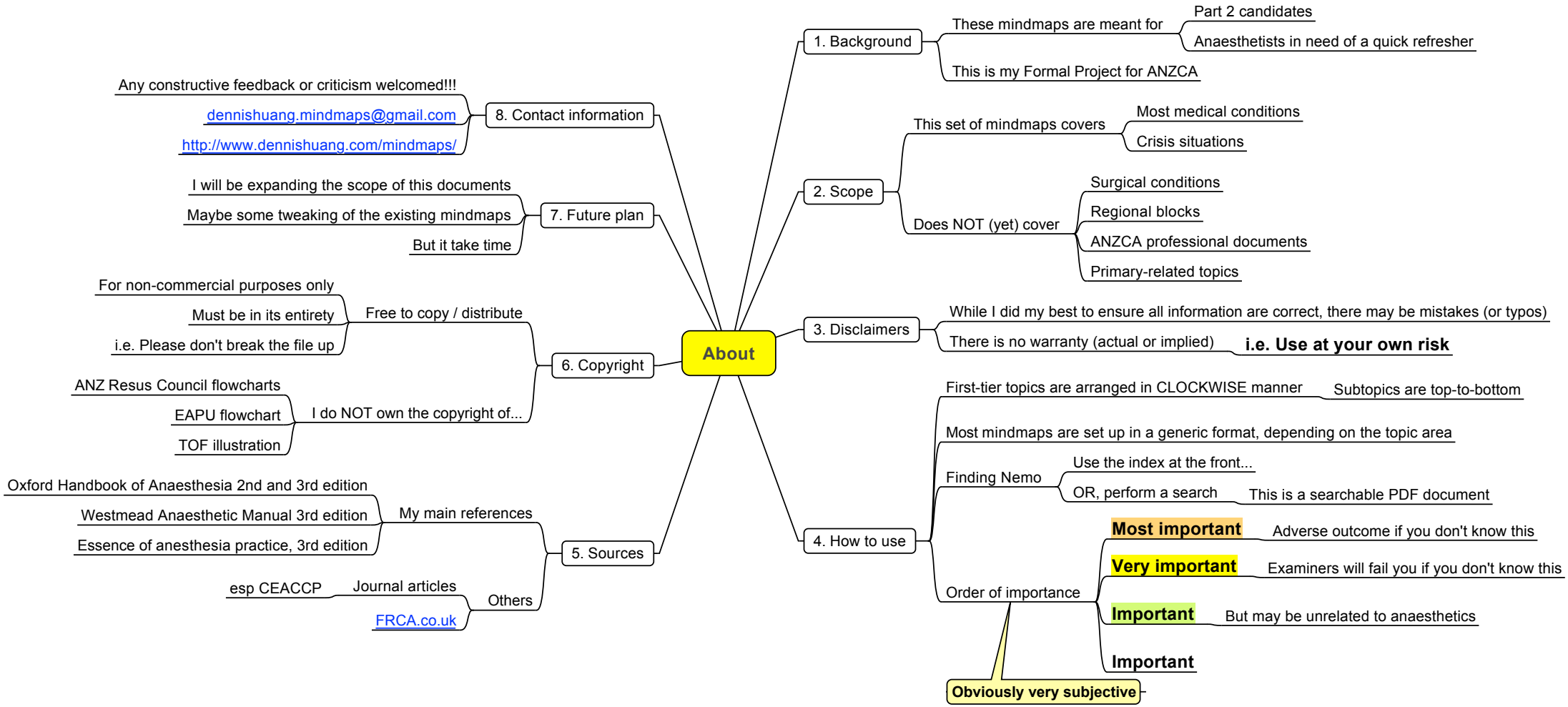
Mindmaps for anaesthetists

By Dennis L Huang

December 2013


About
1. Background
These mindmaps are meant for
Part 2 candidates
Anaesthetists in need of a quick refresher
This is my Formal Project for ANZCA
2. Scope
This set of mindmaps covers
Most medical conditions
Crisis situations
Does NOT (yet) cover
Surgical conditions
Regional blocks
ANZCA professional documents
Primary-related topics
3. Disclaimers
While I did my best to ensure all information are correct, there may be mistakes (or typos)
There is no warranty (actual or implied)
i.e. Use at your own risk
4. How to use
First-tier topics are arranged in CLOCKWISE manner
Subtopics are top-to-bottom
Most mindmaps are set up in a generic format, depending on the topic area
Finding Nemo
Use the index at the front...
OR, perform a search
This is a searchable PDF document
Order of importance
Most important
Adverse outcome if you don't know this
Very important
Examiners will fail you if you don't know this
Important
But may be unrelated to anaesthetics
Important
Obviously very subjective
5. Sources
My main references
Oxford Handbook of Anaesthesia 2nd and 3rd edition
Westmead Anaesthetic Manual 3rd edition
Essence of anesthesia practice, 3rd edition
Others
Journal articles
esp CEACCP
FRCA.co.uk
6. Copyright
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For non-commercial purposes only
Must be in its entirety
i.e. Please don't break the file up
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ANZ Resus Council flowcharts
EAPU flowchart
TOF illustration
7. Future plan
I will be expanding the scope of this documents
Maybe some tweaking of the existing mindmaps
But it take time
8. Contact information
Any constructive feedback or criticism welcomed!!!
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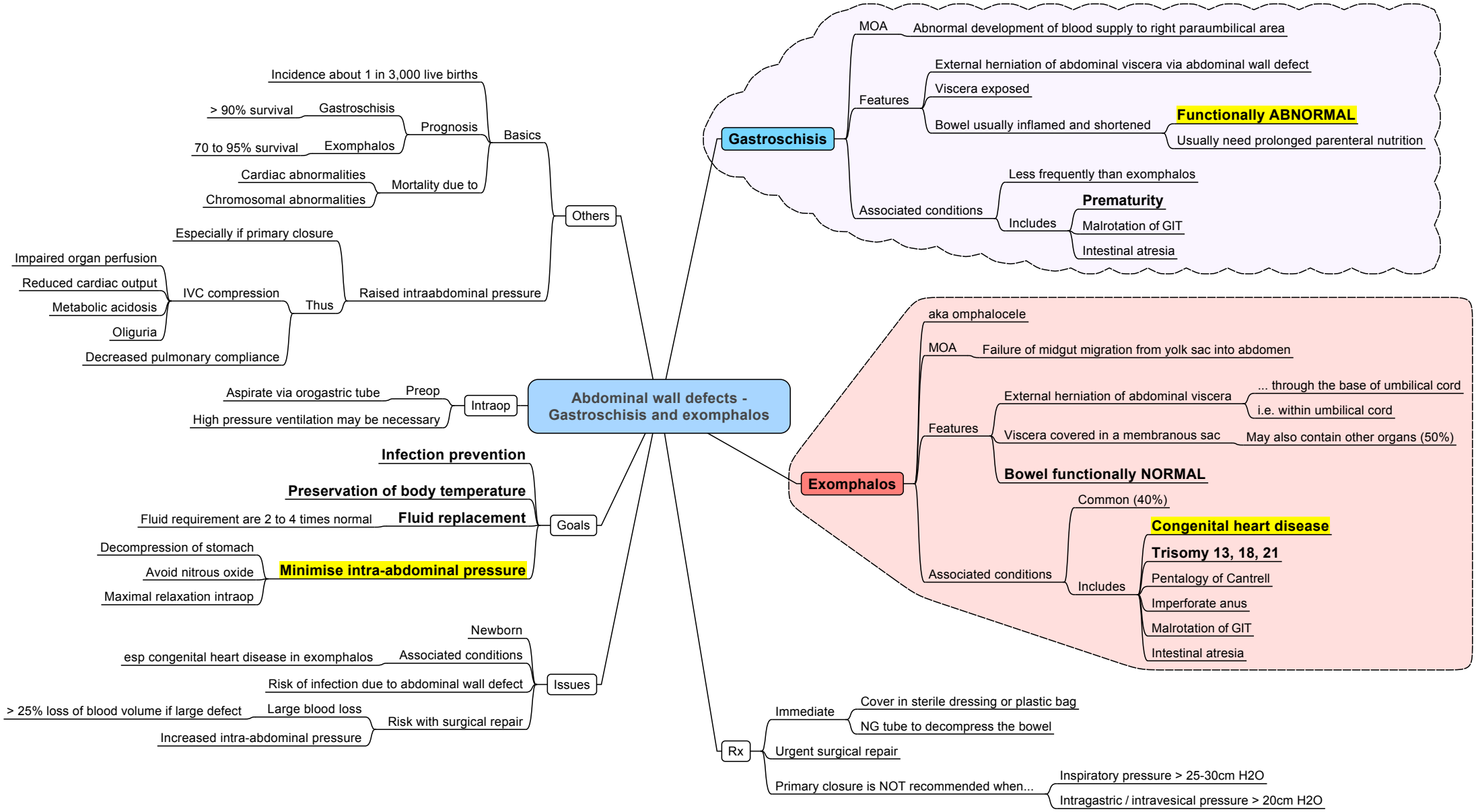
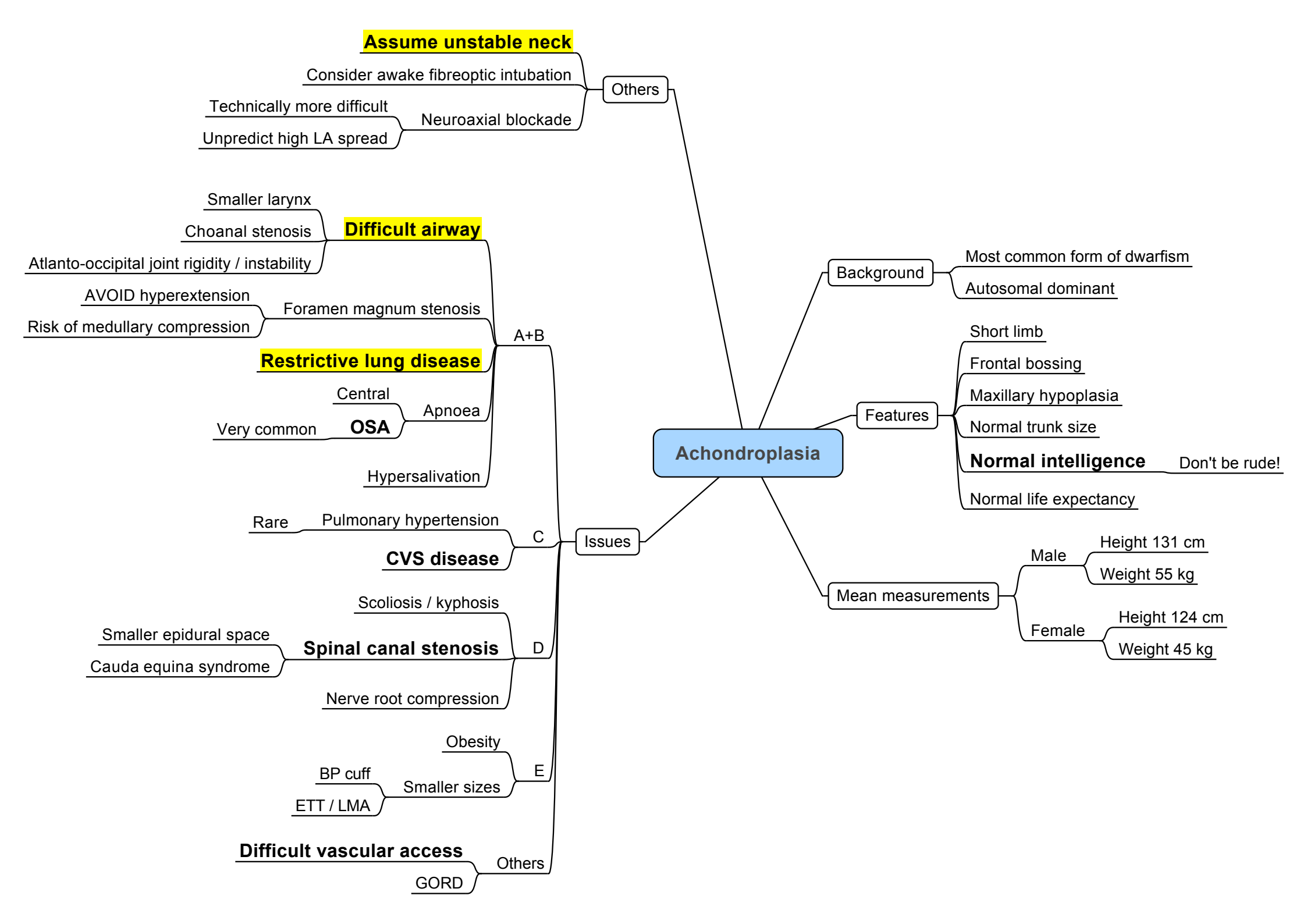
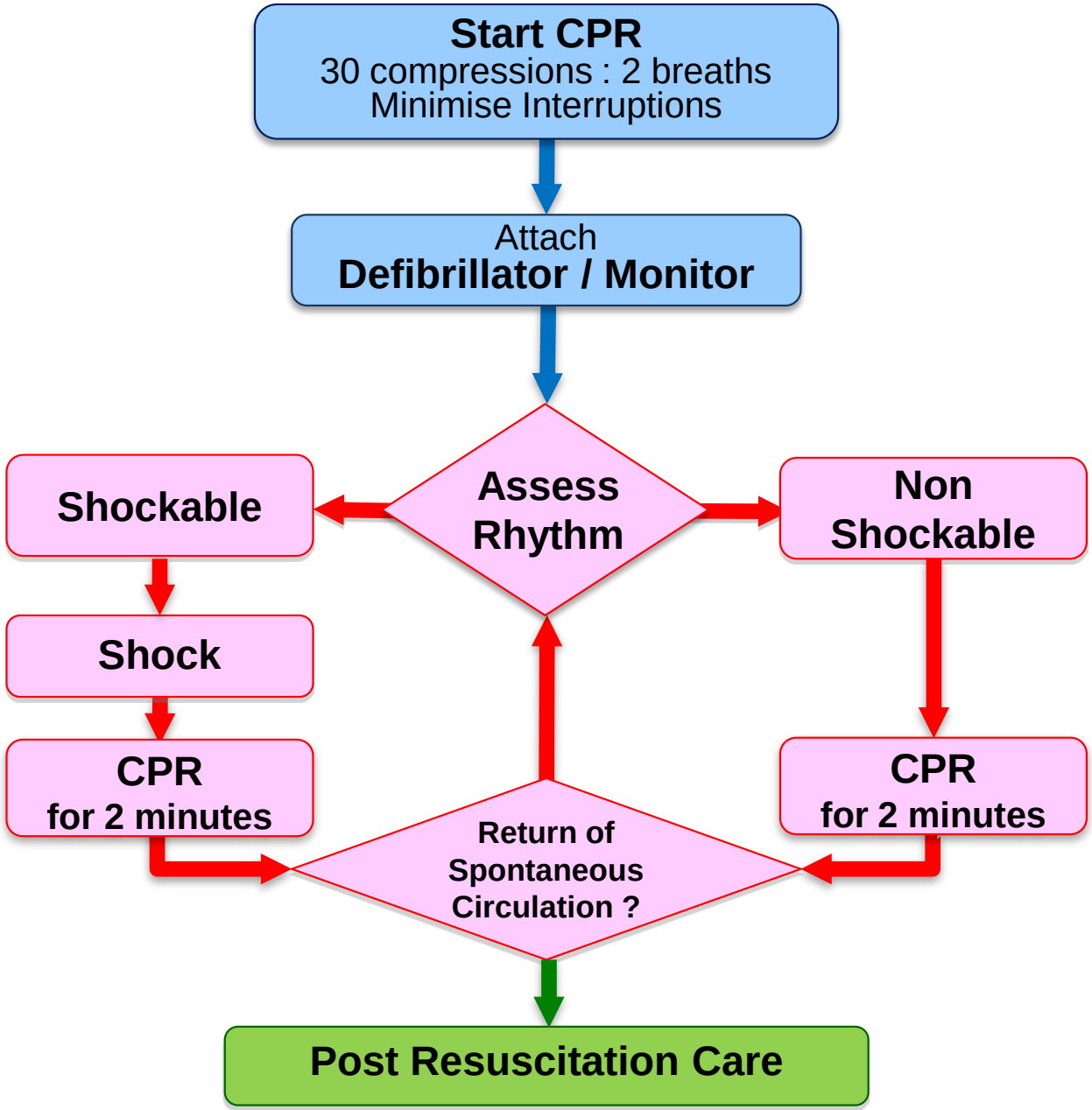
http://www.dennishuang.com/mindmaps/

Contents


Abdominal wall defects - Gastroschisis and exomphalos
Basics
Incidence about 1 in 3,000 live births
Prognosis
Gastroschisis
> 90% survival
Exomphalos
70 to 95% survival
Mortality due to
Cardiac abnormalities
Chromosomal abnormalities
Others
Raised intraabdominal pressure
Especially if primary closure
Thus
IVC compression
Impaired organ perfusion
Reduced cardiac output
Metabolic acidosis
Oliguria
Decreased pulmonary compliance
Intraop
Preop
Aspirate via orogastric tube
High pressure ventilation may be necessary
Goals
Infection prevention
Preservation of body temperature
Fluid replacement
Fluid requirement are 2 to 4 times normal
Minimise intra-abdominal pressure
Decompression of stomach
Avoid nitrous oxide
Maximal relaxation intraop
Issues
Newborn
Associated conditions
esp congenital heart disease in exomphalos
Risk of infection due to abdominal wall defect
Risk with surgical repair
Large blood loss
> 25% loss of blood volume if large defect
Increased intra-abdominal pressure
Gastroschisis
MOA
Abnormal development of blood supply to right paraumbilical area
Features
External herniation of abdominal viscera via abdominal wall defect
Viscera exposed
Bowel usually inflamed and shortened
Functionally ABNORMAL
Usually need prolonged parenteral nutrition
Associated conditions
Less frequently than exomphalos
Includes
Prematurity
Malrotation of GIT
Intestinal atresia
Exomphalos
aka omphalocele
MOA
Failure of midgut migration from yolk sac into abdomen
Features
External herniation of abdominal viscera
... through the base of umbilical cord
i.e. within umbilical cord
Viscera covered in a membranous sac
May also contain other organs (50%)
Bowel functionally NORMAL
Associated conditions
Common (40%)
Congenital heart disease
Includes
Trisomy 13, 18, 21
Pentalogy of Cantrell
Imperforate anus
Malrotation of GIT
Intestinal atresia
Rx
Immediate
Cover in sterile dressing or plastic bag
NG tube to decompress the bowel
Urgent surgical repair
Primary closure is NOT recommended when...
Inspiratory pressure > 25-30cm H2O
Intragastric / intravesical pressure > 20cm H2O


Achondroplasia
Background
Most common form of dwarfism
Autosomal dominant
Features
Short limb
Frontal bossing
Maxillary hypoplasia
Normal trunk size
Normal intelligence
Don't be rude!
Normal life expectancy
Mean measurements
Male
Height 131 cm
Weight 55 kg
Female
Height 124 cm
Weight 45 kg
Issues
Others
Assume unstable neck
Consider awake fibreoptic intubation
Neuroaxial blockade
Technically more difficult
Unpredict high LA spread
A+B
Difficult airway
Smaller larynx
Choanal stenosis
Atlanto-occipital joint rigidity / instability
Foramen magnum stenosis
AVOID hyperextension
Risk of medullary compression
Restrictive lung disease
Apnoea
Central
OSA
Very common
Hypersalivation
C
Pulmonary hypertension
Rare
CVS disease
D
Spinal canal stenosis
Scoliosis / kyphosis
Smaller epidural space
Cauda equina syndrome
Nerve root compression
E
Smaller sizes
Obesity
BP cuff
ETT / LMA
Others
Difficult vascular access
GORD

Advanced Life Support for Adults



During CPR

Airway adjuncts (LMA / ETT)
Oxygen
Waveform capnography
IV / IO access
Plan actions before interrupting compressions
(e.g. charge manual defibrillator)
Drugs
Shockable
* Adrenaline 1 mg after 2nd shock
(then every 2nd cycle)
* Amiodarone 300 mg after 3rd shock
Non Shockable
* Adrenaline 1 mg immediately
(then every 2nd cycle)

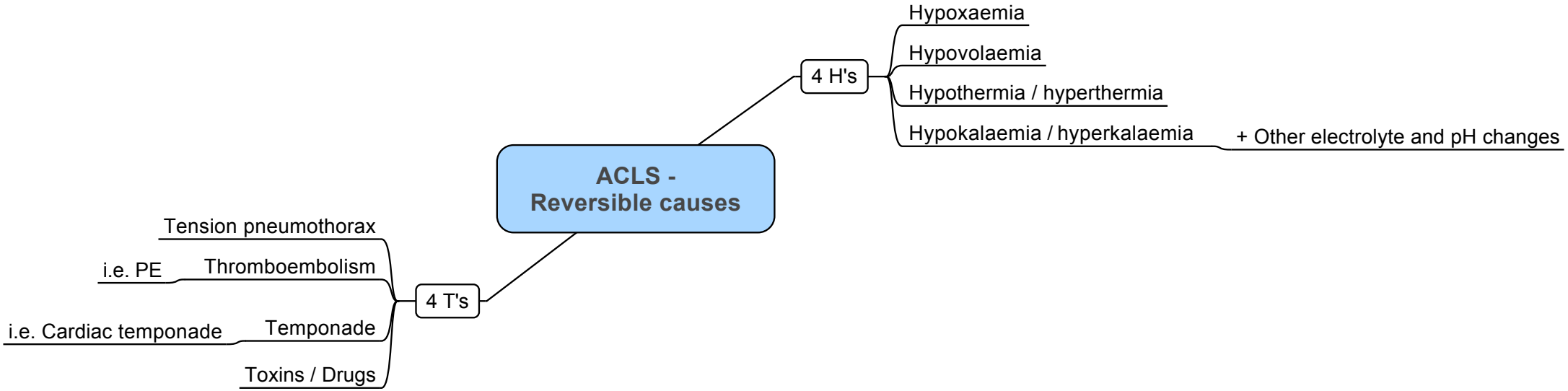
Consider and Correct

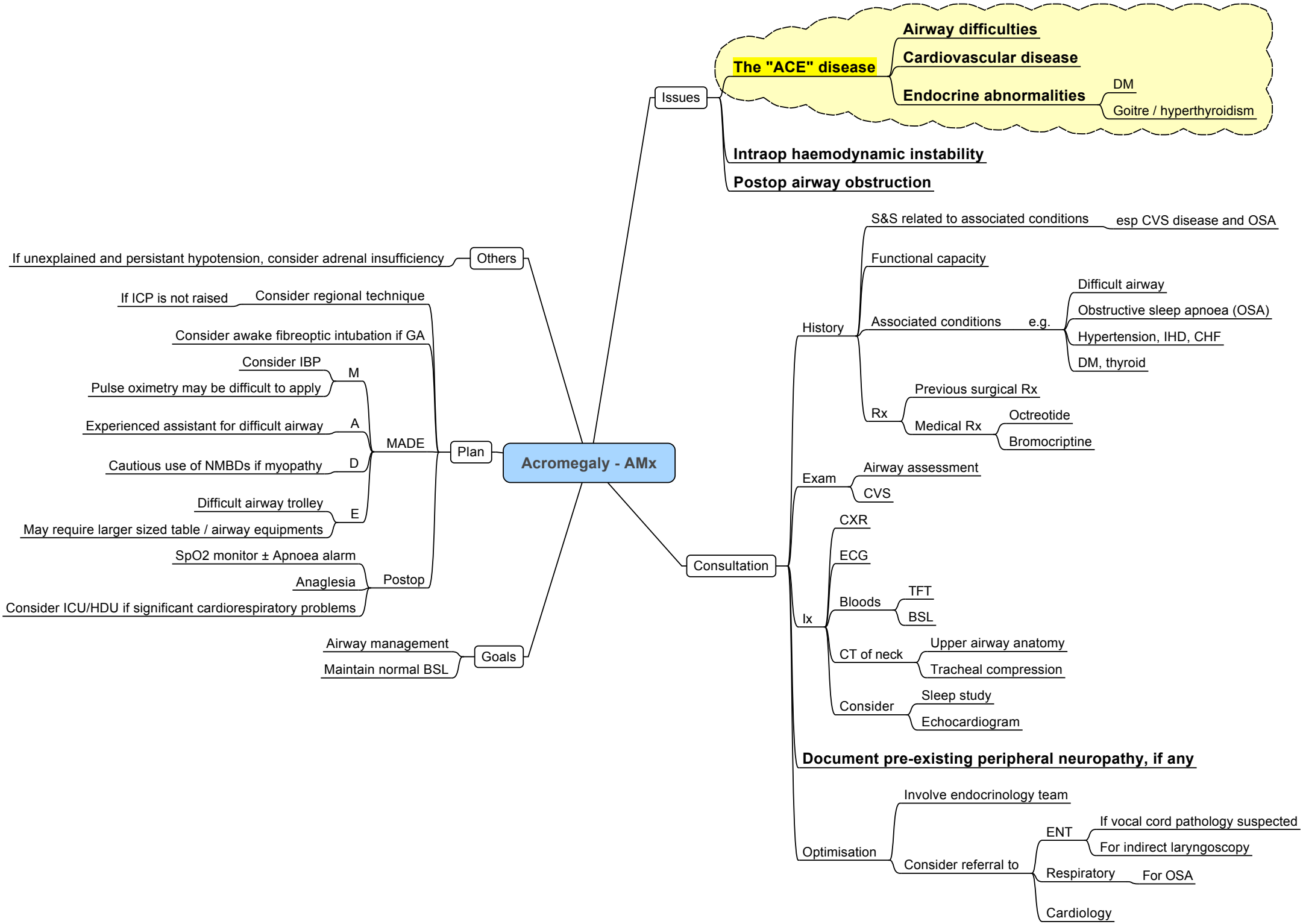
Hypoxia
Hypovolaemia
Hyper / hypokalaemia / metabolic disorders
Hypothermia / hyperthermia
Tension pneumothorax
Tamponade
Toxins
Thrombosis (pulmonary / coronary)

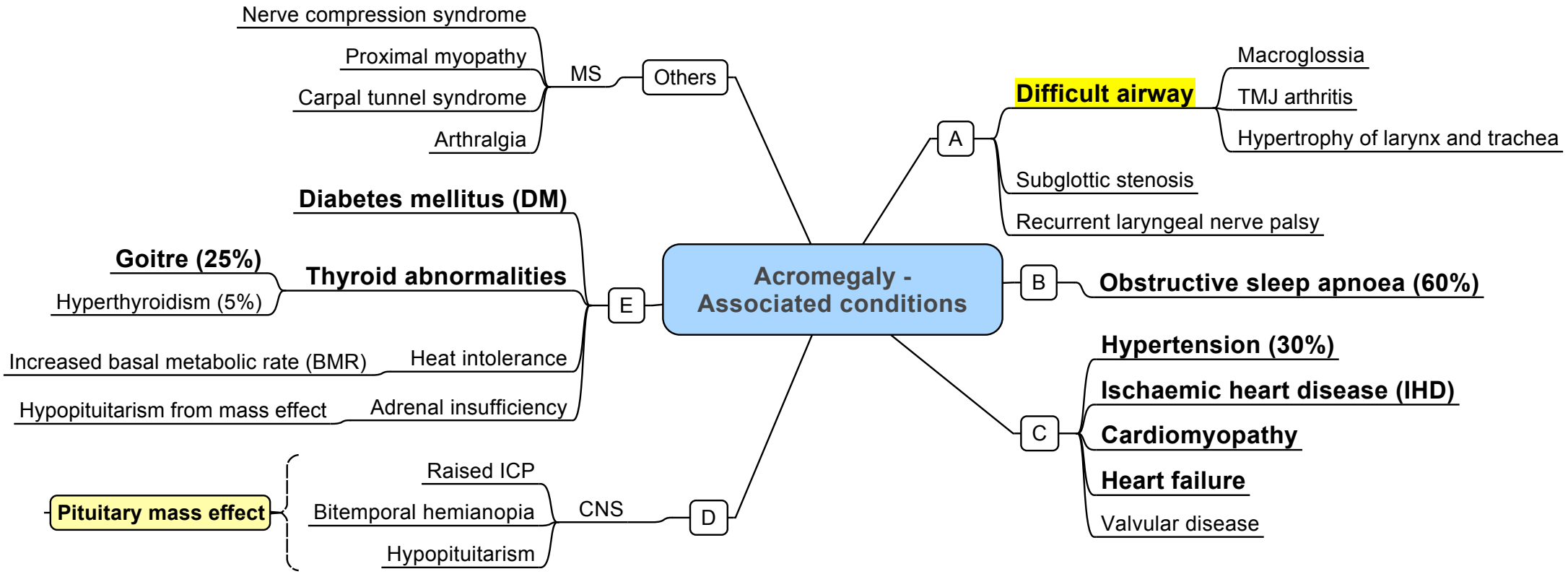
Post Resuscitation Care

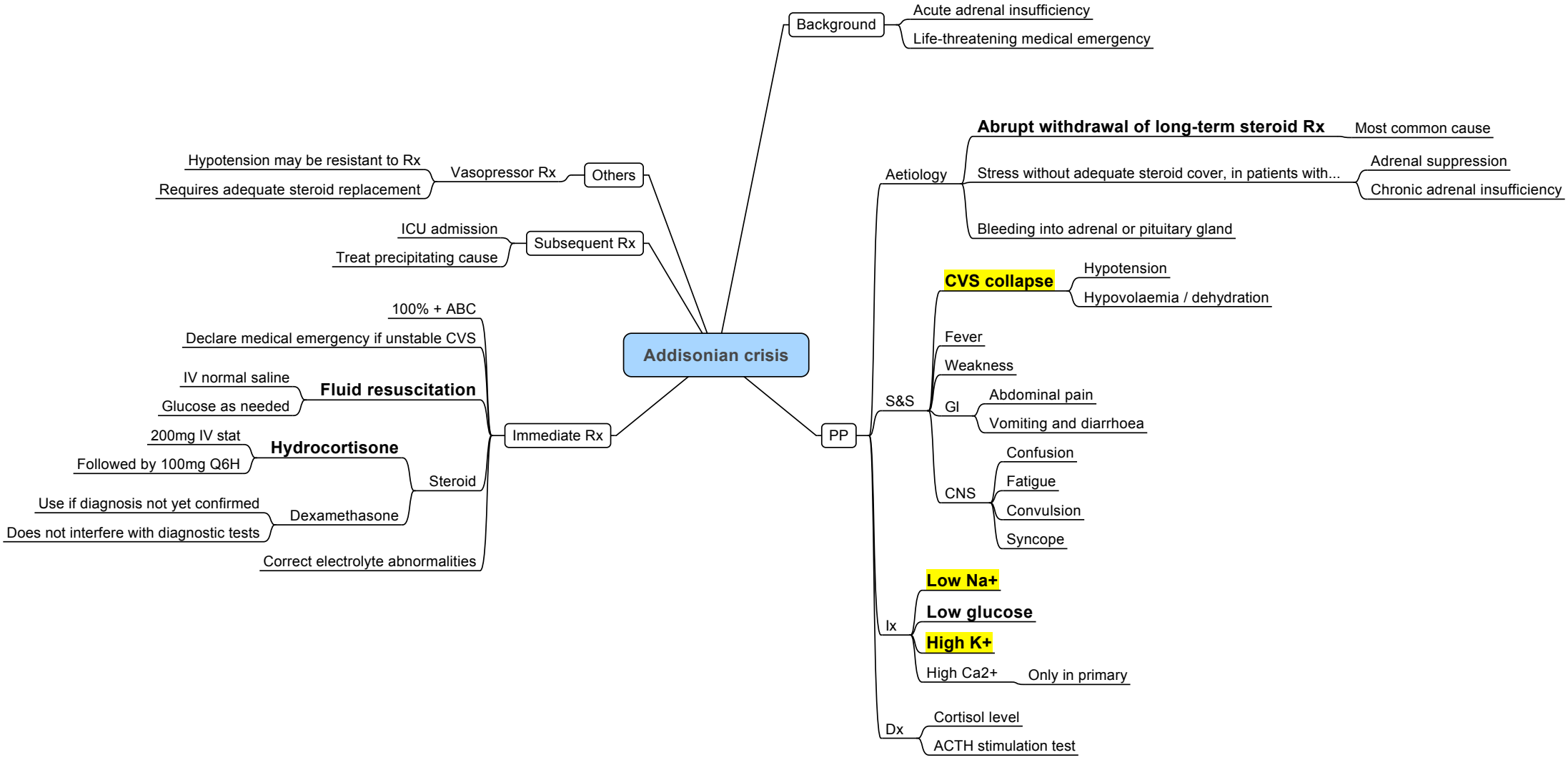
Re-evaluate ABCDE
12 lead ECG
Treat precipitating causes
Re-evaluate oxygenation and ventilation
Temperature control (cool)

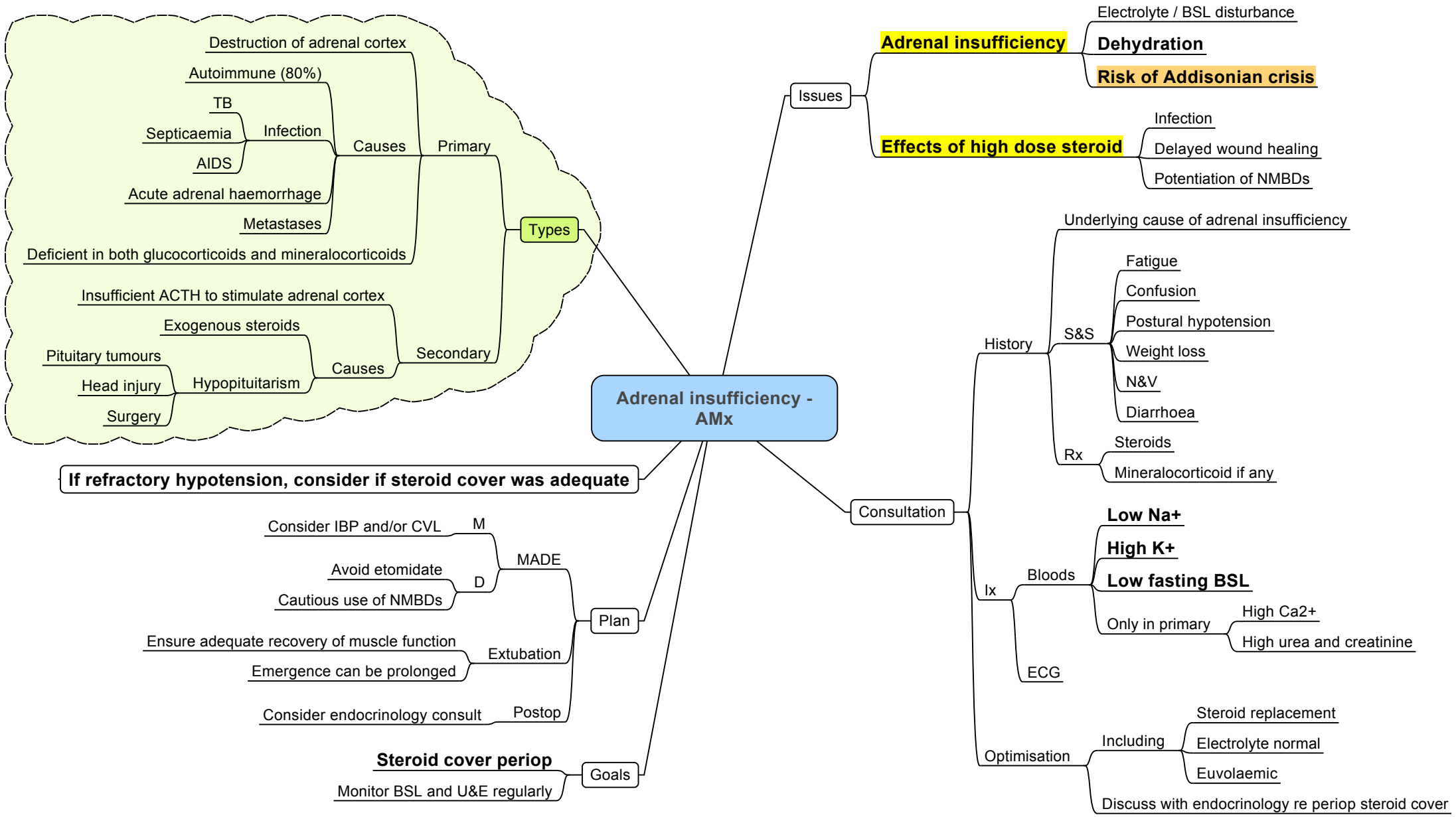
December 2010

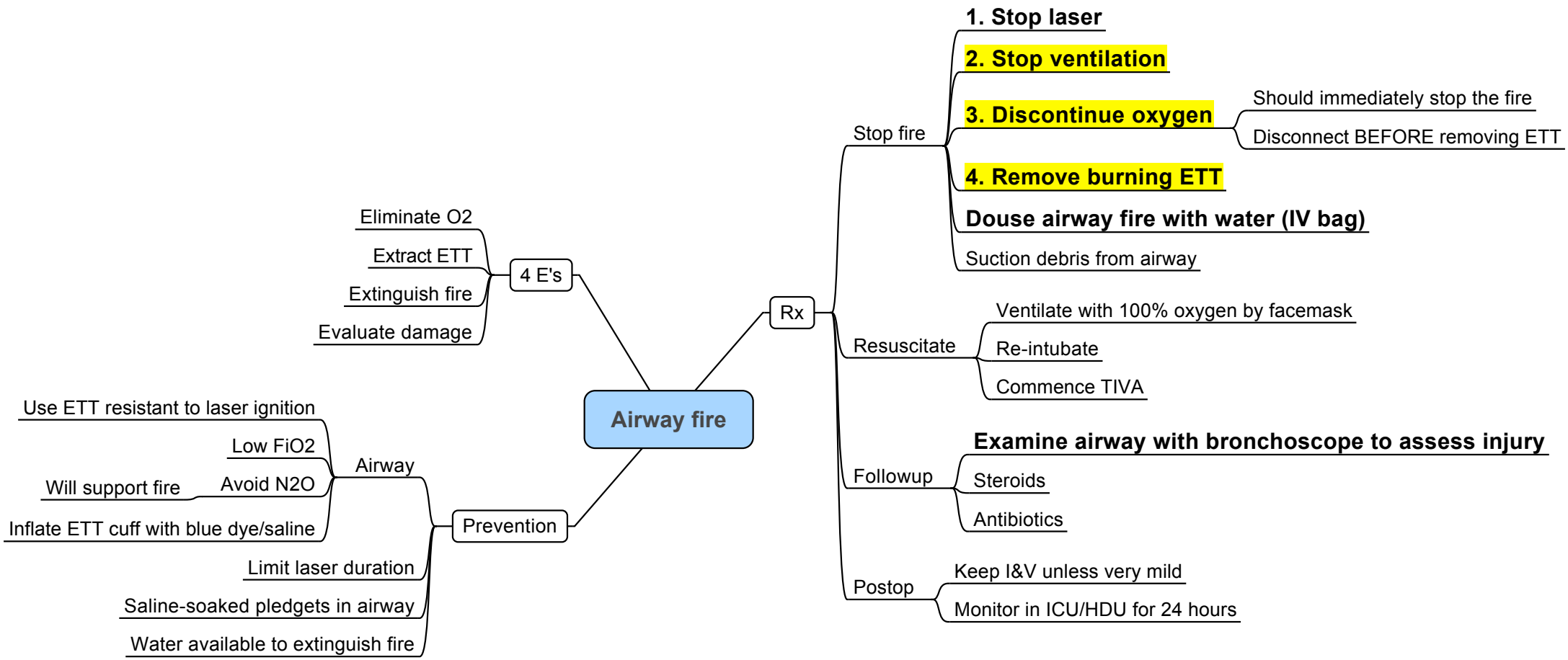

ACLS -
Reversible causes
4 H's
Hypoxaemia
Hypovolaemia
Hypothermia / hyperthermia
Hypokalaemia / hyperkalaemia
+ Other electrolyte and pH changes
4 T's
Tension pneumothorax
Thromboembolism
i.e. PE
Temponade
i.e. Cardiac temponade
Toxins / Drugs

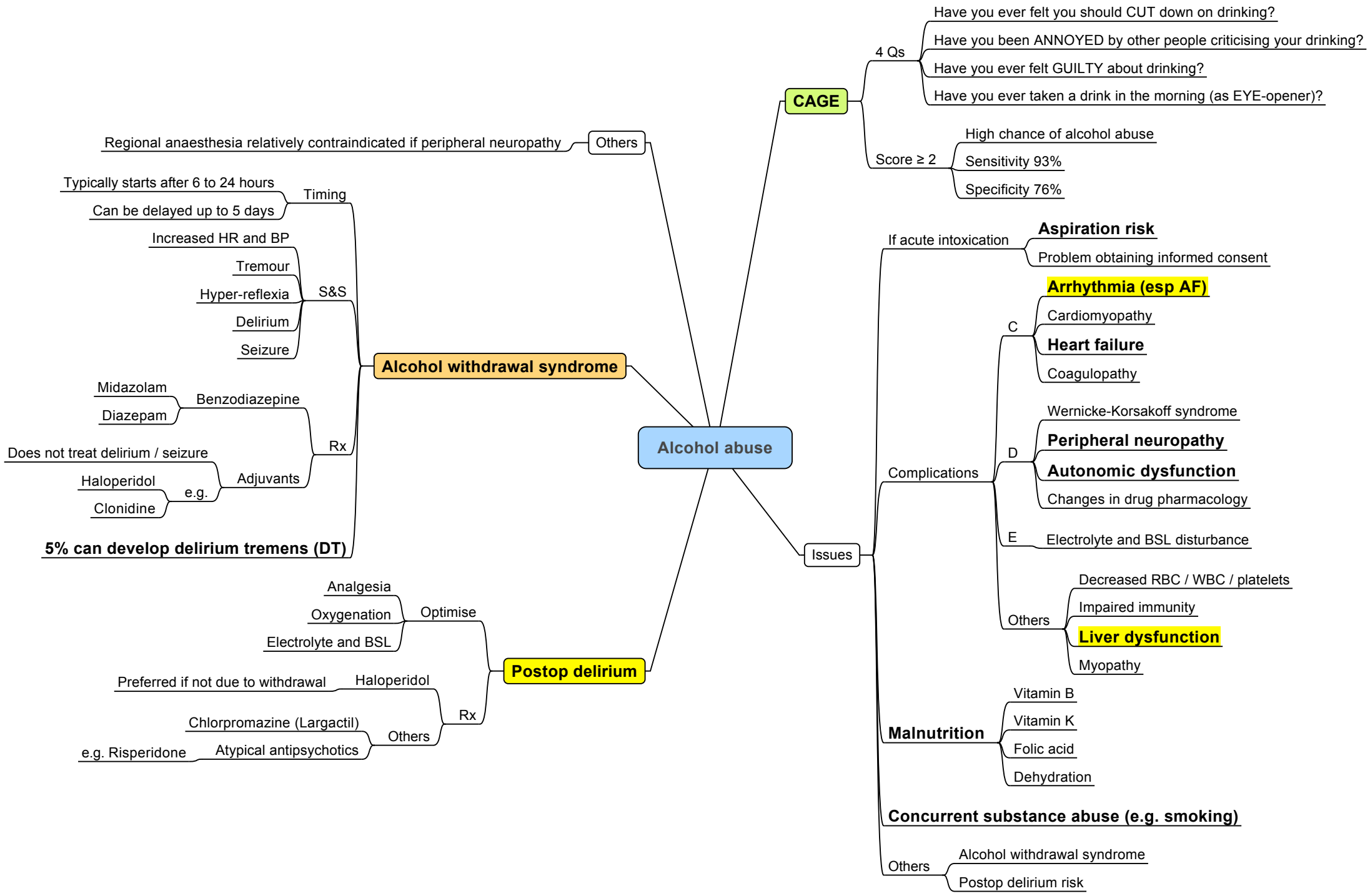

Acromegaly - AMx
Issues
The "ACE" disease
Airway difficulties
Cardiovascular disease
Endocrine abnormalities
DM
Goitre / hyperthyroidism
Intraop haemodynamic instability
Postop airway obstruction
Consultation
History
S&S related to associated conditions
esp CVS disease and OSA
Functional capacity
Associated conditions
e.g.
Difficult airway
Obstructive sleep apnoea (OSA)
Hypertension, IHD, CHF
DM, thyroid
Rx
Previous surgical Rx
Medical Rx
Octreotide
Bromocriptine
Exam
Airway assessment
CVS
Ix
CXR
ECG
Bloods
TFT
BSL
CT of neck
Upper airway anatomy
Tracheal compression
Consider
Sleep study
Echocardiogram
Document pre-existing peripheral neuropathy, if any
Optimisation
Involve endocrinology team
Consider referral to
ENT
If vocal cord pathology suspected
For indirect laryngoscopy
Respiratory
For OSA
Cardiology
Plan
MADE
M
Consider IBP
Pulse oximetry may be difficult to apply
A
Experienced assistant for difficult airway
D
Cautious use of NMBDs if myopathy
E
Difficult airway trolley
May require larger sized table / airway equipments
If ICP is not raised
Consider regional technique
Consider awake fibreoptic intubation if GA
Postop
SpO2 monitor ± Apnoea alarm
Anaglesia
Consider ICU/HDU if significant cardiorespiratory problems
Goals
Airway management
Maintain normal BSL
Others
If unexplained and persistant hypotension, consider adrenal insufficiency

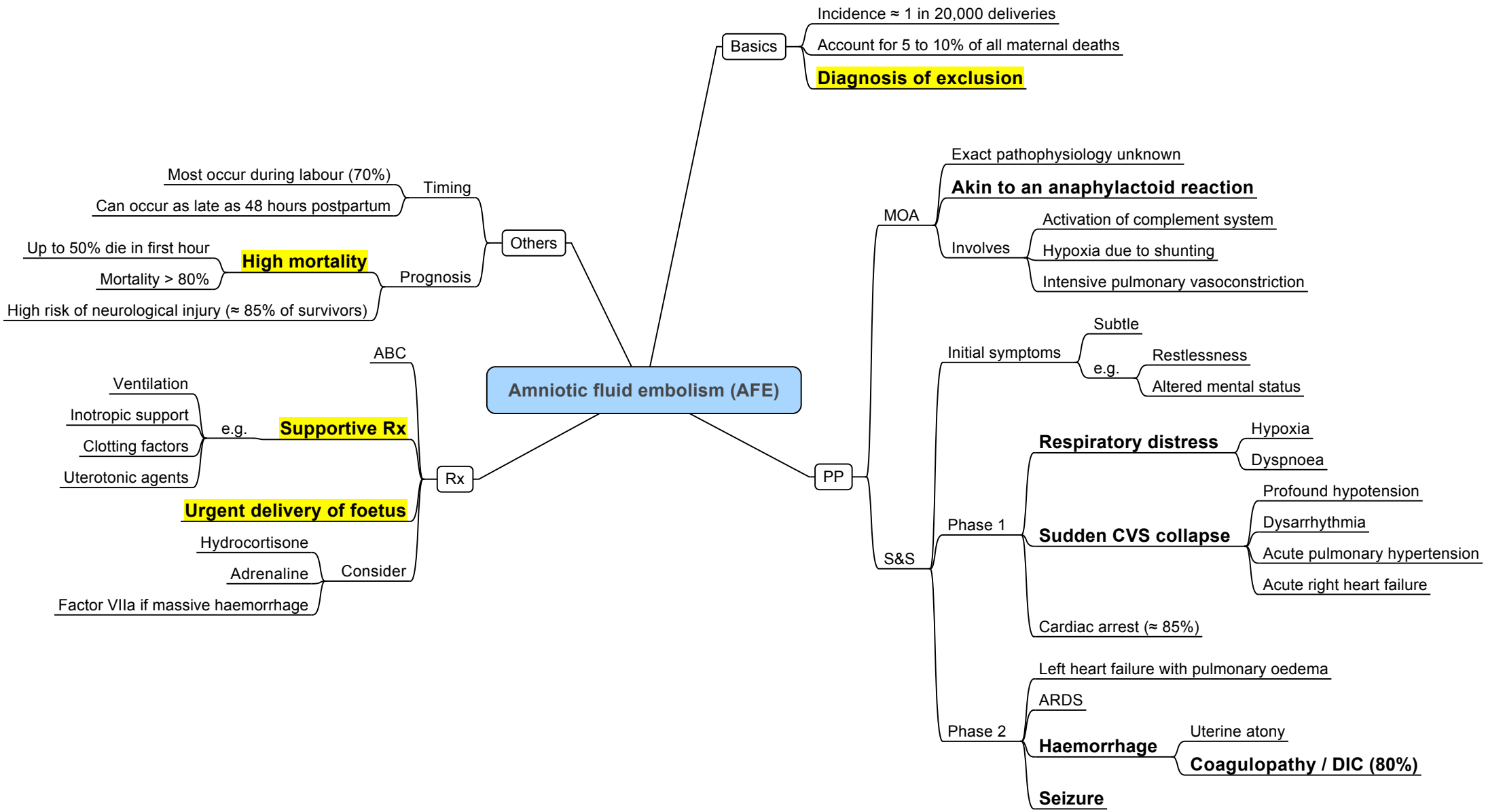

Acromegaly - Associated conditions
A
Difficult airway
Macroglossia
TMJ arthritis
Hypertrophy of larynx and trachea
Subglottic stenosis
Recurrent laryngeal nerve palsy
B
Obstructive sleep apnoea (60%)
C
Hypertension (30%)
Ischaemic heart disease (IHD)
Cardiomyopathy
Heart failure
Valvular disease
D
CNS
Raised ICP
Bitemporal hemianopia
Hypopituitarism
Pituitary mass effect
E
Diabetes mellitus (DM)
Thyroid abnormalities
Goitre (25%)
Hyperthyroidism (5%)
Heat intolerance
Increased basal metabolic rate (BMR)
Adrenal insufficiency
Hypopituitarism from mass effect
Others
MS
Nerve compression syndrome
Proximal myopathy
Carpal tunnel syndrome
Arthralgia

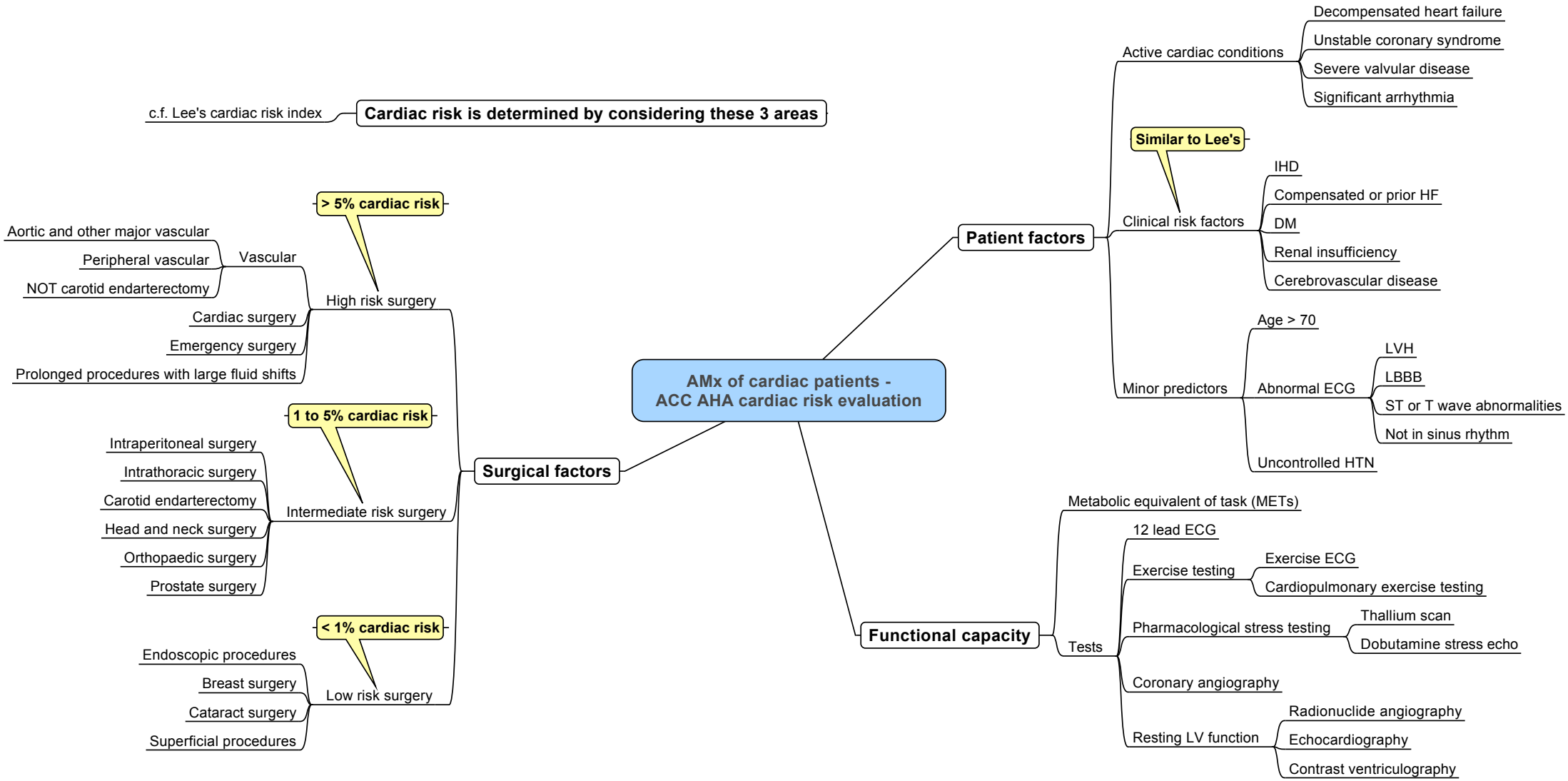

Addisonian crisis
Background
Acute adrenal insufficiency
Life-threatening medical emergency
PP
Aetiology
Abrupt withdrawal of long-term steroid Rx
Most common cause
Stress without adequate steroid cover, in patients with...
Adrenal suppression
Chronic adrenal insufficiency
Bleeding into adrenal or pituitary gland
S&S
CVS collapse
Hypotension
Hypovolaemia / dehydration
Fever
Weakness
GI
Abdominal pain
Vomiting and diarrhoea
CNS
Confusion
Fatigue
Convulsion
Syncope
Ix
Low Na+
Low glucose
High K+
High Ca2+
Only in primary
Dx
Cortisol level
ACTH stimulation test
Immediate Rx
100% + ABC
Declare medical emergency if unstable CVS
Fluid resuscitation
IV normal saline
Glucose as needed
Steroid
Hydrocortisone
200mg IV stat
Followed by 100mg Q6H
Dexamethasone
Use if diagnosis not yet confirmed
Does not interfere with diagnostic tests
Correct electrolyte abnormalities
Subsequent Rx
ICU admission
Treat precipitating cause
Others
Vasopressor Rx
Hypotension may be resistant to Rx
Requires adequate steroid replacement


Adrenal insufficiency - AMx
Types
Primary
Destruction of adrenal cortex
Causes
Autoimmune (80%)
Infection
TB
Septicaemia
AIDS
Acute adrenal haemorrhage
Metastases
Deficient in both glucocorticoids and mineralocorticoids
Secondary
Insufficient ACTH to stimulate adrenal cortex
Causes
Exogenous steroids
Hypopituitarism
Pituitary tumours
Head injury
Surgery
If refractory hypotension, consider if steroid cover was adequate
Issues
Adrenal insufficiency
Electrolyte / BSL disturbance
Dehydration
Risk of Addisonian crisis
Effects of high dose steroid
Infection
Delayed wound healing
Potentiation of NMBDs
Consultation
History
Underlying cause of adrenal insufficiency
S&S
Fatigue
Confusion
Postural hypotension
Weight loss
N&V
Diarrhoea
Rx
Steroids
Mineralocorticoid if any
Ix
Bloods
Low Na+
High K+
Low fasting BSL
Only in primary
High Ca2+
High urea and creatinine
ECG
Optimisation
Including
Steroid replacement
Electrolyte normal
Euvolaemic
Discuss with endocrinology re periop steroid cover
Plan
MADE
M
Consider IBP and/or CVL
D
Avoid etomidate
Cautious use of NMBDs
Extubation
Ensure adequate recovery of muscle function
Emergence can be prolonged
Postop
Consider endocrinology consult
Goals
Steroid cover periop
Monitor BSL and U&E regularly

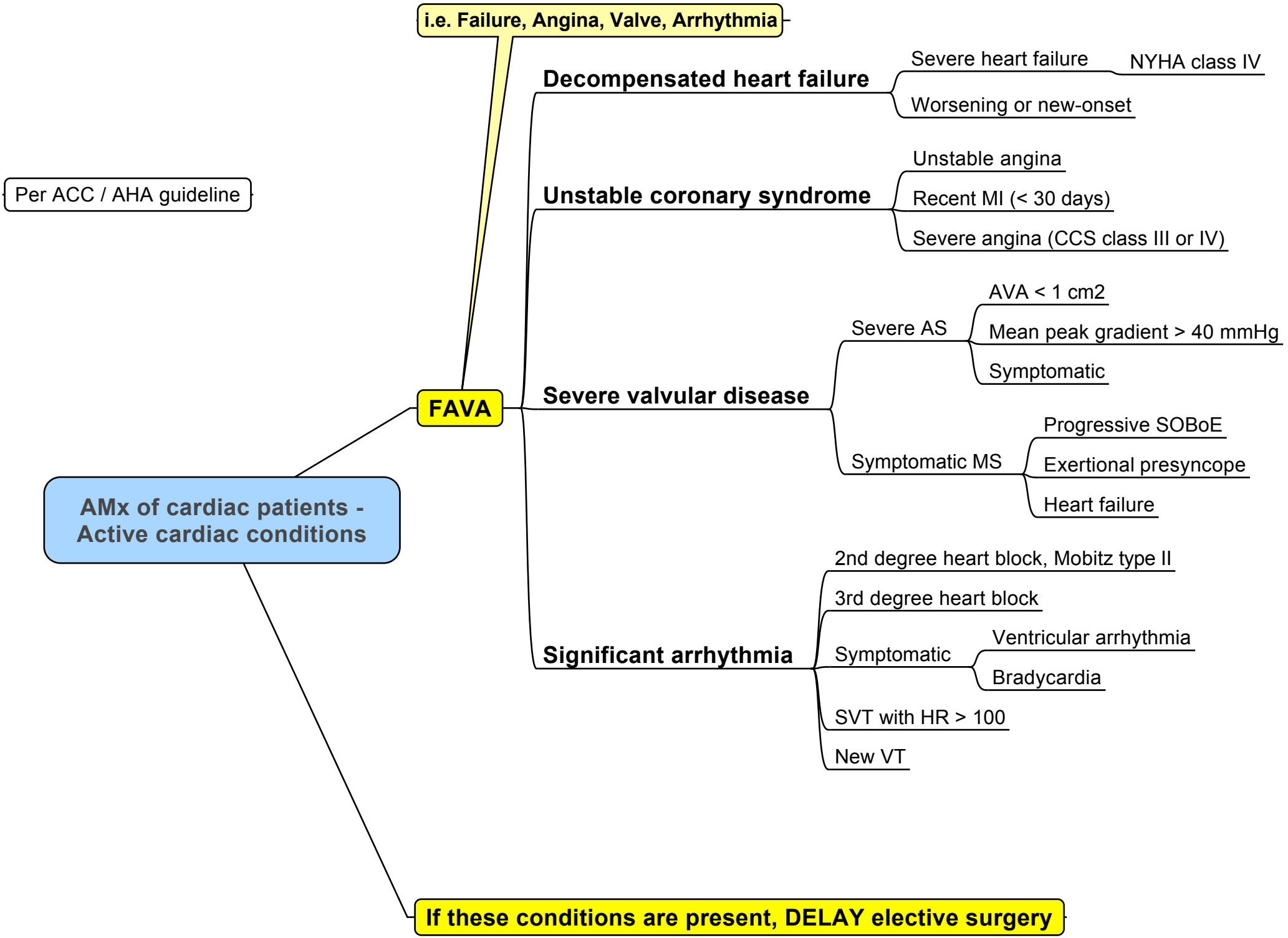

Airway fire
Rx
Stop fire
1. Stop laser
2. Stop ventilation
3. Discontinue oxygen
Should immediately stop the fire
Disconnect BEFORE removing ETT
4. Remove burning ETT
Douse airway fire with water (IV bag)
Suction debris from airway
Resuscitate
Ventilate with 100% oxygen by facemask
Re-intubate
Commence TIVA
Followup
Examine airway with bronchoscope to assess injury
Steroids
Antibiotics
Postop
Keep I&V unless very mild
Monitor in ICU/HDU for 24 hours
Prevention
4 E's
Eliminate O2
Extract ETT
Extinguish fire
Evaluate damage
Airway
Use ETT resistant to laser ignition
Low FiO2
Avoid N2O
Will support fire
Inflate ETT cuff with blue dye/saline
Limit laser duration
Saline-soaked pledgets in airway
Water available to extinguish fire

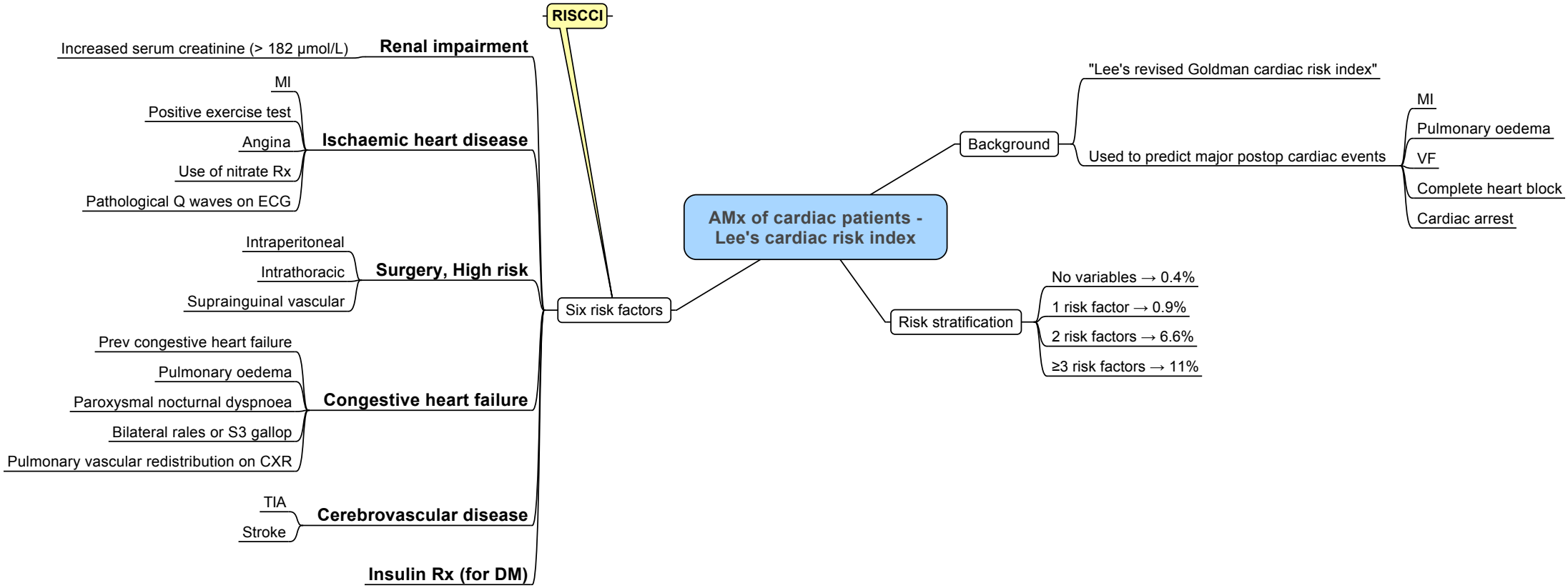

Alcohol abuse
CAGE
4 Qs
Have you ever felt you should CUT down on drinking?
Have you been ANNOYED by other people criticising your drinking?
Have you ever felt GUILTY about drinking?
Have you ever taken a drink in the morning (as EYE-opener)?
Score ≥ 2
High chance of alcohol abuse
Sensitivity 93%
Specificity 76%
Issues
If acute intoxication
Aspiration risk
Problem obtaining informed consent
Complications
C
Arrhythmia (esp AF)
Cardiomyopathy
Heart failure
Coagulopathy
D
Wernicke-Korsakoff syndrome
Peripheral neuropathy
Autonomic dysfunction
Changes in drug pharmacology
E
Electrolyte and BSL disturbance
Others
Decreased RBC / WBC / platelets
Impaired immunity
Liver dysfunction
Myopathy
Malnutrition
Vitamin B
Vitamin K
Folic acid
Dehydration
Concurrent substance abuse (e.g. smoking)
Others
Alcohol withdrawal syndrome
Postop delirium risk
Alcohol withdrawal syndrome
Others
Regional anaesthesia relatively contraindicated if peripheral neuropathy
Timing
Typically starts after 6 to 24 hours
Can be delayed up to 5 days
S&S
Increased HR and BP
Tremour
Hyper-reflexia
Delirium
Seizure
Rx
Benzodiazepine
Midazolam
Diazepam
Adjuvants
Does not treat delirium / seizure
e.g.
Haloperidol
Clonidine
5% can develop delirium tremens (DT)
Postop delirium
Optimise
Analgesia
Oxygenation
Electrolyte and BSL
Rx
Haloperidol
Preferred if not due to withdrawal
Others
Chlorpromazine (Largactil)
Atypical antipsychotics
e.g. Risperidone

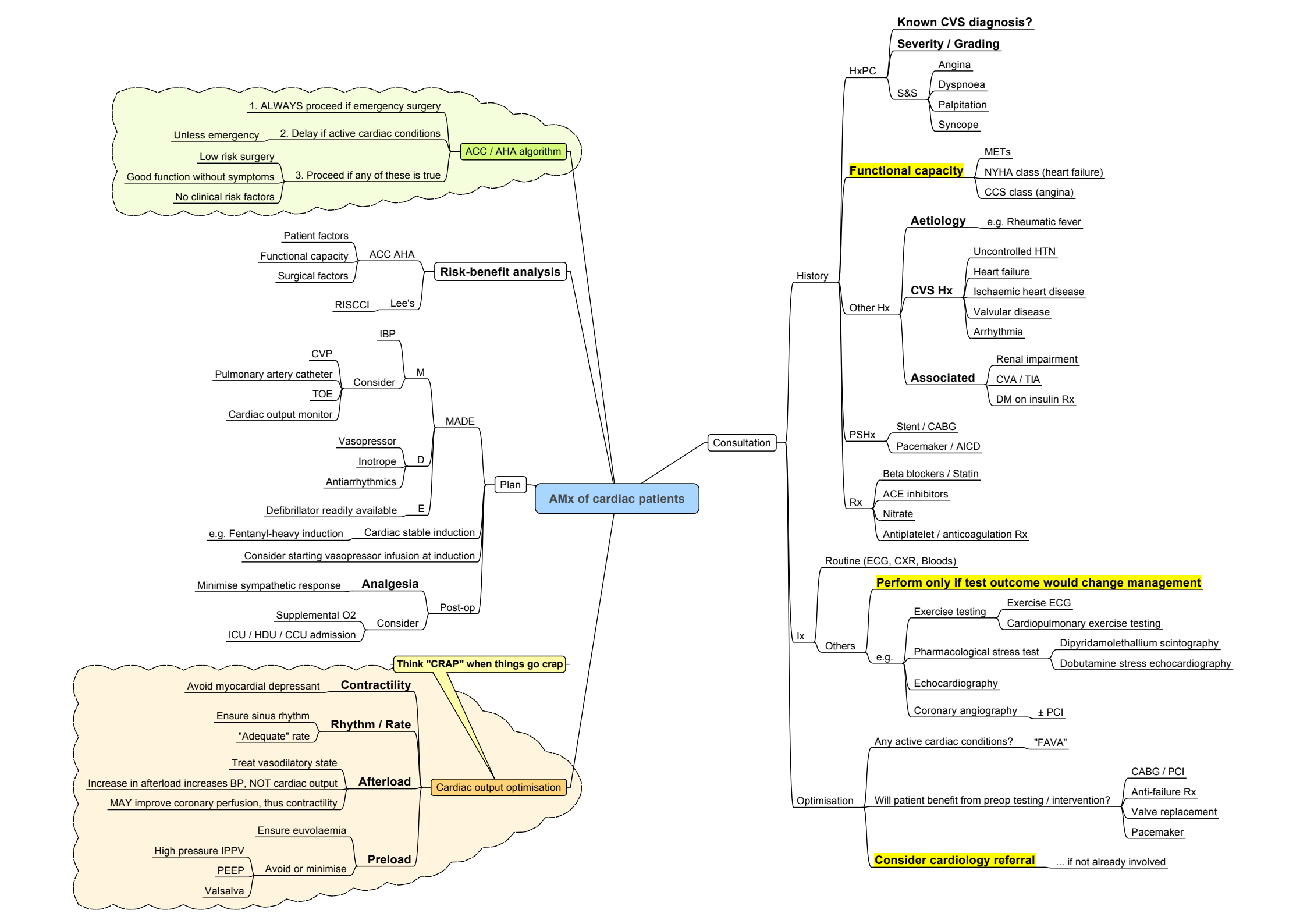

Amniotic fluid embolism (AFE)
Basics
Incidence ≈ 1 in 20,000 deliveries
Account for 5 to 10% of all maternal deaths
Diagnosis of exclusion
PP
MOA
Exact pathophysiology unknown
Akin to an anaphylactoid reaction
Involves
Activation of complement system
Hypoxia due to shunting
Intensive pulmonary vasoconstriction
S&S
Initial symptoms
Subtle
e.g.
Restlessness
Altered mental status
Phase 1
Respiratory distress
Hypoxia
Dyspnoea
Sudden CVS collapse
Profound hypotension
Dysarrhythmia
Acute pulmonary hypertension
Acute right heart failure
Cardiac arrest (≈ 85%)
Phase 2
Left heart failure with pulmonary oedema
ARDS
Haemorrhage
Uterine atony
Coagulopathy / DIC (80%)
Seizure
Rx
ABC
e.g.
Supportive Rx
Ventilation
Inotropic support
Clotting factors
Uterotonic agents
Urgent delivery of foetus
Consider
Hydrocortisone
Adrenaline
Factor VIIa if massive haemorrhage
Others
Timing
Most occur during labour (70%)
Can occur as late as 48 hours postpartum
Prognosis
High mortality
Up to 50% die in first hour
Mortality > 80%
High risk of neurological injury (≈ 85% of survivors)

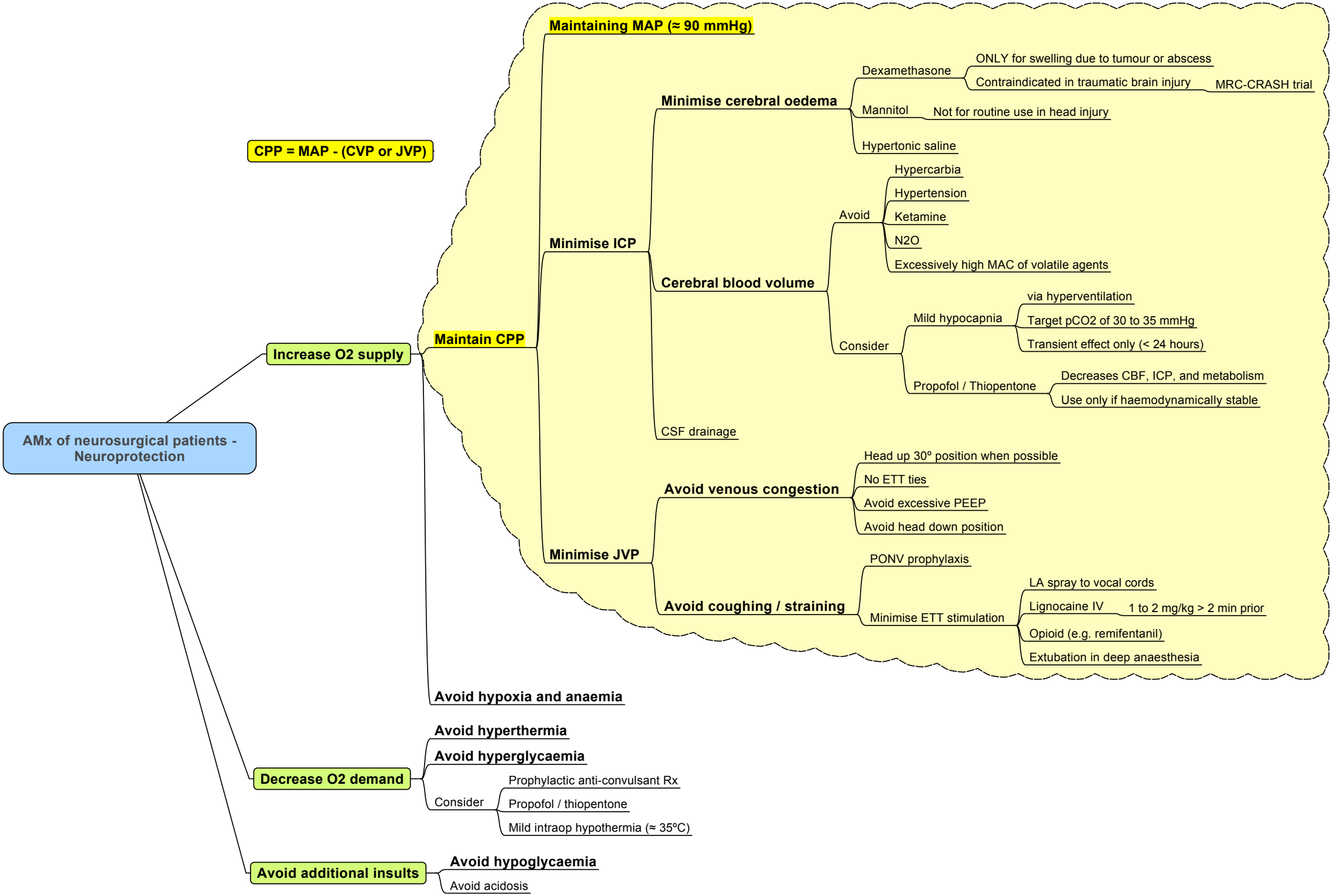

AMx of cardiac patients -
ACC AHA cardiac risk evaluation
Cardiac risk is determined by considering these 3 areas
c.f. Lee's cardiac risk index
Patient factors
Active cardiac conditions
Decompensated heart failure
Unstable coronary syndrome
Severe valvular disease
Significant arrhythmia
Similar to Lee's
Clinical risk factors
IHD
Compensated or prior HF
DM
Renal insufficiency
Cerebrovascular disease
Minor predictors
Age > 70
Abnormal ECG
LVH
LBBB
ST or T wave abnormalities
Not in sinus rhythm
Uncontrolled HTN
Surgical factors
> 5% cardiac risk
High risk surgery
Vascular
Aortic and other major vascular
Peripheral vascular
NOT carotid endarterectomy
Cardiac surgery
Emergency surgery
Prolonged procedures with large fluid shifts
1 to 5% cardiac risk
Intermediate risk surgery
Intraperitoneal surgery
Intrathoracic surgery
Carotid endarterectomy
Head and neck surgery
Orthopaedic surgery
Prostate surgery
< 1% cardiac risk
Low risk surgery
Endoscopic procedures
Breast surgery
Cataract surgery
Superficial procedures
Functional capacity
Metabolic equivalent of task (METs)
Tests
12 lead ECG
Exercise testing
Exercise ECG
Cardiopulmonary exercise testing
Pharmacological stress testing
Thallium scan
Dobutamine stress echo
Coronary angiography
Resting LV function
Radionuclide angiography
Echocardiography
Contrast ventriculography

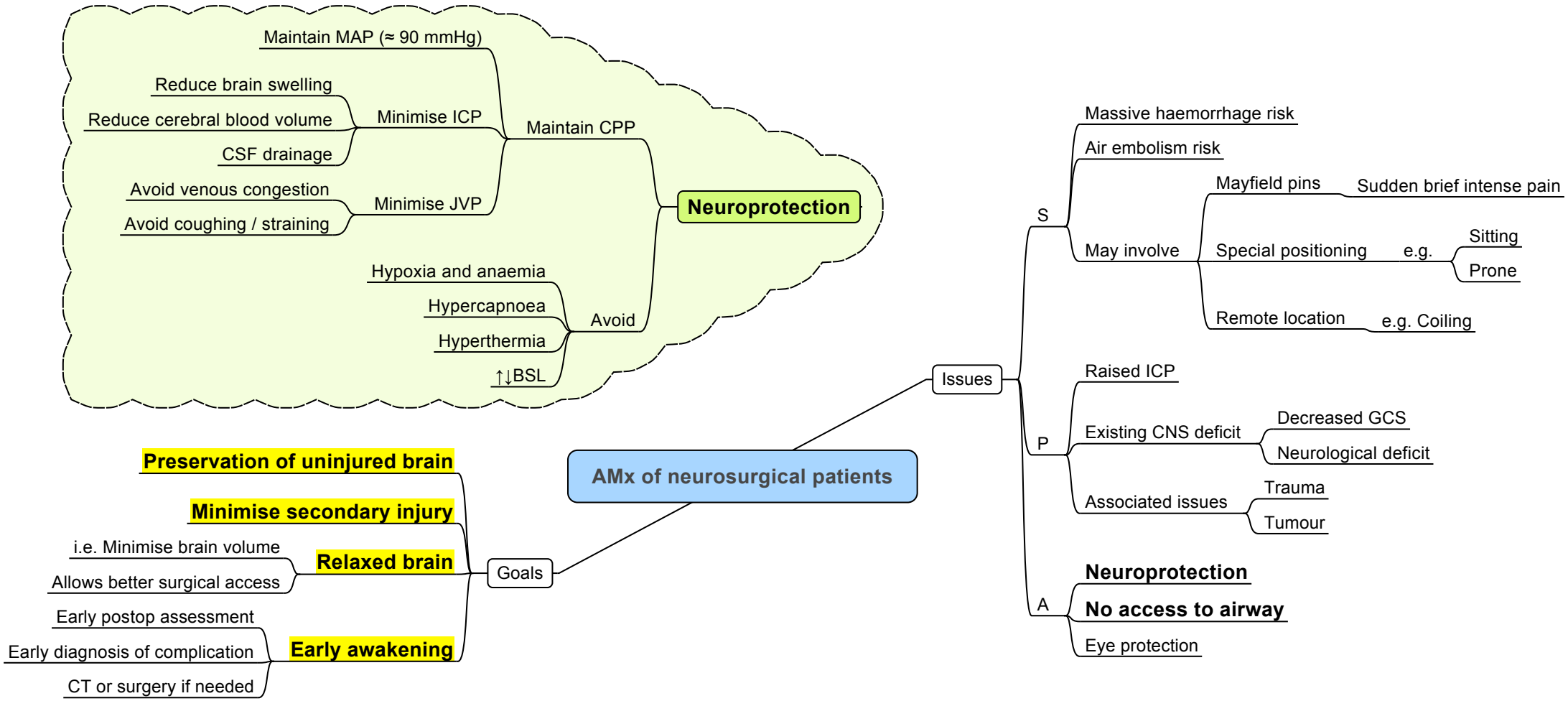

Emergency surgery?
Yes
No
Active cardiac condition?
Yes
Delay surgery
Workup + optimise
Proceed
No
Good functional capacity
… without symptoms?
Yes
No
Low risk surgery?
Yes
No
No clinical risk factors?
Yes
i.e. No clinical risk factors
No
Further workup needed
High risk surgery
AND
At least 3 clinical risk factors?
Yes
Consider testing
No
Consider non-invasive testing

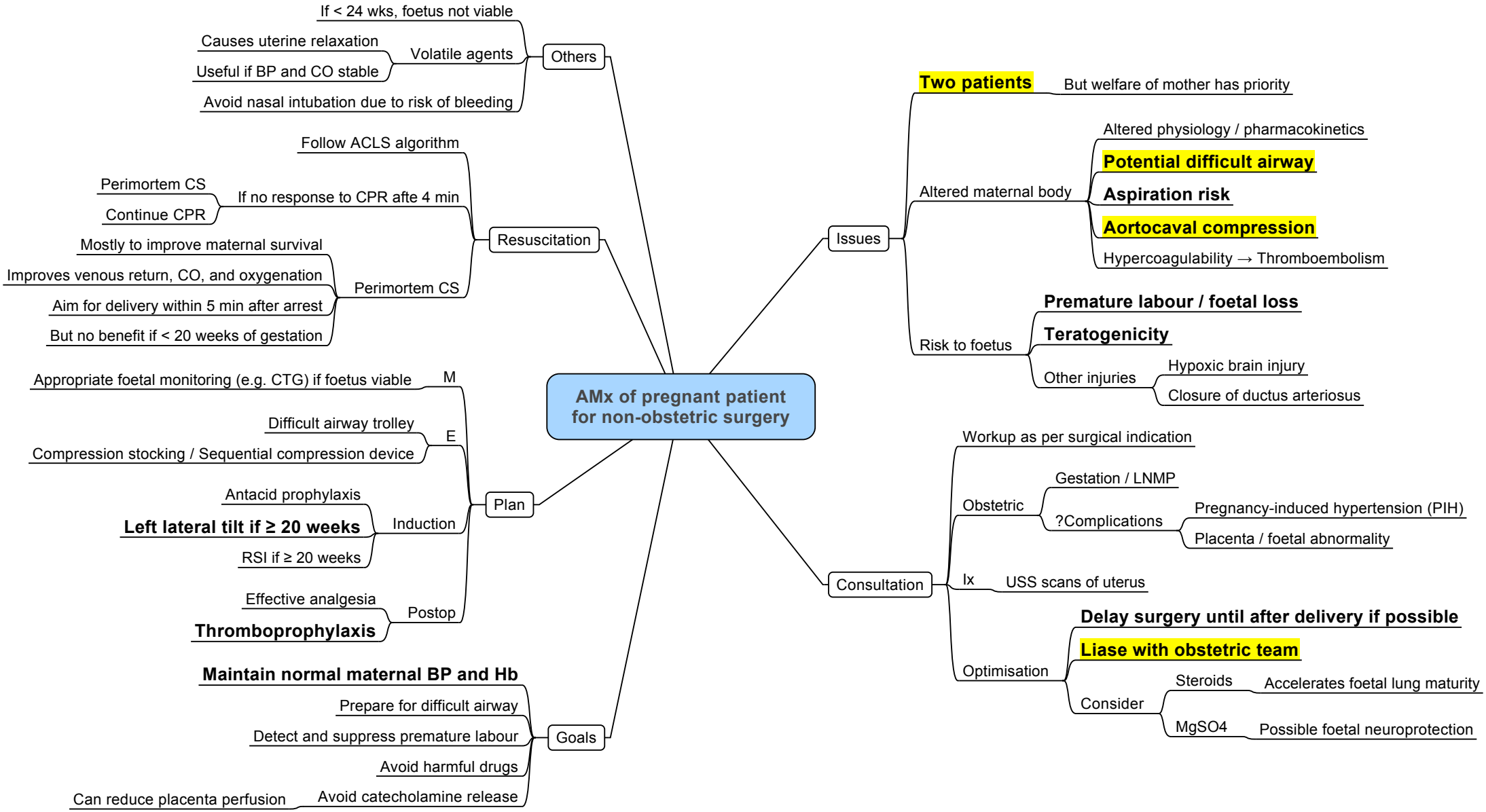

AMx of cardiac patients - Active cardiac conditions
Per ACC / AHA guideline
FAVA
i.e. Failure, Angina, Valve, Arrhythmia
Decompensated heart failure
Severe heart failure
NYHA class IV
Worsening or new-onset
Unstable coronary syndrome
Unstable angina
Recent MI (< 30 days)
Severe angina (CCS class III or IV)
Severe valvular disease
Severe AS
AVA < 1 cm2
Mean peak gradient > 40 mmHg
Symptomatic
Symptomatic MS
Progressive SOBoE
Exertional presyncope
Heart failure
Significant arrhythmia
2nd degree heart block, Mobitz type II
3rd degree heart block
Symptomatic
Ventricular arrhythmia
Bradycardia
SVT with HR > 100
New VT
If these conditions are present, DELAY elective surgery

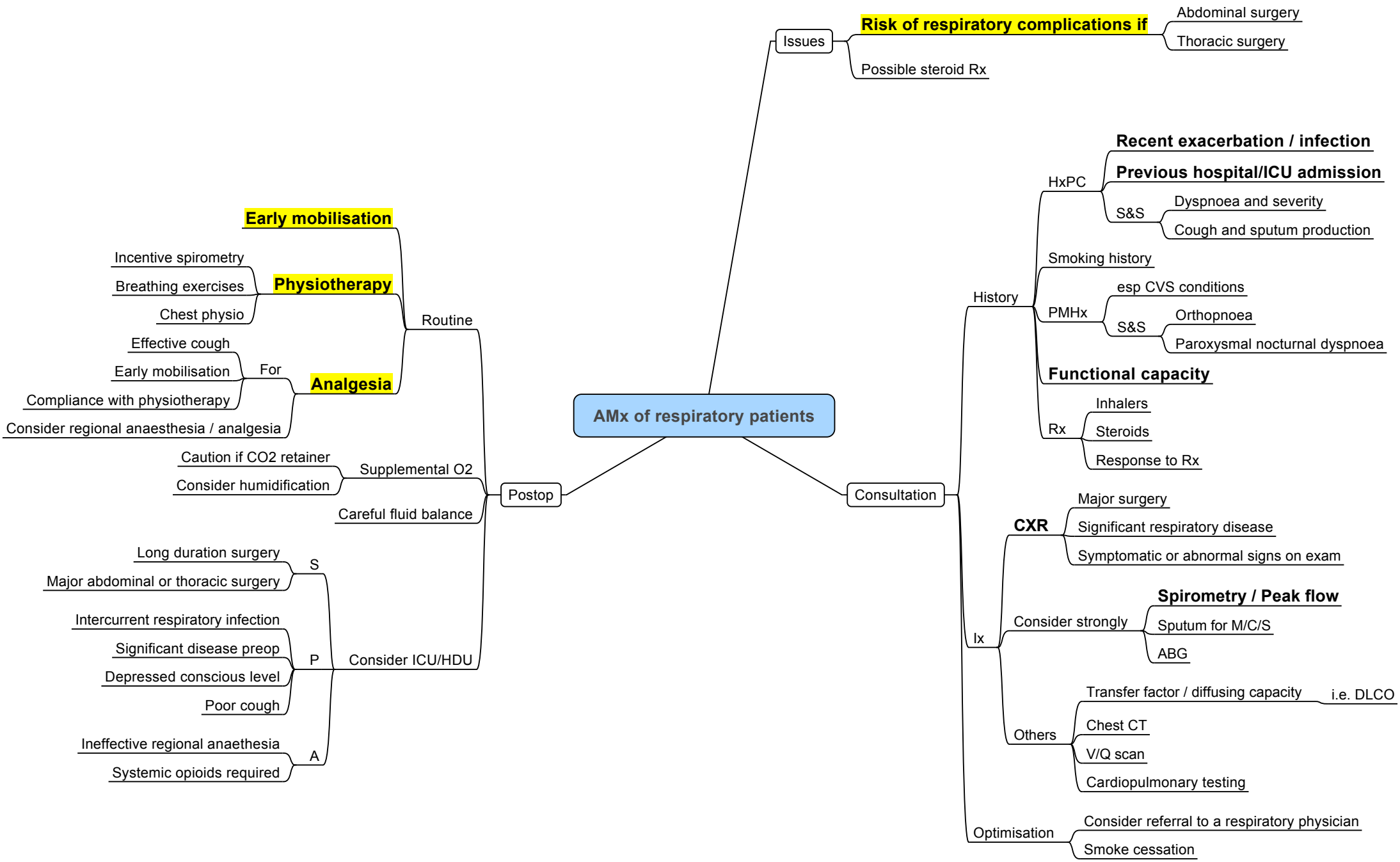

AMx of cardiac patients - Lee's cardiac risk index
Background
"Lee's revised Goldman cardiac risk index"
Used to predict major postop cardiac events
MI
Pulmonary oedema
VF
Complete heart block
Cardiac arrest
Risk stratification
No variables → 0.4%
1 risk factor → 0.9%
2 risk factors → 6.6%
≥3 risk factors → 11%
Six risk factors
RISCCI
Renal impairment
Increased serum creatinine (> 182 µmol/L)
Ischaemic heart disease
MI
Positive exercise test
Angina
Use of nitrate Rx
Pathological Q waves on ECG
Surgery, High risk
Intraperitoneal
Intrathoracic
Suprainguinal vascular
Congestive heart failure
Prev congestive heart failure
Pulmonary oedema
Paroxysmal nocturnal dyspnoea
Bilateral rales or S3 gallop
Pulmonary vascular redistribution on CXR
Cerebrovascular disease
TIA
Stroke
Insulin Rx (for DM)

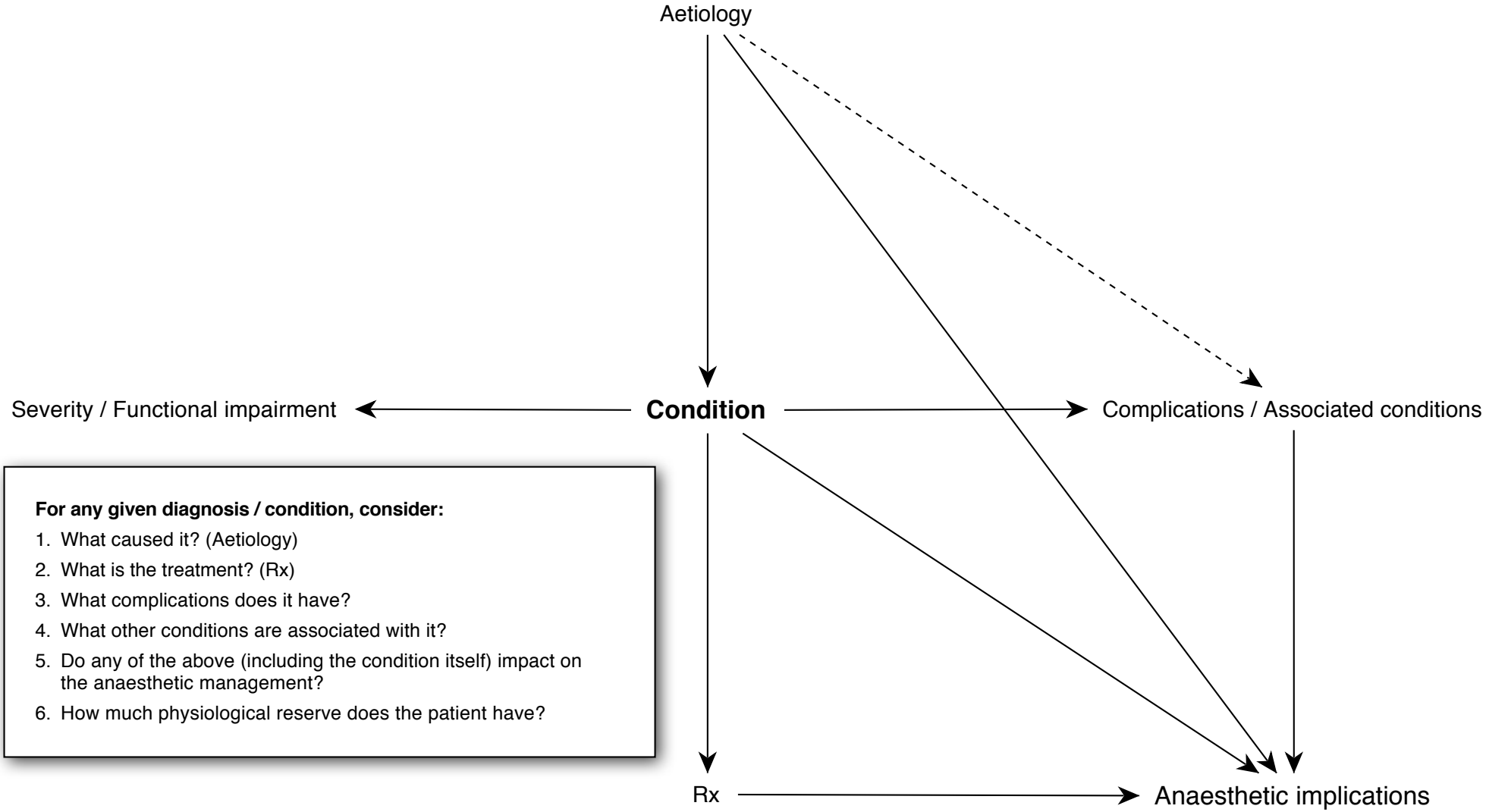

AMx of cardiac patients
Consultation
History
HxPC
Known CVS diagnosis?
Severity / Grading
S&S
Angina
Dyspnoea
Palpitation
Syncope
Functional capacity
METs
NYHA class (heart failure)
CCS class (angina)
Other Hx
Aetiology
e.g. Rheumatic fever
CVS Hx
Uncontrolled HTN
Heart failure
Ischaemic heart disease
Valvular disease
Arrhythmia
Associated
Renal impairment
CVA / TIA
DM on insulin Rx
PSHx
Stent / CABG
Pacemaker / AICD
Rx
Beta blockers / Statin
ACE inhibitors
Nitrate
Antiplatelet / anticoagulation Rx
Ix
Routine (ECG, CXR, Bloods)
Others
Perform only if test outcome would change management
e.g.
Exercise testing
Exercise ECG
Cardiopulmonary exercise testing
Pharmacological stress test
Dipyridamolethallium scintography
Dobutamine stress echocardiography
Echocardiography
Coronary angiography
± PCI
Optimisation
Any active cardiac conditions?
"FAVA"
Will patient benefit from preop testing / intervention?
CABG / PCI
Anti-failure Rx
Valve replacement
Pacemaker
Consider cardiology referral
... if not already involved
Plan
MADE
M
Consider
IBP
CVP
Pulmonary artery catheter
TOE
Cardiac output monitor
D
Vasopressor
Inotrope
Antiarrhythmics
E
Defibrillator readily available
Cardiac stable induction
e.g. Fentanyl-heavy induction
Consider starting vasopressor infusion at induction
Post-op
Analgesia
Minimise sympathetic response
Consider
Supplemental O2
ICU / HDU / CCU admission
Risk-benefit analysis
ACC AHA
Patient factors
Functional capacity
Surgical factors
Lee's
RISCCI
ACC / AHA algorithm
1. ALWAYS proceed if emergency surgery
2. Delay if active cardiac conditions
Unless emergency
3. Proceed if any of these is true
Low risk surgery
Good function without symptoms
No clinical risk factors
Cardiac output optimisation
Think "CRAP" when things go crap
Contractility
Avoid myocardial depressant
Rhythm / Rate
Ensure sinus rhythm
"Adequate" rate
Afterload
Treat vasodilatory state
Increase in afterload increases BP, NOT cardiac output
MAY improve coronary perfusion, thus contractility
Preload
Ensure euvolaemia
Avoid or minimise
High pressure IPPV
PEEP
Valsalva


AMx of neurosurgical patients - Neuroprotection
Increase O2 supply
CPP = MAP - (CVP or JVP)
Maintain CPP
Maintaining MAP (≈ 90 mmHg)
Minimise ICP
Minimise cerebral oedema
Dexamethasone
ONLY for swelling due to tumour or abscess
Contraindicated in traumatic brain injury
MRC-CRASH trial
Mannitol
Not for routine use in head injury
Hypertonic saline
Cerebral blood volume
Avoid
Hypercarbia
Hypertension
Ketamine
N2O
Excessively high MAC of volatile agents
Consider
Mild hypocapnia
via hyperventilation
Target pCO2 of 30 to 35 mmHg
Transient effect only (< 24 hours)
Propofol / Thiopentone
Decreases CBF, ICP, and metabolism
Use only if haemodynamically stable
CSF drainage
Minimise JVP
Avoid venous congestion
Head up 30º position when possible
No ETT ties
Avoid excessive PEEP
Avoid head down position
Avoid coughing / straining
PONV prophylaxis
Minimise ETT stimulation
LA spray to vocal cords
Lignocaine IV
1 to 2 mg/kg > 2 min prior
Opioid (e.g. remifentanil)
Extubation in deep anaesthesia
Avoid hypoxia and anaemia
Decrease O2 demand
Avoid hyperthermia
Avoid hyperglycaemia
Consider
Prophylactic anti-convulsant Rx
Propofol / thiopentone
Mild intraop hypothermia (≈ 35ºC)
Avoid additional insults
Avoid hypoglycaemia
Avoid acidosis


AMx of neurosurgical patients
Goals
Preservation of uninjured brain
Minimise secondary injury
Relaxed brain
i.e. Minimise brain volume
Allows better surgical access
Early awakening
Early postop assessment
Early diagnosis of complication
CT or surgery if needed
Neuroprotection
Maintain CPP
Maintain MAP (≈ 90 mmHg)
Minimise ICP
Reduce brain swelling
Reduce cerebral blood volume
CSF drainage
Minimise JVP
Avoid venous congestion
Avoid coughing / straining
Avoid
Hypoxia and anaemia
Hypercapnoea
Hyperthermia
↑↓BSL
Issues
S
Massive haemorrhage risk
Air embolism risk
May involve
Mayfield pins
Sudden brief intense pain
Special positioning
e.g.
Sitting
Prone
Remote location
e.g. Coiling
P
Raised ICP
Existing CNS deficit
Decreased GCS
Neurological deficit
Associated issues
Trauma
Tumour
A
Neuroprotection
No access to airway
Eye protection

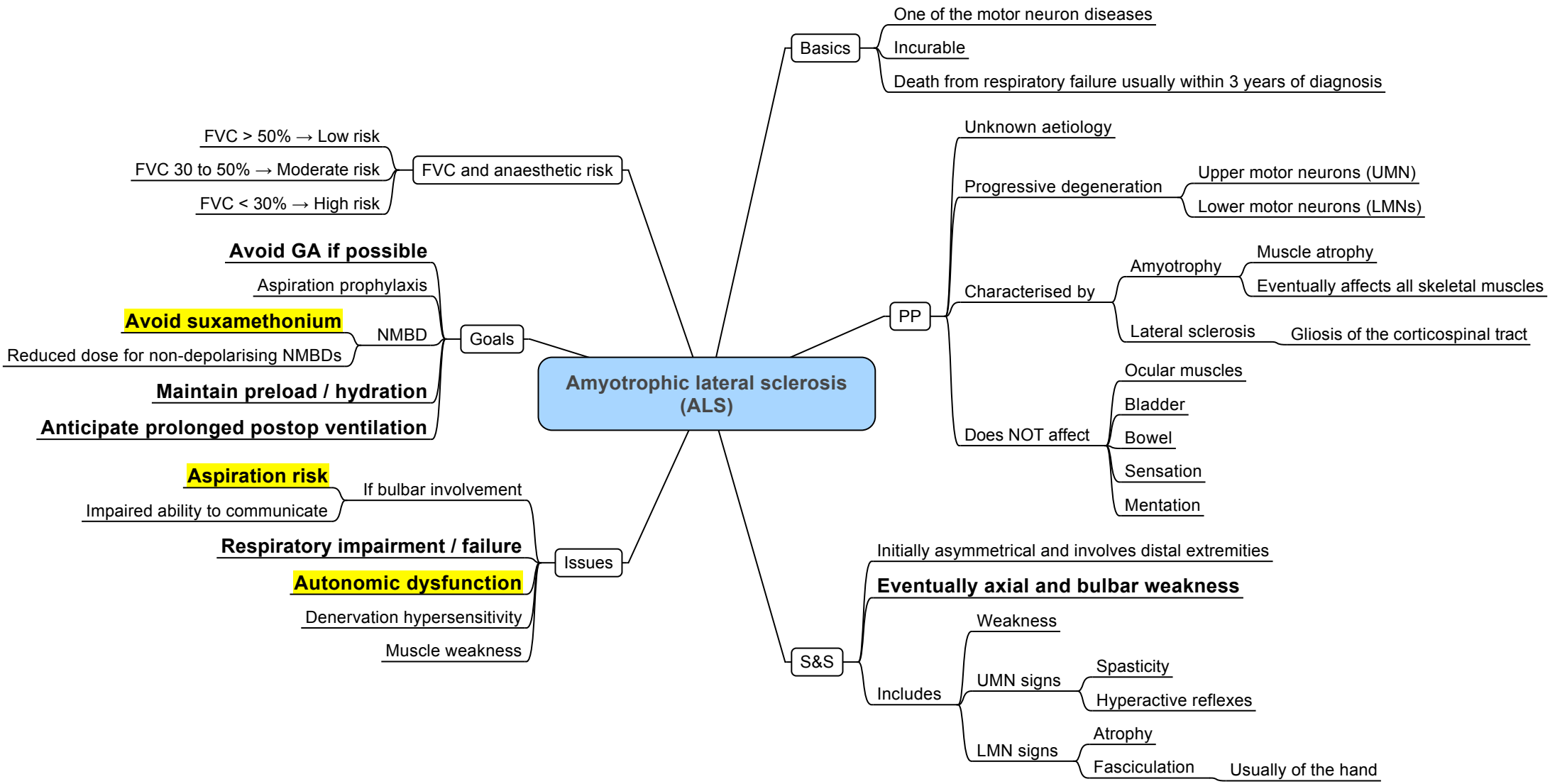

AMx of pregnant patient for non-obstetric surgery
Issues
Two patients
But welfare of mother has priority
Altered maternal body
Altered physiology / pharmacokinetics
Potential difficult airway
Aspiration risk
Aortocaval compression
Hypercoagulability → Thromboembolism
Risk to foetus
Premature labour / foetal loss
Teratogenicity
Other injuries
Hypoxic brain injury
Closure of ductus arteriosus
Consultation
Workup as per surgical indication
Obstetric
Gestation / LNMP
?Complications
Pregnancy-induced hypertension (PIH)
Placenta / foetal abnormality
Ix
USS scans of uterus
Optimisation
Delay surgery until after delivery if possible
Liase with obstetric team
Consider
Steroids
Accelerates foetal lung maturity
MgSO4
Possible foetal neuroprotection
Goals
Maintain normal maternal BP and Hb
Prepare for difficult airway
Detect and suppress premature labour
Avoid harmful drugs
Avoid catecholamine release
Can reduce placenta perfusion
Plan
M
Appropriate foetal monitoring (e.g. CTG) if foetus viable
E
Difficult airway trolley
Compression stocking / Sequential compression device
Induction
Antacid prophylaxis
Left lateral tilt if ≥ 20 weeks
RSI if ≥ 20 weeks
Postop
Effective analgesia
Thromboprophylaxis
Resuscitation
Follow ACLS algorithm
If no response to CPR afte 4 min
Perimortem CS
Continue CPR
Perimortem CS
Mostly to improve maternal survival
Improves venous return, CO, and oxygenation
Aim for delivery within 5 min after arrest
But no benefit if < 20 weeks of gestation
Others
If < 24 wks, foetus not viable
Volatile agents
Causes uterine relaxation
Useful if BP and CO stable
Avoid nasal intubation due to risk of bleeding

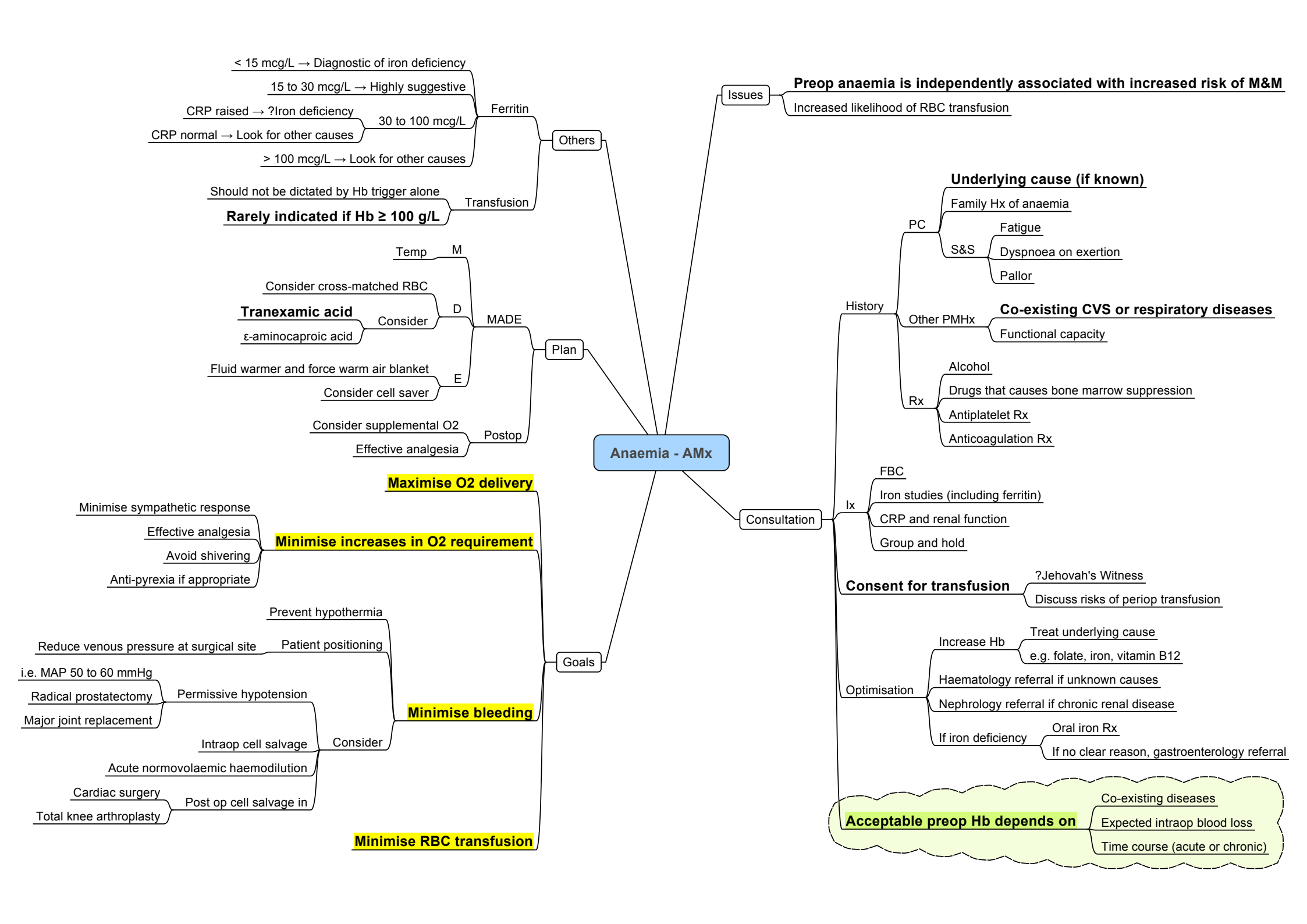

AMx of respiratory patients
Issues
Risk of respiratory complications if
Abdominal surgery
Thoracic surgery
Possible steroid Rx
Consultation
History
HxPC
Recent exacerbation / infection
Previous hospital/ICU admission
S&S
Dyspnoea and severity
Cough and sputum production
Smoking history
PMHx
esp CVS conditions
S&S
Orthopnoea
Paroxysmal nocturnal dyspnoea
Functional capacity
Rx
Inhalers
Steroids
Response to Rx
Ix
CXR
Major surgery
Significant respiratory disease
Symptomatic or abnormal signs on exam
Consider strongly
Spirometry / Peak flow
Sputum for M/C/S
ABG
Others
Transfer factor / diffusing capacity
i.e. DLCO
Chest CT
V/Q scan
Cardiopulmonary testing
Optimisation
Consider referral to a respiratory physician
Smoke cessation
Postop
Routine
Early mobilisation
Physiotherapy
Incentive spirometry
Breathing exercises
Chest physio
For
Analgesia
Effective cough
Early mobilisation
Compliance with physiotherapy
Consider regional anaesthesia / analgesia
Supplemental O2
Caution if CO2 retainer
Consider humidification
Careful fluid balance
Consider ICU/HDU
S
Long duration surgery
Major abdominal or thoracic surgery
P
Intercurrent respiratory infection
Significant disease preop
Depressed conscious level
Poor cough
A
Ineffective regional anaesthesia
Systemic opioids required

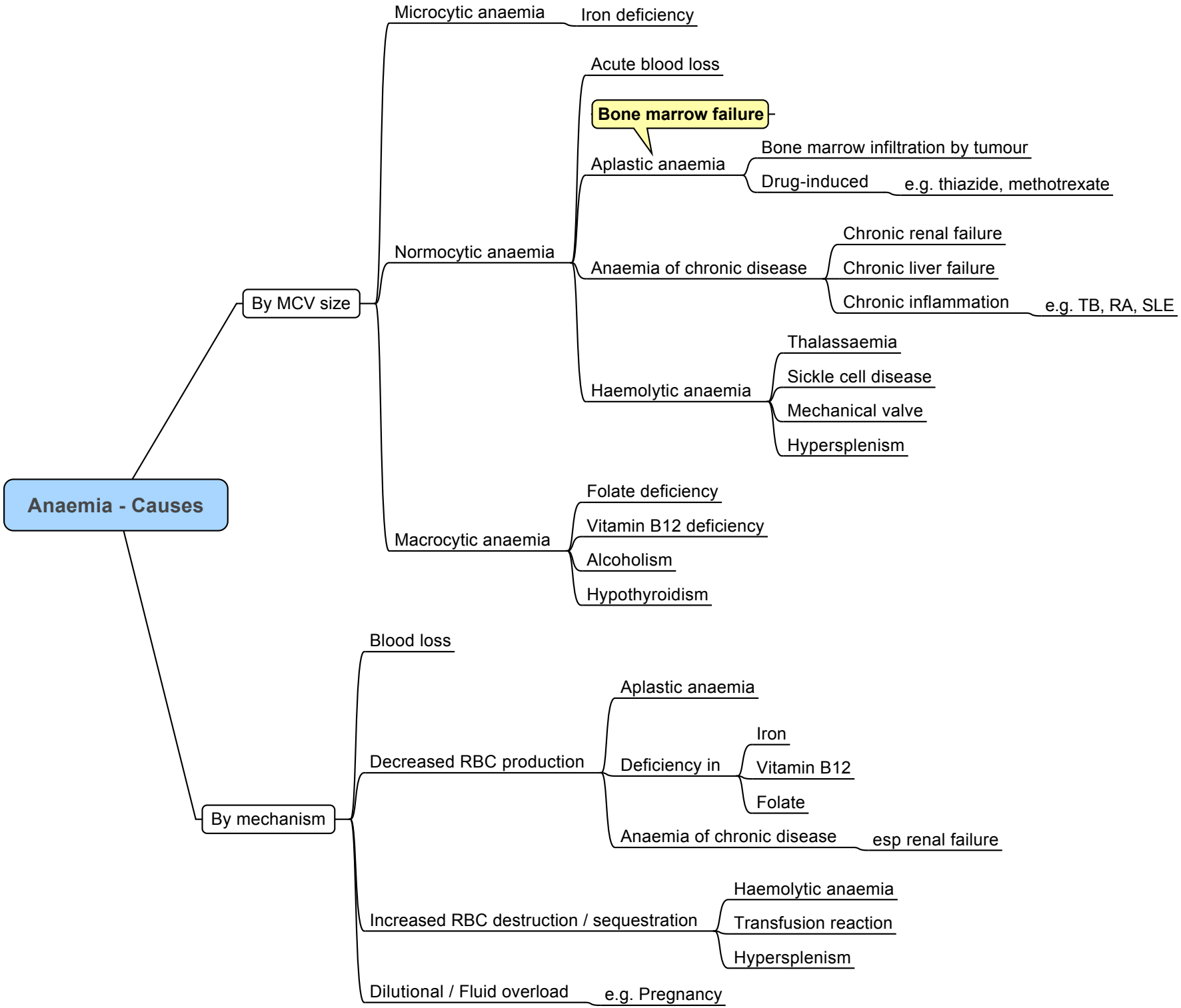


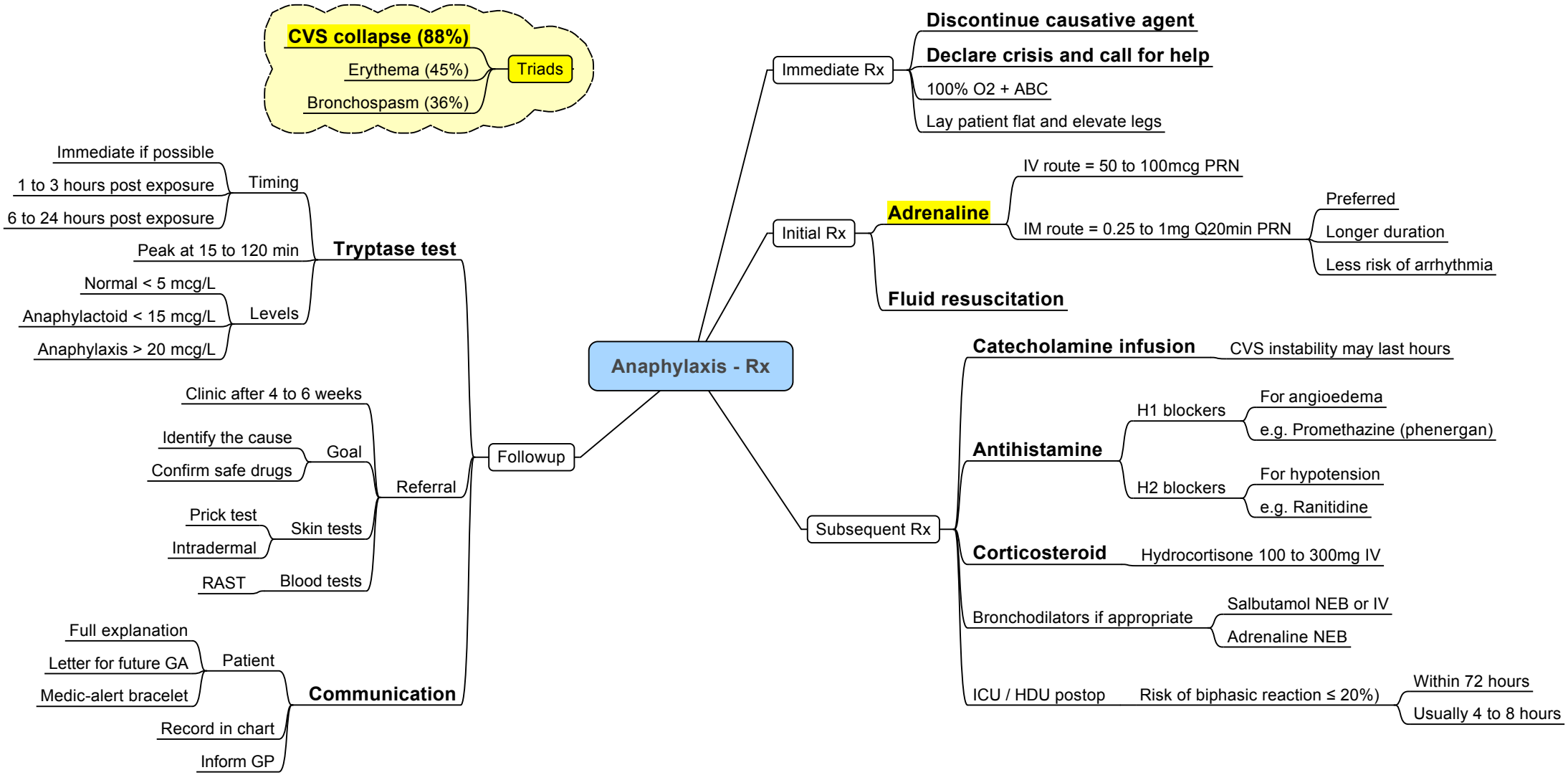
For any given diagnosis / condition, consider:

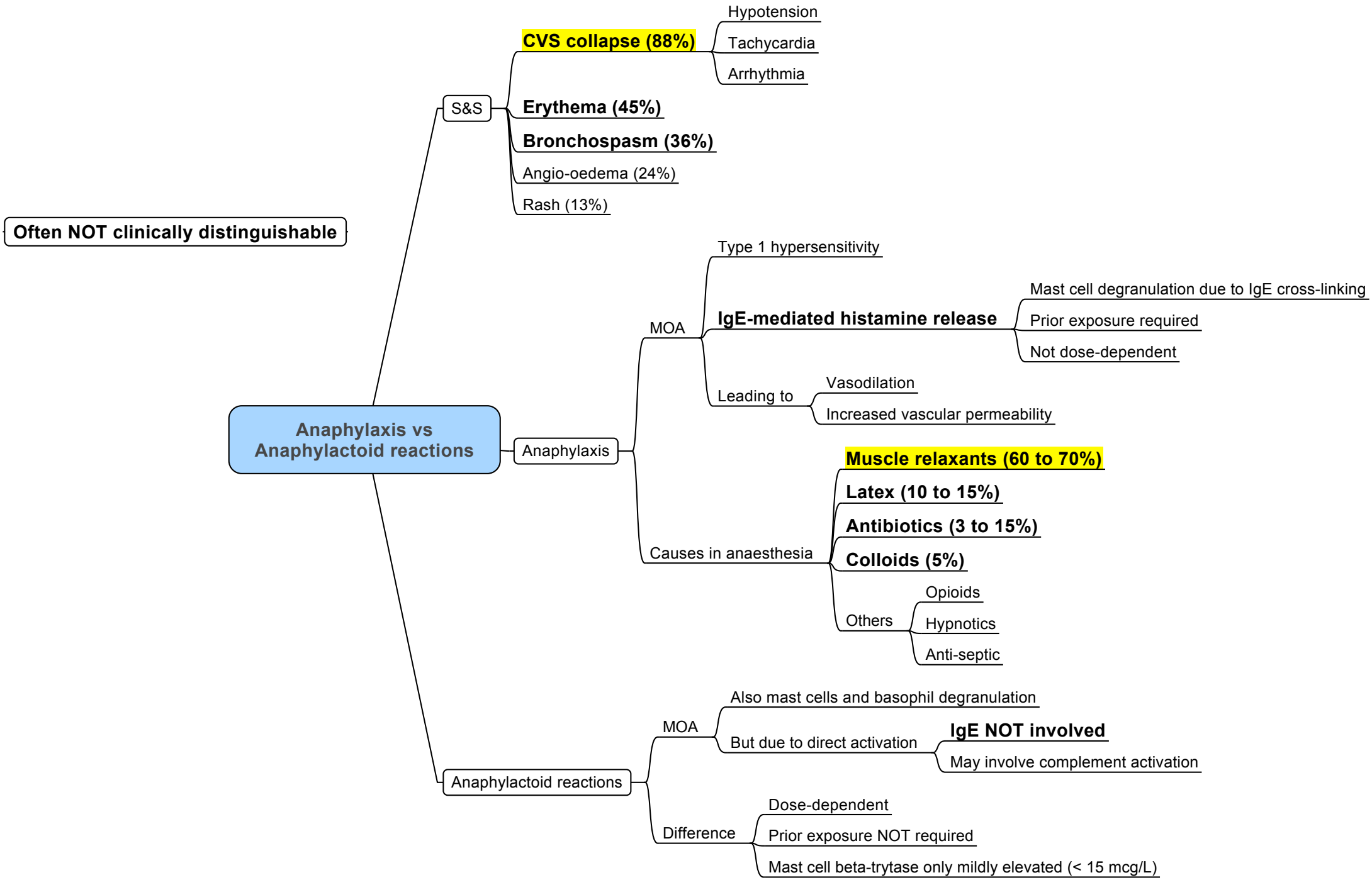
1. What caused it? (Aetiology)
2. What is the treatment? (Rx)
3. What complications does it have?
4. What other conditions are associated with it?
5. Do any of the above (including the condition itself) impact on the anaesthetic management?
6. How much physiological reserve does the patient have?

