients, the diseae and destructive process is often advanced before the diagnosis is made. Since cartilage has limited potential for regeneration, irreversible damage of this tissue may result. The viability of the adjacent epiphysis and growthplates are at risk in children.

*S. aureus* remains the commonest and most destructive of organisms causing infective arthritis. Other common types of infective arthritis are gonococcal arthritis in adolescent females and infections aroud artificial joints in the increasing population of persons where a degenerated joint has been replaced by an artificial joint.

Infection distant from joints, ie, bowel or throat infections, may provoke inflammatory reactions in joints. These joint reactions are probably the result of immune complexes that find their way to tissues specific to joints, ie, cartilage.

# **Definitions and Classification**

- Infective arthritis refers to a joint reaction following the abnormal multiplication of organisms in the body.

- In bacterial arthritis the micro-organisms can be demonstrated in the joint fluids or tissues. Those caused by pyogenic organisms are generally known as *septic arthritis*.

- In post-infective arthritis the organism itself can no longer be isolated from the joint. Antigens arising from the organisms may still be present. The inflammatory response triggered by the infection may persist for a few days or weeks after the organism has been eradicated.

- In reactive arthritis the nature of the causative organism and the relationship between infections by that organism and joint reactions are proved. At no time can the organism itself or antigens arising from the organism be found within the joint, joint fluid or tissues.

- In inflammatory syndromes of joints, ie, rheumatoid arthritis, there is a possibility that the condition may be associated with a concurrent or preceding infection, but the causative infective agent has not yet been identified.

### Etiology

When virulent micro-organisms or a large number of micro-organisms enter a joint, local defence mechanisms may be overcome temporarily or for longer periods. An inflammatory reaction affecting all the tissues of the joints will follow and this may persist for a considerable time after the invading organism has been eradicated.

The organisms may enter the joint:

- directly

- by way of haematogenous spread via vascular components of the joint.

Direct Inoculation results from:

- Spreading from periarticular infections.

The barriers against spreading of haematogenous osteitis towards joints have been discussed in chapter 7.4. As indicated the barriers are less effective in the very young and in certain joints of which the hip joint is the most important.

- Iatrogenic inoculation of joints during arthrocentesis.

It has been shown that minute quantities of infected blood is enough to cause septic arthritis if it is introduced into a joint. Collecting blood from the inguinal region during a phase of bacteraemia should therefore be prohibited because of the potential risk of infecting the adjacent hip joint.

If a joint has to be aspirated for diagnostic purposes and there is a possibility that the surrounding tissues are infected, intravenous administration of appropriate antibiotics ten minutes to one hour before the procedure will virtually eliminate the risk of causing a septic arthritis. The antibiotics will not interfere with the identification and culture of organism from joints that are infected.

## - Trauma.

Open injuries seldom lead to destructive arthritis if efficient wound management prevents local infection. If wound infection does occur, the joint may become infected.

Thorough debridement of wounds in the vicinity of joints and the covering of joints by viable tissues is therefore essential. Local skin flaps or in severe cases free microvascular grafts may be required.

### Haematogenous Infective Arthritis

The more severe and destructive types of arthritis result from haematogenous spread. The site of the primary infection is not always detectable. The type of organisms, the competence of the host defence mechanisms, and local joint factors determine the risk of the specific joints becoming infected during a phase of bacteraemia. *S. aureus* is the most common organism leading to haematogenous septic arthritis in all ages (the only exception is the young adolescent female where gonococcal arthritis is more common).

Certain strains of *S. aureus* have a great affinity for articular and epiphyseal cartilage and these strains can be used for producing septic arthritis in experimental animals. The focus of infection is the junction of epiphyseal bone with articular cartilage from where it spreads intra-articularly. Alternatively an intra-articular leak of the epiphyseal circulation during septicaemia can lead to septic arthritis.

Following gonococcal bacteraemia, 80% of patients will develop a joint infection. Only 1% of pneumococcal septicaemia is complicated by a septic arthritis.

In tuberculous arthritis, it is likely that the haematogenous spread is to periarticular subchondral bone, where the organism may lie dormant for long periods. When multiplication does occur, the infection can spread to the joint where focal infection of the synovium results.

### Pathology

*S. aureus* and *M. tuberculosis* are the micro-organisms that most often destroy joints. Gonococcal infection, although relatively common, virtually never destroys joints even if left untreated.

The precise mechanisms whereby staphylococcus rapidly destroys articular cartilage are unknown. Since the experimental model for the production of haematogenous infective arthritis has become available, it was learned that certain strains of *S. aureus* have the ability to bind themselves to the cartilage by way of glycocalyx. This may be an important factor since the degeneration of articular collagen and the secondary invasion of the *S. aureus* in the deeper layers of the cartilage have been observed.

Previously proteolytic enzymes, the liberation of lysozyme and staphylokinases were proposed as factors that lead to the degeneration of cartilage. None of these have been proved.

The mechanism whereby cartilage destruction occurs in tuberculosis of joints is not clear. The overgrowth of synovium that occurs in this disease is of importance. The hypertrophied synovium adheres to cartilage. Such pannus formation interferes with cartilage nutrition and results in focal degeneration of cartilage.

When the infection in a joint involves epiphyseal circulation, sequestration of a part or the whole bony epiphysis may result. The germinal layers of the growth plate depend on the epiphyseal circulation for their nutritional needs. If the germinal layer of chondrocytes in the growthplate does not survive, growth disturbance will follow. Severe growth disturbance and also destruction of the whole proximal epiphysis of the femur are not an uncommon sequel of infantile septic arthritis of the hip.

# **Clinical Course, Symptoms and SIgns**

The presentation depends on the age and immune competency of the patient, the joint involved and the type and virulence of infective agent.

In neonates and infants non-specific signs of septicaemia, ie, failure to thrive, irritability, anaemia and other signs are usually present for a few days before it is recognized that the child does not move a joint or limb spontaneously and dislikes passive movement thereof. This sign is known as "pseudoparalysis".

In children with septic arthritis the symptoms and signs are usually acute and all movement of the joints are extremely painful. In superficial joints, effusion and local signs of infection are easily detectable. A single joint is usually infected except in children under five years where more joints can be affected simultaneously.

Gonococcal arthritis localizes in one or more joints after preceding polyarthralgia. The adolescent or young adult female is most often affected. Half of the patients have skin lesions, namely pink or red macules that blanch on pressure. Effusions are often large but with little pain or other signs of inflammation. Knees, wrists and hips are most often involved.

# Diagnosis

Every painful swollen joint is an infective arthritis until proved otherwise.

All joints should be examined in patients with pyrexia of unknown origin. Pain on movement and limitation of hip joint movement are often overlooked unless hip joint movement is tested specifically. This requires the immobilization of the pelvis by maximally flexing the other hip (the Thomas test).

In septic arthritis all movements of the involved joint are usually painful. Repetitive movements or "warming up" do not improve the symptoms as it does in inflammatory syndromes.

Joint effusions are easily detectable in superficial joints but difficult or impossible to detect in other joints, ie, hip and shoulder. In these joints X-rays are of great value. The distention of the capsule displaces the extra-articular fat and muscle lines on X-ray pictures. Subluxation is indicated by the increase in distance between the bones articulating at the involved joint. Comparison with the contralateral joint X-rayed in exactly the same position is essential.

A definite diagnosis depends on the demonstration of the organism in the joint fluid or in biopsy of synovial and other joint tissues. In tuberculosis and gonococcal infections the isolation of the organism is seldom achieved. In the latter condition intracellular diplococci is diagnostic but is not always found.

A clear microscopic appearance of joint aspirate does not exclude infective arthritis. Cell counts of 25000 to 250000 cells per cubic millimeter are usually obtained in cases of septic arthritis. If the cell count is more than 50000 with more than 90% neutrophils, infective arthritis must be diagnosed even if no culture is obtained.

