Unconsciousness

Professor Michael Veltman MBBS FANZCA FASE FFPMANZCA University of Notre Dame

Deputy Director Medical Services Joondalup Health Campus

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Outline & Learning Objectives

- Definitions of key terms
- Physiology of consciousness
- Assessment & Differential Diagnosis
- Management
- Medically Induced Coma

Definitions

Definitions

Consciousness
Sleep
Delerium
Dementia
Coma

Consciousness

- Self-awareness
- Access to memories
- Ability to manipulate abstract ideas
- Focus of attention



A state of reduced interaction with the environment

Reversible

Different from anaesthesia/coma.

Delirium

- Acute condition with altered mental state
 Organic basis
 Syndrome (not a diagnosis)
 Multiple causes.
 inability to focus attention & mental confusion
 - impairments in awareness & temporal and spatial orientation
 - Many similarities to coma.



Unrousable unresponsivenss.

- No response to pain.
- Reflects a lack of CNS function

Usually has preserved brainstem function

Brain mediated reflexes are preserved.

Brain Death

Irreversible state
Loss of all brain function
No response to pain
No brainstem reflexes
Exclusions
Temperature, drugs

Pathology with normal conscious states

Dementia

- Not the same as alteration of consciousness
- Alert mental state
 - Anosognosia (unawareness of illness)

Ischaemic Stroke

 Ischaemic stroke will only affect consciousness if brainstem affected
 Hemorrhagic stroke is different.

Usually has focal neurology.

Locked in syndrome

Awareness, sleep-wake cycles
 May have some meaningful behaviour

Due to

- High level spinal injury
- Guillain-Barré syndrome
- Parkinson's disease (severe) or similar

Pathology with altered conscious states



- Epilepsy is a condition of uncontrolled discharge of neurones
 - Generalised seizures are associated with a loss of consciousness
 - Partial seizures are associated with an altered conscious state

Narcolepsy

Due to the lack of orexin in the hypothalamus

Loss of stabilising switch

Sudden onset of sleep

Physiology of Consciousness

The Diencephalon



"Interbrain"
 Region of the embryonic vertebrate neural tube

 Gives rise to posterior forebrain structures

Role of Thalamus



 Directing sensory input (except olfaction)
 Motor function control
 Autonomic and endocrine function control
 Homeostasis

Reticular Activating System



Very Broad term Several nuclei: Midbrain Reticular Formation Mesencephalic nuclei Pontine Tegmentum Thalamic intralaminar nucleus Hypothalamus Is not the sole component of alertness

Physiology of consciousness



Within the diencephalon Thalamus Hypothalamus Epithalamus Ventral/Pre-Thalamus Third Ventricle

Transmitter systems associated with alertness

Monoamine systems
 Serotonin (raphe)
 Histamine (TMN)
 Noradrenaline (locus ceruleus)



Cholinergic systems AcetylCholine

Brain stem (LDT and PPT) project to thalamus

Forebrain (basal nucleus of Meynert) to cortex

Sleep/Wake Regulation



Electroencephalography



EEG patterns



Spectral Frequency



Consciousness is associated with:

- Higher frequency firing of neurones
- Synchronised discharges across larger areas of the brain.

Assessment and Diagnosis

Assessment

Start from basics. Is the person unresponsive Airway Breathing Circulation If so ALS or BLS algorithmns apply

ALS causes of unconsciousness

These are emergencies
 Need to treat quickly
 Usually within a few minutes

The H's of ALS

Cause	Altered mental state	
Hypovolaemia	When MAP < 60mmHg (≈80/50)	
Hypoxia	When $DO_2 < 400 \text{ml/min} (SaO_2 < 60\%)$	
H+ (Acidosis)	With pH < 7.0 or > 7.6	
Hyper/hypokalaemia	Only with arrythmias (K+ < 3.0 or > 8.0)	
Hypoglycaemia	When BSL < 3.0 or > 30	
Hypothermia	When core temp < 28° Celcius	

The T's of ALS

Cause	Altered mental state	
Toxins	Depends on drug	
Tamponade	When MAP < 60mmHg (≈80/50)	
Tension Pneumothorax	When MAP < 60mmHg (≈80/50)	
Thrombosis (AMI, PE)	When MAP < 60mmHg (≈80/50)	
Trauma (Head)	Need raised pressure (CPP > 60 mmHg) *Head injuries may cause hypertension*	

Cerebral Perfusion Pressure

Cerebral Perfusion Pressure
 Mean Arterial Pressure - Intracranial Pressure
 Needs to be > 60 with acute head injuries
 Normal ICP 11 mmHg
 Mild head injury 20 mmHg
 Severe head injury >40 mmHg

Assess Conscious State

Glascow coma scale probably the best
 Three areas of assessment
 Eye movement (Scale of 1-4)
 Verbal responses (Scale of 1-5)
 Motor responses (Scale of 1-6)

Assess Conscious State

	Eye	Verbal	Motor
1	Closed	Silent	Immobile
2	Opens to pain	Incomprehensible sounds	Extension to pain Decerebrate
3	Opens to voice	Inappropriate words	Flexion to pain Decorticate
4	Opens spontaneously	Confused or disoriented	Flex/Withdraw to pain
5		Oriented, conversations normal	Localises pain
6			Obeys command

Interpreting the GCS

Assessing head injury
 Severe, with GCS < 8 (coma)
 Moderate, GCS 8 –12
 Minor, GCS ≥ 13.

