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10th Edition

Medical Emergencies in General Practice

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Foreword
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Contents

Chapter 1: Cardiovascular Emergencies 1

- Pain in Chest *1*
- Ischemic Heart Disease *5*
- Acute Myocardial Infarction *10*
- Non-ST-Elevated Myocardial Infarction (NSTEMI) and Unstable Angina (UA) *29*
- Heart Failure *33*
- Acute Pulmonary Edema *35*
- Cardiogenic Shock *39*
- Pulmonary Embolism *45*
- Hypertensive Crisis *51*
- Hypertensive Emergency *51*
- Cardiac Arrhythmias *55*
- Antiarrhythmic Agents *56*
- Tachyarrhythmias *60*
- Bradyarrhythmias *74*
- Stokes–Adams Syndrome *77*
- Sudden Cardiac Death and Cardiac Arrest *79*
- Acute Cardiac Tamponade *84*
- Appendix *86*

Chapter 2: Respiratory Emergencies 88

- Life-threatening Hemoptysis *88*
- Foreign Body Airway Obstruction *91*
- Acute Severe Bronchial Asthma *93*
- Spontaneous Pneumothorax *101*
- Acute Severe Pneumonia *104*
- Pneumonia in Immunocompromised Patients *112*
- Respiratory Failure *113*
- Acute Respiratory Distress Syndrome *118*

Chapter 3: Gastrointestinal Emergencies 123

- Acute Vomiting *123*
- Acute Diarrheal Diseases *124*
- Gastrointestinal Perforation *128*

- Acute Gastrointestinal Bleeding *129*
- Lower Gastrointestinal Bleeding *135*
- Acute Hepatic Failure *137*
- Acute Pancreatitis *146*

Chapter 4: Neurological Emergencies **153**

- Acute Confusional State *153*
- The Unconscious Patient *157*
- Cerebrovascular Catastrophes (Stroke) *164*
- Ischemic Stroke *167*
- Hemorrhagic Stroke *173*
- Hypertensive Encephalopathy *179*
- Cerebral Venous Thrombosis *181*
- Acute Bacterial (Pyogenic) Meningitis *182*
- Tubercular Meningitis *188*
- Aseptic Meningitis *191*
- Acute Viral Encephalitis *192*
- Seizures *194*
- Status Epilepticus *197*
- Acute Headache *199*
- Migraine *201*
- Cluster Headache *203*
- Acute Vertigo *204*

Chapter 5: Renal Emergencies **207**

- Clinical Fluid, Electrolyte, and Acid-base Abnormalities *207*
- Sodium Abnormalities *207*
- Potassium Abnormalities *210*
- Calcium Abnormalities *215*
- Metabolic Acidosis *222*
- Metabolic Alkalosis *224*
- Respiratory Acidosis *226*
- Respiratory Alkalosis *226*
- Acute Renal Failure *228*

Chapter 6: Hematological Emergencies **241**

- Classification *241*
- Severe Anemia *241*
- Hemostatic Disorders *247*
- Platelet Disorders *248*

- Henoch–Schonlein Purpura (Anaphylactoid Purpura) *254*
- Hemophilia *255*
- Disseminated Intravascular Coagulation *259*
- Neutropenia *261*

Chapter 7: Endocrinal and Metabolic Emergencies 263

- Thyroid Gland *263*
- Thyroid Crisis ("Storm") *263*
- Thyroid-associated Ophthalmopathy *266*
- Thyrotoxic Hypokalemic Periodic Paralysis *267*
- Myxedema Coma *268*
- Acute Adrenocortical Insufficiency *270*
- Diabetic Coma *272*
- Hyperosmolar Hyperglycemic Nonketotic Coma *279*
- Lactic Acidosis *282*
- Hypoglycemia *284*
- Acute Gouty Arthritis *287*

Chapter 8: Emergencies in Infectious and Tropical Diseases 290

- Dengue Fever *290*
- Chikungunya Fever *293*
- Ebola Virus Disease *295*
- Influenza A: Virus Infection *296*
- Leptospirosis *297*
- Enteric (Typhoid) Fever *301*
- Cholera *303*
- Mumps *305*
- Tetanus *307*
- Rabies *310*
- Malaria *314*
- Cerebral Malaria *319*
- Amebiasis *322*
- Amebic Liver Abscess *322*
- Coronavirus Disease (COVID-2019) *325*

