ebsite www.jaypeebrothers.com, for detailed information on medicine books, visit our website www.jaypeebrothers.com



Step by Step®

Critical Care

Arun Kumar Paul

2nd Edition

Step by Step®

Contents

Chapter 1. **Respiratory Disorders** **1–28**

Adult respiratory distress syndrome (ARDS) *1*
Severe asthma: Status asthmaticus *5*
Acute exacerbations of chronic obstructive pulmonary disease *8*
Pulmonary embolism *10*
Pneumothorax *15*
Aspiration pneumonitis *20*
Upper airway obstruction *24*
Airway burn *26*
Flail chest *27*

Chapter 2. **Cardiovascular Disorders** **29–58**

Acute myocardial infarction *29*
Congestive cardiac failure *34*
Acute pulmonary edema *39*
Hypertensive crisis *44*
Shock *46*
Cardiac tamponade *53*

Chapter 3. **Neurologic and Central Nervous System Disorders** **59–96**

Head injury *59*
Cerebrovascular accident *65*
Cerebral edema *66*
Status epilepticus *69*
Poliomyelitis *72*
Tetanus *74*
Increased intracranial pressure *77*
Brain death *82*
Guillain-Barré syndrome *84*

Coma *86*
Traumatic spinal cord injury *90*
Myasthenia gravis *94*

Chapter 4. **Liver Diseases** **97–106**
Acute liver failure *97*
Chronic hepatic failure *102*

Chapter 5. **Renal Diseases** **107–119**
Acute renal failure *107*
Chronic renal failure *112*
Some nephrotoxic drugs *118*
Diabetic nephropathy *118*

Chapter 6. **Endocrine Dysfunctions** **120–133**
Thyroid storm/Thyrotoxic crisis *120*
Myxedema coma *122*
Diabetic ketoacidosis 125
Hyperglycemic hyperosmolar nonketotic coma *127*
Hypoglycemia *130*
Acute adrenocortical insufficiency *132*

Chapter 7. **Temperature Illness** **134–141**
Heat stroke *134*
Malignant hyperthermia *135*
Hypothermia *138*

Chapter 8. **Poisoning** **142–159**
Self-poisoning *142*
Barbiturate poisoning *145*
Salicylate poisoning *146*
Paracetamol poisoning *147*

Tricyclic antidepressants poisoning *148*
Lithium poisoning *149*
Narcotic poisoning *150*
Poisoning with various tranquilizers *151*
Organophosphorus poisoning *151*
Alcohol poisoning *152*
Carbon monoxide poisoning *154*
Cyanide poisoning *155*
Poisoning with amphetamines *155*
Cocaine poisoning *156*
Poisoning by toxic irritant gases *157*
Poisoning with caustics *158*

Chapter 9. **Cardiopulmonary Resuscitation 160–168**
Cardiopulmonary arrest *160*

Chapter 10. **Fluid, Electrolyte and Acid-Base Disorders 169–193**
Dehydration *169*
Pure water-deficiency *170*
Water intoxication (excess) *171*
Hypercalcemia *172*
Hypocalcemia *174*
Hyperkalemia *175*
Hypokalemia *177*
Hypernatremia *179*
Hyponatremia *181*
Hyperphosphatemia *184*
Hypophosphatemia *185*
Acid-base disturbances *186*
Metabolic acidosis *187*
Metabolic alkalosis *188*
Respiratory acidosis *190*
Respiratory alkalosis *191*

Chapter 3

Neurologic and Central Nervous System Disorders

JAYPEE BROTHERS

Head Injury

Head injury may be defined as craniocerebral trauma due to injury of skull or brain or both. It is mostly due to a motor vehicle accident, a fall or a direct impact on head and is frequently associated with other injuries such as cervical spinal injury, thoracoabdominal injury and so on.