Any reduction in GCS from 15 is abnormal
 GCS falling over time is an emergency.

Differential Diagnosis

- Very long list of causes
 - All disease states end in coma and death
- Focus on ones where there is no obvious other cause

"AEIOU TIPPSSS"

Alcohol
Epilepsy
Insulin & glycaemic changes
Overdosage of drugs
Uraemia & metabolic causes

TIPPSSS

Trauma to head Infection (esp in elderly or if intracranial) Raised intracranial pressure Psychiatric disorder Stroke Simple Feint Stokes-Adams (cardiac arrythmia)

Predisposing Causes

Cognitive impairment / dementia

- Comorbidity
 - Older Age
 - Dehydration / Malnutrition
 - Drug and alcohol use
 - Psychiatric e.g. depression
- Sensory impairment (vision, hearing)
- Functional dependence

How to assess

History and examination, basic observations
Basic chemistry
EUC, BSL, FBP, Ca++, ABG's
Toxicology
TFT's, LFT's
Imaging - CT Head (vs MRI)

Management

Basics

Remember ABC's - fix these first
Assess Glasgow Coma Scale
If rapidly falling GCS, or if GCS < 12
Medical Emergency - minutes count
If stable (over hours)
Requires urgent investigation.



- General management
 - Support basic organ systems
 - Obtain a diagnosis
 - Manage specific problems and complications

Rapidly Falling GCS

ALS algorithmn
 IV access (+ take bloods)
 Intubate & mildly hyperventilate

 Aim for PaCO₂ = 30mmHg
 Investigation (including brain imaging)
 Only do lumbar puncture after imaging

Emergency Management

Conditions you need to have a plan for

- Seizures
- Altered plasma glucose
- Raised intracranial pressure
- Head trauma
- Sepsis



Management (after ABC's)
 Midazolam 0.1 mg /kg or Diazepam 0.15mg/kg
 Phenytoin 15mg/kg over 30 minutes

Can consider Thiopentone/Propofol

Hypoglycaemia

Glucose 50%
 25-50 mL
 Need larger bore needles and cannula

Raised ICP

Imaging essential to exclude masses
 Medical management

 Mannitol (0.25-0.5 g/kg) ≈ 150ml 20% mannitol
 Hypertonic saline
 Prevent hypercapnia

 Surgical management

Trauma

Primary & Secondary injury
Aim to minimise secondary injury
Aim to normalise
CO2, BSL, ICP, CPP
Aim to prevent
Seizures, Hyponatraemia.



Time to intervention is important:
 Get samples (blood/urine/sputum/etc)
 Antibiotics

Supportive management (eg for BP)

Induced Coma

Rationale for Benefit

Anaesthesia (surgery and procedures)
 Mostly for amnesia - different from coma
 Induced coma in ICU
 To allow hypothermia
 Trauma

Management of head injuries

Induced coma

Benefit in non traumatic head injuries
 Reduce core temperature to < 35°
 Must be done shortly after brain injury
 No benefit with trauma
 Need to stop normal thermoregulation



Mostly to control airway and PaCO₂
 Usually requires paralysis
 Thiopentone/Propofol + suxamethonium
 Midazolam + rocuronium

Occasionally for oxygenation.



Inhalational anaesthetics
 Intravenous anaesthetics
 Benzodiazepines
 Other sedatives

Inhalational Agents

Nitrous Oxide, Methoxyflurane
 Desflurane, Sevoflurane, Isoflurane
 Advantages
 Easy to administer

- Measure depth of anaesthesia
- Minimal tolerance

Analgesia with N₂O and Methoxyflurane

Intravenous agents

Proprofol

- 2 mg/kg induction, 30-50 mg/kg/hr (maintence)
- Fast onset and offset
- Thiopentone

3-5 mg/kg induction, maintence more complex
 Reduces ICP and Cerebral O₂ requirements
 IV agents match O₂ with blood flow.

Benzodiazepines

MidazolamDiazepam

Note differential effects on amnesia versus coma.

Other Agents

Analgesics: Opiates

 μ receptor agonists

 Sedatives: Clonidine, Dexmetomidine

 α2 receptor agonists

 Ketamine

 NMDA receptor antagonists

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