Chapter 9: Acute Poisoning 326

- Principles of Management *326*
- Sedative-hypnotic Toxicity *329*
- Salicylate Poisoning *330*
- Acute Opiate Poisoning *332*

- Carbon Monoxide Poisoning *333*
- Acute Alcoholic (Ethanol) Intoxication *335*
- Acute Withdrawal (Abstinence) Syndrome *336*
- Methyl Alcohol (Methanol) Poisoning *337*
- Corrosive Poisoning *338*
- Snake Bite Poisoning *339*
- Insecticide (Pesticide) Poisoning *342*
- Carbamate Insecticides *346*
- Aluminum Phosphide Poisoning *348*
- Less Common Types of Poisoning *351*

Chapter 10: Iatrogenic Emergencies 356

- Procedure-related Emergencies *356*
- Intravenous Infusion related Emergencies *359*
- Drug Interactions and Drug-induced Emergencies *363*

Chapter 11: Shock Syndrome and Critical Allergic Reactions 376

- Shock Syndrome *376*
- Septic Shock *382*
- Critical Allergic Reactions *387*

Chapter 12: Environmental Emergencies 391

Acute Heat Reactions 391
- Heat Edema *391*
- Heat Syncope *392*
- Heat Cramps *392*
- Heat Exhaustion *392*

Heat Stroke and Heat Hyperpyrexia 393
- Heat Hyperpyrexia *393*
- Heat Stroke *394*

Hypothermia 398

Medical Emergencies in the Air 400
- Adverse Effects of Altitude Hypoxia *401*
- Adverse Effects of Pressure Changes *402*

Near Drowning 404

Electrical Injuries 406

High Altitude Sickness 407

Frostbite 409

Chapter 13: Miscellaneous Emergencies **410**

- Generalized Anxiety and Panic Disorders *410*
- Syncope *415*
- Prophylactic Immunization in Infectious Diseases *419*
- General *422*
- Appendix *422*

Index ***425***

4 CHAPTER Neurological Emergencies

ACUTE CONFUSIONAL STATE

Definition

Confusion implies *an altered state of consciousness* characterized by inability to sustain a normal level of attention *(attention deficit)* coupled with disordered perception and thinking. It represents the first step in the "disorders of consciousness", which range from confusion through drowsiness and stupor to coma. Synonymous terms include delirium, toxic psychosis, and toxic-metabolic encephalopathy. The key feature is that, in contrast to dementia, *confusional states are short lived and reversible.*

Etiology

Causes of acute confusion can be divided into four groups: (1) Intracranial diseases, (2) Medication/intoxication, (3) Systemic diseases secondarily affecting brain, and (4) Withdrawal syndromes **(Table 4.1)**.

Pathophysiology

The exact pathophysiology of most confusional states is not understood. The *key feature of confusion is disturbance of arousal and attention.* These functions are located in: (1) the *reticular core of the brainstem and thalamus* from where this activating system projects into the cortex (responsible for arousal), and (2) *the cortex* itself where the incoming impulses are constantly sorted out and stratified, allowing selected focusing on a stimulus to the exclusion of extraneous stimuli ("Attention"). A disruption of these functions at any level results in an inability to maintain a coherent stream of thought or activity. The result may be inattention or fluctuations of attention, or distraction (so that relevant and unrelated stimuli have equal value).

Clinical Features

Confusion is most common in the elderly and in the hospitalized patients, usually manifesting as delirium. The condition is usually seen in the setting of surgery, multiple drug therapy, or systemic disease. It is typically marked by clouding of consciousness, inattentiveness, and inability to register immediate

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Table 4.1: Causes of acute confusional state.