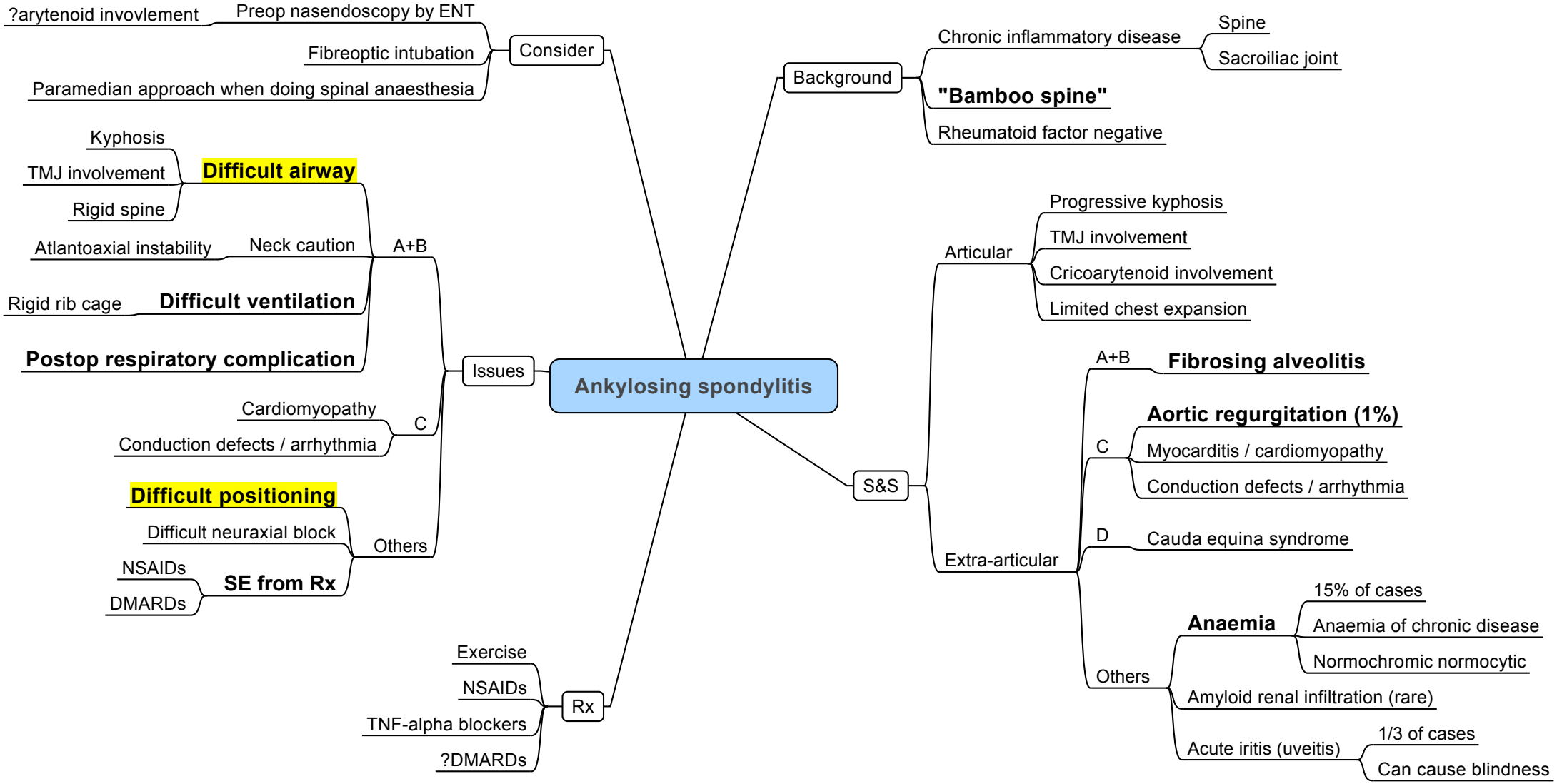

Amyotrophic lateral sclerosis (ALS)
Basics
One of the motor neuron diseases
Incurable
Death from respiratory failure usually within 3 years of diagnosis
PP
Unknown aetiology
Progressive degeneration
Upper motor neurons (UMN)
Lower motor neurons (LMNs)
Characterised by
Amyotrophy
Muscle atrophy
Eventually affects all skeletal muscles
Lateral sclerosis
Gliosis of the corticospinal tract
Does NOT affect
Ocular muscles
Bladder
Bowel
Sensation
Mentation
S&S
Initially asymmetrical and involves distal extremities
Eventually axial and bulbar weakness
Includes
Weakness
UMN signs
Spasticity
Hyperactive reflexes
LMN signs
Atrophy
Fasciculation
Usually of the hand
Issues
If bulbar involvement
Aspiration risk
Impaired ability to communicate
Respiratory impairment / failure
Autonomic dysfunction
Denervation hypersensitivity
Muscle weakness
Goals
NMBD
Avoid GA if possible
Aspiration prophylaxis
Avoid suxamethonium
Reduced dose for non-depolarising NMBDs
Maintain preload / hydration
Anticipate prolonged postop ventilation
FVC and anaesthetic risk
FVC > 50% → Low risk
FVC 30 to 50% → Moderate risk
FVC < 30% → High risk

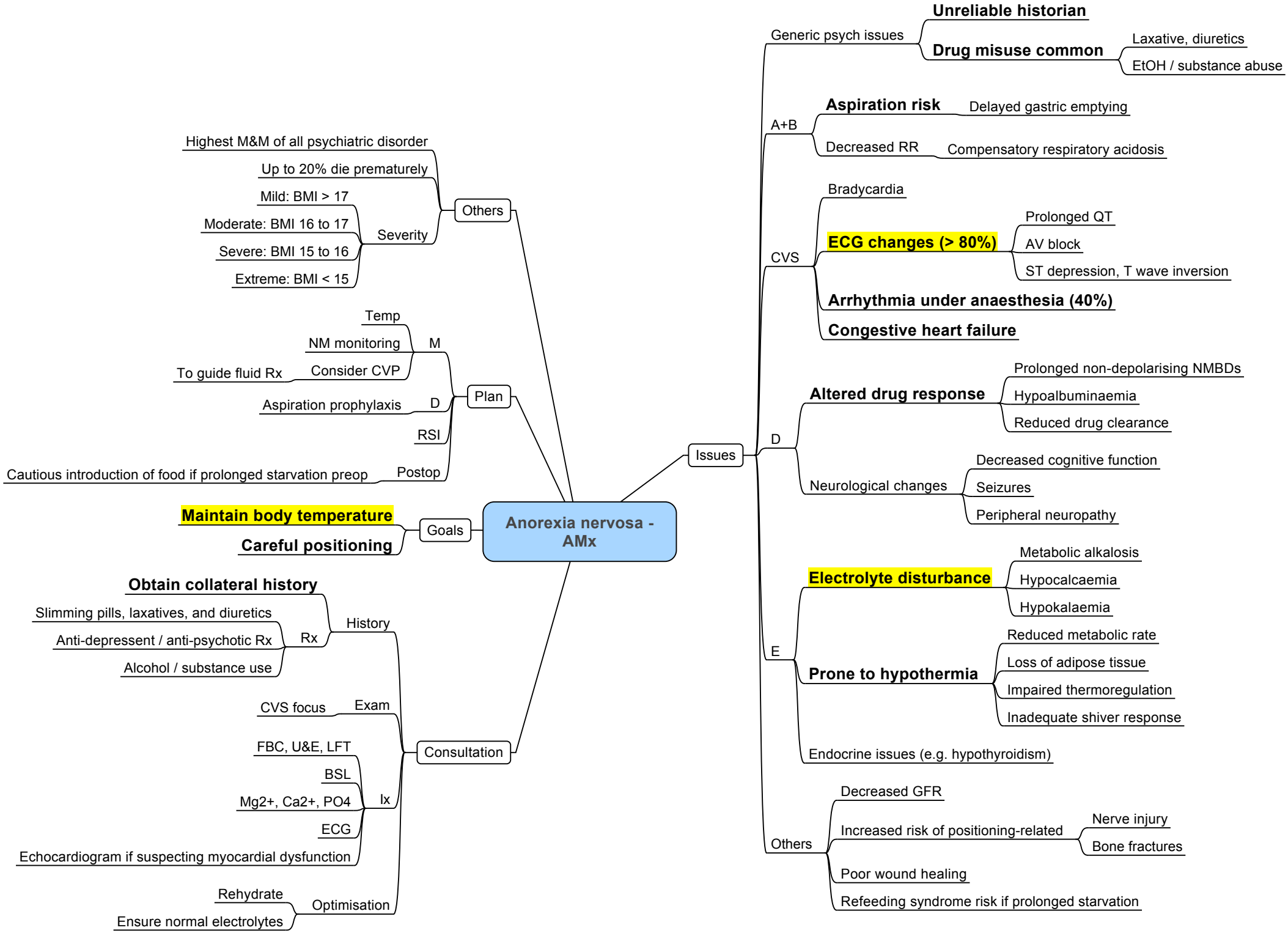

Anaemia - AMx
Consultation
History
PC
Underlying cause (if known)
Family Hx of anaemia
S&S
Fatigue
Dyspnoea on exertion
Pallor
Other PMHx
Co-existing CVS or respiratory diseases
Functional capacity
Rx
Alcohol
Drugs that causes bone marrow suppression
Antiplatelet Rx
Anticoagulation Rx
Ix
FBC
Iron studies (including ferritin)
CRP and renal function
Group and hold
Consent for transfusion
?Jehovah's Witness
Discuss risks of periop transfusion
Optimisation
Increase Hb
Treat underlying cause
e.g. folate, iron, vitamin B12
Haematology referral if unknown causes
Nephrology referral if chronic renal disease
If iron deficiency
Oral iron Rx
If no clear reason, gastroenterology referral
Acceptable preop Hb depends on
Co-existing diseases
Expected intraop blood loss
Time course (acute or chronic)
Issues
Preop anaemia is independently associated with increased risk of M&M
Increased likelihood of RBC transfusion
Plan
MADE
M
Temp
D
Consider
Consider cross-matched RBC
Tranexamic acid
ε-aminocaproic acid
E
Fluid warmer and force warm air blanket
Consider cell saver
Postop
Consider supplemental O2
Effective analgesia
Others
Ferritin
< 15 mcg/L → Diagnostic of iron deficiency
15 to 30 mcg/L → Highly suggestive
30 to 100 mcg/L
CRP raised → ?Iron deficiency
CRP normal → Look for other causes
> 100 mcg/L → Look for other causes
Transfusion
Should not be dictated by Hb trigger alone
Rarely indicated if Hb ≥ 100 g/L
Goals
Maximise O2 delivery
Minimise increases in O2 requirement
Minimise sympathetic response
Effective analgesia
Avoid shivering
Anti-pyrexia if appropriate
Minimise bleeding
Prevent hypothermia
Patient positioning
Reduce venous pressure at surgical site
Consider
Permissive hypotension
i.e. MAP 50 to 60 mmHg
Radical prostatectomy
Major joint replacement
Intraop cell salvage
Acute normovolaemic haemodilution
Post op cell salvage in
Cardiac surgery
Total knee arthroplasty
Minimise RBC transfusion

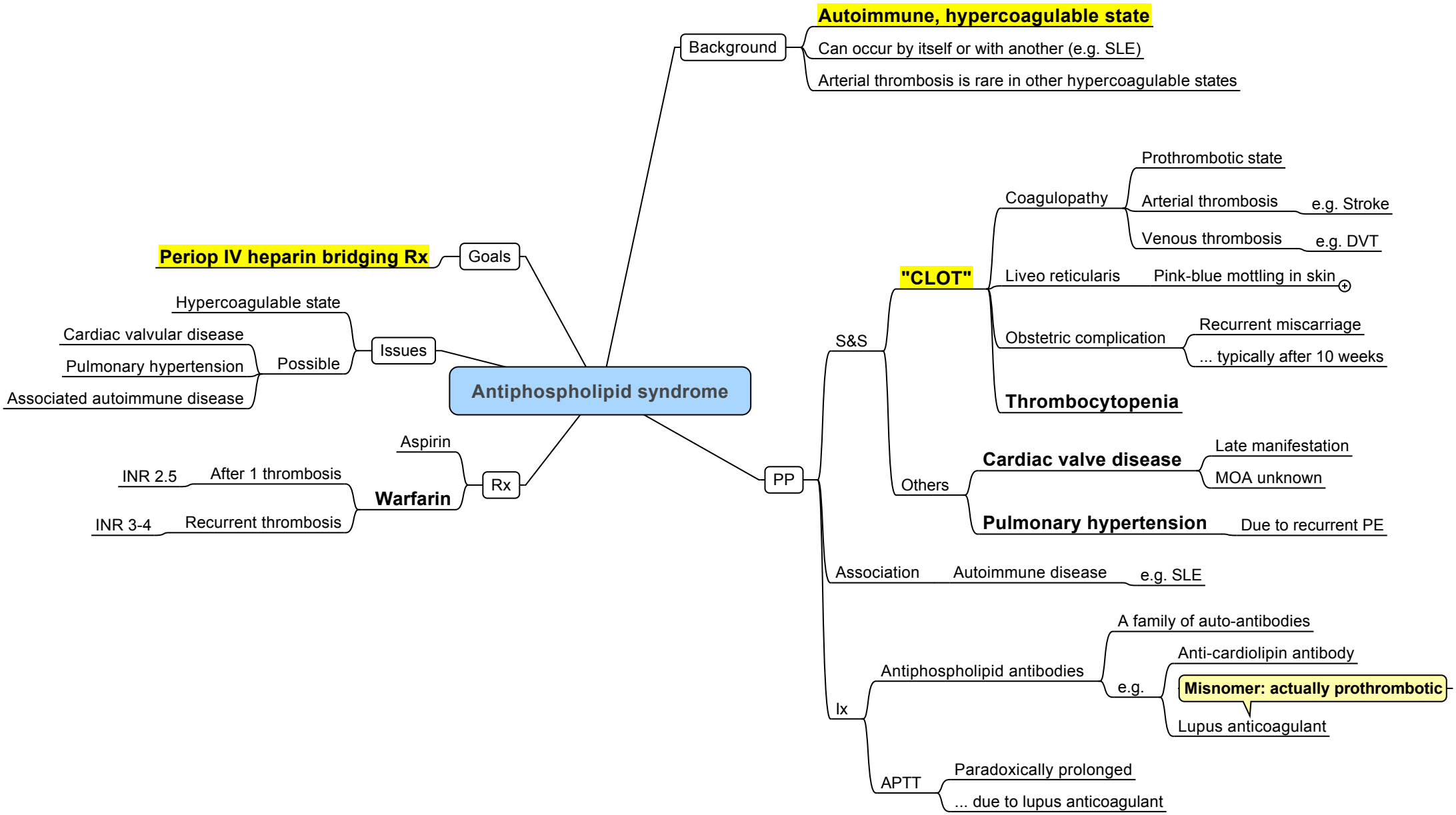

Anaemia - Causes
By MCV size
Microcytic anaemia
Iron deficiency
Normocytic anaemia
Acute blood loss
Bone marrow failure
Aplastic anaemia
Bone marrow infiltration by tumour
Drug-induced
e.g. thiazide, methotrexate
Anaemia of chronic disease
Chronic renal failure
Chronic liver failure
Chronic inflammation
e.g. TB, RA, SLE
Haemolytic anaemia
Thalassaemia
Sickle cell disease
Mechanical valve
Hypersplenism
Macrocytic anaemia
Folate deficiency
Vitamin B12 deficiency
Alcoholism
Hypothyroidism
By mechanism
Blood loss
Decreased RBC production
Aplastic anaemia
Deficiency in
Iron
Vitamin B12
Folate
Anaemia of chronic disease
esp renal failure
Increased RBC destruction / sequestration
Haemolytic anaemia
Transfusion reaction
Hypersplenism
Dilutional / Fluid overload
e.g. Pregnancy

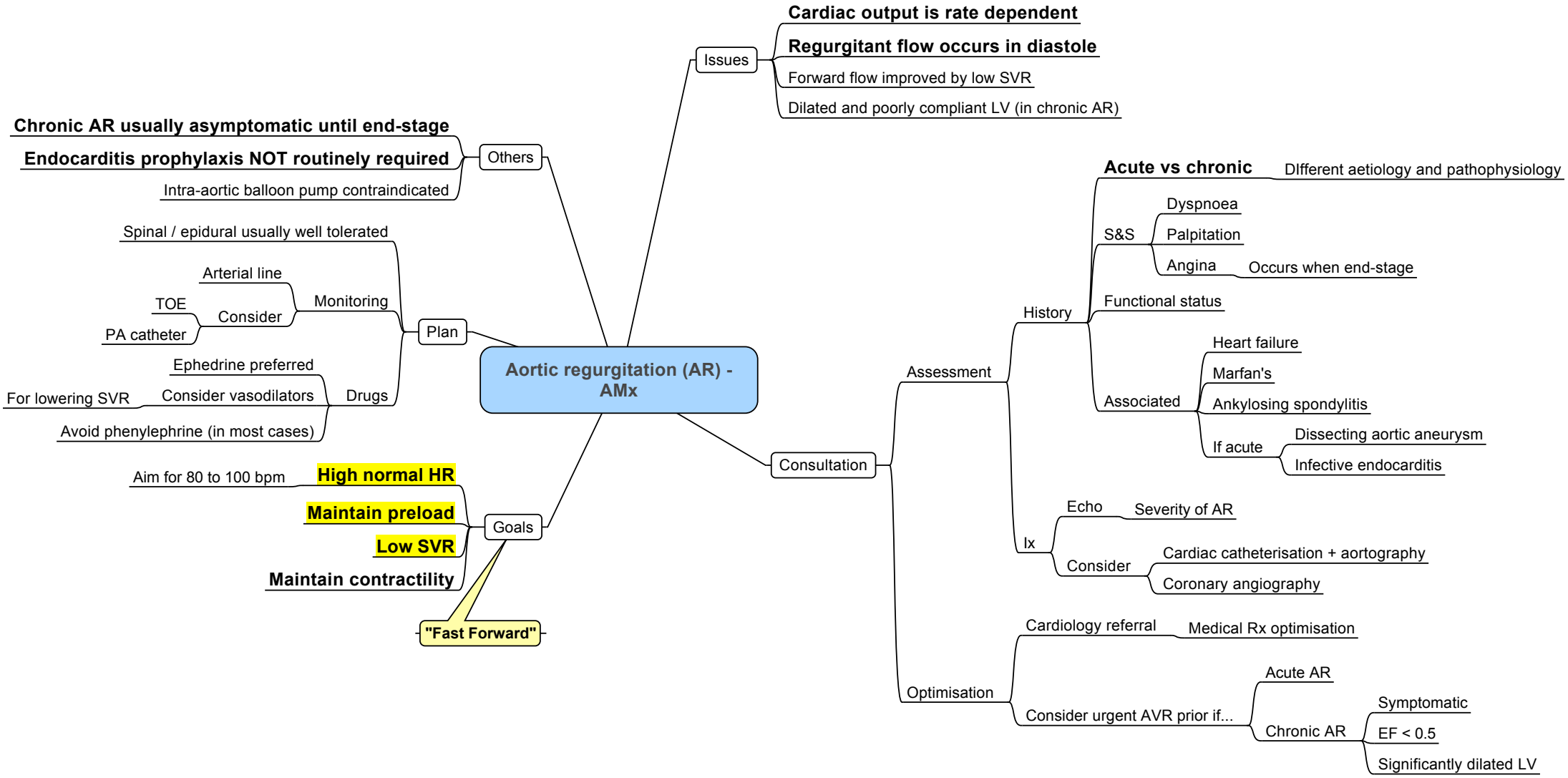

Anaphylaxis - Rx
Triads
CVS collapse (88%)
Erythema (45%)
Bronchospasm (36%)
Immediate Rx
Discontinue causative agent
Declare crisis and call for help
100% O2 + ABC
Lay patient flat and elevate legs
Initial Rx
Adrenaline
IV route = 50 to 100mcg PRN
IM route = 0.25 to 1mg Q20min PRN
Preferred
Longer duration
Less risk of arrhythmia
Fluid resuscitation
Subsequent Rx
Catecholamine infusion
CVS instability may last hours
Antihistamine
H1 blockers
For angioedema
e.g. Promethazine (phenergan)
H2 blockers
For hypotension
e.g. Ranitidine
Corticosteroid
Hydrocortisone 100 to 300mg IV
Bronchodilators if appropriate
Salbutamol NEB or IV
Adrenaline NEB
ICU / HDU postop
Risk of biphasic reaction ≤ 20%)
Within 72 hours
Usually 4 to 8 hours
Followup
Tryptase test
Timing
Immediate if possible
1 to 3 hours post exposure
6 to 24 hours post exposure
Peak at 15 to 120 min
Levels
Normal < 5 mcg/L
Anaphylactoid < 15 mcg/L
Anaphylaxis > 20 mcg/L
Referral
Clinic after 4 to 6 weeks
Goal
Identify the cause
Confirm safe drugs
Skin tests
Prick test
Intradermal
Blood tests
RAST
Communication
Patient
Full explanation
Letter for future GA
Medic-alert bracelet
Record in chart
Inform GP

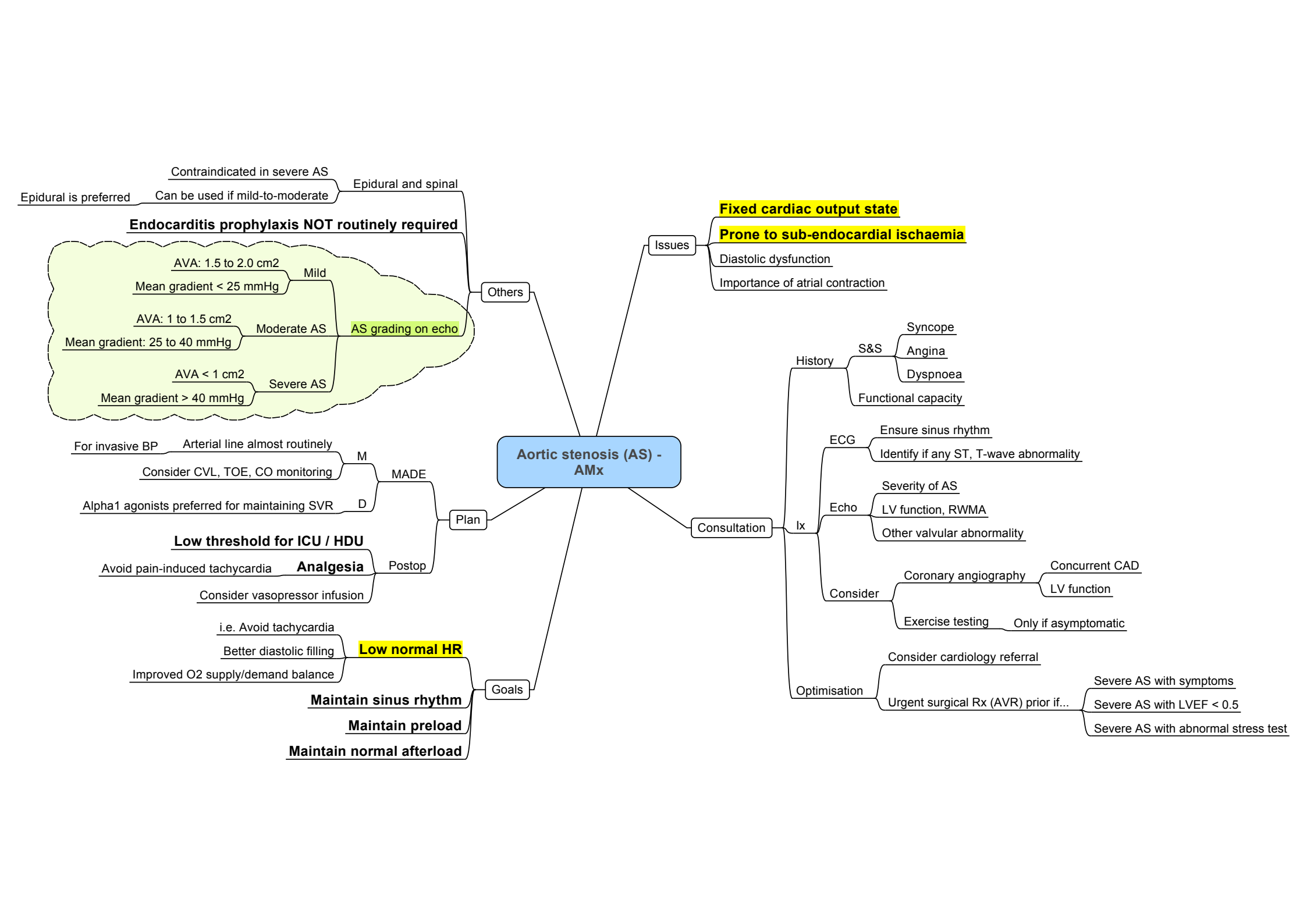

Anaphylaxis vs Anaphylactoid reactions
Often NOT clinically distinguishable
S&S
CVS collapse (88%)
Hypotension
Tachycardia
Arrhythmia
Erythema (45%)
Bronchospasm (36%)
Angio-oedema (24%)
Rash (13%)
Anaphylaxis
MOA
Type 1 hypersensitivity
IgE-mediated histamine release
Mast cell degranulation due to IgE cross-linking
Prior exposure required
Not dose-dependent
Leading to
Vasodilation
Increased vascular permeability
Causes in anaesthesia
Muscle relaxants (60 to 70%)
Latex (10 to 15%)
Antibiotics (3 to 15%)
Colloids (5%)
Others
Opioids
Hypnotics
Anti-septic
Anaphylactoid reactions
MOA
Also mast cells and basophil degranulation
But due to direct activation
IgE NOT involved
May involve complement activation
Difference
Dose-dependent
Prior exposure NOT required
Mast cell beta-trytase only mildly elevated (< 15 mcg/L)

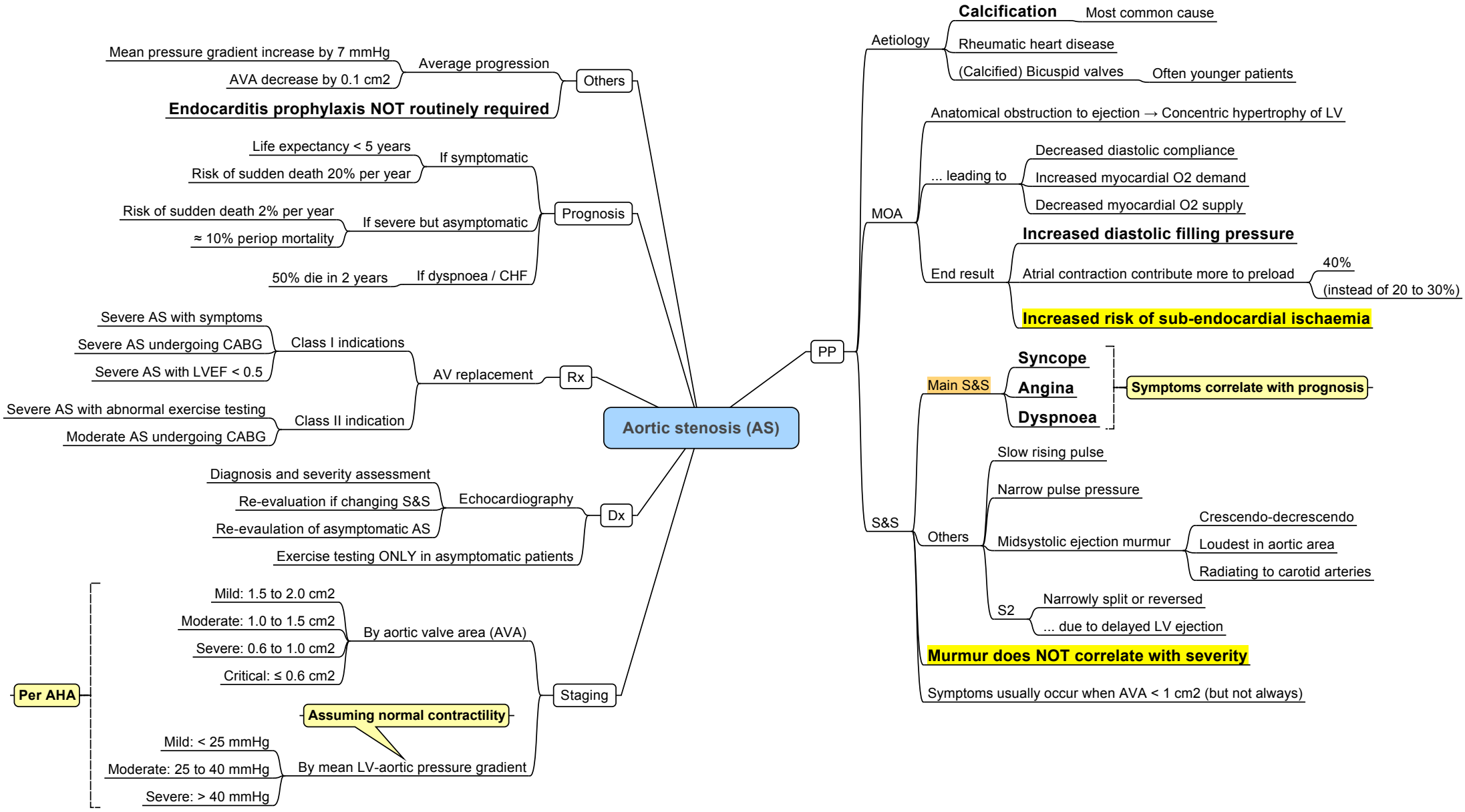

Ankylosing spondylitis
Background
Chronic inflammatory disease
Spine
Sacroiliac joint
"Bamboo spine"
Rheumatoid factor negative
S&S
Articular
Progressive kyphosis
TMJ involvement
Cricoarytenoid involvement
Limited chest expansion
Extra-articular
A+B
Fibrosing alveolitis
C
Aortic regurgitation (1%)
Myocarditis / cardiomyopathy
Conduction defects / arrhythmia
D
Cauda equina syndrome
Others
Anaemia
15% of cases
Anaemia of chronic disease
Normochromic normocytic
Amyloid renal infiltration (rare)
Acute iritis (uveitis)
1/3 of cases
Can cause blindness
Issues
A+B
Difficult airway
Kyphosis
TMJ involvement
Rigid spine
Neck caution
Atlantoaxial instability
Difficult ventilation
Rigid rib cage
Postop respiratory complication
C
Cardiomyopathy
Conduction defects / arrhythmia
Others
Difficult positioning
Difficult neuraxial block
SE from Rx
NSAIDs
DMARDs
Consider
Preop nasendoscopy by ENT
?arytenoid invovlement
Fibreoptic intubation
Paramedian approach when doing spinal anaesthesia
Rx
Exercise
NSAIDs
TNF-alpha blockers
?DMARDs


Anorexia nervosa - AMx
Issues
Generic psych issues
Unreliable historian
Drug misuse common
Laxative, diuretics
EtOH / substance abuse
A+B
Aspiration risk
Delayed gastric emptying
Decreased RR
Compensatory respiratory acidosis
CVS
Bradycardia
ECG changes (> 80%)
Prolonged QT
AV block
ST depression, T wave inversion
Arrhythmia under anaesthesia (40%)
Congestive heart failure
D
Altered drug response
Prolonged non-depolarising NMBDs
Hypoalbuminaemia
Reduced drug clearance
Neurological changes
Decreased cognitive function
Seizures
Peripheral neuropathy
E
Electrolyte disturbance
Metabolic alkalosis
Hypocalcaemia
Hypokalaemia
Prone to hypothermia
Reduced metabolic rate
Loss of adipose tissue
Impaired thermoregulation
Inadequate shiver response
Endocrine issues (e.g. hypothyroidism)
Others
Decreased GFR
Increased risk of positioning-related
Nerve injury
Bone fractures
Poor wound healing
Refeeding syndrome risk if prolonged starvation
Consultation
History
Rx
Obtain collateral history
Slimming pills, laxatives, and diuretics
Anti-depressent / anti-psychotic Rx
Alcohol / substance use
Exam
CVS focus
Ix
FBC, U&E, LFT
BSL
Mg2+, Ca2+, PO4
ECG
Echocardiogram if suspecting myocardial dysfunction
Optimisation
Rehydrate
Ensure normal electrolytes
Goals
Maintain body temperature
Careful positioning
Plan
M
Temp
NM monitoring
Consider CVP
To guide fluid Rx
D
Aspiration prophylaxis
RSI
Postop
Cautious introduction of food if prolonged starvation preop
Others
Severity
Highest M&M of all psychiatric disorder
Up to 20% die prematurely
Mild: BMI > 17
Moderate: BMI 16 to 17
Severe: BMI 15 to 16
Extreme: BMI < 15

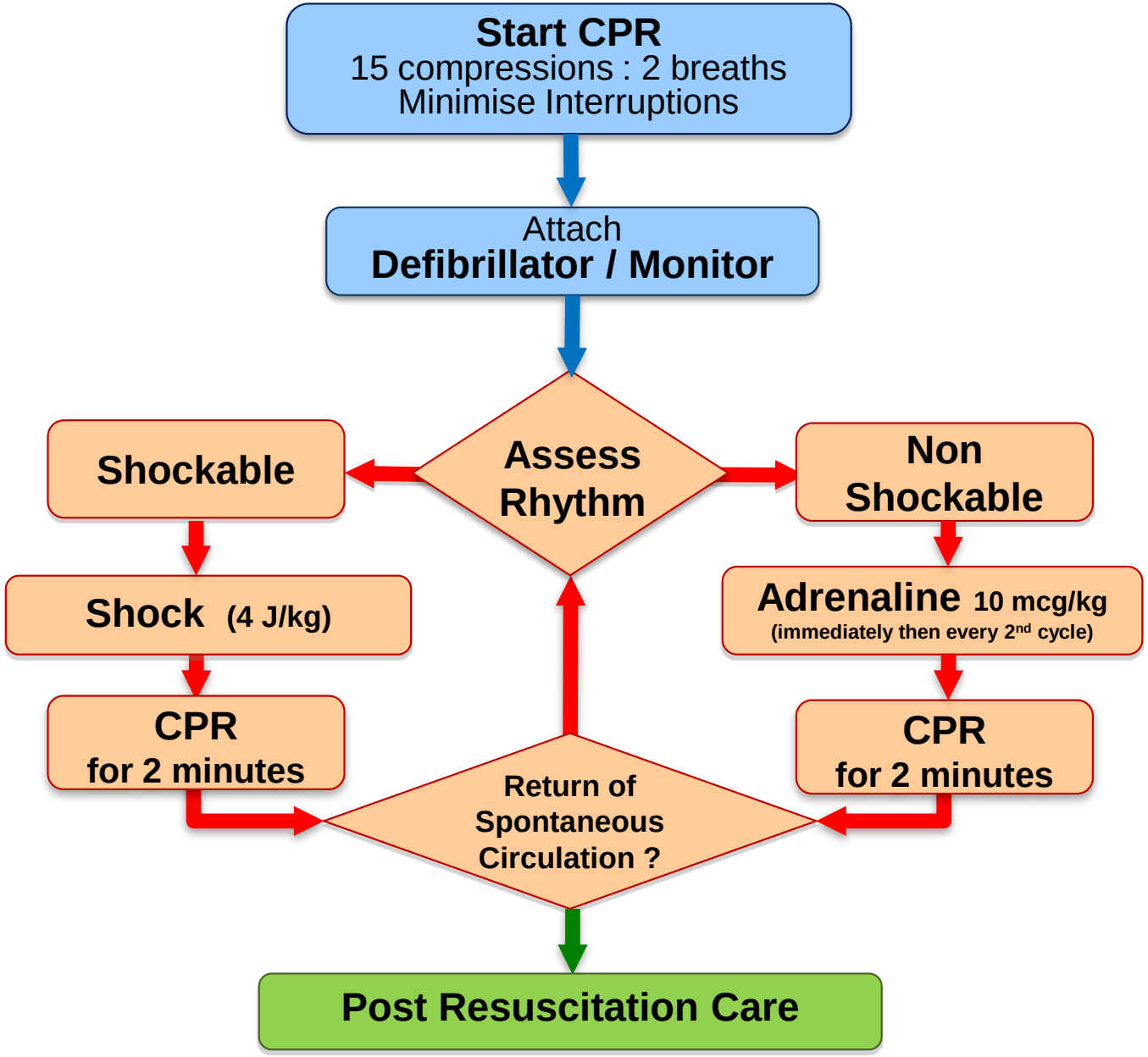

Antiphospholipid syndrome
Background
Autoimmune, hypercoagulable state
Can occur by itself or with another (e.g. SLE)
Arterial thrombosis is rare in other hypercoagulable states
PP
S&S
"CLOT"
Coagulopathy
Prothrombotic state
Arterial thrombosis
e.g. Stroke
Venous thrombosis
e.g. DVT
Liveo reticularis
Pink-blue mottling in skin
Obstetric complication
Recurrent miscarriage
... typically after 10 weeks
Thrombocytopenia
Others
Cardiac valve disease
Late manifestation
MOA unknown
Pulmonary hypertension
Due to recurrent PE
Association
Autoimmune disease
e.g. SLE
Ix
Antiphospholipid antibodies
A family of auto-antibodies
e.g.
Anti-cardiolipin antibody
Misnomer: actually prothrombotic
Lupus anticoagulant
APTT
Paradoxically prolonged
... due to lupus anticoagulant
Rx
Aspirin
Warfarin
After 1 thrombosis
INR 2.5
Recurrent thrombosis
INR 3-4
Goals
Periop IV heparin bridging Rx
Issues
Possible
Hypercoagulable state
Cardiac valvular disease
Pulmonary hypertension
Associated autoimmune disease


Aortic regurgitation (AR) - AMx
Issues
Cardiac output is rate dependent
Regurgitant flow occurs in diastole
Forward flow improved by low SVR
Dilated and poorly compliant LV (in chronic AR)
Others
Chronic AR usually asymptomatic until end-stage
Endocarditis prophylaxis NOT routinely required
Intra-aortic balloon pump contraindicated
Plan
Spinal / epidural usually well tolerated
Monitoring
Arterial line
Consider
TOE
PA catheter
Drugs
Ephedrine preferred
Consider vasodilators
For lowering SVR
Avoid phenylephrine (in most cases)
Goals
Aim for 80 to 100 bpm
High normal HR
Maintain preload
Low SVR
Maintain contractility
"Fast Forward"
Consultation
Assessment
History
Acute vs chronic
DIfferent aetiology and pathophysiology
S&S
Dyspnoea
Palpitation
Angina
Occurs when end-stage
Functional status
Associated
Heart failure
Marfan's
Ankylosing spondylitis
If acute
Dissecting aortic aneurysm
Infective endocarditis
Ix
Echo
Severity of AR
Consider
Cardiac catheterisation + aortography
Coronary angiography
Optimisation
Cardiology referral
Medical Rx optimisation
Consider urgent AVR prior if...
Acute AR
Chronic AR
Symptomatic
EF < 0.5
Significantly dilated LV


Aortic stenosis (AS) - AMx
Consultation
History
S&S
Syncope
Angina
Dyspnoea
Functional capacity
Ix
ECG
Ensure sinus rhythm
Identify if any ST, T-wave abnormality
Echo
Severity of AS
LV function, RWMA
Other valvular abnormality
Consider
Coronary angiography
Concurrent CAD
LV function
Exercise testing
Only if asymptomatic
Optimisation
Consider cardiology referral
Urgent surgical Rx (AVR) prior if...
Severe AS with symptoms
Severe AS with LVEF < 0.5
Severe AS with abnormal stress test
Issues
Fixed cardiac output state
Prone to sub-endocardial ischaemia
Diastolic dysfunction
Importance of atrial contraction
Others
Epidural and spinal
Contraindicated in severe AS
Can be used if mild-to-moderate
Epidural is preferred
Endocarditis prophylaxis NOT routinely required
AS grading on echo
Mild
AVA: 1.5 to 2.0 cm2
Mean gradient < 25 mmHg
Moderate AS
AVA: 1 to 1.5 cm2
Mean gradient: 25 to 40 mmHg
Severe AS
AVA < 1 cm2
Mean gradient > 40 mmHg
Plan
MADE
M
Arterial line almost routinely
For invasive BP
Consider CVL, TOE, CO monitoring
D
Alpha1 agonists preferred for maintaining SVR
Postop
Low threshold for ICU / HDU
Analgesia
Avoid pain-induced tachycardia
Consider vasopressor infusion
Goals
Low normal HR
i.e. Avoid tachycardia
Better diastolic filling
Improved O2 supply/demand balance
Maintain sinus rhythm
Maintain preload
Maintain normal afterload


Aortic stenosis (AS)
PP
Aetiology
Calcification
Most common cause
Rheumatic heart disease
(Calcified) Bicuspid valves
Often younger patients
MOA
Anatomical obstruction to ejection → Concentric hypertrophy of LV
... leading to
Decreased diastolic compliance
Increased myocardial O2 demand
Decreased myocardial O2 supply
End result
Increased diastolic filling pressure
Atrial contraction contribute more to preload
40%
(instead of 20 to 30%)
Increased risk of sub-endocardial ischaemia
S&S
Main S&S
Syncope
Angina
Dyspnoea
Symptoms correlate with prognosis
Others
Slow rising pulse
Narrow pulse pressure
Midsystolic ejection murmur
Crescendo-decrescendo
Loudest in aortic area
Radiating to carotid arteries
S2
Narrowly split or reversed
... due to delayed LV ejection
Murmur does NOT correlate with severity
Symptoms usually occur when AVA < 1 cm2 (but not always)
Rx
AV replacement
Class I indications
Severe AS with symptoms
Severe AS undergoing CABG
Severe AS with LVEF < 0.5
Class II indication
Severe AS with abnormal exercise testing
Moderate AS undergoing CABG
Dx
Echocardiography
Diagnosis and severity assessment
Re-evaluation if changing S&S
Re-evaulation of asymptomatic AS
Exercise testing ONLY in asymptomatic patients
Staging
Per AHA
By aortic valve area (AVA)
Mild: 1.5 to 2.0 cm2
Moderate: 1.0 to 1.5 cm2
Severe: 0.6 to 1.0 cm2
Critical: ≤ 0.6 cm2
Assuming normal contractility
By mean LV-aortic pressure gradient
Mild: < 25 mmHg
Moderate: 25 to 40 mmHg
Severe: > 40 mmHg
Prognosis
If symptomatic
Life expectancy < 5 years
Risk of sudden death 20% per year
If severe but asymptomatic
Risk of sudden death 2% per year
≈ 10% periop mortality
If dyspnoea / CHF
50% die in 2 years
Others
Average progression
Mean pressure gradient increase by 7 mmHg
AVA decrease by 0.1 cm2
Endocarditis prophylaxis NOT routinely required

Advanced Life Support for Infants and Children



During CPR

Airway adjuncts (LMA / ETT)
Oxygen
Waveform capnography
IV / IO access
Plan actions before interrupting compressions
(e.g. charge manual defibrillator to 4 J/kg)
Drugs
Shockable
* Adrenaline 10 mcg/kg after 2nd shock (then every 2nd cycle)
* Amiodarone 5mg/kg after 3rd shock
Non Shockable
* Adrenaline 10 mcg/kg immediately (then every 2nd cycle)

Consider and Correct

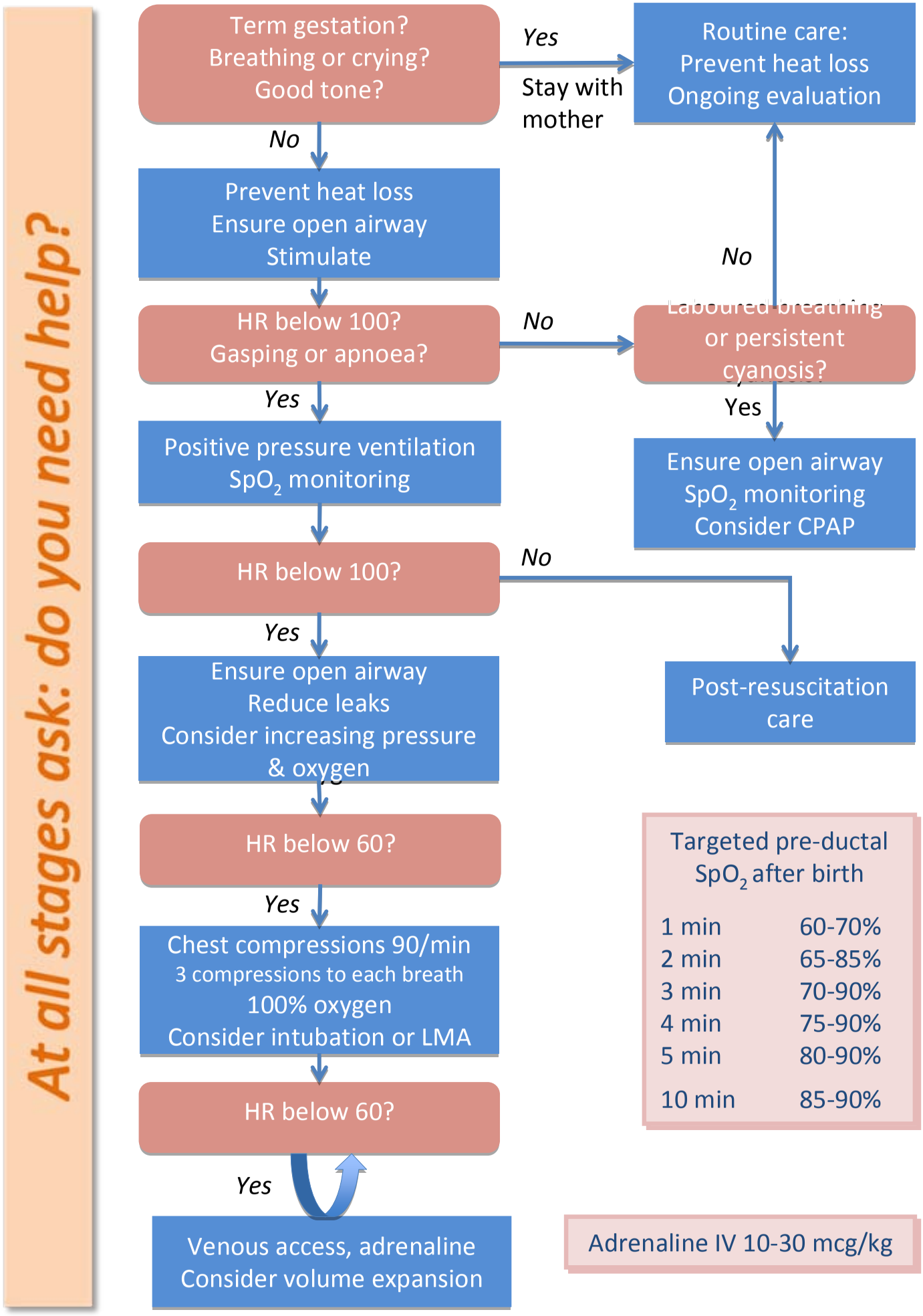
Hypoxia
Hypovolaemia
Hyper / hypokalaemia / metabolic disorders
Hypothermia / hyperthermia
Tension pneumothorax
Tamponade
Toxins
Thrombosis (pulmonary / coronary)

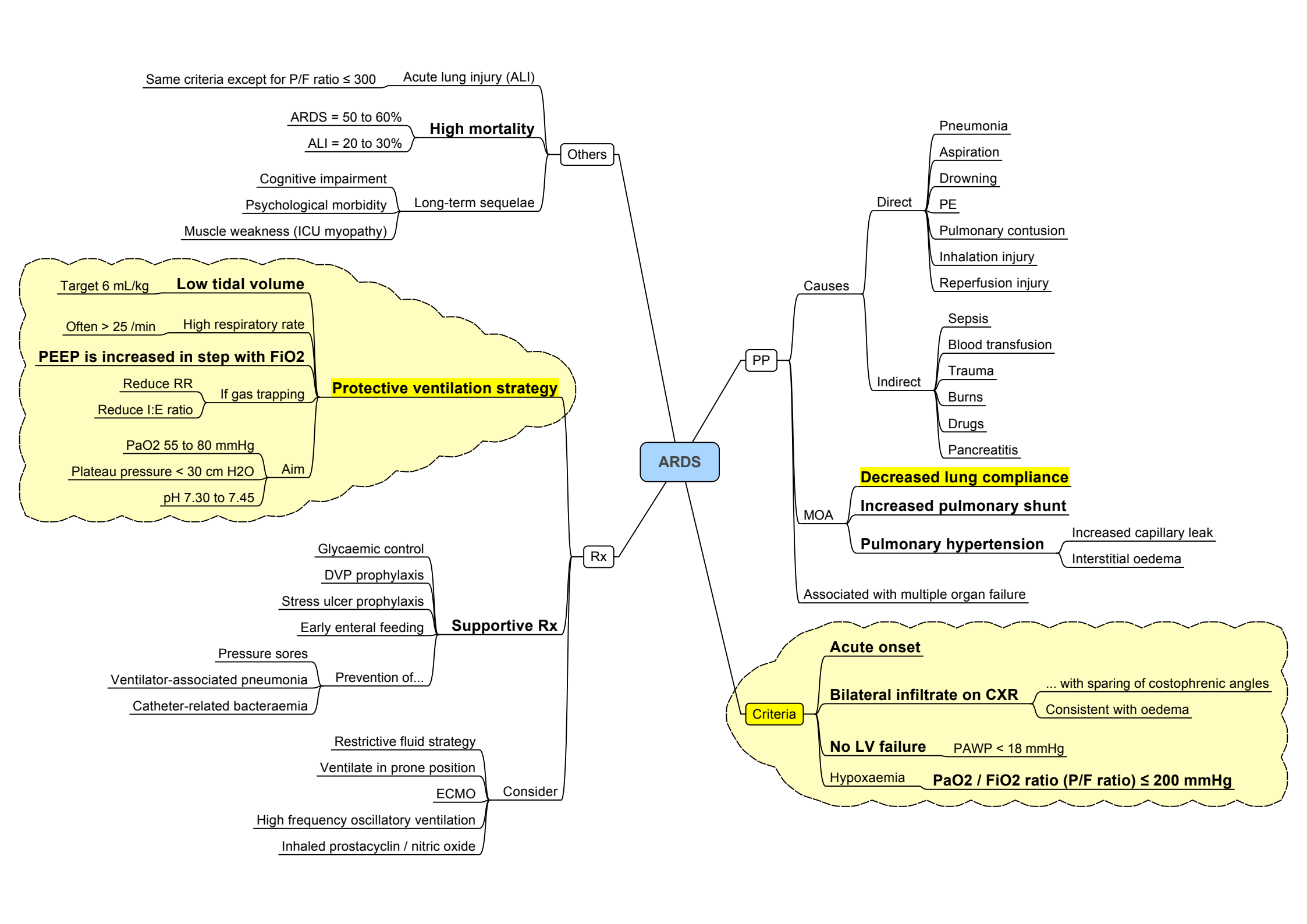
Post Resuscitation Care

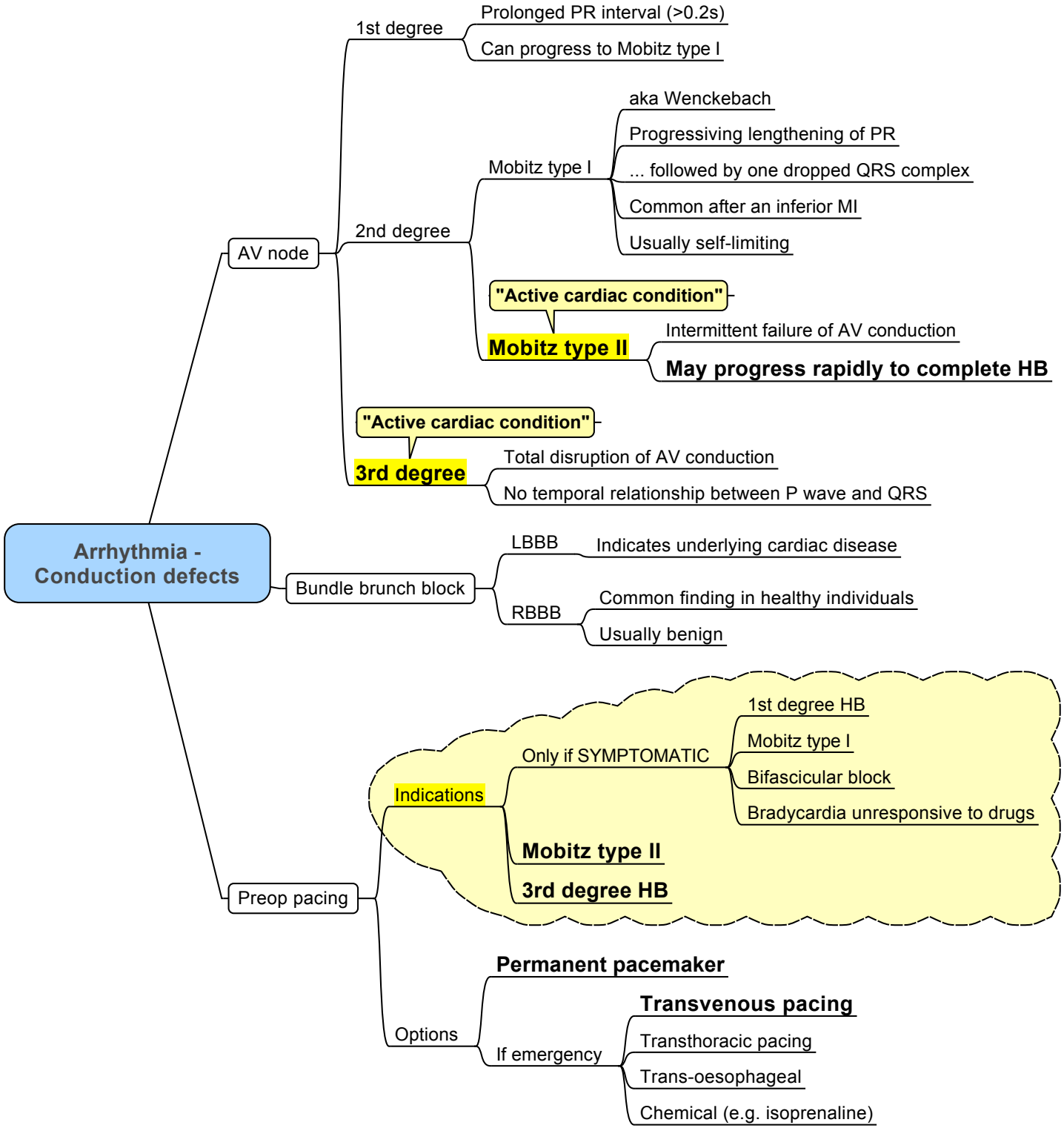
Re-evaluate ABCDE
12 lead ECG
Treat precipitating causes
Re-evaluate oxygenation and ventilation
Temperature control (cool)

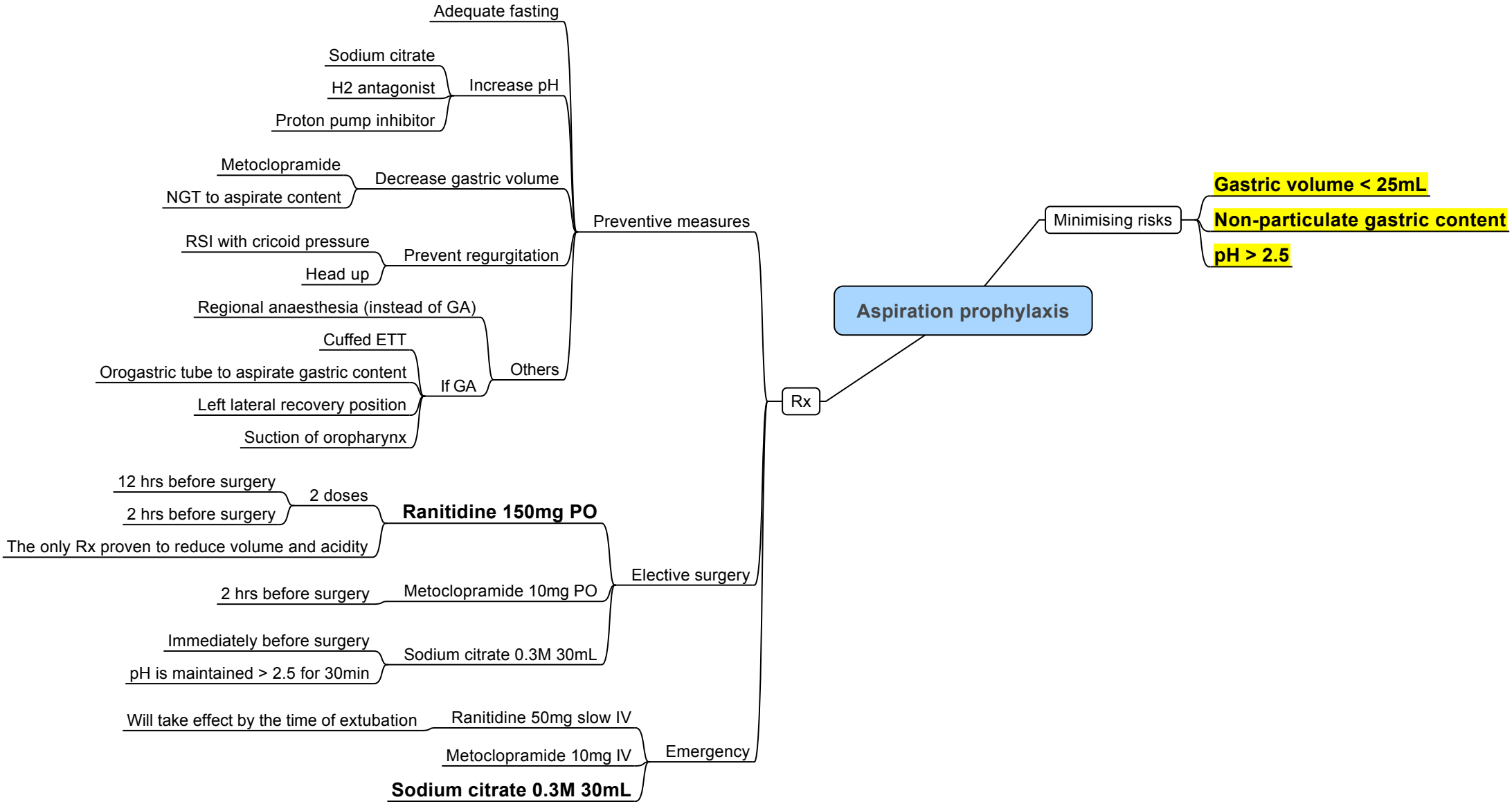
December 2010

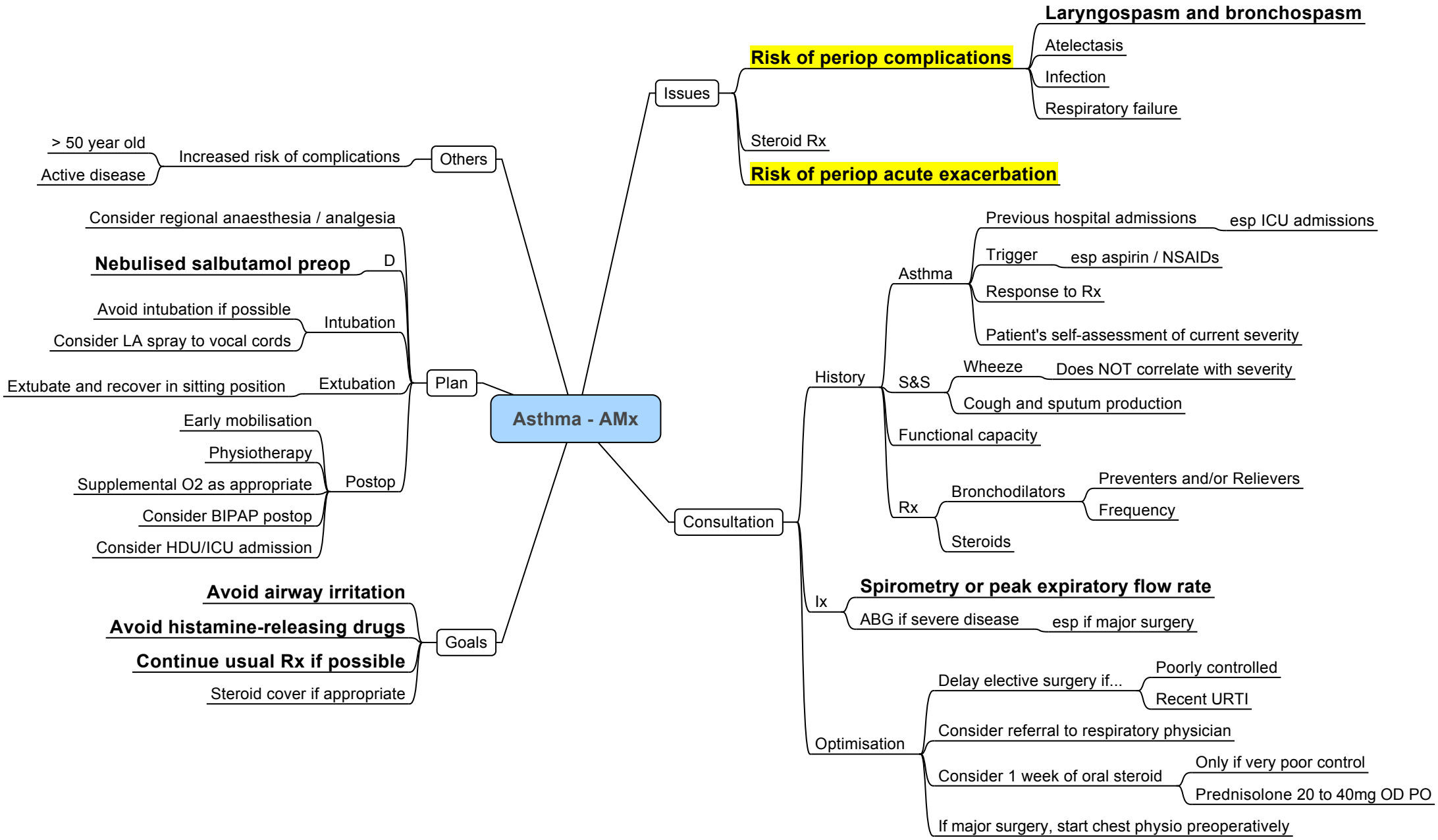
Newborn Life Support

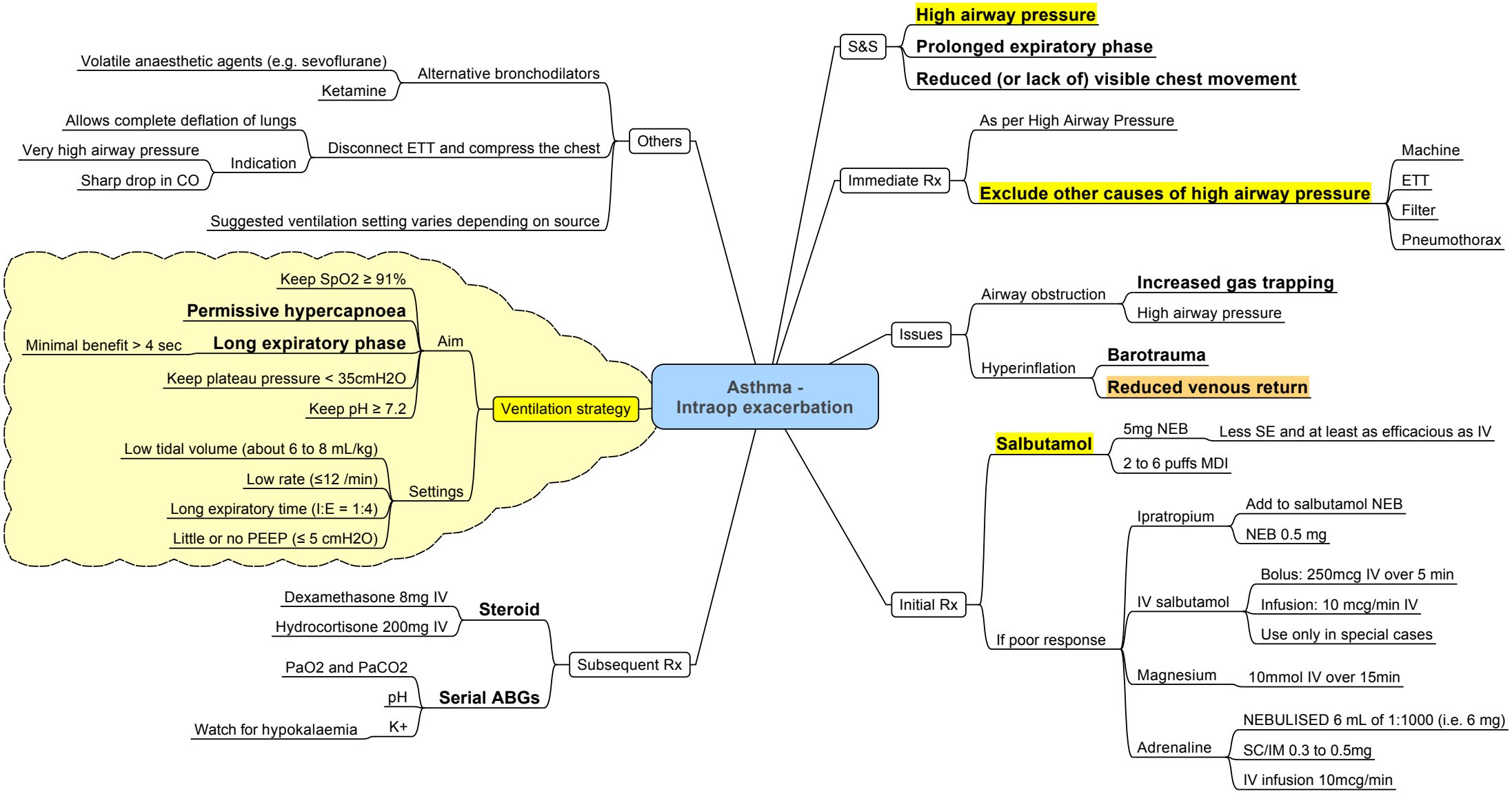


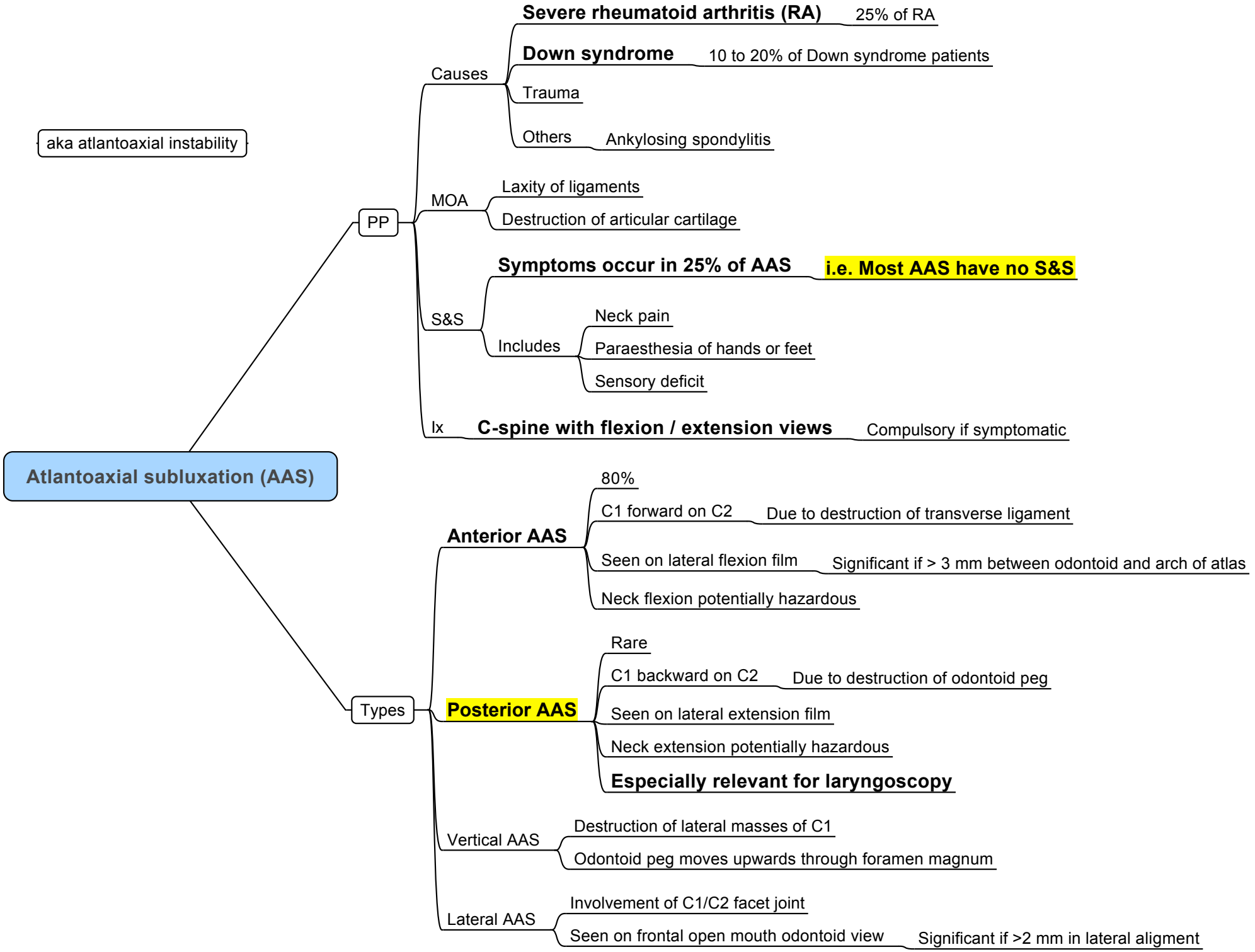

ARDS
PP
Causes
Direct
Pneumonia
Aspiration
Drowning
PE
Pulmonary contusion
Inhalation injury
Reperfusion injury
Indirect
Sepsis
Blood transfusion
Trauma
Burns
Drugs
Pancreatitis
MOA
Decreased lung compliance
Increased pulmonary shunt
Pulmonary hypertension
Increased capillary leak
Interstitial oedema
Associated with multiple organ failure
Criteria
Acute onset
Bilateral infiltrate on CXR
... with sparing of costophrenic angles
Consistent with oedema
No LV failure
PAWP < 18 mmHg
Hypoxaemia
PaO2 / FiO2 ratio (P/F ratio) ≤ 200 mmHg
Rx
Protective ventilation strategy
Low tidal volume
Target 6 mL/kg
High respiratory rate
Often > 25 /min
PEEP is increased in step with FiO2
If gas trapping
Reduce RR
Reduce I:E ratio
Aim
PaO2 55 to 80 mmHg
Plateau pressure < 30 cm H2O
pH 7.30 to 7.45
Supportive Rx
Glycaemic control
DVP prophylaxis
Stress ulcer prophylaxis
Early enteral feeding
Prevention of...
Pressure sores
Ventilator-associated pneumonia
Catheter-related bacteraemia
Consider
Restrictive fluid strategy
Ventilate in prone position
ECMO
High frequency oscillatory ventilation
Inhaled prostacyclin / nitric oxide
Others
Acute lung injury (ALI)
Same criteria except for P/F ratio ≤ 300
High mortality
ARDS = 50 to 60%
ALI = 20 to 30%
Long-term sequelae
Cognitive impairment
Psychological morbidity
Muscle weakness (ICU myopathy)

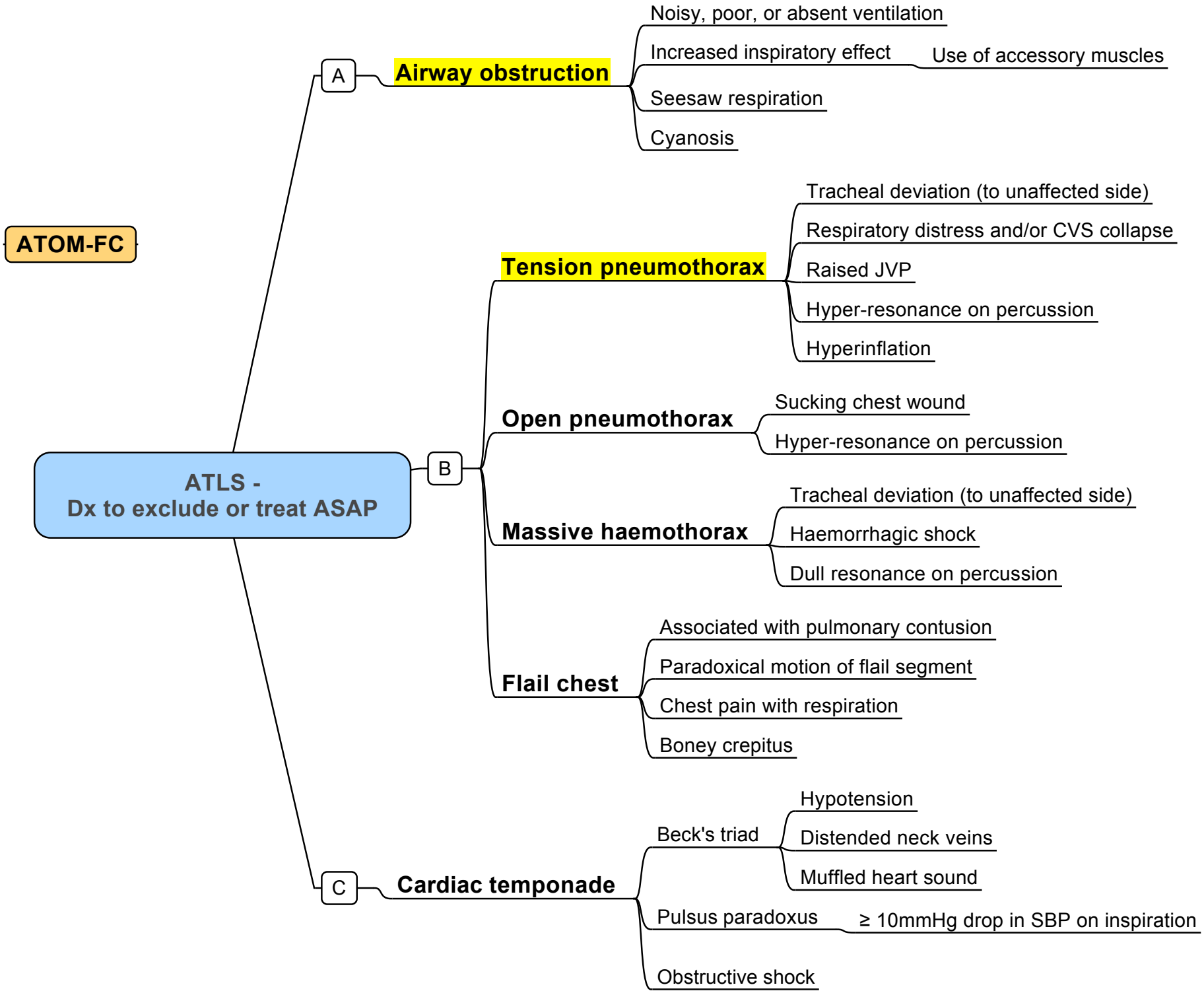

Arrhythmia - Conduction defects
AV node
1st degree
Prolonged PR interval (>0.2s)
Can progress to Mobitz type I
2nd degree
Mobitz type I
aka Wenckebach
Progressiving lengthening of PR
... followed by one dropped QRS complex
Common after an inferior MI
Usually self-limiting
"Active cardiac condition"
Mobitz type II
Intermittent failure of AV conduction
May progress rapidly to complete HB
"Active cardiac condition"
3rd degree
Total disruption of AV conduction
No temporal relationship between P wave and QRS
Bundle brunch block
LBBB
Indicates underlying cardiac disease
RBBB
Common finding in healthy individuals
Usually benign
Preop pacing
Indications
Only if SYMPTOMATIC
1st degree HB
Mobitz type I
Bifascicular block
Bradycardia unresponsive to drugs
Mobitz type II
3rd degree HB
Options
Permanent pacemaker
If emergency
Transvenous pacing
Transthoracic pacing
Trans-oesophageal
Chemical (e.g. isoprenaline)

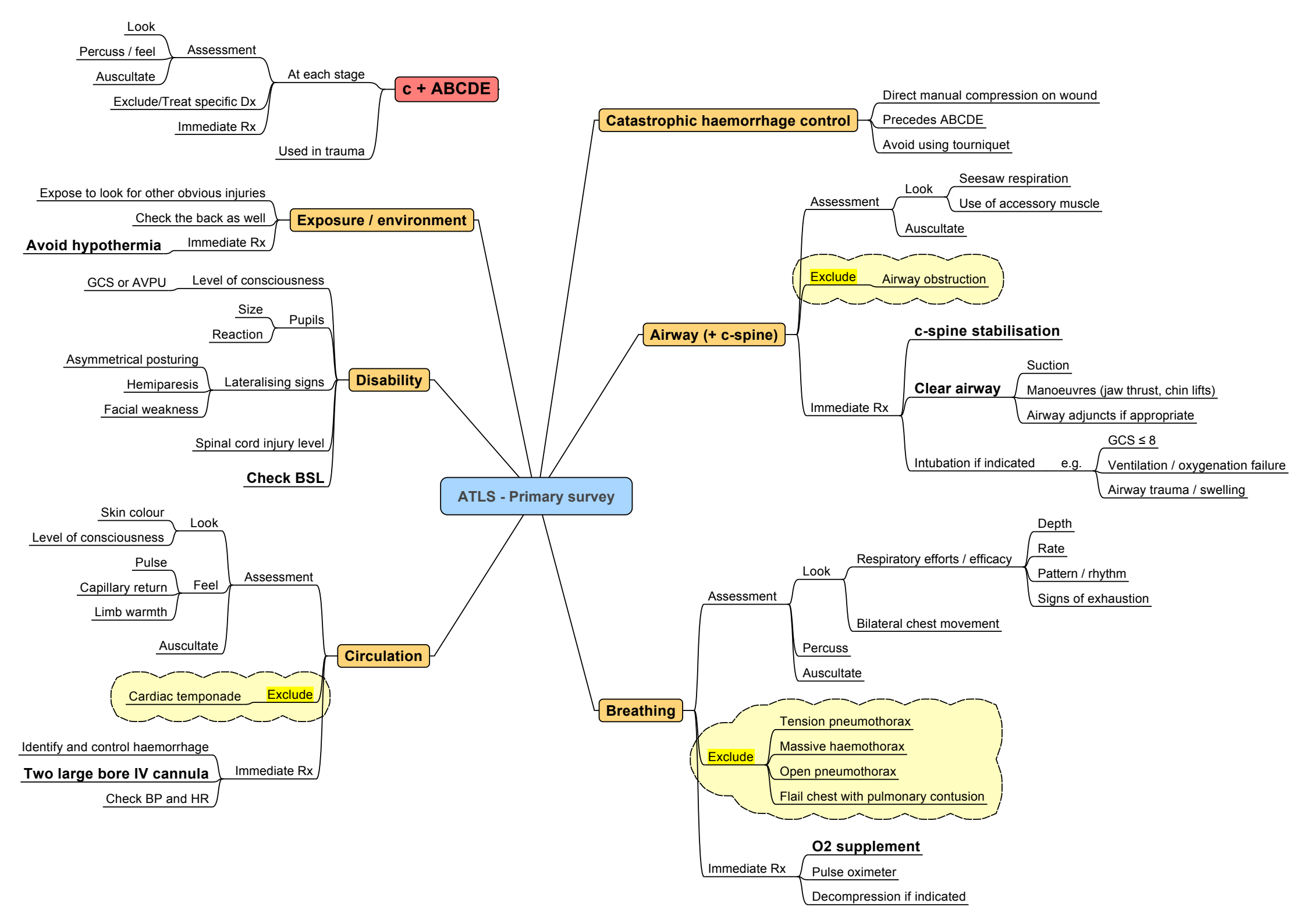

Aspiration prophylaxis
Minimising risks
Gastric volume < 25mL
Non-particulate gastric content
pH > 2.5
Rx
Preventive measures
Adequate fasting
Increase pH
Sodium citrate
H2 antagonist
Proton pump inhibitor
Decrease gastric volume
Metoclopramide
NGT to aspirate content
Prevent regurgitation
RSI with cricoid pressure
Head up
Others
Regional anaesthesia (instead of GA)
If GA
Cuffed ETT
Orogastric tube to aspirate gastric content
Left lateral recovery position
Suction of oropharynx
Elective surgery
Ranitidine 150mg PO
2 doses
12 hrs before surgery
2 hrs before surgery
The only Rx proven to reduce volume and acidity
Metoclopramide 10mg PO
2 hrs before surgery
Sodium citrate 0.3M 30mL
Immediately before surgery
pH is maintained > 2.5 for 30min
Emergency
Ranitidine 50mg slow IV
Will take effect by the time of extubation
Metoclopramide 10mg IV
Sodium citrate 0.3M 30mL

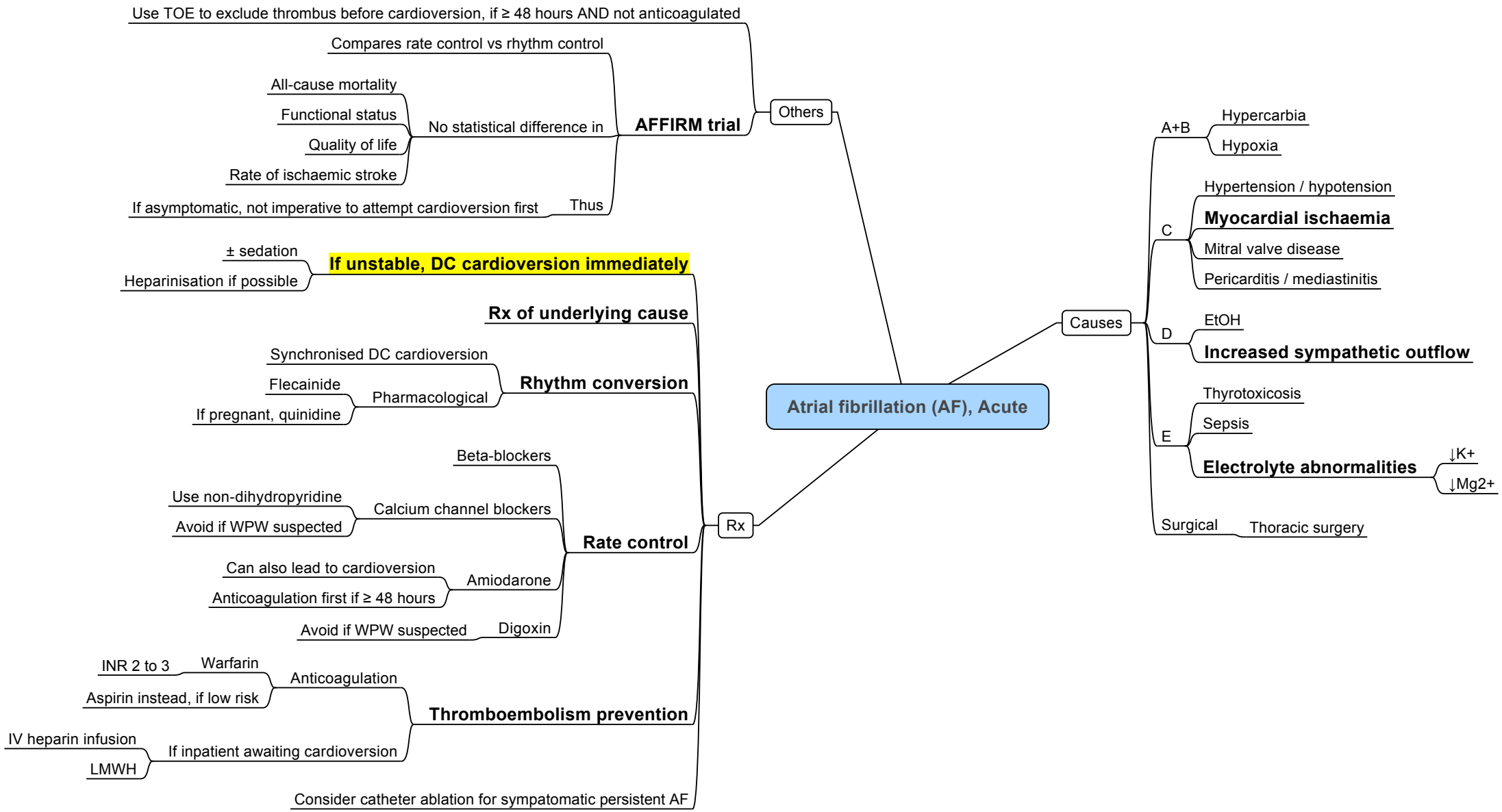

Asthma - AMx
Consultation
History
Asthma
Previous hospital admissions
esp ICU admissions
Trigger
esp aspirin / NSAIDs
Response to Rx
Patient's self-assessment of current severity
S&S
Wheeze
Does NOT correlate with severity
Cough and sputum production
Functional capacity
Rx
Bronchodilators
Preventers and/or Relievers
Frequency
Steroids
Ix
Spirometry or peak expiratory flow rate
ABG if severe disease
esp if major surgery
Optimisation
Delay elective surgery if...
Poorly controlled
Recent URTI
Consider referral to respiratory physician
Consider 1 week of oral steroid
Only if very poor control
Prednisolone 20 to 40mg OD PO
If major surgery, start chest physio preoperatively
Issues
Risk of periop complications
Laryngospasm and bronchospasm
Atelectasis
Infection
Respiratory failure
Steroid Rx
Risk of periop acute exacerbation
Others
Increased risk of complications
> 50 year old
Active disease
Plan
D
Nebulised salbutamol preop
Consider regional anaesthesia / analgesia
Intubation
Avoid intubation if possible
Consider LA spray to vocal cords
Extubation
Extubate and recover in sitting position
Postop
Early mobilisation
Physiotherapy
Supplemental O2 as appropriate
Consider BIPAP postop
Consider HDU/ICU admission
Goals
Avoid airway irritation
Avoid histamine-releasing drugs
Continue usual Rx if possible
Steroid cover if appropriate

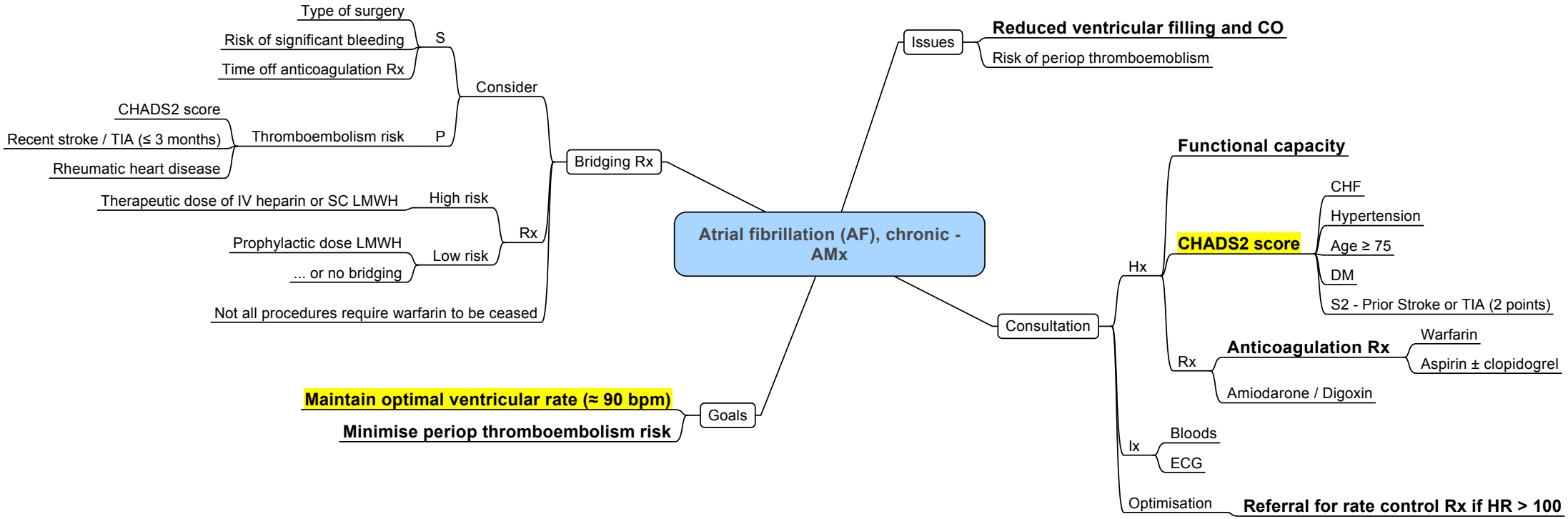

Asthma - Intraop exacerbation
S&S
High airway pressure
Prolonged expiratory phase
Reduced (or lack of) visible chest movement
Immediate Rx
As per High Airway Pressure
Exclude other causes of high airway pressure
Machine
ETT
Filter
Pneumothorax
Issues
Airway obstruction
Increased gas trapping
High airway pressure
Hyperinflation
Barotrauma
Reduced venous return
Initial Rx
Salbutamol
5mg NEB
Less SE and at least as efficacious as IV
2 to 6 puffs MDI
If poor response
Ipratropium
Add to salbutamol NEB
NEB 0.5 mg
IV salbutamol
Bolus: 250mcg IV over 5 min
Infusion: 10 mcg/min IV
Use only in special cases
Magnesium
10mmol IV over 15min
Adrenaline
NEBULISED 6 mL of 1:1000 (i.e. 6 mg)
SC/IM 0.3 to 0.5mg
IV infusion 10mcg/min
Subsequent Rx
Steroid
Dexamethasone 8mg IV
Hydrocortisone 200mg IV
Serial ABGs
PaO2 and PaCO2
pH
K+
Watch for hypokalaemia
Ventilation strategy
Aim
Keep SpO2 ≥ 91%
Permissive hypercapnoea
Long expiratory phase
Minimal benefit > 4 sec
Keep plateau pressure < 35cmH2O
Keep pH ≥ 7.2
Settings
Low tidal volume (about 6 to 8 mL/kg)
Low rate (≤12 /min)
Long expiratory time (I:E = 1:4)
Little or no PEEP (≤ 5 cmH2O)
Others
Alternative bronchodilators
Volatile anaesthetic agents (e.g. sevoflurane)
Ketamine
Disconnect ETT and compress the chest
Indication
Allows complete deflation of lungs
Very high airway pressure
Sharp drop in CO
Suggested ventilation setting varies depending on source

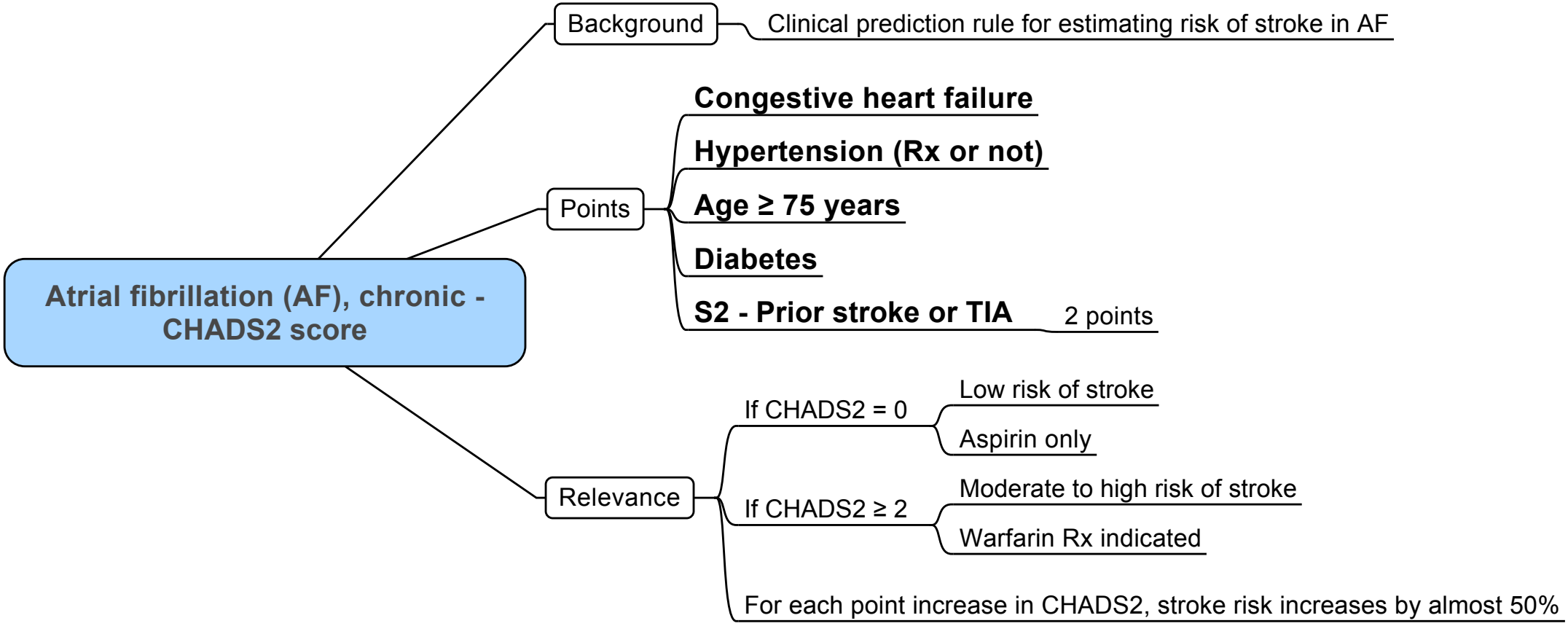

Atlantoaxial subluxation (AAS)
aka atlantoaxial instability
PP
Causes
Severe rheumatoid arthritis (RA)
25% of RA
Down syndrome
10 to 20% of Down syndrome patients
Trauma
Others
Ankylosing spondylitis
MOA
Laxity of ligaments
Destruction of articular cartilage
S&S
Symptoms occur in 25% of AAS
i.e. Most AAS have no S&S
Includes
Neck pain
Paraesthesia of hands or feet
Sensory deficit
Ix
C-spine with flexion / extension views
Compulsory if symptomatic
Types
Anterior AAS
80%
C1 forward on C2
Due to destruction of transverse ligament
Seen on lateral flexion film
Significant if > 3 mm between odontoid and arch of atlas
Neck flexion potentially hazardous
Posterior AAS
Rare
C1 backward on C2
Due to destruction of odontoid peg
Seen on lateral extension film
Neck extension potentially hazardous
Especially relevant for laryngoscopy
Vertical AAS
Destruction of lateral masses of C1
Odontoid peg moves upwards through foramen magnum
Lateral AAS
Involvement of C1/C2 facet joint
Seen on frontal open mouth odontoid view
Significant if >2 mm in lateral aligment

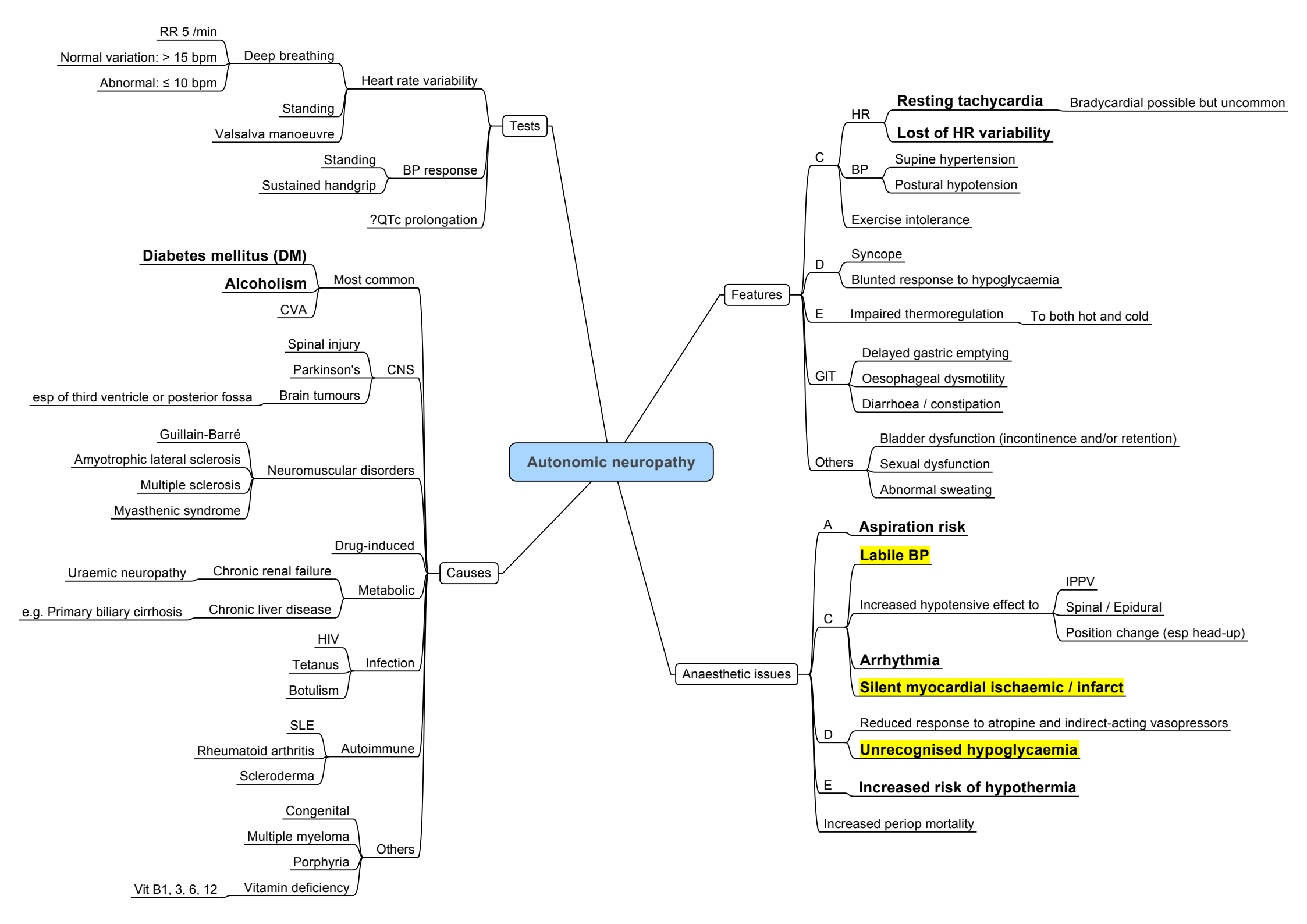

ATOM-FC
ATLS -
Dx to exclude or treat ASAP
A
Airway obstruction
Noisy, poor, or absent ventilation
Increased inspiratory effect
Use of accessory muscles
Seesaw respiration
Cyanosis
B
Tension pneumothorax
Tracheal deviation (to unaffected side)
Respiratory distress and/or CVS collapse
Raised JVP
Hyper-resonance on percussion
Hyperinflation
Open pneumothorax
Sucking chest wound
Hyper-resonance on percussion
Massive haemothorax
Tracheal deviation (to unaffected side)
Haemorrhagic shock
Dull resonance on percussion
Flail chest
Associated with pulmonary contusion
Paradoxical motion of flail segment
Chest pain with respiration
Boney crepitus
C
Cardiac temponade
Beck's triad
Hypotension
Distended neck veins
Muffled heart sound
Pulsus paradoxus
≥ 10mmHg drop in SBP on inspiration
Obstructive shock

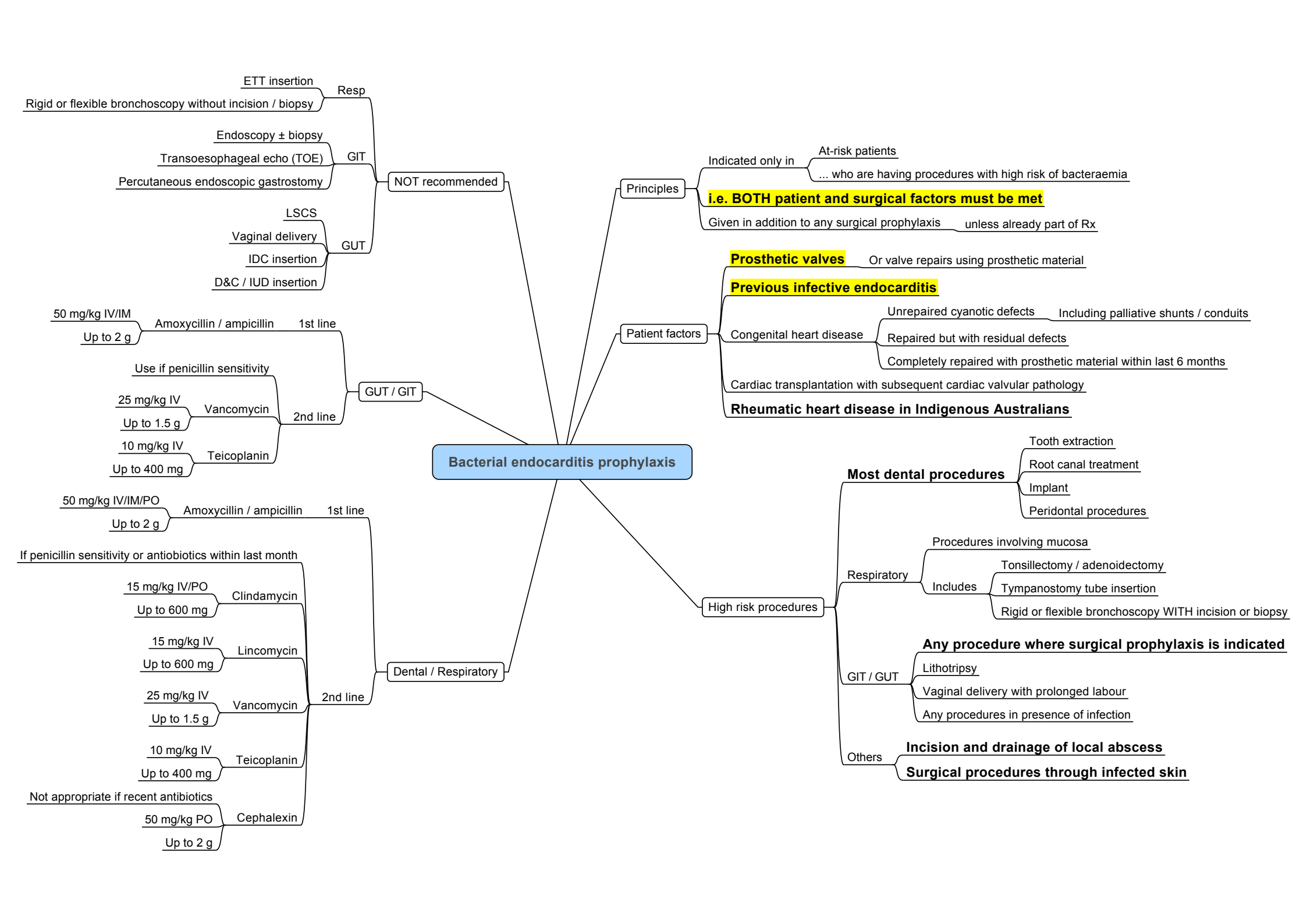

ATLS - Primary survey
c + ABCDE
Look
Percuss / feel
Auscultate
Assessment
Exclude/Treat specific Dx
Immediate Rx
At each stage
Used in trauma
Catastrophic haemorrhage control
Direct manual compression on wound
Precedes ABCDE
Avoid using tourniquet
Exposure / environment
Expose to look for other obvious injuries
Check the back as well
Avoid hypothermia
Immediate Rx
Airway (+ c-spine)
Assessment
Look
Seesaw respiration
Use of accessory muscle
Auscultate
Exclude
Airway obstruction
c-spine stabilisation
Immediate Rx
Clear airway
Suction
Manoeuvres (jaw thrust, chin lifts)
Airway adjuncts if appropriate
Intubation if indicated
e.g.
GCS ≤ 8
Ventilation / oxygenation failure
Airway trauma / swelling
Disability
GCS or AVPU
Level of consciousness
Size
Pupils
Reaction
Asymmetrical posturing
Hemiparesis
Lateralising signs
Facial weakness
Spinal cord injury level
Check BSL
Circulation
Skin colour
Look
Level of consciousness
Pulse
Capillary return
Feel
Limb warmth
Assessment
Auscultate
Cardiac temponade
Exclude
Identify and control haemorrhage
Two large bore IV cannula
Immediate Rx
Check BP and HR
Breathing
Assessment
Look
Respiratory efforts / efficacy
Depth
Rate
Pattern / rhythm
Signs of exhaustion
Bilateral chest movement
Percuss
Auscultate
Exclude
Tension pneumothorax
Massive haemothorax
Open pneumothorax
Flail chest with pulmonary contusion
Immediate Rx
O2 supplement
Pulse oximeter
Decompression if indicated

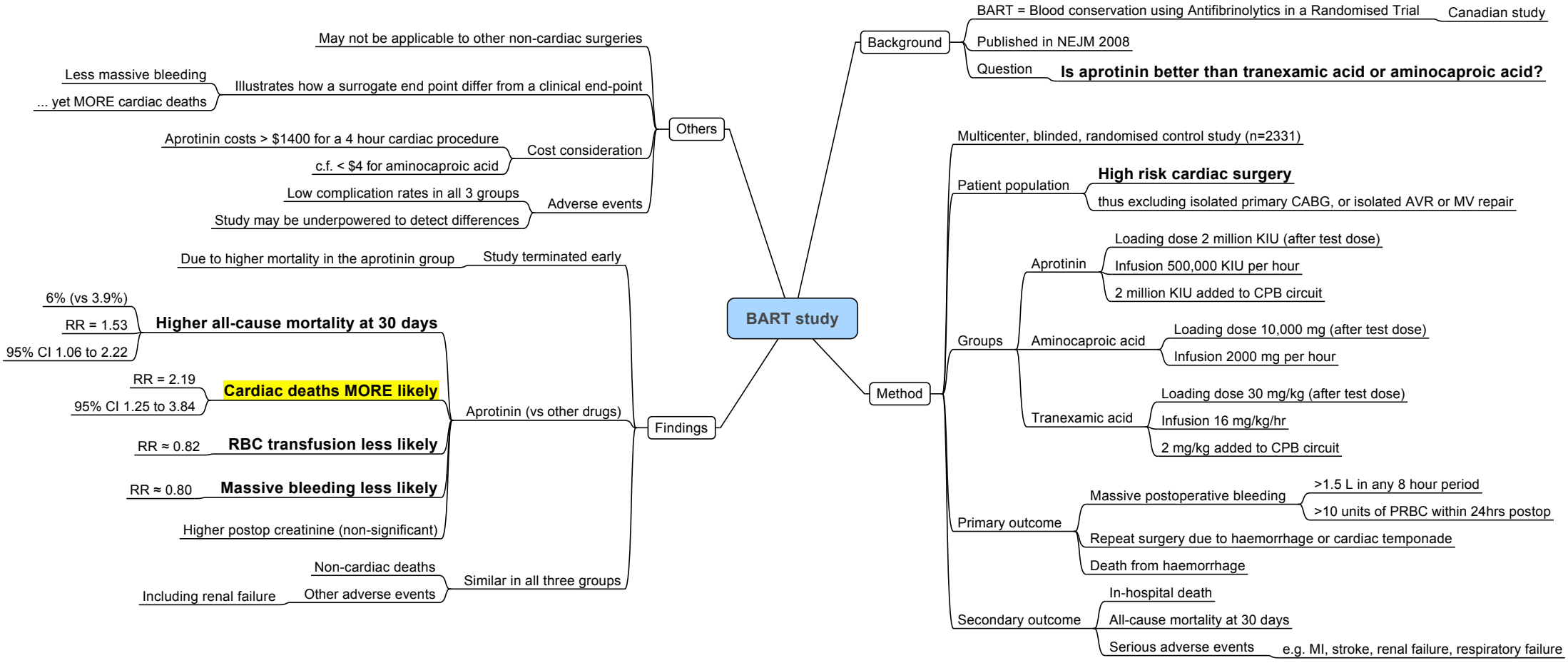

Atrial fibrillation (AF), Acute
Causes
A+B
Hypercarbia
Hypoxia
C
Hypertension / hypotension
Myocardial ischaemia
Mitral valve disease
Pericarditis / mediastinitis
D
EtOH
Increased sympathetic outflow
E
Thyrotoxicosis
Sepsis
Electrolyte abnormalities
↓K+
↓Mg2+
Surgical
Thoracic surgery
Rx
Others
Use TOE to exclude thrombus before cardioversion, if ≥ 48 hours AND not anticoagulated
AFFIRM trial
Compares rate control vs rhythm control
No statistical difference in
All-cause mortality
Functional status
Quality of life
Rate of ischaemic stroke
Thus
If asymptomatic, not imperative to attempt cardioversion first
If unstable, DC cardioversion immediately
± sedation
Heparinisation if possible
Rx of underlying cause
Rhythm conversion
Synchronised DC cardioversion
Pharmacological
Flecainide
If pregnant, quinidine
Rate control
Beta-blockers
Calcium channel blockers
Use non-dihydropyridine
Avoid if WPW suspected
Amiodarone
Can also lead to cardioversion
Anticoagulation first if ≥ 48 hours
Digoxin
Avoid if WPW suspected
Thromboembolism prevention
Anticoagulation
Warfarin
INR 2 to 3
Aspirin instead, if low risk
If inpatient awaiting cardioversion
IV heparin infusion
LMWH
Consider catheter ablation for sympatomatic persistent AF

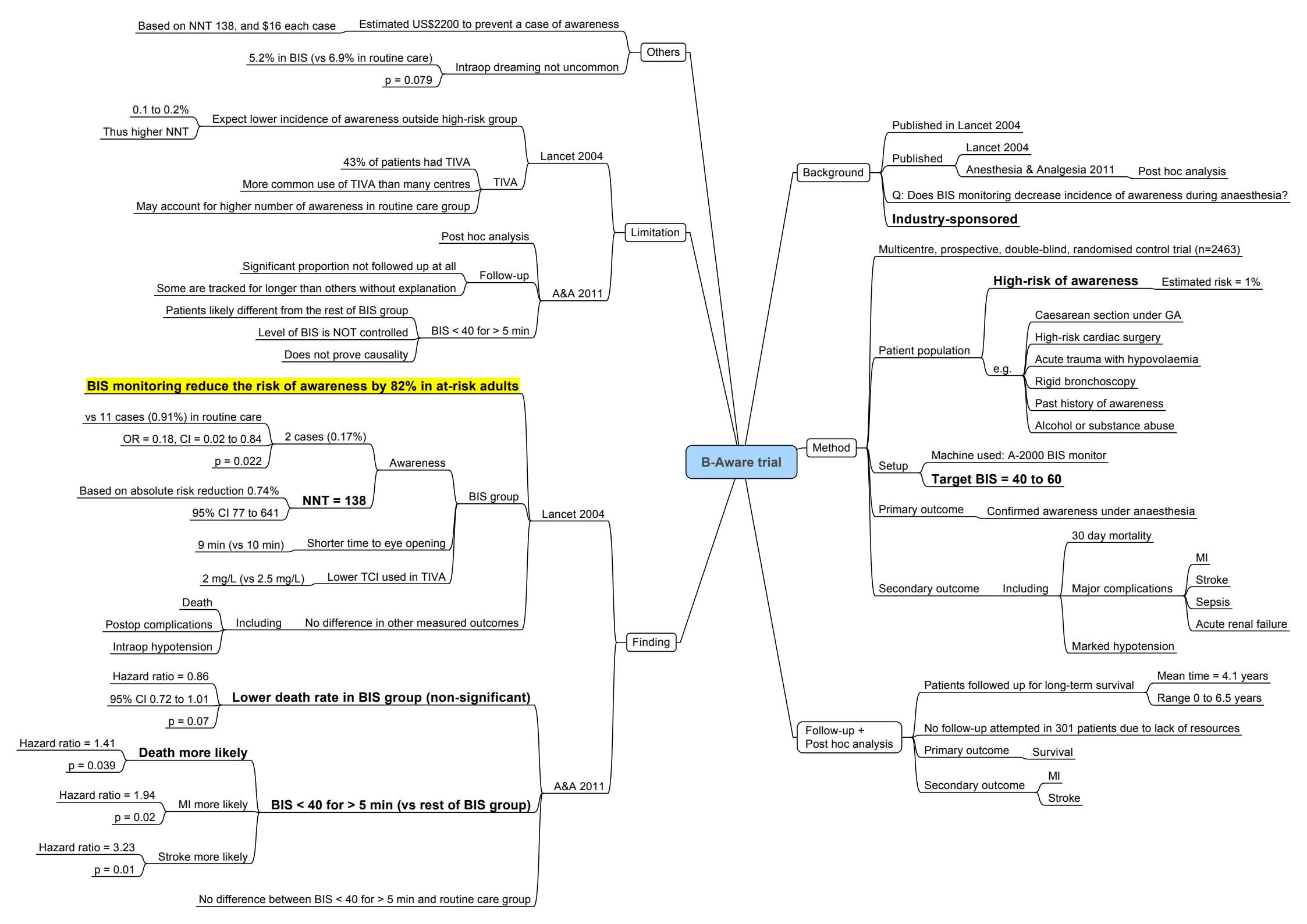

Atrial fibrillation (AF), chronic - AMx
Issues
Reduced ventricular filling and CO
Risk of periop thromboemoblism
Consultation
Hx
Functional capacity
CHADS2 score
CHF
Hypertension
Age ≥ 75
DM
S2 - Prior Stroke or TIA (2 points)
Rx
Anticoagulation Rx
Warfarin
Aspirin ± clopidogrel
Amiodarone / Digoxin
Ix
Bloods
ECG
Optimisation
Referral for rate control Rx if HR > 100
Goals
Maintain optimal ventricular rate (≈ 90 bpm)
Minimise periop thromboembolism risk
Bridging Rx
Consider
S
Type of surgery
Risk of significant bleeding
Time off anticoagulation Rx
P
Thromboembolism risk
CHADS2 score
Recent stroke / TIA (≤ 3 months)
Rheumatic heart disease
Rx
High risk
Therapeutic dose of IV heparin or SC LMWH
Low risk
Prophylactic dose LMWH
... or no bridging
Not all procedures require warfarin to be ceased

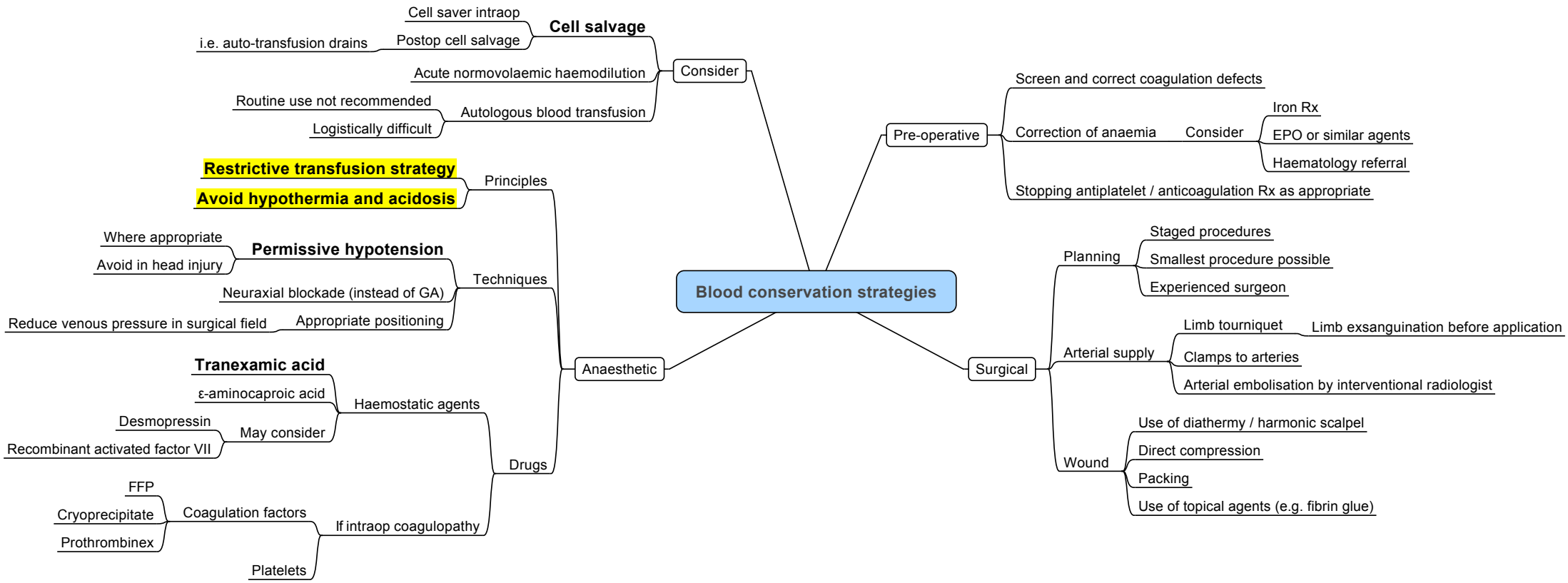

Atrial fibrillation (AF), chronic - CHADS2 score
Background
Clinical prediction rule for estimating risk of stroke in AF
Points
Congestive heart failure
Hypertension (Rx or not)
Age ≥ 75 years
Diabetes
S2 - Prior stroke or TIA
2 points
Relevance
If CHADS2 = 0
Low risk of stroke
Aspirin only
If CHADS2 ≥ 2
Moderate to high risk of stroke
Warfarin Rx indicated
For each point increase in CHADS2, stroke risk increases by almost 50%

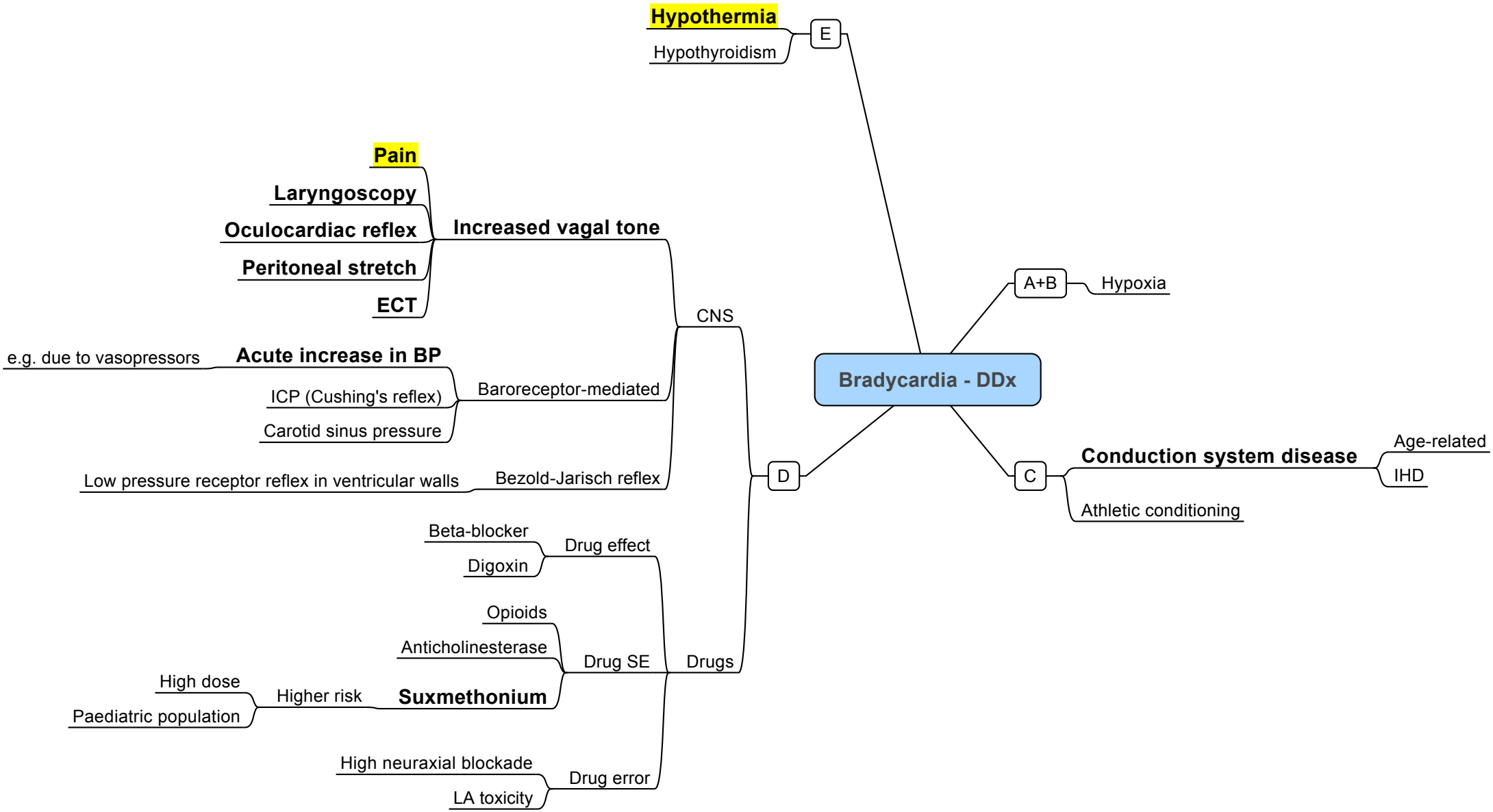

Autonomic neuropathy
Tests
Heart rate variability
Deep breathing
RR 5 /min
Normal variation: > 15 bpm
Abnormal: ≤ 10 bpm
Standing
Valsalva manoeuvre
BP response
Standing
Sustained handgrip
?QTc prolongation
Features
C
HR
Resting tachycardia
Bradycardial possible but uncommon
Lost of HR variability
BP
Supine hypertension
Postural hypotension
Exercise intolerance
D
Syncope
Blunted response to hypoglycaemia
E
Impaired thermoregulation
To both hot and cold
GIT
Delayed gastric emptying
Oesophageal dysmotility
Diarrhoea / constipation
Others
Bladder dysfunction (incontinence and/or retention)
Sexual dysfunction
Abnormal sweating
Causes
Most common
Diabetes mellitus (DM)
Alcoholism
CVA
CNS
Spinal injury
Parkinson's
Brain tumours
esp of third ventricle or posterior fossa
Neuromuscular disorders
Guillain-Barré
Amyotrophic lateral sclerosis
Multiple sclerosis
Myasthenic syndrome
Drug-induced
Metabolic
Chronic renal failure
Uraemic neuropathy
Chronic liver disease
e.g. Primary biliary cirrhosis
Infection
HIV
Tetanus
Botulism
Autoimmune
SLE
Rheumatoid arthritis
Scleroderma
Others
Congenital
Multiple myeloma
Porphyria
Vitamin deficiency
Vit B1, 3, 6, 12
Anaesthetic issues
A
Aspiration risk
Labile BP
C
Increased hypotensive effect to
IPPV
Spinal / Epidural
Position change (esp head-up)
Arrhythmia
Silent myocardial ischaemic / infarct
D
Reduced response to atropine and indirect-acting vasopressors
Unrecognised hypoglycaemia
E
Increased risk of hypothermia
Increased periop mortality

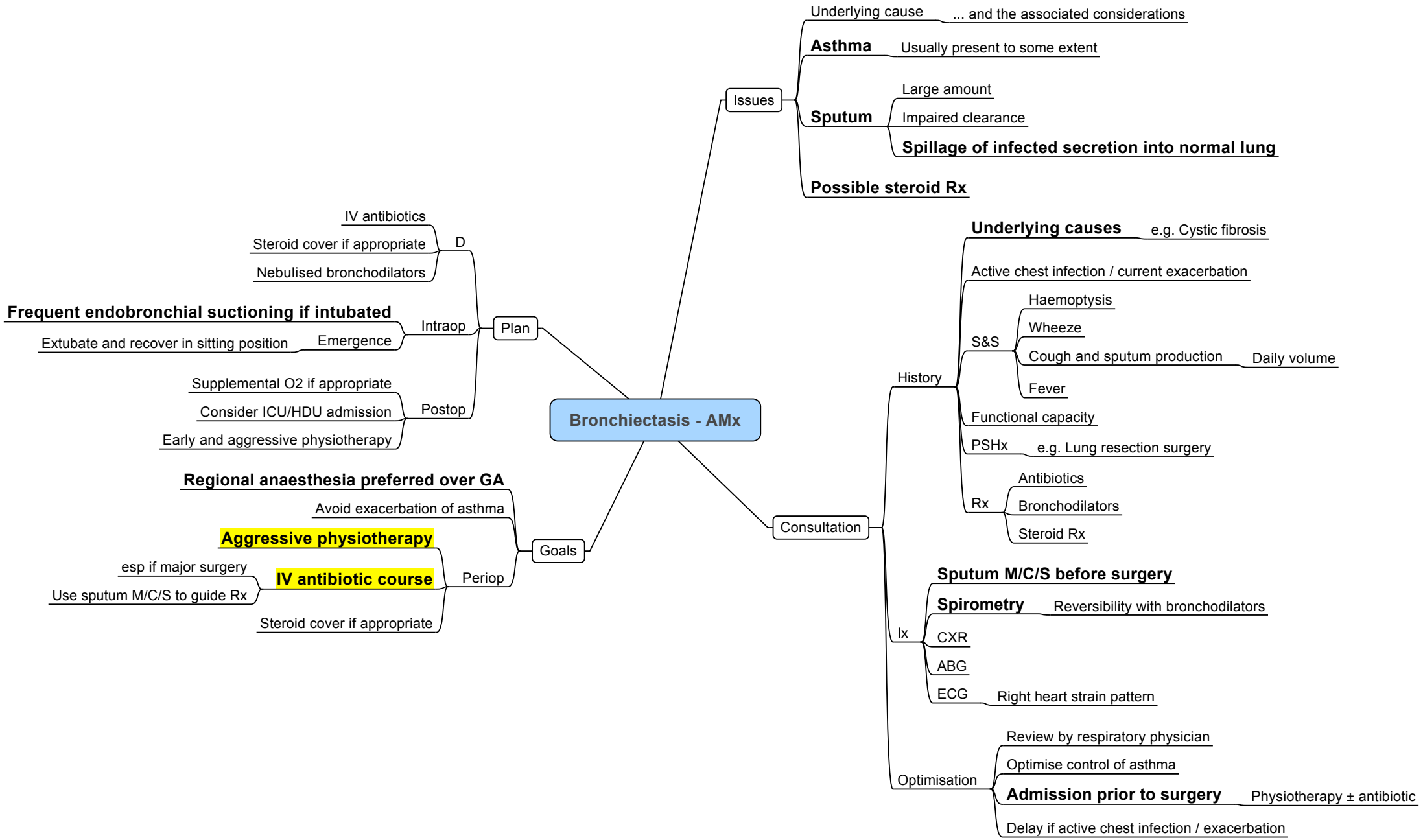

Bacterial endocarditis prophylaxis
Principles
Indicated only in
At-risk patients
... who are having procedures with high risk of bacteraemia
i.e. BOTH patient and surgical factors must be met
Given in addition to any surgical prophylaxis
unless already part of Rx
Patient factors
Prosthetic valves
Or valve repairs using prosthetic material
Previous infective endocarditis
Congenital heart disease
Unrepaired cyanotic defects
Including palliative shunts / conduits
Repaired but with residual defects
Completely repaired with prosthetic material within last 6 months
Cardiac transplantation with subsequent cardiac valvular pathology
Rheumatic heart disease in Indigenous Australians
High risk procedures
Most dental procedures
Tooth extraction
Root canal treatment
Implant
Peridontal procedures
Respiratory
Procedures involving mucosa
Includes
Tonsillectomy / adenoidectomy
Tympanostomy tube insertion
Rigid or flexible bronchoscopy WITH incision or biopsy
GIT / GUT
Any procedure where surgical prophylaxis is indicated
Lithotripsy
Vaginal delivery with prolonged labour
Any procedures in presence of infection
Others
Incision and drainage of local abscess
Surgical procedures through infected skin
NOT recommended
Resp
ETT insertion
Rigid or flexible bronchoscopy without incision / biopsy
GIT
Endoscopy ± biopsy
Transoesophageal echo (TOE)
Percutaneous endoscopic gastrostomy
GUT
LSCS
Vaginal delivery
IDC insertion
D&C / IUD insertion
GUT / GIT
1st line
Amoxycillin / ampicillin
50 mg/kg IV/IM
Up to 2 g
2nd line
Use if penicillin sensitivity
Vancomycin
25 mg/kg IV
Up to 1.5 g
Teicoplanin
10 mg/kg IV
Up to 400 mg
Dental / Respiratory
1st line
Amoxycillin / ampicillin
50 mg/kg IV/IM/PO
Up to 2 g
2nd line
If penicillin sensitivity or antiobiotics within last month
Clindamycin
15 mg/kg IV/PO
Up to 600 mg
Lincomycin
15 mg/kg IV
Up to 600 mg
Vancomycin
25 mg/kg IV
Up to 1.5 g
Teicoplanin
10 mg/kg IV
Up to 400 mg
Cephalexin
Not appropriate if recent antibiotics
50 mg/kg PO
Up to 2 g

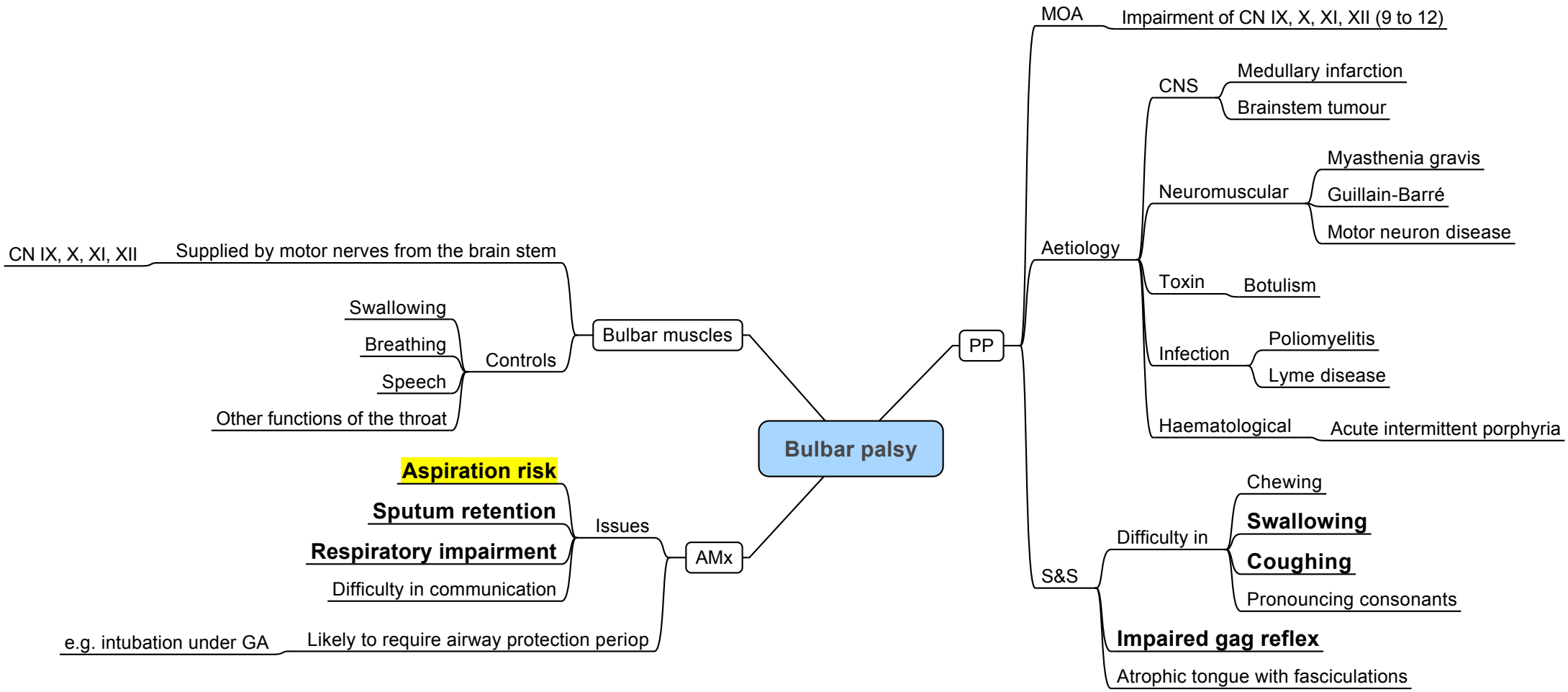

BART study
Background
BART = Blood conservation using Antifibrinolytics in a Randomised Trial
Canadian study
Published in NEJM 2008
Question
Is aprotinin better than tranexamic acid or aminocaproic acid?
Method
Multicenter, blinded, randomised control study (n=2331)
Patient population
High risk cardiac surgery
thus excluding isolated primary CABG, or isolated AVR or MV repair
Groups
Aprotinin
Loading dose 2 million KIU (after test dose)
Infusion 500,000 KIU per hour
2 million KIU added to CPB circuit
Aminocaproic acid
Loading dose 10,000 mg (after test dose)
Infusion 2000 mg per hour
Tranexamic acid
Loading dose 30 mg/kg (after test dose)
Infusion 16 mg/kg/hr
2 mg/kg added to CPB circuit
Primary outcome
Massive postoperative bleeding
>1.5 L in any 8 hour period
>10 units of PRBC within 24hrs postop
Repeat surgery due to haemorrhage or cardiac temponade
Death from haemorrhage
Secondary outcome
In-hospital death
All-cause mortality at 30 days
Serious adverse events
e.g. MI, stroke, renal failure, respiratory failure
Findings
Study terminated early
Due to higher mortality in the aprotinin group
Aprotinin (vs other drugs)
Higher all-cause mortality at 30 days
6% (vs 3.9%)
RR = 1.53
95% CI 1.06 to 2.22
Cardiac deaths MORE likely
RR = 2.19
95% CI 1.25 to 3.84
RBC transfusion less likely
RR ≈ 0.82
Massive bleeding less likely
RR ≈ 0.80
Higher postop creatinine (non-significant)
Similar in all three groups
Non-cardiac deaths
Other adverse events
Including renal failure
Others
May not be applicable to other non-cardiac surgeries
Illustrates how a surrogate end point differ from a clinical end-point
Less massive bleeding
... yet MORE cardiac deaths
Cost consideration
Aprotinin costs > $1400 for a 4 hour cardiac procedure
c.f. < $4 for aminocaproic acid
Adverse events
Low complication rates in all 3 groups
Study may be underpowered to detect differences

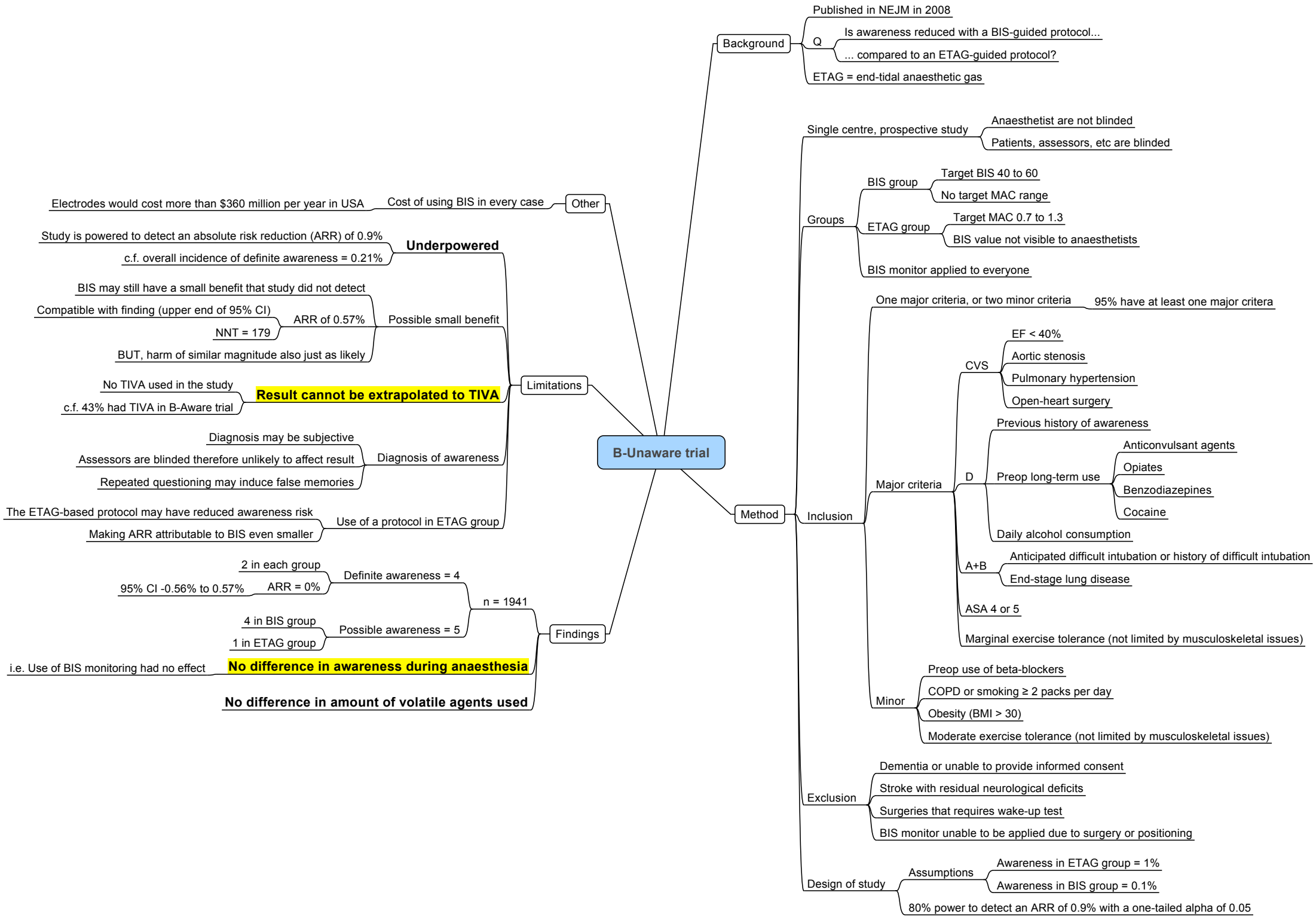

B-Aware trial
Background
Published in Lancet 2004
Published
Lancet 2004
Anesthesia & Analgesia 2011
Post hoc analysis
Q: Does BIS monitoring decrease incidence of awareness during anaesthesia?
Industry-sponsored
Method
Multicentre, prospective, double-blind, randomised control trial (n=2463)
Patient population
High-risk of awareness
Estimated risk = 1%
e.g.
Caesarean section under GA
High-risk cardiac surgery
Acute trauma with hypovolaemia
Rigid bronchoscopy
Past history of awareness
Alcohol or substance abuse
Setup
Machine used: A-2000 BIS monitor
Target BIS = 40 to 60
Primary outcome
Confirmed awareness under anaesthesia
Secondary outcome
Including
30 day mortality
Major complications
MI
Stroke
Sepsis
Acute renal failure
Marked hypotension
Follow-up + Post hoc analysis
Patients followed up for long-term survival
Mean time = 4.1 years
Range 0 to 6.5 years
No follow-up attempted in 301 patients due to lack of resources
Primary outcome
Survival
Secondary outcome
MI
Stroke
Finding
Lancet 2004
BIS monitoring reduce the risk of awareness by 82% in at-risk adults
BIS group
Awareness
2 cases (0.17%)
vs 11 cases (0.91%) in routine care
OR = 0.18, CI = 0.02 to 0.84
p = 0.022
NNT = 138
Based on absolute risk reduction 0.74%
95% CI 77 to 641
Shorter time to eye opening
9 min (vs 10 min)
Lower TCI used in TIVA
2 mg/L (vs 2.5 mg/L)
No difference in other measured outcomes
Including
Death
Postop complications
Intraop hypotension
A&A 2011
Lower death rate in BIS group (non-significant)
Hazard ratio = 0.86
95% CI 0.72 to 1.01
p = 0.07
BIS < 40 for > 5 min (vs rest of BIS group)
Death more likely
Hazard ratio = 1.41
p = 0.039
MI more likely
Hazard ratio = 1.94
p = 0.02
Stroke more likely
Hazard ratio = 3.23
p = 0.01
No difference between BIS < 40 for > 5 min and routine care group
Limitation
Lancet 2004
Expect lower incidence of awareness outside high-risk group
0.1 to 0.2%
Thus higher NNT
TIVA
43% of patients had TIVA
More common use of TIVA than many centres
May account for higher number of awareness in routine care group
A&A 2011
Post hoc analysis
Follow-up
Significant proportion not followed up at all
Some are tracked for longer than others without explanation
BIS < 40 for > 5 min
Patients likely different from the rest of BIS group
Level of BIS is NOT controlled
Does not prove causality
Others
Based on NNT 138, and $16 each case
Estimated US$2200 to prevent a case of awareness
Intraop dreaming not uncommon
5.2% in BIS (vs 6.9% in routine care)
p = 0.079

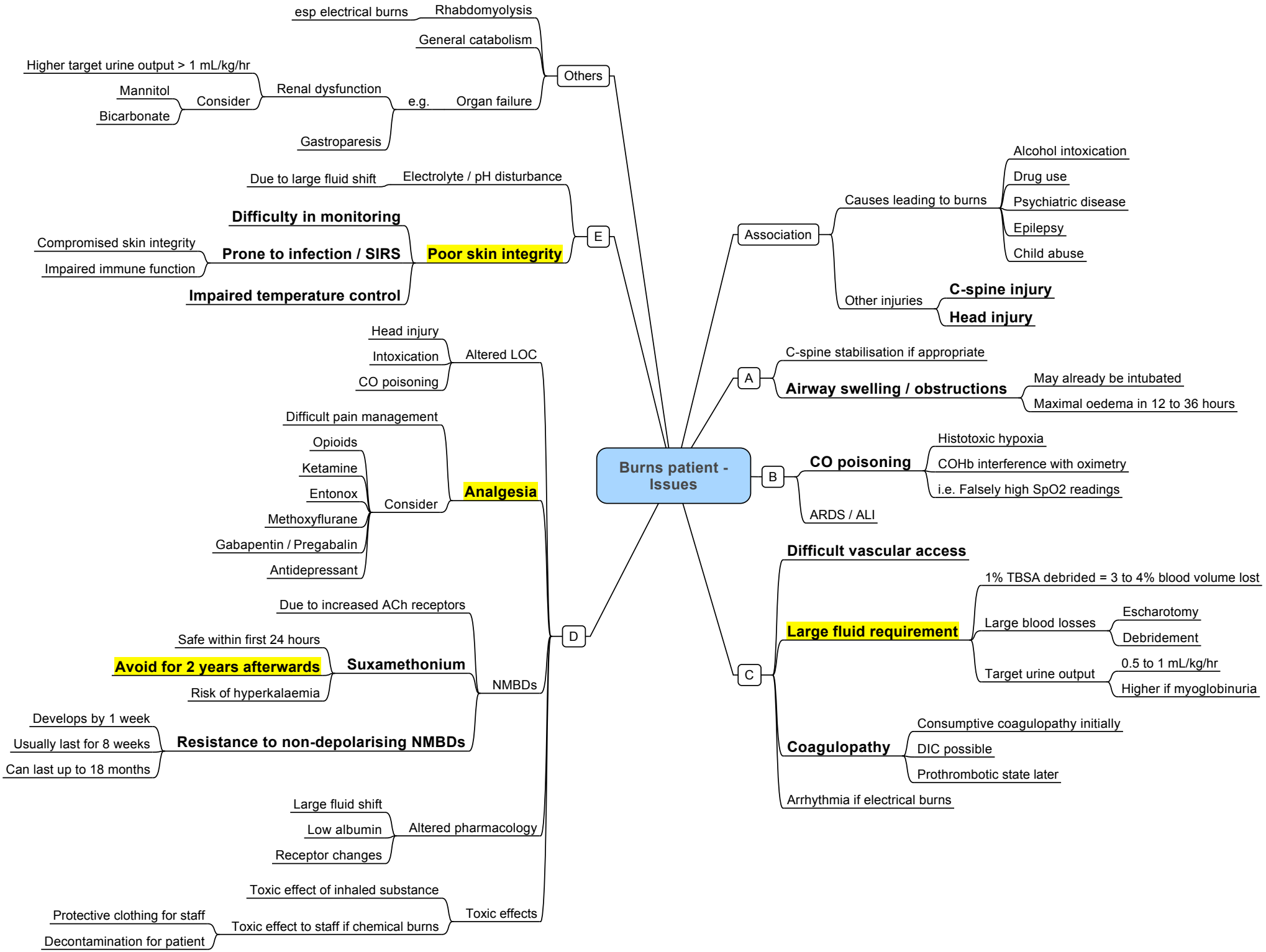

Blood conservation strategies
Anaesthetic
Consider
Cell salvage
Cell saver intraop
Postop cell salvage
i.e. auto-transfusion drains
Acute normovolaemic haemodilution
Autologous blood transfusion
Routine use not recommended
Logistically difficult
Principles
Restrictive transfusion strategy
Avoid hypothermia and acidosis
Techniques
Permissive hypotension
Where appropriate
Avoid in head injury
Neuraxial blockade (instead of GA)
Appropriate positioning
Reduce venous pressure in surgical field
Drugs
Haemostatic agents
Tranexamic acid
ε-aminocaproic acid
May consider
Desmopressin
Recombinant activated factor VII
If intraop coagulopathy
Coagulation factors
FFP
Cryoprecipitate
Prothrombinex
Platelets
Pre-operative
Screen and correct coagulation defects
Correction of anaemia
Consider
Iron Rx
EPO or similar agents
Haematology referral
Stopping antiplatelet / anticoagulation Rx as appropriate
Surgical
Planning
Staged procedures
Smallest procedure possible
Experienced surgeon
Arterial supply
Limb tourniquet
Limb exsanguination before application
Clamps to arteries
Arterial embolisation by interventional radiologist
Wound
Use of diathermy / harmonic scalpel
Direct compression
Packing
Use of topical agents (e.g. fibrin glue)

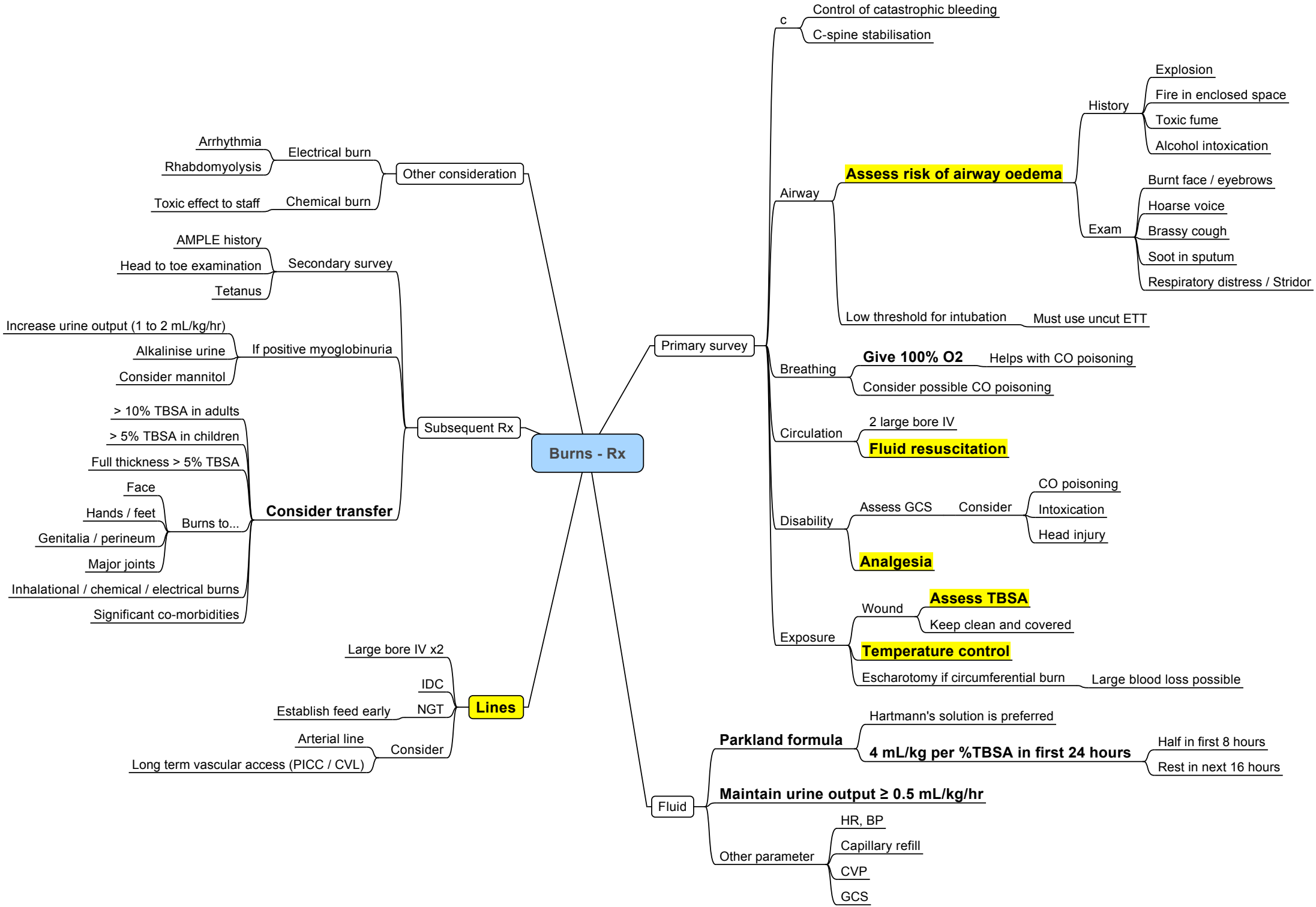

Bradycardia - DDx
A+B
Hypoxia
C
Conduction system disease
Age-related
IHD
Athletic conditioning
D
CNS
Increased vagal tone
Pain
Laryngoscopy
Oculocardiac reflex
Peritoneal stretch
ECT
Baroreceptor-mediated
Acute increase in BP
e.g. due to vasopressors
ICP (Cushing's reflex)
Carotid sinus pressure
Bezold-Jarisch reflex
Low pressure receptor reflex in ventricular walls
Drugs
Drug effect
Beta-blocker
Digoxin
Drug SE
Opioids
Anticholinesterase
Higher risk
Suxmethonium
High dose
Paediatric population
Drug error
High neuraxial blockade
LA toxicity
E
Hypothermia
Hypothyroidism

