
# Hospital At Night

**On Call Medicine Algorithms**

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5. **Neurology**
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   - ❖ TIA
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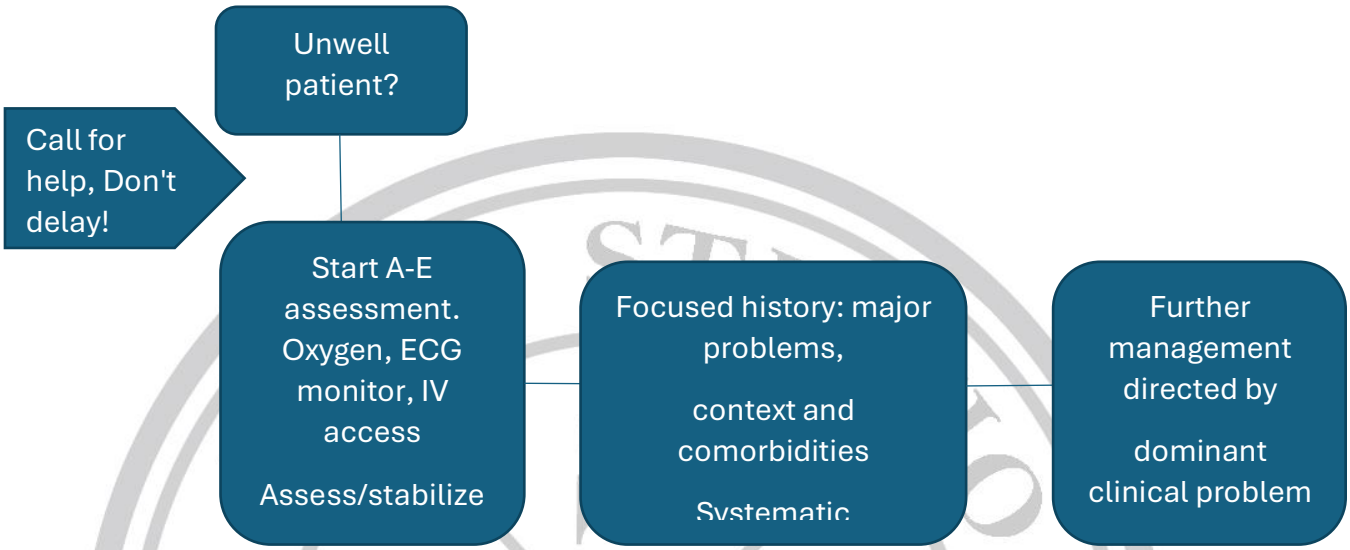
Section 1

# Common presentations

**Approaching an unwell patient:**



| Physiological parameter | Score 3 | 2 | 1 | 0 | 1 | 2 | 3 |
|---|---|---|---|---|---|---|---|
| Respiration rate (per minute) | ≤8 | | 9–11 | 12–20 | | 21–24 | ≥25 |
| $SpO_2$ Scale 1 (%) | ≤91 | 92–93 | 94–95 | ≥96 | | | |
| $SpO_2$ Scale 2 (%) | ≤83 | 84–85 | 86–87 | 88–92 ≥93 on air | 93–94 on oxygen | 95–96 on oxygen | ≥97 on oxygen |
| Air or oxygen? | | Oxygen | | Air | | | |
| Systolic blood pressure (mmHg) | ≤90 | 91–100 | 101–110 | 111–219 | | | ≥220 |
| Pulse (per minute) | ≤40 | | 41–50 | 51–90 | 91–110 | 111–130 | ≥131 |
| Consciousness | | | | Alert | | | CVPU |
| Temperature (°C) | ≤35.0 | | 35.1–36.0 | 36.1–38.0 | 38.1–39.0 | ≥39.1 | |

Table 1.1. Adult NEWS2 score

| NEW score | Clinical risk | Response |
|---|---|---|
| Aggregate score 0–4 | Low | Ward-based response |
| Red score<br>Score of 3 in any individual parameter | Low–medium | Urgent ward-based response* |
| Aggregate score 5–6 | Medium | Key threshold for urgent response* |
| Aggregate score 7 or more | High | Urgent or emergency response** |

* Response by a clinician or team with competence in the assessment and treatment of acutely ill patients and in recognising when the escalation of care to a critical care team is appropriate.

**The response team must also include staff with critical care skills, including airway management.

Table 1.2. Risk assessment using NEWS2 score

| Assessment | Signs | Cause | Action |
|---|---|---|---|
| **Air way** | Upper airway obstruction | Inhalation of Foreign body/secretions/ blood/vomit<br>Anaphylaxis<br>Angioedema | **-Conscious**<br>Sit the patient up<br>Give high flow<br>Call for help from anesthetic/ENT<br><br>**-Unconscious**<br>Head tilt and lift chin lift<br>Remove dentures<br>Suction<br>Call for help from anesthetic/ENT<br>Before intubation, ventilate the patient using a bag-mask device with 100% oxygen |
| **Breathing** | Respiratory rate <8 or >30/min<br><br>Oxygen saturation under 92% | Exacerbation of Underlying lung disease<br>Pulmonary embolus<br>pneumothorax<br>Pleural effusion | Supplemental oxygen<br>Increase inspired oxygen concentration if needed to achieve arterial oxygen saturation >90% (>88% in acute exacerbation of COPD)<br>Diagnose and treat underlying cause and contributory factors<br>If feasible, sit the patient up to improve diaphragmatic descent and increase tidal volume<br>Clear secretions: encourage cough, physiotherapy, aspiration<br>Drain large pleural effusion if present<br>Drain pneumothorax if present<br>Optimize cardiac output: treat hypotension and heart failure<br>Consider ventilatory support |
| **Circulatory** | Heart rate <40 or >130 bpm<br>SBP <90mmHg<br>CRT <2sec | Cardiac tamponade<br>Hypovolemic shock<br>Septic shock<br>Electrolyte abnormalities<br>Arrythmias | IV access<br>ECG-Correct major arrhythmia<br>Fluid resuscitation to correct hypovolemia (e.g. from acute blood loss or severe sepsis<br>Consider/exclude cardiac tamponade<br>Use inotropic vasopressor agent if there is pulmonary edema, or refractory |

| | | | |
|---|---|---|---|
| | | | hypotension despite fluid resuscitation<br>Diagnose and treat underlying cause<br>Correct major metabolic abnormalities (e.g. derangements of electrolytes) |
| **Disability** | AVPU<br>Temperature<br>pupils<br>Pain<br>Blood sugars | Stroke<br>Hyperglycemia<br>hypoglycemia | Correct hyper/hypoglycemia<br>Stroke/TIA management<br>Anti-pyrectics/bear huggers<br>Analgesia |
| **Exposure** | Bladder distention<br>Abdominal distention<br>PR bleed<br>DVT | Urinary retention<br>Abdominal emergency (section 4)<br>DVT | Bladder scan<br>Urinary catheterization<br>Imaging for abdomen<br>Doppler scan/ anticoagulation |

Table 1.3 summary of A-E assessment

| Investigations for critically unwell | |
|---|---|
| Immediate | Urgent |
| 1. Arterial blood gases<br>2. ECG<br>3. Blood glucose<br>4. Electrolytes and Renal profile<br>5. Full blood count | 1. Chest X-ray<br>2. Head CT if reduced GCS or focal signs<br>3. Coagulation screen if low platelet count, suspected coagulation disorder, jaundice or purpura<br>4. Biochemical profile<br>5. Amylase if abdominal pain or tenderness<br>6. C-reactive protein<br>7. Blood culture if suspected sepsis<br>8. Urine stick test<br>9. Toxicology screen (serum 10 ml and urine 50 ml) if suspected poisoning |

Table 1.4 Investigations to be requested for the unwell.