Head injury may have certain types and basic pathophysiology are as follows:

1. **Closed head injury**: No penetration of the skull and skull cavity not exposed to external environment.
2. **Open head injury**: Cranial contents exposed to external environment.
3. **Coup injury**: Injury of brain directly at the site of impact.
4. **Contrecoup injury**: Injury of brain opposite to the site of impact.
5. **Diffuse injury**: Injury involved the entire brain.
6. **Primary impact damage**: It is mostly due to accleration/deceleration forces applied to the skull contents. There may be contusions of the cortex both beneath the site of impact and contrecoup affecting widely in the brain.

7. **Secondary head injury**: It is due to hemorrhage and edema. Intracerebral and subdural bleeding may occur at the time of injury. Hematoma within the rigid skull may result to increased intracranial pressure and brain shift. Infection may also occur.

   Increased intracranial presure can also cause decreased cerebral perfusion pressure and ischemia of the brain.
8. **Skull fractures**:
   a. Linear fracture: Crack in the skull.
   b. Comminuted fracture: Fragmentation of skull bones.
   c. Depressed fracture: An inward depresion of skull fragments. Compound fracture may be associated with infection.
   d. Basilar skull fracture: Fracture of the base of the skull. It may be associated with bleeding into the nasopharynx with the consequent danger of inhalation of clots. It carries a high risk for cerebrospinal leakage and infection through a dural tear in the ear or nose.
9. **Extradural hematoma**:
   a. History of a blow to the temporal region with loss of consciousness. It is followed by a lucid interval and then again unconsciousness.
   b. Increasing drowsiness, dilatation of pupil on the affected side and hemiparesis on opposite side.
   c. Apnea, other pupil dilates and death occurs.
   d. Evacuation of blood clot through a burr hole is the lifesaving treatment.
10. **Cerebral edema**: Following trauma, there may be cerebral edema increasing gradually. It is usually associated with increased intracranial tension, increasing coma, fixed dilated pupils, and ultimately respiratory failure.

JAYPEE BROTHERS

11. **Changes in cerebral hemodynamics**: Localized brain injury produces local tissue acidosis and vasodilation. These blood vessels become unresponsive to changes in arterial PCO_2 as in normal areas of brain. The effect of hypercarbia is thus to cause vasodilation in normal areas of brain and divert blood from the damaged areas which have a luxury perfusion. Loss of local autoregulation resulting in 'steal phenomenon' if there is hypoxia, hypercarbia, and hypotension. Treatment with IPPV results in hypocapnia and the reverse situation develops. Intracranial pressure is reduced and blood is diverted to perfuse the damaged brain tissue.
12. Brainstem injury interferes with the vital centers and may cause decerebrate rigidity and hyperthermia.
13. **Airway obstruction**: It may be due to unconsciousness, and falling back of tongue, associated injuries such as fracture of mandible or fracture of roof of pharynx resulting in hemorrhage in the pharynx and depression of reflexes and thus more risk of aspiration. Airway obstruction causes straining, hypoxia, and hypercarbia and thus raised intracranial tension.
14. Cardiovascular collapse.
15. Damaged brain cannot tolerate minor insults, fat embolism, hypoxia, hypercarbia, and hypotension.
16. Acute head injury is frequently associated with other injuries including cervical spine and thoracoabdominal injuries. These need careful attention.

Clinical Assessment

1. A full history of trauma.
2. General physical examination. Any other significant trauma.
3. Neurological examination: Mental status, skull and spine need careful examination.

JAYPEE BROTHERS

4. Headache, tingling, paresthesia, and paralysis.
5. Level of consciousness: Fully conscious, confusion, lethargy, stupor, and coma. Rising ICP may often be associated with papilledema and rapidly deteriorating conscious level.
6. Any dysfunction of cranial nerves.
7. The Glasgow coma scale is a standardized scale, accepted all over to assess the severity of head injuries. It is based on eye opening, verbal, and motor response (Table 3.1). Each response on the scale is given a number, high for normal and low for impaired response.