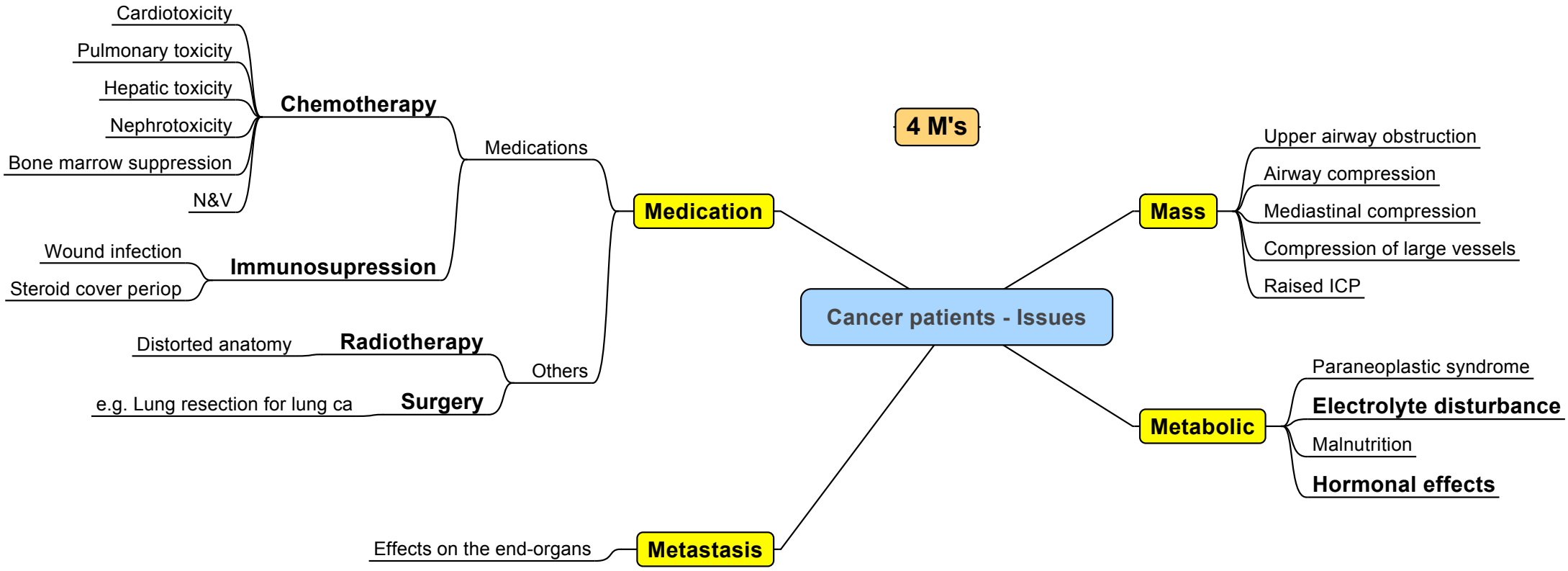

Bronchiectasis - AMx
Issues
Underlying cause
... and the associated considerations
Asthma
Usually present to some extent
Sputum
Large amount
Impaired clearance
Spillage of infected secretion into normal lung
Possible steroid Rx
Plan
D
IV antibiotics
Steroid cover if appropriate
Nebulised bronchodilators
Intraop
Frequent endobronchial suctioning if intubated
Emergence
Extubate and recover in sitting position
Postop
Supplemental O2 if appropriate
Consider ICU/HDU admission
Early and aggressive physiotherapy
Goals
Regional anaesthesia preferred over GA
Avoid exacerbation of asthma
Periop
Aggressive physiotherapy
IV antibiotic course
esp if major surgery
Use sputum M/C/S to guide Rx
Steroid cover if appropriate
Consultation
History
Underlying causes
e.g. Cystic fibrosis
Active chest infection / current exacerbation
S&S
Haemoptysis
Wheeze
Cough and sputum production
Daily volume
Fever
Functional capacity
PSHx
e.g. Lung resection surgery
Rx
Antibiotics
Bronchodilators
Steroid Rx
Ix
Sputum M/C/S before surgery
Spirometry
Reversibility with bronchodilators
CXR
ABG
ECG
Right heart strain pattern
Optimisation
Review by respiratory physician
Optimise control of asthma
Admission prior to surgery
Physiotherapy ± antibiotic
Delay if active chest infection / exacerbation

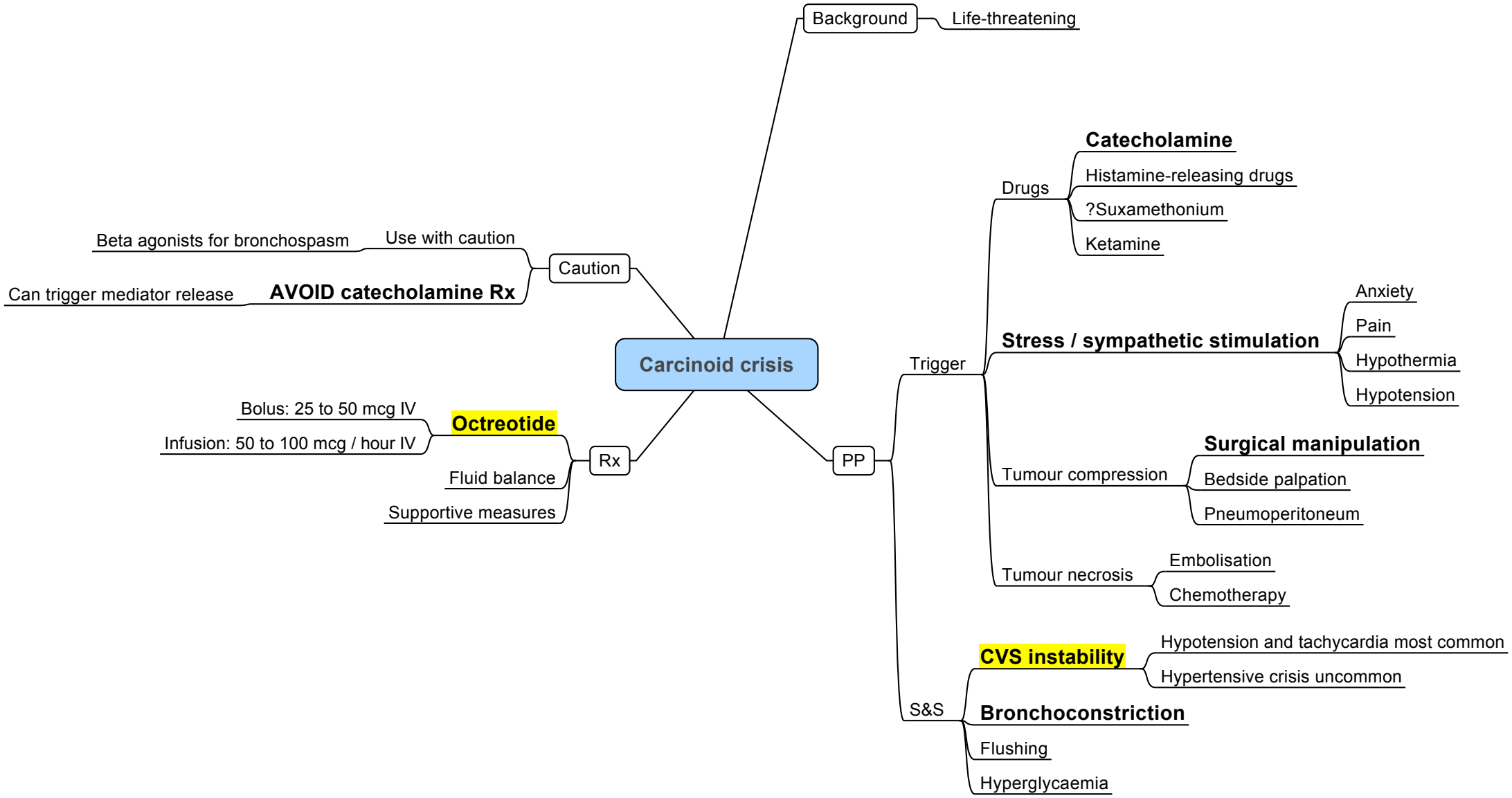

Bulbar palsy
PP
MOA
Impairment of CN IX, X, XI, XII (9 to 12)
Aetiology
CNS
Medullary infarction
Brainstem tumour
Neuromuscular
Myasthenia gravis
Guillain-Barré
Motor neuron disease
Toxin
Botulism
Infection
Poliomyelitis
Lyme disease
Haematological
Acute intermittent porphyria
S&S
Difficulty in
Chewing
Swallowing
Coughing
Pronouncing consonants
Impaired gag reflex
Atrophic tongue with fasciculations
Bulbar muscles
Supplied by motor nerves from the brain stem
CN IX, X, XI, XII
Controls
Swallowing
Breathing
Speech
Other functions of the throat
AMx
Issues
Aspiration risk
Sputum retention
Respiratory impairment
Difficulty in communication
Likely to require airway protection periop
e.g. intubation under GA

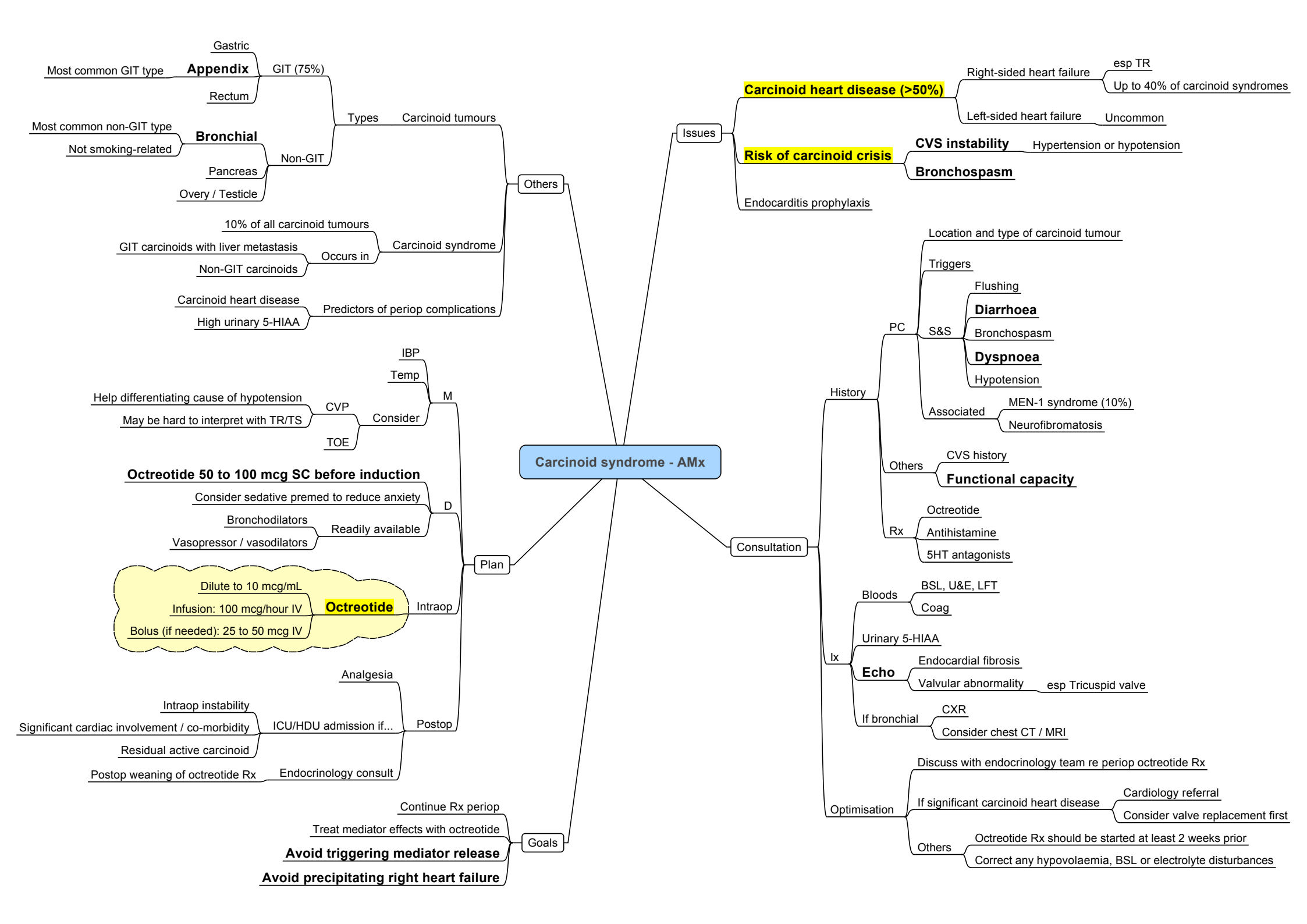

B-Unaware trial
Background
Published in NEJM in 2008
Q
Is awareness reduced with a BIS-guided protocol...
... compared to an ETAG-guided protocol?
ETAG = end-tidal anaesthetic gas
Method
Single centre, prospective study
Anaesthetist are not blinded
Patients, assessors, etc are blinded
Groups
BIS group
Target BIS 40 to 60
No target MAC range
ETAG group
Target MAC 0.7 to 1.3
BIS value not visible to anaesthetists
BIS monitor applied to everyone
Inclusion
One major criteria, or two minor criteria
95% have at least one major critera
Major criteria
CVS
EF < 40%
Aortic stenosis
Pulmonary hypertension
Open-heart surgery
D
Previous history of awareness
Preop long-term use
Anticonvulsant agents
Opiates
Benzodiazepines
Cocaine
Daily alcohol consumption
A+B
Anticipated difficult intubation or history of difficult intubation
End-stage lung disease
ASA 4 or 5
Marginal exercise tolerance (not limited by musculoskeletal issues)
Minor
Preop use of beta-blockers
COPD or smoking ≥ 2 packs per day
Obesity (BMI > 30)
Moderate exercise tolerance (not limited by musculoskeletal issues)
Exclusion
Dementia or unable to provide informed consent
Stroke with residual neurological deficits
Surgeries that requires wake-up test
BIS monitor unable to be applied due to surgery or positioning
Design of study
Assumptions
Awareness in ETAG group = 1%
Awareness in BIS group = 0.1%
80% power to detect an ARR of 0.9% with a one-tailed alpha of 0.05
Findings
n = 1941
Definite awareness = 4
2 in each group
ARR = 0%
95% CI -0.56% to 0.57%
Possible awareness = 5
4 in BIS group
1 in ETAG group
i.e. Use of BIS monitoring had no effect
No difference in awareness during anaesthesia
No difference in amount of volatile agents used
Limitations
Other
Cost of using BIS in every case
Electrodes would cost more than $360 million per year in USA
Underpowered
Study is powered to detect an absolute risk reduction (ARR) of 0.9%
c.f. overall incidence of definite awareness = 0.21%
Possible small benefit
BIS may still have a small benefit that study did not detect
ARR of 0.57%
Compatible with finding (upper end of 95% CI)
NNT = 179
BUT, harm of similar magnitude also just as likely
Result cannot be extrapolated to TIVA
No TIVA used in the study
c.f. 43% had TIVA in B-Aware trial
Diagnosis of awareness
Diagnosis may be subjective
Assessors are blinded therefore unlikely to affect result
Repeated questioning may induce false memories
Use of a protocol in ETAG group
The ETAG-based protocol may have reduced awareness risk
Making ARR attributable to BIS even smaller

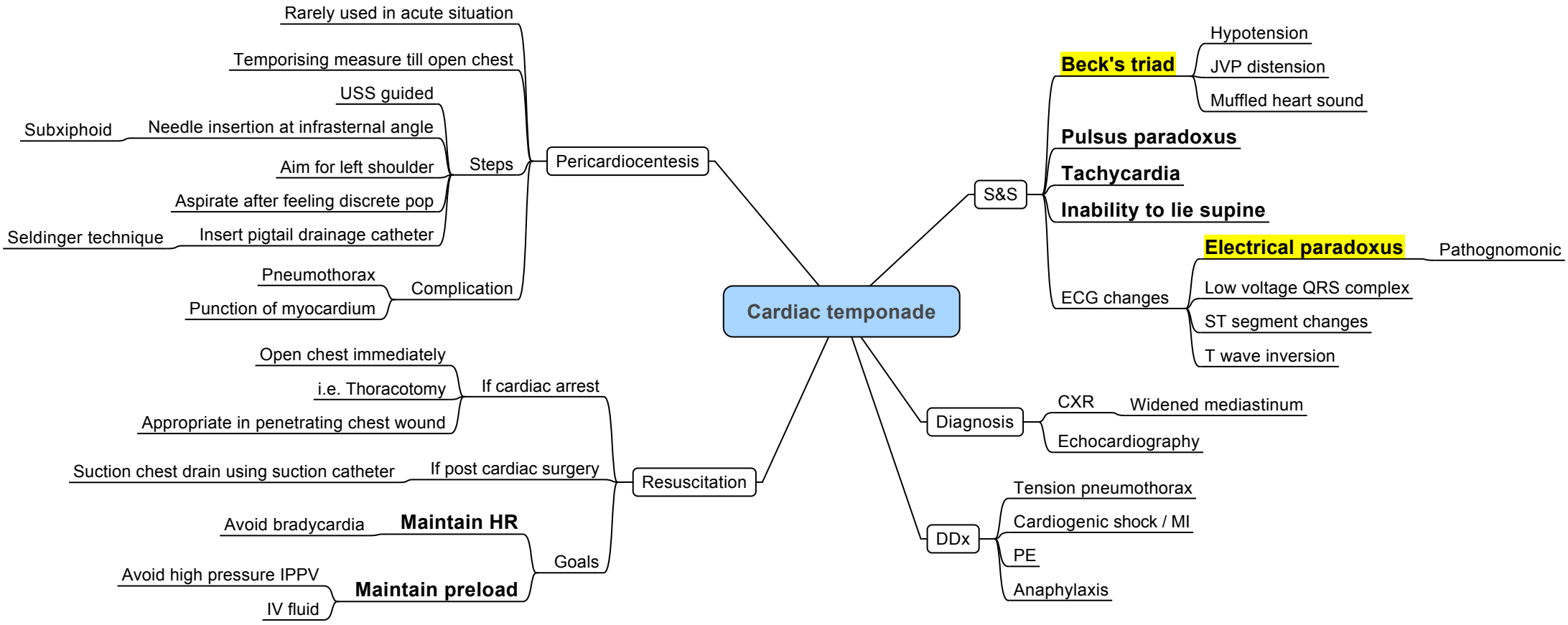

Burns patient - Issues
A
Airway swelling / obstructions
C-spine stabilisation if appropriate
May already be intubated
Maximal oedema in 12 to 36 hours
B
CO poisoning
Histotoxic hypoxia
COHb interference with oximetry
i.e. Falsely high SpO2 readings
ARDS / ALI
C
Difficult vascular access
Large fluid requirement
1% TBSA debrided = 3 to 4% blood volume lost
Large blood losses
Escharotomy
Debridement
Target urine output
0.5 to 1 mL/kg/hr
Higher if myoglobinuria
Coagulopathy
Consumptive coagulopathy initially
DIC possible
Prothrombotic state later
Arrhythmia if electrical burns
D
Altered LOC
Head injury
Intoxication
CO poisoning
Analgesia
Difficult pain management
Consider
Opioids
Ketamine
Entonox
Methoxyflurane
Gabapentin / Pregabalin
Antidepressant
NMBDs
Due to increased ACh receptors
Suxamethonium
Safe within first 24 hours
Avoid for 2 years afterwards
Risk of hyperkalaemia
Resistance to non-depolarising NMBDs
Develops by 1 week
Usually last for 8 weeks
Can last up to 18 months
Altered pharmacology
Large fluid shift
Low albumin
Receptor changes
Toxic effects
Toxic effect of inhaled substance
Toxic effect to staff if chemical burns
Protective clothing for staff
Decontamination for patient
E
Poor skin integrity
Electrolyte / pH disturbance
Due to large fluid shift
Difficulty in monitoring
Prone to infection / SIRS
Compromised skin integrity
Impaired immune function
Impaired temperature control
Others
Rhabdomyolysis
esp electrical burns
General catabolism
Renal dysfunction
e.g.
Organ failure
Consider
Higher target urine output > 1 mL/kg/hr
Mannitol
Bicarbonate
Gastroparesis
Association
Causes leading to burns
Alcohol intoxication
Drug use
Psychiatric disease
Epilepsy
Child abuse
Other injuries
C-spine injury
Head injury

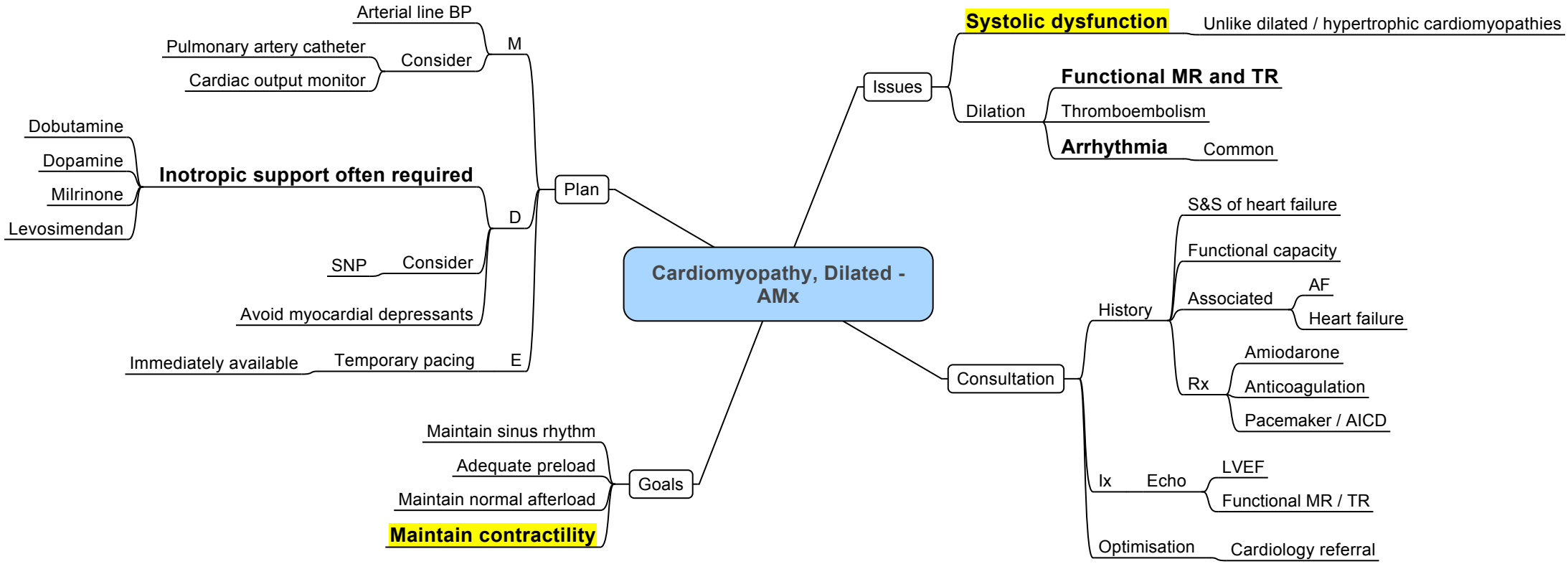

Burns - Rx
Primary survey
c
Control of catastrophic bleeding
C-spine stabilisation
Airway
Assess risk of airway oedema
History
Explosion
Fire in enclosed space
Toxic fume
Alcohol intoxication
Exam
Burnt face / eyebrows
Hoarse voice
Brassy cough
Soot in sputum
Respiratory distress / Stridor
Low threshold for intubation
Must use uncut ETT
Breathing
Give 100% O2
Helps with CO poisoning
Consider possible CO poisoning
Circulation
2 large bore IV
Fluid resuscitation
Disability
Assess GCS
Consider
CO poisoning
Intoxication
Head injury
Analgesia
Exposure
Wound
Assess TBSA
Keep clean and covered
Temperature control
Escharotomy if circumferential burn
Large blood loss possible
Fluid
Parkland formula
Hartmann's solution is preferred
4 mL/kg per %TBSA in first 24 hours
Half in first 8 hours
Rest in next 16 hours
Maintain urine output ≥ 0.5 mL/kg/hr
Other parameter
HR, BP
Capillary refill
CVP
GCS
Lines
Large bore IV x2
IDC
NGT
Establish feed early
Consider
Arterial line
Long term vascular access (PICC / CVL)
Subsequent Rx
Other consideration
Electrical burn
Arrhythmia
Rhabdomyolysis
Chemical burn
Toxic effect to staff
Secondary survey
AMPLE history
Head to toe examination
Tetanus
If positive myoglobinuria
Increase urine output (1 to 2 mL/kg/hr)
Alkalinise urine
Consider mannitol
Consider transfer
> 10% TBSA in adults
> 5% TBSA in children
Full thickness > 5% TBSA
Burns to...
Face
Hands / feet
Genitalia / perineum
Major joints
Inhalational / chemical / electrical burns
Significant co-morbidities

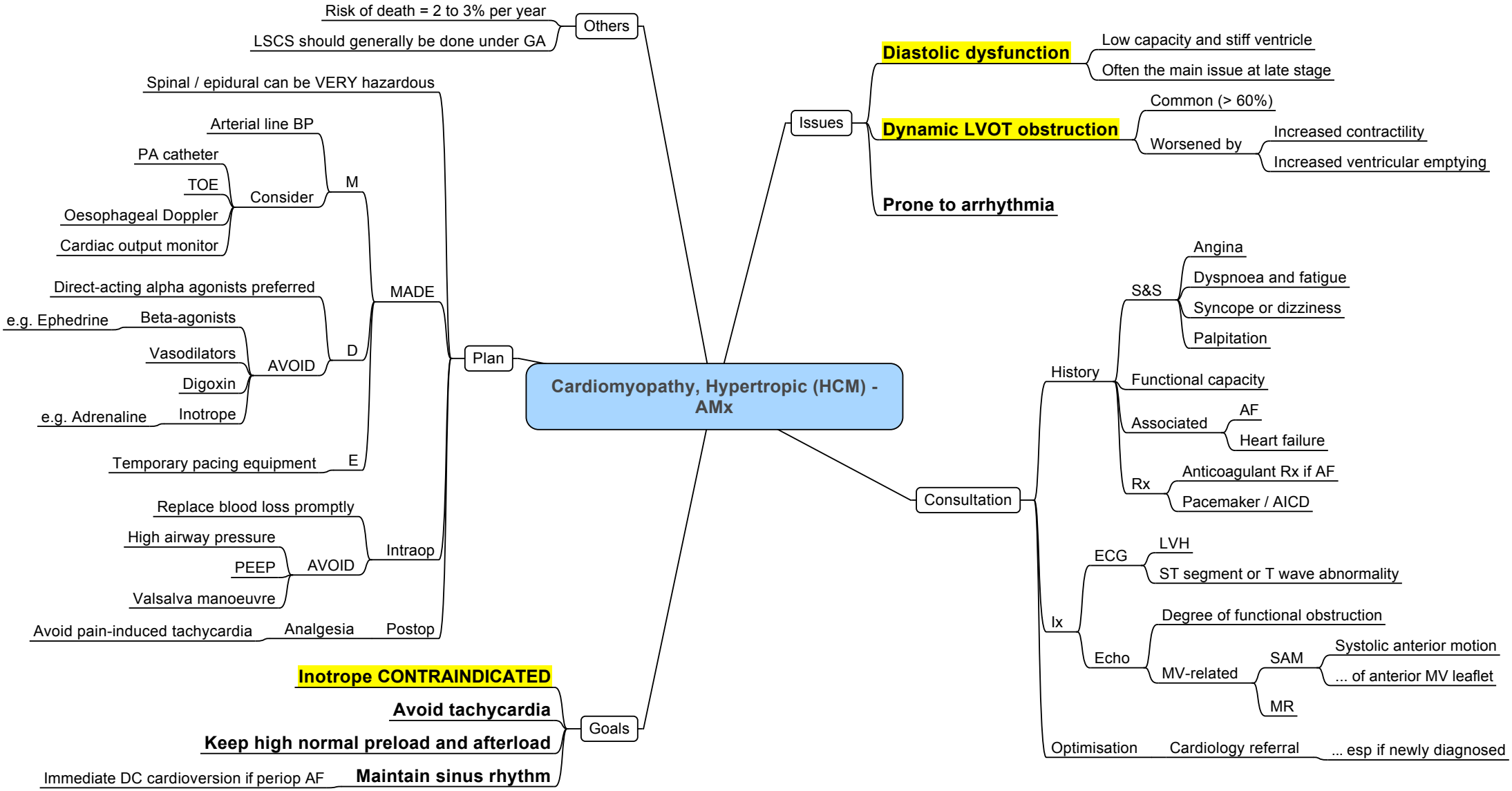

Cancer patients - Issues
4 M's
Medication
Medications
Chemotherapy
Cardiotoxicity
Pulmonary toxicity
Hepatic toxicity
Nephrotoxicity
Bone marrow suppression
N&V
Immunosupression
Wound infection
Steroid cover periop
Others
Radiotherapy
Distorted anatomy
Surgery
e.g. Lung resection for lung ca
Mass
Upper airway obstruction
Airway compression
Mediastinal compression
Compression of large vessels
Raised ICP
Metabolic
Paraneoplastic syndrome
Electrolyte disturbance
Malnutrition
Hormonal effects
Metastasis
Effects on the end-organs

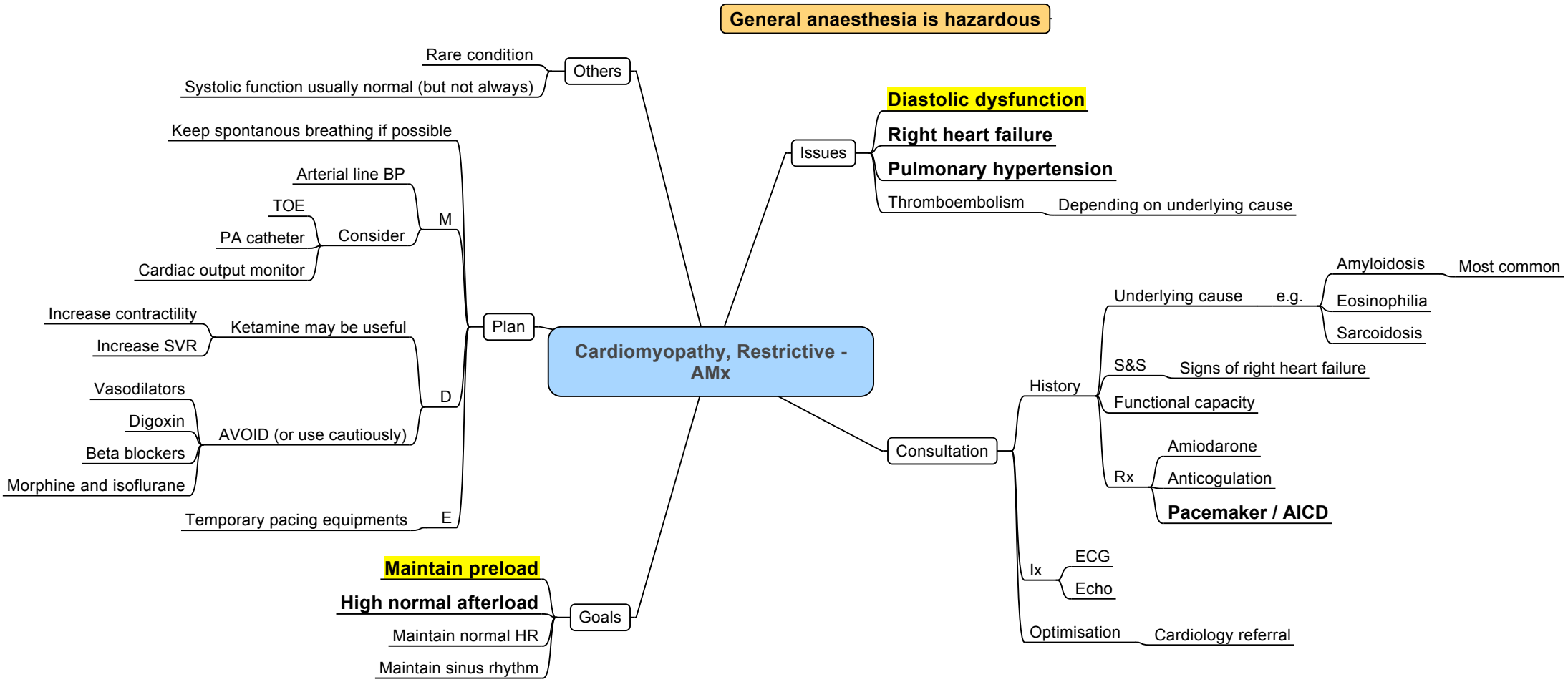

Carcinoid crisis
Background
Life-threatening
Caution
Use with caution
Beta agonists for bronchospasm
AVOID catecholamine Rx
Can trigger mediator release
Rx
Octreotide
Bolus: 25 to 50 mcg IV
Infusion: 50 to 100 mcg / hour IV
Fluid balance
Supportive measures
PP
Trigger
Drugs
Catecholamine
Histamine-releasing drugs
?Suxamethonium
Ketamine
Stress / sympathetic stimulation
Anxiety
Pain
Hypothermia
Hypotension
Tumour compression
Surgical manipulation
Bedside palpation
Pneumoperitoneum
Tumour necrosis
Embolisation
Chemotherapy
S&S
CVS instability
Hypotension and tachycardia most common
Hypertensive crisis uncommon
Bronchoconstriction
Flushing
Hyperglycaemia

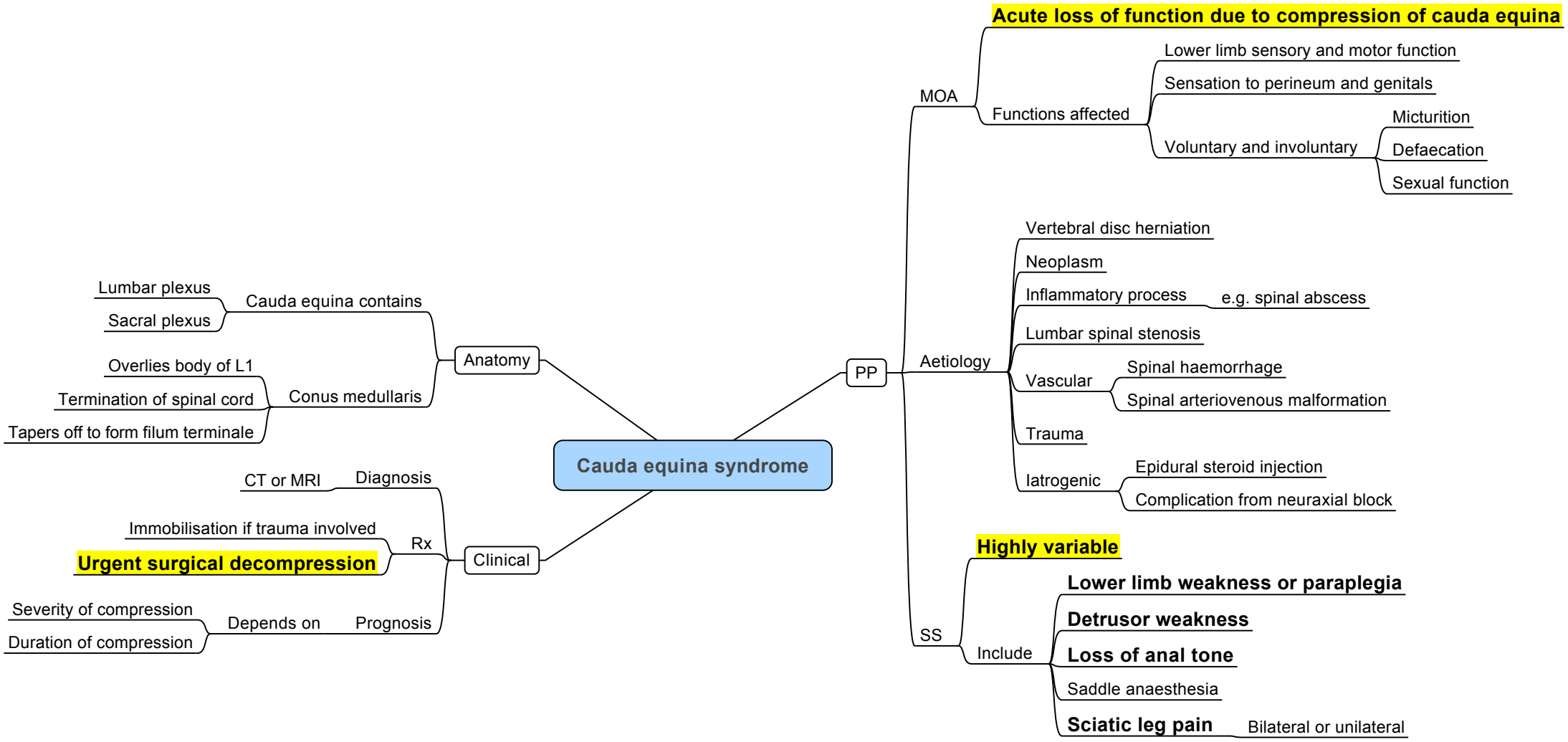

Carcinoid syndrome - AMx
Consultation
History
PC
Location and type of carcinoid tumour
Triggers
S&S
Flushing
Diarrhoea
Bronchospasm
Dyspnoea
Hypotension
Associated
MEN-1 syndrome (10%)
Neurofibromatosis
Others
CVS history
Functional capacity
Rx
Octreotide
Antihistamine
5HT antagonists
Ix
Bloods
BSL, U&E, LFT
Coag
Urinary 5-HIAA
Echo
Endocardial fibrosis
Valvular abnormality
esp Tricuspid valve
If bronchial
CXR
Consider chest CT / MRI
Optimisation
Discuss with endocrinology team re periop octreotide Rx
If significant carcinoid heart disease
Cardiology referral
Consider valve replacement first
Others
Octreotide Rx should be started at least 2 weeks prior
Correct any hypovolaemia, BSL or electrolyte disturbances
Issues
Carcinoid heart disease (>50%)
Right-sided heart failure
esp TR
Up to 40% of carcinoid syndromes
Left-sided heart failure
Uncommon
Risk of carcinoid crisis
CVS instability
Hypertension or hypotension
Bronchospasm
Endocarditis prophylaxis
Plan
M
IBP
Temp
Consider
CVP
Help differentiating cause of hypotension
May be hard to interpret with TR/TS
TOE
D
Octreotide 50 to 100 mcg SC before induction
Consider sedative premed to reduce anxiety
Readily available
Bronchodilators
Vasopressor / vasodilators
Intraop
Octreotide
Dilute to 10 mcg/mL
Infusion: 100 mcg/hour IV
Bolus (if needed): 25 to 50 mcg IV
Postop
Analgesia
ICU/HDU admission if...
Intraop instability
Significant cardiac involvement / co-morbidity
Residual active carcinoid
Endocrinology consult
Postop weaning of octreotide Rx
Goals
Continue Rx periop
Treat mediator effects with octreotide
Avoid triggering mediator release
Avoid precipitating right heart failure
Others
Carcinoid tumours
Types
GIT (75%)
Gastric
Most common GIT type
Appendix
Rectum
Non-GIT
Most common non-GIT type
Bronchial
Not smoking-related
Pancreas
Overy / Testicle
Carcinoid syndrome
10% of all carcinoid tumours
Occurs in
GIT carcinoids with liver metastasis
Non-GIT carcinoids
Predictors of periop complications
Carcinoid heart disease
High urinary 5-HIAA

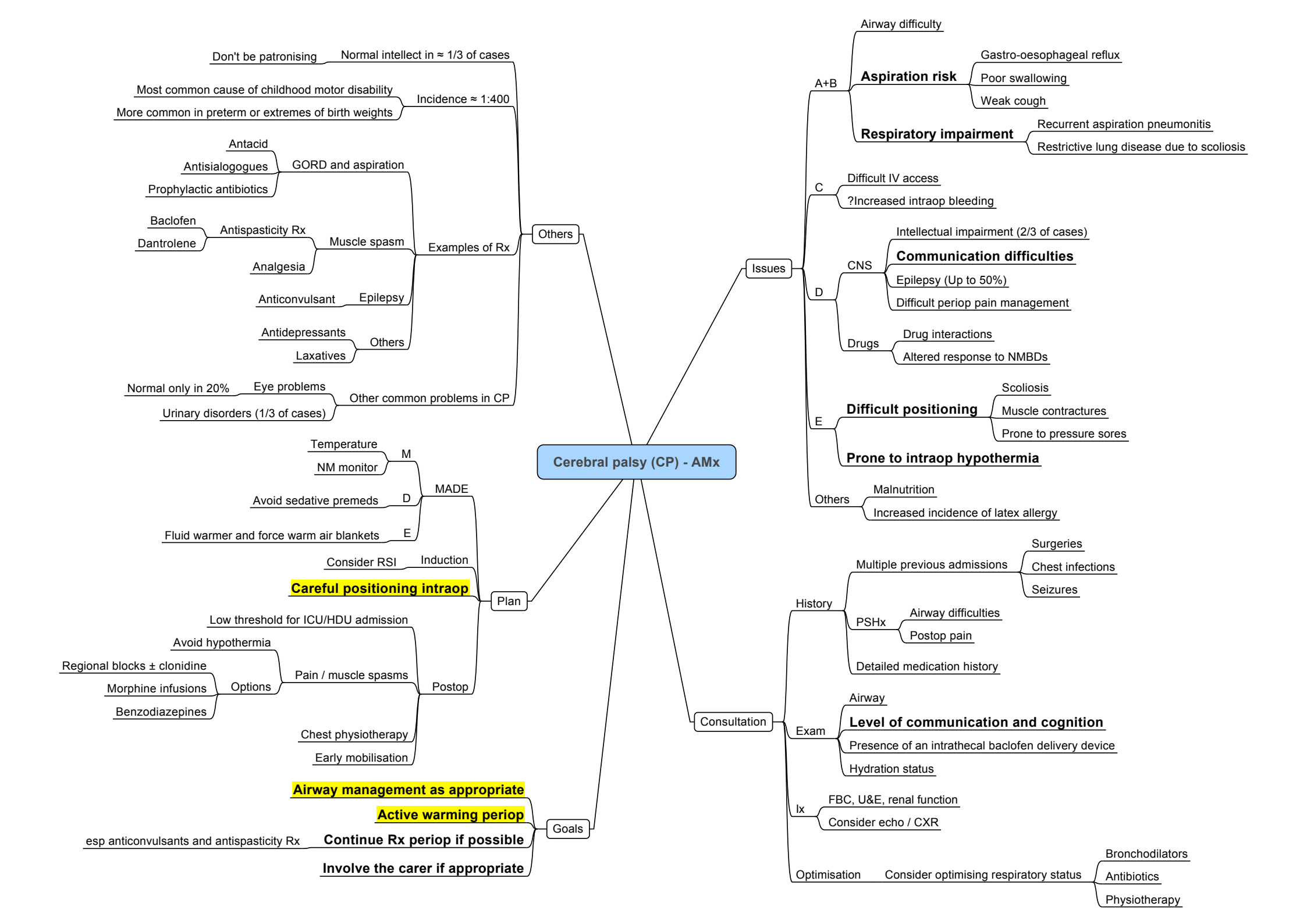

Cardiac temponade
Pericardiocentesis
Steps
Rarely used in acute situation
Temporising measure till open chest
USS guided
Needle insertion at infrasternal angle
Subxiphoid
Aim for left shoulder
Aspirate after feeling discrete pop
Insert pigtail drainage catheter
Seldinger technique
Complication
Pneumothorax
Punction of myocardium
S&S
Beck's triad
Hypotension
JVP distension
Muffled heart sound
Pulsus paradoxus
Tachycardia
Inability to lie supine
ECG changes
Electrical paradoxus
Pathognomonic
Low voltage QRS complex
ST segment changes
T wave inversion
Diagnosis
CXR
Widened mediastinum
Echocardiography
DDx
Tension pneumothorax
Cardiogenic shock / MI
PE
Anaphylaxis
Resuscitation
If cardiac arrest
Open chest immediately
i.e. Thoracotomy
Appropriate in penetrating chest wound
If post cardiac surgery
Suction chest drain using suction catheter
Goals
Maintain HR
Avoid bradycardia
Maintain preload
Avoid high pressure IPPV
IV fluid

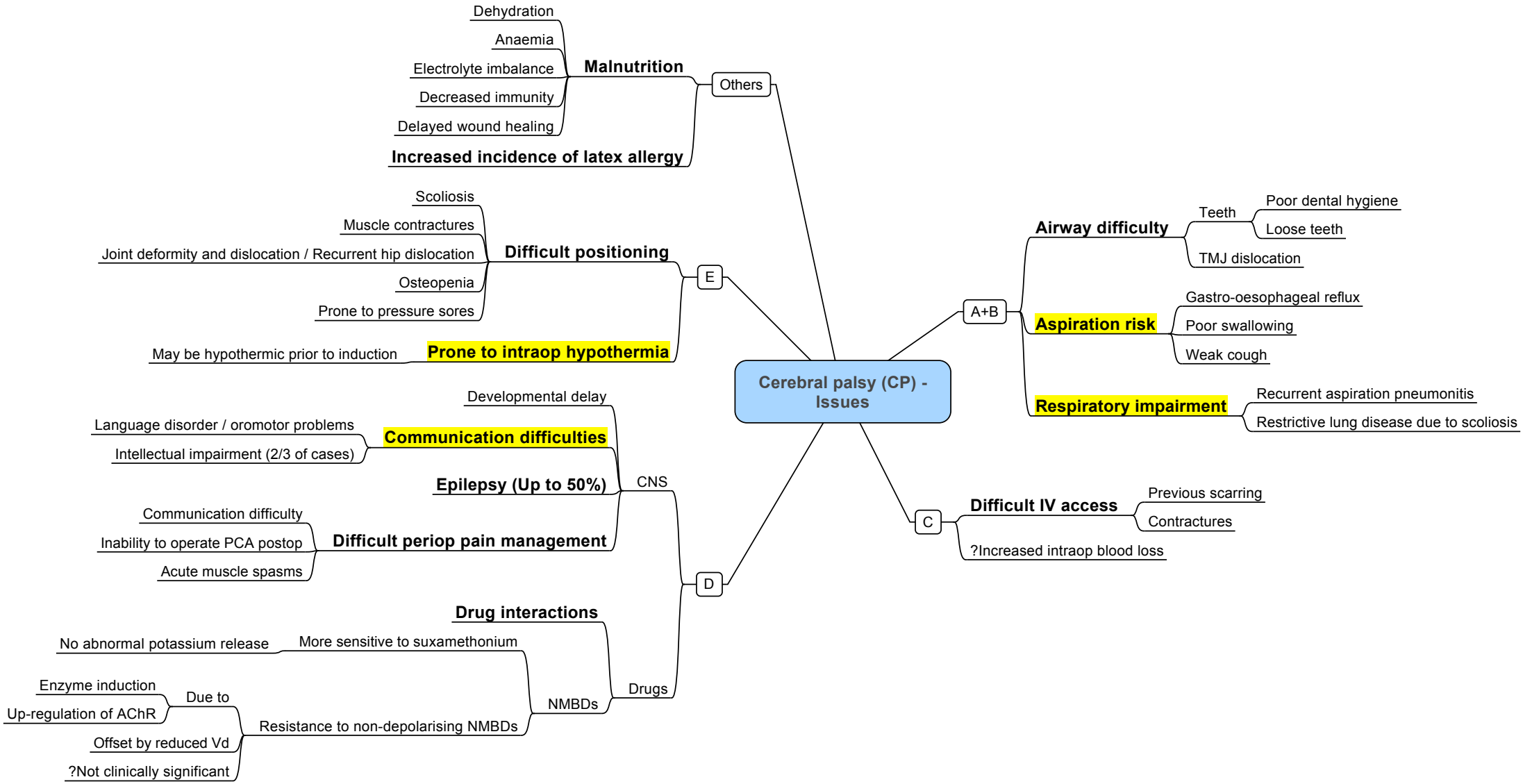

Cardiomyopathy, Dilated - AMx
Plan
M
Consider
Arterial line BP
Pulmonary artery catheter
Cardiac output monitor
D
Inotropic support often required
Dobutamine
Dopamine
Milrinone
Levosimendan
Consider
SNP
Avoid myocardial depressants
E
Temporary pacing
Immediately available
Goals
Maintain sinus rhythm
Adequate preload
Maintain normal afterload
Maintain contractility
Issues
Systolic dysfunction
Unlike dilated / hypertrophic cardiomyopathies
Dilation
Functional MR and TR
Thromboembolism
Arrhythmia
Common
Consultation
History
S&S of heart failure
Functional capacity
Associated
AF
Heart failure
Rx
Amiodarone
Anticoagulation
Pacemaker / AICD
Ix
Echo
LVEF
Functional MR / TR
Optimisation
Cardiology referral

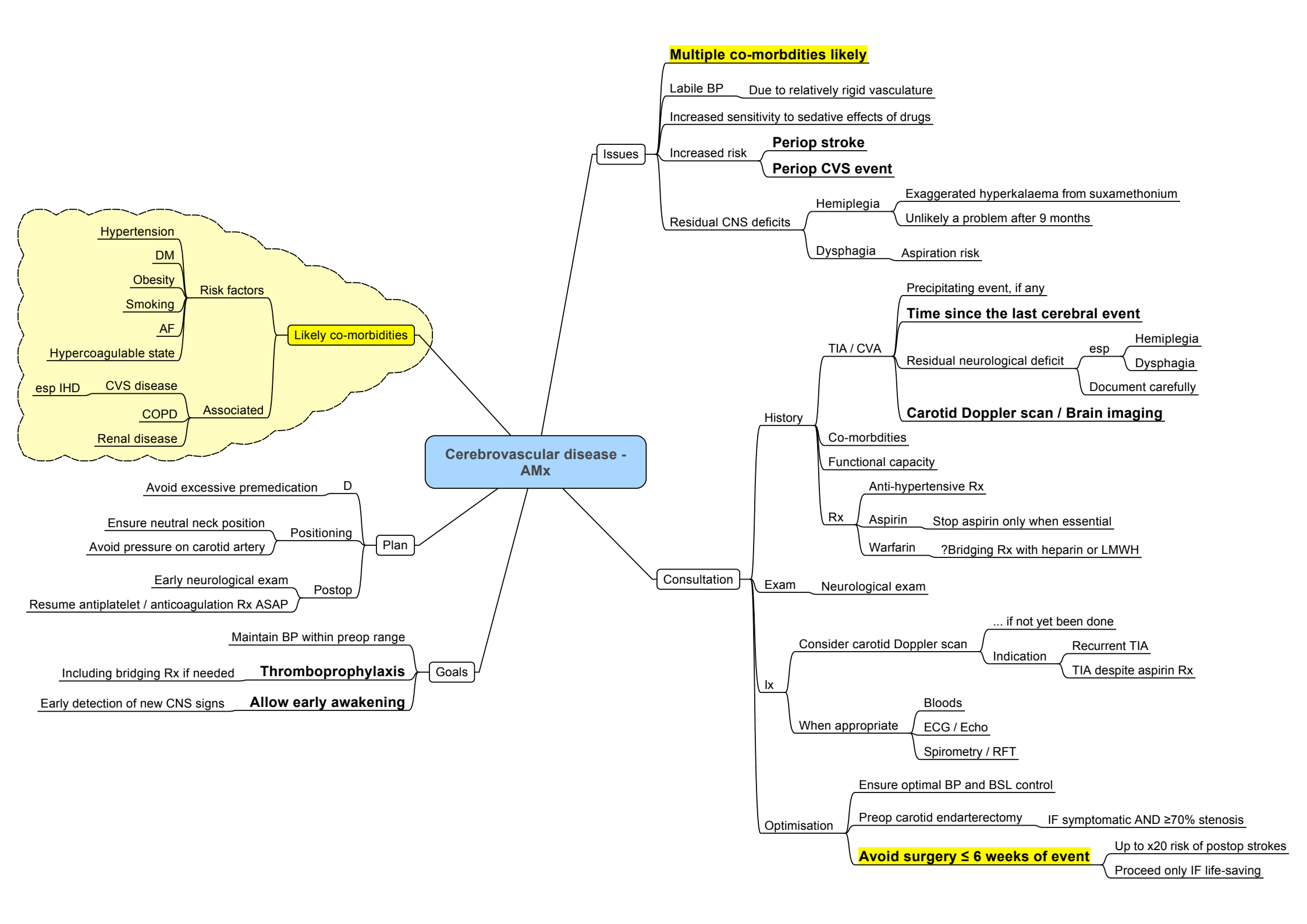

Cardiomyopathy, Hypertropic (HCM) - AMx
Consultation
History
S&S
Angina
Dyspnoea and fatigue
Syncope or dizziness
Palpitation
Functional capacity
Associated
AF
Heart failure
Rx
Anticoagulant Rx if AF
Pacemaker / AICD
Ix
ECG
LVH
ST segment or T wave abnormality
Echo
Degree of functional obstruction
MV-related
SAM
Systolic anterior motion
... of anterior MV leaflet
MR
Optimisation
Cardiology referral
... esp if newly diagnosed
Issues
Diastolic dysfunction
Low capacity and stiff ventricle
Often the main issue at late stage
Dynamic LVOT obstruction
Common (> 60%)
Worsened by
Increased contractility
Increased ventricular emptying
Prone to arrhythmia
Plan
Others
Risk of death = 2 to 3% per year
LSCS should generally be done under GA
MADE
M
Consider
Arterial line BP
PA catheter
TOE
Oesophageal Doppler
Cardiac output monitor
D
AVOID
Direct-acting alpha agonists preferred
Beta-agonists
e.g. Ephedrine
Vasodilators
Digoxin
Inotrope
e.g. Adrenaline
E
Temporary pacing equipment
Spinal / epidural can be VERY hazardous
Intraop
AVOID
Replace blood loss promptly
High airway pressure
PEEP
Valsalva manoeuvre
Postop
Analgesia
Avoid pain-induced tachycardia
Goals
Inotrope CONTRAINDICATED
Avoid tachycardia
Keep high normal preload and afterload
Maintain sinus rhythm
Immediate DC cardioversion if periop AF

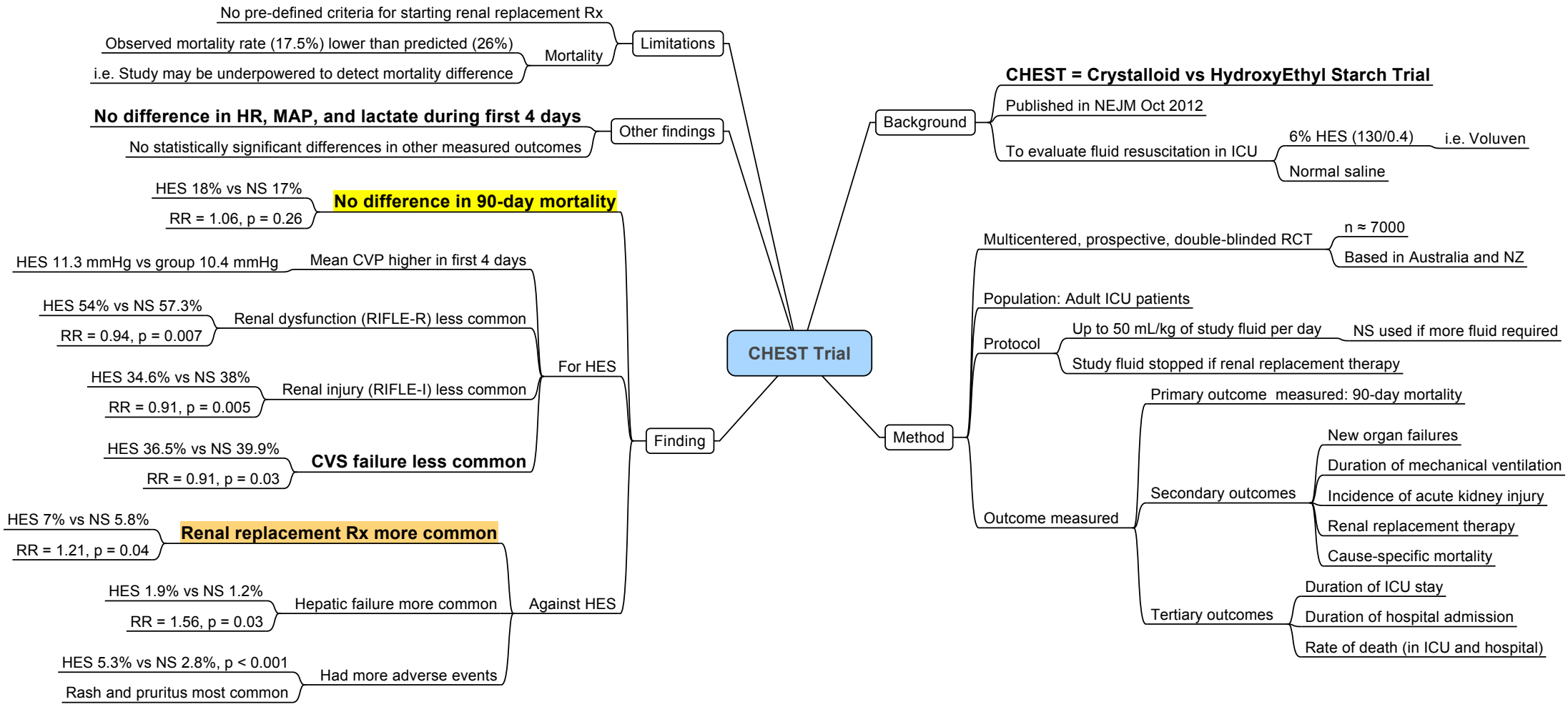

General anaesthesia is hazardous
Cardiomyopathy, Restrictive - AMx
Others
Rare condition
Systolic function usually normal (but not always)
Issues
Diastolic dysfunction
Right heart failure
Pulmonary hypertension
Thromboembolism
Depending on underlying cause
Plan
M
Keep spontanous breathing if possible
Consider
Arterial line BP
TOE
PA catheter
Cardiac output monitor
D
Ketamine may be useful
Increase contractility
Increase SVR
AVOID (or use cautiously)
Vasodilators
Digoxin
Beta blockers
Morphine and isoflurane
E
Temporary pacing equipments
Goals
Maintain preload
High normal afterload
Maintain normal HR
Maintain sinus rhythm
Consultation
History
Underlying cause
e.g.
Amyloidosis
Most common
Eosinophilia
Sarcoidosis
S&S
Signs of right heart failure
Functional capacity
Rx
Amiodarone
Anticogulation
Pacemaker / AICD
Ix
ECG
Echo
Optimisation
Cardiology referral

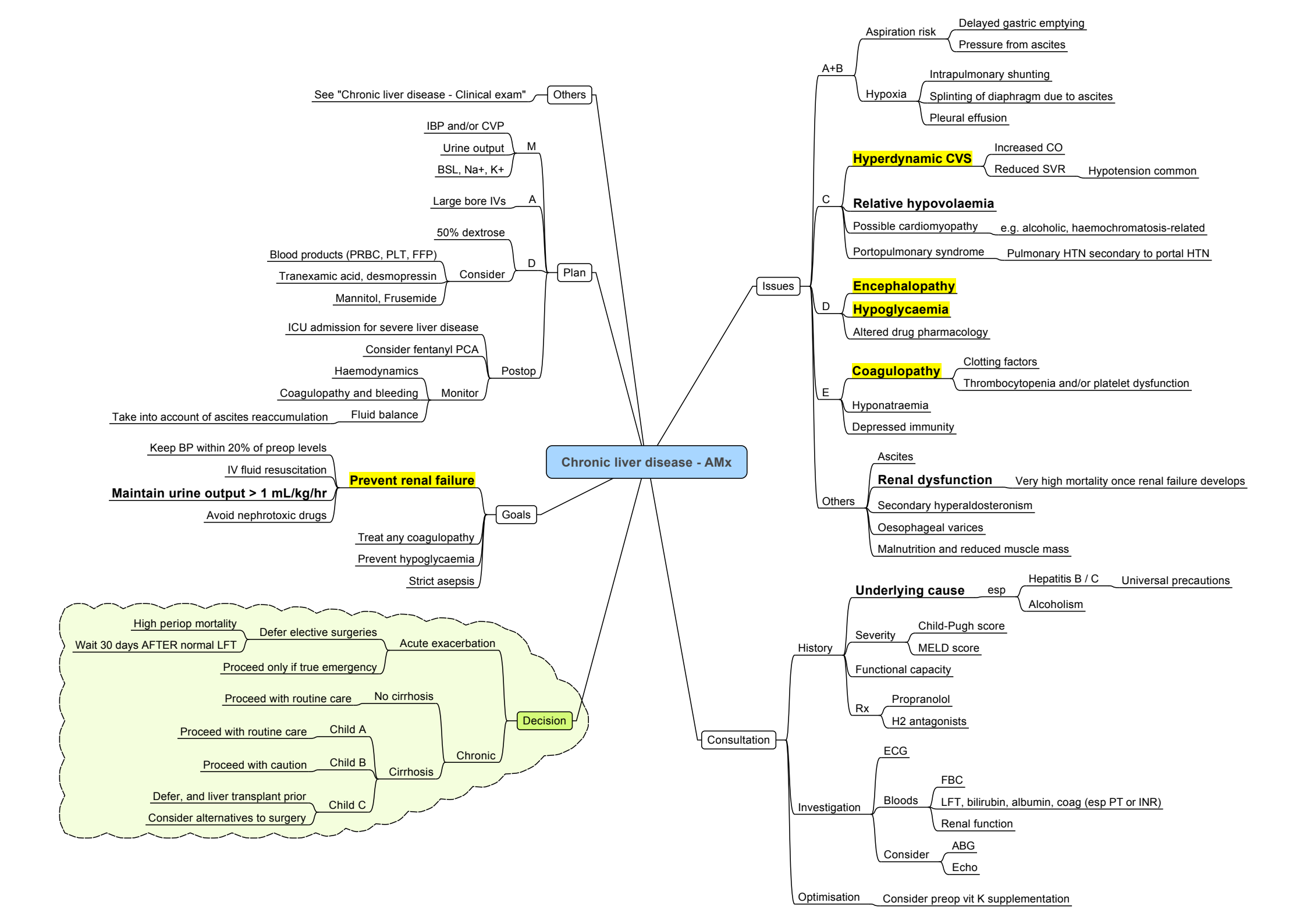

Cauda equina syndrome
PP
MOA
Acute loss of function due to compression of cauda equina
Functions affected
Lower limb sensory and motor function
Sensation to perineum and genitals
Voluntary and involuntary
Micturition
Defaecation
Sexual function
Aetiology
Vertebral disc herniation
Neoplasm
Inflammatory process
e.g. spinal abscess
Lumbar spinal stenosis
Vascular
Spinal haemorrhage
Spinal arteriovenous malformation
Trauma
Iatrogenic
Epidural steroid injection
Complication from neuraxial block
SS
Highly variable
Include
Lower limb weakness or paraplegia
Detrusor weakness
Loss of anal tone
Saddle anaesthesia
Sciatic leg pain
Bilateral or unilateral
Anatomy
Cauda equina contains
Lumbar plexus
Sacral plexus
Conus medullaris
Overlies body of L1
Termination of spinal cord
Tapers off to form filum terminale
Clinical
Diagnosis
CT or MRI
Rx
Immobilisation if trauma involved
Urgent surgical decompression
Prognosis
Depends on
Severity of compression
Duration of compression

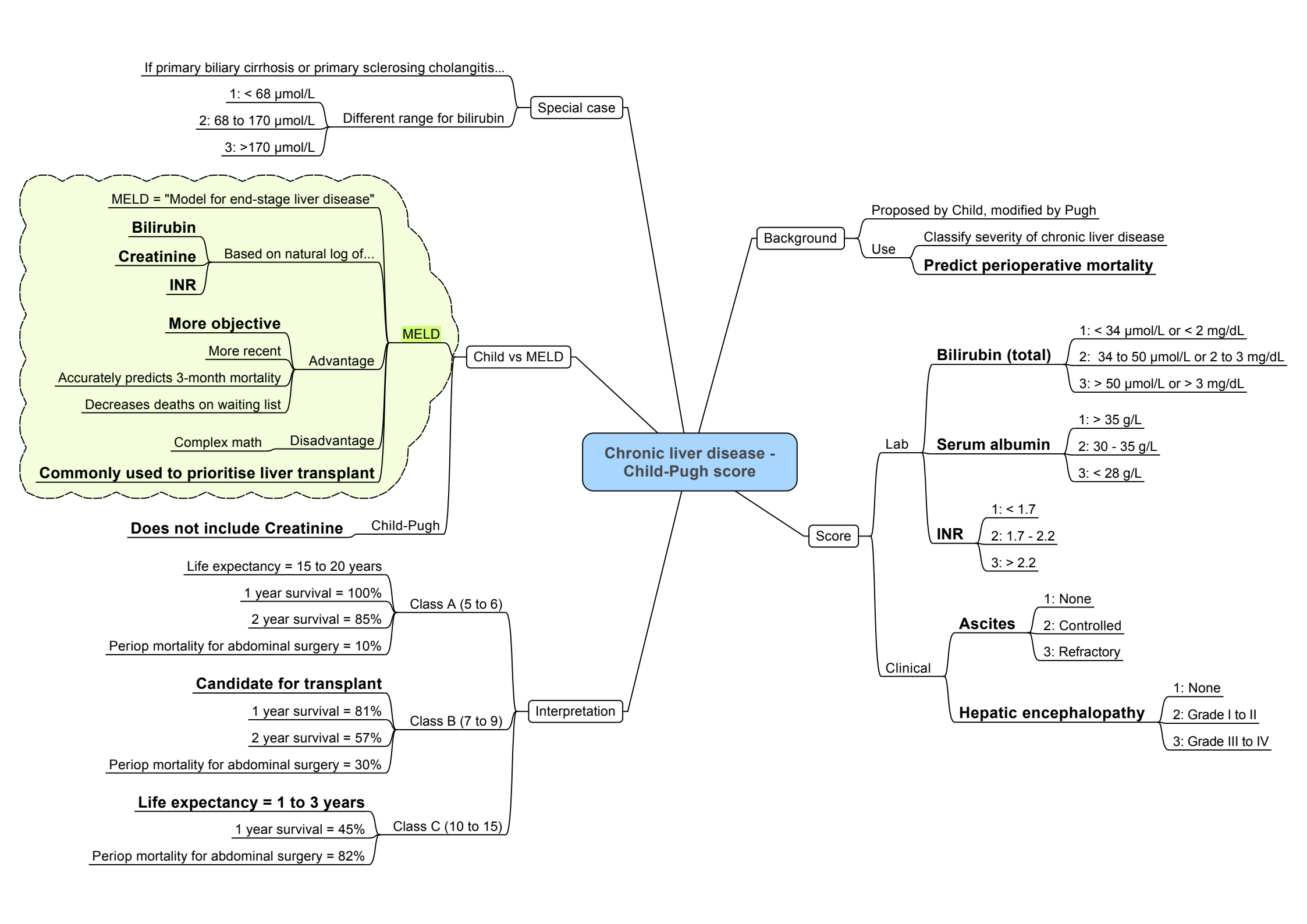

Cerebral palsy (CP) - AMx
Issues
A+B
Airway difficulty
Aspiration risk
Gastro-oesophageal reflux
Poor swallowing
Weak cough
Respiratory impairment
Recurrent aspiration pneumonitis
Restrictive lung disease due to scoliosis
C
Difficult IV access
?Increased intraop bleeding
D
CNS
Intellectual impairment (2/3 of cases)
Communication difficulties
Epilepsy (Up to 50%)
Difficult periop pain management
Drugs
Drug interactions
Altered response to NMBDs
E
Difficult positioning
Scoliosis
Muscle contractures
Prone to pressure sores
Prone to intraop hypothermia
Others
Malnutrition
Increased incidence of latex allergy
Consultation
History
Multiple previous admissions
Surgeries
Chest infections
Seizures
PSHx
Airway difficulties
Postop pain
Detailed medication history
Exam
Airway
Level of communication and cognition
Presence of an intrathecal baclofen delivery device
Hydration status
Ix
FBC, U&E, renal function
Consider echo / CXR
Optimisation
Consider optimising respiratory status
Bronchodilators
Antibiotics
Physiotherapy
Goals
Airway management as appropriate
Active warming periop
Continue Rx periop if possible
esp anticonvulsants and antispasticity Rx
Involve the carer if appropriate
Plan
MADE
M
Temperature
NM monitor
D
Avoid sedative premeds
E
Fluid warmer and force warm air blankets
Induction
Consider RSI
Careful positioning intraop
Postop
Low threshold for ICU/HDU admission
Pain / muscle spasms
Avoid hypothermia
Options
Regional blocks ± clonidine
Morphine infusions
Benzodiazepines
Chest physiotherapy
Early mobilisation
Others
Incidence ≈ 1:400
Most common cause of childhood motor disability
More common in preterm or extremes of birth weights
Normal intellect in ≈ 1/3 of cases
Don't be patronising
Examples of Rx
GORD and aspiration
Antacid
Antisialogogues
Prophylactic antibiotics
Muscle spasm
Antispasticity Rx
Baclofen
Dantrolene
Analgesia
Epilepsy
Anticonvulsant
Others
Antidepressants
Laxatives
Other common problems in CP
Eye problems
Normal only in 20%
Urinary disorders (1/3 of cases)

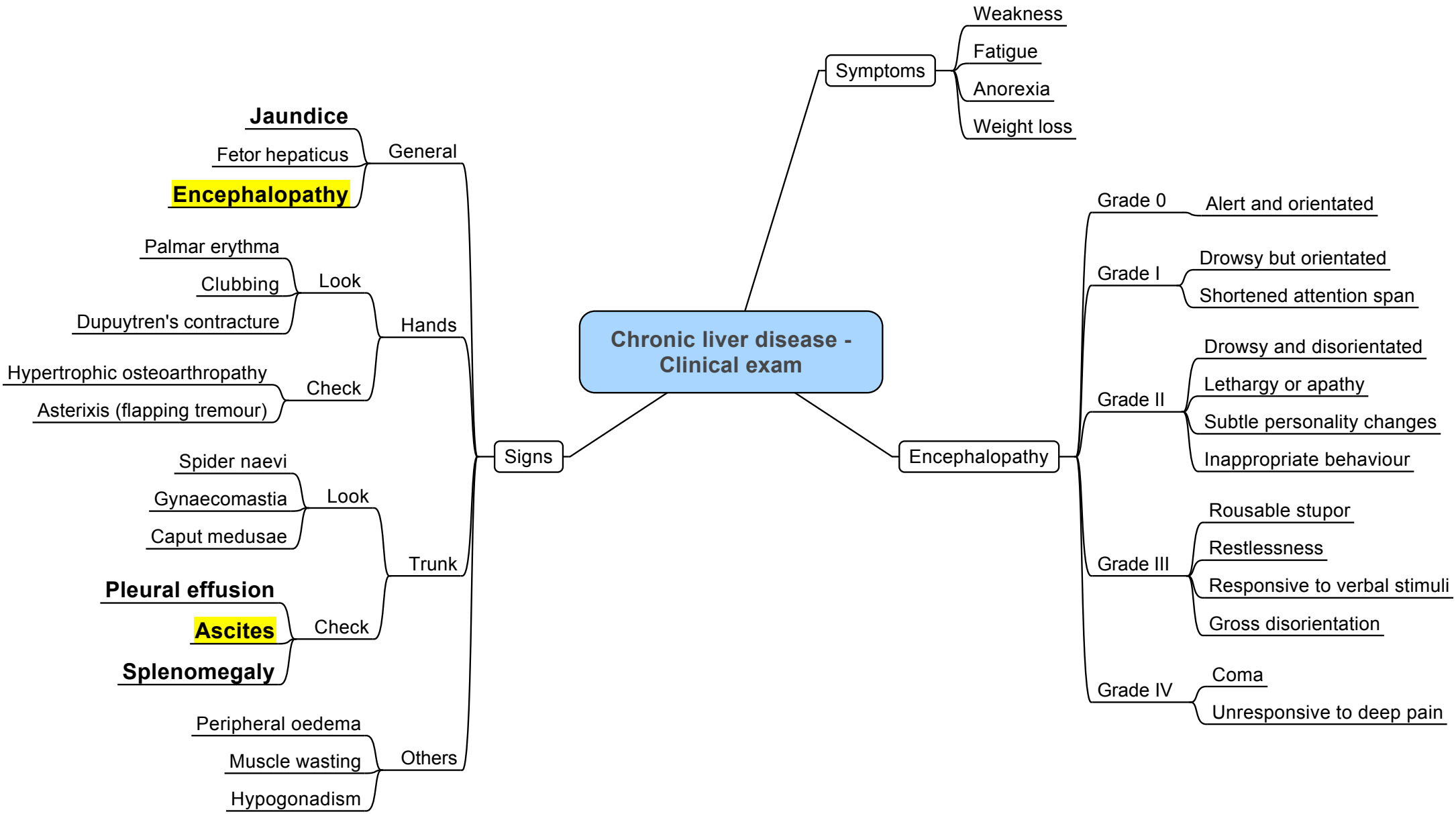

Cerebral palsy (CP) - Issues
A+B
Airway difficulty
Teeth
Poor dental hygiene
Loose teeth
TMJ dislocation
Aspiration risk
Gastro-oesophageal reflux
Poor swallowing
Weak cough
Respiratory impairment
Recurrent aspiration pneumonitis
Restrictive lung disease due to scoliosis
C
Difficult IV access
Previous scarring
Contractures
?Increased intraop blood loss
D
CNS
Epilepsy (Up to 50%)
Communication difficulties
Language disorder / oromotor problems
Intellectual impairment (2/3 of cases)
Developmental delay
Difficult periop pain management
Communication difficulty
Inability to operate PCA postop
Acute muscle spasms
Drugs
NMBDs
Drug interactions
More sensitive to suxamethonium
No abnormal potassium release
Resistance to non-depolarising NMBDs
Due to
Enzyme induction
Up-regulation of AChR
Offset by reduced Vd
?Not clinically significant
E
Difficult positioning
Scoliosis
Muscle contractures
Joint deformity and dislocation / Recurrent hip dislocation
Osteopenia
Prone to pressure sores
Prone to intraop hypothermia
May be hypothermic prior to induction
Others
Malnutrition
Dehydration
Anaemia
Electrolyte imbalance
Decreased immunity
Delayed wound healing
Increased incidence of latex allergy

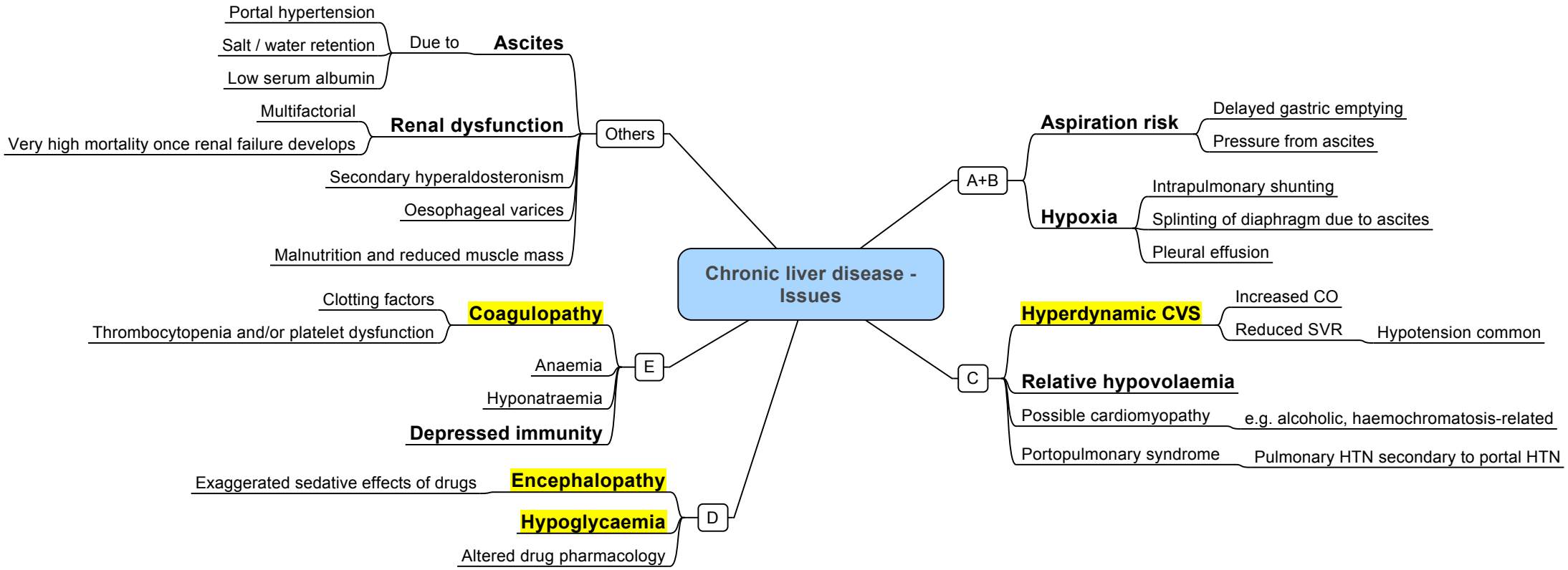

Cerebrovascular disease - AMx
Likely co-morbidities
Risk factors
Hypertension
DM
Obesity
Smoking
AF
Hypercoagulable state
Associated
esp IHD
CVS disease
COPD
Renal disease
Plan
D
Avoid excessive premedication
Positioning
Ensure neutral neck position
Avoid pressure on carotid artery
Postop
Early neurological exam
Resume antiplatelet / anticoagulation Rx ASAP
Goals
Maintain BP within preop range
Thromboprophylaxis
Including bridging Rx if needed
Allow early awakening
Early detection of new CNS signs
Issues
Multiple co-morbdities likely
Labile BP
Due to relatively rigid vasculature
Increased sensitivity to sedative effects of drugs
Increased risk
Periop stroke
Periop CVS event
Residual CNS deficits
Hemiplegia
Exaggerated hyperkalaema from suxamethonium
Unlikely a problem after 9 months
Dysphagia
Aspiration risk
Consultation
History
TIA / CVA
Precipitating event, if any
Time since the last cerebral event
Residual neurological deficit
esp
Hemiplegia
Dysphagia
Document carefully
Carotid Doppler scan / Brain imaging
Co-morbdities
Functional capacity
Rx
Anti-hypertensive Rx
Aspirin
Stop aspirin only when essential
Warfarin
?Bridging Rx with heparin or LMWH
Exam
Neurological exam
Ix
Consider carotid Doppler scan
... if not yet been done
Indication
Recurrent TIA
TIA despite aspirin Rx
When appropriate
Bloods
ECG / Echo
Spirometry / RFT
Optimisation
Ensure optimal BP and BSL control
Preop carotid endarterectomy
IF symptomatic AND ≥70% stenosis
Avoid surgery ≤ 6 weeks of event
Up to x20 risk of postop strokes
Proceed only IF life-saving

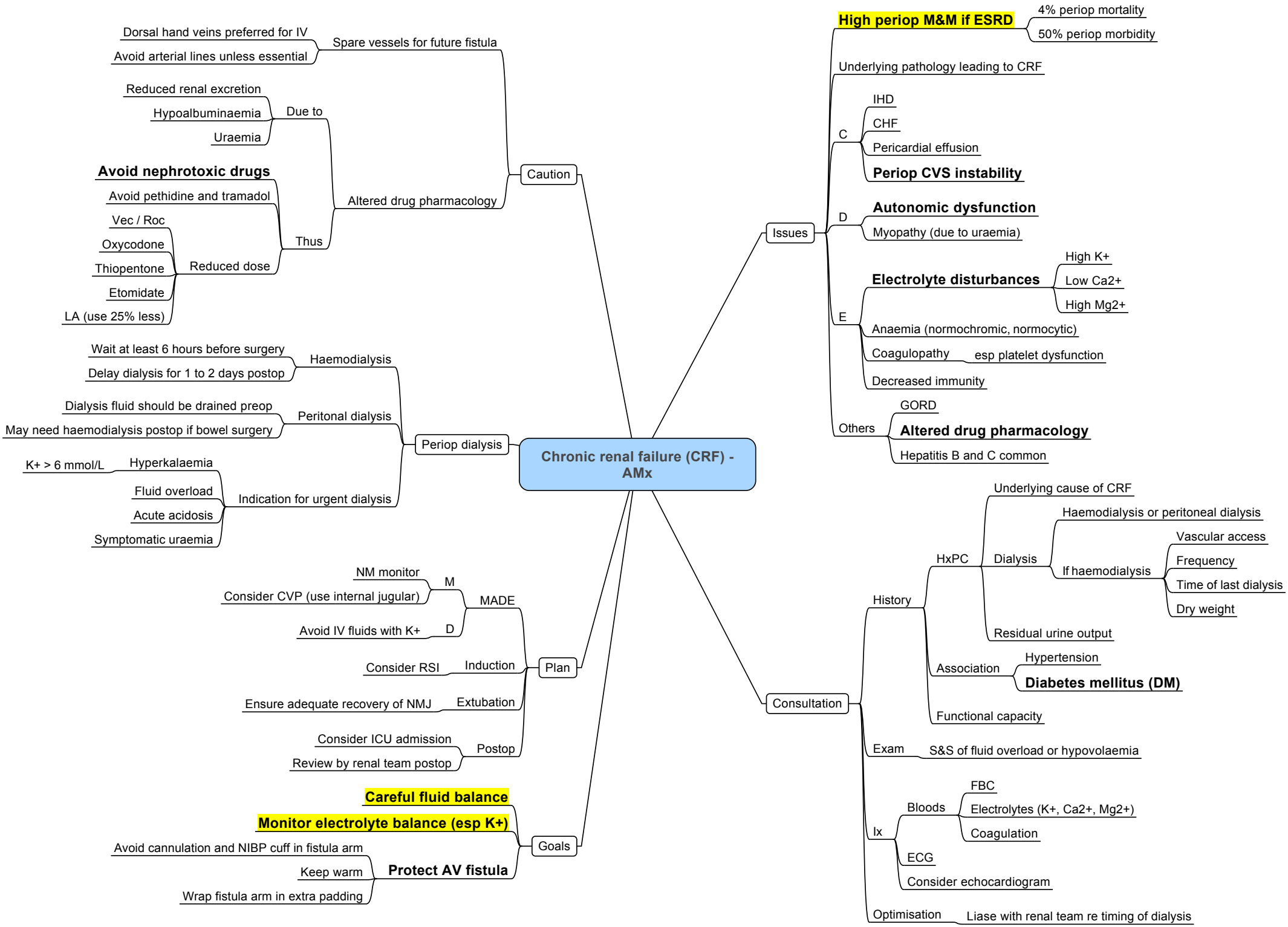

CHEST Trial
Background
CHEST = Crystalloid vs HydroxyEthyl Starch Trial
Published in NEJM Oct 2012
To evaluate fluid resuscitation in ICU
6% HES (130/0.4)
i.e. Voluven
Normal saline
Method
Multicentered, prospective, double-blinded RCT
n ≈ 7000
Based in Australia and NZ
Population: Adult ICU patients
Protocol
Up to 50 mL/kg of study fluid per day
NS used if more fluid required
Study fluid stopped if renal replacement therapy
Outcome measured
Primary outcome measured: 90-day mortality
Secondary outcomes
New organ failures
Duration of mechanical ventilation
Incidence of acute kidney injury
Renal replacement therapy
Cause-specific mortality
Tertiary outcomes
Duration of ICU stay
Duration of hospital admission
Rate of death (in ICU and hospital)
Finding
No difference in 90-day mortality
HES 18% vs NS 17%
RR = 1.06, p = 0.26
For HES
Mean CVP higher in first 4 days
HES 11.3 mmHg vs group 10.4 mmHg
Renal dysfunction (RIFLE-R) less common
HES 54% vs NS 57.3%
RR = 0.94, p = 0.007
Renal injury (RIFLE-I) less common
HES 34.6% vs NS 38%
RR = 0.91, p = 0.005
CVS failure less common
HES 36.5% vs NS 39.9%
RR = 0.91, p = 0.03
Against HES
Renal replacement Rx more common
HES 7% vs NS 5.8%
RR = 1.21, p = 0.04
Hepatic failure more common
HES 1.9% vs NS 1.2%
RR = 1.56, p = 0.03
Had more adverse events
HES 5.3% vs NS 2.8%, p < 0.001
Rash and pruritus most common
Limitations
Mortality
No pre-defined criteria for starting renal replacement Rx
Observed mortality rate (17.5%) lower than predicted (26%)
i.e. Study may be underpowered to detect mortality difference
Other findings
No difference in HR, MAP, and lactate during first 4 days
No statistically significant differences in other measured outcomes

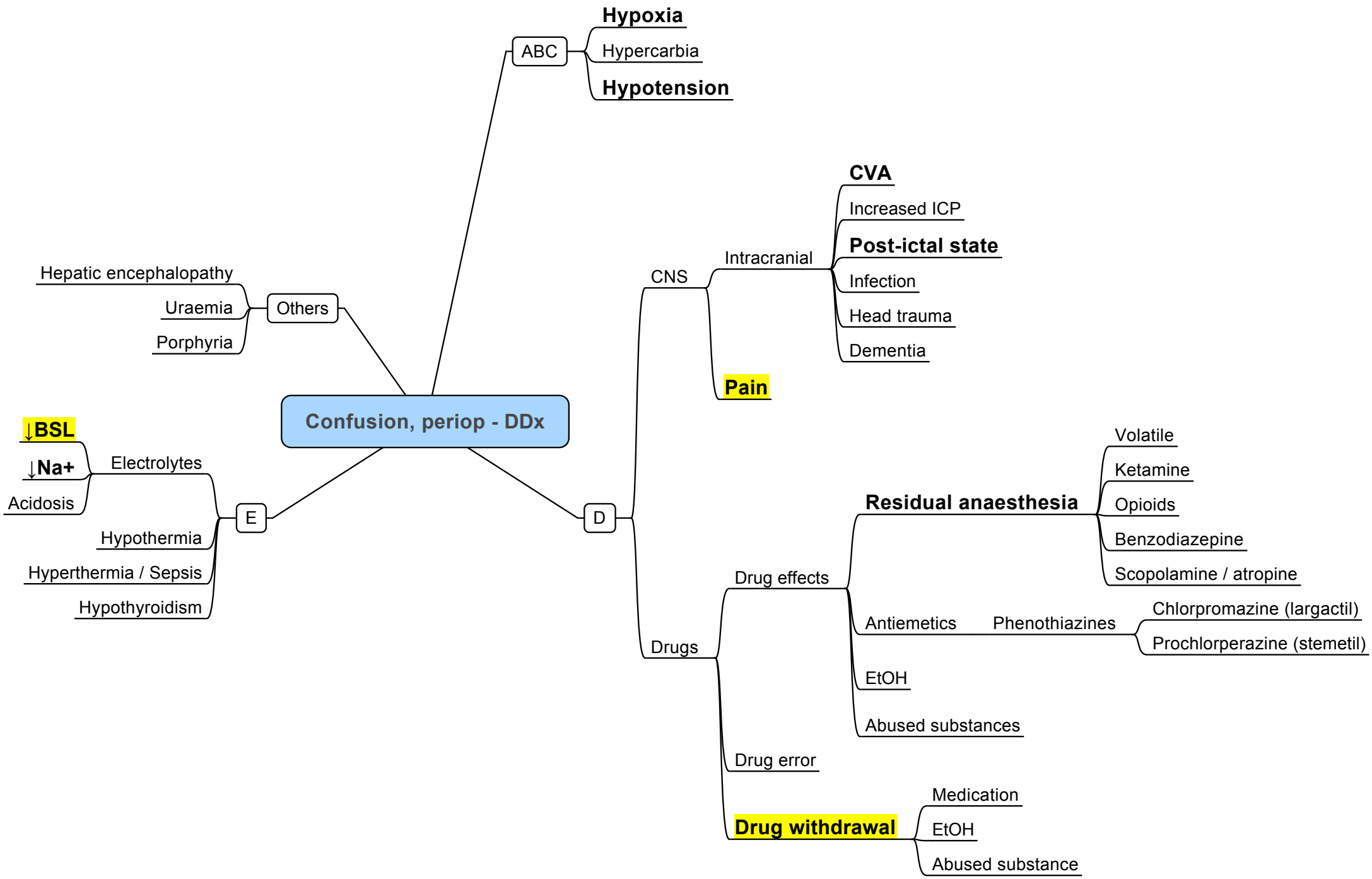

Chronic liver disease - AMx
Issues
A+B
Aspiration risk
Delayed gastric emptying
Pressure from ascites
Hypoxia
Intrapulmonary shunting
Splinting of diaphragm due to ascites
Pleural effusion
C
Hyperdynamic CVS
Increased CO
Reduced SVR
Hypotension common
Relative hypovolaemia
Possible cardiomyopathy
e.g. alcoholic, haemochromatosis-related
Portopulmonary syndrome
Pulmonary HTN secondary to portal HTN
D
Encephalopathy
Hypoglycaemia
Altered drug pharmacology
E
Coagulopathy
Clotting factors
Thrombocytopenia and/or platelet dysfunction
Hyponatraemia
Depressed immunity
Others
Ascites
Renal dysfunction
Very high mortality once renal failure develops
Secondary hyperaldosteronism
Oesophageal varices
Malnutrition and reduced muscle mass
Consultation
History
Underlying cause
esp
Hepatitis B / C
Universal precautions
Alcoholism
Severity
Child-Pugh score
MELD score
Functional capacity
Rx
Propranolol
H2 antagonists
Investigation
ECG
Bloods
FBC
LFT, bilirubin, albumin, coag (esp PT or INR)
Renal function
Consider
ABG
Echo
Optimisation
Consider preop vit K supplementation
Plan
Others
See "Chronic liver disease - Clinical exam"
M
IBP and/or CVP
Urine output
BSL, Na+, K+
A
Large bore IVs
D
50% dextrose
Consider
Blood products (PRBC, PLT, FFP)
Tranexamic acid, desmopressin
Mannitol, Frusemide
Postop
ICU admission for severe liver disease
Consider fentanyl PCA
Monitor
Haemodynamics
Coagulopathy and bleeding
Fluid balance
Take into account of ascites reaccumulation
Goals
Prevent renal failure
Keep BP within 20% of preop levels
IV fluid resuscitation
Maintain urine output > 1 mL/kg/hr
Avoid nephrotoxic drugs
Treat any coagulopathy
Prevent hypoglycaemia
Strict asepsis
Decision
Acute exacerbation
High periop mortality
Defer elective surgeries
Wait 30 days AFTER normal LFT
Proceed only if true emergency
Chronic
No cirrhosis
Proceed with routine care
Cirrhosis
Child A
Proceed with routine care
Child B
Proceed with caution
Child C
Defer, and liver transplant prior
Consider alternatives to surgery

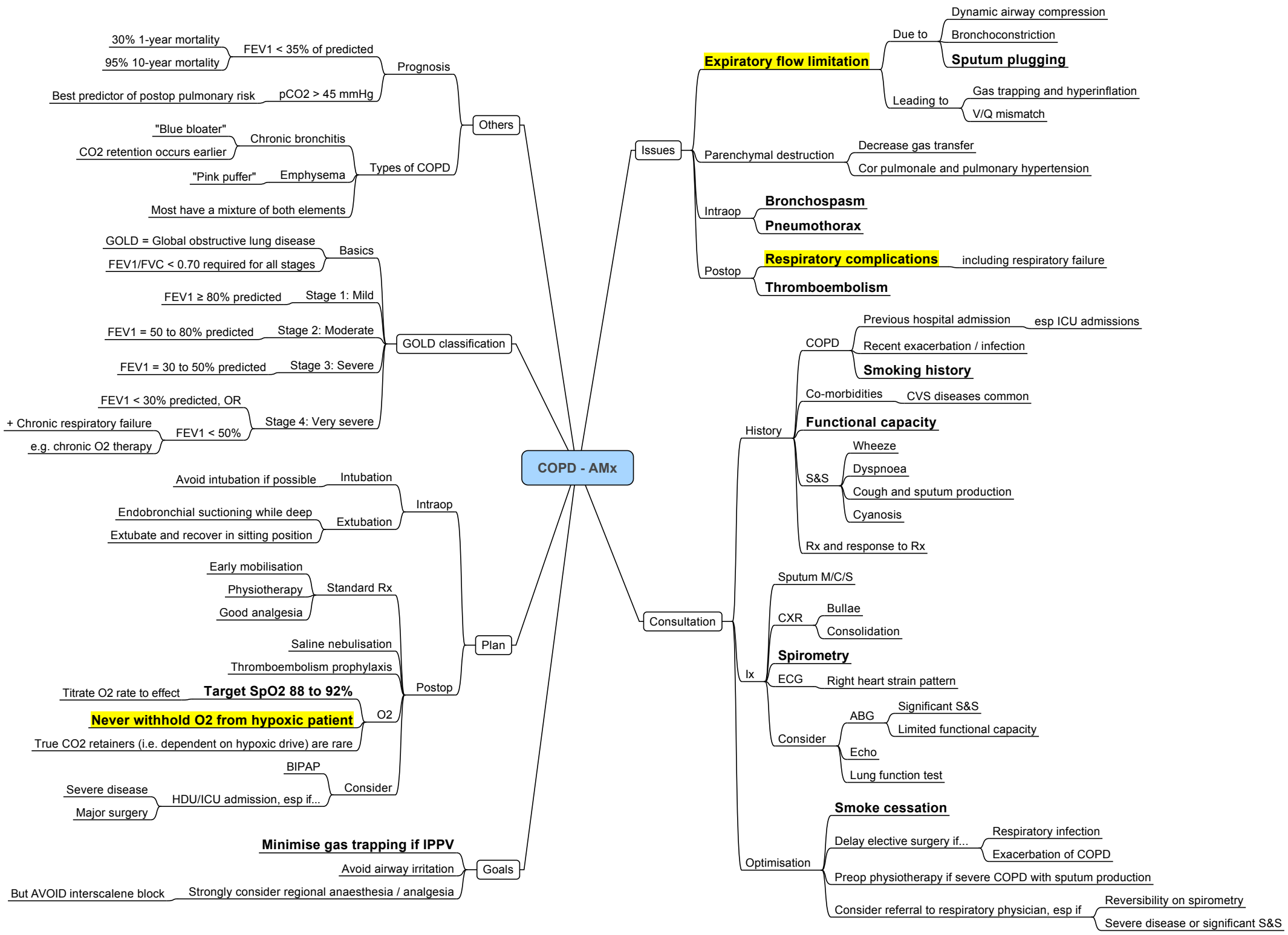

Chronic liver disease - Child-Pugh score
Background
Proposed by Child, modified by Pugh
Use
Classify severity of chronic liver disease
Predict perioperative mortality
Score
Lab
Bilirubin (total)
1: < 34 µmol/L or < 2 mg/dL
2: 34 to 50 µmol/L or 2 to 3 mg/dL
3: > 50 µmol/L or > 3 mg/dL
Serum albumin
1: > 35 g/L
2: 30 - 35 g/L
3: < 28 g/L
INR
1: < 1.7
2: 1.7 - 2.2
3: > 2.2
Clinical
Ascites
1: None
2: Controlled
3: Refractory
Hepatic encephalopathy
1: None
2: Grade I to II
3: Grade III to IV
Interpretation
Class A (5 to 6)
Life expectancy = 15 to 20 years
1 year survival = 100%
2 year survival = 85%
Periop mortality for abdominal surgery = 10%
Class B (7 to 9)
Candidate for transplant
1 year survival = 81%
2 year survival = 57%
Periop mortality for abdominal surgery = 30%
Class C (10 to 15)
Life expectancy = 1 to 3 years
1 year survival = 45%
Periop mortality for abdominal surgery = 82%
Child vs MELD
MELD
MELD = "Model for end-stage liver disease"
Based on natural log of...
Bilirubin
Creatinine
INR
Advantage
More objective
More recent
Accurately predicts 3-month mortality
Decreases deaths on waiting list
Disadvantage
Complex math
Commonly used to prioritise liver transplant
Child-Pugh
Does not include Creatinine
Special case
Different range for bilirubin
If primary biliary cirrhosis or primary sclerosing cholangitis...
1: < 68 µmol/L
2: 68 to 170 µmol/L
3: >170 µmol/L

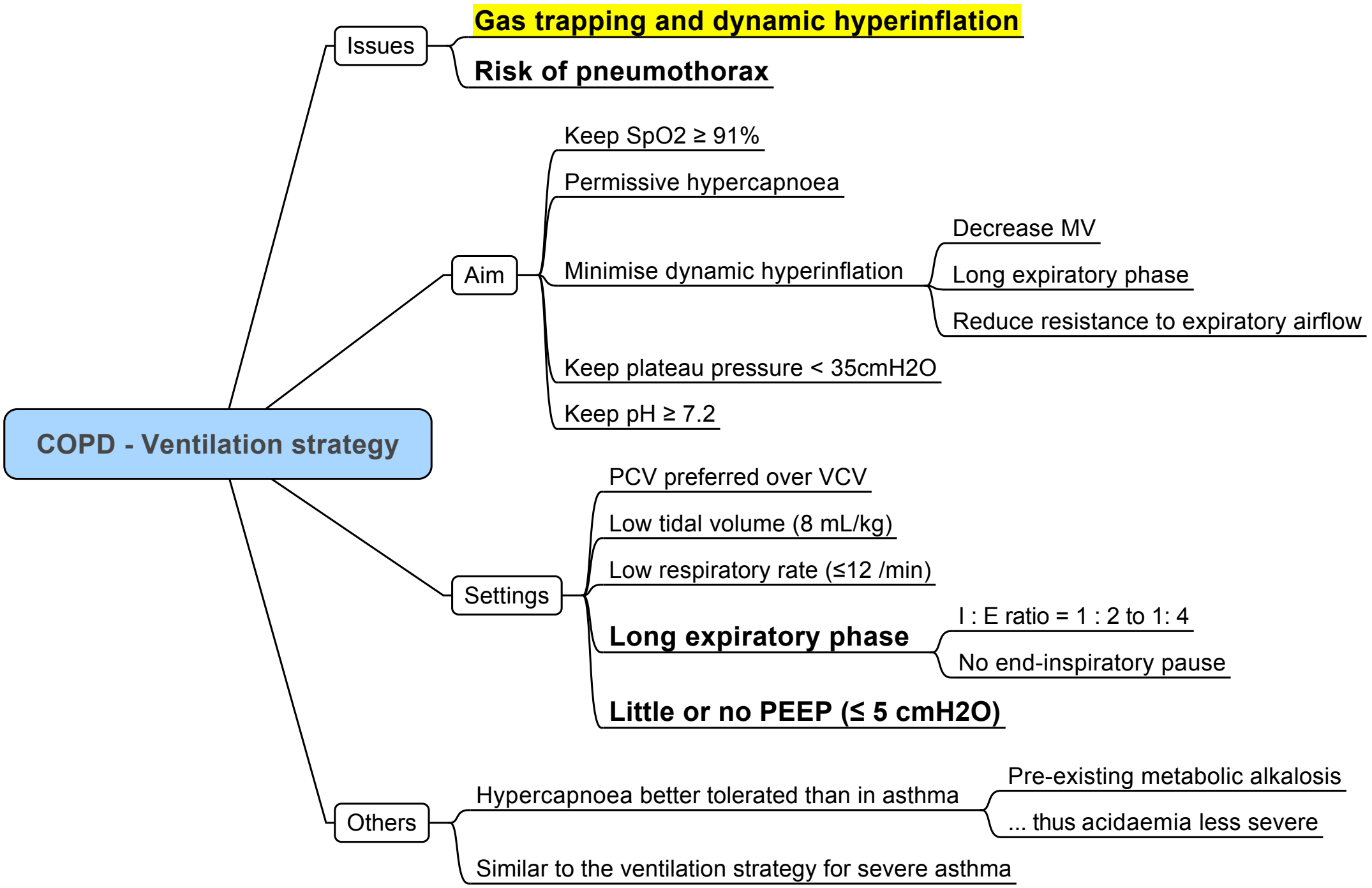

Chronic liver disease - Clinical exam
Symptoms
Weakness
Fatigue
Anorexia
Weight loss
Signs
General
Jaundice
Fetor hepaticus
Encephalopathy
Hands
Look
Palmar erythma
Clubbing
Dupuytren's contracture
Check
Hypertrophic osteoarthropathy
Asterixis (flapping tremour)
Trunk
Look
Spider naevi
Gynaecomastia
Caput medusae
Check
Pleural effusion
Ascites
Splenomegaly
Others
Peripheral oedema
Muscle wasting
Hypogonadism
Encephalopathy
Grade 0
Alert and orientated
Grade I
Drowsy but orientated
Shortened attention span
Grade II
Drowsy and disorientated
Lethargy or apathy
Subtle personality changes
Inappropriate behaviour
Grade III
Rousable stupor
Restlessness
Responsive to verbal stimuli
Gross disorientation
Grade IV
Coma
Unresponsive to deep pain

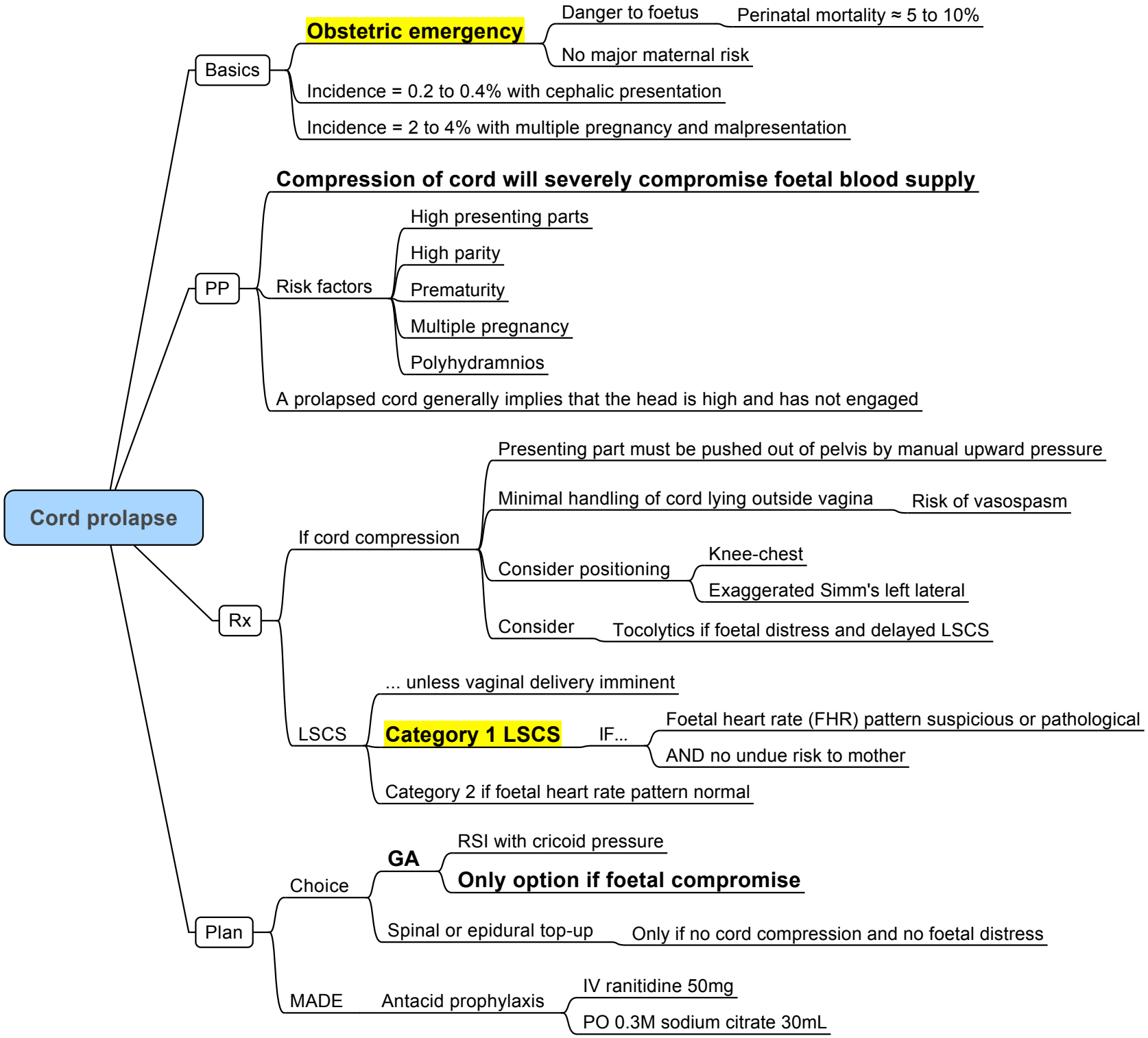

Chronic liver disease - Issues
A+B
Aspiration risk
Delayed gastric emptying
Pressure from ascites
Hypoxia
Intrapulmonary shunting
Splinting of diaphragm due to ascites
Pleural effusion
C
Hyperdynamic CVS
Increased CO
Reduced SVR
Hypotension common
Relative hypovolaemia
Possible cardiomyopathy
e.g. alcoholic, haemochromatosis-related
Portopulmonary syndrome
Pulmonary HTN secondary to portal HTN
D
Encephalopathy
Exaggerated sedative effects of drugs
Hypoglycaemia
Altered drug pharmacology
E
Coagulopathy
Clotting factors
Thrombocytopenia and/or platelet dysfunction
Anaemia
Hyponatraemia
Depressed immunity
Others
Ascites
Due to
Portal hypertension
Salt / water retention
Low serum albumin
Renal dysfunction
Multifactorial
Very high mortality once renal failure develops
Secondary hyperaldosteronism
Oesophageal varices
Malnutrition and reduced muscle mass

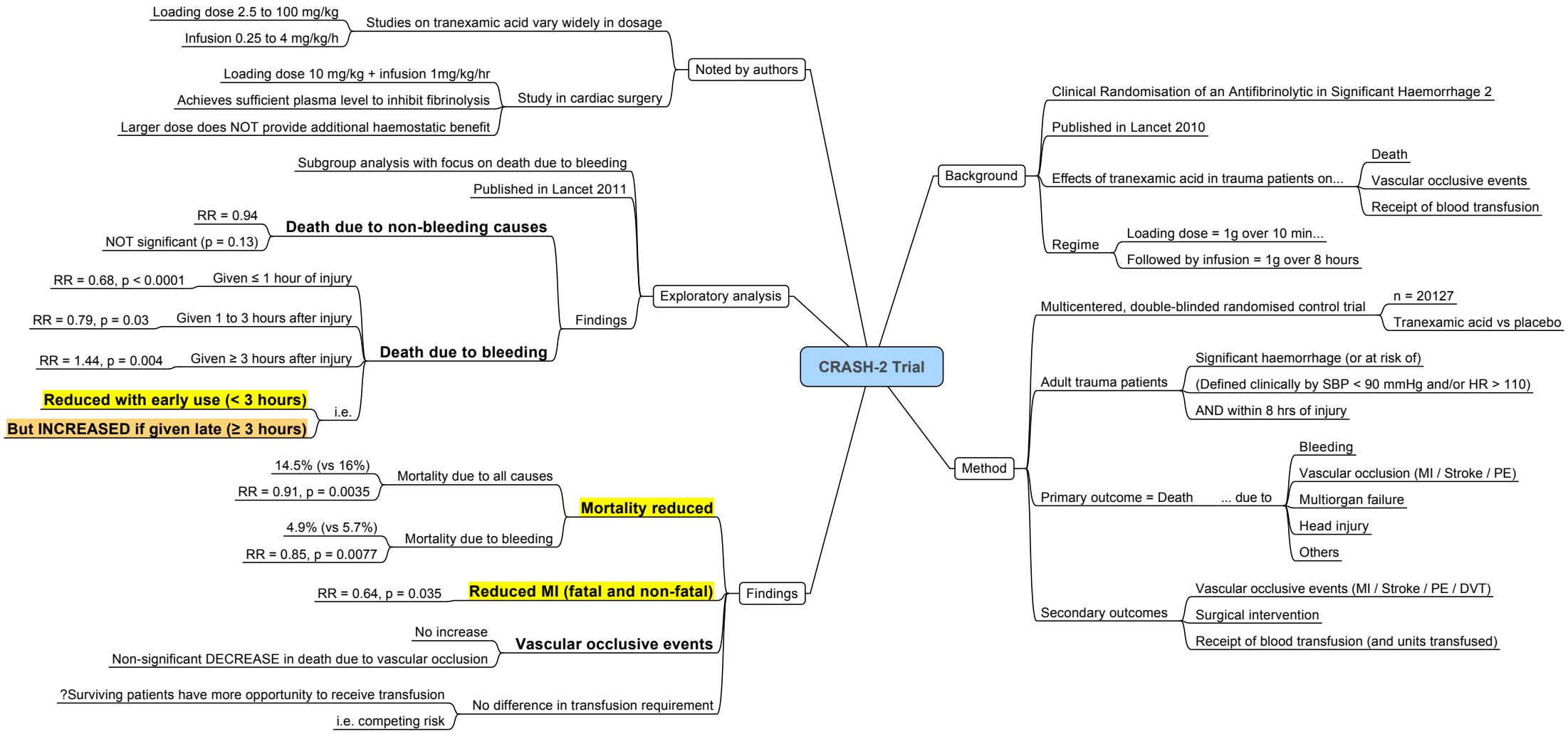

Chronic renal failure (CRF) - AMx
Caution
Spare vessels for future fistula
Dorsal hand veins preferred for IV
Avoid arterial lines unless essential
Altered drug pharmacology
Due to
Reduced renal excretion
Hypoalbuminaemia
Uraemia
Thus
Avoid nephrotoxic drugs
Avoid pethidine and tramadol
Reduced dose
Vec / Roc
Oxycodone
Thiopentone
Etomidate
LA (use 25% less)
Periop dialysis
Haemodialysis
Wait at least 6 hours before surgery
Delay dialysis for 1 to 2 days postop
Peritonal dialysis
Dialysis fluid should be drained preop
May need haemodialysis postop if bowel surgery
Indication for urgent dialysis
Hyperkalaemia
K+ > 6 mmol/L
Fluid overload
Acute acidosis
Symptomatic uraemia
Issues
High periop M&M if ESRD
4% periop mortality
50% periop morbidity
Underlying pathology leading to CRF
C
IHD
CHF
Pericardial effusion
Periop CVS instability
D
Autonomic dysfunction
Myopathy (due to uraemia)
E
Electrolyte disturbances
High K+
Low Ca2+
High Mg2+
Anaemia (normochromic, normocytic)
Coagulopathy
esp platelet dysfunction
Decreased immunity
Others
GORD
Altered drug pharmacology
Hepatitis B and C common
Consultation
History
HxPC
Underlying cause of CRF
Dialysis
Haemodialysis or peritoneal dialysis
If haemodialysis
Vascular access
Frequency
Time of last dialysis
Dry weight
Residual urine output
Association
Hypertension
Diabetes mellitus (DM)
Functional capacity
Exam
S&S of fluid overload or hypovolaemia
Ix
Bloods
FBC
Electrolytes (K+, Ca2+, Mg2+)
Coagulation
ECG
Consider echocardiogram
Optimisation
Liase with renal team re timing of dialysis
Plan
MADE
M
NM monitor
Consider CVP (use internal jugular)
D
Avoid IV fluids with K+
Induction
Consider RSI
Extubation
Ensure adequate recovery of NMJ
Postop
Consider ICU admission
Review by renal team postop
Goals
Careful fluid balance
Monitor electrolyte balance (esp K+)
Protect AV fistula
Avoid cannulation and NIBP cuff in fistula arm
Keep warm
Wrap fistula arm in extra padding

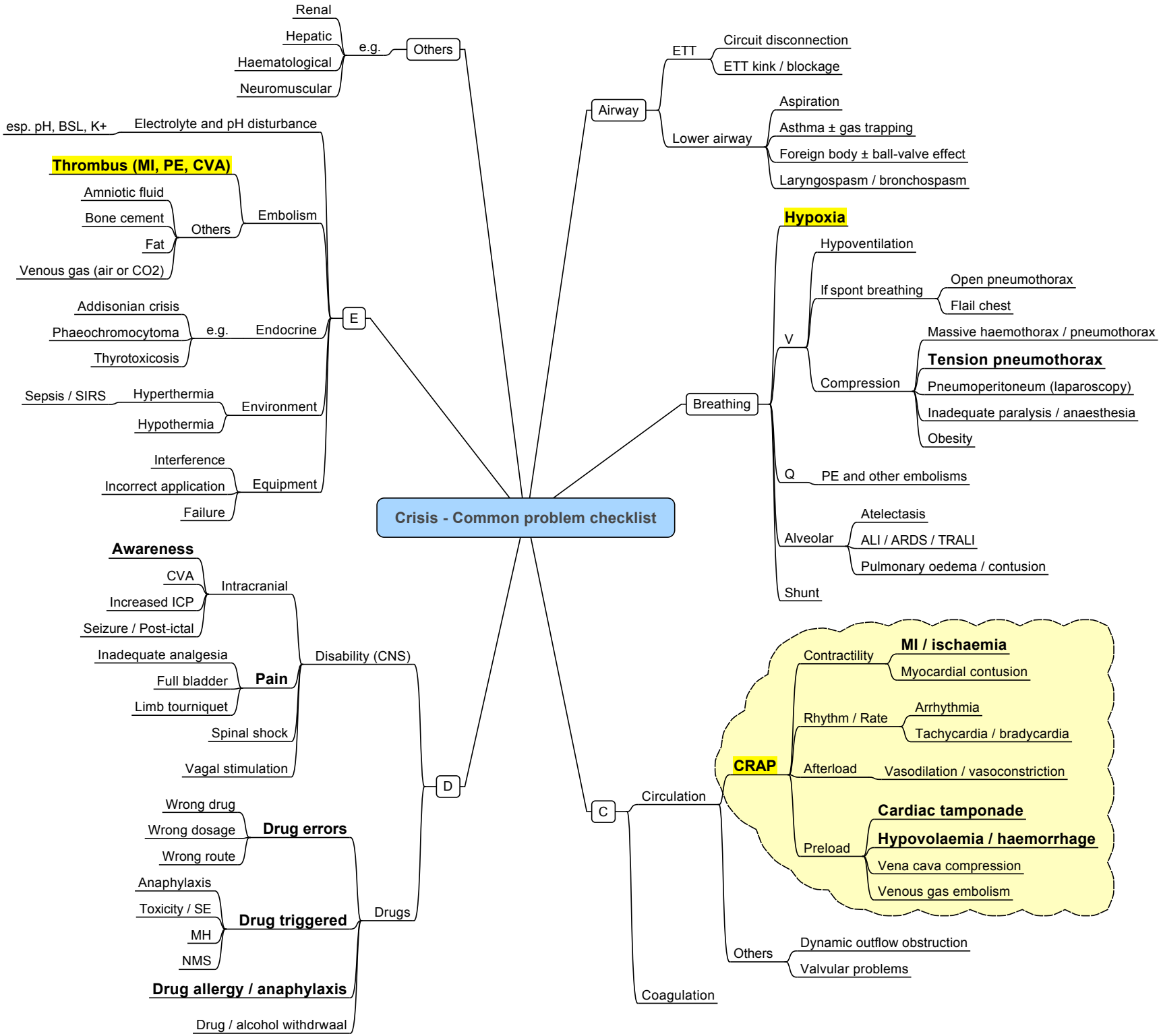

Confusion, periop - DDx
ABC
Hypoxia
Hypercarbia
Hypotension
D
CNS
Intracranial
CVA
Increased ICP
Post-ictal state
Infection
Head trauma
Dementia
Pain
Drugs
Drug effects
Residual anaesthesia
Volatile
Ketamine
Opioids
Benzodiazepine
Scopolamine / atropine
Antiemetics
Phenothiazines
Chlorpromazine (largactil)
Prochlorperazine (stemetil)
EtOH
Abused substances
Drug error
Drug withdrawal
Medication
EtOH
Abused substance
E
Electrolytes
↓BSL
↓Na+
Acidosis
Hypothermia
Hyperthermia / Sepsis
Hypothyroidism
Others
Hepatic encephalopathy
Uraemia
Porphyria

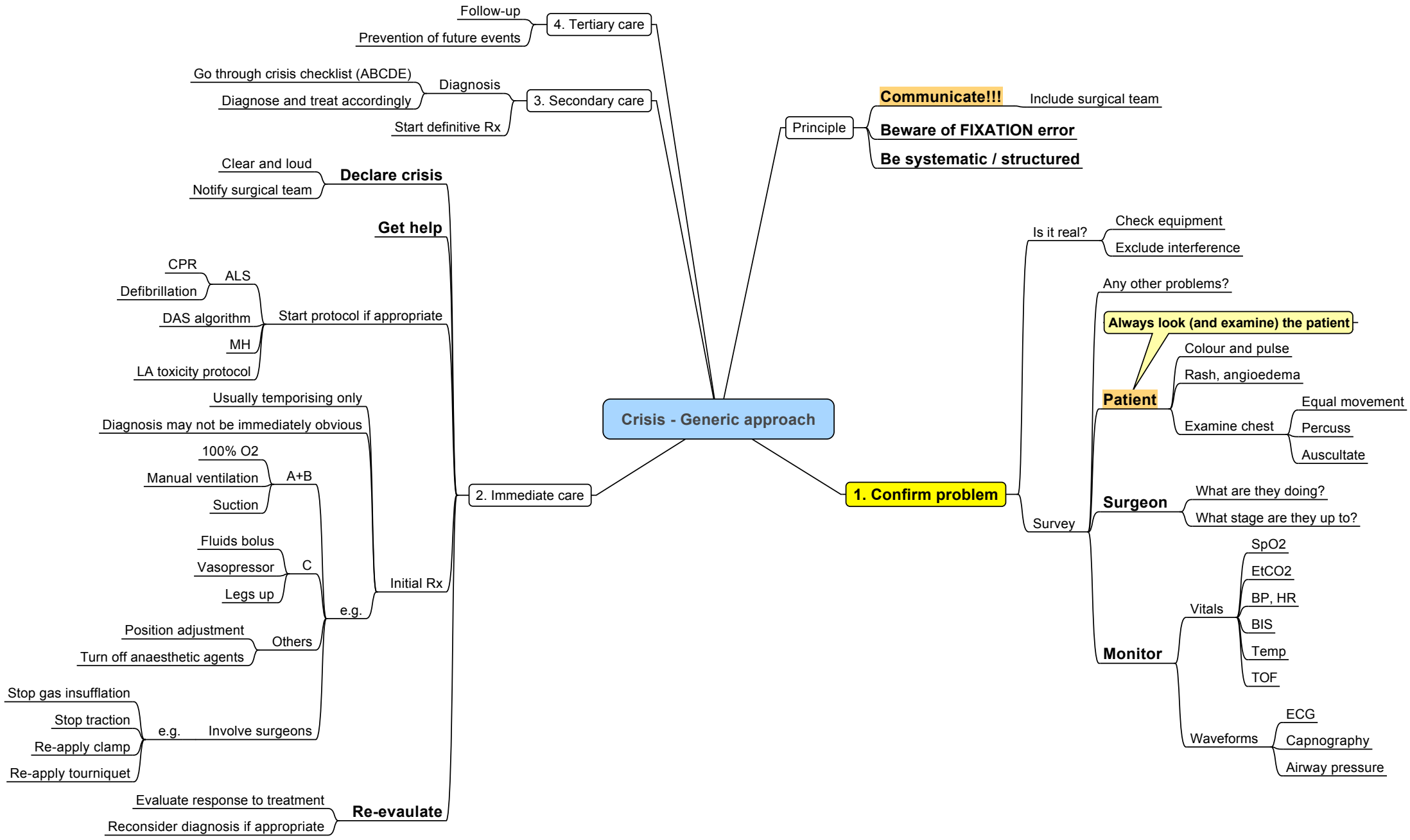

COPD - AMx
Consultation
History
COPD
Previous hospital admission
esp ICU admissions
Recent exacerbation / infection
Smoking history
Co-morbidities
CVS diseases common
Functional capacity
S&S
Wheeze
Dyspnoea
Cough and sputum production
Cyanosis
Rx and response to Rx
Ix
Sputum M/C/S
CXR
Bullae
Consolidation
Spirometry
ECG
Right heart strain pattern
Consider
ABG
Significant S&S
Limited functional capacity
Echo
Lung function test
Optimisation
Smoke cessation
Delay elective surgery if...
Respiratory infection
Exacerbation of COPD
Preop physiotherapy if severe COPD with sputum production
Consider referral to respiratory physician, esp if
Reversibility on spirometry
Severe disease or significant S&S
Issues
Expiratory flow limitation
Due to
Dynamic airway compression
Bronchoconstriction
Sputum plugging
Leading to
Gas trapping and hyperinflation
V/Q mismatch
Parenchymal destruction
Decrease gas transfer
Cor pulmonale and pulmonary hypertension
Intraop
Bronchospasm
Pneumothorax
Postop
Respiratory complications
including respiratory failure
Thromboembolism
Plan
Intraop
Intubation
Avoid intubation if possible
Extubation
Endobronchial suctioning while deep
Extubate and recover in sitting position
Postop
Standard Rx
Early mobilisation
Physiotherapy
Good analgesia
Saline nebulisation
Thromboembolism prophylaxis
O2
Target SpO2 88 to 92%
Titrate O2 rate to effect
Never withhold O2 from hypoxic patient
True CO2 retainers (i.e. dependent on hypoxic drive) are rare
Consider
BIPAP
HDU/ICU admission, esp if...
Severe disease
Major surgery
Goals
Minimise gas trapping if IPPV
Avoid airway irritation
Strongly consider regional anaesthesia / analgesia
But AVOID interscalene block
GOLD classification
Basics
GOLD = Global obstructive lung disease
FEV1/FVC < 0.70 required for all stages
Stage 1: Mild
FEV1 ≥ 80% predicted
Stage 2: Moderate
FEV1 = 50 to 80% predicted
Stage 3: Severe
FEV1 = 30 to 50% predicted
Stage 4: Very severe
FEV1 < 30% predicted, OR
FEV1 < 50%
+ Chronic respiratory failure
e.g. chronic O2 therapy
Others
Prognosis
FEV1 < 35% of predicted
30% 1-year mortality
95% 10-year mortality
pCO2 > 45 mmHg
Best predictor of postop pulmonary risk
Types of COPD
Chronic bronchitis
"Blue bloater"
CO2 retention occurs earlier
Emphysema
"Pink puffer"
Most have a mixture of both elements

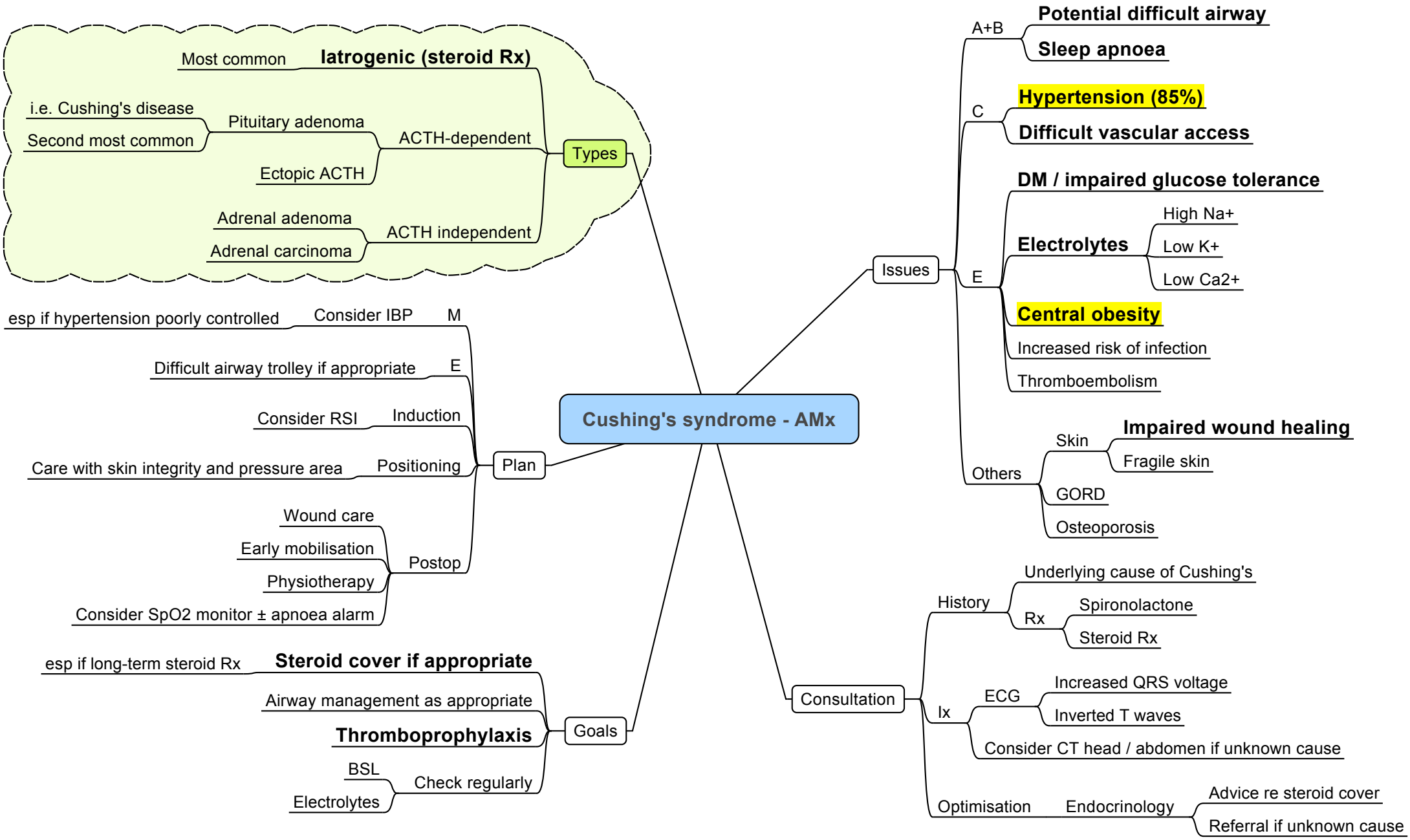

COPD - Ventilation strategy
Issues
Gas trapping and dynamic hyperinflation
Risk of pneumothorax
Aim
Keep SpO2 ≥ 91%
Permissive hypercapnoea
Minimise dynamic hyperinflation
Decrease MV
Long expiratory phase
Reduce resistance to expiratory airflow
Keep plateau pressure < 35cmH2O
Keep pH ≥ 7.2
Settings
PCV preferred over VCV
Low tidal volume (8 mL/kg)
Low respiratory rate (≤12 /min)
Long expiratory phase
I : E ratio = 1 : 2 to 1: 4
No end-inspiratory pause
Little or no PEEP (≤ 5 cmH2O)
Others
Hypercapnoea better tolerated than in asthma
Pre-existing metabolic alkalosis
... thus acidaemia less severe
Similar to the ventilation strategy for severe asthma

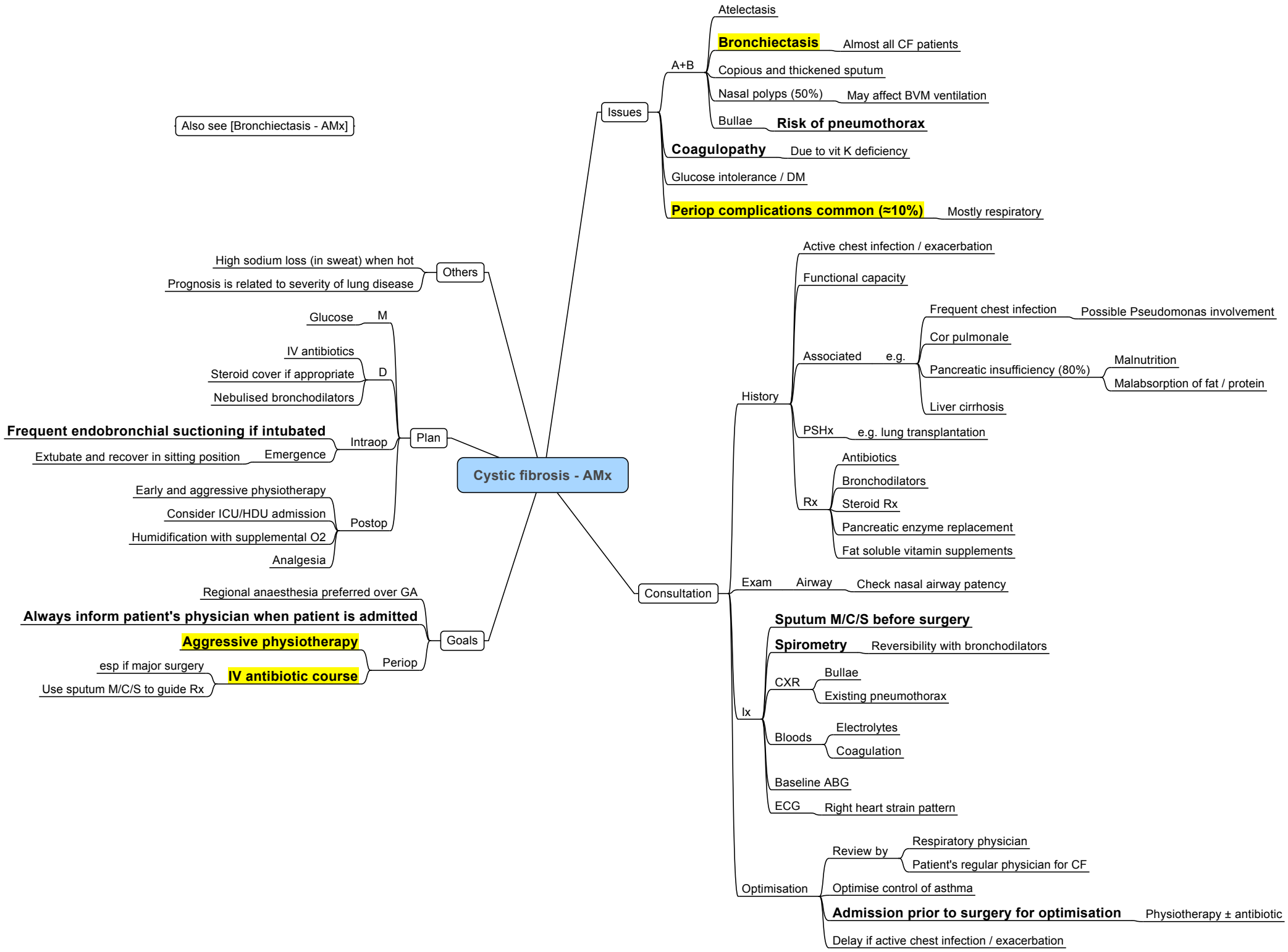

Cord prolapse
Basics
Obstetric emergency
Danger to foetus
Perinatal mortality ≈ 5 to 10%
No major maternal risk
Incidence = 0.2 to 0.4% with cephalic presentation
Incidence = 2 to 4% with multiple pregnancy and malpresentation
PP
Compression of cord will severely compromise foetal blood supply
Risk factors
High presenting parts
High parity
Prematurity
Multiple pregnancy
Polyhydramnios
A prolapsed cord generally implies that the head is high and has not engaged
Rx
If cord compression
Presenting part must be pushed out of pelvis by manual upward pressure
Minimal handling of cord lying outside vagina
Risk of vasospasm
Consider positioning
Knee-chest
Exaggerated Simm's left lateral
Consider
Tocolytics if foetal distress and delayed LSCS
LSCS
... unless vaginal delivery imminent
Category 1 LSCS
IF...
Foetal heart rate (FHR) pattern suspicious or pathological
AND no undue risk to mother
Category 2 if foetal heart rate pattern normal
Plan
Choice
GA
RSI with cricoid pressure
Only option if foetal compromise
Spinal or epidural top-up
Only if no cord compression and no foetal distress
MADE
Antacid prophylaxis
IV ranitidine 50mg
PO 0.3M sodium citrate 30mL

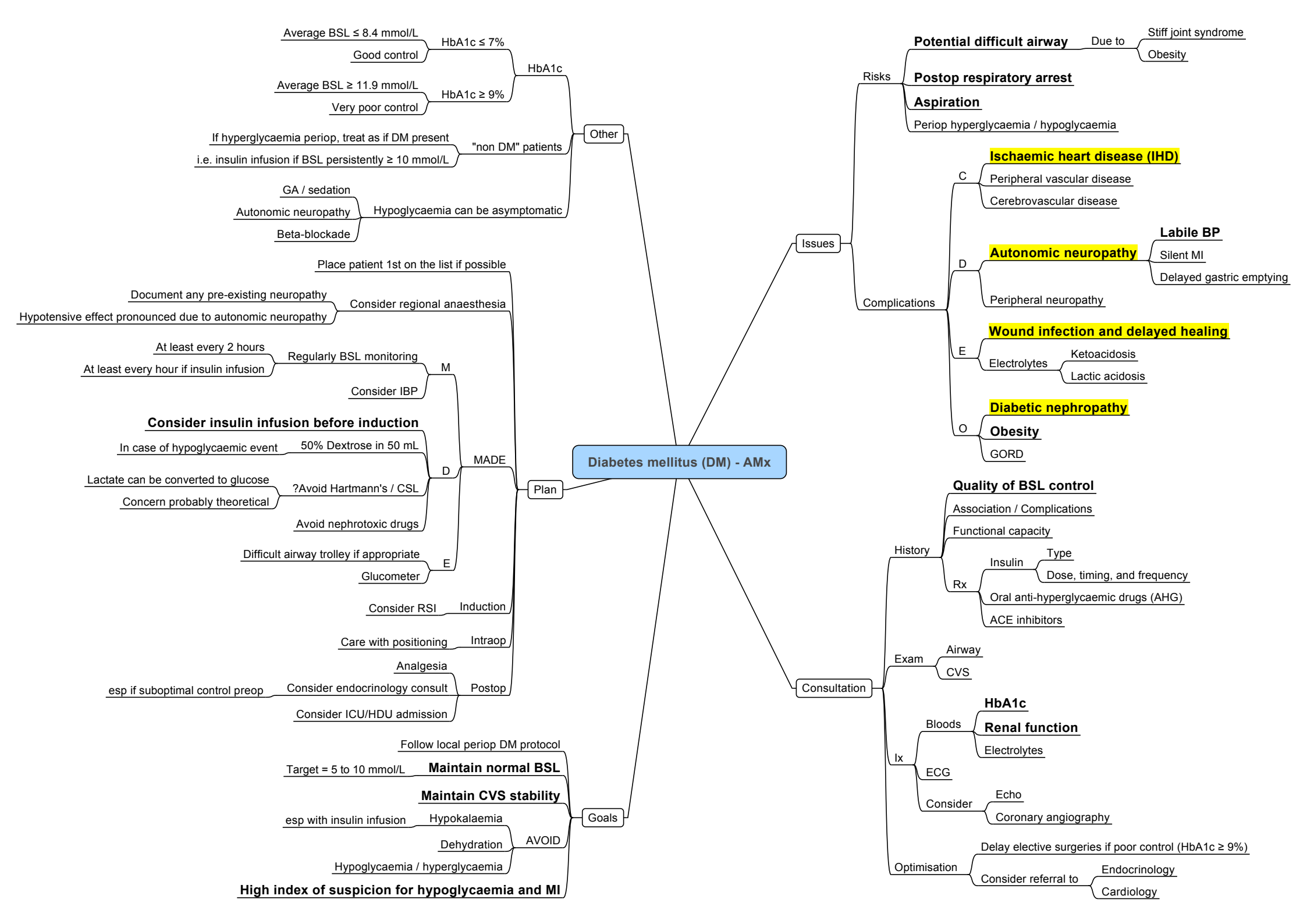

CRASH-2 Trial
Background
Clinical Randomisation of an Antifibrinolytic in Significant Haemorrhage 2
Published in Lancet 2010
Effects of tranexamic acid in trauma patients on...
Death
Vascular occlusive events
Receipt of blood transfusion
Regime
Loading dose = 1g over 10 min...
Followed by infusion = 1g over 8 hours
Method
Multicentered, double-blinded randomised control trial
n = 20127
Tranexamic acid vs placebo
Adult trauma patients
Significant haemorrhage (or at risk of)
(Defined clinically by SBP < 90 mmHg and/or HR > 110)
AND within 8 hrs of injury
Primary outcome = Death
... due to
Bleeding
Vascular occlusion (MI / Stroke / PE)
Multiorgan failure
Head injury
Others
Secondary outcomes
Vascular occlusive events (MI / Stroke / PE / DVT)
Surgical intervention
Receipt of blood transfusion (and units transfused)
Findings
Mortality reduced
Mortality due to all causes
14.5% (vs 16%)
RR = 0.91, p = 0.0035
Mortality due to bleeding
4.9% (vs 5.7%)
RR = 0.85, p = 0.0077
Reduced MI (fatal and non-fatal)
RR = 0.64, p = 0.035
Vascular occlusive events
No increase
Non-significant DECREASE in death due to vascular occlusion
No difference in transfusion requirement
?Surviving patients have more opportunity to receive transfusion
i.e. competing risk
Exploratory analysis
Findings
Death due to bleeding
Given ≤ 1 hour of injury
RR = 0.68, p < 0.0001
Given 1 to 3 hours after injury
RR = 0.79, p = 0.03
Given ≥ 3 hours after injury
RR = 1.44, p = 0.004
i.e.
Reduced with early use (< 3 hours)
But INCREASED if given late (≥ 3 hours)
Death due to non-bleeding causes
RR = 0.94
NOT significant (p = 0.13)
Subgroup analysis with focus on death due to bleeding
Published in Lancet 2011
Noted by authors
Studies on tranexamic acid vary widely in dosage
Loading dose 2.5 to 100 mg/kg
Infusion 0.25 to 4 mg/kg/h
Study in cardiac surgery
Loading dose 10 mg/kg + infusion 1mg/kg/hr
Achieves sufficient plasma level to inhibit fibrinolysis
Larger dose does NOT provide additional haemostatic benefit

