Volume

1

25 Years
Silver Jubilee Edition

Manipal Manual of SURGERY

As per the latest **NMC** Guidelines | **Competency Based Medical Education (CBME)** Curriculum under Graduate Medical Education Regulation

Seventh Edition

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25 Years Silver Jubilee Edition

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Seventh Edition

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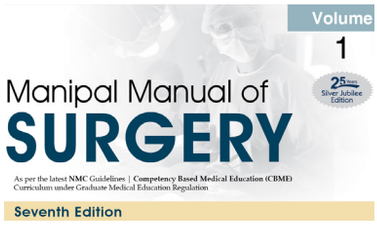

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to
our children
Ranjan
and
Rachana

Forewords

It is both an honor and a privilege to write this foreword for the *'Silver Jubilee Celebration'* and the seventh edition of the *Manipal Manual of Surgery* by Dr K Rajgopal Shenoy. Over the last 25 years, this manual has established itself as an indispensable resource for medical students, surgical residents, and practising surgeons, alike across the India.

The *Manipal Manual of Surgery* has always stood out for its clarity, comprehensiveness, and practicality. It reflects Dr Shenoy's vast clinical expertise and his passion for teaching, which resonates in every chapter. This new edition builds on the strong foundation of its predecessors, incorporating the latest advancements in surgical science and technology while maintaining its hallmark accessibility and relevance to real-world clinical practice.

Incorporation of many videos of clinical examinations and procedures is really something very exciting, and I am sure will help to pass on the art and science of clinical medicine to the next generations. The use of illustrations, flowcharts, and case-based discussions further enhances its value as a learning tool to provide a practical approach in day-to-day clinical settings.

This edition is particularly significant as it comes at a time when surgery is witnessing rapid transformation, with innovations in minimally invasive techniques, robotic surgery, artificial intelligence, and enhanced recovery protocols shaping the way we care for patients. Dr Shenoy has ensured that these advancements are seamlessly integrated into the book, making it a truly contemporary and future-ready guide for students and professionals.

As the President of the Association of Surgeons of India, I take great pride in witnessing the impact of this manual on shaping the future of surgery in our country. Resources like the *Manipal Manual of Surgery* play a pivotal role in upholding the standards of surgical education, ensuring that our young surgeons are well-prepared to tackle the challenges of modern healthcare.

I congratulate Dr Rajgopal Shenoy and his team for their relentless dedication to advancing surgical education. This edition is a testament to their commitment to excellence, and I have no doubt that it will continue to inspire and guide countless readers in their journey towards becoming skilled and compassionate surgeons.

With best wishes for continued success.

Dr Pravin Suryawanshi
President, Association of Surgeons of India
Professor and Head, Department of Surgery, Deputy Dean, CEO
MGM Medical College and Hospital, Aurangabad, Maharashtra

I am truly delighted and immensely proud to see my close friend and well-wisher, Prof Rajgopal Shenoy, coming out with this edition of his renowned book, *Manipal Manual of Surgery*.

Looking back, I fondly recall my journey with him as a surgical colleague—participating in numerous academic programs, serving together as co-examiners in various examinations, and cherishing our camaraderie as true friends in the Association of Surgeons of India for over two decades.

Having had the privilege of reviewing some chapters of the latest edition, I must highlight the way he has tackled the topic of postoperative complications. His approach is remarkably simple yet methodical, ensuring that even the most junior medical graduate can grasp these critical issues with ease and clarity.

I take pride in the fact that the first edition of this book was launched in 2000 on my native soil, Madurai. Prof. Shenoy's inherent talent for teaching and his unique style of imparting knowledge have always been remarkable.

As the Vice President of Association of Surgeons of India, I deeply appreciate his invaluable contributions to academic events conducted for postgraduate students across the country. His dedication has significantly enriched surgical education in every form.

I am confident that this edition will be a boon to undergraduate students, postgraduate trainees, and junior surgeons alike.

Prof (Dr) D Maruthu Pandian
Former Dean, Madurai Medical College, Tamil Nadu
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I am extremely glad to note that the seventh edition of *Manipal Manual of Surgery* is about to get published. A book with seven editions and lasting for more than 25 years stands in testimony to its popularity and acceptance amidst the readers of science of surgery. It is difficult to keep pace with the exponential increase in the knowledge and hence bringing out this edition at frequent intervals, and including contemporary concepts in every edition and making it updated remains a huge task. The contributors and editors of the book have done constant and effective work to make the seventh edition a truly new edition.

The 21st century has witnessed a huge change in accrual of knowledge, its understanding and assimilation and onward communication. Influx of electronic media has almost shown the print media its grave. Today knowledge is available to all at any time and at any place. Use of high resolution pictures and videos provide real-time learning.

Reading texts and journals, usage of libraries and preparing notes have gone out of fashion. Yet, *Manipal Manual of Surgery* finds its unique place in the minds of its readers.

The book conforms to the CBME criteria laid down by the National Medical Council. The editors have added chapters on communication skills, research topics, bio-hazard disposals, postoperative complications, clinical evaluation of acute abdomen. At the end there are many attempts to pose clinically oriented problems which stimulates the readers to apply their minds to solve them.

I hope this book will be received with more acceptance by the scientific world as in the past.

Dr Santhosh John Abraham
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It is an honor and great pleasure for me to pen a few words regarding the seventh edition of the *Manipal Manual of Surgery* Prof Rajgopal Shenoy, a doyen in surgery and one of the most popular teachers across the globe.

To remain relevant, concise, up to date with crisp revision of the contents and meeting the expectations of learners and teachers alike is indeed a great challenge, that too consecutively for seventh time. I bow to his enthusiasm and hard work. Consistency, pursuance and perseverance are reflected in this edition. Though considered as a book for mainly undergraduates, this manual has always met the needs of postgraduates too. We, as Surgeons need to acknowledge contribution of Dr Anita Nileshwar in all editions. This edition will have video snippets and many more innovative learning contents which will be of significant use to all the learners. I congratulate Prof Rajgopal Shenoy for his contribution and wish him all the success.