Table 3.1: The Glasgow coma scale

Sign	Evaluation	Score
Eye opening	Nil	1
	To pain	2
	To speech	3
	Spontaneous	4
Verbal response	Nil	1
	Garbled	2
	Inappropriate	3
	Confused	4
	Oriented	5
Motor response	Nil	1
	Extension	2
	Abnormal flexion	3
	Flexion withdrawal	4
	Localizes pain	5
	Obeys	6

The lowest score is 3 and maximum is 15. The responsiveness of the patient is indicated by the total number obtained. In severe head injury a score may be 7 or less.

JAYPEE BROTHERS

8. Size and reaction of pupil:
   a. Pinpoint: Pontine hemorrhage and narcotic overdose.
   b. Small: Drug-related, metabolic coma, and pontine hemorrhage.
   c. Midposition and nonreactive: Midbrain damage.
   d. Dilated bilateral and fixed: Severe anoxia, and death.
   e. Pupil shape—Ovoid or slightly oral: Increased ICP; irregular in trauma to the pupil.
9. Convulsion.
10. Leakage of CSF: Basilar skull fracture.
11. Other findings: Bradycardia and abnormal respiration. Rising blood pressure and falling pulse rate indicate rising ICP.
12. Hyperthermia.
13. Abnormalities in motor function: Paresis, paralysis, abnormal gait, and involuntary movements.

Investigations

1. Skull X-ray.
2. Computed tomography: It is helpful to detect epidural hematoma, subdural hematoma, intracranial shift, herniation, etc.
3. Magnetic resonance imaging: Posterior fossa lesions and brainstem lesions.

Management

1. **Establishment and maintenance of airway and adequate ventilation**:
   a. Cleaning of airway, tracheobronchial toilet, and care of oropharyngeal airway.
   b. Endotracheal intubation and IPPV.
   c. Tracheostomy may be needed.
   d. Fracture of cervical spine may be associated. Laryngoscopy and intubation or any head movement

JAYPEE BROTHERS

may be dangerous in such cases. An axial or in-line manual traction should be applied throughout.

2. **Resuscitation of cardiovascular collapse**: IV infusion of fluids, colloids or blood may be indicated. Vasopressors, and dopamine may be helpful for fluid and electrolyte balance.
3. **Assessment and management of associated injuries**: Facial trauma, neck trauma, and chest injury may be there.
4. **Monitor and promote neurologic functions**: Monitoring of vital signs is essential.
5. **Supportive treatment**:
   a. Indwelling urinary catheter.
   b. Nasogastric tube.
   c. IV infusion of fluids.
   d. Nutrition: Parenteral nutrition may be indicated.
   e. CVP or pulmonary artery catheter is essential for hemodynamic monitoring.
   f. Skin care.
   g. Prevention and treatment of infection: Antibiotics.
   h. Chest physiotherapy.
6. Reduce or prevent increased ICP:
   a. Dexamethasone.
   b. Osmotic diuretics.
   c. Hyperventilation.
7. **Control and prevention of convulsions**:
   a. Anticonvulsants: Diazepam, phenytoin, and phenobarbitone.
8. **Management of complications**:
   a. Hyperthermia.
   b. Coma.
9. Consultation with other specialists like neurosurgeon, etc. may be indicated.

JAYPEE BROTHERS

Cerebrovascular Accident

Cerebrovascular accident refers to an area of brain damage resulting from a circulatory disturbance such as thrombosis, embolism or hemorrhage. It may be either minor with recovery to normal or major with severe and permanent disability or even death.

Causes

1. **Cerebral thrombosis**:
   a. Essential hypertension with atherosclerosis.
   b. Hypotension as following myocardial infarction.
   c. Inflammatory diseases of blood vessels such as temporal arterities, polyarteritis nodosa, systemic lupus erythematous, etc.
   d. Hematological disorders: Polycythemia.
   e. Use of oral contraceptives.
2. **Cerebral embolism**:
   a. Mitral valve disease with atrial fibrillation.
   b. Prosthetic heart valve.
   c. Subacute bacterial endocarditis.
   d. Fat, air or malignant cells.
3. **Intracranial hemorrhage**: It may be either cerebral or subarachnoid.
   a. Rupture of small arterioles or microaneurysms.
   b. Rupture of a congenital aneurysm.
   c. Mycotic aneurysm caused by weakening of the arterial wall around a septic emboli.
   d. Arteriovenous aneurysms.
   e. Anticoagulant therapy.