## Cardiac Arrest Algorithm

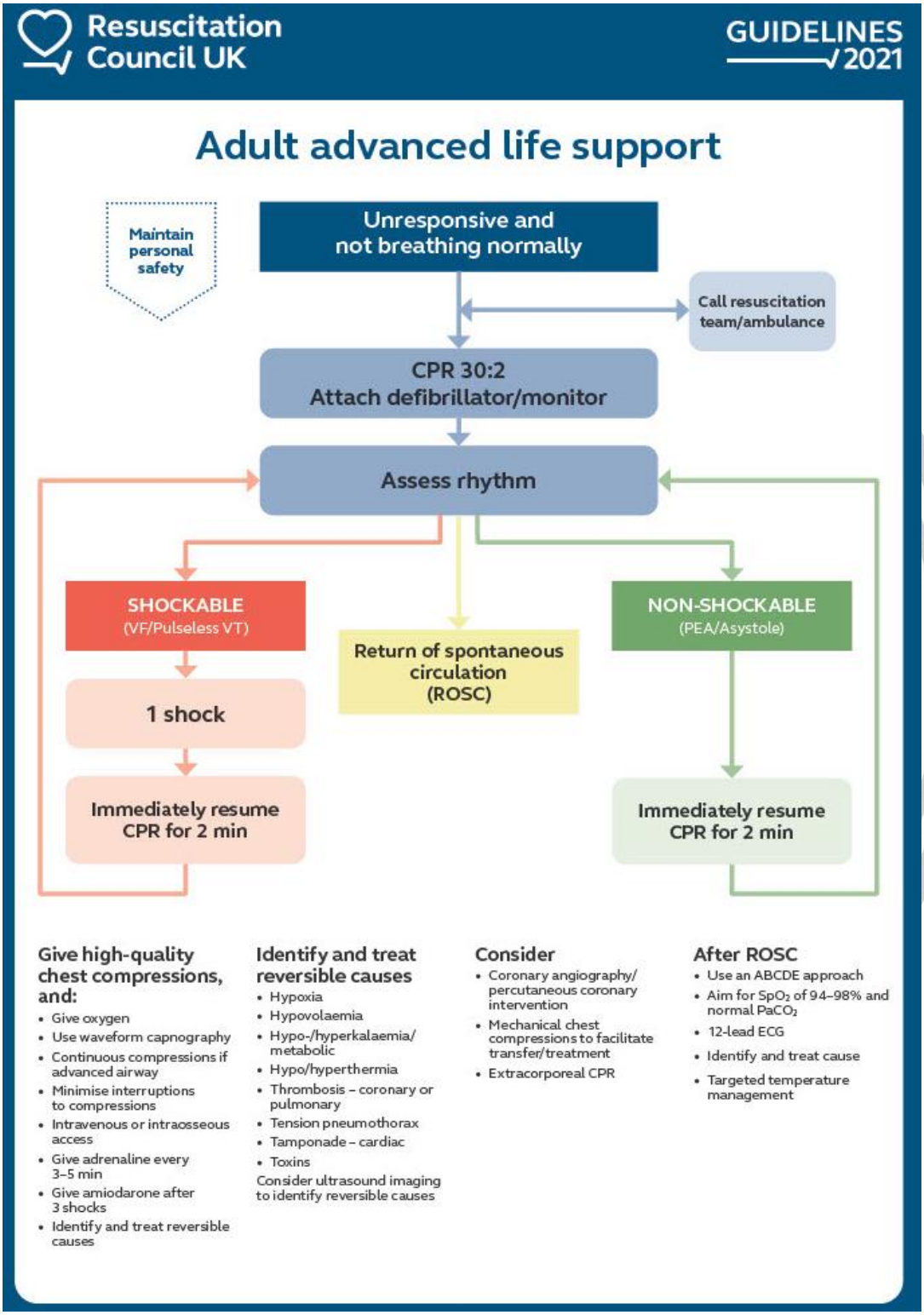



Figure: 1.1. Cardiac arrest algorithm by Resus-UK council

# Tachyarrhythmia

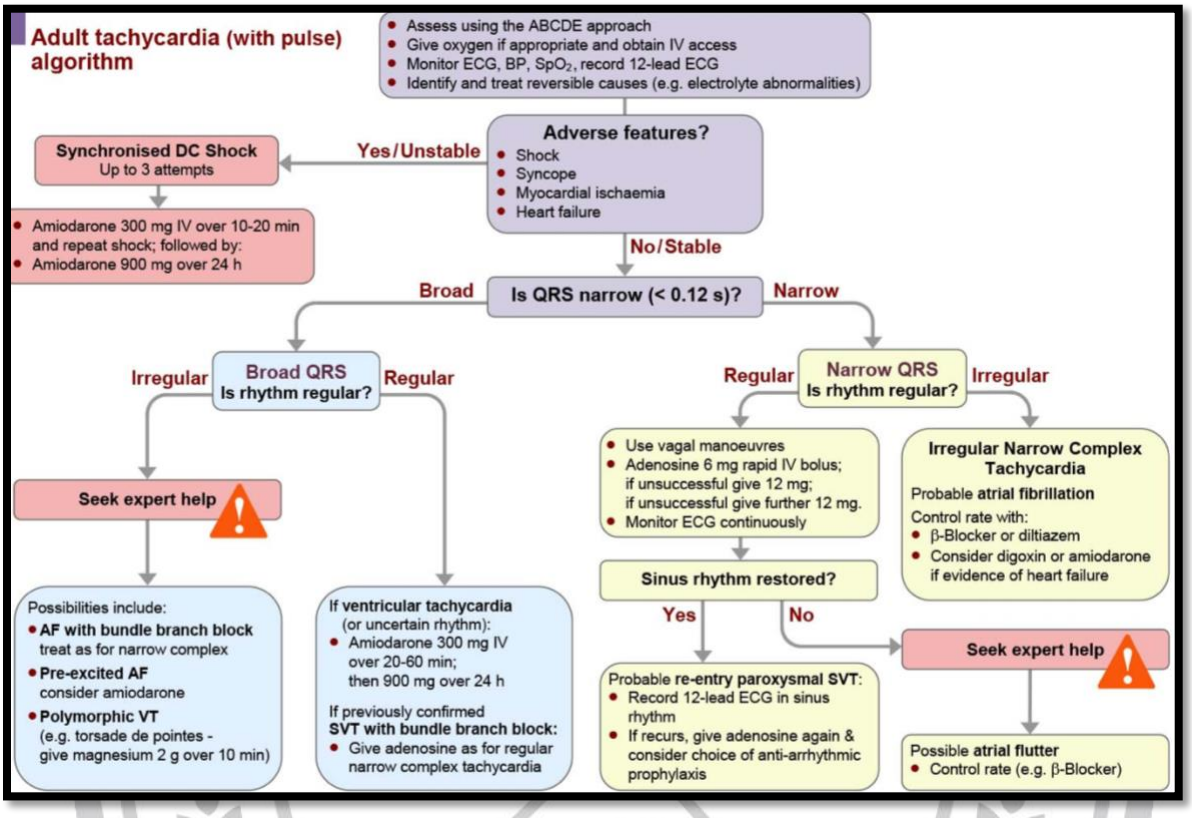



Figure 1.2. Tachyarrhythmia with pulse management

## Bradyarrythemia

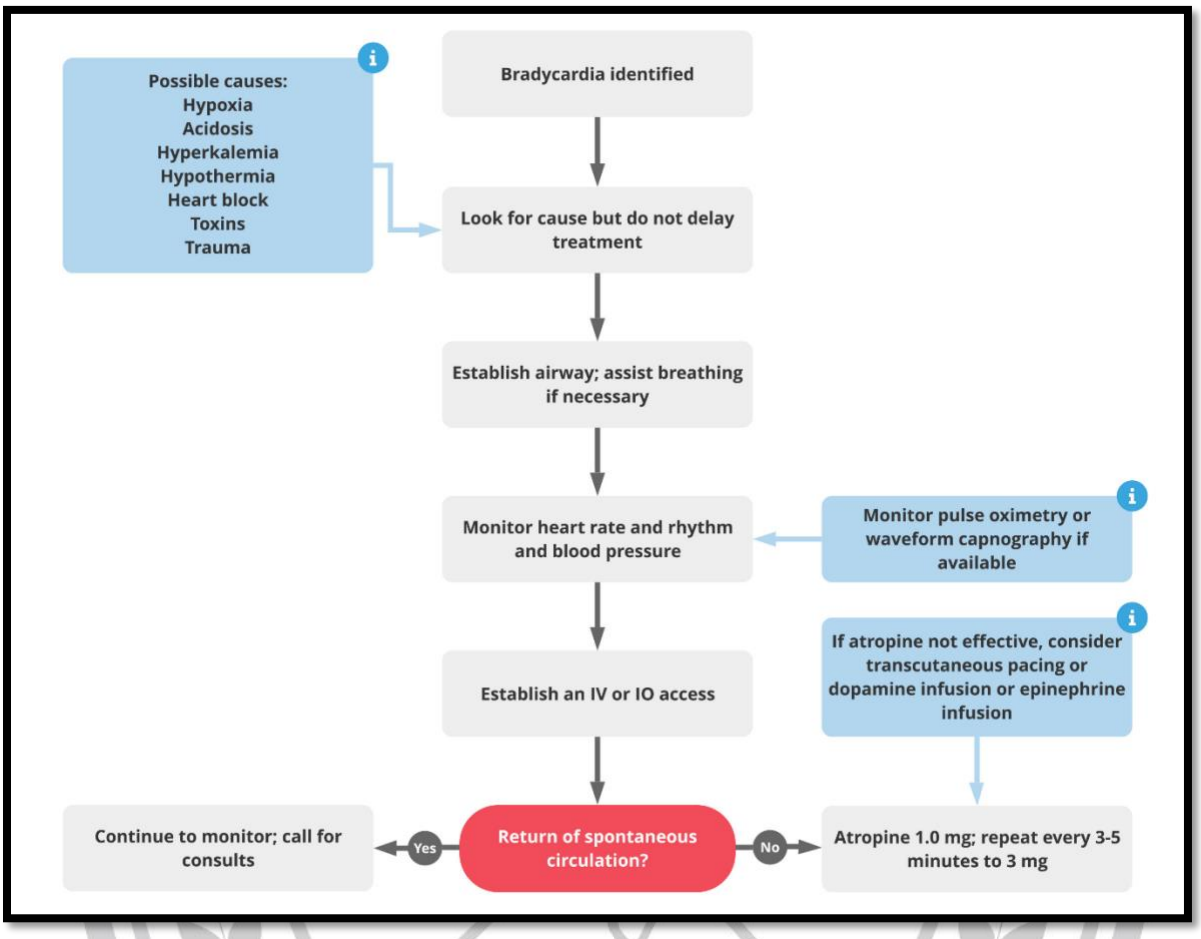



Figure 1.3. Bradyarrhythmia management

## Hypotension

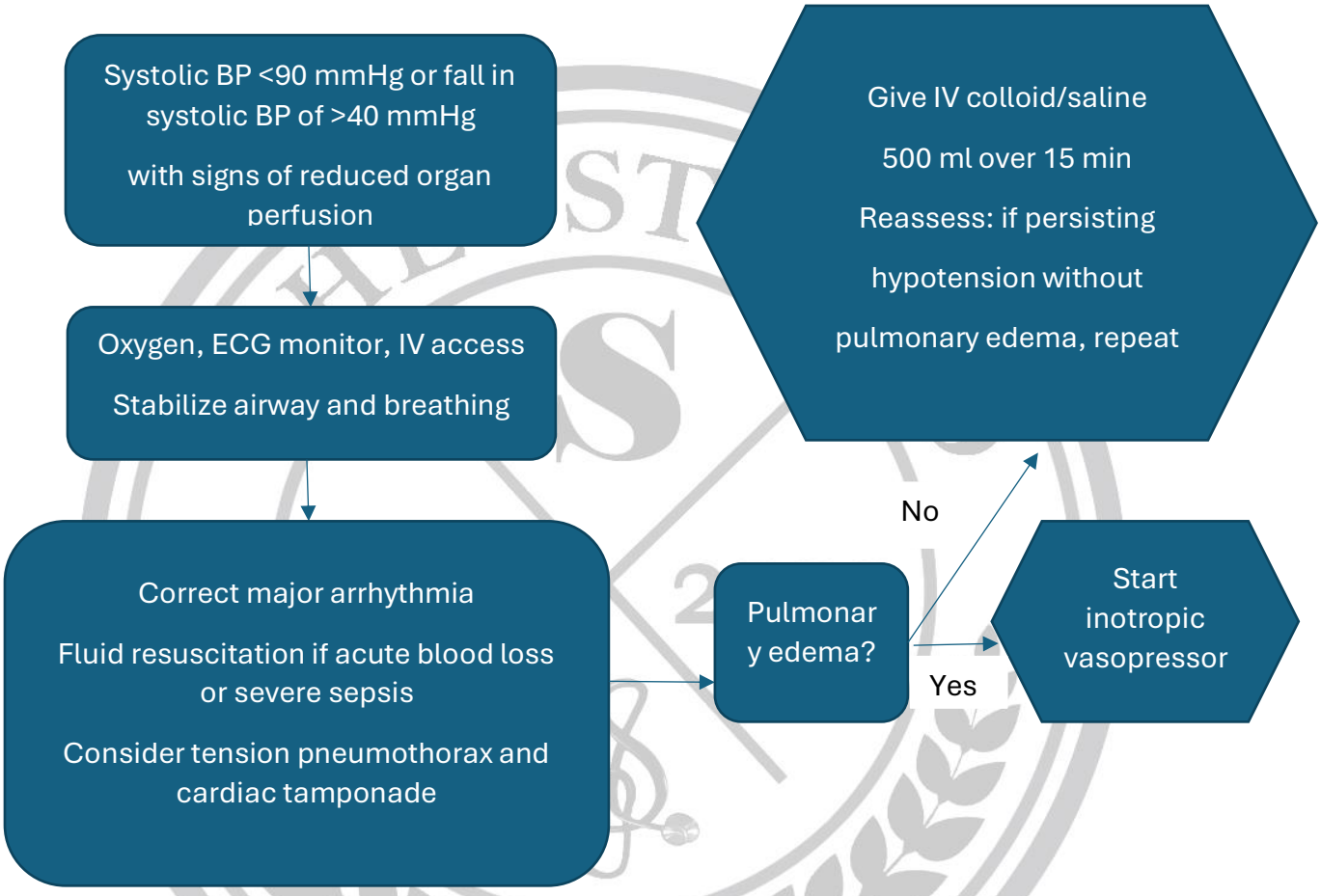



Figure 1.4. Acute management of hypotension.

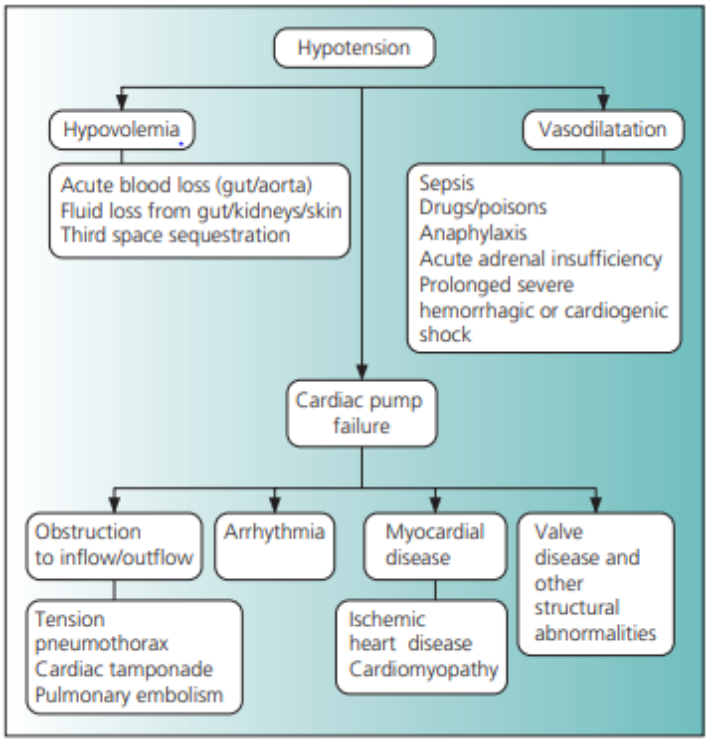



Figure 1.5. Causes of hypotension.

| Indications of ECHO |
|---|
| 1. Suspected cardiac tamponade<br>2. Hypotension and breathlessness following placement of central venous cannula or pacing lead, or in a patient with known cancer – Raised jugular venous pressure – Pulsus paradoxus >10 mmHg<br>3. Suspected acute major acute pulmonary embolism. Risk factors for venous thromboembolism – Raised jugular venous pressure<br>4. Hypotension with pulmonary edema<br>5. Unexplained severe hypotension |

Table 1.5. Indications for ECHO in hypotension

| Cause | IVC | LV diameter | LV diameter (Contraction) | RV diameter | RV diameter (Contraction) |
|---|---|---|---|---|---|
| Hypovolemia | flat | small | ↑ | small | ↑ |
| sepsis | flat | Normal/large | Normal/ ↓ | Normal/large | Normal/ ↓ |
| Ischemia | Normal or dilated | large | ↓ Regional and global | Normal | Normal |
| Acute PE | dilated | normal/small | Normal/ ↑ | Large | ↓ |
| Cardiac tamponade | dilated | normal | Normal/ ↑ | Normal | Diastolic free wall collapse |
| RV infarct | dilated | normal/large | Normal/↓ | Large | ↓ |

Table 1.6. ECHO findings in hypotension

| **Drugs** | **dose** | **Mechanism of action** |
|---|---|---|
| Dobutamine | 5–40 | Beta-1 inotropism and beta-2 vasodilatation |
| Dopamine | 5–10<br>10–40 | Beta-1 inotropism<br>Alfa-1 vasoconstriction |
| Epinephrine | 0.05<br>0.05–5 | Beta-1 inotropism and beta-2 vasodilatation<br>Beta-1 inotropism and alfa-1 vasoconstriction |
| Norepinephrine | 0.05–5 | Alfa-1 vasoconstriction and beta-1 inotropism |

Table 1.7. Inotropic drugs, dose and Mechanism of action in management of acute hypotension.

# Hypertension

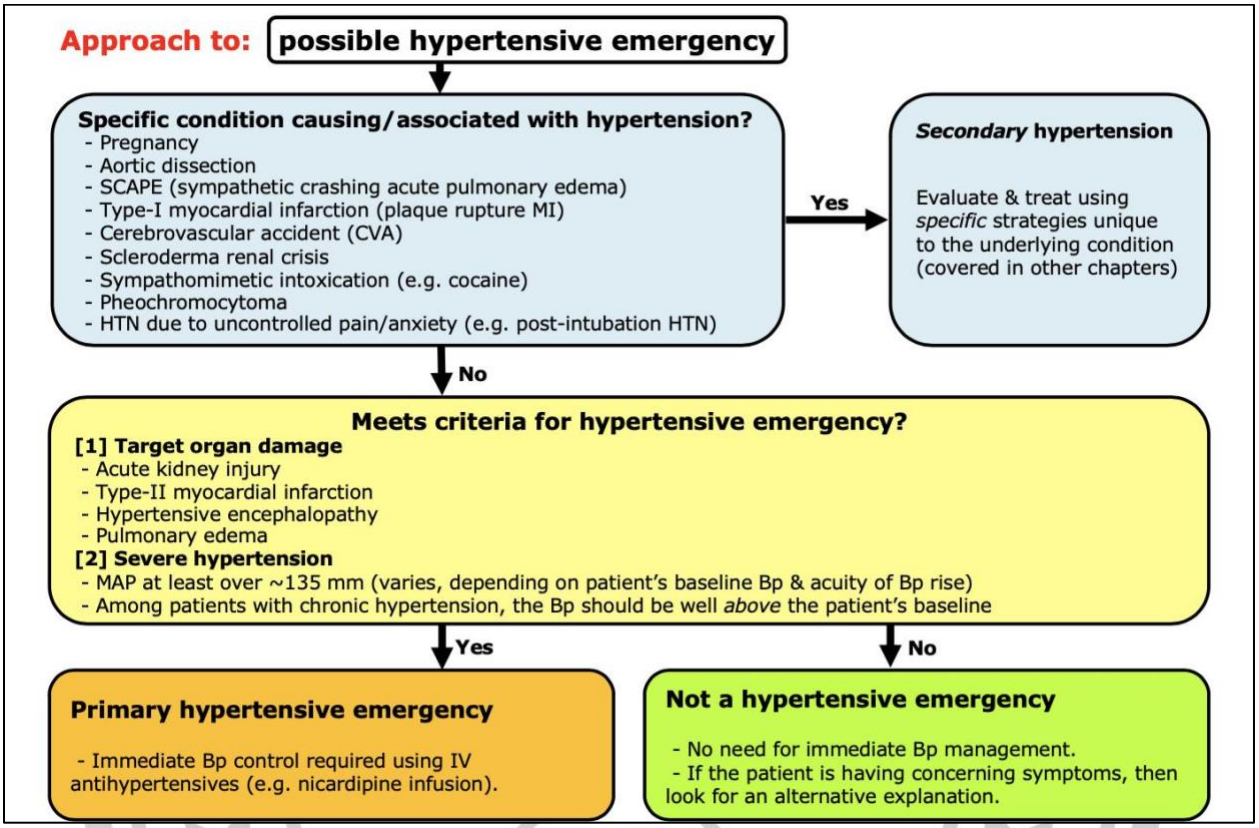



Figure 1.6. approach to management of Hypertension

**Focused History:**

- Previous treatment/compliance
- Previous investigations
- Known cardiac/renal disease
- Recent stroke or subarachnoid hemorrhage
- Features of hypertensive encephalopathy

**Examination:**

- Blood pressure in both arms
- Presence and symmetry of the major pulses, check for pulse delay
- Carotid, abdominal and femoral bruits
- Check for signs of heart failure
- Abdominal mass
- Fundi: retinal hemorrhages, exudates or papilledema (not due to other causes)

**Investigations**

- Blood glucose
- Sodium, potassium and creatinine (check daily)
- Full blood count
- Plasma renin/aldosterone (for later analysis)
- Urine stick test and microscopy
- Ultrasound of kidneys and urinary tract
- Urinary catecholamine excretion
- Urinary free cortisol excretion if suspected Cushing syndrome
- Chest X-ray
- ECG

| Clinical features | Initial drug therapy |
|---|---|
| **Pheochromocytoma suspected** | Labetalol 100–200 mg 12-hourly PO |
| **Renal artery stenosis suspected** | Amlodipine 5–10 mg daily PO *plus* Bisoprolol 2.5–5 mg daily PO |
| **Other patients** | Amlodipine 5–10 mg daily *plus* Bisoprolol 2.5–5 mg daily PO |
| **If there is pulmonary edema or other signs of fluid retention** | Add furosemide 20–40 mg daily PO to one of the above regimens |

Table 1.8. Drug management for hypertension in secondary causes.

## Acute Chest pain

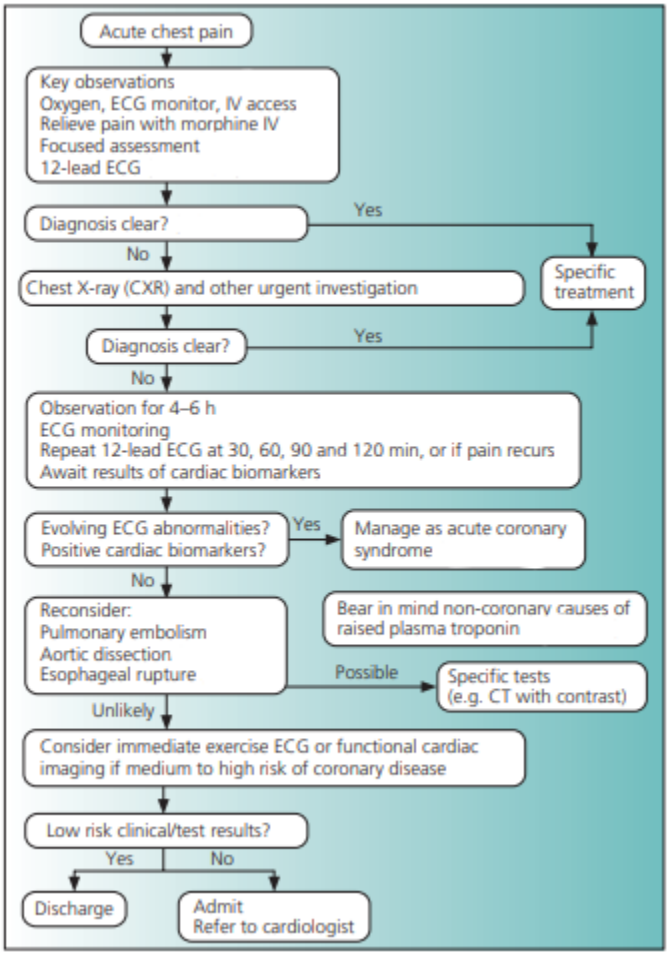



Figure 1.7. Acute chest pain management

| Causes of chest pain |
|---|
| 1. Acute coronary syndrome |
| 2. Pulmonary embolism |
| 3. GERD |
| 4. Pneumonia |
| 5. Pleurisy |
| 6. Costochondritis |
| 7. Panic Attack |

Table 1.9. common causes of Chest pain.

