Title **Assessing Disorders of Consciousness**

Introduction

Assessing patients with a reduced conscious level is a core skill in intensive care. The diagnosis in such cases is often straightforward but can be challenging in the face of initial normal investigations. Many causes of reduced conscious level are clinical emergencies which require immediate remedial action.

The vocabulary of reduced conscious level is varied and sometimes confusing and overlapping. Reduced conscious level, encephalopathy, altered arousal, altered mental state etc are all terms used with subtly differing meanings – it is best to try and be precise in your descriptions.

Guideline Scope Assessing adult with non-traumatic coma – a guide for critical care doctors in training.

Evidence Review Not applicable

Main Guidance

Pathphysiology

Although perhaps simplistic, it is known that the proper function of two connected 'structures' in the brain is necessary to maintain usual consciousness. These are the

i) *Cerebral Cortex*. The thin grey matter covering of the brain, dense with the cell bodies of motor, sensory and other neurons must be functioning as a global whole to maintain consciousness.

Processes which impact on this global network will reduce conscious level. These tend to be diffuse processes, often affecting the physiological surrounding of cortical neurons – eg hypoxia, hyponatraemia, seizures.

ii) *Ascending Reticular Activating System(ARAS)*. This is best thought of as a large network of ascending neurons found in the upper brainstem (upper rostral pons) and projecting to the midbrain, thalamus, basal forebrain and cortex. Proper function of this dense network within the upper brainstem is needed to maintain arousal and alertness.

Disease processes which impact on this part of the brainstem will reduce conscious level by interrupting normal function of the ARAS. This tends to be caused by a *structural* brain injury, either local to the brain stem (eg a brain stem stroke) or by herniation and compression of the brain stem (eg large supratenttorial ICH with raised intra-cranial pressure).

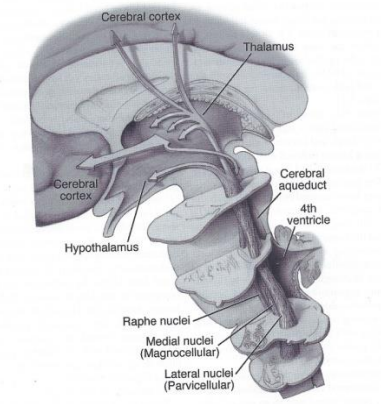


Figure 8.1 Ascending reticular activating system. By permission of Mayo Foundation.

A diagnostic approach should consider the likely pathophysiological processes which may be impacting on one or other of these structures.

The many aetiologies of coma may be very broadly grouped into three classes:

1. Diseases that produce focal or lateralizing signs (usual structural lesion with impact on ARAS)
2. Coma without lateralizing signs but with signs of *meningism*(implying infection or sub-arachnoid haemorrhage)
3. Coma without lateralizing signs nor signs of meningism (implying a'medical' cause of coma – a process impacting globally on cortical function). This group may be broadly termed encephalopathy which implies a process outwith the brain impacting on its function.

<u>Assessment</u>

History Although by definition there will not be a history from the patient, other witnesses etc will often provide very key and often diagnostic information (eg witnessed overdose, prolonged seizure etc).

General Examination Before a detailed neurological assessment, a general examination of the patient may point to the aetiology of coma.

- Smell on breath – eg ketones, alcohol.
- Skin changes – CO poisoning, IV drug use, signs of ALD.
- Changes associated with endocrine disease – eg hypopituitarism, hypothyroidism.
- Body temperature – CNS or other infection, extreme hyper or hypothermia as outright cause.
- Sources of systemic infection.
- Respiratory pattern.
- Heart rate and blood pressure.

Neurological Examination

- GCS – seek asymmetry of motor response indicating a focal lesion.
- Pupillary response.
    - Unilateral fixed, dilated – uncal herniation.