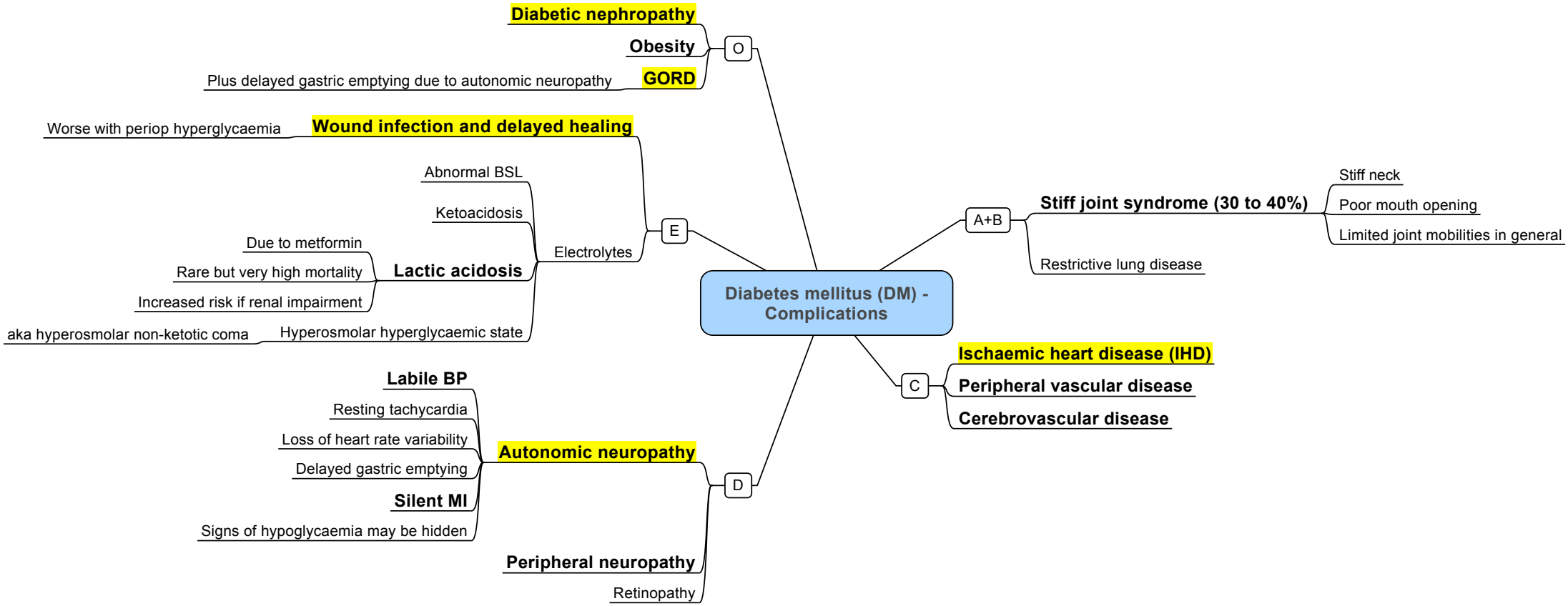

Crisis - Common problem checklist
A
Airway
ETT
Circuit disconnection
ETT kink / blockage
Lower airway
Aspiration
Asthma ± gas trapping
Foreign body ± ball-valve effect
Laryngospasm / bronchospasm
B
Breathing
Hypoxia
V
Hypoventilation
If spont breathing
Open pneumothorax
Flail chest
Compression
Massive haemothorax / pneumothorax
Tension pneumothorax
Pneumoperitoneum (laparoscopy)
Inadequate paralysis / anaesthesia
Obesity
Q
PE and other embolisms
Alveolar
Atelectasis
ALI / ARDS / TRALI
Pulmonary oedema / contusion
Shunt
C
Circulation
CRAP
Contractility
MI / ischaemia
Myocardial contusion
Rhythm / Rate
Arrhythmia
Tachycardia / bradycardia
Afterload
Vasodilation / vasoconstriction
Preload
Cardiac tamponade
Hypovolaemia / haemorrhage
Vena cava compression
Venous gas embolism
Others
Dynamic outflow obstruction
Valvular problems
Coagulation
D
Disability (CNS)
Intracranial
Awareness
CVA
Increased ICP
Seizure / Post-ictal
Pain
Inadequate analgesia
Full bladder
Limb tourniquet
Spinal shock
Vagal stimulation
Drugs
Drug errors
Wrong drug
Wrong dosage
Wrong route
Drug triggered
Anaphylaxis
Toxicity / SE
MH
NMS
Drug allergy / anaphylaxis
Drug / alcohol withdrwaal
E
Electrolyte and pH disturbance
esp. pH, BSL, K+
Embolism
Thrombus (MI, PE, CVA)
Others
Amniotic fluid
Bone cement
Fat
Venous gas (air or CO2)
Endocrine
e.g.
Addisonian crisis
Phaeochromocytoma
Thyrotoxicosis
Environment
Sepsis / SIRS
Hyperthermia
Hypothermia
Equipment
Interference
Incorrect application
Failure
Others
e.g.
Renal
Hepatic
Haematological
Neuromuscular

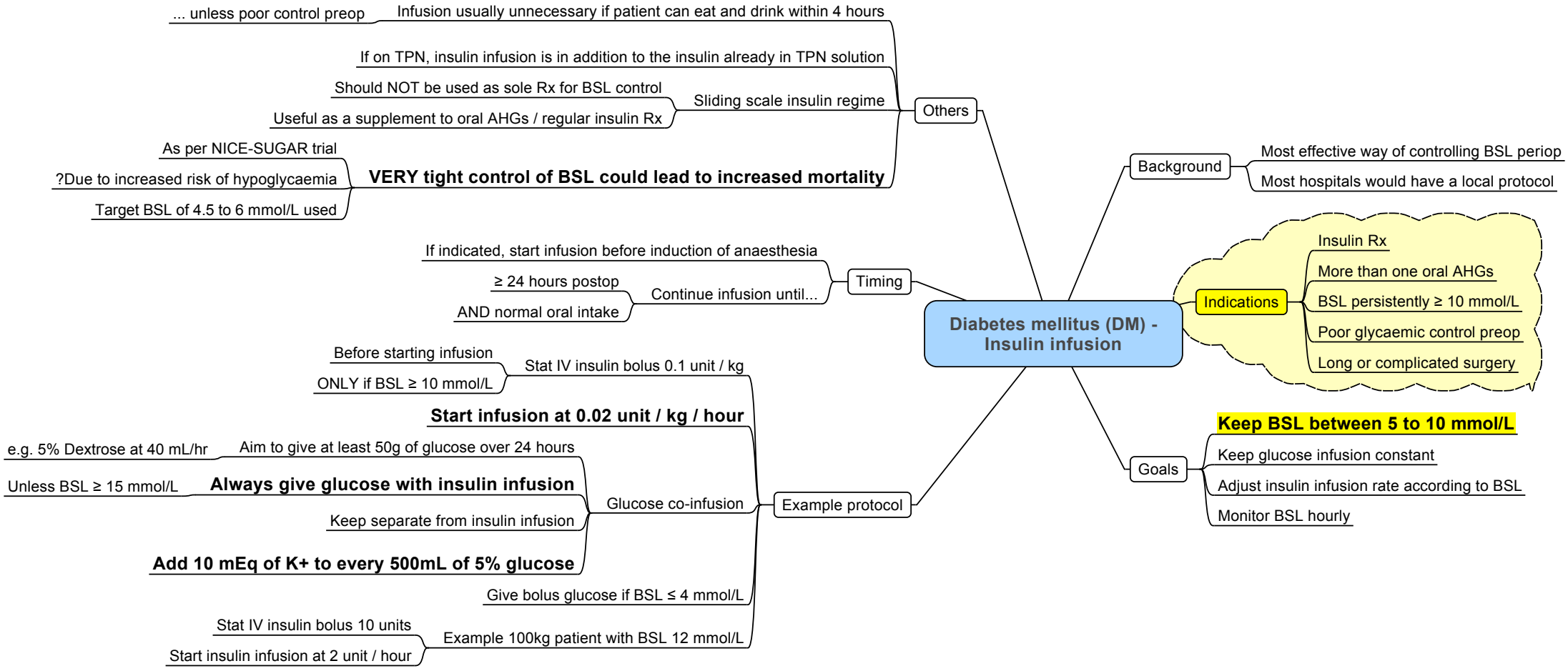

Crisis - Generic approach
Principle
Communicate!!!
Include surgical team
Beware of FIXATION error
Be systematic / structured
1. Confirm problem
Is it real?
Check equipment
Exclude interference
Survey
Any other problems?
Always look (and examine) the patient
Patient
Colour and pulse
Rash, angioedema
Examine chest
Equal movement
Percuss
Auscultate
Surgeon
What are they doing?
What stage are they up to?
Monitor
Vitals
SpO2
EtCO2
BP, HR
BIS
Temp
TOF
Waveforms
ECG
Capnography
Airway pressure
2. Immediate care
Declare crisis
Clear and loud
Notify surgical team
Get help
Start protocol if appropriate
ALS
CPR
Defibrillation
DAS algorithm
MH
LA toxicity protocol
Initial Rx
Usually temporising only
Diagnosis may not be immediately obvious
e.g.
A+B
100% O2
Manual ventilation
Suction
C
Fluids bolus
Vasopressor
Legs up
Others
Position adjustment
Turn off anaesthetic agents
Involve surgeons
e.g.
Stop gas insufflation
Stop traction
Re-apply clamp
Re-apply tourniquet
Re-evaulate
Evaluate response to treatment
Reconsider diagnosis if appropriate
3. Secondary care
Diagnosis
Go through crisis checklist (ABCDE)
Diagnose and treat accordingly
Start definitive Rx
4. Tertiary care
Follow-up
Prevention of future events

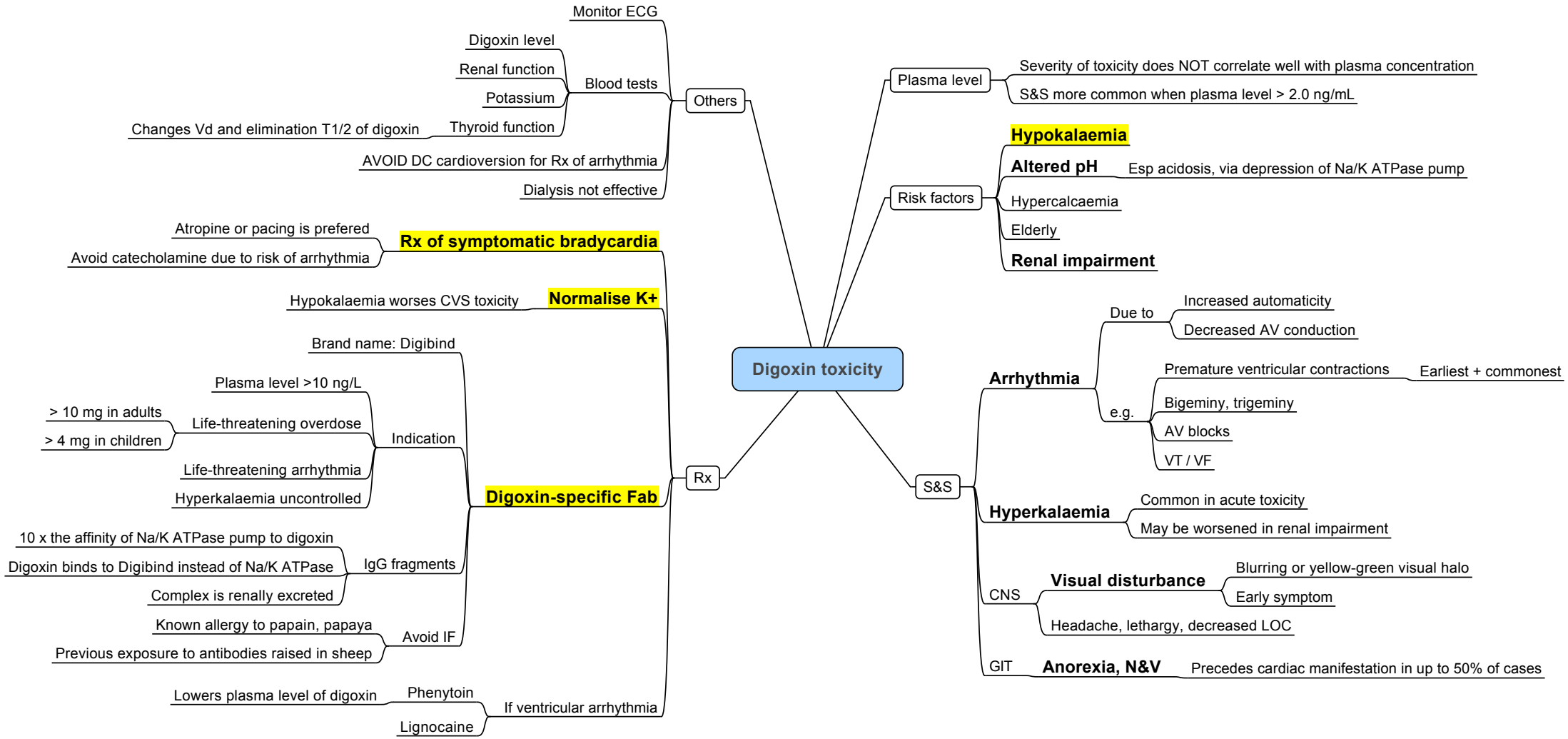

Cushing's syndrome - AMx
Types
Most common
Iatrogenic (steroid Rx)
ACTH-dependent
i.e. Cushing's disease
Second most common
Pituitary adenoma
Ectopic ACTH
ACTH independent
Adrenal adenoma
Adrenal carcinoma
Issues
A+B
Potential difficult airway
Sleep apnoea
C
Hypertension (85%)
Difficult vascular access
E
DM / impaired glucose tolerance
Electrolytes
High Na+
Low K+
Low Ca2+
Central obesity
Increased risk of infection
Thromboembolism
Others
Impaired wound healing
Skin
Fragile skin
GORD
Osteoporosis
Plan
M
esp if hypertension poorly controlled
Consider IBP
E
Difficult airway trolley if appropriate
Induction
Consider RSI
Positioning
Care with skin integrity and pressure area
Postop
Wound care
Early mobilisation
Physiotherapy
Consider SpO2 monitor ± apnoea alarm
Goals
esp if long-term steroid Rx
Steroid cover if appropriate
Airway management as appropriate
Thromboprophylaxis
Check regularly
BSL
Electrolytes
Consultation
History
Underlying cause of Cushing's
Rx
Spironolactone
Steroid Rx
Ix
ECG
Increased QRS voltage
Inverted T waves
Consider CT head / abdomen if unknown cause
Optimisation
Endocrinology
Advice re steroid cover
Referral if unknown cause

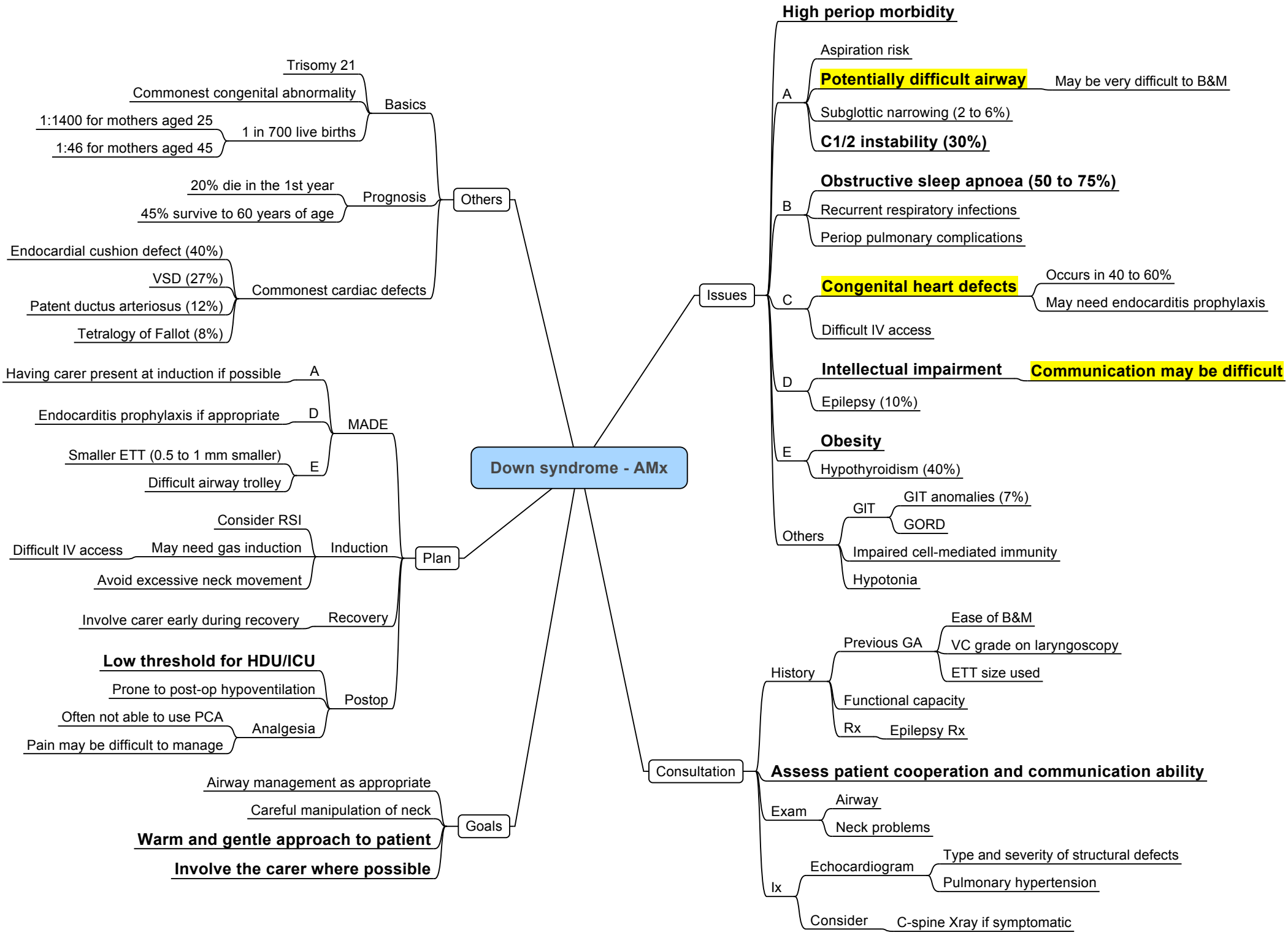

Cystic fibrosis - AMx
Also see [Bronchiectasis - AMx]
Issues
A+B
Atelectasis
Bronchiectasis
Almost all CF patients
Copious and thickened sputum
Nasal polyps (50%)
May affect BVM ventilation
Bullae
Risk of pneumothorax
Coagulopathy
Due to vit K deficiency
Glucose intolerance / DM
Periop complications common (≈10%)
Mostly respiratory
Consultation
History
Active chest infection / exacerbation
Functional capacity
Associated
e.g.
Frequent chest infection
Possible Pseudomonas involvement
Cor pulmonale
Pancreatic insufficiency (80%)
Malnutrition
Malabsorption of fat / protein
Liver cirrhosis
PSHx
e.g. lung transplantation
Rx
Antibiotics
Bronchodilators
Steroid Rx
Pancreatic enzyme replacement
Fat soluble vitamin supplements
Exam
Airway
Check nasal airway patency
Ix
Sputum M/C/S before surgery
Spirometry
Reversibility with bronchodilators
CXR
Bullae
Existing pneumothorax
Bloods
Electrolytes
Coagulation
Baseline ABG
ECG
Right heart strain pattern
Optimisation
Review by
Respiratory physician
Patient's regular physician for CF
Optimise control of asthma
Admission prior to surgery for optimisation
Physiotherapy ± antibiotic
Delay if active chest infection / exacerbation
Plan
M
Glucose
D
IV antibiotics
Steroid cover if appropriate
Nebulised bronchodilators
Intraop
Frequent endobronchial suctioning if intubated
Emergence
Extubate and recover in sitting position
Postop
Early and aggressive physiotherapy
Consider ICU/HDU admission
Humidification with supplemental O2
Analgesia
Goals
Periop
Regional anaesthesia preferred over GA
Always inform patient's physician when patient is admitted
Aggressive physiotherapy
IV antibiotic course
esp if major surgery
Use sputum M/C/S to guide Rx
Others
High sodium loss (in sweat) when hot
Prognosis is related to severity of lung disease

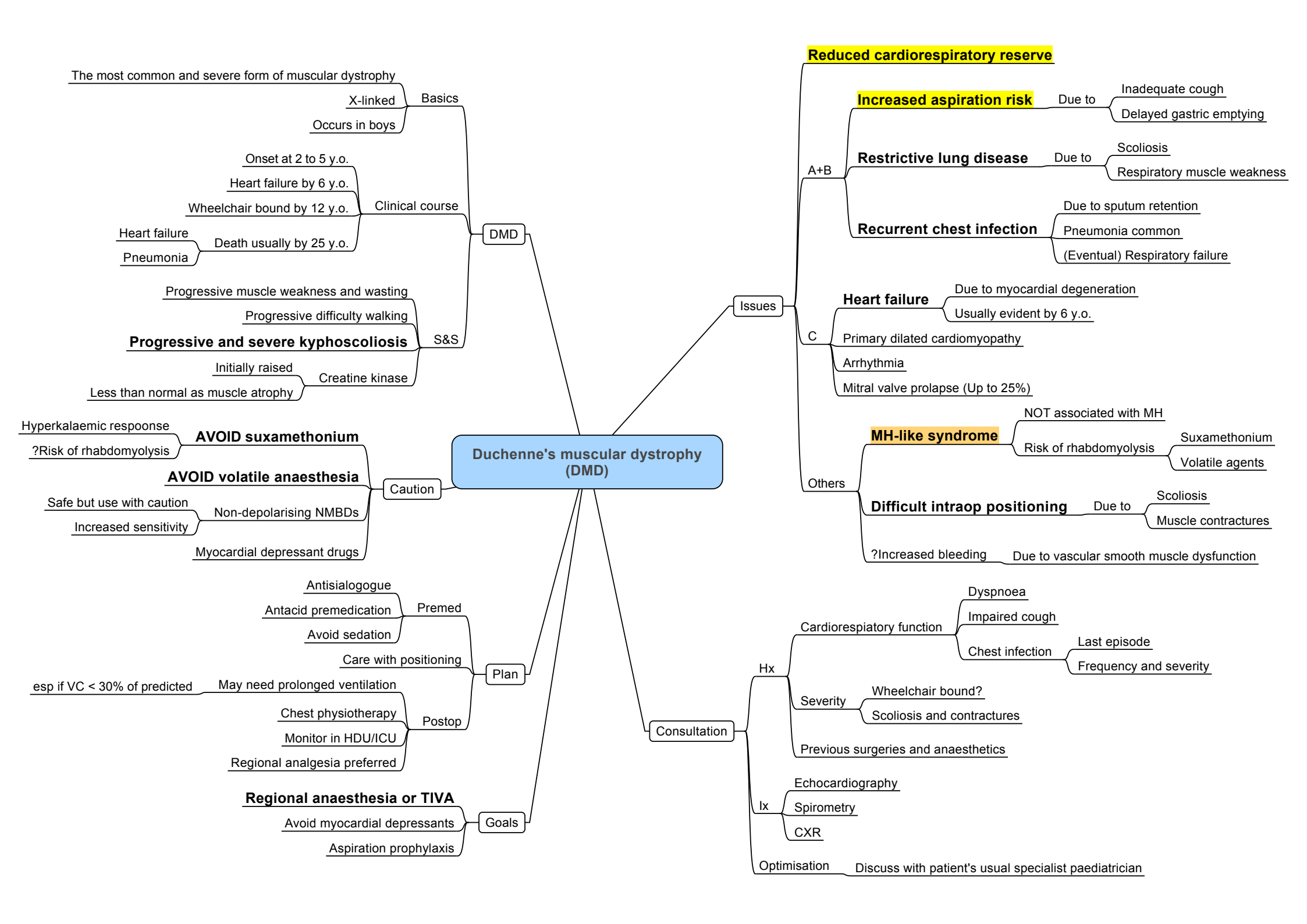

Diabetes mellitus (DM) - AMx
Issues
Risks
Potential difficult airway
Due to
Stiff joint syndrome
Obesity
Postop respiratory arrest
Aspiration
Periop hyperglycaemia / hypoglycaemia
Complications
C
Ischaemic heart disease (IHD)
Peripheral vascular disease
Cerebrovascular disease
D
Autonomic neuropathy
Labile BP
Silent MI
Delayed gastric emptying
Peripheral neuropathy
E
Wound infection and delayed healing
Electrolytes
Ketoacidosis
Lactic acidosis
O
Diabetic nephropathy
Obesity
GORD
Consultation
History
Quality of BSL control
Association / Complications
Functional capacity
Rx
Insulin
Type
Dose, timing, and frequency
Oral anti-hyperglycaemic drugs (AHG)
ACE inhibitors
Exam
Airway
CVS
Ix
Bloods
HbA1c
Renal function
Electrolytes
ECG
Consider
Echo
Coronary angiography
Optimisation
Delay elective surgeries if poor control (HbA1c ≥ 9%)
Consider referral to
Endocrinology
Cardiology
Goals
Follow local periop DM protocol
Target = 5 to 10 mmol/L
Maintain normal BSL
Maintain CVS stability
AVOID
esp with insulin infusion
Hypokalaemia
Dehydration
Hypoglycaemia / hyperglycaemia
High index of suspicion for hypoglycaemia and MI
Plan
MADE
M
Regularly BSL monitoring
At least every 2 hours
At least every hour if insulin infusion
Consider IBP
D
Consider insulin infusion before induction
In case of hypoglycaemic event
50% Dextrose in 50 mL
Lactate can be converted to glucose
?Avoid Hartmann's / CSL
Concern probably theoretical
Avoid nephrotoxic drugs
E
Difficult airway trolley if appropriate
Glucometer
Induction
Consider RSI
Intraop
Care with positioning
Postop
Analgesia
Consider endocrinology consult
esp if suboptimal control preop
Consider ICU/HDU admission
Consider regional anaesthesia
Document any pre-existing neuropathy
Hypotensive effect pronounced due to autonomic neuropathy
Place patient 1st on the list if possible
Other
HbA1c
HbA1c ≤ 7%
Average BSL ≤ 8.4 mmol/L
Good control
HbA1c ≥ 9%
Average BSL ≥ 11.9 mmol/L
Very poor control
"non DM" patients
If hyperglycaemia periop, treat as if DM present
i.e. insulin infusion if BSL persistently ≥ 10 mmol/L
Hypoglycaemia can be asymptomatic
GA / sedation
Autonomic neuropathy
Beta-blockade

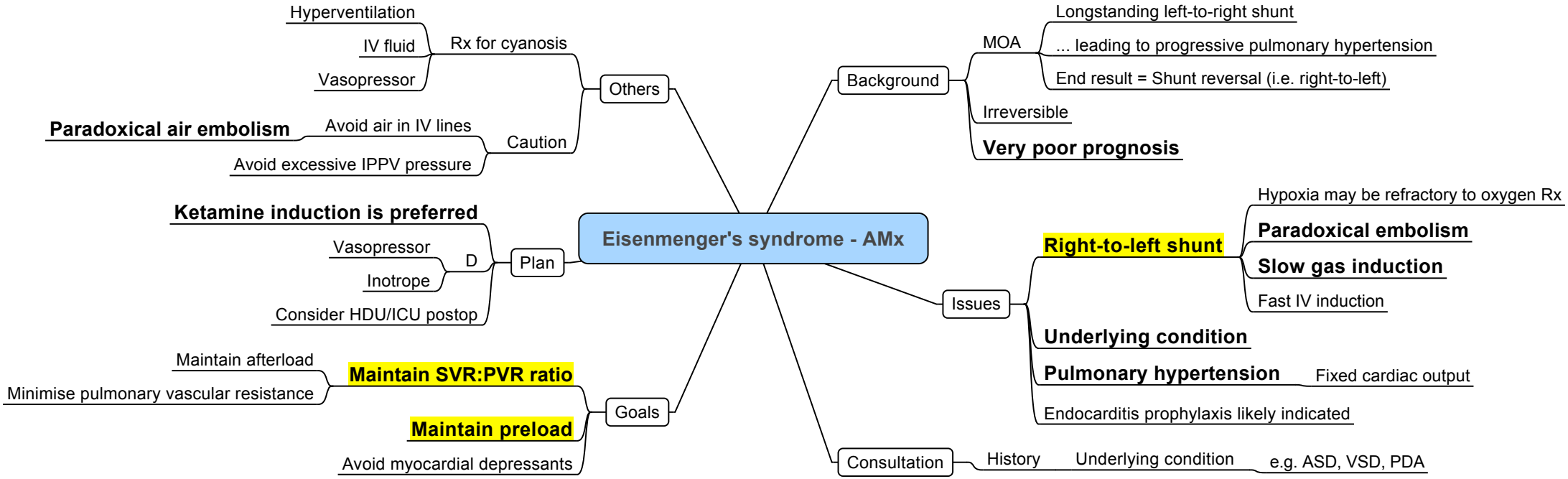

Diabetes mellitus (DM) - Complications
O
Diabetic nephropathy
Obesity
GORD
Plus delayed gastric emptying due to autonomic neuropathy
A+B
Stiff joint syndrome (30 to 40%)
Stiff neck
Poor mouth opening
Limited joint mobilities in general
Restrictive lung disease
C
Ischaemic heart disease (IHD)
Peripheral vascular disease
Cerebrovascular disease
D
Autonomic neuropathy
Labile BP
Resting tachycardia
Loss of heart rate variability
Delayed gastric emptying
Silent MI
Signs of hypoglycaemia may be hidden
Peripheral neuropathy
Retinopathy
E
Wound infection and delayed healing
Worse with periop hyperglycaemia
Electrolytes
Abnormal BSL
Ketoacidosis
Lactic acidosis
Due to metformin
Rare but very high mortality
Increased risk if renal impairment
Hyperosmolar hyperglycaemic state
aka hyperosmolar non-ketotic coma

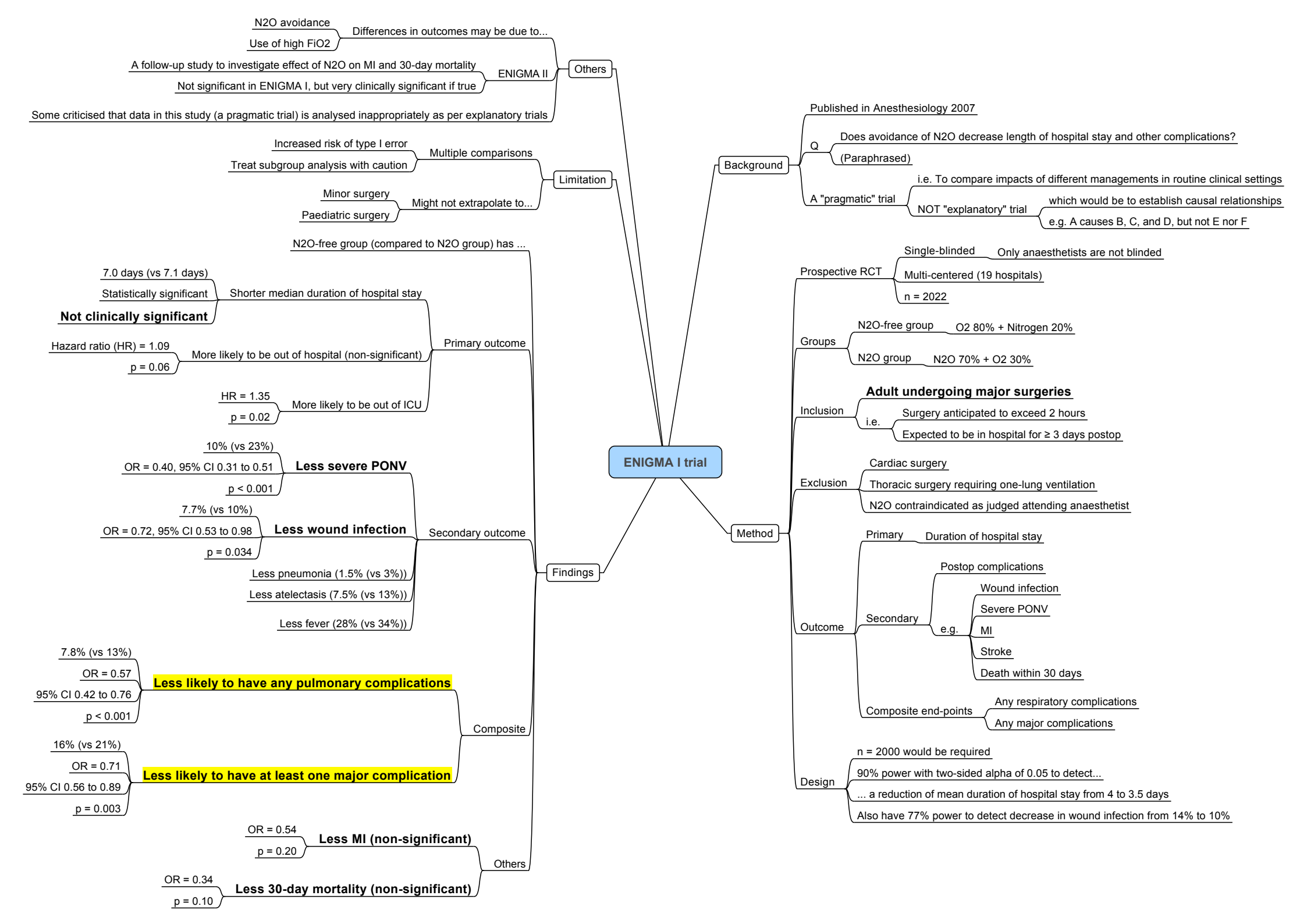

Diabetes mellitus (DM) - Insulin infusion
Background
Most effective way of controlling BSL periop
Most hospitals would have a local protocol
Indications
Insulin Rx
More than one oral AHGs
BSL persistently ≥ 10 mmol/L
Poor glycaemic control preop
Long or complicated surgery
Goals
Keep BSL between 5 to 10 mmol/L
Keep glucose infusion constant
Adjust insulin infusion rate according to BSL
Monitor BSL hourly
Example protocol
Stat IV insulin bolus 0.1 unit / kg
Before starting infusion
ONLY if BSL ≥ 10 mmol/L
Start infusion at 0.02 unit / kg / hour
Glucose co-infusion
Aim to give at least 50g of glucose over 24 hours
e.g. 5% Dextrose at 40 mL/hr
Always give glucose with insulin infusion
Unless BSL ≥ 15 mmol/L
Keep separate from insulin infusion
Add 10 mEq of K+ to every 500mL of 5% glucose
Give bolus glucose if BSL ≤ 4 mmol/L
Example 100kg patient with BSL 12 mmol/L
Stat IV insulin bolus 10 units
Start insulin infusion at 2 unit / hour
Timing
If indicated, start infusion before induction of anaesthesia
Continue infusion until...
≥ 24 hours postop
AND normal oral intake
Others
Infusion usually unnecessary if patient can eat and drink within 4 hours
... unless poor control preop
If on TPN, insulin infusion is in addition to the insulin already in TPN solution
Sliding scale insulin regime
Should NOT be used as sole Rx for BSL control
Useful as a supplement to oral AHGs / regular insulin Rx
VERY tight control of BSL could lead to increased mortality
As per NICE-SUGAR trial
?Due to increased risk of hypoglycaemia
Target BSL of 4.5 to 6 mmol/L used

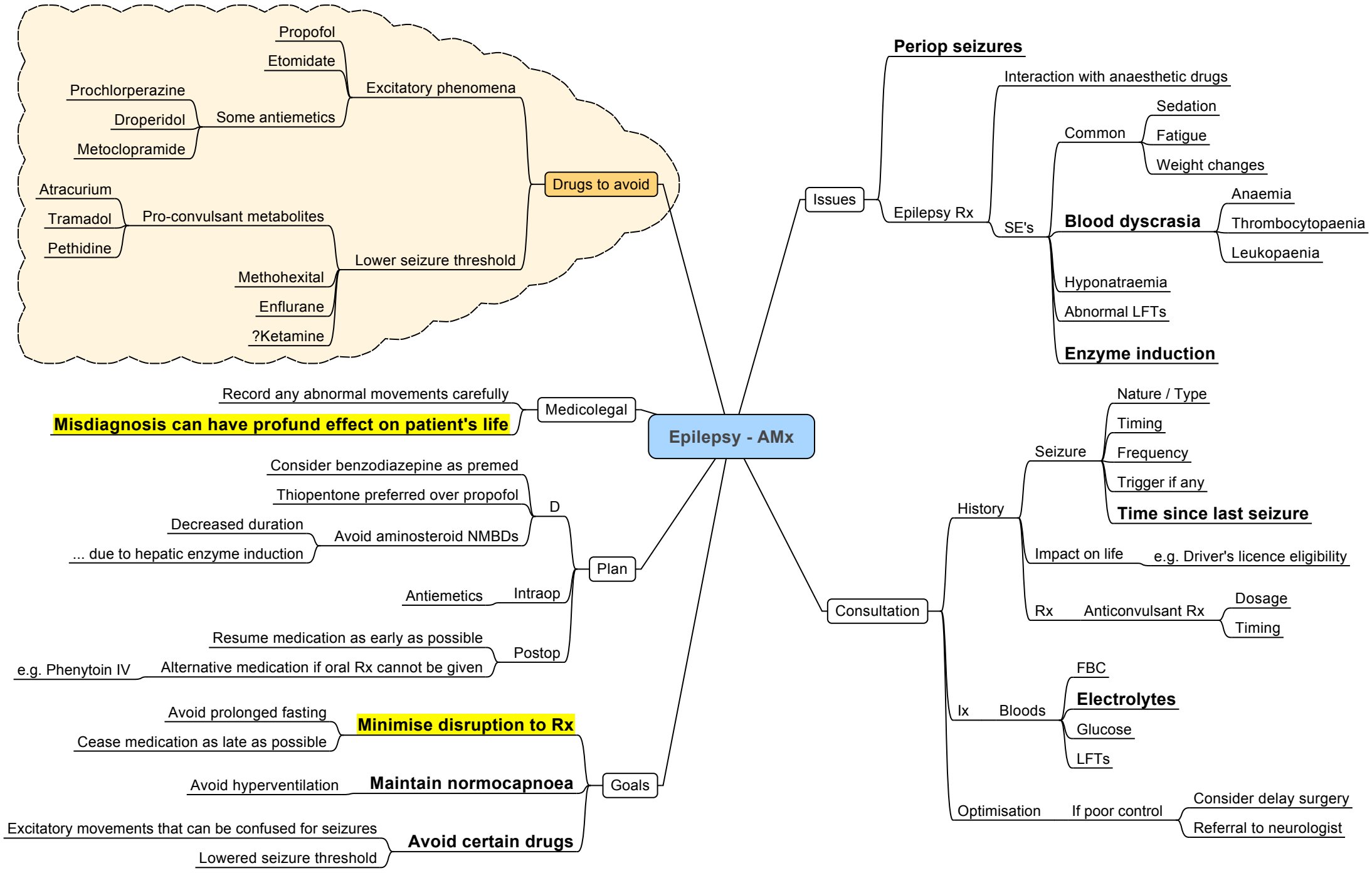

Digoxin toxicity
Rx
Others
Monitor ECG
Blood tests
Digoxin level
Renal function
Potassium
Thyroid function
Changes Vd and elimination T1/2 of digoxin
AVOID DC cardioversion for Rx of arrhythmia
Dialysis not effective
Rx of symptomatic bradycardia
Atropine or pacing is prefered
Avoid catecholamine due to risk of arrhythmia
Normalise K+
Hypokalaemia worses CVS toxicity
Digoxin-specific Fab
Brand name: Digibind
Indication
Plasma level >10 ng/L
Life-threatening overdose
> 10 mg in adults
> 4 mg in children
Life-threatening arrhythmia
Hyperkalaemia uncontrolled
IgG fragments
10 x the affinity of Na/K ATPase pump to digoxin
Digoxin binds to Digibind instead of Na/K ATPase
Complex is renally excreted
Avoid IF
Known allergy to papain, papaya
Previous exposure to antibodies raised in sheep
If ventricular arrhythmia
Lowers plasma level of digoxin
Phenytoin
Lignocaine
S&S
Plasma level
Severity of toxicity does NOT correlate well with plasma concentration
S&S more common when plasma level > 2.0 ng/mL
Risk factors
Hypokalaemia
Altered pH
Esp acidosis, via depression of Na/K ATPase pump
Hypercalcaemia
Elderly
Renal impairment
Arrhythmia
Due to
Increased automaticity
Decreased AV conduction
e.g.
Premature ventricular contractions
Earliest + commonest
Bigeminy, trigeminy
AV blocks
VT / VF
Hyperkalaemia
Common in acute toxicity
May be worsened in renal impairment
CNS
Visual disturbance
Blurring or yellow-green visual halo
Early symptom
Headache, lethargy, decreased LOC
GIT
Anorexia, N&V
Precedes cardiac manifestation in up to 50% of cases


Down syndrome - AMx
Issues
High periop morbidity
A
Aspiration risk
Potentially difficult airway
May be very difficult to B&M
Subglottic narrowing (2 to 6%)
C1/2 instability (30%)
B
Obstructive sleep apnoea (50 to 75%)
Recurrent respiratory infections
Periop pulmonary complications
C
Congenital heart defects
Occurs in 40 to 60%
May need endocarditis prophylaxis
Difficult IV access
D
Intellectual impairment
Communication may be difficult
Epilepsy (10%)
E
Obesity
Hypothyroidism (40%)
Others
GIT
GIT anomalies (7%)
GORD
Impaired cell-mediated immunity
Hypotonia
Consultation
History
Previous GA
Ease of B&M
VC grade on laryngoscopy
ETT size used
Functional capacity
Rx
Epilepsy Rx
Assess patient cooperation and communication ability
Exam
Airway
Neck problems
Ix
Echocardiogram
Type and severity of structural defects
Pulmonary hypertension
Consider
C-spine Xray if symptomatic
Goals
Airway management as appropriate
Careful manipulation of neck
Warm and gentle approach to patient
Involve the carer where possible
Plan
MADE
A
Having carer present at induction if possible
D
Endocarditis prophylaxis if appropriate
E
Smaller ETT (0.5 to 1 mm smaller)
Difficult airway trolley
Induction
Consider RSI
Difficult IV access
May need gas induction
Avoid excessive neck movement
Recovery
Involve carer early during recovery
Postop
Low threshold for HDU/ICU
Prone to post-op hypoventilation
Analgesia
Often not able to use PCA
Pain may be difficult to manage
Others
Basics
Trisomy 21
Commonest congenital abnormality
1 in 700 live births
1:1400 for mothers aged 25
1:46 for mothers aged 45
Prognosis
20% die in the 1st year
45% survive to 60 years of age
Commonest cardiac defects
Endocardial cushion defect (40%)
VSD (27%)
Patent ductus arteriosus (12%)
Tetralogy of Fallot (8%)

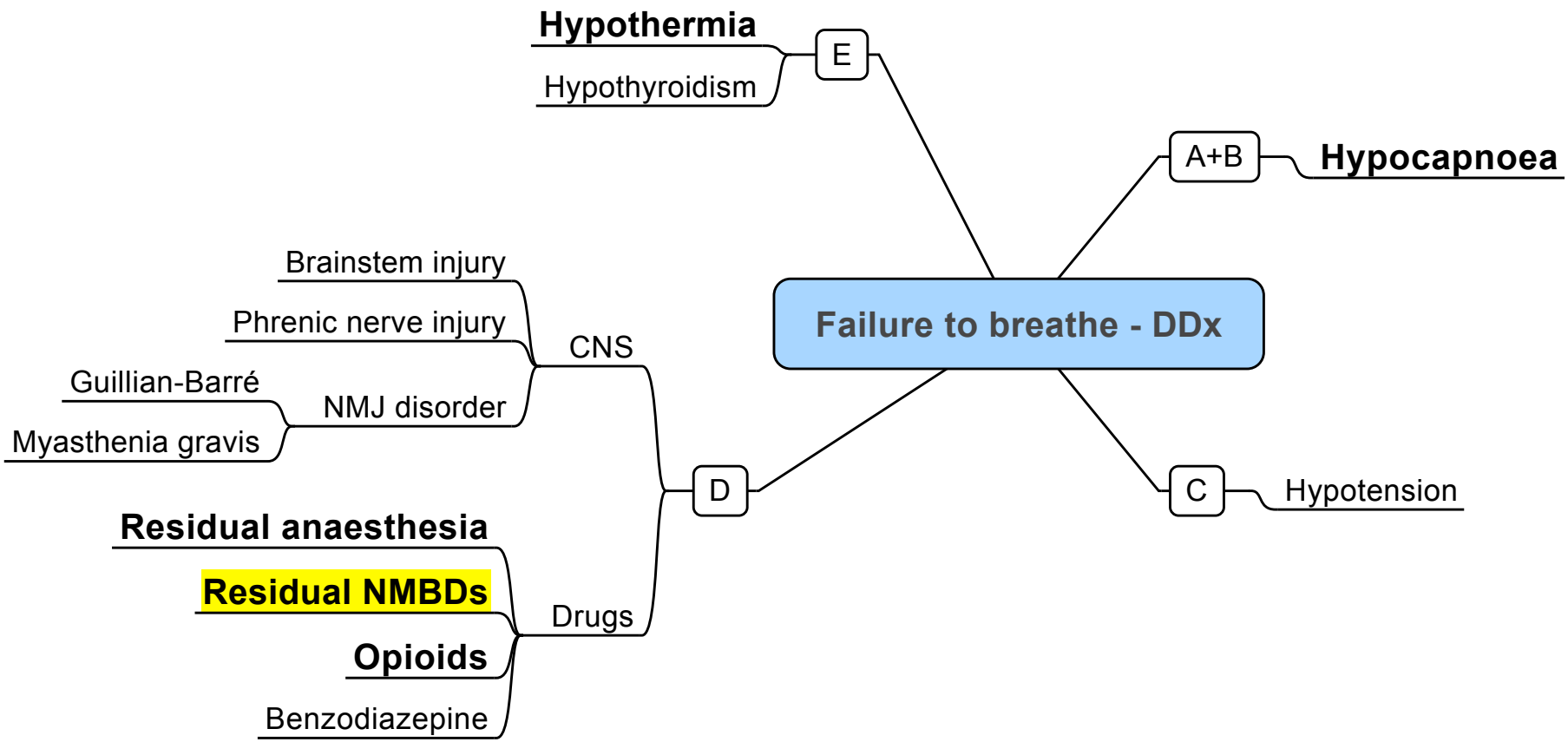

Duchenne's muscular dystrophy (DMD)
DMD
Basics
The most common and severe form of muscular dystrophy
X-linked
Occurs in boys
Clinical course
Onset at 2 to 5 y.o.
Heart failure by 6 y.o.
Wheelchair bound by 12 y.o.
Death usually by 25 y.o.
Heart failure
Pneumonia
S&S
Progressive muscle weakness and wasting
Progressive difficulty walking
Progressive and severe kyphoscoliosis
Creatine kinase
Initially raised
Less than normal as muscle atrophy
Caution
AVOID suxamethonium
Hyperkalaemic respoonse
?Risk of rhabdomyolysis
AVOID volatile anaesthesia
Non-depolarising NMBDs
Safe but use with caution
Increased sensitivity
Myocardial depressant drugs
Plan
Premed
Antisialogogue
Antacid premedication
Avoid sedation
Care with positioning
Postop
esp if VC < 30% of predicted
May need prolonged ventilation
Chest physiotherapy
Monitor in HDU/ICU
Regional analgesia preferred
Goals
Regional anaesthesia or TIVA
Avoid myocardial depressants
Aspiration prophylaxis
Issues
A+B
Reduced cardiorespiratory reserve
Increased aspiration risk
Due to
Inadequate cough
Delayed gastric emptying
Restrictive lung disease
Due to
Scoliosis
Respiratory muscle weakness
Recurrent chest infection
Due to sputum retention
Pneumonia common
(Eventual) Respiratory failure
C
Heart failure
Due to myocardial degeneration
Usually evident by 6 y.o.
Primary dilated cardiomyopathy
Arrhythmia
Mitral valve prolapse (Up to 25%)
Others
MH-like syndrome
NOT associated with MH
Risk of rhabdomyolysis
Suxamethonium
Volatile agents
Difficult intraop positioning
Due to
Scoliosis
Muscle contractures
?Increased bleeding
Due to vascular smooth muscle dysfunction
Consultation
Hx
Cardiorespiatory function
Dyspnoea
Impaired cough
Chest infection
Last episode
Frequency and severity
Severity
Wheelchair bound?
Scoliosis and contractures
Previous surgeries and anaesthetics
Ix
Echocardiography
Spirometry
CXR
Optimisation
Discuss with patient's usual specialist paediatrician

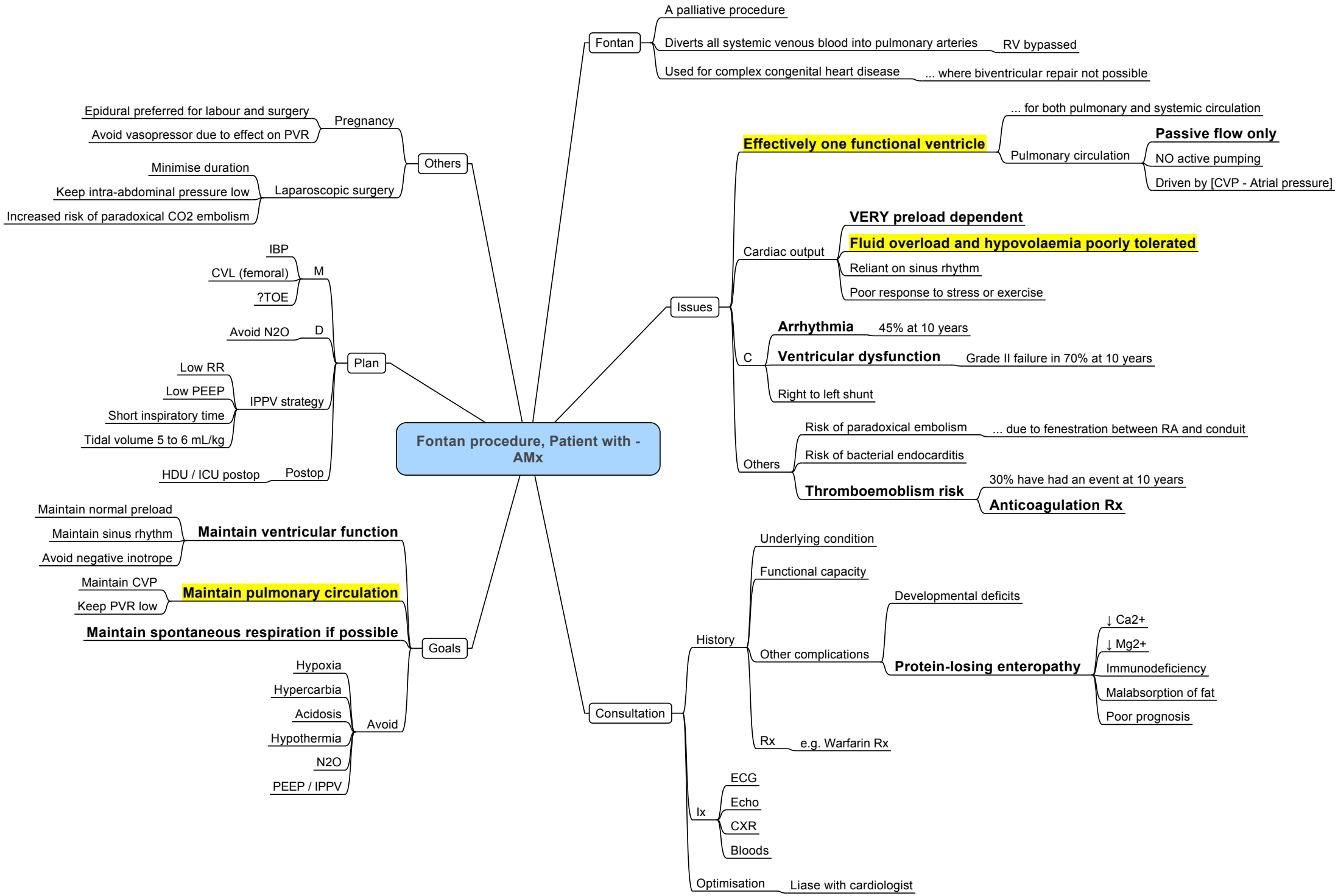

Eisenmenger's syndrome - AMx
Background
MOA
Longstanding left-to-right shunt
... leading to progressive pulmonary hypertension
End result = Shunt reversal (i.e. right-to-left)
Irreversible
Very poor prognosis
Issues
Right-to-left shunt
Hypoxia may be refractory to oxygen Rx
Paradoxical embolism
Slow gas induction
Fast IV induction
Underlying condition
Pulmonary hypertension
Fixed cardiac output
Endocarditis prophylaxis likely indicated
Consultation
History
Underlying condition
e.g. ASD, VSD, PDA
Goals
Maintain SVR:PVR ratio
Maintain afterload
Minimise pulmonary vascular resistance
Maintain preload
Avoid myocardial depressants
Plan
Ketamine induction is preferred
D
Vasopressor
Inotrope
Consider HDU/ICU postop
Others
Rx for cyanosis
Hyperventilation
IV fluid
Vasopressor
Caution
Paradoxical air embolism
Avoid air in IV lines
Avoid excessive IPPV pressure

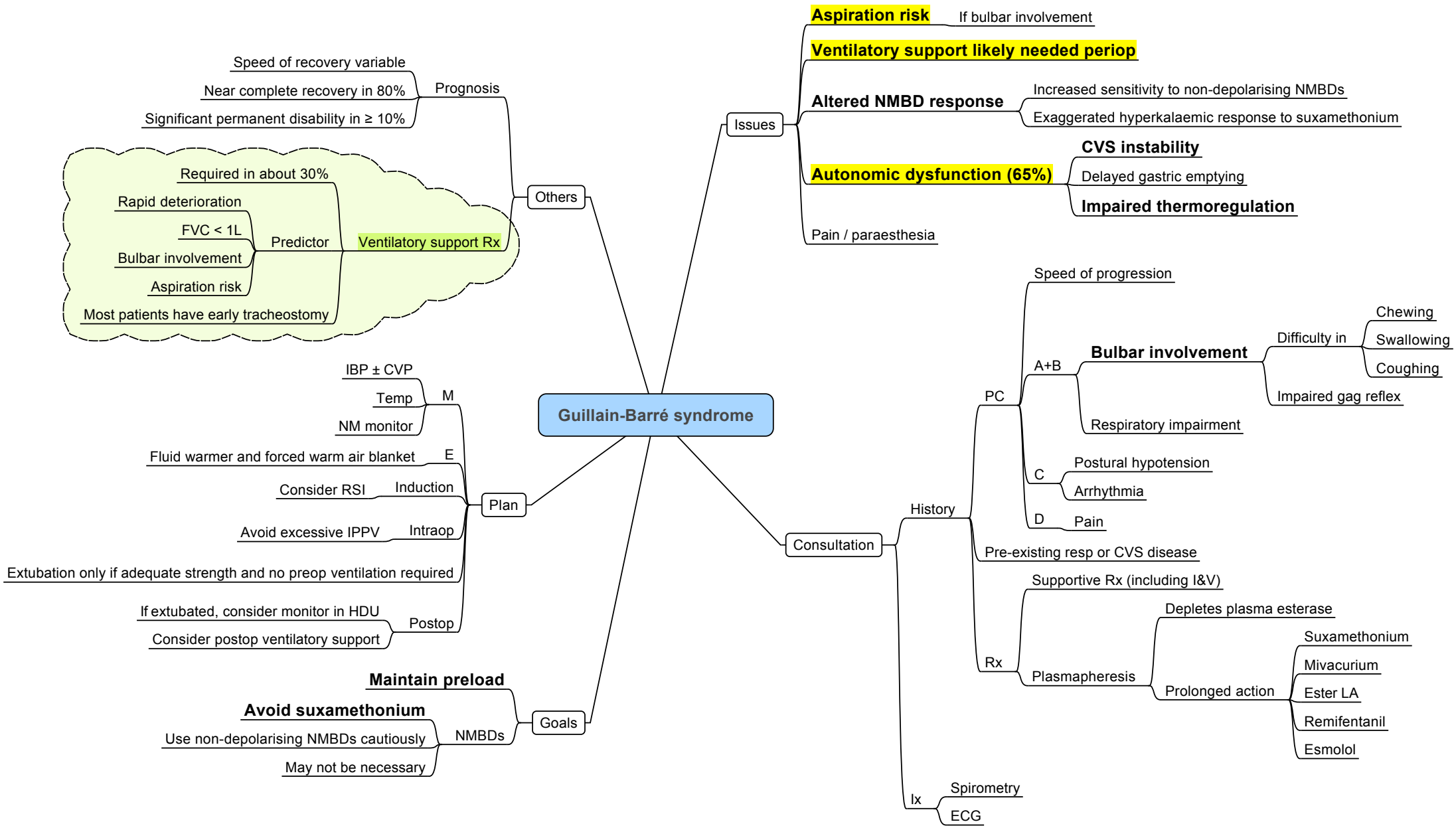

ENIGMA I trial
Background
Published in Anesthesiology 2007
Q
Does avoidance of N2O decrease length of hospital stay and other complications?
(Paraphrased)
A "pragmatic" trial
i.e. To compare impacts of different managements in routine clinical settings
NOT "explanatory" trial
which would be to establish causal relationships
e.g. A causes B, C, and D, but not E nor F
Method
Prospective RCT
Single-blinded
Only anaesthetists are not blinded
Multi-centered (19 hospitals)
n = 2022
Groups
N2O-free group
O2 80% + Nitrogen 20%
N2O group
N2O 70% + O2 30%
Inclusion
Adult undergoing major surgeries
i.e.
Surgery anticipated to exceed 2 hours
Expected to be in hospital for ≥ 3 days postop
Exclusion
Cardiac surgery
Thoracic surgery requiring one-lung ventilation
N2O contraindicated as judged attending anaesthetist
Outcome
Primary
Duration of hospital stay
Secondary
Postop complications
e.g.
Wound infection
Severe PONV
MI
Stroke
Death within 30 days
Composite end-points
Any respiratory complications
Any major complications
Design
n = 2000 would be required
90% power with two-sided alpha of 0.05 to detect...
... a reduction of mean duration of hospital stay from 4 to 3.5 days
Also have 77% power to detect decrease in wound infection from 14% to 10%
Findings
N2O-free group (compared to N2O group) has ...
Primary outcome
Shorter median duration of hospital stay
7.0 days (vs 7.1 days)
Statistically significant
Not clinically significant
More likely to be out of hospital (non-significant)
Hazard ratio (HR) = 1.09
p = 0.06
More likely to be out of ICU
HR = 1.35
p = 0.02
Secondary outcome
Less severe PONV
10% (vs 23%)
OR = 0.40, 95% CI 0.31 to 0.51
p < 0.001
Less wound infection
7.7% (vs 10%)
OR = 0.72, 95% CI 0.53 to 0.98
p = 0.034
Less pneumonia (1.5% (vs 3%))
Less atelectasis (7.5% (vs 13%))
Less fever (28% (vs 34%))
Composite
Less likely to have any pulmonary complications
7.8% (vs 13%)
OR = 0.57
95% CI 0.42 to 0.76
p < 0.001
Less likely to have at least one major complication
16% (vs 21%)
OR = 0.71
95% CI 0.56 to 0.89
p = 0.003
Others
Less MI (non-significant)
OR = 0.54
p = 0.20
Less 30-day mortality (non-significant)
OR = 0.34
p = 0.10
Limitation
Multiple comparisons
Increased risk of type I error
Treat subgroup analysis with caution
Might not extrapolate to...
Minor surgery
Paediatric surgery
Others
Differences in outcomes may be due to...
N2O avoidance
Use of high FiO2
ENIGMA II
A follow-up study to investigate effect of N2O on MI and 30-day mortality
Not significant in ENIGMA I, but very clinically significant if true
Some criticised that data in this study (a pragmatic trial) is analysed inappropriately as per explanatory trials

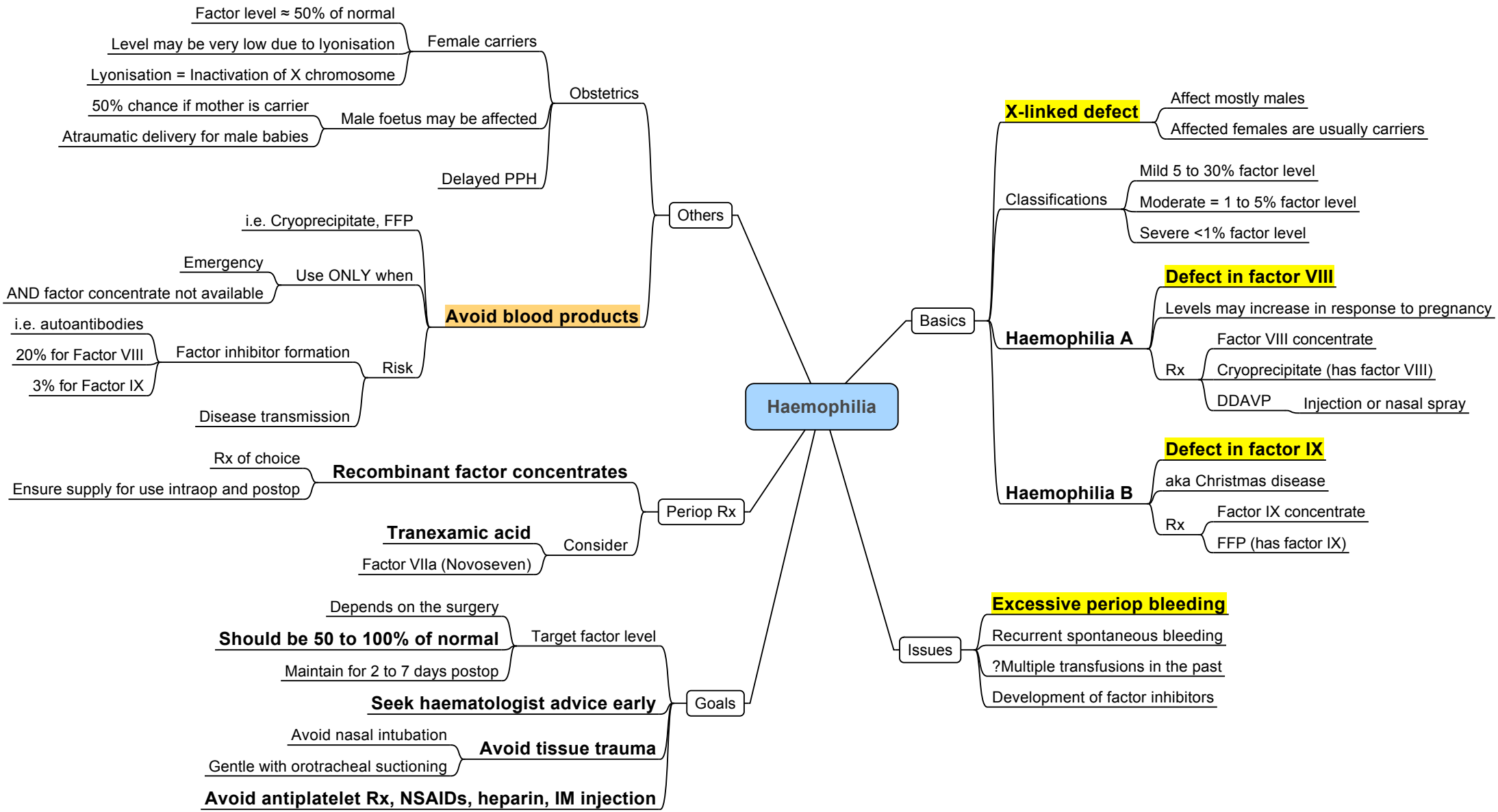

Epilepsy - AMx
Issues
Periop seizures
Epilepsy Rx
Interaction with anaesthetic drugs
SE's
Common
Sedation
Fatigue
Weight changes
Blood dyscrasia
Anaemia
Thrombocytopaenia
Leukopaenia
Hyponatraemia
Abnormal LFTs
Enzyme induction
Drugs to avoid
Excitatory phenomena
Propofol
Etomidate
Some antiemetics
Prochlorperazine
Droperidol
Metoclopramide
Lower seizure threshold
Pro-convulsant metabolites
Atracurium
Tramadol
Pethidine
Methohexital
Enflurane
?Ketamine
Medicolegal
Record any abnormal movements carefully
Misdiagnosis can have profund effect on patient's life
Plan
D
Consider benzodiazepine as premed
Thiopentone preferred over propofol
Avoid aminosteroid NMBDs
Decreased duration
... due to hepatic enzyme induction
Intraop
Antiemetics
Postop
Resume medication as early as possible
Alternative medication if oral Rx cannot be given
e.g. Phenytoin IV
Goals
Minimise disruption to Rx
Avoid prolonged fasting
Cease medication as late as possible
Maintain normocapnoea
Avoid hyperventilation
Avoid certain drugs
Excitatory movements that can be confused for seizures
Lowered seizure threshold
Consultation
History
Seizure
Nature / Type
Timing
Frequency
Trigger if any
Time since last seizure
Impact on life
e.g. Driver's licence eligibility
Rx
Anticonvulsant Rx
Dosage
Timing
Ix
Bloods
FBC
Electrolytes
Glucose
LFTs
Optimisation
If poor control
Consider delay surgery
Referral to neurologist

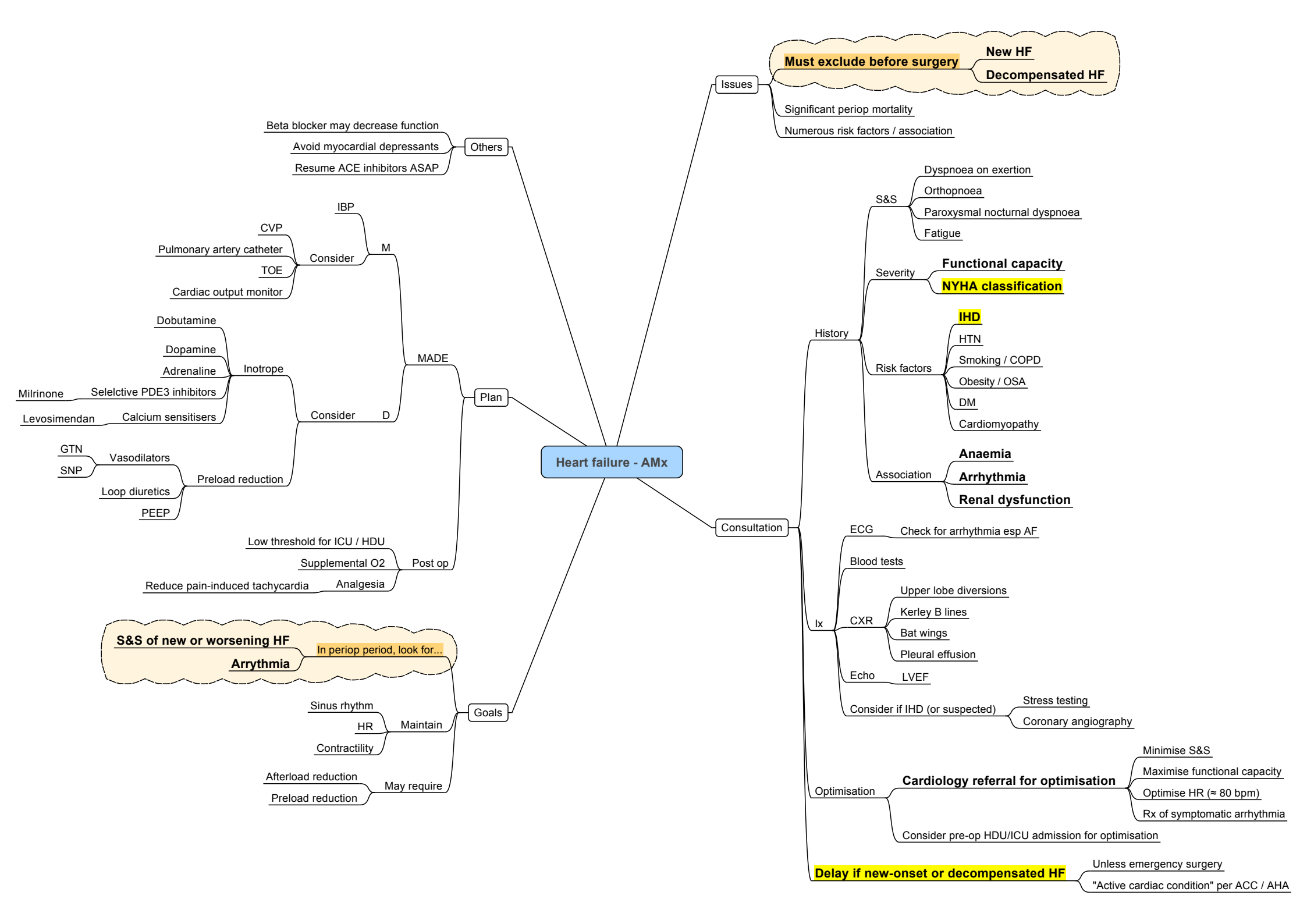
For intubated non-hypoxic patient, at end of a GA

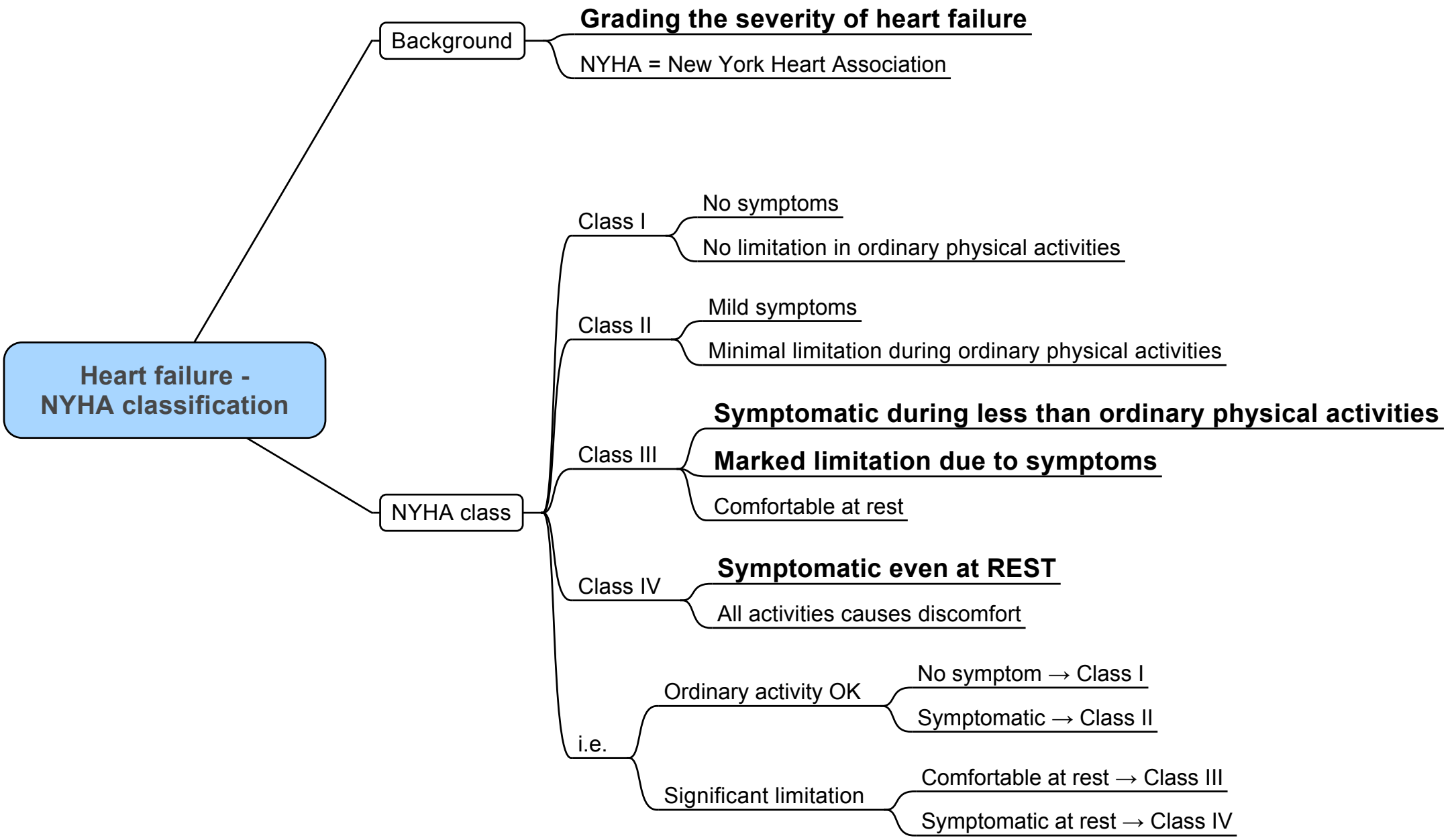


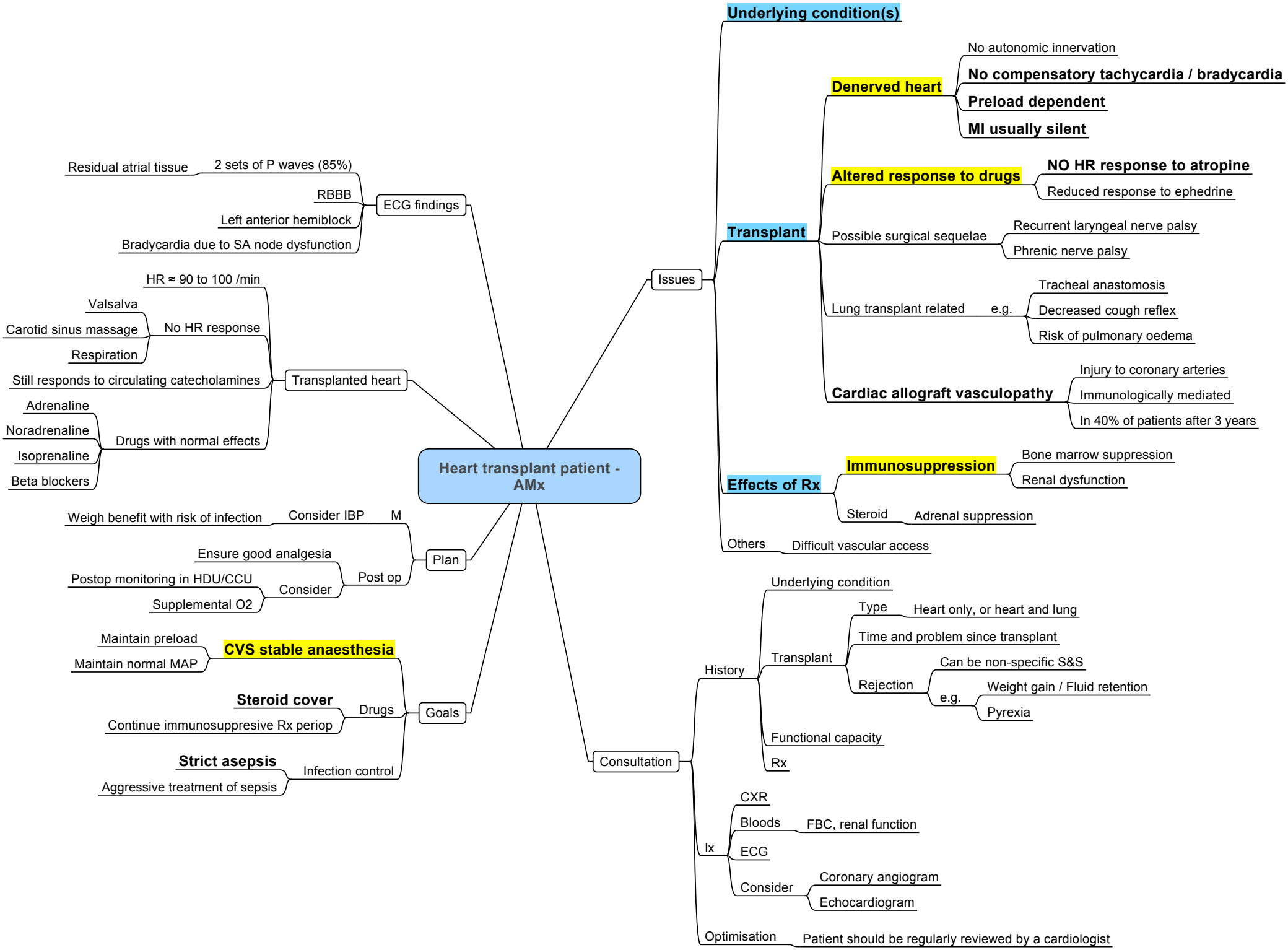

Fontan procedure, Patient with - AMx
Fontan
A palliative procedure
Diverts all systemic venous blood into pulmonary arteries
RV bypassed
Used for complex congenital heart disease
... where biventricular repair not possible
Issues
Effectively one functional ventricle
... for both pulmonary and systemic circulation
Pulmonary circulation
Passive flow only
NO active pumping
Driven by [CVP - Atrial pressure]
Cardiac output
VERY preload dependent
Fluid overload and hypovolaemia poorly tolerated
Reliant on sinus rhythm
Poor response to stress or exercise
C
Arrhythmia
45% at 10 years
Ventricular dysfunction
Grade II failure in 70% at 10 years
Right to left shunt
Others
Risk of paradoxical embolism
... due to fenestration between RA and conduit
Risk of bacterial endocarditis
Thromboemoblism risk
30% have had an event at 10 years
Anticoagulation Rx
Consultation
History
Underlying condition
Functional capacity
Other complications
Developmental deficits
Protein-losing enteropathy
↓ Ca2+
↓ Mg2+
Immunodeficiency
Malabsorption of fat
Poor prognosis
Rx
e.g. Warfarin Rx
Ix
ECG
Echo
CXR
Bloods
Optimisation
Liase with cardiologist
Goals
Maintain ventricular function
Maintain normal preload
Maintain sinus rhythm
Avoid negative inotrope
Maintain pulmonary circulation
Maintain CVP
Keep PVR low
Maintain spontaneous respiration if possible
Avoid
Hypoxia
Hypercarbia
Acidosis
Hypothermia
N2O
PEEP / IPPV
Plan
M
IBP
CVL (femoral)
?TOE
D
Avoid N2O
IPPV strategy
Low RR
Low PEEP
Short inspiratory time
Tidal volume 5 to 6 mL/kg
Postop
HDU / ICU postop
Others
Pregnancy
Epidural preferred for labour and surgery
Avoid vasopressor due to effect on PVR
Laparoscopic surgery
Minimise duration
Keep intra-abdominal pressure low
Increased risk of paradoxical CO2 embolism

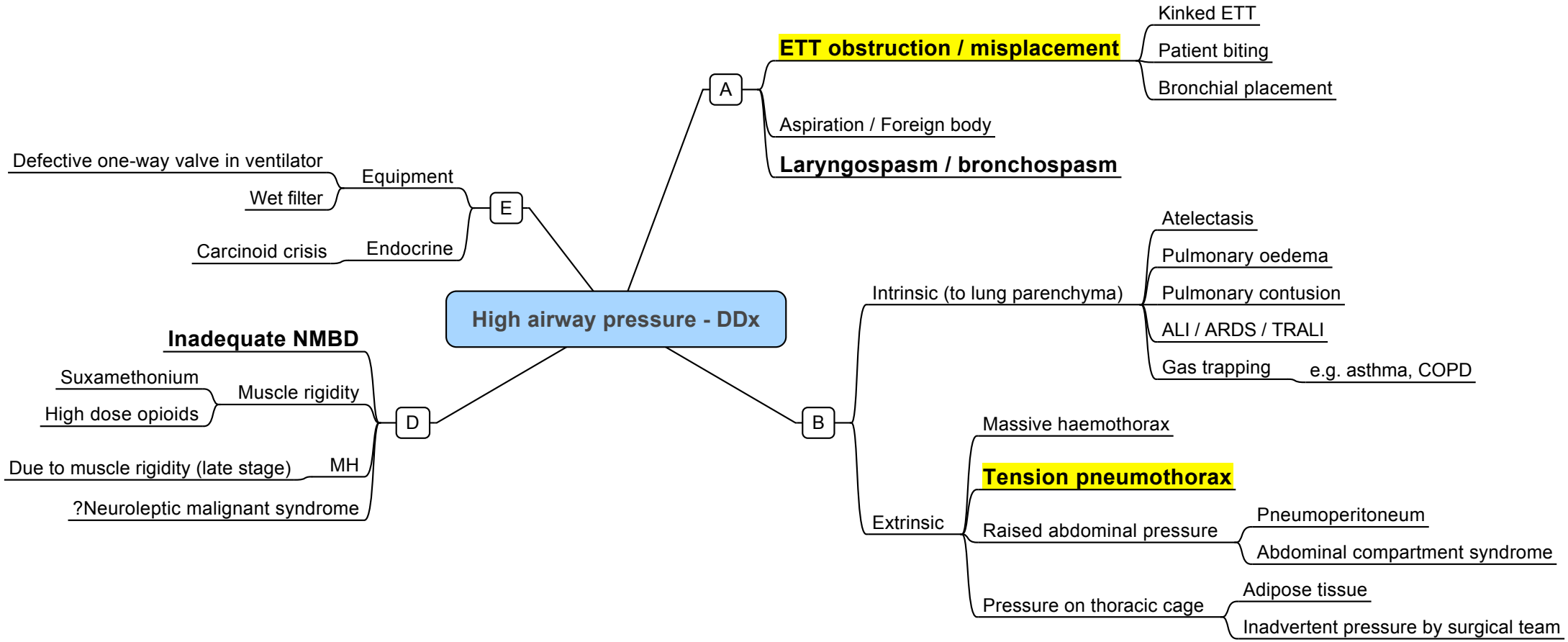

Guillain-Barré syndrome
Consultation
History
PC
Speed of progression
A+B
Bulbar involvement
Difficulty in
Chewing
Swallowing
Coughing
Impaired gag reflex
Respiratory impairment
C
Postural hypotension
Arrhythmia
D
Pain
Pre-existing resp or CVS disease
Rx
Supportive Rx (including I&V)
Plasmapheresis
Depletes plasma esterase
Prolonged action
Suxamethonium
Mivacurium
Ester LA
Remifentanil
Esmolol
Ix
Spirometry
ECG
Issues
Aspiration risk
If bulbar involvement
Ventilatory support likely needed periop
Altered NMBD response
Increased sensitivity to non-depolarising NMBDs
Exaggerated hyperkalaemic response to suxamethonium
Autonomic dysfunction (65%)
CVS instability
Delayed gastric emptying
Impaired thermoregulation
Pain / paraesthesia
Others
Prognosis
Speed of recovery variable
Near complete recovery in 80%
Significant permanent disability in ≥ 10%
Ventilatory support Rx
Required in about 30%
Predictor
Rapid deterioration
FVC < 1L
Bulbar involvement
Aspiration risk
Most patients have early tracheostomy
Plan
M
IBP ± CVP
Temp
NM monitor
E
Fluid warmer and forced warm air blanket
Induction
Consider RSI
Intraop
Avoid excessive IPPV
Extubation only if adequate strength and no preop ventilation required
Postop
If extubated, consider monitor in HDU
Consider postop ventilatory support
Goals
Maintain preload
NMBDs
Avoid suxamethonium
Use non-depolarising NMBDs cautiously
May not be necessary

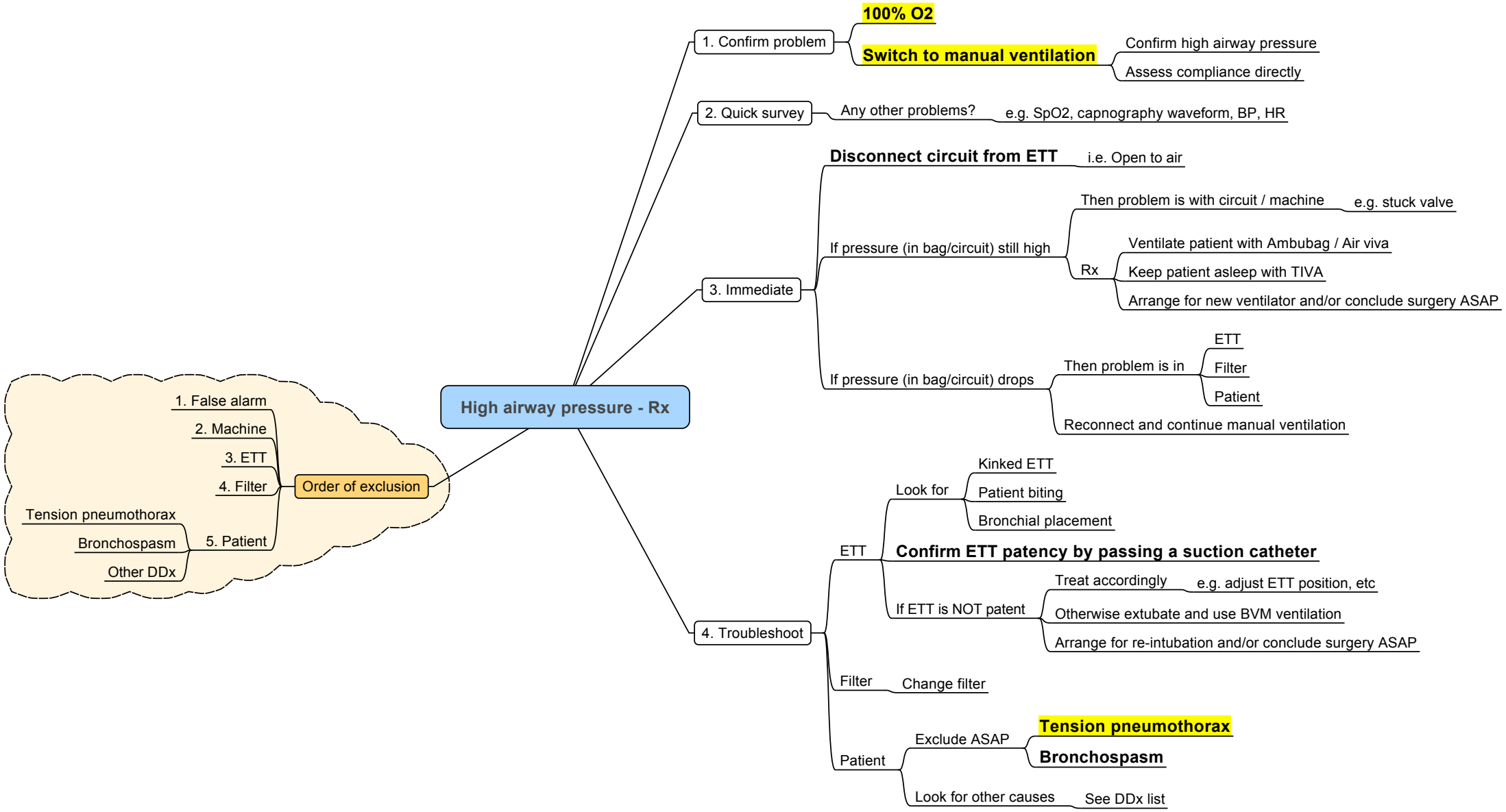

Haemophilia
Basics
X-linked defect
Affect mostly males
Affected females are usually carriers
Classifications
Mild 5 to 30% factor level
Moderate = 1 to 5% factor level
Severe <1% factor level
Haemophilia A
Defect in factor VIII
Levels may increase in response to pregnancy
Rx
Factor VIII concentrate
Cryoprecipitate (has factor VIII)
DDAVP
Injection or nasal spray
Haemophilia B
Defect in factor IX
aka Christmas disease
Rx
Factor IX concentrate
FFP (has factor IX)
Issues
Excessive periop bleeding
Recurrent spontaneous bleeding
?Multiple transfusions in the past
Development of factor inhibitors
Goals
Target factor level
Depends on the surgery
Should be 50 to 100% of normal
Maintain for 2 to 7 days postop
Seek haematologist advice early
Avoid tissue trauma
Avoid nasal intubation
Gentle with orotracheal suctioning
Avoid antiplatelet Rx, NSAIDs, heparin, IM injection
Periop Rx
Recombinant factor concentrates
Rx of choice
Ensure supply for use intraop and postop
Consider
Tranexamic acid
Factor VIIa (Novoseven)
Others
Obstetrics
Female carriers
Factor level ≈ 50% of normal
Level may be very low due to lyonisation
Lyonisation = Inactivation of X chromosome
Male foetus may be affected
50% chance if mother is carrier
Atraumatic delivery for male babies
Delayed PPH
Avoid blood products
Use ONLY when
Emergency
AND factor concentrate not available
Risk
Factor inhibitor formation
i.e. autoantibodies
20% for Factor VIII
3% for Factor IX
Disease transmission
i.e. Cryoprecipitate, FFP

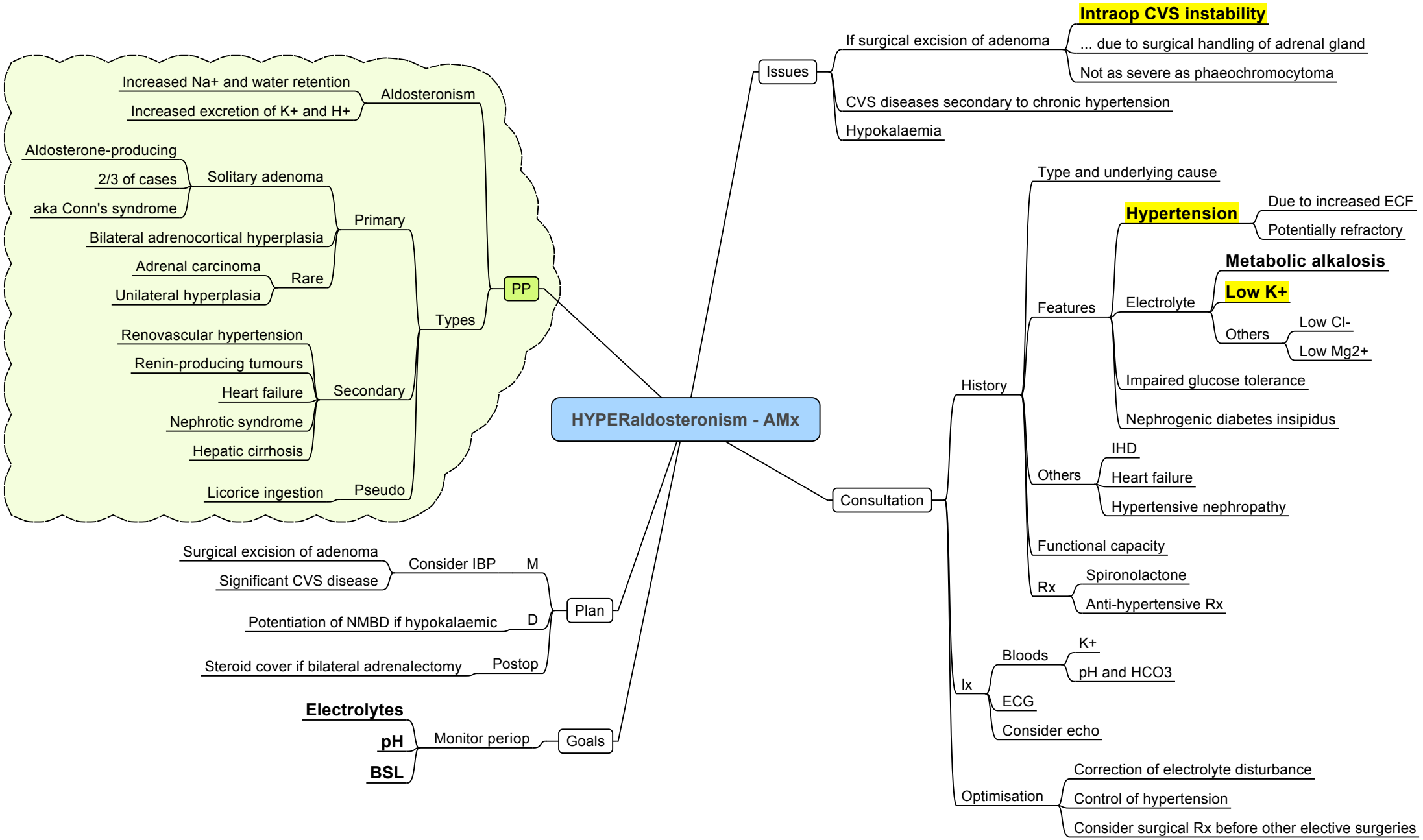

Heart failure - AMx
Consultation
History
S&S
Dyspnoea on exertion
Orthopnoea
Paroxysmal nocturnal dyspnoea
Fatigue
Severity
Functional capacity
NYHA classification
Risk factors
IHD
HTN
Smoking / COPD
Obesity / OSA
DM
Cardiomyopathy
Association
Anaemia
Arrhythmia
Renal dysfunction
Ix
ECG
Check for arrhythmia esp AF
Blood tests
CXR
Upper lobe diversions
Kerley B lines
Bat wings
Pleural effusion
Echo
LVEF
Consider if IHD (or suspected)
Stress testing
Coronary angiography
Optimisation
Cardiology referral for optimisation
Minimise S&S
Maximise functional capacity
Optimise HR (≈ 80 bpm)
Rx of symptomatic arrhythmia
Consider pre-op HDU/ICU admission for optimisation
Delay if new-onset or decompensated HF
Unless emergency surgery
"Active cardiac condition" per ACC / AHA
Issues
Must exclude before surgery
New HF
Decompensated HF
Significant periop mortality
Numerous risk factors / association
Plan
Others
Beta blocker may decrease function
Avoid myocardial depressants
Resume ACE inhibitors ASAP
MADE
M
Consider
IBP
CVP
Pulmonary artery catheter
TOE
Cardiac output monitor
D
Consider
Inotrope
Dobutamine
Dopamine
Adrenaline
Selelctive PDE3 inhibitors
Milrinone
Calcium sensitisers
Levosimendan
Preload reduction
Vasodilators
GTN
SNP
Loop diuretics
PEEP
Post op
Low threshold for ICU / HDU
Supplemental O2
Analgesia
Reduce pain-induced tachycardia
Goals
S&S of new or worsening HF
Arrythmia
In periop period, look for...
Maintain
Sinus rhythm
HR
Contractility
May require
Afterload reduction
Preload reduction

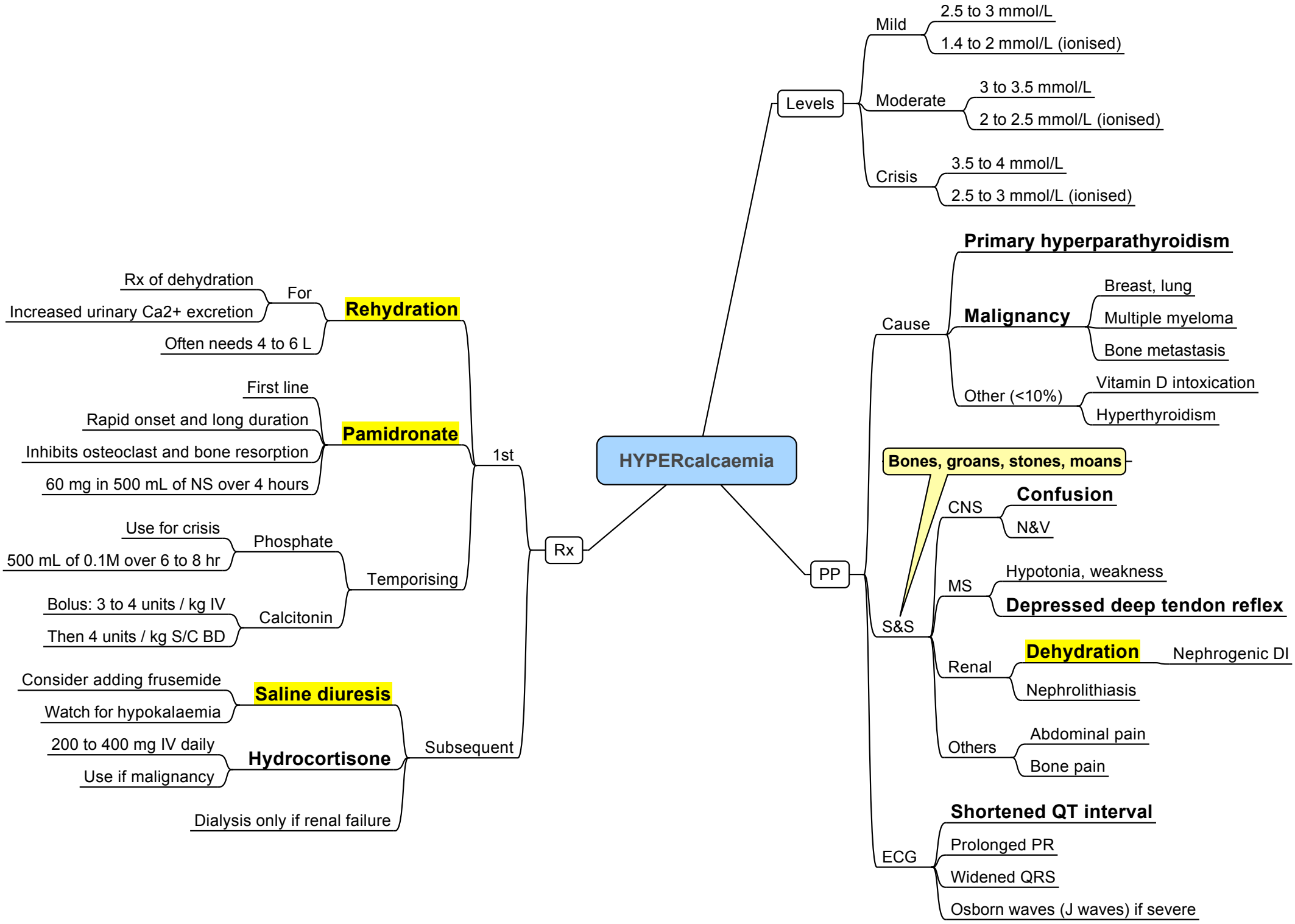

Heart failure - NYHA classification
Background
Grading the severity of heart failure
NYHA = New York Heart Association
NYHA class
Class I
No symptoms
No limitation in ordinary physical activities
Class II
Mild symptoms
Minimal limitation during ordinary physical activities
Class III
Symptomatic during less than ordinary physical activities
Marked limitation due to symptoms
Comfortable at rest
Class IV
Symptomatic even at REST
All activities causes discomfort
i.e.
Ordinary activity OK
No symptom → Class I
Symptomatic → Class II
Significant limitation
Comfortable at rest → Class III
Symptomatic at rest → Class IV

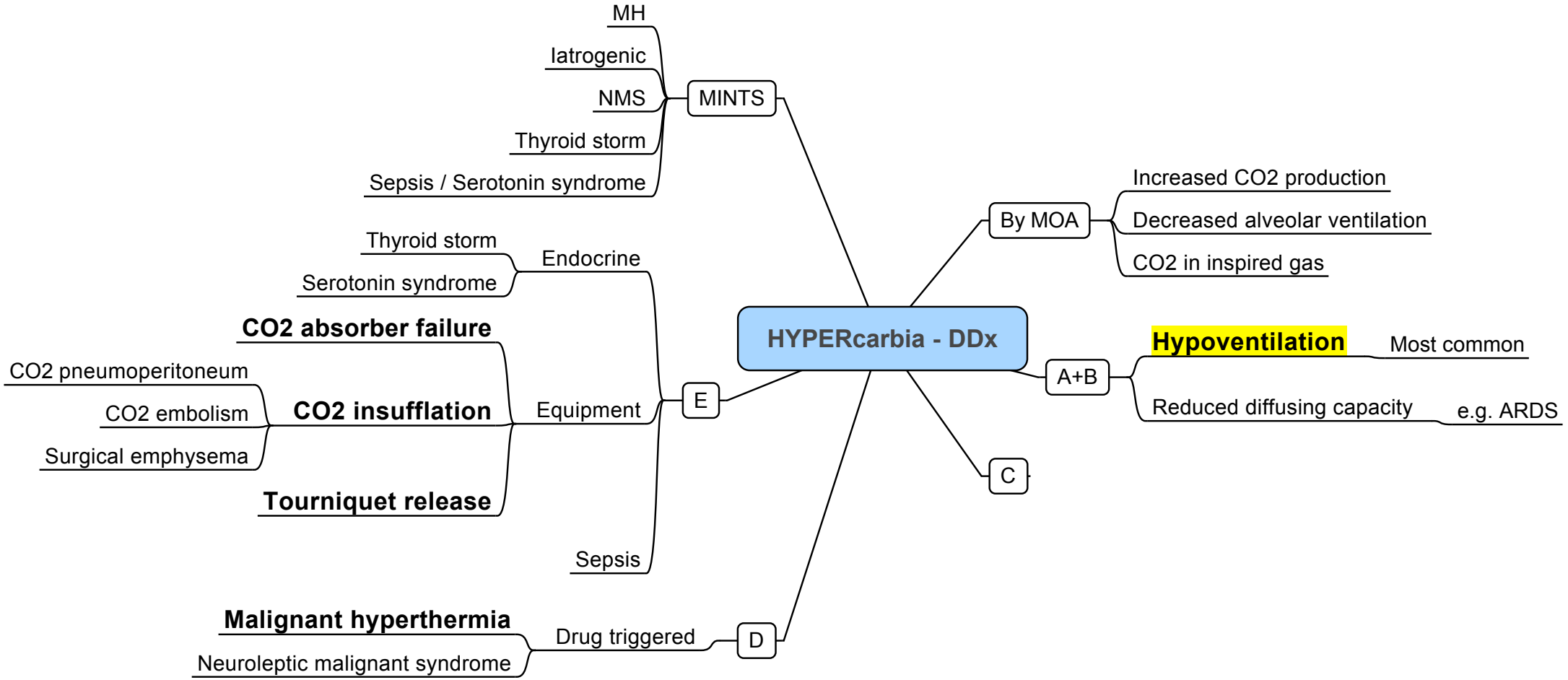

Heart transplant patient - AMx
Issues
Underlying condition(s)
Transplant
Denerved heart
No autonomic innervation
No compensatory tachycardia / bradycardia
Preload dependent
MI usually silent
Altered response to drugs
NO HR response to atropine
Reduced response to ephedrine
Possible surgical sequelae
Recurrent laryngeal nerve palsy
Phrenic nerve palsy
Lung transplant related
e.g.
Tracheal anastomosis
Decreased cough reflex
Risk of pulmonary oedema
Cardiac allograft vasculopathy
Injury to coronary arteries
Immunologically mediated
In 40% of patients after 3 years
Effects of Rx
Immunosuppression
Bone marrow suppression
Renal dysfunction
Steroid
Adrenal suppression
Others
Difficult vascular access
ECG findings
Residual atrial tissue
2 sets of P waves (85%)
RBBB
Left anterior hemiblock
Bradycardia due to SA node dysfunction
Transplanted heart
HR ≈ 90 to 100 /min
No HR response
Valsalva
Carotid sinus massage
Respiration
Still responds to circulating catecholamines
Drugs with normal effects
Adrenaline
Noradrenaline
Isoprenaline
Beta blockers
Plan
Weigh benefit with risk of infection
Consider IBP
M
Post op
Ensure good analgesia
Consider
Postop monitoring in HDU/CCU
Supplemental O2
Goals
CVS stable anaesthesia
Maintain preload
Maintain normal MAP
Drugs
Steroid cover
Continue immunosuppresive Rx periop
Infection control
Strict asepsis
Aggressive treatment of sepsis
Consultation
History
Underlying condition
Transplant
Type
Heart only, or heart and lung
Time and problem since transplant
Rejection
Can be non-specific S&S
e.g.
Weight gain / Fluid retention
Pyrexia
Functional capacity
Rx
Ix
CXR
Bloods
FBC, renal function
ECG
Consider
Coronary angiogram
Echocardiogram
Optimisation
Patient should be regularly reviewed by a cardiologist

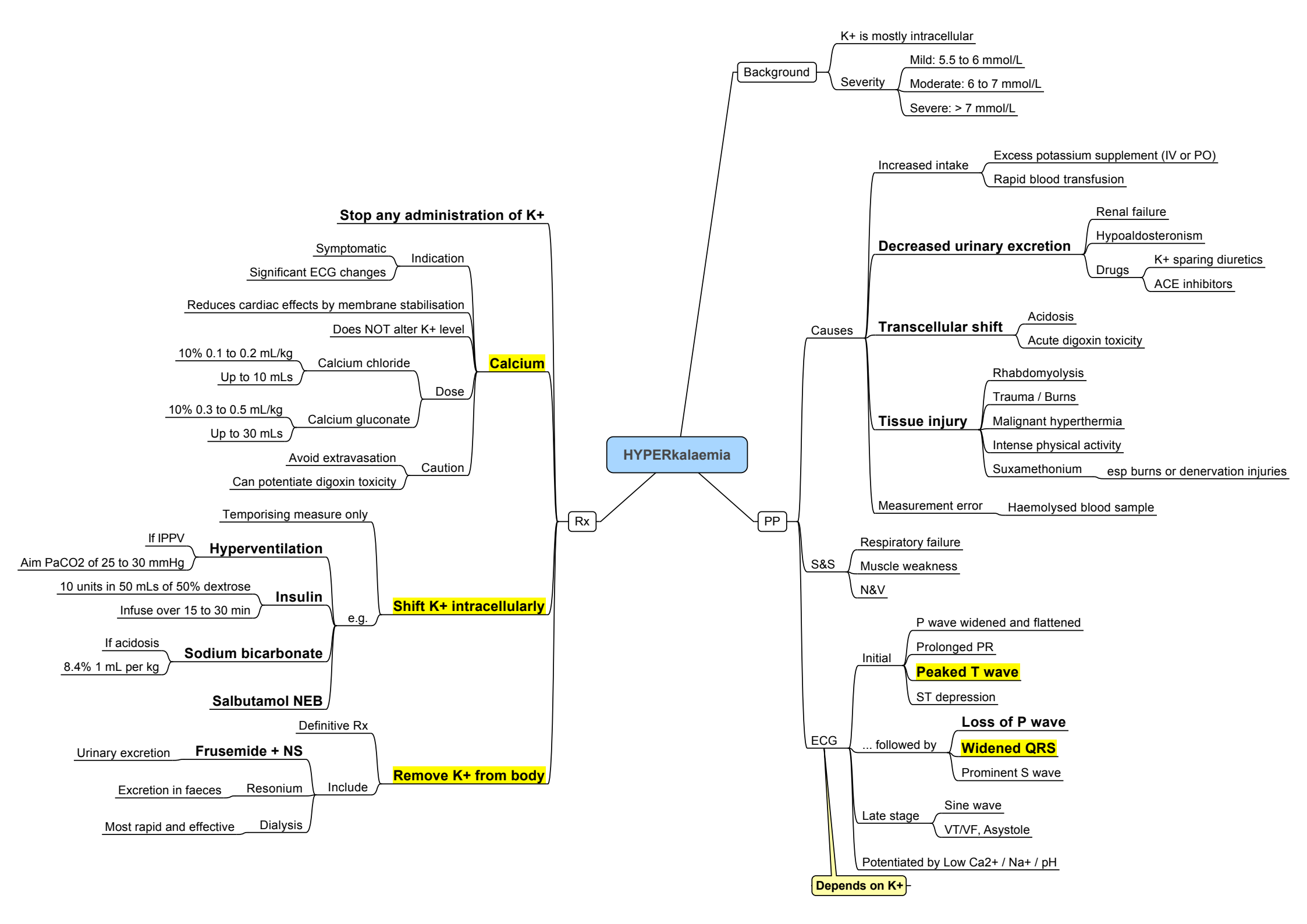

High airway pressure - DDx
A
ETT obstruction / misplacement
Kinked ETT
Patient biting
Bronchial placement
Aspiration / Foreign body
Laryngospasm / bronchospasm
B
Intrinsic (to lung parenchyma)
Atelectasis
Pulmonary oedema
Pulmonary contusion
ALI / ARDS / TRALI
Gas trapping
e.g. asthma, COPD
Extrinsic
Massive haemothorax
Tension pneumothorax
Raised abdominal pressure
Pneumoperitoneum
Abdominal compartment syndrome
Pressure on thoracic cage
Adipose tissue
Inadvertent pressure by surgical team
D
Inadequate NMBD
Muscle rigidity
Suxamethonium
High dose opioids
MH
Due to muscle rigidity (late stage)
?Neuroleptic malignant syndrome
E
Equipment
Defective one-way valve in ventilator
Wet filter
Endocrine
Carcinoid crisis

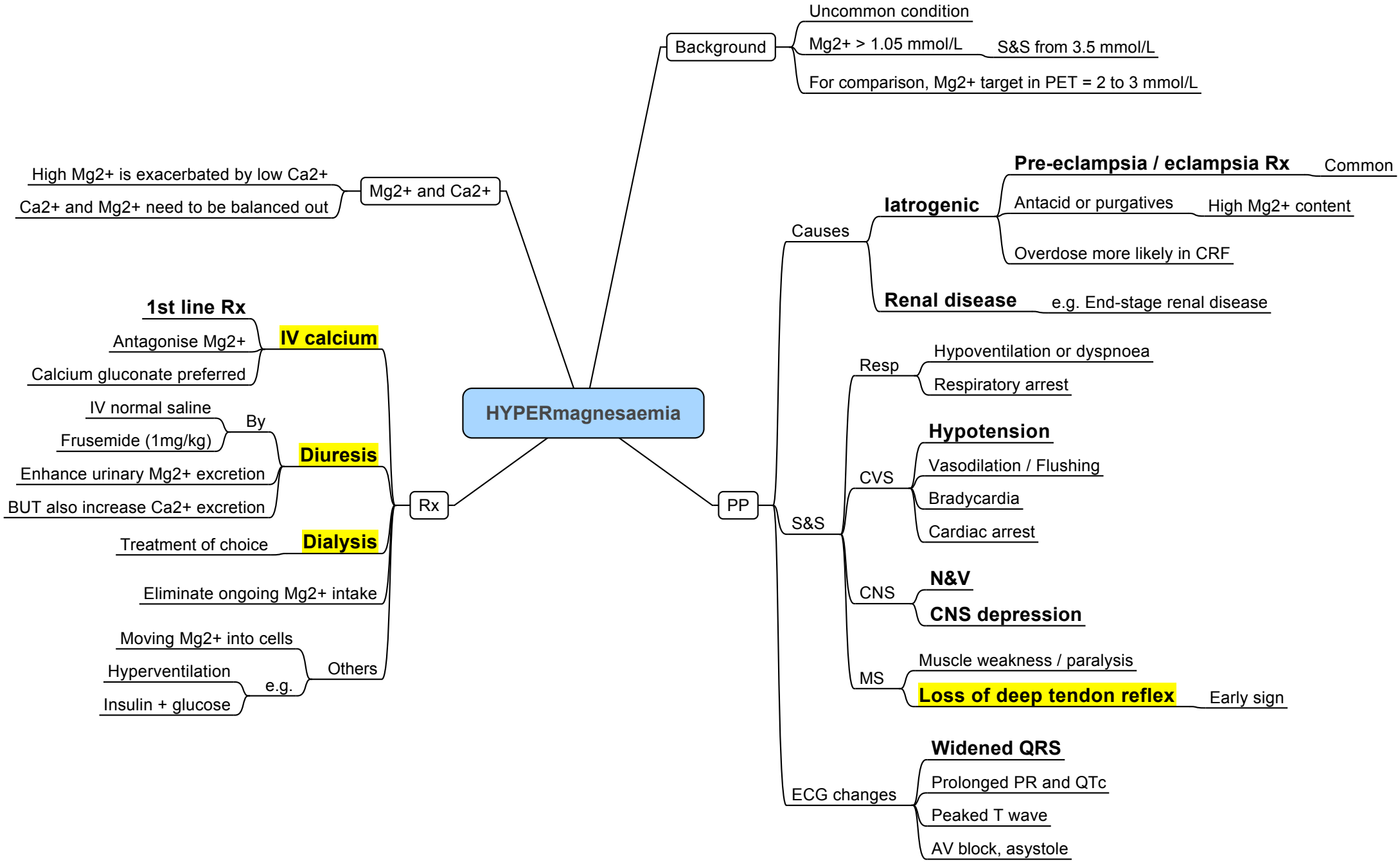

High airway pressure - Rx
1. Confirm problem
100% O2
Switch to manual ventilation
Confirm high airway pressure
Assess compliance directly
2. Quick survey
Any other problems?
e.g. SpO2, capnography waveform, BP, HR
3. Immediate
Disconnect circuit from ETT
i.e. Open to air
If pressure (in bag/circuit) still high
Then problem is with circuit / machine
e.g. stuck valve
Rx
Ventilate patient with Ambubag / Air viva
Keep patient asleep with TIVA
Arrange for new ventilator and/or conclude surgery ASAP
If pressure (in bag/circuit) drops
Then problem is in
ETT
Filter
Patient
Reconnect and continue manual ventilation
4. Troubleshoot
ETT
Look for
Kinked ETT
Patient biting
Bronchial placement
Confirm ETT patency by passing a suction catheter
If ETT is NOT patent
Treat accordingly
e.g. adjust ETT position, etc
Otherwise extubate and use BVM ventilation
Arrange for re-intubation and/or conclude surgery ASAP
Filter
Change filter
Patient
Exclude ASAP
Tension pneumothorax
Bronchospasm
Look for other causes
See DDx list
Order of exclusion
1. False alarm
2. Machine
3. ETT
4. Filter
5. Patient
Tension pneumothorax
Bronchospasm
Other DDx

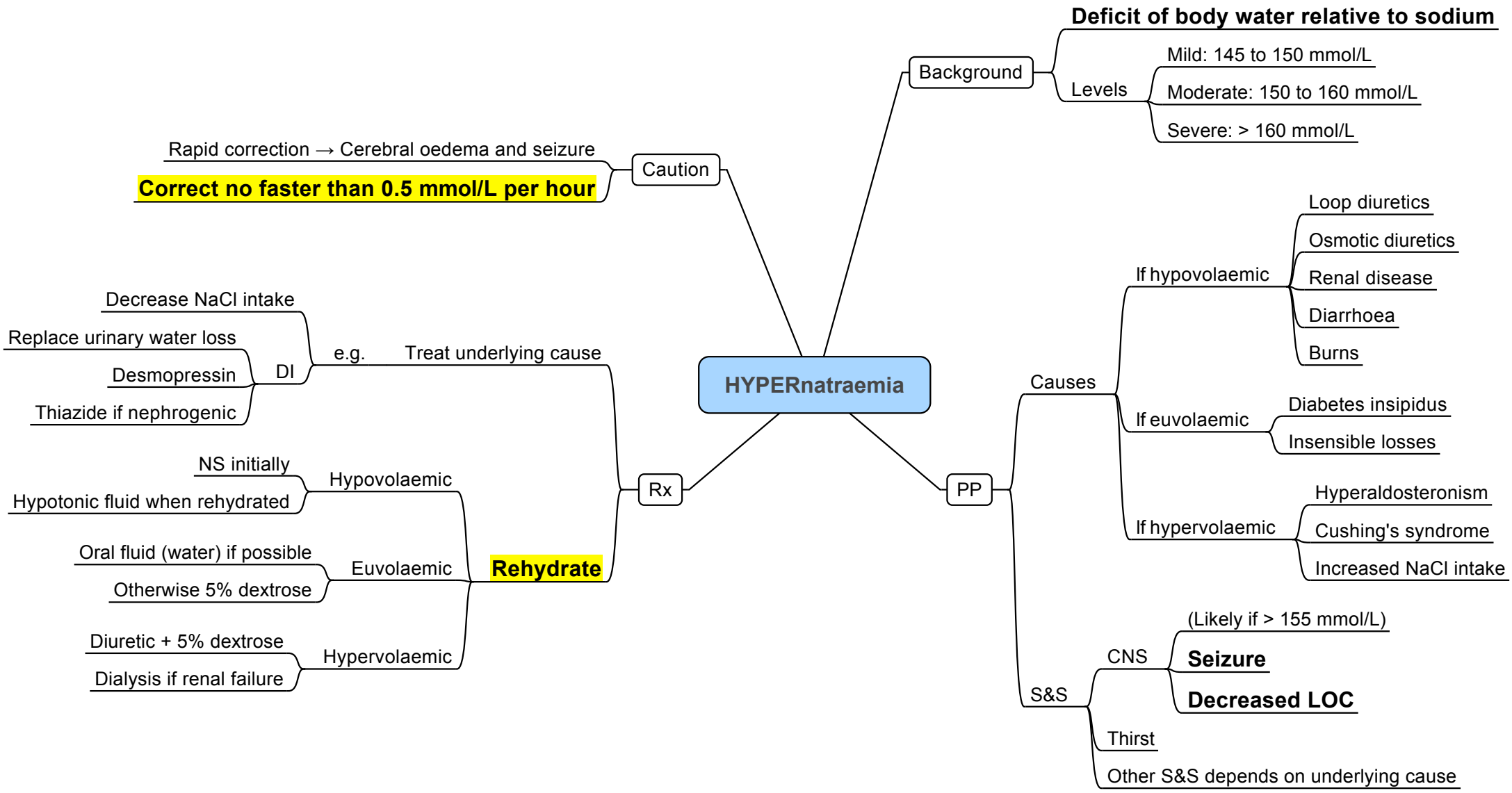

HYPERaldosteronism - AMx
PP
Aldosteronism
Increased Na+ and water retention
Increased excretion of K+ and H+
Types
Primary
Solitary adenoma
Aldosterone-producing
2/3 of cases
aka Conn's syndrome
Bilateral adrenocortical hyperplasia
Rare
Adrenal carcinoma
Unilateral hyperplasia
Secondary
Renovascular hypertension
Renin-producing tumours
Heart failure
Nephrotic syndrome
Hepatic cirrhosis
Pseudo
Licorice ingestion
Issues
If surgical excision of adenoma
Intraop CVS instability
... due to surgical handling of adrenal gland
Not as severe as phaeochromocytoma
CVS diseases secondary to chronic hypertension
Hypokalaemia
Consultation
History
Type and underlying cause
Features
Hypertension
Due to increased ECF
Potentially refractory
Electrolyte
Metabolic alkalosis
Low K+
Others
Low Cl-
Low Mg2+
Impaired glucose tolerance
Nephrogenic diabetes insipidus
Others
IHD
Heart failure
Hypertensive nephropathy
Functional capacity
Rx
Spironolactone
Anti-hypertensive Rx
Ix
Bloods
K+
pH and HCO3
ECG
Consider echo
Optimisation
Correction of electrolyte disturbance
Control of hypertension
Consider surgical Rx before other elective surgeries
Plan
Consider IBP
M
Surgical excision of adenoma
Significant CVS disease
D
Potentiation of NMBD if hypokalaemic
Postop
Steroid cover if bilateral adrenalectomy
Goals
Monitor periop
Electrolytes
pH
BSL

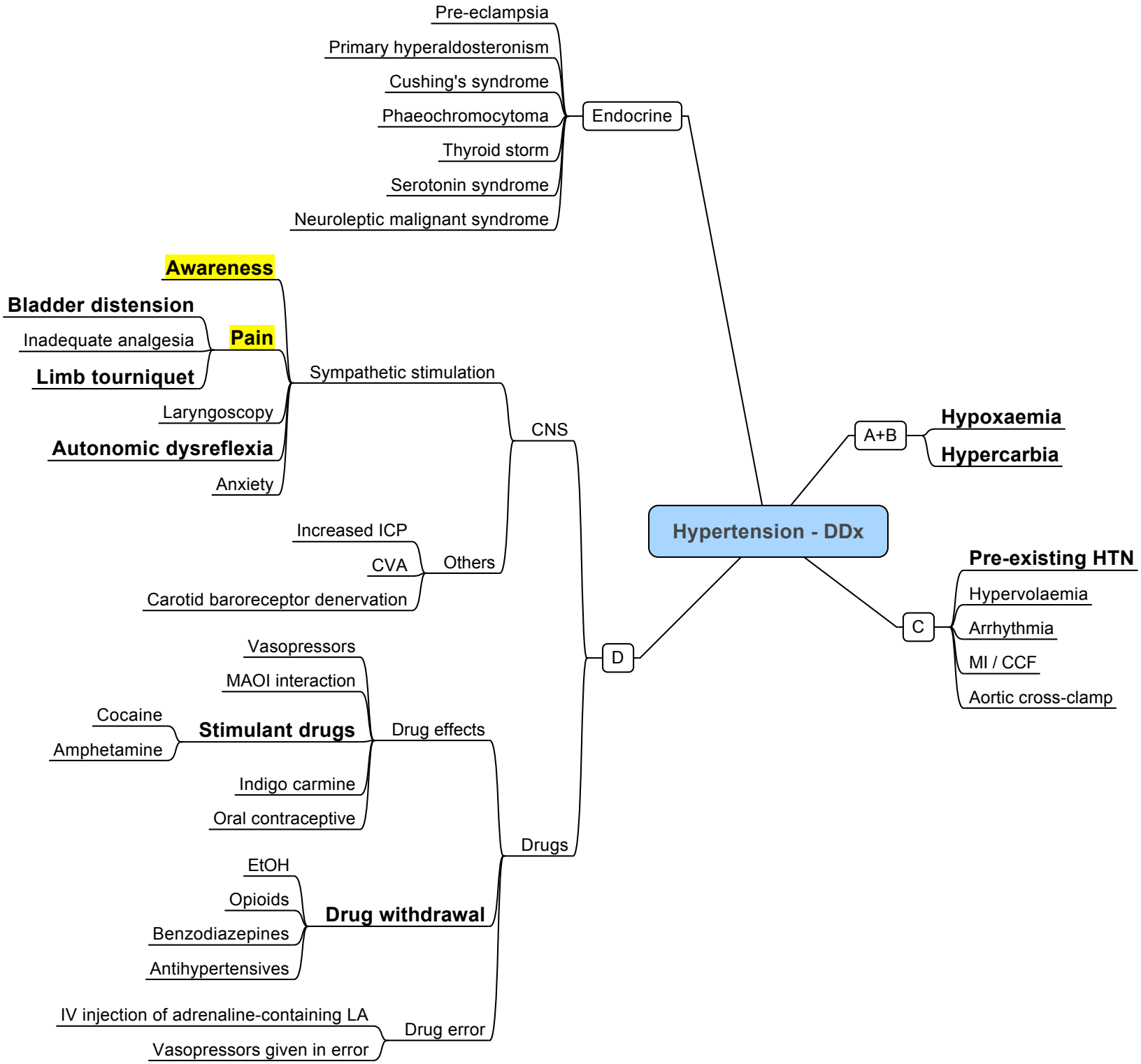

HYPERcalcaemia
Levels
Mild
2.5 to 3 mmol/L
1.4 to 2 mmol/L (ionised)
Moderate
3 to 3.5 mmol/L
2 to 2.5 mmol/L (ionised)
Crisis
3.5 to 4 mmol/L
2.5 to 3 mmol/L (ionised)
Rx
1st
Rehydration
For
Rx of dehydration
Increased urinary Ca2+ excretion
Often needs 4 to 6 L
Pamidronate
First line
Rapid onset and long duration
Inhibits osteoclast and bone resorption
60 mg in 500 mL of NS over 4 hours
Temporising
Phosphate
Use for crisis
500 mL of 0.1M over 6 to 8 hr
Calcitonin
Bolus: 3 to 4 units / kg IV
Then 4 units / kg S/C BD
Subsequent
Saline diuresis
Consider adding frusemide
Watch for hypokalaemia
Hydrocortisone
200 to 400 mg IV daily
Use if malignancy
Dialysis only if renal failure
PP
Cause
Primary hyperparathyroidism
Malignancy
Breast, lung
Multiple myeloma
Bone metastasis
Other (<10%)
Vitamin D intoxication
Hyperthyroidism
S&S
Bones, groans, stones, moans
CNS
Confusion
N&V
MS
Hypotonia, weakness
Depressed deep tendon reflex
Renal
Dehydration
Nephrogenic DI
Nephrolithiasis
Others
Abdominal pain
Bone pain
ECG
Shortened QT interval
Prolonged PR
Widened QRS
Osborn waves (J waves) if severe

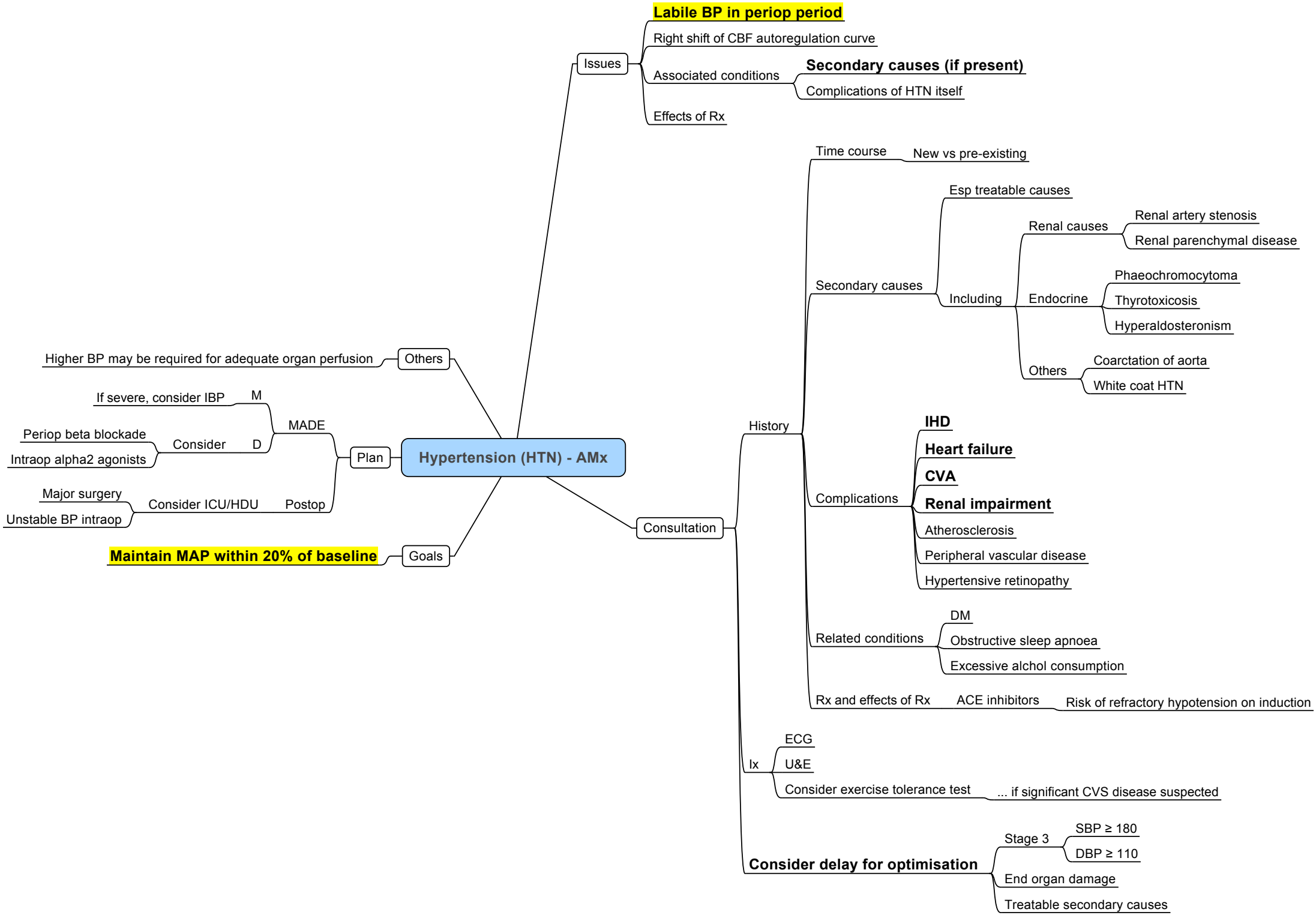

HYPERcarbia - DDx
By MOA
Increased CO2 production
Decreased alveolar ventilation
CO2 in inspired gas
A+B
Hypoventilation
Most common
Reduced diffusing capacity
e.g. ARDS
C
D
Drug triggered
Malignant hyperthermia
Neuroleptic malignant syndrome
E
Equipment
CO2 insufflation
CO2 pneumoperitoneum
CO2 embolism
Surgical emphysema
CO2 absorber failure
Tourniquet release
Endocrine
Thyroid storm
Serotonin syndrome
Sepsis
MINTS
MH
Iatrogenic
NMS
Thyroid storm
Sepsis / Serotonin syndrome

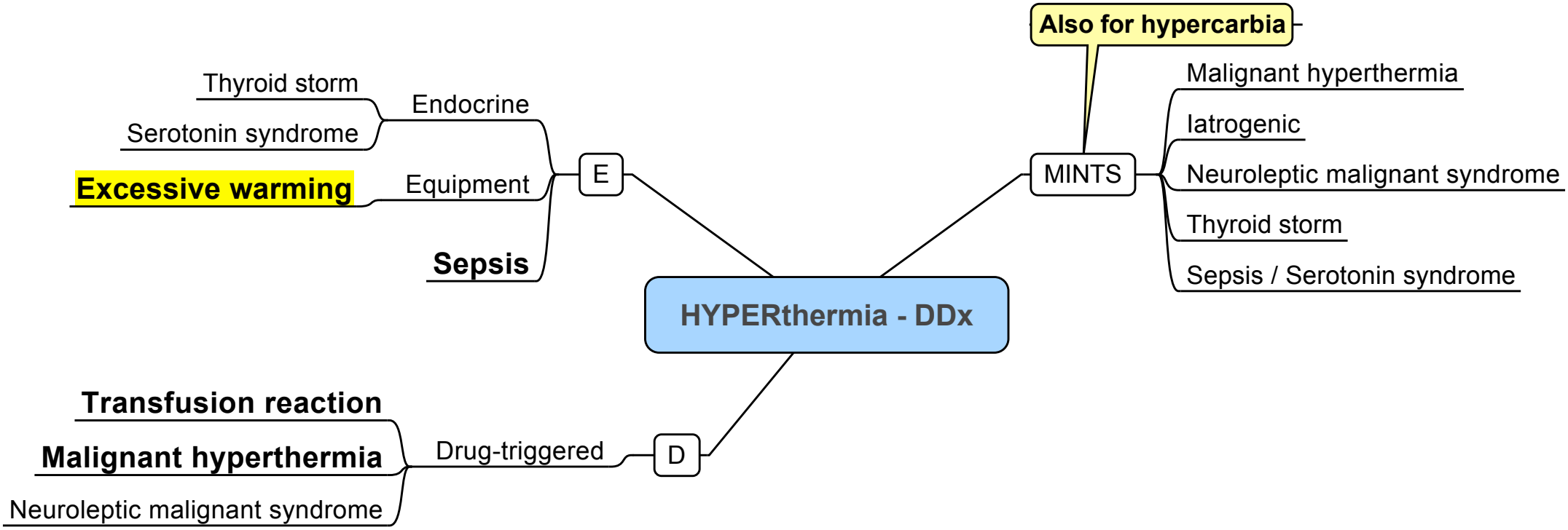

HYPERkalaemia
Background
K+ is mostly intracellular
Severity
Mild: 5.5 to 6 mmol/L
Moderate: 6 to 7 mmol/L
Severe: > 7 mmol/L
PP
Causes
Increased intake
Excess potassium supplement (IV or PO)
Rapid blood transfusion
Decreased urinary excretion
Renal failure
Hypoaldosteronism
Drugs
K+ sparing diuretics
ACE inhibitors
Transcellular shift
Acidosis
Acute digoxin toxicity
Tissue injury
Rhabdomyolysis
Trauma / Burns
Malignant hyperthermia
Intense physical activity
Suxamethonium
esp burns or denervation injuries
Measurement error
Haemolysed blood sample
S&S
Respiratory failure
Muscle weakness
N&V
ECG
Initial
P wave widened and flattened
Prolonged PR
Peaked T wave
ST depression
... followed by
Loss of P wave
Widened QRS
Prominent S wave
Late stage
Sine wave
VT/VF, Asystole
Potentiated by Low Ca2+ / Na+ / pH
Depends on K+
Rx
Stop any administration of K+
Calcium
Indication
Symptomatic
Significant ECG changes
Reduces cardiac effects by membrane stabilisation
Does NOT alter K+ level
Dose
Calcium chloride
10% 0.1 to 0.2 mL/kg
Up to 10 mLs
Calcium gluconate
10% 0.3 to 0.5 mL/kg
Up to 30 mLs
Caution
Avoid extravasation
Can potentiate digoxin toxicity
Shift K+ intracellularly
Temporising measure only
e.g.
Hyperventilation
If IPPV
Aim PaCO2 of 25 to 30 mmHg
Insulin
10 units in 50 mLs of 50% dextrose
Infuse over 15 to 30 min
Sodium bicarbonate
If acidosis
8.4% 1 mL per kg
Salbutamol NEB
Remove K+ from body
Definitive Rx
Include
Frusemide + NS
Urinary excretion
Resonium
Excretion in faeces
Dialysis
Most rapid and effective

