CLINICAL PROTOCOL
ON
TRAUMA CARE

Karnataka Health Systems Development Project,
Bangalore
Government of Karnataka

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Foreword

Dr. S.N. Halbhavi, Specialist, District Hospital, Belgaum, conducted the first District Level Training Programme at District Hospital, Belgaum, to the group of Surgeons of Belgaum District from 10-03-1997 to 19-03-1997.

In this booklet, an attempt has been made to provide a diagnostic and management guidelines on Trauma care, which may be informative to all the Doctors taking care of Trauma patients in all the primary and secondary level hospitals.

With the present trend of increasing major road accidents, especially with the increased number of two wheelers on road, younger patients with major injuries and multiple fractures are reported to all casualty units of Govt. Hospitals, where a sound knowledge of either managing on modern lines or to refer to higher center without enhancing the damage to the existing conditions, is required. In this direction this booklet will be of some help either to the trainers who have to carryout the training programmes for the doctors working at Community Health Centres or to others who are facing the problems in their day-to-day life in hospitals. I would like to mention that 45 Trauma Centres are being established all over Karnataka to take care of the victims of Trauma especially of accidents.

I also request the clinicians to express their views and suggestions to improve upon the modalities involved in taking care of large number of Trauma victims.

Dr. S. Subramanya, IAS
Project Administrator &
E/o Additional Secretary to Govt.
Health & F W Department,
Bangalore.

PREFACE

I was informed by the Superintendent and District Surgeon, District Hospital, Belgaum, in the first week of March 1997, that I should conduct training programme to the group of Surgeons from Belgaum District. I had lot of apprehension at that moment, because the group consisted of Surgeons who were seniors and had plenty of practical experience and some were my contemporaries. This training programme was the after effect of my training in St. John Medical College, Belgaum, during the month of November '96. My sincere attendance and the notes that I had made during the training were of much help to me. Dr. S.L. Mokhashi, all the time encouraged me to go ahead and this was the source of inspiration to me.

The duration of the training was of 10 days from 10-03-97 to 19-03-97. I contacted the Senior Professors and Specialist in Anatomy, Surgery, Gasto-enterology, Dental & ENT Surgery in J.N. Medical College and District Hospital, Belgaum and arranged few lectures. Met Dr. Joshi, former Professor of Radiology and presently working in Disha Diagnostic Centre, Belgaum and arranged for lecturer and demonstration in Ultrasound Scanning. (I would succeed in getting the overhead projector and slide projector from the District Health Officer, Belgaum) The training consisted of pre-test on the first day, post-test on the last day of training and lectures by me and the invitee professors with discussion and visit to K.L.E. Hospitals and Disha Diagnostic Centre for observation in Endoscopy and Ultrasound respectively. The trainees also visited for 2 days Surgical Operation theatres of District Hospital, Belgaum, and observed various operations and anaesthesia procedures.

During the course of training, Dr. G.V. Vijayalaxmi, Additional Director, KHSDP and Dr. J.A. Krishna Swamy, Joint Director (Hospitals), KHSDP, Bangalore, visited the District Hospital and participated in the training and explained the themes of KHSDP. Her kind words of appreciation made me forget all the trouble that I had taken to organise the training programme and conduct it successfully.

On the last day of training, in the Valedictory function, the trainees expressed their satisfaction and told that they could learn so many new things, which was a very high moral boost to me.

After the training, I visited the KHSDP Office, Bangalore. I was introduced to ~~Dr.~~ Dr. Subramanya, Project Administrator, by the Additional Director. He gave me letter of appreciation and asked me to prepare the training module on Trauma, sharing my experiences. Thus, this booklet originated.

I prepared the notes topic-wise which I had taught in the training period and submitted to the Project Administrator. It took sometime to include the notes in the computer and copy was ready. This copy was handed over to Dr. Manjunath, Senior Specialist (Orthopaedic Surgeon, K.C. General Hospital, Bangalore), who scrutinized and suggested to add few chapters in Orthopaedics.

During the month of March 1998, I was deputed to training in Ultrasound and I took this opportunity to meet Dr. Vijayalaxmi. She readily handed over the entire file to me and requested me to go through the whole file and include whatever needed and feed in the computer. In the file I could find the typed notes on multiple fractures from Dr. Farooque, Professor and Head of Department of Orthopaedics, AIIMS, New Delhi. Some of the topics are included in this booklet. I hope this booklet will be best guide for Doctors who treat Trauma patients.

Dr. Subhash N. Halbhavi
M.S. (Gen. Surgery)
Specialist, District Hospital,
Belgaum.

Goals

1. To familiarise Surgeons and Casuality Medical Officers, the force acting in Trauma victims.

2. To anticipate and find out the injuries inflicted in traumatised patients.

3. To make ABC resuscitations as part of routine examination.

4. To diagnose correctly and TREAT or REFER the patient in the GOLDEN HOUR.

5. MINIMISE MORTALITY IN THE PRODUCTIVE AGE AND PREVENT DOWN FALL OF ECONOMY OF THE COUNTRY.

We thank

All the Officers and Staff who have contributed their time and energy in various ways in bringing out this book.

We acknowledge the services of

1. Teaching Faculty of J.N. Medical College, Belgaum.
2. Teaching Faculty of St. John's Medical College, Bangalore.
3. All India Institute of Medical Sciences, New Delhi.
4. Health Systems Project I, Andhra Pradesh and
5. Orthopaedic Department, K.C. General Hospital, Bangalore.

Dr. G.V. Vijayalakshmi,
Consultant,
KHSDP, Bangalore.

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TRAUMA AND ITS MANAGEMENT

In the present decade of this century, there is marked increase in the number of trauma patients, mainly due to Road Traffic Accidents, because of (1) rapid increase in the number of vehicles on the road and (2) introduction of high speed vehicles. The condition of existing roads is not good and there is no strict implementation of rules. Alcohol is also one of the major contributors.

These patients are mainly brought to the Causality Department of District and Taluka Hospitals and Major Hospitals and to out patient department of Primary Health Centres and Units. Here the causality Medical Officer is assisted by Staff Nurses and sometimes by a House Surgeon in the hospitals attached to Medical Colleges. He has two tasks to perform (1) To examine, assess and render emergency treatment and (2) to record the history and findings which may be required to be produced in the Courts of Law in future. Hence thorough knowledge of accident and emergency medicine is mandatory to these doctors.

After initial resuscitation, which is highly decision making to save the life of the patient, he may be treated in the same centre or may have to be referred to higher centre depending upon the existing condition of the patient.

Trauma is the leading cause of mortality and disability during the first 4 decades of life. It is the third most common cause of death overall. It has been calculated that more than 4 million trauma cases are hospitalised in the USA annually. In the United Kingdom there are 14,000 deaths annually due to trauma; of these 5000 to 6000 result from road traffic accidents. For each trauma death, there are more than 10 other persons that are seriously injured, and some of these are permanently disabled.

The direct cost to society in caring for the victims of trauma is enormous, for in many instances trauma affects young individuals, and their loss of productivity at work is immense.

MECHANISM OF INJURY

Trauma may be broadly classified into : (1) blunt or nonpenetrating and (2) penetrating varieties.

Injury occurs when tissues are deformed. The severity of the damage is related to the amount of energy released and the area over which it is applied.

In penetrating trauma of low velocity, the energy is usually focused over a small area, e.g. injuries caused by bullets from hand guns, or due to knives, sharp instruments, spikes of glass, wood or metal. However, in high velocity missile injuries, the energy may be dissipated over a wide area.

In blunt injuries, the damage may be caused by acceleration, deceleration, rotational or shearing forces. Victims of motor vehicle accidents may be injured by either rapid deceleration or by deformation of the vehicle itself. As deceleration occurs in a crash, the occupant's body is thrown against the interior of the vehicle, whereas the vehicular deformation is caused by inward movement of the shell, the person may be crushed and trapped inside. Ejection of an occupant may occur, in which case rapid deceleration of the body occurs when it strikes the ground or another vehicle.

When a pedestrian is struck by a moving vehicle, there is often an acceleration injury in addition to the direct trauma at the sites of impact.

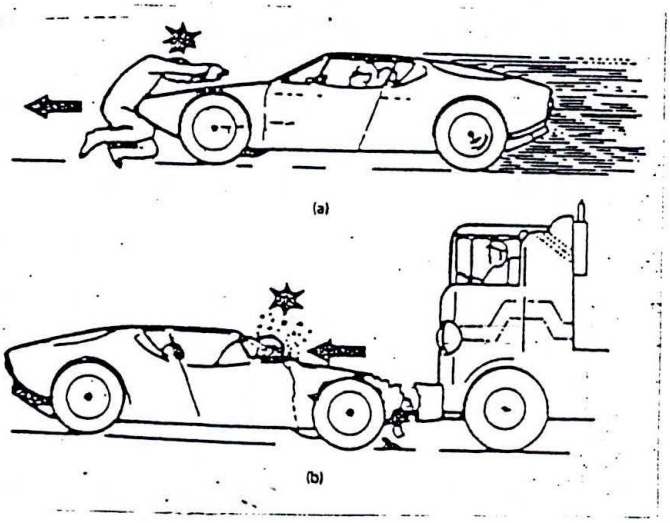


Fig 1: (a) A victim struck by a fast-moving vehicle suffers blunt trauma (*) and acceleration (→) injuries. (b) In head-on impaction type injuries, the victim suffers blunt trauma (*) and deceleration injuries (←).

Patterns of injuries in road traffic accidents :

While the variety of injuries that may occur in a road traffic accident is vast, there is a tendency for certain patterns of associated injuries to emerge. There may be a combination of head and cervical injuries; cervical whiplash and sternal injuries; sternal fracture and dorsal spinal injuries; fracture of the lower ribs and rupture of the spleen or liver; intra-abdominal and diaphragmatic injuries; pelvic fracture and urinary tract trauma and lower limb fracture associated with either dislocation of the hip or spinal fracture.

Disaster Planning :

Disasters may be classified as being either natural or artificial. Natural disasters embrace events such as earthquakes, typhoons and floods. Artificial disasters include major road, rail or air crashes, industrial accidents, rioting and explosions associated with civil disorder.

When a disaster occurs, large numbers of casualties are likely to arrive at hospital within a short period of time and may overwhelm the facilities and services available. It is therefore important to set priorities, not only in the management of the individual, but for organisation of care of the totality of the injured group.

Triage :

Triage means 'sorting'. The term was used during warfare when sorting out casualties at forward clearing stations but, nowadays is frequently applied to disaster situations. Triage may be necessary both in the field and in the hospital setting. In hospital triage may be used in three situations :

1. On arrival of patients at Causality Department.
2. In the x-ray department.
3. In determining priorities for operative intervention.

TRAUMA SEVERITY SCORES

Glasgow Coma Scale :

This scale relates specifically to head injury patients. The three components of this scale are as follows :

1. Eye opening.
2. Best verbal response.
3. Best motor response.

The coding for each of these is shown as follows :

Eye Opening	
Spontaneous	4
To voice	3
To pain	2
None	1
Verbal response	
Oriented	5
Confused	4
Inappropriate words	3
Incomprehensible sounds	2
None	1
Motor response	
Obeys command	6
Localises pain	5
Withdraws (pain)	4
Flexion (pain)	3
Extension (pain)	2
None	1
Total	**3 - 15**

Revised Trauma Score :

Of the many severity scoring system in use, the Revised Trauma Score as devised by Champion is widely accepted and applied. This incorporates the Glasgow Coma Scale. Variables on three organ systems that are vital to survival are thus combined into a single score as shown below. High scoring equates with prognosis.

Glasgow coma scale	*Systolic blood pressure*	*Respiratory rate*	*Points*
13 - 15	> 89	10 -19	4
9 - 12	76 - 89	> 29	3
6 - 8	50 - 75	6 - 9	2
4 - 5	1 - 49	1 - 5	1
3	0	0	0

Trimodal pattern of trauma deaths :

Trunkey has pointed out that death due to trauma fall broadly into three groups giving a distinct trimodal pattern.

1. Immediate deaths (50%) - those occurring within the first few minutes of injury and usually due to extensive trauma to the following :
    - brain or upper spinal cord,
    - the heart or major blood vessels,
    - rupture of major airway.

This first peak is due to injuries which are generally so lethal that little that can be done in their management that is likely to affect the outcome. Hence, it is only accident prevention or measures taken to reduce the severity of the injury, such as wearing of seat belts.

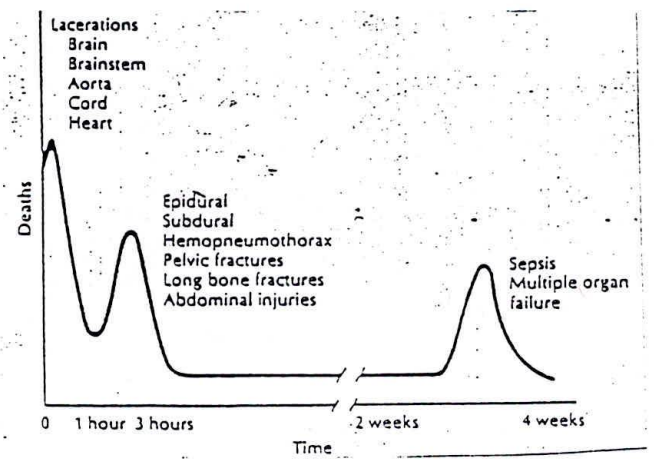


Fig 2: Trimodal patterns of death following trauma based on 1,500 autopsies.
(From Bailey & Love's Short Practice of Surgery)

2. Early deaths (30%) - those occurring within the first few hours after injury due to space-occupying collections of blood within the skull, or massive blood loss into the chest or abdominal cavities from major vessels and organ disruption, or into soft tissues from extensive fractures of pelvic or long bones.

3. Late deaths (20%) - those occurring some weeks after injury, generally due to sepsis and multiple organ failure. Organ failure may involve the heart, kidney,. liver, lung, brain and haemopoietic systems.

It is amongst those cases represented by the second and third peaks that potentially preventable deaths occur.

Pre-hospital management :

The aim should be for the rapid and smooth transfer or patients from the scene of the accident to a hospital.

Attention is paid to securing an adequate airway. Gloves are worn and a two-fingers 'sweep' is used to clear solid material from the mouth and pharynx combined with suction to remove fluid and debris. Patency is maintained by lifting the mandible forward and, if appropriate, inserting an airway. For emergency use, a mini-tracheostomy set is invaluable. Cervical spine should be protected by the use of a balloon, sandbags or forehead strapping. Other measures include ensuring adequate ventilation and oxygenation, covering and sealing open 'sucking' chest wounds, controlling external bleeding by direct pressure and monitoring the neurological status. If there is an obvious fracture of an extremity with gross deformity, the limb should be gently drawn into alignment as axial traction is maintained. As axial traction is maintained, the fracture is splinted.

If delay is anticipated intravenous fluids may be administered.

Hospital Care :

A team approach by those trained in trauma management, preferably by an Advanced Trauma Life Support (ATLS) course, is ideal. The aim of this course, which was developed by the American College of Surgeons and is now being introduced in many centres around the UK and elsewhere, is to instruct doctors in the recognition of problems and in the provision of expert resuscitative measures during the 'golden hour' following trauma. Since the severely injured patient has many problems, swift and co-ordinate management requires a team approach. Factors indicating that a patient has a high risk of multiple injuries are outlined below : (After Champion)

1. A penetrating injury to the chest, abdomen, head, neck or groin.
2. Two or more proximal long bone fractures.
3. Burns > 15% combined with facial injury or airway problems.
4. A flail chest.
5. Evidence of a high energy impact :

- Falls of 20 feet or more.
- Change in velocity in crash of 20 m.p.h. or more estimated from outward deformity of car.
- Rearward displacement of front axle.
- Sideward instrusion of 35 cm or more on the patient's side of the car.
- Ejection of patient.
- Rollover.
- Death of another person in the same car.
- Pedestrian hit at more than 20 m.p.h.

Primary survey and resuscitation :

The initial evaluation of the patient proceeds in parallel with the ABC of resuscitation, i.e., *airway, breathing, circulation*, which must be attended to in that order. Endotracheal intubation may be required depending on the local anatomical injury and the level of consciousness. It is important to check up by inspection and auscultation that both sides of the chest are being ventilated and oxygenated. Blood is taken for haematocrit estimation, blood grouping and cross-matching, electrolytes and urea analysis and blood gas analysis. Mini-tracheostomy may be required.

Two peripheral intravenous lines using size 14 cannulae are set up. Central venous cannulation and bladder catheterisation are undertaken to aid in the control of fluid balance. Vital signs are measured and recorded. A rapid initial assessment of the brain and spinal cord is made by requesting the patient (if conscious) to put out the tongue, move the toes and squeeze the examiner's fingers.

Secondary survey :

The adequacy of resuscitation is judged by the patient's clinical response.

Increasing tachycardia, decreasing blood pressure and increasing pallor suggest continuing blood loss into one of the body cavities.

In order to carry out a detailed head-to-toe examination the patient should be completely disrobed. It may be necessary to cut off the clothing to avoid undue disturbance of the patient.

Head *:* A thorough check is undertaken for external signs of injury such as bruising, lacerations or bony deformity. Depressed skull fractures may or may not be palpable. Deterioration of the neurological status may indicate rising intracranial pressure, or it may be due to hypoxia and hypoperfusion. If a patient with closed head injury becomes hypotensive, a careful search should be made for blood loss with particular attention to the abdominal and thoracic cavities. Hypoxia and hypercarbia both tend to compound the initial brain damage.

The nostrils and external auditory meatus are examined for any evidence of rhinorrhea or otorrhoea. It should be remembered that the cerebrospinal fluid emerging from these orifices is

mixed with blood and produces a double ring if dropped on a sheet. Clotting is also delayed by the presence of the CSF fluid.

Face : The eyes are checked for the presence of foreign bodies, perforation, visual acuity, pupillary reflex and corneal reflex. Subconjunctival haemorrhage may suggest basal skull fracture. An assessment is made for the stability of the mandible. Fracture of the bones of the middle third of the face with displacement can compromise the airway, which may be improved by pulling the mobile segment forward. The mouth is checked for retropharyngeal haematoma and foreign bodies.

Neck : Palpate for subcutaneous emphysema. The cervical spinous processes are carefully palpated for any 'step' deformity. Examination of the neck may reveal venous distension or tracheal deviation. A lateral x-ray showing all seven cervical vertebrae is essential in patients with multiple trauma. Care must be taken to avoid lesions at C_1, C_2 and C_7 levels going undetected. Downward traction on the arms while the film is being taken will enhance the demonstration of the lower cervical vertebrae.

Thorax : Inspection of the anterior and posterior chest wall is undertaken to check for any penetrating or sucking chest wound, or for paradoxical movement of a flail segment. Impact against an automobile steering wheel may have caused sternal fracture and cardiac contusion. Palpation of the ribs reveal tenderness or crepitus in the case of fractures. If the lower ribs are fractured, the possibility of intra-abdominal organ injury, esepecially hepatic or splenic, must be borne in mind.

Percussion and auscultation may confirm the presence of pneumothorax or haemothorax. Tension pneumothorax needs immediate release. This can be achieved in the first instance using a large-bore intravenous cannula inserted in the 2nd intercostal space in the midclavicular line on the affected side. Subsequently a 32 French gauge chest drain can be inserted just anterior to the midaxillary line in the 5th intercostal space. Likewise, an open chest wound or haemothorax will require the insertion of a chest drain. While all patients who sustain severe blunt trauma require chest x-ray, this may have to be deferred until life-threatening priorities have been dealt with appropriately.

Penetrating chest wounds may injure the heart. Not uncommonly, the patient reaches hospital alive, especially if the lesion involves the right side of the heart. Distant heart sounds, distended neck veins, a rapid, thready pulse and falling blood pressure may herald the development of cardiac tamponade. Rapid deceleration as a result of a car crash or fall from a height may result in tearing of the thoracic aorta.

Abdomen : The use of a nasogastric tube reduces the risk of aspiration of regurgitated gastric content, and is prophylactic against acute gastric dilatation. The abdominal wall and back should be carefully inspected for signs of injury or ecchymosis. Any laceration is covered with sterile gauze. Eviscerated bowel is covered with a large sterile pack soaked in warm saline. Pain in the shoulder during inspiration may suggest subdiaphragmatic irritation from blood or leaked gastrointestinal content. Palpation of the abdomen may reveal abdominal distension and

voluntary guarding. Blunt abdominal trauma may produce severe intraperitoneal and retroperitoneal injuries with relatively few physical signs. Diminished or absent bowel sounds may also be due to peritoneal irritation from intestinal contents. However, at the initial examination, bowel sounds may still be audible in up to 30 per cent of cases with significant intra-abdominal lesions.

Initial clinical assessment of the abdomen in blunt trauma is accurate at best in only 70-80 per cent of cases. Accuracy is likely to be less in patients with multiple trauma or concomitant head injuries, or altered levels of consciousness due to alcohol or drugs. Diagnostic peritoneal lavage is particularly helpful in patients with an altered level of consciousness from head injury, alcohol intoxication or drug ingestion, in whom clinical assessment of the abdomen may yield few clinical signs. False negative results may be obtained in patients with extraperitoneal injuries to duodenum, pancreas, bladder or colon.

It must be stressed that extensive investigations should not be undertaken at the expense of resuscitation. In some critically ill patients, urgent resuscitation and, in some cases, emergency laparotomy must take precedence over investigative procedures.

Pelvis : The pelvis is compressed and distracted manually to check for pain enhancement and pelvic stability. Blood at the penile meatus may indicate urethral damage, either partial or complete, and no attempt at urethral catheterisation should be undertaken. If there is suspicion of urethral injury and a urethrogram has not yet been undertaken to clarify the situation, the suprapubic placement of a urinary catheter is undertaken. Following severe blunt trauma, the pelvis should always be x-rayed.

Spinal injuries : Tests are made for peripheral sensory and motor defects. In spinal injuries with unstable fractures, further neurological damage can be caused by moving the patient inappropriately. However, if no spinal cord injury occurred as a result of the initial trauma when forces at the moment of impact were greatest, then the patient can be carefully log rolled to examine the back.

Extremities : Extremities should be inspected and examined for signs of trauma. A check is made for the presence of peripheral pulses and also that the nerves are intact. If fractures are suspected, x-rays are taken while maintaining splintage.