    - Bilateral fixed dilated Massive midbrain injury, TCAs, sympathomimetics.
    - Unilateral constricted – Horners.
    - Bilateral constricted – Opiates, metabolic encephalopathies, pontine or thalamic lesions.
- Fundoscopy – papilloedema, changes of hypertension, vireous haemorrhage in SAH.
- Eye Movements.
- Neck stiffness/ meningism.
- Limb movements and postural change.

Immediate management and baseline investigations will occur simultaneously.

Immediate management

- A,B,C interventions and appropriate monitoring.
- Treating clear cut aetiologies – eg hypoglycaemia, use of naloxone etc.
- Thiamine (Pabrinex) if Wernicke's in differential.

Baseline Investigations

- Immediate blood glucose +/- ketones
- FBC, U+E, LFT, Coag, Calcium, serum osmo, paracetamol/salicylate levels, consider urine toxicology
- Blood Gas inc carbon monoxide.
- ECG and CXR.
- +/- Blood culture where infection suspected.
- All patients without a clear cut diagnosis at initial presentation rendering imaging unnecessary, will undergo CT brain (+/- CT angio).

Often, the clinical picture, baseline investigations and CT will provide a diagnosis. If not the clinical assessment will direct second line investigations.

Where the diagnosis is not clear, early involvement of the Wd 18 consultant +/- neurology is mandated.

Second Line Investigations

<u>CT angiogram</u> – this should be thought of early at the time of original CT scanning if a neurovascular problem is within the likely differential (e.g. post ictal anterior circulation CVA, posterior circulation stroke). This should also be considered where the plain scan has demonstrated a bleed. CT venogram should be considered also where venous sinus thrombosis is a possibility.

<u>Lumbar Puncture</u> – ensure safe (no raised ICP suggested on CT), normal clotting and platelets. Remember HSV PCR in addition to baseline CSF tests.

<u>EEG</u> - where non-convulsive status epilepticus (NCSE) or other seizure disorder is possible, try and obtain an urgent EEG. If not possible, consider use of empiric anti-convulsant and discuss use of BIS or Phillips Monitor EEG.

MRI – this may be warranted as an urgent investigation for example to confirm brainstem CVA.