Intracranial diseases • Vascular • Neoplastic • Infections (meningitis/encephalitis) • Subdural hematoma • Epilepsy • Trauma	**Drugs/Intoxications*** • Phenothiazines, benzodiazepines • Sedatives, hypnotics, narcotics • Anticholinergics • Aminophylline • Antidepressants • Antihistamines
Systemic diseases • Renal failure • Hypoglycemia • Electrolyte disorder • Myocardial infarction • Congestive heart failure • Infections, e.g., pneumonia • Diabetic ketoacidosis • Hypo-or-hyperthyroidism • Non-ketotic hyperosmolar diabetic state • Hypo-or-hyperthermia	• Antihypertensives (methyldopa, clonidine, captopril) • Beta-blockers • Cimetidine, ranitidine • Corticosteroids • Digitalis Quinidine • Isoniazide • Antibiotics (quinolones, penicillin, cephaloporins • Alcohol intoxication • Lidocaine • Mefloquine • Metoclopramide • Metronidazole
Withdrawal syndromes • Alcohol withdrawal (delirium tremens) • Barbiturate and non-barbiturate sedative drug withdrawal	Nonsteroidal anti-inflammatory drugs

***Note:** This is not an all-inclusive list of medicines which can cause acute confusional state.

events and recall them a few minutes later. Because of disruption of attention there is *impairment of cognitive functions,* which involve logic, comprehension, judgment, and memory. Since neuronal involvement is diffuse in nature, all aspects of intellectual functions are disordered including orientation in relation to time and place (but usually not to person).

In addition to all the above-mentioned negative features of quiet confusion, delirium may supervene and is marked by autonomic and psychomotor overactivity. The resulting signs and symptoms may include restlessness**,** anxiety, irrational motor behavior, tremors, diaphoresis, dilated pupils, and sometimes, hallucinations. Rapid fluctuations may occur from day-to-day and even from 1 hour to the next. Symptoms often worsen in the evening because of further reduction in visual and auditory inputs into the reticular system, or addition of sleeping pill *("sun-downing syndrome").*

Diagnosis

Arriving at a diagnosis of delirium involves two steps. The first step is *to exclude dementia and acute transient psychiatric disorders.* The second step is *to define the cause of confusion/delirium* specially to identify any underlying

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Table 4.2: Important differences between ACS and acute functional psychosis.

Features	*Acute confusional state*	*Acute functional psychosis*
Onset	Sudden	Sudden
Course	Fluctuating	Stable
Consciousness	Clouded	Clear
Hallucinations	None or visual	None or auditory
Involuntary movements	Common (tremor or asterixis)	Usually absent
Systemic disease/drug toxicity	Often present	Usually absent

Table 4.3: Acute confusional state/delirium versus dementia.

Features	*Delirium*	*Dementia*
Onset	Acute	Slow
Course	Fluctuating in character and severity	Stable
Duration	Days to weeks	Months to years
Attention	Disordered	Usually normal
Thinking	Disorganized	Limited
Precipitating factor(s)	Often present	Usually absent

neurological disease or associated systemic illness. The exercise can be, at times, complex.

The process of diagnosis should begin with a thorough history and physical examination including neurological check-up. Suitable laboratory tests and other diagnostic studies should then be planned.

Hallucination can occur in both delirium and schizophrenia but are usually visual in the former and auditory in the latter. There may also be evidence of *"conversion reaction"* and dissociative states in the past. Other important differences between confusion and acute functional psychosis are given at **Table 4.2**.

Every case of ACS/delirium must also be *distinguished from dementia,* since the two conditions carry different prognosis **(Table 4.3)**. Important varieties of dementia syndrome are given at **Table 4.4**. Further discussion of the subject is beyond the scope of this text.

Investigations

These are planned to establish the cause of confusion, especially to rule out reversible/treatable etiologic factor(s). The laboratory tests should include estimation of hematocrit, white cell count, blood glucose, electrolytes, calcium, and hepatic and renal function studies besides routine X-ray of the chest and an ECG. Additionally, blood and urine cultures, blood gases, ammonia level, and thyroid function studies may be carried out when clinically indicated.

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Table 4.4: Important varieties of dementia syndrome.

• **Cortical dementia** ➢ Senile dementia of Alzheimer type (SDAT) ➢ Multi-infarct dementia ➢ Encephalitis	• **Frontal dementia** ➢ Subdural hematoma ➢ Space occupying lesion ➢ Hydrocephalus
• **Subcortical dementia** ➢ Parkinson's disease ➢ Huntington's chorea ➢ Multi-infarct dementia ➢ Demyelinating diseases ➢ Post-traumatic	• **Amnesic dementia** ➢ Ischemic encephalopathy ➢ Post-traumatic encephalopathy ➢ Korsakoff syndrome • **Pseudodementia** ➢ Depression ➢ Confusional state

Almost every patient in confusional state needs a computerized tomographic (CT) scan or magnetic resonance imaging (MRI) of the brain, especially when a focal lesion is suspected, or asymmetries are observed on examination of nervous system. In selected cases, EEG may also be desirable. Blood level of the drug may be required when intoxication is suspected. Lumbar puncture is essential when CNS infections are suspected or there is a strong suggestion of subarachnoid hemorrhage even though CT scan is normal (15% cases). As far as possible, lumbar puncture should be done after cranial CT.