## Acute breathlessness

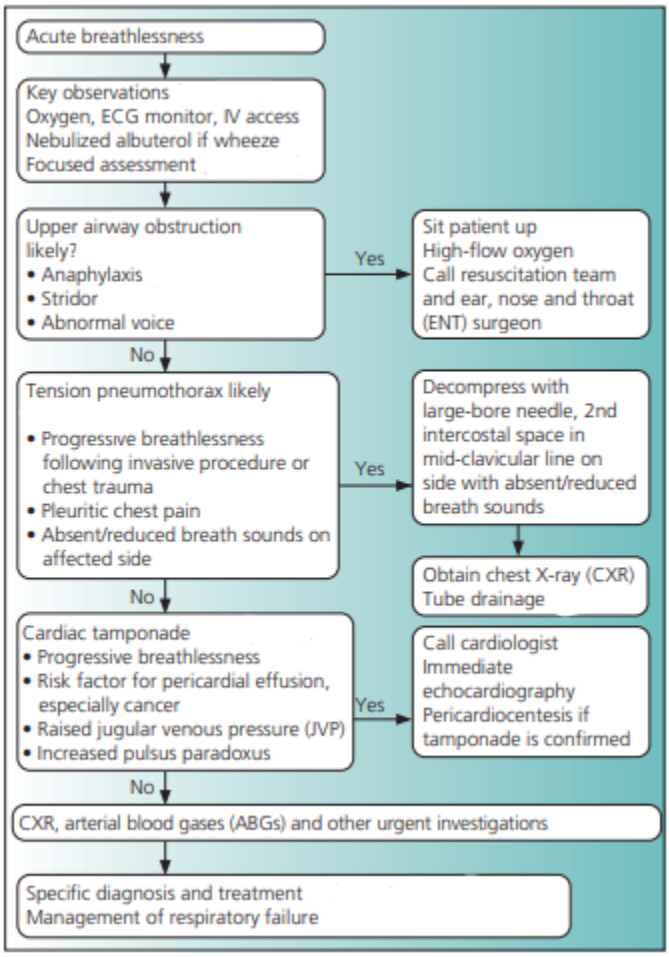



Figure 1.8. acute management of breathlessness.

**Focused History questions:**

1. Breathlessness: usual and recent change?
2. Wheeze: usual and recent change?
3. Sputum: usual volume/purulence and recent change
4. Chest pain: pleuritic/non-pleuritic
5. Effort tolerance: usual (e.g. distance walked on the flat; number of stairs climbed without stopping; ability to manage activities of daily living unaided) and recent change
6. Known respiratory disease? – Previous acute exacerbations requiring hospital admission/ventilation?

– Previous lung function tests and arterial blood gases (check notes): an FEV1 60–80% of predicted signifies mild COPD; 40–60%, moderate COPD; less than 40%, severe COPD
– Requirement for home nebulized bronchodilator and/or oxygen therapy?

7. Hisotry of cardiac disease?
8. Coronary disease (check notes for angiography report)?
9. Myocardial or valve disease (check notes for echo report)?
10. Risk factors for venous thromboembolism?

**Investigations:**

1. CXR
2. ABG
3. ECG
4. FBC, electrolytes and renal profile
5. Biomarkers: D dimers, troponin, and BNP

**Differential diagnosis:**

1. Acute respiratory distress syndrome
2. Acute exacerbation of underlying lung disease (COPD, Asthma, bronchiectasis and cystic fibrosis)
3. Pneumonia
4. Pneumothorax
5. Pulmonary embolism
6. Cardiac tamponade
7. Pleural effusion
8. Foreign body causing obstruction

| Cause | PaO2 | PaCO2 | PH |
|---|---|---|---|
| Acute Asthma | Normal/ ↓ | ↓ | ↑ |
| Acute COPD | ↓ | ↑ | Normal/ ↓ |
| Pulmonary embolism | Normal/ ↓ | ↓ | ↑ |
| Sepsis | Normal/ ↓ | ↓ | ↓ |
| Panic attack | ↑ /Normal | ↓ | ↑ |

Table 1.10. Arterial blood gas in different cases of acute shortness of breath.

# Acute Abdomen

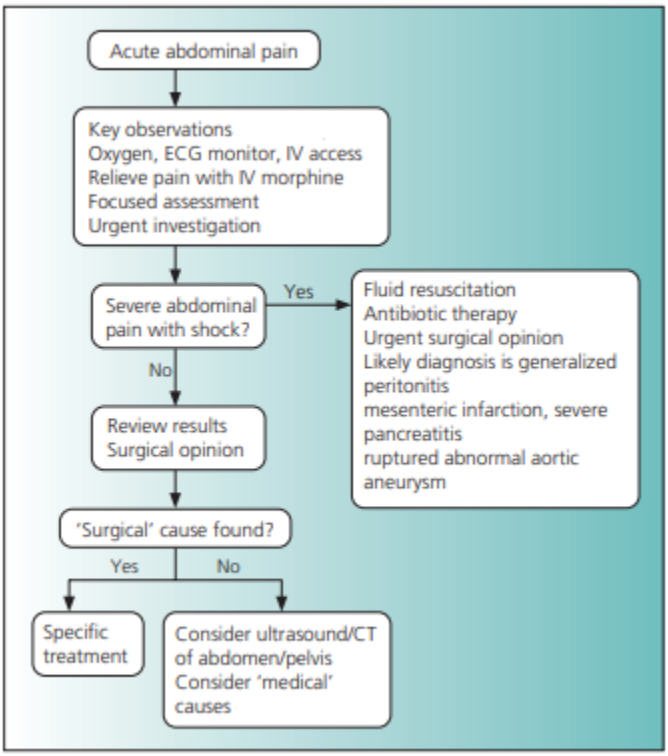



Figure 1.8. Acute management of Abdominal pain

**Focused History questions:**

1. Onset of pain- sudden or gradual?
2. Location? Has it moved since onset?
   - Visceral pain arising from the gut, biliary tract or pancreas is poorly localized.
   -Peritoneal pain (due to inflammation or infection) is well localized (unless there is generalized peritonitis).
3. Severity of pain?
4. Associated nausea/vomiting?
5. Previous similar episodes?
6. Abdominal surgeries?
7. Relevant Medical history?

**Examination:**

1. Abdominal distention?
2. Bowel sounds? Soft/absent?

3. Presence of abdominal scars- previous surgeries?
4. Palpable organs/mass?
5. Is tenderness localized or generalized?
6. Hernia? Any signs of infection/strangulation?
7. Femoral pulse?
8. Rectal examination

**Investigations:**

1. Full blood count
2. Clotting profile, group and screen.
3. Blood glucose
4. Serum electrolytes and renal profile
5. Liver function tests
6. Serum amylase (raised in pancreatitis, perforated ulcer, mesenteric ischemia and severe sepsis)
7. ABG
8. Urine and blood cultures
9. Urine dip
10. ECG
11. Imaging includes abdominal X ray, CT abdomen and pelvis to look for obstruction of large and/or small bowel; ischemic bowel (dilated and thickened loops of small bowel); cholangitis (gas in biliary tree); gall stones; urinary tract stones.

**Differential Diagnosis:**

1. <u>Right upper quadrant</u>
   • Pneumonia
   • Hepatic congestion due to congestive heart failure
   • Alcoholic hepatitis
   • Viral hepatitis
   • Acute gonococcal perihepatitis (Fitz–Hugh–Curtis syndrome)
2. <u>Epigastric</u>
   • Acute myocardial infarction
   • Pericarditis
   • Pancreatitis
3. <u>Left upper quadrant</u>
   • Pneumonia
4. <u>Central</u>
   • Mesenteric ischemia/infarction
   • Diabetic ketoacidosis
   • Aortic dissection
   • Acute intermittent porphyria

• Vaso-occlusive crisis of sickle cell disease
• Henoch–Schonlein purpura
• Typhoid/ Malaria
• Retroperitoneal hemorrhage (complicating heparin or warfarin therapy, or due to a bleeding disorder, leaking abdominal aortic aneurysm or vertebral fracture)

# Sepsis

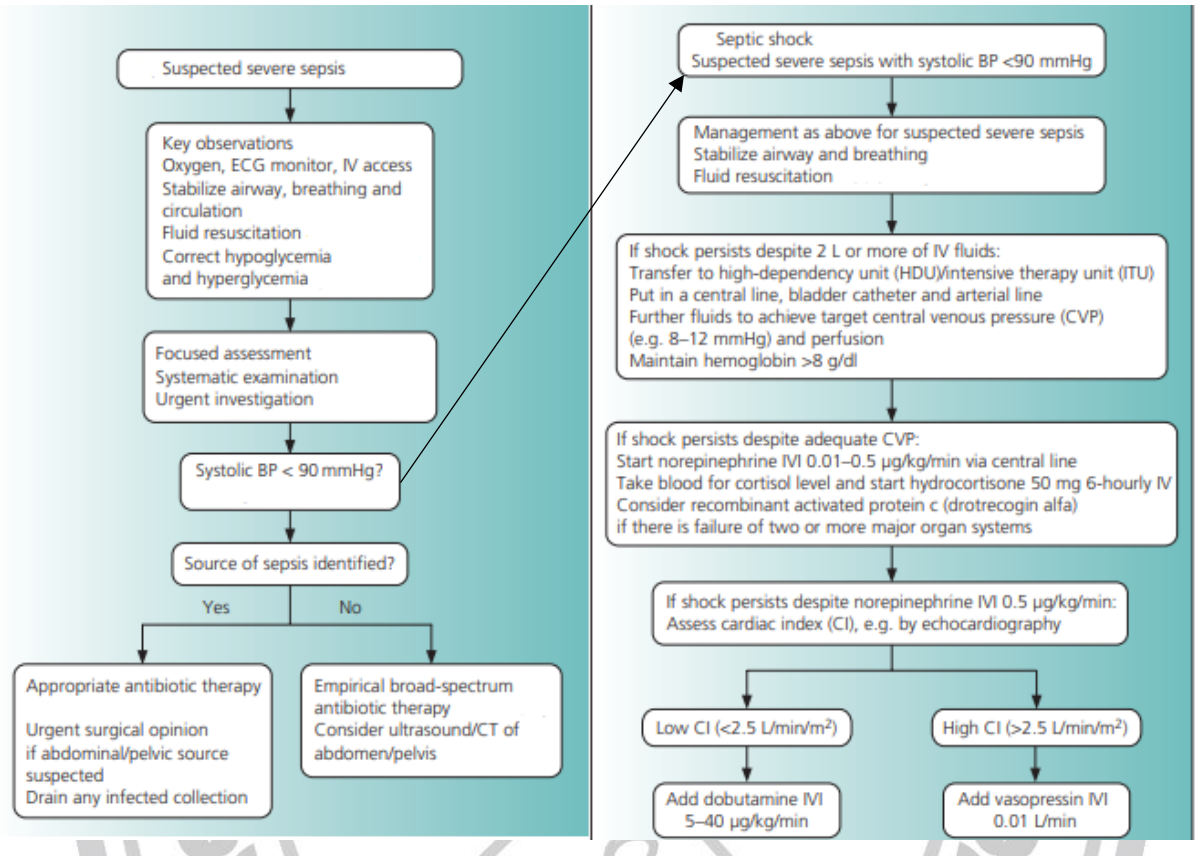



Figure 1.9. Acute management of sepsis.

**Fluid management in Sepsis:**

• 1 L of normal saline over 30 min
• 1 L of sodium lactate (Hartmann solution; Ringer lactate solution) over 30 min
• 1 L of sodium lactate over 30 min
• Start norepinephrine infusion if shock persists despite 2 L or more of IV fluid
• Give further maintenance/bolus fluid guided by clinical condition and hemodynamic monitoring (e.g. maintenance 200 ml/h Ringer lactate solution)

**Focused History Questions:**

• Age, sex, comorbidities, medications, hospital or community acquired
• Current major symptoms and their time course

• Risk factors for sepsis? Consider immunosuppressive therapy, AIDS, cancer, renal failure, liver failure, diabetes, malnutrition, splenectomy, IV drug use, prosthetic heart valve, other prosthetic material, peripheral IV cannula, central venous cannula, bladder catheter
• Recent culture results?
• Recent surgery or invasive procedures?
• Recent foreign travel?
• Contact with infectious disease?

**Investigations:**
• Full blood count
• Coagulation screen if there is purpura or jaundice, prolonged oozing from puncture sites, bleeding from surgical wounds or low platelet count
• Blood glucose
• Sodium, potassium and creatinine
• C-reactive protein
• Blood culture
• Urinalysis and urine microscopy/culture
• Chest X-ray
• Arterial blood gases and pH
• Additional investigation directed by the clinical picture (e.g. lumbar puncture, aspiration of pleural effusion or ascites, joint aspiration).

# Falls

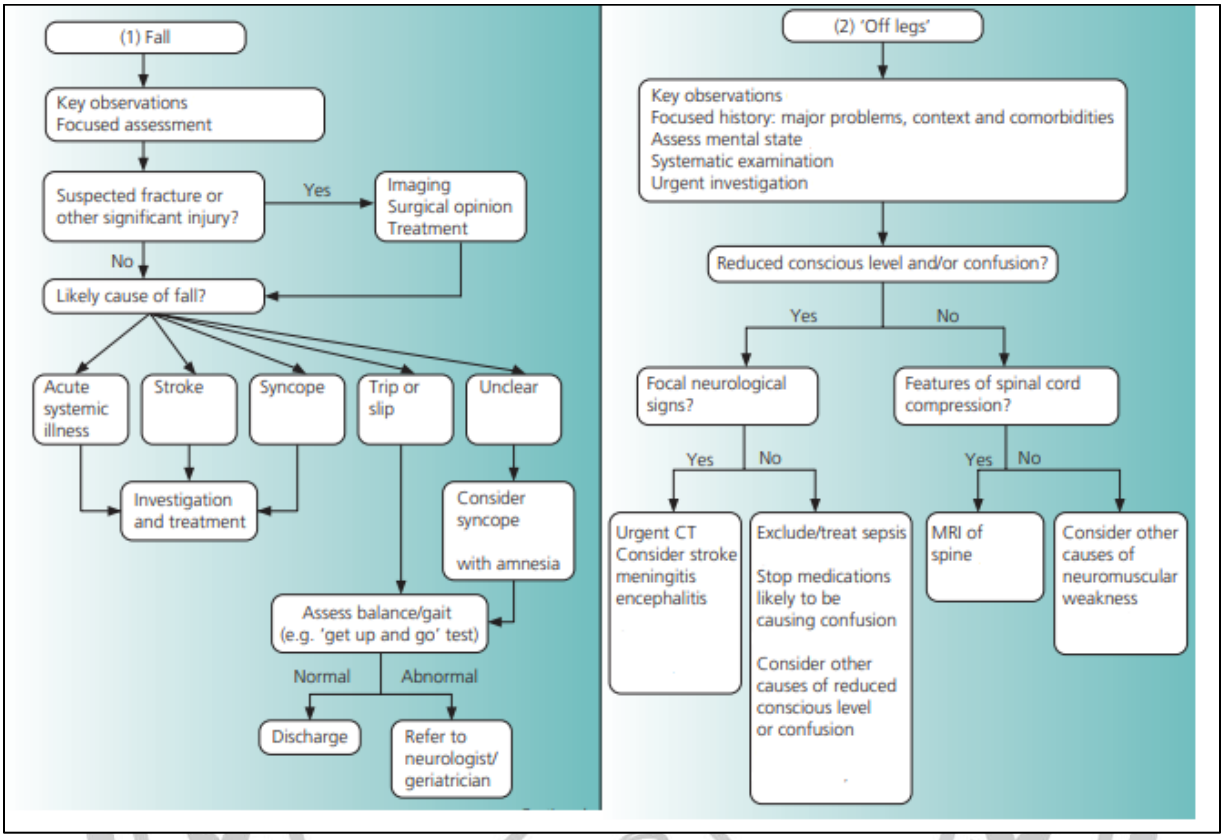



Figure 1.10. Acute management of falls.

**Focused history questions:**

• Circumstances of fall (e.g. place, time of day, witnessed)
• Symptoms before fall (e.g. presyncope/syncope, palpitations)
• Injuries sustained
• Contributory factors (e.g. dementia, previous stroke, parkinsonism, lower limb joint disorders, foot disorders)
• Previous falls (how many in past year?)
• Previous syncope
• Usual effort tolerance (e.g. able to climb stairs; able to walk on fl at; able to manage activities of daily living)
• Walking aids used
• If fall at home, are there environmental hazards (ask family/carer), e.g. loose rugs, poor lighting?
• Current medications (e.g. sedatives, hypnotics, antidepressants, antihypertensives, multiple drugs)
• Alcohol history
• Social history: living at home or residential/nursing home resident?

**Examination**

• Key observations and systematic examination
• Injuries sustained (check for head injury, fracture, joint dislocation, soft tissue bruising and laceration)
• Assess mental state (e.g. abbreviated mental status examination of the elderly
• If the patient does not have evidence of acute illness or injury, screen for neurological and musculoskeletal disorders with the ‘get up and go’ test: ask the patient to stand up from a chair without using the arms, walk several paces and return: can this be done without difficulty or unsteadiness?

