Emergency Medicine

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Introduction

This volume is intended to cover the diagnosis and initial management of the major emergencies likely to be encountered in medical practice. The significant complications which may occur in the acute period are discussed, but the long-term care of these conditions is mentioned briefly only where relevant. Where appropriate, a problem-orientated approach has been taken, and the book has been roughly subdivided into five sections. The first five chapters deal with patients whose general presentation is with an impairment of their conscious level. The next section discusses the management of the various consequences of trauma, followed by sections on emergencies originating, first, in the abdomen, and, secondly, in the cardiopulmonary systems, this latter being linked with chapters on the management of the important related topics on cardiac arrest and shock. The final section is devoted to a discussion of four important but generally unrelated problems.

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Contents

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Early management of the comatose patient

G. H. Hall

Working definition

A comatose patient is out of touch with his surroundings, cannot communicate sensibly, responds imperfectly or not at all to external stimuli, and cannot be roused to normal waking behaviour. Coma ranges from mild confusional states, through stupor (temporarily rouseable) and delirium (hallucinations) to true coma (unresponsive). These subdivisions are vague, and levels of coma are best assessed by the Glasgow scale (*Table 1.1*). Coma should not be equated with unconsciousness. Unconsciousness does not occur in psychological unresponsiveness (playing possum), akinetic mutism, or the 'locked in' syndrome caused by disturbance of the reticular activating system. Coma always implies serious dysfunction of the cerebral hemispheres and brain stem. It carries a bad prognosis for two

Table 1.1. Glasgow scale (coma)*

Response	*Degree of response*	*Time sequence*			
		Initial	*After 1 h*	*After 2 h*	*After 3 h*
Eye opening	Spontaneous				
	To speech				
	To pain				
	None				
Best verbal response	Orientated				
	Confused				
	Inappropriate				
	Incomprehensible				
	None				
Best motor response	Obeying				
	Localizing				
	Flexing				
	Extending				
	None				

*This is the type of chart that should be completed for the observations on a patient with an impairment of consciousness

reasons: the patient cannot guard himself against common physical dangers (fire, water, asphyxiation and exposure) and the underlying causes are often life-threatening.

Immediate measures

1. Detain those who brought the patient, for detailed questioning later.
2. Establish airway, by intubation if necessary, and ventilate if required.
3. Measure temperature, pulse, respiration and blood pressure. Treat severe deviations from normal.
4. Insert i.v. cannula. Take blood for estimation of sugar, electrolytes, urea and haemoglobin and for blood count; save serum for drug estimation. Set up infusion of saline.
5. Give thiamine 100 mg i.v. followed by 50% glucose 25 ml.
6. Proceed to orderly history and physical examination.

History

This has to be obtained from attendants or relatives: the patient's own doctor should be contacted if possible for information about past history and medication. Particular attention should be directed towards (1) the possibility of trauma, exposure to drugs or toxins: (2) recent personality change, history of diabetes or psychoneurosis. The rate of onset and the duration of coma must be noted and the presence of convulsions, incontinence, tongue-biting or obvious changes in skin colour, sweating and respiration. Ask 'Who by, how, where, when, why, was the patient found?'

Examination

This is directed towards rapid detection of remediable causes of coma, and to provide a baseline to assess subsequent progress. The *best* eye, verbal and motor response to stimulation (according to levels on the Glasgow scale) should be recorded. A routine technique should be followed to avoid omissions. A satisfactory method is to make a rapid survey of all parts of the body externally, then to examine the cardiorespiratory, alimentary and central nervous systems. The CNS should be examined for evidence of the following:

1. Raised intracranial pressure,
2. Localizing signs,
3. Brain-stem involvement.

The most important signs to look for and their interpretation are given in *Table 1.2.* In addition, the age, sex, state of nutrition and of hygiene often provide diagnostic information.