Pseudocoma

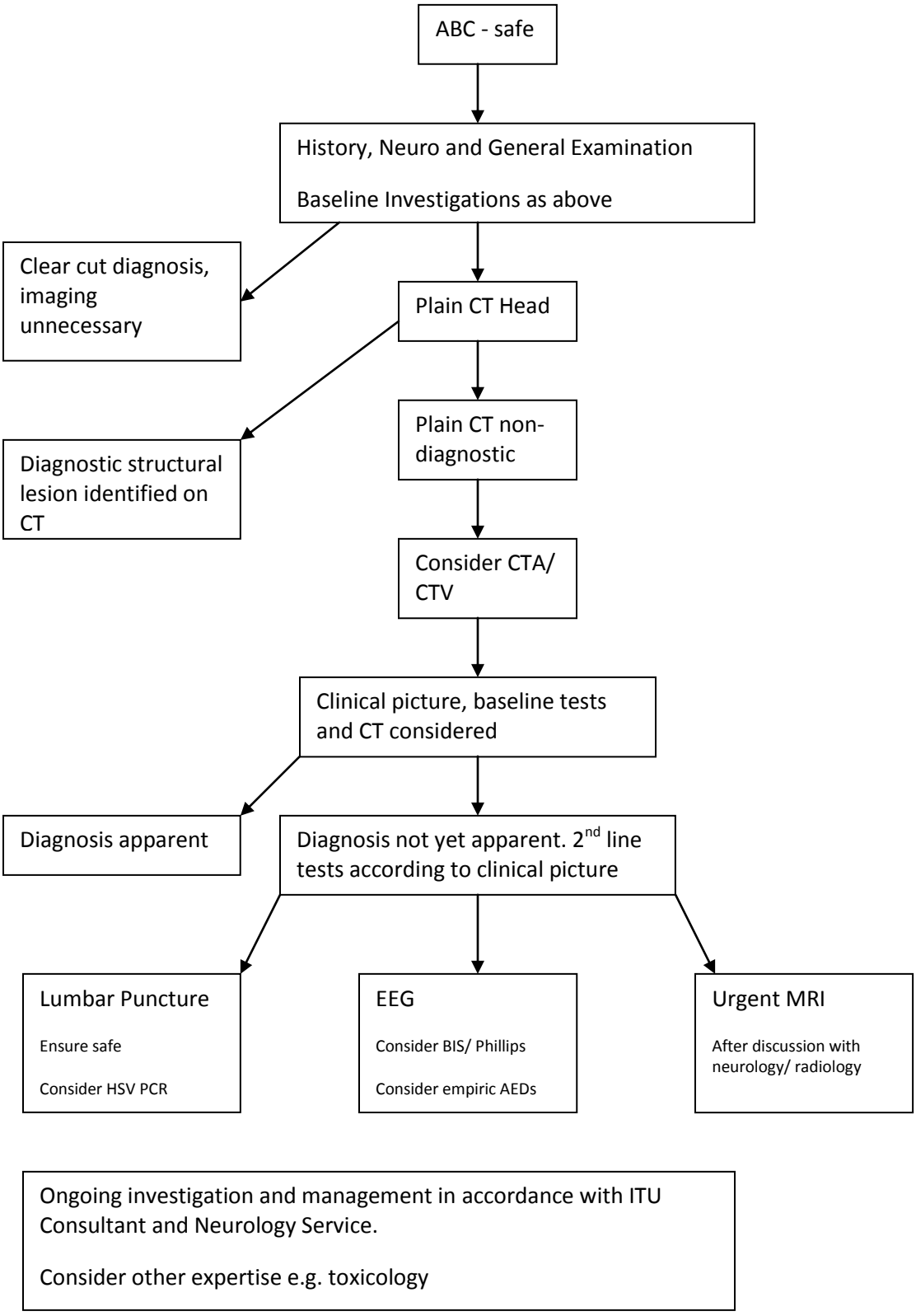
Although rare, patients with mental health issues may present with fictitious coma which may be difficult to spot. An index of suspicion should be raised by exam findings that do not fit with usual neurological presentations. Resistance to eye opening and painful manouevres may be observed.

Encephalopathy - Causes

Category	Examples	Diagnosis
Drugs and toxins	Alcohol Drug overdose (inc non intentional) Recreational Drugs Methanol/Ethylene glycol	Serum osmo/ blood alcohol/osmolar gap Paracetamol/Salicylate Level Urine toxicology (nb no immediate result)
'Gases'	O2 delivery – post arrest (HIE), hypotension, hypoxaemia Hypercarbia Carbon monoxide	Clinical ABG
Sugar/ Diabetes Related	Hypoglycaemia DKA Hyperosmolar Hyperglycaemic State	BM POC ketones
Seizure Related	Post-Ictal NCSE	Clinical EEG
Infection	CNS Infection Systemic Infection and septic encephalopathy	Meningism LP Systems findings
Metabolic	Sodium disorders Hypercalcaemia Unusual metabolic disorders	Blood tests
Organ Failure Associated	Uraemia Hepatic Encephalopathy	Blood tests

Endocrine	Hypopituitarism/ low cortisol Hypothyroid coma	Blood tests
Temperature	Hyperthermia Hypothermia	Clinical
Neurovascular – normal plain CT	Hypertensive encephalopathy and related – PRES Brainstem stroke Venous sinus thrombosis	Clinical CT angio/venogram MRI/ MRA/ MRV
Less Common Encephalitides	Autoimmune and Paraneoplastic	After exclusion of more common causes Neurology Consult

Broad Outline Flow chart for investigation and assessment of the unconscious patient



Notes

Even if infection strongly suspected form the outset, an unconscious patient should be imaged before LP.

Training, Implementation and Resource Implications

This guidance forms the basis for teaching critical care doctors in training.

Monitoring

Assessment of trainees performance in gaining this competency.

A.Vincent March 2014 Review March 2017