## Acute headache

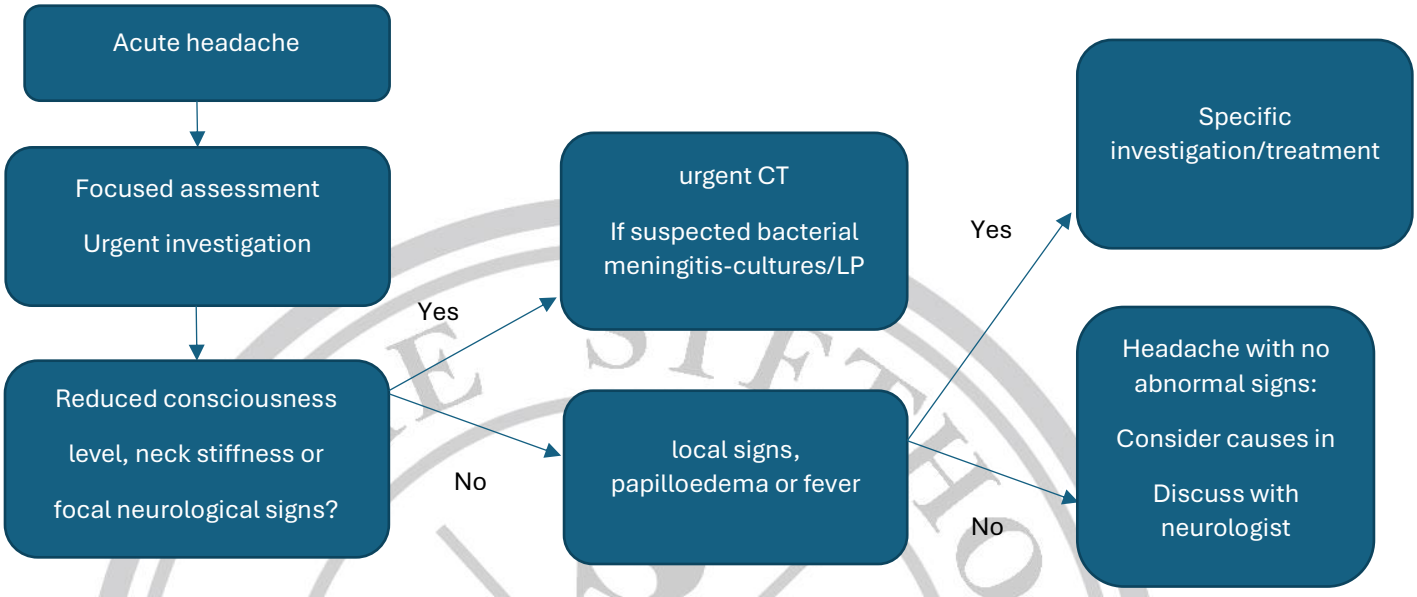



Figure 1.11. Acute management of headache.

**Focused History questions:**

1. Onset: Sudden or gradual?
2. How did the headache start? Duration?
3. Syncope at onset?
4. Severity? Worst headache ever?
5. Distribution (unilateral, diffuse, localized)
6. Associated systemic, neurological or visual symptoms (e.g. syncope/ presyncope, limb weakness, speech disturbance, blurring of vision, transient blindness, diplopia, scotomata, fortification spectra). Did these precede or follow the headache?

**Examination**

1. Observations: airway, respiratory rate, arterial oxygen saturation, heart rate, blood pressure, perfusion, consciousness level, temperature, blood glucose
2. Neck stiffness (in both flexion and extension)?
3. Focal neurological signs? -Horner syndrome (partial ptosis and constricted pupil: if present, consider carotid artery dissection
4. Visual acuity and fields. Fundi (papilledema or retinal hemorrhage?) • Signs of dental, ENT or ophthalmic disease? Temporal artery tenderness or loss of pulsation?

**Investigations:**

1. Full blood count
2. Coagulation screen if suspected intracranial hemorrhage
3. ESR and C-reactive protein
4. Blood glucose
5. Sodium, potassium and creatinine
6. Skull X-ray if suspected sinus infection
7. Blood culture if suspected bacterial meningitis
8. CT scan if suspected intracranial hemorrhage or meningitis/encephalitis with contraindication to LP
9. LP if suspected subarachnoid hemorrhage or meningitis/ encephalitis, if there are no contraindications

**Differential diagnosis:**

• Stroke

• Subarachnoid hemorrhage

• Chronic subdural hematoma

• Raised intracranial pressure

• Meningitis

• Encephalitis

• Cerebral malaria

• Hypertensive encephalopathy

**Acute headache and papilledema:**

• Meningitis

• Encephalitis

• Subarachnoid hemorrhage

• Systemic infectious disease (including malaria and typhoid in patients who have returned from abroad

• Local infection (e.g. sinusitis)

## Vomiting

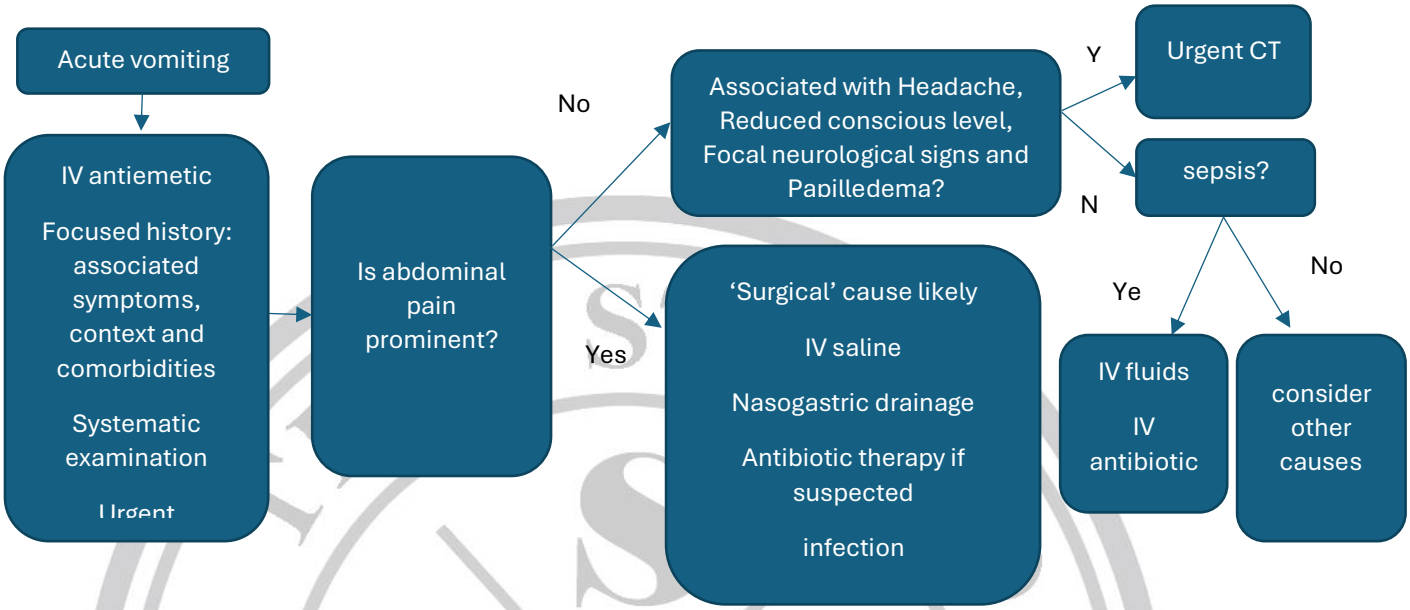



Figure 1.12. Acute management of vomiting.

**Causes:**

1. Cranial
   • Raised intracranial pressure
   • Intracranial hemorrhage (subarachnoid hemorrhage or intracerebral hemorrhage)
   • Cerebellar hemorrhage or infarction
   • Acute labyrinthitis • Acute migraine
2. Intrathoracic
   • Inferior myocardial infarction
3. Intra-abdominal
   • Acute gastroenteritis • Gastroparesis • Intestinal obstruction • Ileus • Biliary tract and pancreatic disorders • After abdominal and pelvic surgery
4. Systemic
   • Sepsis • Drugs and toxins (including anesthetic agents and chemotherapy) • Diabetic ketoacidosis • Adrenal insufficiency • Uremia and Hypercalcemia • Acute intermittent porphyria • Pregnancy

| Drug | Comment | Dose |
|---|---|---|
| **Cyclizine** | Antihistamine<br>Can be coadministered with opioid to prevent vomiting | 50 mg 8-hourly IV/IM/PO |
| **Prochlorperazine** | Phenothiazine<br>Can be coadministered with opioid to prevent vomiting | 12.5 mg IM followed if needed after 6 h by oral dose of 10 mg |
| **Domperidone** | Dopamine antagonist<br>Less likely to cause sedation and dystonic reaction than prochlorperazine and metoclopramide<br>Can be used to treat vomiting associated with chemotherapy | 12.5 mg IM |
| **Metoclopramide** | Has prokinetic as well as antiemetic effect and so may be more effective than phenothiazines for vomiting due to intra-abdominal disease<br>May cause acute dystonic reaction (treat with procyclidine 5 mg IV) | 5–10 mg 8-hourly IV/IM/PO |
| **Ondansetron** | $5HT_3$ antagonist<br>Used to treat vomiting associated with chemotherapy/ radiotherapy and postoperative vomiting | 4 mg IV |

Figure 1.13. Drugs used for management of vomiting.

Section 2

# Cardiovascular System

## Acute coronary system

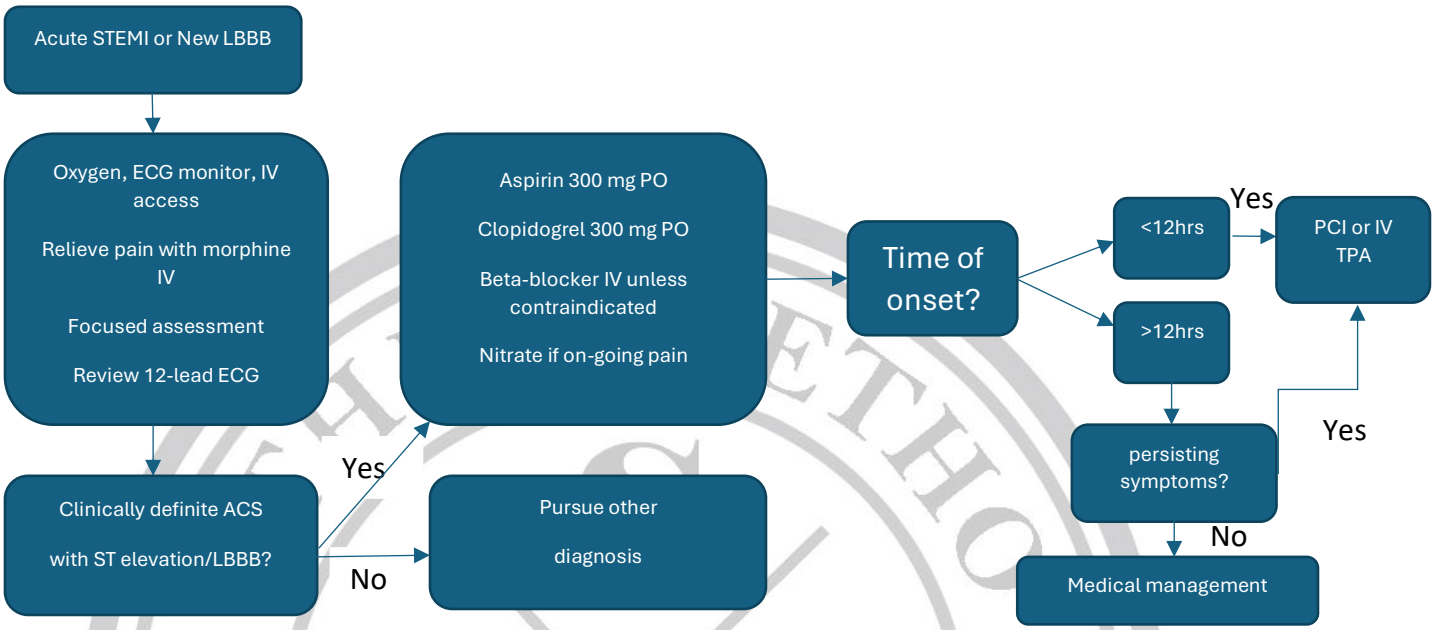



Figure 2.1. Acute management of STEMI

**Focused history questions:**

1. Time since onset of symptoms
2. Are symptoms consistent with myocardial ischemia/infarction?
3. Does ECG show persistent ST segment elevation consistent with acute coronary syndrome or left bundle branch block not known to be old?
4. Are there signs of heart failure or shock?
5. Which reperfusion strategy: primary percutaneous coronary intervention or fibrinolysis

**Investigations:**

1. ECG (repeat 60 and 90 min after fibrinolysis to assess reperfusion; a reduction in ST elevation by more than 70% in the leads with maximal elevation is associated with the most favorable outcomes)
2. Chest X-ray and Troponins
3. Echocardiography if: – There is cardiogenic shock – The diagnosis is in doubt and chest pain still present (to establish whether there is a left ventricular regional wall motion abnormality consistent with acute coronary syndrome)
4. Blood glucose (management of diabetes in acute coronary syndrome)
5. Sodium, potassium and creatinine

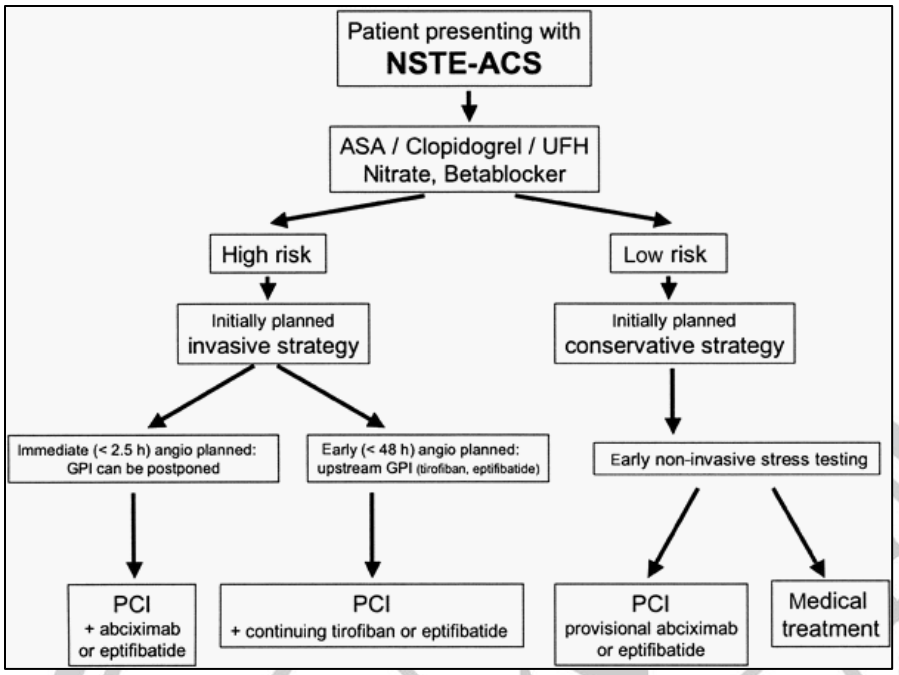



Figure 2.2 Acute management of NSTEMI

**Drug therapy:**

| Drug | Dose |
|---|---|
| Morphine | 5–10 mg IV, with additional doses of 2.5 mg IV every 5 to 10 min until pain-free. |
| Metoclopramide | 10 mg IV if needed for nausea/vomiting |
| Aspirin | 300 mg PO stat followed by 75 mg PO daily |
| Clopidogrel | 300 mg PO stat followed by 75 mg PO daily for 1 month |
| ACEI | Start unless contraindicated by low BP |
| Nitrate | Indicated in case of on-going chest pain/pulmonary edema<br>Can be given IV or Buccal 2–5 mg every 8 hourly |
| Beta-blocker | If not contraindicated by hypotension, heart failure, asthma and bradycardia. Start on metoprolol 5 mg IV over 5 min, then repeat as needed up to 15 mg with target HR 60 bpm, followed by oral switch |

Table 2.1. Initial drug therapy in ST elevation Myocardial infarction

**Absolute contraindications**

- Prior intracranial hemorrhage
- Known structural cerebrovascular lesion (e.g. arteriovenous malformation)
- Known malignant intracranial neoplasm (primary or metastatic)
- Ischemic stroke within 3 months (except acute ischemic stroke within 3 h)
- Suspected aortic dissection
- Active bleeding or bleeding tendency (excluding menses)
- Significant closed head trauma or facial trauma within 3 months

**Relative contraindications and cautions**

- History of chronic, severe, poorly controlled hypertension
- Severe uncontrolled hypertension on presentation (systolic BP >180 mmHg or diastolic BP >110 mmHg) (could be an absolute contraindication in low risk patient)
- History of prior ischemic stroke >3 months, dementia or known intracranial pathology not covered in absolute contraindications
- Traumatic or prolonged (>10 min) cardiopulmonary resuscitation or major surgery within 3 weeks
- Recent (within 2–4 weeks) internal bleeding
- Non-compressible vascular punctures
- For streptokinase/anistreplase: prior exposure (>5 days ago) or prior allergic reaction to these agents
- Pregnancy
- Active peptic ulcer
- Current use of anticoagulants: the higher the INR, the higher the risk of bleeding

Figure 2.2. Contraindications to fibrinolytic treatment.