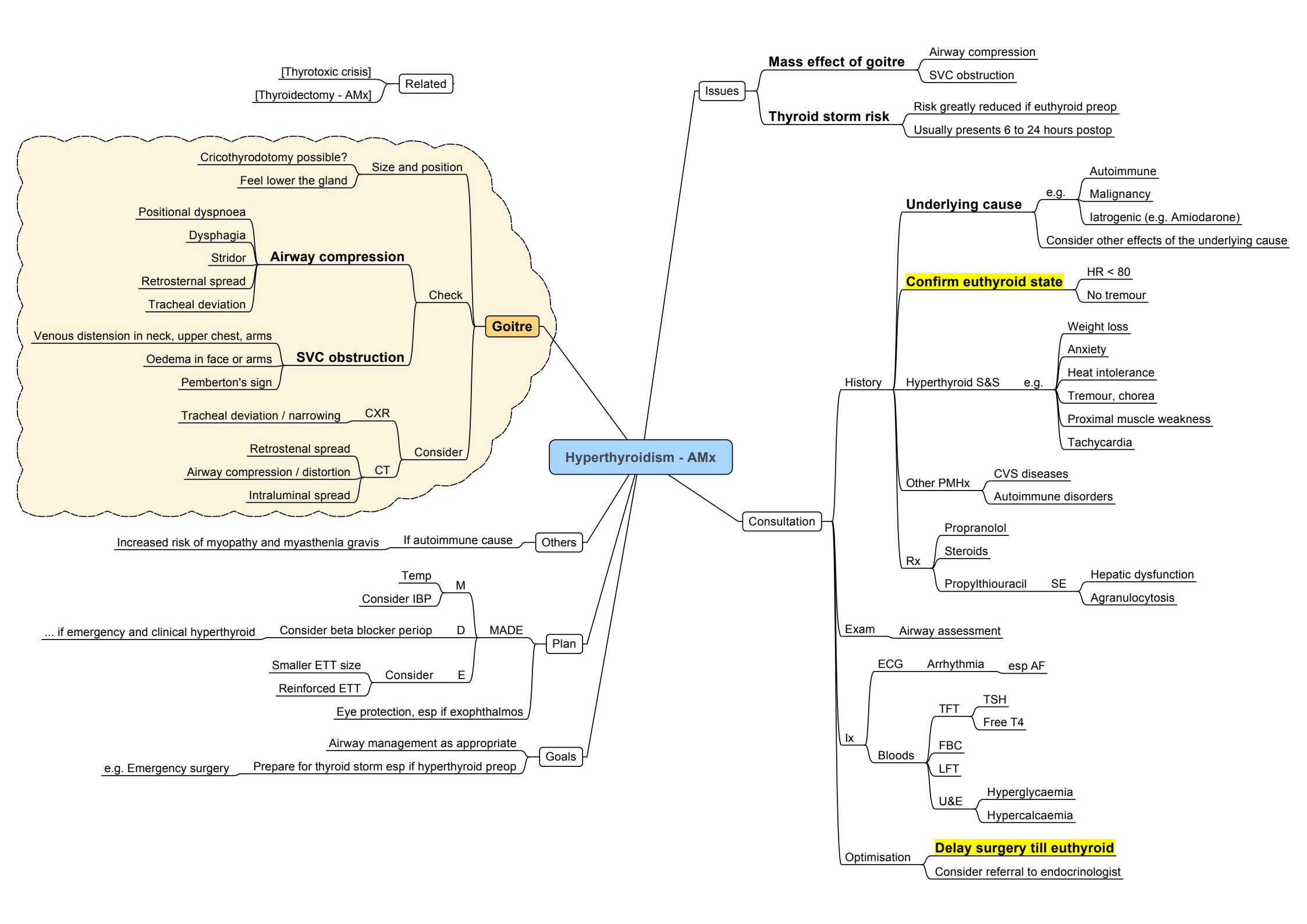

HYPERmagnesaemia
Background
Uncommon condition
Mg2+ > 1.05 mmol/L
S&S from 3.5 mmol/L
For comparison, Mg2+ target in PET = 2 to 3 mmol/L
Mg2+ and Ca2+
High Mg2+ is exacerbated by low Ca2+
Ca2+ and Mg2+ need to be balanced out
Rx
1st line Rx
IV calcium
Antagonise Mg2+
Calcium gluconate preferred
Diuresis
By
IV normal saline
Frusemide (1mg/kg)
Enhance urinary Mg2+ excretion
BUT also increase Ca2+ excretion
Dialysis
Treatment of choice
Eliminate ongoing Mg2+ intake
Others
e.g.
Moving Mg2+ into cells
Hyperventilation
Insulin + glucose
PP
Causes
Iatrogenic
Pre-eclampsia / eclampsia Rx
Common
Antacid or purgatives
High Mg2+ content
Overdose more likely in CRF
Renal disease
e.g. End-stage renal disease
S&S
Resp
Hypoventilation or dyspnoea
Respiratory arrest
CVS
Hypotension
Vasodilation / Flushing
Bradycardia
Cardiac arrest
CNS
N&V
CNS depression
MS
Muscle weakness / paralysis
Loss of deep tendon reflex
Early sign
ECG changes
Widened QRS
Prolonged PR and QTc
Peaked T wave
AV block, asystole

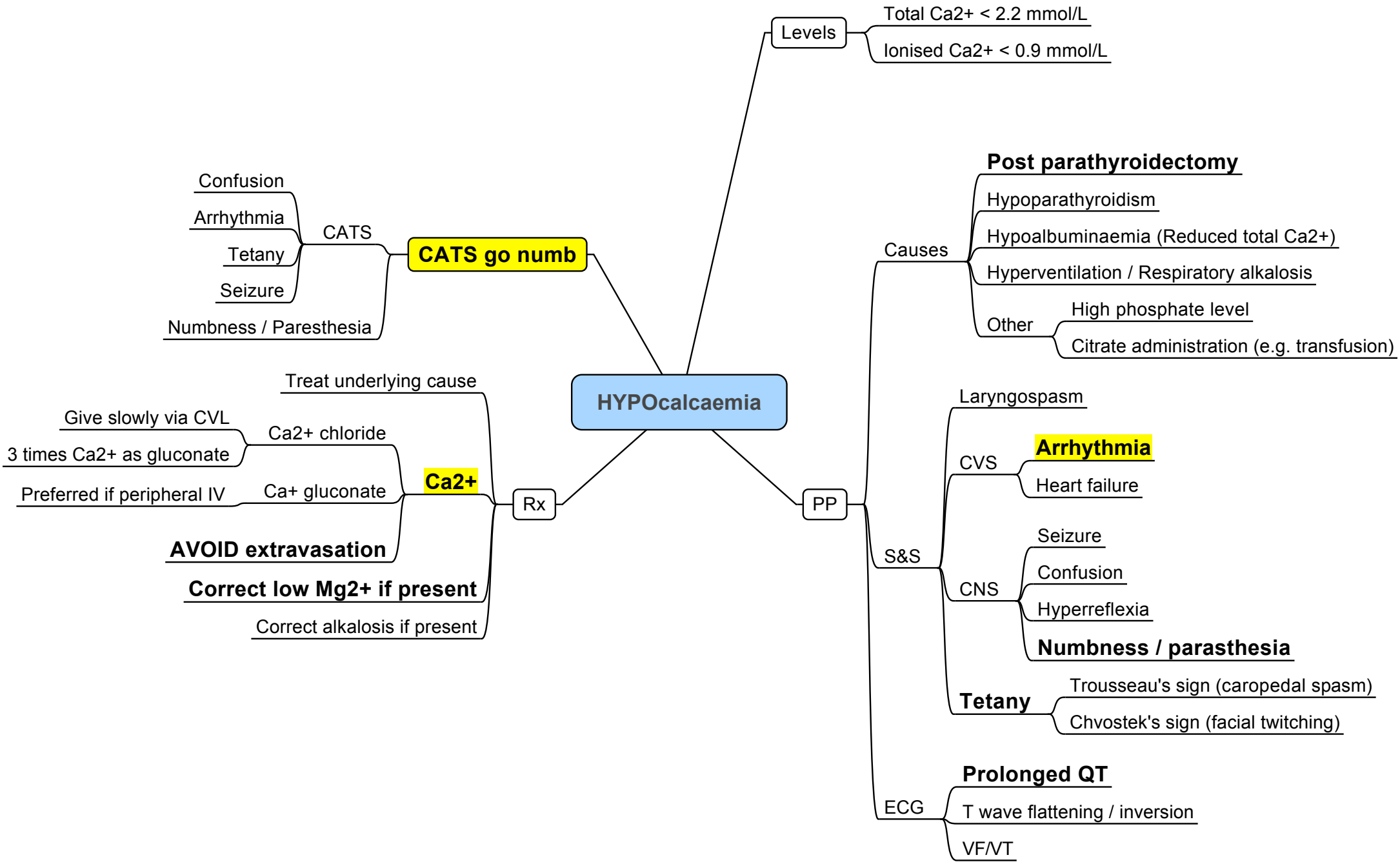

HYPERnatraemia
Background
Deficit of body water relative to sodium
Levels
Mild: 145 to 150 mmol/L
Moderate: 150 to 160 mmol/L
Severe: > 160 mmol/L
PP
Causes
If hypovolaemic
Loop diuretics
Osmotic diuretics
Renal disease
Diarrhoea
Burns
If euvolaemic
Diabetes insipidus
Insensible losses
If hypervolaemic
Hyperaldosteronism
Cushing's syndrome
Increased NaCl intake
S&S
CNS
(Likely if > 155 mmol/L)
Seizure
Decreased LOC
Thirst
Other S&S depends on underlying cause
Rx
Caution
Rapid correction → Cerebral oedema and seizure
Correct no faster than 0.5 mmol/L per hour
Treat underlying cause
e.g.
DI
Decrease NaCl intake
Replace urinary water loss
Desmopressin
Thiazide if nephrogenic
Rehydrate
Hypovolaemic
NS initially
Hypotonic fluid when rehydrated
Euvolaemic
Oral fluid (water) if possible
Otherwise 5% dextrose
Hypervolaemic
Diuretic + 5% dextrose
Dialysis if renal failure

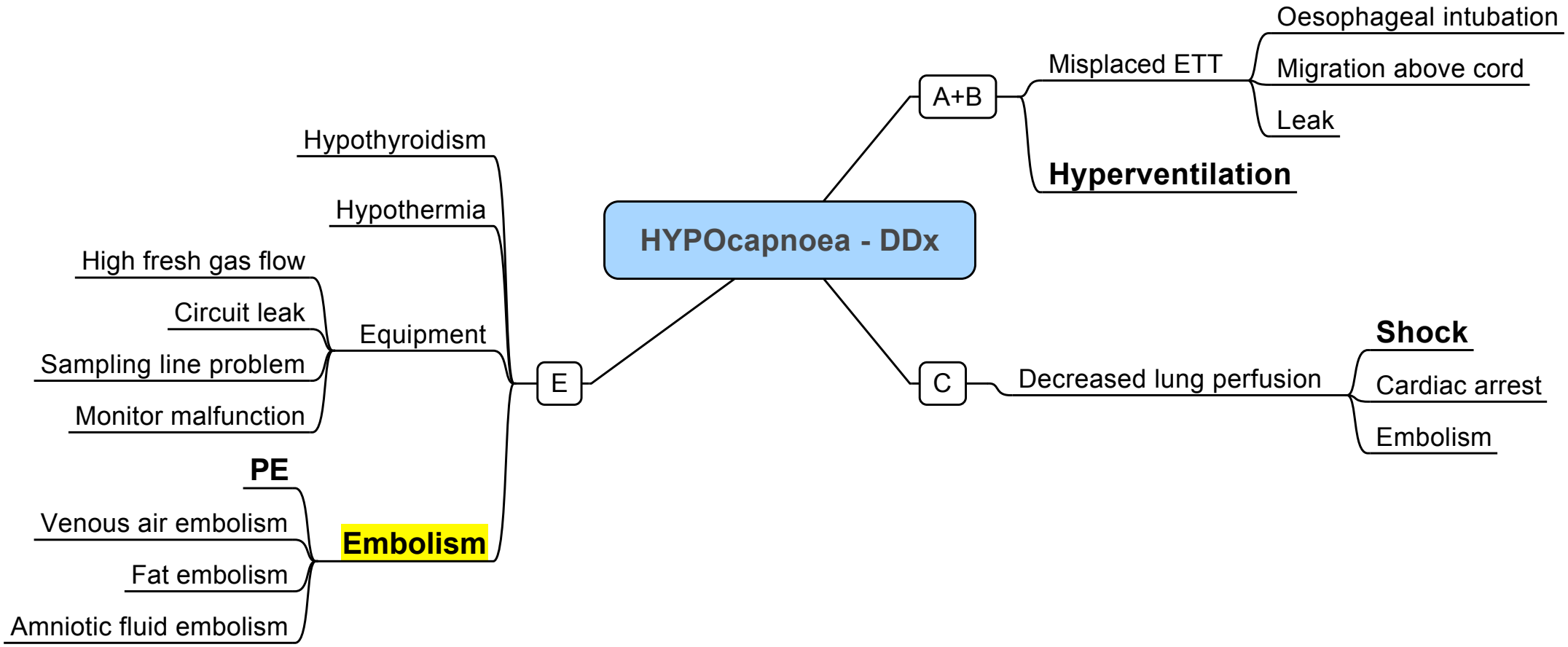

Hypertension - DDx
A+B
Hypoxaemia
Hypercarbia
C
Pre-existing HTN
Hypervolaemia
Arrhythmia
MI / CCF
Aortic cross-clamp
D
CNS
Sympathetic stimulation
Awareness
Bladder distension
Pain
Inadequate analgesia
Limb tourniquet
Laryngoscopy
Autonomic dysreflexia
Anxiety
Others
Increased ICP
CVA
Carotid baroreceptor denervation
Endocrine
Pre-eclampsia
Primary hyperaldosteronism
Cushing's syndrome
Phaeochromocytoma
Thyroid storm
Serotonin syndrome
Neuroleptic malignant syndrome
Drugs
Drug effects
Vasopressors
MAOI interaction
Stimulant drugs
Cocaine
Amphetamine
Indigo carmine
Oral contraceptive
Drug withdrawal
EtOH
Opioids
Benzodiazepines
Antihypertensives
Drug error
IV injection of adrenaline-containing LA
Vasopressors given in error

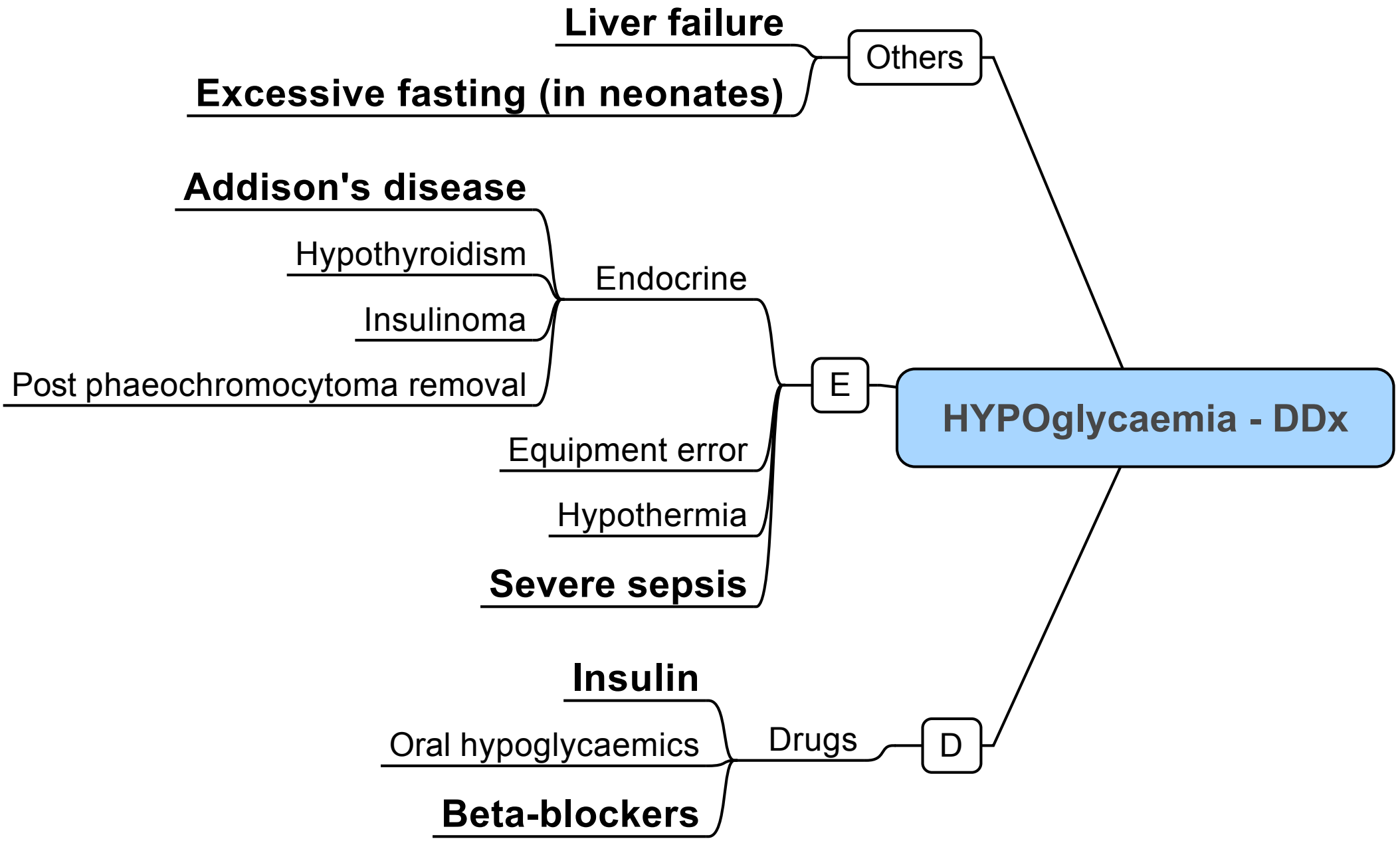

Hypertension (HTN) - AMx
Issues
Labile BP in periop period
Right shift of CBF autoregulation curve
Associated conditions
Secondary causes (if present)
Complications of HTN itself
Effects of Rx
Consultation
History
Time course
New vs pre-existing
Secondary causes
Esp treatable causes
Including
Renal causes
Renal artery stenosis
Renal parenchymal disease
Endocrine
Phaeochromocytoma
Thyrotoxicosis
Hyperaldosteronism
Others
Coarctation of aorta
White coat HTN
Complications
IHD
Heart failure
CVA
Renal impairment
Atherosclerosis
Peripheral vascular disease
Hypertensive retinopathy
Related conditions
DM
Obstructive sleep apnoea
Excessive alchol consumption
Rx and effects of Rx
ACE inhibitors
Risk of refractory hypotension on induction
Ix
ECG
U&E
Consider exercise tolerance test
... if significant CVS disease suspected
Consider delay for optimisation
Stage 3
SBP ≥ 180
DBP ≥ 110
End organ damage
Treatable secondary causes
Plan
Others
Higher BP may be required for adequate organ perfusion
MADE
M
If severe, consider IBP
Consider
D
Periop beta blockade
Intraop alpha2 agonists
Postop
Consider ICU/HDU
Major surgery
Unstable BP intraop
Goals
Maintain MAP within 20% of baseline

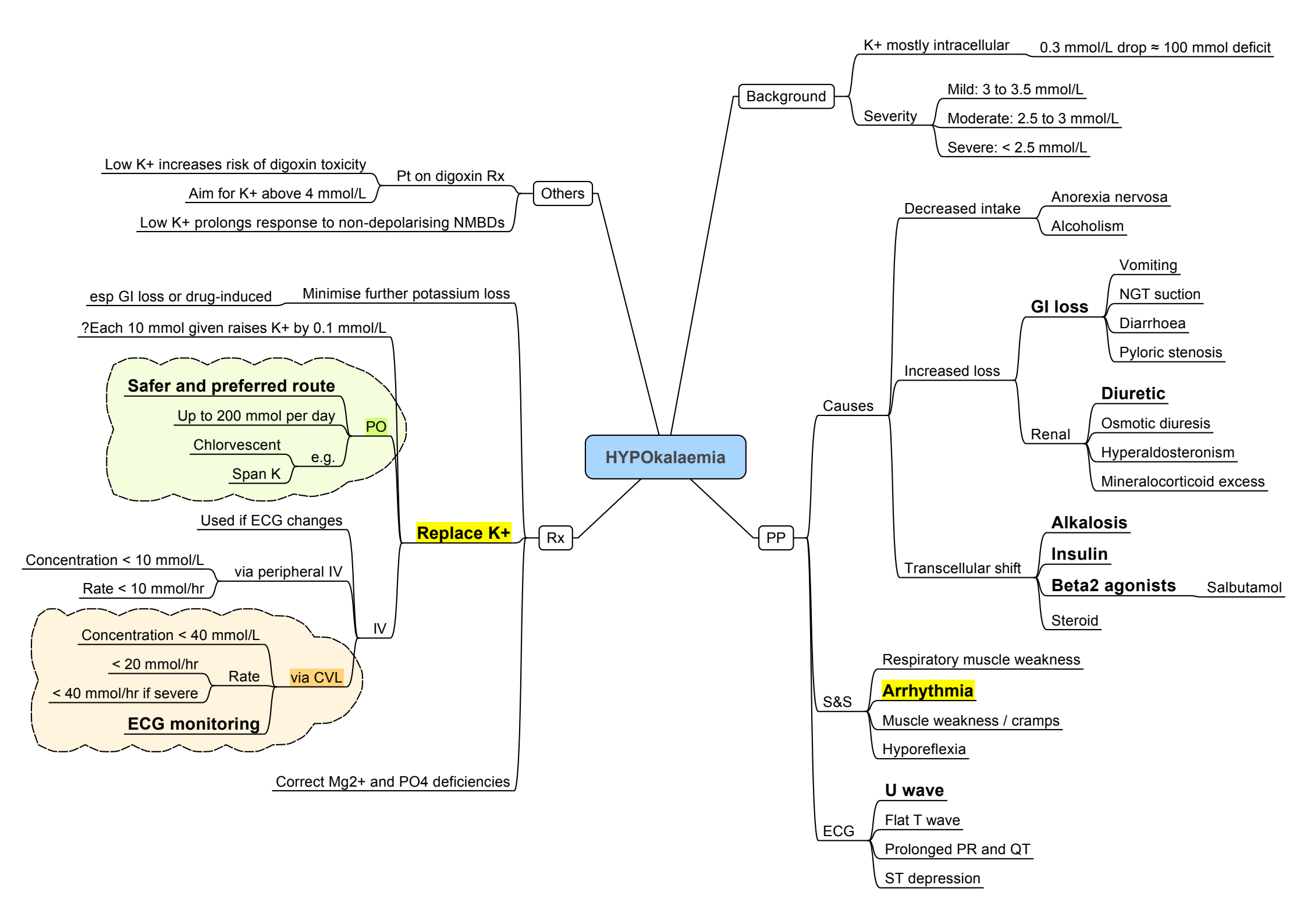

HYPERthermia - DDx
E
Endocrine
Thyroid storm
Serotonin syndrome
Equipment
Excessive warming
Sepsis
D
Drug-triggered
Transfusion reaction
Malignant hyperthermia
Neuroleptic malignant syndrome
MINTS
Also for hypercarbia
Malignant hyperthermia
Iatrogenic
Neuroleptic malignant syndrome
Thyroid storm
Sepsis / Serotonin syndrome

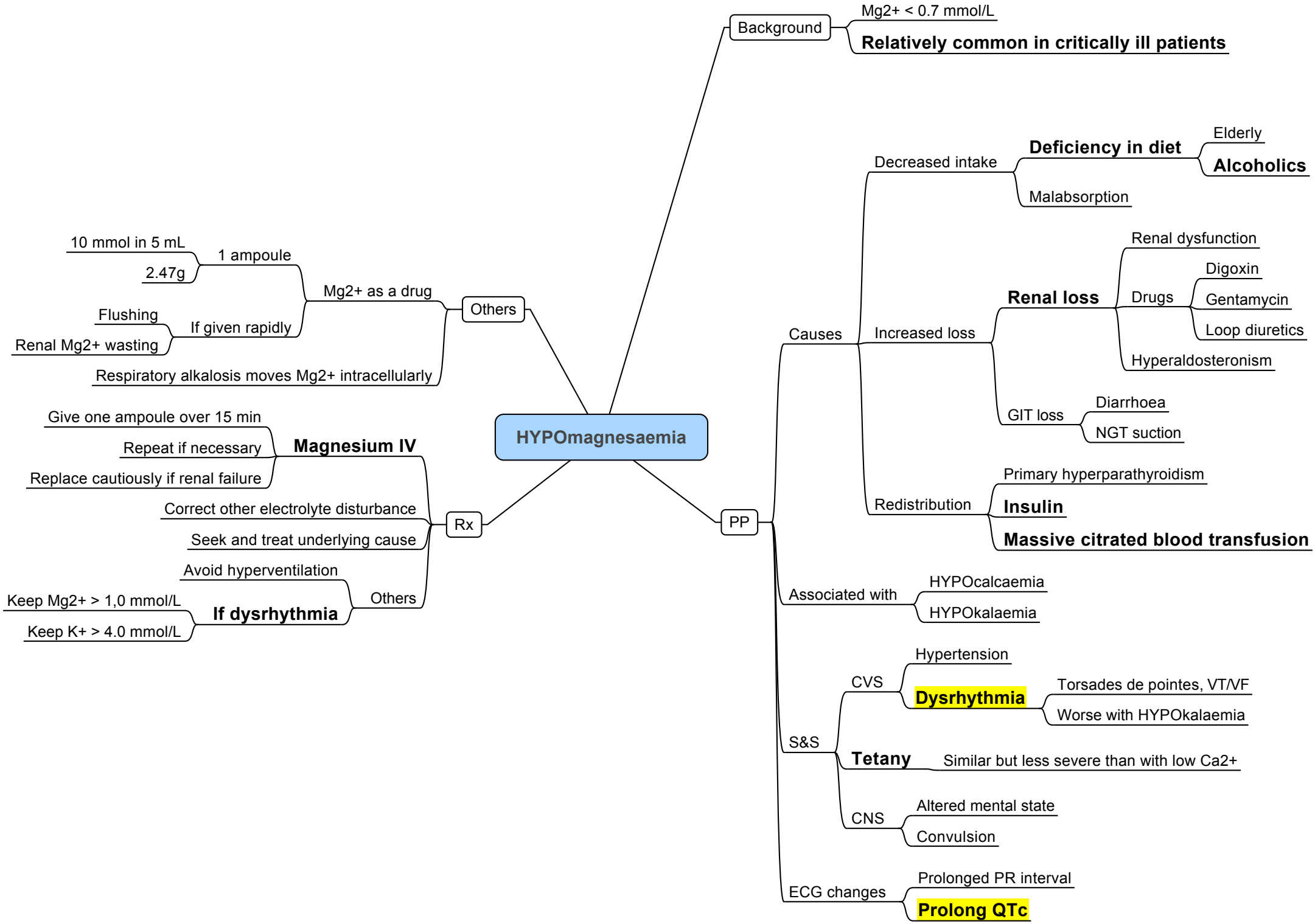

Hyperthyroidism - AMx
Related
[Thyrotoxic crisis]
[Thyroidectomy - AMx]
Goitre
Size and position
Cricothyrodotomy possible?
Feel lower the gland
Check
Airway compression
Positional dyspnoea
Dysphagia
Stridor
Retrosternal spread
Tracheal deviation
SVC obstruction
Venous distension in neck, upper chest, arms
Oedema in face or arms
Pemberton's sign
Consider
CXR
Tracheal deviation / narrowing
CT
Retrostenal spread
Airway compression / distortion
Intraluminal spread
Others
If autoimmune cause
Increased risk of myopathy and myasthenia gravis
Plan
MADE
M
Temp
Consider IBP
D
Consider beta blocker periop
... if emergency and clinical hyperthyroid
E
Consider
Smaller ETT size
Reinforced ETT
Eye protection, esp if exophthalmos
Goals
Airway management as appropriate
Prepare for thyroid storm esp if hyperthyroid preop
e.g. Emergency surgery
Issues
Mass effect of goitre
Airway compression
SVC obstruction
Thyroid storm risk
Risk greatly reduced if euthyroid preop
Usually presents 6 to 24 hours postop
Consultation
History
Underlying cause
e.g.
Autoimmune
Malignancy
Iatrogenic (e.g. Amiodarone)
Consider other effects of the underlying cause
Confirm euthyroid state
HR < 80
No tremour
Hyperthyroid S&S
e.g.
Weight loss
Anxiety
Heat intolerance
Tremour, chorea
Proximal muscle weakness
Tachycardia
Other PMHx
CVS diseases
Autoimmune disorders
Rx
Propranolol
Steroids
Propylthiouracil
SE
Hepatic dysfunction
Agranulocytosis
Exam
Airway assessment
Ix
ECG
Arrhythmia
esp AF
Bloods
TFT
TSH
Free T4
FBC
LFT
U&E
Hyperglycaemia
Hypercalcaemia
Optimisation
Delay surgery till euthyroid
Consider referral to endocrinologist

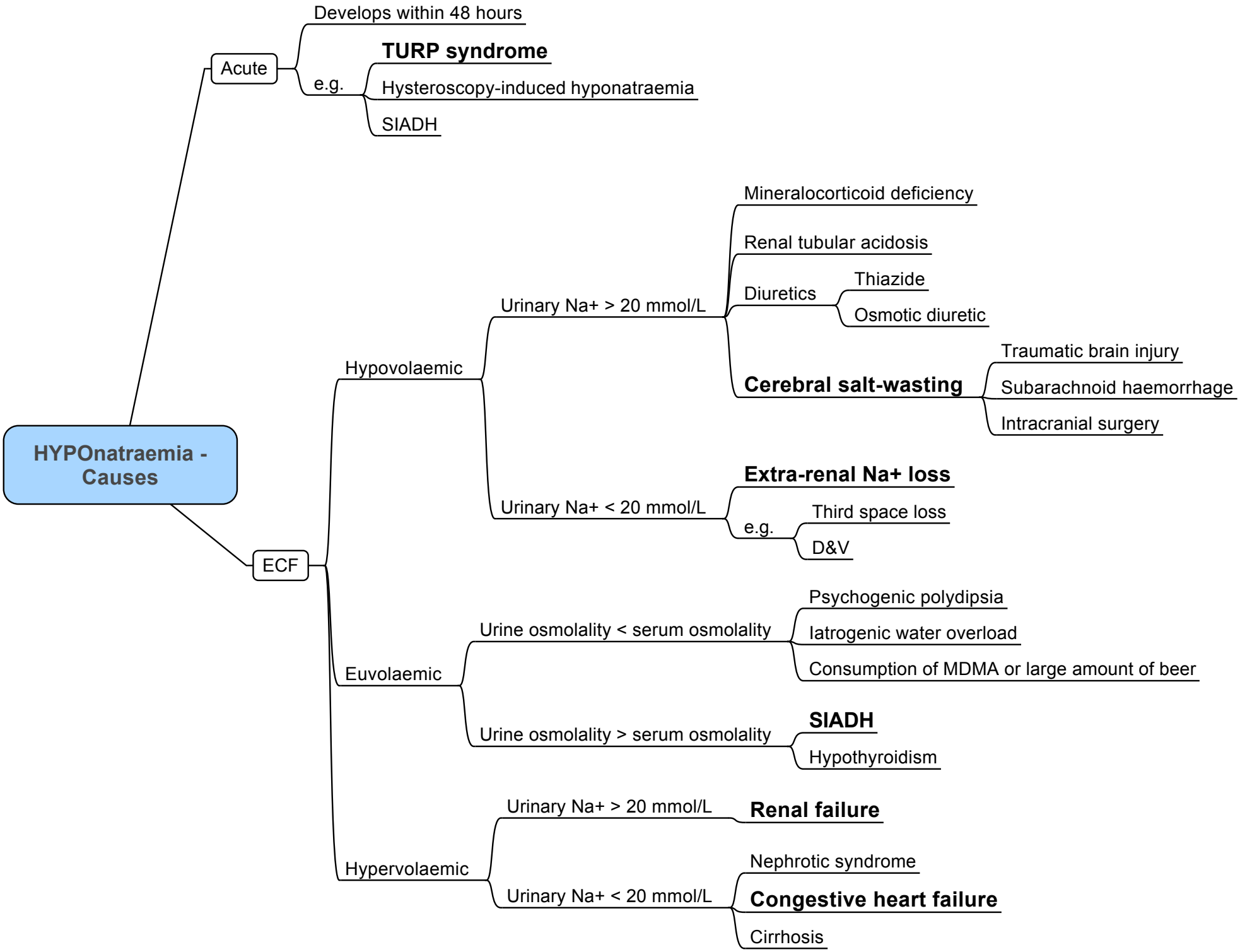

HYPOcalcaemia
Levels
Total Ca2+ < 2.2 mmol/L
Ionised Ca2+ < 0.9 mmol/L
CATS go numb
CATS
Confusion
Arrhythmia
Tetany
Seizure
Numbness / Paresthesia
Rx
Treat underlying cause
Ca2+
Ca2+ chloride
Give slowly via CVL
3 times Ca2+ as gluconate
Ca+ gluconate
Preferred if peripheral IV
AVOID extravasation
Correct low Mg2+ if present
Correct alkalosis if present
PP
Causes
Post parathyroidectomy
Hypoparathyroidism
Hypoalbuminaemia (Reduced total Ca2+)
Hyperventilation / Respiratory alkalosis
Other
High phosphate level
Citrate administration (e.g. transfusion)
S&S
Laryngospasm
CVS
Arrhythmia
Heart failure
CNS
Seizure
Confusion
Hyperreflexia
Numbness / parasthesia
Tetany
Trousseau's sign (caropedal spasm)
Chvostek's sign (facial twitching)
ECG
Prolonged QT
T wave flattening / inversion
VF/VT

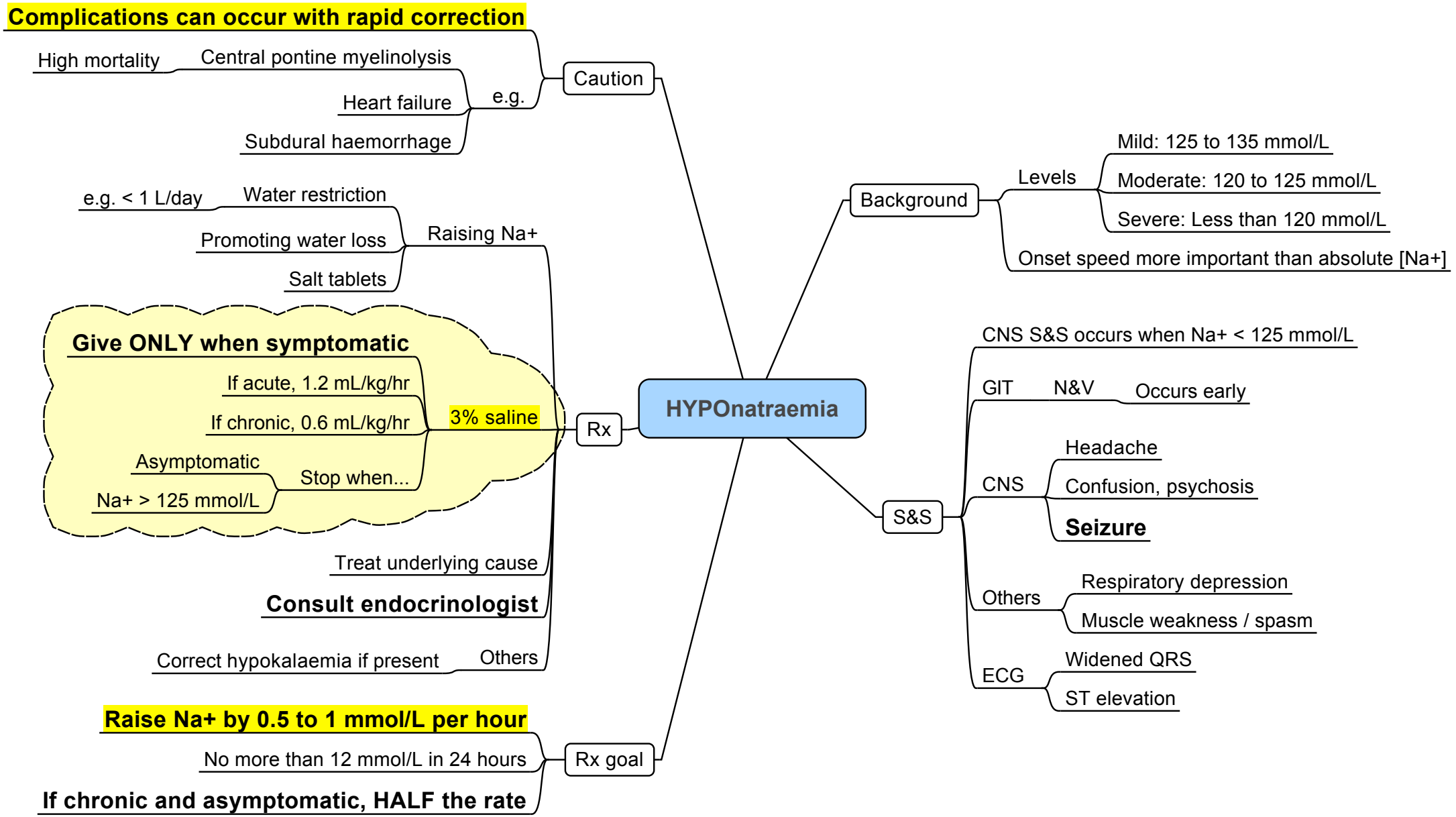

HYPOcapnoea - DDx
A+B
Misplaced ETT
Oesophageal intubation
Migration above cord
Leak
Hyperventilation
C
Decreased lung perfusion
Shock
Cardiac arrest
Embolism
E
Hypothyroidism
Hypothermia
Equipment
High fresh gas flow
Circuit leak
Sampling line problem
Monitor malfunction
Embolism
PE
Venous air embolism
Fat embolism
Amniotic fluid embolism

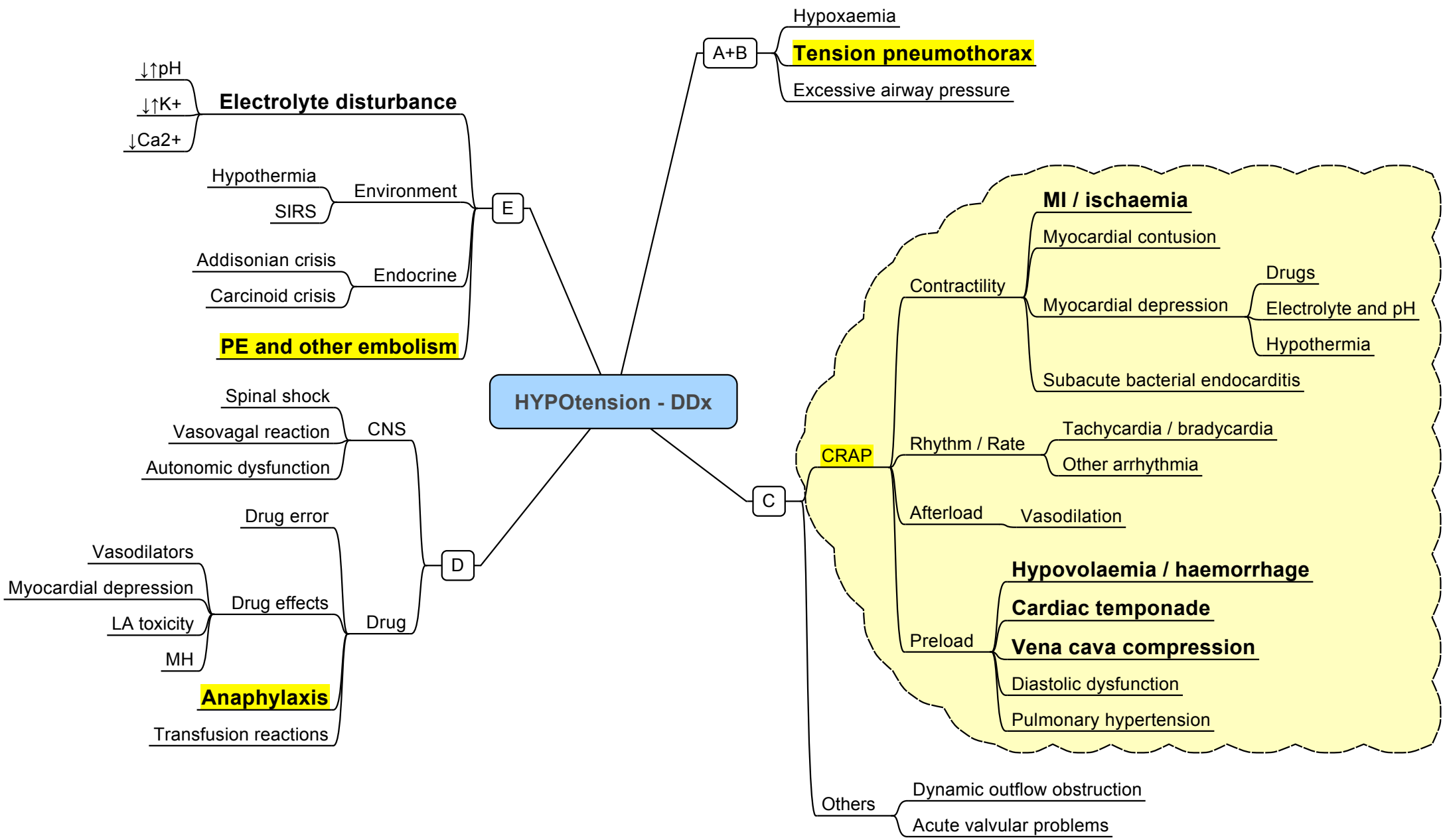

HYPOglycaemia - DDx
E
Endocrine
Addison's disease
Hypothyroidism
Insulinoma
Post phaeochromocytoma removal
Equipment error
Hypothermia
Severe sepsis
Others
Liver failure
Excessive fasting (in neonates)
D
Drugs
Insulin
Oral hypoglycaemics
Beta-blockers

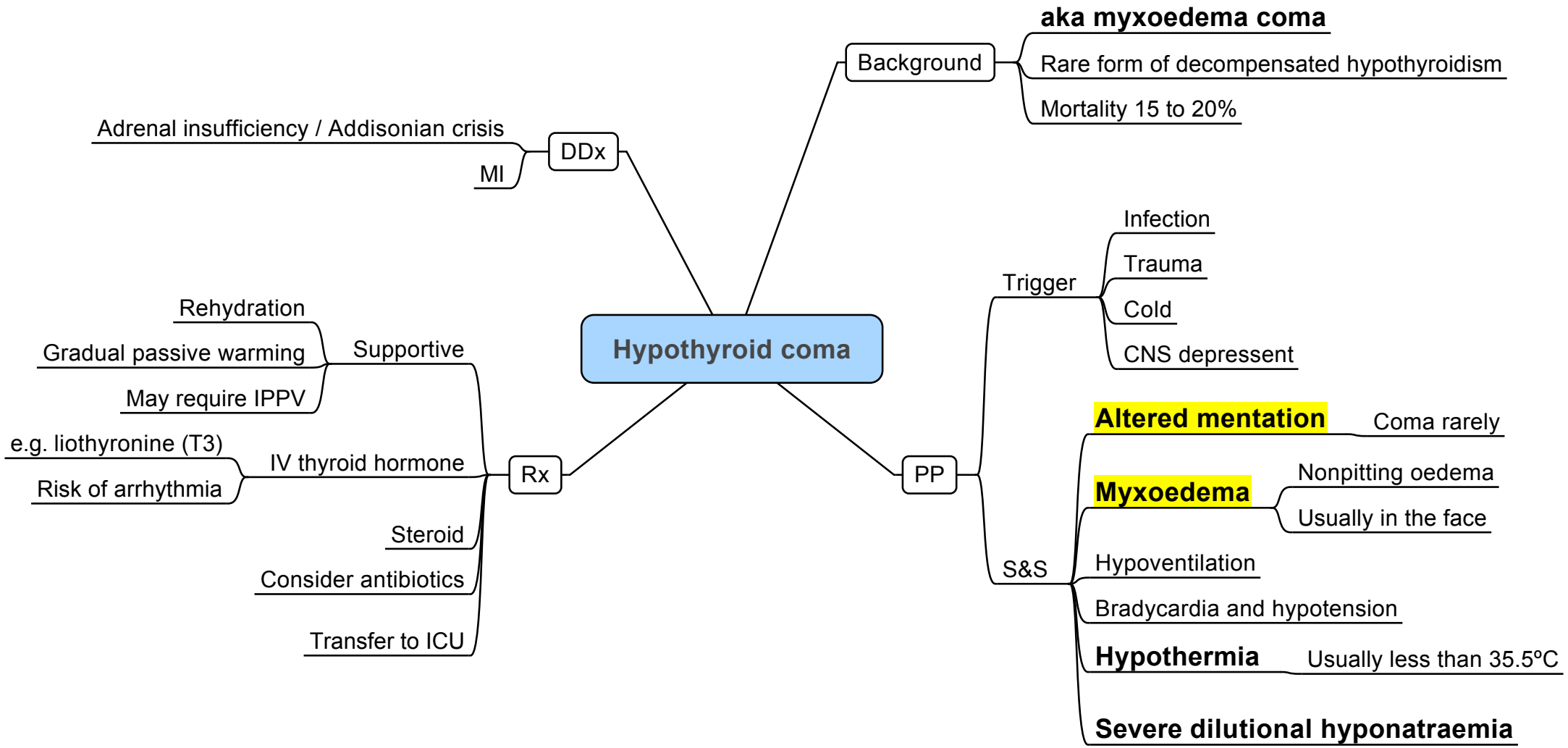

HYPOkalaemia
Background
K+ mostly intracellular
0.3 mmol/L drop ≈ 100 mmol deficit
Severity
Mild: 3 to 3.5 mmol/L
Moderate: 2.5 to 3 mmol/L
Severe: < 2.5 mmol/L
PP
Causes
Decreased intake
Anorexia nervosa
Alcoholism
Increased loss
GI loss
Vomiting
NGT suction
Diarrhoea
Pyloric stenosis
Renal
Diuretic
Osmotic diuresis
Hyperaldosteronism
Mineralocorticoid excess
Transcellular shift
Alkalosis
Insulin
Beta2 agonists
Salbutamol
Steroid
S&S
Respiratory muscle weakness
Arrhythmia
Muscle weakness / cramps
Hyporeflexia
ECG
U wave
Flat T wave
Prolonged PR and QT
ST depression
Rx
Others
Pt on digoxin Rx
Low K+ increases risk of digoxin toxicity
Aim for K+ above 4 mmol/L
Low K+ prolongs response to non-depolarising NMBDs
Minimise further potassium loss
esp GI loss or drug-induced
?Each 10 mmol given raises K+ by 0.1 mmol/L
Replace K+
Safer and preferred route
PO
Up to 200 mmol per day
e.g.
Chlorvescent
Span K
IV
Used if ECG changes
via peripheral IV
Concentration < 10 mmol/L
Rate < 10 mmol/hr
via CVL
Concentration < 40 mmol/L
Rate
< 20 mmol/hr
< 40 mmol/hr if severe
ECG monitoring
Correct Mg2+ and PO4 deficiencies

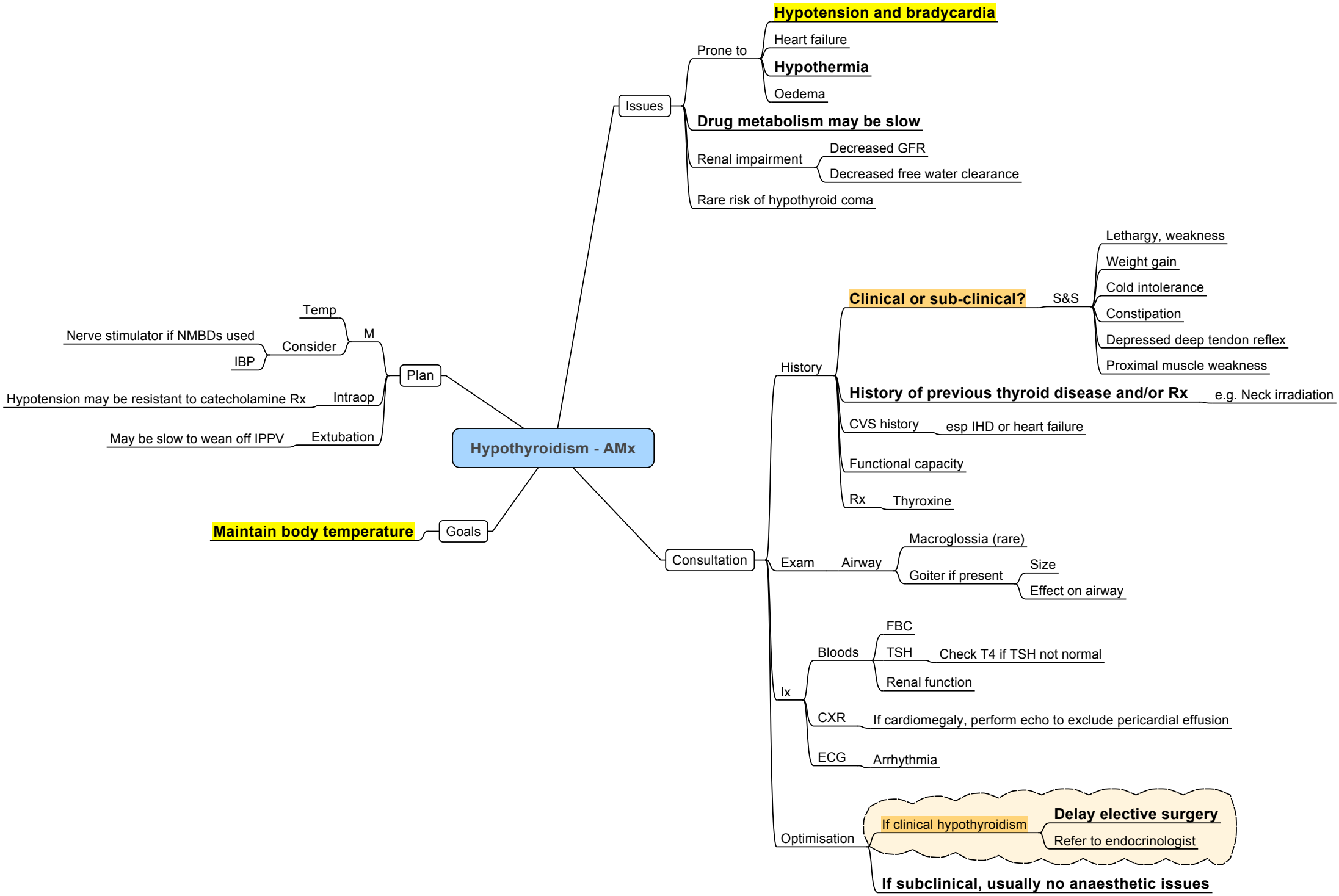

HYPOmagnesaemia
Background
Mg2+ < 0.7 mmol/L
Relatively common in critically ill patients
PP
Causes
Decreased intake
Deficiency in diet
Elderly
Alcoholics
Malabsorption
Increased loss
Renal loss
Renal dysfunction
Drugs
Digoxin
Gentamycin
Loop diuretics
Hyperaldosteronism
GIT loss
Diarrhoea
NGT suction
Redistribution
Primary hyperparathyroidism
Insulin
Massive citrated blood transfusion
Associated with
HYPOcalcaemia
HYPOkalaemia
S&S
CVS
Hypertension
Dysrhythmia
Torsades de pointes, VT/VF
Worse with HYPOkalaemia
Tetany
Similar but less severe than with low Ca2+
CNS
Altered mental state
Convulsion
ECG changes
Prolonged PR interval
Prolong QTc
Rx
Others
Mg2+ as a drug
1 ampoule
10 mmol in 5 mL
2.47g
If given rapidly
Flushing
Renal Mg2+ wasting
Respiratory alkalosis moves Mg2+ intracellularly
Magnesium IV
Give one ampoule over 15 min
Repeat if necessary
Replace cautiously if renal failure
Correct other electrolyte disturbance
Seek and treat underlying cause
Others
If dysrhythmia
Avoid hyperventilation
Keep Mg2+ > 1,0 mmol/L
Keep K+ > 4.0 mmol/L

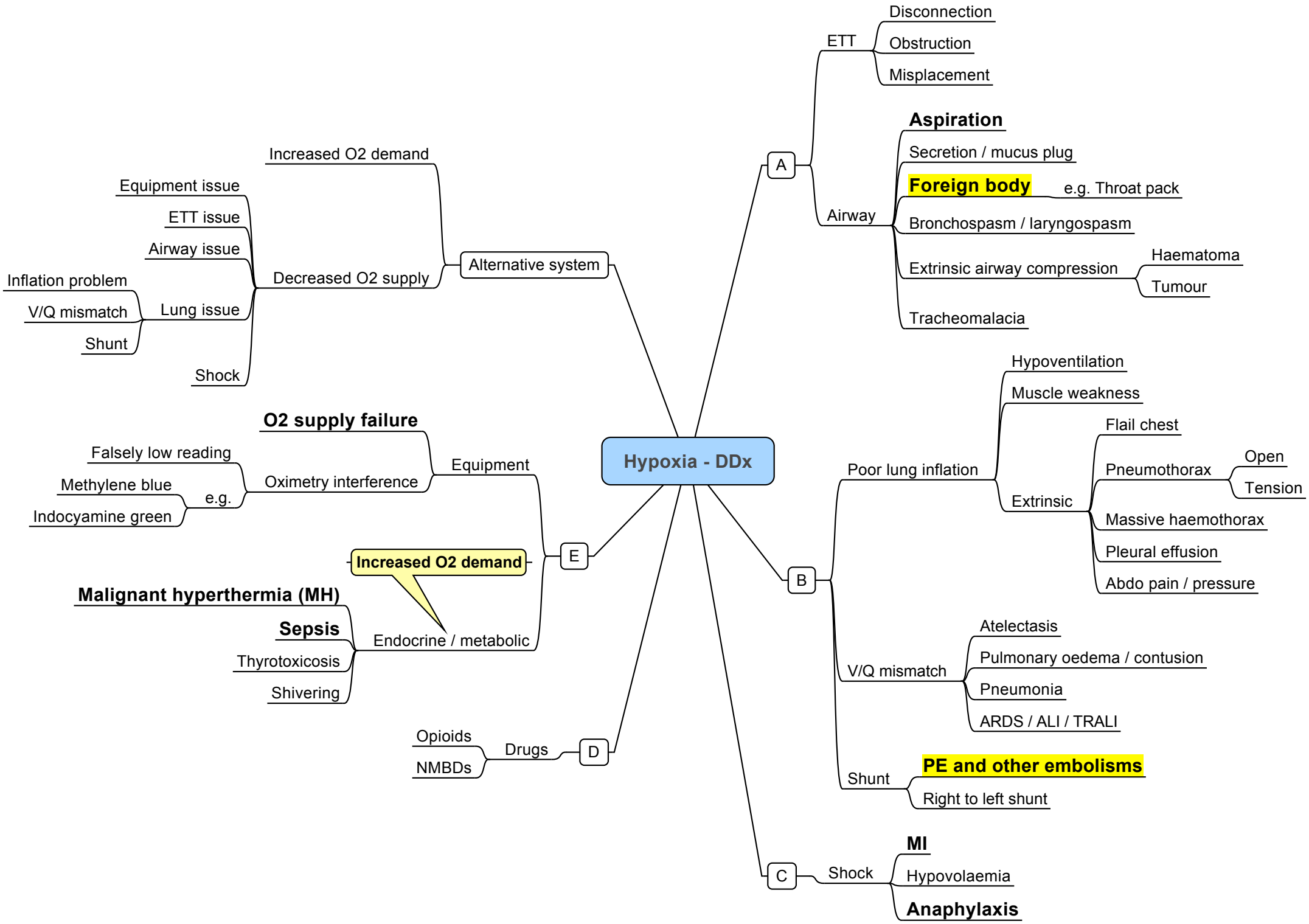

HYPOnatraemia - Causes
Acute
Develops within 48 hours
e.g.
TURP syndrome
Hysteroscopy-induced hyponatraemia
SIADH
ECF
Hypovolaemic
Urinary Na+ > 20 mmol/L
Mineralocorticoid deficiency
Renal tubular acidosis
Diuretics
Thiazide
Osmotic diuretic
Cerebral salt-wasting
Traumatic brain injury
Subarachnoid haemorrhage
Intracranial surgery
Urinary Na+ < 20 mmol/L
Extra-renal Na+ loss
e.g.
Third space loss
D&V
Euvolaemic
Urine osmolality < serum osmolality
Psychogenic polydipsia
Iatrogenic water overload
Consumption of MDMA or large amount of beer
Urine osmolality > serum osmolality
SIADH
Hypothyroidism
Hypervolaemic
Urinary Na+ > 20 mmol/L
Renal failure
Urinary Na+ < 20 mmol/L
Nephrotic syndrome
Congestive heart failure
Cirrhosis


HYPOnatraemia
Background
Levels
Mild: 125 to 135 mmol/L
Moderate: 120 to 125 mmol/L
Severe: Less than 120 mmol/L
Onset speed more important than absolute [Na+]
S&S
CNS S&S occurs when Na+ < 125 mmol/L
GIT
N&V
Occurs early
CNS
Headache
Confusion, psychosis
Seizure
Others
Respiratory depression
Muscle weakness / spasm
ECG
Widened QRS
ST elevation
Rx
Caution
Complications can occur with rapid correction
e.g.
High mortality
Central pontine myelinolysis
Heart failure
Subdural haemorrhage
Raising Na+
e.g. < 1 L/day
Water restriction
Promoting water loss
Salt tablets
3% saline
Give ONLY when symptomatic
If acute, 1.2 mL/kg/hr
If chronic, 0.6 mL/kg/hr
Stop when...
Asymptomatic
Na+ > 125 mmol/L
Others
Treat underlying cause
Consult endocrinologist
Correct hypokalaemia if present
Rx goal
Raise Na+ by 0.5 to 1 mmol/L per hour
No more than 12 mmol/L in 24 hours
If chronic and asymptomatic, HALF the rate

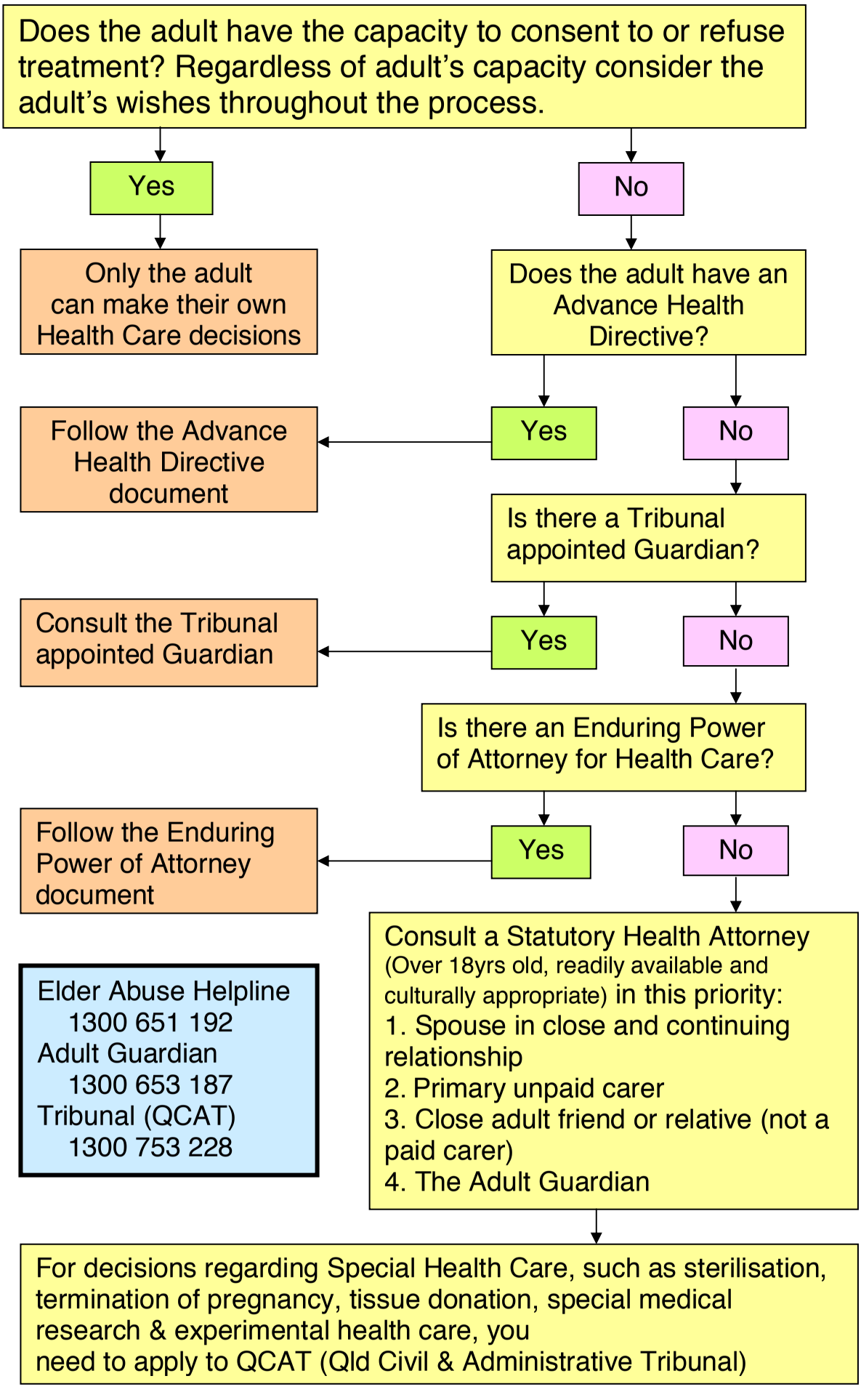

HYPOtension - DDx
A+B
Hypoxaemia
Tension pneumothorax
Excessive airway pressure
E
↓↑pH
↓↑K+
↓Ca2+
Electrolyte disturbance
Hypothermia
SIRS
Environment
Addisonian crisis
Carcinoid crisis
Endocrine
PE and other embolism
C
CRAP
Contractility
MI / ischaemia
Myocardial contusion
Myocardial depression
Drugs
Electrolyte and pH
Hypothermia
Subacute bacterial endocarditis
Rhythm / Rate
Tachycardia / bradycardia
Other arrhythmia
Afterload
Vasodilation
Preload
Hypovolaemia / haemorrhage
Cardiac temponade
Vena cava compression
Diastolic dysfunction
Pulmonary hypertension
Others
Dynamic outflow obstruction
Acute valvular problems
D
CNS
Spinal shock
Vasovagal reaction
Autonomic dysfunction
Drug
Drug error
Drug effects
Vasodilators
Myocardial depression
LA toxicity
MH
Anaphylaxis
Transfusion reactions


Hypothyroid coma
Background
aka myxoedema coma
Rare form of decompensated hypothyroidism
Mortality 15 to 20%
DDx
Adrenal insufficiency / Addisonian crisis
MI
PP
Trigger
Infection
Trauma
Cold
CNS depressent
S&S
Altered mentation
Coma rarely
Myxoedema
Nonpitting oedema
Usually in the face
Hypoventilation
Bradycardia and hypotension
Hypothermia
Usually less than 35.5ºC
Severe dilutional hyponatraemia
Rx
Supportive
Rehydration
Gradual passive warming
May require IPPV
IV thyroid hormone
e.g. liothyronine (T3)
Risk of arrhythmia
Steroid
Consider antibiotics
Transfer to ICU


Hypothyroidism - AMx
Issues
Prone to
Hypotension and bradycardia
Heart failure
Hypothermia
Oedema
Drug metabolism may be slow
Renal impairment
Decreased GFR
Decreased free water clearance
Rare risk of hypothyroid coma
Consultation
History
Clinical or sub-clinical?
S&S
Lethargy, weakness
Weight gain
Cold intolerance
Constipation
Depressed deep tendon reflex
Proximal muscle weakness
History of previous thyroid disease and/or Rx
e.g. Neck irradiation
CVS history
esp IHD or heart failure
Functional capacity
Rx
Thyroxine
Exam
Airway
Macroglossia (rare)
Goiter if present
Size
Effect on airway
Ix
Bloods
FBC
TSH
Check T4 if TSH not normal
Renal function
CXR
If cardiomegaly, perform echo to exclude pericardial effusion
ECG
Arrhythmia
Optimisation
If clinical hypothyroidism
Delay elective surgery
Refer to endocrinologist
If subclinical, usually no anaesthetic issues
Goals
Maintain body temperature
Plan
M
Temp
Consider
Nerve stimulator if NMBDs used
IBP
Intraop
Hypotension may be resistant to catecholamine Rx
Extubation
May be slow to wean off IPPV


Hypoxia - DDx
A
ETT
Disconnection
Obstruction
Misplacement
Airway
Aspiration
Secretion / mucus plug
Foreign body
e.g. Throat pack
Bronchospasm / laryngospasm
Extrinsic airway compression
Haematoma
Tumour
Tracheomalacia
B
Poor lung inflation
Hypoventilation
Muscle weakness
Extrinsic
Flail chest
Pneumothorax
Open
Tension
Massive haemothorax
Pleural effusion
Abdo pain / pressure
V/Q mismatch
Atelectasis
Pulmonary oedema / contusion
Pneumonia
ARDS / ALI / TRALI
Shunt
PE and other embolisms
Right to left shunt
C
Shock
MI
Hypovolaemia
Anaphylaxis
D
Drugs
Opioids
NMBDs
E
Equipment
O2 supply failure
Oximetry interference
e.g.
Falsely low reading
Methylene blue
Indocyamine green
Increased O2 demand
Endocrine / metabolic
Malignant hyperthermia (MH)
Sepsis
Thyrotoxicosis
Shivering
Alternative system
Increased O2 demand
Decreased O2 supply
Equipment issue
ETT issue
Airway issue
Lung issue
Inflation problem
V/Q mismatch
Shunt
Shock

WHO CAN MAKE HEALTH CARE DECISIONS in QLD?



This flowchart is a visual representation of The Guardianship and Administration Act 2000, Chapter 5 (Health matters and special health matters) Division 2 (Health care and special health care – consent) sections 65 and 66.

It is also recommended to refer to the following section of the act

Health care principle (when making a decision)

Copied from Guardianship and Administration Act 2000 (Schedule 1, Part 2 Section 12)

(1) The ***health care principle*** means power for a health matter, or special health matter, for an adult should be exercised by a guardian, the adult guardian, the tribunal, or for a matter relating to prescribed special health care, another entity—

(a) In the way least restrictive of the adult's rights; and

(b) Only if the exercise of power—

(i) Is necessary and appropriate to maintain or promote the adult's health or wellbeing; or

(ii) Is, in all the circumstances, in the adult's best interests.

Example of exercising power in the way least restrictive of the adult's rights— If there is a choice between a more or less intrusive way of meeting an identified need, the less intrusive way should be adopted.

(2) In deciding whether the exercise of a power is appropriate, the guardian, the adult guardian, tribunal or other entity must, to the greatest extent practicable—

(a) Seek the adult's views and wishes and take them into account; and

(b) Take the information given by the adult's health provider into account. (See section 76 of the G&A Act 2000 re Health providers to give information).

(3) The adult's views and wishes may be expressed—

(a) Orally; or

(b) In writing, for example, in an advance health directive; or

(c) In another way, including, for example, by conduct.

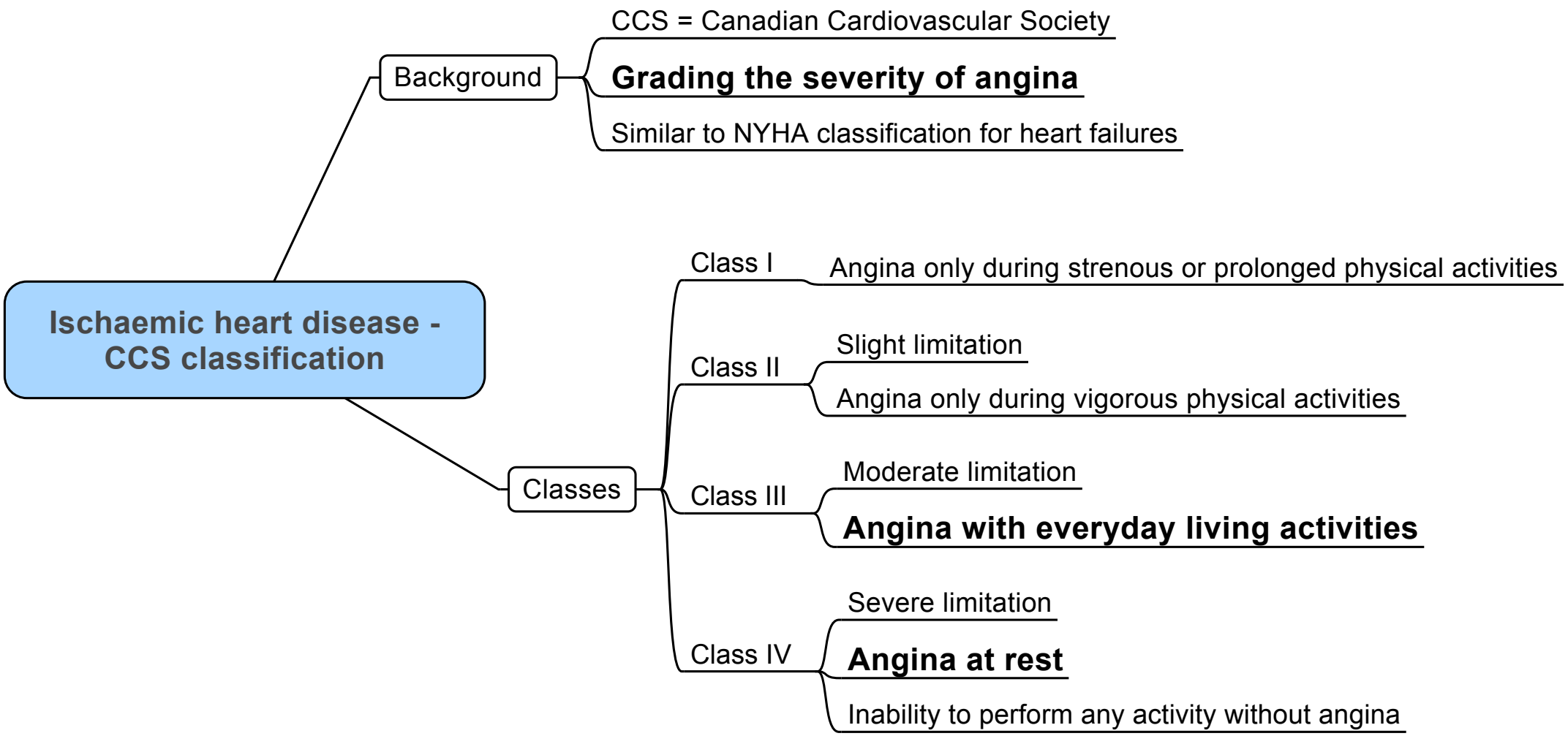
(4) The health care principle does not affect any right an adult has to refuse health care.

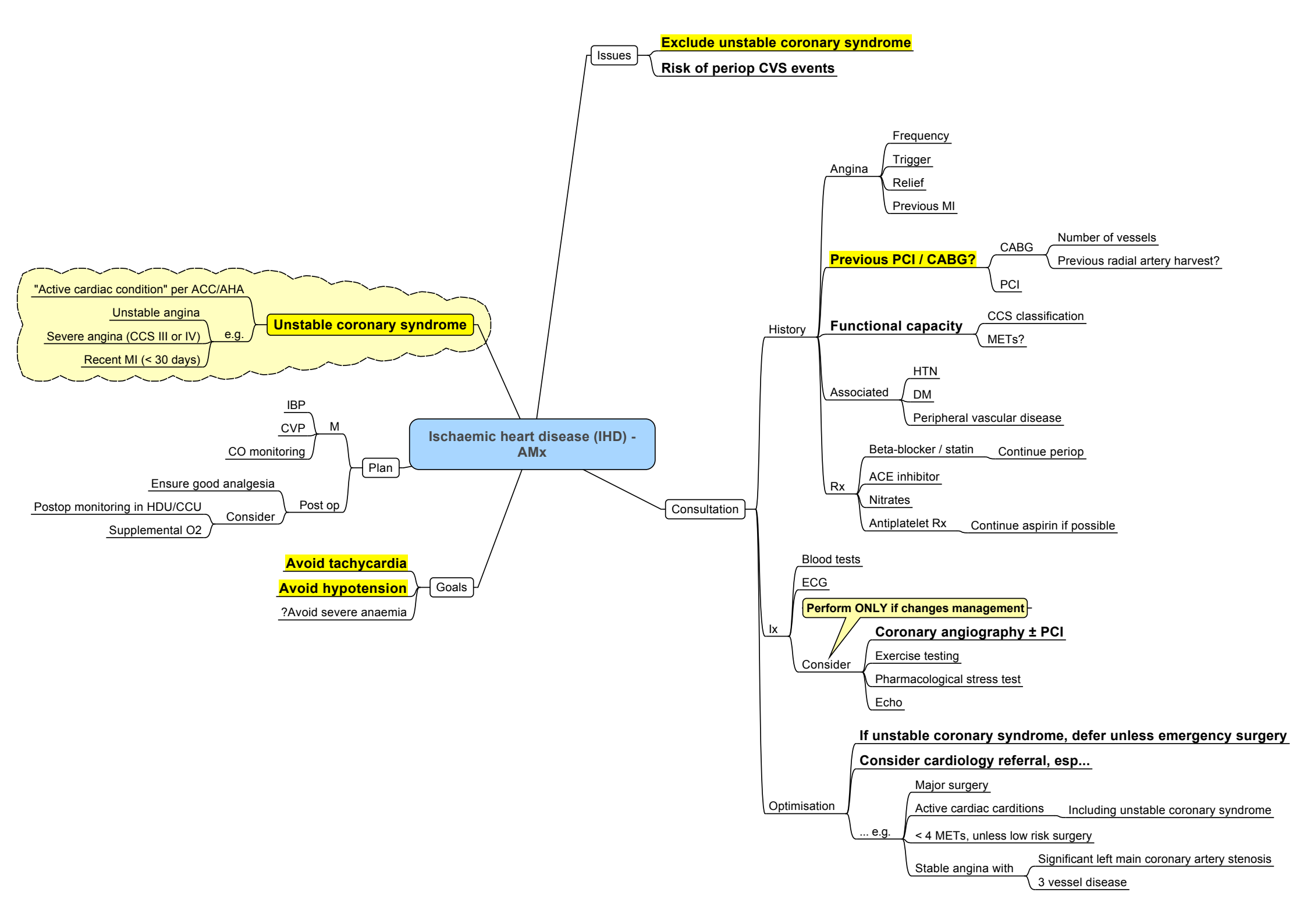
(5) In deciding whether to consent to special health care for an adult, the tribunal or other entity must, to the greatest extent practicable, seek the views of the following person and take them into account—

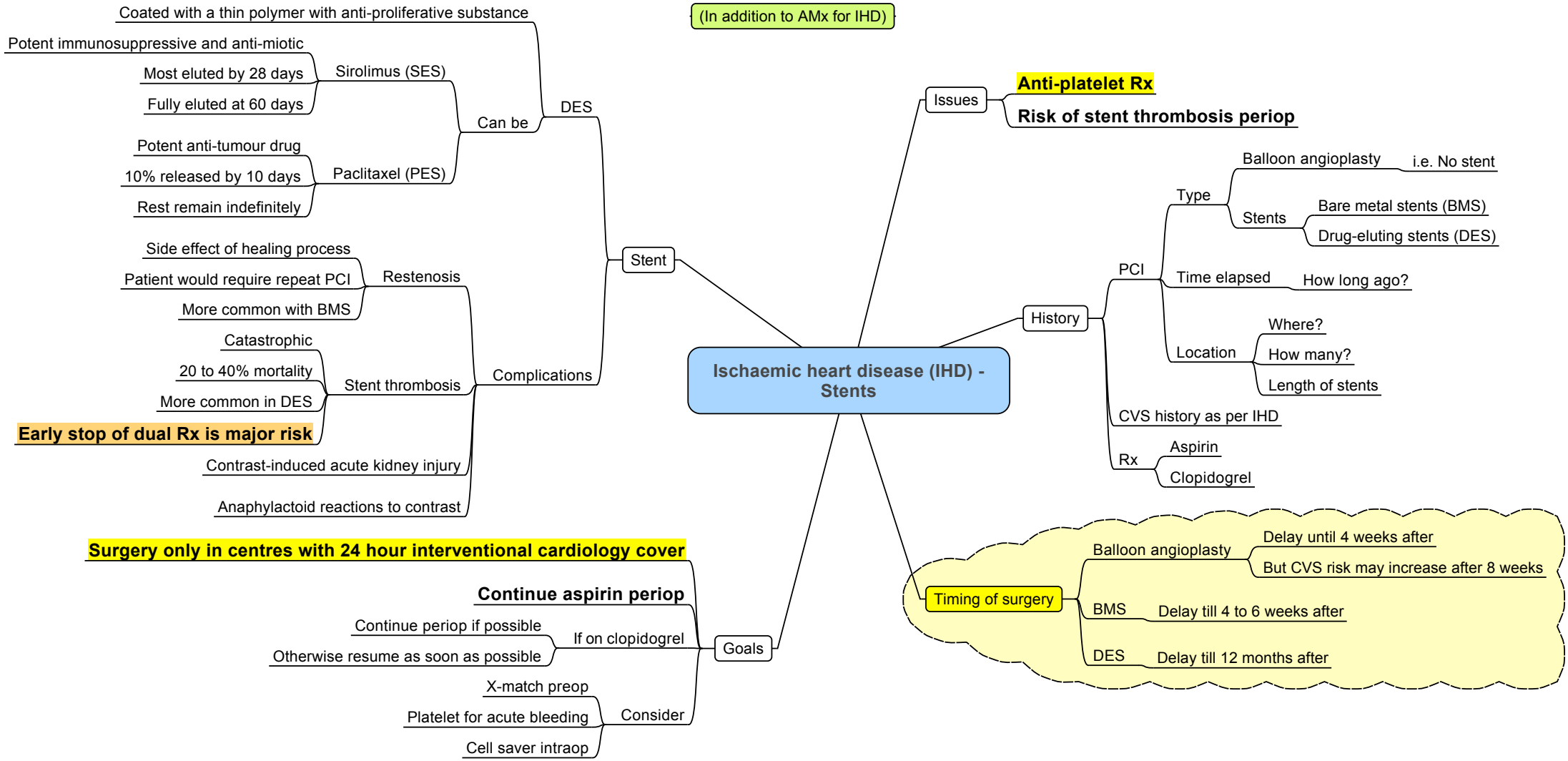
(a) A guardian appointed by the tribunal for the adult;

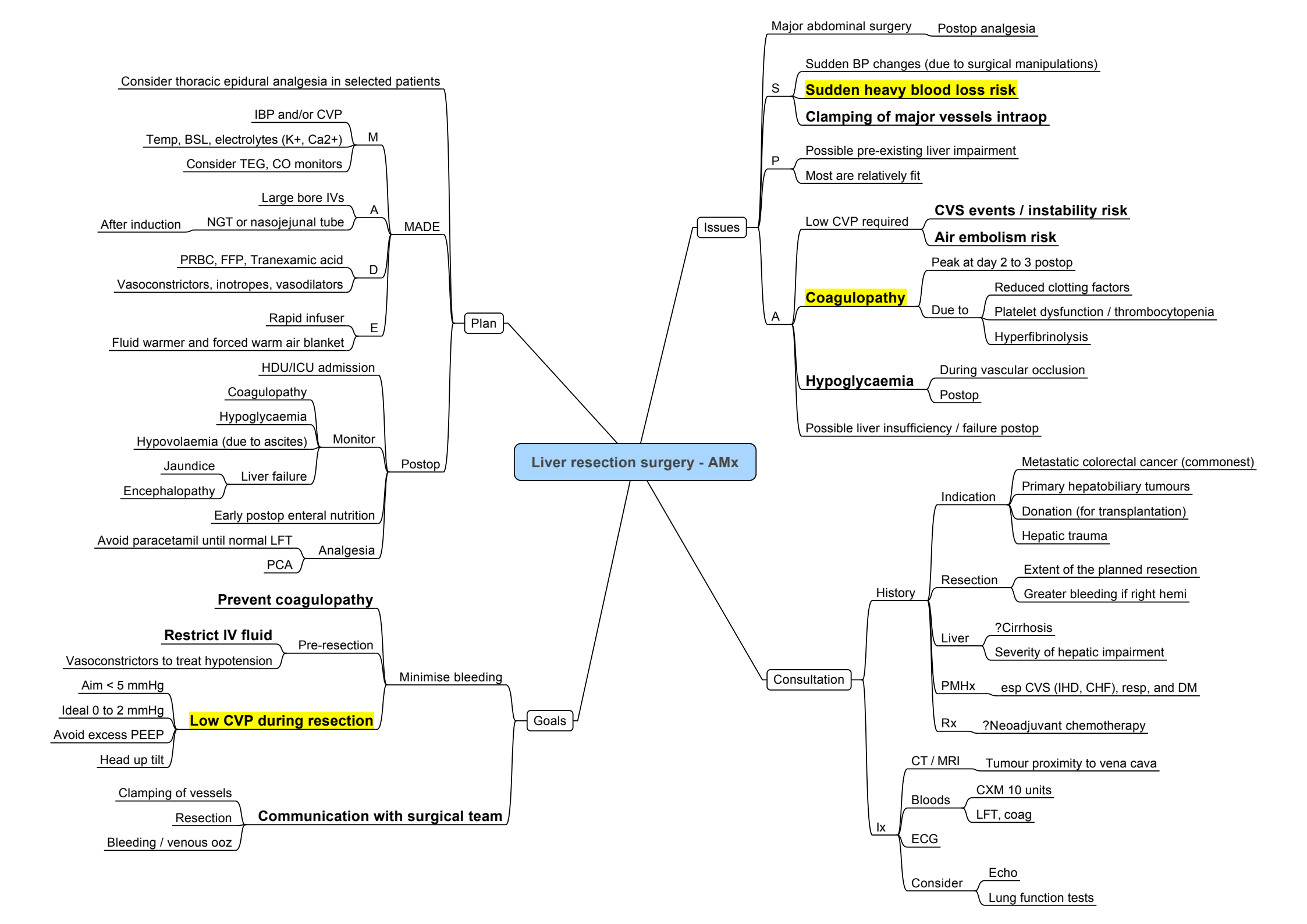
(b) If there is no guardian mentioned in paragraph (a), an attorney for a health matter appointed by the adult;

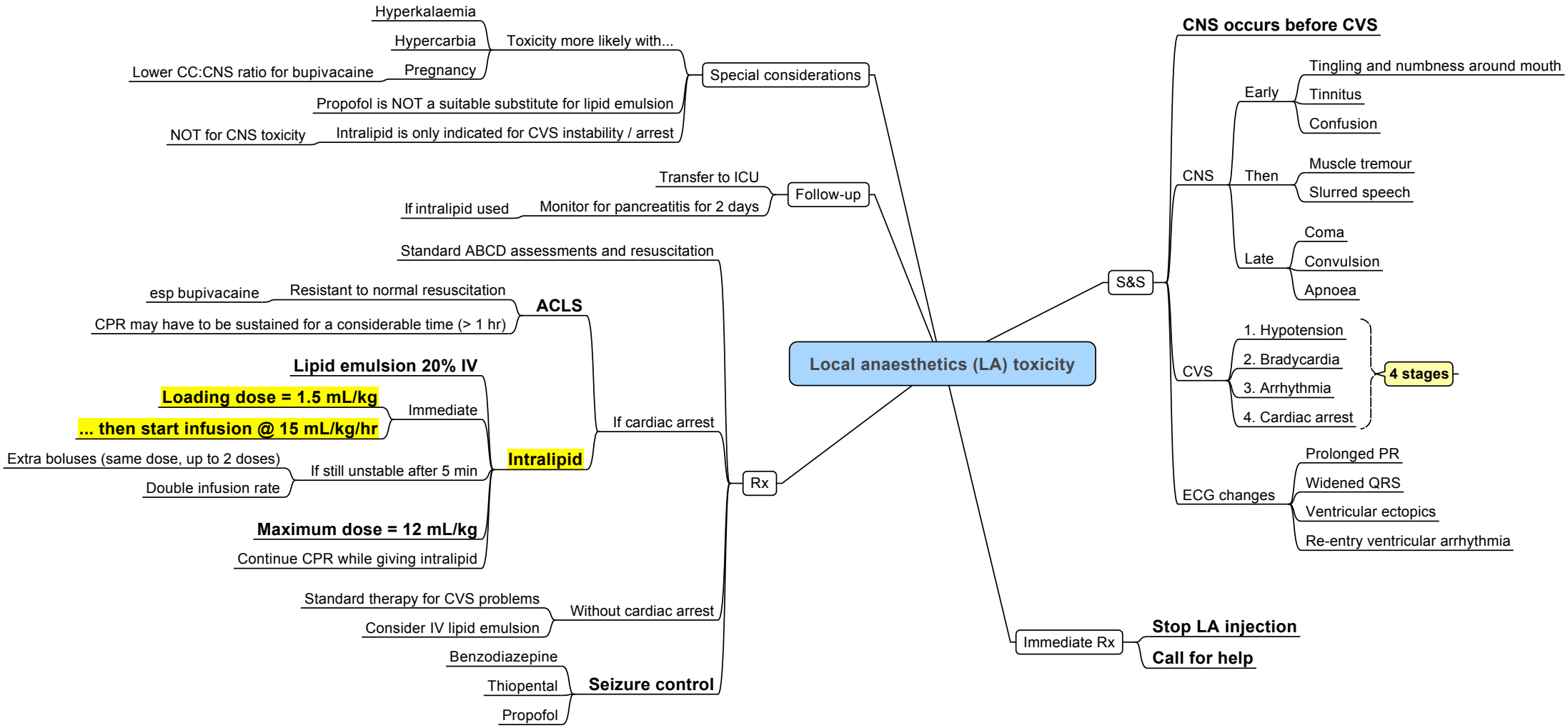
(c) If there is no guardian or attorney mentioned in paragraph (a) or (b), the statutory health attorney for the adult.

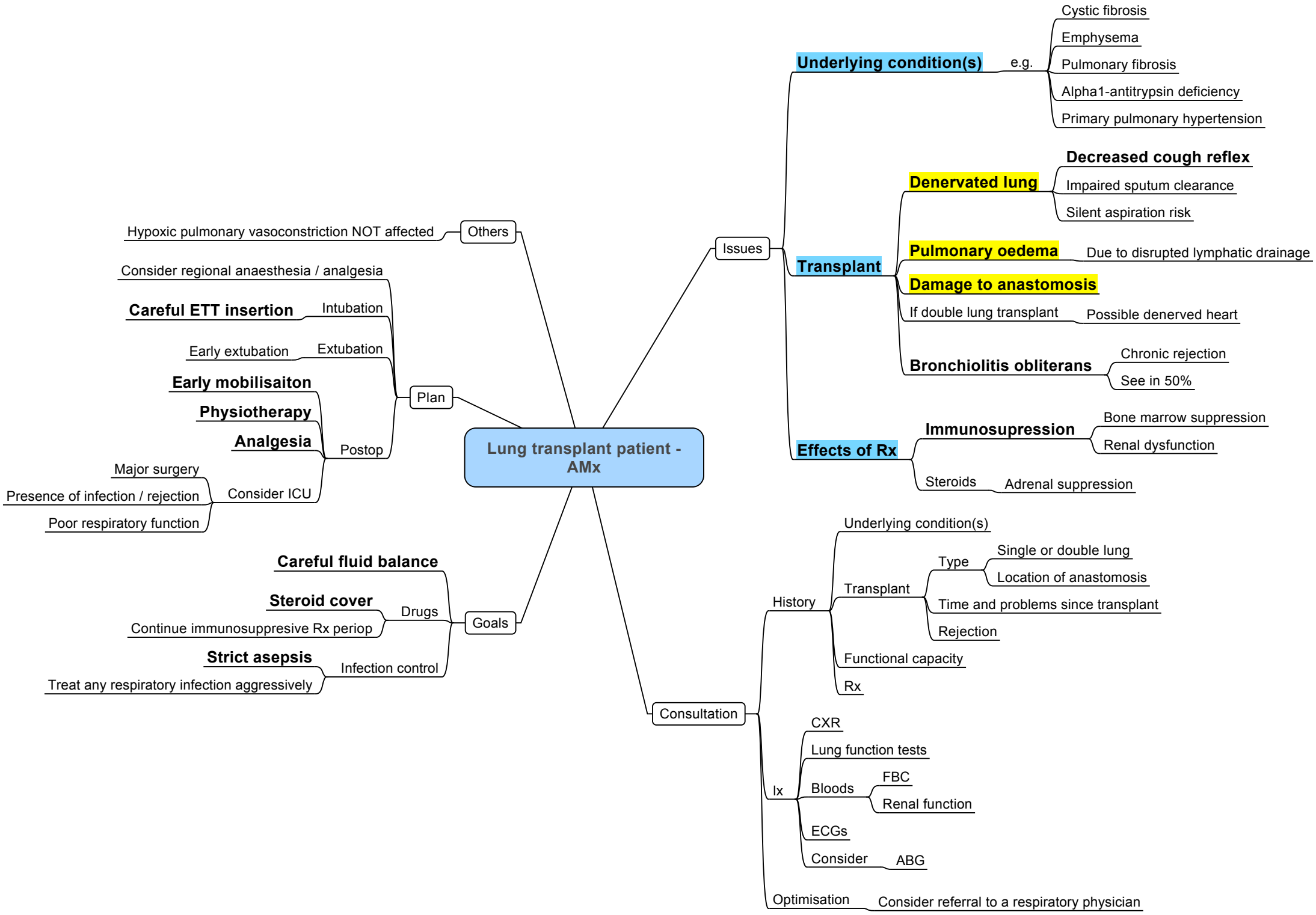

Ischaemic heart disease - CCS classification
Background
CCS = Canadian Cardiovascular Society
Grading the severity of angina
Similar to NYHA classification for heart failures
Classes
Class I
Angina only during strenous or prolonged physical activities
Class II
Slight limitation
Angina only during vigorous physical activities
Class III
Moderate limitation
Angina with everyday living activities
Class IV
Severe limitation
Angina at rest
Inability to perform any activity without angina

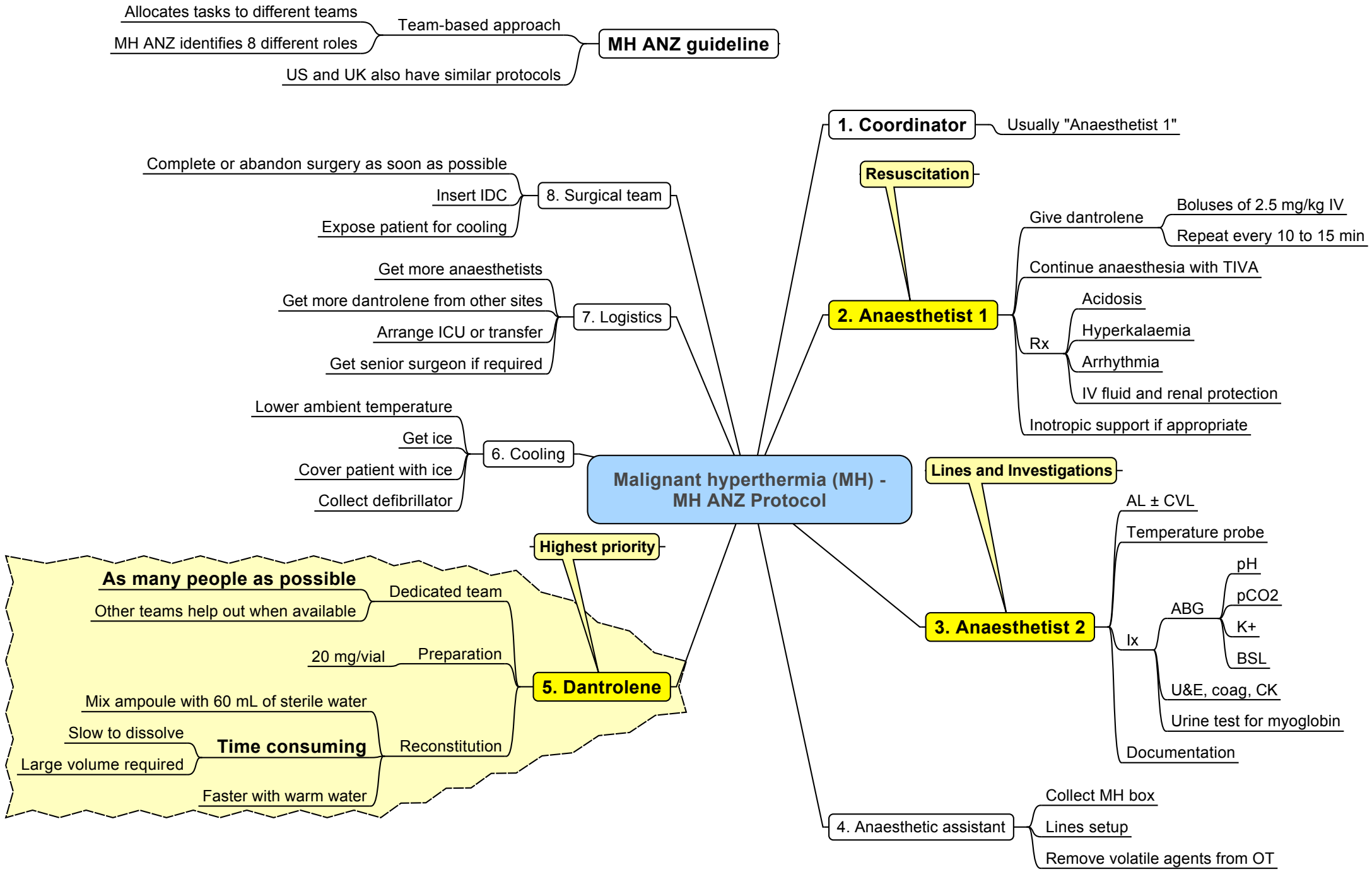

Ischaemic heart disease (IHD) - AMx
Issues
Exclude unstable coronary syndrome
Risk of periop CVS events
"Active cardiac condition" per ACC/AHA
e.g.
Unstable angina
Severe angina (CCS III or IV)
Recent MI (< 30 days)
Unstable coronary syndrome
Plan
M
IBP
CVP
CO monitoring
Post op
Consider
Ensure good analgesia
Postop monitoring in HDU/CCU
Supplemental O2
Goals
Avoid tachycardia
Avoid hypotension
?Avoid severe anaemia
Consultation
History
Angina
Frequency
Trigger
Relief
Previous MI
Previous PCI / CABG?
CABG
Number of vessels
Previous radial artery harvest?
PCI
Functional capacity
CCS classification
METs?
Associated
HTN
DM
Peripheral vascular disease
Rx
Beta-blocker / statin
Continue periop
ACE inhibitor
Nitrates
Antiplatelet Rx
Continue aspirin if possible
Ix
Blood tests
ECG
Perform ONLY if changes management
Consider
Coronary angiography ± PCI
Exercise testing
Pharmacological stress test
Echo
Optimisation
If unstable coronary syndrome, defer unless emergency surgery
Consider cardiology referral, esp...
... e.g.
Major surgery
Active cardiac carditions
Including unstable coronary syndrome
< 4 METs, unless low risk surgery
Stable angina with
Significant left main coronary artery stenosis
3 vessel disease

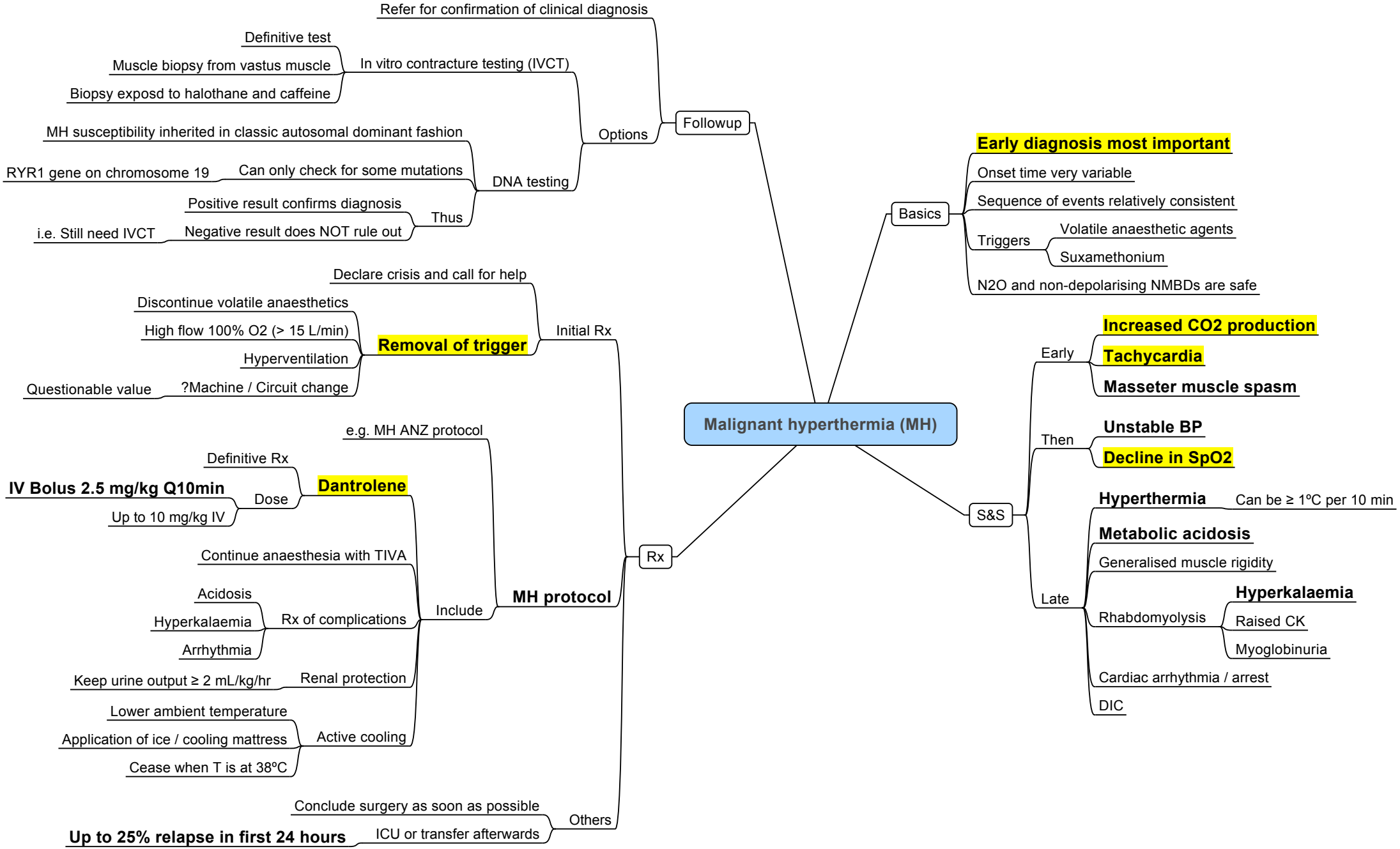

Ischaemic heart disease (IHD) - Stents
(In addition to AMx for IHD)
Stent
DES
Can be
Coated with a thin polymer with anti-proliferative substance
Sirolimus (SES)
Potent immunosuppressive and anti-miotic
Most eluted by 28 days
Fully eluted at 60 days
Paclitaxel (PES)
Potent anti-tumour drug
10% released by 10 days
Rest remain indefinitely
Complications
Restenosis
Side effect of healing process
Patient would require repeat PCI
More common with BMS
Stent thrombosis
Catastrophic
20 to 40% mortality
More common in DES
Early stop of dual Rx is major risk
Contrast-induced acute kidney injury
Anaphylactoid reactions to contrast
Surgery only in centres with 24 hour interventional cardiology cover
Goals
Continue aspirin periop
If on clopidogrel
Continue periop if possible
Otherwise resume as soon as possible
Consider
X-match preop
Platelet for acute bleeding
Cell saver intraop
Issues
Anti-platelet Rx
Risk of stent thrombosis periop
History
PCI
Type
Balloon angioplasty
i.e. No stent
Stents
Bare metal stents (BMS)
Drug-eluting stents (DES)
Time elapsed
How long ago?
Location
Where?
How many?
Length of stents
CVS history as per IHD
Rx
Aspirin
Clopidogrel
Timing of surgery
Balloon angioplasty
Delay until 4 weeks after
But CVS risk may increase after 8 weeks
BMS
Delay till 4 to 6 weeks after
DES
Delay till 12 months after

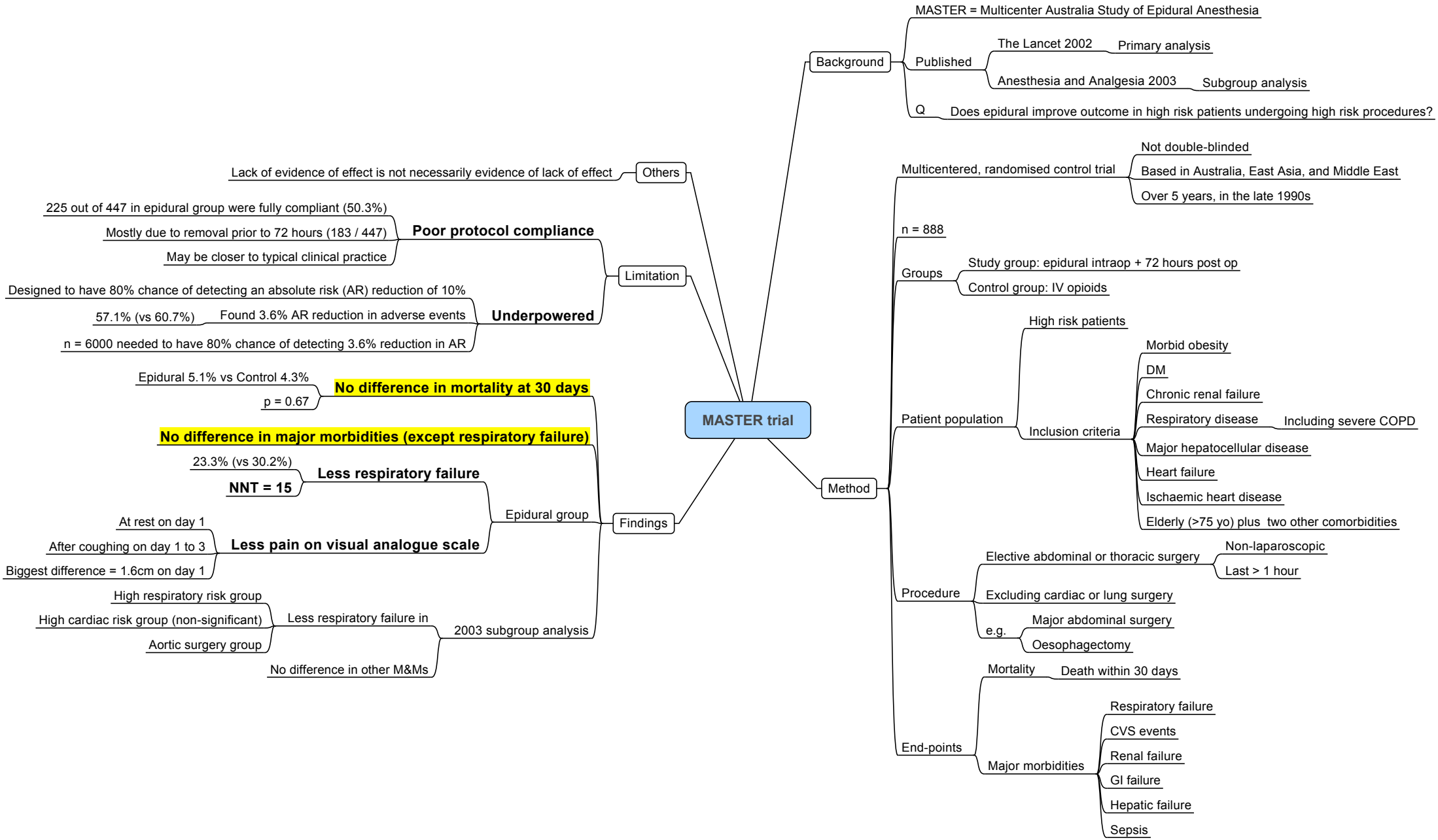

Liver resection surgery - AMx
Consultation
History
Indication
Metastatic colorectal cancer (commonest)
Primary hepatobiliary tumours
Donation (for transplantation)
Hepatic trauma
Resection
Extent of the planned resection
Greater bleeding if right hemi
Liver
?Cirrhosis
Severity of hepatic impairment
PMHx
esp CVS (IHD, CHF), resp, and DM
Rx
?Neoadjuvant chemotherapy
Ix
CT / MRI
Tumour proximity to vena cava
Bloods
CXM 10 units
LFT, coag
ECG
Consider
Echo
Lung function tests
Issues
Major abdominal surgery
Postop analgesia
S
Sudden BP changes (due to surgical manipulations)
Sudden heavy blood loss risk
Clamping of major vessels intraop
P
Possible pre-existing liver impairment
Most are relatively fit
A
Low CVP required
CVS events / instability risk
Air embolism risk
Coagulopathy
Peak at day 2 to 3 postop
Due to
Reduced clotting factors
Platelet dysfunction / thrombocytopenia
Hyperfibrinolysis
Hypoglycaemia
During vascular occlusion
Postop
Possible liver insufficiency / failure postop
Goals
Minimise bleeding
Pre-resection
Prevent coagulopathy
Restrict IV fluid
Vasoconstrictors to treat hypotension
Low CVP during resection
Aim < 5 mmHg
Ideal 0 to 2 mmHg
Avoid excess PEEP
Head up tilt
Communication with surgical team
Clamping of vessels
Resection
Bleeding / venous ooz
Plan
MADE
M
IBP and/or CVP
Temp, BSL, electrolytes (K+, Ca2+)
Consider TEG, CO monitors
A
Large bore IVs
After induction
NGT or nasojejunal tube
D
PRBC, FFP, Tranexamic acid
Vasoconstrictors, inotropes, vasodilators
E
Rapid infuser
Fluid warmer and forced warm air blanket
Consider thoracic epidural analgesia in selected patients
Postop
HDU/ICU admission
Monitor
Coagulopathy
Hypoglycaemia
Hypovolaemia (due to ascites)
Liver failure
Jaundice
Encephalopathy
Early postop enteral nutrition
Analgesia
Avoid paracetamil until normal LFT
PCA

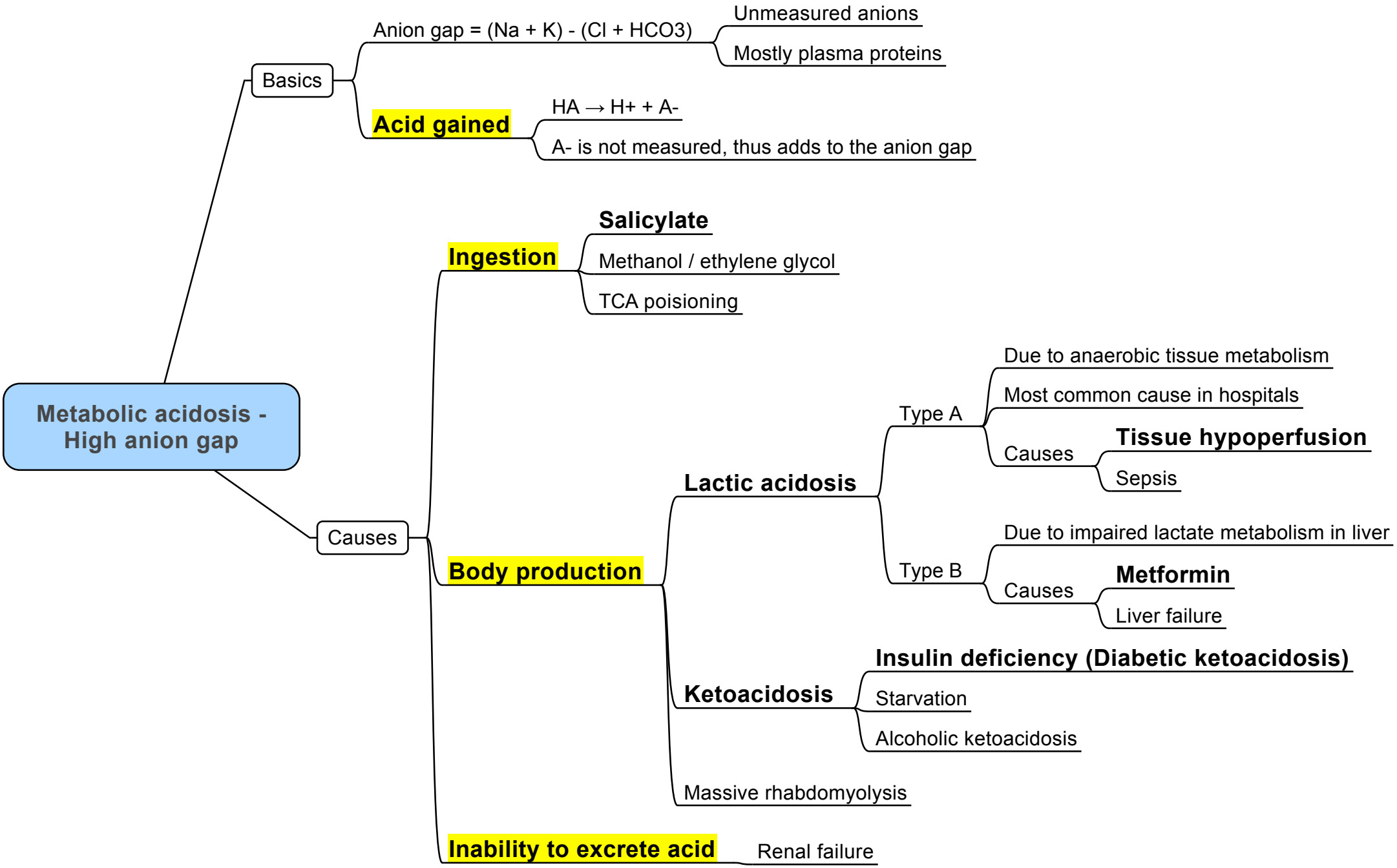

Local anaesthetics (LA) toxicity
S&S
CNS occurs before CVS
CNS
Early
Tingling and numbness around mouth
Tinnitus
Confusion
Then
Muscle tremour
Slurred speech
Late
Coma
Convulsion
Apnoea
CVS
1. Hypotension
2. Bradycardia
3. Arrhythmia
4. Cardiac arrest
4 stages
ECG changes
Prolonged PR
Widened QRS
Ventricular ectopics
Re-entry ventricular arrhythmia
Immediate Rx
Stop LA injection
Call for help
Rx
Special considerations
Toxicity more likely with...
Hyperkalaemia
Hypercarbia
Pregnancy
Lower CC:CNS ratio for bupivacaine
Propofol is NOT a suitable substitute for lipid emulsion
Intralipid is only indicated for CVS instability / arrest
NOT for CNS toxicity
Follow-up
Transfer to ICU
Monitor for pancreatitis for 2 days
If intralipid used
Standard ABCD assessments and resuscitation
If cardiac arrest
ACLS
Resistant to normal resuscitation
esp bupivacaine
CPR may have to be sustained for a considerable time (> 1 hr)
Intralipid
Lipid emulsion 20% IV
Immediate
Loading dose = 1.5 mL/kg
... then start infusion @ 15 mL/kg/hr
If still unstable after 5 min
Extra boluses (same dose, up to 2 doses)
Double infusion rate
Maximum dose = 12 mL/kg
Continue CPR while giving intralipid
Without cardiac arrest
Standard therapy for CVS problems
Consider IV lipid emulsion
Seizure control
Benzodiazepine
Thiopental
Propofol

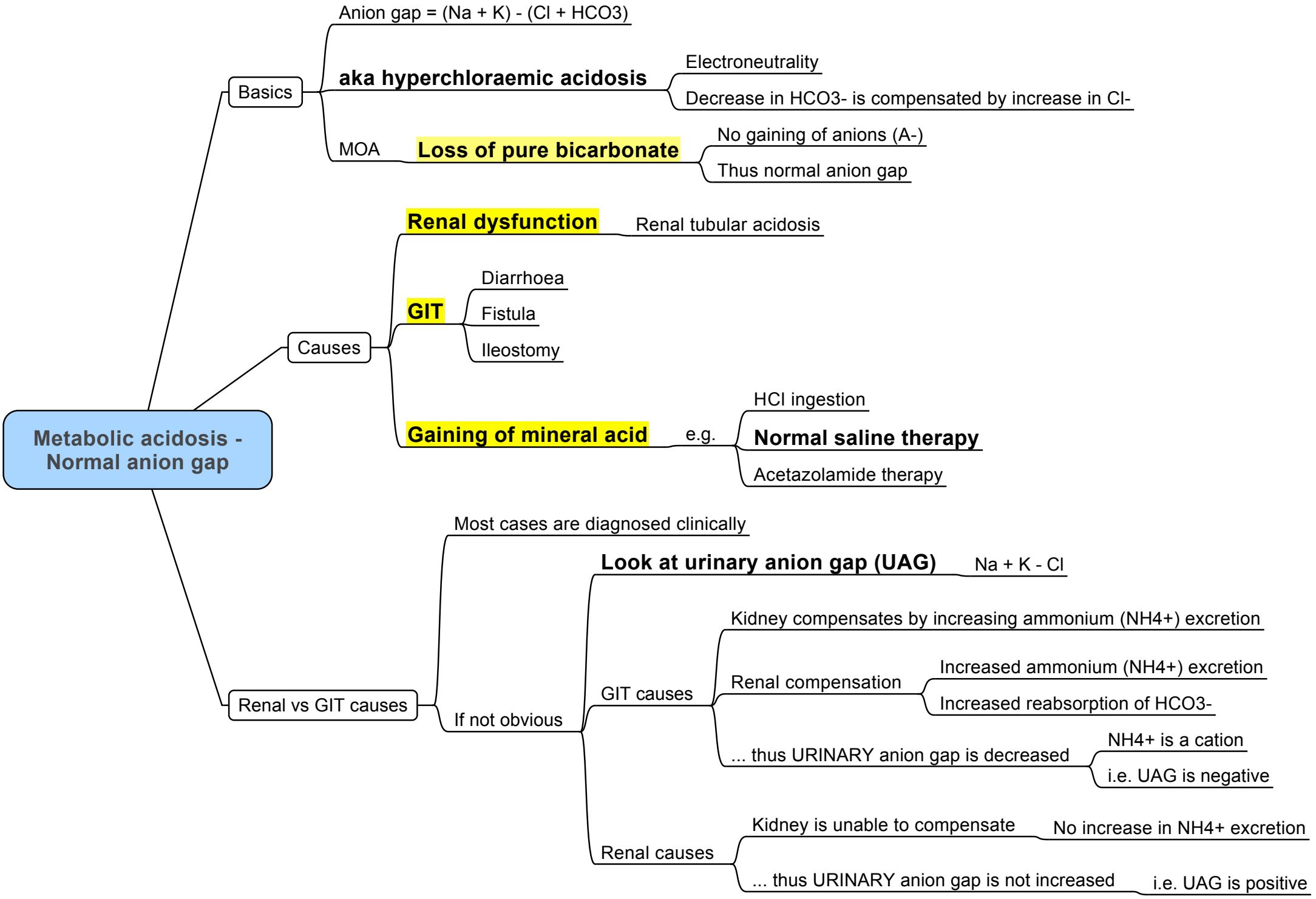

Lung transplant patient - AMx
Issues
Underlying condition(s)
e.g.
Cystic fibrosis
Emphysema
Pulmonary fibrosis
Alpha1-antitrypsin deficiency
Primary pulmonary hypertension
Transplant
Denervated lung
Decreased cough reflex
Impaired sputum clearance
Silent aspiration risk
Pulmonary oedema
Due to disrupted lymphatic drainage
Damage to anastomosis
If double lung transplant
Possible denerved heart
Bronchiolitis obliterans
Chronic rejection
See in 50%
Effects of Rx
Immunosupression
Bone marrow suppression
Renal dysfunction
Steroids
Adrenal suppression
Consultation
History
Underlying condition(s)
Transplant
Type
Single or double lung
Location of anastomosis
Time and problems since transplant
Rejection
Functional capacity
Rx
Ix
CXR
Lung function tests
Bloods
FBC
Renal function
ECGs
Consider
ABG
Optimisation
Consider referral to a respiratory physician
Goals
Careful fluid balance
Drugs
Steroid cover
Continue immunosuppresive Rx periop
Infection control
Strict asepsis
Treat any respiratory infection aggressively
Plan
Intubation
Careful ETT insertion
Consider regional anaesthesia / analgesia
Extubation
Early extubation
Postop
Early mobilisaiton
Physiotherapy
Analgesia
Consider ICU
Major surgery
Presence of infection / rejection
Poor respiratory function
Others
Hypoxic pulmonary vasoconstriction NOT affected

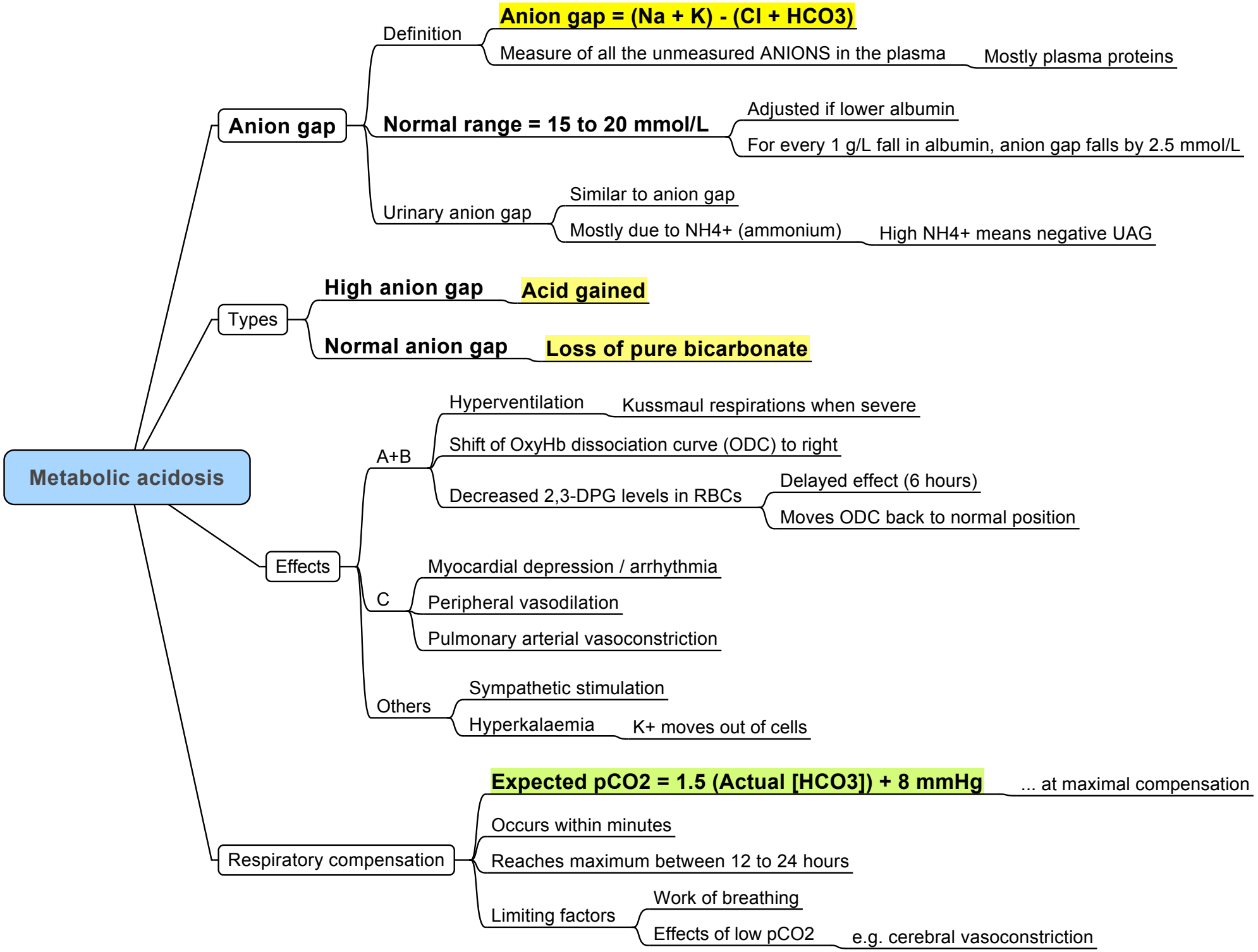

MH ANZ guideline
Team-based approach
Allocates tasks to different teams
MH ANZ identifies 8 different roles
US and UK also have similar protocols
Malignant hyperthermia (MH) - MH ANZ Protocol
1. Coordinator
Usually "Anaesthetist 1"
2. Anaesthetist 1
Resuscitation
Give dantrolene
Boluses of 2.5 mg/kg IV
Repeat every 10 to 15 min
Continue anaesthesia with TIVA
Rx
Acidosis
Hyperkalaemia
Arrhythmia
IV fluid and renal protection
Inotropic support if appropriate
3. Anaesthetist 2
Lines and Investigations
AL ± CVL
Temperature probe
Ix
ABG
pH
pCO2
K+
BSL
U&E, coag, CK
Urine test for myoglobin
Documentation
4. Anaesthetic assistant
Collect MH box
Lines setup
Remove volatile agents from OT
5. Dantrolene
Highest priority
Dedicated team
As many people as possible
Other teams help out when available
Preparation
20 mg/vial
Reconstitution
Mix ampoule with 60 mL of sterile water
Time consuming
Slow to dissolve
Large volume required
Faster with warm water
6. Cooling
Lower ambient temperature
Get ice
Cover patient with ice
Collect defibrillator
7. Logistics
Get more anaesthetists
Get more dantrolene from other sites
Arrange ICU or transfer
Get senior surgeon if required
8. Surgical team
Complete or abandon surgery as soon as possible
Insert IDC
Expose patient for cooling

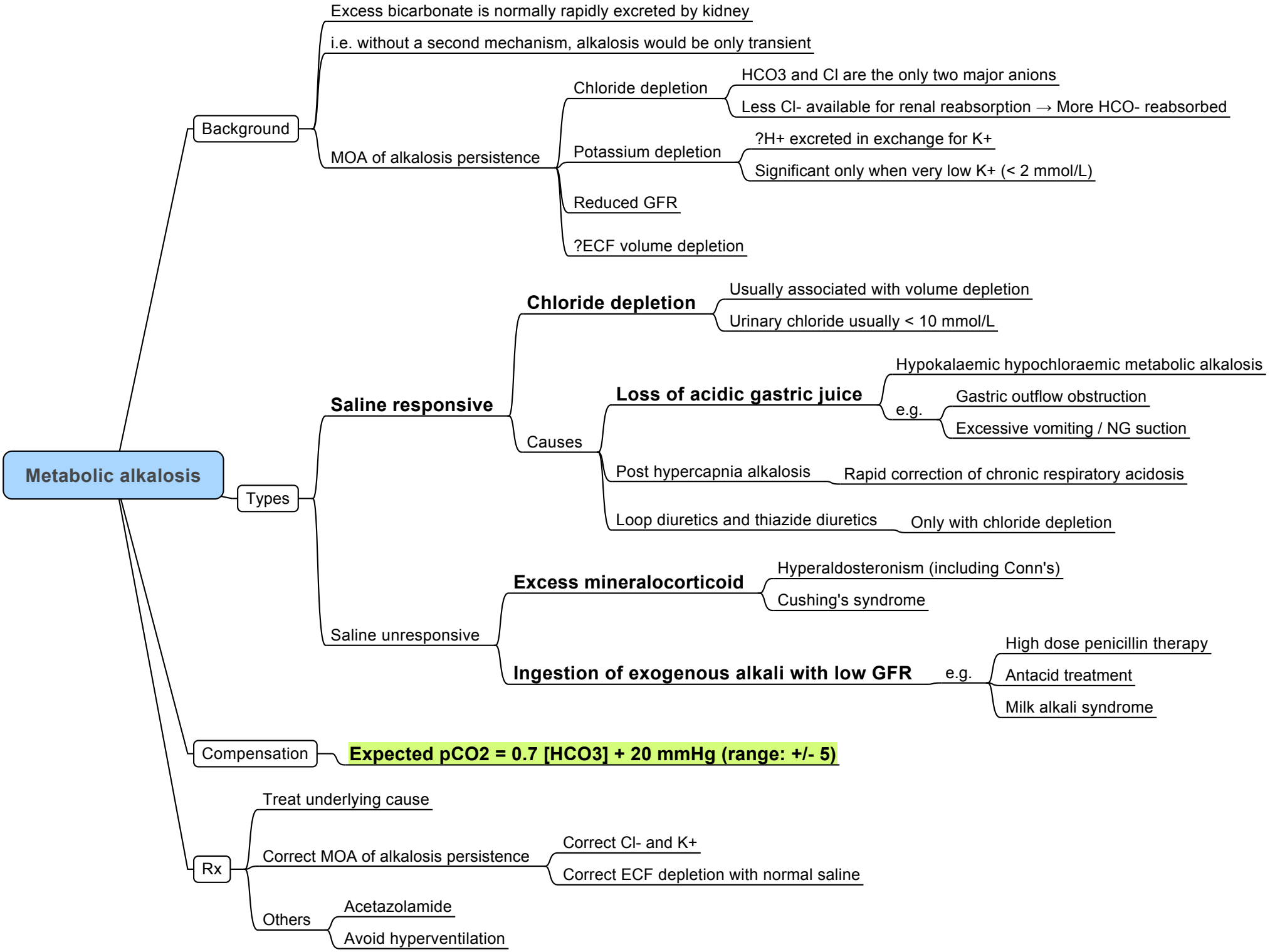

Malignant hyperthermia (MH)
Basics
Early diagnosis most important
Onset time very variable
Sequence of events relatively consistent
Triggers
Volatile anaesthetic agents
Suxamethonium
N2O and non-depolarising NMBDs are safe
S&S
Early
Increased CO2 production
Tachycardia
Masseter muscle spasm
Then
Unstable BP
Decline in SpO2
Late
Hyperthermia
Can be ≥ 1ºC per 10 min
Metabolic acidosis
Generalised muscle rigidity
Rhabdomyolysis
Hyperkalaemia
Raised CK
Myoglobinuria
Cardiac arrhythmia / arrest
DIC
Rx
Initial Rx
Declare crisis and call for help
Removal of trigger
Discontinue volatile anaesthetics
High flow 100% O2 (> 15 L/min)
Hyperventilation
?Machine / Circuit change
Questionable value
MH protocol
e.g. MH ANZ protocol
Include
Dantrolene
Definitive Rx
Dose
IV Bolus 2.5 mg/kg Q10min
Up to 10 mg/kg IV
Continue anaesthesia with TIVA
Rx of complications
Acidosis
Hyperkalaemia
Arrhythmia
Renal protection
Keep urine output ≥ 2 mL/kg/hr
Active cooling
Lower ambient temperature
Application of ice / cooling mattress
Cease when T is at 38ºC
Others
Conclude surgery as soon as possible
ICU or transfer afterwards
Up to 25% relapse in first 24 hours
Followup
Refer for confirmation of clinical diagnosis
Options
In vitro contracture testing (IVCT)
Definitive test
Muscle biopsy from vastus muscle
Biopsy exposd to halothane and caffeine
DNA testing
MH susceptibility inherited in classic autosomal dominant fashion
Can only check for some mutations
RYR1 gene on chromosome 19
Thus
Positive result confirms diagnosis
Negative result does NOT rule out
i.e. Still need IVCT

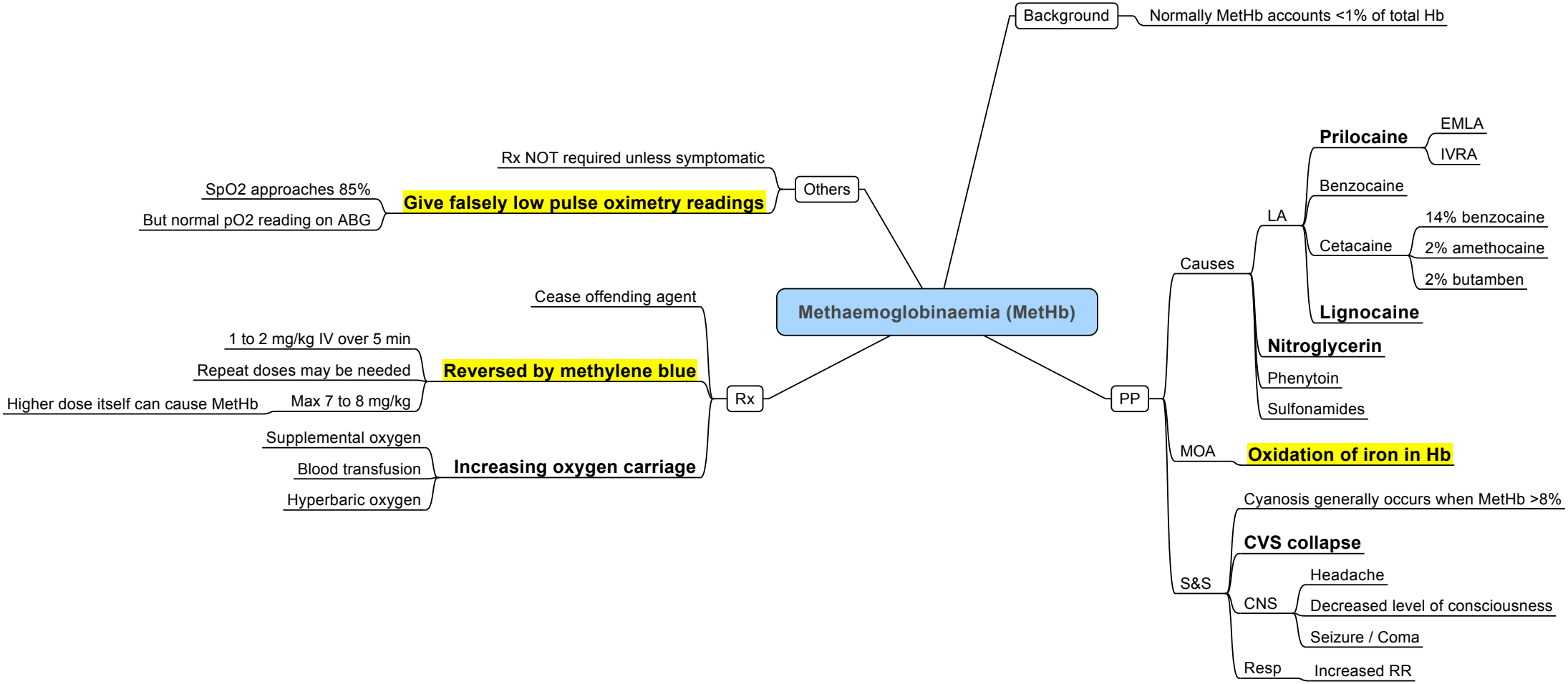

MASTER trial
Background
MASTER = Multicenter Australia Study of Epidural Anesthesia
Published
The Lancet 2002
Primary analysis
Anesthesia and Analgesia 2003
Subgroup analysis
Q
Does epidural improve outcome in high risk patients undergoing high risk procedures?
Method
Multicentered, randomised control trial
Not double-blinded
Based in Australia, East Asia, and Middle East
Over 5 years, in the late 1990s
n = 888
Groups
Study group: epidural intraop + 72 hours post op
Control group: IV opioids
Patient population
Inclusion criteria
High risk patients
Morbid obesity
DM
Chronic renal failure
Respiratory disease
Including severe COPD
Major hepatocellular disease
Heart failure
Ischaemic heart disease
Elderly (>75 yo) plus two other comorbidities
Procedure
Elective abdominal or thoracic surgery
Non-laparoscopic
Last > 1 hour
Excluding cardiac or lung surgery
e.g.
Major abdominal surgery
Oesophagectomy
End-points
Mortality
Death within 30 days
Major morbidities
Respiratory failure
CVS events
Renal failure
GI failure
Hepatic failure
Sepsis
Findings
Epidural group
No difference in mortality at 30 days
Epidural 5.1% vs Control 4.3%
p = 0.67
No difference in major morbidities (except respiratory failure)
Less respiratory failure
23.3% (vs 30.2%)
NNT = 15
Less pain on visual analogue scale
At rest on day 1
After coughing on day 1 to 3
Biggest difference = 1.6cm on day 1
2003 subgroup analysis
Less respiratory failure in
High respiratory risk group
High cardiac risk group (non-significant)
Aortic surgery group
No difference in other M&Ms
Limitation
Poor protocol compliance
225 out of 447 in epidural group were fully compliant (50.3%)
Mostly due to removal prior to 72 hours (183 / 447)
May be closer to typical clinical practice
Underpowered
Designed to have 80% chance of detecting an absolute risk (AR) reduction of 10%
Found 3.6% AR reduction in adverse events
57.1% (vs 60.7%)
n = 6000 needed to have 80% chance of detecting 3.6% reduction in AR
Others
Lack of evidence of effect is not necessarily evidence of lack of effect