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Preface to the Seventh Edition

Manipal Manual of Surgery (MMS) started with humble beginning of notes which got cyclostyled, then Xeroxed, and then saw the first edition in 2000 which was then called the *millennium edition*. In 2025, we celebrate 25 years of *MMS* in print which students read all over India and in over 20 other countries too. Change in the pattern of understanding the syllabus and the requirements by the authorities who regulate medical colleges, we also made a few changes three years back, released sixth edition and it was aptly titled as CBME edition. Thousands of readers and ardent followers of *MMS* have appreciated the simplicity, originality and easy to understand language, scoring very high marks too in the examinations. *Then why this new edition and what are the changes?*

"Those who cannot change their minds cannot change anything."

"Change is inevitable". —George Bernard Shaw

Post-Covid, we have seen tremendous change amongst students in their preference to read digital content. However, it remains a fact that as of now learning that takes place best when it is taught effectively and energetically in the classrooms and in the bedside.

In this new edition, we have corrected a few mistakes. We have edited the book effectively based on the requirements of undergraduate and postgraduate students. Topics have been rearranged to get a better understanding of the subject. Management-oriented discussions are included. A new chapter on congenital anomalies of the genitourinary tract and postoperative complications is added for a comprehensive understanding of the subject. High quality images have been added. "A picture is worth one thousand words"– someone said, and it is true. From the very first edition of the book, one of the major strengths of the book is high quality original photographs which explain so much about the disease and presentation. Clinical case capsules are added with the aim of improving interpretation skills.

Problem-based cases are also given in digital contents. A good volume of digital content has been incorporated in this book which will help students in theory and clinical examination also. Digital content includes video recording of topics, PowerPoint presentations and videos of clinical signs and a few operative conditions. Let this silver jubilee edition bring better hope and inspiration for your future studies and competitive examinations. We are sure you will emerge with flying colours not only in the MBBS but also in other competitive examinations.

Enjoy reading *Manipal Manual of Surgery* seventh edition with digital content, the access to which is given free to the buyers of this book through scratch code.

K Rajgopal Shenoy

Anitha Shenoy (Nileshwar)

Preface to the First Edition

I developed interest in surgery and teaching due to my eminent teachers (role models) in my undergraduate days at Government Medical College, Bellary (1977–1983) and postgraduate days at Kasturba Medical College, Mangalore (1983–1986). Kasturba Medical College, Manipal, gave me enough opportunities, encouragement, and confidence to develop interest in teaching. The genesis of the manual is attributed to the tremendous response to my lecture notes in surgery. The book has been named as a manual as it is complete, comprehensive, precise and simple.

Manipal Manual of Surgery is written, keeping in mind the time constraint and vastness of the subject, that the student has to face before an examination. Reference books are not essential to learn basic facts of surgery. The standard books are undoubtedly necessary to gain in-depth knowledge as well as understanding of the subject. Every student should refer to them whenever necessary. This manual is best utilized as a revision and handbook that gives precise facts in a clear, concise way that makes a student feel that surgery is after all not an insurmountable obstacle to pass the final MBBS examination.

One may wonder what is the necessity of another book when you have so many established books in the market? With the voluminous advances in surgery the **textbooks have virtually become jungles** where students get lost. I have made this book simple, easily understandable with flow charts, tables and key boxes. The manual covers the text, clinics and coloured photographs. Relevant clinical cases kept for clinical examination are discussed similar to bedside clinics. At the same time, some important and illustrative clinical problems discussed in this manual are actual case reports. Speciality topics which are written by senior faculty members of our institution add glory to the book. We have tried to shed light through the jungles so that one can come out safely.

I call this as a complete book in surgery, but do not believe in including orthopaedics in it because it is a subject that has developed as a separate speciality. By merely adding some more pages the purpose will not be served when good orthopaedics books are available in the market. The *viva voce* examination is a unique section which helps in reducing your ***anxiety*** before you appear for viva.

I take this opportunity to express my gratitude to all the staff from the department of surgery and specialities who have helped me in one or more ways in the outcome of the book. I should make a special mention of Prof U Santosh Pai who has been a constant source of inspiration to me while I was preparing this manual. It is him who gave me an appropriate title for this book. My wife, Dr Anitha, not only contributed anaesthesiology topics but also helped me in editing this manual.

My close friend and classmate, Dr L Ramachandra, Associate Professor in the department of surgery who has helped me in editing many chapters and has given me valuable suggestions. Dr Gopinath Pai, Dr Sreedharan. Dr Shashidhar Matta (Assistant Professor), Dr Shashikumar, (Urologist), Dr Gautham Prakash, Dr Divya Shenoy, Dr Juliana Samuel, Dr Krishnamurthy, Dr Anand Nadkarni (Interns) also have contributed to the completion of the book.

I sincerely thank Mr Umesh Acharya from dental college for drawing simple and beautiful diagrams, Mr Parashuram Bhat and Kiran from photography section for the coloured photographs. **I believe that students should spend more time in looking at those simple line diagrams and photographs which can often illustrate more than the text.**

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I greatly appreciate the constant support and encouragement given to me by Mr SK Jain, Managing Director, Late Mr BR Sharma of CBS Publishers & Distributors, New Delhi and Mr VK Jain, Production Director, deserves special thanks for the excellent production of the book.