Treatment

Almost all patients presenting with acute confusion will require hospitalization. As a domiciliary measure following steps should be practiced: (1) Treatment of any identifiable and reversible medical disorder, (2) Optimal control of fluid and electrolyte balance, blood volume, urinary output, oxygenation, any infection, and (3) Drug therapy. The first two aspects are not discussed further.

Drug Therapy

This should preferably be decided in consultation with a psychiatrist. The first principle is to *avoid polypharmacy* and *discontinue all nonessential drugs.* All possibly incriminating drugs **(Table 4.1)** should be withdrawn, and (if absolutely essential) replaced by drugs which do not have psychoactive or anticholinergic properties. Even sedatives and hypnotics should be avoided unless one is treating alcohol or benzodiazepine withdrawal.

The drug of choice for managing severe acute confusion/delirium is haloperidol, but even this should be used only if patient is agitated. The usual dose is 1–2 mg orally or IM two or three times a day. The dosage should be adjusted so as to blunt the agitation permitting nursing care and avoiding exhaustion. The aim should be not to totally suppress agitation since this may require very large doses, which can impair vital functions.

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Other drugs should be used with caution. These include chlorpromazine and thioridazine for agitation in general, and diazepam for withdrawal symptoms.

THE UNCONSCIOUS PATIENT

Normal state of consciousness is dependent upon integrity of the reticular system which is chiefly located in the thalamus and upper brainstem. It receives collateral axons from the main spinothalamic and specific sensory pathways, and projects them to the whole of the cerebral cortex. Interruption of activity in this system results in a state of unconsciousness. Pathophysiologically this may be due to:

- **Metabolic and toxic/infective disorders:** Such disorders impair cerebral functions bilaterally, and are amongst the commonest causes of coma **(Table 4.1)**.
- **Supratentorial mass lesions,** such as massive cerebral infarct, brain abscess, and brain tumor, which secondarily encroach upon deep diencephalic structures. These are common conditions, which result in unconsciousness through the mechanism of herniation of brain tissue (either uncal or central transtentorial) with consequent compression and ischemia of the brainstem structures.
- **Subtentorial lesions** arising from the substance of the brainstem and involving reticular formation directly, e.g., brainstem tumor or infarct. Such diseases are relatively uncommon and virtually untreatable.
- **Psychiatric disorders,** which may resemble coma but are physiologically different.

Etiology

It may be mentioned right at the beginning that like jaundice, coma is only one manifestation of the whole symptom complex and is not a disease by itself. The syndrome of coma, which may range from drowsiness to deep unconsciousness, may be the result of many conditions **(Box 4.1)**. A general approach to arrive at a correct diagnosis in a case of coma is also shown schematically in **Flowchart 4.1**.

Evaluation of the Unconsciousness Patient

A correct diagnosis of the cause of coma may be obvious (as in hepatic, diabetic, or uremic coma), or may be reached only after a detailed examination of the patient and relevant investigations. However, priority should be to ensure that all vital functions are stable otherwise appropriate resuscitative measures should be immediately instituted. Any bleeding from site of trauma, if present, should also be quickly controlled. Following systematic approach is recommended:

A detailed history should be obtained from the relations, friends and witnesses accompanying the patient especially regarding: (1) Mode of onset

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Box 4.1: Causes of coma.