## Acute pulmonary oedema

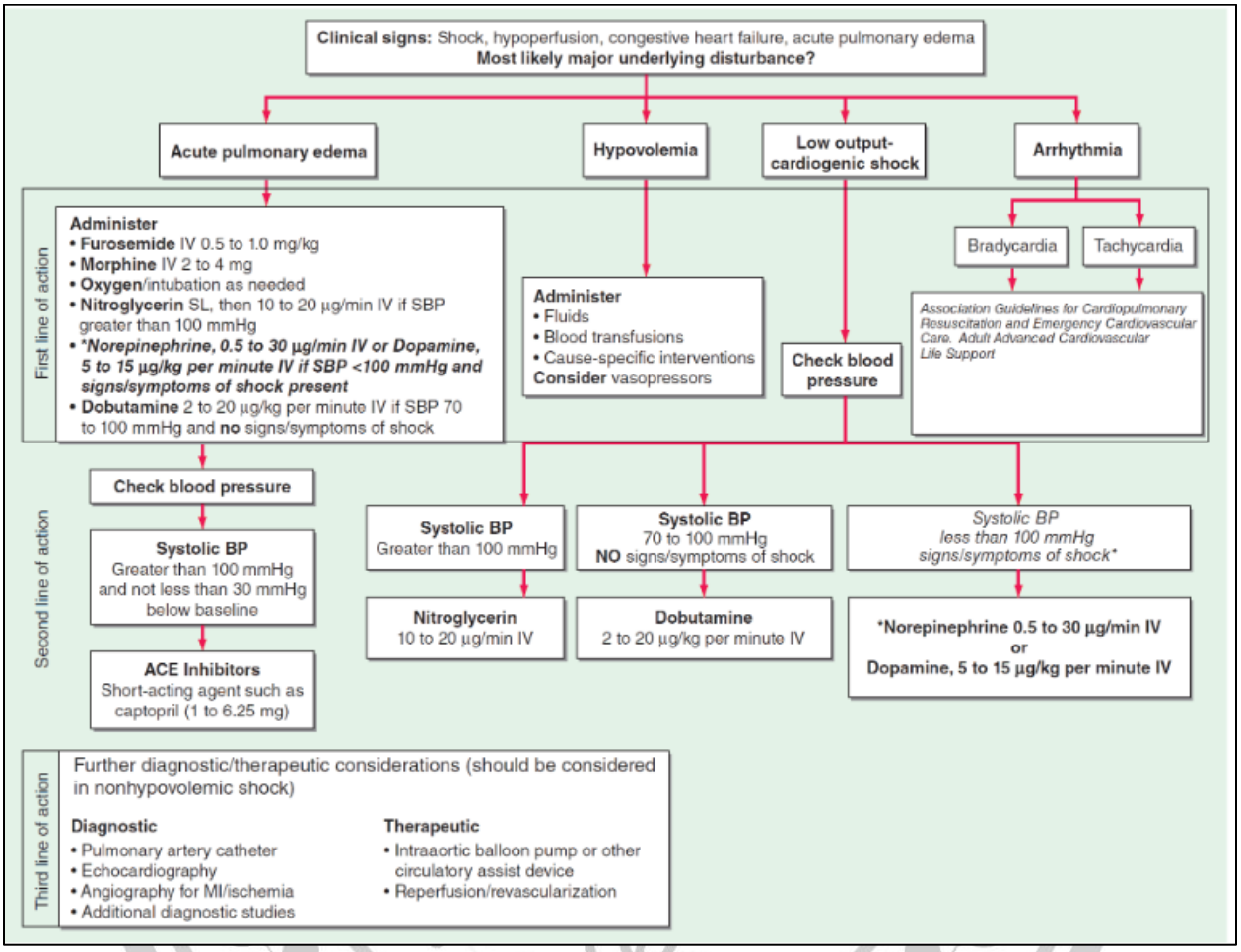



Figure 2.3. acute management of pulmonary edema

**Investigations:**

1. ECG (Arrhythmia? acute myocardial infarction or ischemia? other cardiac diseases?)
2. Chest X-ray
3. Arterial blood gases, Blood glucose, full blood count, renal profile, electrolytes Erythrocyte sedimentation rate (ESR) or C-reactive protein
4. Transthoracic echocardiography, acute valve lesion or ventricular septal rupture? Differentiate between cardiogenic/non-cardiogenic pulmonary edema
5. Cardiac biomarkers: troponin and BNP

| Causes of pulmonary oedema | |
|---|---|
| Raised pressure pulmonary oedema | Negative pressure oedema |
| • Cardiac causes:<br>Acute myocardial infarction, Severe aortic stenosis, myocarditis, aortic dissection, infective endocarditis, chest trauma, Acute mitral regurgitation (infective endocarditis, ruptured chordae or papillary muscle, chest trauma) | • Early postoperative period |
| • Renal causes:<br>Acute or chronic renal failure and Renal artery stenosis | • Forced inspiration in the presence of upper airway obstruction (e.g. laryngospasm after extubating) |
| • Central nervous system:<br>Subarachnoid hemorrhage | • After laryngospasm relief, patients develop features of pulmonary edema |
| • Iatrogenic | • Cardiogenic pulmonary edema should be excluded y |

Table 2.2. Causes of pulmonary oedema.

| Mode of ventilation | Indications | Contraindications | Disadvantages and complications |
|---|---|---|---|
| **Non-invasive ventilatory support with continuous positive airways pressure (CPAP)** | Oxygenation failure: oxygen saturation <92% despite $FiO_2$ > 40%<br>Ventilatory failure: mild to moderate respiratory acidosis, arterial pH 7.25–7.35 | Recent facial, upper airway or upper gastrointestinal tract surgery<br>Vomiting or bowel obstruction<br>Copious secretions<br>Hemodynamic instability<br>Impaired consciousness, confusion or agitation | Discomfort from tightly fitting facemask<br>Discourages coughing and clearing of secretions |
| **Endotracheal intubation and mechanical ventilation** | Upper airway obstruction<br>Impending respiratory arrest<br>Airway at risk because of neurological disease or coma (GCS 8 or lower)<br>Oxygenation failure: $PaO_2$ <7.5–8 kPa despite supplemental oxygen/NIV<br>Ventilatory failure: moderate to severe respiratory acidosis, arterial pH < 7.25 | Severely impaired functional capacity and/or severe comorbidity<br>Cardiac disorder not remediable<br>Patient has expressed wish not to be ventilated | Adverse hemodynamic effects<br>Pharyngeal, laryngeal and tracheal injury<br>Pneumonia<br>Ventilator-induced lung injury (e.g. pneumothorax)<br>Complications of sedation and neuromuscular blockade |

Figure 2.4. Ventilatory support in acute pulmonary oedema

# Acute pericarditis

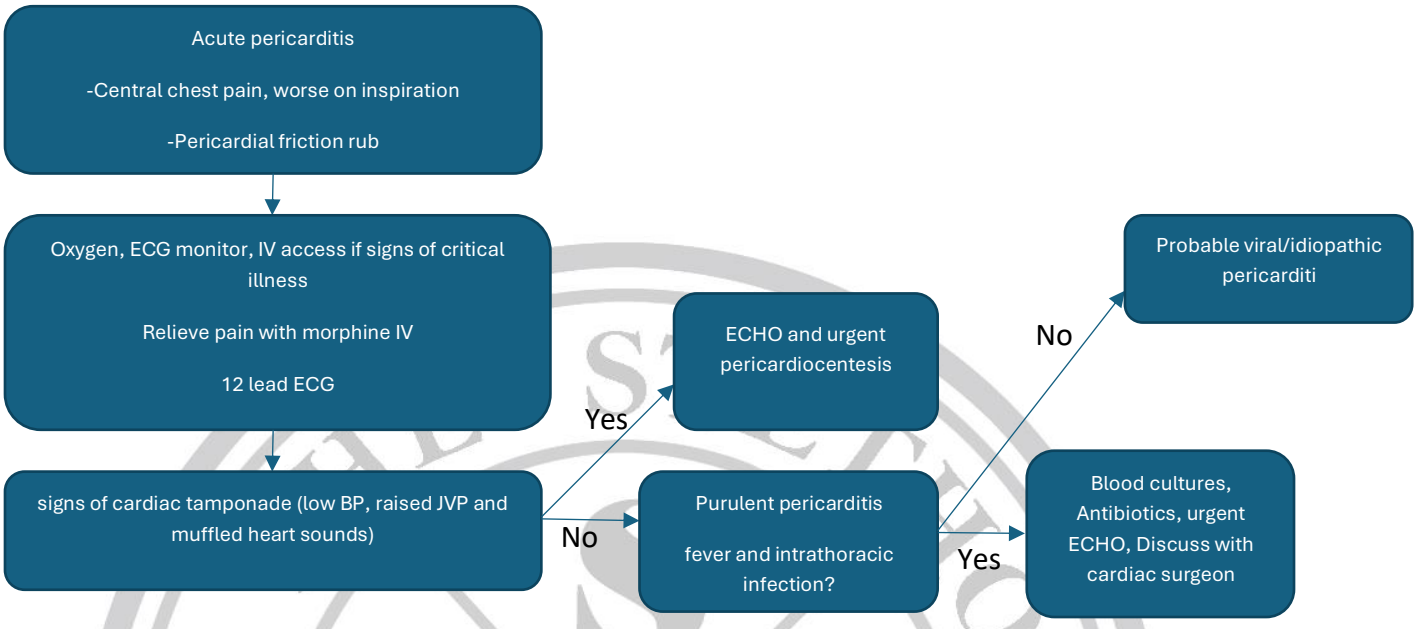



Figure 2.5. Acute management of pulmonary oedema

## Investigations

1. XR chest
2. FBC, renal profile, ESR and electrolytes
3. Troponins
4. ECHO
5. Blood Culture and virology
6. ANA

## Causes of acute pericarditis

•Idiopathic (85–90%)

• Infectious diseases (viral, bacterial, fungal, tuberculous) (7%)

• Acute myocardial infarction (pericarditis occurs in 5–10% of patients with myocardial infarction) • Malignancy (7%)

• Rheumatic diseases, e.g. systemic lupus erythematosus (3–5%)

• Aortic dissection (<1%)

• Advanced renal failure (pericarditis occurs in 5% of patients before renal replacement therapy)

• Pericardial surgery or trauma (Dressler/post cardiotomy syndrome) (<1%)

• Adverse drug reaction (<1%)

**Dressler syndrome:**

• Occurs 2–4 weeks after open heart surgery

• Recognized but rare complication of acute myocardial infarction

• Acute self-limiting illness with fever, pericarditis and pleuritis

• ECG usually shows only non-specific ST/T abnormalities

• Chest X-ray shows: – Large cardiac silhouette (due to pericardial effusion) – Pleural effusions – Transient pulmonary infiltrates (occasionally seen)

• White cells count and ESR raised (often >70 mm/h)

• Treat with NSAID or colchicine

## Deep vein thrombosis

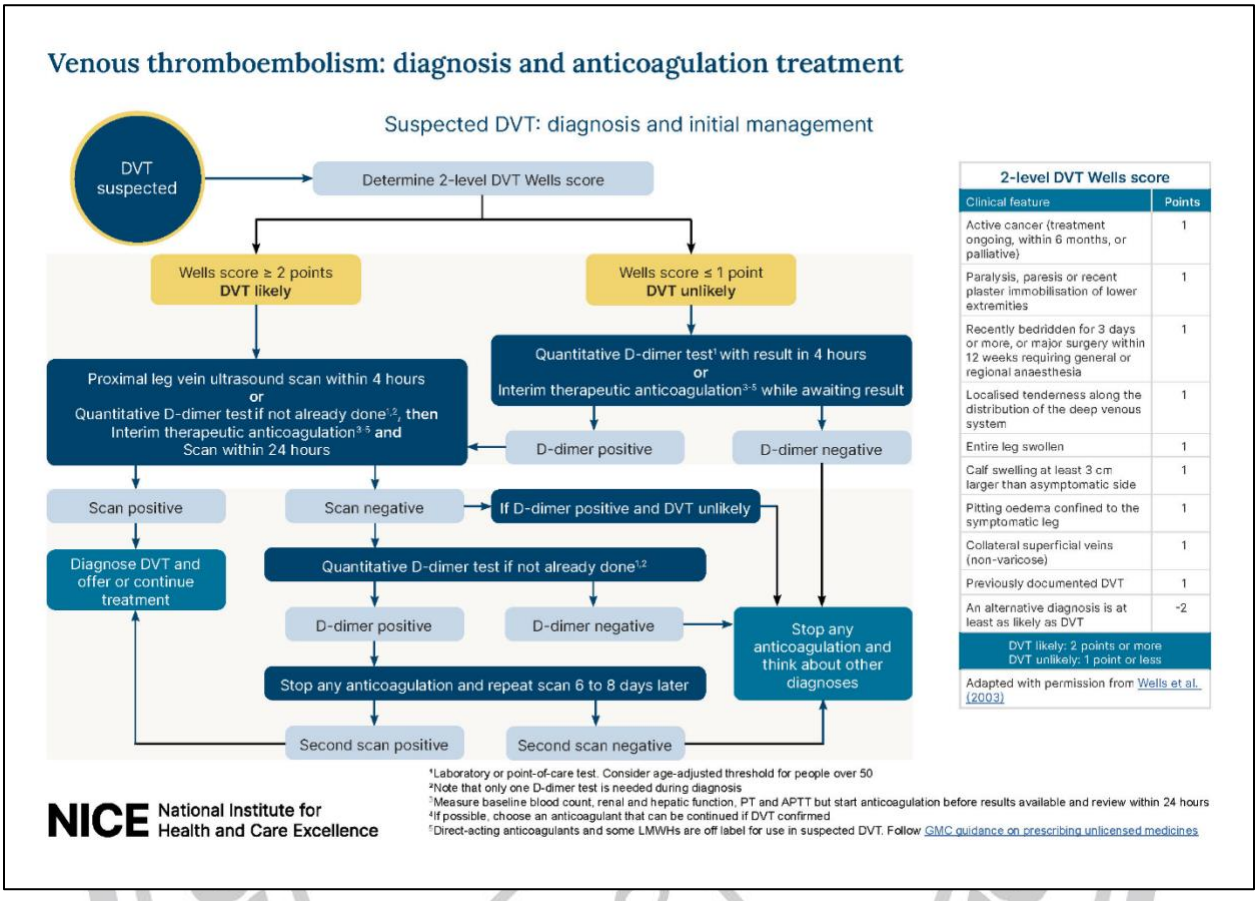



Figure 2.6. NICE guidelines to management of suspected DVT

| Causes of leg swelling |
|---|
| 1. **lymphatic /Venous**<br>• Deep vein thrombosis • Superficial thrombophlebitis • Inferior vena cava (IVC) obstruction (e.g. by tumor) • Varicose veins with chronic venous hypertension • post-phlebitis syndrome • Congenital lymphedema |
| 2. **Musculoskeletal**<br>• Calf hematoma • Ruptured Baker (popliteal) cyst (which may complicate rheumatoid arthritis or osteoarthritis of the knee) • Muscle tear |
| 3. **Skin** • Cellulitis |
| 4. **Systemic**<br>• Congestive heart failure • Liver failure • Renal failure • Nephrotic syndrome • Hypoalbuminemia • Chronic respiratory failure • Pregnancy • Idiopathic edema |

Table 2.3. Causes of leg swelling.