I sincerely hope that this *Manual* will be useful to you. Any criticisms, and contributions to this book are welcome. **I wish all my dear students the very best. Do enjoy reading this book.**

K Rajgopal Shenoy

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K Rajgopal Shenoy
Anitha Shenoy (Nileshwar)

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Volume 1

Basic Principles of Surgery
General Surgery
Urology
Specialities

Chapter
48

Neurosurgery

- Head injuries
  - Classification
  - Primary lesions
  - Secondary lesions
- Extradural/epidural haematoma
- Chronic subdural haematoma
- Raised intracranial pressure
- Fracture skull
- CSF rhinorrhoea
- Pott's puffy tumour
- Hydrocephalus
- Brain tumours
- Trigeminal neuralgia
- Brainstem death

Introduction

Head injuries derive their importance because of the fact that many patients who die or who are disabled belong to the younger age groups. Head injuries account for 1% of all deaths, one-fourth of deaths due to trauma and they are responsible for **half of all deaths** from road traffic accidents. Majority of the patients are young, adult males.

PATHOPHYSIOLOGY AND MECHANISM OF HEAD INJURIES

SU17.4: Describe pathophysiology, mechanism of head injuries.

Classification

I. Based on clinical type

1. Open
2. Closed

II. Based on type of injury

1. Blunt injury—acceleration, deceleration
2. Missile injuries

- The term **open head injury** is used to denote a type of injury in which there is a **fracture of the skull associated with tear of the dura and arachnoid**, resulting in cerebrospinal fluid leak either to the external environment or into one of the **potentially infective areas in the base of the skull, e.g. CSF rhinorrhoea or otorrhoea**.
- **A closed head injury** is one where there is **no such leakage**. The advantage of this classification is that it helps the treating physician to recognise a group of patients who are likely to develop an infective complication following the head injury and he can initiate measures to prevent it.
- **Blunt injuries,** depending on the severity of impact, can result in an open or closed head injury. Missile injuries tend to result in an open head injury most often.
- The brain is protected by a bony box which has a **vault** and **base of the skull.** The **base** of skull in contrast to the vault is a **rough terrain** due to the various **bony prominences, ridges** and **foramina**.
- This factor is important in causing extensive brain damage to the brain in acceleration/deceleration type of injuries. In addition to the linear acceleration/deceleration, **rotational acceleration** is also capable of producing damage to the brain as the **brain swirls about** inside the skull. Such injuries result in maximal damage at interfaces between structures of different densities such as **grey matter–white matter junctions**.

Pathology

The pathological changes due to trauma to the brain can be classified into primary and secondary.

I. Primary lesions

- Diffuse axonal injury
- Shearing lesions
- Contusions and burst lobe

II. Secondary lesions

- Swelling, haemorrhage
- Extradural haematoma
- Subdural, intracerebral haematoma
- Infection, SAH

I. Primary Lesions (Key Box 48.1)

A few important primary lesions are discussed below.

- **Diffuse neuronal damage is the most constant** feature of blunt injuries. Immediately after an injury, no changes may be seen but changes begin **after 14 hours of injury and maximum effects** may last up to **one week. Prolonged unconsciousness** may follow injuries which produce only diffuse neuronal damage without any obvious macroscopic changes. Shearing lesions of the nerve fibres account for some severe injuries without any conspicuous changes to naked eye examination of the brain.
- **Cerebral concussion:** Alteration in consciousness without structural damage as a result of non-penetrating traumatic brain injury. There might be loss of consciousness, confusion and amnesia. These are the features. Widespread degeneration of white matter occurs without much changes in the nervous system cortex or brainstem. These patients have spasticity in all four limbs after injury and **when they regain consciousness**, they are found to be severely demented (Key Box 48.2).
- **Contusion and burst lobe** are the obvious naked eye changes seen after injuries and were thought to be the main injuries before diffuse neuronal damage and shearing lesions were described. Contusions are seen on the summit of the gyri which get injured against the bone. **The overlying pia is torn and the blood seeps into the subarachnoid space.** A bleeding cortical vessel may result in the formation of the **acute subdural haematoma or intracerebral haemorrhage. Brain oedema** which develops surrounding the contusion and lacerations is the one that determines the outcome. Most often contusions are seen at the **poles of the frontal and temporal lobes, under surface of frontal and temporal lobes, over corpus callosum, superior and anterior surfaces of cerebellum and anterior surface of brainstem**.

Key Box 48.1

Primary Lesions

- Diffuse neuronal damage
- Cerebral contusion
- Cerebral laceration

Key Box 48.2

Cerebral Concussion

- Temporary physiological paralysis of the nervous system
- Loss of consciousness
- Post-traumatic amnesia
- Recovery may be complete
- Some can develop complications

II. Secondary Lesions (Key Box 48.3)

- **Brain swelling:** This is a vague term applied to increase in brain bulk due to both oedema and venous congestion. It is aggravated by hypoxia or respiratory insufficiency which may be due to associated lung injury or obstruction to upper respiratory passages. Sometimes such a swelling can lead to severe brain compression which is difficult to relieve, since there is no single mass lesion.

Pearls of Wisdom

Malignant cerebral oedema has close to 100% mortality. This is more common in children.

- **Intracranial haemorrhage:** Extradural or subdural haemorrhages may develop as a clean cut secondary event, even though bleeding may have started at the time of injury. These cause compression of brain, secondary rise in intracranial pressure and can cause death, if not detected and treated early.
- **Infections:** All open head injuries are liable to result in intracranial infection either as generalised meningitis or focal infection such as **subdural empyema or brain abscess, osteomyelitis of skull**. After closed head injuries, infection of a subpericranial blood clot may result in **Pott's puffy tumour.** When infection supervenes on an already injured brain, it may retard the recovery or may even lead to death. Hence, it becomes mandatory to treat all infections vigorously.