- **Metabolic/toxic/infective disorders**
  - Poisonings, especially barbiturate, opium, and alcohol
  - Diabetic coma
  - Hypoglycemia
  - Sepsis
  - Hepatic failure
  - Renal failure
  - Cerebral malaria
  - Sunstroke and heat stroke
  - Encephalitis/meningitis
  - Postictal states
  - Electrolyte imbalance
  - Hypoxia/hypercapnia
- **Supratentorial mass lesions**
  - Cerebral vascular catastrophes
    - Cerebral infarct
    - Cerebral hemorrhage
    - Hypertensive encephalopathy
  - Intracranial space occupying lesions
    - Brain tumor
    - Brain abscess
  - Epidural, subdural, or intracerebral hematoma
- **Subtentorial (brainstem) lesions**
  - Brainstem tumor
  - Brainstem infarct or hemorrhage
  - Cerebellar hemorrhage
- **Psychogenic**
  - Hysteria
  - Malingering

of coma, (2) History of any drug addiction, (3) Any possibility of head injury or foul play, and (4) Any significant past illness. If no clues are available, the circumstances under which the patient was discovered should be enquired and a specific search of the environments as well as the patient's clothes made to locate any letter (possible suicide) or drugs which might have been consumed.

A thorough physical examination should then be conducted especially for any signs of trauma or needle marks on the extremities, any systemic organ disease or metabolic disease. Any peculiar odor of the breath should also be noted. Evidence of meningeal irritation should be sought by passive flexion of the neck and by Kernig and Brudzinski's signs. However its absence, in a patient in *deep* coma, does not rule out meningeal pathology. Funduscopy is also desirable.

An attempt should be made to separate out cases of coma due to conditions listed in group 1 from other pathologic states (**Box 4.1**). This is important because *metabolic/toxic encephalopathies involve the cortex and brainstem diffusely, and do not exhibit abrupt or progressive rostrocaudal dysfunction*

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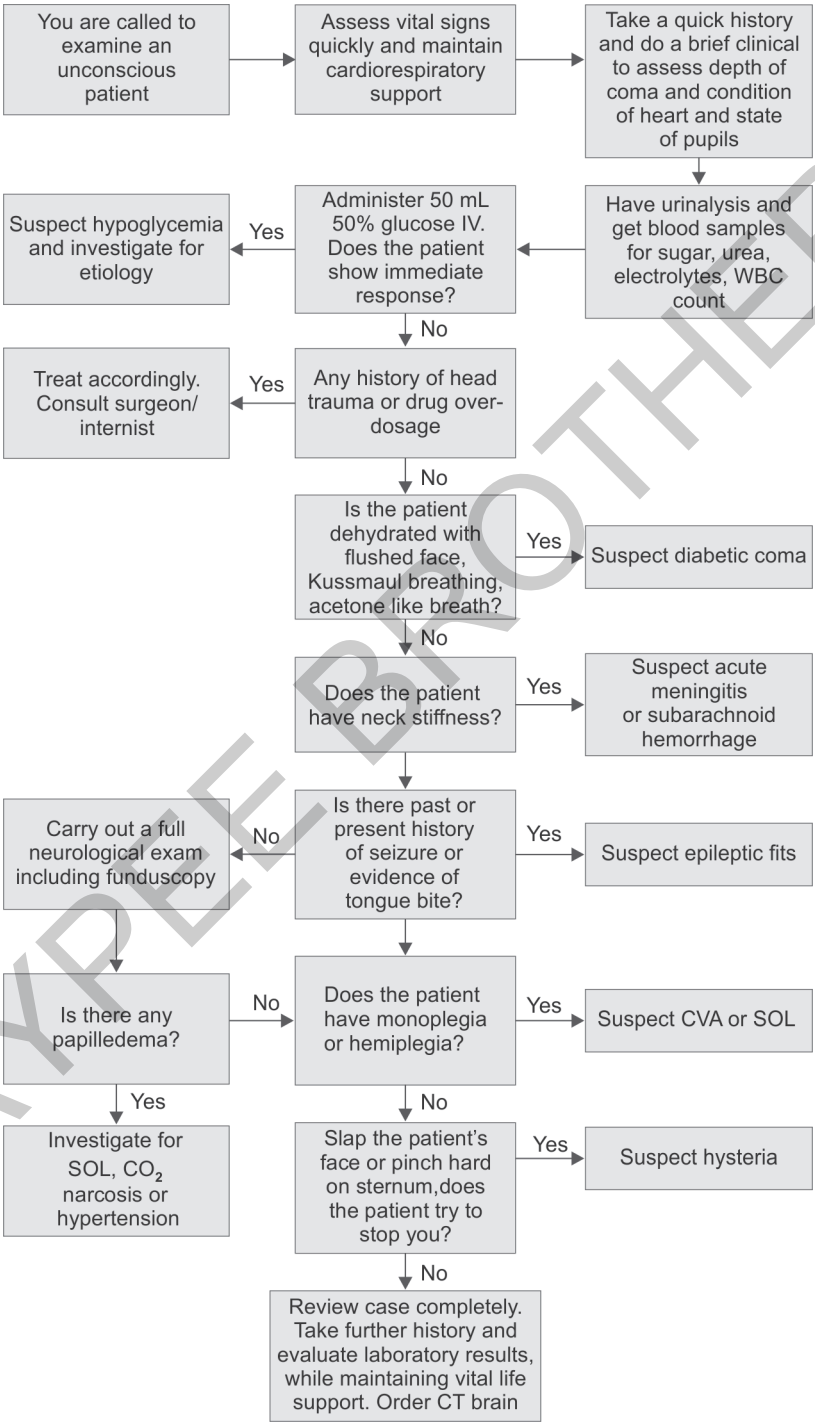
Flowchart 4.1: Evaluation of unconscious patient.