Table 1.2. Important signs and their interpretation

System	*Finding*	*Interpretation*
General	Trauma	Concussion; intracranial damage and bleeding.
	Convulsions	Epilepsy; hypoglycaemia, hypocalcaemia, hypokalaemia.
	Hemiplegia	Stroke.
	Opisthotonus; photophobia	Meningitis, subarachnoid haemorrhage.
	Cyanosis	Asphyxia; methaemoglobinaemia.
	Pallor	Shock (cardiac, haemorrhagic, septicaemic); uraemia; myxoedema; pernicious anaemia.
	Fast respiration	Ketoacidosis, uraemia; pontine haemorrhage; shock.
	Slow respiration	Drugs; brain-stem compression.
	Breath smell	Alcohol; ketones; uraemia; foetor hepaticus; melaena.
	Jaundice	Drug abuse; liver failure.
	Flushing	Ketoacidosis; septicaemia; atropine; pontine haemorrhage; CO_2 narcosis.
	Sweating	Fever; hypoglycaemia.
	'Sleeping'	Drugs; hypothermia
	Rash	Drugs; blood dyscrasia; SLE.
Cardiovascular	Bradycardia	Heart block; raised intracranial pressure; hypothermia; drugs; myxoedema.
	Tachycardia	Shock; cardiac arrhythmia; drugs (tricyclic antidepressants).
	Hypotension	Shock; pulmonary embolus; adrenal insufficiency; medullary failure; cardiac arrest.
	Hypertension	Malignant hypertension; uraemia; raised IC pressure; phaeochromocytoma; reaction to MAOs.
	Murmurs	SBE; mycotic aneurysm; cerebral embolism.
	Heart failure	Myxoedema; electrolyte disturbance; cardiac arrest.
Respiratory	Clubbing	Ca bronchus with cerebral secondaries. SBE; hyponatraemia.
	Bronchitis and emphysema	CO_2 narcosis; hypoxia.
Abdominal	Melaena	GI bleeding.
	Hepatosplenomegaly	Liver failure.
	Ascites	GI bleeding; carcinomatosis.
	Peritonitis	Septicaemic shock; ruptured aneurysm.

Table 1.2. Important signs and their interpretation

System	*Finding*	*Interpretation*
Central nervous*	Neck stiffness	Meningitis; SAH.
	Papilloedema	Raised IC pressure; malignant HT.
	Pupils unequal	Uncal herniation.
	Pupils equal, small, reactive	Lesion above brain stem.
	Pupils dilated, unreactive	Brain-stem lesion.
	Conjugate deviation	To side of unaffected hemisphere in cerebral infarction.
	Oculocephalic response absent	Pontine or medullary depression.
	Oculovestibular response absent	Pontine or medullary depression.
	Hemiplegia	Stroke.
	Decorticate (arms adducted and flexed; legs extended)	Upper brain-stem lesion.
	Decerebrate (arms and legs extended; internal rotation of forearm on sternal pressure)	Lower brain-stem lesion.

* Notes on brain-stem reflexes:
Oculocephalic response: rotate head in horizontal and vertical planes. Eyes should remain looking ahead as if fixed on one spot.
Oculovestibular response: irrigate auditory canal (must be free of wax and no perforation of drum) with 5–20 ml *ice cold* water. Eyes should deviate to side of irrigation (conjugate): nystagmus is a normal response but may be present in light coma.
These are the most reliable signs of brain-stem function.

Investigations

The preliminary examination should have provided enough information to decide whether the coma is likely to be caused by a localized intracranial abnormality or a general toxic-metabolic-endocrine disturbance. Appropriate further investigations will then be obvious. Certain tests should be done anyway, and these are listed in *Table 1.3.*

Further investigations

Urgent investigation by a neuroradiological or neurosurgical team must be considered if a rapidly expanding intracranial lesion is suspected, particularly after trauma (except dural or subdural haematoma). It is unlikely at present that CT scan, angiographic or EEG facilities will be available for use in the emergency room. Toxicological analysis of stomach washings, blood and urine samples may be necessary in cases of obscure poisoning.

