

Coma + Delirium

consciousness: state of awareness of self + surroundings
alterations = coma: arousal affected
= delirium/dementia: content of consciousness altered
sleep.

Coma : unarousable unresponsiveness

Anatomy: Reticular activating system "alerting system"

located:
• upper brainstem tegmentum ^{upper part of brain}
• lower diencephalon: certain thalamic nuclei

↓ projects to whole cerebral cortex + receives recurrent cortex modifier received info. innervation.

Disruption of this system = disruption of consciousness.

2 Broad Groups = structural
metabolic + other

• Structural: Mass lesion =
tumor
abscess
hemorrhage
infarct
granuloma

Lesion in RAS = brainstem stroke
thalamic hemorrhage.

Widespread bilateral damage to cortex + white matter

Mass lesion causes:

- distortion of deep structures
 - ± herniation of medial temporal lobe through tentorium cerebelli
 - deep, ^{central} structures laterally disl result in = compression
irrhaemia
hemorrhage } in consciousness producing structures
- cerebellar mass lesions similarly distort part.
Fossa structures → leads to ↓ consciousness

↳ trauma
meningitis
encephalitis
hypoxia
ischaemia

Metabolic

hypoxia: NB trauma pt "drunk"
hypercapnia
hypernatremia
hypoglycaemia: NB
hypothermia
uremia
hepatic F
DKA

Drugs

sedatives
barbiturates
EtOH
opiates
salicylates
anticholinergics
amphetamines

Toxins

methanol
ethylene glycol
CO
mushrooms

Infx: bact. meningitis ✓
viral encephalitis
sepsis
typhoid
malaria NB travel hx.

Epilepsy

ictal | post ictal | drugs

Psych - catatonia

Other

diffuse cerebral isch.
fat embolism
hypotension
hypertension

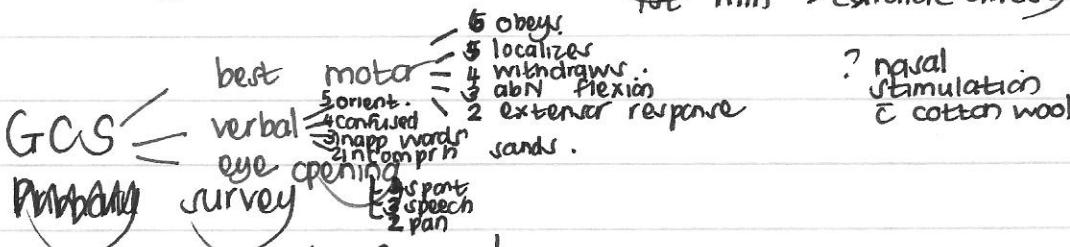
Approach

30:2 if alone

- Stabilize {
- C : check pulse + start compressions if none
 - A : open airway = ^{head tilt chin lift} NB stabilize spine
 - B : ^{Jaw thrust} ^{(may be present in} ^{1st min > cardiac arrest)}
NB agonal gasps = x breathing

Glucose

Disability



Exposure | Environmental Control

Adjuncts to 1° survey = ECG leads
catheters (urine / NG)
sats
BP.

Now: special investigations based on findings | ddx.

NB Hx.

Delirium

Neuropsychiatric \$ \leftarrow acute onset - usually hours \rightarrow days.
fluctuating course - may have lucid intervals
reduced ability to sustain attention

DSM 4

At least 2 of

- perceptual disturbance - (misinterpretations / illusions / hallucinations)
- incoherent speech
- disturbance of sleep-wake cycle
- ↑ / ↓ psychomotor activity

Pts range from hypoactive + blunted to raging uncontrollable maniac
often worse @ night.

- Attention deficit: cardinal sx. Distractable
- Unable to maintain stream of thought.
- Disorientated to time most common.
- Loss of personal identity unusual.
- Recent memory impaired + worsened by attentional deficit
- Often persecutory delusions.
- Insight + judgement impaired: may confabulate
- May appear to be depressed: esp elderly pts.
- Writing may be impaired by motor + spatial impairment + spelling + syntax errors.

NB to recognise the delirium

Any sudden cognitive problem, particularly arising in hospital /
should presumed to be delirium

Special Invest : bloods NB drug screen \leftarrow blood
urine. cap. elderly!!! ↑
OT
CXR. (NB)
LP.

Mx: ID + Rx cause

Environmental + supportive measures

orientate pt every time you see him
educate staff + family to do the same

light (also @ night)

noise

warmth + nutrition

adequate environment safe for pt + others.

LD avoid restraint!!

Drugs (only when essential)

Antipsychotics: Haloperidol
(low dose)
can give parenterally
can also use other APsHs

Benzos: 1st line if seizures / EtOH inv.
useful adjunct to
pt manageable not obtunded.
Watch BP / resp.

Lorazepam
(↓ elderly / hepatic D)

Watch out:
hypoxia
UTI / pneumonia in elderly

Gullion Barre

Acute inflammatory demyelinating polyneuropathy

- cell mediated immunological reaction directed @ periph nerves
- reduced suppressor T-cell response
 - occasionally s- antibodies to myelin components found (X rationally measured helpful reflexes)
 - Term for a collection of clinical syndromes, although classic variant
 - description is a demyelinating neuropathy & ascending weakness
 - segmental n. damage
 - lymphocytes infiltr. n. roots + release cytotoxic substances that damage Schwann cells + myelin
 - myelin removed by microglia/macrophages
 - If axon damaged n. cell dies + can't regenerate

Clinical

- 3w after onset viral/bact illness - resp most freq GvH secondary Ag in capsule shared w/ nerves.
- pain suggestive of n. root irritation (50%)
 - paresthesia lower limb

CN may be affected : facial, n. most common. may be 1st finding often unilateral undistinguishable from Bell's Palsy

Weakness develops over 3 → 21d, clinically ascending, symmetrical

may be generalized

May worsen over commences distally + ascend

Loss tendon reflexes, plantar responses remain normal. (↓ tone can be significant weakness)

May be autonomic inv - fluctuating BP, facial flushing, tachycardia / bradycardia, anhidrosis / diaphoresis, urinary retention / constipation.

Sensory signs infrequent, usually digital, limb changes

most pts report paresthesia, numbness or similar sensory changes

Investigations

CSF prot ↑ > 2nd/3rd wk of illness. (speculated due to widespread inflammation)

(gamma globulin fraction ↑, cells can't)

EMG: prolonged conduction velocities (late)

Virology: occasionally rising titres to specific viruses

Comptibacter aerology: + = poorer px

May do stool culture for C. jejuni

NB

↑ short acting
e.g. beta block ↓ BP
IV fluids
supine posture
tachy
brady

A B C arrhythmias

Autonomic dysfunction
be prepared!

arrhythmias
temp paeng for
2° 3° heart block.

Any degree of resp F
Intubate

Possible resp failure NB rarer late sign.

- hypoxia
- rapidly declining resp funct
- poor/weak cough
- suspected aspiration

NB 1F deltoids + biceps weak : diaphr next.
next flexion weak / CN inv : danger sign.
- forced vital capacity < 15 mL/kg

See pt tchr! Do peak flows!

1/3 = ICU labile dysautonomia.
FVC < 20 ml/kg
res - bulbar palsy

Rx pain carbamazepine
gabapentin

NB

prevent thromboembolism
+ → sepsis
- physio → pneumonia

Only plasma exchange proven effective → shortens recovery time
Polygam IV immunoglobulin have

± 50% most experience long-term residual neuropathy
↓ QOL + physical functioning