Microscopy of joint fluid, after gram staining, often gives early valuable clues to the type of organism before cultures are available.

The differential diagnosis includes a large number of conditions affecting joints. Many of the polyarthritides commence with monoarthritis. Polyarthritis patients may also develop septic arthritis in any one of their affected joints. In the differential diagnosis the following should be considered: osteomyelitis, rheumatic fever and haemophilia in children and crystal arthropathy (ie, gout) in adults.

### Treatment

Treatment should be commenced as soon as septic arthritis is suspected. Early appropriate antibiotics in optimal dosages is the factor that can favourably change the prognosis. Preferably joint aspiration and blood cultures should be taken before antibiotics is started, but if aspiration has to be delayed for some reason, antibiotic treatment should not be delayed.

*S. aureus* septic arthritis remains the most common septic arthritis in all ages, except adolescents. Appropriate therapy against *S. aureus* should therefore always be included in the antibiotic régime. If left untreated, complete destruction of the articular cartilage of a joint may occur within days. The anti-staphylococcal therapy is similar to that of osteitis (see previous chapter) namely 100 mg/kg Cloxacillin followed by 200 mg/kg/24. The anti-staphylococcal potency of broad spectrum antibiotics is not sufficient and it should not be used against staphylococcus.

In neonates, gram-negative organisms and in neonates and young infants streptococcal infections are not uncommon. It is therefore important to obtain a gram stain of the pus. For gram-negative infections ampicillin or an aminoglycoside should be added to the anti-staphylococcal treatment. Antibiotics are continued until the culture indicates that the infection is caused by an organism other than *S. aureus*. If gram-positive cocci are seen, Cloxacillin therapy combined with penicillin G is given until the culture indicates which organism is responsible for the arthritis.

Between six months and four years, a drug effective against *H. influenzae* should always be given until the culture proves another organism. The sensitivity spectrum of this organism is constantly changing. This should be considered before a specific drug is chosen. Consult your microbiology department. *H. influenzae* seldom destroys bone or joint but other sequela of *H. influenza* bacteraemia, ie, meningitis, is often fatal.

Between six years and adolescence, most acute osteites are due to *S. aureus* and a single antibiotic (Cloxacillin) may be used. The same holds for very acute septic arthritis at any age. A less acute presentation in young females is likely to be gonococcal and penicillin may be used as a single drug.

In older patients, or joints damaged by disease, ie, rheumatoid arthritis, a variety of organisms may be responsible. Gram staining and culture are important.

Tuberculous arthritis is difficult to diagnose and to differentiate from monoarticular or pauciarticular chronic juvenile arthritis or adult inflammatory arthritis syndromes. Careful imaging of the periarticular bone often indicates a cystic lesion. From these lesions tuberculosis can usually be demonstrated on histology or culture. Synovial biopsies often indicate non-specific chronic synovitis and joint aspiration seldom yields positive cultures. Since the synovial infection is focal, improved positive yields on histology can be obtained if multiple biopsies are taken representing the total synovial lining of the affected joint.

The diagnosis often depends on exclusion of other causes of chronic synovitis associated with regional osteoporosis. A therapeutic trial on anti-tuberculous drugs is helpful and if positive rapidly leads to a decrease in synovitis and an increased range of motion. The test is, however, non-specific since rifampicin is also an anti-staphylococcal antibiotic.

If fluid reaccumulation occurs after the diagnostic aspiration in septic arthritis, repeated aspirations are indicated in superficial joints. If aspiration fails to completely drain the infected joint effusion, surgical drainage should be performed. Loculation or thick pus is then usually found and these loculations should be broken down.

In the hip joint a negative aspiration or the aspiration of pus should always be followed by surgical drainage. Any of the described surgical approaches to the hip can be used. In contrast to other joints the capsule of the hip joint is not closed after surgical drainage. The skin is always closed. Continuous irrigation has no place in septic arthritis since it is ineffective to wash away organisms such as *S. aureus* or *M. tuberculosis*. The danger of secondary infection is always high.

No antibacterial chemicals or antibiotics should be instilled into joints. These substances will aggravate the synovitis. Antibiotics given intravenously reach the synovial fluid in sufficient quantities in the presence of a synovitis.

Infected joints should be splinted and protected. Complete immobilization should be avoided. Traction is a suitable form of immobilization and protects the joints which, because of the distended capsule, are at risk of being dislocated.

Movement is an essential part of joint physiology. It is beneficial for the preservation of the cartilage and should be encouraged.

The duration of protection of the joints and antibiotic therapy in septic arthritis is empirical. It is suggested that antibiotic therapy in the case of bacterial septic arthritis should be continued for seven to fourteen days and for tuberculosis for three months provided that rifampicin is part of the therapy. Whenever there is a relapse of symptoms, or an increasing synovitis, the antibiotic therapy should be repeated.

Normal function can be resumed as soon as the inflammatory phases subside and the joint capsule and ligaments have regained their normal tension. Physiotherapy may be required to regain a normal range of motion and to rehabilitate muscles after a period of relative immobilization.

## **Prognosis and Complications**

Unless irreversible damage was done to articular and growthplate cartilage, joint function can return to normal. Recovery is often slow and may take months in tuberculosis.

When cartilage is completely destroyed, a bony or fibrous ankylosis follows. Bony ankylosis is more common after staphylococcal arthritis while fibrous ankylosis often follows tuberculosis. If the ankylosis is in a functional position, the patient may have relatively little disability. The natural tendency for ankylosis is, however, to be in unacceptable positions. Positioning of the affected joint during the acute phase of the disease is therefore of great importance. Fibrous ankylosis with a tendency to go into deformity is best treated by arthrodesis in a functional position. Once the joint is arthrodesed, the sepsis usually clears up completely. In selected patients a joint previously destroyed by infection can be converted to a total joint replacement provided that all the prerequisites and indications for total joint replacement of that specific joint are met.

## Comment

## **Infective Arthritis**

# I D Learmonth

Septic arthritis represents an orthopaedic emergency and is still responsible for an untenable legacy of morbidity and mortality. Early diagnosis is essential if the therapeutic measures are to be effective.

In considering the clinical presentation, I think it should be stressed that the child may be acutely toxic and present with paralytic ileus, pneumonitis or meningeal irritation. However, diligent clinical examination will identify the involved joint.

It is equally important to recognize that in the infant the diagnosis may be complicated by the absence of recognized systemic response to infection, the sum of which may merely manifest itself as a failure to thrive. Radiological examination of the hip may be particularly useful in diagnosing septic arthritis of the hip in the neonates.

I believe that *haemophilus influenza* is the most important causative organism in the neonate. In a prospective study of 18 cases of septic arthritis at the Red Cross Children's Hospital *H. influenza* was responsible for the only two poor results. Blockey and McAllister reported a similar experience.

*H. influenza* is not sensitive to the traditional "best guess" empirical antibiotic régime. Immediate gram-staining of pus is therefore essential, and all patients should have blood taken for culture and sensitivity prior to commencing intravenous antibiotic therapy. In the patient under three years of age, it is probably wise to add chloramphenicol to the antibiotic régime until the results of the culture and sensitivity are available.

A rational choice of antibiotic is based on data collected regarding the causal organisms and their antibiograms. Changing sensitivities necessitate constant reviews of the antibiotics used. It is important to give an adequate dose of Cloxacillin - at least 150 mg/kg/day - to achieve bactericidal levels, as it readily binds to protein in the bloodstream.

Antibiotics should be continued for a miminum of three weeks in the presence of a favourable clinical response. Further continuation of therapy is indicated if there is any clinical or laboratory evidence of persistence of infection.

A negative aspiration at the hip is no cause for complacency. Patterson reported repeated failure to aspirate fro a directly visualized bulging hip capsule which subsequently discharged pus under considerable pressure upon opening the joint.