Key Box 48.3

Secondary Lesions

1. Brain swelling
   - Oedema
   - Venous congestion
   - Hypoxia
2. Intracranial haemorrhage
   - Extradural
   - Subdural SAH
3. Infections
   - A. Open head injury
     - Generalised meningitis/fulminant meningitis
     - Subdural empyema
   - B. Closed head injury
     - Pott's puffy tumour

Cause of Death in Head Injuries

- It is instructive to consider the pathological findings in fatal cases and to speculate the **deaths** which might have been prevented. For example, earlier many deaths which had occurred as a result of aggravation of brain swelling **due to hypoxia** could have been prevented by **ventilation** and **anti-oedema measures**. It should

be emphasised that the role of the treating physician is to anticipate and take appropriate measures to prevent the patient from succumbing to the secondary injuries (damages—seizures, hypoxia). **In extensive primary damage to the brain, apart from supportive treatment, one may have to wait and hope**.

- Extensive injury to vital areas such as diencephalon, or patients with **diffuse damage** are **not likely to survive**. These are patients who are unconscious from the time of injury with bilateral, dilated, fixed pupils, flaccidity in all 4 limbs and autonomic disturbances.
- Sometimes a head injury associated with extensive injuries to chest, abdomen or the limbs by their sheer severity can cause death.
- Intracranial complications such as haematomas, brain swelling, infection, and extracranial complications such as chest injury/metabolic abnormalities, if recognised and treated early, can go a long way in saving the life of the patients.

INTRACRANIAL HAEMATOMA

- Most of the head injuries are mild or minor and irrespective of how they are managed, the patient recovers on his own. All those who are unconscious, even if briefly, run the risk of respiratory obstruction. Some of the so-called trivially injured run the risk of developing an intracranial haematoma. Hence, all head injuries must be taken seriously. A **complicated head injury** is one where anyone of the **secondary pathological changes** may occur and threaten the life of the patient. **Uncomplicated** head injury is one where no **such events** occur. However, it could be a severe one where the unconsciousness is prolonged.
- These haematomas could develop in any one of the planes intracranially. Extradural (epidural), subdural, intracerebral haematoma, or a haemorrhagic contusion.
- The clinical presentations of these haematomas are due to either increase in the intracranial pressure or due to signs of cerebral compression. In the case of acute subdural haematoma or intracerebral haematoma, the clinical picture and the outcome of treatment is also dependent on associated brain damage, age of the patient, time of presentation and GCS at presentation.

EXTRADURAL/EPIDURAL HAEMATOMA (Fig. 48.1)

The clot collects between the dura and the inner table of skull. A majority of them occur in the **middle cranial fossa,** since injury to **middle meningeal vessels (vein and artery) is the commonest cause.** However, about 20–25% of the extradural haematomas can occur in the frontal, parietal regions, at the vertex or in the posterior fossa. Injuries to the dural venous sinuses or a large diploic venous channel are the other causes for

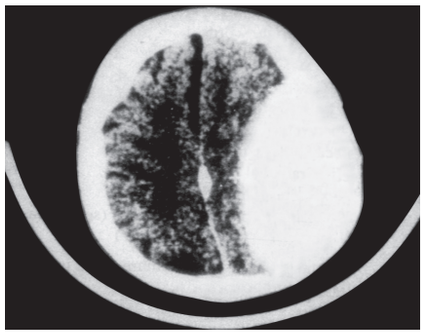

Fig. 48.1: CT scan showing extradural haematoma

the formation of a haematoma. Depending upon the source of bleeding, the haematoma could collect rapidly (hyperacute type) or slowly over a period of a few hours to a few days and present as a **chronic calcified lesion**. 60–80% of these patients have an associated fracture of the skull bone and only a few of them may present with classical symptoms with **lucid interval**. In the remaining patients, the initial picture can vary from an unconscious state to a fully conscious person with or without a history of post-traumatic amnesia. With the widespread availability and use of CT scan, the diagnosis has become much simpler nowadays. However, a few clinical features are worth mentioning.

SU17.5: Describe clinical features for neurological assessment and GCS in head injuries.

Neurological assessment by Glasgow Coma Scale

1. Eyes open
   - Spontaneously 4
   - To speech 3
   - To pain 2
   - None 1
2. Best verbal response
   - Oriented 5
   - Confused 4
   - Inappropriate words 3
   - Incomprehensible sounds 2
   - None 1
3. Best motor response
   - Obeys commands 6
   - Localises the pain 5
   - Withdrawal to pain 4
   - Flexion to pain 3
   - Extension to pain 2 (severe damage with increase of ICP)
   - None 1

- Total score is 15; minimum score is 3. Any patient who has a coma score of 8 or less than 8 is said to be in coma.

1. Deteriorating Consciousness Level

- This is one of the hallmarks for the diagnosis of intracranial haematoma. The term **'lucid interval'** is used when a patient recovers from an initial period

of unconscious state. Though in earlier days, this was said to be associated with intracranial haematomas, it can occur in other conditions such as brain oedema, multiple contusions. To assess the consciousness level properly, instead of using vaguely defined terms such as semiconscious, obtunded, etc. the **'Glasgow Coma Score'** is widely used, to avoid observer errors in the observation of such patients.

- **Restlessness** in a previously quiet patient indicates **increasing intracranial pressure**, which again needs to be investigated. At the earliest appearance of focal neuronal deficit, the patient has to be taken up for exploratory burr holes.
- Progressive neurological deficit indicates cerebral compression and the manifestation may depend on the area of the brain affected.

