The Unconscious Patient

A Practical Approach

DR. HAMID AHMED MRCP (UK)
PHYSICIAN SENIOR ASSISTANT

Causes

Coma is an emergency situation representing either a primary insult to the brain tissues or announcing an underlying systemic disorder.

(A) ***Structural/Primary Insults:***

supra- or infra-tentorial lesions.

i. Trauma:
— open and closed head injury.
ii. Vascular:
— hypertensive encephalopathy;
— subarachnoid haemorrhage;
— intracerebral haemorrhage;
— thrombosis;
— subdural haematoma;
— cerebral vasculitis e.g. SLE.
iii. Infections:
— meningitis-encephalitis;
— cerebral malaria;
— Reye syndrome;
— septic emboli, e.g. (SBE, Bronchiectasis)
iv. Epilepsy:
— postictal state
— Eclampsia
v. Space-occupying lesions:
— Abscess or neoplasm causing acute herniation.

(B) ***Metabolic/Systemic Disorders:***

represent 75% of cases

i. Lack or deficiency of essential substrates:
1. Glucose:
The brain utilises 30 - 50 mg per minute and so hypoglycaemia is not surprisingly damaging to the brain.
Thiamine is a Cofactor for glucose metabolism. It acutely decreases in Wernicke's encephalopathy, and can be a cause of confusion progressing to coma.
2. O_2:
The glucose is metabolized aerobically so hypoxia will be seriously deleterious.
3. Blood:
About 15% of total body circulation is diverted to the brain.
Cerebral blood flow can be compromised in:
(a) prolonged hypotension as in shock states.
(b) cardiac arrest.
(c) transient arrest e.g. Stokes - Adam's attack, arrhythmias.
(d) increased viscosity, e.g. polycytaemia.

ii. Excess Toxins:
1. Drug overdose:
— Heavy metals;
— Narcotics;
— hypnotics (Barbiturate, non barb);
— tricyclics;
— salicylates;
— alcohol;
— anticholinergics.
2. Hepatic failure
3. Uraemia

iii. Altered optimum environment for neuronal functions:
1. CO_2:
(a) Hypercapnoea
(b) Hypocapnoea with resp. alkalosis (e.g. hyper-ventilation).
2. Glucose:
Hyperglycaemia:
(a) DKA (metabolic acidosis is contributing)
(b) NKDHS. (Non-Ketotic Diabetic hyperosmolar syndrome).
3. Sodium:
Hypernatraemia (esp. in children or infants) Hyponatraemia: IADHSS. (Inappropriate ADH secretion syndrome).
4. Calcium
Hypercalcaemia or hypocalcaemia.
5. Temperature
Hypothermia—hyperthemia
6. Hormonal integrity dysfunction
— Myxoedema
— thyrotoxicosis
— adrenal insufficiency
— hypopituitarism (pituitary apoplexy).

The list is exhaustive and what has been mentioned is just examples of common and less common entities. It should be noted also that in the one case, many factors are contributing e.g. the alcoholic patient who presents in coma, excess alcohol with or without drug overdose, head injury, subdural haematoma (from repeated falls), acute thiamine deficiency, hypoglycaemia, metabolic acidosis and/or liver failure all can be operating to produce the coma .....!

Examination

Unlike other situations, the direct doctor-patient relationship is lost and the physician has to rely on keen observations for whatever could be a key to the cause. History from relatives, friends or witnesses bringing the patient to hospital can be enormously helpful. A nurse who checks the patient's belongings may find cards indentifying the patient as diabetic, epileptic or she may find a drug prescription....!

Before wasting time in full general and neurological examination, one should:

1. *Ensure an open airway:*
remove any F.B., dentures; suck out secretion; ensure a patent oropharyngeal airway, otherwise if competent, intubate.

2. *Ensure proper supply of metabolic substrates:*
i) Oxygen: supply 8-10 L/min.
Attach to ventilator when indicated (associated chest injuries - respiratory failure as shown by ABG's).

ii) *Glucose:* 30-50 ml, 50% i.v.
Glucose can be life saving in cases of hypoglycaemia and, in other conditions, is never harmful. Thiamine 100 mg if the patient looks malnourished as in the alcoholic. Thiamine is preferably given before glucose.

iii) *Blood:* When pulse rate and B.P. denote shock, look for evidence of hypovolaemia or haemorrhage in case of injury (concealed abd. trauma is usually missed in the shadow of head injury.)