As part of the early management of the traumatised patient, consideration may have to be given to the need for pain relief, antibiotic administration and tetanus prophylaxis.

TEACHING PROGRAMME FOR GENERAL SURGEONS

Topic : Diagnosis and Management of Head Injuries

1. Define Concussion / cerebral contusion
   compound depressed fracture
   Acute subdural haematoma
   Complications
2. How to take history
3. To assess level of consciousness
4. How to diagnose each as one (1)
5. To judge who needs surgical intervention
6. How to transport
7. How to write reference letter
8. Prevention

NOTE : Doctors working below 100 beds.
Ref : C.G.S. less than 13 to higher centres.

HEAD INJURY

	Objectives		**Contents**	**Activities and AIDS**
1.	An account	(a) Modes of Transport	RTA	(a) Airwary Metal
	of accident		FALL	(b) Endotracheal tube
			ASSAULT	(c) X-rays
			MISSILE	(d) C.T. Scan
			CALAMITIES	(e) Craniotomy Set
		(b) Time & Date		(f) Fibre Optic Head Light
		(c) Conscious	No Loss	(g) Suction Apparatus
			Loss	(h) Bipolar Coagulator
			Lost but recovered	(i) Cervical Collar
			Continued to deteriorate	(j) Thoraco-lumbar Brace
		(d) Vomiting		(k) Knee Hammer
		(e) Seizures		(l) A/v. Aid
		(f) Increased Headache		(m) O.H. Projector
		(g) ENT bleeding		(n) Slide Projector
		(h) Alcohol		(o) View Box
		(i) Helmet		(p) Torch
2.	Diagnosis	(a) Concussion		
		(b) Cerebral Contusion		
		(c) Extradural Haematoma		

		(d) Acute Subdural Haematoma		
		(e) Intra-Cerebral Haematoma		
		(f) Compound depressed Fracture		
3.	Transport	(a) Semi-prone Position		
		(b) Immobilization of neck with Cervical Collar		
4.	Examination	(a) Air way		
		(b) Breathing		
		(c) Pulse / BP		
		(d) Assessment of Consciousness by G.C. Scale		
		(e) Pupillary changes		
		(f) Focal deficit, sensory level		

CRANIOCEREBRAL INJURIES

Introduction : In most countries, the primary and continuing care of patients who have sustained a head injury is the responsibility of general and Orthopaedic surgeons rather than neurosurgeons. Despite the expansion of neurosurgical services, this situation is likely to continue and therefore the general surgeon requires a knowledge of principles and practice in this field. Since the majority of patients do not require any neurosurgery, the major steps in the care of such patients are medical, diagnostic and nursing, and it is these steps which will determine the outcome far more frequently than any surgical manoeuvres. Indeed, the avoidance of unnecessary cranial surgery is an important principle, because such surgery may be harmful. At the same time the general surgeon must be aware of the indications for specialised intracranial investigation, which usually requires transferring the patient to a neurosurgical department. Finally, he or she must be able to decide when there is a clear indication for urgent surgery without specialised investigation, either because the rate of deterioration does not allow any further delay, or because the facilities for intracranial investigation do not exist. Thus, in many parts of the world, patients must still be managed without CT scanning, although this situation should become less common in future.

Injuries of the brain

Primary Brain Injury (Type I)

a. Due to movement of the brain within the skull box causing shearing forces due to deceleration and acceleration. Ex. Road traffic Accident - when the moving head strikes against the immovable object like foot path or road.

b. In acceleration injury the stationary skull is struck by a moving object. Ex. Assault in which the brain accelerates against the stationary mid brain.

Head Struck from front	**Head Struck from behind or in front at one side or the other**	**Head struck far away from site of blow to the head**
In this both the cerebral hemispheres are displaced posteriorly against static mid-brain, Hypothalamus, Brain-stem	In this one cerebral hemisphere moves against the other so that junctional tissues of commissures and corpus collasum are damaged.	In this grey matter of cortex moves over the white matter leading to tear of nerve cells and axon fibres of that region. Injury is of diffuse in nature.

Secondary Brain Leision (Type II)

It is due to localised injury to the brain against rough internal configuration of skull and flax tentorium. When the internal portion of the skull is rough, damage to the brain is more and when it is smooth damage to brain is less

Temporal lobe of the brain	Damaged against Sharp edged sphenoid ridge.
Frontal lobe (Inferior surface)	Damaged by rough surface of anterior cranial fossa floor.
Occipital lobe	Damaged due to injury to Flax - Tentorium.
Corpus callosum	Damaged against sharp edge of Falx.
Cerebral Peduncle	Damaged by sharp edge of tentorium.

TYPE OF BRAIN DAMAGE IS INDICATED BY THREE TERMS

1. ***CEREBRAL CONCUSSION:***

It is a brief physiological paralysis function of the brain without structural organic brain damage.

Clinical Signs and Symptoms:

1. Transient loss of impairment of consciousness.
2. Dizziness or mild confusion followed by complete recovery. It may be short losting from one minute to ½ an hour or may be long lasting prolonged for hours or even a day.
3. Patient looks pale, pulse feeble, Temperature increases and later on pulse becomes full and bounding.

Cause for loss of consciousness:

Cerebral Anemia is due to compression of the brain causing emptying of vessels or abrupt stretching of anterior and posterior perforating arteries causing transient ischemia of brain.

2. **CEREBRAL CONTUSION:**

In this there is damage to nerve cells and axons with bruise and swelling of cortial gyri, Brain odema and congestion contribute to swelling of brain.

3. **CEREBRAL LACERATION:**

In this brain surface is torn or lacerated with effusion of blood into CSF fluid. Often laceration is most severe on the side opposite to that on which the blow is struck. This condition is known an contracoup injury.

Changes in the brain:

Damage to the nerve cells and axons causes brain odema which leeds to giosis and atropic changes in the brain. The damaged portion of the brain shrinks and fluid accumulates in the subarchanoid space. Therefore it causes mental impairment. After some years scar formation and later on causes epilepsy (Traumatic epilepsy of Jaksonean type)

CEREBRAL IRRITATION

It may occur within 48 hours of injury.

Signs and symptoms:

1. Patient curls up on his side with knee drawn up and arm flexed (an attitude of flexion)
2. Avoids light, interference, No interest in surroundings.
3. Recovery is complete.

Patient complaints on headache, irritability, depression, lack of concentration, defective memory and change of personality.

Aetiology - is due to localised odems which occurs around the area of contusion or laceration in brain.

SEQUALE OF CEREBRAL CONTUSION OR LACERATION

1. Post traumatic amnesia (PTA). This is the time between the head injury and return of continuous memory.

    - If it is less than 1 hour, injury is regarded as slight.
    - Between 1 and 2 hours injury is regarded as moderate.
    - Between 1 and 7 days injury is regarded as severe.

2. Retrograde traumatic amnesia (RTA). This means loss of memory of events before the occurrence of accident.
3. Cerebral Irritation - this is called as post contutional syndrome (after recovery patient complaints of headache, photo phobia, nausea, vomiting, depression, delerium or lack of concentration).
4. Traumatic Epilepsy.
5. Permanent damage to an improtanf focal centre of cerebral cortex.
    - Anosmia - injury to the inferior aspect of frontal lobe.
    - Hemianopia, Blindness - occipetal lobe lesion.
    - Ataxia and Nystagnus - cerebellar lesion.

6. Cerebral compression features due to

    - Acute extra dural hematoma of arterial origin.
    - Acute extra dural hematoma of venus origin.
    - Acute sub dural hematoma.
    - Acute intra cerebral hematoma.

Mechanism and pathology :

At the moment of impact, a diffuse neuronal lesion is inflicted on the brain, which is responsible for the immediate clinical picture of brain injury. Secondary changes of brain swelling or intracranial haemorrhage take time to develop. The rise in pressure resulting from these causes leads to a deterioration in the patient's level of consicousness a few hours after injury; the clinical picture in the early stages results from the neuronal lesion alone. All degrees of brain injury resulting in loss of consciousness, concussion, contusion, or laceration of the brain are produced by one mechanism, namely displacement and distortion of the cerebral tissues occurring at the moment of impact.

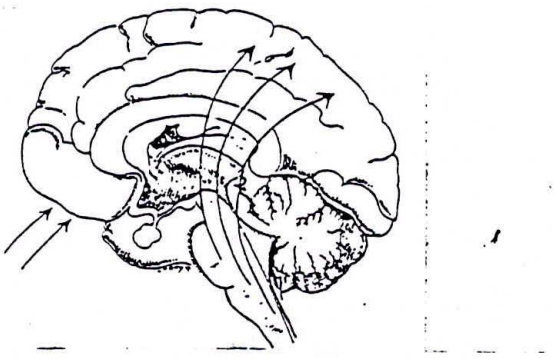


Fig 3: Lines of force acting on the hypothalamus and brain stem as the result of posterior displacement of the hemisphere.

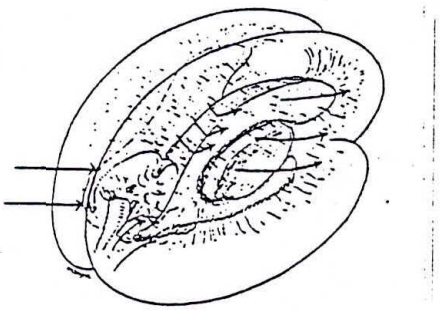


Fig 4: Lines of force acting on the corpus callosum and one peduncle as the result of posterior displacement of one hemisphere.

Early secondary pathology

1. Intracranial factors :

Brain swelling : The brain often reacts to any insult, be it ischaemic, neoplastic inflammatory or traumatic, by swelling due to oedema. The accumulation of fluid is both extracellular and intracellular. Macroscopically oedema may be localised at least initially to the part of the brain most damaged, but it may extend rapidly throughout one or even both cerebral hemispheres, causing a severe rise in intracranial pressure and, at an earlier stage, features of a relatively localised space-occupying or compressing lesion which may mimic a compressing haematoma.

Brain necrosis : This is seen most frequently in the so-called burst temporal lobe syndrome. By a combination of swelling (oedema and venous engorgement), ischaemia leading to haemorrhagic infarction, and necrosis, there may be localised mass occupying the anterior and middle parts of one of the temporal lobes; similar changes may also occur in one or both anterieor poles of the frontal lobe. This produces all the clinical features of a lateralised supratentorial mass lesion, usually presenting some hours or even a few days after injury, and may require urgent craniotomy and temporal or frontal lobectomy.

Haematoma : The surgically remediable intracranial haematomas may be extradural, subdural or intracerebral. They may be the result of arterial or venous bleeding, and therefore vary in their time of presentation in relation to the injury. The deeper the haematoma, the more likely it is to be associated with primary brain damage, oedema, and necrosis and therefore the results of surgical evacuation are poorer. It is rare to have an intracerebral haematoma due solely to the rupture of one major vessel; usually such haematomas are due to the tearing of many small vessels, and it is often difficult to determine the relative part played by the haematoma in the clinical problem.

Vascular changes : Consequent to rising intracranial pressure, disturbance in cerebral blood flow may occur which causes further brain damage through ischaemia, and the ischaemia itself may produce an increase in brain oedema. In normal life there are autoregulatory mechanisms, which to some extent protect the cerebral circulation despite changes in blood pressure and intracranial pressure. When the intracranial pressure rises severely, the autoregulatory mechanism fails and irreversible ischaemic changes occur. In addition, following brain injury there may be dilatation and engorgement of the venous system which will further increase the brain swelling, and therefore the intracranial pressure, and increase the brain oedema by impeding the normal drainage of tissue fluid and worsen the cellular ischaemia.

Coning, or herniation of the contents of the supratentorial compartment through the tentorial hiatus, or the contents of the infratentorial compartment through the foramen magnum, may accompany a rise in pressure in the appropriate compartment. The pathological sequelae at the tentorial hiatus include herniation of the medial part of the temporal lobe (the uncus) on the side of the supratentorial mass, which causes pressure upon the ipsilateral 3rd cranial nerve, and upon the midbrain. The midbrain is distorted and displaced away from the side of the mass, where the free edge of the tentorium indents or 'notches' the cerebral peduncle and interferes with the descending motor pathways from the hemisphere opposite to that which is being compressed. In addition, the compression of the midbrain contributes to obstruction of the CSF flow in the aqueduct, which contributes further to the rise of intracranial pressure. These changes produce a deterioration in the level of consciousness, dilatation of the pupil on the side of the compressing mass, and a hemiparesis on the same side as the mass. This situation is called 'Kernohan's notch'.

Coup and contre-coup : These words are used to indicate the types of craniocerebral damage which may occur either on the side of the blow to the head (coup), or opposite (and often diagonally opposite) to the position of the blow (centre-coup). Provided it is clear where, and where alone, the head was struck, this knowledge can provide a useful guide to pathology when used in conjunction with the lateralising signs of a compressing lesion (see Coning, above).

2. Extracranial factors :

Respiration : Respiratory failure or inadequacy is one of the most potent factors which can aggravate or precipitate the development of severe brain oedema and venous congestion, leading rapidly to irreversible changes. Of the two factors, the arterial pO_2 and pCO_2, a rise in pCO_2 is the more potent. It is therefore essential that pCO_2 and pO_2 are maintained at normal

levels in any patient who has sustained a severe head injury. Although at times hyperventilation has been employed in order to reduce intracranial pressure, abnormally low pCO_2 levels may cause a reduction in cerebral blood flow and subsequent cerebral ischaemic damage, and it is therefore safer to achieve normal levels.

Systemic blood pressure and blood volume : When the brain is at risk from ischaemic damage following injury it is obviously essential that a normal cardiac output is maintained and that blood loss from other injuries is adequately replaced. Recent work has confirmed that when the intracranial pressure is high, protective cerebral blood flow regulatory mechanisms are impaired and a fall in cardiac output is particularly likely to result in irreversible cerebral ischaemic damage.

Fluids : Although the onset of brain oedema is usually the result of the primary brain injury and ischaemia, the state of the vascular compartment will influence its course. Thus, if hypotonic fluids are given intravenously, the plasma osmolality may be lowered, particularly if there is any delay or defect in renal excretion of the excess 'water', and therefore by an osmotic effect fluid may be drawn into the brain tissue and brain swelling increased. With disturbance of the normal blood-brain barrier (which is poorly understood), these effects may be increased. Therfore intravenous fluids should be isotonic, and the volumes used (apart from replacement of blood loss by blood) should be dictated by normal requirements and renal function. Problems with fluid and electrolyte balance may be exacerbated by inappropriate secretion of pituitary antidiuretic hormone as a cerebral response to the cerebral injury. This may produce dangerously low levels of plasma sodium.

Temperature : When the body temperature rises, the metabolic demands of all tissues rise and the accumulation of catabolites increases. When cells or particular groups of cells are damaged, increased metabolic demands will aggravate the cellular failure or aggravate the manifestations of poor function. This is well seen following severe head injury when a rise in body temperature will cause a further deterioration in neurological state.

THE ACUTE COMPRESSING INTRACRANIAL HAEMATOMAS

These are (1) acute extradural haematoma, (2) acute subdural haematoma, and (3) acute intracerebral haematoma.

Acute extradural haematoma : The majority of cases of acute extradural haematoma are due to an injury associated with fracture of the temporal or parietal bone whose bleeding results in an extracranial extravasation producing a boggy swelling deep to the temporal muscle. Although intracranial haematomas are more common (x30) when a skull fracture is present, a remediable extradural haematoma may occur in the absence of a fracture.

The classical syndrome of extradural haemorrhage results from injuries of the anterior or posterior branches of the middle meningeal artery. In such cases, the injuring force, which comes from a lateral direction, is often relatively trivial, such as a blow from a golf or cricket ball which strikes the thin bone of the temporal plate, inflicting a fracture which drives the dura

inwards and tears a meningeal artery or vein. In the elastic skull of children, the injury may occur without fracture, as it may, (but less commonly), in adults.

From the torn vessel blood passes in three directions :

1. Outward through the fracture to form a boggy swelling under the temporal muscle, the finding of which is an additional indication for the admission for observation of a conscious patient.
2. Downwards into the middle fossa.
3. Upwards over the parietal region.

The development of the extradural compressing mass depends upon the ease with which the dura can be stripped away from the inner surface of the skull. With advancing age the dura becomes more adherent to the skull and, with the 'elasticity' of the skull, is one factor in the greater incidence of extradural haematoma in children and young adults. During the 'lucid' interval (below) the haematoma gradually enlarges and it is not until it has reached a size sufficient to cause a severe rise in intracranial pressure and critical distortion of the midbrain at the tentorial hiatus, that the conscious level deteriorates, often rapidly, with dilatation first of the pupil on the side of the haematoma, then of the contralateral pupil as the stage of decerebrate rigidity is reached.

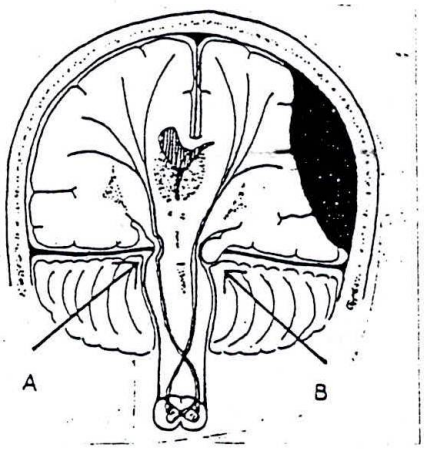


Fig 5: Acute extradural haematoma. (A) Impaction of the opposite crus against the opposite rim of the tentorial opening. (B) Displacement of the inner edge of the temporal lobe (uncus) descending into the tentorial opening (midbrain cone).

Clinical features : Usually, a laterally directed blow of small magnitude causes a short initial period of concussion, followed by a characteristic **lucid interval**, during which the haematoma is collecting intracranially and also forming a swelling under the temporal muscle. An example is an unconscious footballer who has carried off the field but later recovered sufficiently to finish the match; headache and drowsiness supervened so that he retired to bed early and was later found dead from middle meningeal haemorrhage.

Although the 'lucid' interval is one of the 'classical' features of an acute extradural haematoma, the 'lucidity' may be only relative compared to the subsequent deterioration. In these circumstances, the clinical diagnosis is less obvious, and the consequent delay in diagnosis

may reduce the prospect of a favourable outcome. The practical difficulty in such cases is to decide what degree of primary cerebral injury may be present with persisting low level of consciousness, but nevertheless an acute extradural haematoma may yet supervene. It is the awareness of this possible 'nonclassical' presentation which has prompted the move towards greater use of the CT scan.

The next change is in the level of consciousness. The patient becomes confused and irritable, and at this stage is in danger of being arrested if found wandering and smelling of alcohol; therefore, beware of the patient brought to casualty in a confused state with a bruise in the temporal region. Persons alleged to be found drunk have been locked in cells only to be found dead in the morning, the unconsciousness and stertorous breathing of compression being mistaken for a drunken stupor. Confusion changes to drowsiness, and there may be evidence of a contralateral hemiparesis due to direct pressure on the cerebral cortex. At the same time, inward displacement of the temporal lobe causes the inner portion of the lobe to press against the 3rd nerve above the edge of the tentorium, causing constriction (rarely observed), rapidly followed by dilatation of the pupil on the side of the haemorrhage. If the pressure is not relieved, displacement of the brain stem at the tentorial opening forces the opposite crus against the rim of the tentorium, producing a hemiparesis, which this time occurs on the same side as the haematoma.

Extradural haemorrhage of venous origin is produced by fractures which injure the major sinuses or by tearing of a meningeal vein. Injury of the superior longitudinal sinus produces a massive subgaleal haematoma, together with evidence of clot compression, causing rapid deterioration of consciousness with unilateral or bilateral leg weakness if the clot is over the upper end of one or both motor cortices.

Acute subdural haematoma : Following severe head injury there is commonly a thin layer of clot over the brain in the subdural space. This does not usually constitute a significant compressing lesion, which is more likely to be produced by the underlying cerebral swelling and contusion. A significant subdural haematoma is produced either by rupture of a large cortical vein as it crosses the potential subdural space to reach the fixed dural venous sinuses or by laceration of the cortex and subsequent venous or arterial haemorrhage.

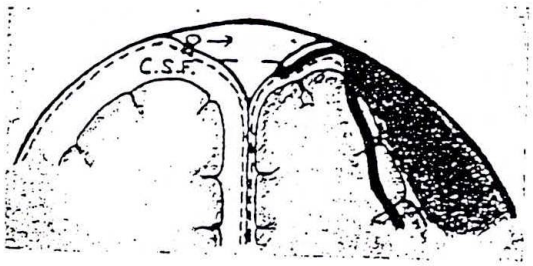


Fig 6: Position of a subdural haematoma produced by tearing of the superior cerebral veins at the level of the arachnoid. The diagram also shows the partial or occasionally complete septum which divides the superior sinus. Cerebrospinal fluid drains into the upper compartment through the arachnoid granulations. The superior cerebral veins drain into the lower. (G.B. Northcroft, FRCS, London.)

The presentation and management of acute subdural haematoma may be similar to that of acute extradural haematoma, but tend to differ in certain respect.

1. There is often severe primary brain damage (e.g., laceration) and therefore initial and persisting loss of consciousness. A truly lucid interval is unusual.
2. Deterioration tends to occur sooner than in extradural haematoma.
3. The haematoma may be 'coup' or 'contre-coup'.
4. The haematoma is often extensive and may cover an entire hemisphere, extending both subfrontally and subtemporally.
5. Effective surgical action is unlikely without special investigations (CT scan or arteriogram, see below).
6. Effective surgery involves an extensive craniotomy; even then the results are poor due to the primary and often widespread brain damage and brain oedema.

When the presentation is less dramatic and delayed by several days (so-called subacute subdural haematoma) it is very difficult to determine the part played by the haematoma as opposed to the underlying brain damage and especially increasing brain oedema. Without the CT scan surgical opinions are frequently without foundation, and surgery ineffective.

7. Very rarely a large acute subdural haematoma may occur in the elderly following a trivial injury or shaking of the brain with rupture of a 'bridging' vein.

Acute intracerebral haematoma : This is the least common of the siginificant and remediable compressing intracranial haematomas. It is rare for a major intracerebral vessel to rupture as a result of trauma and produce an expanding haematoma that is the sole cause of cerebral compression. When there is totally lucid interval before such an event, it is more likely that haemorrhage has occurred from a pre-existing vascular lesion such as an aneurysm or arteriovenous malformation. If time permits, arteriography before surgery is advisable.