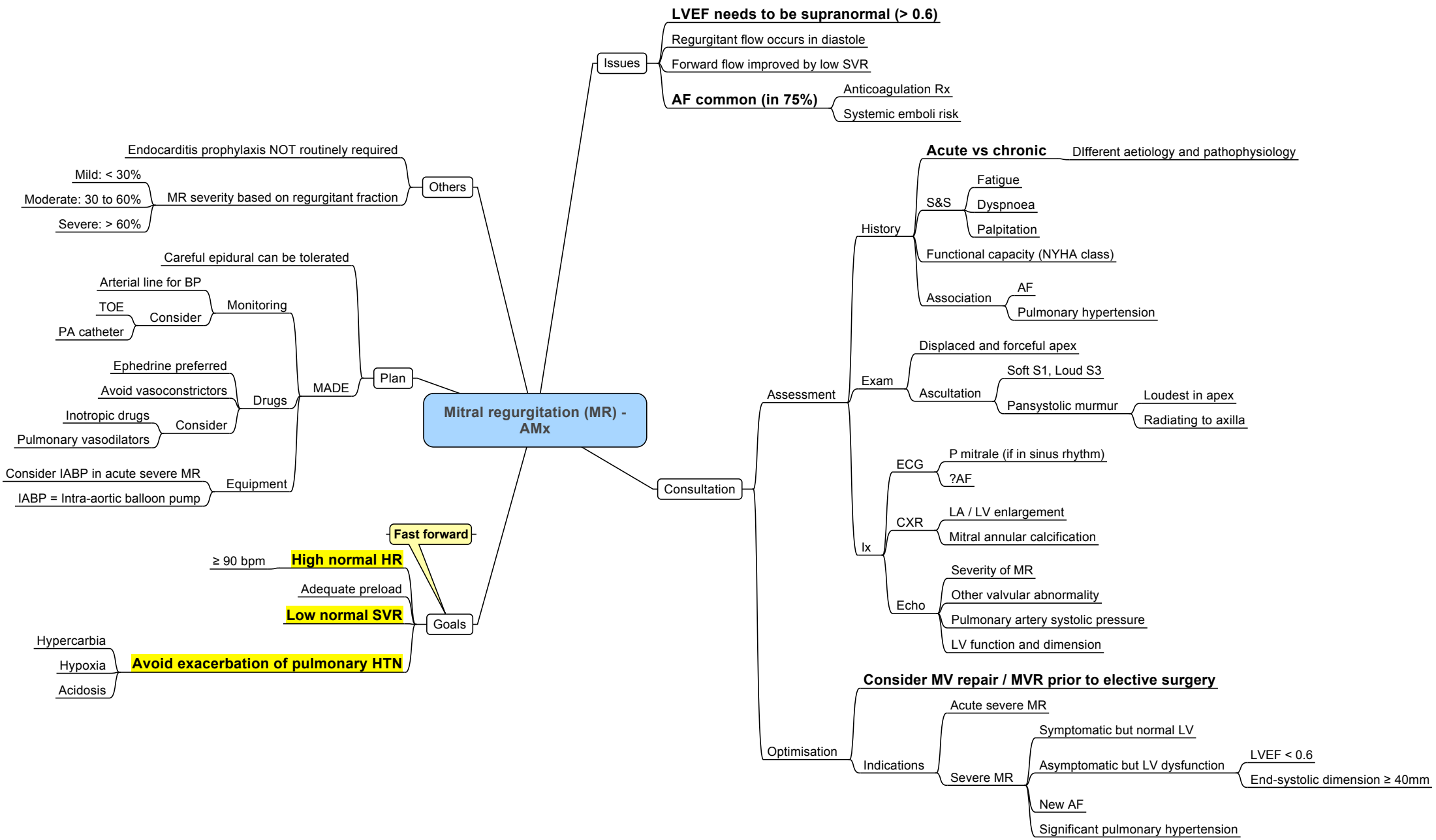

Metabolic acidosis - High anion gap
Basics
Anion gap = (Na + K) - (Cl + HCO3)
Unmeasured anions
Mostly plasma proteins
Acid gained
HA → H+ + A-
A- is not measured, thus adds to the anion gap
Causes
Ingestion
Salicylate
Methanol / ethylene glycol
TCA poisioning
Body production
Lactic acidosis
Type A
Due to anaerobic tissue metabolism
Most common cause in hospitals
Causes
Tissue hypoperfusion
Sepsis
Type B
Due to impaired lactate metabolism in liver
Causes
Metformin
Liver failure
Ketoacidosis
Insulin deficiency (Diabetic ketoacidosis)
Starvation
Alcoholic ketoacidosis
Massive rhabdomyolysis
Inability to excrete acid
Renal failure

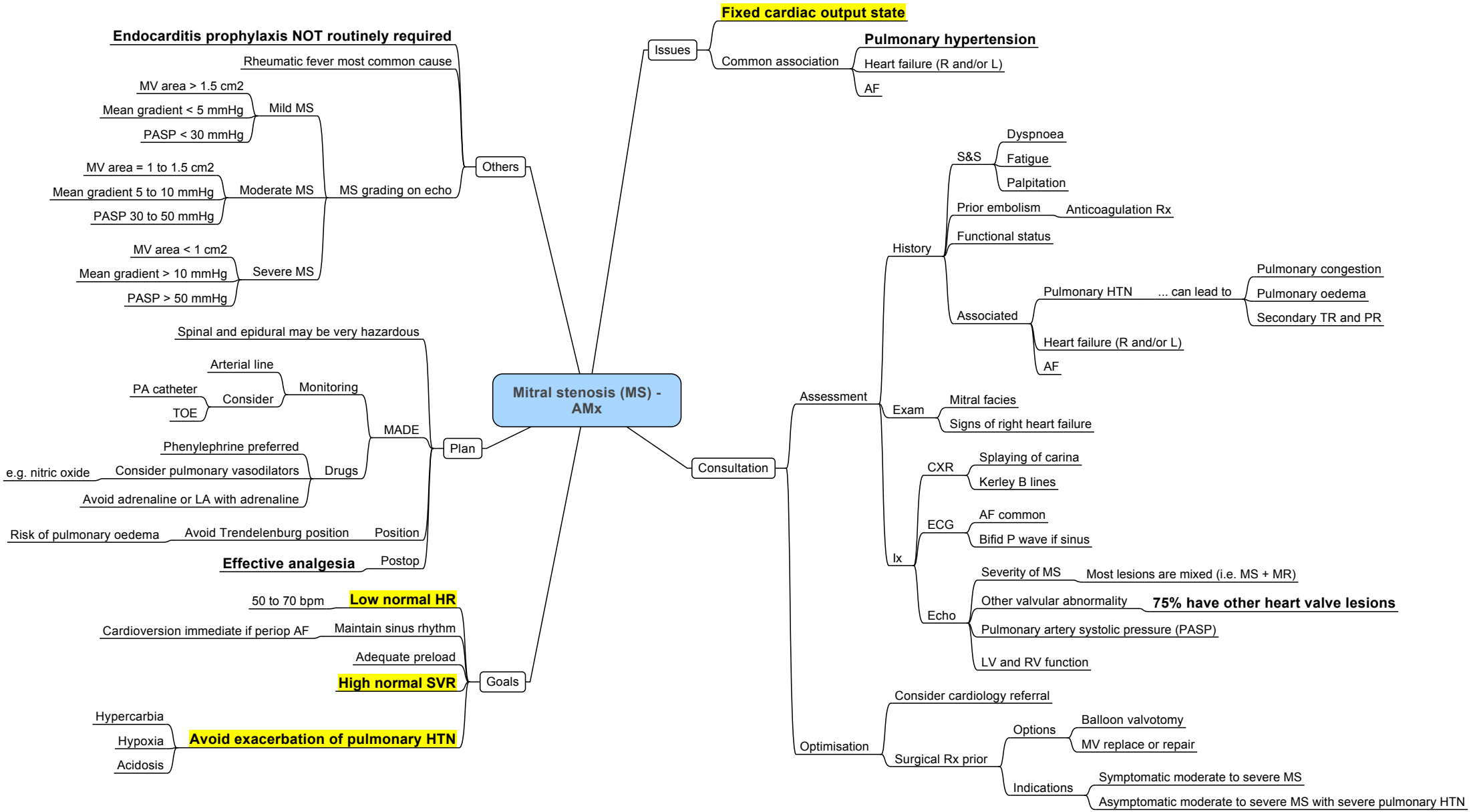

Metabolic acidosis - Normal anion gap
Basics
Anion gap = (Na + K) - (Cl + HCO3)
aka hyperchloraemic acidosis
Electroneutrality
Decrease in HCO3- is compensated by increase in Cl-
MOA
Loss of pure bicarbonate
No gaining of anions (A-)
Thus normal anion gap
Causes
Renal dysfunction
Renal tubular acidosis
GIT
Diarrhoea
Fistula
Ileostomy
Gaining of mineral acid
e.g.
HCl ingestion
Normal saline therapy
Acetazolamide therapy
Renal vs GIT causes
Most cases are diagnosed clinically
If not obvious
Look at urinary anion gap (UAG)
Na + K - Cl
GIT causes
Kidney compensates by increasing ammonium (NH4+) excretion
Renal compensation
Increased ammonium (NH4+) excretion
Increased reabsorption of HCO3-
... thus URINARY anion gap is decreased
NH4+ is a cation
i.e. UAG is negative
Renal causes
Kidney is unable to compensate
No increase in NH4+ excretion
... thus URINARY anion gap is not increased
i.e. UAG is positive

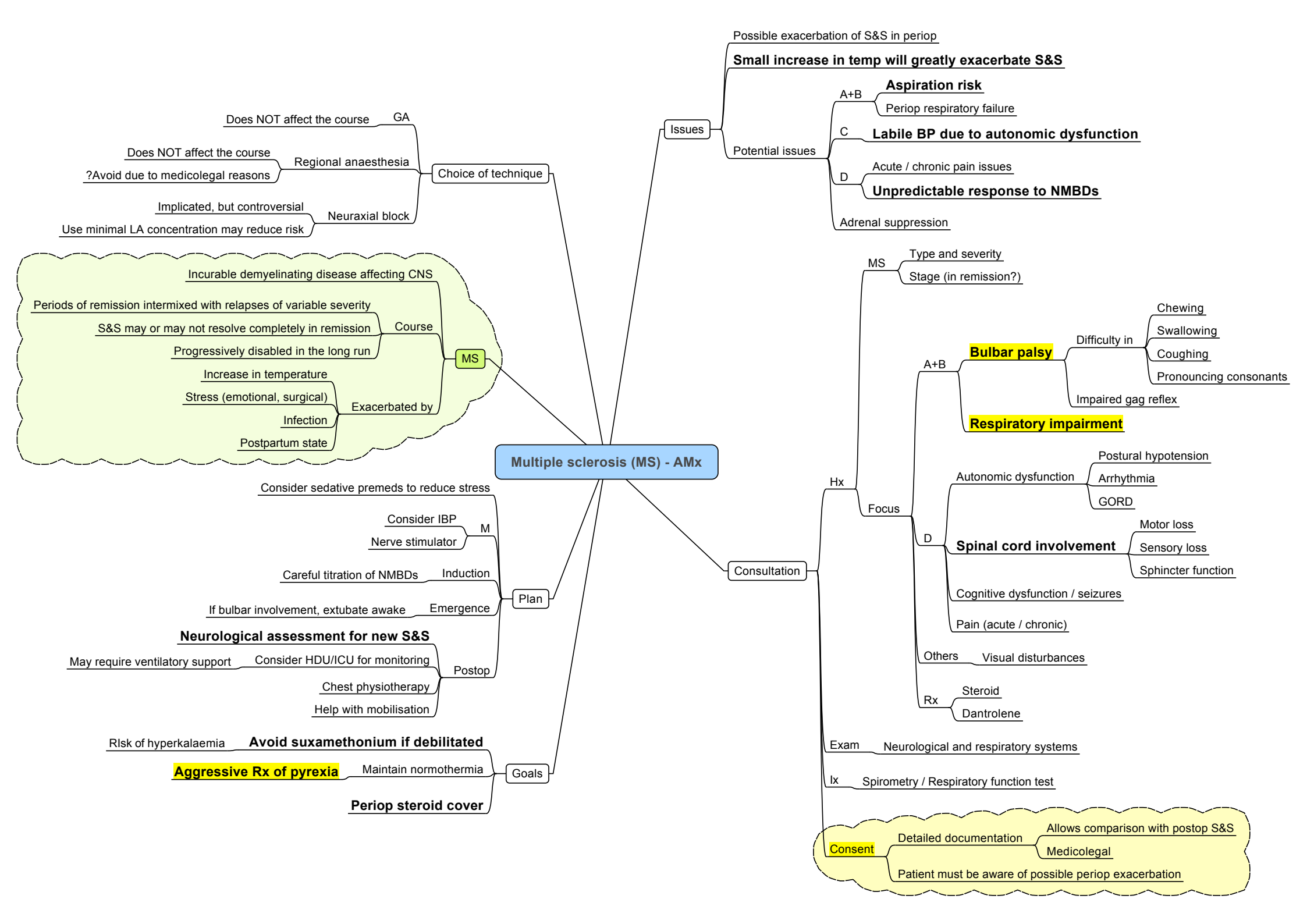

Metabolic acidosis
Anion gap
Definition
Anion gap = (Na + K) - (Cl + HCO3)
Measure of all the unmeasured ANIONS in the plasma
Mostly plasma proteins
Normal range = 15 to 20 mmol/L
Adjusted if lower albumin
For every 1 g/L fall in albumin, anion gap falls by 2.5 mmol/L
Urinary anion gap
Similar to anion gap
Mostly due to NH4+ (ammonium)
High NH4+ means negative UAG
Types
High anion gap
Acid gained
Normal anion gap
Loss of pure bicarbonate
Effects
A+B
Hyperventilation
Kussmaul respirations when severe
Shift of OxyHb dissociation curve (ODC) to right
Decreased 2,3-DPG levels in RBCs
Delayed effect (6 hours)
Moves ODC back to normal position
C
Myocardial depression / arrhythmia
Peripheral vasodilation
Pulmonary arterial vasoconstriction
Others
Sympathetic stimulation
Hyperkalaemia
K+ moves out of cells
Respiratory compensation
Expected pCO2 = 1.5 (Actual [HCO3]) + 8 mmHg
... at maximal compensation
Occurs within minutes
Reaches maximum between 12 to 24 hours
Limiting factors
Work of breathing
Effects of low pCO2
e.g. cerebral vasoconstriction

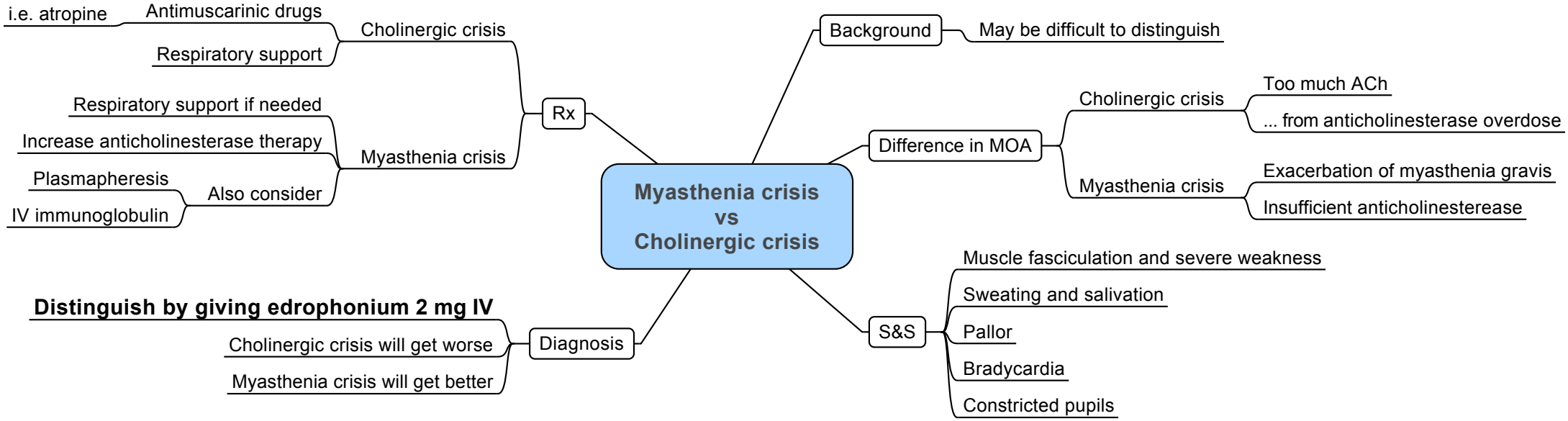

Metabolic alkalosis
Background
Excess bicarbonate is normally rapidly excreted by kidney
i.e. without a second mechanism, alkalosis would be only transient
MOA of alkalosis persistence
Chloride depletion
HCO3 and Cl are the only two major anions
Less Cl- available for renal reabsorption → More HCO- reabsorbed
Potassium depletion
?H+ excreted in exchange for K+
Significant only when very low K+ (< 2 mmol/L)
Reduced GFR
?ECF volume depletion
Types
Saline responsive
Chloride depletion
Usually associated with volume depletion
Urinary chloride usually < 10 mmol/L
Causes
Loss of acidic gastric juice
Hypokalaemic hypochloraemic metabolic alkalosis
e.g.
Gastric outflow obstruction
Excessive vomiting / NG suction
Post hypercapnia alkalosis
Rapid correction of chronic respiratory acidosis
Loop diuretics and thiazide diuretics
Only with chloride depletion
Saline unresponsive
Excess mineralocorticoid
Hyperaldosteronism (including Conn's)
Cushing's syndrome
Ingestion of exogenous alkali with low GFR
e.g.
High dose penicillin therapy
Antacid treatment
Milk alkali syndrome
Compensation
Expected pCO2 = 0.7 [HCO3] + 20 mmHg (range: +/- 5)
Rx
Treat underlying cause
Correct MOA of alkalosis persistence
Correct Cl- and K+
Correct ECF depletion with normal saline
Others
Acetazolamide
Avoid hyperventilation

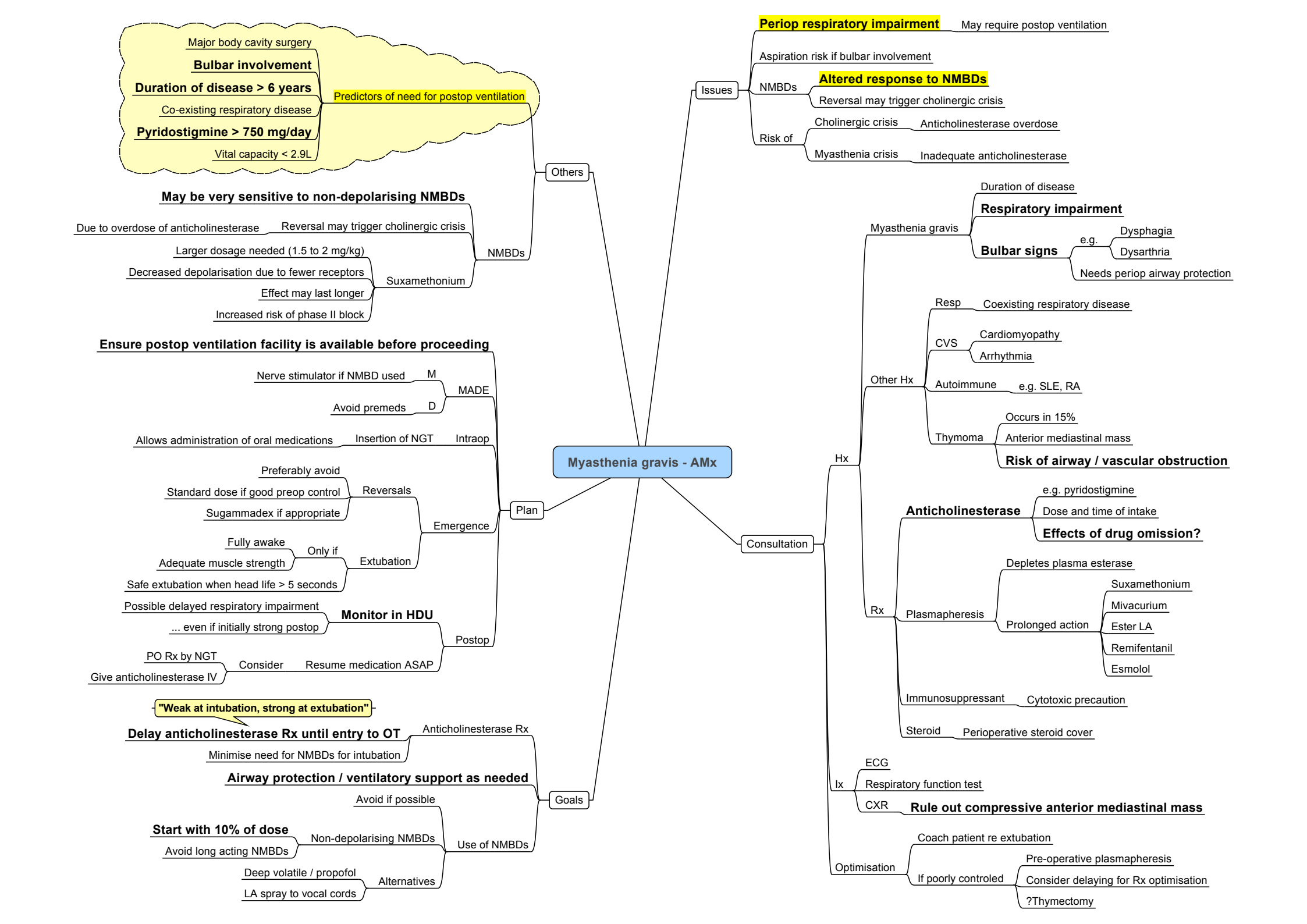

Methaemoglobinaemia (MetHb)
Background
Normally MetHb accounts <1% of total Hb
PP
Causes
LA
Prilocaine
EMLA
IVRA
Benzocaine
Cetacaine
14% benzocaine
2% amethocaine
2% butamben
Lignocaine
Nitroglycerin
Phenytoin
Sulfonamides
MOA
Oxidation of iron in Hb
S&S
Cyanosis generally occurs when MetHb >8%
CVS collapse
CNS
Headache
Decreased level of consciousness
Seizure / Coma
Resp
Increased RR
Rx
Cease offending agent
Reversed by methylene blue
1 to 2 mg/kg IV over 5 min
Repeat doses may be needed
Max 7 to 8 mg/kg
Higher dose itself can cause MetHb
Increasing oxygen carriage
Supplemental oxygen
Blood transfusion
Hyperbaric oxygen
Others
Rx NOT required unless symptomatic
Give falsely low pulse oximetry readings
SpO2 approaches 85%
But normal pO2 reading on ABG

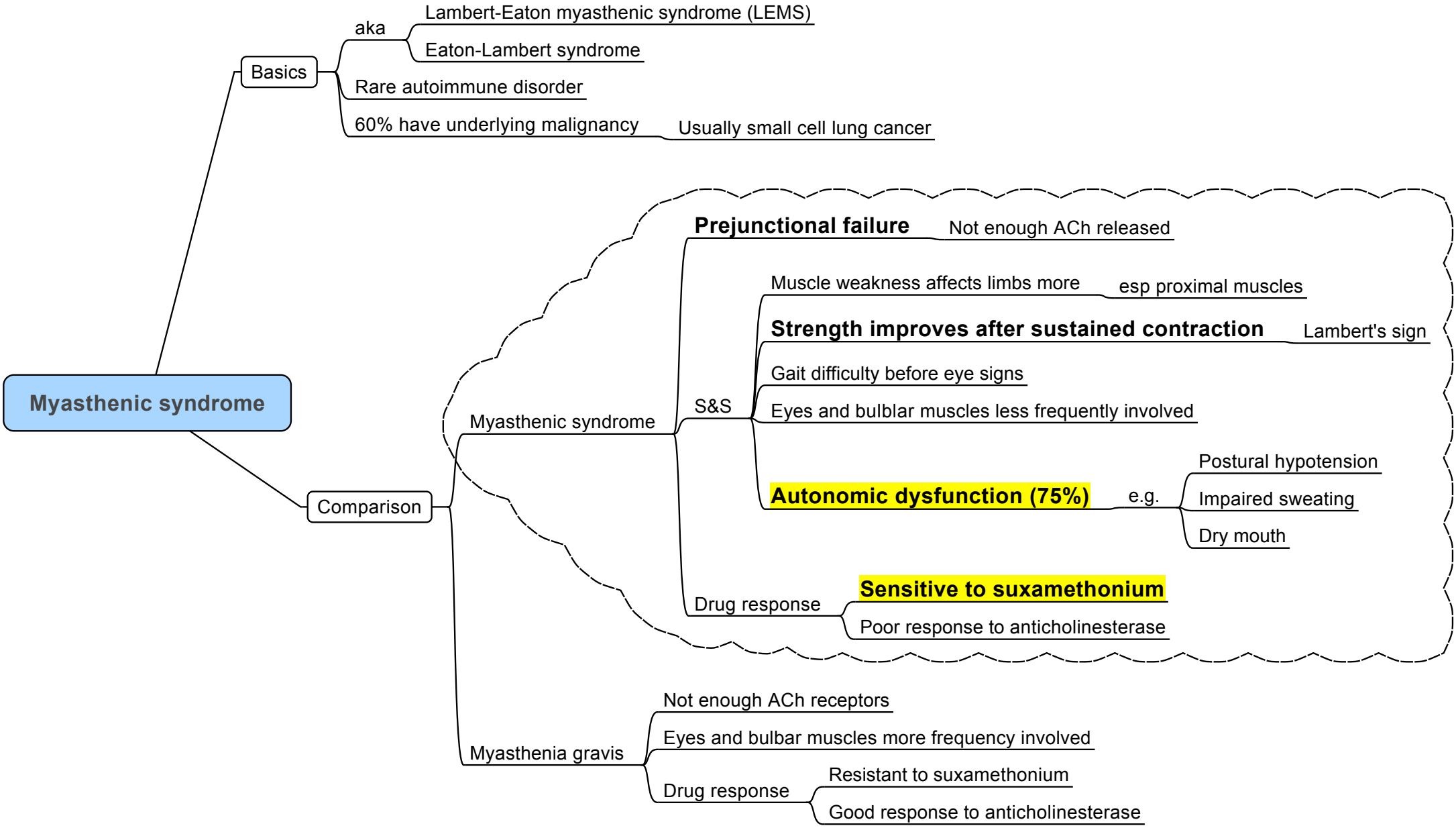

Mitral regurgitation (MR) - AMx
Issues
LVEF needs to be supranormal (> 0.6)
Regurgitant flow occurs in diastole
Forward flow improved by low SVR
AF common (in 75%)
Anticoagulation Rx
Systemic emboli risk
Others
Endocarditis prophylaxis NOT routinely required
MR severity based on regurgitant fraction
Mild: < 30%
Moderate: 30 to 60%
Severe: > 60%
Plan
MADE
Careful epidural can be tolerated
Monitoring
Consider
Arterial line for BP
TOE
PA catheter
Drugs
Ephedrine preferred
Avoid vasoconstrictors
Consider
Inotropic drugs
Pulmonary vasodilators
Equipment
Consider IABP in acute severe MR
IABP = Intra-aortic balloon pump
Goals
Fast forward
≥ 90 bpm
High normal HR
Adequate preload
Low normal SVR
Avoid exacerbation of pulmonary HTN
Hypercarbia
Hypoxia
Acidosis
Consultation
Assessment
Acute vs chronic
DIfferent aetiology and pathophysiology
History
S&S
Fatigue
Dyspnoea
Palpitation
Functional capacity (NYHA class)
Association
AF
Pulmonary hypertension
Exam
Displaced and forceful apex
Ascultation
Soft S1, Loud S3
Pansystolic murmur
Loudest in apex
Radiating to axilla
Ix
ECG
P mitrale (if in sinus rhythm)
?AF
CXR
LA / LV enlargement
Mitral annular calcification
Echo
Severity of MR
Other valvular abnormality
Pulmonary artery systolic pressure
LV function and dimension
Optimisation
Consider MV repair / MVR prior to elective surgery
Indications
Acute severe MR
Severe MR
Symptomatic but normal LV
Asymptomatic but LV dysfunction
LVEF < 0.6
End-systolic dimension ≥ 40mm
New AF
Significant pulmonary hypertension

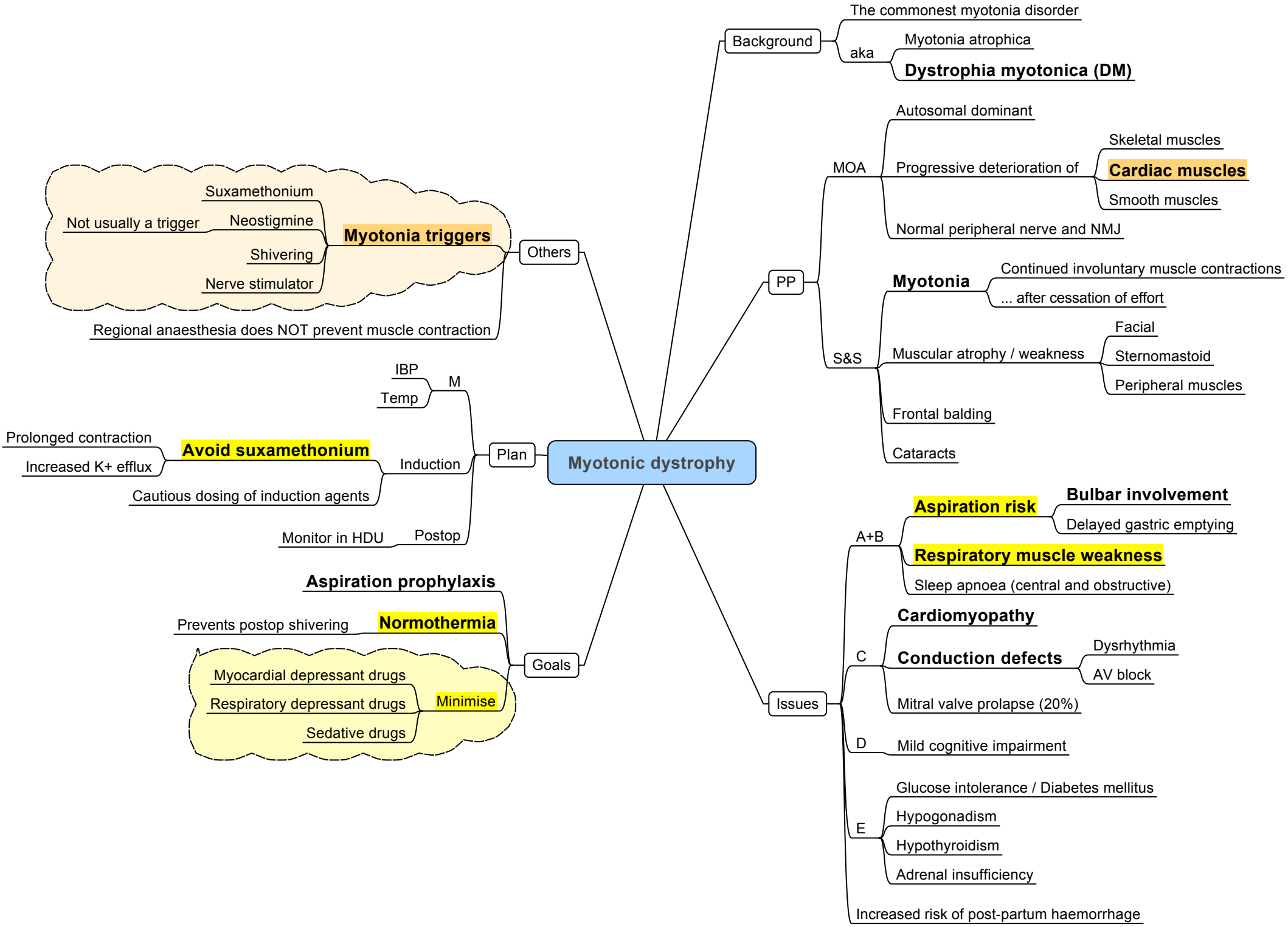

Mitral stenosis (MS) - AMx
Consultation
Assessment
History
S&S
Dyspnoea
Fatigue
Palpitation
Prior embolism
Anticoagulation Rx
Functional status
Associated
Pulmonary HTN
... can lead to
Pulmonary congestion
Pulmonary oedema
Secondary TR and PR
Heart failure (R and/or L)
AF
Exam
Mitral facies
Signs of right heart failure
Ix
CXR
Splaying of carina
Kerley B lines
ECG
AF common
Bifid P wave if sinus
Echo
Severity of MS
Most lesions are mixed (i.e. MS + MR)
Other valvular abnormality
75% have other heart valve lesions
Pulmonary artery systolic pressure (PASP)
LV and RV function
Optimisation
Consider cardiology referral
Surgical Rx prior
Options
Balloon valvotomy
MV replace or repair
Indications
Symptomatic moderate to severe MS
Asymptomatic moderate to severe MS with severe pulmonary HTN
Issues
Fixed cardiac output state
Common association
Pulmonary hypertension
Heart failure (R and/or L)
AF
Others
Endocarditis prophylaxis NOT routinely required
Rheumatic fever most common cause
MS grading on echo
Mild MS
MV area > 1.5 cm2
Mean gradient < 5 mmHg
PASP < 30 mmHg
Moderate MS
MV area = 1 to 1.5 cm2
Mean gradient 5 to 10 mmHg
PASP 30 to 50 mmHg
Severe MS
MV area < 1 cm2
Mean gradient > 10 mmHg
PASP > 50 mmHg
Plan
Spinal and epidural may be very hazardous
MADE
Monitoring
Consider
Arterial line
PA catheter
TOE
Drugs
Phenylephrine preferred
Consider pulmonary vasodilators
e.g. nitric oxide
Avoid adrenaline or LA with adrenaline
Position
Avoid Trendelenburg position
Risk of pulmonary oedema
Postop
Effective analgesia
Goals
Low normal HR
50 to 70 bpm
Maintain sinus rhythm
Cardioversion immediate if periop AF
Adequate preload
High normal SVR
Avoid exacerbation of pulmonary HTN
Hypercarbia
Hypoxia
Acidosis

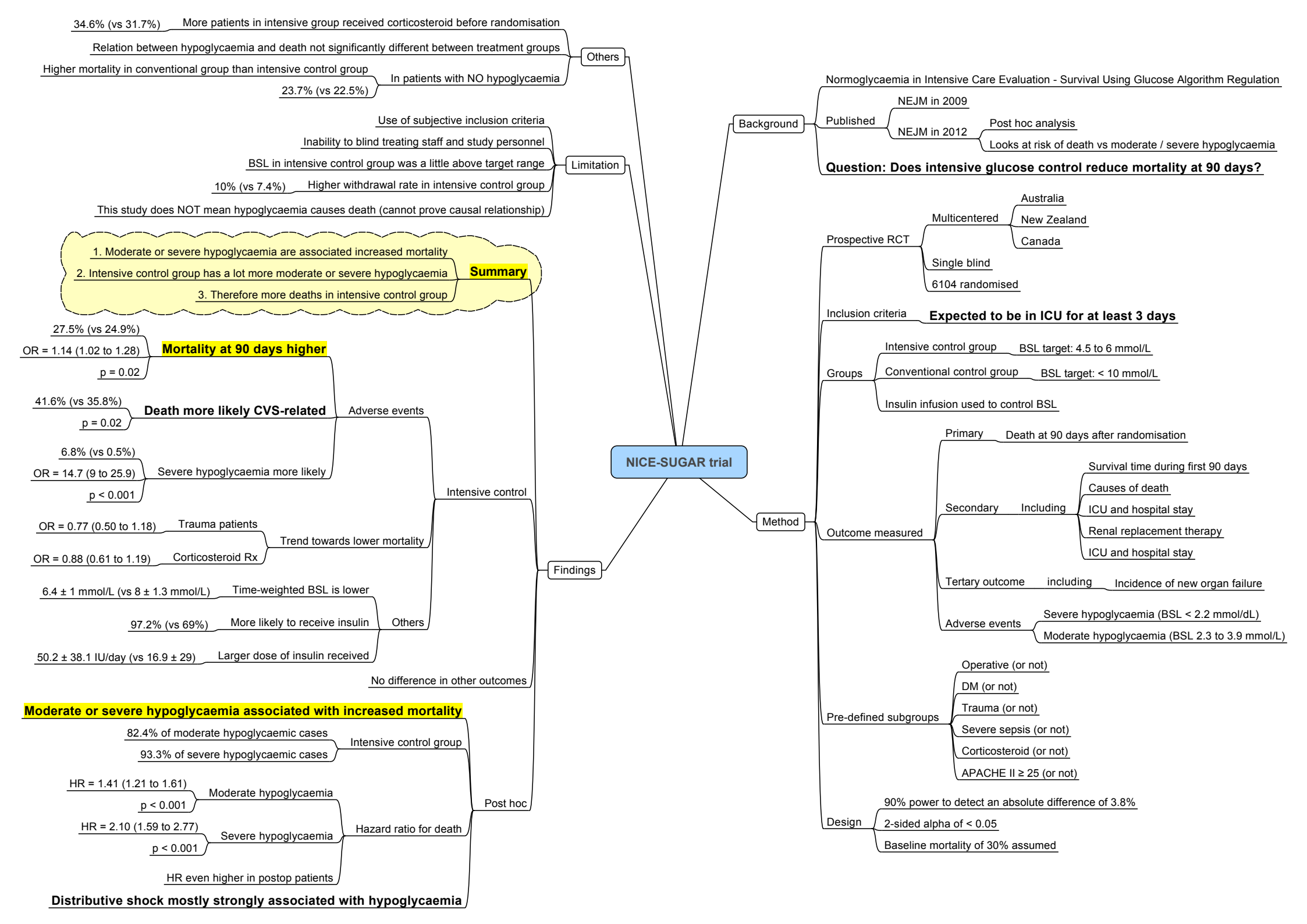

Multiple sclerosis (MS) - AMx
Issues
Possible exacerbation of S&S in periop
Small increase in temp will greatly exacerbate S&S
Potential issues
A+B
Aspiration risk
Periop respiratory failure
C
Labile BP due to autonomic dysfunction
D
Acute / chronic pain issues
Unpredictable response to NMBDs
Adrenal suppression
Choice of technique
GA
Does NOT affect the course
Regional anaesthesia
Does NOT affect the course
?Avoid due to medicolegal reasons
Neuraxial block
Implicated, but controversial
Use minimal LA concentration may reduce risk
MS
Incurable demyelinating disease affecting CNS
Course
Periods of remission intermixed with relapses of variable severity
S&S may or may not resolve completely in remission
Progressively disabled in the long run
Exacerbated by
Increase in temperature
Stress (emotional, surgical)
Infection
Postpartum state
Consultation
Hx
MS
Type and severity
Stage (in remission?)
Focus
A+B
Bulbar palsy
Difficulty in
Chewing
Swallowing
Coughing
Pronouncing consonants
Impaired gag reflex
Respiratory impairment
D
Autonomic dysfunction
Postural hypotension
Arrhythmia
GORD
Spinal cord involvement
Motor loss
Sensory loss
Sphincter function
Cognitive dysfunction / seizures
Pain (acute / chronic)
Others
Visual disturbances
Rx
Steroid
Dantrolene
Exam
Neurological and respiratory systems
Ix
Spirometry / Respiratory function test
Consent
Detailed documentation
Allows comparison with postop S&S
Medicolegal
Patient must be aware of possible periop exacerbation
Plan
Consider sedative premeds to reduce stress
M
Consider IBP
Nerve stimulator
Induction
Careful titration of NMBDs
Emergence
If bulbar involvement, extubate awake
Postop
Neurological assessment for new S&S
May require ventilatory support
Consider HDU/ICU for monitoring
Chest physiotherapy
Help with mobilisation
Goals
Risk of hyperkalaemia
Avoid suxamethonium if debilitated
Aggressive Rx of pyrexia
Maintain normothermia
Periop steroid cover

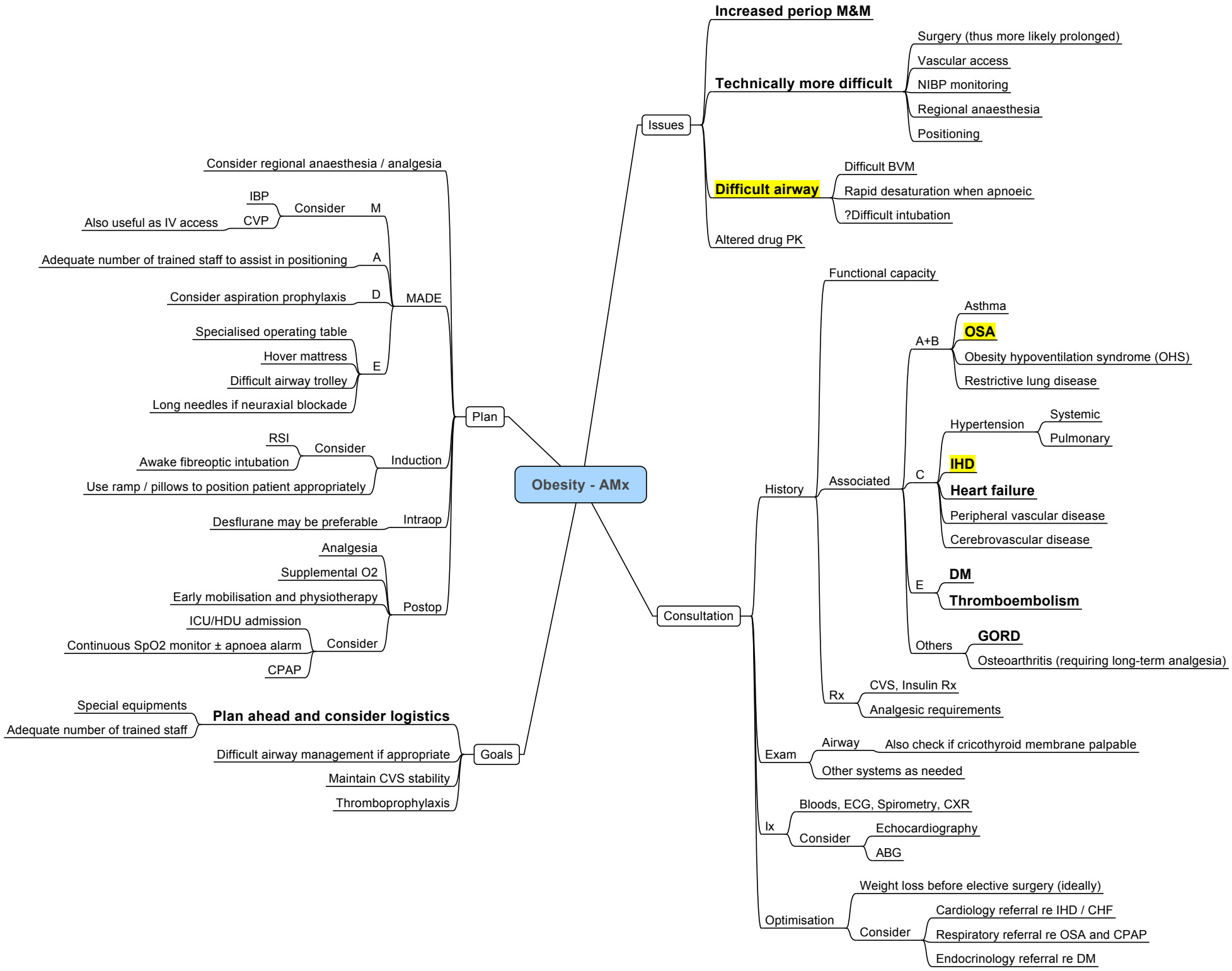

Myasthenia crisis
vs
Cholinergic crisis
Background
May be difficult to distinguish
Difference in MOA
Cholinergic crisis
Too much ACh
... from anticholinesterase overdose
Myasthenia crisis
Exacerbation of myasthenia gravis
Insufficient anticholinesterase
S&S
Muscle fasciculation and severe weakness
Sweating and salivation
Pallor
Bradycardia
Constricted pupils
Diagnosis
Distinguish by giving edrophonium 2 mg IV
Cholinergic crisis will get worse
Myasthenia crisis will get better
Rx
Cholinergic crisis
Antimuscarinic drugs
i.e. atropine
Respiratory support
Myasthenia crisis
Respiratory support if needed
Increase anticholinesterase therapy
Also consider
Plasmapheresis
IV immunoglobulin

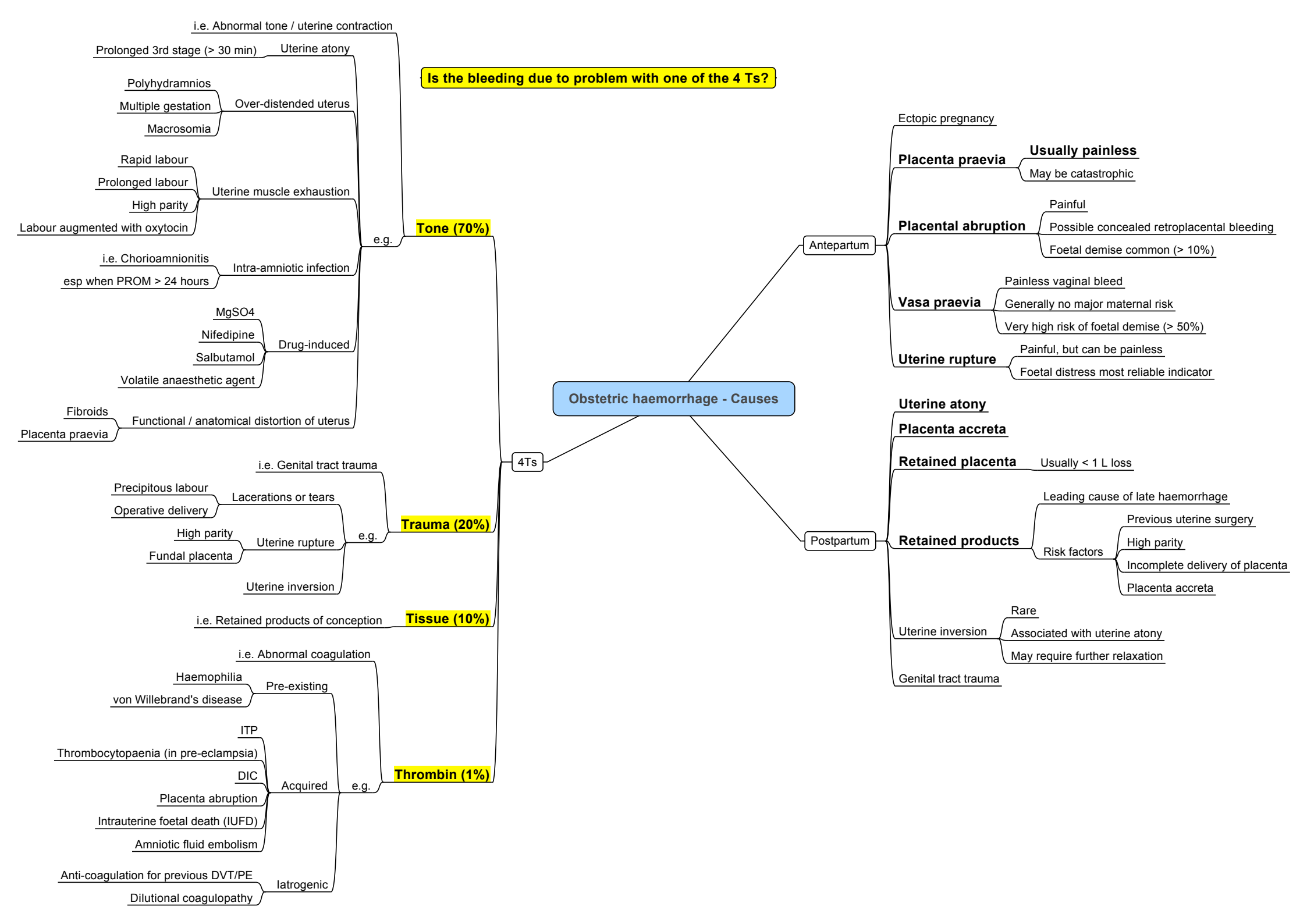

Myasthenia gravis - AMx
Consultation
Hx
Myasthenia gravis
Duration of disease
Respiratory impairment
Bulbar signs
e.g.
Dysphagia
Dysarthria
Needs periop airway protection
Other Hx
Resp
Coexisting respiratory disease
CVS
Cardiomyopathy
Arrhythmia
Autoimmune
e.g. SLE, RA
Thymoma
Occurs in 15%
Anterior mediastinal mass
Risk of airway / vascular obstruction
Rx
Anticholinesterase
e.g. pyridostigmine
Dose and time of intake
Effects of drug omission?
Plasmapheresis
Depletes plasma esterase
Prolonged action
Suxamethonium
Mivacurium
Ester LA
Remifentanil
Esmolol
Immunosuppressant
Cytotoxic precaution
Steroid
Perioperative steroid cover
Ix
ECG
Respiratory function test
CXR
Rule out compressive anterior mediastinal mass
Optimisation
Coach patient re extubation
If poorly controled
Pre-operative plasmapheresis
Consider delaying for Rx optimisation
?Thymectomy
Issues
Periop respiratory impairment
May require postop ventilation
Aspiration risk if bulbar involvement
NMBDs
Altered response to NMBDs
Reversal may trigger cholinergic crisis
Risk of
Cholinergic crisis
Anticholinesterase overdose
Myasthenia crisis
Inadequate anticholinesterase
Others
Predictors of need for postop ventilation
Major body cavity surgery
Bulbar involvement
Duration of disease > 6 years
Co-existing respiratory disease
Pyridostigmine > 750 mg/day
Vital capacity < 2.9L
NMBDs
May be very sensitive to non-depolarising NMBDs
Due to overdose of anticholinesterase
Reversal may trigger cholinergic crisis
Suxamethonium
Larger dosage needed (1.5 to 2 mg/kg)
Decreased depolarisation due to fewer receptors
Effect may last longer
Increased risk of phase II block
Plan
Ensure postop ventilation facility is available before proceeding
MADE
M
Nerve stimulator if NMBD used
D
Avoid premeds
Intraop
Insertion of NGT
Allows administration of oral medications
Emergence
Reversals
Preferably avoid
Standard dose if good preop control
Sugammadex if appropriate
Extubation
Only if
Fully awake
Adequate muscle strength
Safe extubation when head life > 5 seconds
Postop
Monitor in HDU
Possible delayed respiratory impairment
... even if initially strong postop
Consider
PO Rx by NGT
Give anticholinesterase IV
Resume medication ASAP
Goals
Anticholinesterase Rx
"Weak at intubation, strong at extubation"
Delay anticholinesterase Rx until entry to OT
Minimise need for NMBDs for intubation
Airway protection / ventilatory support as needed
Use of NMBDs
Avoid if possible
Non-depolarising NMBDs
Start with 10% of dose
Avoid long acting NMBDs
Alternatives
Deep volatile / propofol
LA spray to vocal cords

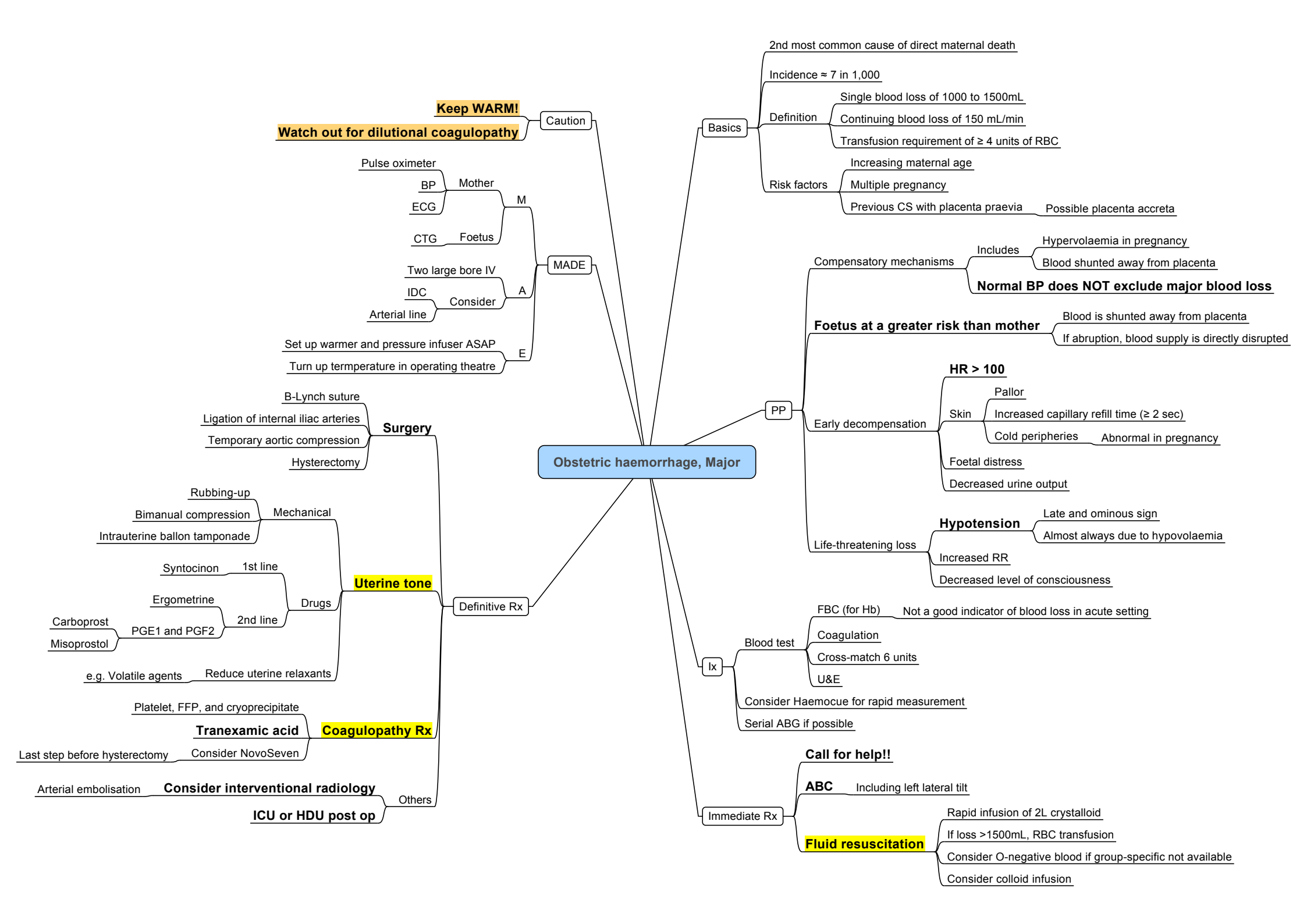

Myasthenic syndrome
Basics
aka
Lambert-Eaton myasthenic syndrome (LEMS)
Eaton-Lambert syndrome
Rare autoimmune disorder
60% have underlying malignancy
Usually small cell lung cancer
Comparison
Myasthenic syndrome
Prejunctional failure
Not enough ACh released
S&S
Muscle weakness affects limbs more
esp proximal muscles
Strength improves after sustained contraction
Lambert's sign
Gait difficulty before eye signs
Eyes and bulblar muscles less frequently involved
Autonomic dysfunction (75%)
e.g.
Postural hypotension
Impaired sweating
Dry mouth
Drug response
Sensitive to suxamethonium
Poor response to anticholinesterase
Myasthenia gravis
Not enough ACh receptors
Eyes and bulbar muscles more frequency involved
Drug response
Resistant to suxamethonium
Good response to anticholinesterase

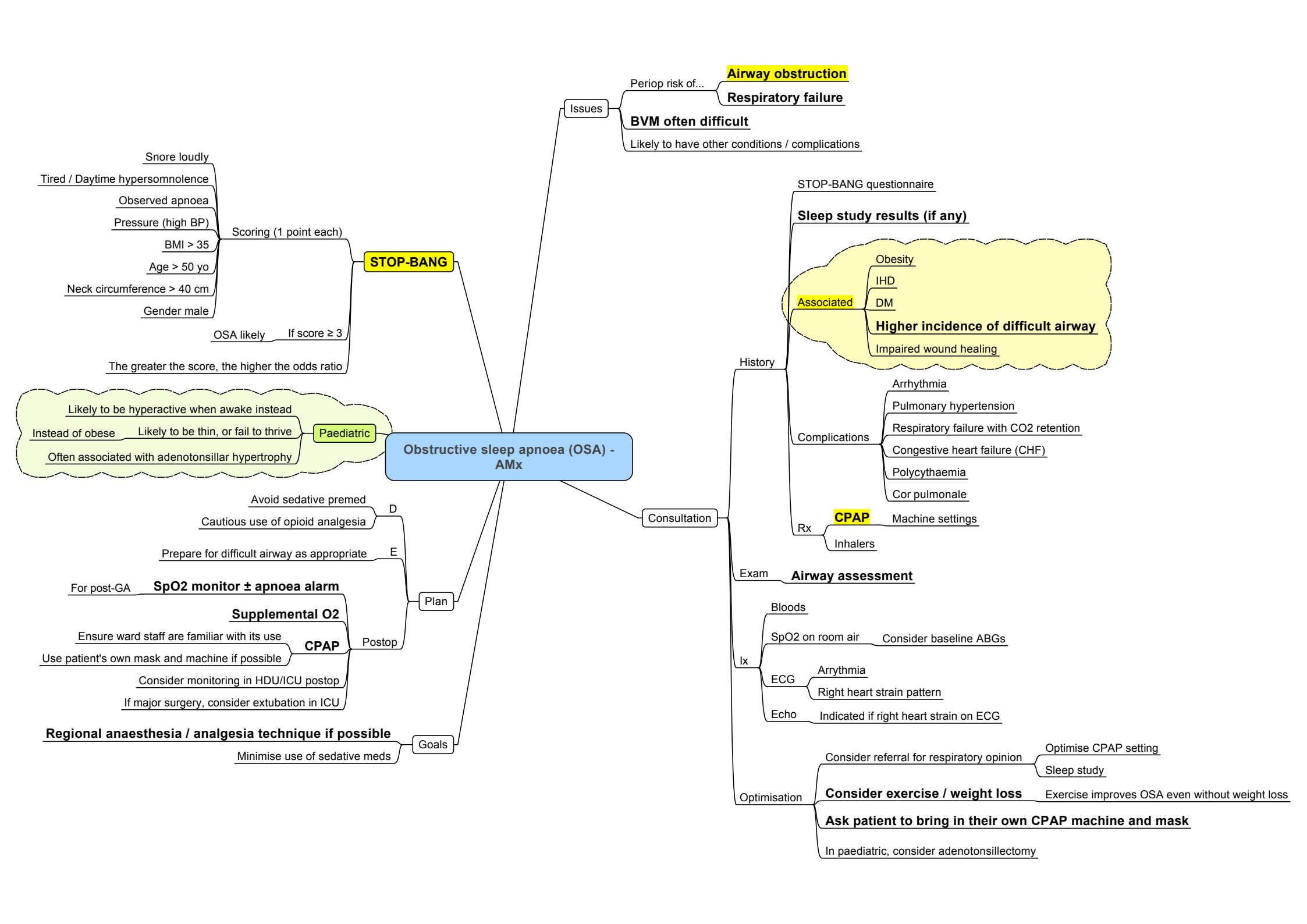

Myotonic dystrophy
Background
The commonest myotonia disorder
aka
Myotonia atrophica
Dystrophia myotonica (DM)
PP
MOA
Autosomal dominant
Progressive deterioration of
Skeletal muscles
Cardiac muscles
Smooth muscles
Normal peripheral nerve and NMJ
S&S
Myotonia
Continued involuntary muscle contractions
... after cessation of effort
Muscular atrophy / weakness
Facial
Sternomastoid
Peripheral muscles
Frontal balding
Cataracts
Issues
A+B
Aspiration risk
Bulbar involvement
Delayed gastric emptying
Respiratory muscle weakness
Sleep apnoea (central and obstructive)
C
Cardiomyopathy
Conduction defects
Dysrhythmia
AV block
Mitral valve prolapse (20%)
D
Mild cognitive impairment
E
Glucose intolerance / Diabetes mellitus
Hypogonadism
Hypothyroidism
Adrenal insufficiency
Increased risk of post-partum haemorrhage
Goals
Aspiration prophylaxis
Prevents postop shivering
Normothermia
Minimise
Myocardial depressant drugs
Respiratory depressant drugs
Sedative drugs
Plan
M
IBP
Temp
Induction
Prolonged contraction
Avoid suxamethonium
Increased K+ efflux
Cautious dosing of induction agents
Postop
Monitor in HDU
Others
Myotonia triggers
Suxamethonium
Not usually a trigger
Neostigmine
Shivering
Nerve stimulator
Regional anaesthesia does NOT prevent muscle contraction

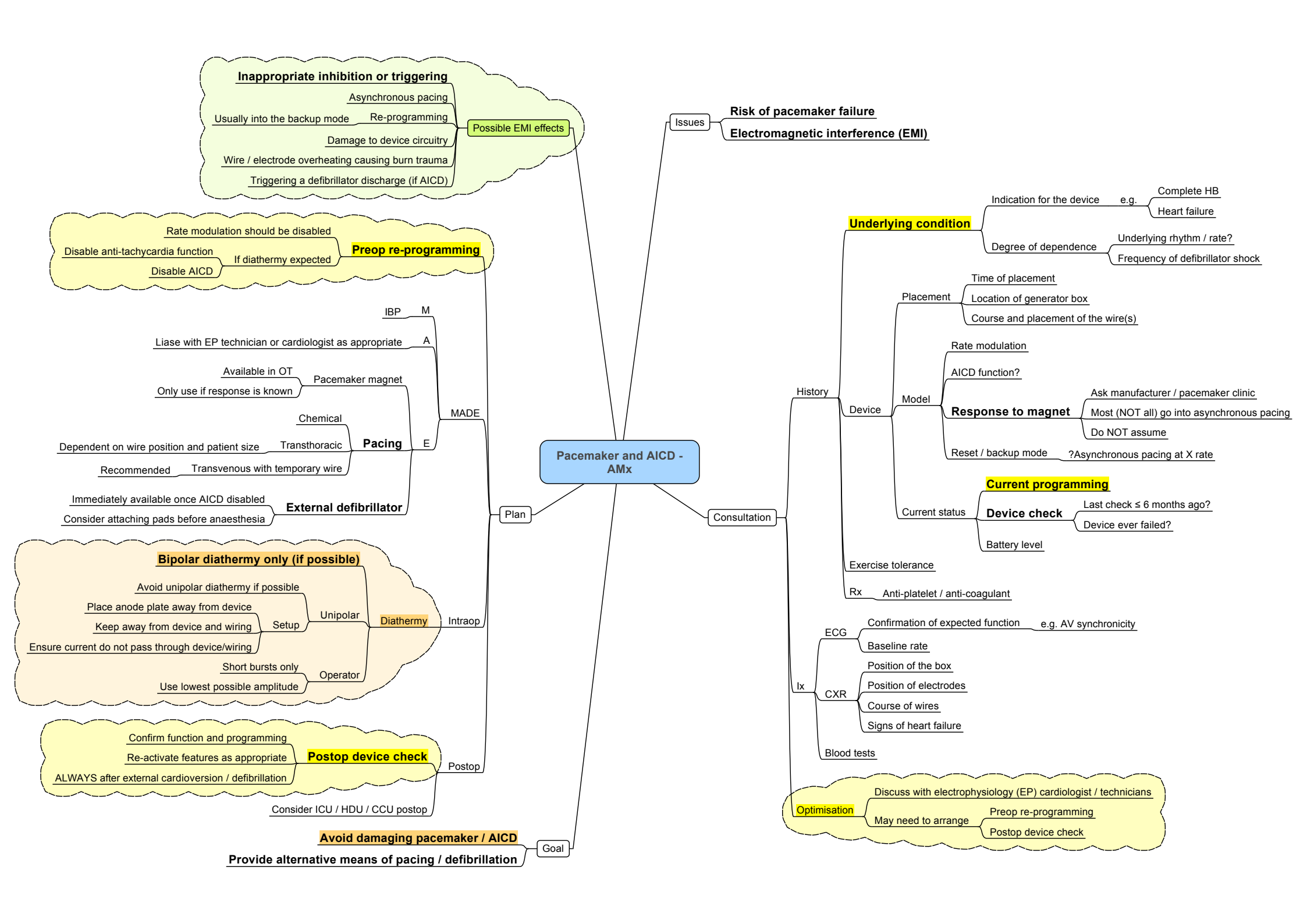

NICE-SUGAR trial
Background
Normoglycaemia in Intensive Care Evaluation - Survival Using Glucose Algorithm Regulation
Published
NEJM in 2009
NEJM in 2012
Post hoc analysis
Looks at risk of death vs moderate / severe hypoglycaemia
Question: Does intensive glucose control reduce mortality at 90 days?
Method
Prospective RCT
Multicentered
Australia
New Zealand
Canada
Single blind
6104 randomised
Inclusion criteria
Expected to be in ICU for at least 3 days
Groups
Intensive control group
BSL target: 4.5 to 6 mmol/L
Conventional control group
BSL target: < 10 mmol/L
Insulin infusion used to control BSL
Outcome measured
Primary
Death at 90 days after randomisation
Secondary
Including
Survival time during first 90 days
Causes of death
ICU and hospital stay
Renal replacement therapy
ICU and hospital stay
Tertary outcome
including
Incidence of new organ failure
Adverse events
Severe hypoglycaemia (BSL < 2.2 mmol/dL)
Moderate hypoglycaemia (BSL 2.3 to 3.9 mmol/L)
Pre-defined subgroups
Operative (or not)
DM (or not)
Trauma (or not)
Severe sepsis (or not)
Corticosteroid (or not)
APACHE II ≥ 25 (or not)
Design
90% power to detect an absolute difference of 3.8%
2-sided alpha of < 0.05
Baseline mortality of 30% assumed
Findings
Summary
1. Moderate or severe hypoglycaemia are associated increased mortality
2. Intensive control group has a lot more moderate or severe hypoglycaemia
3. Therefore more deaths in intensive control group
Adverse events
Intensive control
Mortality at 90 days higher
27.5% (vs 24.9%)
OR = 1.14 (1.02 to 1.28)
p = 0.02
Death more likely CVS-related
41.6% (vs 35.8%)
p = 0.02
Severe hypoglycaemia more likely
6.8% (vs 0.5%)
OR = 14.7 (9 to 25.9)
p < 0.001
Trend towards lower mortality
Trauma patients
OR = 0.77 (0.50 to 1.18)
Corticosteroid Rx
OR = 0.88 (0.61 to 1.19)
Others
Time-weighted BSL is lower
6.4 ± 1 mmol/L (vs 8 ± 1.3 mmol/L)
More likely to receive insulin
97.2% (vs 69%)
Larger dose of insulin received
50.2 ± 38.1 IU/day (vs 16.9 ± 29)
No difference in other outcomes
Post hoc
Moderate or severe hypoglycaemia associated with increased mortality
Intensive control group
82.4% of moderate hypoglycaemic cases
93.3% of severe hypoglycaemic cases
Hazard ratio for death
Moderate hypoglycaemia
HR = 1.41 (1.21 to 1.61)
p < 0.001
Severe hypoglycaemia
HR = 2.10 (1.59 to 2.77)
p < 0.001
HR even higher in postop patients
Distributive shock mostly strongly associated with hypoglycaemia
Limitation
Use of subjective inclusion criteria
Inability to blind treating staff and study personnel
BSL in intensive control group was a little above target range
Higher withdrawal rate in intensive control group
10% (vs 7.4%)
This study does NOT mean hypoglycaemia causes death (cannot prove causal relationship)
Others
More patients in intensive group received corticosteroid before randomisation
34.6% (vs 31.7%)
Relation between hypoglycaemia and death not significantly different between treatment groups
In patients with NO hypoglycaemia
Higher mortality in conventional group than intensive control group
23.7% (vs 22.5%)

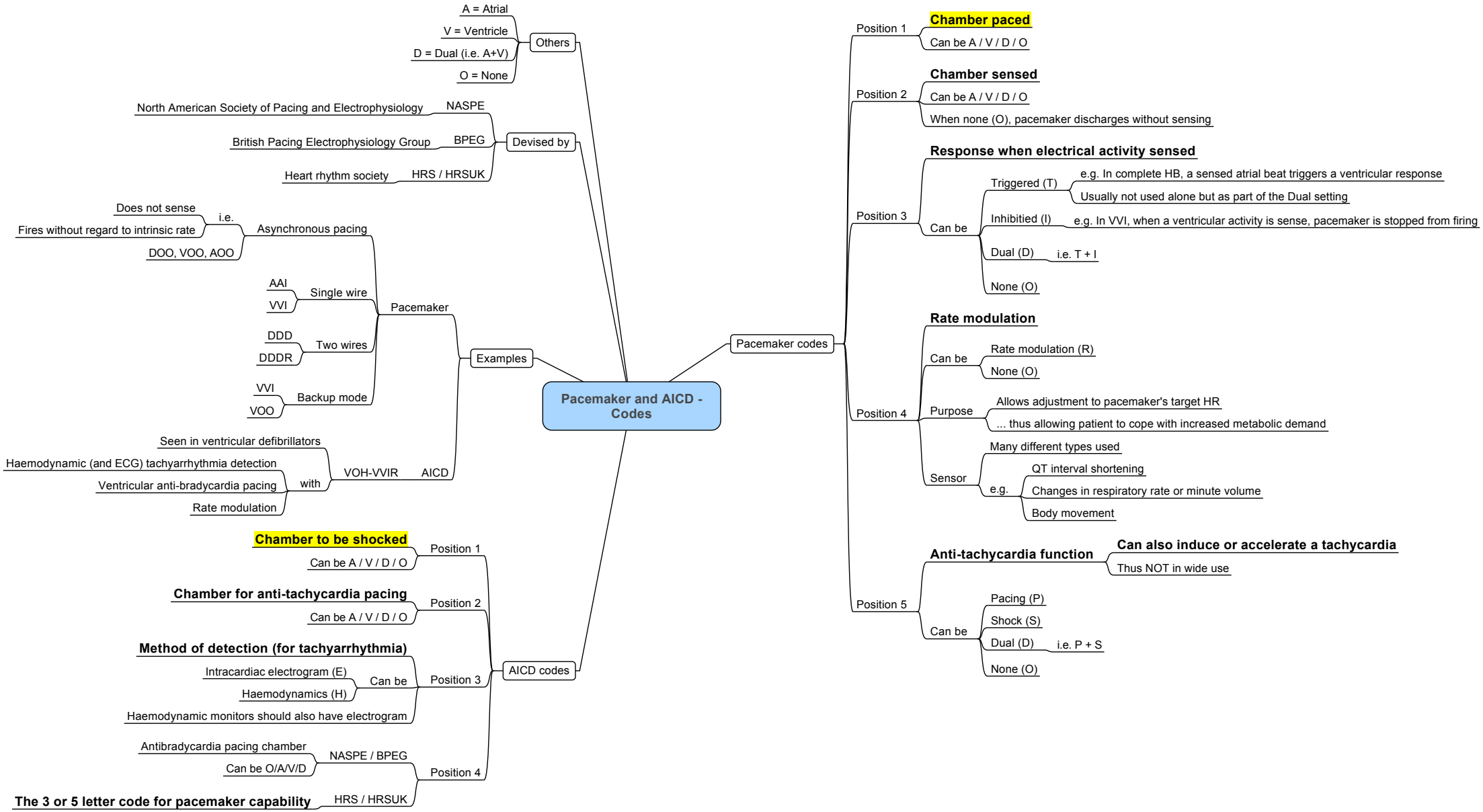

Obesity - AMx
Consultation
History
Functional capacity
Associated
A+B
Asthma
OSA
Obesity hypoventilation syndrome (OHS)
Restrictive lung disease
C
Hypertension
Systemic
Pulmonary
IHD
Heart failure
Peripheral vascular disease
Cerebrovascular disease
E
DM
Thromboembolism
Others
GORD
Osteoarthritis (requiring long-term analgesia)
Rx
CVS, Insulin Rx
Analgesic requirements
Exam
Airway
Also check if cricothyroid membrane palpable
Other systems as needed
Ix
Bloods, ECG, Spirometry, CXR
Consider
Echocardiography
ABG
Optimisation
Weight loss before elective surgery (ideally)
Consider
Cardiology referral re IHD / CHF
Respiratory referral re OSA and CPAP
Endocrinology referral re DM
Issues
Increased periop M&M
Technically more difficult
Surgery (thus more likely prolonged)
Vascular access
NIBP monitoring
Regional anaesthesia
Positioning
Difficult airway
Difficult BVM
Rapid desaturation when apnoeic
?Difficult intubation
Altered drug PK
Plan
MADE
M
Consider
IBP
CVP
Also useful as IV access
A
Adequate number of trained staff to assist in positioning
D
Consider aspiration prophylaxis
E
Specialised operating table
Hover mattress
Difficult airway trolley
Long needles if neuraxial blockade
Induction
Consider
RSI
Awake fibreoptic intubation
Use ramp / pillows to position patient appropriately
Intraop
Desflurane may be preferable
Postop
Analgesia
Supplemental O2
Early mobilisation and physiotherapy
Consider
ICU/HDU admission
Continuous SpO2 monitor ± apnoea alarm
CPAP
Consider regional anaesthesia / analgesia
Goals
Plan ahead and consider logistics
Special equipments
Adequate number of trained staff
Difficult airway management if appropriate
Maintain CVS stability
Thromboprophylaxis

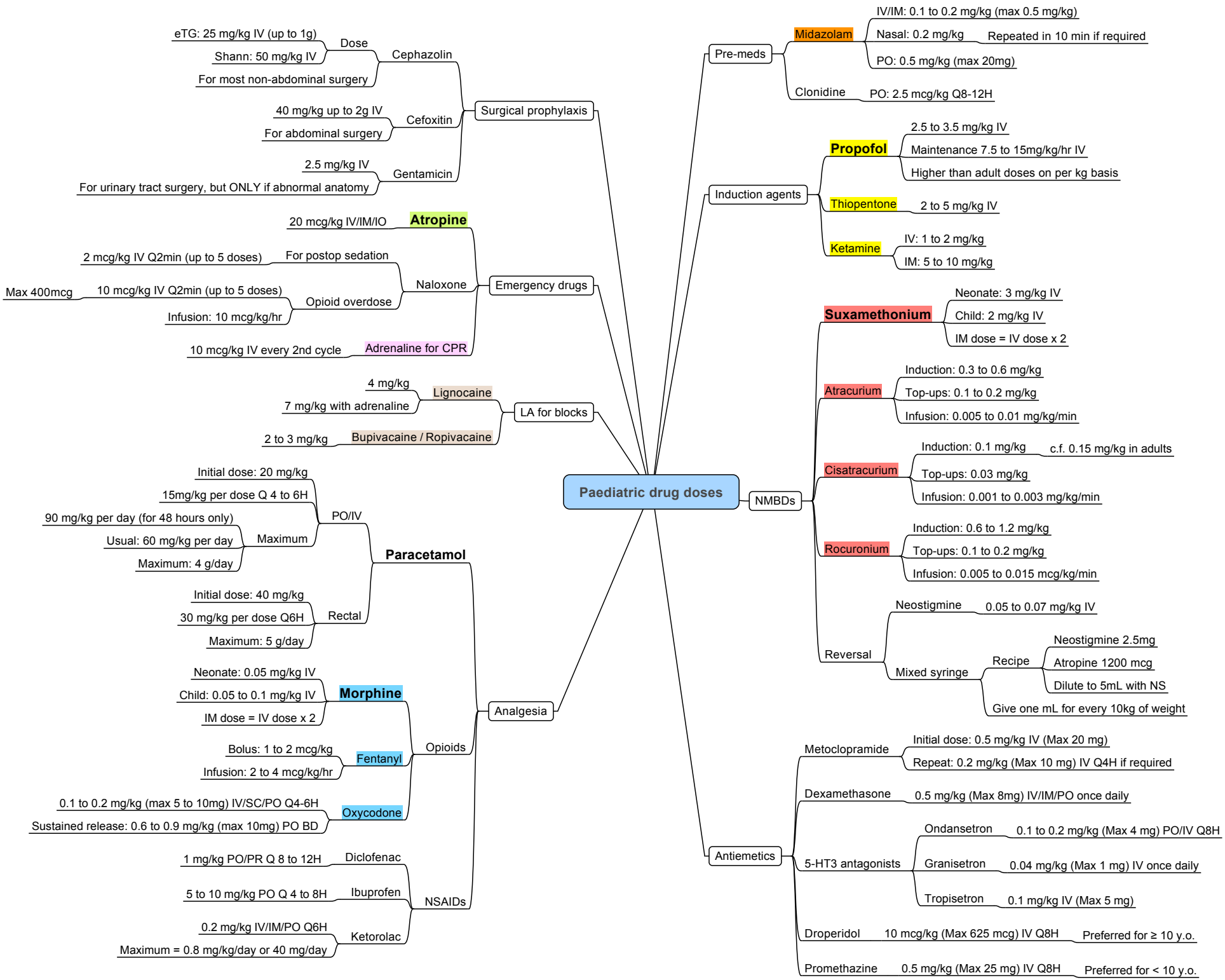

Obstetric haemorrhage - Causes
Is the bleeding due to problem with one of the 4 Ts?
4Ts
Tone (70%)
e.g.
i.e. Abnormal tone / uterine contraction
Uterine atony
Prolonged 3rd stage (> 30 min)
Over-distended uterus
Polyhydramnios
Multiple gestation
Macrosomia
Uterine muscle exhaustion
Rapid labour
Prolonged labour
High parity
Labour augmented with oxytocin
Intra-amniotic infection
i.e. Chorioamnionitis
esp when PROM > 24 hours
Drug-induced
MgSO4
Nifedipine
Salbutamol
Volatile anaesthetic agent
Functional / anatomical distortion of uterus
Fibroids
Placenta praevia
Trauma (20%)
e.g.
i.e. Genital tract trauma
Lacerations or tears
Precipitous labour
Operative delivery
Uterine rupture
High parity
Fundal placenta
Uterine inversion
Tissue (10%)
i.e. Retained products of conception
Thrombin (1%)
e.g.
i.e. Abnormal coagulation
Pre-existing
Haemophilia
von Willebrand's disease
Acquired
ITP
Thrombocytopaenia (in pre-eclampsia)
DIC
Placenta abruption
Intrauterine foetal death (IUFD)
Amniotic fluid embolism
Iatrogenic
Anti-coagulation for previous DVT/PE
Dilutional coagulopathy
Antepartum
Ectopic pregnancy
Placenta praevia
Usually painless
May be catastrophic
Placental abruption
Painful
Possible concealed retroplacental bleeding
Foetal demise common (> 10%)
Vasa praevia
Painless vaginal bleed
Generally no major maternal risk
Very high risk of foetal demise (> 50%)
Uterine rupture
Painful, but can be painless
Foetal distress most reliable indicator
Postpartum
Uterine atony
Placenta accreta
Retained placenta
Usually < 1 L loss
Retained products
Leading cause of late haemorrhage
Risk factors
Previous uterine surgery
High parity
Incomplete delivery of placenta
Placenta accreta
Uterine inversion
Rare
Associated with uterine atony
May require further relaxation
Genital tract trauma

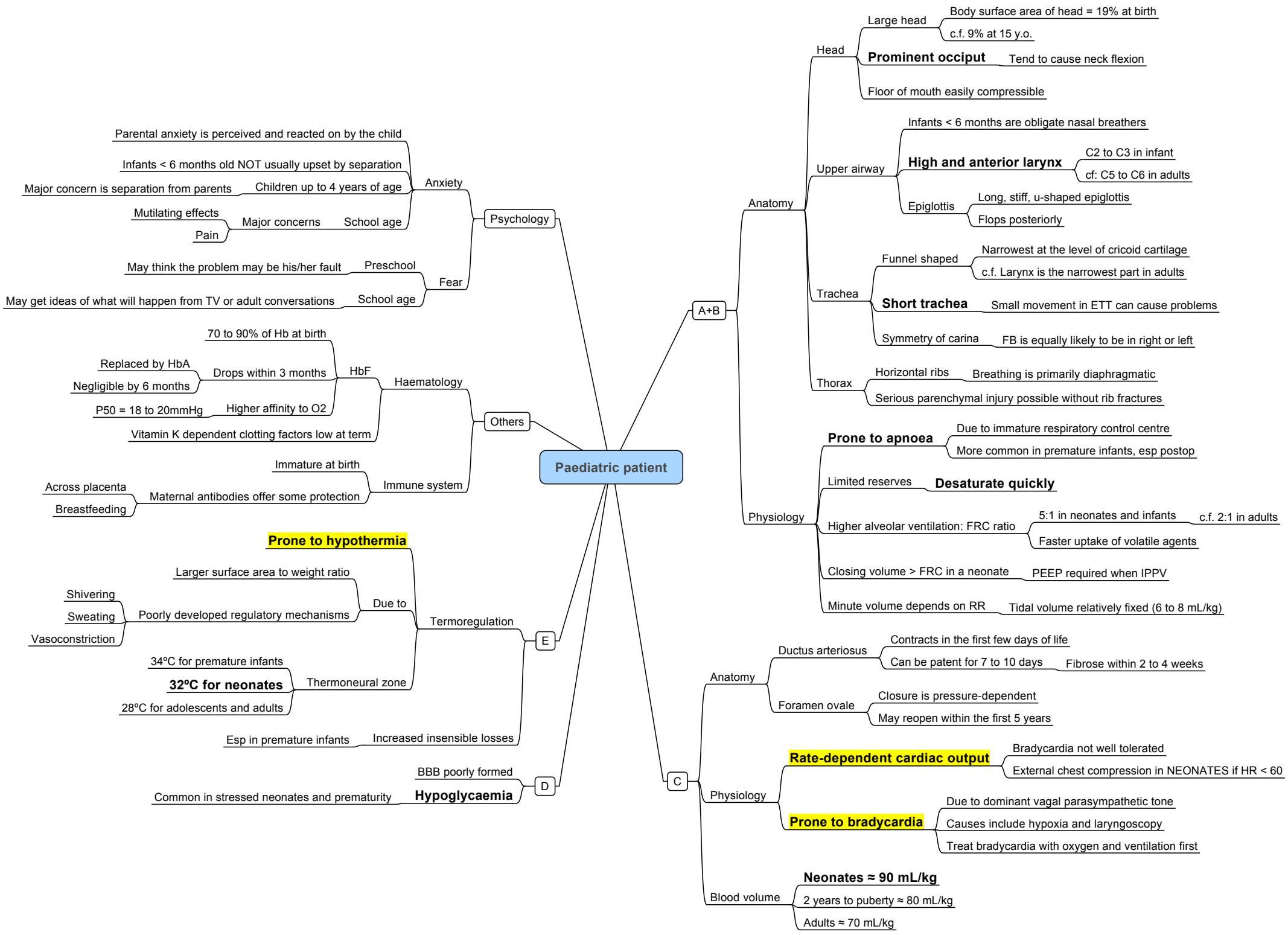

Obstetric haemorrhage, Major
Basics
2nd most common cause of direct maternal death
Incidence ≈ 7 in 1,000
Definition
Single blood loss of 1000 to 1500mL
Continuing blood loss of 150 mL/min
Transfusion requirement of ≥ 4 units of RBC
Risk factors
Increasing maternal age
Multiple pregnancy
Previous CS with placenta praevia
Possible placenta accreta
PP
Compensatory mechanisms
Includes
Hypervolaemia in pregnancy
Blood shunted away from placenta
Normal BP does NOT exclude major blood loss
Foetus at a greater risk than mother
Blood is shunted away from placenta
If abruption, blood supply is directly disrupted
Early decompensation
HR > 100
Skin
Pallor
Increased capillary refill time (≥ 2 sec)
Cold peripheries
Abnormal in pregnancy
Foetal distress
Decreased urine output
Life-threatening loss
Hypotension
Late and ominous sign
Almost always due to hypovolaemia
Increased RR
Decreased level of consciousness
Ix
Blood test
FBC (for Hb)
Not a good indicator of blood loss in acute setting
Coagulation
Cross-match 6 units
U&E
Consider Haemocue for rapid measurement
Serial ABG if possible
Immediate Rx
Call for help!!
ABC
Including left lateral tilt
Fluid resuscitation
Rapid infusion of 2L crystalloid
If loss >1500mL, RBC transfusion
Consider O-negative blood if group-specific not available
Consider colloid infusion
Definitive Rx
Caution
Keep WARM!
Watch out for dilutional coagulopathy
MADE
M
Mother
Pulse oximeter
BP
ECG
Foetus
CTG
A
Consider
Two large bore IV
IDC
Arterial line
E
Set up warmer and pressure infuser ASAP
Turn up termperature in operating theatre
Surgery
B-Lynch suture
Ligation of internal iliac arteries
Temporary aortic compression
Hysterectomy
Uterine tone
Mechanical
Rubbing-up
Bimanual compression
Intrauterine ballon tamponade
Drugs
1st line
Syntocinon
2nd line
Ergometrine
PGE1 and PGF2
Carboprost
Misoprostol
Reduce uterine relaxants
e.g. Volatile agents
Coagulopathy Rx
Platelet, FFP, and cryoprecipitate
Tranexamic acid
Consider NovoSeven
Last step before hysterectomy
Others
Consider interventional radiology
Arterial embolisation
ICU or HDU post op

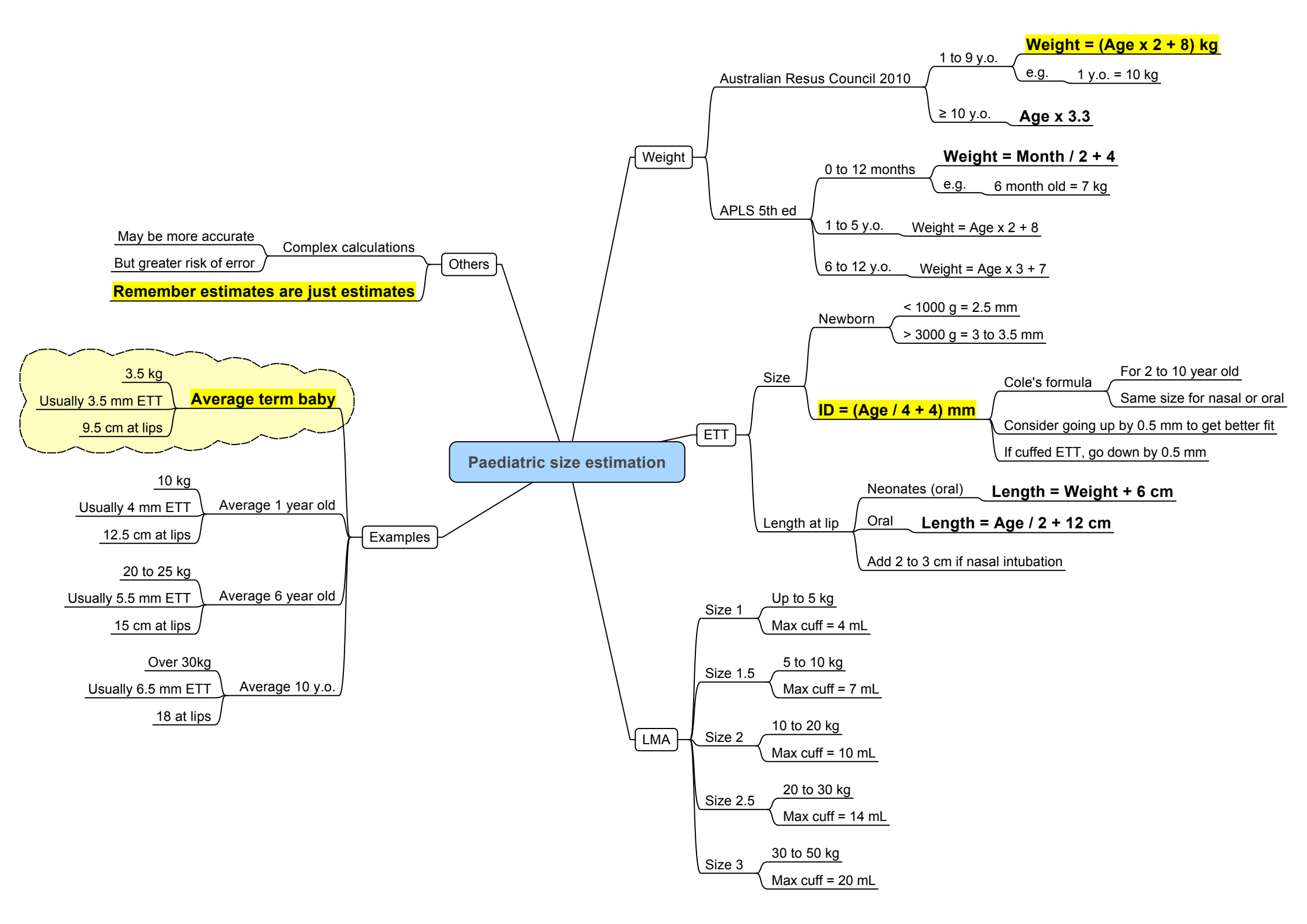

Obstructive sleep apnoea (OSA) - AMx
Issues
Periop risk of...
Airway obstruction
Respiratory failure
BVM often difficult
Likely to have other conditions / complications
STOP-BANG
Scoring (1 point each)
Snore loudly
Tired / Daytime hypersomnolence
Observed apnoea
Pressure (high BP)
BMI > 35
Age > 50 yo
Neck circumference > 40 cm
Gender male
OSA likely
If score ≥ 3
The greater the score, the higher the odds ratio
Paediatric
Likely to be hyperactive when awake instead
Instead of obese
Likely to be thin, or fail to thrive
Often associated with adenotonsillar hypertrophy
Plan
D
Avoid sedative premed
Cautious use of opioid analgesia
E
Prepare for difficult airway as appropriate
Postop
For post-GA
SpO2 monitor ± apnoea alarm
Supplemental O2
CPAP
Ensure ward staff are familiar with its use
Use patient's own mask and machine if possible
Consider monitoring in HDU/ICU postop
If major surgery, consider extubation in ICU
Goals
Regional anaesthesia / analgesia technique if possible
Minimise use of sedative meds
Consultation
History
STOP-BANG questionnaire
Sleep study results (if any)
Associated
Obesity
IHD
DM
Higher incidence of difficult airway
Impaired wound healing
Complications
Arrhythmia
Pulmonary hypertension
Respiratory failure with CO2 retention
Congestive heart failure (CHF)
Polycythaemia
Cor pulmonale
Rx
CPAP
Machine settings
Inhalers
Exam
Airway assessment
Ix
Bloods
SpO2 on room air
Consider baseline ABGs
ECG
Arrythmia
Right heart strain pattern
Echo
Indicated if right heart strain on ECG
Optimisation
Consider referral for respiratory opinion
Optimise CPAP setting
Sleep study
Consider exercise / weight loss
Exercise improves OSA even without weight loss
Ask patient to bring in their own CPAP machine and mask
In paediatric, consider adenotonsillectomy

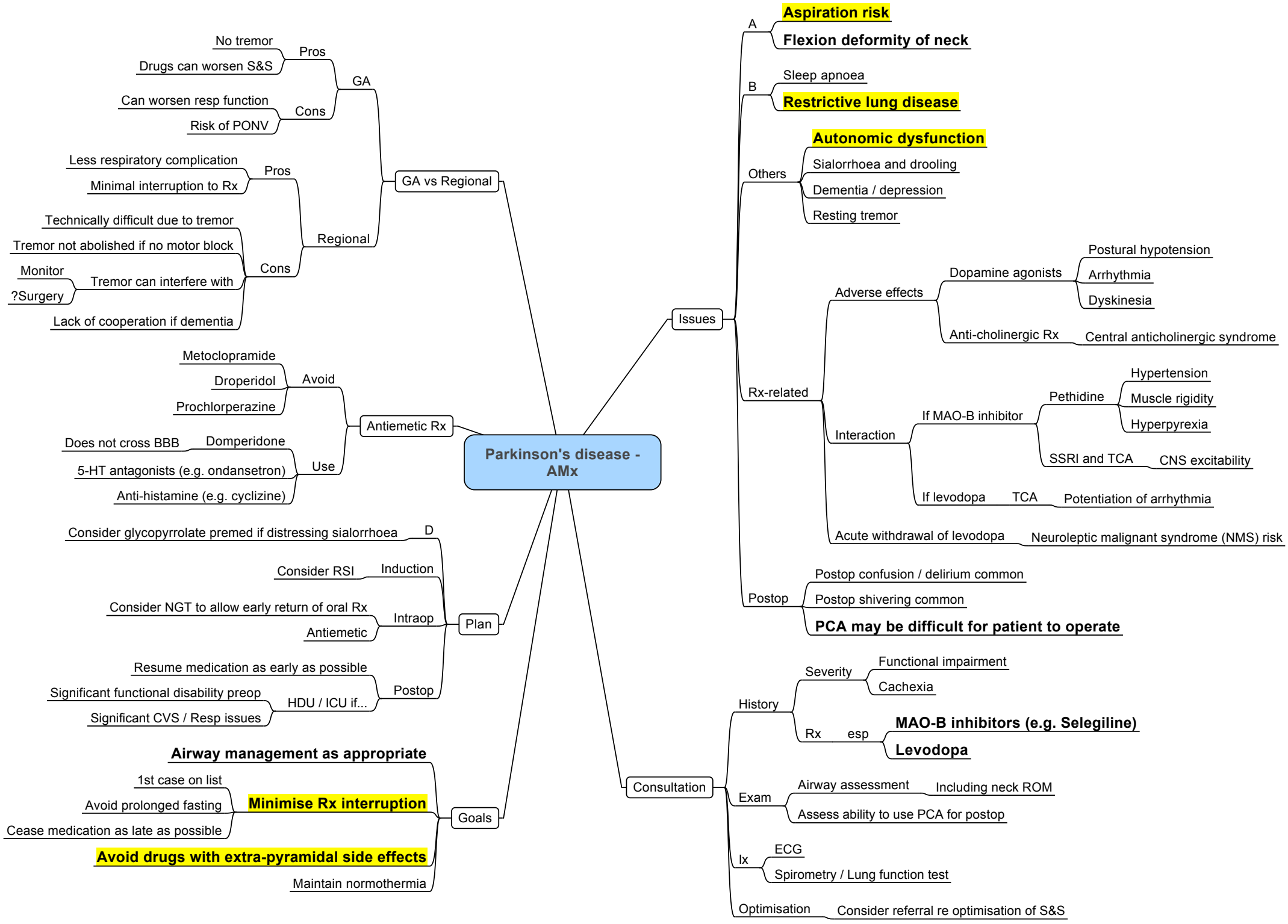

Pacemaker and AICD - AMx
Issues
Risk of pacemaker failure
Electromagnetic interference (EMI)
Possible EMI effects
Inappropriate inhibition or triggering
Asynchronous pacing
Re-programming
Usually into the backup mode
Damage to device circuitry
Wire / electrode overheating causing burn trauma
Triggering a defibrillator discharge (if AICD)
Consultation
History
Underlying condition
Indication for the device
e.g.
Complete HB
Heart failure
Degree of dependence
Underlying rhythm / rate?
Frequency of defibrillator shock
Device
Placement
Time of placement
Location of generator box
Course and placement of the wire(s)
Model
Rate modulation
AICD function?
Response to magnet
Ask manufacturer / pacemaker clinic
Most (NOT all) go into asynchronous pacing
Do NOT assume
Reset / backup mode
?Asynchronous pacing at X rate
Current status
Current programming
Device check
Last check ≤ 6 months ago?
Device ever failed?
Battery level
Exercise tolerance
Rx
Anti-platelet / anti-coagulant
Ix
ECG
Confirmation of expected function
e.g. AV synchronicity
Baseline rate
CXR
Position of the box
Position of electrodes
Course of wires
Signs of heart failure
Blood tests
Optimisation
Discuss with electrophysiology (EP) cardiologist / technicians
May need to arrange
Preop re-programming
Postop device check
Plan
Preop re-programming
Rate modulation should be disabled
Disable anti-tachycardia function
If diathermy expected
Disable AICD
MADE
M
IBP
A
Liase with EP technician or cardiologist as appropriate
E
Pacemaker magnet
Available in OT
Only use if response is known
Pacing
Chemical
Transthoracic
Dependent on wire position and patient size
Transvenous with temporary wire
Recommended
External defibrillator
Immediately available once AICD disabled
Consider attaching pads before anaesthesia
Intraop
Diathermy
Bipolar diathermy only (if possible)
Unipolar
Setup
Avoid unipolar diathermy if possible
Place anode plate away from device
Keep away from device and wiring
Ensure current do not pass through device/wiring
Operator
Short bursts only
Use lowest possible amplitude
Postop
Postop device check
Confirm function and programming
Re-activate features as appropriate
ALWAYS after external cardioversion / defibrillation
Consider ICU / HDU / CCU postop
Goal
Avoid damaging pacemaker / AICD
Provide alternative means of pacing / defibrillation

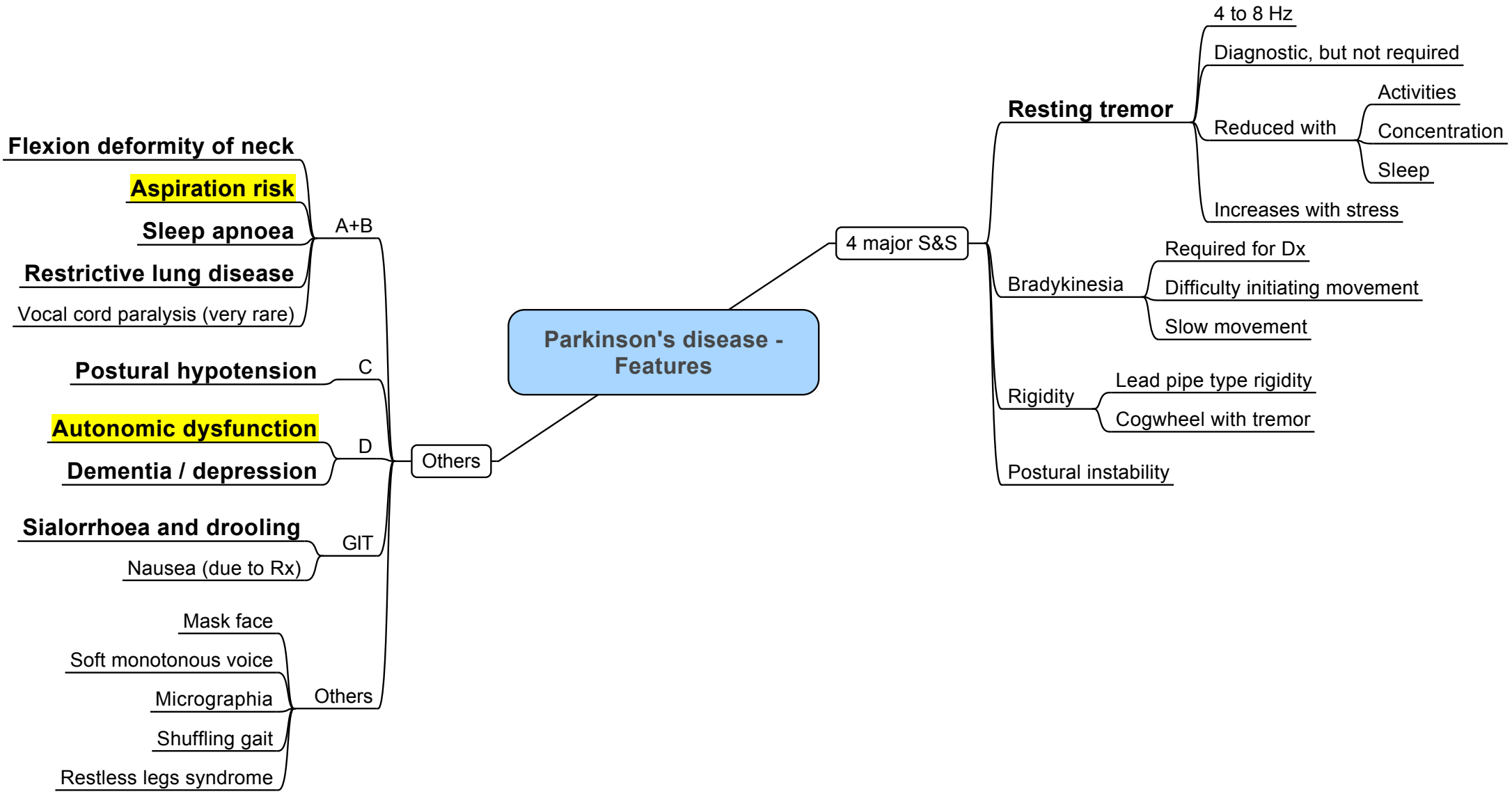

Pacemaker and AICD - Codes
Pacemaker codes
Position 1
Chamber paced
Can be A / V / D / O
Position 2
Chamber sensed
Can be A / V / D / O
When none (O), pacemaker discharges without sensing
Position 3
Response when electrical activity sensed
Can be
Triggered (T)
e.g. In complete HB, a sensed atrial beat triggers a ventricular response
Usually not used alone but as part of the Dual setting
Inhibitied (I)
e.g. In VVI, when a ventricular activity is sense, pacemaker is stopped from firing
Dual (D)
i.e. T + I
None (O)
Position 4
Rate modulation
Can be
Rate modulation (R)
None (O)
Purpose
Allows adjustment to pacemaker's target HR
... thus allowing patient to cope with increased metabolic demand
Sensor
Many different types used
e.g.
QT interval shortening
Changes in respiratory rate or minute volume
Body movement
Position 5
Anti-tachycardia function
Can also induce or accelerate a tachycardia
Thus NOT in wide use
Can be
Pacing (P)
Shock (S)
Dual (D)
i.e. P + S
None (O)
Examples
Pacemaker
Asynchronous pacing
i.e.
Does not sense
Fires without regard to intrinsic rate
DOO, VOO, AOO
Single wire
AAI
VVI
Two wires
DDD
DDDR
Backup mode
VVI
VOO
AICD
VOH-VVIR
with
Seen in ventricular defibrillators
Haemodynamic (and ECG) tachyarrhythmia detection
Ventricular anti-bradycardia pacing
Rate modulation
Others
A = Atrial
V = Ventricle
D = Dual (i.e. A+V)
O = None
Devised by
NASPE
North American Society of Pacing and Electrophysiology
BPEG
British Pacing Electrophysiology Group
HRS / HRSUK
Heart rhythm society
AICD codes
Position 1
Chamber to be shocked
Can be A / V / D / O
Position 2
Chamber for anti-tachycardia pacing
Can be A / V / D / O
Position 3
Method of detection (for tachyarrhythmia)
Can be
Intracardiac electrogram (E)
Haemodynamics (H)
Haemodynamic monitors should also have electrogram
Position 4
Antibradycardia pacing chamber
Can be O/A/V/D
NASPE / BPEG
The 3 or 5 letter code for pacemaker capability
HRS / HRSUK

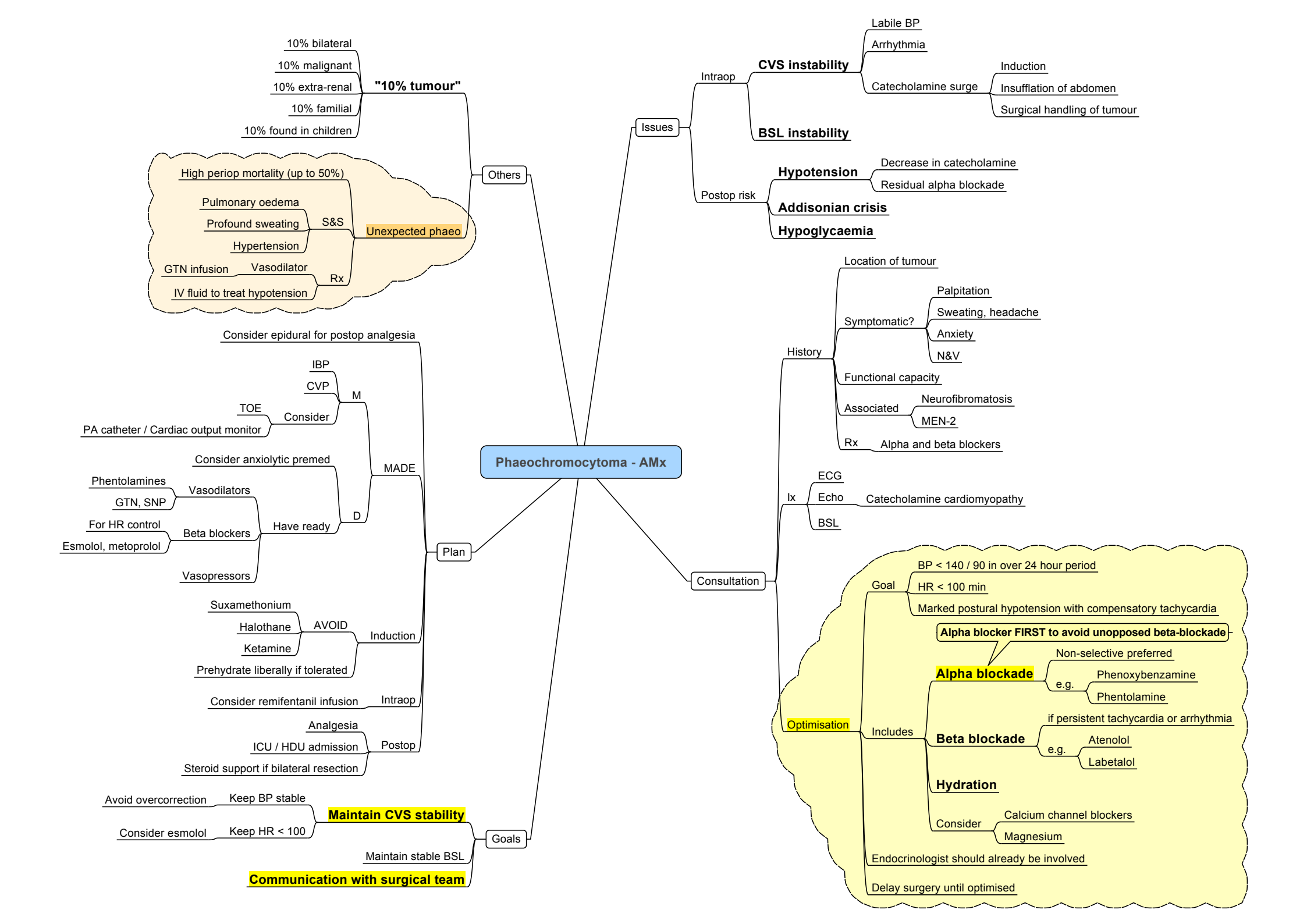

Paediatric drug doses
Pre-meds
Midazolam
IV/IM: 0.1 to 0.2 mg/kg (max 0.5 mg/kg)
Nasal: 0.2 mg/kg
Repeated in 10 min if required
PO: 0.5 mg/kg (max 20mg)
Clonidine
PO: 2.5 mcg/kg Q8-12H
Induction agents
Propofol
2.5 to 3.5 mg/kg IV
Maintenance 7.5 to 15mg/kg/hr IV
Higher than adult doses on per kg basis
Thiopentone
2 to 5 mg/kg IV
Ketamine
IV: 1 to 2 mg/kg
IM: 5 to 10 mg/kg
NMBDs
Suxamethonium
Neonate: 3 mg/kg IV
Child: 2 mg/kg IV
IM dose = IV dose x 2
Atracurium
Induction: 0.3 to 0.6 mg/kg
Top-ups: 0.1 to 0.2 mg/kg
Infusion: 0.005 to 0.01 mg/kg/min
Cisatracurium
Induction: 0.1 mg/kg
c.f. 0.15 mg/kg in adults
Top-ups: 0.03 mg/kg
Infusion: 0.001 to 0.003 mg/kg/min
Rocuronium
Induction: 0.6 to 1.2 mg/kg
Top-ups: 0.1 to 0.2 mg/kg
Infusion: 0.005 to 0.015 mcg/kg/min
Reversal
Neostigmine
0.05 to 0.07 mg/kg IV
Mixed syringe
Recipe
Neostigmine 2.5mg
Atropine 1200 mcg
Dilute to 5mL with NS
Give one mL for every 10kg of weight
Antiemetics
Metoclopramide
Initial dose: 0.5 mg/kg IV (Max 20 mg)
Repeat: 0.2 mg/kg (Max 10 mg) IV Q4H if required
Dexamethasone
0.5 mg/kg (Max 8mg) IV/IM/PO once daily
5-HT3 antagonists
Ondansetron
0.1 to 0.2 mg/kg (Max 4 mg) PO/IV Q8H
Granisetron
0.04 mg/kg (Max 1 mg) IV once daily
Tropisetron
0.1 mg/kg IV (Max 5 mg)
Droperidol
10 mcg/kg (Max 625 mcg) IV Q8H
Preferred for ≥ 10 y.o.
Promethazine
0.5 mg/kg (Max 25 mg) IV Q8H
Preferred for < 10 y.o.
Analgesia
Paracetamol
PO/IV
Initial dose: 20 mg/kg
15mg/kg per dose Q 4 to 6H
Maximum
90 mg/kg per day (for 48 hours only)
Usual: 60 mg/kg per day
Maximum: 4 g/day
Rectal
Initial dose: 40 mg/kg
30 mg/kg per dose Q6H
Maximum: 5 g/day
Opioids
Morphine
Neonate: 0.05 mg/kg IV
Child: 0.05 to 0.1 mg/kg IV
IM dose = IV dose x 2
Fentanyl
Bolus: 1 to 2 mcg/kg
Infusion: 2 to 4 mcg/kg/hr
Oxycodone
0.1 to 0.2 mg/kg (max 5 to 10mg) IV/SC/PO Q4-6H
Sustained release: 0.6 to 0.9 mg/kg (max 10mg) PO BD
NSAIDs
Diclofenac
1 mg/kg PO/PR Q 8 to 12H
Ibuprofen
5 to 10 mg/kg PO Q 4 to 8H
Ketorolac
0.2 mg/kg IV/IM/PO Q6H
Maximum = 0.8 mg/kg/day or 40 mg/day
Emergency drugs
Atropine
20 mcg/kg IV/IM/IO
Naloxone
For postop sedation
2 mcg/kg IV Q2min (up to 5 doses)
Opioid overdose
Max 400mcg
10 mcg/kg IV Q2min (up to 5 doses)
Infusion: 10 mcg/kg/hr
Adrenaline for CPR
10 mcg/kg IV every 2nd cycle
LA for blocks
Lignocaine
4 mg/kg
7 mg/kg with adrenaline
Bupivacaine / Ropivacaine
2 to 3 mg/kg
Surgical prophylaxis
Cephazolin
Dose
eTG: 25 mg/kg IV (up to 1g)
Shann: 50 mg/kg IV
For most non-abdominal surgery
Cefoxitin
40 mg/kg up to 2g IV
For abdominal surgery
Gentamicin
2.5 mg/kg IV
For urinary tract surgery, but ONLY if abnormal anatomy

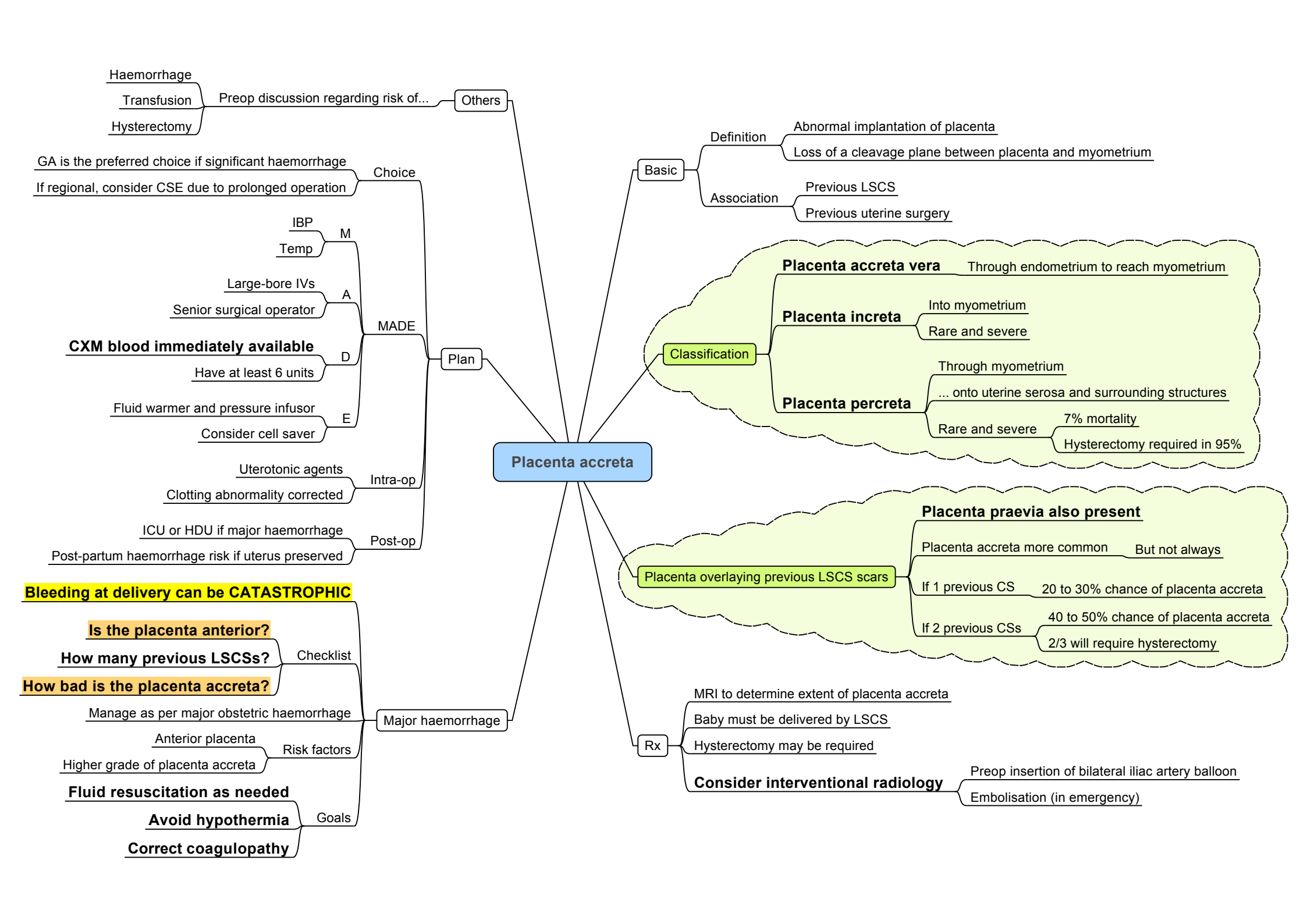

Paediatric patient
A+B
Anatomy
Head
Large head
Body surface area of head = 19% at birth
c.f. 9% at 15 y.o.
Prominent occiput
Tend to cause neck flexion
Floor of mouth easily compressible
Upper airway
Infants < 6 months are obligate nasal breathers
High and anterior larynx
C2 to C3 in infant
cf: C5 to C6 in adults
Epiglottis
Long, stiff, u-shaped epiglottis
Flops posteriorly
Trachea
Funnel shaped
Narrowest at the level of cricoid cartilage
c.f. Larynx is the narrowest part in adults
Short trachea
Small movement in ETT can cause problems
Symmetry of carina
FB is equally likely to be in right or left
Thorax
Horizontal ribs
Breathing is primarily diaphragmatic
Serious parenchymal injury possible without rib fractures
Physiology
Prone to apnoea
Due to immature respiratory control centre
More common in premature infants, esp postop
Limited reserves
Desaturate quickly
Higher alveolar ventilation: FRC ratio
5:1 in neonates and infants
c.f. 2:1 in adults
Faster uptake of volatile agents
Closing volume > FRC in a neonate
PEEP required when IPPV
Minute volume depends on RR
Tidal volume relatively fixed (6 to 8 mL/kg)
C
Anatomy
Ductus arteriosus
Contracts in the first few days of life
Can be patent for 7 to 10 days
Fibrose within 2 to 4 weeks
Foramen ovale
Closure is pressure-dependent
May reopen within the first 5 years
Physiology
Rate-dependent cardiac output
Bradycardia not well tolerated
External chest compression in NEONATES if HR < 60
Prone to bradycardia
Due to dominant vagal parasympathetic tone
Causes include hypoxia and laryngoscopy
Treat bradycardia with oxygen and ventilation first
Blood volume
Neonates ≈ 90 mL/kg
2 years to puberty ≈ 80 mL/kg
Adults ≈ 70 mL/kg
D
BBB poorly formed
Common in stressed neonates and prematurity
Hypoglycaemia
E
Prone to hypothermia
Termoregulation
Due to
Larger surface area to weight ratio
Poorly developed regulatory mechanisms
Shivering
Sweating
Vasoconstriction
Thermoneural zone
34ºC for premature infants
32ºC for neonates
28ºC for adolescents and adults
Increased insensible losses
Esp in premature infants
Others
Haematology
HbF
70 to 90% of Hb at birth
Drops within 3 months
Replaced by HbA
Negligible by 6 months
Higher affinity to O2
P50 = 18 to 20mmHg
Vitamin K dependent clotting factors low at term
Immune system
Immature at birth
Maternal antibodies offer some protection
Across placenta
Breastfeeding
Psychology
Anxiety
Parental anxiety is perceived and reacted on by the child
Infants < 6 months old NOT usually upset by separation
Children up to 4 years of age
Major concern is separation from parents
Major concerns
School age
Mutilating effects
Pain
Fear
Preschool
May think the problem may be his/her fault
School age
May get ideas of what will happen from TV or adult conversations

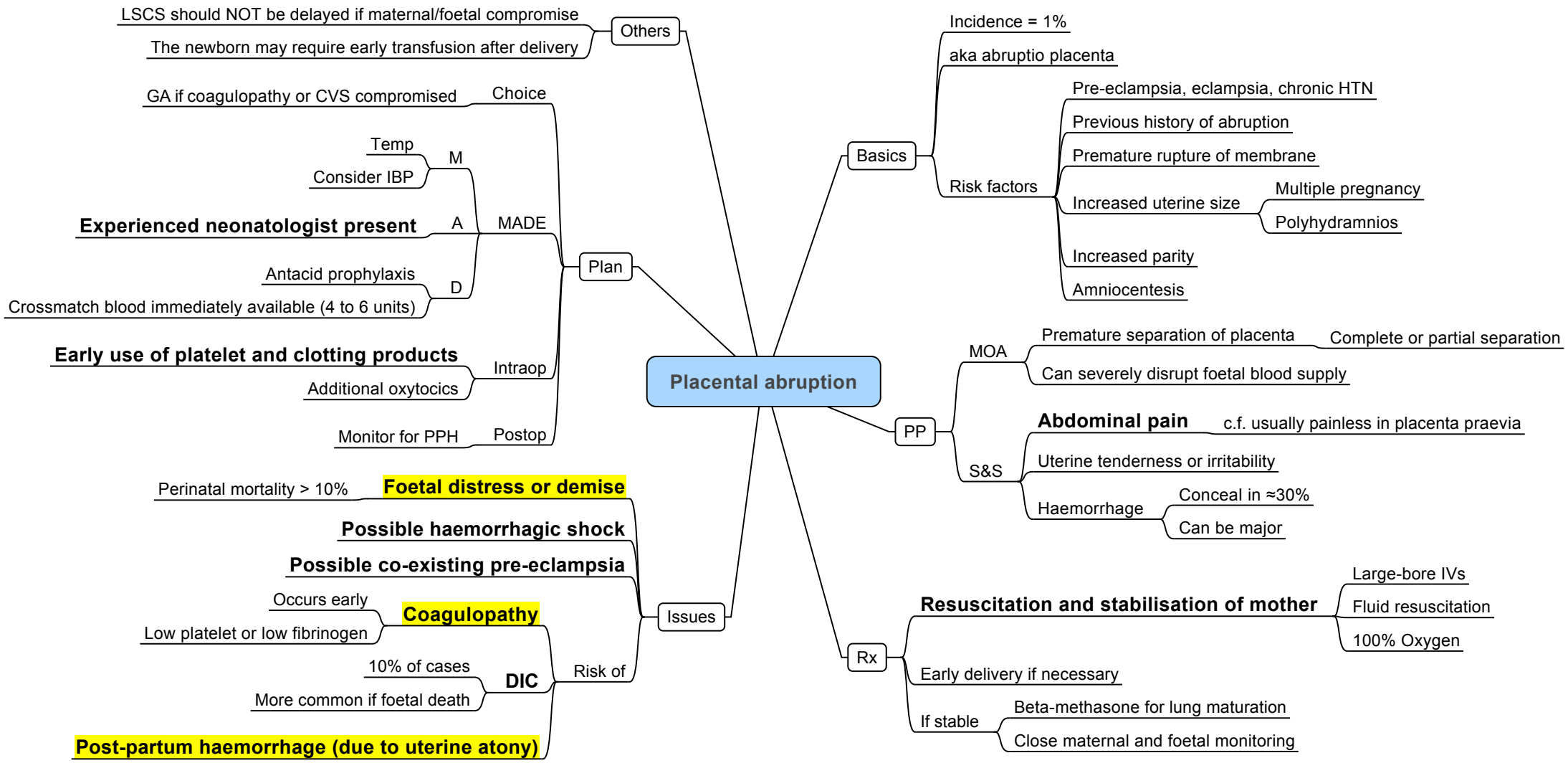

Paediatric size estimation
Weight
Australian Resus Council 2010
1 to 9 y.o.
Weight = (Age x 2 + 8) kg
e.g.
1 y.o. = 10 kg
≥ 10 y.o.
Age x 3.3
APLS 5th ed
0 to 12 months
Weight = Month / 2 + 4
e.g.
6 month old = 7 kg
1 to 5 y.o.
Weight = Age x 2 + 8
6 to 12 y.o.
Weight = Age x 3 + 7
ETT
Size
Newborn
< 1000 g = 2.5 mm
> 3000 g = 3 to 3.5 mm
ID = (Age / 4 + 4) mm
Cole's formula
For 2 to 10 year old
Same size for nasal or oral
Consider going up by 0.5 mm to get better fit
If cuffed ETT, go down by 0.5 mm
Length at lip
Neonates (oral)
Length = Weight + 6 cm
Oral
Length = Age / 2 + 12 cm
Add 2 to 3 cm if nasal intubation
LMA
Size 1
Up to 5 kg
Max cuff = 4 mL
Size 1.5
5 to 10 kg
Max cuff = 7 mL
Size 2
10 to 20 kg
Max cuff = 10 mL
Size 2.5
20 to 30 kg
Max cuff = 14 mL
Size 3
30 to 50 kg
Max cuff = 20 mL
Others
Complex calculations
May be more accurate
But greater risk of error
Remember estimates are just estimates
Examples
Average term baby
3.5 kg
Usually 3.5 mm ETT
9.5 cm at lips
Average 1 year old
10 kg
Usually 4 mm ETT
12.5 cm at lips
Average 6 year old
20 to 25 kg
Usually 5.5 mm ETT
15 cm at lips
Average 10 y.o.
Over 30kg
Usually 6.5 mm ETT
18 at lips

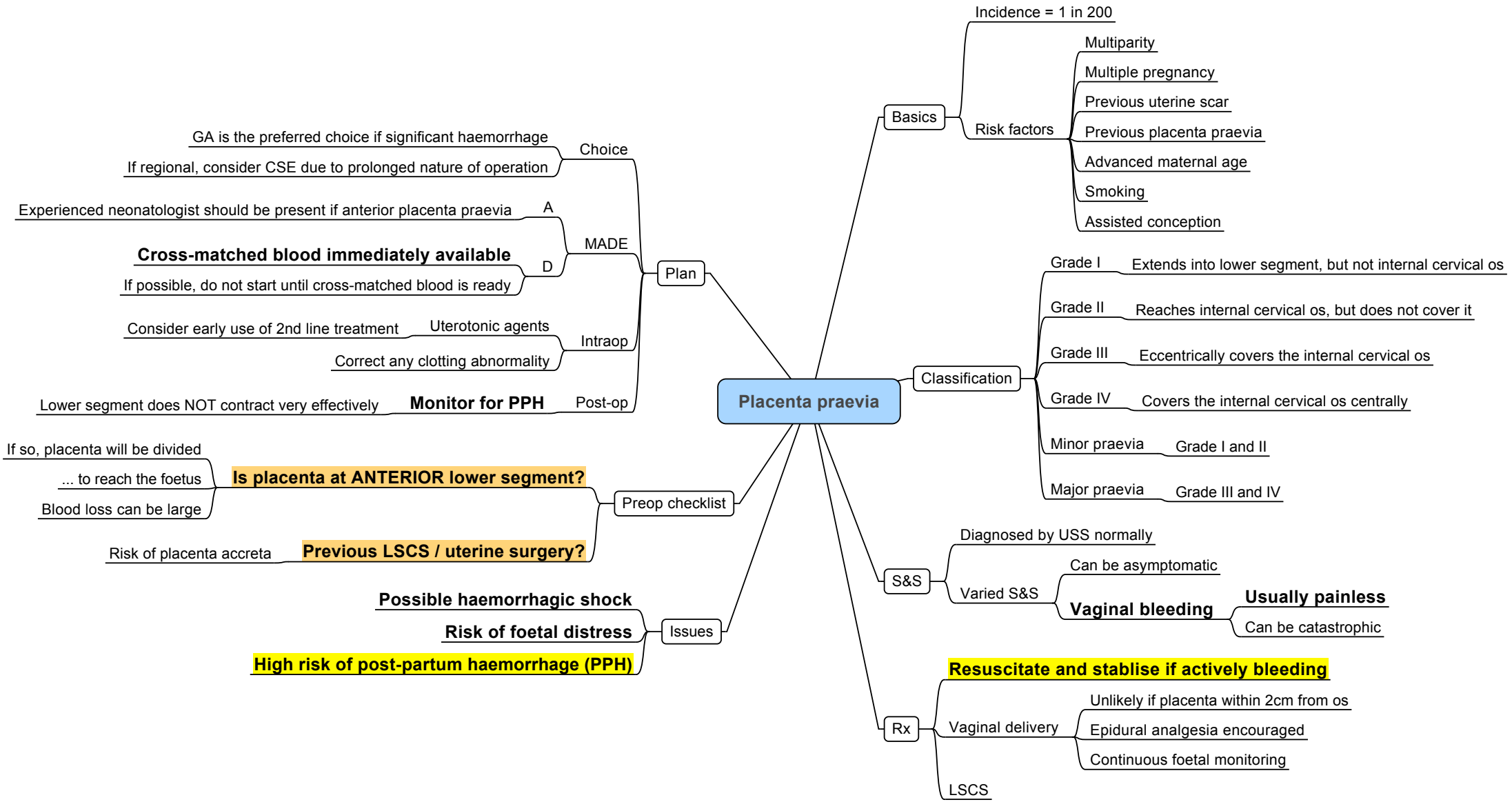

Parkinson's disease - AMx
GA vs Regional
GA
Pros
No tremor
Drugs can worsen S&S
Cons
Can worsen resp function
Risk of PONV
Regional
Pros
Less respiratory complication
Minimal interruption to Rx
Cons
Technically difficult due to tremor
Tremor not abolished if no motor block
Tremor can interfere with
Monitor
?Surgery
Lack of cooperation if dementia
Antiemetic Rx
Avoid
Metoclopramide
Droperidol
Prochlorperazine
Use
Domperidone
Does not cross BBB
5-HT antagonists (e.g. ondansetron)
Anti-histamine (e.g. cyclizine)
Plan
D
Consider glycopyrrolate premed if distressing sialorrhoea
Induction
Consider RSI
Intraop
Consider NGT to allow early return of oral Rx
Antiemetic
Postop
HDU / ICU if...
Resume medication as early as possible
Significant functional disability preop
Significant CVS / Resp issues
Goals
Airway management as appropriate
Minimise Rx interruption
1st case on list
Avoid prolonged fasting
Cease medication as late as possible
Avoid drugs with extra-pyramidal side effects
Maintain normothermia
Issues
A
Aspiration risk
Flexion deformity of neck
B
Sleep apnoea
Restrictive lung disease
Others
Autonomic dysfunction
Sialorrhoea and drooling
Dementia / depression
Resting tremor
Rx-related
Adverse effects
Dopamine agonists
Postural hypotension
Arrhythmia
Dyskinesia
Anti-cholinergic Rx
Central anticholinergic syndrome
Interaction
If MAO-B inhibitor
Pethidine
Hypertension
Muscle rigidity
Hyperpyrexia
SSRI and TCA
CNS excitability
If levodopa
TCA
Potentiation of arrhythmia
Acute withdrawal of levodopa
Neuroleptic malignant syndrome (NMS) risk
Postop
Postop confusion / delirium common
Postop shivering common
PCA may be difficult for patient to operate
Consultation
History
Severity
Functional impairment
Cachexia
Rx
esp
MAO-B inhibitors (e.g. Selegiline)
Levodopa
Exam
Airway assessment
Including neck ROM
Assess ability to use PCA for postop
Ix
ECG
Spirometry / Lung function test
Optimisation
Consider referral re optimisation of S&S

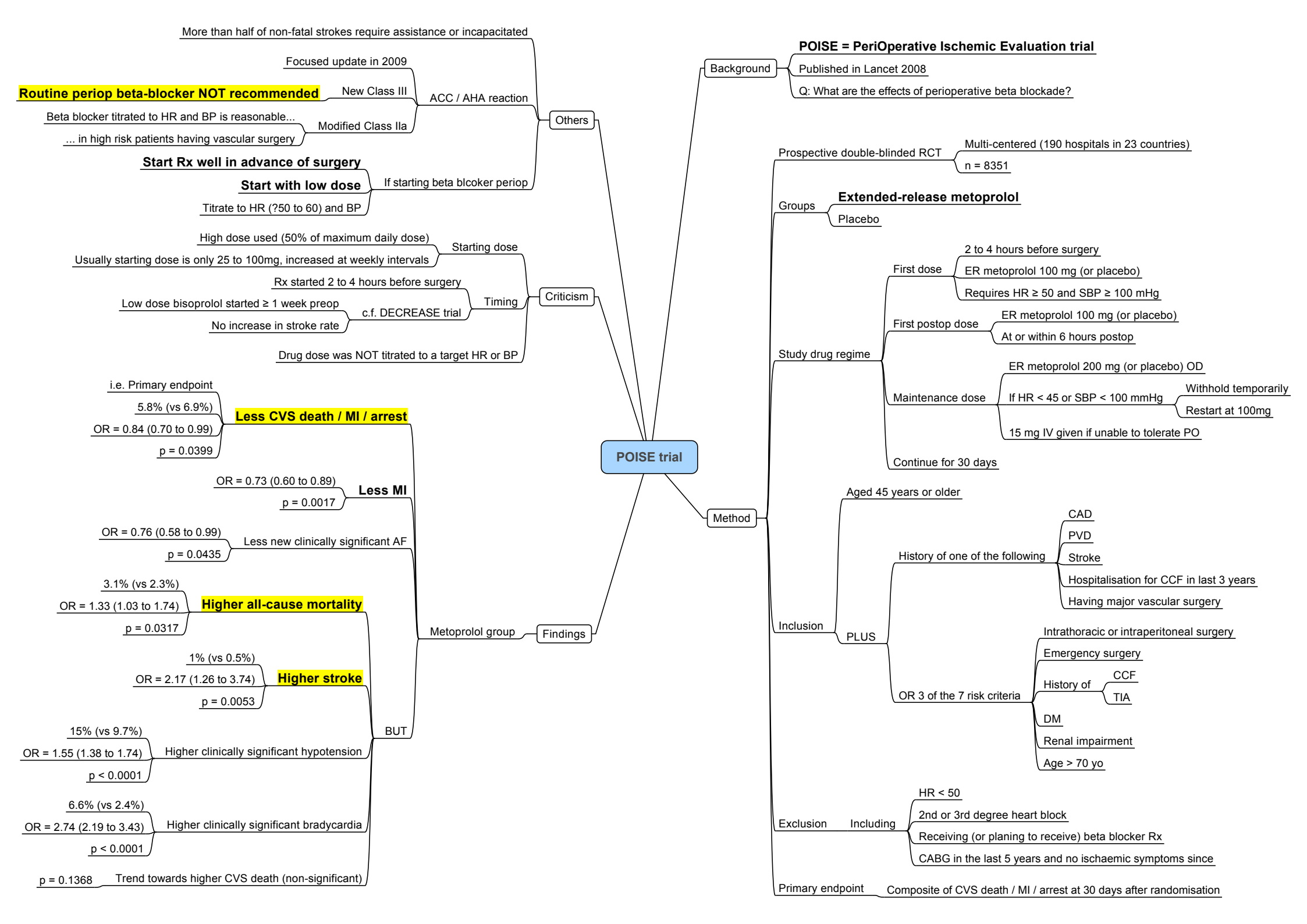

Parkinson's disease - Features
4 major S&S
Resting tremor
4 to 8 Hz
Diagnostic, but not required
Reduced with
Activities
Concentration
Sleep
Increases with stress
Bradykinesia
Required for Dx
Difficulty initiating movement
Slow movement
Rigidity
Lead pipe type rigidity
Cogwheel with tremor
Postural instability
Others
A+B
Flexion deformity of neck
Aspiration risk
Sleep apnoea
Restrictive lung disease
Vocal cord paralysis (very rare)
C
Postural hypotension
D
Autonomic dysfunction
Dementia / depression
GIT
Sialorrhoea and drooling
Nausea (due to Rx)
Others
Mask face
Soft monotonous voice
Micrographia
Shuffling gait
Restless legs syndrome


Phaeochromocytoma - AMx
Others
"10% tumour"
10% bilateral
10% malignant
10% extra-renal
10% familial
10% found in children
Unexpected phaeo
High periop mortality (up to 50%)
S&S
Pulmonary oedema
Profound sweating
Hypertension
Rx
Vasodilator
GTN infusion
IV fluid to treat hypotension
Issues
Intraop
CVS instability
Labile BP
Arrhythmia
Catecholamine surge
Induction
Insufflation of abdomen
Surgical handling of tumour
BSL instability
Postop risk
Hypotension
Decrease in catecholamine
Residual alpha blockade
Addisonian crisis
Hypoglycaemia
Consultation
History
Location of tumour
Symptomatic?
Palpitation
Sweating, headache
Anxiety
N&V
Functional capacity
Associated
Neurofibromatosis
MEN-2
Rx
Alpha and beta blockers
Ix
ECG
Echo
Catecholamine cardiomyopathy
BSL
Optimisation
Goal
BP < 140 / 90 in over 24 hour period
HR < 100 min
Marked postural hypotension with compensatory tachycardia
Includes
Alpha blockade
Alpha blocker FIRST to avoid unopposed beta-blockade
Non-selective preferred
e.g.
Phenoxybenzamine
Phentolamine
Beta blockade
if persistent tachycardia or arrhythmia
e.g.
Atenolol
Labetalol
Hydration
Consider
Calcium channel blockers
Magnesium
Endocrinologist should already be involved
Delay surgery until optimised
Plan
Consider epidural for postop analgesia
MADE
M
IBP
CVP
Consider
TOE
PA catheter / Cardiac output monitor
D
Consider anxiolytic premed
Have ready
Vasodilators
Phentolamines
GTN, SNP
Beta blockers
For HR control
Esmolol, metoprolol
Vasopressors
Induction
AVOID
Suxamethonium
Halothane
Ketamine
Prehydrate liberally if tolerated
Intraop
Consider remifentanil infusion
Postop
Analgesia
ICU / HDU admission
Steroid support if bilateral resection
Goals
Maintain CVS stability
Keep BP stable
Avoid overcorrection
Keep HR < 100
Consider esmolol
Maintain stable BSL
Communication with surgical team

