Emergency viva topics

Contents

Cardiac arrest

Response, breathing, pulse, call for help

Chest compressions

100-120/min
Over middle of the lower half of the sternum
5-6 cm depth
Allow complete recoil
ETCO2 > 10 mmHg indicate adequate compressions

Oxygenation – ventilations 30:2

Triple manouvre
OT airway (philtrum to tragus / angle of mandible) or LMA. Quick intubation by an experienced person (LMA does not prevent aspiration)
Continuous ventilation possible only after ETT

Defibrillator – rhythm check AFAP, < 10s
Shockable – pulseless VT, VF 200 J
Non shockable – PEA / asystole adrenaline 1:10000 10 mL IV push, repeated every 4 minutes (every other rhythm check)

Amiodarone 300 mg IV push and **adrenaline** 1 mg (1: 10 000 in 10 mL) after the 3rd consecutive shock
Can repeat after the 5th shock at 150 mg dose as IV push
Lidocaine 1mg/kg is an alternative

Look for reversible causes
Hypoxia
Hypovolemia
Hypothermia
Hyper/hypokalaemia, Ca

Thrombosis – PE, MI
Tension
Tamponade
Toxin

ROSC
Pulse, BP, SpO2 / arterial BP waveform
Sustained rise in ETCO2 over 35-40 mmHg is the best marker

Post arrest

Ventilation, SpO2 94
Circulation dobutamine
Higher care

Hypothermia 4 C NS / Hartman IV 1-2 L

If following commands – cath lab???

SVT	50-100	
AF	120-200	200
VT	100	
VF	100-200	360

Acute chest pain

1. MI
2. PE
3. AD
4. Pneumothorax
5. GORD
6. Acute pericarditis

Acute SOB

1. LVF/MI
2. Acute severe asthma / COPD
3. PE
4. Pneumothorax
5. Cardiac tamponade
6. Anaphylaxis
7. Acidosis metabolic

Pulmonary embolism

Old classes : Massive – hypotension / Submassive (high markers) / Low risk
New classes : high risk (hypotension) / IM risk (PESI III/IV) / low risk (PESI I/II)

Dead space surfactant resorption collapse. Infarct rare, if occurs Hptysis, pleuritic pain

Well's score – to determine probability of PE

PESI score – for risk assessment
Background (age, cancer, CCF, lung disease), presentation (HR, SpO2, RR, SBP, T, mentation)

Hestia score – to determine which patients can be managed as outpat

Dx & Rx
Hypotensive patient CTPA / TTE : RV overload fibrinolysis
Normotensive patient
Confirm Dx

PE likely (Well) □ CTPA □ confirm
PE unlikely (Well) □ D-dimer □ CTPA □ confirm

Assess risk

PESI III/IV : intermediate

RV strain AND high TnI/BNP-high : AC, consider rescue reperfusion

Neither : IM-low risk : AC

PESI I/II : low risk : Consider early Discharge on OACs. Subsegmental PEs w/o DVTs may not benefit from ACs at all

Supportive

ECG tachy, AF, arrhythmia, RV strain, RBBB, S1Q3T3

2DE RV strain, empty left heart. Typical – McConnell sign – akinetic mid wall and normal apex of RV.

Fibrinolysis

SK

250 000 IU over 30 min □ 100 000 IU/h IVI for 24h OR
1 500 000 IU over 2h

rtPA

urokinase

other - TNK

	SK	rtPA	
	Binds to plasminogen and converts to plasmin	Binds to fibrin and activates plasminogen within the clot	
	250 000 IU over 30 min □ 100 000 IU/h IVI for 24h OR 1 500 000 IU over 2h	100 mg IV over 2h OR 0.6 mg/kg (max 50 mg) over 15 min	
UFH	Stop during SK infusion	Continue during rtPA infusion	Stop UK infusion
LMWH / fonda			
	Hypotension, anaphylaxis, bleeding		

Shorter duration infusions achieve faster T'lysis, but not FDA approved,
Small scale head to head comparisons shows equal efficacy. rtPA preferred due to short duration of infusion

Anticoagulation
LMWH / fonda
Give T'lysis Start UFH short term infusion after 12h from enox or 24h from fonda dose (risk of bleeding and need for rapid reversal) before restarting LMWH

Half dose – weak recommendation
Low dose catheter directed reperfusion : effective but needs expertise

Surgical embolectomy – when F'lysis is CIn or fails

PEITHO (Pul EMb Thrombolysis trial)
UFH + TNK bolus Vs UFH + placebo for patients with **submassive PE** (intermediate risk PE)
TNK group had less deaths / HD collapse within 7d but greater major bleeding, ICH. Risk greater than benefit. Therefore, thrombolysis not recommended for submassive PE (PE without hypotension)

Anticoagulation
UFH / enox / fonda
VKA / NOACs – RECOVER, EINSTEIN, AMPLIFY, Hokusai et al

Assess for the cause
Cancer
Thrombophilia – APLS, familial, PRV, CTD
Immobility – air travel, post Sx

Treatment outline
1. Hypotension ?
2. Wells, D dimer, CTPA, TTE, PESI
3. Anticoagulation
4. T'lysis SK / rtPA / UK- if this fails, surgery preferred over re T'lysis
5. Surgical embolectomy
6. Catheter directed T'lysis
7. IVC filter

PE in pregnancy – same management. If peripartum consider surgical options over T'lysis

ACS

STEMI equivalents
deWinter
Wellens – dynamic. Poor response to Tlysis, needs angio and reperfusion. 75% get STEMI within 1 week
STE in aVR
LBBB with Sgarbossa
Isolated posterior MI
Upright T in V1

LBBB and RBBB in a pt with CFx of ongoing ischaemic pain – need angio coz STEMI cannot be ruled out

Acute LVF

Hx – orthopnea, chest pain
Ex –rapid thread pulse, cold clammy peripheries, high JVP, gallop, cardiomegaly, basal crackles, dependent edema

Classification
Cold / Warm / wet / dry
Killip

I	no signs
II	rales and gallop
III	frank pulmonary edema
IV	hypoperfusion

Management

Congestion – prop up, O2, freusemide, GTN, CPAP, Ultrafiltration, ventilation
Hypoperfusion – inotropes, fluid challenge if dry, LVAD, IABP
Treat the cause
Prevent DVT

1. Monitor
    a. HR BP SpO2, IPOP, Cr, Sy, Signs
2. Ix
    a. ECG, 2DE
    b. BNP / NT pro BNP / MR proANP -
    c. Cr, SE, LFT
    d. Procalcitonin / CRP, FBC, UFR, CXR
    e. TSH – both hypo hyper cause acHF. Recommended to be done
3. Oxygen if < 90% / PaO2 < 60 mmHg
4. IV frusemide : bolus / infusion. Min 20-40mg IV bolus. If already on frusemide, give a doubled dose. Can repeat 2 hourly, doubling the dose. Max dose per 24h : 400 mg. IV 5-10 mg/hour
5. GTN as long as SBP > 90 (ESC 2016)
    a. Reduce pre and after load – may increase SV
    b. Reduce pulmonary congestion
    c. Caution in MS and AS
    d. 10-20 mcg/min, max 200 mcg/min
6. CPAP / BiPAP – SpO2 < 90%, RR > 25/min. Caution : hypotension
7. Ventilation
   In : hypoxia (< 60 mmHg) / hypercapnia (> 50 mmHg) / acidosis (< 7.35 - ?resp)
8. Inotropes – for symptomatic hypotension
    a. Dob / Dop / levosi / milrinone (latter 2 circumvents BB effect. Both are inodilators – hypotension may worsen)
    b. Vasopressor – Norad
9. Mechanical circulatory support
    a. IABP – ACS, peri op, myocarditis
    b. LVAD
10. DVT prevention

11. Rx the ppt cause – CHAMP
    a. ACS – revasc
    b. HTN – vasodilator IV
    c. Arrhythmia – BB / digoxin / amoidraone / TPM
    d. Mechanical – IE, post MI mech complication, valve disease
    e. PE
    f. Infection- Abtc

Dob B1 B2 5-20 micg/kg/min
Adr B > alfa
NA alfa > B
Dop DA > B > alfa
Levosi calcium sensitizer (by modulating TnC) and vasodilator (by activating K channels)
Milri PDE-3i. increase cAMP inotropy and vasodil

DOSE trial - frusemide
High dose (2.5 x home dose) > low dose in efficacy. High dose caused more renal impairment
Continuous = intermittent in efficacy

HTN emergencies

Emergency	BP target	1st line	Alternative	Try to avoid
malignant HTN	20-25% MAP reduction Several hours	Labetalol (relatively less reduction of cerebal perfusion and edema cf nitrates) Nicadipine	Nitroprusside	GTN – cerebral vasodilation
Encephalopathy	20-25% MAP reduction Immediately (U2D: 10-20% by 1h. don't exceed 25% by 24h)	Labetalol, nicardipine	Nitroprusside	GTN – cerebral vasodilation
Ischemic stroke	< 220/120. If higher, reduce MAP by 15% over 24h (faster for other In – MI, AD etc) For T/lysis: < 185/110 & keep < 180-105 If none of above, no need to reduce within 1st 5-7 days	Labetalol	Nicardipine, nitroprusside	GTN – cerebral vasodilation
ICH	Debatable < 180 immediately (target 130-180: no consensus) (AHA: IV Rx if SBP > 220)	Labetalol	Nicardipine, nitroprusside	GTN – cerebral vasodilation
ACS	< 140 immediately	GTN, labetalol GTN may cause tachy, control with labetalol	Urapidil	**nitroprusside** reduces perfusion through diseased coronaries & worsen damage.