2. Pupillary Abnormalities

These are to be considered as a late manifestation. It is due to pressure by the **herniating** uncus of the **temporal lobe on the ipsilateral third nerve at the tentorial hiatus**. In the **early stages** due to irritation of the nerve, there is a sluggish response to the light source. This is called early third nerve and is associated with neurological obtundation, bradycardia and constriction. Since, it is a transient phenomenon, the early constriction goes unnoticed most of time. The patients are often detected in the **next stage**, i.e. **pupillary dilatation,** caused by **paralysis of pupilloconstrictor fibres in the third nerve**. However, if the cerebral compression is unrelieved, this may go onto bilateral. Pupillary dilatation is due to **ischaemia of third nerve nucleus** at the midbrain which is caused by pressure on the posterior cerebral artery. These series of pupillary changes have been termed **Hutchinsonian pupils.** The dilated pupil has a definite localising value in that if an exploratory burr hole has been decided upon, it should be done on the side of the initially dilated pupil.

3. Autonomic Disturbances

Bradycardia, though said to be a definite sign, is a late and not an early sign. Initially, there may be a rise in the pulse rate (tachycardia) which may progress to bradycardia, when the systolic blood pressure increases. At times there may be a rise in the diastolic pressure also. These **changes occur due to changes in the cerebral blood flow as a consequence of increased intracranial pressure.** Respirations become deep and slow rate (bradypnoea) and later patients may develop Cheyne-Stokes ventilation due to brainstem ischaemia.

4. Non-localising Signs

Kernonhan's notch—contralateral pupillary dilatation 6th nerve and 8th nerve palsy in posterior fossa lesions and CVJ anomalies.

Pearls of Wisdom

Cushing's triad of increased intracranial pressure (ICP)

- Bradycardia
- Hypertension
- Irregular respiration

- Local scalp swelling is seen in more than half of the cases. Thus, examination of the head for any such swelling becomes important.
- Some of these patients may have a stiff neck either due to increased intracranial pressure or due to associated injury to neck muscles. Mild fever may, at times, occur and this sometimes confuses the observer. In such a case, the patients must be investigated with a definitive investigation like CT scan. If much time is not available, one should not hesitate to proceed to exploratory burr holes or a 'trauma craniotomy flap' has to be employed to rule out a haematoma.
- Though in adults, 'shock' is a rare complication of head injury, in children with intracranial haematoma and associated cephalohaematomas, due to volume depletion 'shock' may be encountered. Even in adults, if there is a large scalp injury which is not sutured immediately, shock can occur.
- Posterior fossa haematomas in any plane are dangerous because of the lesser space available for the haematomas. As a result of this, rapid brainstem compression can occur which may prove fatal. The availability of CT scan has made detection of these so-called 'unusual haematomas' more frequent. In a suspected case, even if **facilities are not available,** the treating physician should **explore the posterior fossa**, if the clinical features suggest haematoma, or if the skull X-rays show a fracture line extending across the occipital bone towards the foramen magnum.

Investigations (Key Box 48.4)

SU17.6: Choose appropriate investigations and discuss the principles of management of head injuries.

As has been pointed out earlier, the advent of CT scan of the head has made the diagnosis easier and more specific. However, it should be emphasised that **in the absence**

Key Box 48.4

Indications for Skull Radiology

- Loss of consciousness
- Obvious depression on the skull
- Compound fracture
- Laceration or contusion of the scalp
- Focal neurological signs

of CT scan, if adequate clinical features point out to the **possibility of an intracranial haematoma**, the patient must be taken up immediately for an exploratory surgery rather than wait and allow him to develop irreversible brainstem damage. Since 60–80% of patients with an intracranial haematoma have a skull bone fracture, irrespective of his consciousness level has to be observed for at least 24–48 hours. Occasionally, one may have to resort to old investigations such as angiography not only to establish the haematoma but also to rule out associated vascular anomalies.

TREATMENT OF HEAD INJURIES IN GENERAL

I. Resuscitation and Support

1. Admission is indicated when

a. Definite history of unconsciousness
b. Fracture temporal bone
c. Person who cannot be attended by the doctors immediately, i.e. no medical facilities nearby.
d. Post-traumatic seizures—patient should be admitted.

2. Casualty reception

a. Airway

- Mouth gag—to prevent tongue falling backwards.
- Endotracheal intubation with positive pressure ventilation. Hypoxia is an important cause of cerebral oedema which worsens the level of consciousness.

b. General assessment of patient

- To rule out abdominal injuries such as splenic rupture.
- Haemothorax—may need an intercostal tube.
- Long bone fractures

c. General assessment of the degree of shock by pulse, blood pressure monitoring and treatment.

d. Neurological assessment by Glasgow Coma Scale

Total score is 15; minimum score is 3. Any patient who has a coma score of 8 or less than 8 is said to be in coma.

II. Care of the Unconscious

a. Ryle's tube aspiration or feeding (in the absence of skull base fractures)
b. Care of the eyes—padding
c. Catheter for drainage of urine
d. Change of position to avoid bedsores.
e. Intubation for elective ventilation/airway protection

III. Surgical Treatment for Extradural Haematoma

Immediate surgery for removal of haematoma and relief of cerebral compression is a must. Extradural haematoma, in particular, is a neurosurgical emergency and patient survival will depend upon the speed with which the compression is relieved. It is not an exaggeration to state that even if decompression has to be done with unsterile instruments, at the bedside it may be worth the effort. In every neurosurgeon's career, at least one such situation might have occurred and a live patient may justify the means employed. Once consciousness is lost, pupils are dilated and decerebrate rigidity and periodic breathing develop, it may be only a few minutes that may be available to save the life of the patient and one should not wait and waste time. In the case of extradural haematoma, the **outcome is dependent on the size of the haematoma and the stage in which** the patient was taken up for surgery. In the case of acute subdural and intracerebral haematoma, it depends on associated brain damage. If the associated brain damage is very severe, patients succumbs to the brain damage (Fig. 48.2 and Key Box 48.5).