It is necessary to stress that the basic surgical tenet "If there's pus, let it out" pertains. Despite antibiotics, if the pus is not evacuated, toxicity may persist, and I would suggest that exploration is mandatory in the presence of clinical suspicion of an underlying purulent septic arthritis. A limited scar is a small price to pay for the confidence that the underlying insidious destructive process is not continuing unchecked. Morrey et al reviewed 37 patients with septic

arthritis of the hip treated at the Mayo Clinic and ascribed the improved result in recent years to a more vigorous policy of early arthrotomy.

In those joints in which there is an intracapsular metaphysis (such as the hip, the ankle and the shoulder) the adjacent metaphysis should be drilled if the exploratory arthrotomy is negative.

Immobilization is a useful adjunct to the control and eradication of infection. Methods allowing ready access to the involved area are to be preferred.

It is believed that early circumspect mobilization reduces the occurrence of chondrolysis and osteoporosis.

# **Chapter 7.6: Principles of Musculockeletal Tumour Surgery**

# **B G P Lindeque**

### Introduction

Musculoskeletal tumours grow along pathways of least resistance, directed by natural barriers. The medullary canal of long bones offers no restriction to growth of marrow cell tumours, ie, Ewing sarcomas and lymphomas, and very little restriction to primary bone tumours of mesenchymal origin, ie, osteogenic sarcomas. Periosteum is an effective barrier and often constrains an aggressive benign tumour or a low-grade malignant tumour. Cortical bone is only modest in its function as a barrier; it is readily penetrated by benign and malignant tumours alike. Cartilage, both epiphyseal and articular, is very effective in preventing local tumour spread. Cartilage is only rarely penetrated and usually late in the course of the disease. Primary bone tumours invading adjacent joints do so by ligamentous, capsular or synovial penetration rather than through articular cartilage. In soft tissues the major fascial boundaries between compartments act as an effective barrier for local tumour spread contrary to muscle fascia (epimysium) which offers little resistance to local tumour spread.

The local behaviour of tumours is more accurately assessed by the clinical course, radiologic appearance and histological grading than by histological examination alone.

# Definitions

The different stages in biological behaviour of musculoskeletal lesions are as follows:

- Localized, latent, static, inactive benign lesions (stage 1).

These lesions are usually asymptomatic, and are discovered accidentally, seldom causing mechanical dysfunction. They remain completely encapsulated. Radiographic appearance reveals a lesion well marginated by a mature shell of cortical-like reactive bone, without deformation or expansion of the encasing bone.

Histologic characteristics include:

- benign cytology
- a well-differentiated matrix
- intact bony or fibrous capsule
- a low cell to matrix ration
- an absent or narrow inflammatory zone.

- Localized active benign lesions (stage 2).

These lesions are mildly symptomatic and usually discovered due to a pathological fracture or mechanical dysfunction. They grow steadily and continue to enlarge during observation. They remain encapsulated. Radiographic characteristics include margin of thin mature cancellous bone rather than a cortical shell, and the inner aspect is often irregular or corrugated. Expansion and deformation of the overlying cortex is frequently observed.

Histologic characteristics include:

- benign cytology
- a well-differentiated matrix
- intact bony or fibrous capsule
- a narrow inflammatory zone
- bone resorption by osteoclasts rather than neoplastic cells

- Aggressive benign lesions (stage 3).

They are often symptomatic and discovered because of pain, a pathological fracture or a growing mass. They penetrate the capsulew into the surrounding tissues. The reactive zone is thick, oedematous and appears inflammatory. They destroy and permeate surrounding tissues. Radiographic appearance include cortical bone destruction, rapid soft tissue extension, periosteal reaction and endosteal buttressing.

Histologic characteristics include:

- a high cell to matrix ratio

- benign cytology
- fingerlike projections of tumour penetrating the tumour capsule

- a thick inflammatory reactive zone between the tumour capsule and normal healthy tissue

- Low-grade sarcomas (Stage 1).

These present as a slow-growing mass with or without pain. Surrounding tissues therefore have ample time to form reactive tissue and often with a false impression of encapsulation. They grow by gradual erosion of neighbouring tissue. The radiographic features of low-grade sarcomas are less ominous than many aggressive benign lesions. They present with a reactive rim of cancellous bone mixed with areas of "breakthrough" and soft-tissue extension of tumour. Buttressing, Codman's triangles and endosteal scalloping are frequently encountered. They have a low risk for distant metastases.

Histologic characteristics include:

- an even proportion of cells to matrix

- a well-differentiated mature matrix

- malignant cytology (anaplasia, pleomorphism, hyperchromasia, a modest number of mitoses, abnormal mitosis)

- a varying degree of necrosis, haemorrhage and vascular invasion

- numerous extensions of the tumour through the capsule into the inflammatory zone that forms a pseudocapsule.

The tumour may gradually extend into draining venous vessels. Regional lymph node metastases is uncommon and distant lung metastases occur late in the course of the disease.

- High-grade sarcomas (Stage 2).

They present as painful, fast-growing inflammatory masses and are frequently associated with pathological fractures. They stimulate a large rim of inflammatory tissue and grow too rapidly for encapsulation to take place. They are uninhibited by natural barriers and quickly extend extra-compartmentally, involving adjacent neurovascular bundles. They cross epiphyseal growth-plates and even extend intra-articullarly. Tumour extension into draining venous vessels occur rapidly. Radiographic features include bony destruction, permeation of surrounding tissues, poor margination between the lesion and the surrounding bone and illdefined intramedullary extension of the tumour.

Histologic characteristics include:

- a high cell to matrix ration

- poor differentiation

- immature matrices

- high-grade cytology (abundant mitosis, abundant abnormal mitosis, vascular invasion, necrosis, haemorrhage, direct destruction of normal tissue by tumour cells).

- little or no encapsulation

- isolated satellite nodules in the pseudo-capsule (inflammatory reactive zone)

- skip metastasis (isolated nodules of tumour in the normal tissue beyond the reactive lymph node metastases) occur soon.

### The Staging System

A staging system is necessary for the following reasons: to meaningfully evaluate significant prognostic factors, to evaluate factors influencing local spread, local recurrence, and distant metastases. It serves to standardize treatment modalities (surgical and other) for different stages and it provides guidelines for adjunctive therapies.

The final staging of any tumour should precede any treatment schedule. No treatment should be administered before a tumour is properly staged. Staging of a tumour does not rest

in the hands of the surgeon alone but it is done in colaboration with radiologists (well trained in the interpretation of plain X-rays, arteriograms, isotope scans, CT and MRI scans), a cytologist and a pathologist. The site of the biopsy is also determined by the team as a whole in order to maximize its accuracy.

The staging system used in this chapter was adopted by the Musculoskeletal Society of the USA and subsequently by the American Joint Committee for Cancer Staging and Endresult Reporting (AJC). It applies only to lesions of connective tissue histogenesis and not to lesions of round-cell origin, ie, Ewing sarcoma, leukaemias, lymphomas, myleomas or metastatic lesions. The staging system is not based on histological aggressiveness nor on radiological appearance per se, but results in a comprehensive evaluation of the tumour as a whole on grounds of its:

- Grade: Biologic aggressiveness including the histologic aggressiveness, radiographic assessment and clinical behaviour in terms of growth rate, size, temperature and biochemical markers.

- Site: Whether it presents as an intracapsular, intracompartmental or extracompartmental mass.

- Metastasis: Whether lymph node or distant metastasis is present.

Staging modalities, ie, special investigations, should be completed *before* a biopsy is performed. Surgery, post-operative bleeding, oedema and inflammatory reaction distorts tissue planes and render staging modalities inaccurate in illuminating the real extent of the tumour. The biopsy site and type of biopsy is vital to the final outcome of the disease.