Table 1.3. Routine investigations

Test	*Finding*	*Interpretation*
Urine	Sugar	Diabetes.
	Protein	Uraemia.
	Ketones	Diabetes; starvation.
	Bile	Liver failure.
	Drugs	
	Cells, organisms	Gram-negative septicaemia.
Blood	Sugar: high	Diabetes.
	low	Hypoglycaemia (use paper test strips).
	Na low	SIADH; fatty serum.
	high	Dehydration; incorrect i.v. therapy.
	K low	Diuretics; endocrine; liver failure; steroids; diarrhoea.
	HCO_3 low	Acidosis (diabetes, uraemia); lactic acidosis.
	high	Hypokalaemia; hypoventilation.
Haemoglobin	High	Dehydration; polycythaemia; stroke.
	Low	Haemorrhage; blood dyscrasia.
	WBC high	Infection; leukaemia.
	low	Blood dyscrasia; viruses.
Blood film	Malarial parasites	Cerebral malaria.
CSF*	Leucocytes > 3 mm^3	Meningitis; encephalitis; brain abscess.
	Blood	Subarachnoid haemorrhage.
	Xanthochromia	Haemorrhage.
	Organisms	Meningitis (pyogenic).
	Low sugar	Meningitis.
Chest X-ray	Numerous	Numerous.
Skull X-ray	Fracture	Trauma.
	Sepsis in paranasal sinuses	Cerebral abscess.
	Deposits in skull	Carcinomatosis.
	Raised intracranial pressure	Expanding lesion
	(Thumbprinting: erosion posterior clinoids)	
	Enlarged pituitary fossa	Pituitary lesion

* CSF examination is useful in most cases of coma of doubtful origin provided that papilloedema is not present.

Conditions for diagnosis of brain death (Royal College Criteria)

1. No suspicion that cause is due to drugs.
2. Not hypothermic (>35°C).
3. No metabolic or endocrine cause detected.
4. No possibility of muscle relaxant causing apnoea.
5. Brain-stem reflexes absent as follows:
   (a) Pupils fixed and not reacting to light.
   (b) Corneal reflexes absent.
   (c) Oculovestibular reflexes absent.
   (d) Absent motor responses in cranial nerve distribution, including gag reflex.
   (e) Apnoea persisting for more than 10 min either during intratracheal administration of 5 per cent CO_2 and 95 per cent O_2; *or* during administration of 100 per cent O_2 and with a P_aCO_2 of more than 50 mmHg (6.65 kPa).

The tests may be repeated before stopping artificial respiration.

Agreement should be reached regarding the findings, between the doctor in charge of the patient and one other senior doctor, who should not be concerned with any transplantation procedures that may be envisaged.

2

Overdoses and poisonings
I. General management

James D. P. Graham

Summary

1. Poisoning in adults is largely restricted to medicinal products – sedatives, analgesics and psychoactive drugs.
2. Emergency treatment is an advanced form of first aid:
   (a) Detect and relieve obstruction of the airway; ensure oxygenation.
   (b) Confirm the probability of poisoning and try to establish type (sedative, analgesic etc.), and site of action (ingestion, inhalation or external).
   (c) If the patient is unconscious grade the coma (I and II detain and observe, III and IV admit to intensive care).
   (d) Antidotes are specific: if relevant, use them at once, but there are few.
   (e) Take steps to reduce absorption, e.g. decontamination, emesis and gastric lavage.
   (f) General principles of good care: safeguard respiration, circulation, body heat, and fluid and electrolyte balance; nursing care; and patient supervision.
   (g) Increase rate of clearance if desirable and feasible: diuresis, forced diuresis; haemoperfusion and haemodialysis rarely.
   (h) Follow-up. Exclude organ damage (liver, kidney and heart). Arrange social and psychiatric care.

The problem

The trend in poisoning in the United Kingdom (UK) has been steadily upwards for more than 20 years and only recently shows some signs of levelling off. In England and Wales in 1976, with a population at that time of approximately 49 million, there were in round figures 3000 deaths, 100 000 hospital admissions (Hospital Inpatients Enquiry, 1977) and 25 000 enquiries to the Poisons Information Services from this cause alone. A survey in Cardiff hospitals made that year showed that of 9000 inpatients, 19 per cent suffered from acute poisoning, which may be compared with 15 per cent with acute ischaemic heart conditions.