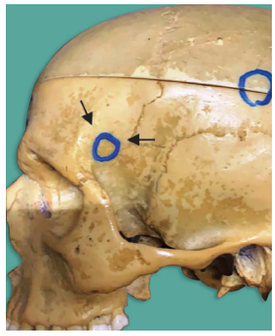

Fig. 48.2: Burr hole site

Key Box 48.5

Extradural Haematoma

- 3 cm vertical incision immediately above the midpoint of zygoma
- Strip the pericranium
- Burr hole with Hudson's brace
- Evacuate 'black-currant jelly' clot
- Extend the burr hole and control bleeding middle-meningeal artery by bipolar diathermy
- Dural hitch sutures to prevent stripping of dura

Acute Subdural Haematoma

Impact damage is more as compared to epidural haematoma. There is associated underlying brain injury. Symptoms are due to compression of underlying brain with midline shift, in addition to parenchymal brain injury.

Causes

- Parenchymal laceration bleed
- Torn cortical bridging vessel

May occur in people who are on anticoagulant therapy.
Mortality—50–90%
Outcome is better, if surgery is done within 4 hours.

CHRONIC SUBDURAL HAEMATOMA

- Common in old people.
- In the elderly, the distance between the dura and the brain increases because of the shrinkage of the

brain. Even a minor trauma can tear the cortical veins resulting in collection of blood.
- Bleeding is never progressive and the blood in the subdural space slowly forms membranes and the inflammatory cytokines cause seepage of fluid into this potential space causing increase in ICP and symptoms compress the brain causing features of raised intracranial pressure (ICP).

Clinical Features
- Elderly patients with history of minor trauma
- Bilateral headache, mental apathy
- Slowness, confusion—later alteration in the level of consciousness may progress to unconsciousness
- Waxing and waning of the level of consciousness is seen in some patients. If such a history is elicited, one should always suspect chronic subdural haematoma.
- Unilateral weakness (contralateral), alterations in speech, seizures.

Diagnosis
CT scan or, if feasible, MRI scan are the ideal investigations (cerebral angiography had been used and is still being used in some centres where access to the latest imaging facilities are not available).

Treatment
- Burr hole and drainage of the haematoma usually under local anaesthesia or occasionally under general anaesthesia is often the practised mode of treatment.
- At times, the patient may need two or more burr holes to ensure adequate evacuation.
- If the brain fails to expand and obliterate the cavity, especially in older people or in persons with a very thick inner membrane, a large craniotomy and wide excision of the subdural membrane has to be carried out to remove the constricting effect.
- Adequate bedrest and plenty of fluid administration are also important postoperative measures.
- Ideally, 2 burr holes and a posterior wick drain is placed for 24 hours, with 3 days of bed rest and adequate hydration.

RAISED INTRACRANIAL PRESSURE
Normal ICP is 8–12 mmHg.

Measures to Reduce the Raised ICP
Aim is to keep ICP 20 mmHg
- Head and elevation up to 30°
- Hyperventilation
- Sedation—with or without muscle relaxant
- Use of diuretics—furosemide, mannitol
- Thermoregulation
- Use of barbiturates—thiopentone—reduces brain metabolic rate
- Maintaining fluid and electrolyte balance
- Seizure control
- Steroids in severe head injury are associated with increased mortality and should not be used. (On the contrary, they are used for reducing vasogenic oedema secondary to tumours).

FRACTURE SKULL

Anterior Fossa Fracture
1. Fracture cribriform plate can result in CSF rhinorrhoea.
2. Fracture may extend to the orbit—subconjunctival haemorrhage.
3. Olfactory nerve involvement—partial anosmia.
4. Optic nerve may be contused or fracture may involve the optic foramen resulting in partial or total loss of vision.
5. Rarely, 3rd nerve palsy gives rise to dilated pupil. Traumatic mydriasis
6. Raccoon eyes.

Middle Cranial Fossa Fracture
1. Epistaxis due to fracture venous/sphenoid sinuses.
2. CSF from the ear: Blood mixes with CSF and so, does not clot.
3. 7th nerve palsy.
4. Rarely 6th and 8th nerves are also involved.
5. Battle sign: Discolouration of skin and haemosinus within the mastoid air cells.

Posterior Cranial Fossa Fracture
1. Extravasation of blood in the suboccipital region causing boggy swelling in the nape of the neck.
2. 9th, 10th, and 11th cranial nerves may be involved.
3. **Battle sign:** Discolouration of skin and collection of blood occur in the region of mastoid process.

CSF RHINORRHOEA
- There should be a communication between the intradural cavity (subarachnoid space) and the nose.
- It indicates tear of the dura mainly in the basal region and a fracture involving paranasal sinuses—frontal, ethmoidal or sphenoidal.
- There is always an injury to a small portion of the brain. It (the portion of brain) plugs the tear, preventing the dura from healing. Thus, the rhinorrhoea persists for many days.
- This leads to complication, i.e. infection and meningitis.

- **Two types**
  1. **Traumatic:** It can be iatrogenic following surgery or can be post-traumatic (62 to 80%).
  2. **Nontraumatic spontaneous:** It can be due to high pressure (hydrocephalus) or congenital.
- Confirmation of CSF rhinorrhoea is on the bedside by following clinical signs.
  1. **Ring sign:** Onto linen, ring of blood with 1 layer ring of clear fluid. Halo or target sign.
  2. **Reservoir sign:** Gush of CSF in certain position of the head.
- β_2 transferrin is the most accurate method.
- CSF may be collected and fluid glucose can be assessed to differentiate from nasal discharge.

Treatment

- Acetazolamide 250 mg three times a day.
- Lumbar drainage can be done in the absence of raised ICP.
- Prophylactic antibiotics.
- If the rhinorrhoea persists, repair of the dural defect alone, (or) at times with a shunt procedure will be needed.
- Conservative management may be tried for up to 2 weeks before resorting to anterior cranial fossa floor repair.