## Cardiac Tamponade

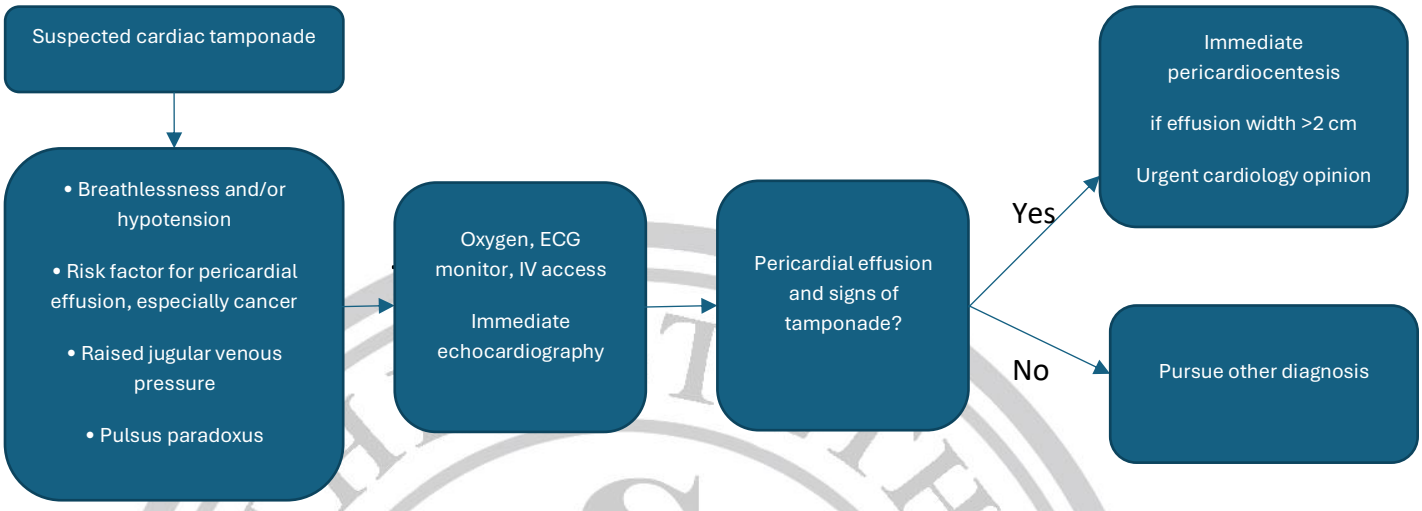



Figure 2.7. acute management of cardiac tamponade

| **Causes:** |
|---|
| 1. Bleeding in pericardial space<br>• Penetrating and blunt chest trauma<br>• Bleeding from cardiac chamber or coronary artery caused by perforation/laceration as a complication of cardiac catheterization, percutaneous coronary intervention, pacemaker insertion, pericardiocentesis or central venous cannulation<br>• Bleeding after cardiac surgery<br>• Cardiac rupture after myocardial infarction<br>• Aortic dissection with retrograde extension into pericardial space |
| 2. Serous pericardial effusion<br>• Metastasis to pericardium (carcinoma of breast/bronchus/lymphoma)<br>• Pericarditis complicating connective tissue diseases (e.g. systemic lupus erythematosus, rheumatoid arthritis)<br>• Post cardiotomy syndrome pericarditis<br>• Tuberculous and viral pericarditis • Uremic pericarditis • Idiopathic pericarditis |
| 3. Purulent pericarditis<br>• Pyogenic bacterial infection |

Table 2.4. Causes of pericardial effusion.

## Cardiogenic shock

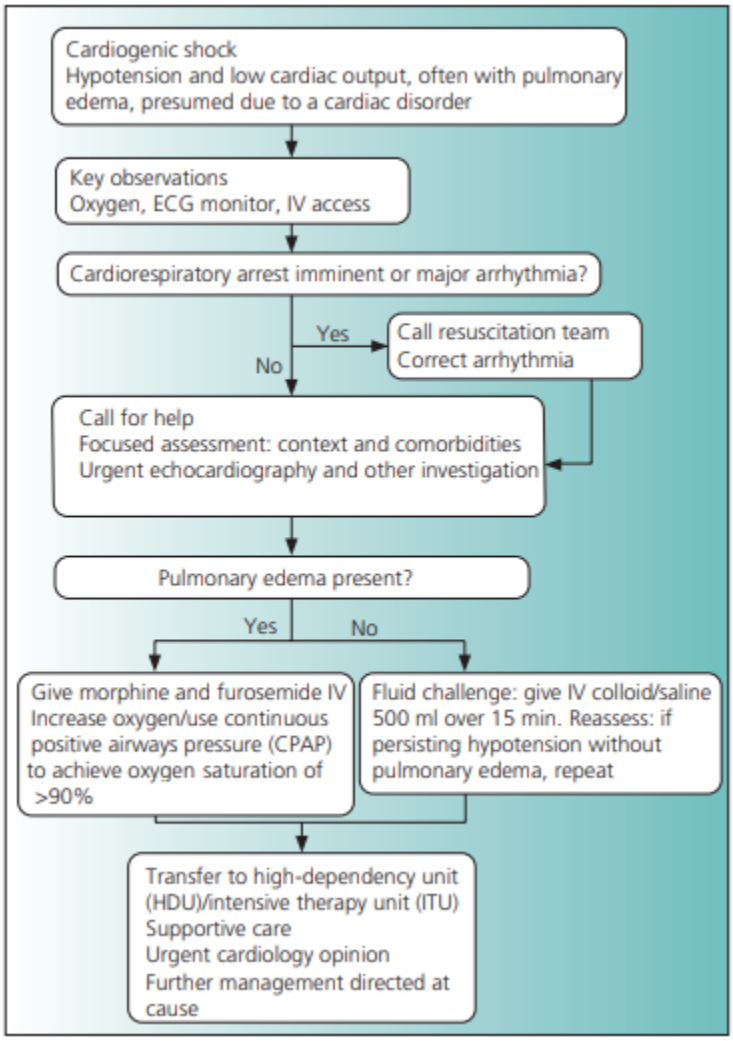



Figure 2.8. Acute management of Cardiogenic shock

**Investigations:**

• ECG • Chest X-ray • Echocardiography • Arterial blood gases and pH • Blood glucose • Sodium, potassium and creatinine • Cardiac biomarkers (for later analysis) • Full blood count • Blood culture.

| Supportive Treatment |
| --- |
| 1. Airway and breathing:<br>• Maintain airway<br>• Treat pulmonary edema<br>• Increase inspired oxygen concentration to achieve arterial oxygen saturation >90%<br>• Ventilatory support (CPAP) or endotracheal intubation and mechanical ventilation if appropriate |
| 2. Circulation<br>• Central line to monitor CVP and for medication administration<br>• Inotropic vasopressor agent if pulmonary edema<br>• ECG or echocardiography |
| 3. Renal function<br>• Input and out monitor<br>• Daily function tests<br>• Catheterize |
| 4. Blood glucose<br>Insulin infusion to control blood glucose >11 mmol/L |
| 5. Symptom control<br>• Morphine to relieve distress/ breathlessness<br>• palliative care review<br>• Consider syringe driver |

Table 2.5. Supportive care in cardiogenic shock

## Pulmonary embolism

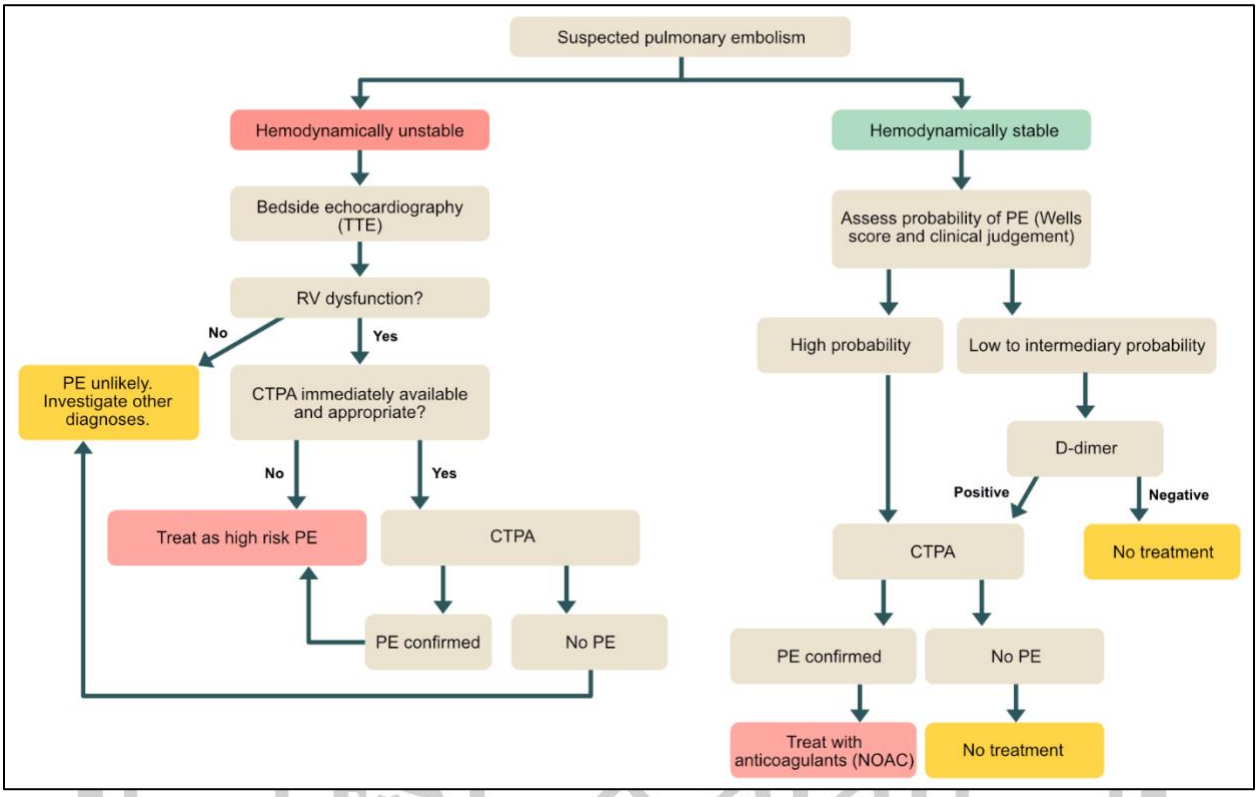



Figure 2.9. Acute management of pulmonary embolism

| ECHO suggestive of Massive Pulmonary embolus |
|---|
| • Dilated right heart (Atrium and ventricle)<br>• Moderate or severe tricuspid regurgitation<br>• Dilated inferior vena cava with little to no change with breathing<br>• Increased pulmonary artery pressure<br>• Absence of significant abnormalities of left heart to cause pulmonary hypertension<br>• Free floating thrombus<br>• Time to pulmonary artery velocity peak is <60 milliseconds<br>• Hypokinetic right ventricle + systolic septal flattening |

Section 3

# Respiratory System

## Acute asthma

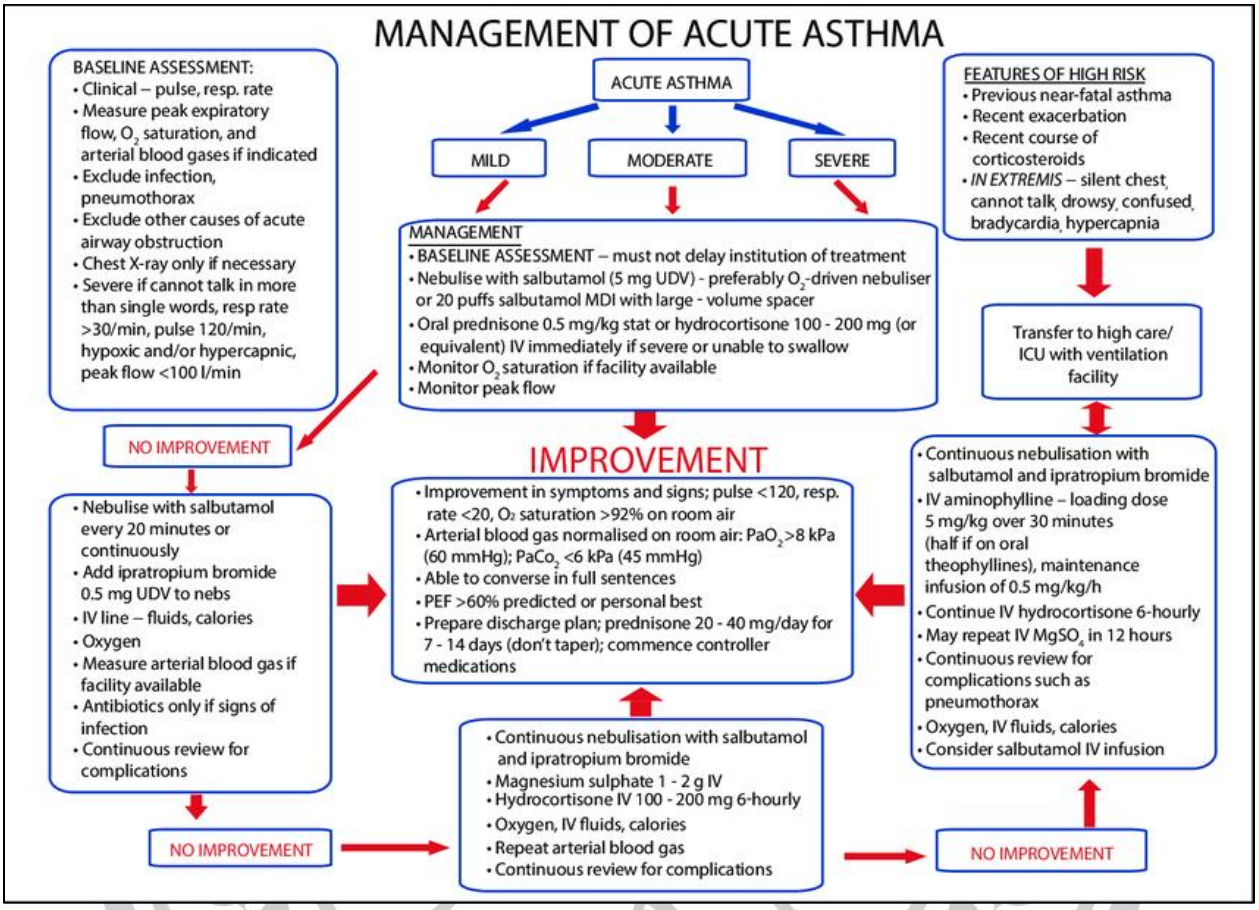



Figure 3.1. acute management of Asthma

| MODERATE ASTHMA | LIFE-THREATENING ASTHMA |
|---|---|
| ▪ increasing symptoms<br>▪ PEF >50-75% best or predicted<br>▪ no features of acute severe asthma | In a patient with severe asthma any one of:<br>• PEF <33% best or predicted<br>• $SpO_2$ <92%<br>• $PaO_2$ <8 kPa<br>• normal $PaCO_2$ (4.6-6.0 kPa)<br>• silent chest<br>• cyanosis<br>• poor respiratory effort<br>• arrhythmia<br>• exhaustion, altered conscious level<br>• hypotension |
| **ACUTE SEVERE ASTHMA** | **NEAR-FATAL ASTHMA** |
| Any one of:<br>• PEF 33-50% best or predicted<br>• respiratory rate ≥25/min<br>• heart rate ≥110/min<br>• inability to complete sentences in one breath | Raised $PaCO_2$ and/or requiring mechanical ventilation with raised inflation pressures |

Figure 3.2. Severity of asthma

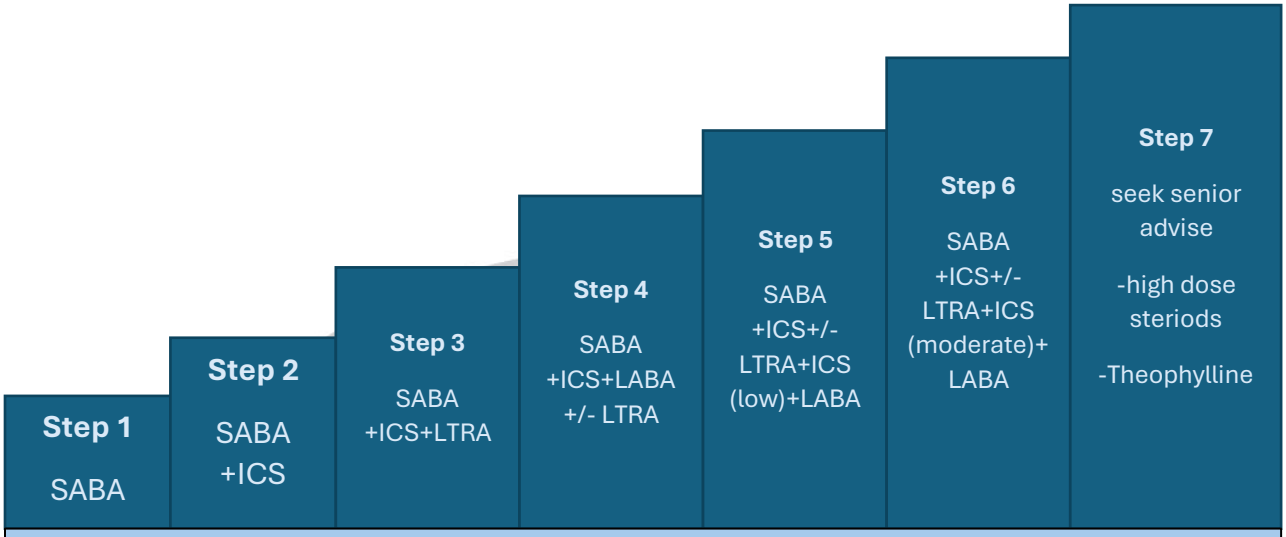



Figure 3.3. Stepwise guideline for Management of Asthma.