Poisoning in adults may be classified as *self-poisoning*, which is deliberate but not necessarily suicidal, and *accidental poisoning*, which includes the relatively small number of domestic and works accidents. The contrast with poisoning in children is marked, because most episodes in the latter age group are held to be free of intent and the causes are most diverse. Mortality and serious morbidity from all causes has declined during the last decade, whereas the number of these due to medicinal substances has increased substantially. Improvement has resulted primarily from a replacement of the formerly lethal carbon monoxide-based gas by the methane-based contemporary town gas, the decline in prescribing of barbiturates and Mandrax (diphenhydramine plus methaqualone), the tendency away from aspirin as a self-medication for minor illnesses and the practice in hospital of screening and selective intensive care. It is not attributable to the development of specific antidotes but has been helped by increased understanding of drug kinetics and clinical pharmacology. Nevertheless, many unexpected acute toxic reactions to drugs, which may lead to the patient's admission to hospital, result from drug–drug interactions.

The age distribution of patients in the poisoning cohort parallels that of the population as a whole, most occurring in those who are 15–50 years of age. Approximately 75 per cent of all poisoned inpatients are adult. The male:female ratio, which was formerly markedly biased in favour of females, is moving towards unity.

Common causes of poisoning in adults

The causes of acute poisoning in adults admitted to hospital are largely, but not exclusively, medicinal substances, and the episode is deliberate. Accidental poisoning has more varied causes. The principal causes in inpatients are as follows:

1. Drugs affecting the central nervous system.
   - (a) Sedatives
     - (i) Minor (benzodiazepine) and major (phenothiazine) tranquillizers.
     - (ii) Barbiturates and other hypnotics.
     - (iii) Alcohol.
   - (b) Antidepressants
     - (i) Tricyclic
     - (ii) Other
     - (iii) Antipsychotics.
   - (c) Analgesics
     - (i) Aspirin
     - (ii) Paracetamol
     - (iii) Mixtures, e.g. with D-propoxyphene
     - (iv) Opioids.
2. Miscellaneous drugs.
   - (a) Digitalis
   - (b) Antibiotics
   - (c) Others

3. Pesticides, e.g. paraquat and diquat.
4. Industrial poisons.
   (a) Metals
   (b) Gases and fumes
   (c) Cyanide
   (d) Corrosives
   (e) Solvents.

Diagnosis – general principles

Diagnosis is frequently simple in acute adult poisoning. The patient, relative or attendant may give a clear history of overdosage with one or more drugs, or there may be a sample available. If in doubt as to its identity, refer it to the hospital pharmacy (or Drug Information Service if available) for identification. If there is still doubt about case management, drug interaction or clinical toxicology, consult the regional Poisons Information Services (*see* page 22 for priority telephone numbers). A useful history may not be forthcoming because of anxiety, fear, fantasy or mental dulling in the patient, or similar inhibitions in the relatives. A large proportion of patients are delivered to hospital casualty departments without previous attendance by a doctor. Frequently the casualty medical officer can proceed on empirical grounds only, and many 'overdoses' remain uncharacterized. If the acute onset of the illness is accompanied by diarrhoea and vomiting, a differential diagnosis of food poisoning by bacteria or their toxins, or the acute effect of irritant poisons has to be considered. If the patient is unconscious, an intracerebral disaster has to be excluded. Then, *decide whether or not the case is, as alleged, one of poisoning.* If this is probable, then:

1. Is the cause *external* – an irritant or corrosive poison or one which may be absorbed through the skin (unusual; obvious signs; nature of chemical substances known): or is it *internal*, after absorption (most commonly; ingestion, inhalation)?
2. *Is there obstruction to breathing?* If so, correct it at once.
3. Is the patient conscious? Assess the level of consciousness and grade it as follows:
   Grade I Drowsy, confused, responds to command, reflexes brisk.
   Grade II Unconscious, does not respond to command, responds to minimal painful stimulus.
   Grade III Deeper, responds only to severe stimulus, respiration depressed.
   Grade IV Coma, no responses, hypotension, severe respiratory depression or apnoea, absent bowel sounds.
   Limb and pupil reflexes are deceptive. The preferred minimal 'painful' stimulus is firm pressure on the skin, sweeping up from above the ankle to below the knee three times. A positive response is withdrawal of the limb, restless movement or a sound. The preferred 'severe' stimulus is to rub the knuckles of the clenched fist heavily on the sternum, with a similar response.
4. *Check for evidence of injury* or severe organic illness.