Pul edema	< 140 immediately	Nitroprusside (better pre and afterload reduction than GTN – will need GTN in a high dose) + frusemide	GTN + frusemide Urapidil + frusemide	**Labetalol** – negative inotrope
Aortic dissection	SBP < 120 and HR < 60 immediately	Esomolol + GTN / nitroprusside / nicardipine (BB is the cornerstone: reduce both HR, BP) Esomolol has faster onset and offset of action than labetalol. So easy to titrate	Labetalol	**Nitroprusside/GTN alone** -> SNS □ high ventricular force & rate **Hydralazine** □ direct arterial dilator, increase aortic shear stress, erratic BP reduction
Eclampsia / pre eclampsia / HELLP	< 160/105 immediately	Labetalol, hydralazine		

Although high RAAM is the driving force, degree of RAAM activation shows a marked variability among individuals. RAAM blockers may cause too rapid to no reduction in BP. Therefore are not suitable for hypertensive emergencies

IV labetalol dosing: IV 2-4mg/min until target is achieved. Then 5-20 mg/h

Hypertensive encephalopathy

Loss of autoregulation – posterior brain is more vulnerable due ot weaker SNS innervation □ PRES
Cerebral edema, microhemorrhages, microinfarcts. Focal signs are unusual, if present think stroke.

MDMA

HTN : BDZ, phentolamine (alfa), nicardipine, GTN. Avoid BB – unopposed alfa □ worsen coronary ischemia
Ischemia : GTN, aspirin, angio
Tachy : dilt, verap

Pheo : phentolamine (alfa), nitroprusside, nicardipine

With AR – avoid BB, DIltiazem etc □ low HR □ increase time for regurg □ worsens AR!

INTERACT-2: Rapid BP reduction (< 140 within 1h Vs < 180) in ICH did not improve 90d functional outcome or death. Vairety of antiHTN used – their individual influences not known

ATACH-2: 110-140 Vs 140-180 no difference (achieved SBPs: 129 Vs 141) in functional outcome or mortality. Insignificant reduction in hematoma expansion

Shock

Inadequate tissue perfusion due to absolute or relative reduction in cardiac output

Causes

With high JVP

cardiogenic,	MI, myocarditis, valve accident, aortic dissection, tachycardia
obstructive	PE, tamponade, Tension pneumothorax

with low JVP

with warm peripheries		
	distributive	septic, anaphylactic, high output HF
with cold peripheries		
	hypovolaemic	hemorrhagic (GI, hemothorax, girdle #, AAA), 3rd spacing (pacnreatitis, rhabdo, diarrhea / vomiting)
mixed		Addison, septic

mgt

ABCDE
Keep flat
Wide bore cannulae / IO cannulation
Blood for – FBC, CRP, SE, RBS, cortisol, cultures RFT, LFT, VBG (lactate), DT, amylase
Attach to monitor – HR, BP, SpO2, temperature

Rx

IV NS 200 mL over 5-10 min – safe even in cardiogenic shock. If responding give further fluids. For large volumes, prefer Hartman (hyperchloremic acidosis with NS)
Further mgt according to cause

Cardiogenic	MI Rx, dobutamine, adr, norad, levosi, milrinone, IABP, LVAD. Specific Rx (PCI, DCC)
Septic	Abtcs, fluids, source eradication
Hypovolemia	fluids, blood (Hb 8)
Anaphylactic	IM adrenaline 0.5 mg
PE	T'lysis
Tension	needle ICT
Tamponade	pericardiocentesis
Addison	HC

Problems with inotropes

Tachyarrhythmia
Myocardial strain
Digital ischemia with vasopressors
Tachyphylaxis

Monitoring the critically ill

Circulation

Ventilation / oxygenation – ARDS, VAP
Fluid balance / hydration
Nutrition
DVT, stress ulcer, sepsis

Anaphylactic shock

Diagnosing anaphylaxis

After exposure to a known allergen
    Hypotension alone OR
    2 from – resp / skin / GI / hypotension

With or without exposure / allergy history, acute onset
    Skin AND
    resp / hypotension

management

ABC
IM adrenaline 0.5 mg.

- can repeat the same dose every 5min. usually responds within 1-2 doses
- IV : 1: 10 000. Or more dilution. Rate 0.1-0.2 micg/kg/min. under ICU monitoing – tachyarrhythmia, severe HTN, coronary ischemia (Counis Xd)
- SC: avoid. Erratic absorption
- Nebulized adrenaline – can be considered for laryngeal edema
- How does adrenaline act? – alfa – vasoconstriction, B1 increase CO, B2 – reduce bronchospasm, mast cell stabilization

Remove allergen
2nd line Rx : IV HC 200mg, IV chlopheniramine 10 mg stat, consider salbutamol nebs
Oxygen, IV fluids 500 mL over 5-10 min
Keep supine, raise legs. DO NOT SIT UP SUDDENLY empty ventricle Xd : sudden death!

Refractory anaphylaxis

Rule out:

- Adrenaline errors - injected too late, wrong dose, short needle, expired / light exposed vial
- Patient suddenly stands or sits or is placed in the upright position after adrenaline injection
- Patient taking a beta-adrenergic blocker which interferes with effect of adrenaline
- Kounis Xd -
- Error in diagnosis

Mgt of refractory anaphylaxis

Adrenaline IVI
Other vasopressors
Glucagon (bypasses B receptors – useful if on BBs), ipratropium (useful if on BB)

???? duration of steroids ????

Arrhythmia

Tachy

BP, GCS, LVF, chest pain
- Yes cardioversion. DCC sync. Can consider adenosine for regular narrow
- No
  - Broad complex – IV, ECG, amiodarone infusion. Adenosine if SVT+BBB/aberrancy
  - Narrow complex – IV access, ECG< vagal, adenosine, BB/CCB – definitive

Adenosine 6mg bolus, NS flush – 12mg bolus and flush
Amiodarone 150 mg over 10 min and infusion 1mg/min over 6h (ACLS tachy algo), (for VT with pulse) procainamide, flecainide

amiodarone for AF – 300mg over 20-30min, 900 mg over 23h

VT MI, K, Mg, pH, ECG, TnI, TTE
Monomorphic VT
Pulseless cardiac arrest mgt
Unstable sync DCC
Stable BP IV amiodarone (alternatives – IV sotalol, flecainide, lignocaine)
Consider MgSO4 (8 mmol over 2-5min 60 mmol in 50 mL of 5%D over 24h)
Sync DCC if refractory
Overdrive pacing

Polymorphic VT

Atrial fibrillation
HD unstable – DCC ? with UFH bolus

HD stable acute rate control with BB / CCB / digoxin
Then plan long term plan – rate Vs rhythm
- Rhythm – electrical or chemical
- If AF < 48h – start AC, cardiovert, continue OAC at least 3 weeks
- If > 48h – AC x 3 weeks – cardiovert, continue OAC
  - Or do TOE rule out clot and cardiovert