The natural progression of benign or malignant connective tissue tumours is the same, lesion for lesion, whether the tumour arises in soft tissues or in bone. A malignant fibrous histiocytoma behaves the same whether it arises in soft tissues and invades the bone or vice versa.

Tumours in certain locations do not lie within well-defined anatomical compartments, due to anatomical structures that meet each other without the formation of proper barriers between them.

Tumours in these areas are always considered extracompartmental (T2). Examples include:

- the popliteal fossa
- the femoral triangle
- pelvis
- obturator foramen
- mid foot
- hind foot
- mid-palmar space of the hand
- periarticular region of the knee
- sciatic notch

- intrapelvic lesions
- periclavicular region
- axilla
- periarticular region of the elbow
- fossa antecubitus
- paraspinal region
- head and neck region

- tumours originating from venous or arterial walls that traverse compartments, ie, a leiomyosarcoma of a venous wall where the vein traverse more than one compartment.

Certain anatomical areas, although not bound by definite barriers, act oncologically as an anatomical compartment. Lesions in these areas are considered intracompartmental (T1).

They include:

- a ray in the hand or foot
- the posterior aspect of the calf
- medial thigh
- posterior aspect of the thigh
- pericapsular regions with the exceptions of the elbow and knee joints (vide supra).

Certain tumours with benign cytology may metastasize to the lungs, ie, a benign giantcell tumour of the bone. These patients have a long survival unlike those with malignant metastases. Lung or bone metastasis does not render a tumour malignant.

## **Staging System**

The staging system is based on three factors, as mentioned above:

Grade (G), site (T) and metastasis (M)

G0 - biologically benign lesions

G1 - low-grade malignant lesions as determined by all staging modalities including a biopsy

G2 - biological high-grade malignant lesions

T0 - the lesion is confined to the intracapsular space

T1 - the lesion has extracapsular extensions but both the lesion and the reactive zone are contained within the anatomic compartment bounded by natural barriers for instance cortical bone, articular cartilage or fascial septae. The lesion is extracapsular bu intracompartmental.

T2 - the lesion or its reactive zone extends beyond compartmental barriers. It is extracompartmental. An intracompartmental lesion may be rendered extracompartmental due to inadequate incomplete surgical excision or by performing a large indiscreet incisional biopsy.

M0 - indicates no evidence of regional or distant metastasis

M1 - signifies either regional (for instance local lymph node) metastases or distant (for instance lung) metastases. Tumour extending into the venous system beyond the compartment may also be regarded as metastatic disease.

Benign lesions are staged as follows:

Stage 1: G0 to M0 Stage 2: G0 to M0 Stage 3: G0 T1-2 M0 to M1

Malignant lesions are staged as follows:

Stage IA: G1 T1 M0 Stage IB: G1 T2 M0 Stage IIA: G2 T1 M0 Stage IIB: G2 T2 M0 Stage IIIA: G1-2 T1 M1 Stage IIIB: G1-2 T2 M1

### **Examples of the Grading of Certain Tumours**

Low Grade (G1)

Parosteal osteogenic sarcoma Clear cell sarcoma Chordoma Myxoid liposarcoma

#### High Grade (G2)

Classic osteogenic sarcoma Malignant fibrous histiocytoma Synovioma Pleomorphic liposarcoma Epithelioid sarcoma (vide infra)

#### **Difficulty in the Grading of Certain Sarcomas**

Certain malignant soft-tissue sarcomas are difficult to grade either as low grade or high grade. An example is epithelioid sarcoma. Epithelioid sarcoma is deceptive, both in making a diagnosis and in grading and staging. Histologically it has low-grade malignant characteristics but clinically it is an extremely malignant lesion.

Lymph node, lymphatic channel and vascular invasion occur readily in the course of epithelioid sarcomas. If the distal parts of a limb is swollen, it is known that the tumour has already infiltrated veins and lymphtic channels alike. It tends to spread by forming nodular lesions proximal in a limb. This tumour is extremely difficult to stage and to cure. In spite of its low-grade cytologic characteristics, it should be treated as a highly malignant lesion.

For lesions in the elbow region a forequarter amputation is contemplated. For lesions in the hand a prophylactic axillary lymphnode dissection is necessary for proper staging. If positive, one should proceed with a forequarter amputation.

Patients with lymphatic metastases, ie, presenting with lymphnode involvement due to osteogenic sarcomas, are unlikely to survive more than a brief period. Patients with fice or more lung metastases also fare poorly (five year survival of 12.5%) as compared to patients presenting with a single pulmonary metastasis amenable to surgery (five year survival of 71%). Stage III lesions should be subdivided or this information should be added when results are analysed to reflect this difference in prognosis.

## **Progression and Regression of Tumours**

Some tumours have the tendency to change their grading. This phenomenon is known as dedifferentiation. An example of this is a chondrosarcoma or even an enchondroma but later develops areas of so-called dedifferentiated fibrosarcoma or osteogenic sarcoma (highgrade malignant areas). This phenomenon is also known as progression. Some cases of socalled dedifferentiated chondrosarcomas have areas of low-grade chondrosarcomatous tissue adjacent to high-grade fibro- or osteosarcoma tissue. These tumours represent sarcomas that develop from two different cell clones, of which one is highly malignant; they are also known as chondrosarcomas with an additional mesenchymal component. These tumours, in fact, do not represent progression. In other cases, a chondrosarcoma progresses from a low-grade lesion to a high-grade sarcoma.

Incomplete excision of lower grade lesions, especially repetitive excisions, may lead to a more aggressive (higher grade) local recurrence.

An example of a benign tumour that progresses to a more aggressive lesion is a fibromatosis that may be indolent for quite some time and suddenly start to be aggressive and infiltrate local tissues. A similar aggressive fibromatosis may regress to a more indolent type after some time. Active stage 2 non-ossifying fibromas and simple bone cysts may regress to latent stage I lesions at skeletal maturity.

## **Staging of Multifocal Sarcomas**

Tumours occasionally present in more than one location. Osteogenic or fibrosarcomas may present in more than one bone without lung metastases. If the lesions are of similar dimensions and arise simultaneously, they are called synchronous multifocal osteogenic sarcomas. If they differ in dimensions (bone of origin' size taken in to account) and arise at different time intervals, it is called metachronous multifocal sarcoma.

Multifocal sarcomas are classified as Stage III disease. They carry a similar grave prognosis compared to other Stage III lesions that present with lung metastases.

## **Staging of Ewing Sarcoma**

Ewing sarcoma is not included in the surgical staging system or musculoskeletal lesions of connective tissue origin. Their natural course is different; it is regarded by some as a systemic disease presenting with multicentric lesions rather than metastases of a primary lesion to other parts of the skeleton.

*All* lesions present as high-grade sarcomas, histologic appearance, therefore, plays no role in the staging of the disease. Factors influencing the course of Ewing sarcoma include:

- extra-osseous extent of a local tumour
- multicentricity
- distant metastases (including bone marrow infiltration).

Ordinary 99m Technetium bone scintigraphy in our experience is not sensitive to demonstrate bone lesions (primary or metastatic) due to Ewing sarcoma. Ewing sarcoma cells may infiltrate bone marrow diffusely without showing up on 99m Technetium scintigraphy. Bone marrow infiltration away from the main tumour mass is regarded as metastatic disease and will show up on Tc-99m sulphur colloid (SC) scans. It is difficult to demonstrate lumbodorsal vertebral infiltration with SC scans due to the high uptake of isotope in the liver. It is also difficult to demonstrate the skull, ribs and forearms because of poor uptake. We believe a total body skeletal MRI scan to be the most sensitive and accurate to demonstrate marrow infiltration.

The stages in Ewing sarcoma are:

- (Ew I) solitary intraosseous
- (Ew II) solitary with extraosseous extent
- (Ew III) multicentric skeletal
- (Ew IV) distant metastases.