Diagnosis

1. Computed tomography
2. Lumbar puncture

JAYPEE BROTHERS

3. Cerebral angiography
4. Hematologic studies
5. Blood biochemistry
6. Chest radiography
7. ECG.

Management

1. General management and nursing care of an unconscious patient.
2. **Supportive measures**:
   a. Bed rest and sedation.
   b. Respiratory care, tracheobronchial toilet, adequate oxygenation, and IPPV.
   c. IV infusion, fluid electrolyte balance, and nutrition.
3. Monitoring of vital functions is essential.
4. **Steroids**: Dexamethasone.
5. Hypertension should be controlled.
6. **Specific treatment**:
   a. Cerebral thrombosis or embolism:
      i. Anticoagulant therapy.
      ii. Aspirin.
   b. Cerebral embolism:
      i. Control of atrial fibrillation.
      ii. Treatment of subacute bacterial endocarditis.
   c. Cerebral hemorrhage:
      i. Reduction of raised intracranial tension.
      ii. Aminocaproic acid.
      iii. Calcium channel blockers to reduce cerebral vasospasm.
      iv. Surgical management.

Cerebral Edema

Cerebral edema implies the swelling of the brain mass mostly either due to accumulation of water or brain

JAYPEE BROTHERS

congestion. There is an increase in brain water content both in intracellular and extracellular compartments. Intracellular fluid accumulation generally occurs in gray matter and extracellular fluid accumulation is mainly in white matter.

Types

1. **Vasogenic**: It is due to disruption of blood-brain barrier as in cases of trauma, ischemia, pressure effect of space occupying lesion, malignant arterial hypertension, etc. Extent and spread of vasogenic edema is mostly determined by the site of lesion, size of vascular leak and pressure force to move the fluid outwards.
2. **Hydrostatic**: Here the cerebral edema is mostly due to high cerebrovascular transmural pressure.
3. **Cytotoxic**: The usual causes are ischemic hypoxia, local ischemia due to intracranial vascular lesion, general systemic ischemia affecting the whole brain, bactericidal agents like hexachlorphene, etc. Here the edema is mostly widespread with severe intracranial tension.
4. **Interstitial**: It is usually of obstructive in nature as in high pressure hydrocephalus.
5. **Hypo-osmotic**: If plasma osmolality is reduced by 12%, there may be brain edema with rise of intracranial pressure. Here the cell membrane is intact and water alone shifts from the vascular compartment following an osmotic gradient.

Clinical Features

1. History of head injury
2. Hypoxia

JAYPEE BROTHERS

3. Coma: Level of consciousness gradually declines
4. Focal signs of hemispheric disturbance must develop
5. Fixed dilated pupil and respiratory failure.

Investigations

1. CT scan.
2. Cerebral angiography.
3. Lumbar puncture.
4. Assess ICP through an extradural pressure transducer and calculate cerebral perfusion pressure (CPP).
   CPP = Mean arterial pressure – ICP.
5. Blood biochemistry, serum electrolytes, blood gas analysis, chest radiograph, ECG, etc. are often helpful.

Management

1. Primary aim should be to reduce the intracranial hypertension, to restore brain distortion and shift, and to increase cerebral blood flow in regions of ischemia.
2. General supportive measures:
   a. Adequate oxygenation—IPPV.
   b. IV fluids and vasopressors.
   c. Care of the unconscious patient.
   d. Care of the skin, eyes, bowel, and bladder.
   e. Normal body temperature should be maintained.
   f. Antibiotics to treat infection.
   g. Nutritional care.
   h. Monitor the vital signs, and ICP.
3. Hypoxemia should be treated promptly. Adequate oxygenation and ventilatory assistance are needed. Tracheostomy is often helpful.
4. Arterial perfusion should be maintained with systolic pressure between 110 and 140 mmHg. Hypotension causes further ischemia and hypertension augments brain edema.