200mg tds 6 weeks, 200mg bd 4 weeks, 200mg daily thereafter

bradycardia

hypotension, LVF, chest pain, confusion

no – W & W

yes – atropine 0.6mg repeat 3-5min upto 5mg

transcutaneous pacing

epinephrine, dopamine, isoprenaline

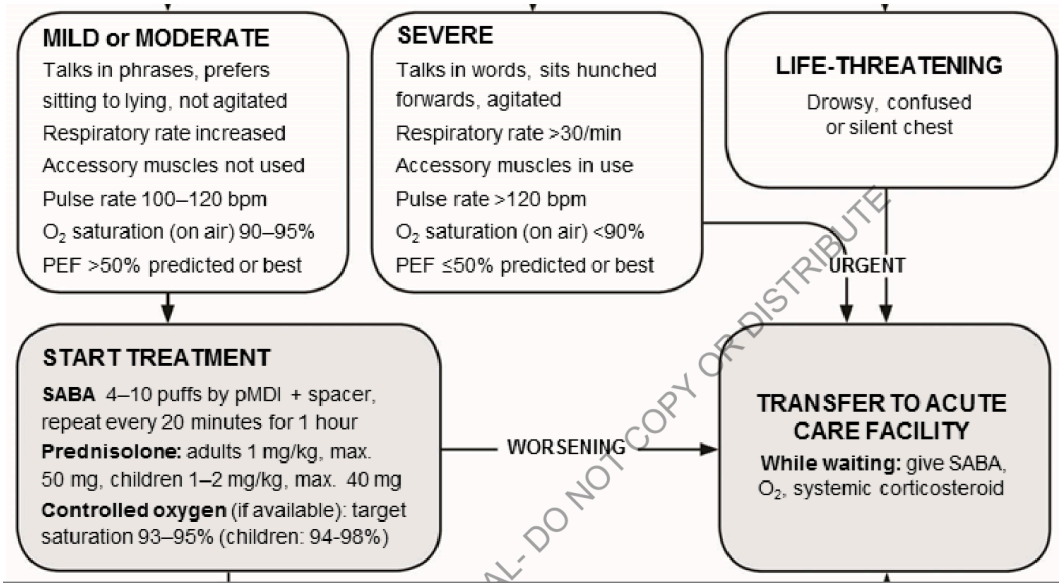
TPM, PPM

Acute severe asthma

ASA phrases, HR > 100, RR > 25 PEFR 33-50%

LTA resp failure (cyanosis, silent chest, NL CO2, SpO2 < 92%), hypotension/arrhythmia, confusion/agitation, pEFR < 33%

NFA high PaCO2



Assessment

Severity

DD HF, pneumothorax, PE, pneumonia

Ppt allergen, compliance, infection (URTI, LRTI)

Rx

Sit up

High flow oxygen
Nebulize salbutamol 5mg, O2 driven, 15-30 min / back to back 4 hourly
Nebulize ipratropium
IV HC 200 mg stat 100mg qds
IV MgSO4 1.2-2g single dose. Do not repeat – muscle / respiratory paralysis
Rule out pneumothorax, PE, HF, pneumonia
ICU
- CPAP cautiously
- Intubation with ketamine for dissociative anesthesia with BD properties. RSI
- Inhalational halothane etc are also helpful
- Maintain sedation and paralysis
- Allow prolonged exp, low TV (6-10 mL/kg), low pressure
- Watchout for barotrauma and hypotension

Treat ppt cause – Abtcs
Support – hydration 2-3 L/day
Prevent DVT and stress ulcers

?? any anesthetic agent to avoid????
Permissive hypercapnia essential ?? LITFL

Taper Rx with clinical improvement
Neb salbutamol 4 6 8 hourly SOS
Neb ipra 6h off
IV HC PO pred 30-60 mg/d – total of 14d
Complete Abtcs

Discharge criteria
1. Home therapy for > 24h without need for rescue therapy
2. PEFR > 75% of best, diurnal variation < 25%
3. Technique checked and corrected, PEFR chart
4. High dose ICS started, LABA started
5. Ppt identified, written plan for acute care
6. Review arranged (2d with local care provider, 1-2 week at clinic)

COPD exacerbation

DD
Infective or non infective
Pneumothorax – CXR, CT Chest
Pneumonia
Other – MI/LVF, PE

Classification for hospitalized pt

Parameter	Mild	Mod	Sev
Respiratory rate	20 -30 bpm	>30 bpm	>30 bpm

Use of accessory muscles	No	Yes	Yes
Mental status	No change	No change	Acute change
Improvement of hypoxemia with oxygen	improved with supplemental oxygen given via Venturi mask 28-35% inspired oxygen (FiO2)	improved with supplemental oxygen via Venturi mask 25-30% FiO2	hypoxemia not improved with supplemental oxygen via Venturi mask or requiring FiO2 > 40%

Oxygen

- 88-92% (60-70mmHg) – higher SpO2 remove hypoxic drive
- Controlled through venturi

BDs

- SABA +/- SAMA
- Each session not > 6min (tachyphylaxis of beta receptors)
- 2-4 hourly
- Air driven (not oxygen dirven – inadvertently remove hypoxic drive)

Steroids

- Oral pred 40mg/d 5-7d
- Higher doses – no added benefit

Antibiotics

- Increasing sputum purulence
- Coamoxiclav 5-7 days
- Pseudo cover if – PHx of pseudo infection, recent hospitalization, concomitant bronchiectasis

NIV

1. T2RF / pH < 7.35,
2. Severe hypoxia - P:F < 300
3. Severe dyspnea with exhaustion RR > 25

- Decide on ceiling
- CIn – resp arrest, low GCS, unRx pneumothorax, hypotension, uncear air way, fascial deformity
- Preferred setting
  - ST mode
  - Back up rate 2-6 below pts rate
  - ePAP 4, iPAP 10-12. Increase iPAP 2cm every 5min until target TV is achieved (6-10 ml/kg)
- reassess in 1h
- persistent hypercapnia : increase iPAP / RR / insp time / decrease insp rise time
- persistent hypoxia : increase ePAP, increase FiO2. When ePAP is increased, increase iPAP too to maintain PS

- timed weaning (D1 24h, D2 16h, D3 12h, D4 2h + 2h + 6h)

Other

- Hydration
- DVT Px
- Rx co mobidities

Discharge plan

Prevent exacerbations
Consider LTOT, vaccines, rehab
Inhaler technique

2ry pneumothorax

- > 2cm or SOB – ICT
- 1-2cm – aspirate
- < 1cm – 24h O2

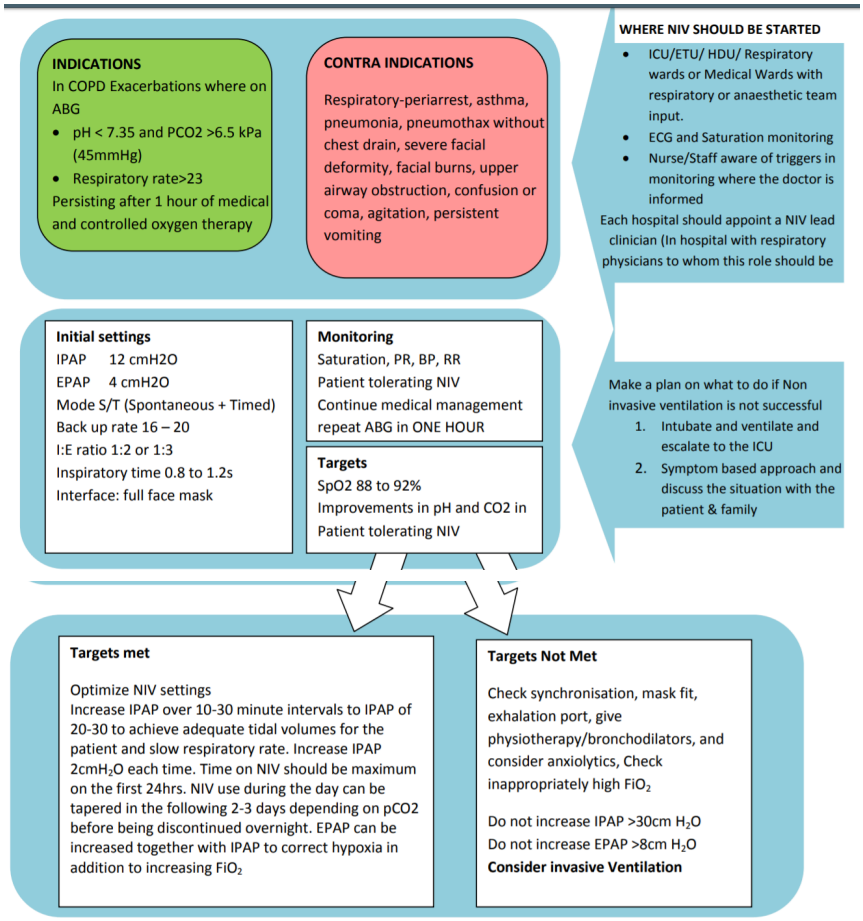
BL or tension pneumothorax – need ICT

MV
Arrest, ventricular arrhythmia, unconscious, HD unstable, NIV failure, respiratory pauses, gasping, agitation, requiring sedation, severe aspiration, secretions

NIV
Mask types – mild to severe disease
Nasal
Orofacial
Fullface
helmet
Other

Successful NIV
Decrease CO2 by 8 pH increase by 0.6

SL guidelines on NIV



Pneumothorax

	PSP	SSP
1. Discharge	< 2cm ,Asy	-
2. 24h obs + O2	-	< 1cm, Asy
3. Needle asp	> 2cm / SOB	1-2cm, Asy
4. ICD	Failing 3 Tension	> 2cm OR SOB Failing 3 Tension