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usually seen in neurosurgical setting. The former also have a more gradual onset, and reveal global impairment without any focal lesions. Certain quick laboratory investigations are valuable in such a situation. These include estimation of blood sugar, electrolytes, hepatic and renal functions, and arterial blood gas study. It should be remembered that *altered electrolytes/blood gases and sepsis* constitute important causes of impairment of consciousness, especially in the elderly, in patients who do not have any focal neurological deficit, and who have normal brain CT.

Neuroanatomic Localization

In states of coma due to conditions listed in Group II and III (**Box 4.1**), a progressive deterioration in nervous system functions tends to occur in a rostrocaudal manner, involving the thalamus, midbrain, pons, and ultimately the medulla, in that order **("herniation syndromes").** Neuroanatomic localization of the pathology (**Table 4.5**) in such cases helps in determining the site as well as severity of the disease process. Further, their periodic evaluation helps in determining the course of the disease since such changes

Table 4.5: Neuroanatomic syndromes in coma: Salient features.

Types of herniation	*Pupils*	*Respiration*	*Eye movements*	*Motor signs*
I. Central Herniation				
a. Thalamic	Small reactive	Normal or Cheyne-Stokes	Usually normal	Usually normal
b. Midbrain	Mid or widely dilated, non-reacting	Central neurogenic hyperventilation	Absent ciliospinal reflex	Decorticate or decerebrate posture
c. Pontine	Pinpoint, non-reacting	Central neurogenic or irregular	Absent oculocephalic and oculovestibular reflexes	Decerebrate posture
d. Medullary	Dilated, fixed (ataxic)	Slow irregular	Absent	Flaccidity
II. Uncal				
a. Early	Ipsilateral pupil dilated but often reactive	Usually normal	Usually normal	Usually normal
b. Moderate/ advanced	Dilated and usually fixed	Cheyne-Stokes or central neurogenic	HI nerve palsy	Hemiparesis (ipsilateral or contralateral) or decerebrate posture

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often progress in a systematic manner. Following parameters are employed for such assessment:

- **State of consciousness:** This is classified as follows:
  a. *Grade I: Normal* alert wakefulness with intact reflexes, sensory as well as motor.
  b. *Grade II (Drowsiness):* The patient is less aware of his environments, appears disinterested, and his responses to verbal commands are slow and delayed, but appropriate.
  c. *Grade III (Stupor):* Patient can be aroused only by intense stimulation such as loud shouts and deep pressure over the supraorbital notch on both sides. Even this stimulation will evoke, in a stuporous patient, only minor nonverbal reaction of semi-purposeful withdrawal movement. Reflex reactions are usually preserved at this stage.
  d. *Grade IV (Coma):* Patient is unaware of his environments, and even intense painful stimuli evoke either minimum (moderately deep coma) or no response at all (deep coma). All reflex reactions are lost at this stage.