Cardiac failure will necessitate inotropics and when available CVP-PCWP should be monitored.

3. *Ensure optimum environment for neuronal function:*
As osmolality, pH, temperature and electrolyte integrity are essential for proper cell life, any abnormalities should be corrected whenever possible. In the majority of cases, $^1/_2$ Ringer's lactate is sufficient and can be rated 500 c.c. 8 hrly. for the adult unless cases of DKA, IADHSS will necessitate otherwise.

The traditional low head position is unnecessary when the patient is intubated, a N.G. tube inserted and haemodynamically stable. Trendelenburg's position is advisable when in shock. In case of head injury, head up position minimises brain engorgement and 2 sand bags around will stabilize the neck movement in case an associated cervical spine injury cannot be excluded.

4. *Treat aggressively any fit* as epilepsy is deleterious to brain tissues; no exception are allowed not to treat epilepsy right away: Diazepam 10mg to be repeated if necessary, is recommended as the ideal treatment.

5. *Investigation planning:*

A. Blood: whenever you are getting IV access, take blood samples for U+E, RBG, Calcium, LFT, CBC, ABG, and toxicology screening and observe any venepuncture denoting drug abuse.

B. Urine: catheterise aseptically and send urine for C&S, microscopy and toxicology screening. NG tube aspirate should be sent for toxicology as well when drug overdose is suspected.

C. Radiology: Signs of ↑I.C.P. may be seen on lat. skull X-ray. In case of head injury, x-ray skull, and a cervical spine will be fundamental. CXR is a routine in all cases. CAT scan whenever possible as in suspected structural causes, is highly informative.

D. EEG may bring to light the presence of an organic lesion, identify epilepsy, define the degree of the metabolic encephalopathy.

E. Lumbar puncture: whenever SAH or meningitis are suspected L.P. is crucial to diagnosis and management.

Now examine your patient

1. Define the *degree of the coma:* the best parameters are those of GLASGOW COMA SCALE:

	Parameter	Action	Score
A	Response to verbal commentary	oriented	5
		confused conversation	4
		inappropriate words	3
		incomprehensive sounds	2
		None	1
B	Eye opening	spontaneous	4
		to sound	3
		to pain to limbs	2
		None	1
C	Motor response	obeys commands	6
		localises pain	5
		flexion a. withdrawal	4
		b. abnormal	3
		extension	2
		None	1

A patient who scores 7 is certainly deeply comatose and the less score he gets on examination, the more gloomy the prognosis will be.

2. *General Examination:*
A. Head & Neck:
Ascertain the presence of head injury or its absence. Palpation of skull may reveal bogginess which indicates an underlying injury not seen by inspection.

Examination of the ear by otoscopy is crucial in any case of head injury. Neck stiffness indicates meningeal irritation by infection or bleeding. Neck examination is to be meticulously attempted with due care when head injury is present so that a cervical spine fracture would not worsen the condition. Kernig's sign is a variant of neck rigidity. The smell of alcohol, acetone, ammonia (hepatic fetor) can be informative of systemic disorder. The face may show stigmata of hypothyroidism, uraemic colour and frost, spider naevi, cherry red colour of CO poisoning, etc.

B. Skin:
look for venepuncture, bullous eruptions, boils, lipodystrophy, insulin injection sites, lipodystrophic diabeticum ulceration, etc. Profuse sweating is characteristic for hypoglycaemia especially when associated with muscle twitches.

C. Chest - Abdomen:
Make sure the presence of any concealed injuries in case of head trauma is not overlooked. Look for evidence of Chronic resp. failure, liver cirrhosis, uraemia, ...etc.

3 *Specific Neurological Exam:* attempting to localise the lesion:

A. *Ocular:*

i Pupil: size (normally 4-6 mm in dim light) symmetry and reaction to light. The pupil signs yield a lot of information, e.g.
unilat. dilated fixed pupil with contralateral (or less commonly ipsilat. hemiplegia) suggest uncal herniation of supratentorial lesions.
Bilat. fixed pupils indicate the final stage of rostral-caudal deterioration i.e. medullary failure.
Pin point pupil characterise opiate overdosage, or otherwise pontine lesions. Generalised brain oedema with mid-line herniation may cause symmetrical small pupils, but still reactive to light, a point which favours the diencephalon dysfunction in toxic-metabolic encephalopathy state.