ICT

Safe triangle - 5^{th} rib upper border, MAL, AAL

Indications

1. Tension pneumothorax
2. BL pneumothoraces
3. Failure of needle aspiration
4. Traumatic pneumothorax

Post ICT SOB

Re expansion pulmonary edema

Tube block
SC emphysema at neck
MI
Pain

Removing
Repeat CXR
Observe

Prevent recurrent
Pleurodesis / pleurectomy / other procedures after 1st SSP (in PSP – after 2nd Pnthx)

Hemoptysis

Massive > 400 mL in 3h or > 600 mL in 24h

Causes of massive hemoptysis
Cancer
TB
Bronchiectasis
Mycetoma

Other causes of hemoptysis
PE
DAH
Pneumonia
Left heart disease

Assessment
Bleeding severity
Oxygenation
Hemodynamics – rarely compromised

Rx

- Oxygenation
    - Oxygen
    - Intubation – single lung intubation in to heathy lung or intubate withdouble lumen ETT
        - Prefer a broad tube that allows bronchoscopy
- Correct coagulopathy, consider Abtcs if infection suspected. Role of tranexaemic – controversial. Few small-scale trials showed reduced hemoptysis volume and duration. Risks are aggravation of PE and facilitation of clotting within airway
- Early bronchoscopy

- Will show the bleeder or at least localize the site
- Adrenaline injection, balloon occlusion of the airway

- Bronchial angiogram /- BAE

- Surgery

Ix for cause
CXR, CT chest
ANA, ANCA, anti GBM, UFR, RFT, LFT, coagulation
Sputum – TB, bacterial culture, aspergillus studies
TTE

Pneumonia

Fever cough SOB - DD
Respiratory infection
Bronchitis
Pneumonia – CAP – T/A, HAP
TB, atypical (PCP, aspergillous)

DPLD – COP, COPD, IPF exacerbation / AIP, subacute HP, E-LD
PE
Cardiac disease
CKD

Lobar patch – RV IE with emboli, PE, pneumonia

HAP – pneumonia developing after 48h of hospital stay, netither present nor incubating on admission
VAP -

HCAP – within 3m, >2d hospital stays / / nursing home / home IV drugs / immunosuppressed / chronic HD within 30d

Mgt
ABC
PCM, hydration
Abtc
Chest PT / sputum clearance – no clear
DM control etc
Vaccination – influenza

HAP
Early – early detected, low risk – ceftriax + clarithromycin/levoflox

Late / early with high risk
Pseudo cover – piptaz / ceftazidime
G - - amikacin / genta / cipro
+/- MRSA cover – vanco

Vanco, AG – post HD

CAP
CURB-65, BU > 7 mmol/L, RR > 30, BP 90/60, age > 65 □ > 4-5: ICU
0-1 : influenza testing if suspected amox +/- clarithro clari, doxy
2 : admit
IV Coam (SL - IV. Others oral) + clari doxy
3-5 IV ceftriax / IV coam + clari

Steroids
Pneumonia

CRB-65 if zero go home

CBS, FBC, CRP, Na, Cr, LFT, ECG, CXR, blood culture, sputum culture
Coamoxiclav / ceftriax, clarithromycin

Poorly responding pneumonia
Drug failure
Disease complication – abscess, collapse, empyema, HF, ARDS
Cancer / underlying lung disease
Alternative Dx - COP

Tamiflu??

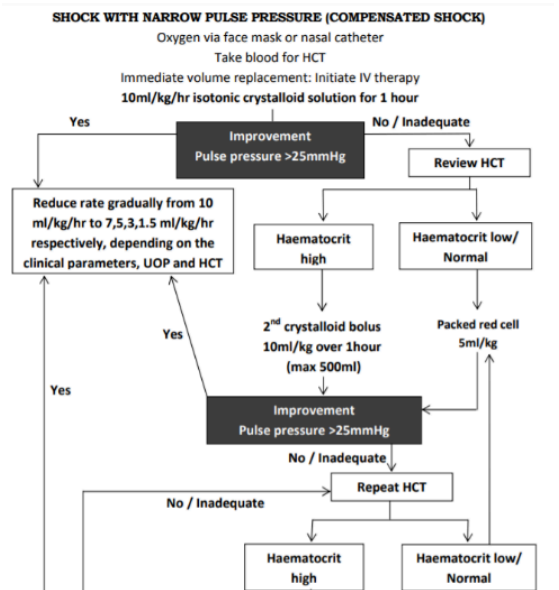
Sepsis

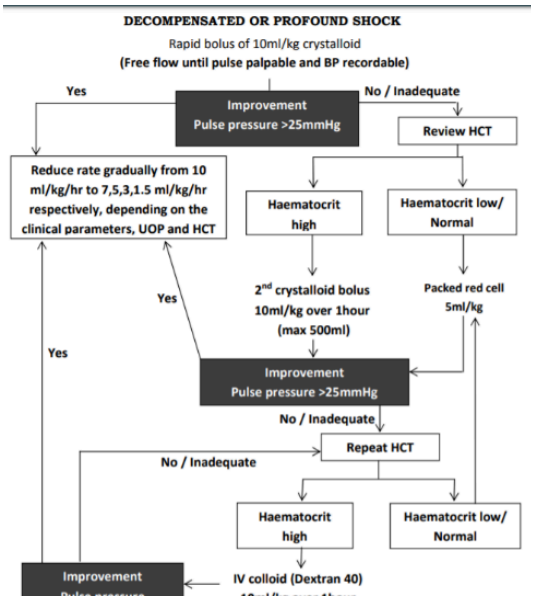
Acute fever

Infection
Poison
Thyrotoxic crisis

Dengue

	Febrile (plt < 130)	Critical	Preshock	Compensated shock	Decomp & Profound shock	Convalescent
Fx		Pl effusion Ascites HCT rise by 20% Heralded by defervesc. , Rapid plt fall	Sweating Abd pain Persistent vomiting Restlessness Postural dizziness UOP <0.5 Cold periph CRFT>2s High DBP postural SBP drop > 20 High HR/RR	PP ≤ 20	Decomp: SBP < 90 or 20% reduction or MAP < 60 Profound: Unrecordable BP	Fx of overload periorbital oedema, cough, wheeze tachypnoea, high SBP, DBP and wide PP. crepts and rhonchi.
Monitoring						
Vitals	3h	1h		15min		
IPOP	6h	3h. in high risk, consider catheter		Hourly. Consider catheter		
HCT	6h	3h		Before and 15min after boluses. 2-3h thereafter		
FBC	bd	Bd		Bd or more		
Mgt	2.5 L/d or less PO +/- IV. Extra for D/V	Fluid quota	Increase infusion rate	Oxygen Bolus (1h) Crystalloid x2 Colloid blood	Oxygen Bolus (15min)	





Blood transfusions in DF

Blood – when bleeding is evident / suspected

1. Overt bleeding > 6-8 mL/kg
2. Refractory shock despite 40-60 mL/kg of fluid
3. Acidosis / organ dysfunction
4. Relatively low HCT, dropping HCT

5mL/kg bolus. Should raise PCV by 5 points

On going bleeding assessment

? DIC
? liver failure
Consider – plt, FFP, TA, PPI

Prevention or treatment of bleeding

Plt, plasma, fVIIa, IVIG, antiD Ab, IL-11 tried in RCTs
None showed benefit (Nipuns review) – all studies have excluded severe bleeding.
??? f VIIa may show benefit

Fx of fluid overload

Cough DD – pneumonia, myocarditis
Tachycardia
Tachypnea
Orthopnea
Increased BP
Mgt – frusemide small bolus out of CP. Infusion with colloid infusion in latter half ???

Place of frusemide

Not in leakage phase
May be at the end of CP, with colloid infusion in an overloaded pt vulnerable to hypotension
Or with blood in bleeding and overload

shock with overlaod □
high HCT □ colloid bolus + frusemide 10-20 mid way, repeat SOS

low HCT blood + ???? frusemide
overload + with no shock
NL / low HCT restrict fluid, monitor – should improve within hours
consider concealed bleeding
pul edema fursemide 10-20

high HCT monitor

Addisonian crisis

Adrenal crisis

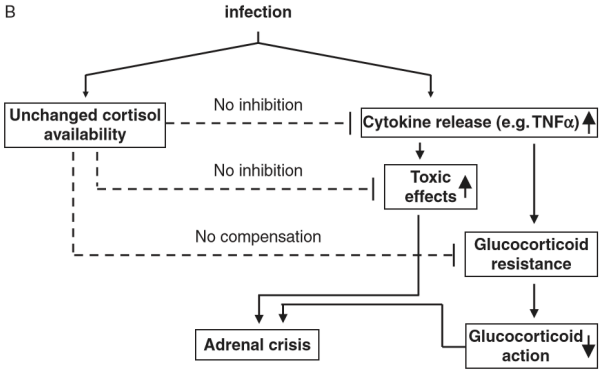


Table 1 Definition and grading of adrenal crisis (modified from (30)).