Usually, traumatic intracerebral haematomas are associated with cerebral laceration, contusion, oedema and necrosis, the compressing lesion being formed by all these elements. Therefore in these circumstances the benefit of removing the clot is unpredictable and often disappointing. Occasionally, the removal of such traumatic masses in the anterior part of the temporal lobe or in the frontal pole may be effective, but preoperative diagnosis with CT scan or arteriogram is essential.

CLINICAL DIAGNOSIS AND MANAGEMENT

Primary and later neurosurgical management must include the following, which will be considered in clinical sequence :

1. Casualty reception.
2. Indications for admission.
3. Radiography.
4. Continuing care and observations.
5. Deterioration.
6. Indications for surgery without special investigation (decision making).
7. Special investigations.
8. Nonsurgical therapy : osmotic diuretics, steroids.
9. Miscellaneous early complications.

1) Casualty reception : The reception of the severely injured patient is a large subject which has already dealt with. In the following paragraphs, the essential points relate to patients whose main reason for admission is a major head injury. There are certain general principles :

(a) Patients are more likely to die from airway obstruction than from any remediable intacranial lesions.

(b) Surgically remediable intrathoracic and intra-abdominal lesions take precedence over any intracranial procedures, as does general resuscitation and correction of blood loss. It is dangerous to take decisions above active (especially surgical) intracranial management before this has been done.

(c) In normal civilian practice patients are brought rapidly to a casualty department, therefore compressing intracranial haematomas are unlikely to present when the patient is first seen.

(d) The initial clinical assessment and its recording, especially of the level of consciousnes, is of crucial importance if any later deterioration is to be gauged accurately.

(e) The immediate institution of the correct care of the unconscious patient does more to lower the morbidity and mortality from major head injury than any other single measure.

In receiving and assessing a patient in casualty the essential steps in chronological order can be summarised as follows :

(i) Protection of the airway : All the usual measures for the protection of the airway in an unconscious patient should be taken immediately; removal of false teeth, positioning prone or on one side with the head low, mouth suction and insertion of a pharyngeal airway. The presence of a compound skull fracture with exuding brain tissue is not a contraindication to lowering the head. In the majority of patients, these measures will suffice. However, if they do

not a cuffed endotracheal tube should be inserted, which is in itself a test of the need for that manoeuvre.

Specific indications for endotracheal intubation are :

(a) Absence of gag reflex.
(b) Maxillofacial injury; orophrayngeal bleeding; facial or oropharyngeal burns.
(c) Uncleared vomitus and / or the need for controlled ventilation.

(ii) General assessment : The general assessment is often more important than the neurological because intrathoracic and intra-abdominal injury may require immediate action. These aspects are dealt in the preceding section, but it should be stressed that the picture of 'surgical shock' should not be ascribed to the head injury unless the presence of another major injury has been excluded.

(i) Neurological assessment :

(a) ***Level of Consciousness :*** This is the single most important neurological observation, and one that causes greatest difficulty in accurate description and recording. Terms and gradings which require definition (e.g., 'coma' or 'stupor') should be avoided. A brief description in simple words which include the degree of alertness, ability to communicate or obey commands, and type of spontaneous or reflex limb movements is often satisfactory. The Glasgow Coma Scale is a valuable method of recording consecutive observations, and of observing progressive changes but without requiring previous definition of terms used.

(b) ***Pupils :*** When both pupils are small on admission and later one dilates, that provides the most accurate clinical guide to the lateralisation of a supratentorial compressing lesion. If, however, one pupil was known to be dilated immediately after the injury it is usually due to direct injury related to the orbit, and neither pupil can be used later as aguide to lateralisation. Therefore the first assessment and recording of pupillary size is of extreme importance; size and equality rather than reaction to light is the more important feature, and should be recored graphically or verbally.

(c) ***Limbs :*** The types of spontaneous or reflex limb movements are an essential part of the assessment of the level of consciousness (see above). Hemiplegia is as likely to be due to a 'stroke' prior to injury as to trauma; in the acute state it is very rarely due to a compressing intracranial haematoma, but may be due to the primary cerebral damage. Alone it is never an indication for urgent surgery. Of more immediate significance is the direction of paraplegia or quadriplegia indicative of a spinal injury.

(d) ***Cranium :*** The position and nature of the external trauma should be noted accurately, the wound over an obvious fracture should be cleaned with saline and surrounding hair removed for a distance of at least 5 cm, and a head dressing applied. The presence of CSF rhinorrhoea, or otorrhoea in noted, and special note made of any mandibular or facial fracture which might lead to further difficulties with the airway.

(e) ***History of injury :*** With the severely injured the initial assessment and resuscitation is usually done before a history of the injury can be obtained. However, that information is important, particularly if a preceding medical condition has led to the injury. Information from witnesses, ambulance attendants and relatives should be sought. Alcohol poses particular problems, which are often greater in those who have been less severely injured, and it makes accurate assessment of neurological lesions more difficult, and may be misleading when deterioration in conscious level due to an intracranial haematoma occurs.

2. Indications for admission : Following head injury, patients are admitted to hospital for two main reasons : (1) For continuing hospital care for those who have suffered serious injury and who are in obvious need of care and possibly surgery. (2) For observation in case a complication should develop, despite the patient being perfectly well at the time of admission. The first category presents little difficulty about the indication for admission, but the second presents considerable difficulty. Remediable complications, e.g., acute extradural haematoma, may occur following very trival injury, but there is good evidence that they are more likely to occur after transient change in consciousness, when a skull fracture is present and when the patient complains of persisting headache. However, although the incidence of haematoma is very low in the absence of these features, it is currently a matter of debate whether transient clouding of consciousness alone should remain an indication for admission. Accepting that there are these grounds for debate, the following are reasonable guidelines :

(a) ***Indication for skull radiographs after recent injury :***
- (i) Loss of consciousness or amnesia at any time.
- (ii) Focal neurological symptoms or abnormal signs.
- (iii) Suspected penetrating injury.
- (iv) Scalp bruising or swelling.
- (v) Alcohol intoxication.
- (vi) Difficulty in assessing the patient.

(b) ***Indications for admission :***
- (i) Any degree of depression of level of consciousness on examination.
- (ii) Skull fracture.
- (iii) Focal neurological signs.
- (iv) Persistent headache or vomiting.
- (v) Other medical conditions such as patients on anticoagulants, haemophilia.
- (vi) Alcohol intoxication.
- (vii) Circumstances of injury unknown.
- (viii) Crime.
- (ix) Absence of responsible relatives or friends.

If the absence of a skull fracture is used as one of the reasons for not admitting a patient, the radiographs must be of acceptable quality. If patients are not admitted, a responsible relative or friend should be given written instructions about the possible complications and appropriate action.

The duration of observation is arbitrary, but since it is exceptional for acute extradural haematoma to present more than 18 hours after injury, overnight admission is usually sufficient. In general, children should be admitted even more readily than adults because of the increased incidence of extradural haematoma. Alcohol intoxication can lead to great difficulties because it can mimic or mask the signs of intracranial haematomas to which alcoholics are generally more prone.

3. Radiography : The purpose of skull radiographs includes :

(a) The demonstration of a skull fracture whose position may provide a valuable guide to the site of an extradural haematoma which might develop later.

(b) The presence of a skull fracture provides some indication of the severity of the injury, and is one of the indications for admission.

(c) The demonstration of a depressed fracture which may require surgery.

(d) The demonstration of a calcified pineal gland and its position relative to the midline, which may change in subsequent radiographs, and indicate the presence of a lateralised supratentorial mass lesion.

(e) The presence of intracranial air.

Fig 7: Skull radiograph of poor quality fails to show fracture (arrow) properly.

Fig 8: Good quality radiograph shows fracture (arrow).

Therefore the radiograph on admission may provide an essential baseline when later deterioration occurs, and in the early management it can be argued that the radiographs are more important in patients who are relatively well than in those who are deeply unconscious. Unless radiographs are of reasonable quality, they are valueless and may be dangerously misleading (figs. above), and in those circumstances it is wiser to defer radiography until films of good quality can be obtained. The skull radiographs usually required are the right and / or left lateral, tghe half-axial (35°) and the anteroposterior (20°). However, a chest radiograph is essential, as

is a lateral radiograph of the cervical spine to demonstrate clinically undetectable fractures or fracture dislocation. If patients are in urgent need of intensive care, time should not be wasted in trying to obtain skull radiographs.

4. Continuing Care : The objects of further care are : (a) by nursing and other intensive care measures to enable the patients to survive a period of 'unconsciousness'; (b) by repeated observation to detect at the earliest possible moment the development of complications which may need surgical or medical action.

Care of the 'unconscious' patient : The quality of care given, which largely depends upon the availability of experienced nursing staff, will determine the outcome in the majority of patients. Attention to airway, skin, eyes, mouth, bladder and bowels, limb joints, nasogastric tube feeding and intravenous infusions are very demanding and the surgeon must take a personal interest in these matters if morale is to be maintained. Controlled ventilation is used widely if spontaneous respiration is inadequate to maintain normal levels of pCO_2 and pO_2. If the ventilation is controlled, full sedation and total paralysis is essential because if the patient struggles to breathe the intracranial pressure will rise. Tracheostomy is very rarely necessary in the first few days after injury because an endotracheal tube may be used for up to 7 days; thereafter if the airway or respiration is inadequate, tracheostamy may be required. As the level of consciousness improves, there may be a stage of irritability which is difficult to control; drugs are best avoided because all sedatives tend to depress respiration to some extent, which may lead to a gradual rise in the pCO_2 and deterioration due to brain swelling even 10 days after injury. Clearly if the patient is conscious enough to experience severe pain from other injuries (e.g., limbs) it is inhumane to withhold analgesics such as codeine phosphate (100 mg.), but morphia is best avoided. Because epilepsy is a major cause of deterioration (see below) all patients should be on prophylactic anticonvulsants; adult drugs and dosages are phenobarbitone 60 mg 8-hourly, or phenytoin 100 mg 8-hourly, and neither will depress the level of consciousness.

5. Deterioration : The most important indication of deterioration following head injury is a decline in the level of consciousness. Its recognition requires accurate observation and accurate sequential recording (see Glasgow Coma Scale, fig.). The major causes of deterioration are :

(a) Airway obstruction / hypoventilation causing brain swelling causing rising intracranial pressure.
(b) Brain swelling causing rising intracranial pressure.
(c) Intracranial haematoma.
(d) Epilepsy.
(e) Fluids and dehydration.
(f) Fever - infection - meningitis.
(g) Blood loss from other injuries.
(h) Aerocele.

Although swelling is a more frequent cause of deterioration than compressing intracranial haematoma, it must be assumed that the deterioration is due to a haematoma until proved

otherwise. The exceptions to this rule are : epilepsy, fluid imbalance, infection and fever, and blood loss. Unfortunately, airway obstruction and inadequate ventilation may not only cause increased brain oedema, but also the addition of brain oedema in the presence of a haematoma and, therefore, it is not always safe to assume that oedema alone is the cause of the deterioration following airway obstruction.

Epilepsy may cause rapid deterioration in level of consciousness and if a convulsion is not witnessed it may be difficult to differentiate this from the occasionally rapid effects of cerebral compression. A convulsion does not necessarily indicate the presence of a compressing haematoma.

Fever from any cause will produce deterioration. *Meningitis* may follow injury, especially with basal skull fractures, and tends to present at the 3rd or 5th day after injury. At that time, in the presence of fever and neck stiffness, diagnostic lumbar puncture is indicated.

Fluid imbalance and particularly dehydration may produce a gradual deterioration over the course of 48 hours; clinical examination, review of fluid charts, and plasma electrolytes should detect the imbalance, and the level of consciousness improves with correction.

6. Indications for surgery (decision making) :

THE SIGNS OF CEREBRAL COMPRESSION are DETERIORATION IN LEVEL OF CONSCIOUSNESS, SLOWING PULSE, RISING BLOOD PRESSURE and SLOWING RESPIRATION.

In the presence of these signs, and having reasonably excluded other causes (see above), the conclusion must be that :

This patient has a compressing haematoma until proved otherwise.

The *course of action* to be taken in these circumstances will depend upon :

(a) Availability of neurosurgical advice and facilities, and safety of transfer.
(b) Availability of CT scanning in the district general hospital or accident service.
(c) Speed of degree of clinical deterioration.
(d) Surgical expertise available.

Although in the majority of cases, and especially in more developed countries, surgery on the basis of skull radiographs and clinical signs is rarely necessary, it is essential that all surgeons should be aware of the indications for and the technique of such surgery. If there are clear indications for urgent surgery, delay for CT scan or transfer to a neurosurgical department may be disastrous.

Surgery without special investigations : The prime purpose of such surgery is to locate and evacuate, or exclude an acute extradural haematoma, an essentially remediable lesion, in a patient who is rapidly deteriorating. (The clinicopathological aspects of acute extradural

haematoma are described above and the reader may need to refer to this section before that which follows.)

The major decisions before embarking upon urgent exploratory surgery concern :

(1) *The presence or not of a haematoma* (determined by the signs of rising pressure, see above).

(2) *The side for exploration* : For this the essential guides are, in order of reliability, (a) the shift of a calcified pineal on x-ray and (b) the side of the first pupil to dilate, provided it was not dilated immediately after injury.

(3) *The position of exploratory burr holes on the appropriate side. In order of importance the guides are*, (a) the position of a vault or squamous temporal fracture, (b) the site of external trauma, and (c) the standard temporal position.

Preoperative : Ideally general anaesthesia with endotracheal intubation and controlled ventilation should be used. However, if that is not available, time should not be lost, an endotracheal tube should be inserted, and operation done under local anaesthesia. Having determined the likely side of the compressing lesion (see above), the patient is positioned supine with a sandbag under the appropriate shoulder and the face turned to the opposite side. It is wise to remove all the hair, or at least that of the appropriate half of the head, and that side is shaved. The sterile towels should be arranged in a way that allows access to frontal, temporal and parietal regions. Blood should be available, but surgery should not be delayed if it is not.

OPERATION

The exploratory burr hole : A 3 cm vertical incision is made down to bone; a self-retaining mastoid-type retractor is inserted and opened forcefully thus controlling scalp and muscle bleeding. The incision for the *standard temporal burr hole* is immediately above the zygomatic arch midway between the posterior margin of the orbit and the external auditory meatus. At that level the squamous temporal bone lies relatively deeply, but no damage can be done in reaching it, and it is essential to use the self-retaining retractor to control the scalp and muscle bleeding. Using a brace and bit with first the sharp point, and, when the inner table of the skull is pierced, the reamer which has a protective flange, a blurr hole is made. If a significant extradural clot is present the dura will not be seen because the dura will have been separated from the brain by the clot, which has the appearance of blackcurrant jelly. For the same reason the responsible bleeding dural artery e.g., middle meningeal will not be seen.

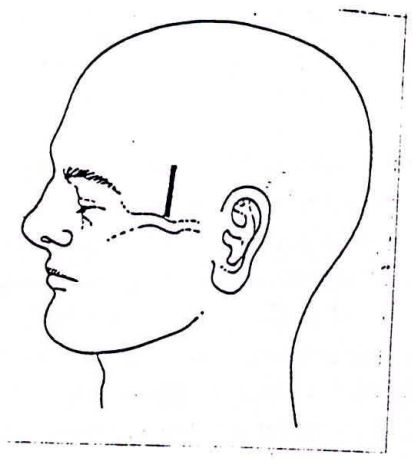


Fig 9: Incision for standard temporal burr hole.

Further action :

1. **Extradural clot is not present.** Bearing in mind that the essential purpose of this exploratory surgery is the exclusion or evacuation of an extradural clot it is unwise to open the dura at this stage. If the first burr hole has been placed over or next to the fracture, it is reasonable to make a second burr hole in the standard temporal position. If that also is negative it is reasonable to make a frontal or parietal burr hole if the original guides have not already included those positions. If none of these burr holes on one side have revealed an extradual clot two further questions arise :

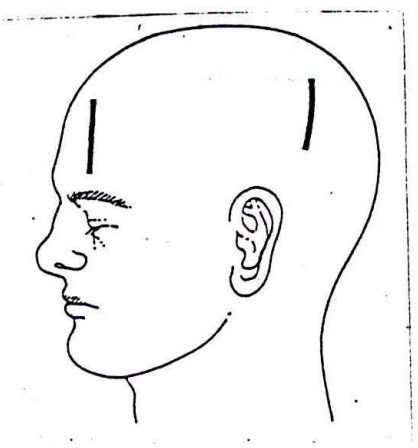


Fig 10: Incision for frontal and parietal burr holes.

(a) ***Should the dura be opened ?*** The assessment of the thickness and therefore significance of an acute subdural clot by inspection through a burr hole is extremely difficult. Only specialised radiology can assess this accurately and show the extent as well as the thickness of the lesion. Whenever there is primary brain damage (contusion, laceration) there is some clot in the subdural space, but this is rarely a significant compressing lesion. Furthermore the satisfactory evacuation of a significant subdural clot requires a major and often larger craniotomy (osteoplastic flap) with a wide dural opening; these procedures require particular neurosurgical

expertise and facilities, and the surgery itself may lead to further technical problems such as herniation of a swollen brain. Therefore if an extradural haematoma is not found, the dura should not be opened, ventilation should be continued, an osmotic diuretic given (mannitol, see below) and the patient transferred for special radiology.

(b) ***Should a burr hole be made on the other side ?*** If the radiologically demonstrated shift of the pineal has been used to determine the side of the compressing lesion nothing will be gained by making a burr hole on the opposite side. If the side of the first dilating pupil and the side of the fracture and / or site of external trauma conicide, a contralateral burr hole need not be made. If, however, the fracture or evidence of external trauma, e.g., bruising and swelling on the temporal region, is on the side opposite to the first dilating pupil, the compressing lesion is likely to be contre-coup (see above). However, very rarely the pupil may mislead and therefore a final burr hole should be made over the fracture and / or site of external bruising or swelling.

2. Extradural clot present. Assessment of thickness of the clot is made by using a blunt-nosed brain cannula with centimetre marks and feeling the resistance of the dura; any clot greater than 0.5 cm thick is significant. Sometimes the burr hole will have been placed just at the edge of the haematoma which is not, therefore, immediately visible; only by gently inserting a dissector and depressing the dura will the clot be detected.

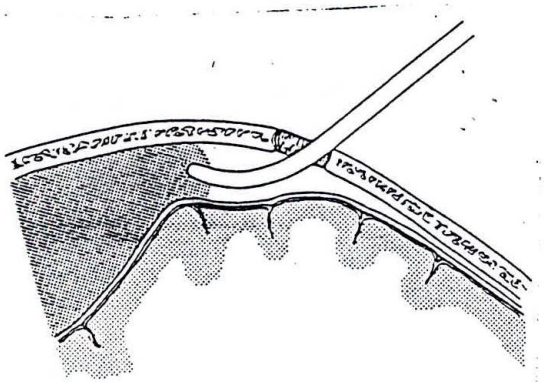


Fig 11: Burr hole near edge of extradural haematoma.

Evacuation of clot :

(1) By craniectomy : The burr hole incision is extended in a straight line to a length of about 8 cm, a second self-retaining retractor is inserted and with bone nibblers the burr hole is enlarged to a craniectomy of at least 7 cm diameter. The clot is then gently lifted off, this and the bone removal decompressing the brain. Small fragments of clot adherent to the dura are left because their removal only produces troublesome oozing from the dura to which 'Oxycel' may be applied.

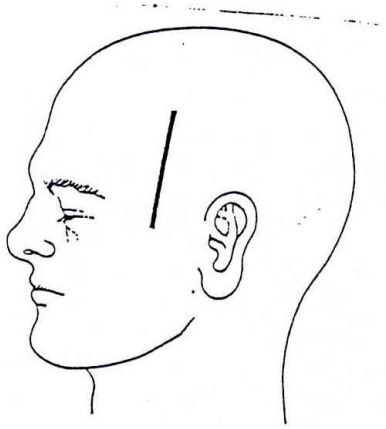


Fig 12: Extension of incision for craniectomy.

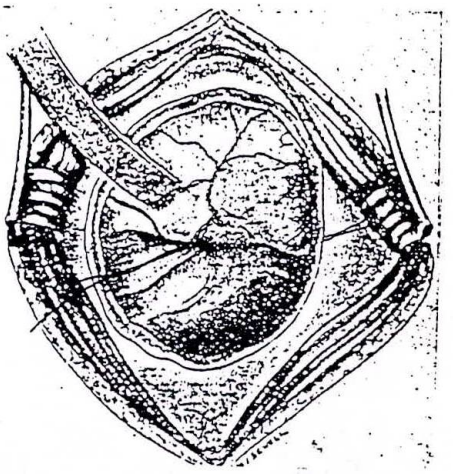


Fig 13: Craniectomy and control of meningeal artery by dural stitch.

If a major dural vessel is bleeding it is controlled either by diathermy or by a stitch passed under the vessel. The dura is then 'hitched' to the surrounding bone by passing sutures, e.g., black silk through the superficial layer of the dura and the surrounding muscle or pericranium; this step is essential to prevent the reaccumulation of a haematoma. The muscles, temporal fascia, galea and skin are then closed in layers without drainage and a full head dress using gauze and crepe bandage is applied.

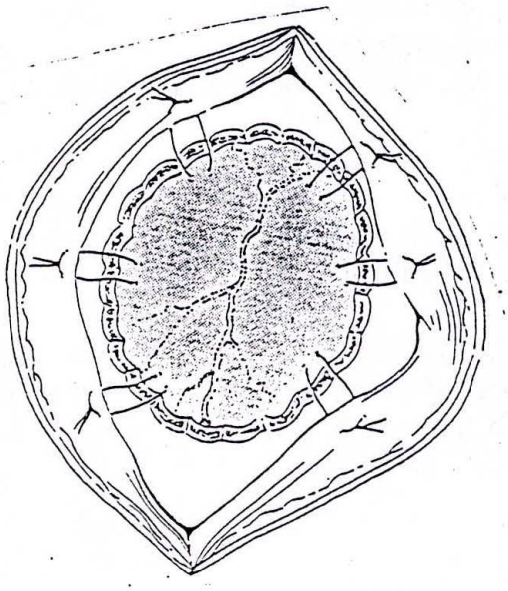


Fig 14: Dural 'hitching' stitches.