JAYPEE BROTHERS

5. Specific measures:
   - Dehydration therapy.
     a. Hyperventilation: Moderate hypocapnia is always beneficial to reduce cerebral blood flow and thereby lower the ICP.
     b. Hypertonic solutions:
        i. IV 20% mannitol.
        ii. 50% glucose.
        iii. Urea.
     c. Steroids: It reduces brain water content, reduces ICP and brain shift, and improves cerebral blood flow.
     d. Diuretics: Frusemide.
6. Monitoring of vital signs and blood biochemistry, estimation of serum electrolytes, ECG, blood gas analysis, and frequent neurological assessment are essential to determine the prognosis and for better therapeutic management.

Status Epilepticus

Status epilepticus is a clinical condition characterized by rapid repetitive recurrence of any type of seizure without recovery between attacks. It constitutes a grave risk of life by causing airway obstruction, hypoxia, and consequent cerebral damage and cardiac arrhythmia. Seizures may occur alone or as a clinical manifestation of other diseases. However, generalized tonic-clonic or grand mal seizures which are convulsive, are the most dangerous.

Causes

1. Noncompliance or inadequate dosage of anticonvulsants in cases of known epilepsy.
2. Sudden withdrawal of anticonvulsant drugs.

JAYPEE BROTHERS

3. Brain tumor (often frontal lobe) and cerebral abscess.
4. Head injury/cerebrovascular disease/encephalitis/meningitis.
5. Liver/kidney disease.
6. Metabolic disorders:
   a. Hypoglycemia
   b. Hypercarbia
   c. Decreased cerebral oxygen
   d. Electrolyte imbalance
   e. Acid-base imbalance.
7. Withdrawal or overdose of some drugs: Phencyclidine, and cocaine.
8. Alcohol withdrawal.

Pathophysiology

Seizures are produced by abnormally hyperactive neurones which form an epileptogenic focus, rapidly and repeatedly depolarizing the cells involved. These may remain localized or spread throughout the entire cortex. Oxygen and nutritive sources are exhausted. The body tries to compensate with an increase in cerebral blood flow. With repeated seizures, low blood glucose level and oxygen content occur. Anaerobic metabolism starts and cellular lactate levels increase. All these complicate the condition of the patient.

Clinical Findings

1. **Aura phase**: The patient feels uneasiness, visual or auditory sensation which may occur several hours or even days before the actual seizure. Nausea and confusion may occur.
2. **Tonic phase**: Consciousness decreases with excessive muscular contraction and even apnea can occur. Pupils become dilated and nonreactive to light.

JAYPEE BROTHERS

or detailed information on medicine books, visit our website www.jaypeebrothers.com, for detailed information on medicin

Step by Step®
Critical Care

Salient Features

- It is a thoroughly revised, updated, and informative second edition
- An amazing book which highlights the importance and need of the 'human-touch' for patients who are in dire need of clinical care
- Comprehensive in nature but multifaceted in approach, the contents are tailormade to cover all types of critical cases under one roof
- Each disease discussed with relevant aspects of day-to-day care thereby manifold in ways of diagnosis
- Teaches through textual support and concise coverage of critical care medicine.

Arun Kumar Paul BSc MBBS DA MS (Anesth) is Ex-Professor, Department of Anesthesiology, Medical College and Hospitals, Kolkata, West Bengal, India, with vast experience of teaching for more than 35 years. He is the well-known author of several books on anesthesiology.



Available at all medical book stores
or buy directly from Jaypee Brothers through online shopping
at www.jaypeebrothers.com

JAYPEE BROTHERS
Medical Publishers (P) Ltd.
www.jaypeebrothers.com
Join us on facebook.com/jaypeebrothers

Shelving
Recommendation
MEDICINE

ISBN 978-93-5152-540-0