Definition:
(A): Major impairment of general health with at least two of the following signs/symptoms
Hypotension (systolic blood pressure <100 mmHg)
Nausea or vomiting
Severe fatigue
Fever
Somnolence
Hyponatraemia (≤132 mmol/l) or hyperkalaemia
Hypoglycaemia
(B): Parenteral glucocorticoid (hydrocortisone) administration followed by clinical improvement
Grading:
Grade 1: outpatient care only
Grade 2: hospital care (general ward)
Grade 3: admission to intensive care unit
Grade 4: death from adrenal crisis (with or without parenteral glucocorticoid administration)

Treatment of crisis
Draw bood for cortisol
IV HC 100mg stat 100-200mg/d gradual taper
IV fluids / dextrose if hypoglycemic (50%D 20-50 mL)
Start FC when HC dose in < 50mg/d (higher HC doses have FC effect)
Find the cause – TB (other infection - fungal), AI adrenalitis, mets, hemorrhage, steroid withdrawal

Thyrotoxic crisis

Myxedema coma

DKA HHS

	DKA	HHS
Incidence	Commoner	Rarer

R/F	T1DM	T2DM
Path	Severe insulin defi and very high counter reg Hmns High hepatic gluconeogenesis And KB synthesis Osmotic diuresis (Gc, KB)	Less severe insulin defi and less elevated counter reg Hmnss High hepatic gluconeo and peripheral uptake But enough insulin to inhibit HSL (& therofore ketogenesis)
Mortality	Less. 2-5%	Higher. 15%
Ppt	Infection – UTI, RTI MI, CVA, PE, pancreatitis Trauma Steroids, HCT, pentamidine, SNS agonists, alcohol Non compliance / eating disroders	Same as DKA Same drugs (also included dilantin)
Presentation	Acute Polyuria, polydipsia, weight loss Vomiting, abdominal pain (related to acidosis and alcohol / cocaine, but not to Glc level) Kussmaul breathing, acetone smell	Polyuria, polydyspsia Altered conscious levels, FNS (without a stroke), fits No abdominal pain / Kussmaul breathing
Typical deficits on presentation	Water (ml/kg) 50-70? Sodium (mmol/kg) 7-10 Chloride (mmol/kg) 3-5 Potassium (mmol/kg)) 3-5	Water 100-220
Dx	Hyperglycemia> 11 (198) Hyperketoneamia (BHB > 3mmol/L) Metabolic acidosis (VENOUS pH < 7.3 or HCO3 < 18) Severe = pH < 7.1	Hyperglycemia > 30 mmol/L Hyperosmolarity > 320 mOsm/L Dehydration No ketonemia (< 3 mmol/L) No acidosis (HCO3 > 15, pH > 7.3)
Complications	K, P Cerebral edema in children Hypoglycemia DVT	Cerebral edema ODS K, P DVT, pressure sores

Management

	DKA	HHS
Fluid	- deficit 50-70 mL/kg - Fill up to euvolemia - Then maintain 150-250mL/h so that 50% of the body water deficit is replenished within 12-24h - in addition, replace ongoing urinary losses - Start with NS: 1L over 1h,2h,2h,4h,4h,6h - ~~if hyperNa, use N/2~~ - when RBS < 250mg/dL (300 in HHS) ~~use, N/2 + 5%D ,~~ add 10%D 125mL/h and continue NS to replenish volume	Deficit : 100-220 mL/kg NS Intial Na rise is expected (Glc dilution □ fall in osm □ fluid shift to cells) N/2 only if osm fails to falld despite good positive balance. 1L 1st hr □ 0.5-1 L 2nd hr □ adjust Target osm reduction 3-8 msom/kg/h Target Na reduction < 10 / day
Insulin	0.15 U/kg IV bolus (not recommended in JBDS 2010) 0.1 U/kg/hr	Start insulin when Glc cease to fall with fluids (start early if coexisting KB+) Target Glc reduction 90mg/dl/h

	Continue basal long acting insulin dose for those who were on it (facilitates maintenance and return to premorbid doses) Target RBS reduction 3mmol/L/h; KB reduction 0.5mmol/L/h; HCO3 3mmol/L/h; osm reduction < 3 mosm/h When RBS < 300 add 10%D 125mL/h Maintain RBS 150-200	0.05U/kg/h (higher rates faster fluid shift out of vessles hypovol, cerebral edema) When RBS < 300 add 10%D 125mL/h Maintain RBS 150-200 by adjusting insulin rate until osm is corrected and mental status returned
K	Total body k always low. Serum can be high due to redistribution. When levels < 5 – replace 2/3 as KCl, 1/3 as K3PO4 Target serum K 4-5 If starting K is < 3.3 – give IV 40 mEq/h and delay insulin until K > 3.3	
NaHCO3	Worsen hypoK Intracellular acidosis (CO2 accumulation) CNS acidosis Only if pH < 7.0	Not indicated
Definition of resolution	Venous pH > 7.3, HCO3 > 18 Plasma KB < 0.3 AG < 12 RBS < 200	Improved mental status Osm < 320 RBS < 300
SC regular insulin	After resolution 0.6U/kg/d or previous dose 2/3in the morning, 1/3 in the evening Basal bolus (night dose = basal + bolus = 1/3 of total daily dose)	

Comparison of guidelines

	DKA		HHS	
	ADA 2009	**JBDS 2013**	**ADA 2009**	**JBDS 2015**
Definition	Glc > 250 pH < 7.3 HCO3 < 15 (?18)	Glc > 200 pH < 7.3 HCO3 < 15	Glc > 600 Osm > 320 pH > 7.3, HCO3 > 15	Glc > 600 Osm > 320 hypovol pH > 7.3, HCO3 > 15
IVF type	NS N/2 if Na is NL or high	NS	NS N/2 if Na is NL or high	NS N/2 only if osm fails to fall despite good +ve balance
	N/2+5%D when CBS < 250	Cont NS. Ad 10%D 125mL/h when CBS < 250	N/2+5%D when CBS < 300	
Insulin	Loading bolus 0.15 U/kg	No loading 0.1U/kg/h	Loading 0.1 U/kg	No loading 0.05 U/kg/h

Estimating total daily dose of insulin (JBDS)
0.5 – 0.75 x BW (kg). Use 0.75 if insulin resistance is suspected (teens, obese)
Basal bolus regimen: 50% of TDD as basal insulin in the evening. Rest divided equally across meals

Change in LoC is not a feature of DKA. Search for an alternative cause

- meningoencephalitis
- stroke / CVST

- cerebral edema
- hypophosphatemia (rhabdo, HF, enceph, H'lysis), ODS, WE

Alcohol intoxication

Pituitary apoplexy

Hyponatraemia

Outline
Assessment – symptoms severity, volume status, acute Vs chronic, cause
Treatment

Targets

Urgently increase by	5
1st 24h do not exceed	10
Subsequent each 24h –	8
Maximum correction up to	130

3%S 2mL/kg in 20 min, check Na, repeat twice, not meeting goals infusion 1 mL/kg/hr till Sy improvement / total rise 10 mmoL / Na 130 – whichever comes earlier

Evaluation

Symptoms

1. Infants (high brain : intracranial space ratio)
2. Premenopausal women (inhibitory effect of Egn and high ADH level)
3. Chronic hypoxic patients (strongest risk factor) are at increased risk for hyponatremia encephalopathy

Severe	Vomiting Fits coma Cardiorespiratory distress Deep somnolescecne
Moderately severe	Nausea without vomiting Headache Confusion
Chronic subtle	Concentration deficits Gait disturbance Falls, Osteoporosis, fractures

Volume status

Cause

Pseudohyponatremia – high lipids / paraproteins / LpX (PBC)
Hypertonic – mannitol, glycine, hyperglycemia
Isotonic – pseudohypoNa, sometimes mannitol / glycine / glucose
Hypotonic
Hypovolaemic – CSW, Addison, dehydration (diarrhea, vomiting), diuretics, 3rd spacing
Euvolaemic – SIADH, hypothyroidism, hypocortisolism, 1ry polydipsia
Hypervolaemic – CCF, CKD, CLCD, Nephrotic

Investigations

Serum and urine Na and osm
SE, Cr, BU, FBC, LFT, RBS, cortisol, TSH, 2DE
SIADH : CXR, CRP/ESR, infection screen, NCCT,