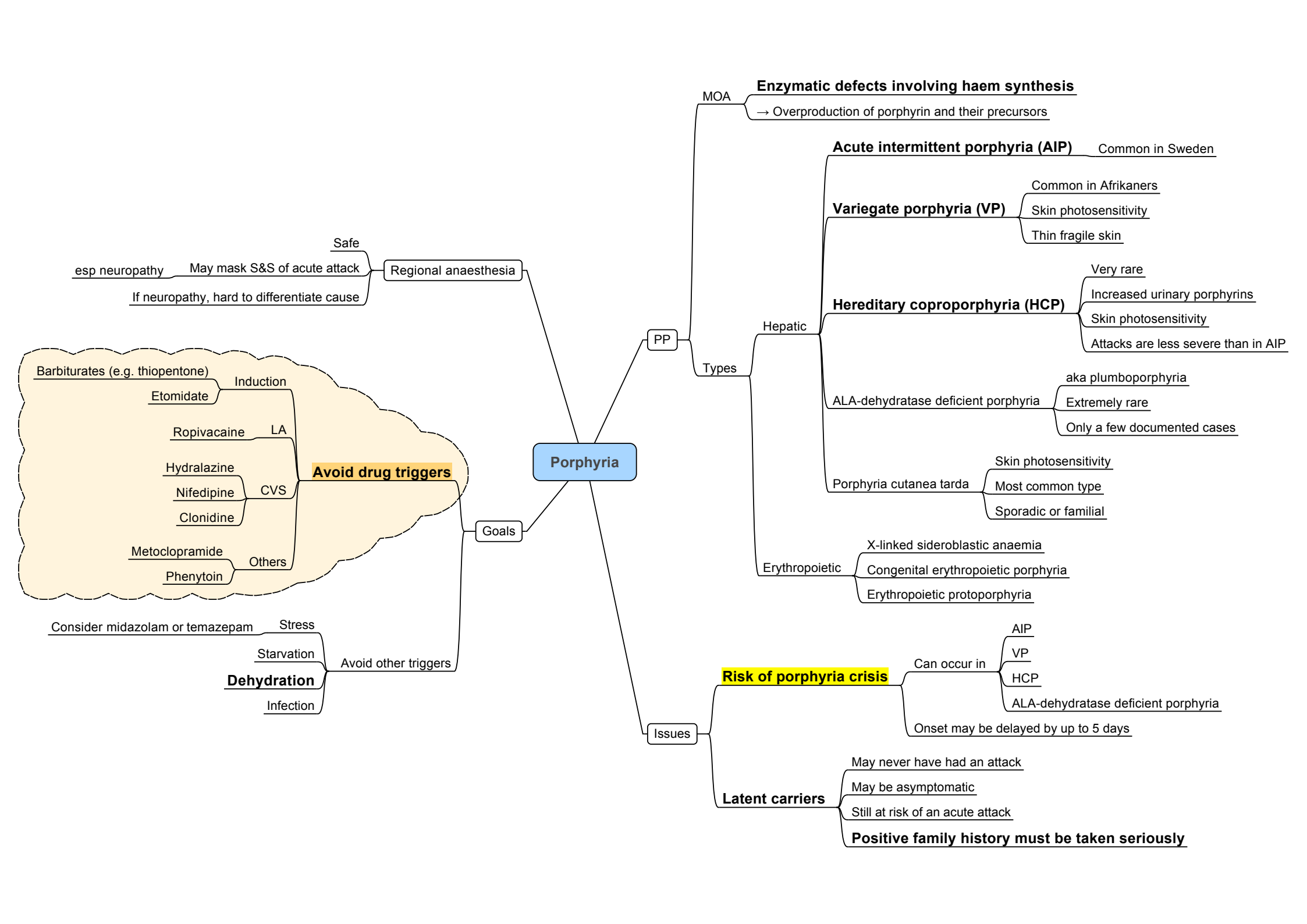

Placenta accreta
Basic
Definition
Abnormal implantation of placenta
Loss of a cleavage plane between placenta and myometrium
Association
Previous LSCS
Previous uterine surgery
Classification
Placenta accreta vera
Through endometrium to reach myometrium
Placenta increta
Into myometrium
Rare and severe
Placenta percreta
Through myometrium
... onto uterine serosa and surrounding structures
Rare and severe
7% mortality
Hysterectomy required in 95%
Placenta overlaying previous LSCS scars
Placenta praevia also present
Placenta accreta more common
But not always
If 1 previous CS
20 to 30% chance of placenta accreta
If 2 previous CSs
40 to 50% chance of placenta accreta
2/3 will require hysterectomy
Rx
MRI to determine extent of placenta accreta
Baby must be delivered by LSCS
Hysterectomy may be required
Consider interventional radiology
Preop insertion of bilateral iliac artery balloon
Embolisation (in emergency)
Major haemorrhage
Bleeding at delivery can be CATASTROPHIC
Checklist
Is the placenta anterior?
How many previous LSCSs?
How bad is the placenta accreta?
Manage as per major obstetric haemorrhage
Risk factors
Anterior placenta
Higher grade of placenta accreta
Goals
Fluid resuscitation as needed
Avoid hypothermia
Correct coagulopathy
Plan
Choice
GA is the preferred choice if significant haemorrhage
If regional, consider CSE due to prolonged operation
MADE
M
IBP
Temp
A
Large-bore IVs
Senior surgical operator
D
CXM blood immediately available
Have at least 6 units
E
Fluid warmer and pressure infusor
Consider cell saver
Intra-op
Uterotonic agents
Clotting abnormality corrected
Post-op
ICU or HDU if major haemorrhage
Post-partum haemorrhage risk if uterus preserved
Others
Preop discussion regarding risk of...
Haemorrhage
Transfusion
Hysterectomy

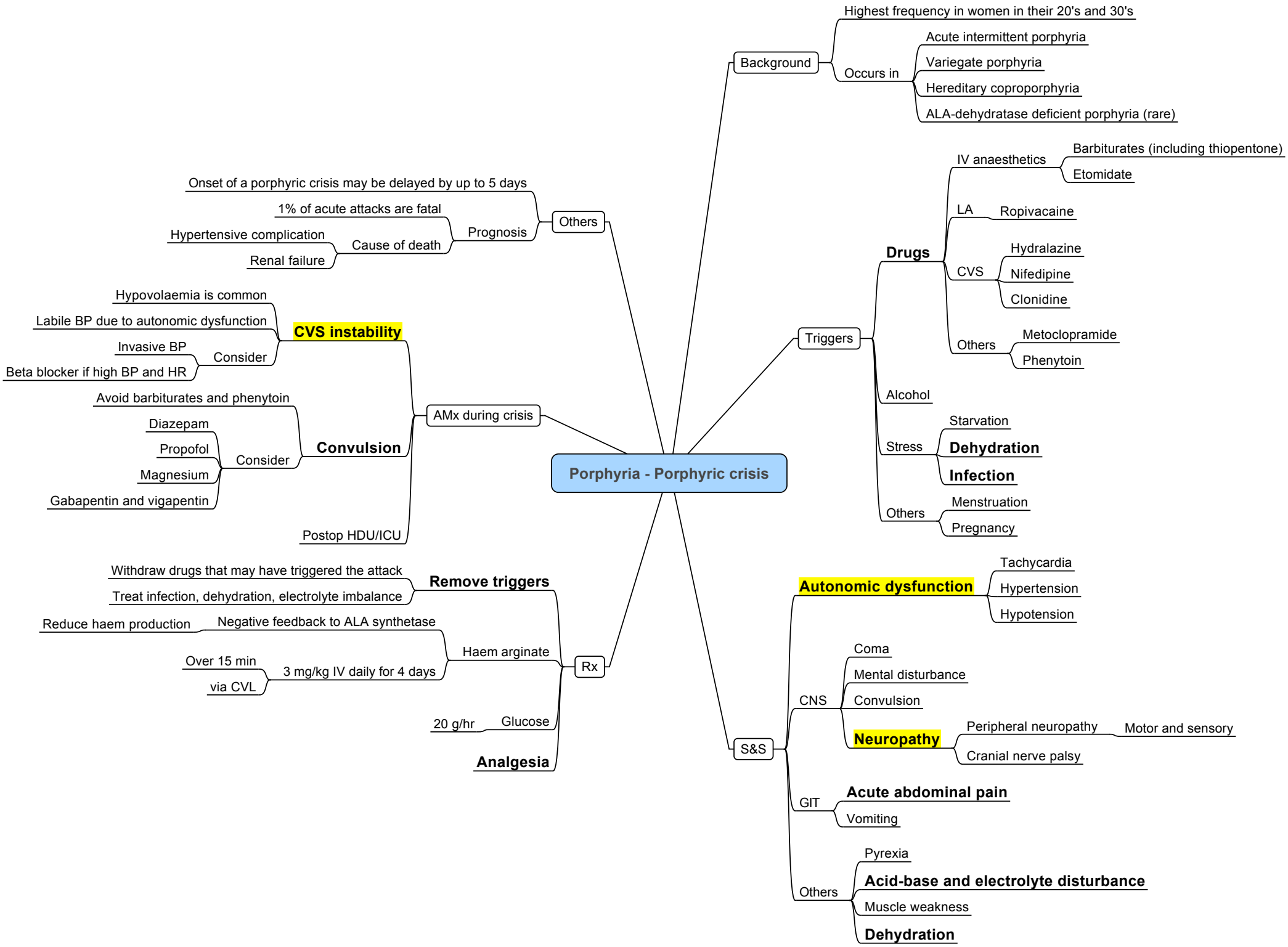

Placental abruption
Basics
Incidence = 1%
aka abruptio placenta
Risk factors
Pre-eclampsia, eclampsia, chronic HTN
Previous history of abruption
Premature rupture of membrane
Increased uterine size
Multiple pregnancy
Polyhydramnios
Increased parity
Amniocentesis
PP
MOA
Premature separation of placenta
Complete or partial separation
Can severely disrupt foetal blood supply
S&S
Abdominal pain
c.f. usually painless in placenta praevia
Uterine tenderness or irritability
Haemorrhage
Conceal in ≈30%
Can be major
Rx
Resuscitation and stabilisation of mother
Large-bore IVs
Fluid resuscitation
100% Oxygen
Early delivery if necessary
If stable
Beta-methasone for lung maturation
Close maternal and foetal monitoring
Issues
Foetal distress or demise
Perinatal mortality > 10%
Possible haemorrhagic shock
Possible co-existing pre-eclampsia
Risk of
Coagulopathy
Occurs early
Low platelet or low fibrinogen
DIC
10% of cases
More common if foetal death
Post-partum haemorrhage (due to uterine atony)
Plan
Choice
GA if coagulopathy or CVS compromised
MADE
M
Temp
Consider IBP
A
Experienced neonatologist present
D
Antacid prophylaxis
Crossmatch blood immediately available (4 to 6 units)
Intraop
Early use of platelet and clotting products
Additional oxytocics
Postop
Monitor for PPH
Others
LSCS should NOT be delayed if maternal/foetal compromise
The newborn may require early transfusion after delivery

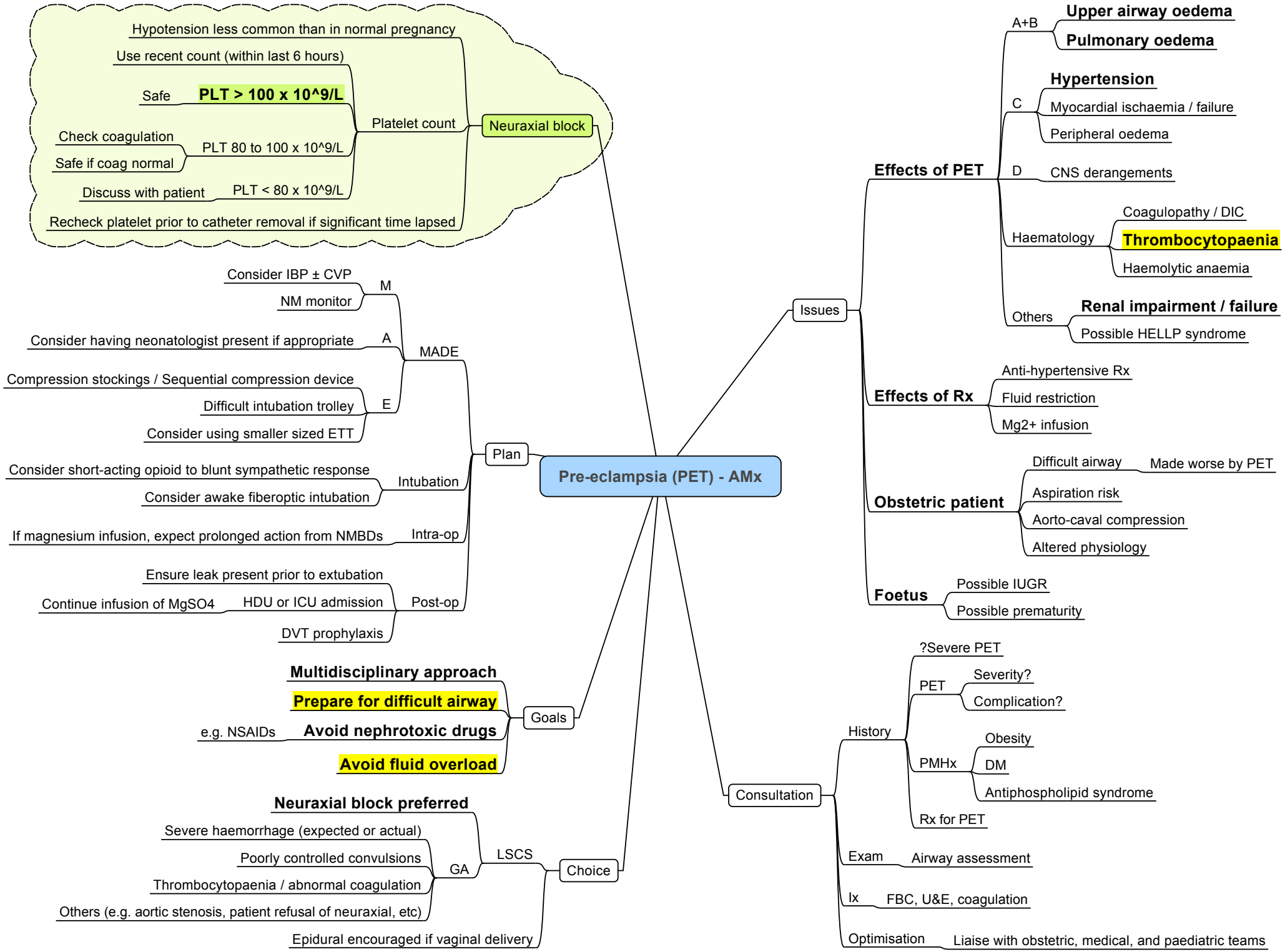

Placenta praevia
Basics
Incidence = 1 in 200
Risk factors
Multiparity
Multiple pregnancy
Previous uterine scar
Previous placenta praevia
Advanced maternal age
Smoking
Assisted conception
Classification
Grade I
Extends into lower segment, but not internal cervical os
Grade II
Reaches internal cervical os, but does not cover it
Grade III
Eccentrically covers the internal cervical os
Grade IV
Covers the internal cervical os centrally
Minor praevia
Grade I and II
Major praevia
Grade III and IV
S&S
Diagnosed by USS normally
Varied S&S
Can be asymptomatic
Vaginal bleeding
Usually painless
Can be catastrophic
Rx
Resuscitate and stablise if actively bleeding
Vaginal delivery
Unlikely if placenta within 2cm from os
Epidural analgesia encouraged
Continuous foetal monitoring
LSCS
Issues
Possible haemorrhagic shock
Risk of foetal distress
High risk of post-partum haemorrhage (PPH)
Preop checklist
Is placenta at ANTERIOR lower segment?
If so, placenta will be divided
... to reach the foetus
Blood loss can be large
Previous LSCS / uterine surgery?
Risk of placenta accreta
Plan
Choice
GA is the preferred choice if significant haemorrhage
If regional, consider CSE due to prolonged nature of operation
MADE
A
Experienced neonatologist should be present if anterior placenta praevia
D
Cross-matched blood immediately available
If possible, do not start until cross-matched blood is ready
Intraop
Uterotonic agents
Consider early use of 2nd line treatment
Correct any clotting abnormality
Post-op
Monitor for PPH
Lower segment does NOT contract very effectively

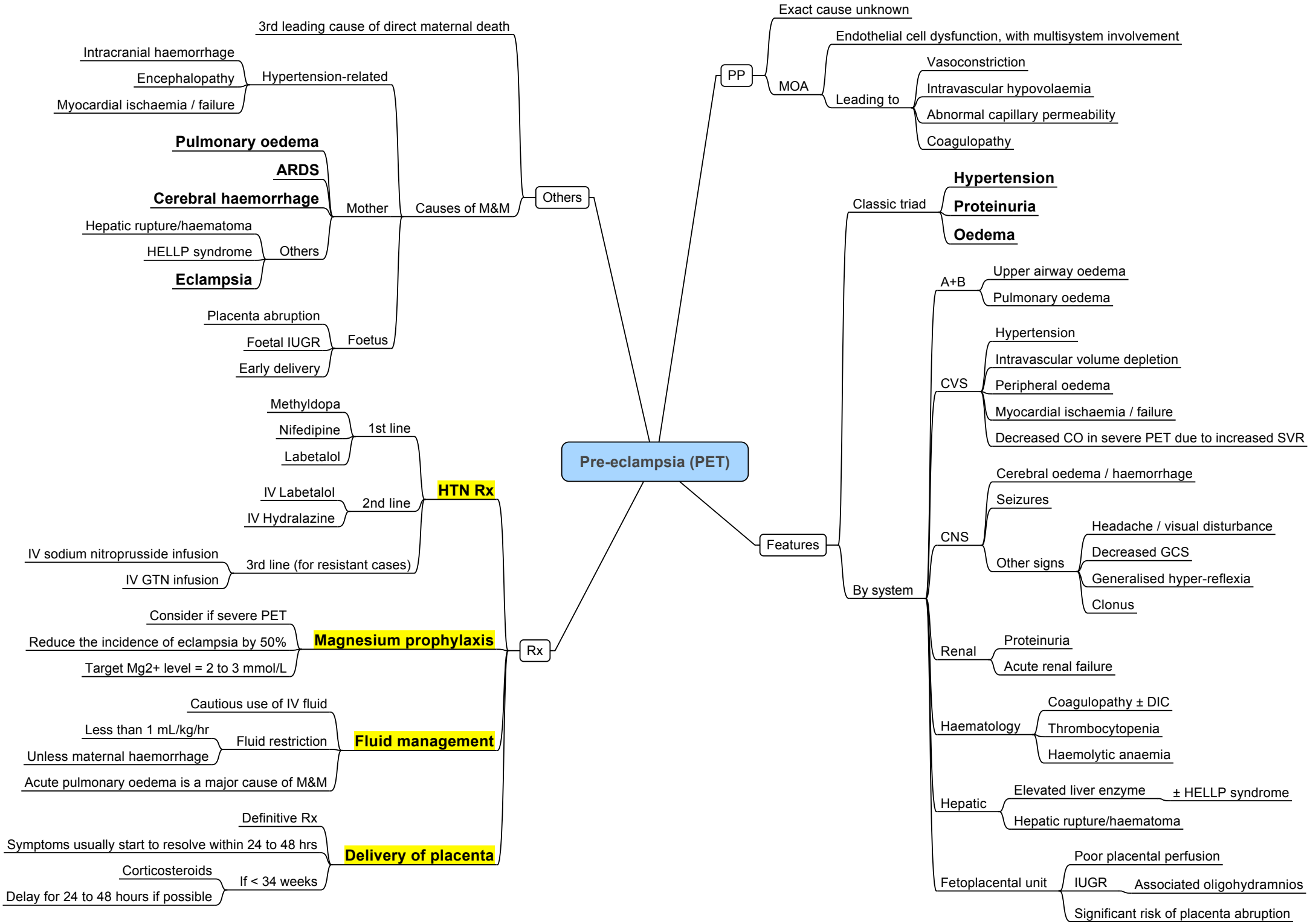

POISE trial
Background
POISE = PeriOperative Ischemic Evaluation trial
Published in Lancet 2008
Q: What are the effects of perioperative beta blockade?
Method
Prospective double-blinded RCT
Multi-centered (190 hospitals in 23 countries)
n = 8351
Groups
Extended-release metoprolol
Placebo
Study drug regime
First dose
2 to 4 hours before surgery
ER metoprolol 100 mg (or placebo)
Requires HR ≥ 50 and SBP ≥ 100 mHg
First postop dose
ER metoprolol 100 mg (or placebo)
At or within 6 hours postop
Maintenance dose
ER metoprolol 200 mg (or placebo) OD
If HR < 45 or SBP < 100 mmHg
Withhold temporarily
Restart at 100mg
15 mg IV given if unable to tolerate PO
Continue for 30 days
Inclusion
Aged 45 years or older
PLUS
History of one of the following
CAD
PVD
Stroke
Hospitalisation for CCF in last 3 years
Having major vascular surgery
OR 3 of the 7 risk criteria
Intrathoracic or intraperitoneal surgery
Emergency surgery
History of
CCF
TIA
DM
Renal impairment
Age > 70 yo
Exclusion
Including
HR < 50
2nd or 3rd degree heart block
Receiving (or planing to receive) beta blocker Rx
CABG in the last 5 years and no ischaemic symptoms since
Primary endpoint
Composite of CVS death / MI / arrest at 30 days after randomisation
Findings
Metoprolol group
Less CVS death / MI / arrest
i.e. Primary endpoint
5.8% (vs 6.9%)
OR = 0.84 (0.70 to 0.99)
p = 0.0399
Less MI
OR = 0.73 (0.60 to 0.89)
p = 0.0017
Less new clinically significant AF
OR = 0.76 (0.58 to 0.99)
p = 0.0435
BUT
Higher all-cause mortality
3.1% (vs 2.3%)
OR = 1.33 (1.03 to 1.74)
p = 0.0317
Higher stroke
1% (vs 0.5%)
OR = 2.17 (1.26 to 3.74)
p = 0.0053
Higher clinically significant hypotension
15% (vs 9.7%)
OR = 1.55 (1.38 to 1.74)
p < 0.0001
Higher clinically significant bradycardia
6.6% (vs 2.4%)
OR = 2.74 (2.19 to 3.43)
p < 0.0001
Trend towards higher CVS death (non-significant)
p = 0.1368
Criticism
Others
ACC / AHA reaction
New Class III
Routine periop beta-blocker NOT recommended
Focused update in 2009
Modified Class IIa
Beta blocker titrated to HR and BP is reasonable...
... in high risk patients having vascular surgery
More than half of non-fatal strokes require assistance or incapacitated
If starting beta blcoker periop
Start Rx well in advance of surgery
Start with low dose
Titrate to HR (?50 to 60) and BP
Timing
Starting dose
High dose used (50% of maximum daily dose)
Usually starting dose is only 25 to 100mg, increased at weekly intervals
c.f. DECREASE trial
Rx started 2 to 4 hours before surgery
Low dose bisoprolol started ≥ 1 week preop
No increase in stroke rate
Drug dose was NOT titrated to a target HR or BP

	Safe	Controversial	AVOID
Volatile and N2O	N2O	Halothane	Enflurane
		Isoflurane	
		Sevoflurane	
Induction agents	Propofol (bolus)	Propofol (infusion)	**Barbiturates**
		Ketamine	**Thiopentone**
			Etomidate
NMBDs	Suxamethonium	Pancuronium	Alcuronium
	Vecuronium	Rocuronium	
		Atracurium	
		Mivacurium	
NMBD reversal	Neostigmine		
	Atropine		
	Glycopyrrolate		
Local anaesthetics	Bupivacaine	Lignocaine	**Ropivacaine**
	Prilocaine	Cocaine	Mepivacaine
	Procaine		
	Procainamide		
Analgesics	Aspirin	Diclofenac	Pentazocine
	Paracetamol	Ketorolac	
	Codeine	Sufentanil	
	Morphine		
	Fentanyl		
	Alfentanil		
	Pethidine		
	Buprenorphine		
	Naloxone		
Sedatives	Midazolam	Diazepam	Nitrazepam
	Temazepam		
	Lorazepam		
Anti-emetics	Droperiodol	Ondansetron	Cimetidine
	Phenothiazine	Ranitidine	**Metoclopramide**
	Chlorpromazine		
	Prochlorperazine		
CVS drugs	Adrenaline	Diltizem	**Hydralazine**
	Alpha agonists	Verapamil	**Nifedipine**
	Beta agonists	SNP	Phenoxybenzamine
	Phentolamine		Ergometrine
	Procainamide		**Clonidine**
	Magnesium		Methyldopa
Others	Heparin	Steroids	Aminophylline
			Oral contraceptive pill
			Phenytoin
			Sulphonamides

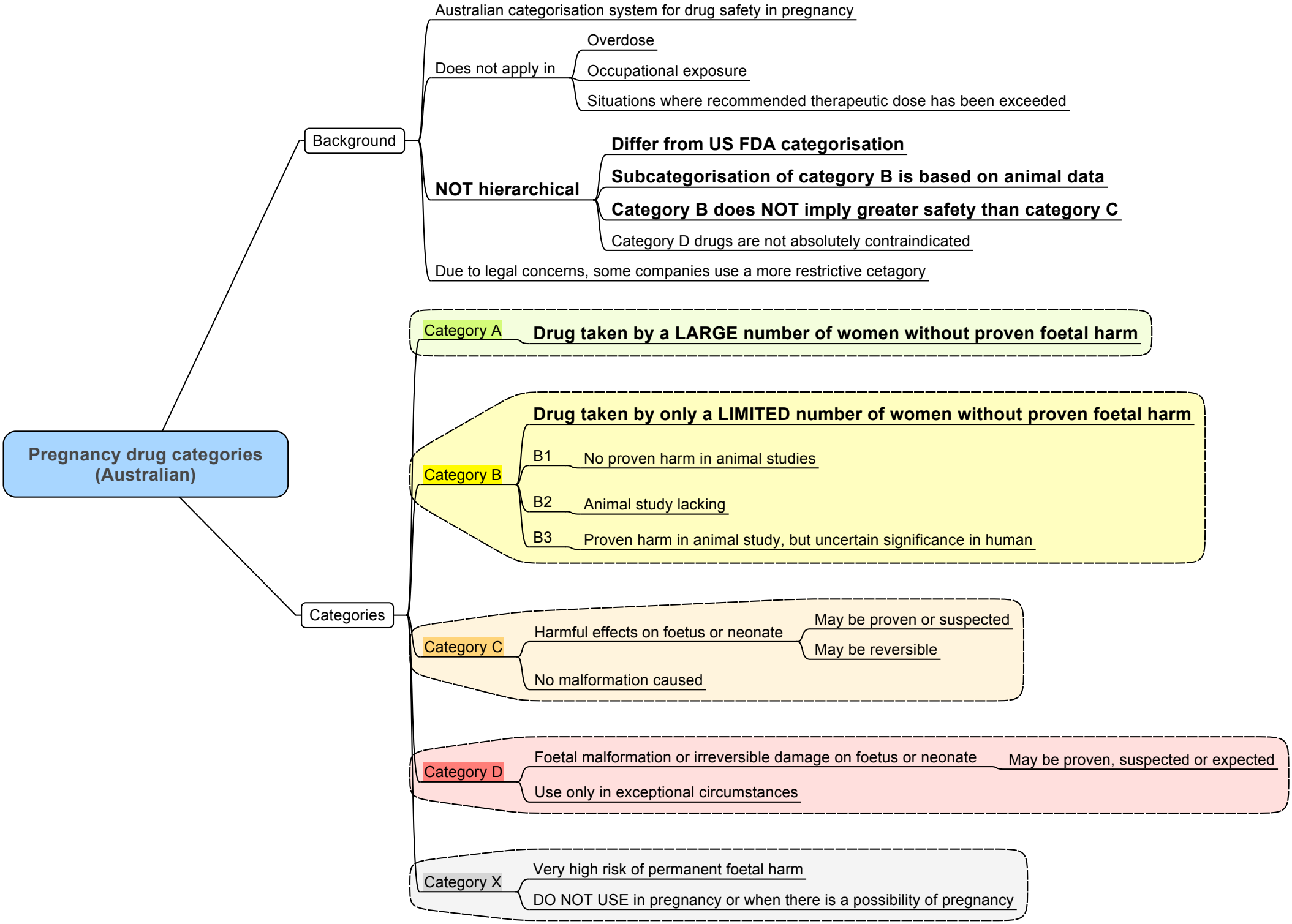

Porphyria
PP
MOA
Enzymatic defects involving haem synthesis
→ Overproduction of porphyrin and their precursors
Types
Hepatic
Acute intermittent porphyria (AIP)
Common in Sweden
Variegate porphyria (VP)
Common in Afrikaners
Skin photosensitivity
Thin fragile skin
Hereditary coproporphyria (HCP)
Very rare
Increased urinary porphyrins
Skin photosensitivity
Attacks are less severe than in AIP
ALA-dehydratase deficient porphyria
aka plumboporphyria
Extremely rare
Only a few documented cases
Porphyria cutanea tarda
Skin photosensitivity
Most common type
Sporadic or familial
Erythropoietic
X-linked sideroblastic anaemia
Congenital erythropoietic porphyria
Erythropoietic protoporphyria
Issues
Risk of porphyria crisis
Can occur in
AIP
VP
HCP
ALA-dehydratase deficient porphyria
Onset may be delayed by up to 5 days
Latent carriers
May never have had an attack
May be asymptomatic
Still at risk of an acute attack
Positive family history must be taken seriously
Goals
Avoid drug triggers
Induction
Barbiturates (e.g. thiopentone)
Etomidate
LA
Ropivacaine
CVS
Hydralazine
Nifedipine
Clonidine
Others
Metoclopramide
Phenytoin
Avoid other triggers
Consider midazolam or temazepam
Stress
Starvation
Dehydration
Infection
Regional anaesthesia
Safe
May mask S&S of acute attack
esp neuropathy
If neuropathy, hard to differentiate cause

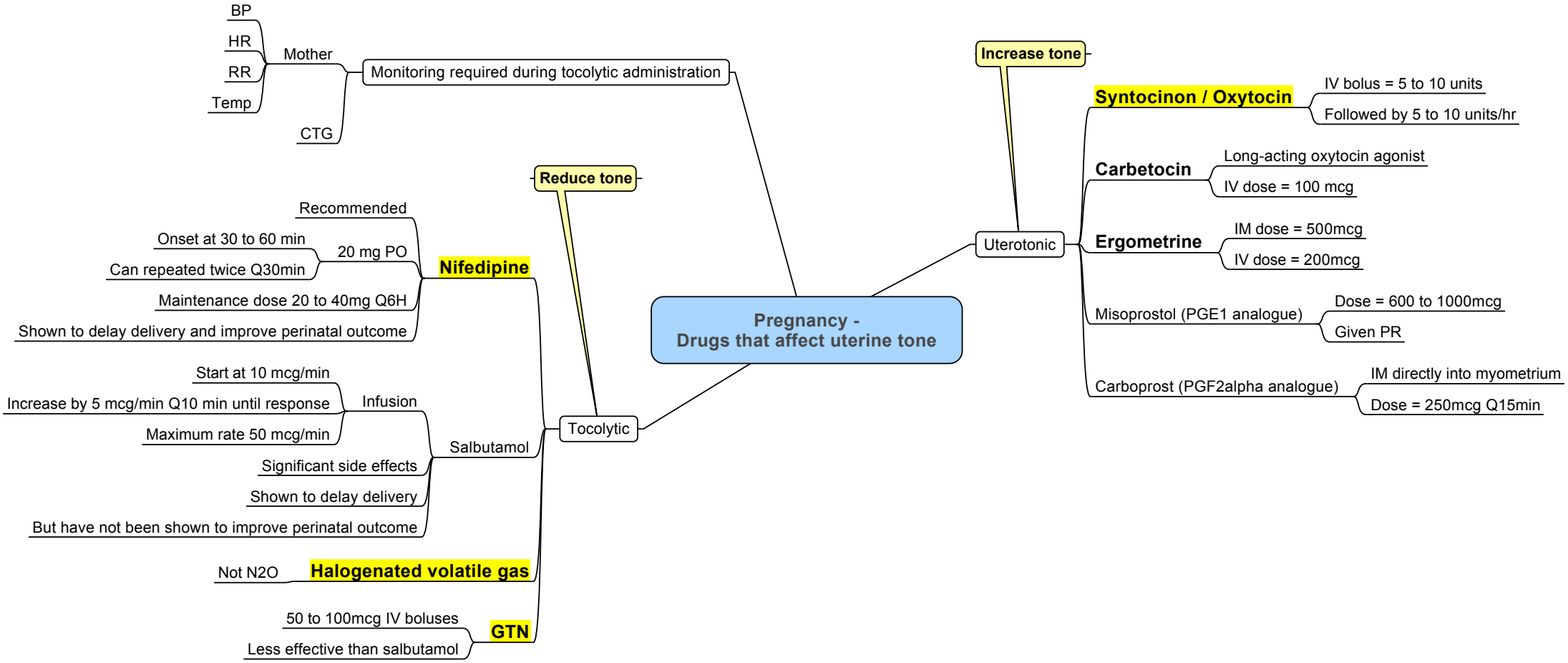

Porphyria - Porphyric crisis
Background
Highest frequency in women in their 20's and 30's
Occurs in
Acute intermittent porphyria
Variegate porphyria
Hereditary coproporphyria
ALA-dehydratase deficient porphyria (rare)
Triggers
Drugs
IV anaesthetics
Barbiturates (including thiopentone)
Etomidate
LA
Ropivacaine
CVS
Hydralazine
Nifedipine
Clonidine
Others
Metoclopramide
Phenytoin
Alcohol
Stress
Starvation
Dehydration
Infection
Others
Menstruation
Pregnancy
S&S
Autonomic dysfunction
Tachycardia
Hypertension
Hypotension
CNS
Coma
Mental disturbance
Convulsion
Neuropathy
Peripheral neuropathy
Motor and sensory
Cranial nerve palsy
GIT
Acute abdominal pain
Vomiting
Others
Pyrexia
Acid-base and electrolyte disturbance
Muscle weakness
Dehydration
Rx
Remove triggers
Withdraw drugs that may have triggered the attack
Treat infection, dehydration, electrolyte imbalance
Haem arginate
Negative feedback to ALA synthetase
Reduce haem production
3 mg/kg IV daily for 4 days
Over 15 min
via CVL
Glucose
20 g/hr
Analgesia
AMx during crisis
CVS instability
Hypovolaemia is common
Labile BP due to autonomic dysfunction
Consider
Invasive BP
Beta blocker if high BP and HR
Convulsion
Avoid barbiturates and phenytoin
Consider
Diazepam
Propofol
Magnesium
Gabapentin and vigapentin
Postop HDU/ICU
Others
Prognosis
Onset of a porphyric crisis may be delayed by up to 5 days
1% of acute attacks are fatal
Cause of death
Hypertensive complication
Renal failure

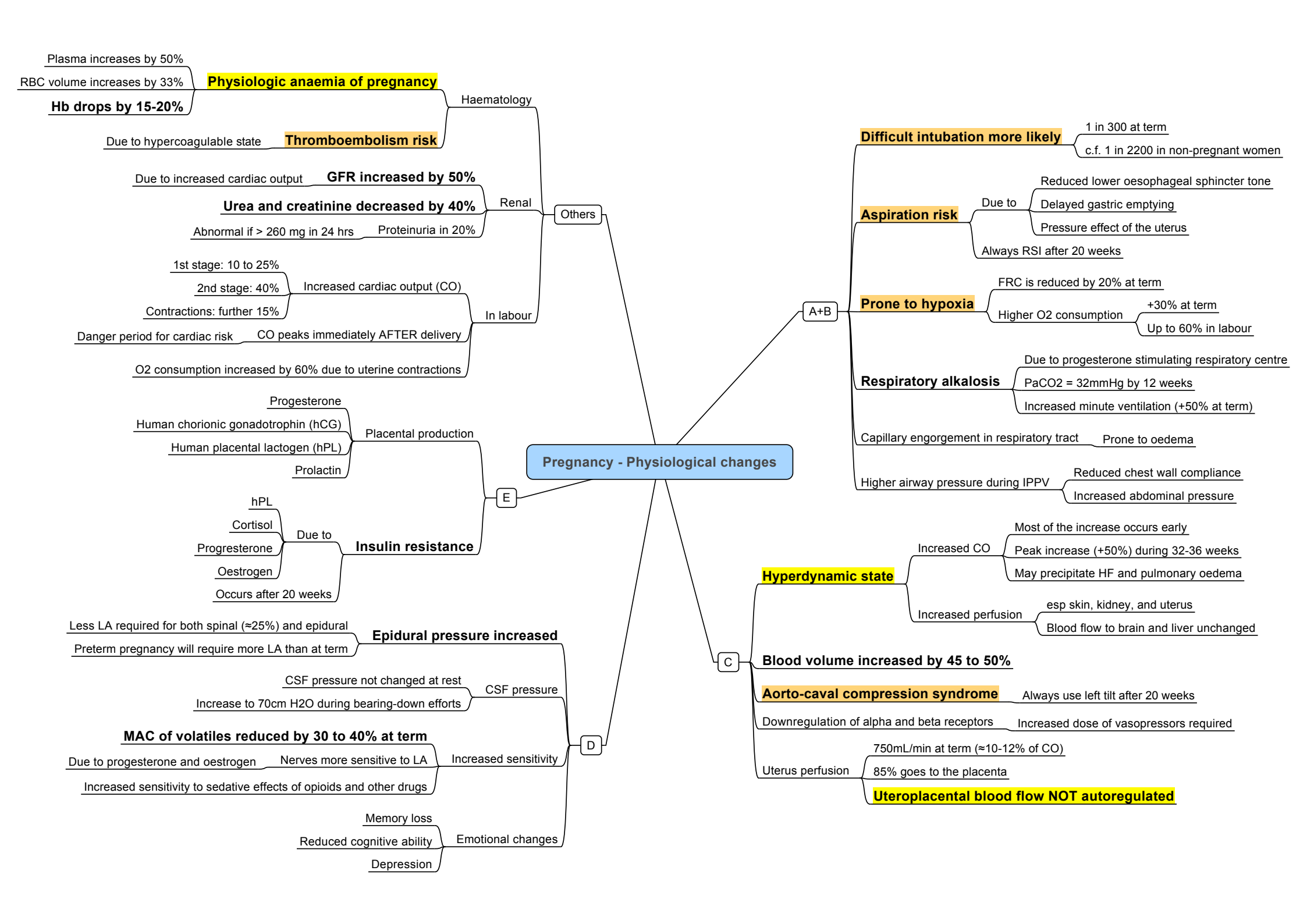

Pre-eclampsia (PET) - AMx
Issues
Effects of PET
A+B
Upper airway oedema
Pulmonary oedema
C
Hypertension
Myocardial ischaemia / failure
Peripheral oedema
D
CNS derangements
Haematology
Coagulopathy / DIC
Thrombocytopaenia
Haemolytic anaemia
Others
Renal impairment / failure
Possible HELLP syndrome
Effects of Rx
Anti-hypertensive Rx
Fluid restriction
Mg2+ infusion
Obstetric patient
Difficult airway
Made worse by PET
Aspiration risk
Aorto-caval compression
Altered physiology
Foetus
Possible IUGR
Possible prematurity
Consultation
History
?Severe PET
PET
Severity?
Complication?
PMHx
Obesity
DM
Antiphospholipid syndrome
Rx for PET
Exam
Airway assessment
Ix
FBC, U&E, coagulation
Optimisation
Liaise with obstetric, medical, and paediatric teams
Choice
LSCS
Neuraxial block preferred
GA
Severe haemorrhage (expected or actual)
Poorly controlled convulsions
Thrombocytopaenia / abnormal coagulation
Others (e.g. aortic stenosis, patient refusal of neuraxial, etc)
Epidural encouraged if vaginal delivery
Goals
Multidisciplinary approach
Prepare for difficult airway
e.g. NSAIDs
Avoid nephrotoxic drugs
Avoid fluid overload
Plan
MADE
M
Consider IBP ± CVP
NM monitor
A
Consider having neonatologist present if appropriate
E
Compression stockings / Sequential compression device
Difficult intubation trolley
Consider using smaller sized ETT
Intubation
Consider short-acting opioid to blunt sympathetic response
Consider awake fiberoptic intubation
Intra-op
If magnesium infusion, expect prolonged action from NMBDs
Post-op
Ensure leak present prior to extubation
Continue infusion of MgSO4
HDU or ICU admission
DVT prophylaxis
Neuraxial block
Platelet count
Safe
PLT > 100 x 10^9/L
Check coagulation
Safe if coag normal
PLT 80 to 100 x 10^9/L
Discuss with patient
PLT < 80 x 10^9/L
Hypotension less common than in normal pregnancy
Use recent count (within last 6 hours)
Recheck platelet prior to catheter removal if significant time lapsed

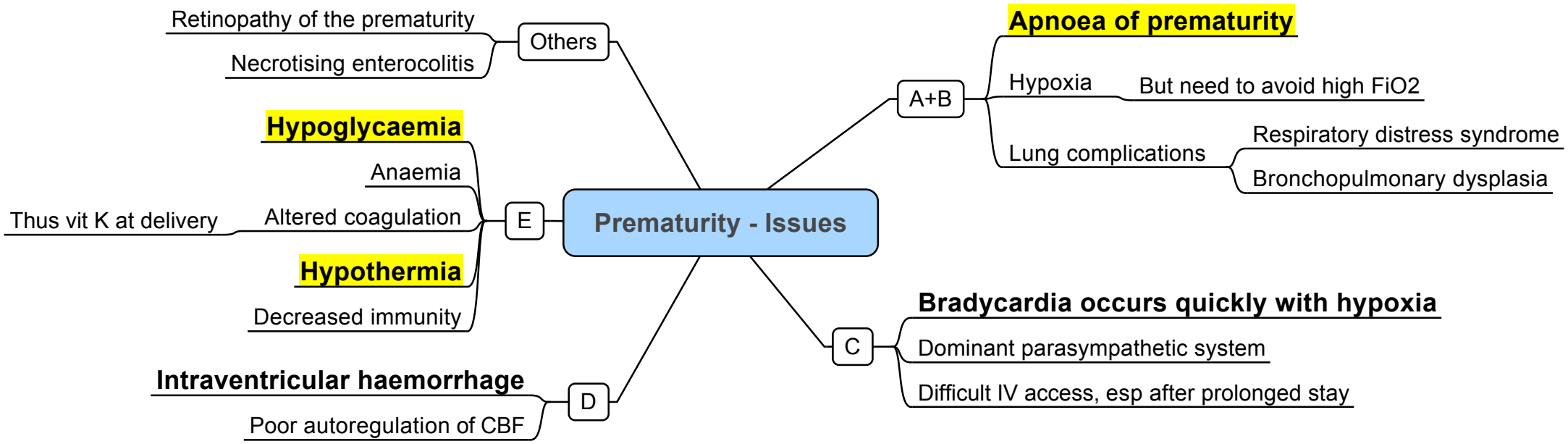

Pre-eclampsia (PET)
Features
Classic triad
Hypertension
Proteinuria
Oedema
By system
A+B
Upper airway oedema
Pulmonary oedema
CVS
Hypertension
Intravascular volume depletion
Peripheral oedema
Myocardial ischaemia / failure
Decreased CO in severe PET due to increased SVR
CNS
Cerebral oedema / haemorrhage
Seizures
Other signs
Headache / visual disturbance
Decreased GCS
Generalised hyper-reflexia
Clonus
Renal
Proteinuria
Acute renal failure
Haematology
Coagulopathy ± DIC
Thrombocytopenia
Haemolytic anaemia
Hepatic
Elevated liver enzyme
± HELLP syndrome
Hepatic rupture/haematoma
Fetoplacental unit
Poor placental perfusion
IUGR
Associated oligohydramnios
Significant risk of placenta abruption
PP
Exact cause unknown
MOA
Endothelial cell dysfunction, with multisystem involvement
Leading to
Vasoconstriction
Intravascular hypovolaemia
Abnormal capillary permeability
Coagulopathy
Others
3rd leading cause of direct maternal death
Causes of M&M
Hypertension-related
Intracranial haemorrhage
Encephalopathy
Myocardial ischaemia / failure
Mother
Pulmonary oedema
ARDS
Cerebral haemorrhage
Others
Hepatic rupture/haematoma
HELLP syndrome
Eclampsia
Foetus
Placenta abruption
Foetal IUGR
Early delivery
Rx
HTN Rx
1st line
Methyldopa
Nifedipine
Labetalol
2nd line
IV Labetalol
IV Hydralazine
3rd line (for resistant cases)
IV sodium nitroprusside infusion
IV GTN infusion
Magnesium prophylaxis
Consider if severe PET
Reduce the incidence of eclampsia by 50%
Target Mg2+ level = 2 to 3 mmol/L
Fluid management
Cautious use of IV fluid
Fluid restriction
Less than 1 mL/kg/hr
Unless maternal haemorrhage
Acute pulmonary oedema is a major cause of M&M
Delivery of placenta
Definitive Rx
Symptoms usually start to resolve within 24 to 48 hrs
If < 34 weeks
Corticosteroids
Delay for 24 to 48 hours if possible

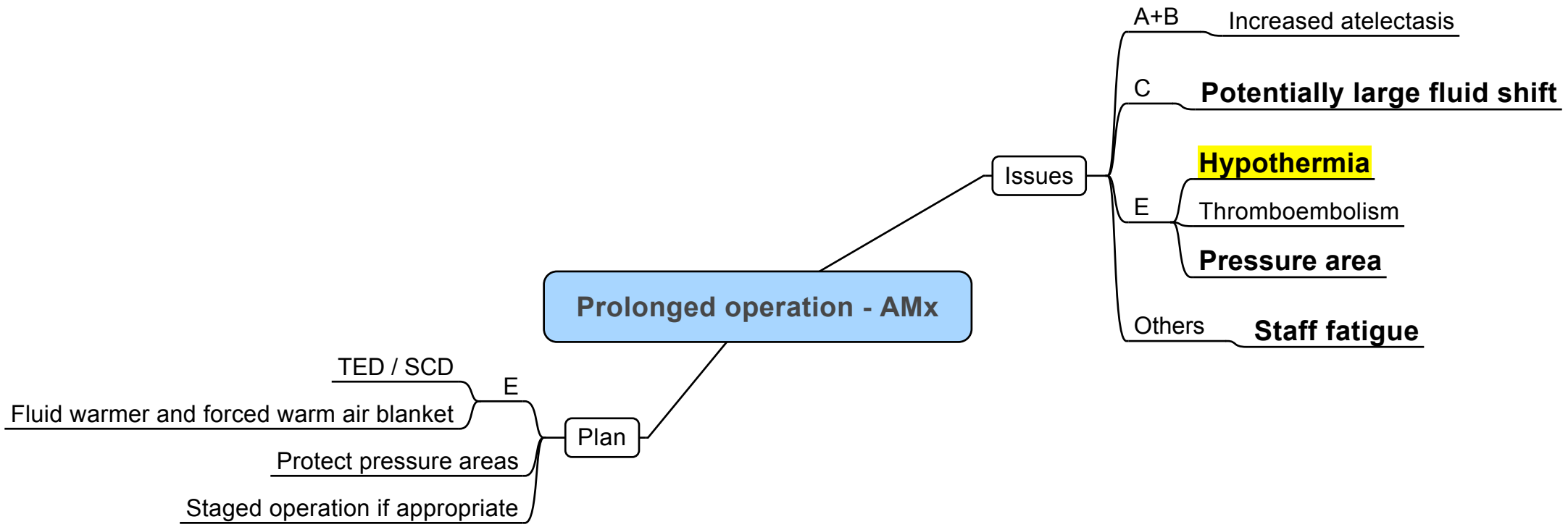
	A	B1	B2	B3	C	D
Volatile and N2O	Halothane		Sevoflurane	Isoflurane	Methoxyflurane	
	Nitrous oxide			Desflurane		
Induction agents	Thiopentone			Ketamine	Propofol	
NMBDs	Suxamethonium		Rocuronium		Cisatracurium	
			Mivacurium		Atracurium	
			Pancuronium		Vecuronium	
			Sugammadex			
Local anaesthetics	Bupivacaine	Ropivacaine		Levobupivacaine		
	Lignocaine					
Analgesics	Paracetamol	Naloxone		Celecoxib	Aspirin	
					NSAIDs (most)	
					Opioids	
					Tramadol	
Anti-emetics	Metoclopramide	Ondansetron	Hyoscine		Droperidol	
		Granisetron			Prochlorperazine	
					Chlorpromazine	
CVS drugs	Atropine	Dexmedetomidine	Glycopyrrolate	Clonidine	Metaraminol	ACE inhibitors
	Ephedrine		Phenylephrine		Metoprolol	
					Esmolol	
Antibiotics	Penicillin	Cephazolin	Cefodizime	Aciclovir		Gentamicin
	Cephalexin	Cefoxitin	Vancomycin			
	Cephalothin	Cefotaxime				
	Erythromycin	Cefotetan				
	Lincomycin	Ceftazidime				
		Ceftriaxone				
		Cefaclor				
Others	Hydrocortisone	Ranitidine		Flumazenil	Midazolam	Phenytoin
	Dexamethasone	Cimetidine		Baclofen	Promethazine	Carbamazepine
		Clopidogrel			Heparin	Valproate
						Warfarin
						Retinoid
						Lithium
						Danazol

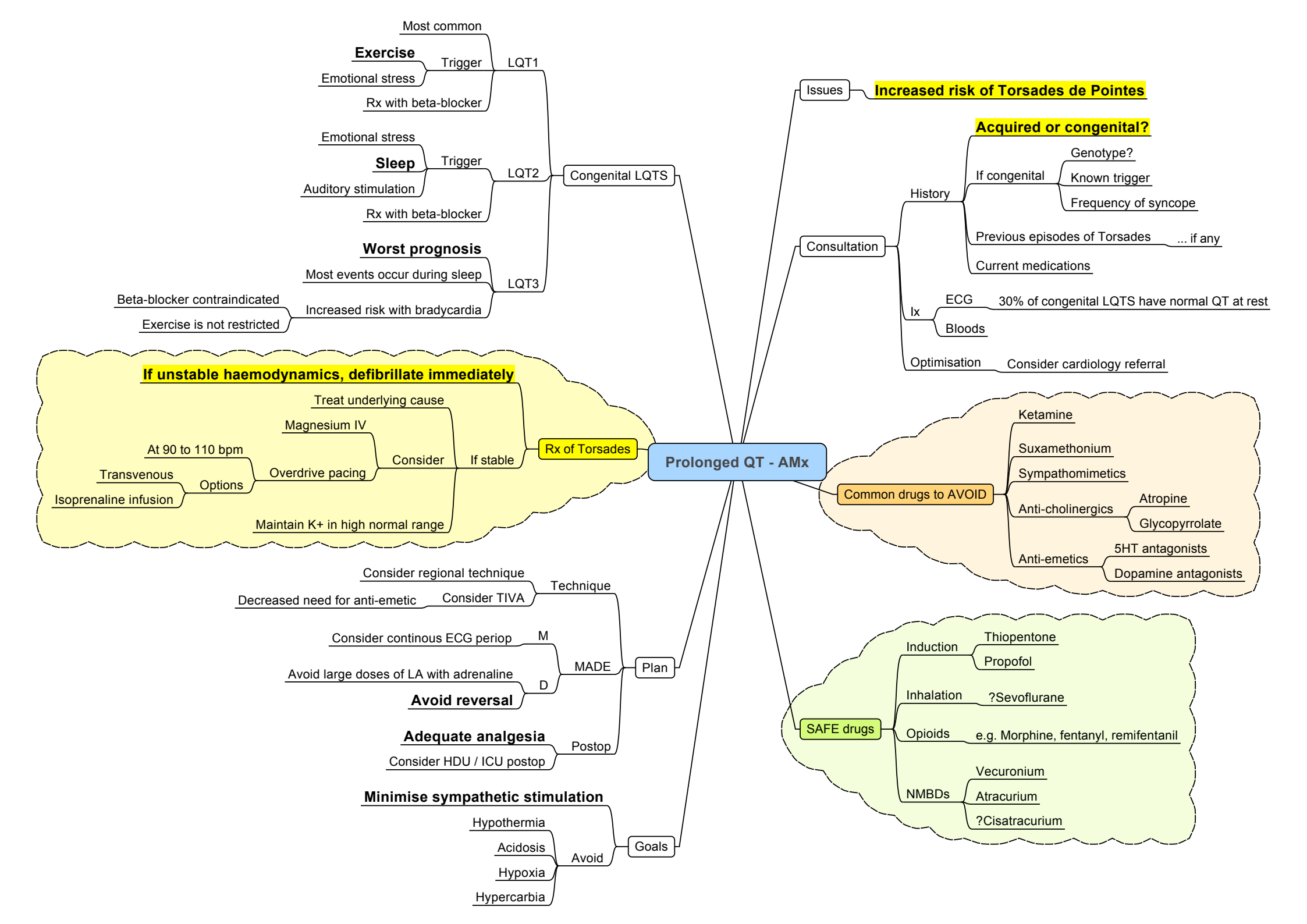

Pregnancy drug categories (Australian)
Background
Australian categorisation system for drug safety in pregnancy
Does not apply in
Overdose
Occupational exposure
Situations where recommended therapeutic dose has been exceeded
NOT hierarchical
Differ from US FDA categorisation
Subcategorisation of category B is based on animal data
Category B does NOT imply greater safety than category C
Category D drugs are not absolutely contraindicated
Due to legal concerns, some companies use a more restrictive cetagory
Categories
Category A
Drug taken by a LARGE number of women without proven foetal harm
Category B
Drug taken by only a LIMITED number of women without proven foetal harm
B1
No proven harm in animal studies
B2
Animal study lacking
B3
Proven harm in animal study, but uncertain significance in human
Category C
Harmful effects on foetus or neonate
May be proven or suspected
May be reversible
No malformation caused
Category D
Foetal malformation or irreversible damage on foetus or neonate
May be proven, suspected or expected
Use only in exceptional circumstances
Category X
Very high risk of permanent foetal harm
DO NOT USE in pregnancy or when there is a possibility of pregnancy

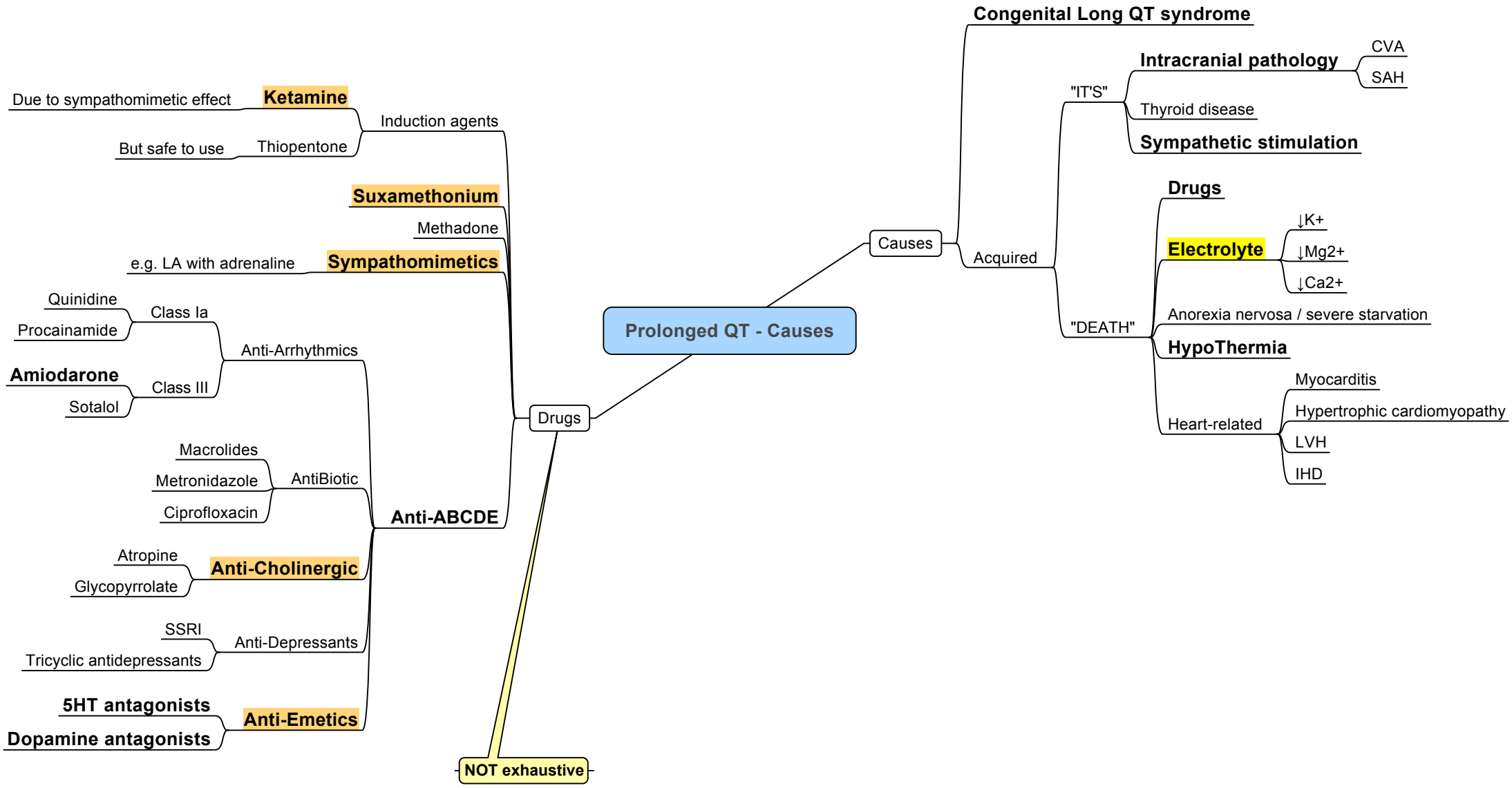

Pregnancy -
Drugs that affect uterine tone
Monitoring required during tocolytic administration
Mother
BP
HR
RR
Temp
CTG
Tocolytic
Reduce tone
Nifedipine
Recommended
20 mg PO
Onset at 30 to 60 min
Can repeated twice Q30min
Maintenance dose 20 to 40mg Q6H
Shown to delay delivery and improve perinatal outcome
Salbutamol
Infusion
Start at 10 mcg/min
Increase by 5 mcg/min Q10 min until response
Maximum rate 50 mcg/min
Significant side effects
Shown to delay delivery
But have not been shown to improve perinatal outcome
Halogenated volatile gas
Not N2O
GTN
50 to 100mcg IV boluses
Less effective than salbutamol
Uterotonic
Increase tone
Syntocinon / Oxytocin
IV bolus = 5 to 10 units
Followed by 5 to 10 units/hr
Carbetocin
Long-acting oxytocin agonist
IV dose = 100 mcg
Ergometrine
IM dose = 500mcg
IV dose = 200mcg
Misoprostol (PGE1 analogue)
Dose = 600 to 1000mcg
Given PR
Carboprost (PGF2alpha analogue)
IM directly into myometrium
Dose = 250mcg Q15min

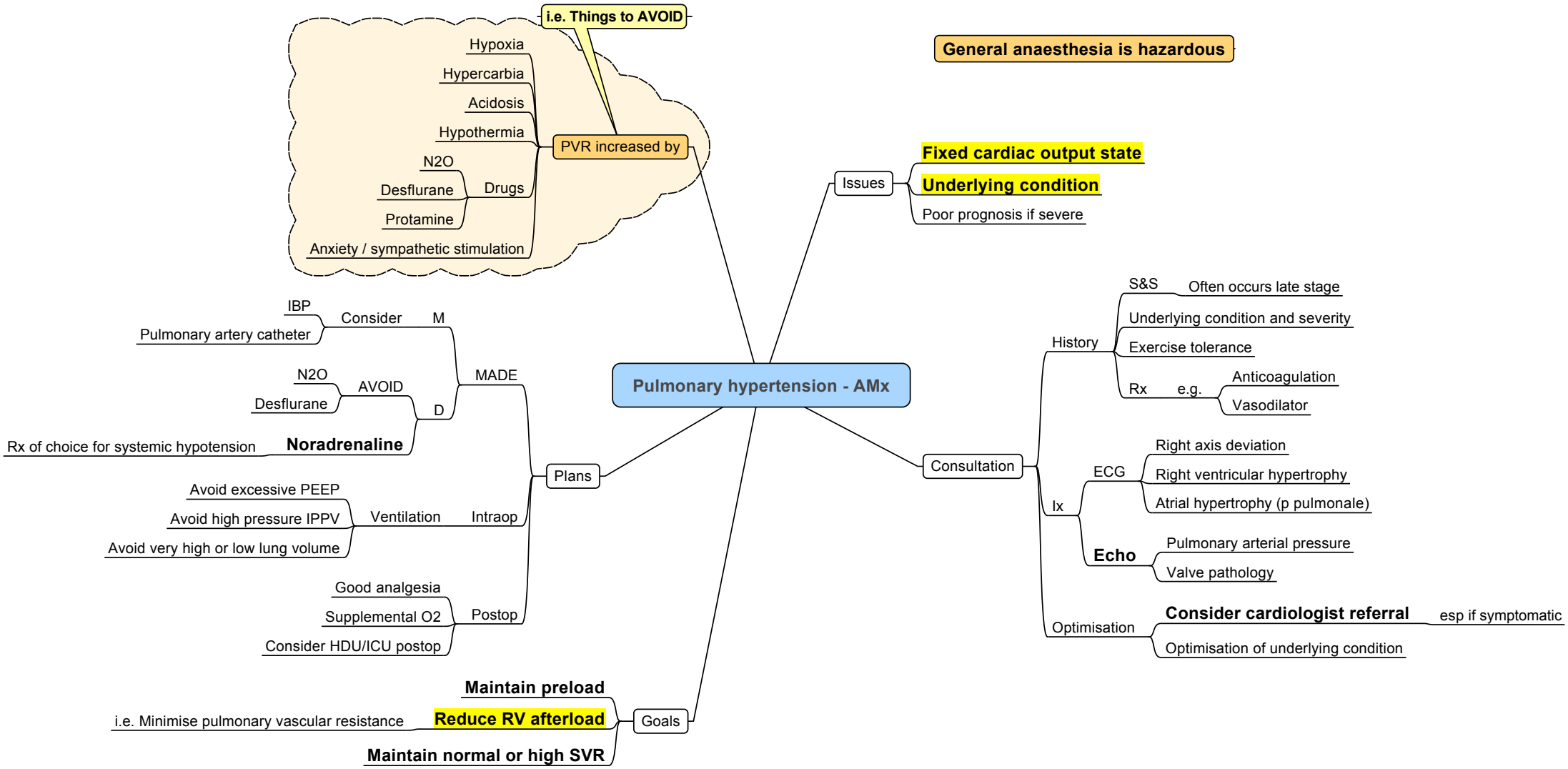

Pregnancy - Physiological changes
A+B
Difficult intubation more likely
1 in 300 at term
c.f. 1 in 2200 in non-pregnant women
Aspiration risk
Due to
Reduced lower oesophageal sphincter tone
Delayed gastric emptying
Pressure effect of the uterus
Always RSI after 20 weeks
Prone to hypoxia
FRC is reduced by 20% at term
Higher O2 consumption
+30% at term
Up to 60% in labour
Respiratory alkalosis
Due to progesterone stimulating respiratory centre
PaCO2 = 32mmHg by 12 weeks
Increased minute ventilation (+50% at term)
Capillary engorgement in respiratory tract
Prone to oedema
Higher airway pressure during IPPV
Reduced chest wall compliance
Increased abdominal pressure
C
Hyperdynamic state
Increased CO
Most of the increase occurs early
Peak increase (+50%) during 32-36 weeks
May precipitate HF and pulmonary oedema
Increased perfusion
esp skin, kidney, and uterus
Blood flow to brain and liver unchanged
Blood volume increased by 45 to 50%
Aorto-caval compression syndrome
Always use left tilt after 20 weeks
Downregulation of alpha and beta receptors
Increased dose of vasopressors required
Uterus perfusion
750mL/min at term (≈10-12% of CO)
85% goes to the placenta
Uteroplacental blood flow NOT autoregulated
D
Epidural pressure increased
Less LA required for both spinal (≈25%) and epidural
Preterm pregnancy will require more LA than at term
CSF pressure
CSF pressure not changed at rest
Increase to 70cm H2O during bearing-down efforts
Increased sensitivity
MAC of volatiles reduced by 30 to 40% at term
Nerves more sensitive to LA
Due to progesterone and oestrogen
Increased sensitivity to sedative effects of opioids and other drugs
Emotional changes
Memory loss
Reduced cognitive ability
Depression
E
Placental production
Progesterone
Human chorionic gonadotrophin (hCG)
Human placental lactogen (hPL)
Prolactin
Insulin resistance
Due to
hPL
Cortisol
Progresterone
Oestrogen
Occurs after 20 weeks
Others
Haematology
Physiologic anaemia of pregnancy
Plasma increases by 50%
RBC volume increases by 33%
Hb drops by 15-20%
Thromboembolism risk
Due to hypercoagulable state
Renal
GFR increased by 50%
Due to increased cardiac output
Urea and creatinine decreased by 40%
Proteinuria in 20%
Abnormal if > 260 mg in 24 hrs
In labour
Increased cardiac output (CO)
1st stage: 10 to 25%
2nd stage: 40%
Contractions: further 15%
CO peaks immediately AFTER delivery
Danger period for cardiac risk
O2 consumption increased by 60% due to uterine contractions

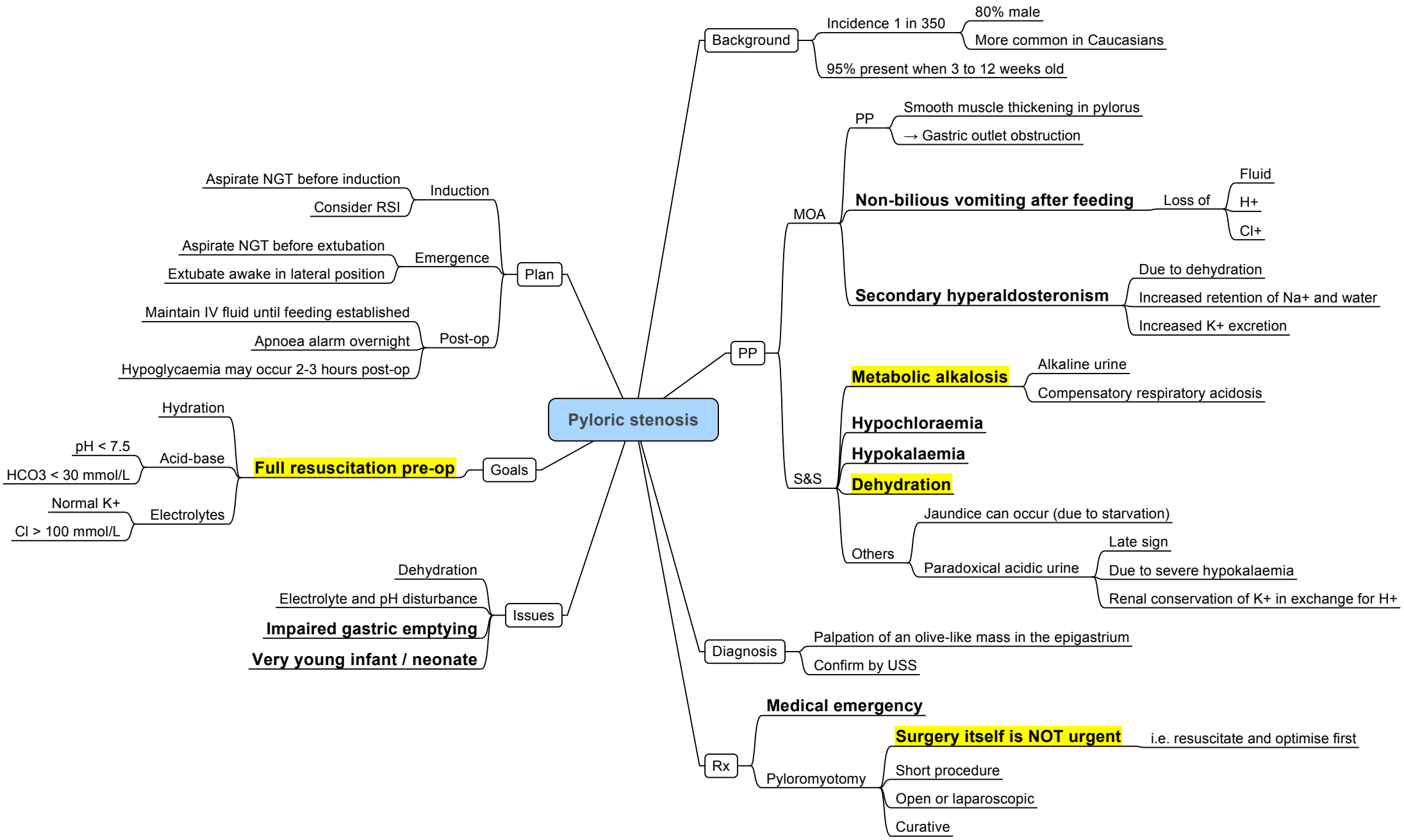

Prematurity - Issues
A+B
Apnoea of prematurity
Hypoxia
But need to avoid high FiO2
Lung complications
Respiratory distress syndrome
Bronchopulmonary dysplasia
C
Bradycardia occurs quickly with hypoxia
Dominant parasympathetic system
Difficult IV access, esp after prolonged stay
D
Intraventricular haemorrhage
Poor autoregulation of CBF
E
Hypoglycaemia
Anaemia
Altered coagulation
Thus vit K at delivery
Hypothermia
Decreased immunity
Others
Retinopathy of the prematurity
Necrotising enterocolitis


Prolonged operation - AMx
Issues
A+B
Increased atelectasis
C
Potentially large fluid shift
Hypothermia
E
Thromboembolism
Pressure area
Others
Staff fatigue
Plan
E
TED / SCD
Fluid warmer and forced warm air blanket
Protect pressure areas
Staged operation if appropriate

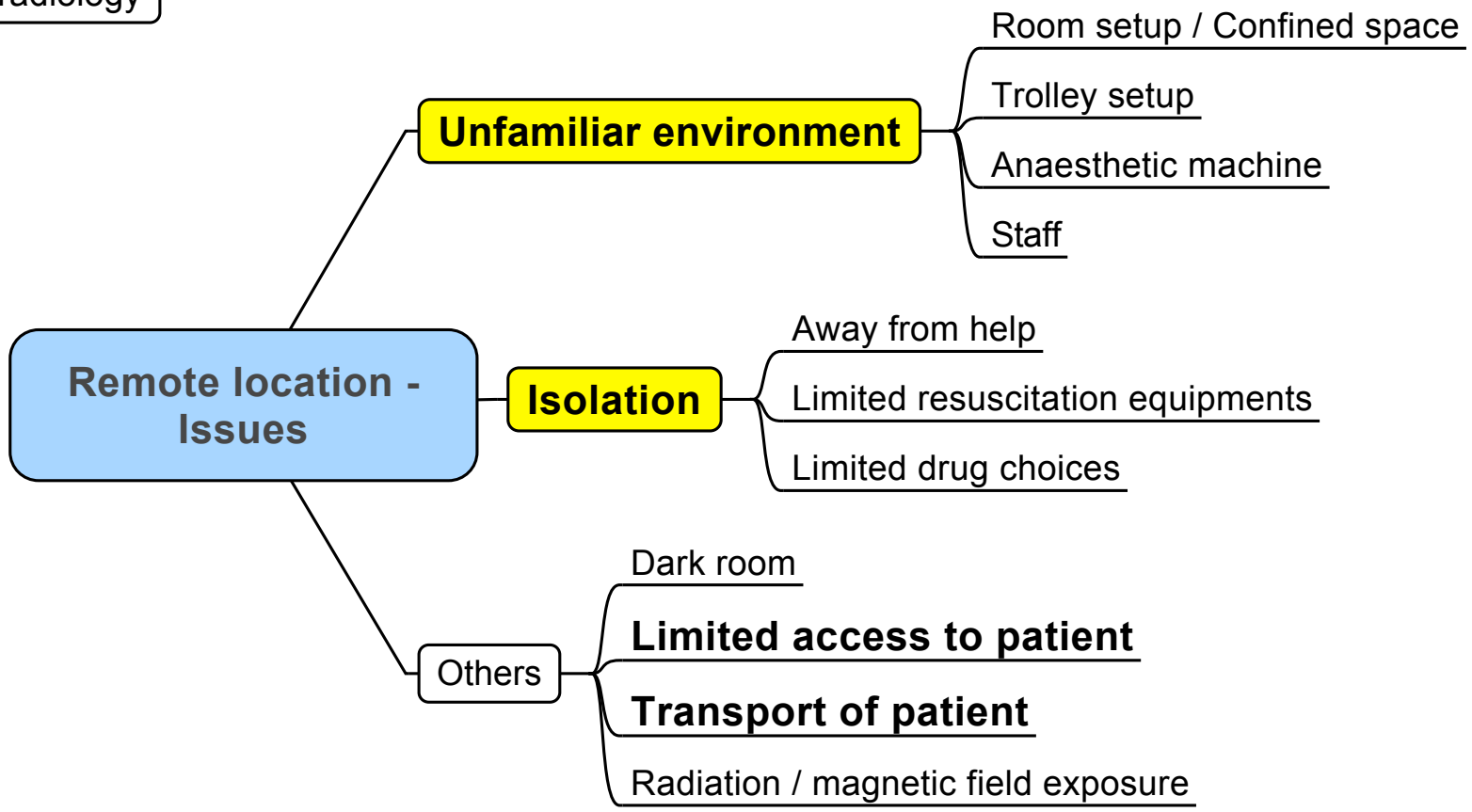

Prolonged QT - AMx
Congenital LQTS
LQT1
Most common
Trigger
Exercise
Emotional stress
Rx with beta-blocker
LQT2
Trigger
Emotional stress
Sleep
Auditory stimulation
Rx with beta-blocker
LQT3
Worst prognosis
Most events occur during sleep
Increased risk with bradycardia
Beta-blocker contraindicated
Exercise is not restricted
Issues
Increased risk of Torsades de Pointes
Consultation
History
Acquired or congenital?
If congenital
Genotype?
Known trigger
Frequency of syncope
Previous episodes of Torsades
... if any
Current medications
Ix
ECG
30% of congenital LQTS have normal QT at rest
Bloods
Optimisation
Consider cardiology referral
Rx of Torsades
If unstable haemodynamics, defibrillate immediately
If stable
Consider
Magnesium IV
Overdrive pacing
At 90 to 110 bpm
Options
Transvenous
Isoprenaline infusion
Treat underlying cause
Maintain K+ in high normal range
Common drugs to AVOID
Ketamine
Suxamethonium
Sympathomimetics
Anti-cholinergics
Atropine
Glycopyrrolate
Anti-emetics
5HT antagonists
Dopamine antagonists
Plan
Technique
Consider regional technique
Consider TIVA
Decreased need for anti-emetic
MADE
M
Consider continous ECG periop
D
Avoid large doses of LA with adrenaline
Avoid reversal
Postop
Adequate analgesia
Consider HDU / ICU postop
SAFE drugs
Induction
Thiopentone
Propofol
Inhalation
?Sevoflurane
Opioids
e.g. Morphine, fentanyl, remifentanil
NMBDs
Vecuronium
Atracurium
?Cisatracurium
Goals
Minimise sympathetic stimulation
Avoid
Hypothermia
Acidosis
Hypoxia
Hypercarbia

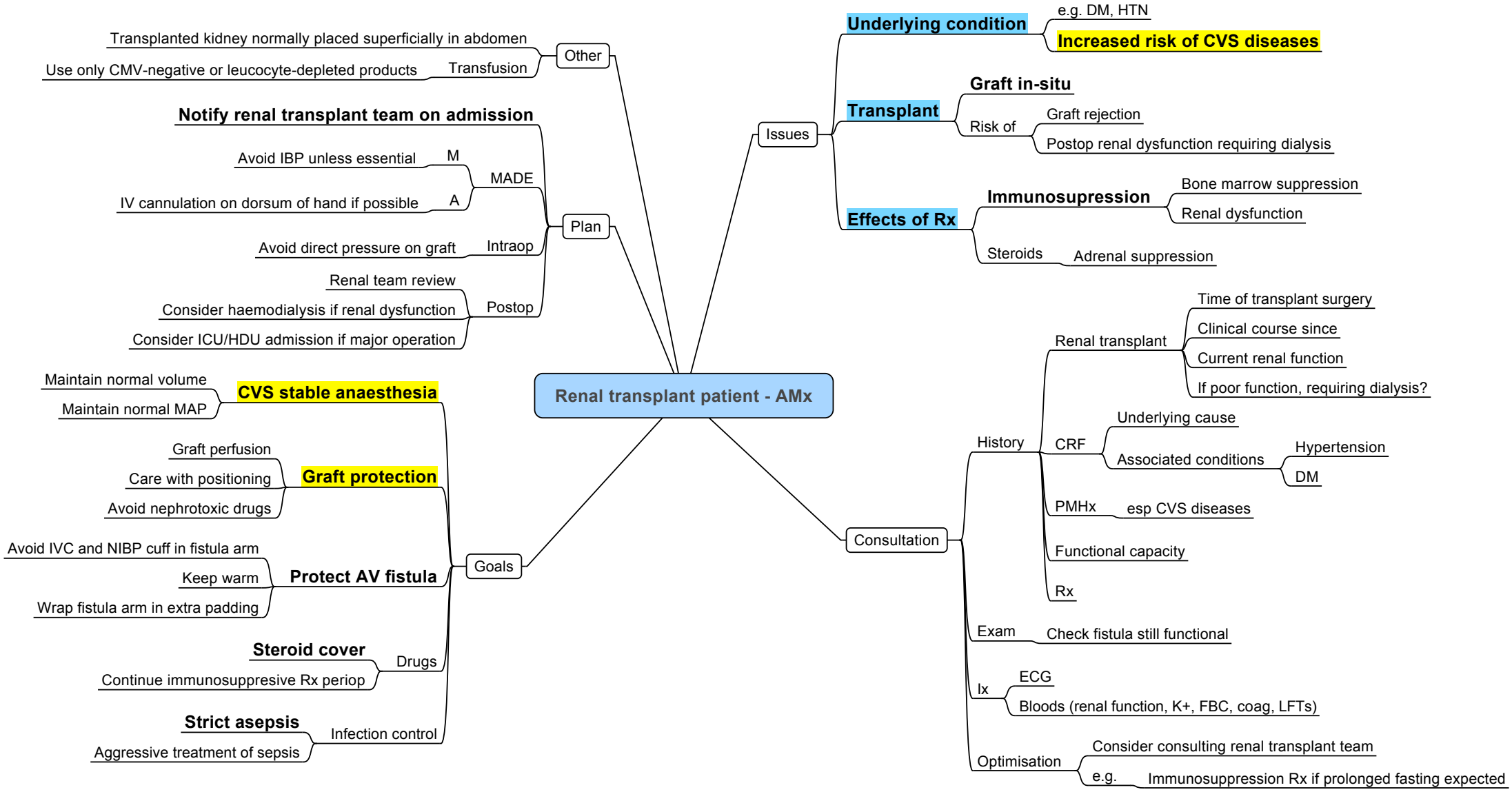

Prolonged QT - Causes
Causes
Congenital Long QT syndrome
Acquired
"IT'S"
Intracranial pathology
CVA
SAH
Thyroid disease
Sympathetic stimulation
"DEATH"
Drugs
Electrolyte
↓K+
↓Mg2+
↓Ca2+
Anorexia nervosa / severe starvation
HypoThermia
Heart-related
Myocarditis
Hypertrophic cardiomyopathy
LVH
IHD
Drugs
Induction agents
Due to sympathomimetic effect
Ketamine
But safe to use
Thiopentone
Suxamethonium
Methadone
e.g. LA with adrenaline
Sympathomimetics
Anti-ABCDE
Anti-Arrhythmics
Class Ia
Quinidine
Procainamide
Class III
Amiodarone
Sotalol
AntiBiotic
Macrolides
Metronidazole
Ciprofloxacin
Anti-Cholinergic
Atropine
Glycopyrrolate
Anti-Depressants
SSRI
Tricyclic antidepressants
Anti-Emetics
5HT antagonists
Dopamine antagonists
NOT exhaustive


Pulmonary hypertension - AMx
General anaesthesia is hazardous
Issues
Fixed cardiac output state
Underlying condition
Poor prognosis if severe
i.e. Things to AVOID
PVR increased by
Hypoxia
Hypercarbia
Acidosis
Hypothermia
Drugs
N2O
Desflurane
Protamine
Anxiety / sympathetic stimulation
Consultation
History
S&S
Often occurs late stage
Underlying condition and severity
Exercise tolerance
Rx
e.g.
Anticoagulation
Vasodilator
Ix
ECG
Right axis deviation
Right ventricular hypertrophy
Atrial hypertrophy (p pulmonale)
Echo
Pulmonary arterial pressure
Valve pathology
Optimisation
Consider cardiologist referral
esp if symptomatic
Optimisation of underlying condition
Plans
MADE
M
Consider
IBP
Pulmonary artery catheter
A
AVOID
N2O
Desflurane
D
Noradrenaline
Rx of choice for systemic hypotension
Intraop
Ventilation
Avoid excessive PEEP
Avoid high pressure IPPV
Avoid very high or low lung volume
Postop
Good analgesia
Supplemental O2
Consider HDU/ICU postop
Goals
Maintain preload
Reduce RV afterload
i.e. Minimise pulmonary vascular resistance
Maintain normal or high SVR

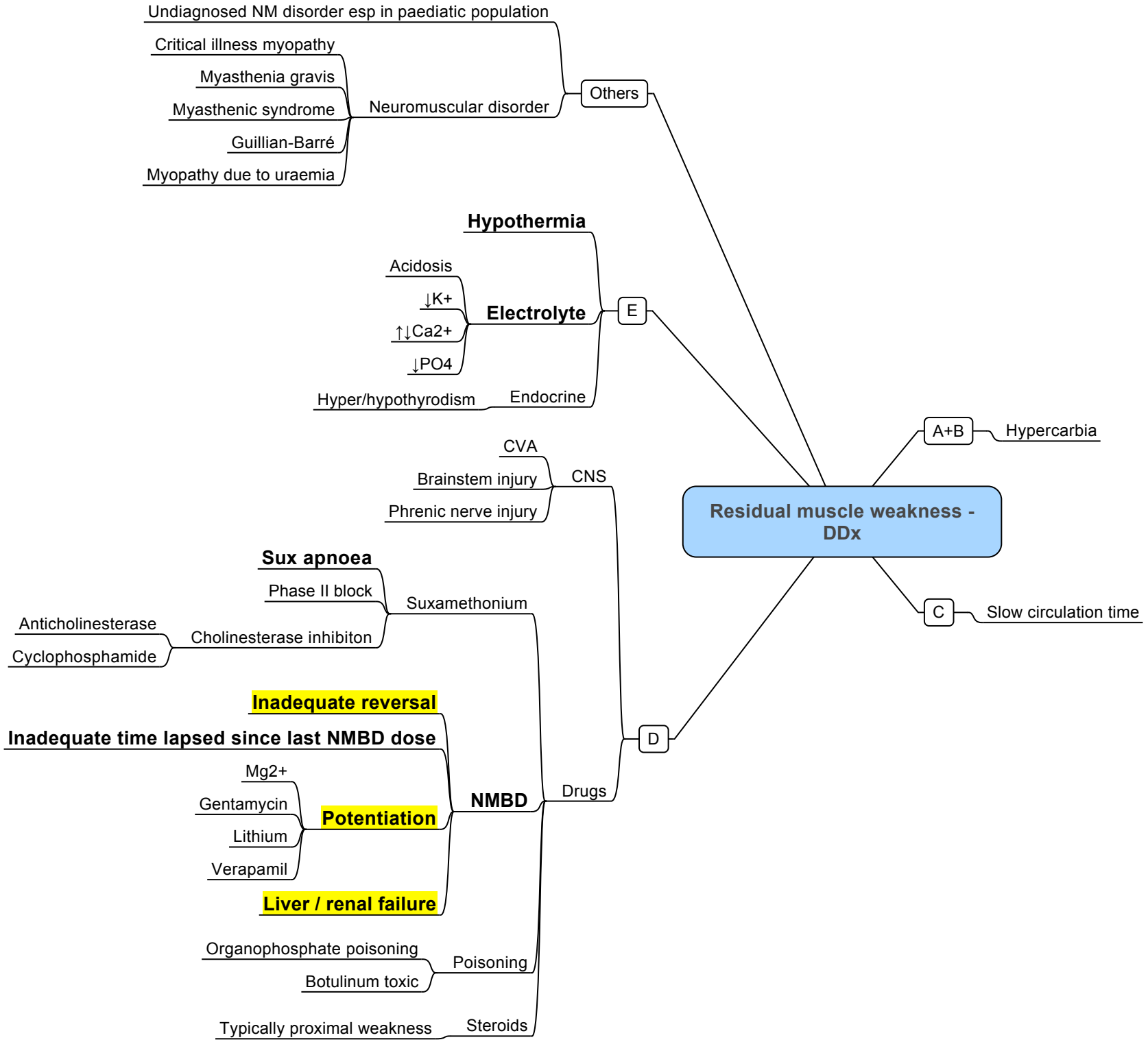

Pyloric stenosis
Background
Incidence 1 in 350
80% male
More common in Caucasians
95% present when 3 to 12 weeks old
PP
MOA
PP
Smooth muscle thickening in pylorus
→ Gastric outlet obstruction
Non-bilious vomiting after feeding
Loss of
Fluid
H+
Cl+
Secondary hyperaldosteronism
Due to dehydration
Increased retention of Na+ and water
Increased K+ excretion
S&S
Metabolic alkalosis
Alkaline urine
Compensatory respiratory acidosis
Hypochloraemia
Hypokalaemia
Dehydration
Others
Jaundice can occur (due to starvation)
Paradoxical acidic urine
Late sign
Due to severe hypokalaemia
Renal conservation of K+ in exchange for H+
Diagnosis
Palpation of an olive-like mass in the epigastrium
Confirm by USS
Rx
Medical emergency
Surgery itself is NOT urgent
i.e. resuscitate and optimise first
Pyloromyotomy
Short procedure
Open or laparoscopic
Curative
Issues
Dehydration
Electrolyte and pH disturbance
Impaired gastric emptying
Very young infant / neonate
Goals
Full resuscitation pre-op
Hydration
Acid-base
pH < 7.5
HCO3 < 30 mmol/L
Electrolytes
Normal K+
Cl > 100 mmol/L
Plan
Induction
Aspirate NGT before induction
Consider RSI
Emergence
Aspirate NGT before extubation
Extubate awake in lateral position
Post-op
Maintain IV fluid until feeding established
Apnoea alarm overnight
Hypoglycaemia may occur 2-3 hours post-op

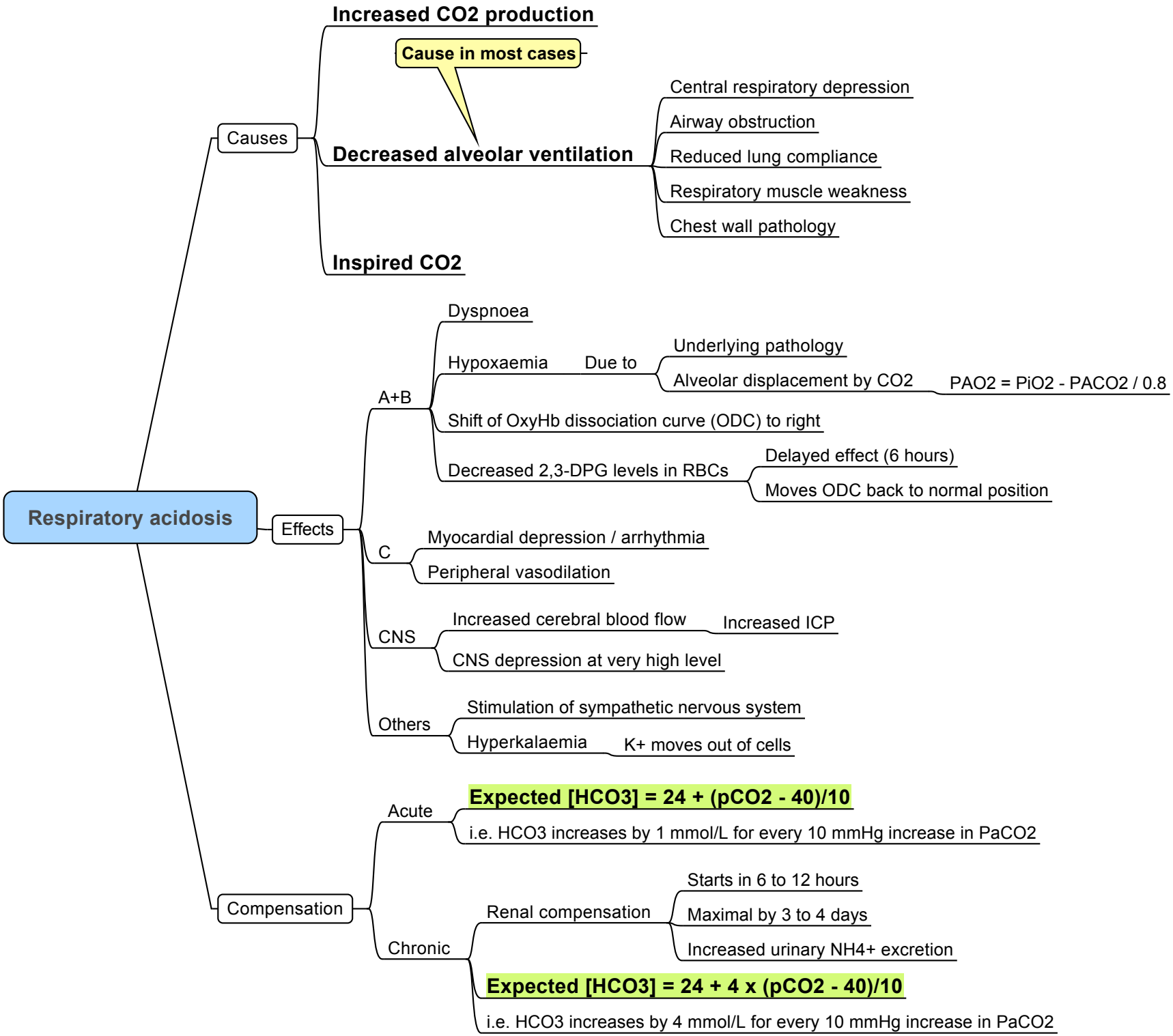
e.g. Cathlab, MRI, Interventional radiology

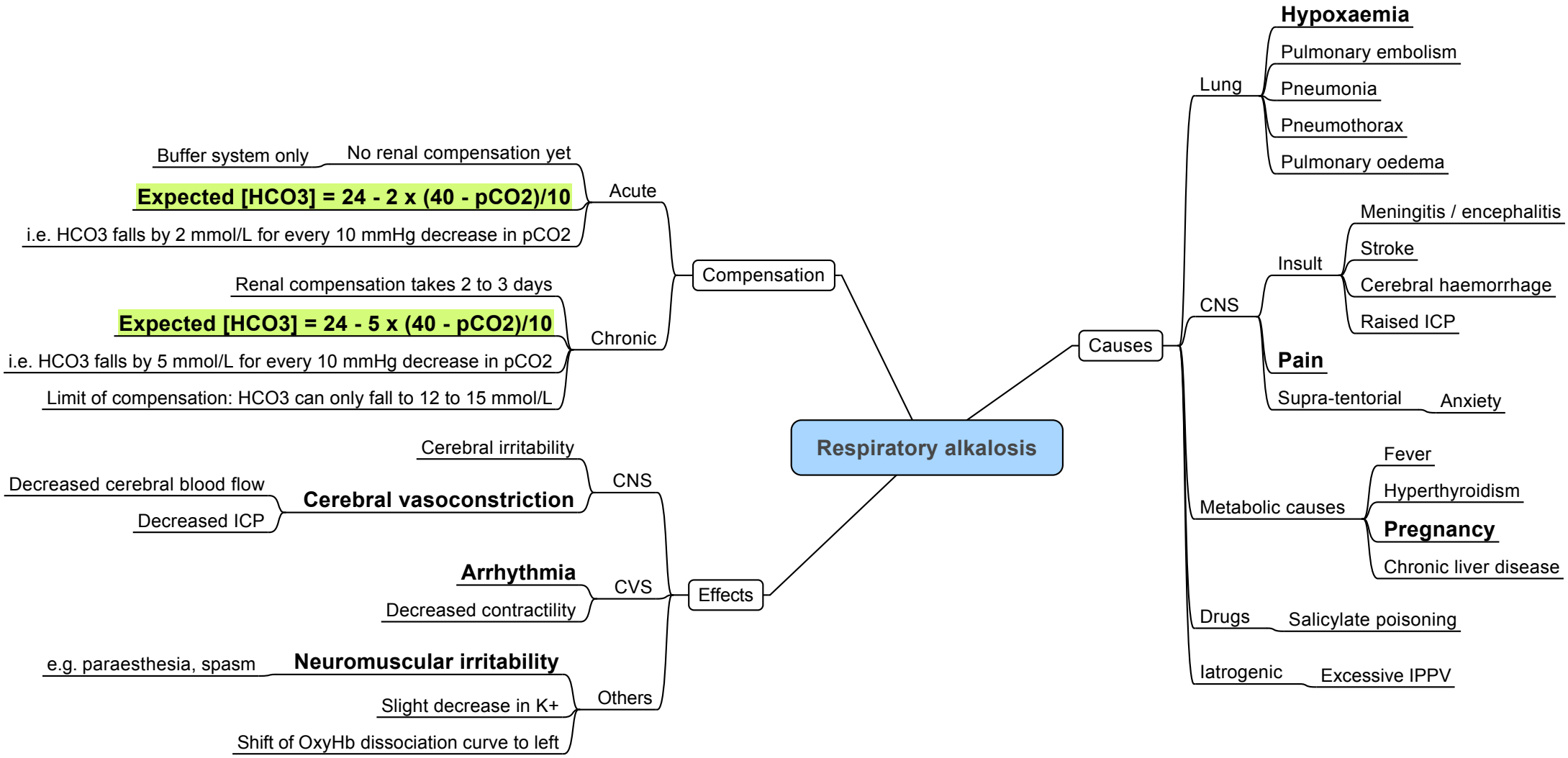


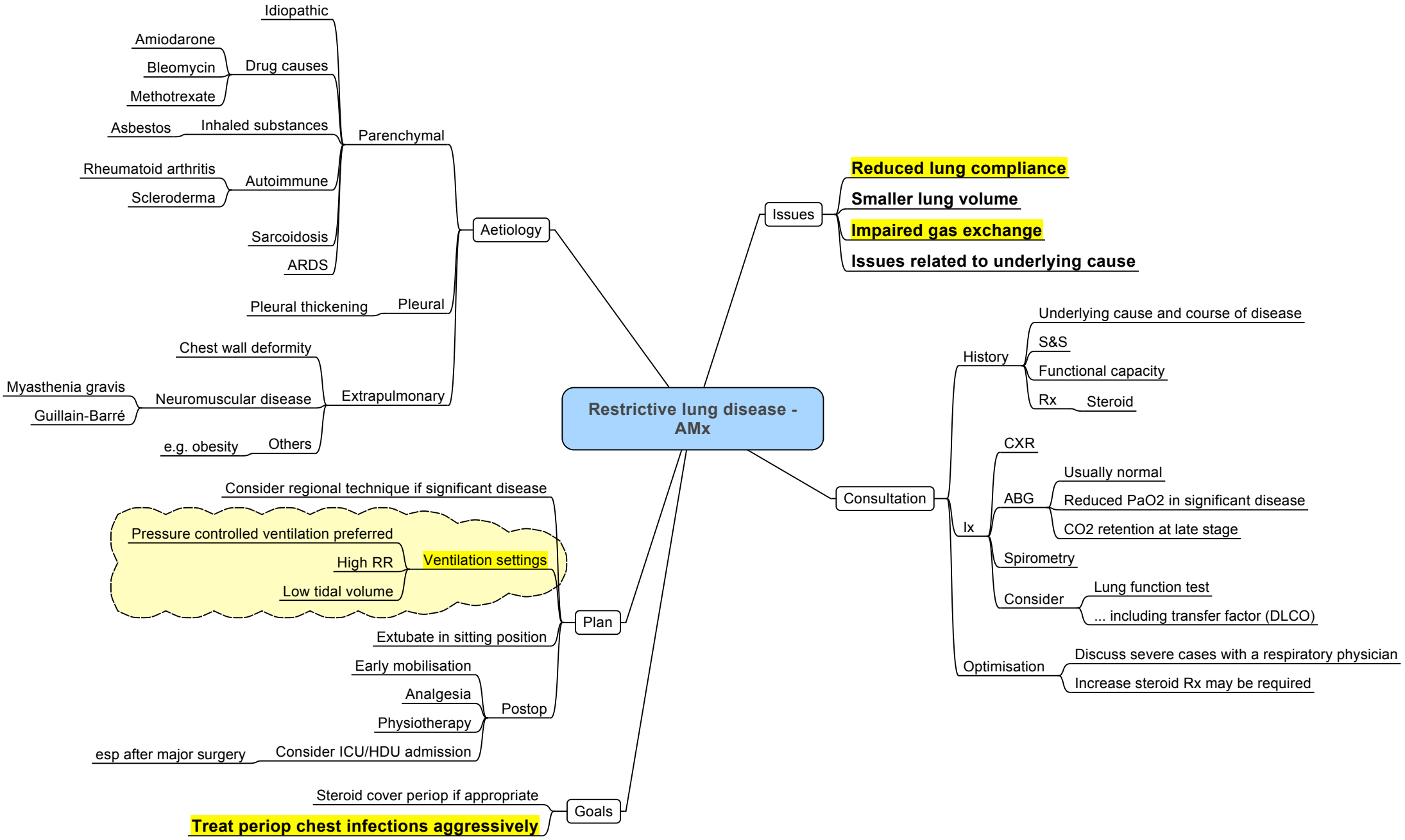

Renal transplant patient - AMx
Consultation
History
Renal transplant
Time of transplant surgery
Clinical course since
Current renal function
If poor function, requiring dialysis?
CRF
Underlying cause
Associated conditions
Hypertension
DM
PMHx
esp CVS diseases
Functional capacity
Rx
Exam
Check fistula still functional
Ix
ECG
Bloods (renal function, K+, FBC, coag, LFTs)
Optimisation
Consider consulting renal transplant team
e.g.
Immunosuppression Rx if prolonged fasting expected
Issues
Underlying condition
e.g. DM, HTN
Increased risk of CVS diseases
Transplant
Graft in-situ
Risk of
Graft rejection
Postop renal dysfunction requiring dialysis
Effects of Rx
Immunosupression
Bone marrow suppression
Renal dysfunction
Steroids
Adrenal suppression
Goals
CVS stable anaesthesia
Maintain normal volume
Maintain normal MAP
Graft protection
Graft perfusion
Care with positioning
Avoid nephrotoxic drugs
Protect AV fistula
Avoid IVC and NIBP cuff in fistula arm
Keep warm
Wrap fistula arm in extra padding
Drugs
Steroid cover
Continue immunosuppresive Rx periop
Infection control
Strict asepsis
Aggressive treatment of sepsis
Plan
MADE
M
Avoid IBP unless essential
A
IV cannulation on dorsum of hand if possible
Intraop
Avoid direct pressure on graft
Postop
Renal team review
Consider haemodialysis if renal dysfunction
Consider ICU/HDU admission if major operation
Notify renal transplant team on admission
Other
Transfusion
Transplanted kidney normally placed superficially in abdomen
Use only CMV-negative or leucocyte-depleted products

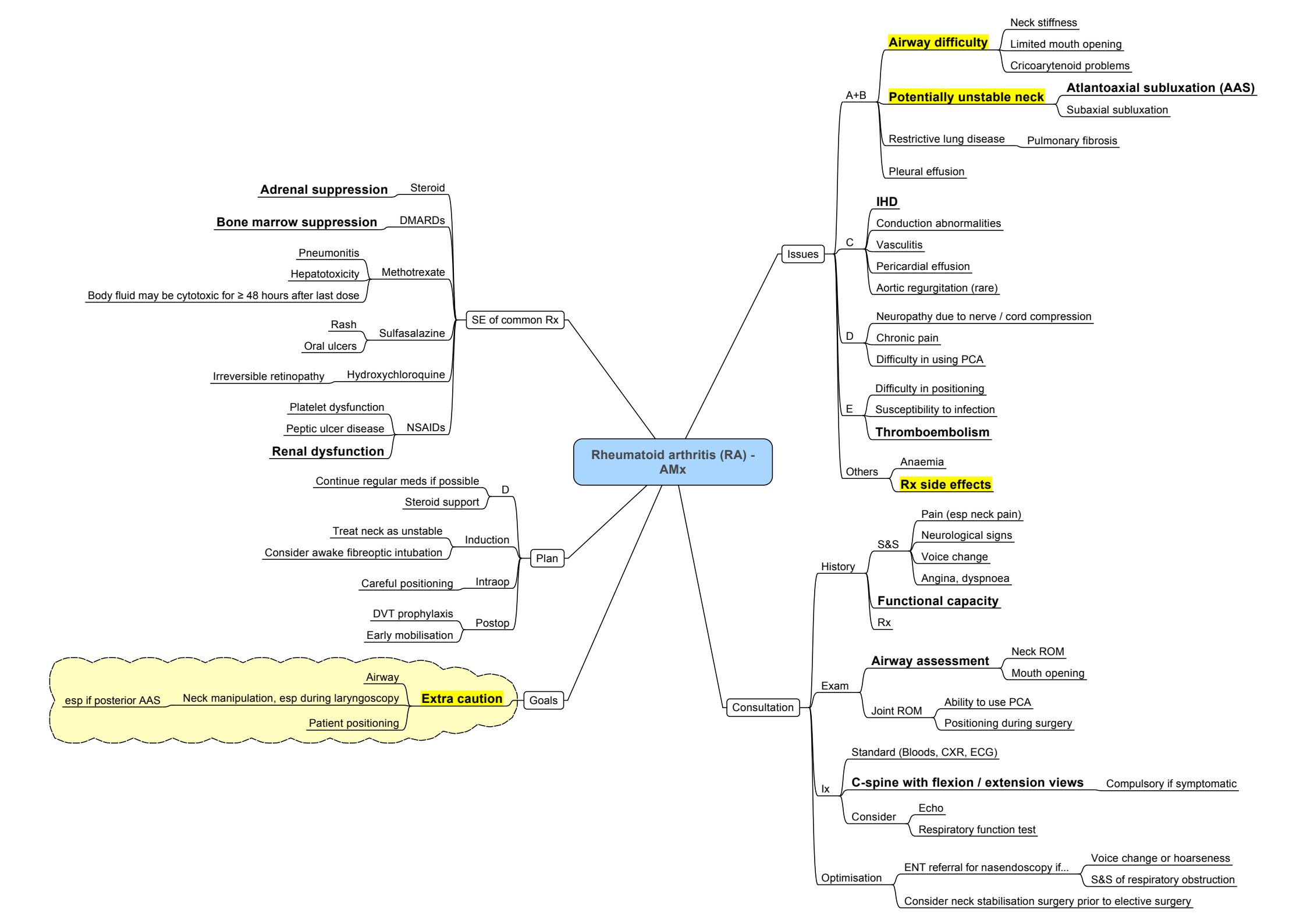

Renal transplant surgery
Plan
MADE
M
CVP
A
Large bore IV
CVL
Generally can be done after induction
D
Antibiotic cover
e.g. cephazolin 1g
Induction
Avoid suxamethonium if hyperkalaemia
Intraop
IV fluid until CVP target reached
Surgical requests
Diuretics
Mannitol
0.5 g/kg
Frusemide
40-80 mg IV
?Dopamine
Inflate bladder
To confirm ureter
Clamp IDC and fill bladder with NS
(± methylene blue)
Extubation
Ensure adequate recovery of NMJ
Post-op
CXR
To exclude pneumothorax, pulmonary oedema
Fentanyl PCA
Renal review postop
esp IV fluid titration
Goals
Graft survival
Early urine output
Saline diuresis
Diuretics
Graft perfusion
Maintain normal to high MAP
?Dopamine
Avoid nephrotoxic drugs
Protect AV fistula
Avoid IVC and NIBP cuff in fistula arm
Keep warm
Wrap fistula arm in extra padding
Maintain high preload
Target CVP 10 to 12mmHg
Often will need 50 to 100 mL/kg of fluids
Treat hypotension with IV fluids
Avoid vasopressor if possible
Consider inotropic agent
Issues
Emergency surgery
But usually have had extensive pre-op workup
ESRF
Including
C
IHD
Periop CVS instability
D
Autonomic dysfunction
E
Electrolyte disturbances
Anaemia (normochromic, normocytic)
Coagulopathy
esp platelet dysfunction
Decreased immunity
Others
GORD
Altered drug pharmacology
Normally on dialysis
Consultation
History
Changes since pre-op workup
Underlying cause leading to ESRF
Dialysis
Vascular access (location and type)
Frequency of dialysis
Time of last dialysis
Dry weight
PMHx
CVS disease
DM
Exercise tolerance
Fasting time
Ix
Check K+ and Hb
Aim for
K+ < 6 mmol/L
pH > 7.25
ECG
Anti-rejection regime
Charted by transplant team
Ensure Rx has been given
e.g.
Prednisone
Cyclosporin
Azothioprine

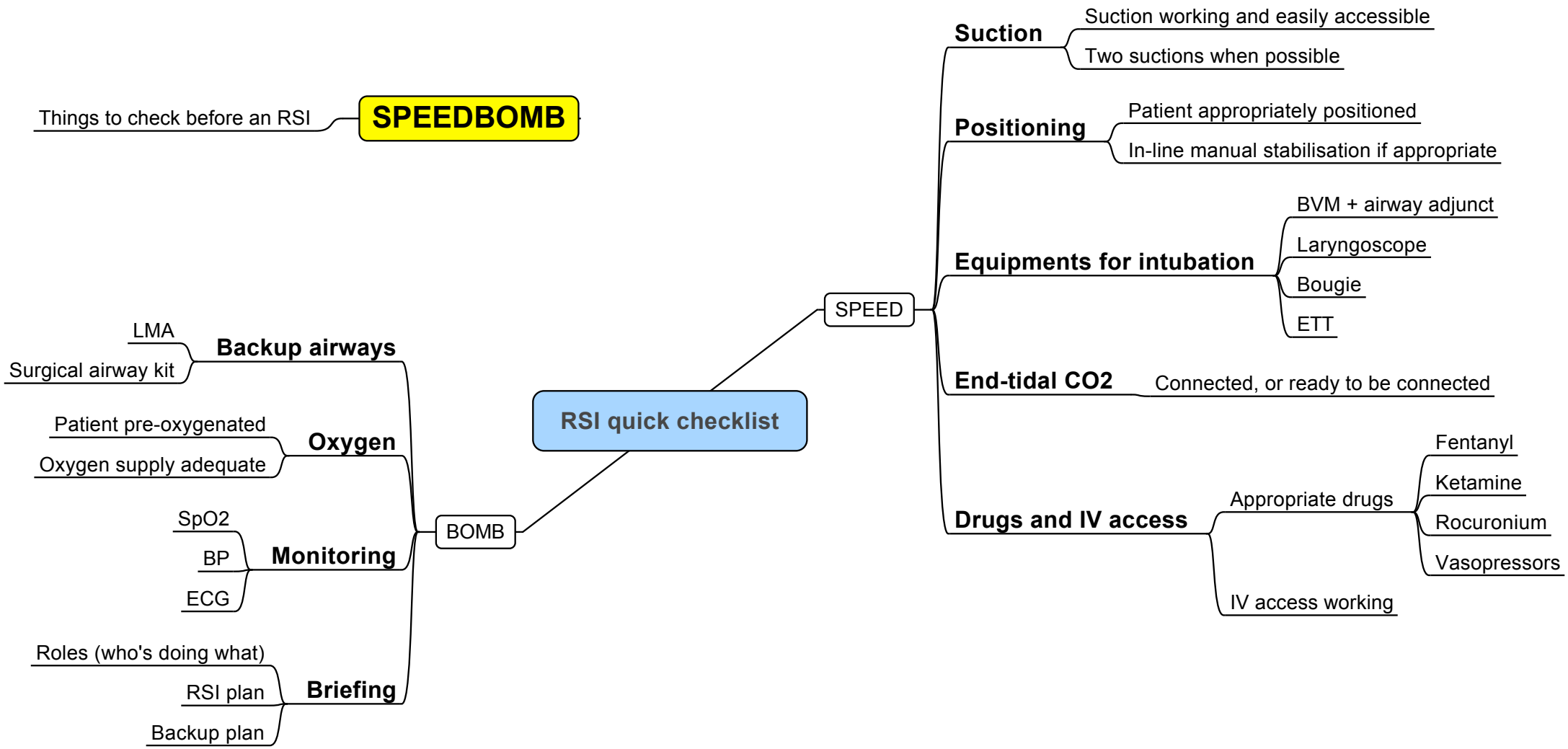

Residual muscle weakness - DDx
A+B
Hypercarbia
C
Slow circulation time
D
Drugs
Others
Undiagnosed NM disorder esp in paediatic population
Neuromuscular disorder
Critical illness myopathy
Myasthenia gravis
Myasthenic syndrome
Guillian-Barré
Myopathy due to uraemia
E
Hypothermia
Electrolyte
Acidosis
↓K+
↑↓Ca2+
↓PO4
Endocrine
Hyper/hypothyrodism
CNS
CVA
Brainstem injury
Phrenic nerve injury
Suxamethonium
Sux apnoea
Phase II block
Cholinesterase inhibiton
Anticholinesterase
Cyclophosphamide
NMBD
Inadequate reversal
Inadequate time lapsed since last NMBD dose
Potentiation
Mg2+
Gentamycin
Lithium
Verapamil
Liver / renal failure
Poisoning
Organophosphate poisoning
Botulinum toxic
Steroids
Typically proximal weakness

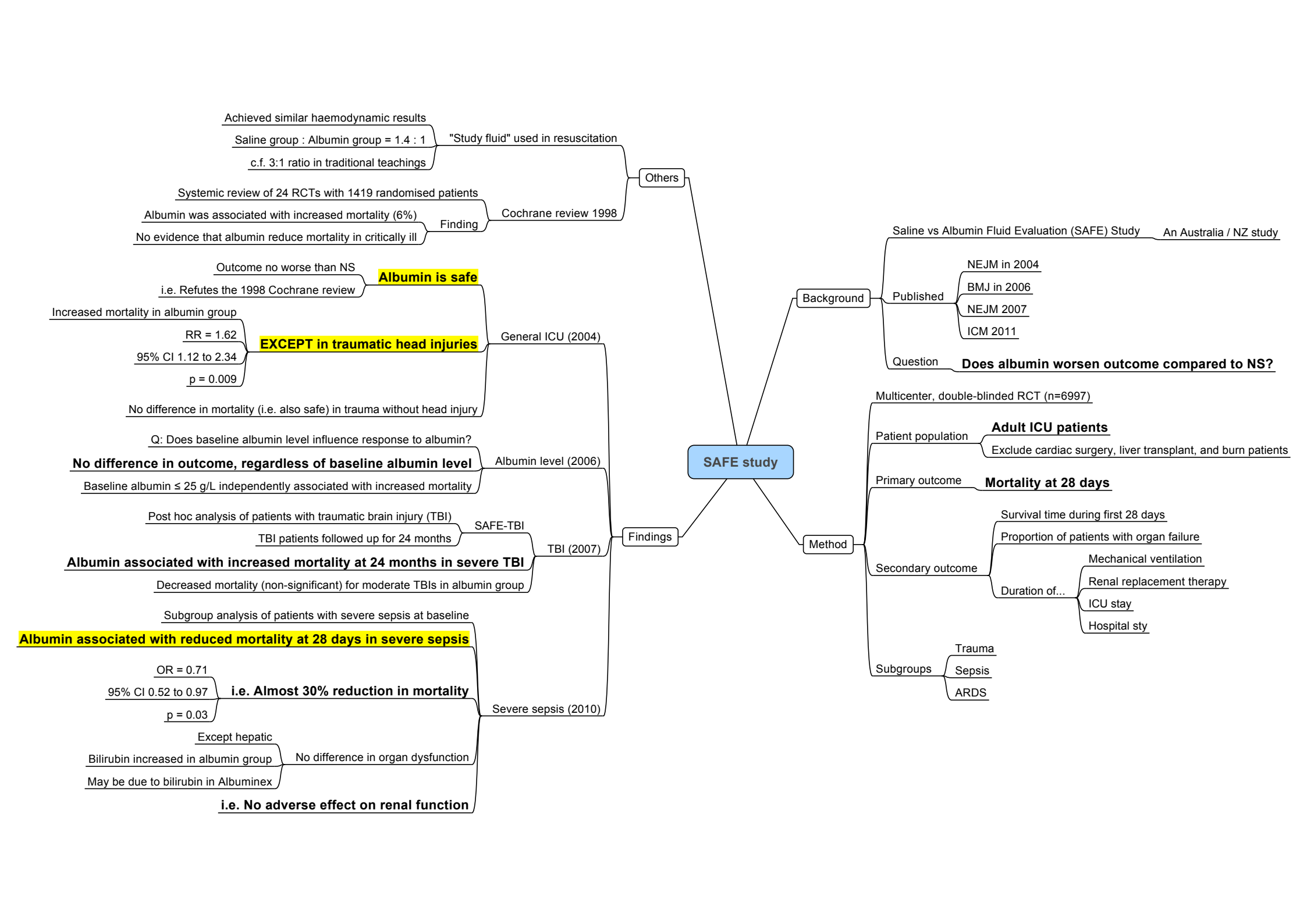

Respiratory acidosis
Causes
Increased CO2 production
Cause in most cases
Decreased alveolar ventilation
Central respiratory depression
Airway obstruction
Reduced lung compliance
Respiratory muscle weakness
Chest wall pathology
Inspired CO2
Effects
A+B
Dyspnoea
Hypoxaemia
Due to
Underlying pathology
Alveolar displacement by CO2
PAO2 = PiO2 - PACO2 / 0.8
Shift of OxyHb dissociation curve (ODC) to right
Decreased 2,3-DPG levels in RBCs
Delayed effect (6 hours)
Moves ODC back to normal position
C
Myocardial depression / arrhythmia
Peripheral vasodilation
CNS
Increased cerebral blood flow
Increased ICP
CNS depression at very high level
Others
Stimulation of sympathetic nervous system
Hyperkalaemia
K+ moves out of cells
Compensation
Acute
Expected [HCO3] = 24 + (pCO2 - 40)/10
i.e. HCO3 increases by 1 mmol/L for every 10 mmHg increase in PaCO2
Chronic
Renal compensation
Starts in 6 to 12 hours
Maximal by 3 to 4 days
Increased urinary NH4+ excretion
Expected [HCO3] = 24 + 4 x (pCO2 - 40)/10
i.e. HCO3 increases by 4 mmol/L for every 10 mmHg increase in PaCO2

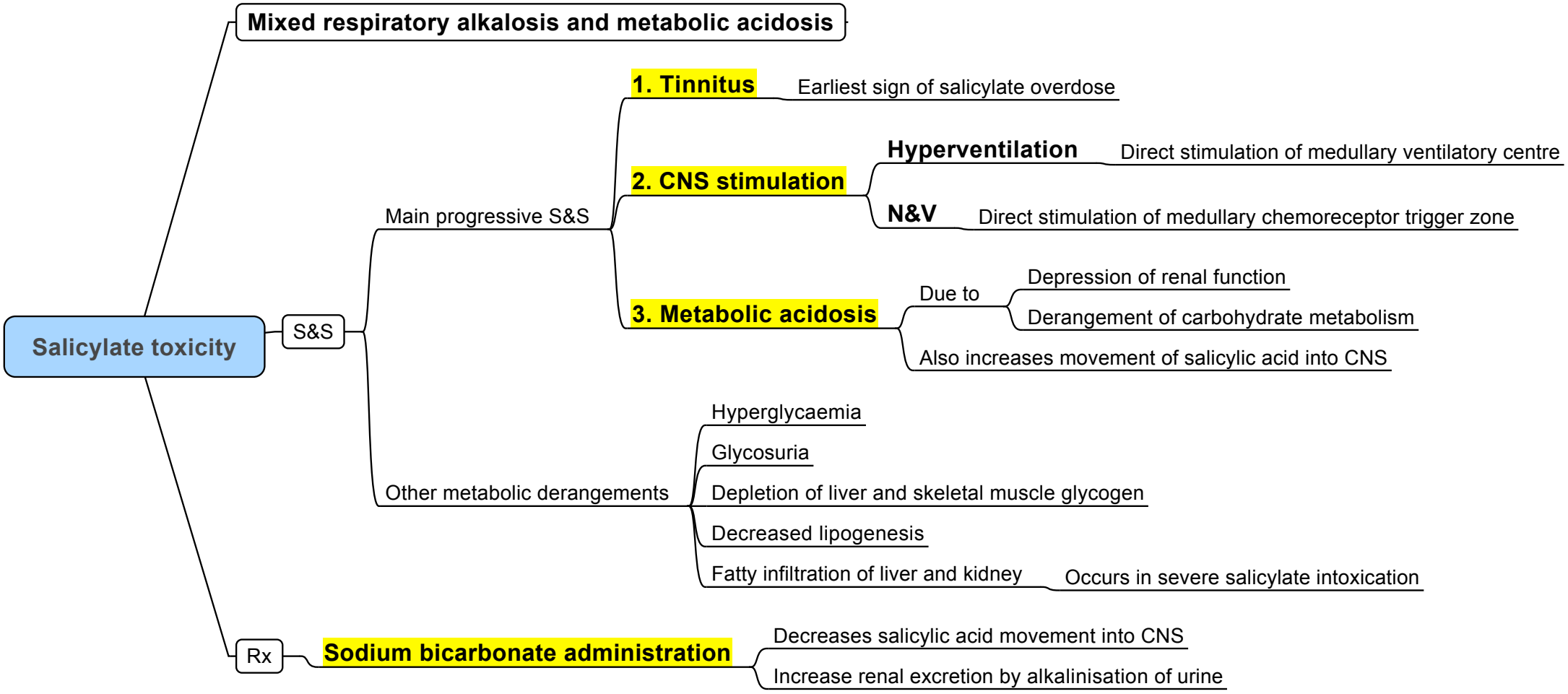

Respiratory alkalosis
Causes
Lung
Hypoxaemia
Pulmonary embolism
Pneumonia
Pneumothorax
Pulmonary oedema
CNS
Insult
Meningitis / encephalitis
Stroke
Cerebral haemorrhage
Raised ICP
Pain
Supra-tentorial
Anxiety
Metabolic causes
Fever
Hyperthyroidism
Pregnancy
Chronic liver disease
Drugs
Salicylate poisoning
Iatrogenic
Excessive IPPV
Compensation
Acute
Buffer system only
No renal compensation yet
Expected [HCO3] = 24 - 2 x (40 - pCO2)/10
i.e. HCO3 falls by 2 mmol/L for every 10 mmHg decrease in pCO2
Chronic
Renal compensation takes 2 to 3 days
Expected [HCO3] = 24 - 5 x (40 - pCO2)/10
i.e. HCO3 falls by 5 mmol/L for every 10 mmHg decrease in pCO2
Limit of compensation: HCO3 can only fall to 12 to 15 mmol/L
Effects
CNS
Cerebral irritability
Cerebral vasoconstriction
Decreased cerebral blood flow
Decreased ICP
CVS
Arrhythmia
Decreased contractility
Others
Neuromuscular irritability
e.g. paraesthesia, spasm
Slight decrease in K+
Shift of OxyHb dissociation curve to left


Restrictive lung disease - AMx
Aetiology
Parenchymal
Idiopathic
Drug causes
Amiodarone
Bleomycin
Methotrexate
Inhaled substances
Asbestos
Autoimmune
Rheumatoid arthritis
Scleroderma
Sarcoidosis
ARDS
Pleural
Pleural thickening
Extrapulmonary
Chest wall deformity
Neuromuscular disease
Myasthenia gravis
Guillain-Barré
Others
e.g. obesity
Issues
Reduced lung compliance
Smaller lung volume
Impaired gas exchange
Issues related to underlying cause
Consultation
History
Underlying cause and course of disease
S&S
Functional capacity
Rx
Steroid
Ix
CXR
ABG
Usually normal
Reduced PaO2 in significant disease
CO2 retention at late stage
Spirometry
Consider
Lung function test
... including transfer factor (DLCO)
Optimisation
Discuss severe cases with a respiratory physician
Increase steroid Rx may be required
Plan
Consider regional technique if significant disease
Ventilation settings
Pressure controlled ventilation preferred
High RR
Low tidal volume
Extubate in sitting position
Postop
Early mobilisation
Analgesia
Physiotherapy
Consider ICU/HDU admission
esp after major surgery
Goals
Steroid cover periop if appropriate
Treat periop chest infections aggressively

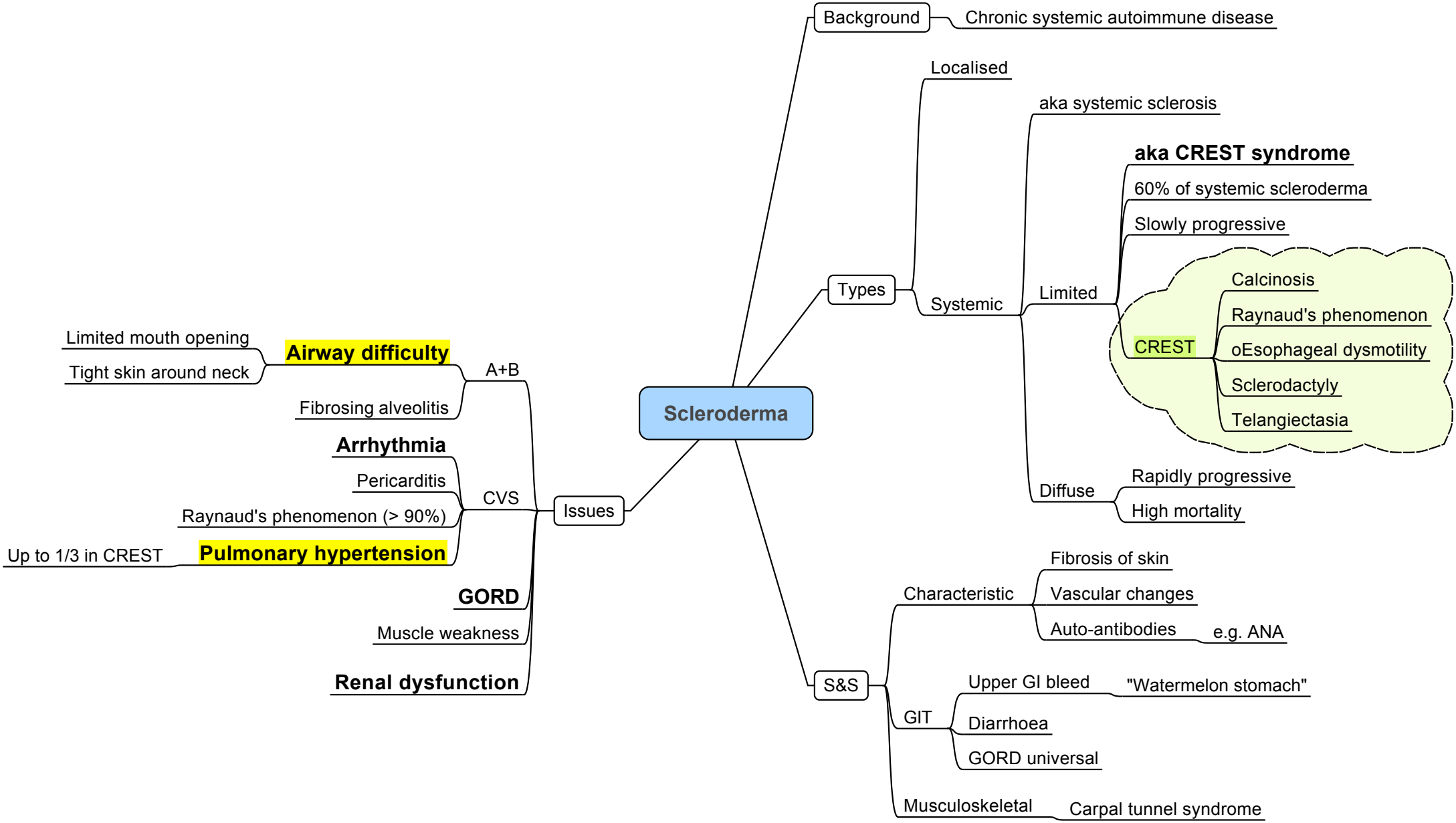

Rheumatoid arthritis (RA) - AMx
Issues
A+B
Airway difficulty
Neck stiffness
Limited mouth opening
Cricoarytenoid problems
Potentially unstable neck
Atlantoaxial subluxation (AAS)
Subaxial subluxation
Restrictive lung disease
Pulmonary fibrosis
Pleural effusion
C
IHD
Conduction abnormalities
Vasculitis
Pericardial effusion
Aortic regurgitation (rare)
D
Neuropathy due to nerve / cord compression
Chronic pain
Difficulty in using PCA
E
Difficulty in positioning
Susceptibility to infection
Thromboembolism
Others
Anaemia
Rx side effects
SE of common Rx
Steroid
Adrenal suppression
DMARDs
Bone marrow suppression
Methotrexate
Pneumonitis
Hepatotoxicity
Body fluid may be cytotoxic for ≥ 48 hours after last dose
Sulfasalazine
Rash
Oral ulcers
Hydroxychloroquine
Irreversible retinopathy
NSAIDs
Platelet dysfunction
Peptic ulcer disease
Renal dysfunction
Plan
D
Continue regular meds if possible
Steroid support
Induction
Treat neck as unstable
Consider awake fibreoptic intubation
Intraop
Careful positioning
Postop
DVT prophylaxis
Early mobilisation
Goals
Extra caution
Airway
Neck manipulation, esp during laryngoscopy
esp if posterior AAS
Patient positioning
Consultation
History
S&S
Pain (esp neck pain)
Neurological signs
Voice change
Angina, dyspnoea
Functional capacity
Rx
Exam
Airway assessment
Neck ROM
Mouth opening
Joint ROM
Ability to use PCA
Positioning during surgery
Ix
Standard (Bloods, CXR, ECG)
C-spine with flexion / extension views
Compulsory if symptomatic
Consider
Echo
Respiratory function test
Optimisation
ENT referral for nasendoscopy if...
Voice change or hoarseness
S&S of respiratory obstruction
Consider neck stabilisation surgery prior to elective surgery

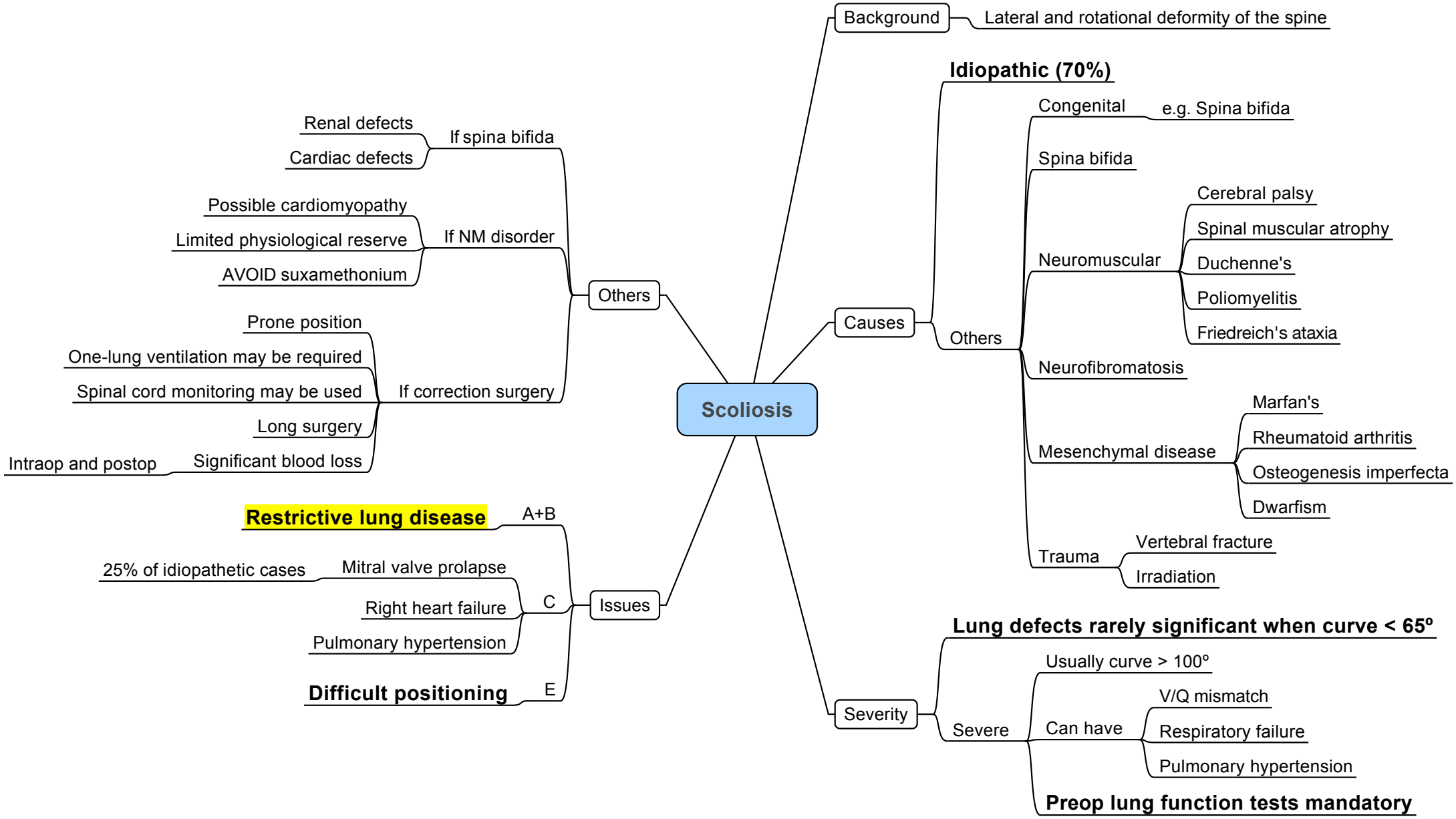

RSI quick checklist
SPEED
Suction
Suction working and easily accessible
Two suctions when possible
Positioning
Patient appropriately positioned
In-line manual stabilisation if appropriate
Equipments for intubation
BVM + airway adjunct
Laryngoscope
Bougie
ETT
End-tidal CO2
Connected, or ready to be connected
Drugs and IV access
Appropriate drugs
Fentanyl
Ketamine
Rocuronium
Vasopressors
IV access working
BOMB
Backup airways
LMA
Surgical airway kit
Oxygen
Patient pre-oxygenated
Oxygen supply adequate
Monitoring
SpO2
BP
ECG
Briefing
Roles (who's doing what)
RSI plan
Backup plan
Things to check before an RSI
SPEEDBOMB

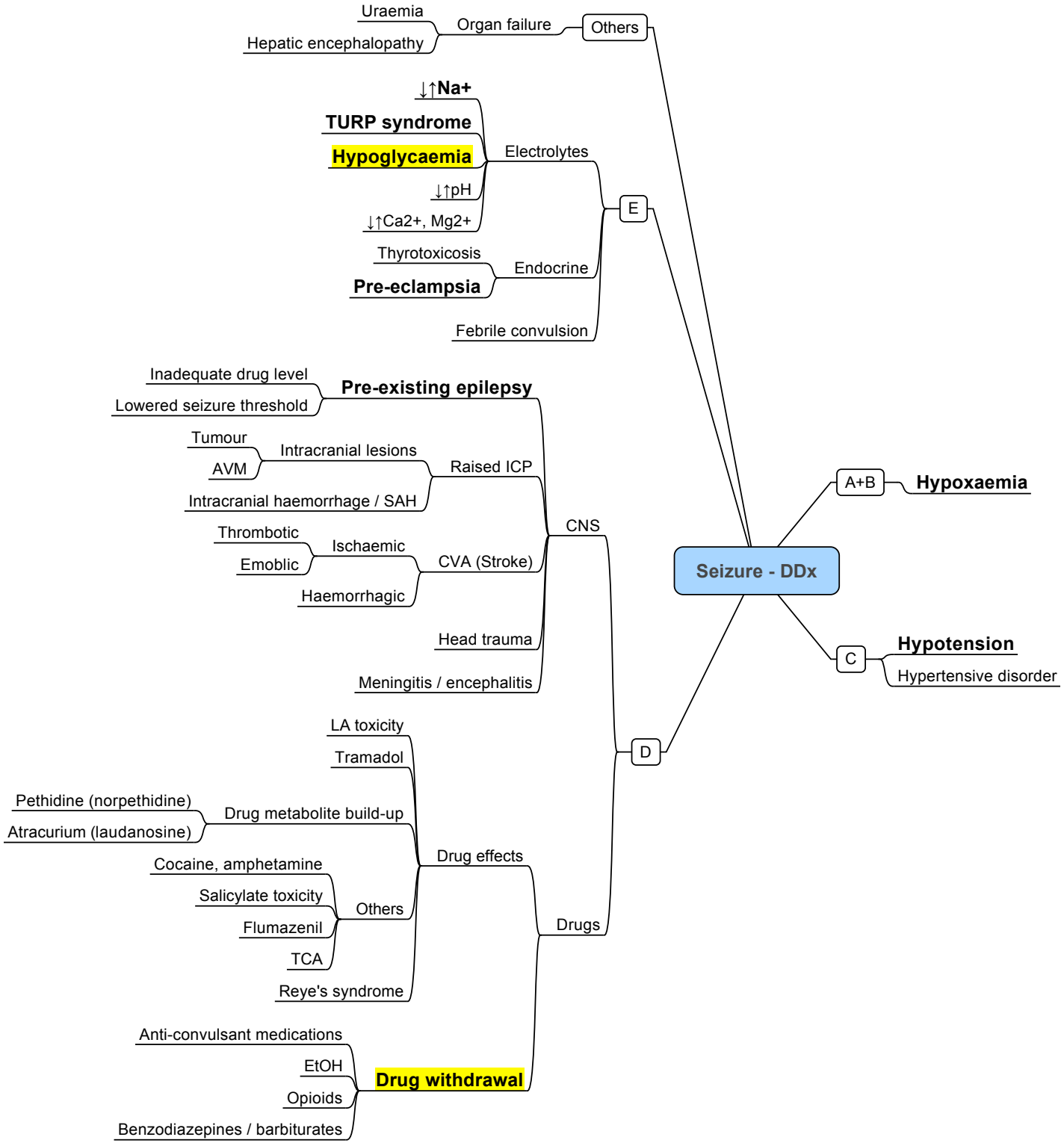

SAFE study
Background
Saline vs Albumin Fluid Evaluation (SAFE) Study
An Australia / NZ study
Published
NEJM in 2004
BMJ in 2006
NEJM 2007
ICM 2011
Question
Does albumin worsen outcome compared to NS?
Method
Multicenter, double-blinded RCT (n=6997)
Patient population
Adult ICU patients
Exclude cardiac surgery, liver transplant, and burn patients
Primary outcome
Mortality at 28 days
Secondary outcome
Survival time during first 28 days
Proportion of patients with organ failure
Duration of...
Mechanical ventilation
Renal replacement therapy
ICU stay
Hospital sty
Subgroups
Trauma
Sepsis
ARDS
Findings
Others
"Study fluid" used in resuscitation
Achieved similar haemodynamic results
Saline group : Albumin group = 1.4 : 1
c.f. 3:1 ratio in traditional teachings
Cochrane review 1998
Systemic review of 24 RCTs with 1419 randomised patients
Finding
Albumin was associated with increased mortality (6%)
No evidence that albumin reduce mortality in critically ill
General ICU (2004)
Albumin is safe
Outcome no worse than NS
i.e. Refutes the 1998 Cochrane review
EXCEPT in traumatic head injuries
Increased mortality in albumin group
RR = 1.62
95% CI 1.12 to 2.34
p = 0.009
No difference in mortality (i.e. also safe) in trauma without head injury
Albumin level (2006)
Q: Does baseline albumin level influence response to albumin?
No difference in outcome, regardless of baseline albumin level
Baseline albumin ≤ 25 g/L independently associated with increased mortality
TBI (2007)
SAFE-TBI
Post hoc analysis of patients with traumatic brain injury (TBI)
TBI patients followed up for 24 months
Albumin associated with increased mortality at 24 months in severe TBI
Decreased mortality (non-significant) for moderate TBIs in albumin group
Severe sepsis (2010)
Subgroup analysis of patients with severe sepsis at baseline
Albumin associated with reduced mortality at 28 days in severe sepsis
OR = 0.71
95% CI 0.52 to 0.97
p = 0.03
i.e. Almost 30% reduction in mortality
No difference in organ dysfunction
Except hepatic
Bilirubin increased in albumin group
May be due to bilirubin in Albuminex
i.e. No adverse effect on renal function