(2) By craniotomy and osteoplastic flap : In experienced hands this gives wider access without any appreciable loss of time in decompressing the brain and is generally used in neurosurgical departments, but does require familiarity and neurosurgical instruments. The stages are the turning of a scalp flap, the placing of multiple burr holes which are then joined by a Gigli saw and the raising of a quadrilateral bone flap hinged at its base upon the temporal muscles.

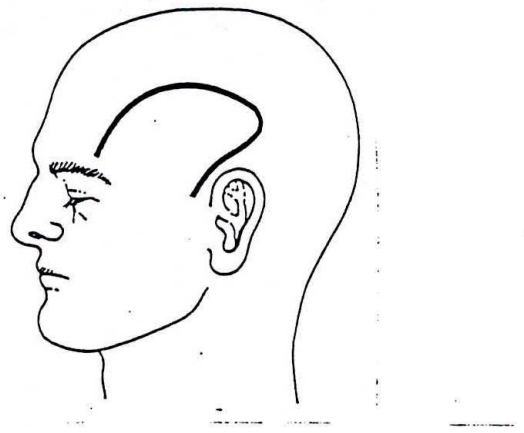


Fig 15: Incision for temporal craniotomy.

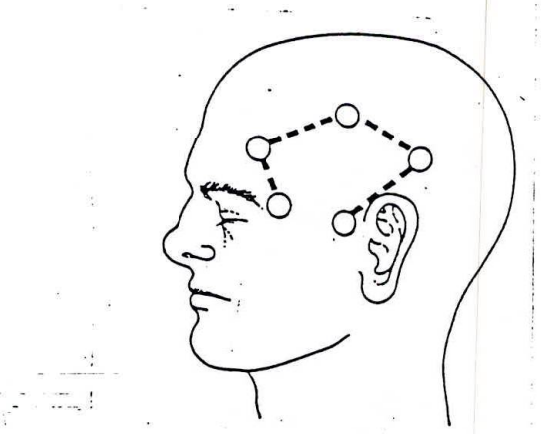


Fig 16: Burr holes and saw cuts for temporal craniotomy.

Postoperative : As soon as the conscious level permits the patient should be mobilised; skin sutures are removed on the 2nd or 3rd day. Antibiotics are not used routinely. Prophylactic anticonvulsant drugs (see Continuing care) should be continued for about 6 weeks in the absence of any other indications for their longer term use.

7. Special Investigations : Once the decision is made to shift the patient to the hospital where neuroradiological facilities are available, it is reasonable to give osmotic diuretics to the patient, to gain temporary improvement during transer.

Computed Axial Tomography (CT scan) : This non-invasive investigation provides the most accurate guide to intracranial pathology following injury. The head is scanned from above downwards in a series of transverse planes like a tomograph from vertex to base of skull by tilting the x-ray tube and detector array within a gantry at each scan.

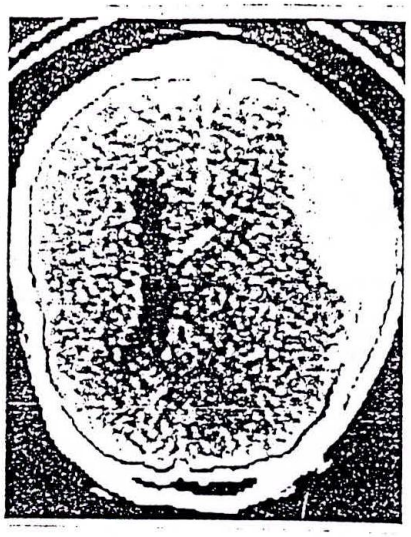


Fig 17: CT scan. Acute extradural haematoma.

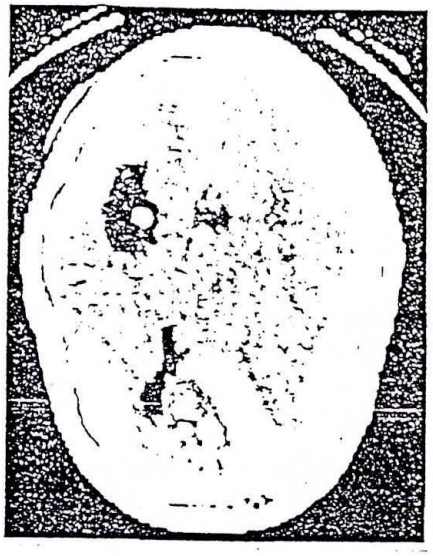


Fig 18: CT scan shows left hemisphere swelling, ventricle displaced to right, contusion, intracerebral blood and subdural clot.

The information is fed to a computer and produces a record in which high density objects such as bone, clot or tumour appear white, the brain substance appears grey, areas of oedema a dark mottled colour and the ventricular fluids black.

Indications for CT Scan : As CT scan facilities are available in non-specialised institutions, there is a need for defining its indications. A safe general rule is : **An indication for CT scanning is also indication for neurosurgical advice prior to CT.** This avoids unwarranted and dangerous delay in transferring to a neurosurgical department.

The indication for CT Scan are still a matter for some debate, but include :

1. Absence of speech or eye-opening after general resuscitation.
2. Deterioration in level of consciousness.
3. Skull fracture with persisting severe headache.
4. Skull fracture with confusion or neurological deficit.

Carotid arteriography, in the abscence of CT scan, remains a valuable method of investigation, particularly in the demonstration of extracerebral haematomas.

8. Nonsurgical therapy

Osmotic diuretics : These substances when administered parentrally, raise the osmolarity of the plasma, fluid will be drawn from the brain extravascular compartment into the blood, there by reducing the brain oedema and swelling.

Mannitol as a 20 per cent solution given intravenously to an adult as a volume of 250 ml the over the course of 20 to 30 minutes is the agent in general use. It may be used repeatedly at 6 or 8 hour intervals but the following must be noted :

1. there should be satisfactory renal function,
2. if a diuresis has not occurred following mannitol, further administration should be delayed,
3. fluid and electrolyte replacement should be meticulous, particularly with the loss of electrolytes with each diuresis,
4. if hyperosmolarity of the blood is to be produced, the rate of administration is critical.

Continued or repeated use of mannitol is only appropriate within neurosurgical departments, in conjunction with continuous recording of intracranial pressure.

There are potential dangers in the use of mannitol before either a diagnosis has been established or a course of actin instituted. Thus although the cause of a patient's deterioration may well be brain oedema, giving mannitol may produce temporary improvement in the presence of a large haematoma, soon to be followed profound deterioration. Therefore mannitol should not be used in the acute stage following injury unless the possibility of an intracranial haematoma has been excluded in a patient who is deteriorating, except to gain time prior to surgery. It should never be used as a diagnostic test to differentiate between oedema and haematoma.

Frusemide (40 - 80 mg by intramuscular injection) is an alternative agent which produces a very rapid diuresis and thereby raises the osmolarity of the vascular compartment. It is nonirritant, but requires good renal function for its effect.

Corticosteroids (dexamethasone and betamethasone). In high doses (up to 24 mg a day) these drugs are effective symptomatically in malignant brain tumours, the main effect being upon surrounding brain oedema. In the management of severe head injuries they have been used widely for some years, often in doses of up to 60 mg a day. Unfortunately this practice is based more upon tradition than objective evidence, because at present there is no evidence that their use improves the outcome in severe head injury.

9. Miscellaneous complications - early

'Brain-stem injury': This term is used loosely to describe a clinical picture which is ascribed to primary damage in the brain-stem (medulla and pons). It should be stressed that the changes may follow unrelieved supratentorial compression. The patients are often children or young adults, the latter being motorcyclists who, wearing crash helmets, suffered an acute flexion/extension movement of the neck at the moment of impact on the vertex. Signs of external trauma may be absent, but the patient is 'unconscious', with spontaneous extensor spasms of all four limbs, arching of the trunk (opisthotonus), a rapid pulse, rapid and often shallow and therefore inadequate respiration, small pupils, pyrexia and sweating. Any stimuli will tend to increase the tendency to extension. If this state has been present from the time of injury the likelihood of a compressing haematoma is remote. Prognosis depends upon intensive nursing

care, and control of respiration; the ultimate intellectual deficit may be considerably less severe than following major supratentorial brain damage, but there is often marked spasticity and incoordination.

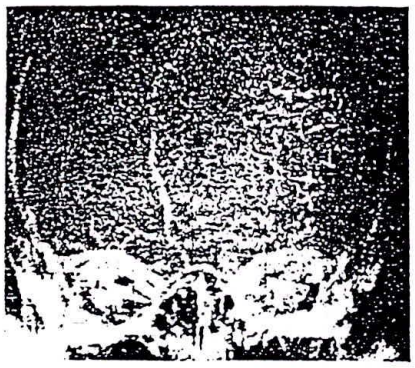


Fig 19: Carotid arteriogram showing midline shift and bare area over cortex due to clot.

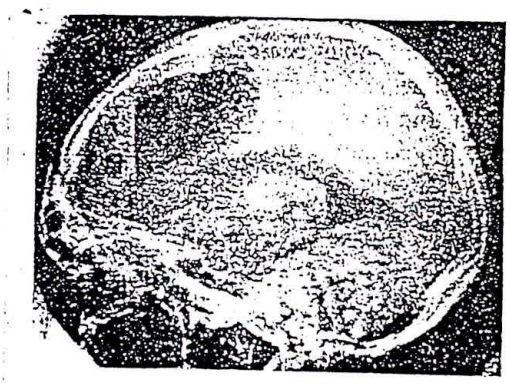


Fig 20: Frontal aerocele and 'spontaneous ventriculogram' fracture through frontal sinus.

Posterior fossa compression : Compression of the cerebellum and medulla by posterior fossa haematoma is rare. However, in the presence of a fracture which passes towards the foramen magnum and therefore crosses the lateral sinus an extradural haematoma may occur. These tend to present somewhat later than the supratentorial extradural haematoma, usually after 48 hours and the most important physical sign is slowing and irregularity of respiration, which may precede other evidence of deterioration. Special investigation by CT scan or ventriculogram is needed prior to a posterior fossa craniectomy.

CSF rhinorrhoea : For CSF rhinorrhoea to occur there must be a communication between the intracranial (intradural) cavity and the nose. Therefore CSF rhinorrhoea indicates a tear of the dura (usually basal) and a fracture involving the paranasal sinuses, frontal, ethmoid or sphenoid. At the moment of impact, not only does the dural tear and fracture occur but also a plug of brain may be forced into the dural tear; although this may temporarily seal the defect it also prevents dural healing and therefore the CSF rhinorrhoea may persist and the patient be at risk from meningitis, which is usually pneumococcal. CSF rhinorrhoea may occur in association with displaced fractures of the middle third of the face.

Although there are different views on the management of CSF rhinorrhoea a reasonabel course is :

1. Initially the patient is given prophylactic antibiotics (penicillin and sulphonamides).
2. Fractures of the middle third of the face are reduced; in many cases the rhinorrhoea may cease.

3. The indications for anterior fossa exploration are CSF rhinorrhoea persisting for more than 10 days, the presence of a fracture involving the frontal or ethmoid sinus, an aerocele, and an attack of meningitis which has been treated. Factors such as age, neurological state and degree of disruption of the anterior fossa floor are taken into consideration.

Aerocele : Entry of air into the cranial cavity usually occurs in association with CSF rhinorrhoea. Air may enter the subarachnoid space and ventricular system (a 'spontaneous ventriculogram') or the substance of the frontal lobe because the brain is adherent to the margin of the dural defect. This occurs particularly if the patient blows his nose. Rarely a frontal aerocele may cause compression and therefore deterioration 2 or more weeks after injury. Surgery is similar to that for CSF rhinorrhoea.

Meningitis : Apart from its association with CSF rhinorrhoea and otorrhoea, meningitis may occur after any major head injury particularly if a basal skull fracture is present but often undetected. Bleeding into the subarachnoid space is common with head injury, and neck stiffness immediately after injury is not an indication for lumbar puncture. Meningitis when it occurs does so after the first 48 to 72 hours and, therefore, provided there are no signs of cerebral compression, fever and neck stiffness are, at that stage, an indication for lumbar puncture. If there are also signs of cerebral compression, the patient should be transferred for urgent intracranial investigation before lumbar puncture.

Pituitary failure : Occasionally, basal fractures may pass across the pituitary fossa causing acute pituitary damage and endocrine failure. This may lead to a profound fall in blood pressure, tachycardia, pallor and hypothermia with deterioration in level of consciousness. Once the condition is recognised high doses of steroids should be given (hydrocortisone 200 mg 6-hourly for the first 24 hours, followed by reduced and then maintenance dosage).

Fat embolism : Systemic fat embolism may cause diagnostic difficulties when patients with multiple injuries, which include a head injury, show neurological deterioration at about 48 hours after injury or surgery for limb fractures. Neurological features which suggest fat embolism rather than an intracranial haematoma are the time of deterioration, the delayed onset of features of a brain-stem injury, pupils which vary in size from moment to moment but which remain equal, the presence of small retinal haemorrhages, and the absence of any firm lateralising signs. The finding of fresh petechiae over the upper part of the trunk and in the axillae is of help. However, in the absence of positive signs of fat embolism, it is usually wise to check the position of the pineal if it is visible, or to obtain a CT scan.

FRACTURES OF THE SKULL

Fractures of the vault and base of the skull are produced : (1) by compression of the sphere, (2) by local indentation, and (3) by tangential injury. A traditional primary classification of skull fractures is :

(a) ***Closed :*** The scalp is not breached, but there may be bruising or grazing of the scalp as distinct from a full thickness laceration.

(b) ***Open :*** There is an open laceration of the scalp with exposure of the underlying fracture.

Fig 21: Sharp bone spicules penetrating the dura. Probing of an open wound to determine the type of fracture is potentially dangerous because a spicule of bone plugging a laceration in a large vein or dural sinus may be dislodged, and cause uncontrollable bleeding.

This simple classification, which is analogous to that of fractures elsewhere, gives some guide to the likelihood of foreign tissues being present in the wound and the relative danger of subsequent infection at any depth. However, given that the primary management of the scalp injury is satisfactory, the differentiation between closed and open fracture is of less surgical importance than the classification of the type of fracture, which can only be established radiologically. Unless there is a very obvious skull defect beneath an open wound in which brain tissue is visible, attempts to a differentiate between simple and depressed fractures clinically are rarely accurate. Furthermore, probing of such wounds to determine the type of fracture is *potentially dangerous*.

1. Simple and comminuted linear fractures of the vault : These are the most common types of fracture, and in themselves do not require surgery. The surgical management is simply that of the overlying laceration if present. The significance of such fractures (apart from those in special sites) is that their presence gives some indication of the severity of the injury, the degree of deceleration or acceleration primary brain injury, and the likelihood of delayed complications. Indeed, extradural haematoma is more common in the presence of a simple linear fracture than with depressed fractures. A linear fracture of the squamous temporal bone is of special significance in that respect, and should make very frequent observation of the patient, particularly a child, obligatory.

2. Linear fractures of the skull base : The clinical indications of the presence of such fractures include bruising within the orbital margins involving the eyelids and conjunctiva (anterior cranial fossa), and bruising in the mastoid region, Battle's sign (middle cranial fossa and petrous bone). Such fractures are the result of skull distortion, and indicate that a considerable force was applied to the skull at the moment of impact. These fractures may be difficult to demonstrate radiologically, unless special skull views are taken. Since accurate diagnosis of such fractures is rarely essential for management, basal views of the skull should be done with circumspection because the position of hyperextension of the neck is potentially dangerous in the presence of any degree of cervical instability, or in the presence of cervical spondylosis.

Skull base fractures may be associated with immediate and often irreversible damage to cranial nerves, e.g., olfactory, optic, oculomotor, facial and auditory. A delayed lower motor neurone facial weakness may be due to contusion and swelling of the facial nerve in continuity within the facial canal. Anterior fossa fractures may be associated with CSF rhinorrhoea. Middle fossa and petrous bone fractures increase the risk of meningitis, which may occur after an interval of several days. CSF otorrhoea indicates a basal fracture with disruption of the dura over the petrous bone, and rupture of the tympanic membrane. Management is by prophylactic antibiotics for Gram-negative and Gram-positive organisms until the otorrhoea stops; meningitis due to continued leakage of CSF from the middle ear through the Eustachian tube is very rare.

3. Linear fractures of the posterior fossa and foramen magnum : These are rare and can be seen clearly only on the half-axial skull radiographs. Their significance lies in the rare association with a posterior fossa extradural haematoma, of which the presenting clinical feature may be a decline in respiration which precedes deterioration in level of consciousness. If such a fracture is present, it is wise to observe the patient for at least 3 days.

4. Linear fractures involving the frontal paranasal sinus : If such fractures are associated with a linear tear of the dura, CSF rhinorrhoea may occur. *Even if CSF rhinorrhoea is not present, it is wise to check that it cannot be provoked by positioning the patient with his head low, before he is discharged from hospital.* The indications for surgery in the absence of CSF rhinorrhoea are debatable, as is the prophylactic use of antibiotics.

5. Depressed fractures : Traditionally, a fracture is said to be 'significantly' depressed if the degree of depression is greater than the depth of the inner table of the skull. The possible complications of depressed fractures are :

(a) ***Dural tear :*** This is the most important indication for surgery. The greater the depression of the bone fragments, and the more the fragments are angled inwards, particularly a spicule, the more likely is the dura to be torn; even in the absence of such features it may be very difficult to exclude a dural laceration by the radiological appearances of a depressed fracture.

(b) ***Underlying haematoma :*** It is unusual to have a significant compressing clot beneath a depressed fracture. If there are clinical indications, investigation, e.g., CT scan, should be done before elevating the fracture.

(c) ***Pressure upon the cerebral cortex :*** In practice this very rarely contributes to the clinical effects of a depressed fracture.

(d) ***Epilepsy :*** A depressed fracture may be one of several factors contributing to early or late post-traumatic epilepsy. Elevation of the fracture may diminish the risk of epilepsy, although in individual cases this may be debatable.

(e) ***Cosmetic defects :*** In the adult this is rarely a problem; but the simple depressed 'pond' fracture of infants following obstetric manoeuvres may need elevation for cosmetic reasons, although such fractures often undergo spontaneous elevation.

(f) ***Pressure upon dural venous sinuses :*** Very rarely a depressed fragment of bone may compress and obstruct the superior sagittal or lateral sinus, leading to raised intracranial pressure. Of greater importance is the risk of severe haemorrhage if depressed fractures over the sinuses are elevated; therefore fractures in these sites should not be elevated.

SURGERY OF DEPRESSED FRACTURES

When the injury is compound prophylactic antibiotics covering Gram-positive and Gram-negative organisms should be started on admission, and continued for 10 days after surgery.

There are different views about the timing of surgery for depressed fractures. Emergency elevation is not required, but as soon as the patient's general condition is stable, especially in respect of other major injuries, operation for the compound fracture should be done. When the fracture is simple, i.e., closed, operation may be delayed for 2 or 3 days, particularly if there is doubt over the patient's neurological progress, and intracranial investigation may become necessary.

Important points in surgical technique are :

1. The hair should be shaved widely round the wound.
2. Care is taken to remove all foreign material from the laceration.
3. A wound may be extended in a linear fashion, or be made part of a skull flap.
4. Because the scalp is the most important of the tissues covering the brain, scalp should not be excised, and debridement should be kept to an absolute minimum.
5. The pericranium is detached from the bone using a rougine, and preserved for closure.
6. A burr hole is made in the normal skull next to the depressed area, but away from the midline of the vault. Bone is nibbled away towards the depression, the underlying dura is gently separated from the overlying bone fragments using an Adson's elevator.
7. The depressed fragments are cautiously lifted out and kept, so that the dura beneath the depression is fully exposed and any dural tear can be seen.
8. If there is a dural tear, the edges of the dura are gently separated from the underlying brain using the Adson's elevator.

9. Any indriven fragments of bone or foreign bodies are cautiously removed, but only obviously necrotic and extruding brain tissue may be sucked away.

10. Any bleeding from the brain can be controlled by diathermy or tantalum clips.

11. The edges of the dural laceration are brought together using interrupted sutures. If there has been loss of dura, a free graft of pericranium may be inserted.

12. If the area of exposed dura is greater than 3 cm diameter, 'hitching' stitches should be inserted.

13. The removal bone fragments should be cleaned and replaced in a mosaic fashion, and the pericranium and scalp closed carefully in layers without drainage.

Skull reconstruction : Areas of skull defect are restored at intervals of 3 to 6 months after injury, by the insertion of moulded tantalum plates or acrylic inlays. Concealed horseshoe or traverse incisions within the hair line are used for these purposes.

Head wounds due to missiles : The outcome depends on the explosive impact of the missile in the cranium, which is the commonest cause of fatality, and the relation of the wound track to the great vessels and the ventricles. Through-and-through tracks, from side to side or front to back, may be survived. Survivable injuries can be transported to a suitable hospital. Projection of the swollen brain through the dura at the site of entry or exit forms a hernia cerebri, which seals off the subarachnoid space during transport. A pressure dressing is applied. Immediate controlled ventilation has reduced the mortality from missile injuries.

Operation consists of the excision of the surface wound and suction, cleansing, and removal of foreign material from the track. The dura is closed by suture or grafting at entry and exit points. CT scanning is of great value in identifying in-driven foreign material and / or bone fragments.

Fig 22: Formation of burr hole beside a fractured area to allow unlocking of the bone fragments.

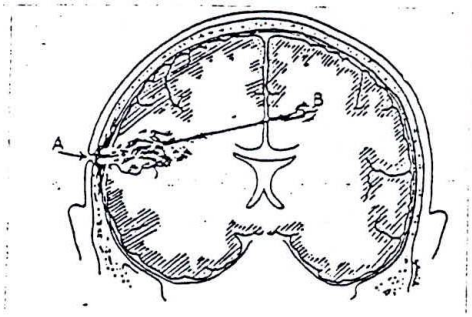


Fig 23: Transverse penetrating wound, entry at (A) showing : (1) protrusion of brain through the dura at the entry site forming a hernia cerebri; (2) pupled brain and bone close to entry; (3) a metal fragment at (B) has crossed the open superior fissure.