An addition to this staging is proposed: (Ew I-IV PNET) - PNET related Ewing sarcoma and (EwS) - Ewing sarcoma of soft-tissue origin.

It is generally accepted that Ewing sarcoma is a tumour of bone which occurs sporadically as a primary tumour of soft tissue. By contrast, a neuroblastoma occurring in bone is metastatic. Yet another category of bone tumours is the peripheral neuroectodermal tumour (PNET) that arises in bone, without involvement of central neuroectodermal tissues. These tumours resemble Ewiung sarcoma histologically, but separation is possible by a variety of characteristics.

# **Staging Modalities**

The modalities used in the staging of musculoskeletal tumours include plain X-ray films, isotope bone scans, arteriography, computed axial tomography scans and MRI (magnetic resonance) scans.

### **Plain X-Rays**

Plain X-rays remain the most important modality in the initial diagnosis of musculoskeletal tumours. Multiple views of the tumour and the chest are mandatory in the diagnosis as staging.

The following points relate to isotopic scans:

(a) Bone lesions:

- Isotope bone scans are of value to demonstrate:

- multiple bony lesions

- large skip metastasis or

- adjacent bone involvement in case of a soft tissue sarcoma.

They are neither specific nor accurate.

- High-grade malignancies may extend beyond the area of increased uptake (hot areas).

- Hot areas may be much more extensive than the real extent of a tumour, due to increased uptake in the epiphyseal areas in children.

- Small skip lesions (1 cm) in the tubular bone marrow are not necessarily potrayed on a technetium scan.

- Most hot spots besides the area of the tumour will turn out to be spurious even after proper X-ray, CT or MRI imaging of that area.

- Hot spots away from the tumour mass in tubular bone marrow should be followed by an MRI scan to rule out occult metastasis. If positive on MRI they should be regarded as metastases and duly biopsied, before staging of the tumour is finalized.

- Superscans - patients with extensive marrow infiltration due to multiple metastases may show a diffusely increased uptake throughout the skeleton. This may be overlooked and regarded as normal, due to its uniform pattern of infiltration.

(b) Soft-tissue lesions:

- Cold scans of bone adjacent to a soft-tissue sarcoma may be misleading and bony involvement of this tumour may still be the case.

- Hot spots about the thorax usually reflects rib metastasis. Pulmonary nodules, even when clearly visible on plain X-ray films, are not seen by a technetium bone scan. Neither will radiologically occult pulmonary metastases show up on a technetium scan.

(c) Isotope scans in complicated circumstances:

- Bone scans are very unreliable following biopsy, fracture or other manipulations. The scan will remain hot until the process has matured; this may take months or years.

- Diffuse infiltration due to myeloma may show no increased uptake on technetium scam, ie, technetium scans are not sensitive in depicting marrow lesions. MR scans are the modality of choice in accurately portraying diffuse marrow infiltration or multiple bony metastases.

Our experience with angiography has led us to believe the following:

- Angiography is as accurate in the localization of the tumour as gross dissection.

- It is very inaccurate in diagnosis.

- It is of great help in planning the biopsy and in anticipating unexpected blood loss. Areas of hypovascularity during the arterial blush phase or early venous phase may be due to necrosis and should be avoided while taking a biopsy. Areas with good arterial blush at the periphery are more suitable for biopsy - these areas represent the active growing part of the tumour.

- Angiography is of limited value in assessing the intraosseous extent of neoplasms.

- For three months or even more after a local excision, the reparative vascular response to surgery may complicate the identification of any but the most gross residual.

- Angiography is very valuable in assessing the intraosseous extent of neoplasms.

- For three months or even more after a local excision, the reparative vascular response to surgery may complicate the identification of any but the most gross residual.

- Angiography is very valuable in differentiating between a soft-tissue osteogenic sarcoma (OGS) and a pseudomalignant myositis ossificans (PMO). In PMO no malignant circulation is seen but only increased normal vasculature, contrary to a soft tissue OGS. Malignant circulation implies: increased vascularity in most cases, incarceration of vessels, venous pooling, A-V fistulae, stump ending and "purposeless" vessels.

- Angiographers specializing in interventional radiography of musculoskeletal tumours produce superior studies compared to those casually involved.

- The interventional radiologist should be present during the pre-biopsy staging meeting. He should be well aware of the reasons for and the particular questions to be answered by the angiographic investigation.

- The early arterial phase demonstrates the relationship of the lesion to the major vessels and the peripheral inflammatory capsule about the lesion. The peripheral inflammatory capsule is similar to the gross pseudocapsule during tumour dissection.

- During the late arterial phase tumour vessels can be demonstrated, ie, incarceration, tortuosity, and an abnormal course of vessels innervating the tumour. These vessels are large and do not relate to any normal anatomic vessels in that region.

- During the capillary phase a tumour blush is demonstrated which denotes the microvascularity of the tumour - a tumour blush is not present in normal tissues, except in inflamed mucous membranes.

- During the early venous phase arteriovenous shunts are demonstrated by fast clearance of contrast from the tumour into the venous circulation. Some tumours may present with sluggish venous clearance because of venous lakes. Telangiectatic osteogenic sarcomas or osteogenic sarcomas with an aneurysmal bone cyst component may demonstrate venous pooling and arteriovenous shunts.

- During the late venous phase the major veins which drain the lesion is demonstrated and should be avoided during the biopsy procedure.

- Angiography depicting anomalous vascular patterns is valuable in designing surgical approaches.

- Angiography is not sensitive enough to show micro-extensions (macro-satellites) or small skip metastases beyond the pseudocapsule. Microsatellites denote microscopic tumour in the inflammatory reactive zone while skip metastases denote the presence of tumour beyond the inflammatory reactive zone in normal surrounding tissue, for instance higher up in the medullary cavity.

- Angiography plays an important role in the evaluation of the tumour response to chemotherapy. If the tumour is responsive, the tumour blush decreases and subsequently disappears, the tortuous tumour vessels disappear and the tumour mass shrinks in size.

- Tumour escape, ie, areas in the tumour not responsive to chemotherapy, is portrayed by the continuous presence of tumour vessels, and a tumour blush, because of increased growth in these areas. By highly selective catheterization of these remaining tumours vessels, intra-arterial chemotherapy can be injected in optimal concentrations to effect maximum response. Areas of persistent hypovascularity denotes a poor blood supply and lead to persistent tumour and poor wound healing.

- Intra-arterial embolization is done prior to definitive surgery in order to decrease intraoperative blood loss - especially in highly vascular tumours. It is sometimes used to obtain tumour control in areas inaccesible to surgery without causing extensive harm, ie, a giant cell tumour of the sacrum.

The following points are relevant to cat scans:

- CAT or MRI scans are usually done prior to angiography. A decision or not to perform a CT or MRI scan is made after the clinical examination and plain X-ray films have been taken into account. In certain instances we favour an MRI and in others we favour a CAT scan (vide infra).

- Using the bone window, intralesional calcification, ossification, extraosseous extension of a bony tumour or "ring" ossifications are vividly demonstrated and are often of diagnostic significance.

- Using the soft-tissue window, tumour masses of 5 mm or more in soft tissue can be identified. Lung metastases 2-5 mm in diameter can be detected. At present CT is the most reliable method in detection of small pulmonary metastases. Extracompartmental extension through major fascial planes is clearly seen, especially when radiolucent fat planes lie along the fascial septae, since their displacement or obliteration is easily seen due to the marked difference in density between fat, muscle and tumour.

- By injecting a radio-opaque contrast medium intravenously, the lesion is seen more clearly due to marked increase (enhancement) in the radio density of the lesion in the venous phase.

- The contrast medium also serves to delineate the vascular bundle in its relation to the tumour. CAT scans with contrast and new-generation MRI scans are most reliable techniques in making judgements about the presence of an adequate surgical plane between a lesion and the large vessels.