  Herniation syndromes result from increased intracranial pressure, and lead to brainstem compression, manifested by arterial hypertension, bradycardia, and respiratory irregularities (Cushing triad). In the initial stages of brainstem compression with only disturbed thalamic functions, there is only confusion, disorientation, and drowsiness. Later, as the midbrain gets involved, drowsiness deepens into stupor and then into coma. As more caudal parts of brainstem get involved, coma deepens, but recognition of the severity of coma is usually not possible at this stage.
- **Pattern of respiration:** In the early thalamic stage, respiration is usually normal but frequent sighing or yawning may occur. As the thalamic dysfunction increases, breathing acquires ***Cheyne-Stokes* character.** Later, with progression into ***midbrain and upper pons,*** Cheyne-Stokes respiration changes to central neurogenic hyperventilation characterized by sustained, deep, rapid and regular hyperpnea. At the *lower pontine and medullary level* compression, **hyperventilation ceases, and gives way to slow respiration, irregular in rate and depth which finally becomes gasping.**
- **Pupillary size and reaction:** The size, equality or inequality of the pupils and their reaction to light provide valuable localizing clues, and should be studied serially, using a strong beam of light.

  At the *thalamic stage,* the pupils are small but react to light. With *early midbrain involvement* the pupils become mid-dilated and nonreactive to light. As damage to the midbrain increases, the pupils become widely dilated and fixed. The *ciliospinal reflex* is also lost at this stage. *Pontine lesion,* usually a hemorrhage, results in very small pupils with minimal reaction to very strong light. Such alterations in pupillary responses generally imply structural brain lesion. *Toxic-metabolic states leave the pupils unaffected with few exceptions* such, as overdose with atropine

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(widely dilated and fixed pupils) or opium (pin-point and barely reacting pupils).

- **Eye movements:** Certain eye movements also assist in localizing the site of lesion. Normally, sudden passive turning of the head to one side produces conjugate deviation of the eyes to the opposite side *(doll's eye movements).* Absence of this *oculocephalic reflex* in a comatose patient implies dysfunction of the pons (where lateral gaze center is located), but does not differentiate between structural and toxic-metabolic pathology.
- **Motor responses:** Presence of local or unilateral paralysis indicates focal structural damage to the brain. This may be detected by alteration of deep reflexes and an extensor planter response on the paralyzed side. However, in the presence of deep coma, planter reflexes lose their significance and may be extensor on both sides.

In the early *thalamic stage* of compression there, may be a generalized increase in tone of all muscles, both flexors and extensors. **Decorticate posture** characterized primarily by fisted upper extremity held in full flexion against the body, occurs with late thalamic and *early midbrain* dysfunction. Later, with *more severe involvement of the midbrain,* **decerebrate state** occurs in which there is extension and adduction of all the four limbs with marked increase in tone. *Pontine-level* dysfunction is invariably associated with extensor posturing. With further deterioration and involvement of *pontomedullary junction,* total flaccidity, and hypotonia result, generally terminal.

A correlation of the observations recorded in respect of the above mentioned five parameters should permit identification and staging of the herniation syndrome in each case of coma. The salient features of these herniation syndromes are summarized in **Table 4.5.**

Critical Evaluation of Neurological Signs

- In a midline *supratentorial mass with rapidly increasing compression* (e.g., diffuse cerebral edema, large subdural or intracerebral hematoma), there is a characteristic rostrocaudal progression of neurological signs starting with features of thalamic dysfunction. Delay in treatment at this stage may convert a recoverable into a nonrecoverable lesion as indicated by appearance of *signs of midbrain involvement,* such as deepening coma, mid-dilated, or widely dilated nonreacting pupils, deep rapid breathing, decerebrate rigidity, and absence of ciliospinal and oculocephalic reflexes.
- When *compression starts from one side* as in laterally placed subdural hematoma, tumor or inflammatory/infective process, the medial portion of the **ipsilateral** temporal lobe (the uncus) is the first to herniate through the tentorial notch. This results in the *uncal herniation syndrome,* characterized by ipsilateral dilated pupil (due to third nerve palsy) and contralateral hemiparesis (due to involvement of ipsilateral pyramidal fibers above the decussation) **(Table 4.5**). In a few cases with rapidly expanding hemispheric lesions, the other pupil may also dilate in due

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course, due to side to side displacement of the brainstem so that the midbrain is grooved by the opposite tentorial edge. Recognition and early treatment of uncal syndrome is most vital since recovery becomes virtually impossible once the opposite pupil dilates. With further increase in compression, the signs mimic those of the central transtentorial herniation.