Penetrating wounds produced by sticks are always to be regarded very seriously. Although scissors or pokers may enter the roof of the orbit when a child falls and thereafter be successfully withdrawn, a stick which goes in through the orbital roof, or backwards behind the orbit into the temporal lobe, cannot be withdrawn intact; portions which are left behind may, from previous contact with the ground, be infected with gas gangrene or tetanus, with probable fatal effects.

LATE EFFECTS OF HEAD INJURY

Chronic subdural haematoma

This is produced by rupture of the veins passing from the cerebral hemispheres to the venous sinuses as the result of displacement of the brain inside the skull. Usually, the superior cerebral veins are ruptured, producing haemorrhage over the convexity of the hemispheres; very rarely veins passing from the temporal lobe to the sphenoid or petrosal sinuses may produce clots which collect on the under-aspect of the brain. This complication, which is potentially fatal, is produced particularly by blows of small magnitude applied to the front or back of the head which may be insufficient to produce even transient concussion, but which are sufficient to move the brain suddenly. Cerebral atrophy renders this displacement easier, and hence the condition becomes commoner with advancing age. The superior cerebral veins pass from the convexity of the hemispheres and pierce the arachnoid membrane before crossing the potential subdural space to joint the inner aspect of the dura 2.5 cm or more from the midline; they then run inwards to drain into the lower compartment of the superior longitudinal sinus. Sudden displacement may snap the vein at the level of the arachnoid, allowing blood to pass downwards into the potential subdural space between the arachnoid and dura. Frequently, corresponding veins on both sides are affected and the condition is bilateral in 50 per cent of cases. The haematomas are often large, bilateral collections up to 60 ml a side or unilateral collections of 120 ml being quite usual. There is a progressive change in the nature of the subdural fluid, which becomes thinner, lighter in colour and eventually is similar to CSF.

Clinical features : The symptoms may follow a concussion, but owing to the slight nature of the force required to produce displacement, this complication may occur without preceding loss of consciousness and without the head being even struck. It can follow a sudden jolt, as when a driver is thrown against the steering-wheel of a car, or be produced by knocking the head against the lintel of a door, or landing heavily on the feet when jumping from a height; it has followed dental extraction and electroconvulsive therapy. The interval between 'trauma' and onset of symptoms may be of weeks or months.

The symptoms are undramatic and consist of mental apathy, slowing of cerebration, slowness of response to questions, merging into stupor. When the stupor develops, it comes and goes as the brain volume varies, the patient being inaccessible at times and then rousing sufficiently to answer questions accurately, but very slow and after a considerable pause. When the level of consciousness deteriorates further the operative mortality rises to 30 per cent; hence the significance of the early symptoms.

Physical signs vary. In older patients, where more room is available and if the fluid collects slowly, there may be no signs, or at most a unilateral or bilateral extensor plantar response from pressure on the motor cortex, or brain-stem displacement. Pupillary changes occur last when the brain-stem is affected and pressure-cone formation is imminent. Lumbar puncture shows a fluid at low pressure with protein increased to 120mg / 100 ml (1.2g/l), often stained yellow from the transudation of pigment, but no cells. Papilloedema is exceptional. Success in treatment comes from acting on suspicion and employing skull radiographs for the

position of the pineal, CT scan, or exploratory burr holes. Carotid arteriography should be avoided in the elderly.

Treatment : Bilateral posterior parietal burr holes are made under local anaeshtesia to expose the dura, which often has a blue-green tinge. On incising the dura there is gush of brown fluid. This should be allowed to flow out of the burr hole spontaneously, aided by lowering the head. The brain surface will frequently remain at a considerable distance from the dura. If the CT scan has shown a large collection of fluid, but only a small amount has been obtained from the posterior parietal burr hole, another burr hole should be made further forward on that side.

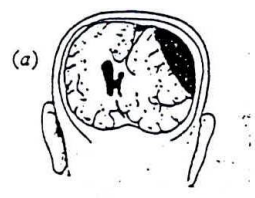


Fig 24: Site of clot

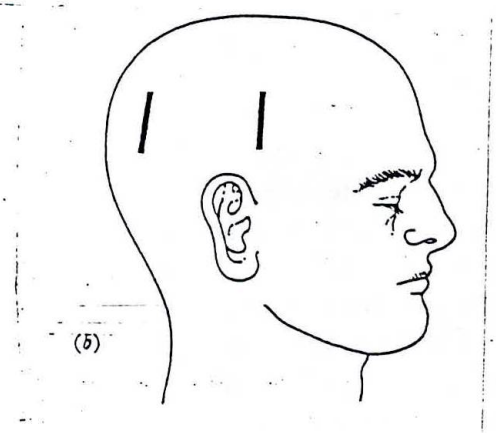


Fig 25: Incisions for burr holes for chronic subdural haematoma.

Postoperatively the patient is nursed flat initially and the subdural space can be re-tapped using a blunt-nosed brain cannula through the closed burr hole wounds.

Burr-hole aspiration of a chronic subdural haematoma, especially in the very elderly, may cause added fresh haemorrhage into the haematoma cavity. Therefore an alternative to surgery is the use of steroids (dexamethasone), but this should be reserved for patients who are not showing rapid or severe neurological deterioration.

Post-traumatic epilepsy : Epilepsy occurring soon after injury does not indicate the presence of a compressing intracranial haematoma. It reflects degrees of primary brain damage, which may be trivial and from which total recovery occurs. Children are more susceptible to early epilepsy than adults, and it is probably a reflection of an inherent epileptic tendency. Epilepsy within one week of injury occurs in about 10 per cent of all cases admitted to hospital with a head injury; factors which tend to increase the incidence are prolonged post-traumatic amnesia, intracranial haematoma and depressed fracture. In these circumstances prophylactic anticonvulsants should be given for about 6 weeks, but for up to 3 years in those who have suffered from any post-traumatic epilepsy.

Late post-traumatic epilepsy has an overall incidence of about 5 per cent, but of up to 25 per cent in those who have suffered from early epilepsy, and therefore similar factors are involved. It probably represents cortical scarring and reactive gliosis. The epilepsy may be focal, general or essentially temporal, and although in the majority of patients its onset is within the first year, in about 20 per cent the onset is delayed for more than 4 years. When the onset is very late, e.g., 10 years, it may be wiser to investigate the patient as a case of 'late onset epilepsy' rather than assume that the epilepsy is post-traumatic. The use of a long-term prophylactic anticonvulsants, e.g., for 3 years, is advisable in those who have suffered early epilepsy, major compound fractures with dural and cortical laceration and intradural compressing haematomas.

Post-traumatic hydrocephalus : Late deterioration with apathy and mental retardation may rarely be caused by post-traumatic hydrocephalus requiring the insertion of a ventricular shunt.

Post-traumatic headache is not always cerebral in origin. Referred pain of spinal origin resulting from associated strains in upper cervical joints may be referred through the great occipital and posterior auricular nerves to the vertex, forehead, or temple. The post-traumatic state, with vertigo, defective memory and concentration, abnormal fatigue, irritability, and defective emotional control, may result from temporary or permanent cerebral lesions.

Patients with minor injuries recover speedily, but in serious cases prolonged convalescence is needed. Usually, some symptoms of the post-contusional state consisting of headache, giddiness, defective memory, defective concentration, irritability, impaired emotional control, impaired sleep, or susceptibility to alcohol, persist for a period of 18 months. Post-traumatic dementia constitutes a permanent handicap sometimes necessitating institutional care. Schizophrenia is a rare complication of even a minor injury.

HEAD INJURIES

1. Maintain airway patency by keeping the patient in the lateral position to prevent the tongue from falling back. Clear the mouth and oropharynx of secretions by means of suction. Introduce an oro-pharyngeal or naso-pharyngeal airway. Perform endotracheal intubation if necessary.

2. Ensure adequate gaseous exchange by providing oxygen. Give positive pressure ventilation using the Ambu bag if necessary.

3. Check the vital signs. The presence of hypo-tension is more likely to be due to injuries other than head injury. These may be external or internal injuries. Therefore it is essential to do full physical examination.

4. Evaluate the severity of the head injury using the Glasgow Coma Scale.

Eye Opening	**Verbal Response**	**Motor Response**
E1 No eye opening	V1 No verbal response	M1 No motor response
E2 Eye opening to pain	V2 Incomprehensible	M2 Abnormal extensor
E3 Eye opening to call	V3 Inappropriate words	M3 Abnormal flexion
E4 Spontaneous eye opening	V4 Confused	M4 Withdraws
	V5 Oriented	M5 Localises
		M6 Obeys commands

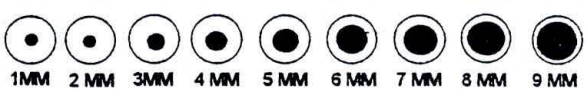
A GCS RATING OF LESS THAN 7 OR 8 DENOTES COMA

5. The presence of pupillary asymmetry denotes incipient herniation. This requires urgent management with anti edema measures. In such a case rule out direct optic nerve or III cranial nerve injury.
6. Start an IV line. Anti edema measures and anticonvulsants may be given in consultation with the neurosurgeon.
7. Raise the head end of the cot by 30 degrees.
8. In case of local scalp injury - shave the area liberally, wash thoroughly with saline and probe the area gently with a gloved finger. Do not use any sharp instruments or probes for this purpose. Do not try to dislodge fractured fragments of skull. Suture the wound after lavaging with hydrogen peroxide and povidone iodine (Betadine / Wokadine).
9. In paraplegic / quadriplegic patients - do not try to extend or move the neck eg., during intubation. Put the patient on a flat board with sand bags on either side of the head to prevent movement. Put a cervical collar before shifting the patient anywhere.
10. Before doing any investigations - Eg. X-ray, CT Scan, consult the Resident on duty in Neurosurgery or the Neurosurgeon, if available or refer the patient to neurosurgeon unit.

Prepared by Neurosurgical Department,
St. John's Medical College Hospital, Bangalore.

NEURO CHART

COMA SCALE		Date and Time															
	EYES OPEN 4	Spontaneously	4														C= eyes closed by swelling
		To Command	3														
		To Pain	2														
		No Response	1														
	BEST MOTOR RESPONSE 6	Obeys Commands	6														Record best arm response
		Localizes Pain	5														
		Flexion-Withdrawal	4														
		Flexion(abnormal)	3														
		Extension (abnormal)	2														
		No Response	1														
	BEST VERBAL RESPONSE 5	Oriented	5														T-Endoracheal Tube or Tracheostomy A= Aphasia
		Confused	4														
		Inappropriate Words	3														
		Incomprehensive Sounds	2														
		No Response	1														
TOTAL SCORE																	
PUPILS	Size	R															B= Brisk S= Strength N= No Reaction C= Closed
	Reaction																
	Size	L															
	Reaction																
	Name of the Nurse																



Courtesy: St. John's Medical College Hospital, Bangalore

SPINAL INJURIES

In recent years spinal injuries have become common as a result of high velocity automobile accidents. Injury to spine involves bony elements, soft tissues (including muscles, ligaments, intervertebral disc and capsule of apophyseal joints) and quite often spinal cord and nerve roots. The aim of treating spinal injury is to restore the painless, stable spine and ensure recovery from neurological damage. The nature of initial injury may at times preclude this achievement in some patients. In order to manage these injuries adequately, comprehensive knowledge of anatomical features, stabilising structures of spine, mechanism of injury and radiological features is necessary. Above all there should be high index of suspicion of presence of spinal injury as the symptoms may be few, develop insidiously later on and quite often patient may be unconscious due to concomitant head injury.

SPINAL STABILITY

Spinal injury can be stable or unstable depending upon whether stabilising structures have been damaged. A stable fracture or dislocation is not liable to displacement greater than that produced at the time of injury, whereas an unstable fracture or dislocation is liable to further displacement with serious consequences to the spinal cord. The unstable injuries therefore require very careful handling of patient, prolonged immobilization, external splintage and sometimes internal fixation also.

Throughout spine, stability between two vertebrae depends upon following structures :

1. Intact ligaments, particularly posterior ligament complex comprising of supraspinous, interspinous, posterior longitudinal ligament, ligamentum flavum and capsule of lateral apophyseal joints. Disruption of posterior longitudinal ligament complex occurs mostly in flexion injuries. Anterior longitudinal ligament also provides stability during extension and is injured in extension injuries of spine.

2. Intervertebral disc is an important stabilizing structure between two adjacent vertebrae and its disruption makes spine, particularly cervical spine, very unstable. Disc may be injured in both flexion and extension injuries.

3. Muscles which are attached to vertebrae not only provide motor power for various movements but also protect spinal column and its contents. Both during flexion and extension injuries muscles get severely attenuated and torn.

Cervical spine is a flexible column with wide range of mobility which has developed at the expense of stability. In cervical spine lateral articulations are small and flat with very little upward inclination and therefore it is easy for the upper face to slip forwards on the lower facet. There is normally some lordosis in cervical spine which disappears in flexion. Greatest mobility occurs in lower cervical spine where intervertebral discs are thicker and wedge shaped. Injuries are also commonest in this area of greater mobility.

The thoracic spine is more stable than the cervical and lumbar spine. The increased stability is due to reinforcement provided by rib case and shape and size of articular processes of facet joints. Articular processes of facet joints are flat and upright facing almost backwards and forwards so that continuous processes lock together securely and dislocation is extremely rare.

Lumbar spine is mobile but less than cervical spine. The articular processes are massive and curved. The are directed inwards and outwards and are vulnerable to dislocation. Lumbar spine is more stable than cervical spine but much less stable when compared to thoracic spine.

CERVICAL SPINE INJURIES

DIAGNOSIS

Cervical spine injury should always be suspected in patients injured in violent road traffic accidents and fall from height, in multiple injury patient and in unconscious patients (due to head injury). Sometimes symptoms develop insidiously and therefore to be aware of possibility of Cervical spine injury is most important factor in the diagnosis. Cervical spine injury should also be suspected in patients complaining of suboccipital, shoulder and arm pain, paraesthesia or motor weakness in upper or lower limbs following injury.

Very careful examination of neck is required. Patient may be holding the neck still or in a deformed position. Neck muscles may be in spasm. There may be localised tenderness over Cervical spine. Do not test for movement. Motor or sensory deficit may be present in limbs. Location of wounds, bruises and abrasions on head and face, along with history of type of accident may give an idea about the direction of damaging force.

Diagnosis is confirmed by X-rays which must be of good quality and show whole of cervical spine. An open mouth AP view for C-1 vertebra is also required. If facet joint dislocation is suspected, oblique X-rays are also taken to decide as to which facet joint is dislocated. When no bony injury is detected on X-rays then in order to identify subuxation without fracture or spontaneously reduced dislocation, lateral X-ray is taken with neck in slight flexion. For this purpose head should be moved very gently by the doctor himself and if patient experiences pain or paraesthesia in limbs, neck movement must be stopped immediately.

EMERGENCY TREATMENT

In suspected cervical spine injuries until the diagnosis has been firmly excluded or when injury is diagnosed, before proper treatment is instituted following care should be taken.

1. Patient should be placed face up on a firm stretcher.
2. Sand bags, rolled bed sheets or blankets are placed on either side of head to maintain neck in neutral position.
3. Never move the neck or test for movements.
4. As soon as possible head halter traction with 5 kg. weight should be applied to keep cervical spine steady.
5. Respiratory embarrassment should be treated by oxygen inhalation and by performing tracheostomy when necessary.

This may be required in cases of cervical spinal cord injury when vital capacity may be markedly reduced due to muscle paralysis.

THORACOLUMBAR SPINE INJURIES

DIAGNOSIS

As with cervical spine injuries awareness to the possibility of thoraco-lumbar spine injury is the most important factor in diagnosis. History is often helpful as the patient may complain of pain in back and (if neurological damage has occurred) inability to move lower limbs and anesthesia. Thorough clinical examination of the patient is necessary. All clothing should be removed so that entire trunk can be inspected. Patient must be rolled on to his side in one piece with the help of 2-3 persons (like log rolling). Inspection may show abrasion or bruising which indicates levels of injury. Spinous processes of entire thoraco-lumbar spine are carefully palpated to detect local tenderness, swelling, gap or step between spinous processes. Normal thoracic kyphosis and lumbar lordosis may be altered with paravertebral muscle spasm. Complete neurological examination is performed to detect any evidence of spinal cord damage.

Following clinical examination X-rays of spine should be in A.P. and lateral planes. After examining these X-rays if necessary oblique views may have to be taken. It must be understood and borne in mind that spinal cord may be transacted without X-ray evidence of fracture or displacement if spontaneous reduction has occurred. In such case clinical examination forms the mainstay of diagnosis.

EMERGENCY MANAGEMENT

In suspected thoraco-lumbar spine injury following care should be taken until the diagnosis has been established or firmly excluded.

1. At the site of accident and while transferring the patient from ambulance to stretcher, patient should be lifted in one piece with the help of at least three persons.
2. Never attempt to test the spinal movements.
3. Chest, abdominal and head injuries should be carefully looked for and (if present) respiratory embarrassment should be promptly treated.
4. Unaccompanied patient should never be sent to radiology department for X-rays. A doctor should accompany the patient. Patient must not leave X-ray department until a thorough X-ray examination has been completed.
5. Neurological assessment should be repeated after 6-12 hours as the signs of cord compression may be delayed at times.

Type of injury varies according to the direction of force which has produced the injury. Here again it is crucial to decide if the injury is stable or unstable. However if in doubt, these cases should be sent to specialized centres for further management. Thus it is important to diagnosis these injuries at primary health centre level or at district level hospital, which not only reduces the mortality, also reduces the morbidity.

Introduction to Thoracic Injury

Trauma is amongst the leading causes of death and disability at all ages. Twenty five percent of all trauma deaths are due to chest trauma alone, and respiratory problems contribute significantly in upto 75% of all trauma deaths. Fortunately, 85% of the life threatening thoracic injuries can be managed by simple interventions which can be carried out easily by the emergency room medical personnel. The chest trauma produces a number of situations which need immediate attention to save life. These are :

THORACIC TRAUMA

Airway Obstruction :

1. Establish Adequate Airway
   - Oral Suction
   - Insert Airway
   - Intubate if necessary (Oro-tracheal or naso-tracheal). If intubation is not possible, an emergency crico-thyroidotomy or tracheostomy may be performed.

2. Look for signs of shock
   - Blood Pressure
   - Pulse
   - Pallor
   - Peripheral pulses
   - Cold extremities.

3. Look for external wounds
   - If sucking wound is present - apply sterile pad and strap
   - Bleeding points if easily seen - to be ligated. If not, apply pressure dressing.

4. Look for associated injuries
   - Head
   - Abdomen
   - Spine
   - Long bones and pelvis.

5. Resuscitate
   - I V line : Using 18 gauge branulae start Ringer Lactate and / or Haemaccel
   - Establish Airway
   - Oxygen mask
   - Prop up the patient (45 degrees).

6. If the patient is extremely dysponeic
    - Do not send for Chest X-Ray
    - Insert an interoostal Drainage Tube in the 4th Intercostal space in the mid-axillary line on the side of diminished air entry.

7. Chest X-ray
    - In the erect position (only if the patient is haemodynamically stable and spinal injury has been ruled out)
    - There is no need for Oblique or Lateral views.

Look for

a) Tension Pneumothorax and Pneumothorax.
b) Haemothorax and Massive Haemothorax.
c) Mediastinal shift and widening.
d) Fractured ribs and Fractured Clavicle.
e) Massive Flail Chest.
f) Subcutaneous emphysema.
g) Massive Air Leak.
h) Increased cardiac size.
i) Elevated diaphragm and Diaphragmatic Injuries.
j) Loss of Aortic definition.

8. Inform the Thoracic ward, Resident Doctor in Cardiothoracic surgery to see the case URGENTLY.

IF THE RESIDENT DOCTOR IS NOT AVAILABLE INFORM THE CARDIOTHORACIC SURGEON ON CALL.

Management of Chest Injuries :

1. Airway Obstruction :
    - Clear the airway of Mechanical Debris and secretions.
    - Oro or Nasotracheal intubation.
    - Tracheostomy.

2. Fractured Ribs and Clavicle :
    - Strapping by adhesive plaster and assurance.

3. Tension Pneumothorax is an extreme emergency which can lead to collapse of the ipsilateral lung, shift of the mediastinum and compression of the large veins which can lead to sudden death due to decreased cardiac output. The immediate release of the tension by placement of a large-bore needle in the second Intercostal space in the Mid Clavicular line followed immediately by insertion of intercostal underwater drainage at second intercostal space is life saving.

4. Open Pneumothorax : The sucking chest wound leads to pneumothorax and collapse of the ipsilateral lung. These patients can be sterilised by any mechanical covering over the open wounds (strap the wound immediately with sterlize gauze and pad with adhesive plaster, and wound to be sutured in the Operation Theater as far as possible. A chest tube is put in simultaneously and connected to an under-water seal. When the patient is sterilised, debridement and formal closure of the chest wound can be performed.

5. Haemo Pneumothorax : Insertion of the Intercostal underwater drainage at $7/8^{th}$ Intercostal space at posterior axillary line.

6. Haemothorax : A small haemothorax that produces little more than blunting of the costophrenic angle on the chest X-ray does not require any treatment. When the haemothorax exceeds an amount that fills the costophrenic sulcus, or when there is associated pneumothorax, intercostal drainage tube may be put and connected to an underwater drainage system.

7. Massive Pneumothorax (> 1.5 liters of blood in pleural cavity on initial insertion of chest tube) should have thoractomy immediately as a surgically correctable cause is likely to be found in a large number of these cases. In addition, after initial chest tube placement, if the bleeding continues at a rate of over 100-200 cc/hr. for over few hours, thoracotomy should be undertaken.

8. Massive Air Leak : Tracheobronchial injuries - often caused by steering wheel compression of the chest in cases of road traffic accidents are increasing in number. The injury is often fatal but may be surprisingly well tolerated for a brief period. Massive air leak is present along with collapse of the lung on the affected side. It needs immediate thoracotomy and repair of the injury.

9. Injury to Lung Parenchyma (by Flial and Stove in chest) : Prevented by strapping with white plaster and refer to higher center.