POTT'S PUFFY TUMOUR

- This is **subperiosteal infection** usually caused by osteomyelitis of the underlying skull.
- It is common in the frontal region and the **frontal bone is commonly involved.**
- The cause of infection is through frontal sinusitis.
- Another common cause of infection of a subpericranial haematoma following needle aspiration.
- It can also follow **chronic suppurative otitis media.**
- Pus collects in the subpericranial space and extradural plane, which communicate with each other (dumb-bell type abscess).
- It causes a **boggy swelling in the frontal region** and tenderness over the scalp.
- Pitting oedema over the scalp is conclusively called **Pott's puffy tumour**.
- Severe headache, vomiting and blurring of vision should clinch the diagnosis.

Treatment

1. CT scan to confirm the diagnosis.
2. A burr hole and aspiration of pus can be done followed by 6–8 weeks of antibiotics.
3. In chronic cases, the wall of the abscess may have to be removed. The associated osteomyelitic skull bone requires a radical removal under cover of antibiotics.

HYDROCEPHALUS

Refer to Digital Content for Brain Tumours.

TRIGEMINAL NEURALGIA

- Superior cerebellar artery or multiple sclerosis plaque can cause this condition.
- Severe episodic lancinating facial pain occurring in the distribution of V cranial nerve.
- Vascular compression of the nerve near the root entry zone, multiple sclerosis may be causative factors.
- Investigation—MRI.

Treatment

- Carbamazepine or gabapentin.
- 75% will not respond to medical treatment.
- Surgery: Refractory cases
  1. Percutaneous glycerol injection, radiofrequency ablation, thermocoagulation, balloon compression, local nerve block.
  2. Microvascular decompression *via* craniotomy.
  3. Stereotactic radiosurgery.

BRAINSTEM DEATH

Irreversible loss of consciousness, loss of brainstem reflexes and apnoea.

Diagnosis of brainstem death is done in three stages:

1. Identification of the cause of irreversible coma.
2. Exclusion of reversible causes of coma.
3. Clinical demonstration of absence of brainstem reflexes.

Brainstem Reflexes

- Pupillary reaction to light, corneal reflexes, vestibular ocular reflex, cough reflex, gag reflex, motor response to pain.
- Apnoea test—apnoea despite a CO_2 increase to >6.65 kPa or 50 mmHg.
- All reflexes must be absent and are tested independently twice by 2 doctors.

VERY IMPORTANT WISDOM LINES IN HEAD INJURY

- If an exploratory burr hole has been decided upon, it should be done on the side of the initially dilated pupil.
- Bradycardia is due to raised intracranial pressure.
- If hypotension responds to volume replacement in trauma centre in a comatose patient, the coma is most likely not due to head injury.
- Admit the patient, if there is fracture temporal bone.
- Hypoxia is an important cause of cerebral oedema which worsens the level of consciousness.
- Steroids in severe head injury are associated with increased mortality and should not be used.
- CSF rhinorrhoea indicates tear of the dura mainly in the basal region and a fracture involving paranasal sinuses.
- Glasgow Coma Score of less than 8 means patient is in coma.

CT SCANS OF HEAD INJURIES (Figs 48.3 to 48.11)

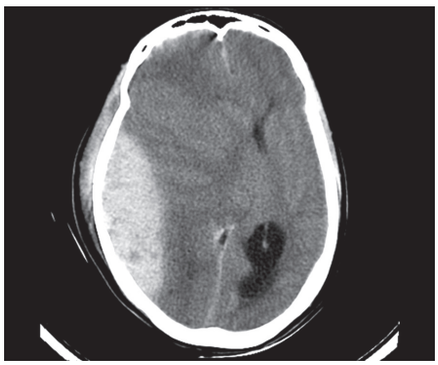

Fig. 48.3: Right temporoparietal extradural haematoma (EDH) showing mass effect with midline shift with subfalcine herniation

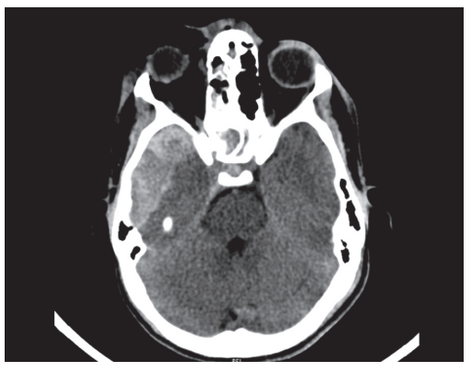

Fig. 48.4: Right temporal extradural haematoma

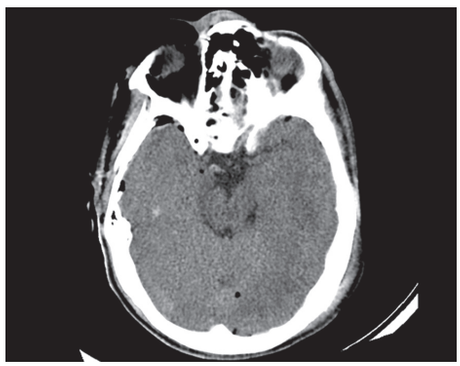

Fig. 48.5: Postoperative scan—no EDH. Post right temporal craniotomy with pneumocephalous

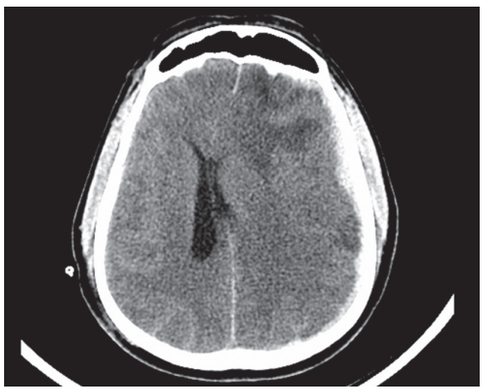

Fig. 48.6: Left frontoparietal acute subdural haematoma. Left frontal and temporal non-haemorrhagic contusion with subfalcine herniation with midline shift