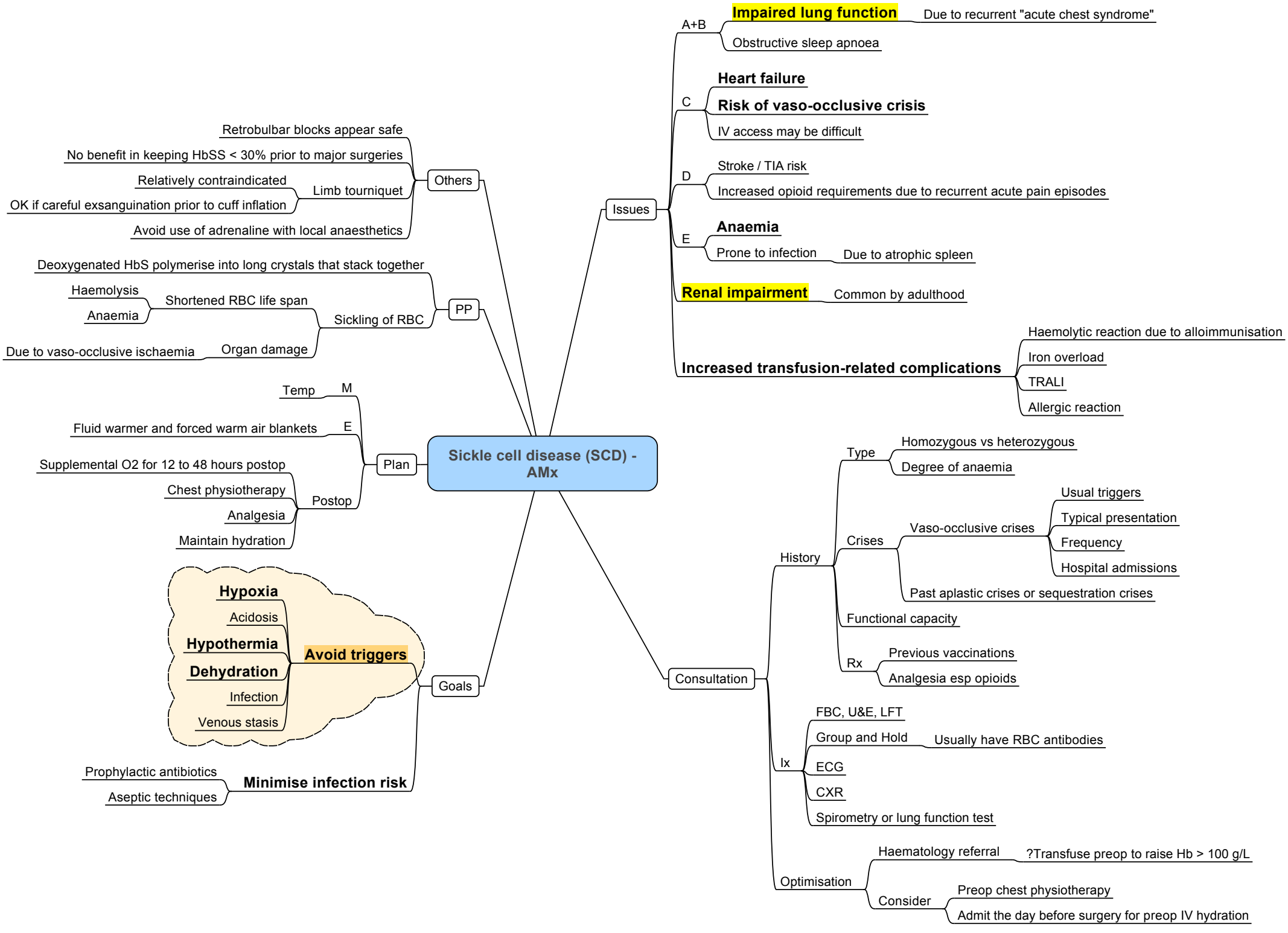

Salicylate toxicity
Mixed respiratory alkalosis and metabolic acidosis
S&S
Main progressive S&S
1. Tinnitus
Earliest sign of salicylate overdose
2. CNS stimulation
Hyperventilation
Direct stimulation of medullary ventilatory centre
N&V
Direct stimulation of medullary chemoreceptor trigger zone
3. Metabolic acidosis
Due to
Depression of renal function
Derangement of carbohydrate metabolism
Also increases movement of salicylic acid into CNS
Other metabolic derangements
Hyperglycaemia
Glycosuria
Depletion of liver and skeletal muscle glycogen
Decreased lipogenesis
Fatty infiltration of liver and kidney
Occurs in severe salicylate intoxication
Rx
Sodium bicarbonate administration
Decreases salicylic acid movement into CNS
Increase renal excretion by alkalinisation of urine

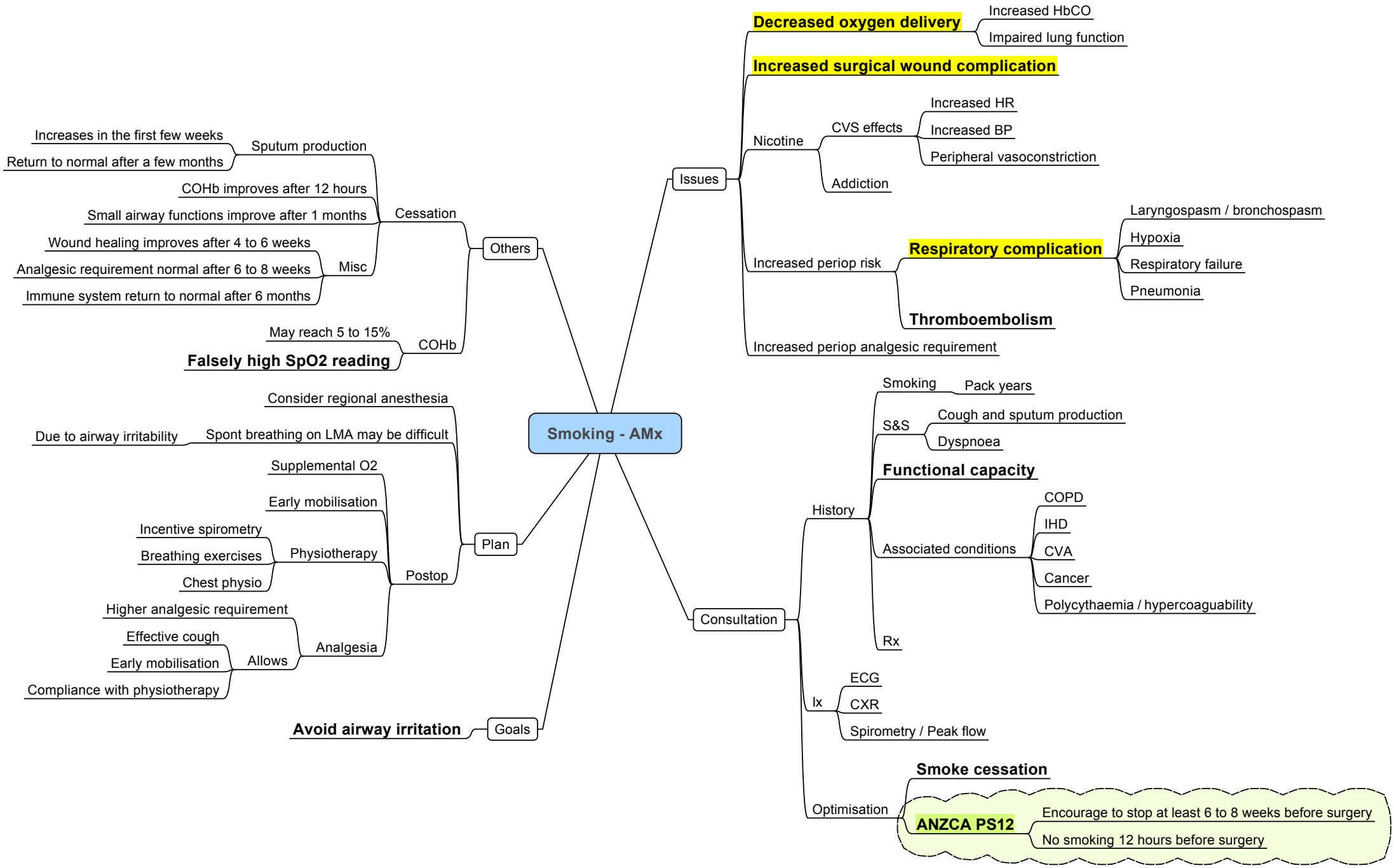

Sarcoidosis - AMx
Issues
A+B
Airway distortion / obstruction
Due to granuloma
Hilar lymphadenopathy
Bronchial obstruction
Distal atelectasis
Interstitial lung disease / pulmonary fibrosis
C
Right heart failure secondary to lung disease
Conduction abnormalities
D
Possible CNS involvement
Usually CN involvements
E
Hypercalcaemia
Endocrine
e.g.
Hypopituitarism
Including diabetes insipidus
Increased vitamin D production
Hyperthyoridism or hypothyroidism
O
Prone to infection
Lymphadenopathy very common
Sarcoidosis
Systemic inflammatory disease
Non-caseating granulomata
Can occur in any body tissues
Heals with fibrosis
10% develop serious disabilities
Poor prognosis if heart involvement
Plan
E
Consider regional anaesthesia if respiratory function impaired
Prepare for difficult airway if airway involvement
Postop
Early mobilisation
Analgesia
Physiotherapy
Goals
Steroid cover periop if necessary
Consultation
History
Previous course
S&S
esp respiratory and CVS
e.g.
Dyspnoea
Palpitation
Functional capacity
Rx
Steroid
Immunosuppressants
e.g. methotrexate
Exam
Airway assessment
Ix
CXR
ECG
Conduction abnormalties
Right ventricular hypertrophy
Blood
LFT
Ca2+
Consider
ABG
If significant respiratory S&S or involvement
Chest CT
If concerned about airway obstruction
Spirometry / Transfer factor (DLCO)

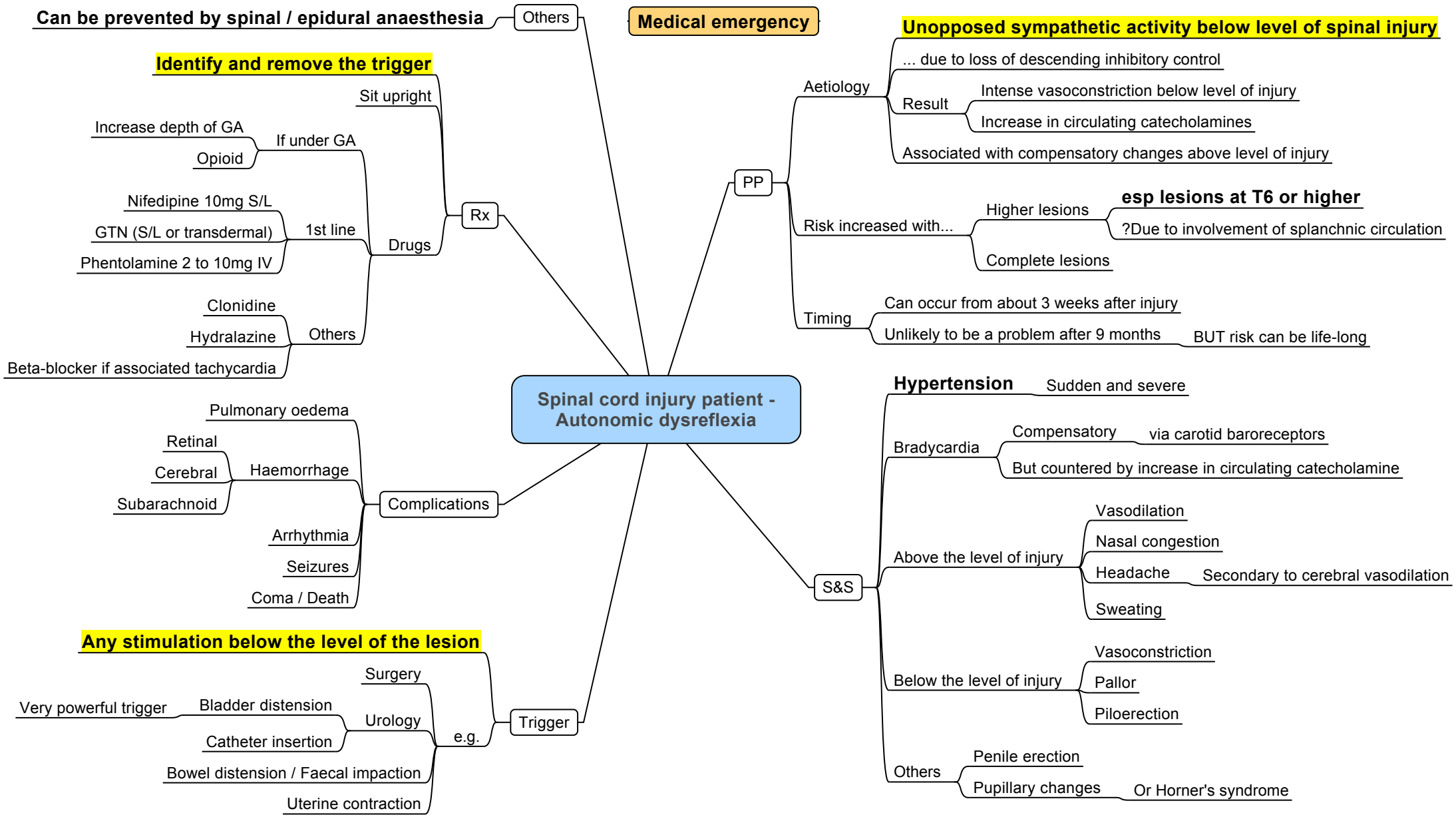

Scleroderma
Background
Chronic systemic autoimmune disease
Types
Localised
Systemic
aka systemic sclerosis
Limited
aka CREST syndrome
60% of systemic scleroderma
Slowly progressive
CREST
Calcinosis
Raynaud's phenomenon
oEsophageal dysmotility
Sclerodactyly
Telangiectasia
Diffuse
Rapidly progressive
High mortality
Issues
A+B
Limited mouth opening
Airway difficulty
Tight skin around neck
Fibrosing alveolitis
CVS
Arrhythmia
Pericarditis
Raynaud's phenomenon (> 90%)
Pulmonary hypertension
Up to 1/3 in CREST
GORD
Muscle weakness
Renal dysfunction
S&S
Characteristic
Fibrosis of skin
Vascular changes
Auto-antibodies
e.g. ANA
GIT
Upper GI bleed
"Watermelon stomach"
Diarrhoea
GORD universal
Musculoskeletal
Carpal tunnel syndrome

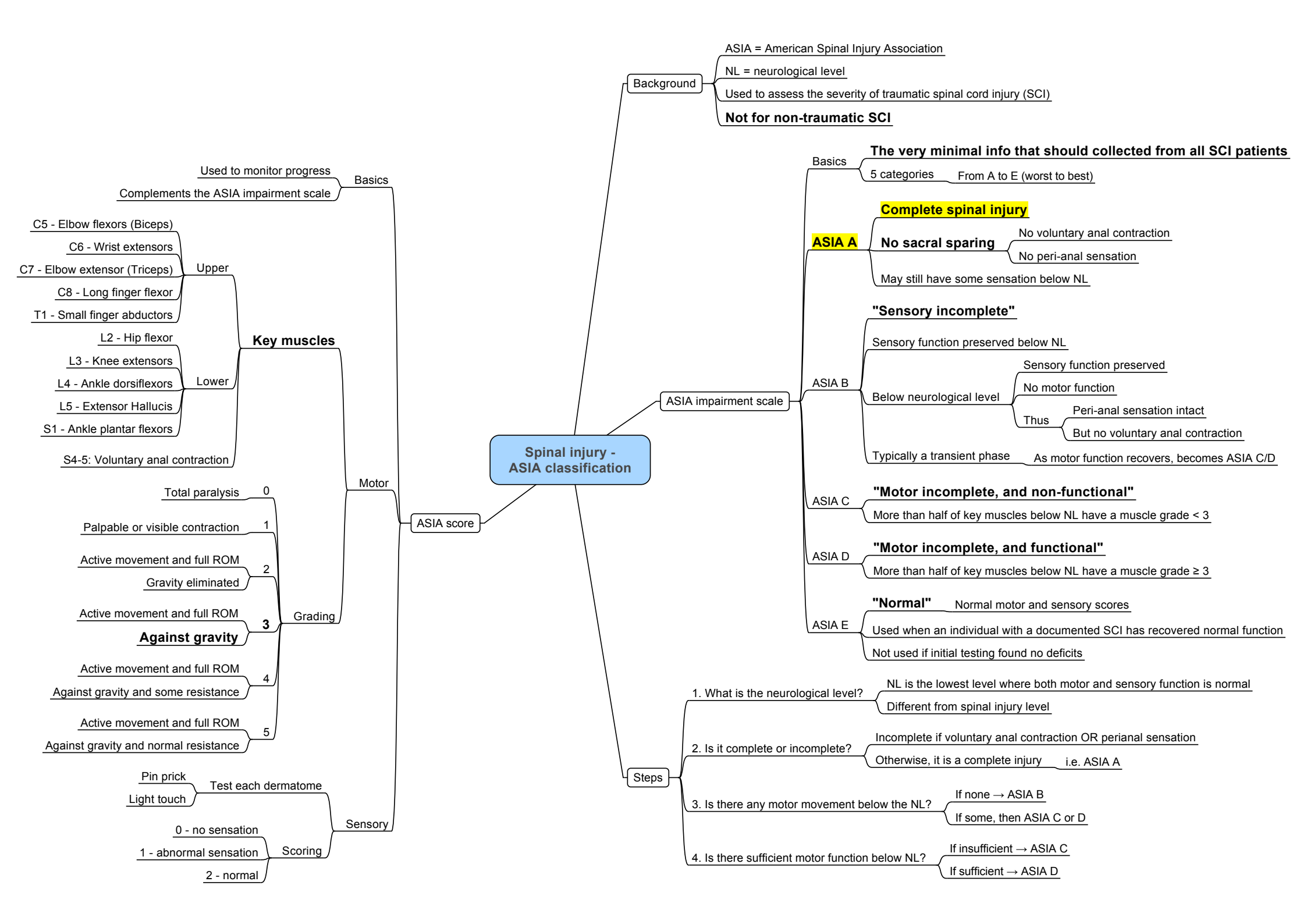

Scoliosis
Background
Lateral and rotational deformity of the spine
Causes
Idiopathic (70%)
Others
Congenital
e.g. Spina bifida
Spina bifida
Neuromuscular
Cerebral palsy
Spinal muscular atrophy
Duchenne's
Poliomyelitis
Friedreich's ataxia
Neurofibromatosis
Mesenchymal disease
Marfan's
Rheumatoid arthritis
Osteogenesis imperfecta
Dwarfism
Trauma
Vertebral fracture
Irradiation
Severity
Lung defects rarely significant when curve < 65º
Severe
Usually curve > 100º
Can have
V/Q mismatch
Respiratory failure
Pulmonary hypertension
Preop lung function tests mandatory
Issues
A+B
Restrictive lung disease
C
25% of idiopathetic cases
Mitral valve prolapse
Right heart failure
Pulmonary hypertension
E
Difficult positioning
Others
If spina bifida
Renal defects
Cardiac defects
If NM disorder
Possible cardiomyopathy
Limited physiological reserve
AVOID suxamethonium
If correction surgery
Prone position
One-lung ventilation may be required
Spinal cord monitoring may be used
Long surgery
Significant blood loss
Intraop and postop

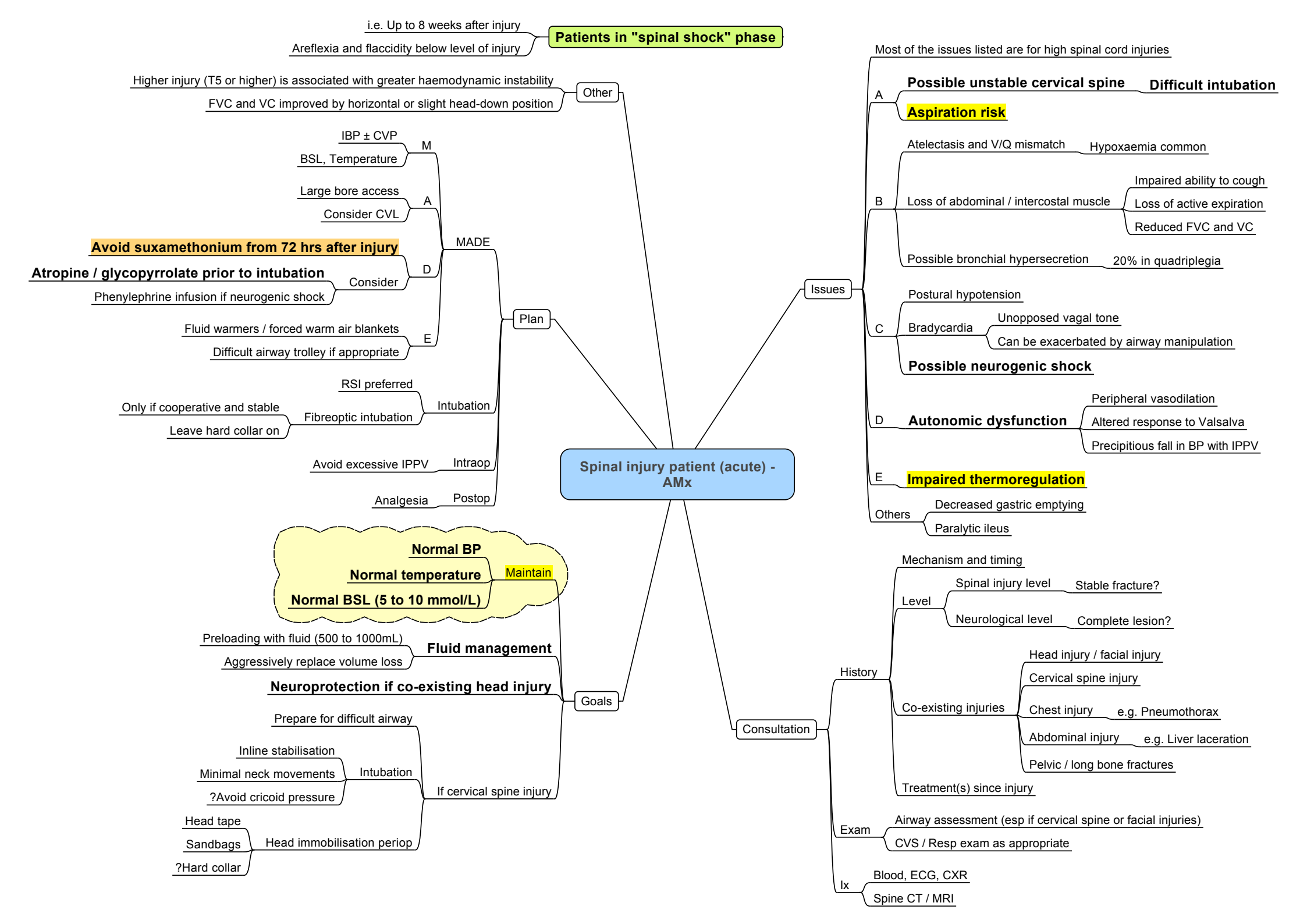

Seizure - DDx
A+B
Hypoxaemia
C
Hypotension
Hypertensive disorder
D
Drugs
Drug withdrawal
Anti-convulsant medications
EtOH
Opioids
Benzodiazepines / barbiturates
Drug effects
Drug metabolite build-up
Pethidine (norpethidine)
Atracurium (laudanosine)
Others
Cocaine, amphetamine
Salicylate toxicity
Flumazenil
TCA
Reye's syndrome
LA toxicity
Tramadol
CNS
Pre-existing epilepsy
Inadequate drug level
Lowered seizure threshold
Raised ICP
Intracranial lesions
Tumour
AVM
Intracranial haemorrhage / SAH
CVA (Stroke)
Ischaemic
Thrombotic
Emoblic
Haemorrhagic
Head trauma
Meningitis / encephalitis
E
Electrolytes
↓↑Na+
TURP syndrome
Hypoglycaemia
↓↑pH
↓↑Ca2+, Mg2+
Endocrine
Thyrotoxicosis
Pre-eclampsia
Febrile convulsion
Others
Organ failure
Uraemia
Hepatic encephalopathy

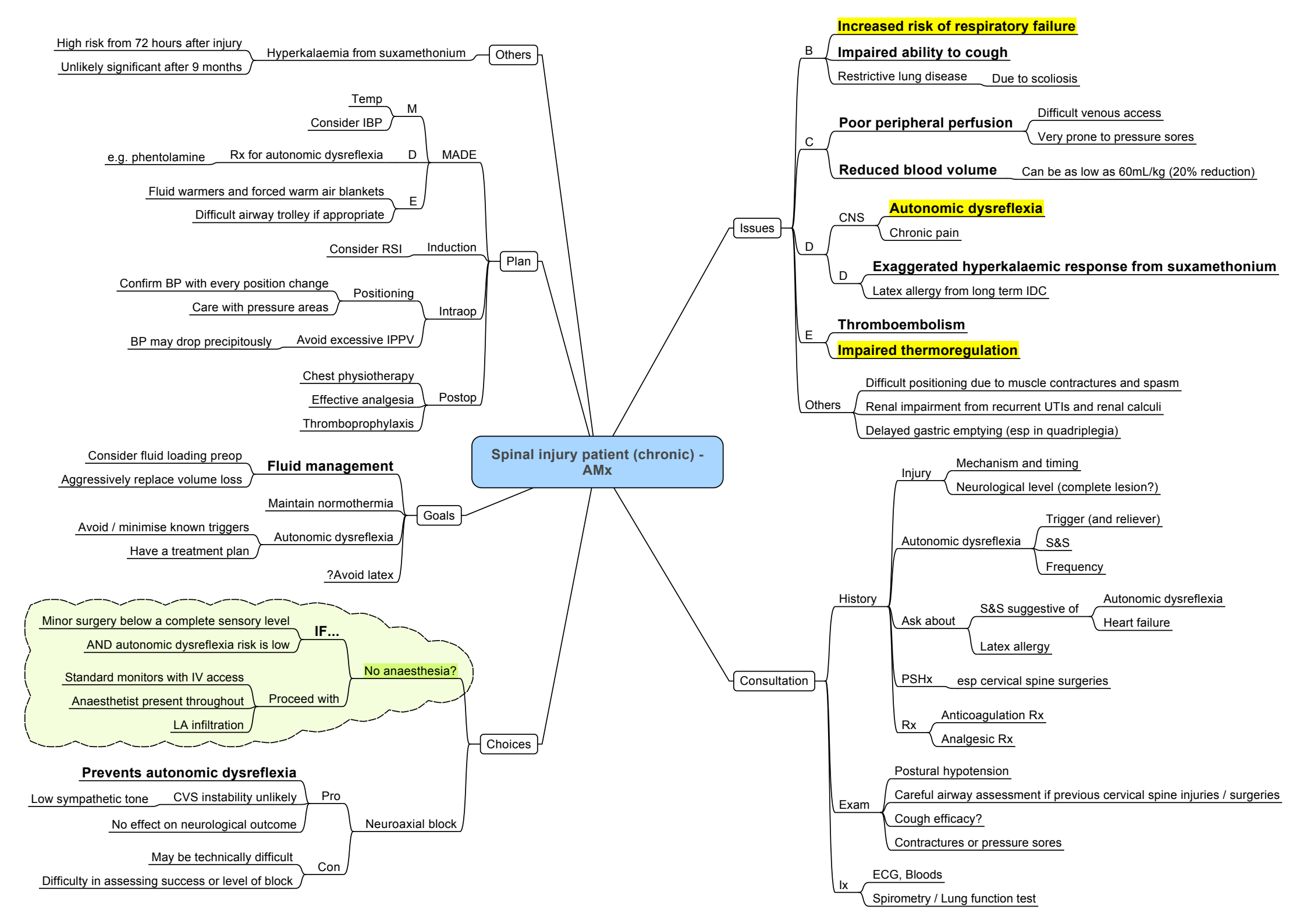

Sickle cell disease (SCD) - AMx
Issues
A+B
Impaired lung function
Due to recurrent "acute chest syndrome"
Obstructive sleep apnoea
C
Heart failure
Risk of vaso-occlusive crisis
IV access may be difficult
D
Stroke / TIA risk
Increased opioid requirements due to recurrent acute pain episodes
E
Anaemia
Prone to infection
Due to atrophic spleen
Renal impairment
Common by adulthood
Increased transfusion-related complications
Haemolytic reaction due to alloimmunisation
Iron overload
TRALI
Allergic reaction
Consultation
History
Type
Homozygous vs heterozygous
Degree of anaemia
Crises
Vaso-occlusive crises
Usual triggers
Typical presentation
Frequency
Hospital admissions
Past aplastic crises or sequestration crises
Functional capacity
Rx
Previous vaccinations
Analgesia esp opioids
Ix
FBC, U&E, LFT
Group and Hold
Usually have RBC antibodies
ECG
CXR
Spirometry or lung function test
Optimisation
Haematology referral
?Transfuse preop to raise Hb > 100 g/L
Consider
Preop chest physiotherapy
Admit the day before surgery for preop IV hydration
Goals
Avoid triggers
Hypoxia
Acidosis
Hypothermia
Dehydration
Infection
Venous stasis
Minimise infection risk
Prophylactic antibiotics
Aseptic techniques
Plan
M
Temp
E
Fluid warmer and forced warm air blankets
Postop
Supplemental O2 for 12 to 48 hours postop
Chest physiotherapy
Analgesia
Maintain hydration
PP
Deoxygenated HbS polymerise into long crystals that stack together
Sickling of RBC
Shortened RBC life span
Haemolysis
Anaemia
Organ damage
Due to vaso-occlusive ischaemia
Others
Retrobulbar blocks appear safe
No benefit in keeping HbSS < 30% prior to major surgeries
Limb tourniquet
Relatively contraindicated
OK if careful exsanguination prior to cuff inflation
Avoid use of adrenaline with local anaesthetics

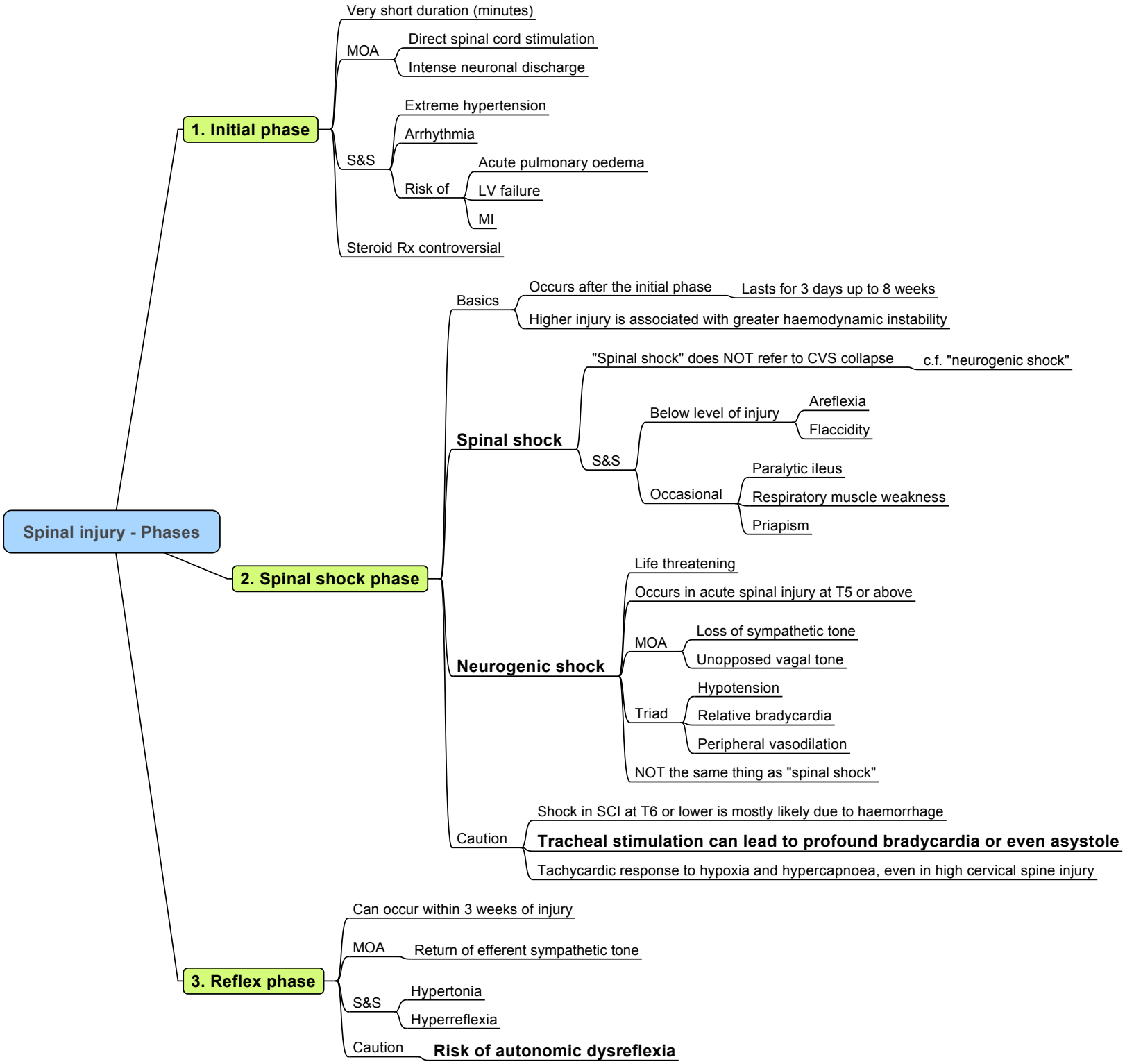

Smoking - AMx
Issues
Decreased oxygen delivery
Increased HbCO
Impaired lung function
Increased surgical wound complication
Nicotine
CVS effects
Increased HR
Increased BP
Peripheral vasoconstriction
Addiction
Increased periop risk
Respiratory complication
Laryngospasm / bronchospasm
Hypoxia
Respiratory failure
Pneumonia
Thromboembolism
Increased periop analgesic requirement
Consultation
History
Smoking
Pack years
S&S
Cough and sputum production
Dyspnoea
Functional capacity
Associated conditions
COPD
IHD
CVA
Cancer
Polycythaemia / hypercoaguability
Rx
Ix
ECG
CXR
Spirometry / Peak flow
Optimisation
Smoke cessation
ANZCA PS12
Encourage to stop at least 6 to 8 weeks before surgery
No smoking 12 hours before surgery
Goals
Avoid airway irritation
Plan
Postop
Physiotherapy
Incentive spirometry
Breathing exercises
Chest physio
Supplemental O2
Early mobilisation
Analgesia
Higher analgesic requirement
Allows
Effective cough
Early mobilisation
Compliance with physiotherapy
Spont breathing on LMA may be difficult
Due to airway irritability
Consider regional anesthesia
Others
Cessation
Sputum production
Increases in the first few weeks
Return to normal after a few months
COHb improves after 12 hours
Small airway functions improve after 1 months
Misc
Wound healing improves after 4 to 6 weeks
Analgesic requirement normal after 6 to 8 weeks
Immune system return to normal after 6 months
COHb
May reach 5 to 15%
Falsely high SpO2 reading

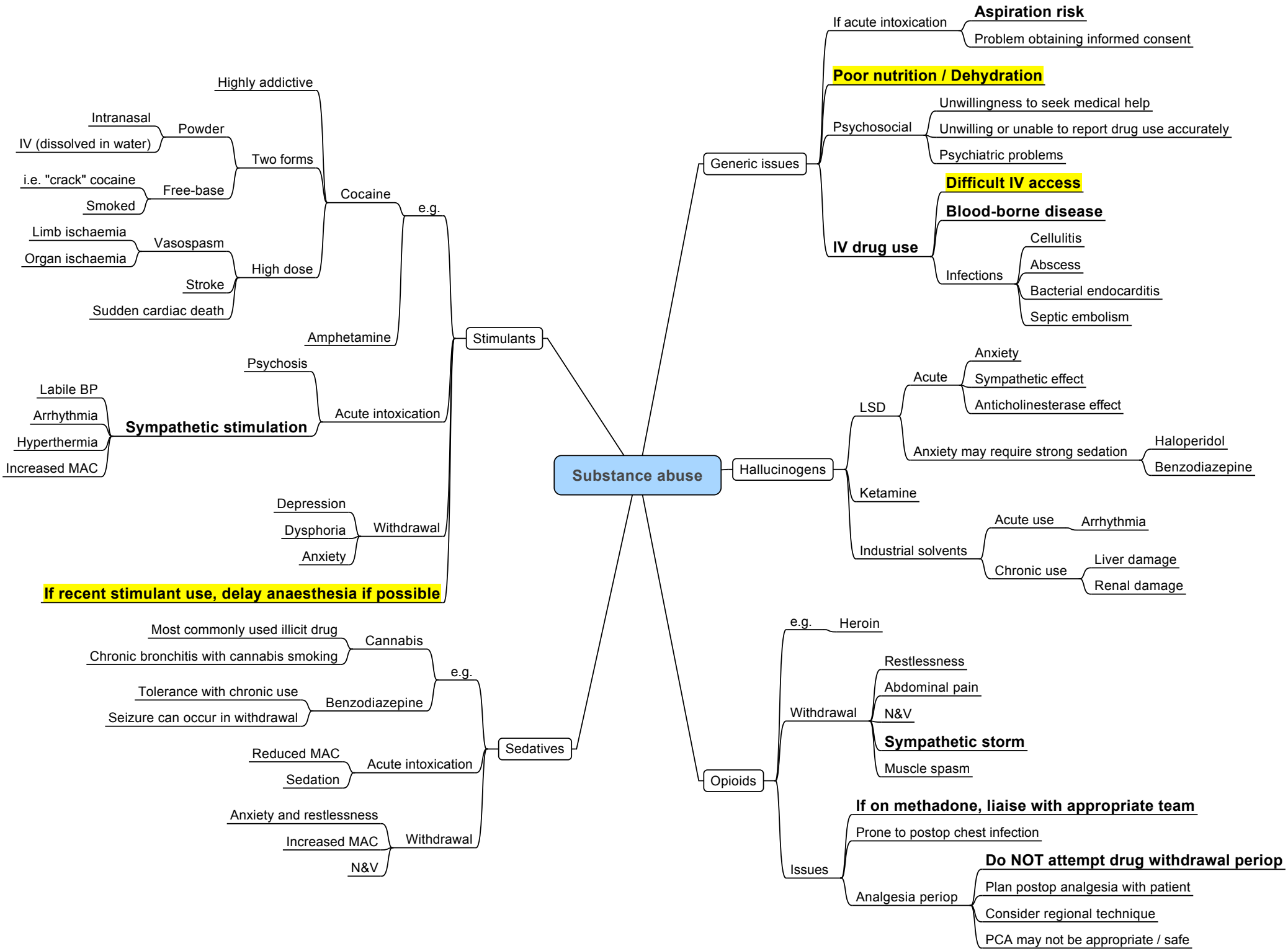

Spinal cord injury patient - Autonomic dysreflexia
Medical emergency
PP
Aetiology
Unopposed sympathetic activity below level of spinal injury
... due to loss of descending inhibitory control
Result
Intense vasoconstriction below level of injury
Increase in circulating catecholamines
Associated with compensatory changes above level of injury
Risk increased with...
Higher lesions
esp lesions at T6 or higher
?Due to involvement of splanchnic circulation
Complete lesions
Timing
Can occur from about 3 weeks after injury
Unlikely to be a problem after 9 months
BUT risk can be life-long
S&S
Hypertension
Sudden and severe
Bradycardia
Compensatory
via carotid baroreceptors
But countered by increase in circulating catecholamine
Above the level of injury
Vasodilation
Nasal congestion
Headache
Secondary to cerebral vasodilation
Sweating
Below the level of injury
Vasoconstriction
Pallor
Piloerection
Others
Penile erection
Pupillary changes
Or Horner's syndrome
Rx
Identify and remove the trigger
Sit upright
If under GA
Increase depth of GA
Opioid
Drugs
1st line
Nifedipine 10mg S/L
GTN (S/L or transdermal)
Phentolamine 2 to 10mg IV
Others
Clonidine
Hydralazine
Beta-blocker if associated tachycardia
Others
Can be prevented by spinal / epidural anaesthesia
Complications
Pulmonary oedema
Haemorrhage
Retinal
Cerebral
Subarachnoid
Arrhythmia
Seizures
Coma / Death
Trigger
Any stimulation below the level of the lesion
e.g.
Surgery
Urology
Bladder distension
Very powerful trigger
Catheter insertion
Bowel distension / Faecal impaction
Uterine contraction

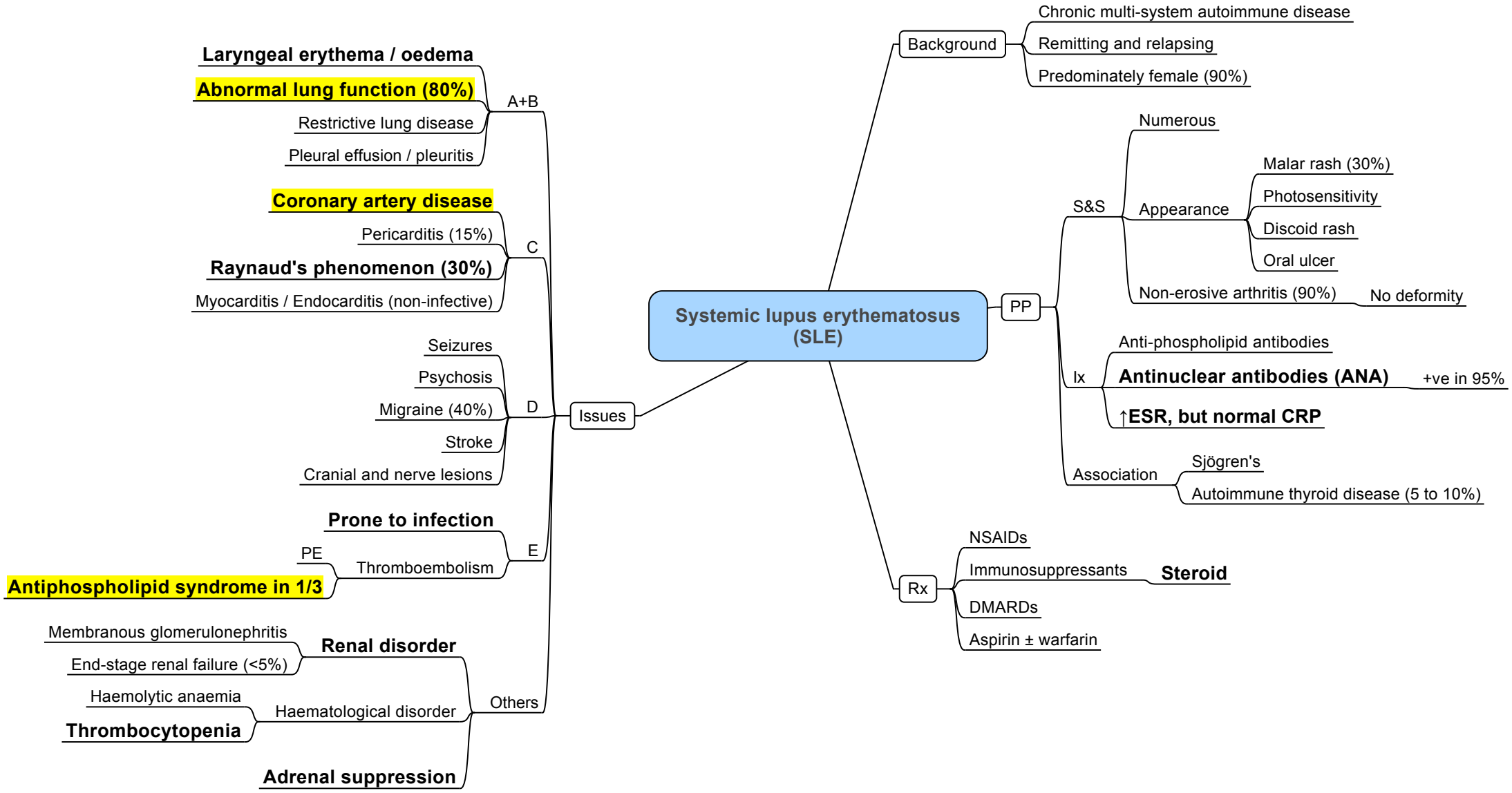

Spinal injury - ASIA classification
Background
ASIA = American Spinal Injury Association
NL = neurological level
Used to assess the severity of traumatic spinal cord injury (SCI)
Not for non-traumatic SCI
ASIA impairment scale
Basics
The very minimal info that should collected from all SCI patients
5 categories
From A to E (worst to best)
ASIA A
Complete spinal injury
No sacral sparing
No voluntary anal contraction
No peri-anal sensation
May still have some sensation below NL
ASIA B
"Sensory incomplete"
Sensory function preserved below NL
Below neurological level
Sensory function preserved
No motor function
Thus
Peri-anal sensation intact
But no voluntary anal contraction
Typically a transient phase
As motor function recovers, becomes ASIA C/D
ASIA C
"Motor incomplete, and non-functional"
More than half of key muscles below NL have a muscle grade < 3
ASIA D
"Motor incomplete, and functional"
More than half of key muscles below NL have a muscle grade ≥ 3
ASIA E
"Normal"
Normal motor and sensory scores
Used when an individual with a documented SCI has recovered normal function
Not used if initial testing found no deficits
Steps
1. What is the neurological level?
NL is the lowest level where both motor and sensory function is normal
Different from spinal injury level
2. Is it complete or incomplete?
Incomplete if voluntary anal contraction OR perianal sensation
Otherwise, it is a complete injury
i.e. ASIA A
3. Is there any motor movement below the NL?
If none → ASIA B
If some, then ASIA C or D
4. Is there sufficient motor function below NL?
If insufficient → ASIA C
If sufficient → ASIA D
ASIA score
Basics
Used to monitor progress
Complements the ASIA impairment scale
Motor
Key muscles
Upper
C5 - Elbow flexors (Biceps)
C6 - Wrist extensors
C7 - Elbow extensor (Triceps)
C8 - Long finger flexor
T1 - Small finger abductors
Lower
L2 - Hip flexor
L3 - Knee extensors
L4 - Ankle dorsiflexors
L5 - Extensor Hallucis
S1 - Ankle plantar flexors
S4-5: Voluntary anal contraction
Grading
0
Total paralysis
1
Palpable or visible contraction
2
Active movement and full ROM
Gravity eliminated
3
Active movement and full ROM
Against gravity
4
Active movement and full ROM
Against gravity and some resistance
5
Active movement and full ROM
Against gravity and normal resistance
Sensory
Test each dermatome
Pin prick
Light touch
Scoring
0 - no sensation
1 - abnormal sensation
2 - normal

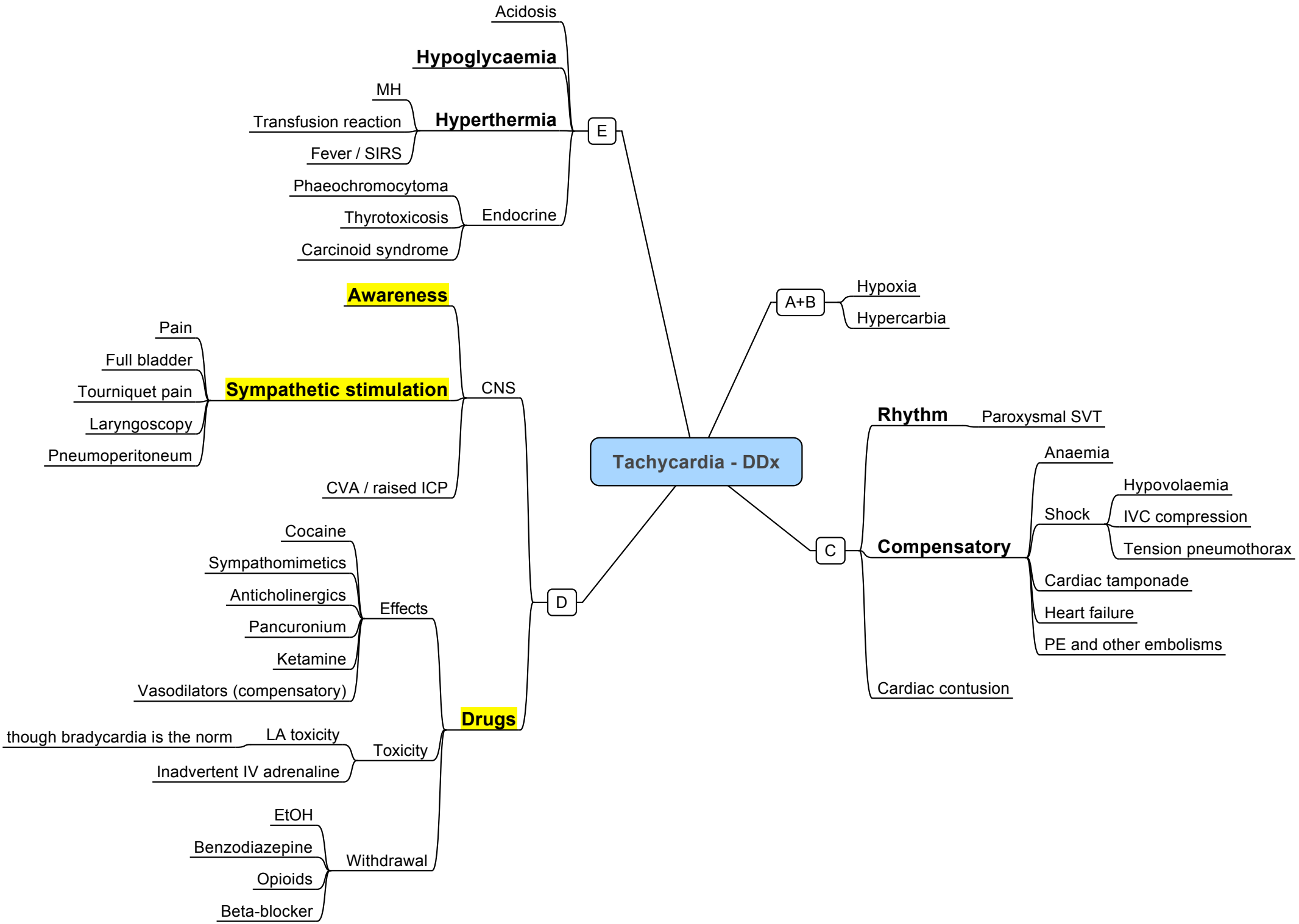

Spinal injury patient (acute) - AMx
Issues
Most of the issues listed are for high spinal cord injuries
A
Possible unstable cervical spine
Difficult intubation
Aspiration risk
B
Atelectasis and V/Q mismatch
Hypoxaemia common
Loss of abdominal / intercostal muscle
Impaired ability to cough
Loss of active expiration
Reduced FVC and VC
Possible bronchial hypersecretion
20% in quadriplegia
C
Postural hypotension
Bradycardia
Unopposed vagal tone
Can be exacerbated by airway manipulation
Possible neurogenic shock
D
Autonomic dysfunction
Peripheral vasodilation
Altered response to Valsalva
Precipitious fall in BP with IPPV
E
Impaired thermoregulation
Others
Decreased gastric emptying
Paralytic ileus
Consultation
History
Mechanism and timing
Level
Spinal injury level
Stable fracture?
Neurological level
Complete lesion?
Co-existing injuries
Head injury / facial injury
Cervical spine injury
Chest injury
e.g. Pneumothorax
Abdominal injury
e.g. Liver laceration
Pelvic / long bone fractures
Treatment(s) since injury
Exam
Airway assessment (esp if cervical spine or facial injuries)
CVS / Resp exam as appropriate
Ix
Blood, ECG, CXR
Spine CT / MRI
Goals
Maintain
Normal BP
Normal temperature
Normal BSL (5 to 10 mmol/L)
Fluid management
Preloading with fluid (500 to 1000mL)
Aggressively replace volume loss
Neuroprotection if co-existing head injury
If cervical spine injury
Intubation
Prepare for difficult airway
Inline stabilisation
Minimal neck movements
?Avoid cricoid pressure
Head immobilisation periop
Head tape
Sandbags
?Hard collar
Plan
MADE
M
IBP ± CVP
BSL, Temperature
A
Large bore access
Consider CVL
D
Consider
Avoid suxamethonium from 72 hrs after injury
Atropine / glycopyrrolate prior to intubation
Phenylephrine infusion if neurogenic shock
E
Fluid warmers / forced warm air blankets
Difficult airway trolley if appropriate
Intubation
RSI preferred
Fibreoptic intubation
Only if cooperative and stable
Leave hard collar on
Intraop
Avoid excessive IPPV
Postop
Analgesia
Other
Higher injury (T5 or higher) is associated with greater haemodynamic instability
FVC and VC improved by horizontal or slight head-down position
Patients in "spinal shock" phase
i.e. Up to 8 weeks after injury
Areflexia and flaccidity below level of injury

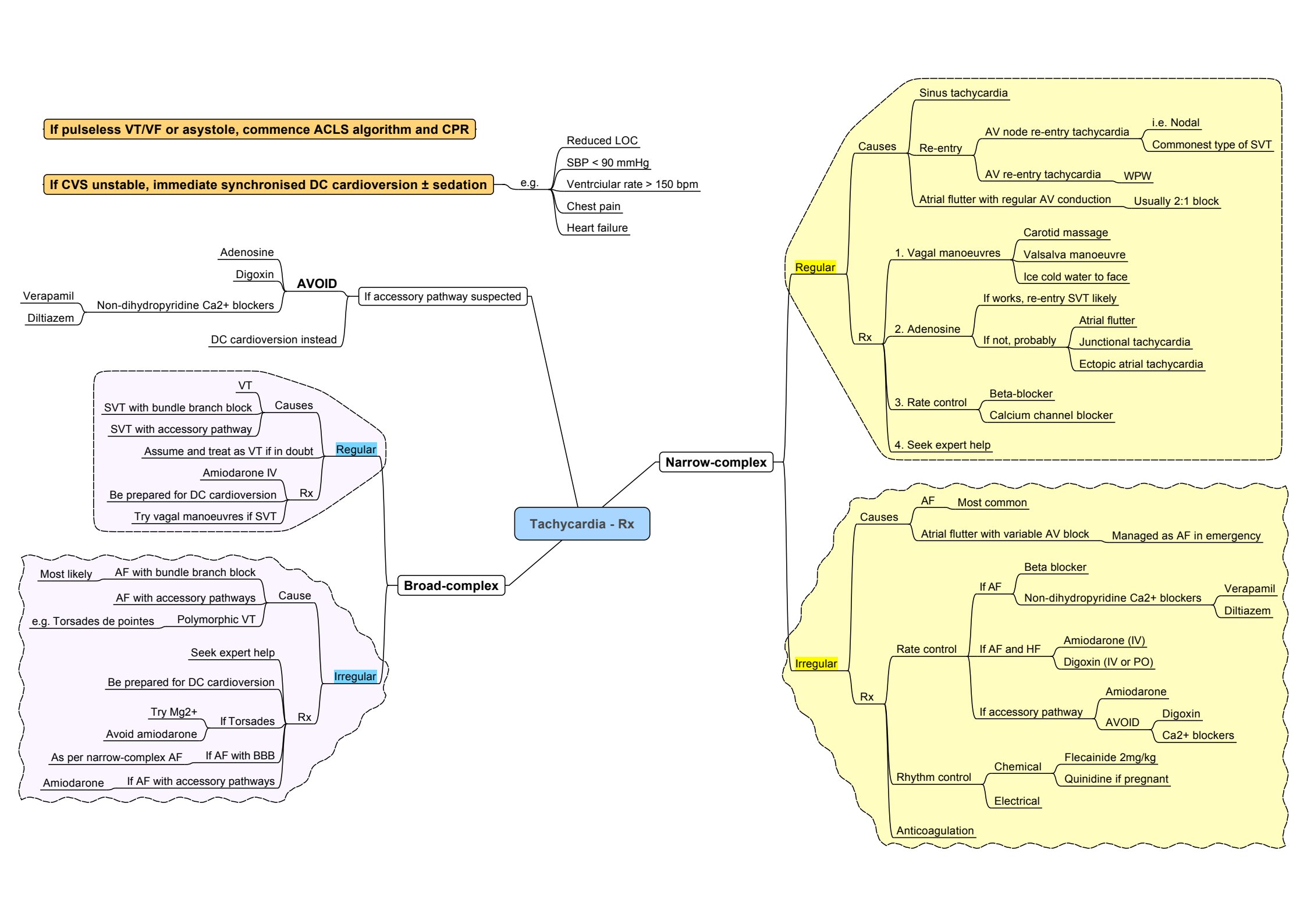

Spinal injury patient (chronic) - AMx
Consultation
History
Injury
Mechanism and timing
Neurological level (complete lesion?)
Autonomic dysreflexia
Trigger (and reliever)
S&S
Frequency
Ask about
S&S suggestive of
Autonomic dysreflexia
Heart failure
Latex allergy
PSHx
esp cervical spine surgeries
Rx
Anticoagulation Rx
Analgesic Rx
Exam
Postural hypotension
Careful airway assessment if previous cervical spine injuries / surgeries
Cough efficacy?
Contractures or pressure sores
Ix
ECG, Bloods
Spirometry / Lung function test
Issues
B
Increased risk of respiratory failure
Impaired ability to cough
Restrictive lung disease
Due to scoliosis
C
Poor peripheral perfusion
Difficult venous access
Very prone to pressure sores
Reduced blood volume
Can be as low as 60mL/kg (20% reduction)
D
CNS
Autonomic dysreflexia
Chronic pain
D
Exaggerated hyperkalaemic response from suxamethonium
Latex allergy from long term IDC
E
Thromboembolism
Impaired thermoregulation
Others
Difficult positioning due to muscle contractures and spasm
Renal impairment from recurrent UTIs and renal calculi
Delayed gastric emptying (esp in quadriplegia)
Goals
Fluid management
Consider fluid loading preop
Aggressively replace volume loss
Maintain normothermia
Autonomic dysreflexia
Avoid / minimise known triggers
Have a treatment plan
?Avoid latex
Choices
No anaesthesia?
IF...
Minor surgery below a complete sensory level
AND autonomic dysreflexia risk is low
Proceed with
Standard monitors with IV access
Anaesthetist present throughout
LA infiltration
Neuroaxial block
Pro
Prevents autonomic dysreflexia
CVS instability unlikely
Low sympathetic tone
No effect on neurological outcome
Con
May be technically difficult
Difficulty in assessing success or level of block
Plan
MADE
M
Temp
Consider IBP
D
Rx for autonomic dysreflexia
e.g. phentolamine
E
Fluid warmers and forced warm air blankets
Difficult airway trolley if appropriate
Induction
Consider RSI
Intraop
Positioning
Confirm BP with every position change
Care with pressure areas
Avoid excessive IPPV
BP may drop precipitously
Postop
Chest physiotherapy
Effective analgesia
Thromboprophylaxis
Others
Hyperkalaemia from suxamethonium
High risk from 72 hours after injury
Unlikely significant after 9 months


Spinal injury - Phases
1. Initial phase
Very short duration (minutes)
MOA
Direct spinal cord stimulation
Intense neuronal discharge
S&S
Extreme hypertension
Arrhythmia
Risk of
Acute pulmonary oedema
LV failure
MI
Steroid Rx controversial
2. Spinal shock phase
Basics
Occurs after the initial phase
Lasts for 3 days up to 8 weeks
Higher injury is associated with greater haemodynamic instability
Spinal shock
"Spinal shock" does NOT refer to CVS collapse
c.f. "neurogenic shock"
S&S
Below level of injury
Areflexia
Flaccidity
Occasional
Paralytic ileus
Respiratory muscle weakness
Priapism
Neurogenic shock
Life threatening
Occurs in acute spinal injury at T5 or above
MOA
Loss of sympathetic tone
Unopposed vagal tone
Triad
Hypotension
Relative bradycardia
Peripheral vasodilation
NOT the same thing as "spinal shock"
Caution
Shock in SCI at T6 or lower is mostly likely due to haemorrhage
Tracheal stimulation can lead to profound bradycardia or even asystole
Tachycardic response to hypoxia and hypercapnoea, even in high cervical spine injury
3. Reflex phase
Can occur within 3 weeks of injury
MOA
Return of efferent sympathetic tone
S&S
Hypertonia
Hyperreflexia
Caution
Risk of autonomic dysreflexia

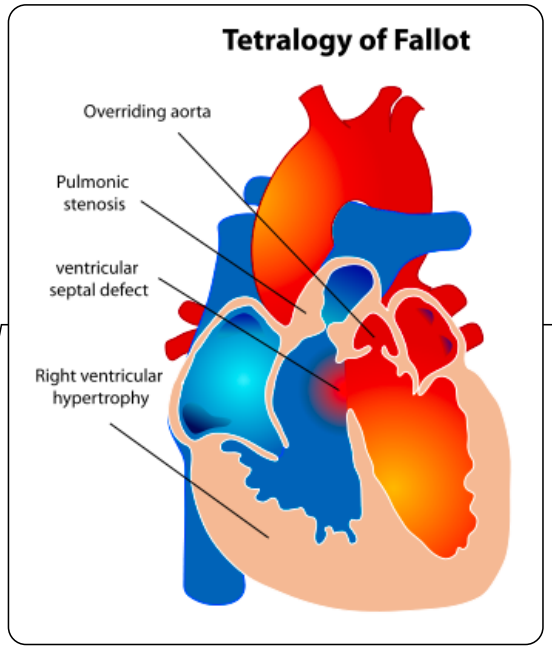

Substance abuse
Stimulants
e.g.
Cocaine
Highly addictive
Two forms
Powder
Intranasal
IV (dissolved in water)
Free-base
i.e. "crack" cocaine
Smoked
High dose
Vasospasm
Limb ischaemia
Organ ischaemia
Stroke
Sudden cardiac death
Amphetamine
Acute intoxication
Psychosis
Sympathetic stimulation
Labile BP
Arrhythmia
Hyperthermia
Increased MAC
Withdrawal
Depression
Dysphoria
Anxiety
If recent stimulant use, delay anaesthesia if possible
Sedatives
e.g.
Cannabis
Most commonly used illicit drug
Chronic bronchitis with cannabis smoking
Benzodiazepine
Tolerance with chronic use
Seizure can occur in withdrawal
Acute intoxication
Reduced MAC
Sedation
Withdrawal
Anxiety and restlessness
Increased MAC
N&V
Generic issues
If acute intoxication
Aspiration risk
Problem obtaining informed consent
Poor nutrition / Dehydration
Psychosocial
Unwillingness to seek medical help
Unwilling or unable to report drug use accurately
Psychiatric problems
IV drug use
Difficult IV access
Blood-borne disease
Infections
Cellulitis
Abscess
Bacterial endocarditis
Septic embolism
Hallucinogens
LSD
Acute
Anxiety
Sympathetic effect
Anticholinesterase effect
Anxiety may require strong sedation
Haloperidol
Benzodiazepine
Ketamine
Industrial solvents
Acute use
Arrhythmia
Chronic use
Liver damage
Renal damage
Opioids
e.g.
Heroin
Withdrawal
Restlessness
Abdominal pain
N&V
Sympathetic storm
Muscle spasm
Issues
If on methadone, liaise with appropriate team
Prone to postop chest infection
Analgesia periop
Do NOT attempt drug withdrawal periop
Plan postop analgesia with patient
Consider regional technique
PCA may not be appropriate / safe


Systemic lupus erythematosus (SLE)
Background
Chronic multi-system autoimmune disease
Remitting and relapsing
Predominately female (90%)
PP
S&S
Numerous
Appearance
Malar rash (30%)
Photosensitivity
Discoid rash
Oral ulcer
Non-erosive arthritis (90%)
No deformity
Ix
Anti-phospholipid antibodies
Antinuclear antibodies (ANA)
+ve in 95%
↑ESR, but normal CRP
Association
Sjögren's
Autoimmune thyroid disease (5 to 10%)
Rx
NSAIDs
Immunosuppressants
Steroid
DMARDs
Aspirin ± warfarin
Issues
A+B
Laryngeal erythema / oedema
Abnormal lung function (80%)
Restrictive lung disease
Pleural effusion / pleuritis
C
Coronary artery disease
Pericarditis (15%)
Raynaud's phenomenon (30%)
Myocarditis / Endocarditis (non-infective)
D
Seizures
Psychosis
Migraine (40%)
Stroke
Cranial and nerve lesions
E
Prone to infection
Thromboembolism
PE
Antiphospholipid syndrome in 1/3
Others
Renal disorder
Membranous glomerulonephritis
End-stage renal failure (<5%)
Haematological disorder
Haemolytic anaemia
Thrombocytopenia
Adrenal suppression


Tachycardia - DDx
A+B
Hypoxia
Hypercarbia
C
Rhythm
Paroxysmal SVT
Compensatory
Anaemia
Shock
Hypovolaemia
IVC compression
Tension pneumothorax
Cardiac tamponade
Heart failure
PE and other embolisms
Cardiac contusion
D
CNS
Awareness
Sympathetic stimulation
Pain
Full bladder
Tourniquet pain
Laryngoscopy
Pneumoperitoneum
CVA / raised ICP
Drugs
Effects
Cocaine
Sympathomimetics
Anticholinergics
Pancuronium
Ketamine
Vasodilators (compensatory)
Toxicity
LA toxicity
though bradycardia is the norm
Inadvertent IV adrenaline
Withdrawal
EtOH
Benzodiazepine
Opioids
Beta-blocker
E
Acidosis
Hypoglycaemia
Hyperthermia
MH
Transfusion reaction
Fever / SIRS
Endocrine
Phaeochromocytoma
Thyrotoxicosis
Carcinoid syndrome


If pulseless VT/VF or asystole, commence ACLS algorithm and CPR
If CVS unstable, immediate synchronised DC cardioversion ± sedation
e.g.
Reduced LOC
SBP < 90 mmHg
Ventrciular rate > 150 bpm
Chest pain
Heart failure
Tachycardia - Rx
Narrow-complex
Regular
Causes
Sinus tachycardia
Re-entry
AV node re-entry tachycardia
i.e. Nodal
Commonest type of SVT
AV re-entry tachycardia
WPW
Atrial flutter with regular AV conduction
Usually 2:1 block
Rx
1. Vagal manoeuvres
Carotid massage
Valsalva manoeuvre
Ice cold water to face
2. Adenosine
If works, re-entry SVT likely
If not, probably
Atrial flutter
Junctional tachycardia
Ectopic atrial tachycardia
3. Rate control
Beta-blocker
Calcium channel blocker
4. Seek expert help
Irregular
Causes
AF
Most common
Atrial flutter with variable AV block
Managed as AF in emergency
Rx
Rate control
If AF
Beta blocker
Non-dihydropyridine Ca2+ blockers
Verapamil
Diltiazem
If AF and HF
Amiodarone (IV)
Digoxin (IV or PO)
If accessory pathway
Amiodarone
AVOID
Digoxin
Ca2+ blockers
Rhythm control
Chemical
Flecainide 2mg/kg
Quinidine if pregnant
Electrical
Anticoagulation
Broad-complex
If accessory pathway suspected
AVOID
Adenosine
Digoxin
Non-dihydropyridine Ca2+ blockers
Verapamil
Diltiazem
DC cardioversion instead
Regular
Causes
VT
SVT with bundle branch block
SVT with accessory pathway
Assume and treat as VT if in doubt
Rx
Amiodarone IV
Be prepared for DC cardioversion
Try vagal manoeuvres if SVT
Irregular
Cause
Most likely
AF with bundle branch block
AF with accessory pathways
e.g. Torsades de pointes
Polymorphic VT
Rx
Seek expert help
Be prepared for DC cardioversion
If Torsades
Try Mg2+
Avoid amiodarone
If AF with BBB
As per narrow-complex AF
If AF with accessory pathways
Amiodarone

Tetralogy of Fallot (TOF) - AMx



- Most common cyanotic congenital heart defects
- Consists of
  - Overriding aorta
  - **RV outflow tract (RVOT) obstruction**
  - Ventricular septal defect (VSD)
  - RV hypertrophy
- Rx
  - Beta blocker
  - Blalock-Taussig shunt — Subclavian artery to pulmonary artery
  - Definitive surgery — Done when 3 to 8 months
    - Includes
      - Patch closure of VSD
      - Enlargement of RVOT

- Issues
  - **Right-to-left shunt**
    - **Cyanosis**
    - **Hypoxia** — Refractory to oxygen Rx
    - Paradoxical embolism
    - Fast IV induction
    - **Slow gas induction**
  - Associated conditions
  - Tet spells
  - Bacterial endocarditis prophylaxis

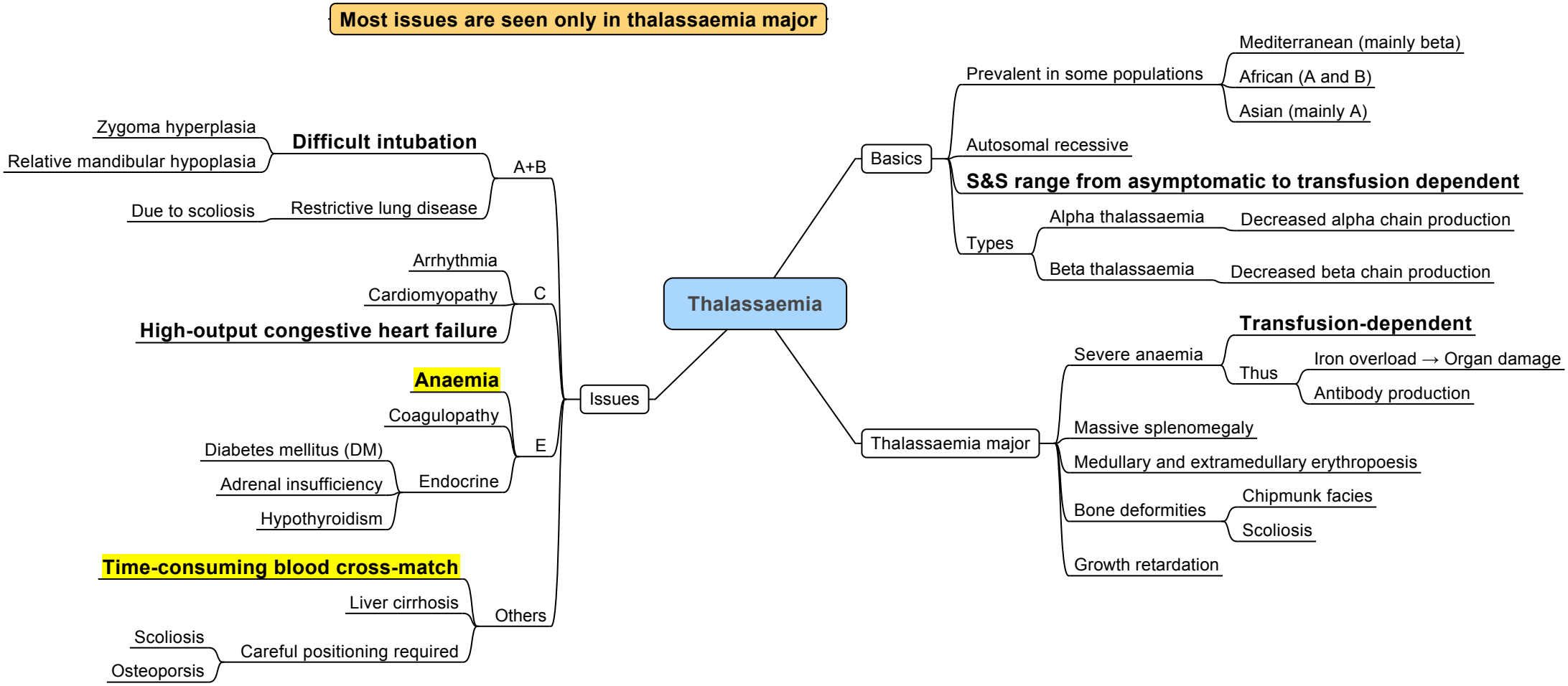
- Consultation
  - History
    - Tet spells
      - Frequency
      - Trigger
      - Resolution
    - Previous surgical repair
    - Associated
      - **Cardiac anomalies (40%)** — e.g.
        - Right aortic arch (25%)
        - Coronary artery abnormalities (9%)
      - **Extra-cardiac anomalies (15%)** — e.g. Down syndrome
  - Ix
    - ECG
      - RV hypertrophy
      - Right axis deviation
    - Bloods — Polycythaemia
    - CXR — Boot-shaped heart
  - Optimisation — Discuss with cardiologist / cardiothoracic surgeon

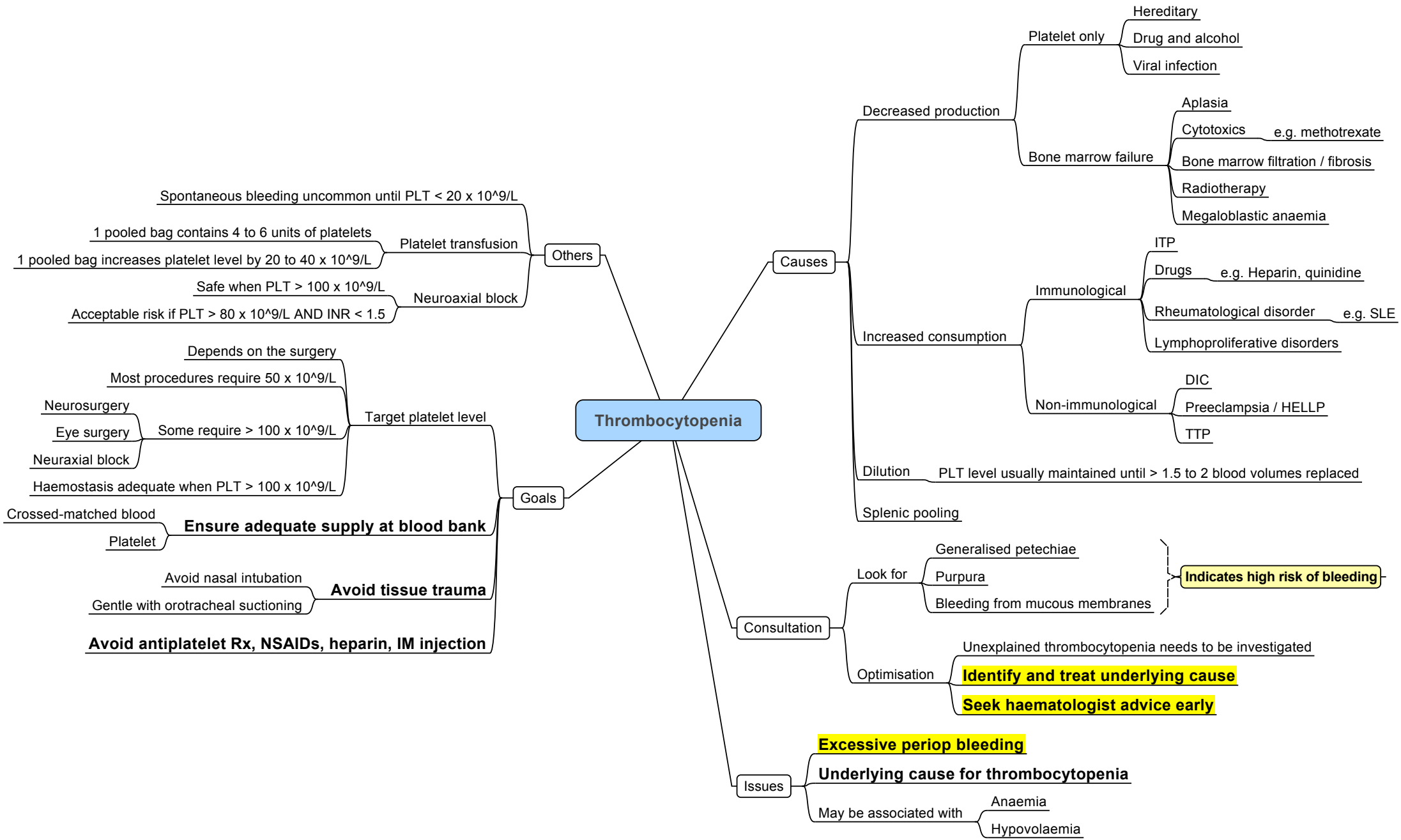
- Goals
  - **Minimise right-to-left shunt** (i.e.)
    - Maintain SVR:PVR ratio
    - Maintain afterload
    - Minimise pulmonary vascular resistance
  - Maintain preload
  - Avoid tet spells
  - Avoid air in IV lines

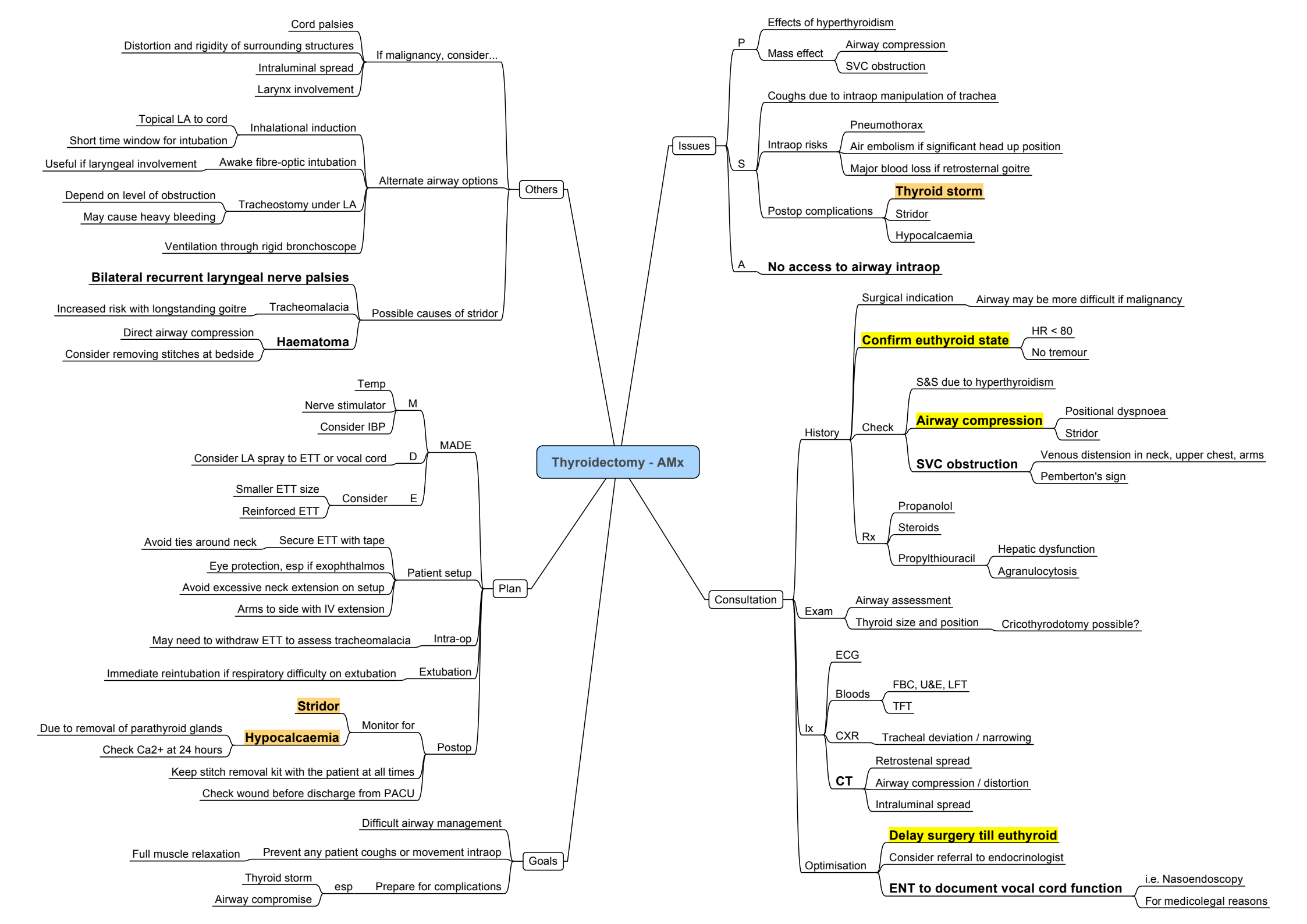
- Plan
  - Preop
    - Maintain oral feeds
    - Continue beta blockers
  - D
    - **Ketamine**
      - Preferred agent
      - SVR preserved
    - Sevoflurane — Acceptable but use with caution

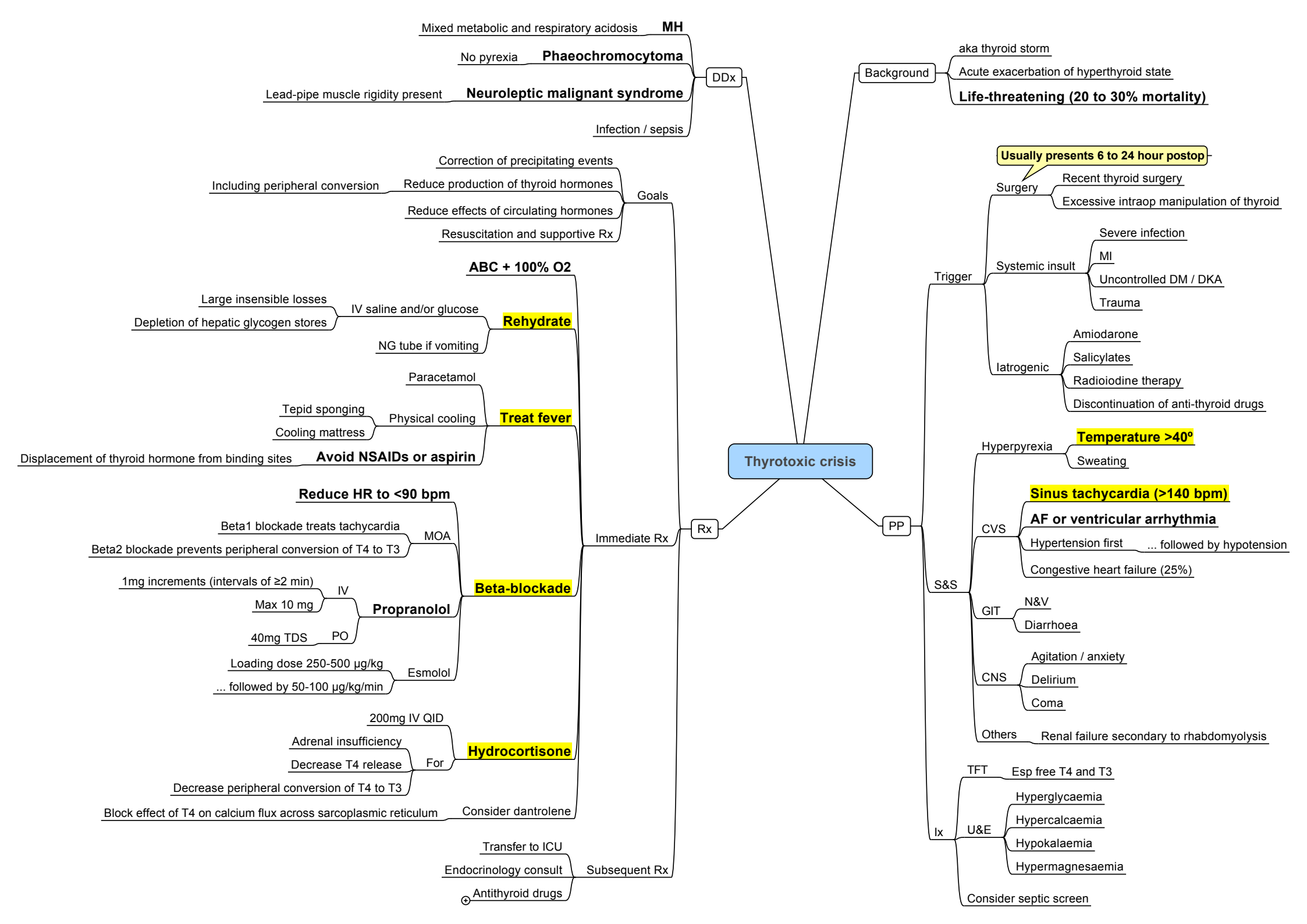
- **Tet spells**
  - ... with rapid deep breathing — Episodic hypercyanosis
  - Peak incidence at 2 to 4 months of age
  - Trigger
    - Crying
    - Exertion — Increased oxygen demand
    - Acidosis
    - Hypercapnoea
  - Rx
    - Oxygen
    - Manoeuvres
      - Abdominal compression
      - Knee-chest position / squatting
      - Flexion of leg
    - IV morphine — MOA unclear
    - Fluid
    - Beta blockers — ?RVOT relaxation
    - Alpha-agonists — e.g. phenylephrine

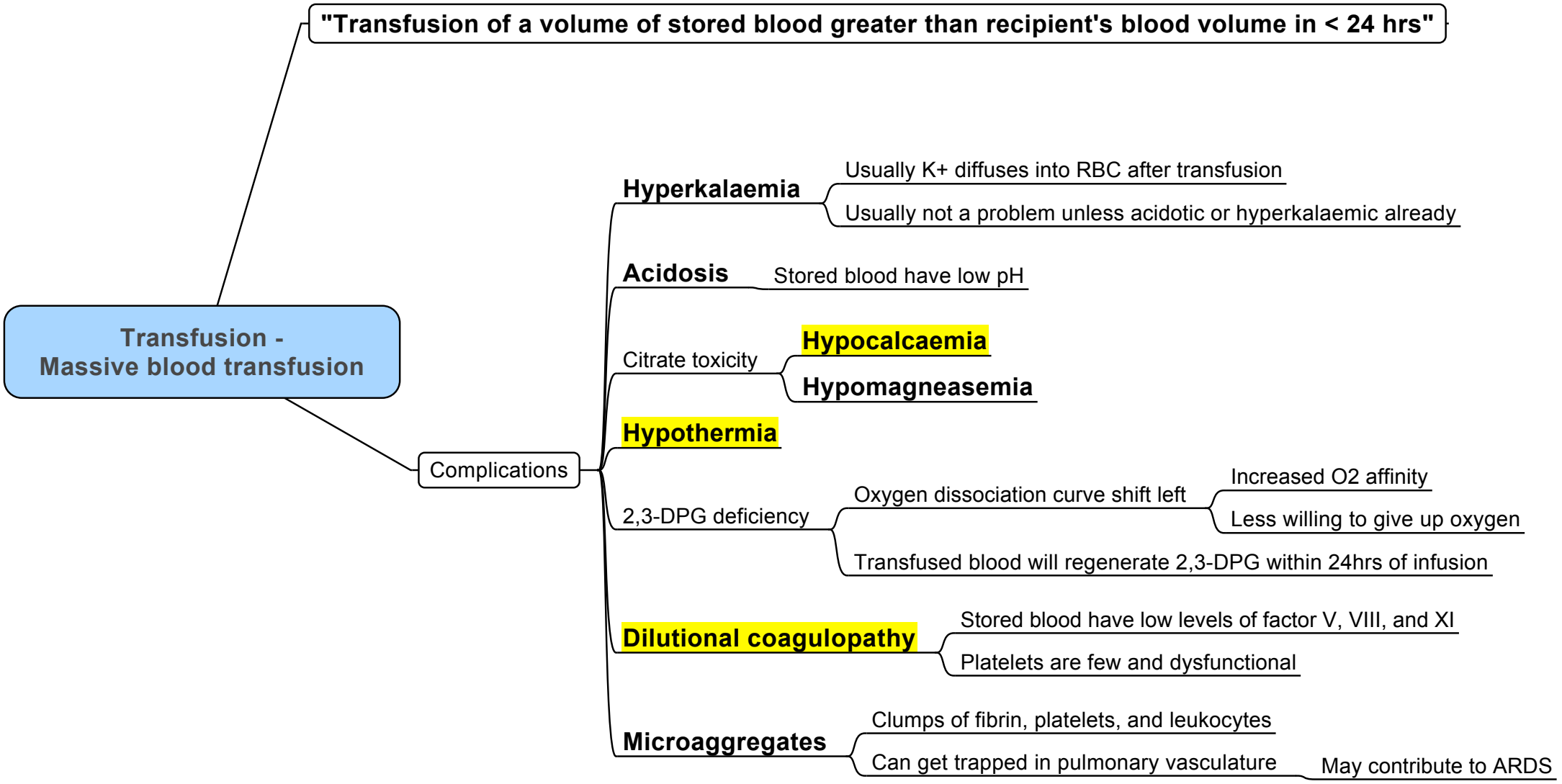
- POST-repair
  - Ventricular arrhythmia
  - **Possible conduction defects**
    - AF
    - RBBB
  - Possible pulmonary valve regurgitation
  - RV wall motion abnormality
  - **Associated conditions**
  - **Bacterial endocarditis prophylaxis**

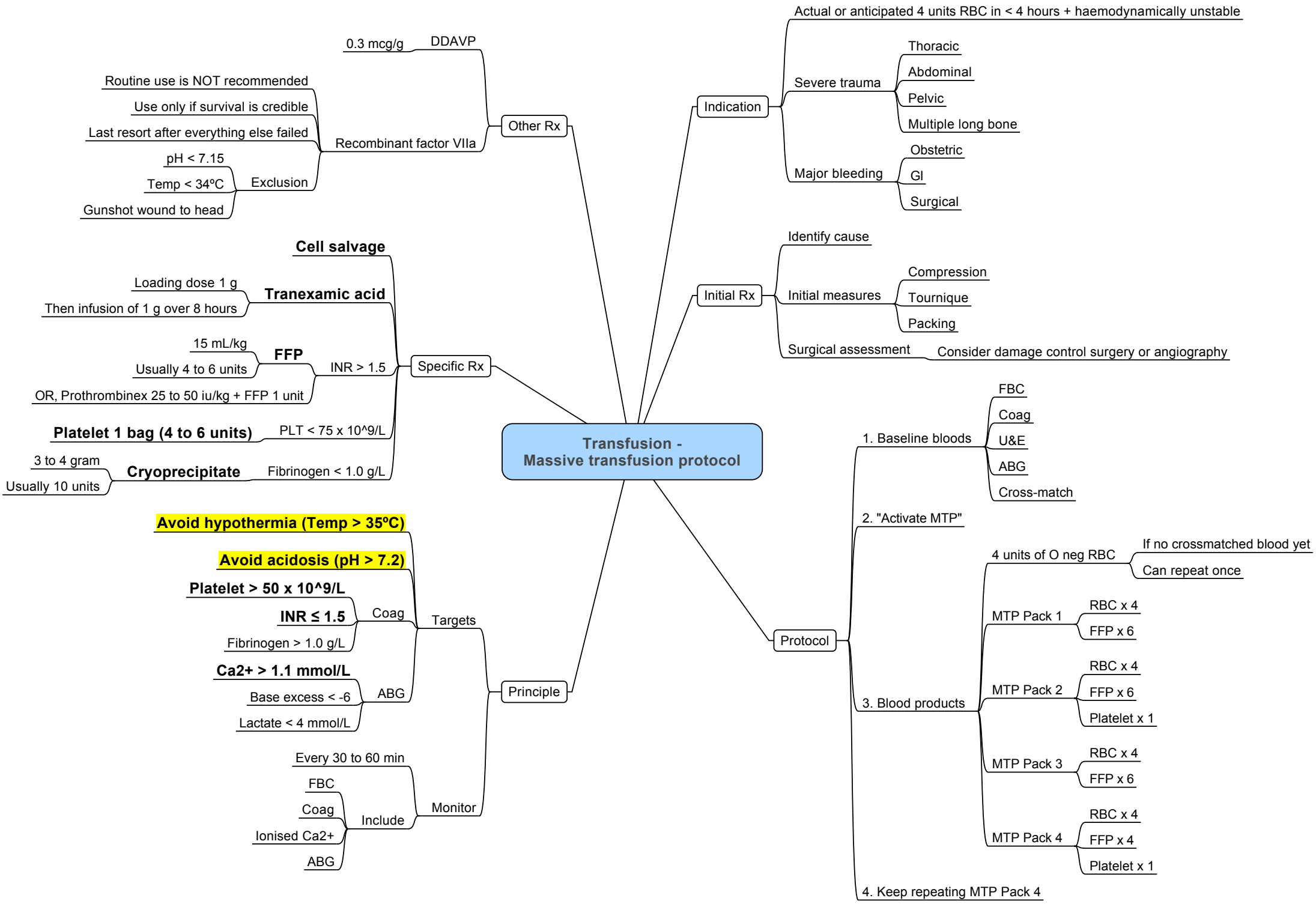

Most issues are seen only in thalassaemia major
Thalassaemia
Basics
Prevalent in some populations
Mediterranean (mainly beta)
African (A and B)
Asian (mainly A)
Autosomal recessive
S&S range from asymptomatic to transfusion dependent
Types
Alpha thalassaemia
Decreased alpha chain production
Beta thalassaemia
Decreased beta chain production
Thalassaemia major
Severe anaemia
Transfusion-dependent
Thus
Iron overload → Organ damage
Antibody production
Massive splenomegaly
Medullary and extramedullary erythropoesis
Bone deformities
Chipmunk facies
Scoliosis
Growth retardation
Issues
A+B
Difficult intubation
Zygoma hyperplasia
Relative mandibular hypoplasia
Restrictive lung disease
Due to scoliosis
C
Arrhythmia
Cardiomyopathy
High-output congestive heart failure
E
Anaemia
Coagulopathy
Endocrine
Diabetes mellitus (DM)
Adrenal insufficiency
Hypothyroidism
Others
Time-consuming blood cross-match
Liver cirrhosis
Careful positioning required
Scoliosis
Osteoporsis

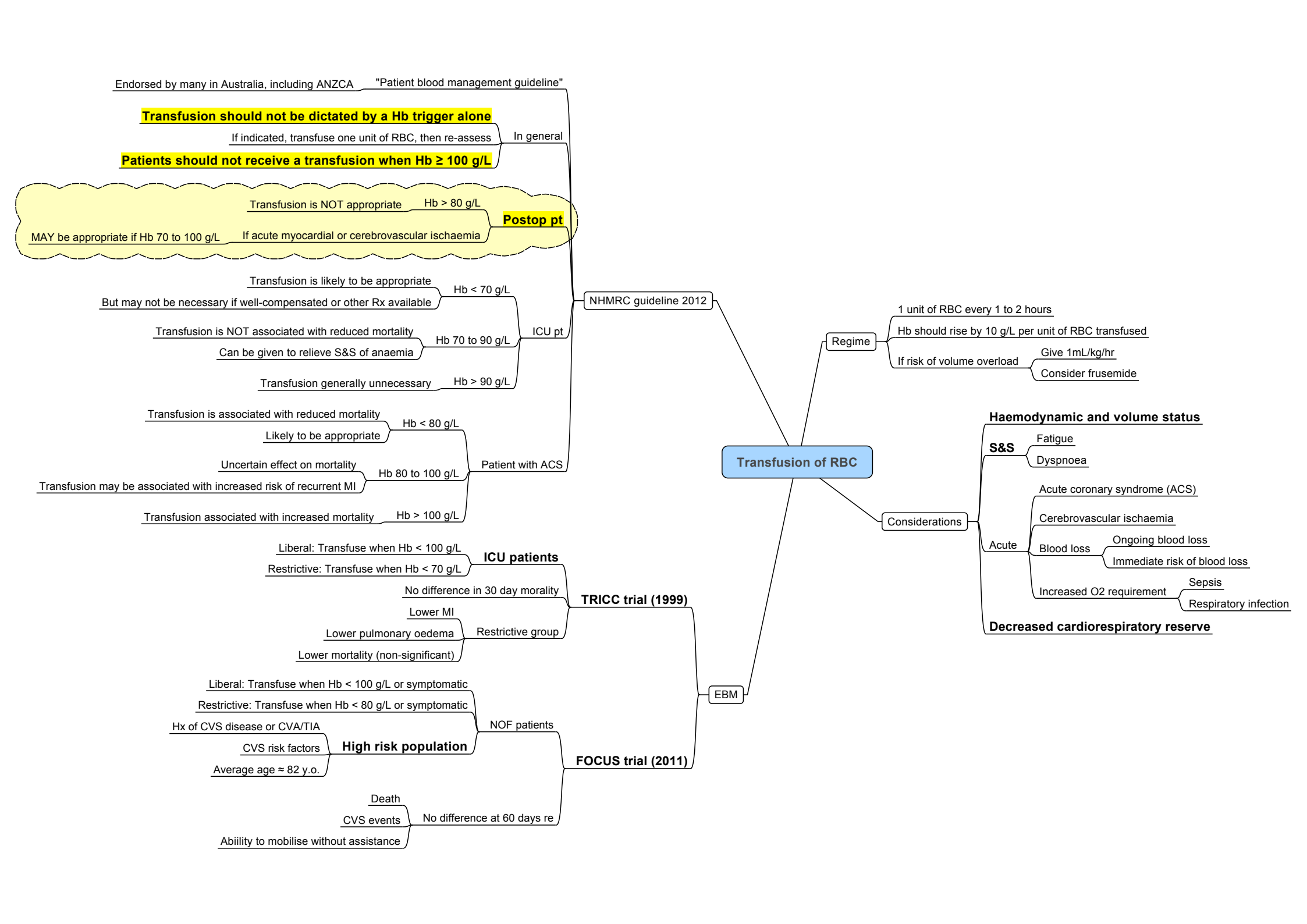

Thrombocytopenia
Causes
Decreased production
Platelet only
Hereditary
Drug and alcohol
Viral infection
Bone marrow failure
Aplasia
Cytotoxics
e.g. methotrexate
Bone marrow filtration / fibrosis
Radiotherapy
Megaloblastic anaemia
Increased consumption
Immunological
ITP
Drugs
e.g. Heparin, quinidine
Rheumatological disorder
e.g. SLE
Lymphoproliferative disorders
Non-immunological
DIC
Preeclampsia / HELLP
TTP
Dilution
PLT level usually maintained until > 1.5 to 2 blood volumes replaced
Splenic pooling
Consultation
Look for
Generalised petechiae
Purpura
Bleeding from mucous membranes
Indicates high risk of bleeding
Optimisation
Unexplained thrombocytopenia needs to be investigated
Identify and treat underlying cause
Seek haematologist advice early
Issues
Excessive periop bleeding
Underlying cause for thrombocytopenia
May be associated with
Anaemia
Hypovolaemia
Goals
Target platelet level
Depends on the surgery
Most procedures require 50 x 10^9/L
Some require > 100 x 10^9/L
Neurosurgery
Eye surgery
Neuraxial block
Haemostasis adequate when PLT > 100 x 10^9/L
Ensure adequate supply at blood bank
Crossed-matched blood
Platelet
Avoid tissue trauma
Avoid nasal intubation
Gentle with orotracheal suctioning
Avoid antiplatelet Rx, NSAIDs, heparin, IM injection
Others
Platelet transfusion
Spontaneous bleeding uncommon until PLT < 20 x 10^9/L
1 pooled bag contains 4 to 6 units of platelets
1 pooled bag increases platelet level by 20 to 40 x 10^9/L
Neuroaxial block
Safe when PLT > 100 x 10^9/L
Acceptable risk if PLT > 80 x 10^9/L AND INR < 1.5

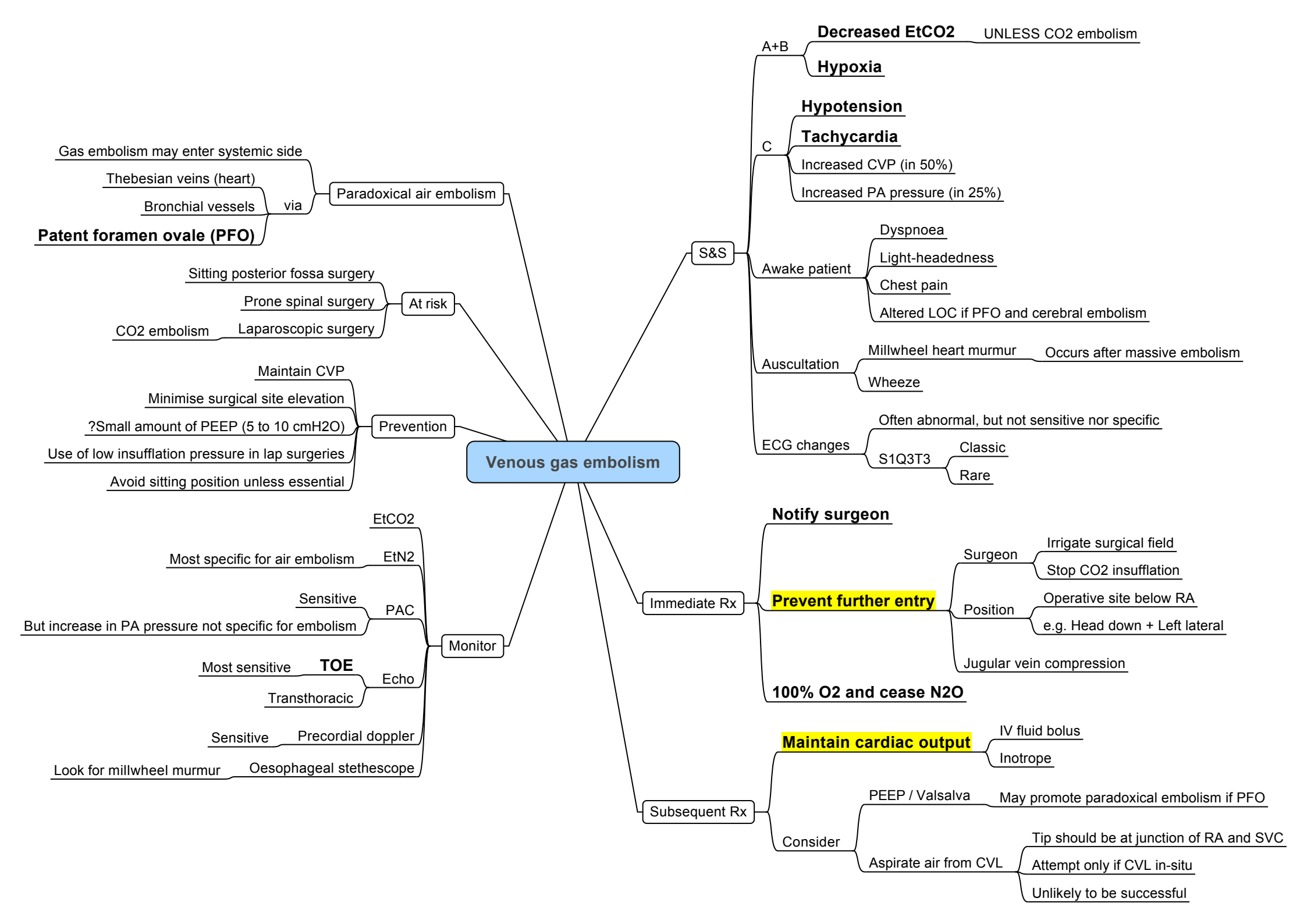

Thyroidectomy - AMx
Consultation
History
Surgical indication
Airway may be more difficult if malignancy
Confirm euthyroid state
HR < 80
No tremour
Check
S&S due to hyperthyroidism
Airway compression
Positional dyspnoea
Stridor
SVC obstruction
Venous distension in neck, upper chest, arms
Pemberton's sign
Rx
Propanolol
Steroids
Propylthiouracil
Hepatic dysfunction
Agranulocytosis
Exam
Airway assessment
Thyroid size and position
Cricothyrodotomy possible?
Ix
ECG
Bloods
FBC, U&E, LFT
TFT
CXR
Tracheal deviation / narrowing
CT
Retrostenal spread
Airway compression / distortion
Intraluminal spread
Optimisation
Delay surgery till euthyroid
Consider referral to endocrinologist
ENT to document vocal cord function
i.e. Nasoendoscopy
For medicolegal reasons
Issues
P
Effects of hyperthyroidism
Mass effect
Airway compression
SVC obstruction
S
Coughs due to intraop manipulation of trachea
Intraop risks
Pneumothorax
Air embolism if significant head up position
Major blood loss if retrosternal goitre
Postop complications
Thyroid storm
Stridor
Hypocalcaemia
A
No access to airway intraop
Plan
MADE
M
Temp
Nerve stimulator
Consider IBP
D
Consider LA spray to ETT or vocal cord
E
Consider
Smaller ETT size
Reinforced ETT
Patient setup
Avoid ties around neck
Secure ETT with tape
Eye protection, esp if exophthalmos
Avoid excessive neck extension on setup
Arms to side with IV extension
Intra-op
May need to withdraw ETT to assess tracheomalacia
Extubation
Immediate reintubation if respiratory difficulty on extubation
Postop
Monitor for
Stridor
Hypocalcaemia
Due to removal of parathyroid glands
Check Ca2+ at 24 hours
Keep stitch removal kit with the patient at all times
Check wound before discharge from PACU
Goals
Difficult airway management
Prevent any patient coughs or movement intraop
Full muscle relaxation
Prepare for complications
esp
Thyroid storm
Airway compromise
Others
If malignancy, consider...
Cord palsies
Distortion and rigidity of surrounding structures
Intraluminal spread
Larynx involvement
Alternate airway options
Inhalational induction
Topical LA to cord
Short time window for intubation
Awake fibre-optic intubation
Useful if laryngeal involvement
Tracheostomy under LA
Depend on level of obstruction
May cause heavy bleeding
Ventilation through rigid bronchoscope
Possible causes of stridor
Bilateral recurrent laryngeal nerve palsies
Tracheomalacia
Increased risk with longstanding goitre
Haematoma
Direct airway compression
Consider removing stitches at bedside

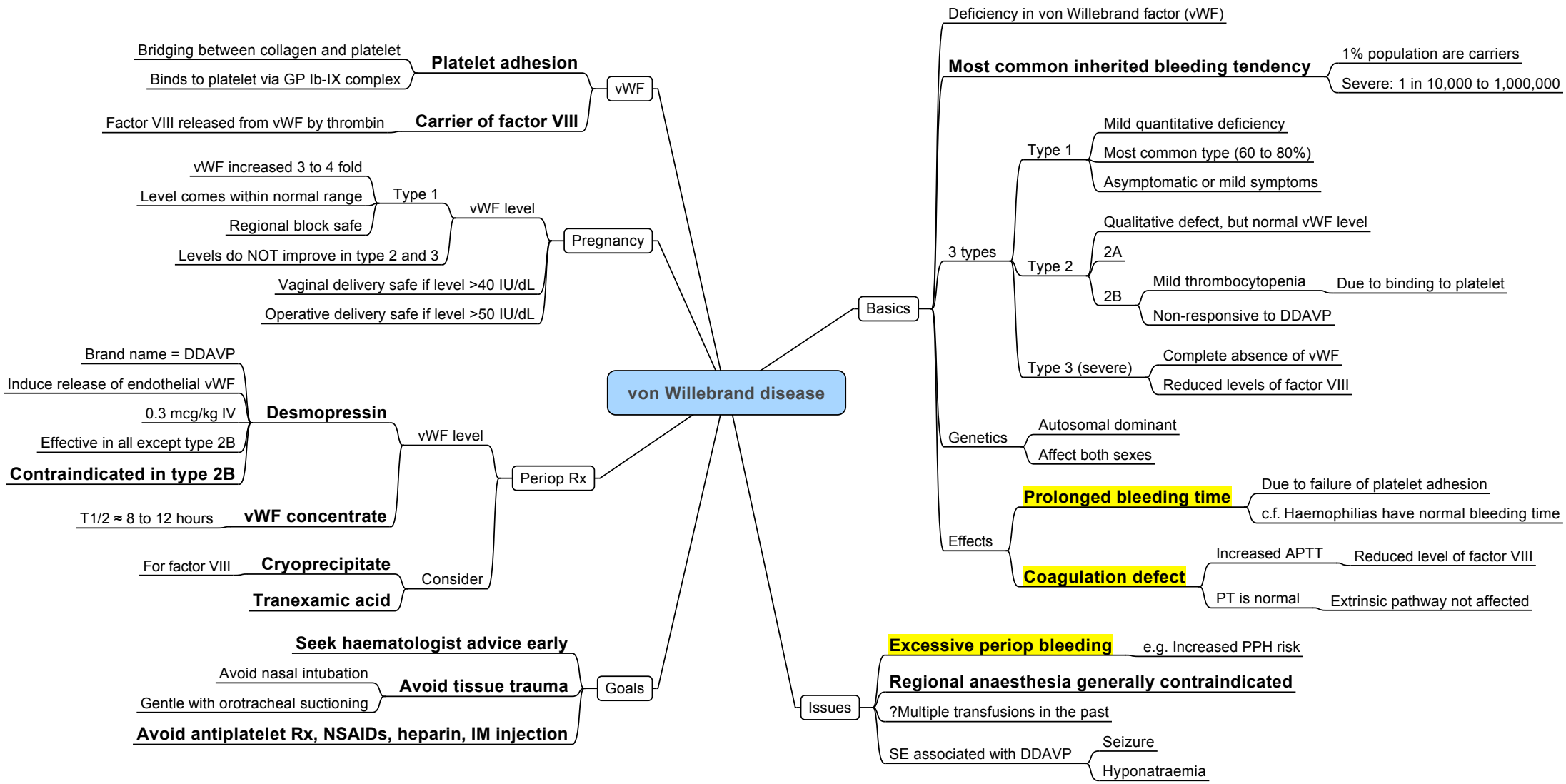

Thyrotoxic crisis
DDx
Mixed metabolic and respiratory acidosis
MH
No pyrexia
Phaeochromocytoma
Lead-pipe muscle rigidity present
Neuroleptic malignant syndrome
Infection / sepsis
Background
aka thyroid storm
Acute exacerbation of hyperthyroid state
Life-threatening (20 to 30% mortality)
PP
Trigger
Surgery
Usually presents 6 to 24 hour postop
Recent thyroid surgery
Excessive intraop manipulation of thyroid
Systemic insult
Severe infection
MI
Uncontrolled DM / DKA
Trauma
Iatrogenic
Amiodarone
Salicylates
Radioiodine therapy
Discontinuation of anti-thyroid drugs
S&S
Hyperpyrexia
Temperature >40º
Sweating
CVS
Sinus tachycardia (>140 bpm)
AF or ventricular arrhythmia
Hypertension first
... followed by hypotension
Congestive heart failure (25%)
GIT
N&V
Diarrhoea
CNS
Agitation / anxiety
Delirium
Coma
Others
Renal failure secondary to rhabdomyolysis
Ix
TFT
Esp free T4 and T3
U&E
Hyperglycaemia
Hypercalcaemia
Hypokalaemia
Hypermagnesaemia
Consider septic screen
Rx
Goals
Correction of precipitating events
Reduce production of thyroid hormones
Including peripheral conversion
Reduce effects of circulating hormones
Resuscitation and supportive Rx
Immediate Rx
ABC + 100% O2
Rehydrate
Large insensible losses
IV saline and/or glucose
Depletion of hepatic glycogen stores
NG tube if vomiting
Treat fever
Paracetamol
Physical cooling
Tepid sponging
Cooling mattress
Avoid NSAIDs or aspirin
Displacement of thyroid hormone from binding sites
Beta-blockade
Reduce HR to <90 bpm
MOA
Beta1 blockade treats tachycardia
Beta2 blockade prevents peripheral conversion of T4 to T3
Propranolol
IV
1mg increments (intervals of ≥2 min)
Max 10 mg
PO
40mg TDS
Esmolol
Loading dose 250-500 µg/kg
... followed by 50-100 µg/kg/min
Hydrocortisone
200mg IV QID
For
Adrenal insufficiency
Decrease T4 release
Decrease peripheral conversion of T4 to T3
Consider dantrolene
Block effect of T4 on calcium flux across sarcoplasmic reticulum
Subsequent Rx
Transfer to ICU
Endocrinology consult
Antithyroid drugs

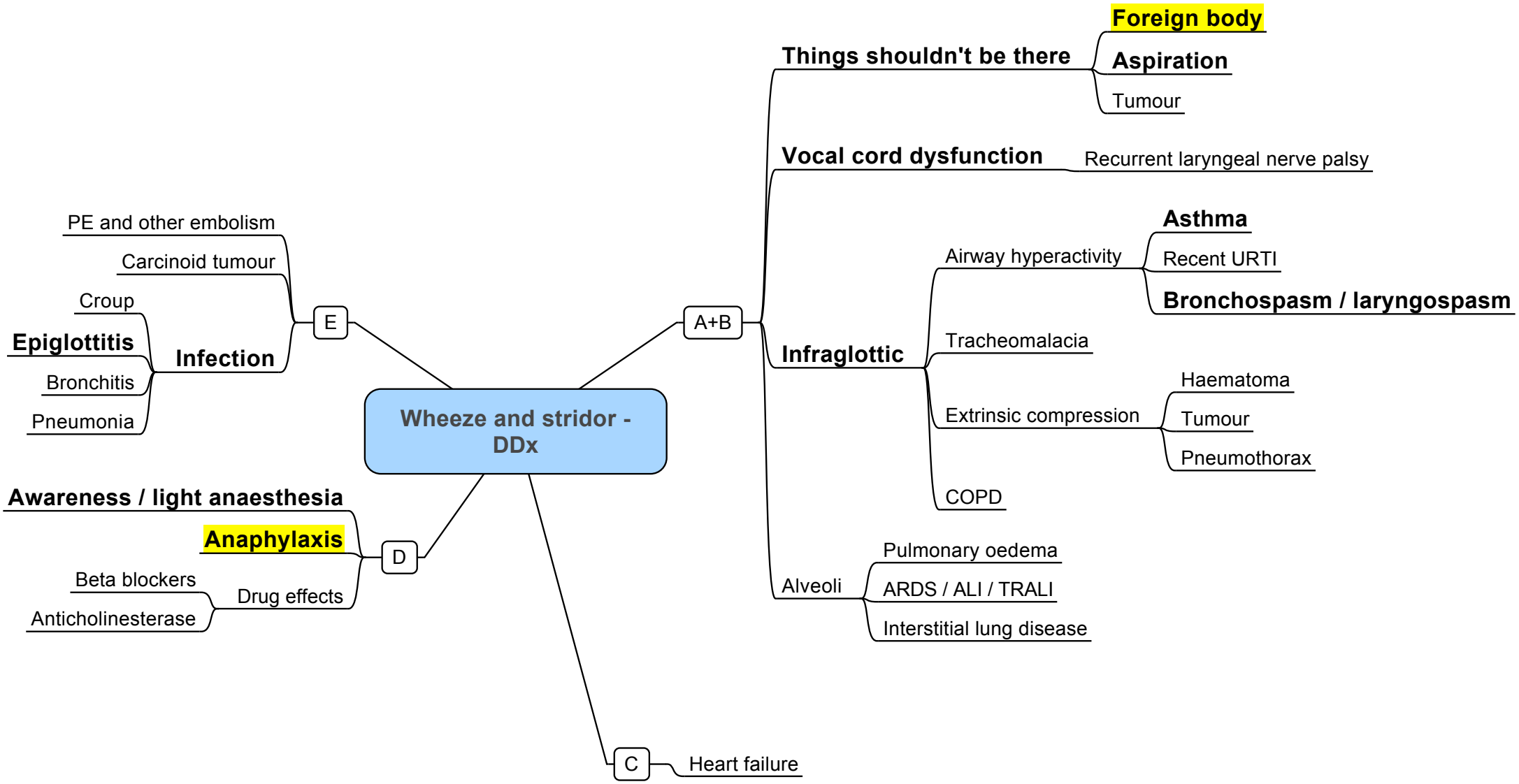

"Transfusion of a volume of stored blood greater than recipient's blood volume in < 24 hrs"
Transfusion - Massive blood transfusion
Complications
Hyperkalaemia
Usually K+ diffuses into RBC after transfusion
Usually not a problem unless acidotic or hyperkalaemic already
Acidosis
Stored blood have low pH
Citrate toxicity
Hypocalcaemia
Hypomagneasemia
Hypothermia
2,3-DPG deficiency
Oxygen dissociation curve shift left
Increased O2 affinity
Less willing to give up oxygen
Transfused blood will regenerate 2,3-DPG within 24hrs of infusion
Dilutional coagulopathy
Stored blood have low levels of factor V, VIII, and XI
Platelets are few and dysfunctional
Microaggregates
Clumps of fibrin, platelets, and leukocytes
Can get trapped in pulmonary vasculature
May contribute to ARDS

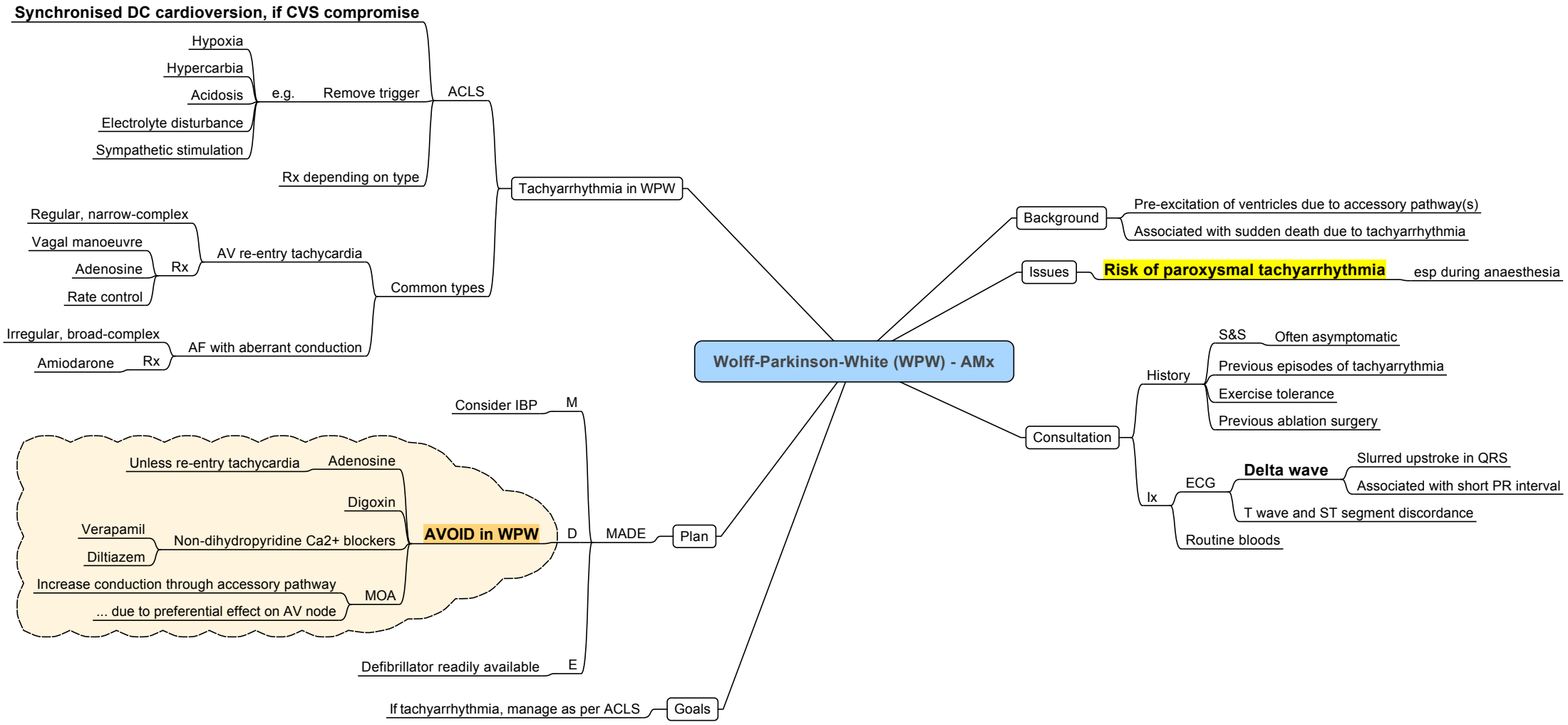

Transfusion -
Massive transfusion protocol
Indication
Actual or anticipated 4 units RBC in < 4 hours + haemodynamically unstable
Severe trauma
Thoracic
Abdominal
Pelvic
Multiple long bone
Major bleeding
Obstetric
GI
Surgical
Initial Rx
Identify cause
Initial measures
Compression
Tournique
Packing
Surgical assessment
Consider damage control surgery or angiography
Protocol
1. Baseline bloods
FBC
Coag
U&E
ABG
Cross-match
2. "Activate MTP"
4 units of O neg RBC
If no crossmatched blood yet
Can repeat once
3. Blood products
MTP Pack 1
RBC x 4
FFP x 6
MTP Pack 2
RBC x 4
FFP x 6
Platelet x 1
MTP Pack 3
RBC x 4
FFP x 6
MTP Pack 4
RBC x 4
FFP x 4
Platelet x 1
4. Keep repeating MTP Pack 4
Principle
Targets
Avoid hypothermia (Temp > 35ºC)
Avoid acidosis (pH > 7.2)
Coag
Platelet > 50 x 10^9/L
INR ≤ 1.5
Fibrinogen > 1.0 g/L
ABG
Ca2+ > 1.1 mmol/L
Base excess < -6
Lactate < 4 mmol/L
Monitor
Every 30 to 60 min
Include
FBC
Coag
Ionised Ca2+
ABG
Specific Rx
Cell salvage
Tranexamic acid
Loading dose 1 g
Then infusion of 1 g over 8 hours
INR > 1.5
FFP
15 mL/kg
Usually 4 to 6 units
OR, Prothrombinex 25 to 50 iu/kg + FFP 1 unit
Platelet 1 bag (4 to 6 units)
PLT < 75 x 10^9/L
Cryoprecipitate
3 to 4 gram
Usually 10 units
Fibrinogen < 1.0 g/L
Other Rx
DDAVP
0.3 mcg/g
Recombinant factor VIIa
Routine use is NOT recommended
Use only if survival is credible
Last resort after everything else failed
Exclusion
pH < 7.15
Temp < 34ºC
Gunshot wound to head


Transfusion of RBC
NHMRC guideline 2012
"Patient blood management guideline"
Endorsed by many in Australia, including ANZCA
In general
Transfusion should not be dictated by a Hb trigger alone
If indicated, transfuse one unit of RBC, then re-assess
Patients should not receive a transfusion when Hb ≥ 100 g/L
Postop pt
Hb > 80 g/L
Transfusion is NOT appropriate
If acute myocardial or cerebrovascular ischaemia
MAY be appropriate if Hb 70 to 100 g/L
ICU pt
Hb < 70 g/L
Transfusion is likely to be appropriate
But may not be necessary if well-compensated or other Rx available
Hb 70 to 90 g/L
Transfusion is NOT associated with reduced mortality
Can be given to relieve S&S of anaemia
Hb > 90 g/L
Transfusion generally unnecessary
Patient with ACS
Hb < 80 g/L
Transfusion is associated with reduced mortality
Likely to be appropriate
Hb 80 to 100 g/L
Uncertain effect on mortality
Transfusion may be associated with increased risk of recurrent MI
Hb > 100 g/L
Transfusion associated with increased mortality
EBM
TRICC trial (1999)
ICU patients
Liberal: Transfuse when Hb < 100 g/L
Restrictive: Transfuse when Hb < 70 g/L
No difference in 30 day morality
Restrictive group
Lower MI
Lower pulmonary oedema
Lower mortality (non-significant)
FOCUS trial (2011)
NOF patients
Liberal: Transfuse when Hb < 100 g/L or symptomatic
Restrictive: Transfuse when Hb < 80 g/L or symptomatic
High risk population
Hx of CVS disease or CVA/TIA
CVS risk factors
Average age ≈ 82 y.o.
No difference at 60 days re
Death
CVS events
Abiility to mobilise without assistance
Regime
1 unit of RBC every 1 to 2 hours
Hb should rise by 10 g/L per unit of RBC transfused
If risk of volume overload
Give 1mL/kg/hr
Consider frusemide
Considerations
Haemodynamic and volume status
S&S
Fatigue
Dyspnoea
Acute
Acute coronary syndrome (ACS)
Cerebrovascular ischaemia
Blood loss
Ongoing blood loss
Immediate risk of blood loss
Increased O2 requirement
Sepsis
Respiratory infection
Decreased cardiorespiratory reserve


Venous gas embolism
S&S
A+B
Decreased EtCO2
UNLESS CO2 embolism
Hypoxia
C
Hypotension
Tachycardia
Increased CVP (in 50%)
Increased PA pressure (in 25%)
Awake patient
Dyspnoea
Light-headedness
Chest pain
Altered LOC if PFO and cerebral embolism
Auscultation
Millwheel heart murmur
Occurs after massive embolism
Wheeze
ECG changes
Often abnormal, but not sensitive nor specific
S1Q3T3
Classic
Rare
Immediate Rx
Notify surgeon
Prevent further entry
Surgeon
Irrigate surgical field
Stop CO2 insufflation
Position
Operative site below RA
e.g. Head down + Left lateral
Jugular vein compression
100% O2 and cease N2O
Subsequent Rx
Maintain cardiac output
IV fluid bolus
Inotrope
Consider
PEEP / Valsalva
May promote paradoxical embolism if PFO
Aspirate air from CVL
Tip should be at junction of RA and SVC
Attempt only if CVL in-situ
Unlikely to be successful
Monitor
EtCO2
EtN2
Most specific for air embolism
PAC
Sensitive
But increase in PA pressure not specific for embolism
Echo
Most sensitive
TOE
Transthoracic
Precordial doppler
Sensitive
Oesophageal stethescope
Look for millwheel murmur
Prevention
Maintain CVP
Minimise surgical site elevation
?Small amount of PEEP (5 to 10 cmH2O)
Use of low insufflation pressure in lap surgeries
Avoid sitting position unless essential
At risk
Sitting posterior fossa surgery
Prone spinal surgery
Laparoscopic surgery
CO2 embolism
Paradoxical air embolism
Gas embolism may enter systemic side
via
Thebesian veins (heart)
Bronchial vessels
Patent foramen ovale (PFO)


von Willebrand disease
Basics
Deficiency in von Willebrand factor (vWF)
Most common inherited bleeding tendency
1% population are carriers
Severe: 1 in 10,000 to 1,000,000
3 types
Type 1
Mild quantitative deficiency
Most common type (60 to 80%)
Asymptomatic or mild symptoms
Type 2
Qualitative defect, but normal vWF level
2A
2B
Mild thrombocytopenia
Due to binding to platelet
Non-responsive to DDAVP
Type 3 (severe)
Complete absence of vWF
Reduced levels of factor VIII
Genetics
Autosomal dominant
Affect both sexes
Effects
Prolonged bleeding time
Due to failure of platelet adhesion
c.f. Haemophilias have normal bleeding time
Coagulation defect
Increased APTT
Reduced level of factor VIII
PT is normal
Extrinsic pathway not affected
Issues
Excessive periop bleeding
e.g. Increased PPH risk
Regional anaesthesia generally contraindicated
?Multiple transfusions in the past
SE associated with DDAVP
Seizure
Hyponatraemia
Goals
Seek haematologist advice early
Avoid tissue trauma
Avoid nasal intubation
Gentle with orotracheal suctioning
Avoid antiplatelet Rx, NSAIDs, heparin, IM injection
Periop Rx
vWF level
Desmopressin
Brand name = DDAVP
Induce release of endothelial vWF
0.3 mcg/kg IV
Effective in all except type 2B
Contraindicated in type 2B
vWF concentrate
T1/2 ≈ 8 to 12 hours
Consider
Cryoprecipitate
For factor VIII
Tranexamic acid
Pregnancy
vWF level
Type 1
vWF increased 3 to 4 fold
Level comes within normal range
Regional block safe
Levels do NOT improve in type 2 and 3
Vaginal delivery safe if level >40 IU/dL
Operative delivery safe if level >50 IU/dL
vWF
Platelet adhesion
Bridging between collagen and platelet
Binds to platelet via GP Ib-IX complex
Carrier of factor VIII
Factor VIII released from vWF by thrombin


Wheeze and stridor - DDx
A+B
Things shouldn't be there
Foreign body
Aspiration
Tumour
Vocal cord dysfunction
Recurrent laryngeal nerve palsy
Infraglottic
Airway hyperactivity
Asthma
Recent URTI
Bronchospasm / laryngospasm
Tracheomalacia
Extrinsic compression
Haematoma
Tumour
Pneumothorax
COPD
Alveoli
Pulmonary oedema
ARDS / ALI / TRALI
Interstitial lung disease
E
Infection
Epiglottitis
Croup
Bronchitis
Pneumonia
PE and other embolism
Carcinoid tumour
D
Awareness / light anaesthesia
Anaphylaxis
Drug effects
Beta blockers
Anticholinesterase
C
Heart failure


Wolff-Parkinson-White (WPW) - AMx
Tachyarrhythmia in WPW
ACLS
Synchronised DC cardioversion, if CVS compromise
Remove trigger
e.g.
Hypoxia
Hypercarbia
Acidosis
Electrolyte disturbance
Sympathetic stimulation
Rx depending on type
Common types
AV re-entry tachycardia
Rx
Regular, narrow-complex
Vagal manoeuvre
Adenosine
Rate control
AF with aberrant conduction
Rx
Irregular, broad-complex
Amiodarone
Plan
MADE
M
Consider IBP
D
AVOID in WPW
Non-dihydropyridine Ca2+ blockers
Verapamil
Diltiazem
Unless re-entry tachycardia
Adenosine
Digoxin
MOA
Increase conduction through accessory pathway
... due to preferential effect on AV node
E
Defibrillator readily available
Goals
If tachyarrhythmia, manage as per ACLS
Background
Pre-excitation of ventricles due to accessory pathway(s)
Associated with sudden death due to tachyarrhythmia
Issues
Risk of paroxysmal tachyarrhythmia
esp during anaesthesia
Consultation
History
S&S
Often asymptomatic
Previous episodes of tachyarrythmia
Exercise tolerance
Previous ablation surgery
Ix
ECG
Delta wave
Slurred upstroke in QRS
Associated with short PR interval
T wave and ST segment discordance
Routine bloods

Abbreviations

? = Possible, query, maybe
AA = Anaesthetic agents
ABG = Arterial blood gas
ACLS = Advanced cardiac life support
ACS = Acute coronary syndrome
AF = Atrial fibrillation
AFM = Amniotic fluid embolism
AI = Aortic incompentence
ALI = Acute lung injury
AMx = Anaesthetic management
ANZCA = Australian and New Zealand College of Anaesthetists
APLS = Advanced paediatric life support
AR = Aortic regurgitation
ARDS = Acute respiratory distress syndrome
ARF = Acute renal failure
AS = Aortic stenosis
ASAP = As soon as possible
ATLS = Advanced trauma life support
AVR = Aortic valve replacement
BBB = Blood-brain barrier / Bundle branch block (rarely)
BLS = Basic life support
BP = Blood pressure
BSL = Blood sugar level
BVM = Bag valve mask (Usually refer to manual ventilation with a bag valve mask without an ETT)
CAD = Coronary artery disease
CBF = Cerebral blood flow
CCF = Congestive cardiac failure
CHF = Congestive heart failure

CNS = Central nervous system (Can also refer to Neurology / Neurological in general)
CO = Cardiac output / Carbon monoxide
Coag = Coagulation
COHb = Carboxyhaemoglobin
COPD = Chronic obstructive pulmonary disease (aka COAD)
CP = Cerebral palsy / coagulation profile
CPAP = Continuous positive airway pressure
CRF = Chronic renal failure
CVA = Cerebrovascular accidents (i.e. Stroke)
CVL = Central venous line
CVP = Central venous pressure
CVS = Cardiovascular
CXM = Cross-match
CXR = Chest X-ray
DBP = Diastolic blood pressure
DDx = Differential diagnosis / List of possible causes
deoxyHb = Deoxyhaemoglobin
DM = Diabetes Mellitus
DMD = Duchenne's muscular dystrophy
ECG = EKG. Do I really have to spell it out?
Echo = Echocardiography
esp = especially
ETT = Endotracheal tube
FBC = Full blood count
FFP = Fresh frozen plasma
G&H = Group and hold
GA = General anaesthesia
GFR = Glomerular filtrate rate
GI = Gastrointestinal
GIT = Gastrointestinal tract

GORD = Gastro-oesophageal reflux disease
GTN = Glyceryl trinitrate
HCM = Hypertrophic cardiomyopathy
HDU = High dependency unit
HOCM = Hypertrophic obstructive cardiomyopathy
HR = Heart rate
HTN = Hypertension
Hx = History
IBP = Invasive BP (measurement)
ICU = Intensive care unit
IHD - Ischaemic heart disease
IJ = Internal jugular (vein)
Intraop = Intraoperative(ly)
IPPV = Intermittent positive pressure ventilation
Ix = Investigation
JVP = Jugular venous pressure
LA = Local anaesthetics / Left atrium
LFT = Liver function test
LOC = Level of consciousness
LV = Left ventricle
LVH = Left ventricular hypertrophy
MADE = Preop preparation (monitor, assistance / vascular access, drugs, equipments)
MetHb = Methaemoglobin / methaemoglobinaemia
MH = Malignant hyperthermia
MI = Myocardial infarction
MOA = Mechanism of Action
MR = Mitral regurgitation
MS = Musculoskeletal / Multiple sclerosis / Mitral stenosis
MTP = Massive transfusion protocol
MVO2 = Myocardial oxygen demand

MVR = Mitral valve replacement
N&V = Nausea and vomiting
NG = Nasogastric
NGT = Nasogastric tube
NM = Neuromuscular
NMBDs = Neuromuscular blocking drugs (i.e. muscle relaxants)
NMJ = Neuromuscular junction
NSAID = Non-steroidal anti-inflammatory drug
ODC = Oxyhaemoglobin dissociation curve
Opt = Optimisation
OSA = Obstructive sleep apnoea
OxyHb = Oxyhaemoglobin
PA = Pulmonary artery
PAC = Pulmonary arterial catheter
PACO2 = Alveolar partial pressure of CO2
PaCO2 = Arterial partial pressure of CO2
PAP = Pulmonary arterial pressure
PASP = Pulmonary artery systolic pressure
PC = Pharmaceutical (Usually refer to packaging / drug formulation)
pCO2 = Partial pressure of CO2
PD = Pharmacodynamics
Periop = Perioperative(ly)
PFO = Patent foramen ovale
PK = Pharmacokinetics
PLT = Platelet
Postop = Postoperative(ly)
PP = Pathophysiology
PPH = Post-partum haemorrhage
Preop = Preoperative(ly)
PVR = Pulmonary vascular resistance

RA = Rheumatoid arthritis, or Right atrium
RAP = Right atrial pressure
RBC = Red blood cell
RF = Risk factors
RSI = Rapid sequence induction
RV = Right ventricular
Rx = Treatment (or medications)
S&S = Symptoms and signs (aka features)
SBP = Systolic blood pressure
SCD = Sequential compression device
SNP = Sodium nitroprusside
SpO2 = O2 saturation as per pulse oximetry
SVC = Superior vena cava
SVR = Systemic vascualr resistance
TBSA = Total body surface area
TED = T.E.D. compression stocking
Temp = Temperature
TFT = Thyroid function test
TIA = Transient ischaemic attack
TOE = Trans-oesophageal echocardiography
TR = Tricuspid regurgitation
TRALI = Transfusion-related acute lung injury
TSH = Thyroid stimulating hormone
U&E = Urea and electrolytes
vWF = von Willebrand factor
WBC = White blood cell
WPW = Wolff-Parkinson-White