Apart from the "life threatening" situations mentioned above, there are many other injuries which require treatment. These are :

Diaphragmatic Injuries :

Urgent repair of massive diaphragmatic injuries may sometimes be required if herniation of abdominal contents into the chest prevents lung expansion - left hemi-diaphragm is ruptured 9 times more often than right hemi-diaphragm. More often, the herniation of the abdominal contents happens slowly and patients present later with various symptoms. Thoracotomy, reduction of abdominal contents and repair of the diaphragm are required in such cases.

MANAGEMENT OF THORACIC TRAUMA

Assessment and Resuscitation

1	2	3	4	5	6
Watch and listen to the patient breathe. Estimate tidal volume. Watch for flail chest. Look for signs of haemothorax or pneumothorax and tracheal shift. For tension pneumothorax, decompress chest by large-bore needle or similar emergency measure in anterior axillary line second or third intercostal space. • Clean out upper airway. • Remove patient's clothing. • Determine injuring mechanism. • Seal sucking wounds.	Monitor vital signs. Restlessness, cyanosis, or bradycardia indicate hypoxia. Confirm that finding with arterial blood gas determination. • Give respiratory support as appropriate : endotracheal intubation with positive pressure assistance.	Start intravenous (IV) fluids through large-bore (10-15 gauge) catheter in arm opposite to chest injury. • Run crystalloid fluids fast enough to keep blood pressure at 80 to 100 mm Hg. • Send blood for type and crossmatch. Use typespecific blood for protracted or recurrent shock after infusion of 2000 ml crystalloid solution.	Obtain upright or lateral decubitus chest X-ray. • Insert large-bore (36F to 40F) chest tube in cephalad direction for haemothorax or pneumothorax in fifth intercostal space (nipple level), just anterior to mid-axillary line (see figure 1). • Avoid trocar chest tubes. • Give systemic antibiotics to patients with penetrating injuries.	Monitor the EKG / CVP.	Perform other diagnostic studies : A. Evaluate persistent air leak with unexpanded lung; confirm bronchial rupture by bronchascopy / gram. B. Conduct radiographic studies for potential great vessel or esophageal injury.

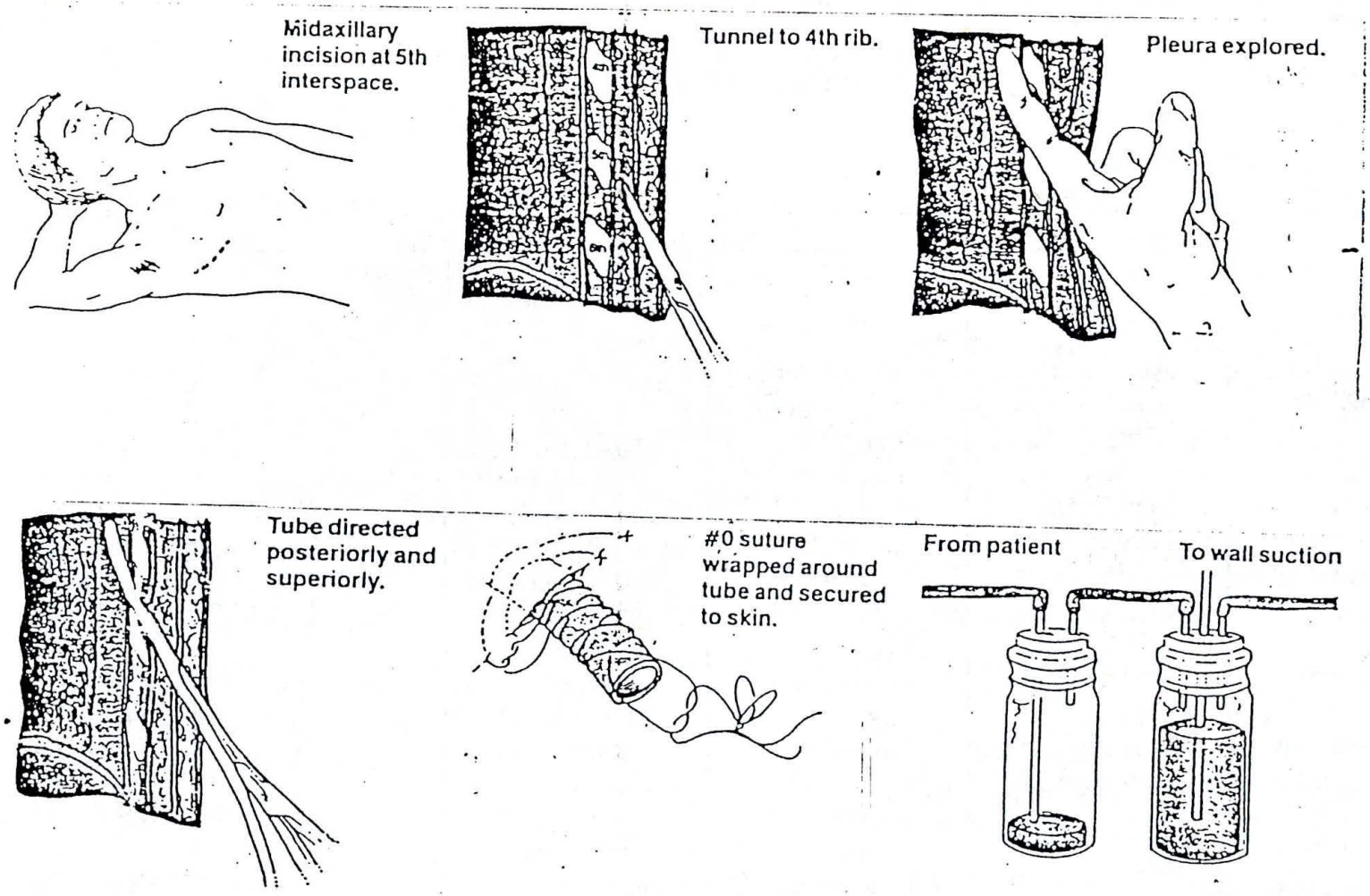


Fig (a) : Insertion of Chest Tube

Indications for urgent thoracotomy in operating room

1. Over 1500 ml blood in chest.
2. Ongoing bleeding at 100 ml/hour.
3. Cardiac tamponade (distended neck veins, supraclavicular cyanosis, low blood pressure)- temporize with aspiration through pericardiocentesis; push IV fluids (see fig. b)
4. Traumatic thoracotomy (loss of chest wall substance)
5. Massive air leak.

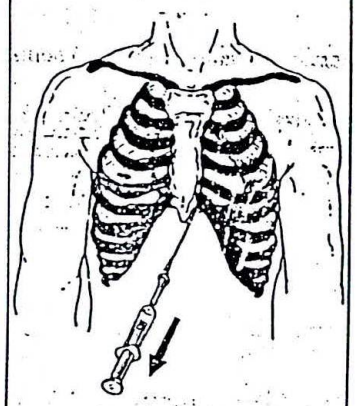


Fig. 6 Pericardiocentesis

Indications for immediate thoracotomy in emergency room by personnel experienced in technique (rarely indicated)

1. Hypovolemic cardiac arrest inspite of blood volume replace-ment, closed chest massage, and defibrillation.
2. Cardiac arrest with penetrating injury to chest.

Relative contra-indications for immediate thora-cotomy in emergency room

1. Obvious, extensive injury to the central nervous system.
2. Prolonged external cardiac massage (>5 to 10 minutes) with electrical asystole and no response to cardiopulmo-nary resuscitation.
3. Blunt chest trauma.
4. Unavailability of surgical personnel for continuing surgical therapy.

Radiographic findings to suggest need for aortography

1. Lateral deviation of nasogastric tube in the esophagus.
2. Widened upper mediastinum greater than 8 cm.
3. Loss of aortic knob.
4. Left apical pleural haematoma.
5. Depressed left main stem bronchus greater than 140 degrees.
6. Right lateral displacement of trachea.
7. Anterior displacement of trachea (lateral x-ray)
8. First and / or second rib fracture.
9. Massive chest trauma with multiple rib fractures.
10. Fracture / dislocation of thoracic spine.

ABDOMINAL TRAUMA

Abdominal injuries are common in patients who have met with road traffic accidents. These are frequently associated with head, chest or limb injuries.

A smaller group of patients present with penetrating abdominal injuries due either to assault or industrial accidents. In both cases, and more so in blunt abdominal injuries, a thorough evaluation of all systems is required. This can be conveniently thought of under two heads.

1. Physiological status.
2. Anatomical injuries.

EVALUATION OF PHYSIOLOGICAL STATUS

The three vital functions can be quickly assessed as

1. Circulation

A fast and thready pulse, pale mucous membranes and a cold and clammy skin, WITH or WITHOUT a fall in blood pressure (note that intense vaso construction can maintain blood pressure in the presence of shock) denotes SHOCK DUE TO HYPOVOLEMIA most often due to INTERNAL OR EXTERNAL BLOOD LOSS.

Once shock is identified (which should be within 2 or 3 minutes of arrival of the patient) the following protocol is to be followed strictly.

Shock Protocol :

a) IMMEDIATELY LARGE SIZE VENOUS ACCESS

It is wise for two persons to start resuscitation, one to start a peripheral line as quickly as possible and another to do a Basilic vein cut-down to insert a large bore catheter.

b) COLLECT BLOOD FOR GROUPING, TYPING AND CROSS MATCH.

c) INFUSE SALINE / RINGER LACTATE / COLLOID.

d) INSERT FOLEY'S CATHETER PER URETHRA.

e) REASSESS AFTER ADEQUATE VOLUME INFUSION FOR URINE OUTPUT.

f) DO NOT SHIFT THE PATIENT TO RADIOLOGY.

Generally this resuscitation procedure corrects shock and provides some more time for evaluation and investigation. However, if SHOCK PERSISTS after initial resuscitation, a major vessel injury or massive solid organ injury is likely. THORACENTESIS (aspiration of the pleural cavity) and ABDOMINAL PARACENTESIS on either side will reveal the possible site of bleeding. Quick transfer to the operation theatre with group specific blood is of essence to control bleeding.

If there is no shock or if shock has been corrected, further evaluation is undertaken.

2. **Respiration**

The respiratory rate and effort, the presence or absence of cyanosis must be noted.

3. **Central Nervous Functions**

The Glasgow Coma Scale is the best evaluator of this and must be used in all cases at initial and subsequent serial observations.

Evaluation of Anatomic Injuries :

This must proceed in sequential and logical order to include the body region divisions accepted internationally viz.,

a) Head and Neck.
b) Thorax.
c) Extremities and Girdle injuries.
d) Skin and superficial tissues (eg., burns, avulsions, etc.,).
e) Abdominal and Pelvic injuries.

Look : For telltale abrasions, contusions which indicate the site of impact and the possible organ injured. For movements with respiration.

Feel : For tenderness, guarding, rigidity and rebound tenderness. The site of maximum tenderness may indicate the possible organ injured. Increasing tenderness or rebound tenderness implies a need for exploration.

Percuss : Is there free air under the diaphragm ?
Is there free fluid in the flanks ?

External Genitalla : Look for the presence of blood at the meatus which indicates urethral injury.

Pelvis : Compression and distraction tests for evidence of pelvic fracture must be done. A pelvic fracture implies that a massive force is involved in the injury and this is frequently associated with injuries to the bladder and urethra, pelvic haematomas and diaphragmatic rupture.

Flank Tap : On either side, is done when bleeding into the peritoneal cavity is suspected. Free flow of non-clotting blood proves haemoperitoneum. A Negative tap is of no clinical significance.

INVESTIGATIONS :

1. X-rays : Do not contribute much to the management of the injured abdomen. Hence, shifting to the Radiology department should be done only if :-

i) The patient is not in shock.
ii) There is no suspicion of internal bleeding.
iii) Accompanied by a doctor.

Remember always that when a spinal injury is suspected the patient should be moved AS LITTLE AS POSSIBLE. A cross table lateral X-ray of the abdomen is the best film to look for free air in the peritoneal cavity. If it can be taken, a Chest X-ray is useful to look for pneumoperitoneum and diaphragmatic hernia.

2. Urine : The presence of macroscopic or microscopic haematuria should alert one to the possibility of injury. If there is no urethral injury, a Foley catheter should be passed. If there is haematuria, the kidneys and bladder should be evaluated.

3. Blood : Hemoglobin and PCV should be done to assess the amount of blood loss.

Serum amylase : To look for pancreatic injury.

Standing Instructions :

1. ALL CASES OF SUSPECTED ABDOMINAL INJURY SHOULD BE EVALUATED BY THE SENIOR RESIDENT ON DUTY IN GENERAL SURGERY BEFORE BEING DECLARED TO BE NORMAL.

2. IN ALL SHOCKED PATIENTS CALL THE GENERAL SURGERY TEAM ON DUTY AFTER STARTING RESUSCITATION.

3. SUTURING OF WOUNDS, MEDICO LEGAL FORMALITIES AND X-RAYS ARE SECONDARY TO RESUSCITATION AND CLINICAL EVALUATION.

ALGORITHM FOR ABDOMINAL TRAUMA

TRAUMA

SHOCK

NO

YES

LARGE VENOUS ACCESS

RUSH RINGER LACTATE / SALINE

BLOOD CROSS MATCH

ASSESS HEAD, NECK, CHEST, EXTREMITIES
PELVIS AND SKIN

ABDOMEN:

- abrasions ?
- Stab wounds ?
- lower rib fractures ?
- respiratory movements ?
- tenderness / rebound tenderness ?
- guarding / rigidity ?
- free fluid ?
- flank tap positive ?
- pelvis / urethra injured ?

DECISION : ABDOMINAL INJURY

YES

ADMIT TO SURGICAL I.C.U.

NO

AWAIT SENIOR RES.

DECISION

MANAGEMENT OF ACUTE ABDOMINAL PROBLEMS

(TRAUMA & NON TRAUMA)

MANAGEMENT OF ABDOMINAL TRAUMA

Initial Assessment :

3 Phases

Primary Survey including resuscitation
Secondary Survey
Definitive management

Primary Survey including resuscitation

To detect lethal injuries immediately and manage at highest priority (ABCDE)

Airway :

To have an open, unobstructed airway free of blood and secretions.

- Jaw thrust
- Chin lift
- Finger sweep

If necessary intubate.

Breathing :

Respiratory rate and depth.
If inadequate intubate.

Take care of Cervical Spine stabilization.

Check for crepitation, flail chest, sucking chest wound, absence if breath sounds, deviation of trachea.

Interventions necessary may be

- insertion of needle to relieve pneumothorax
- occlusive dressing for sucking chest wound.
- insertion of chest tube if large haemothorax or pneumothorax is suspected.

Circulation :

Check pulse and heart.
Cardiac arrest - External cardiac massage.
Exsanginuating bleeding - External compression.

2 large bore (14 - 16 G) intravenous cannulae.
Blood group and cross matching.
2 litres of crystalloids (Ringer Lactate)

Blood pressure is low and there is no evidence of blood loss - rule out cardiac tamponade.

Look for elevated venous pressure, low blood pressure and muffled heart sounds.

Disability :

Level of consciousness.
(Glasgow coma scale)

Exposure :

Adequate exposure of patient from head to toe.

** *Take care of cervical spine.*

Secondary Survey

Detailed history.
Thorough clinical evaluation of patient from head to toe including per rectal examination and examination of back.

Continue Resuscitation.

Definitive Management

It depends on the general condition of patient and nature of injuries detected at primary and secondary survey.

Blunt Abdominal Trauma

Haemodynamic instability inspite of resuscitation
Evidence of hollow viscous injury : Clinical
Radiological

↓

Laparotomy

Haemodynamically stable.
No evidence of hollow viscous injury,
equivocal clinical signs

↓

Diagnostic peritoneal lavage

Diagnostic Peritoneal Lavage (DPL) by Open technique

> 10 ml unclotted blood
> 10,000 RBC / mm^3
> 500 WBC / mm^3
Presence of bile, bacteria vegetable matter

↓

Laparotomy

RBC < 50,000 / mm^3
WBC < 100 / mm^3
Absence of bile, bacteria or vegetable
matter and patient is stable

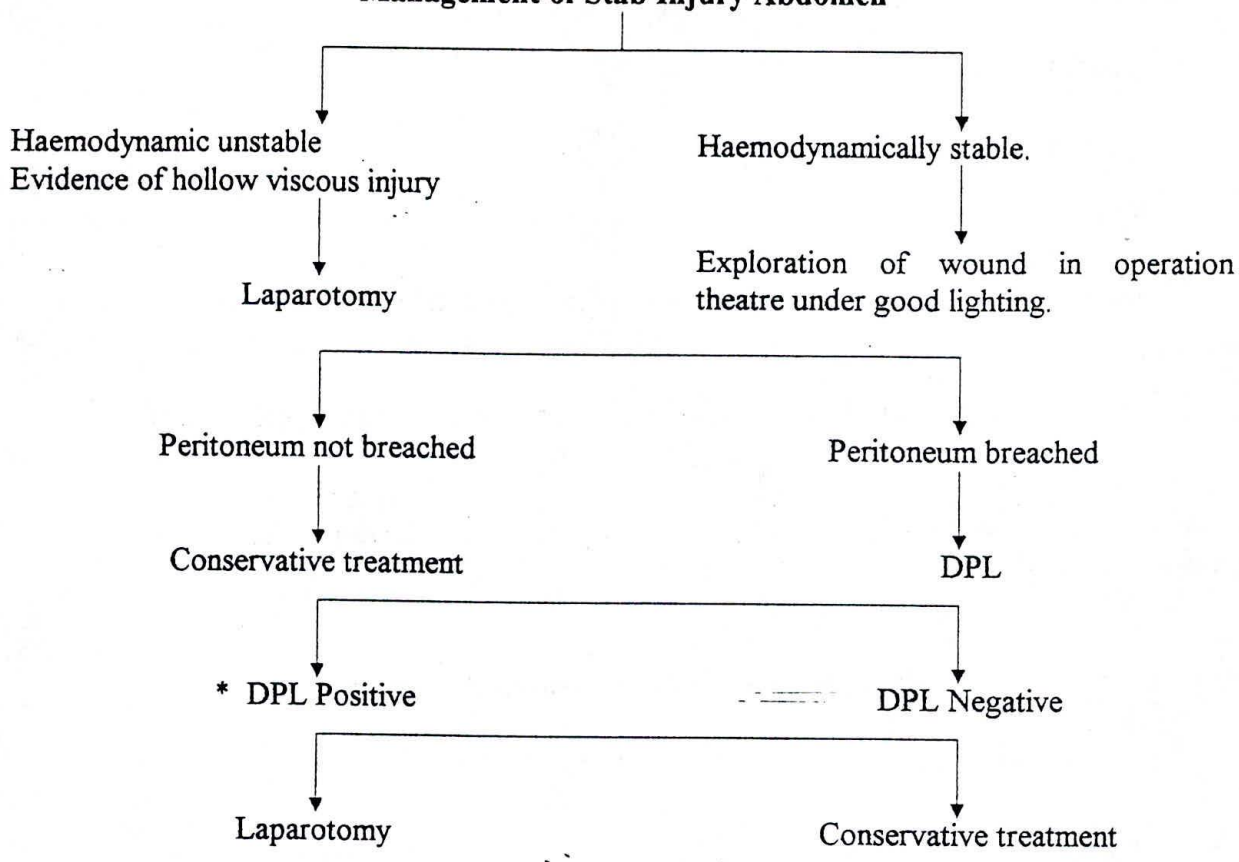
↓

Conservative treatment

Requisites for Conservative Treatment :

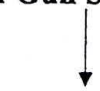
Adequate blood bank facilities.
Adequate personnel for repeated, frequent clinical evaluation of patient.
Intensive care unit monitoring when necessary.

Management of Stab Injury Abdomen

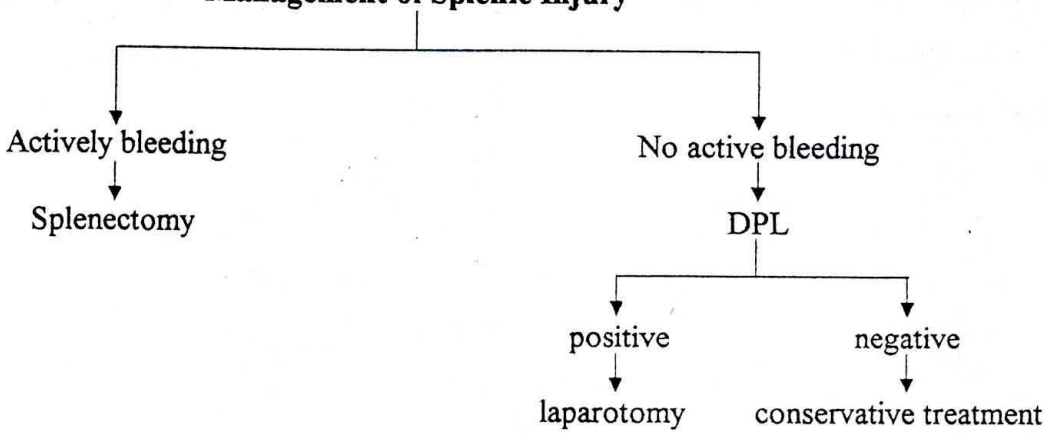


* RBC 1000 / mm^3 is taken as positive for stab injury of abdomen.

Management of Gun Shot injury Abdomen



Management of Splenic Injury



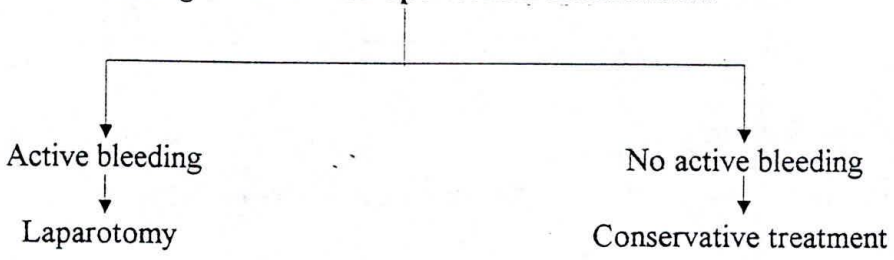
Management of Liver Injury :

Active bleeding
↓
Laparotomy
↓
Pringle's maneuver
↓
Laceration : Ligate individual blood vessels, approximation of laceration edges with horizontal mattress of continuous sutures using absorbable suture material.

More severe injuries : perihepatic packing and referral of a tertiary care centre (needed in 5% of cases).