- CAT scans serve to demonstrate heterogeneity in tumours as well. An example is an osteogenic sarcoma of the pelvis with a large chondroid component. CT will clearly show the osteogenic as well as the chondroid regions. A representative biopsy should include tissue from both these regions.

- CAT scans are also valuable in directing needle placement to a representative area when performing needle biopsies, especially in spinal tumours.

- CAT scans are very valuable in evaluating the response of a tumour to chemotherapeutic drugs. Osteogenic sarcomas responsive to chemotherapy show a decreasing inflammatory zone, increased ossification, improved margination between the reactive inflammatory zone and normal healthy tissue and gross tumour shrinkage. "Tumour escape" signifying areas in the tumour not as responsive as the rest is also portrayed by CT: the tumour may grow in these areas, the inflammatory zone fails to shrink and ossification is absent.

- Cystic lesions are usually entirely homogenous and often display a fluid level if the patient is asked to remain supine for two hours prior to scanninhg. This is particularly true of aneurysmal bone cysts.

- CT scans are not reliable in determining the bony involvement of an adjacent softtissue tumour. The only reliable scan to demonstrate bone involvement is the MRI.

- CT scans are not as reliable as MRI to demonstrate the reactive inflammatory tumour response, or infective changes.

The following points are relevant to MRI scans:

- The MRI is extremely helpful in evaluating intramedullary spread of a tumour. Comparing MRI pictures to the pathological specimen has demonstrated the accuracy of the MRI to the nearest millimeter in our experience. - The MRI is helpful in evaluating soft tissue extension of tumours, the exact margins and relations of soft-tissue sarcomas to major arteries and nerves.

- The contrast between abnormal and normal soft tissue is much more striking by MRI than by CT, but not so in bone. No MR signal is obtained from bone due to the absence of protons, hence corticalbone and calcifications appear as black lines or dots on MR images. Cortical bone appears black and fat appears white. MR imaging is therefore inferior to plain X-rays and CT scans in the evaluation of periosteal reactions, ossification, calcification and cortical destruction.

- MRI is considerably more sensitive than CAT scans or angiography in demonstrating the relationship of soft tissue lesions to fascial planes, adjacent bone, and adjacent nerves.

- New generation MRI scans are even more realiable than CT with contrast or angiography in demonstrating the relationship of the lesion to major vessels. The possibility exists that by means of an MR angiogram, a standard angiogram or CT with contrast can be obviated. This is of special value in patients allergic to the contrast medium or with septic lesions in the vicinity of the tumour.

- MRI is extremely valuable in the three-dimensional portrayal of a tumour and is far superior to CT in this aspect due to the possibility of three-dimensional viewing and a display in all planes, for instance coronal, sagittal, horizontal or even oblique.

- MRI scans are preferred to myelograms or CT with contrast in staging primary tumours of the vertebrae. Contrast material injected intrathecally may cause tumour spread if the needle is inadvertently inserted into the tumour or an occult metastasis.

- MRI scans are extremely sensitive to demonstrate vertebral or other bony metastases.

- It is also the most sensitive method in locating areas of myelomatosis in bone, thereby greatly increasing the possibility of a positive marrow aspiration. Plain X-ray or technetium scans may fail to demonstrate large areas of infiltrative myeloma. This also applies to Ewing sarcoma.

- MRI has become most valuable in evaluating tumour response to chemotherapy. It is not invasive, it obviates the need for repetitive radiation exposure and vividly demonstrates tumour shrinkage.

- It is superior to CT scans in identifying haemorrhage, oedema, reactive inflammation and intralesional necrosis. It demonstrates the extent of potential contamination by the haematoma or granulation tissue from a previous incomplete excision.

- It is a mandatory examination in staging of osteogenic sarcomas, due to its accuracy in demonstrating proximal skip metastasis.

- It is more reliable than CT in the portrayal of liver metastasis.

- It is very helpful in demonstrating lymph gland involvement by sarcomas as well as retroperitoneal metastasis (CT is equally effective in demonstrating mediastinal lymph glands).

- It is also helpful in the evaluation of local control with metallic prosthesis in place. It does not produce artefacts of similar magnitude as CT. Soft-tissue structures adjacent to metallic prosthesis are still visible on MRI.

- It is the most sensitive staging modality in discerning tumour recurrence from postoperative surgical scar tissue or to tell the difference between tumour recurrence and radiotherapy changes.

- It gives valuable information in metastatic cancers. MR is more sensitive than technetium scans in the diagnosis of metastatic disease to the bone. (See section on the staging of metastatic disease).

We have altered our schedule in the staging of musculoskeletal tumours, depending on the clinical examination and plain X-ray films of the tumour and chest. If plain X-rays demonstrate a homogenous radiolucent lesion consistent with a stage 2 or 3 benign lesion, we now proceed with a technetium scan and an MRI investigation.

If plain X-rays demonstrate areas of calcification or ossification, or if the possibility exists of an osteoid-producing lesion in the differential diagnosis, a technetium bone scan and CT scan are done. After re-evaluation the necessity to proceed with MRI and finally angiography is assessed.

Osteogenic sarcomas are staged with plain X-ray films of the tumour and chest, technetium scans, CT scans, MRI scans and angiography (in that order) prior to biopsy.

We investigate soft-tissue sarcomas with plain X-ray films, technetium scans and MRI of the lesion and liver. CT scans of the lungs are performed in all malignant lesions.

### **Biopsies**

The art of taking a biopsy should be refined to perfection. The following rules should strictly be adhered to in order to prevent complications:

- The biopsy should not interfere with the final excision of the tumour.

- The biopsy should be removed en bloc with the tumour.

- The biopsy should be taken preferably by the same surgeon who performs the final excision.

- An incisional biopsy is used for suspected malignant lesions.

- An excisional biopsy (marginal excision) is used for suspected benign lesions.

- The biopsy should always run longitudinally in a limb and should be parallel to the neurovascular bundles. It should be placed far enough from the neurovascular bundle in order that a final excision does not include the bundle.

- The biopsy should be properly land-marked (proximal, distal, medial and lateral) in the operating room in order to facilitate pathological examination.

- The pathologist should be present in the operating room while the biopsy is taken in order to determine the adequacy of an open biopsy by gross inspection and by performing a frozen section on the specimen. The frozen section is done to evaluate the adequacy and representation of tumour tissue in the specimen. A frozen section can be done if the tumour tissue is sectionable with a surgical blade - it is not possible if the tumour is too hard to cut with a surgical blade.

- A portovac exit should always be longitudinal to the biopsy site and in line with the biopsy to prevent an unnecessary inclusion of normal tissue during the final excision of the tumour.

- A biopsy should never be done under local anaesthetic but preferably under general anaesthetic or under a regional bloc. Local anaesthetic distorts the anatomy and cell identification becomes difficult.

- The pathologist attending the frozen section needs to attent the preoperative staging conference in order to familiarize himself with the preoperative differential diagnosis. The surgeon should convey to him in precise detail what he expects from the frozen section. This is absolutely mandatory to achieve acceptable levels of accuracy.

An orthopaedist does not appreciate being asked to operate on a patient without the opportunity to examine the patient and the X-rays before surgery. Similar courtesy should be extended to our pathology colleagues and it is their duty to refuse definitive opinion in its absence.

- A biopsy should be done no longer than seven days after admission. Staging studies should start immediately and should be completed in six days. It is not justifiable to linger on week after week. Survival is dependent on the time from the start of a tumour till treatment is begun.

The rationale of frozen sections in musculoskeletal pathology includes the following:

- The pathologist has the opportunity to evaluate the adequacy and representation or lack thereof of the biopsy specimen. If the tissue obtained is necrotic or uninterpretable, the time to take more tissue is there and then.

- If the process confers more with an inflammatory or infective condition, tissue cultures may be sent immediately.