Mgt

Severe symptoms

- **IV 3% saline 150 mL (2 mL/kg) over 20 min. check Na. repeat up to a total of 3 boluses until Na is increased by 5 mmol/L (rapid correction to prevent cerebral edema)**
- **If symptoms improve** with this much of Na rise, subsequent treatment should be guided by the cause. Check Na at 6h, 12h, 24h and daily thereafter
  - Total increase in Na should not exceed
    10 mmol/L in the first 24h
    8 mmol/L in the subsequent 24h intervals
  - Final Na level should not exceed 130 mmol/L
- **If symptoms do not improve with 5 mmol/L rise of Na,**
  - Continue 3% saline to increase Na by 1 mmol/L every hour (1mL/kg fo 3%S will increase Na by 1 mmol/L if body water is 50% of body weight - KC)
  - Continue this only until
    - Symptoms improve
    - Total Na rise is 10 mmol/L
    - Na reaches 130 mmol/L
      (whichever comes first)
- **Correction of hypoK / hypoMg will also increase serum Na**

With moderate symptoms

Investigate for the cause
Give 1 bolus of 3% saline 150 mL (2mL/kg) over 20 min
Monitor Na at 1h, 6h, 12h and daily
Treat according to the cause, aiming to increase Na by 5 mmol/L within 1st 24h, but not by more than 10 mmol/L.
In subsequent 24h intervals, increase Na by 5-8 mmol/L is desirable

Benefit of bolus over continuous infusion to calculated Na correction rate

1. Avoids calculation errors
2. Simplifies regimen
3. Avoid overcorrection
4. Allows rapid partial correction that is needed to prevent or reverse cerebral edema

ODS

RF

Alcohol
Liver disease
Thiazides
Antidepressants
Duration and severity (Na < 120, greatest risk < 105) of hypoNa
Hypokalemia

CFx

hypoNa enceph improve deteriorate (coma, spastic quadriparesis, pseudobulbar palsy, EPS)

Px

Cautious monitoring and not exceeding the limits
If at high risk, consider desmopressin to stop water diuresis
Proactive strategy – giving dDAVP in pts likely to develop overcorrection
Reactive strategy – giving dDAVP when Na correction trajectory is ominous!
Rescue strategy – giving dDAVP when overcorrection has occurred, along with 5% dextrose

Rx

Discontinue active treatment
Consider;
    5% dextrose 10mL/kg over 1h under strict monitoring of Na and UOP
    IV desmopressin2micg 8hrly or less frequently
ODS is reversible !

Pitfalls in Rx

1. When hypoNa and hypoK and hypovolemia co exist,
    a. Correct hypoK first – it is more life threatening. Its correction itself increases Na a little
    b. Correct hypovolemia with isotonic saline (0.9%) – volume correction also reduces ADH and increases Na level. Giving hypertonic fluid will cause a rapid Na rise ppt ODS
2. Singaporean lecturer said : volume restriction will increase Na by only about 1-2 mmol/L. it seems he likes to use vaptans more liberally

How much of Na correction is offered by K correction?

Hypercalcemia

Ca, P, ALP, Mg
Cr, Na

acute: - saline pamidronate calcitonin HD. Glcc for MM, lymphoma, granuloma, vit D toxicity
Mild < 12 hydration alone
Severe > 15 hydration, Pami, Calcitonin +/- HD, Glcc
IM 12-15 apporpriate combination

Hydration:
3-6 L/day or 200-300 mL/h
Target UOP 100-150 mL/h

Method	OoA	Duration	Problems
Hydration	Hours	Until infused	Overload, Ca calculi
Diuresis	Hours	Until given	IV fluid depletion, hypoK, hypoMg
Calcitonin IV/IM/SC	Hours	1-2d (tachyphylaxis)	Short lasting effect
HD	Hours	1-2d	Low P (needs replacement, if not increase Ca)
Bisphosphonate	1-2d	3 weeks	Low P, Mg, Ca, Jaw necrosis
Steroids	1-2d	Days – weeks	Glc, BP, limited settings

Chronic
Hydration
Suppress PTH
- Calcimimetic – cinacalcet (PO), etelcalcitide (IV)
- Paricalcitol (DHEC – more PTH gland selective)
- Surgery – young, severe hyperCa, stones, bone disease. Pre op Vit D to prevent HBS

Bisphos for OP

Confused elderly

Infection – UTI, pneumonia, cellulitis, AGE, meningoencephalitis
Metabolic – CBS, Na, Ca, Mg, Cr, LFT
CNS – SDH, mets, ODS
MI
Endocrine – thyroid
Poisoning

Variceal bleeding

Resuscitation
- 2 widebore cannulae, blood for Hb, INR, Cr, Na, G & DT
- IV NS 1L in 20-30 min
- Blood (7-9), FFP (INR 1.5?), plt (50)
- Urine catheter – IP/OP

Terlipressin / octreotide
- Terlipressin 2mg stat and 1-2mg 4-6 hourly OR
- Octreotide 50mcg stat and 25-50 mcg/hour

IV omeprazole 80mg stat and 8 mg/hour – sp for PUD, may reduce post EBL bleeding and clot stabilization

Ceftriaxone
Lactulose, rifaximin
UGIE
- Banding
- Sclerotherapy
- Adrenaline for ulcer
- Sengstaken Blakemore
- BRTO – balloon occluded retrograde transvenous obliteration
- TIPS

Post banding
NBM
Abtc
Terlipressin 72h
Hep encephalopathy
Repeat endo in 2 weeks
BB on discharge – CIn.

Scores

Rockall – for mortality and rebleeding risk prediction.
With or without UGIE. Variceal / PUD
Clinical phase – age, shock, comorbidity
UGIE compoenets – MWT / CA / no CA paths and Fx of recent bleeding

Glasgow Blatchford – to determine admission for treatment Vs outpt Mgt
Based on Hb, BU, SBP, other (HR, melena, syncope, heart failure, liver disease).
Does not need UGIE. Had both varicea; pr ppk;
Low risk patients (BU<6.5, Hb>13, SBP>110, HR<100, no above adverse Fx – ie score 0) can be managed as **out patient.**

AIMS-65
(albumin < 3, INR > 1.5, Mental status, SBP < 90 , age > 65)
Less accurate than RS and GBS

PPI – bolus = IVI

Acute liver failure

Hep encephalopathy classification

West heaven	ISHEN	CFx	Rx
Minimal	covert	psychometric	no
I	covert	sleep reversal	no
II	overt	flaps, disorient in time	yes
III	overt	confusion	yes

IV overt coma yes

DD
SDH / EDH / ICH
Wernicke's
Alcohol intoxication / withdrawal
ODS
Hypoglycemia , hypoNa
Post ictal / NCSE
Wilsons neurological disease
Primary psychiatric disorder

Mgt

1. Initial care of drowsy pt
2. Detect and treat reversible causes
3. Identify and correct ppt factors - infection, UGIB, constipation, sedative, hypoK
4. Empiric therapy for HE
    i) **Lactulose**, 2-3 soft /d
    ii) Metro 200mg 6h or rifaximin 550mg bd (equal efficacy for acute HE in an open label RCT with 120 pts – 2018, Arab J of GE). Long term metro not given due to neurotoxicity
    iii) LOLA (IV – AASLD 2014. Cochrane : IV = PO) – improve mortality
    iv) BCAA supplements – faster recovery. No mortality benefit
    v) Albumin – no mortality benefit short term. Improves long term outcomes

Protein < 40g/d ppts malnutrition, increase prot catabolism worsen HE
Vege proteins (high BCAA. Low AAA) are better tolerated than animal proteins (high AAA)
Prot restriction not encouraged, certainly not for > 48h, and if at all, is mainly for animal protein. Give 1.2-1.5 g/kg/d
Probiotics – improves QoL and outcomes of hep enc but not mortality – cochrane

2ry Prevention
Lactulose
Rifaximin after 2nd HE event
LT

New criteria for HRS by International Ascites Club (2015)

- Cirrhosis + ascites
- AKI (Cr 50% rise in 7d or 26 rise in 48h)
- No
    - shock
    - ~~hypovolemia (nodireutics& on albumin x 2d)~~
    - structural kidney disease (P'uria < 500, RC < 50, normalUSS)
    - Nephrotoxic drugs

Rx

Mgt of AKI in cirrhosis

Dx sepsis, drugs, HRS, GN, hypovol
RRT only if LT is planned
Avoid nephrotoxins
Fluid restore BP. Give IV albumin for volume repletion – needed for 2 days before Dx HRS
Treat

Albumin 1g/kg/d + vasopressor (Cochrane 10 RCT review – all are equal – low quality EBM)
(after 2d, albumin 20-40g/d up to 14d)
Vasopressor : terli 0.5-1 mg 4-6h, double every 2 days, max 12mg/d
Or norad or octreo+midodrine