**Checklist prior to discharge:**

• Stable up to 24 hours

• Inhaler technique checked

• Peak flow >75% of predicted

• Oral and inhaled medication prescribed

• General practitioner to follow-up within two working days

• Follow-up in asthma clinic within 4 weeks

## Acute COPD

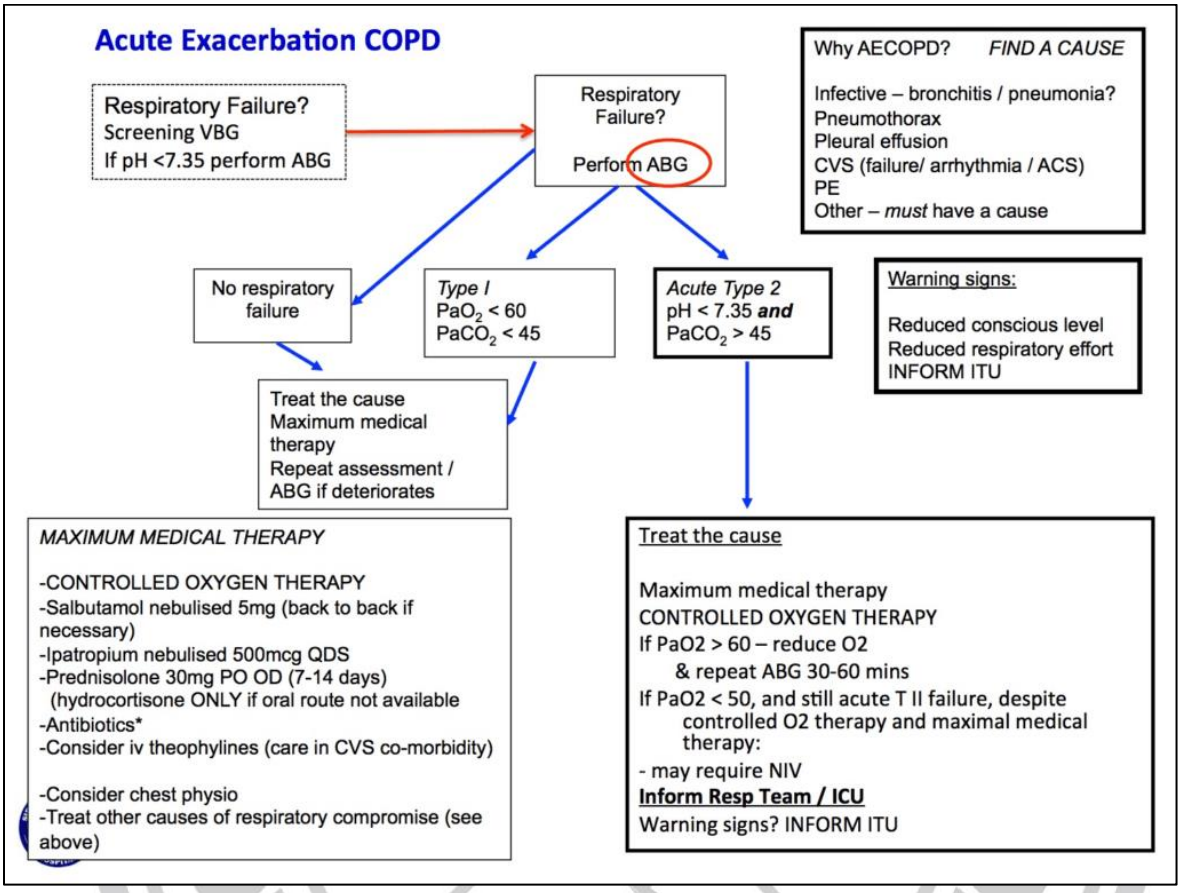



Figure 3.4. Acute management of COPD.

**Checklist prior to discharge:**

- Clinically stable for more than 24 hours
- Bronchodilator therapy not required frequently
- Spirometry checked/arranged
- Inhaler technique checked
- Able to walk unaided for a short distance
- Social support organized at home
- Advice on smoking cessation provided
- General practitioner to follow-up within 1 week
- Follow-up ap

# Pneumothorax

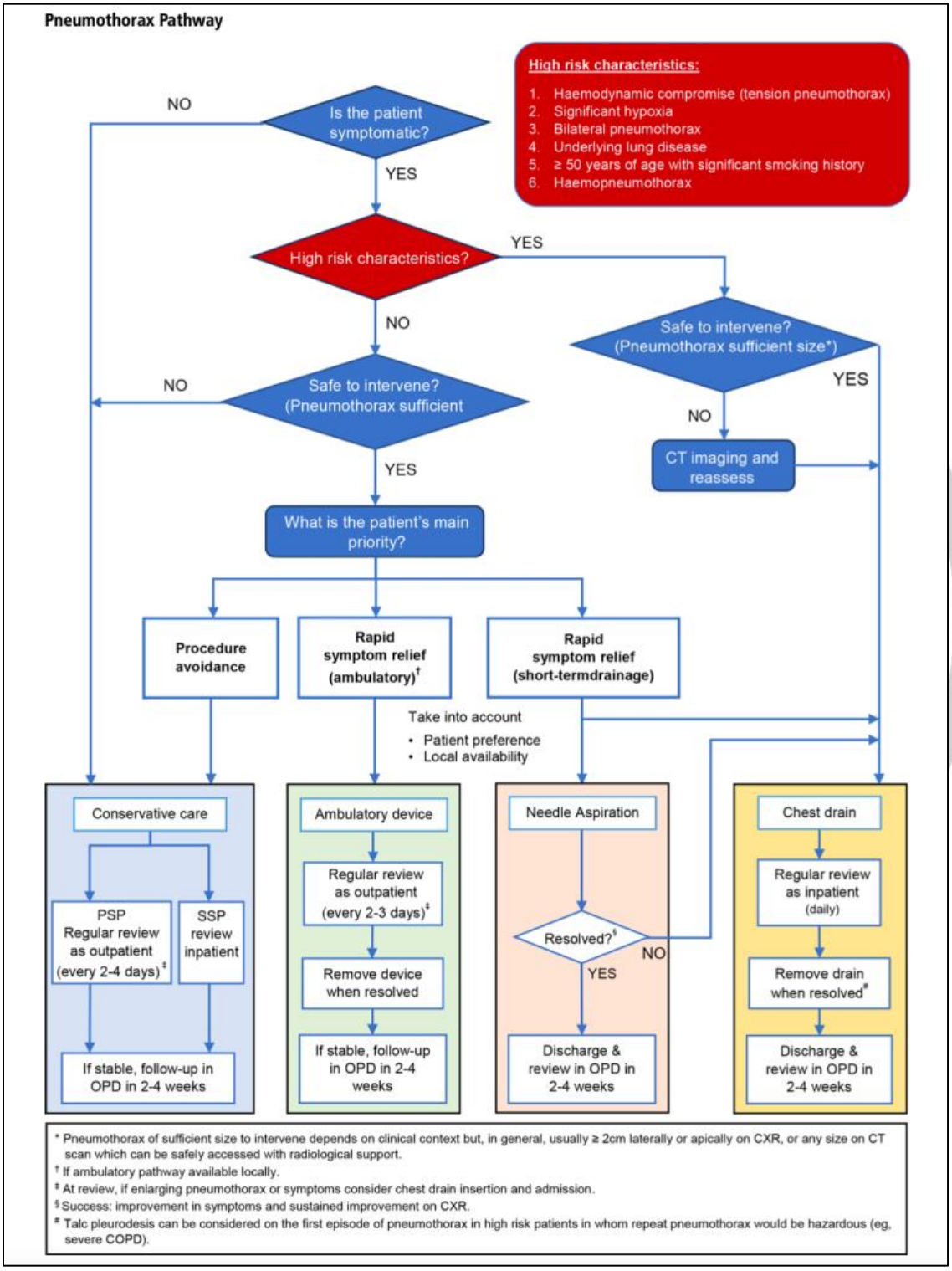



Figure 3.5. British Thoracic society guideline to management of pneumothorax.

**Community Acquired Pneumonia:**

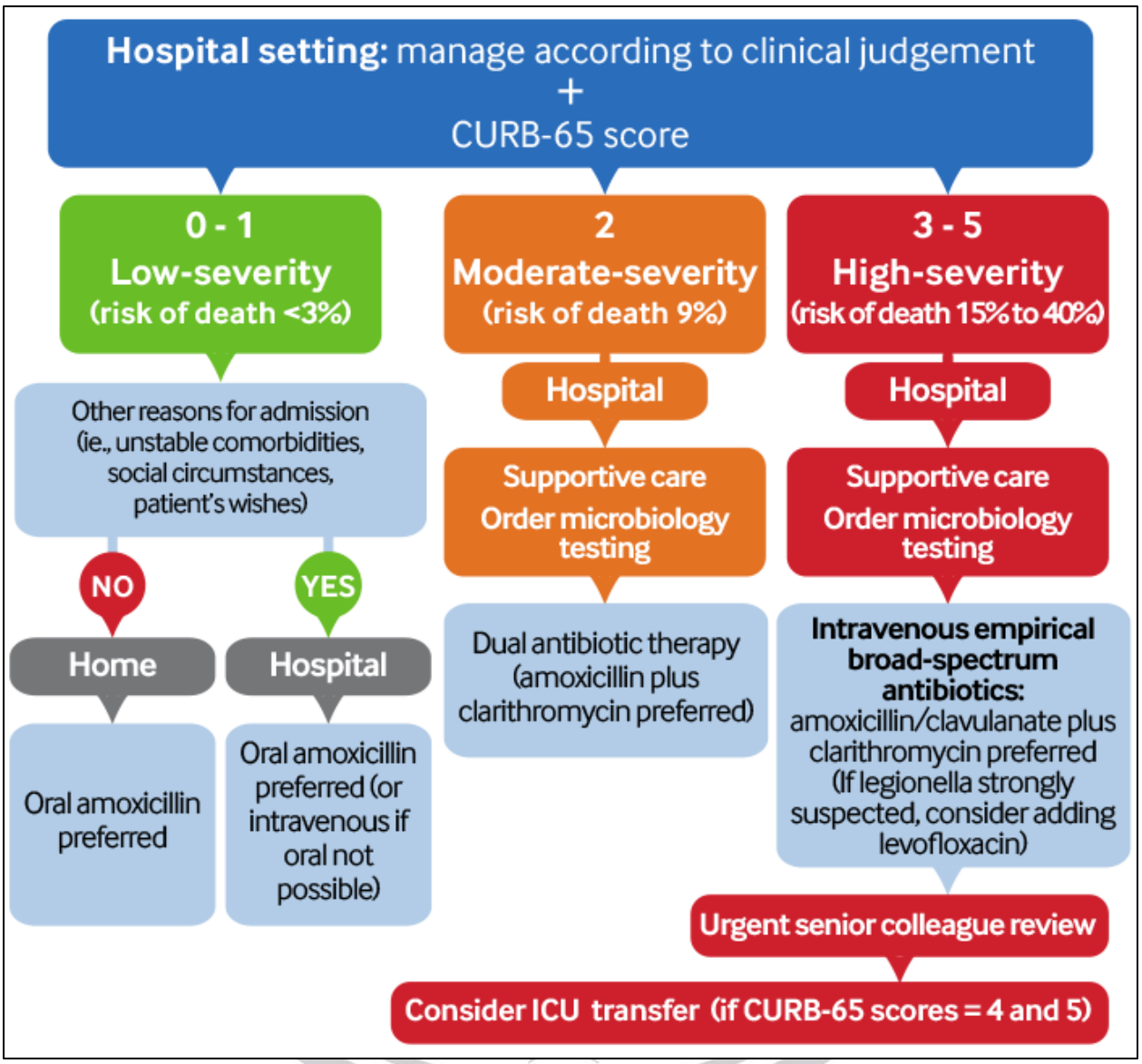



Figure 3.6. BMJ guideline to management of community acquired pneumonia

## Hospital acquired pneumonia

| Hospital acquired pneumonia (Severity) | Non penicillin allergic patients | Penicillin allergic patients |
|---|---|---|
| Non severe HAP | Co-amoxiclav | Clarithromycin |
| Severe HAP | IV tazocin +gentamycin<br>Add clarithromycin if legionella suspected<br>Add Vancomycin/teicoplanin in case of MSRA suspected | Meropenem+gentamycin |

Table 3.1. Antibiotic treatment for HAP.

Section 4

# Gastrointestinal System

# Upper Gastrointestinal bleed

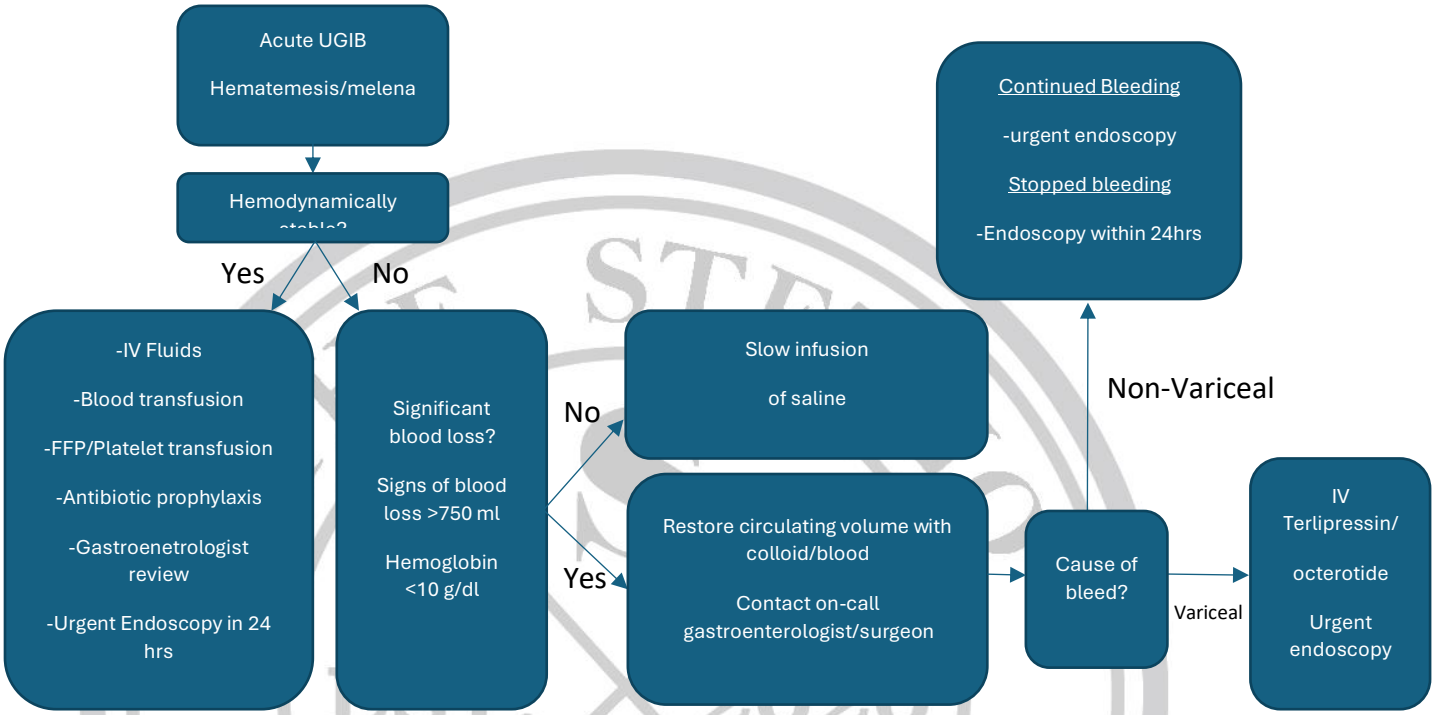



Figure 4.1. Acute management of Upper Gastrointestinal bleed.

| Table 4.1. Estimated Blood loss | |
|---|---|
| Major blood loss<br>(>1500 ml, >30% of blood volume) | • Pulse rate >120/min<br>• Systolic BP <120 mmHg<br>• Cool or cold extremities with slow or absent capillary refill<br>• Respiratory rate >20/min<br>• CNS involvement: agitation, confusion, reduced conscious level |
| Minor blood loss<br>(<750 ml, <15% of blood volume) | • Pulse rate <100/min<br>• Systolic BP >120 mmHg<br>• Normal perfusion of extremities<br>• Normal respiratory rate<br>• Normal mental state |

**Goals prior to Endoscopy**

1. Hemoglobin level >80mg/dl
2. Platelet count >50 x $10^9$/L
3. PT and APTT <1.5 × control
4. fibrinogen >1 g/L

Consider replacement as required.