- If the diagnosis confers with a neoplastic process but a definite diagnosis cannot be made with confidence, an immediate decision should be made as far as fresh tissue is

concerned for electron microscopy, special stains, immunofluorescence or ultraviolete microscopy. This must be done before the remaining tissue is dropped in routine fixatives which would render such special stains impossible.

- In order to reach a verdict as far as a diagnosis is concerned upon which a therapeutic decision can be made, the following questions should be answered:

- Is it a neoplastic, infective or chronic inflammatory process?

- If neoplastic, is it benign or malignant?

- If infective, is it acute or chronic?

- If malignant, is it primary or metastatic?

- If primary, is the frozen section adequate to finalize the diagnosis immediately?

- If the diagnosis can be made with confidence, is it feasible to proceed with definitive curative surgery there and then or should surgery follow other non-surgical treatment modalities, for instance, chemotherapy?

- Does the frozen section confer with one of the pre-operative differentials in diagnosis? If not, is it possible that tissue obtained is not representative?

If the frozen section material does not confer with the clinical and radiological clues, even after large representative sampling, it is best to postpone a therapeutic decision till the final sections, special stains and electron microscopy are available.

In roughly three-fourths of tumour presentations upon which a frozen section is requested in order to clarify immediate therapeutic decisions, will such a section be confirmatory and should a therapeutic decision be taken? To adopt the attitude of: "I will never act on a frozen section", is to lack common sense.

- Frozen sections may prevent unpleasant surprises, for instance, a 12-year old patient presents with a lesion in the distal femur believed to be fibrous dysplasia. A frozen section reveals a round cell tumour. Instead of proceeding with a preplanned curretage and allograft filling, the biopsy site is closed, awaiting confirmation of the diagnosis of Ewing sarcoma.

# Principles in the Treatment of Musculoskeletal Tumours

Traditional terms such as incisional biopsy, excisional biopsy, resection and amputation are difficult to define in biologic and/or anatomic terms. The current method of describing surgical margins include the following:

- Intracapsular - the plane of dissection is within the lesion and tumour is present at the margin.

- Marginal - the plane of dissection is extracapsular within the reactive zone. The margin is sectioned through reactive tissue with or without micro satellite tumour (in case of malignancies) left in situ.

- Wide - the plane of dissection runs beyond the reactive zone through normal healthy tissue within the compartment. Distant skip metastases may be left behind.

- Radical - the plane of dissection runs beyond the compartment. The compartment, for instance the whole femur, is removed en bloc.

An amputation therefore is not a surgical margin. It is incorrect to use the term amputation to state the preferred method of treatment for a certain tumour. For instance, an amputation may be intracapsular if it runs through the tumnour, it may be marginal if it runs through the reactive zone, it may be wide if it runds beyond the reactive zone through normal healthy tissue. A disarticulation may similarly be radical, ie, the whole compartment is removed, and it may be marginal due to a lesion that is adjacent to the joint and the line of dissection is only through the reactive zone and not even through normal healthy tissue.

## Neo-Adjuvant and Adjuvant Chemotherapy

The term "neo adjuvant or primary chemotherapy" denotes the administration of chemotherapy not as an adjuvant to surgery, but as the primary and first line of treatment *before* surgery. Most malignant sarcomas have already seeded miocrometastases to the lungs or other organs at the time of presentation. Surgery ablates the primary but does nothing to the already existent (but not yet apparent) micrometastases. In an attempt to rid a patient of the micrometastases (which will lead to his death if left unattended) primary or neo-adjuvant chemotherapy is administered. Surgery in the form of an amputation or local resection will then follow and chemotherapy will again be administered after surgery (depending on the specific protocol). The term primary chemotherapy is preferred to neo-adjuvant chemotherapy: chemotherapeutic administration is primary and continues throughout the therapeutic régime.

Adjuvant chemotherapy denotes the administration of chemotherapeutic drugs as an adjuvant, secondary to primary surgery. In these cases, the patient receives an amputation followed by the administration of chemotherapeutic drugs.

Current protocols for osteogenic sarcoma show improved five-year and overall survival rates as well as increased disease-free survival rates of patients who received primary chemotherapy followed by surgery as compared to patients that received surgery followed by adjuvant chemotherapy.

The preferred treatment for different stages of tumours is as follows:

# **Benign Lesions**

stage 1 - intracapsular removal

stage 2 - marginal or intracapsular excision with an effective adjuvant

stage 3 - wide or marginal excision with an effective adjuvant.

# **Malignant Lesions**

stage 1 - wide excision

stage 2 - radical resection or a wide excision plus an effective adjuvant

stage 3 - chemotherapy, thoracotomy and radical resection.

An example of a stage 2 benign lesion of bone is the so-called giant-cell tumour. The treatment for a giant-cell tumour is a marginal excision of the tumour or if the tumour is in the region of a joint, an intracapsular curettage may be performed and an effective adjuvant instituted such as cementation with bone cement or cryosurgery.

An example of treatment of a Stage II malignant lesions such as osteogenic sarcoma of the distal femur is a radical resection, which means hip disarticulation or primary chemotherapy, plus an amputation or a wide local resection with a limb salvage procedure.

#### **Down-Staging of Tumours**

Down-staging of a tumour means that after chemotherapy or radiotherapy, a tumour is rendered more "resectable" in that it shrinks in volume so that a less aggressive margin is necessary to rid the patient of the tumour. It may also result in the disappearance of lung metastases or lymph-node metastases in tumours, ie, a Wilms' tumour, which render the tumour surgically resectable. The concept and the term "down-stage" is fundamentally wrong. Primary chemotherapy does not change either the grading or the site of a tumour. This treatment kills all or most of the tumour cells, it delineates or confines the reactive zone to such an extent that staging modalities, ie, MRI or CT, are more accurate in depicting the exact extent of the tumour.

Primary chemotherapy also acts against distant micrometastases, especially of the high grade tumours. A less aggressive surgical procedure is now technically possible due to improved preoperative staging and shrinkage of the tumour (especially marrow cell tumours). There is less chance of leaving microsatellites of the tumour behind because the reactive zone is delineated more accurately. The excision should still take place outside the reactive zone through normal healthy tissue. Instead of doing a radical resection, a local wide excision is done.

Local recurrences developing after excision of "down-staged" lesions are by no means of a lower grade than the original tumour, on the contrary, they are more aggressive and less amenable to conservative local treatment, and lung metastases usually occur concurrently.

Pathological specimens examined by us after primary chemotherapy reveal remaining viable tumour cells (tumour escape) usually at the *periphery* of the tumour in the reactive zone, subchondrally, subsynovially, in the biopsy tract and intramedullary. A conservative (marginal) surgical excision will lead to local recurrences.

"Neo-adjuvant treatment" or, more correctly, "primary chemotherapy" or radiotherapy does not lead to down-staging of a lesion, it leads to improved delineation by MRI and CT besides its effect on tumour cell division. It is more accurate to use the term "improved delineation due to neo-adjuvant treatment".

Stage III lesions with minimle visible lung disease or lymph node metastases may respond to such an extent on chemotherapy that all small nodules either disappear from the lungs, or ossify in cases of osteogenic sarcoma. An amputation or exarticulation is now feasible, not due to down-staging - it still remains stage III disease - but due to adequate tumour response on chemotherapy. It is more accurate to use the term "surgical control of the primary after systemic chemotherapeutic responsiveness in stage III disease" or "surgical control of the primary after neo-adjuvant elimination of metastases". Metastasectomy of calcified (ossified) lung metastases in osteogenic sarcoma is of utmost importance in rendering these patients disease free with an improved survival rate.

## **Metastatic Disease of Bone**

Metastases to the skeleton are the most common of all bone neoplasms. More than 80% of bone metastases originate from the lung, breast, and prostate.