- In cases with *toxic/metabolic coma,* unlike progressive brain compression, *there is no orderly deterioration in brainstem functions.* In fact, large scale disparities may exist between different parameters (e.g., barely reactive pupils in a patient who intermittently regains consciousness), and deep coma may be present out of proportion to neurological findings. Examination of pupillary reflexes is especially important, since these are preserved in all toxic/metabolic encephalopathies except when due to anoxia or certain drugs, which have specific effects on the pupils (vide supra).

Investigations

Certain investigations should be done routinely in all cases of coma, and include urine examination, complete blood count, blood urea, sugar and electrolyte estimation, a CXR, and ECG. Non-enhanced CT scan of the brain should be considered when coma is unlikely to be due to toxic/metabolic disorders. *Lumbar puncture, if indicated, should be done preferably after CT brain.* In addition, in all cases suspected of poisoning a gastric lavage should be done and the stomach contents preserved for chemical examination.

Management

Emergency Treatment

Patient should be hospitalized as early as possible, and measures instituted to provide a clear airway, stable pulse, blood pressure, and respiration. As already stated, these should take precedence over detailed physical examination and laboratory studies.

General Measures

- **Posture:** The patient should be nursed on the side (rather than in flat position), to minimize the risk of respiratory complications, and the position changed every few hours to prevent pressure sores. An air mattress or alfa-bed should be used, if the course of illness is likely to be a prolonged one. The bed clothes should be kept dry and free from creases. The limbs should be kept in the optimum position, lightly massaged, and moved through full range frequently, to avoid contractures and deep vein thrombosis. Care should be taken that the patient does not fall from the bed.
- **Nutrition and fluid balance:** As far as possible, nutrition and fluids should be provided every 2–3 hours through a nasogastric tube. Too hot and too

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cold fluids must be avoided. The main risk is of aspiration pneumonia in such patients but this can be minimized by: (1) Aspirating the stomach contents before each feed to ensure the correct position of the tube, (2) Elevating the head of the patient during and for a short period after the feed, and (3) Restricting the volume of each feed to 200 mL.

The amount of fluid intake during the first few days should be carefully controlled in all cases where cerebral edema is a factor, especially in acute stroke. There is often a breakdown of blood brain barrier in the damaged area of the brain in such cases which is apt to retain fluid and thus develop cerebral edema. Oral route is to be preferred, but if IV fluid administration is necessary, isotonic saline (0.95%) or *(slightly) hypertonic glucose should be given (not 5.0% glucose which is slightly hypotonic,* isotonic glucose solution being 5.25%). Serum electrolytes should also be monitored.

- **Other measures:** Vital functions should be maintained keeping in view that normal autoregulation of blood flow is likely to be lost in the microvasculature in the damaged area especially in cases of ischemic infarction. *Sudden and drastic changes in blood pressure should therefore be avoided,* and hypertension treated only if the blood pressure is >180/110. Episodes of hypotension may be especially dangerous.

Patency of airways and adequate respiration must be ensured. Some patients who may have lost cough reflex and are likely to stay in coma for a long time, may require endotracheal intubation or even tracheostomy for proper removal of secretions. Oxygen therapy is generally beneficial, and some cases with significant respiratory depression may require assisted ventilation. Seizures may occur and are best controlled by phenytoin, since it does not further depress level of consciousness. Neurosurgical consultation should also be considered in suitable cases.

Specific Measures

Treatment of increased intracranial pressure (ICP): Lowering ICP is a vital component of management of comatose patients developing herniation syndromes. This is best reflected by calculating cerebral perfusion pressure (CPP) defined as mean arterial pressure minus ICP. As far as possible, CPP should be maintained >70–80 mm Hg. However, this is possible only in advanced neurological centers. In the absence of ICP monitoring, hyperventilation to a $PaCO_2$ 30–35 mm Hg should be initiated, followed by IV mannitol (0.25–1.0 g/kg). Another measure is use of barbiturates which decrease cerebral metabolism, and thereby reduce cerebral blood flow.

CEREBROVASCULAR CATASTROPHES (STROKE)

Acute vascular disruption to a specific region of the brain ("stroke") is the second most frequent cause of death world-wide, and constitutes one of the commonest medical emergencies. The term "stroke" originates from biblical reference to being "struck down", implying an acute dramatic neurologic

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