Note : Ensure safe transfer of the patient to a higher centre after providing basic medical care and adequate fluid resuscitation.

Management of Retroperitoneal Haematoma



Decision making at Laparotomy :

Location	Blunt Injury	Penetrating Injury
Mid line		
Supramesocolic	Haematoma explored	Haematoma explored
Inframesocolic	Haematoma explored	Haematoma explored
Lateral		
Perirenal	Not explored if IVP shows functioning kidneys.	Not explored if IVP shows functioning kidneys.
Pelvic	Not explored if haematoma is not or slowly expanding, arterial pulsation in groin are normal.	Not explored if haematoma is not or slowly expanding, arterial pulsation in groin are normal.

Management of Small Bowel Injury :

Small perforation	:	Closure in 2 layers
Larger involvement of bowel wall circumference	:	resection and EEA 2 layers
Vertical tear in messentery with viable small bowel	-	suture ligation of bleeding vessel.
Transverse tear in mesentery with non viable bowel	-	resection and EEA.

Management of Colonic Injury :

Small perforation due to stab wounds / low velocity wounds :

< 4 - 6 hours old
No gross contamination
No severe blood loss
No evidence of prolonged shock
Young patient.

Right colon - Freshen the edges
Primary closure.

Trasverse colon - Freshen the edges
Primary closure
Exteriorise the sutured part of colon if necessary.

Left Colon - Freshen the edges
Primary closure with proximal colostomy.

Large perforations due to high velocity or blunt injuries

Right colon - right hemicolectomy

Contamination not much and
other criteria as for primary closure
Ileo transverse anastomosis in 2 layers

Gross contamination prolonged shock
ileostomy and mucous fistula

Left Colon - Resection and Hartman's procedure

Transverse colon - Resection and End Colostomy and mucous fistula.

Management of Stomach Injuries :

Perforation - closure in 2 layers
Look for injury on posterior wall, pancreas etc.

Antibiotic Cover :

All patients operated for trauma should receive perioperative antibiotic cover for 3-5 days consisting of ampicillin, geramycin (if urine output is > 30 ml / hour) and metronidazole.

Management of Duodenal Injuries :

Small perforation (< 2 cm)

without gross contamination - primary closure

Blunt / high velocity injuries / stab injuries

Mild :

Stab injury, < 75% of duodenal wall involvement
Duodenal site 3 or 4th part injury
repair interval < 24 hours, No CBD / Pancreatic injury
Debridement and primary closure with gastrostomy and retrograde jejunostomy.

Severe :

Blunt or missile injury, > 75% wall duodenal wall involvement, 1st or 2nd part, injury repair interval > 24 hours, CBD or pancreatic injury.

Debridement and Roux en Y anastomosis, duodenal diverticulization or pyloric exclusion and gastrojejunostomy (for protection of duodenal repair).

Sever injury with uncontrollable bleeding from associated pancreatic injury pack, attain haemostasis and refer to a higher centre.

Management of Pancreatic Injury :

- Contusion and laceration without duct injury - external drainage.
- Distal transection or distal duct injury - distal pancreatectomy and drainage.
- Proximal transection or duct injury - distal pancreatectomy and Roux en Y pancreatico jejunostomy
- Combined pancreatic and duodenal injury - ampulla / blood supply intact - treatment same as that of severe duodenal injury.
- Ampulla destroyed / devascularisation - pack, attain haemostasis and refer to a higher centre.

GENITOURINARY TRAUMA

MANAGEMENT OF RENAL TRAUMA

Introduction :

Emergency urological management may vary according to type of Trauma :

- Anatomic site
- Documentation or suspicion of associated injuries
- Clinical status
- Stability
- Availability of specialty personnel and facilities.

Criteria for suspected Renal Trauma :

1. Penetrating trauma to the flank or abdomen regardless of the degree of haematuria.
2. Blunt trauma to abdominal with gross haematuria.
3. Blunt trauma associated with microhaematuria and shock (BP < 90 mm Hg)
4. Bruising or tenderness over abdomen.
5. Radiographic demonstration of fracture lower rib or lumbar transverse process fracture.
6. Deceleration trauma, RTA and fall from a height may produce
   - Traction or Avulsion injury to renal pedicle.
   - Blunt injury may cause major or minor renal injury.
   - Perinephric or retroperitoneal haematoma.

CLASSIFICATION OF RENAL TRAUMA

Grade I	Contusion	(70%)
Grade II	Laceration - Extravasation	(20%)
Grade III	Pedicle Injury Shattering of kidney	(10%)

Grade I	- Conservative
Grade II	- Requires removal owing to severe haemorrhage (removal of kidney)
Grade III	- Laceration, extravasation will recover spontaneously and requires operation or embolotherapeutic intervention only for haemorrhagic or extravasation complications.

Investigations required for staging of Renal trauma :

1. Excretary urography.
2. Nephrotomography.
3. Computed tomography (CT).
4. Angiography.

Indications for Renal Exploration :

I
- Absolute-persistent bleeding
- Major parenchymal or renal vessel laceration

II
- Relative-urinary extravasation
- Nonviable tissue
- Incomplete staging and
- Arterial thrombosis usually coexist with bleeding.

Total Renal exposure is necessary to

- Evaluate the injury full.
- Excessive bleeding may require that the renal vessels be clamped. If warm ischemia time is expected to exceed 60 minutes, ice slush should be used for cooling.
- The renal pelvis, vessels, parenchyma and ureter must be inspected carefully, as should the site of injury for nonviable tissue and entry into the collecting system.

A) ***Debridement :*** Removal of nonviable tissue is important and done by sharply incising the margins. Approximately 30% of viable kidney will provide sufficient function to avoid dialysis, a useful guideline while considering renal salvage.

B) ***Haemostasis :*** Blood vessels within the renal parenchyma are sutre-ligated with 4-0 chromic sutures. Large veins coursing within the parenchyma can be ligated without worry, owing to the well developed intrarenal collateral circulation.

C) ***Defect Coverage :*** Ideally, the cut parenchymal surface is covered with renal capsule, which provides a seal over the defect to help, prevent delayed bleeding and urinary extravasation. Failing this a pedicle flap of omentum or free grafts of peritoneum can also be used.

D) ***Partial Nephrectomy :*** When the upper or lower portion of the kidney is extensively damaged, partial nephrectomy is called for.

E) ***Renorrhaphy :*** When the middle portion of the kidney is injured or when polar injuries have resulted in only small amounts of devitalized tissue, renorrhaphy can be

undertaken. The nonviable tissue must be excised, the collecting system closed and haemostasis obtained.

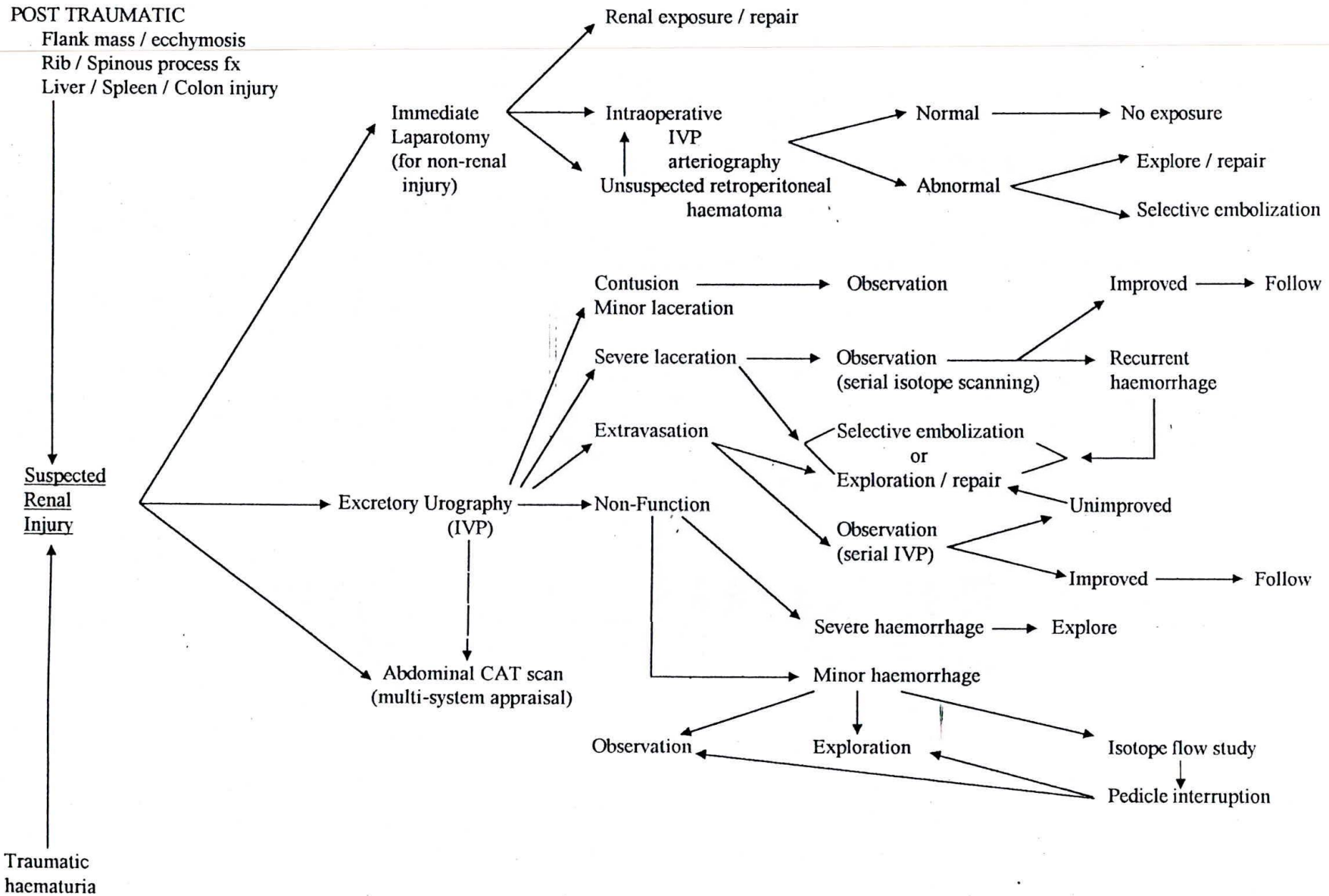
F) ***Vascular Injuries :*** The repair of injuries to the main renal pedicle can be difficult. Venous injuries are difficult to diagnose with current imaging techniques. Injury to the main renal vein can be repaired with 5-0 vascular sutures. There are no symptoms or signs specific to this injury, and haematuria is absent in one third of the cases. Severe multiple associated injuries are present in almost all cases, with a mortality rate of 44%. Radiologic evaluation with intravenous urography shows non-appearance of the involved kidney. CT reveals nonenhancement of the kidney except for the periphery of cortex from collateral circulation (rim sign), and renal arteriography shows occulusion of renal artery or its branches. Even with vascular repair, the function of the kidney is not restored to normal. Even with vascular repair, the function of the kidney is not restored to normal. Late hypertension is found in 50% of patients with main renal artery thrombosis managed conservatively.

Renal Trauma : Management Protocol

	Probable Grade of injury	Management
Haemorrhage (flank, urine)		
Transitory	1 - 2	Observation
Sustained	2 - 3	Embolization / surgery
Intermittent / delayed	2 - 3	Bedrest; embolization / surgery
Extravasation		
Transitory	2	Observation
Sustained	3	Drainage; surgery
Urography		
Function present	1	Observation
Segmental (polar)	2	Observation; embolization / surgery
Function absent	3	Observation; surgery

The potential deleterious consequences of renal trauma are well known. Various complications of renal trauma are as follows :

- Unnecessary exploration / repair; erroneous staging; unexpected retroperitoneal haematoma.
- Haemorrhage (persistent, delayed).
- Extravasation (fistula, urinoma, pseudocyst, hydronephrosis).
- Infection (pyonephrosis, pyelonephritis, abscess, sepsis).



Silhoutte of management of suspected renal trauma.

- Hypertension (Vascular, infarction, compression).
- Renovascular (thrombosis, aneurysm, arteriovenous fistula).
- Infarction (segmental, total).
- Calcification (Calculi, dystrophic).
- Pain.
- Death (Haemorrhage, sepsis) 0.8-4%.
- Association with non renal injury (pancreas, liver, spleen, colon, bowel, vena cava).

Management of Urethral Injuries

Urethral trauma can be divided into two parts : (i) Management of proximal (anterior) rupture of the urethra; (ii) Management of distal (posterior) urethral rupture.

Proximal rupture of the urethra :

Partial tears and complete dismemberment of the prostatomembranous urethra can accompany pelvic fractures or may be without it. When a patient arrives in the emergency room, having suffered enough trauma to fracture the pelvis and tear the posterior urethra, other obvious injuries often take precedence in management, such as shock and intrabdominal injuries. When someone notices blood from the urethral meatus, or is unable to pass a urethral catheter, the possibility of a urethral rupture is appreciated. Since there is a high morbidity associated with rupture of the posterior urethra, the patient deserves the best possible approach and the initial evaluation should be performed with care so that the situation will not be further complicated. Post urethral injury is seen in 5-10% male patients with fracture pelvis. A clinical classical triad to diagnose the urethral injury is blood at the urethral meatus, inability to void, a palpable bladder. The usual mechanism of injury is a violent force that disrupts the soft tissue rather than to laceration of the urethra or prostate by a bony spicule. The posterior urethral injuries are classified in three types :

Type I	Intact, but stretched posterior urethra, may be associated rupture of pubo-prostatic ligament;
Type II	Rupture of membrano-prostatic urethra above the uro-genital diaphragm;
Type III	There is a disruption of membranous urethra and may be extension in to proximal bulbous urethra with disruption of urogenital diaphragm itself or both. In the evaluation of a patient suspected of posterior rupture of urethra, one is often unable to palpate the prostate, since it is in a "high-riding" position on rectal examination. There may be boggyness in pelvis, presence of perineal haematoma and swelling of genitalia. Public and ischial rami are fractured with a shearing action which cleaves off the urethra at the junction of the prostatic urethra and superior edge of the urogenital diaphragm.

Diagnostic catheterization is to be condemned because it may convert partial tear in to complete disruption, particularly if a catheter guide is used. It also increases the risk of haemorrhage in prostatic bed and there is also a possibility of infecting sterile haematoma. The most important diagnostic step is the retrograde urethrogram. If there is doubt of combined bladder and urethral injuries, intravenous urogram followed by cystogram, would be worthwhile. In an isolated urethral injury bladder is often full, therefore trocar cystostomy should be done, so as to divert the urine without instrumenting the urethra. A cystogram can then be performed. Commonly there is a teardrop or parachute deformity of the bladder due to a pelvic haematoma and extravasated urine. There is a greater controversy over whether to pass a catheter, immediately operate to pass a catheter, attempt primary reapproximation, or simply wait for a delayed repair. The devastating morbidity arising from a posterior urethral tear includes stricture formation, incontinence and impotence. The various opinions concerning the proper management of this trauma have arisen from attempts to reduce the incidence of its sequalae.

Stricture :

Stricture formation is common even after skilled and careful management. The usual indications for primary realignment are when prostrate is attached to displaced pubic fragment, bladder lies high in pelvis with large pelvic haematoma and wide urethral gap, concomitant rectal, bladder and bladder neck injuries. Stricture formation results because of a gap between the two ruptured ends of the urethra and a filling-in of fibrous tissue. To prevent this, muscular layers and adventia of the urethra must be brought in to contact. Simple trocar cystostomy and delayed urethroplasty is another option. It is our experience that the difficulty of achieving a satisfactory primary repair, even in the best hands, justifies a no-touch technique.

Incontinence :

Urinary incontinence has been variously reported after this injury with the vesical neck being the primary source of continence. Incontinence may be avoided with correct initial management.

Impotence :

Impotence occurs after rupture to the posterior urethra, but the incidence and exact mechanism are not well known. Patients may experience a return of erections 6 to 19 months after injury, so final assessment should probably delayed at least two years. Emission and ejaculation are even more likely to be impaired than erections. Approximately 25 percent of those with complete posterior urethral tears will end up with permanent impotence and various levels of partial impotence may afflict others. One of the series has compared results of various methods or repair.

Repair of Urethral Rupture :

Method	No. of Patients	Stricture	Incontinence	Impotence
I.	128	11%	16%	26%
II.	119	5%	5%	8%
III.	36	16%	0%	4%

I Two stage urethroplasty in failed cases of conventional realignment or primary E-E anastomosis.
II Initial SPC + Delayed two stage urethroplasty.
III Initial SPC + Delayed one stage urethroplasty.

In AIIMS, we follow the protocol of initial trocar suprapubic cystostomy, followed by assessment of urethral injury at 3-4 weeks and then definitive urethroplasty is performed.

Distal Urethral Rupture :

Trauma to the anterior urethra occurs infrequently. Even with multiple severe injuries to the pelvis, it is unlikely that the distal urethra will be traumatized due to its lack of fixation. Straddle-type injuries account for most trauma to the bulbous portion of the urethra. A laceration or incomplete separation of urethral mucosa allows extravasation of urine during voiding. Buck's fascia represents the first line of compartmentalisation and prevents the wide damage of the periurethral phlegmon due to urine dissecting to the limits of Colle's fascia. When Buck's fascia is intact, the urine is confined to the penile shaft, but if it has been ruptured the extravasation can spread widely in to the scrotum and perianal regions as well as into abdomen. It is prevented from going in to the thighs because of Scarpa's attachment to the fascia lata. Anterior urethral injuries may be due to blunt trauma (straddle) or because of penetrating trauma. These are classified in to three categories :

I. ***Contusion :*** Clinical features of urethral injury but the result of urethrography normal.
II. ***Partial :*** Extravasation of contrast from the mid bulbous urethra but continuity maintained.
III. ***Complete :*** Continuity is completely disrupted.

The usual mechanism in straddle type of injury is that bulbous urethra and surrounding corpus spongiosum get crushed against inferior aspect of pubis. Initial recognition of the extent of injury to the distal urethra is difficult. There may be blood at the tip of penis accompanied by swelling and bruising and voiding is usually difficult, but, frequently a clear urine may appear. Patients often do not seek medical care until the swelling and discomfort are severely disabling, often as long as 72 hours after the trauma, so it is not unusual to be dealing with septic patient by that time. Coverage with antibiotics and drainage of extravasated urine are basic to management. If there is no clinical evidence of extravasation, a retrograde urethrogram may define a small tear and then treatment would consist only of a urethral catheter and antibiotic coverage. If there is a bridge of urethral tissue intact across the laceration or rupture of the urethra, spontaneous

regeneration and reformation of the urethra will occur. Trocar cystostomy to drain the urine will suffice to treat the more extended but nonpenetrating tear. However any penetrating injury with a missile or sharp object deserves open depridement with suture reapproximation of the urethral tissue. Spatulation of the ends of the torn urethra often will prevent stricture formation. Some urologists observe a trocar suprapubic cystostomy is still better alternative for complete urethral disruption and partial tear. After 2 to 3 weeks retrograde urethrogram should be done to assess the situation, then accordingly definite plan should be made. On following strategies subsequent management should be done according to situation :

1. Excision of stricture and end to end anastomosis (≤ 1 cm)
2. Excision with patch graft (1 cm +)
3. Full thickness tube graft (x cm)
4. Two stage urethroplasty
5. Optical Internal urethrotomy / core through visual internal urethrotomy.

The problems involved in definitive urethral reconstruction should never be underestimated. The potentially complicated nature of a pelvic fracture urethral injury is entirely apparent. Thus, surgeons with a general urologic training who do not have both a special additional and ongoing experience of reconstructive procedures and a particular aptitude for the problems involved must be advised that "having a go" is not in the best interests and is essentially a personal matter because many contrarily conceived procedures work quite satisfactorily in the hands of others.

BLADDER TRAUMA

Usually there is a high incidence of associated injuries with bladder rupture from external trauma. In many instances, treatment of other injuries must take precedence over management of the urinary tract.

Extraperitoneal Bladder Ruptures

Extraperitoneal bladder ruptures secondary; to blunt trauma are caused by fractures of the bony pelvis 95 percent of cases.

Blunt Trauma :

In the cases of blunt trauma associated with bladder rupture, the amount of force necessary to cause the injury may be quite small, especially if the bladder was full. Intra-peritoneal rupture usually results with direct trauma to full bladder, but in those cases associated with pelvic fractures, extraperitoneal rupture is more common. The most likely mechanism through which the bladder is ruptured in pelvic fractures is either a sudden increase in intravesical pressure or compression against the bony pelvis with perforation by bony fragments.

Penetrating Injuries :

Penetrating injuries to the bladder are usually intraperitoneal and have a high incidence of associated injuries, the most common being colon and small bowel. It may also be associated with urethral injuries.

Diagonosis :

Usually patient complains of lower abdominal pain and tenderness. Examination often reveals bruising in the lower abdominal region, and on palpation, there is abdominal tenderness and muscle guarding or rigidity. With contusion injury, the patient can void, and haematuria is present. With a bladder rupture, the patient is usually unable to void and a specimen obtained by catheter reveals haematuria. In a severely injured patient who has a fracture pelvis, a urethral catheter should be passed to determine if haematuria. In a severely injured patient who has a fracture pelvis, a urethral catheter should be passed to determine if haematuria is present. If blood is seen at meatus, a retrograde urethrogram should be done to rule out urethral injury.

A delayed presentation may be as acute abdomen, absence of voiding and elevated blood urea nitrogen. Macroscopic or microscopic haematuria is the indication for radiologic evaluation of urinary tract. The intravenous pyelogram is performed first, so that an occult lower urethral injury is recognised before a large amount of dye has extravasated from the bladder obscuring the urethral injury. In male, a retrograde urethrogram should be performed prior to the cystogram to

rule out urethral rupture. A stress cystogram with post emptying films. The technique consists of filling the bladder through a foley catheter under gravity until the contrast material ceases to flow. A picture is taken to evaluate the presence of extravasated dye. If none is present, a further 50 ml is introduced into the bladder, an additional films are made in the anterior-posterior and both oblique projections. The bladder is then copiously irrigated with sterile water or saline, and a repeat film is taken to make sure that no dye has extravasated either in front or behind the bladder. With extraperitoneal ruptures the dye will extravasate around the bladder in the pre and perivesical spaces. With intraperitoneal rupture the dye extravasates in to both paracolic guters and can outline loops of bowel.