Failure to react to light is a poor prognosis.

ii External ocular muscle movements:

A. Fixed lat. conjugate deviation.

1. with ipsilat. hemiplegia, Contralateral Brain stem or Thalamic lesion.

2. With contralat.hemiplegia, Ipsilateral cerebral lesion.

B. Downward tonic conjugate: thalamic lesion

iii Oculo-cephalic reflex:

Doll's Manoeuvre:

Normally passive movement of head towards the right will cause the eye to move to right. When there is diencephalon dysfunction eyes will move away from side of movement, while mesencephalic failure will result in no eye movements.

iv. Oculo-Vestibular reflex

Making sure no tympanic rupture instill 30 ml of iced water in the ear when his head is 30 degrees to the horizontal, normally a nystagmus with fast component away from the side of stimulation is maximally produced on lat. gaze towards the side stimulated. The action is lost when mesencephalon (brainstem) fails.

v. Corneal reflex:

If brisk and intact with fixed dilated pupils, undoubtedly the lesion is at mid-brain at site of oculo-motor N. with intact brain stem below that level.

B. *Motor System:*

Observe the posture of the patient.

i. Hemiplegia is readily seen by inspection, otherwise by failure to respond to pain to limb (pinching flesh, pressing nail-bed or deep pressure).

ii. Decortication: when bilateral diencephalon dysfunction occurs, lower limbs are extended and plantar flexed while upper limbs will show adduction at shoulder, flexion at elbow, wrists and fingers.

iii Decerebration: indicates mid-brain or pontine dysfunctions. Usually it is apparent on response to pain when limbs extend away. Like decortication LL are extended but UL internally rotated at shoulder and hyperpronated at forearms. Unlike decortication it is more likely to be unilat. when the other limb is paralysed.

iv. Flaccid limbs with no movements to any noxious stimulus indicate medullary failure.

v. Observe spontaneous twitches or movements particularly, myoclonus, flapping tremors.

C. *Respiratory Pattern*

i. Chyne Stoke: Alternate long hyperpnoea and apnoea of shorter period. Indicate diencephalon lesion.

ii. Central Neurogenic hyperventilation, with RR more than 40/m. indicates mid-brain lesion.

iii Apneustic breathing: full inspirations, interrupted by a pause, indicate pontine lesions.

iv. Ataxic irregular unpredictable pattern, indicates medullary dysfunction.

Finally *Difference between Metabolic and Structural* Causes (See Table).

References:

Adams, R.D. and Victor M.: Principles of Neurology (2nd Ed.) (1981)
Earnest, M.P.: Neurological Emergencies (1983)
Matthews, W.B.: Practical Neurology (3rd. Ed.) (1975)
Mayo Clinic: Clinical Examinations in Neurology (5th. Ed.) (1981)
Plum and Posner: Diagnosis of Stupor and Coma (3rd. Ed.) (1980)

Differences between Metabolic-Structural Coma

	Metabolic	Structural
Pathogenesis:	Biochemical	Anatomical
Interruption of ARS (ascending activating reticular system)	Toxic, deficiency, of substances and inhibitory effects.	Uncalherniation of supra-tentorial and central herniation of infratentorial lesions.
Site of Lesion of ARS interruption	Diencephalon (Cerebral Cortex)	Mesencephalon (Brain stem)
Pupils, reaction to light	Usually preserved pupil tends to contract (exception: in glutethimide poisoning)	lost unil. or bilat. dilated.
Motor	Decortication flapping tremors (asterixis) myoclonus jerks are common.	Hemiplegia ± decerebration is frequent.
Resp. Patterns	Chyne Stoke	Central hyperventilation, apneustic breathing, ataxic pattern.
EEG	Diffuse changes*	focal changes
CAT Scan	Normal	pathological lesions may be seen

* Massive Barbiturate overdose may cause iso-electric EEG