Almost 20% of patients with metastatic cancer will develop detectable skeletal metastases during their life and if autopsies are done on patients dying of cancer, 70% of them will show evidence of skeletal metastases.

The majority of metastatic lesions become evident after the diagnosis and management of the primary lesion. The metastatic lesions are clinically suspected because of the history.

Metastatic disease to bone is commonly associated with pain. The pain may precede any radiographic evidence of metastases by weeks or even months. The pain is usually unaffected by activity, wakes the patient at night or keeps him awake, it is deep seated and is not relieved by ordinary analgesics. It has the tendency to increase in severity.

Pathologic fracture occurs in 15% of patients with radiographic evidence of skeletal metastases. Destructive lesions are more prone to fracture than mixed or blastic lesions.

Purely destructive metastases are seen with carcinoma of the lung, kidney, breast, thyroid, gastrointestinal tract and neuroblastoma in that order of frequency. Purely blastic metastases are seen with carcinoma of the prostate, breast, bladder and stomach.

Metastases occur by haematogenous dissemination, hence the bones with haematopoetic marrow are the most commonly involved, ie, the spine, ribs, pelvis, metaphyses of long bones, sternum and skull. Spinal metastases occur mainly in the vertebral bodies, affecting the lumbar region most often, followed by the thoracic, cervical and sacral vertebrae.

## The Staging (Work-Up) of Metastatic Disease

The work-up should include thorough history, a complete examination and special investigations. Special attention is paid to patient's age (older than 40 years makes metastatic disease more likely), lymphadenopathy, hepatomegaly and splenomegaly.

Laboratory studies should include a full blood count, sedimentation rate, platelet count, reticulocyte count, urea, uric acid, electrolytes, alkaline phosphatase, acid phosphatase, serum calcium and phosphate and protein electrophoresis.

Anaemia is a frequent findings in carcinoma. Elevated levels of alkaline phosphatase and serum calcium are features of metastases. Elevated levels of acid phosphatase are characteristic of carcinoma of the prostate. A monoclonal spike on electrophoresis indicates the diagnosis of myeloma.

The majority of patients with metastatic disease has a known primary, there is no need to order a whole battery special investigations. Blood investigations are done as previously mentioned, and a radio-isotope skeletal study is done.

Radio-isotope evaluation of the skeletal system is very helpful as a screening investigation for metastatic disease. Areas of increased uptake will receive plain X-ray films and MRI studies if the diagnosis is in doubt. 99m Technetium is currently the most frequently used radionuclide in bone scintigraphy. Bone scintigraphy is 95% accurate for skeletal metastases. The older a patient becomes, the more likely will "hot spots" be due to osteoarthritic changes, or stress fractures to osteoporosis. It may be extremely difficult to tell the difference between osteoporosis and metastatic bone disease. MRI studies are indicated in those situations: they are accurate in differentiating osteoporosis from metastatic bone disease. MRI will also reveal the extent of metastatic infiltration.

One should be aware of the "superscan" entity: metastatic disease infiltrates to such an extent that almost the whole skeleton lights up and this should not be interpreted as a normal scintigraph study.

Multiple myeloma and aggressive metastatic tumours are capable of producing extensive bone destruction without a significant reparative process of new bone formation.

When a patient, 40 years or older, presents with a solitary bone lesion that poses a diagnostic problem, staging studies are indicated prior to biopsy, even though metastatic disease is high up on the differential list.

Needle biopsies or needle aspirations are indicated to confirm the diagnosis of a solitary metastasis. They are also indicated in widespread metastatic disease in which the histologic diagnosis of metastasis has not yet been confirmed. We prefer to confirm the diagnosis in view of the fact that more than one type of tumour may be present. A patient with a mammary carcinoma may suffer from malignant melanoma as well. Almost 9% of patients with an occult primary cancer that presents with a metastasis has a history of at least one other malignancy before their occult primary cancer.

Carcinomas are staged according to their local extent (T), regional lymphatic metastases (N), distant metastases (lung, bone, liver, etc) (M), and occasionally their histologic grade (G). Each of these carcinomas has individual differences in staging. The abovementioned staging modalities, especially the MRI, are extremely helpful in planning the ideal site for biopsy.

The question in solitary bone metastasis may arise: does a biopsy lead to iatrogenic spread of the cancer? Studies have shown an increased number of cancer cells in the blood after manipulation or biopsy but it has also been established that if final surgery follows in no more than 30 days after biopsy, no decrease in survival will ensue.

### **Principles in the Treatment of Metastatic Disease**

The important question to be answered before treatment is instituted is whether the disease is curable or not. If cure is attempted, all the principles as laid out for primary bone tumours apply. Wide excisions or radical resections are necessarry under these circumstances.

If palliation is attempted for a pathological fracture or impending pathological fracture, the following principles apply:

- The first operative procedure has the best chance to be successful. Do rather too much than too little: prevent the possibility of a second procedure.

- Prosthetic replacement is a better concept than open reduction and internal fixation for lesions that involve joints.

- Internal fixation in combination with methylmethacrylate or Ionas bio-active bone cement is a good concept for metaphyseal or diaphyseal lesions. Replace as much defective bone as possible.

- Aim for the soonest return to a mobile and functional state possible. Long periods of immobilization are not well tolerated.

- Minimize the period of hospitalization: the patient's decreased life expectancy must be taken into account.

- Radiation therapy for pathological or impending pathological fractures is not contraindicated. It relieves pain and fracture healing will take place.

- Impending pathological fractures should be fixed by either internal fixation or prosthetic replacement *before* the fracture actually occurs. Patients with actual fractures will survive an average of 4.7 months while those with lesions stabilized prophylactically will survive an average of 13.8 months postoperatively.

# **Cancer of Unknown Primary Site (CUP)**

Metastatic cancer of unknown primary site is a persistent and perplexing problem. The incidence of metastatic malignant disease without a known primary is as high as 7%, and it

may constitute up to 15% of referred patients with solid tumours. Only 15% of patients will have tumours that are responsive to systemic therapy. Despite the usage of modern staging modalities, ie, sonography, mammography, CT, nuclear scans, biochemical and immunological tests, the percentage of cancers that present as occult primary malignancies has remained stable over the years.

The histologic cell types in patients with histologic proven CUP include:

- adenocarcinoma 37%
- carcinoma 16%

- squamous cell carcinoma 12%. Patients with squamous cell carcinoma have a significantly longer median survival (9 months) than patients with adenocarcinoma.

The overall survival of patients with CUP is poor, with a median survival of five months, a year survival of 23% and a five year survival of 6%.

Our strategy in patients with skeletal metastases of unknown origin entails the following: medical history, physical examination, routine laboratory studies, chest radiograph, 99m Technetium bone scintigraphy and CT of the abdomen and pelvis. If this regimen fails to reveal the primary site, it is unlikely that further extensive diagnostic procedure will help to trace the primary. The whole work-up should not take longer than seven days. It is not justifiable to drag on doing special investigations: a needle biopsy is performed immediately after the abovementioned strategy and treatment is started promptly. Instalment of treatment should not be delayed since eventual survival is directly related to treatment.

#### Comment

### **Principles of Musculoskeletal Tumour Surgery**

# J G A du Toit

It should once again be stressed that preoperative chemotherapy in the case of sensitive malignant tumours may eliminate small metastatic lesions from regional lymph nodes and from the lungs, making the definitive surgery treatment more feasible.

With the advent of MRI investigation, the staging of metastatic disease is made much easier. MRI investigation in the case of marrow infiltration by metastatic disease, as well as its placing in showing the soft-tissue extent of bone metastases, is of superior importance. Metastases of unknown primary origin continues to pose a perplexing problem which in spite of new modalities of diagnosis is no nearer to the solution. However, the exact staging of the tumour using all the diagnostic modalities is of great importance to select the cases for treatment with greater accuracy. This has led to not only understanding the problem better, but also to improving the figures.