Treatment :

Isolated extraperitoneal bladder rupture especially small one, can easily be handled by 2 weeks of Foley catheter drainage. If there is large rupture or patient is to be explored for associated injuries; open the bladder, do not disturb pelvic haematoma, repair the rupture, and insert a surprapubic tube. A cystogram should be done after 10-14 days before removing the suprapubic tube.

Intraperitoneal rupture of bladder

This is an injury of violent deceleration. Road traffic accident, penetrating wounds from knives or gunshot injury, comprise the most of intraperitoneal rupture. It may also be associated with pelvic fractures. The abdomen may be diffusely tender. A voiding cystourethrogram in a conscious patient or a static film of bladder with 250 ml of contrast, often reveal the picture. Delayed picture leads to prerenal azotemia, individual may show a slight acidosis with a higher than 10:1 ratio of the blood urea nitrogen to the creatinine. Exploration of bladder and repair in two layers is desired. A cystogram is done at 2 weeks, if no extravasation, the suprapubic tube should be clamped and patient is asked to void normally.

EXTERNAL GENITALIA INJURY

Injuries to the external genitalia (the penis, scrotum and / or testicles), can present complex problems to the urogenital surgeon. Therapeutic goals include adequate cosmetic repair, the preservation of fertility, and the preservation of sexual potency.

Penile Injuries :

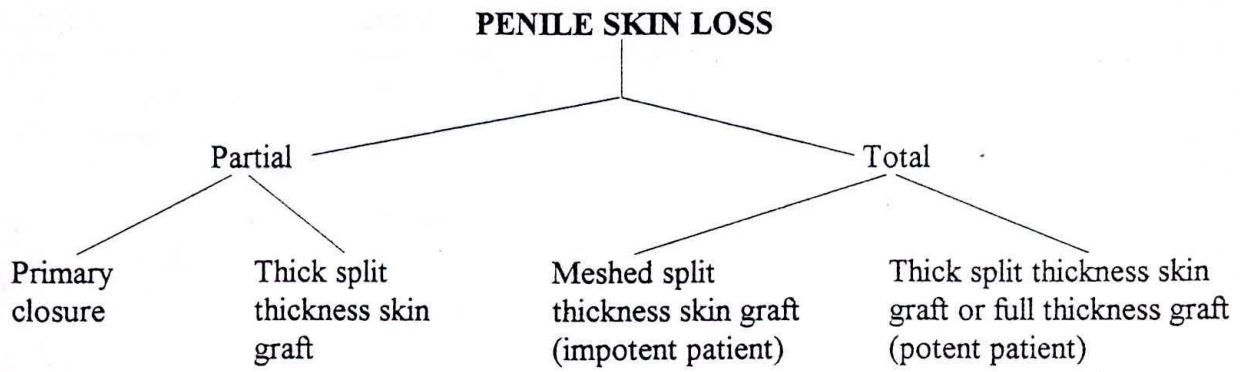
Most traumatic injuries have occured to the erect penis as a result of direct blunt trauma that bends the organ abnormally. It usually occurs during sexual foreplay or intercourse when the penis is suddenly deviated either laterally or downward, in the erect state. A cracking noise is usually heard with subsequent pain and swelling of the shaft of the penis secondary to haematoma formation and deviation of the penis to the side opposite the fracture. In the most of cases, the diagnosis is easily made on the basis of typical history and physical findings. However an accurate

diagnostic tests i.e., cavernosography, ultrasonography, MRI may be required in few cases. Blood at the meatus, any degree of haematuria, and difficulty in urination are suggestive of associated urethral injury, which occurs in 10 to 20% of patients. A review of literature suggest that early surgical repair of the tunica albugenia defect is associated with lower risk of penile angulation, a shorter hospital stay and more rapid functional return. If there is minimal haematoma and no extracorpal extravasation, conservative treatment may be warranted.

Complete urethral disruption is best managed by primary repair, whereas partial disruption can be treated adequately by trocar cystostomy.

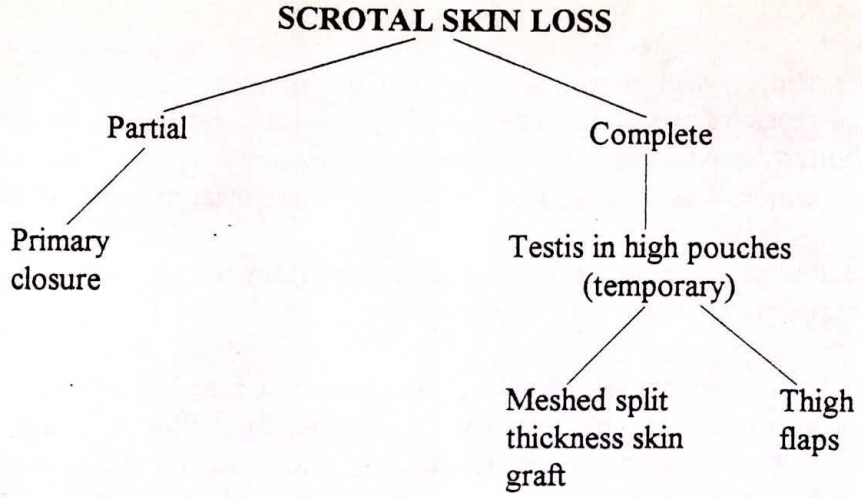
Complete transection of part or all of the penis is a rare occurrence. The treatment depends primarily on the amount of time that has elapsed from time to injury to the time of surgery and how much of the penis has been transected. Microvascular reimplantation procedures give good result with minimum postoperative complications. Although microreplantation offers the best results in the case of penile amputation, if it is not available the older corporal reattachment techniques should be offered.

Avulsion of all or part of the penile skin can occur in mechanical accidents, infections, burns (electrical, chemical or thermal) or be self inflicted. For minor loss of the skin of the penis, a scrotal flap has been used with good results. For more extensive skin loss, split thickness skin grafts have yielded the most consistently satisfactory results. It is important in applying the grafts that the penis be in the erect state or stretched so that a chordee will not develop with inadequate skin coverage.



Scrotal Injuries :

Loss of scrotal skin is found in similar situations to that causing loss of penile skin. When the amount of scrotal skin lost is small and the edges can be approximated over the testicles, primary closure is performed since the scrotum has enormous powers of regeneration. When the scrotal skin has been totally avulsed, several options are available. Firstly, the testicles can be placed in to thigh pockets. Secondly, the testicles with their neurovascular bundle and the vasa deferentia can be pushed up and fixed in the inguinal canals. In both cases, the testes can either be left in position or brought down at a later time and placed in a scrotum reconstructed from split thickness skin grafts or flaps of skin rotated from the medial thighs.



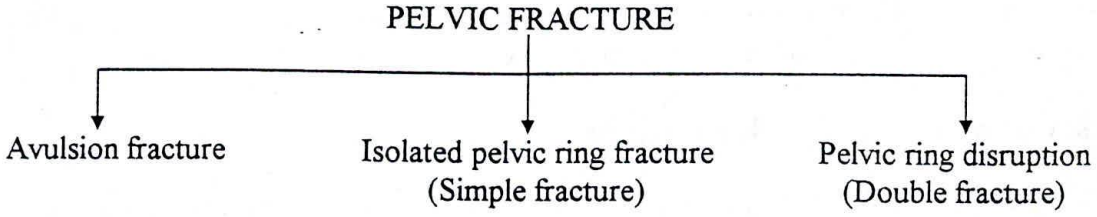
Testicular Injuries :

The tunica albuginea of the testis can be violated with extrusion of the seminiferous tubules by either blunt or penetrating trauma. Any patient with this type of injury should have immediate exploration with excision of the extruded portions of seminiferous tubules and closure of tunica albuginea. Rupture of testis secondary to blunt trauma is a violent and immediately painful event. Nausea and vomiting are frequently associated as occasionally is syncope.

The definitive diagnosis of testicular rupture depends on surgical exploration, which is called for, whenever there is question of a rupture. Scrotal ultrasonography can demonstrate whether disruption has occured (useful for surgery) and it can evaluate the testicular parenchyma. The objectives of surgical exploration are testicular salvage, prevention of infection, control of bleeding and reduction of convalescence. Complete avulsion of one or both testicles can also happen rarely with accidental trauma.

PELVIC FRACTURES

Depending on whether or not the pelvic ring is disrupted to produce a loss of its integrity and capacity as a basin.



Avulsion fracture :

A portion of bone which gives attachment to the concerned muscle becomes fractures and detached from parent bone. It mainly occurs in Athletes and adolescents.

Mechanism : Violent contraction of muscle attached to the bone.

- Anterior-superior iliac spine - Saratorius.
- Anterior-inferior iliac spine - Rectus femoris.
- A portion of Ischeal tuberosity - strong hamstring muscle contraction.

Clinical Features : Local tenderness, pain in the fractured site.

X-ray shows avulsion of apophysis

Treatment : Few days of bed rest.

Isolated pelvic ring fracture (Simple fracture) :

It breaks the continuity of pelvic ring and the integrity of the pelvis is not disrupted.

Sites :

(1) Unilateral fracture of the pubic rami one or both on one side only.
(2) Fracture of the body of ilium.
(3) Minimal sub luxation of symphysis pubis or sacro-ileac joint. Displacement is slight and intrapelvic viscera hardly damaged.
(4) Fracture of the acetabilum with central dislocation of the hip.

Mechanism : Direct blow to the side of pelvis or front of the pelvis.

Clinical features : Patient cannot walk after the injury with or he can walk with extreme pain.

On Examination : Bruising of the affected side, tenderness over the affected region present.

Treatment : (a) Fracture of ilium and pubic rami - bedrest.
(b) With central dislocation of the hip - skeletal traction should be applied.
(c) When acetabular fracture with large portion of the roof or posterior wall detached, open reduction and screw fixation is necessary.

Pelvic ring disruption (Double fracture) :

In this, displacement is considerable, the chance of injury to intrapelvic organs is more.

Mechanism : 3 types of forces cause this injury. They are

(a) Compression force :

- Lateral force from both sides may fracture the side wall of pelvis on both sides. This injury may be associated with medial displacement of hip.

- Anterioposterior compression force may fracture both pubic rami on both sides. The central segment will be pushed back wards damaging the urethra.

(b) Hinge force : This force is applied from front to one side of the pelvis. Such force is seen in RUN OVER ACCIDENTS. One half of the pelvis is usually affected and the symphysis pubis is forced apart in front where as in the back there is usually fracture at Sacro-iliac region, so that the whole pelvis is opened up and the other half of the pelvis remains intact.

(c) Vertical force : Like previous force, this comes from down upwards. Such force usually occurs when an individual FALLS FROM A HEIGHT on ONE LEG. The affected side of the pelvis fractures on two places, the pubis in front and ileum behind. The portion of the pelvis lying in between the two fractures is pushed upwards.

Clinical features :

- Patient is unable to move after injury,
- The patient is not able to pass urine after injury,
- Patient complains of pain on movement even on coughing,
- Internal haemorrhage is considerable so much so to produce SHOCK.

On Examination :

1) Affected area will be seen with bruisings and swelling of the affected region.
2) Tenderness will be diffuse, that is not of diagnostic value, Suprapubic tenderness will be there.

3) The urethra and urinary bladder should be investigated properly to exclude any damage to these organs in those regions.
4) In Hinge force fracture, the gap may be felt in symphysis which is abnormally large.
5) In Vertical force, there is possibility of damage to nerve roots and particularly to Sciatic nerve. This should be excluded.

X-ray pelvis is diagnostic and indicates the type of fracture. From the type of fracture one can assess the type of injury force.

(a) In compression injury, both pubic rami are fractured on both sides.
(b) In Hinge force fracture, there is an abnormal gap at the symphysis pubis and there is a fracture near or sub luxation at the sacro-iliac joint.

In vertical force fracture, the pubic rami and post portion of ilium are fractured on same side with upward shift of the segment of pelvis between the fractures.

(1) Treatment of Shock : Corrected immediately

I V fluids → saline
I V fluids → dextrose
Blood transfusion
Plasma expanders

(2) Treatment of Pelvic vascular injuries : Mainly the urinary bladder and urethra are ruptured, when pubic rami are fractured. This can occur even when the symphysis pubis is forced apart as in the hinged type of injury.

- Patient is asked to pass urine, if can pass urine and if urine is clear - watch the patient for subsequent changes in urine.
- If patient is not able to pass urine - pass a catheter.

Intrapelvic rupture urethra is the commonest injury in intrapelvic in case of pubic rami fractures.

Treatment : Suprapubic cystostomy, sound is passed from above. By rail roading methods a self retaining catheter is introduced into bladder. The ruptured urethra is now approximated and sutured. Bladder closed a d kept retropubic space s closed in layers.

Urinary Bladder supture :

Extraperitoneal → Repair
Introperitoneal → Repair

Treatment of Fracture :

1) Compression type of fracture - only bed rest for 3 weeks, No reduction, hip joint movements allowed, after 3 weeks allowed walking, Later on spinal exercise is allowed.
2) Hinge type of fracture - requires reduction under General Anaesthesia and is rolled to affected hole. A compression force is applied to the affected pelvis so that it gradually closed. After reduction a firm binder or lumbosacral corset applied.

If reduction fails, open reduction, internal fixation. The wire is passed through drill holes or through obtrutor foramine to bring the 2 pubic bones together. Patient remains in bed for 3 weeks. Lower limb movements allowed during this period. Then patient is allowed to walk with crutches. Union take place 6 weeks. Corset is worn for another 3 weeks.

Verticle force fractures

Under general anaesthesia fracture is reduced by pulling the leg of the affected side. This will bring down the fractured segment in line with the other side of pelvis. Skeletal traction is applied and is continued for 6 weeks till the fracture unites. Traction is removed and the patient is allowed to move with crutches. Weight bearing is not allowed for 3 months from the date of injury.

Complications of fracture pelvis :

1) Intrapelvic haemorrhage - oligemic haemorrhage to injury.
2) Paralytic ileus - pelvic veins, shock
3) Urogenital damage (Urinary bladder and urethra damae) most important complication.
4) Persistent sacro iliac pain.
5) Sciatic nerve injury particularly in vertical force fractures. Such nerve injury usually recovers.
6) Malunion of fracture results pelvic deformity and difficulty in labour.

Sacro - coccygeal injury

Fracture sacrum.
Fractur coccyx.
Sprain in SIJ.

Mechanism : Blow from behind usually causes fracture sacrum fall on the buttocks causes fracture of coccyx and sprain of SIJ.

X-ray : Highly significant and diagnostic.

Fracture of sacrum → No displacement

Fracture of sacrum → Displacement → lower segment is pushed forwards.

Fracture of coccyx - the lower segment is angulated forwards.

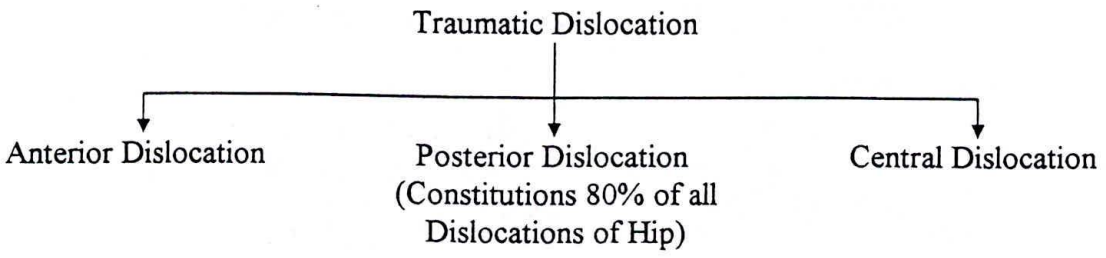
X-ray does not reveal the fracture - then sacro-coccygeal sprain.

Treatment : If displacement present - displacement should be tried per rectum.
Pain during sitting - sorbo cushion used while sitting.
If pain not relieved - local anaesthetic injection to the tender spot or excision of coccyx should be done.

FRACTURE & DISLOCATIONS OF THE LOWER EXTREMITY

TRAUMATIC DISLOCATION OF THE HIP JOINT

It is one of the type of Acquired Dislocations.



Dislocation of hip is relatively uncommon because -

1. The head of the femur is well covered by acetabular cavity, which is further deepened by the acetabular labrum
2. There are strong ligaments reinforce the capsule of hip joint, particularly ILIOFEMORAL LIGAMENT.
3. There are strong muscles outside the joint to protect it.

Mechanism : The posterior dislocation of the hip is flexed and adducted as in this position, the head of the femur lies against the posterior part of the capsule which is relatively weak and posteriorly the acetabular cavity is also deep. In this position, if a force is applied along the long axis of the femoral shaft, the head will be dislocated over the posterior lip of acetobulum, internally rotating the head of femur at the time of impact.

Eg. (1) This type of force may occur in CAR ACCIDENT, when after applying sudden break or after the CAR HAS HIT AGAINST A TREE OR AN IMMOVABLE OBJECT, the FRONT SEAT PASSANGER in a CAR is thrown forwards so that his knee strikes against the dash board and the HEAD OF FEMUR, DISLOCATES POSTERIORLY.. This is known as 'DASH BOARD DISLOCATION'.

If the hip is adducted, dislocation may occur without fracture.

If the hip is in only flexed position and not adducted, the impact is liable to fracture the rim of the acetabulum.

(2) Eg. Posterior dislocation may occur if a weight falls on the back of a person in stooping position, due to fall of roof.

Types of posterior dislocation of hip :

(a) Gluteal Type : In this, head of the femur lies above the piriformis muscle and under cover of glutei.

(b) Sciatic Type : Below the piriformis, near the greater notch. Sciatic nerve may be injured.

(c) Regular Type : In this, the Ileofemoral ligament, (the strongest ligament in the body) may remain intact even, when the dislocation of the hip has taken place.

(d) Irregular Type : In this there is slight tear of the lateral limb of the ileofemoral ligament and when the ileofemoral ligament is COMPLETELY TORN - it is called IRREGULAR TYPE of post dislocation.

Clinical Features of post dislocation :

- Shortening of leg.
- Hip is held slightly FLEXED, adducted and internally rotated.
- Pain is less - if acetabulum is fractured.
  Pain is more - if acetabulum is not fractured.

Signs :

(1) The head of the femur lies on the dorsum ilii. The kidney of the head is noted by the fact, that it moves with the rotation of the shaft. The Medial Condyle looks to the same direction as the head of femur.

(2) Palpation is femeoral artery at groin is difficult in this case due to absence of head of the femur behind the artery.

(3) If the Bryants Traingle is drawn - the base of the triangle is shortened in the posterior dislocation.

(Bryants triangle is drawn by joining the Anterior superior, iliac spine with the greater trochanter and by a line drawn vertically downwards from the anterior superior iliac spine). The greater trochanter is raced towards the anterior superior iliac spine due to internal rotation of the limb.

(4) The tip of greater trochanter lies above the 'NELATONS LINE'. A line drawn from the most prominent part of the ischial tuberosity to the tip of anterior superior iliac spine. Normally this line touches the tip of the greater trochanter.

(5) Moris Bitrochanteric test indicates the distance between the outer border of greater trochanter to the symphysis pubis. This will indicate the medical displacement of the greater trochanter in posterior dislocation of hip.

(6) Sciatic nerve function must be tested as injury to the sciatic nerve may occur in post dislocation of hip, particularly in sciatic type.

(7) Movements at hip are restricted.

X-ray confirms diagnosis. Look for associated fracture of the posterior acetabular rim or not, femoral head, neck, shaft - SHENTONS LINE is always disorted.

Complications :

(a) Avascular necrosis of femoral head occurs with in 8 hours of injury, occur approximately in 15% of cases, incidence increase sharply with delay in reduction. X-ray appearance of avascular necrosis reveals it self about 2 to 3 years after injury.
(b) Sciatic Nerve injury - occurs in 10% of cases, peroneal part of sciatic nerve commonly affected, recovery is usual and treatment is a foot drop splint should be given.
(c) Post dislocation hip with other fractures post (rim of acetabulum, head of femur, neck of the femur), open-reduction and internal fixation.
(d) Late oesteoarthritis of hip following dislocation.
(e) Myositis ossificans - passive movements at hip should be prohibited after injury. Hip joint should be rested in hip spica.
(f) Unreduced dislocation - if not reduced more than a few weeks, its reduction becomes difficult even by operation, and a vascular necrosis of head is inevitable. Then the choice remains between replacement of the head by a prothesis and subtrochanteric oesteolomy.

Treatment :

Reduction :- Anaesthesia - general anaesthesia with muscle relaxant key to success, is complete muscle relaxation.

Position of the patient :- Transfer the patient on a stretcher canvas on the floor.

Procedure :

An assistant studies the pelvis of the patient when the patient is fully relaxed after giving the muscle relaxant. The knee and hip are flexed gently to right angle. At the same time the adduction and internal rotation deformities are corrected, so that the head of the femur is now lying directly behind the acetabulum what is needed is a pull to the thigh vertically upwards. This can be best performed by griping the patients leg between the surgeons knees and resting the surgeons forearms on his thighs with his knees and thigh flexed to 45°. During the pull of the

thigh upwards, an attempt should be made to gradually externally rotate the femur and to abduct is to slightly. Reduction usually occurs with a 'clunk'.

After reduction, it is always necessary to examine the stability by pushing the femur downwards. Gross instability is an indication for open reduction and internal fixation of acetabular fracture.

Check radiographs - patient should always be taken in 2 planes to confirm perfect reduction and to know about the associated fracture in post-reduction film.

Other methods of reduction : If the above method fails, Bigelow's method may be tried. It is popular. After flexing the abduction of the hip joint, it is smoothly externally rotated. It is then gradually extended and pulled steadily. As extension progresses, the externally rotated limb is turned into neutral position.

Immobilisation :

Whether or not the dislocation has been properly reduced, a skeletal traction is applied through the tibial tubercle to give rest to the hip joint. This pull is given usually with 15 pounds weight and is maintained for 3 weeks.

Rehabilitation :

Active use of hip should be encouraged as soon the patient is relieved of pain even with traction. After the traction has been taken off the patient may be allowed up with crutches. Weight bearing is not allowed before 8 weeks.

Oesteo-orthrosis usually develops irrespective of treatment.