Acute / subacute diarrhea in ulcerative colitis

Relapse
Infection – PMC
Drug induced – Abtc associated
Cancer

Status epilepsy

– ABC, CBS, BDZ, AED, anesthesia. > 5min : impending status epilepsy

1. ABC, IV access – CBS, RFT, LFT, FBC, Ca, Mg, Na
2. CBS, thiamine
3. BDZ – diazepam IV 10mg/2min, lorazepam 4mg IV, Midazolam 10mg IM / IN / buccal
   Veterans Affairs Comparative Trial: lorazepam > diazepam, phenytoin, pheny+Diazepam in terminating overt GCSE and preventing recurrence. Probably due to prolonged duration of action (10-12h) as it is not redistributed to adipose tissue like siazepam
4. Repeat BDZ
5. IV AED – phenytoin 20mg/kg at 50mg/min with ECG monitor
6. Consider repeating IV AED (IV levet, NaV)
7. Anesthesia – TPS / propofol / midazolam IV with ventilation and continuous EEG monitoring. If midazolam infusion doesn't control within 45-60min, switch to TPS / propofol

- IVI until EEG is seizure free for 12h. then tail off
- Identify precipitating event / predisposing factors / toxin, infection etc
- Look for complications – rhabdo, drug toxicities, aspiration, bed sores

	Diazepam	Lorazepam	Midazolam
	IV, PR	IV	IM, IN, buccal
	10 mg (0.1-0.2 mg/kg)	4 mg (0.1 mg/kg)	> 40 kg : 10mg < 40 kg : 5 mg Can repeat boluses every 5 min util seizures control, upto 2mg/kg

			Then give the infusion 0.1 mg/kg/h
OoA	20s	2-3 min	1 min
DoA	20 min	10-12h	?
Lipid solubility	High	Low	?
	Thrombophlebitis, resp depression		

	Phenytoin	**Fosphenytoin**
	Active drug	Prodrug
Purple glove	More – ppt due to poor water solublity	Less (due to water solubility, so no ppt during injection)
Hypotension, arrhythmia	More Due to solvent propylene glycol	Less – no propylene glycol
Dose and rate	20 mg/kg 50 mg/min	20 mg/kg 100-150 mg/min
Route	IV	IV Although can be given IM, not recommended in SE due to erratic absorption

Ppt events
Electrolytes – Na, Glc, Ca, Mg
HTN, uremia
Toxins - TCAD
CNS infection
AIE – NMDARE
CVT, abscess, tumor

NCSE
Less aggressive management may be enough because complications are less

AFP

1. GBS
2. Snake bite
3. Botulism
4. MG
5. Stroke

Unconscious patient

Non surgical acute abdomen

DKA
Krait bite
AIP
Inferior MI
Acute pancreatitis

Acute confusion

Endocrine / metabolic
Glc – hypoGlc, DKA, HONK
Myxedema
Thyrotoxic crisis
Hypercalcemia
Hyponatremia, hypernatremia

stroke

acute focal neurological deficit - DD

1. stroke / TIA
2. hypo/hyperglycemia
3. demyelination
4. tumor bleed
5. seizure / Todds
6. focal encephalitis

Resuscitations
ABC
CBS
Swallowing – sit, speech, slurring sip of water. If failing at any point, pass NG
NCCT
NIHSS, timing – eligibility for intervention

T'lysis
In
< 3h, infarct should sensory strokes be T'lsed?
3-4.5h, infarct, NIHSS < 25, stroke size < 1/3 of MCA territory, age < 80y, no PHx of DM+stroke
Exclude CIn
Target time – within 60min from admission
rtPA 0.9 mg/kg, IV infusion 10% of the dose as a bolus in 1 min, rest over 1h
BP < 185/110 at start, keep < 180/105 during infusion. IV labetalol SOS
No antithrombotics for 24h

Thrombectomy
In SL:
< 6h, NIHSS > 6, ASPECT > 6, clot in ICA / MCA1
MRI perfusion scan??

Medical mgt
Aspirin
DAPT for minor stroke for 3 weeks
High intensity statin - SPARCL

Preserve penumbra
BP control, CBS, Na, infection/temperature, O2

Supportive care – PT, ST, OT, prevent pressure sores and DVT,

Screen for DM, DL, FBC (PRV), AF, cardiac, carotid - correct

Specific situations
Improving weakness
Minor stroke – if functionally disabling – give. (eg: visual filed defect). For non-disabling stroke can consider (up to 4.5h)
Extensive clear hypoattenuation – don't give. Established infarct. Minimal benefit. High bleeding risk
frank hypodensity – established stroke. Don't give. Bleeding risk high
Suspected functional – give. No risk of bleeding
Pregnancy
Coagulopathy – plt < 100, INR > 1.7, aPTT > 40, PT > 15s avoid
DOACs - cant give unless not taken within 48h or after confirming fXa activity is normal and clotting profile is normal. can we reverse and give?? – not commented in AHA 2018. Probably not advisable to reverse

Warfarin – INR < 1.7 give
DAPT – give
Therapeutic LMWH within 24h – don't give
DVT Px – IPC stoking reduce DVT and improve mortality at 6m (CLOTS trial)
PPI Px
Physiotherapy
Malignant MCA
Restarting OACs after ischemic stroke

TIA – 1d
Minor NIHSS < 8 - 3d
Mod NIHSS 8-15 - 6d
Severe NIHSS > 1512d, after re imaging

Early CT Fx of stroke

1. Dense MCA, dot , double dot
2. Sulci effacement
3. Loss of gre white demarcation
4. Loss of insular ribbon

Deterioration of LoC post stroke

1. CBS
2. Na
3. ICH
4. Cerebral edema – mannitol 1g/kg, decompressive craniotomy
5. Obstructive hypodrcephalus
6. Reinfarct
7. HTN encephalopathy
8. Sepsis
9. Uremic

Mgt of rtPA induced angioedema (plasmin increase bradykinin and histamine genesis)

1. Stop rtPA and ACEI
2. Consider ETT
3. IV methyl **prednisolone** 125mg + IV **ranitidine** 50mg + IV **diphenhydramine** 50mg (H1RB)
4. Consider **adrenaline** 0.3 mL of 1:1000 as SC or 0.5 mL nebulized
5. Consider **icatibant** 30mg SC stat and repeatable 6 hourly upto 3 doses (Bradyk RB)

Malignant MCA

Stroke unit

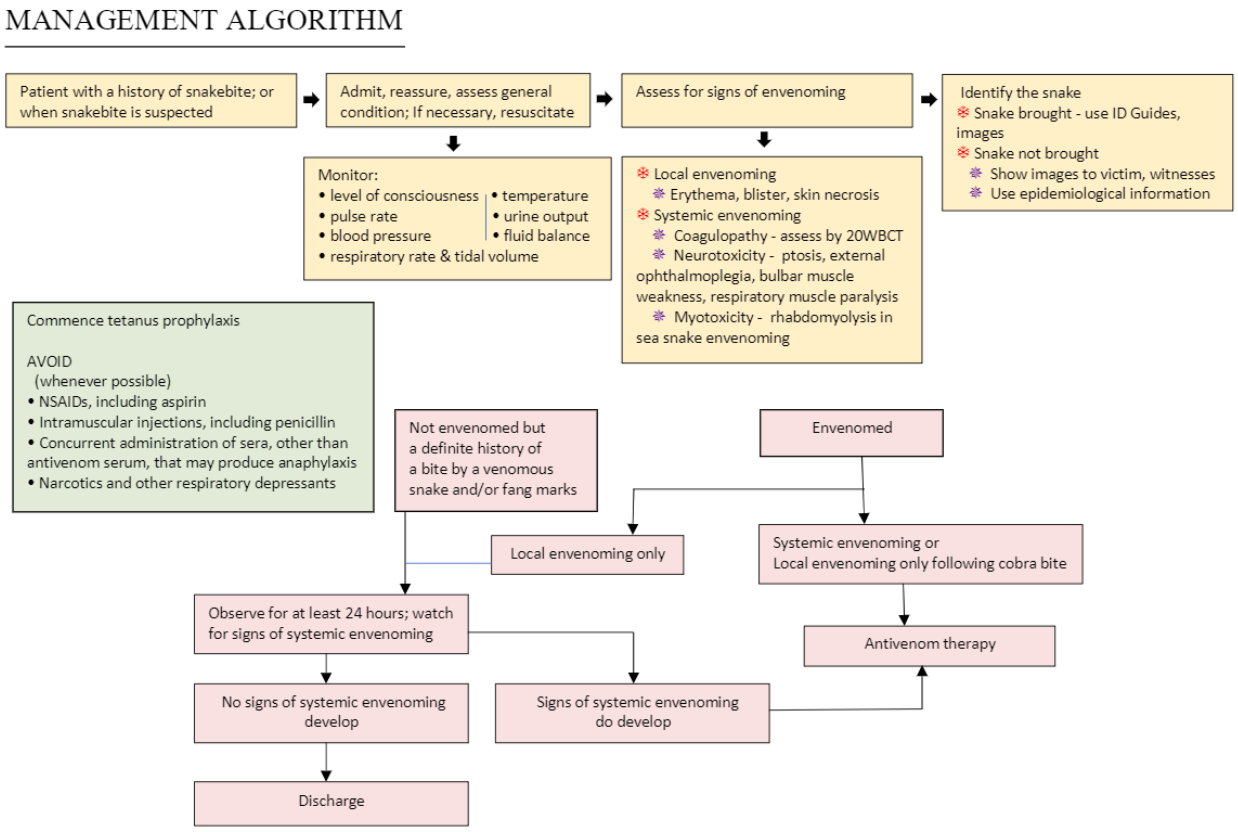
Bacterial meningitis

CSF when to do CSF
Blood culture
DXM
Abtc – Ceftriax +/- vancomycin
Consider – ampicillin, meropenem, vanco