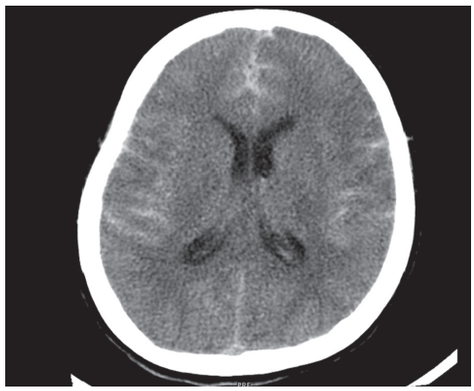

Fig. 48.7: Traumatic right more than left frontoparietal and anterior interhemispheric subarachnoid haemorrhage

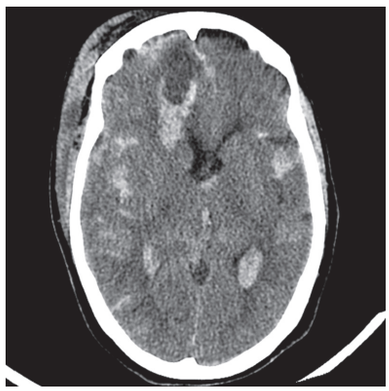

Fig. 48.8: Diffuse axonal injury— right frontal, left temporal contusion, right frontoparietal region—subarachnoid haemorrhage and intraventricular haemorrhage

(*Contributed by* Dr Sunil Upadhyay—Assistant Professor, Department of Neurosurgery, KMC, Manipal)

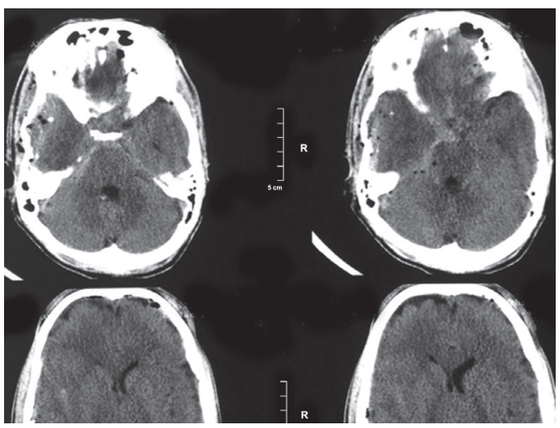

Fig. 48.9: Right temporal depressed fracture with temporal EDH with pneumocephalous

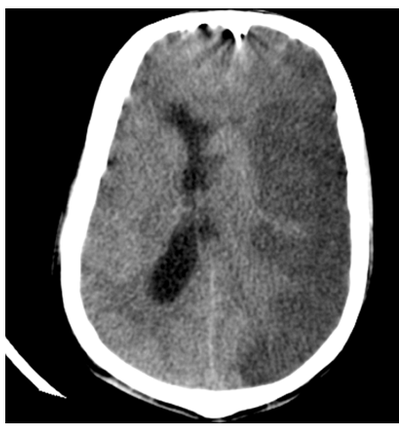

Fig. 48.10: Left frontoparietal hypodence area suggestive of left middle cerebral artery stem infarct

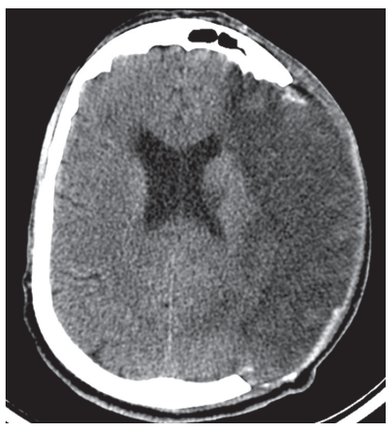

Fig. 48.11: Left FTP decompressive craniectomy brain herniation outside the skull

MULTIPLE CHOICE QUESTIONS

1. Following is not the feature of Glasgow Coma Scale:
A. Eyes opening B. Motor response
C. Verbal response D. Sensory response

2. Closed head injury include following, *except*:
A. Head injury with fracture skull
B. Head injury with black eye
C. Head injury with facial nerve palsy
D. Head injury with CSF rhinorrhoea

3. Which of the following is included under secondary lesions following head injury?
A. Diffuse neuronal damage
B. Contusions
C. Lacerations
D. Swelling

4. Post-traumatic amnesia is a feature of:
A. Raised intracranial tension
B. Fall from a height
C. Fracture skull
D. Cerebral concussion

5. Which is an important cause of brain swelling following head injury?
A. Infection
B. Oedema
C. Acidosis
D. Bleeding

6. The cause of extradural haematoma is bleeding from:
A. Venous sinuses
B. Cavernous sinus bleeding
C. Basal veins bleeding
D. Middle meningeal vessels bleeding

7. Lucid interval is typically seen in:
A. Extradural haematoma
B. Acute subdural haematoma
C. Chronic subdural haematoma
D. Pontine haematoma

8. Which nerve is paralysed after herniation of temporal lobe in extradural haematoma?
A. Oculomotor nerve B. Ophthalmic nerve
C. Trigeminal nerve D. Facial nerve

9. Pupillary dilatation following head injury is due to ischaemia of the third nerve caused by:
A. Middle cerebral artery
B. Posterior cerebral artery
C. Inferior cerebral artery
D. Anterior cerebral artery

10. Following are definite indications for admission in a head injury patient, *except*:
A. Fracture skull
B. CSF rhinorrhoea
C. History of unconsciousness
D. Scalp bleeding

Answers

1. D **2.** D **3.** D **4.** D **5.** B **6.** D **7.** A **8.** B **9.** B **10.** D