Dropping GCS
Edema, hydrocephalus
Abscess
Subdural empyema

Snake bite

2017 SLMA guidelines



1. ABC
2. Wash the bite site
3. Fx of local and systemic envenomation - WBCT
4. TT (IM. on discharge – avoids risk of bleeding?)
5. Identify the snake
6. Monitor for 24h. 48h with cobra and HNV

7. AVS

WBCT – 2 ML of blood in to clean dry glass tube. Free to stand. Check in 20 min

Indications

Commence antivenom therapy immediately for the bites of the following snakes if the indications listed are present:

Russell's viper:
- If coagulopathy is present: positive 20WBCT; incoagulable blood, spontaneous bleeding.
- If no demonstrable coagulopathy, negative 20WBCT BUT proven Russell's viper bite with fang marks, abdominal pain and some local effects such as swelling, or in the presence of any one or more systemic effects such as visual disturbances, dizziness, faintness, collapse, shock, hypotension, cardiac arrhythmias and myocardial damage (reduced ejection fraction) (WHO Guidelines p. 96)

Cobra:
- Any evidence of systemic envenoming or local envenoming.
- "Dry bites" are common; in the presence of fang marks without symptoms of envenoming observe for 48 hours. If any swelling appears give the first dose (10 vials) of AV.

Kraits:
- If neurotoxic effects (ptosis, ophthalmoplegia) are present, for bites by either species of krait OR
- If severe abdominal pain in the absence of neurotoxicity.

Saw-scaled viper:
- Only if coagulopathy is present.

- LR - swelling

  10 vials, each dissolved to 10 mL of diluting solution water 100 mL – dissolve in to 400 mL of NS over 1h
  Max dose 40 vials
  Prevention of anaphylaxis – SC adrenaline 0.25 mg (0.25 mL of 1 : 1000 adrenaline) – Avoid in past stroke / TIA / ICH. Watchout for hypertensive crisis
  Premed with HC / H1RB / H2RB and infusion rate (over 10min vs 120 min) doesn't change the risk of reactions

SL trial on prevention of AVS reactions - 2011

- 1007 pts
- RCT
- All possible combinations of adrenaline 0.25mg SC, HC 200mg IV, promethazine 25 mg IV
- Benefit only with adrenaline. HC negated this benefit. Higher deaths among those receiving HC.
- ASCVD, uncontrolled HTN were excluded due to risks with adrenaline

AVS reactions

Anaphylaxis – within 180min of starting. Skin, GI, lung, hypotension.
Stop. IM adrenaline (what if in coagulopathy?) rpt consider IV. Along with salbutamol. HC and piriton are no longer recommended (research papers 2011, 2013). WHO 2016 guidelines recommend their use

After reaction settles, restart AVS cautiously if indication is strong. Risk of anaphylaxis reduces with repeat dosing.

Febrile reactions – within 2h. IVF, PCM, cooling

SSS – 1-12d. skin rash, LN, MnM, arthralgia, myalgia, proteinuria
Rx – piriton 2mg 6 hourly 5d – if no response at 2d, give prednisolone 5mg 6 hourly 5d

polongaTab – open labelled RCT reported in 2001, 1g Vs Haffkiene 10g, inferior in efficacy, superior in safety. Need further studies with higher doses

cobra – day time near human dwellings, upper limb. Coagulopathy in short lived, often without major bleeding, resolves spontaneously

SSW – finger and toe tips. LR+, caog+. AKI rare. Neuro – no

Green pit viper – LR ++, coagulopathy (Rx – FFP 10 mL/kg/d x 2d)

RV – dusk and night. Daytime in paddyfields
Intial AV dose : 20 vials, (each in to 10 mL of dilution water 200 ML) dissolve in 300 mL of NS 500 mL solution give over 1h. recent research suggest this is adequate to neutralize the total venom dose and further dosing will not offer any added benefit. However, 2017 SL guidelines still suggest to repeat at 10 vial doses every 6h.
FFP was beneficial in Australian study but not in SL study

HNV – daytime, below ankle / fingers
Observe for 48h
LR+ - puncture the blister and give prophylactic Abtcs – fastens recovery
Coagulopathy – do 6h WBCT for 48h. if abn, give FFP

Krait
AVS 10 vials enough
Will not reverse paralysis
Ventilate when TV < 250 mL
Recovery only when new nerve fibres sprout

Ceylon krait bites are rare. Don't know if Haffkeine (Indian) works. Guideline recommends to give, assuming it would cross neutralize

Paracetamol overdose

RF for severe injury – malnutrition, P450 +, fasting, staggered overdose, chronic alcoholics (P450+)

Ix – LFT, RFT, INR

Rx
AC < 1h
Indications for NAC
- Dose > 150mg/kg or unknown or toxic PCM level (staggered: > 7.5g a day or > 4g/d if high risk)
- Hepatitis
- No benefit after 72h of acute ingestion

Dosing
Duration – until PCM level is undetectable and AST ALT normalize
LT

Methionine
Comparable efficacy, cheaper. Oral

NAC reactions
Non IgE mediated
Urticaria, wheezing – give adrenaline – can restart after recovery
Hypotension – give adrenaline. Cant restart

GL
NG
Left lateral
NS 250 mL in room temperature
Funnel or syringe
In and out
Until effluent is clear

AC
50-100g

Salicylate

TCAD

Rx – ABC, GL, O2, fluids
NaHCO3 (8.4%) 50-150 mL bolus infusion. Target pH 7.50-7.55
- QRS > 100, hypotension, SVT or VT

AVOID amiodarone

Theophylline

Kaneru / digoxin

Rx
MDAC
Correct hypoK and Mg
SVT – phenytoin, VT – lidocaine. 50-100J DC only if essential
AVOID – CCB, amiodarone, Cal gluc (even for hyperK), cardioversion
digiFab

Organophosphate

BB/CCB

Respiratory failure

T1RF **PaO2 < 60mmHg with FiO2 50% or PaO2 < 40 mmHg with any FiO2**
Pulmonary edema
Pneumonia
Asthma
PE
Pneumothorax
ARDS
Lung collapse – FB / mucus plug / tumor
Trauma / contusion

T2RF **PaCO2 > 50 mmHg or > 45 mmHg with pH < 7.30**
COPD
Neuromuscular
GBS
Krait bite
MG
Poisoning – opioid, BDZ, organophosphate
Prolong T1RF

Correct hypoxia
Give oxygen
Nasal prong 2L/min, 30%
Face mask 6-8 L/min, 60%

Non rebreathing mask with reservoir bag 15 L/min 70%
Bag valve mask respirator with reservoir bag 15L/min 100%
NIV (ePAP)
Invasive ventilation (increase FiO2, increase TV ..)

Correct hypercapnia
NIV (iPAP)
Mechanical ventilation (increase RR)
Indications – FVC < 15 mL/kg

Mgt of ARDS

- Sedate, paralyse, ventilate – decrease O2 requirement
- Conservative fluid resusc – CVP ~ 4, Hb 7 (negative balance, diuretics, hyperoncotic albumin solutions)
- Lung protective ventilation – TV 6mL/kg, insp plateau pressure < 30 mcH2O, adjusted PEEP (~ 15 mmHg), prone position (12h/d for severe)
- Steroids for mod-sev ARDS early in the course

High flow oxygen through nasal canula improves 90d mortality in acute hypoxic resp failure - FLORALI

AKI

SEPTIC ARTHRITIS

Analgesics
Temporary immobilization
Synovial fluid – FR, gram stain, culture, crystals in polarized microscopy
    Purulent
    W > 25 000, (often 100 000), N > 90%, high CRP
    Protein high, glucose low
USS – fluid, aspiration, synovitis
Antibiotics
    Flucloxacillin
    MRSA – vanco
    Gonococcal – ceftriaxone
Arthroscopic washout

Summaries