**Focused history questions:**

• Estimated blood loss (Table 4.1)

• Variceal bleeding possible?

• History of UGIB and previous endoscopy findings?

• Current medications- using NSAIDs, aspirin and other antiplatelet agents, and warfarin?

• Usual or/and recent alcohol intake?

• Vomiting preceded first or hematemesis. Mallory– Weiss tear?

• Other medical problems- cardiovascular and renal disease?

**Investigations:**

• Full blood count

• Group and save serum (crossmatch 6 units of whole blood if there is shock or signs of major bleed)

• Prothrombin time

• Renal profile, Liver function tests, electrolytes

• CXR

• ECG

**Management for variceal bleed:**

1. Fluid resuscitation
2. Terlipressin 2 mg IV followed by 1–2 mg every 4–6 h until bleeding is controlled, for up to 72 h
3. If bleeding continues, put in a Sengstaken–Blakemore tube
4. Arrange for urgent endoscopy
5. IV PPI (pantoprazole) 80mg bolus followed by 8mg/hour infusion for 72 hours.
6. Antibiotic prophylaxis with IV ciprofloxacin or equivalent IV, followed by oral therapy for a total of 7–10 days.

| Causes of non-variceal bleed | Management |
|---|---|
| 1. Peptic ulcer | IV fluids<br>IV PPI<br>Treatment for possible H. pylori (clarithromycin+ amoxicillin/metronidazole+ omeprazole for 4 weeks)<br>Endoscopy with in 24 hours and repeat in 6-8 weeks |
| 2. Erosive gastritis | Stop offending drugs- NSAIDs, antiplatelets, anticoagulants (warfarin)<br>Management as advised in figure 4.1. |
| 3. Mallory Weis tear | Management as advised in figure 4.1.<br>Bleeding usually stops spontaneously and rebleeding is rare |
| 4. Esophagitis and esophageal ulcer | Give a proton pump inhibitor for 4 weeks, followed by a further 4–8 weeks treatment if not fully healed |
| 5. Upper GI hemorrhage with normal OGD | • Patients who were presented with melena only should be investigated for a small bowel or proximal colonic source of bleeding if no upper GI source is found. A normal blood urea suggests a colonic cause of melena, except in patients with chronic liver disease<br>• Visceral angiography can be useful after two negative endoscopies, but only if done when the patient is actively bleeding |

Table 4.2. Causes of non-variceal GI bleed and their management.

# Acute diarrhea

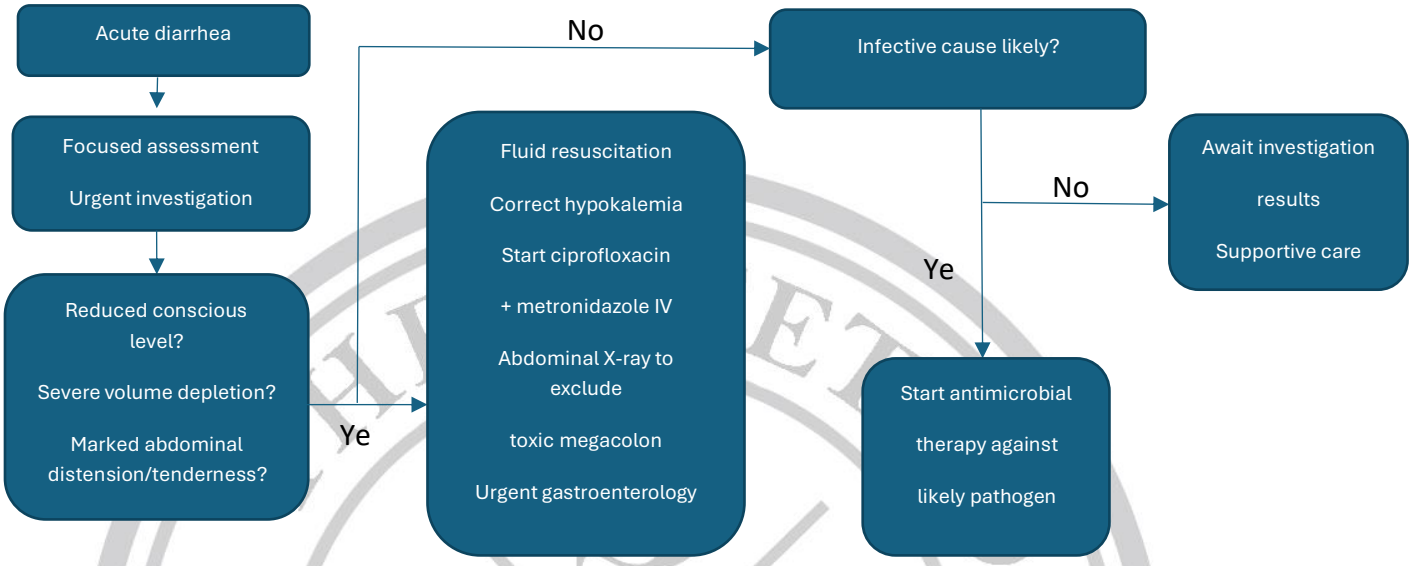



Figure 4.2. Acute management of diarrhea

**Focused history questions:**

1. Onset (sudden or gradual) and Duration
2. Frequency and nature of stools (watery, mucous or blood in stool)
3. Sick contacts?
4. Associated symptoms like fever, vomiting/nausea, abdominal pain, malaise?
5. Recent use of antibiotics?
6. Travel history
7. Any associated medical conditions

**Examination:**

1. Severity? Hemodynamic stability?
2. Toxic megacolon signs/symptoms
3. Extra-abdominal features like rash or arthropathy?

**Investigations:**

1. Routine bloods (Full blood count, serum electrolytes, renal and liver function tests)
2. Stool microscopy, cultures, ova and parasite
3. XR abdomen for toxic colon
4. Erythrocyte sedimentation rate and C-reactive protein
5. Clostridium Difficile test
6. Blood glucose
7. Sigmoidoscopy if bloody diarrhea

| Diarrheal causes | Investigation | Treatment |
|---|---|---|
| 1. Campylobacter Jejuni | Stool Culture | Ciprofloxacin 500 mg 12-hourly PO for 5 days or Erythromycin 500 mg 12-hourly PO for 5 days |
| 2. Non-typhoid Salmonella | Stool Culture | Ciprofloxacin 500 mg 12-hourly PO for 5 days or trimethoprim 200 mg 12-hourly PO for 5 days |
| 3. Escherichia coli O157:H7 | Culture of E. coli (using MacConkey E. coli) Serological tests | Supportive treatment |
| 4. Fecal impaction with overflow diarrhea | Rectal examination | Laxatives/enema |
| 5. Ulcerative colitis | Rectal biopsy | IV steroid/Ciclosporin/surgical review |
| 6. Giardiasis | Stool cyst/trophozoites in stool or jejunal biopsy | Metronidazole 400 mg 8 hourly for 5 days |
| 7. Schistosomiasis | Cyst in stool | Praziquantel single dose |
| 8. Shigellosis | Culture of Shigella from stool | Ciprofloxacin 500 mg 12-hourly PO for 5 days or Trimethoprim 200 mg 12-hourly PO for 5 days |
| 9. Clostridium difficile colitis | Clostridium difficile stool test | Oral vancomycin/oral fidaxomicin/ oral vancomycin + IV metronidazole |
| 10. Amoebic dysentery | Cyst in stool | Metronidazole 400 mg 8 hourly for 5 days |

Table 4.3. Causes of acute diarrhea and their management

# Acute Jaundice

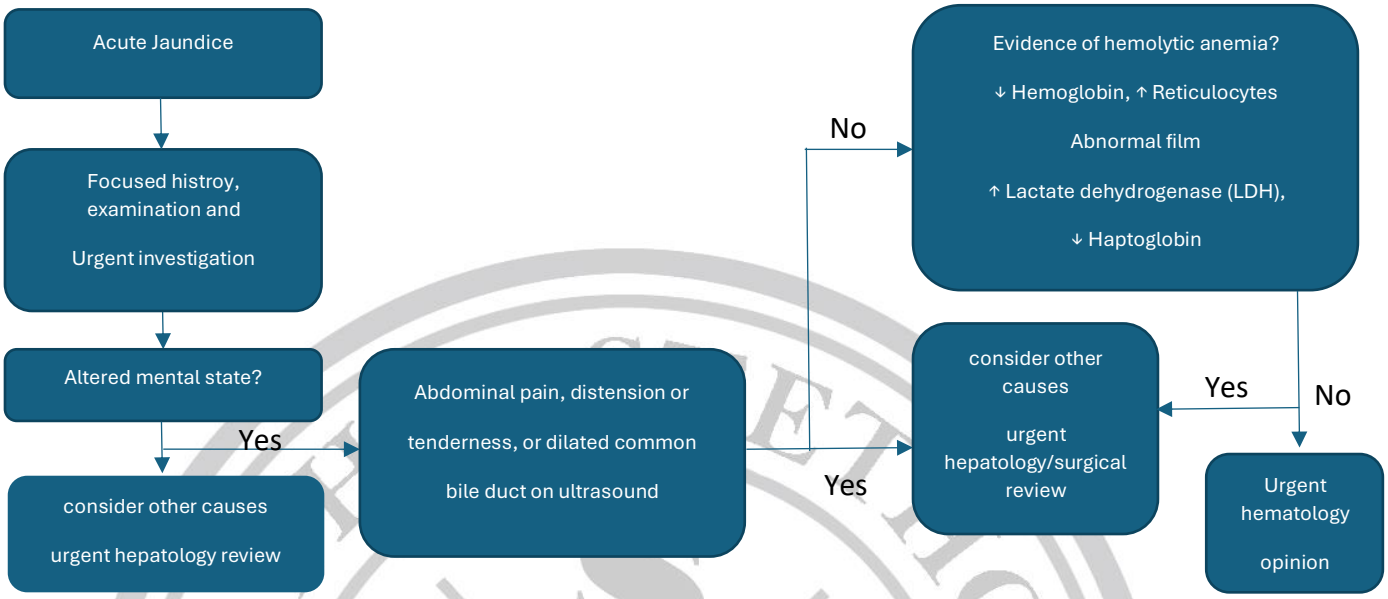



Figure 4.3. Acute management of Jaundice.

**Focused history questions:**

1. Onset, duration and associated symptoms (fever and abdominal pain)

2. Previous liver disease

3. Full drug history, including all prescription and non-prescription drugs, herbal remedies and dietary supplements over the past year

4. Risk factors like foreign travel, IV drug use, MSM (men who have sex with men), multiple sexual partners, body piercing/tattoos, blood transfusion/products, and needle-stick injury?

5. Sexual history Pregnancy?

7. Usual and recent alcohol intake

8. Other medical problems (e.g. cardiovascular, hematological disease, transplant recipient, cancer, HIV/AIDS)?

**Examination:**

• Depth of jaundice? Chronic liver disease signs? Right upper quadrant tenderness? Splenomegaly/Ascites?

• Liver enlargement? (Malignant infiltration, congestive heart failure, acute Budd–Chiari syndrome, early viral hepatitis, alcoholic hepatitis)

| Causes of Jaundice | | |
|---|---|---|
| with abnormal conscious level/mental state | with abdominal pain, distension or tenderness | With intrahepatic cholestasis |
| Fulminant hepatic failure | Acute cholangitis | Viral hepatitis |
| Decompensated chronic liver disease | Intra-abdominal sepsis | End-stage liver disease |
| Postcardiac arrest: ischemic hepatitis plus hypoxic ischemic brain injury | Budd–Chiari syndrome | Syphilitic hepatitis |
| Sepsis with multiple organ failure | HELLP syndrome of pregnancy | Intrahepatic cholestasis of pregnancy |
| Alcoholic hepatitis | Congestive heart failure | Alcoholic hepatitis |
| Falciparum malaria | Acute pancreatitis | Sepsis |
| Severe acute cholangitis | Alcoholic hepatitis | Primary biliary cirrhosis |

Figure 4.4. Causes of jaundice.

## Ascites

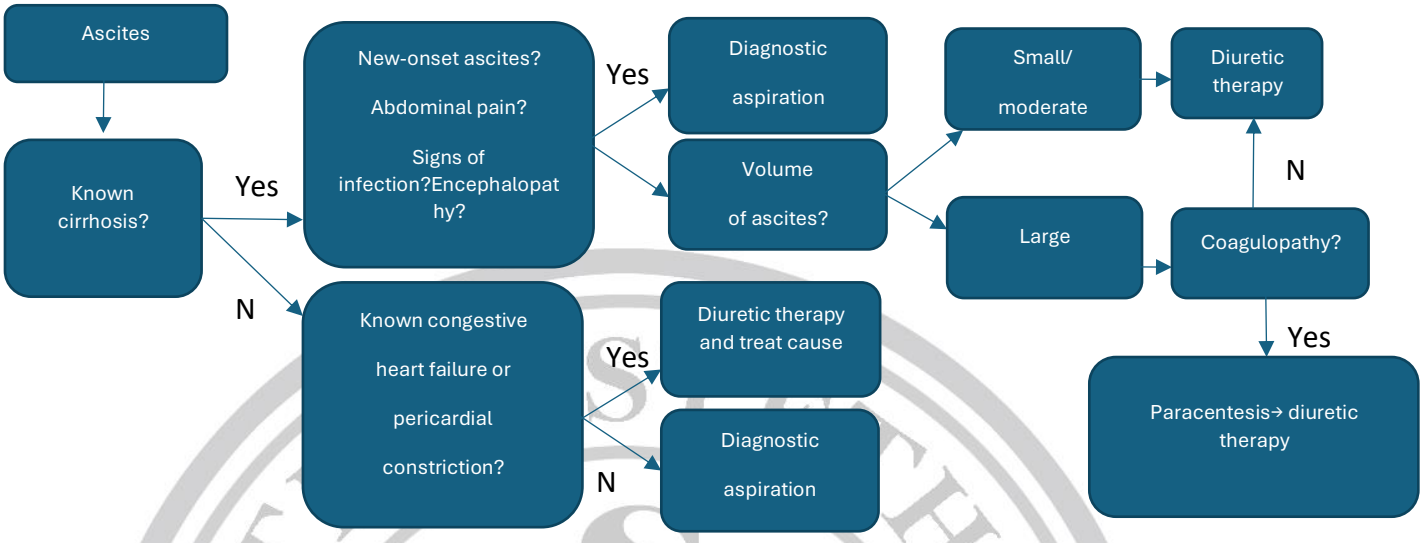



Figure 4.4. acute management of ascites.

| SAAG (11 g/L or greater)<br>Associated with portal hypertension | SAAG (<11 g/L)<br>Associated with peritoneal neoplasms, infection and inflammation |
|---|---|
| Alcoholic hepatitis | Peritoneal carcinomatosis |
| Cirrhosis | Nephrotic syndrome |
| Hepatic outflow obstruction:<br>– Budd–Chiari syndrome<br>– Portal veno-occlusive disease | Pancreatitis |
| | Serositis |
| Cardiac ascites:<br>– Tricuspid regurgitation<br>– Constrictive pericarditis<br>– Right-sided heart failure | Peritoneal tuberculosis |
| | Meig's syndrome |
| | Myxedema |

Table 4.5. Causes of ascites based on SAAG (serum albumin to ascitic gradient)

**Spontaneous bacterial peritonitis:**

1. Diagnosis: >250 neutrophils/mm3 of ascitic fluid
2. Escherichia coli is the most common organism.
3. Treat with IV cefotaxime 2 g QDS for 5 days, followed by quinolone PO for 5 days

**Ascites Management in Cirrhosis:**

1. Salt restriction to ~50 mmol/day
2. Spironolactone 100 mg daily and furosemide 40 mg daily oral
3. Target weight loss is 1kg/day for patients with peripheral edema and 0.5kg/day for without peripheral edema

4. Increase dose of spirolactone (by 100mg) and furosemide (by 40mg) after every 3-4 days, to maximum of 40omg of spirolactone and 160mg of furosemide for achieving target weight loss.
5. Amiloride can be used as an alternative to spirolactone in the case of gynecomastia.
6. Paracentesis- a single paracentesis (to remove 5 L), followed by dietary sodium restriction and diuretic therapy. Albumin solution (8 g albumin per liter of ascites removed) should be given IV during paracentesis. Seek advice from a hepatologist/ gastroenterologist.

# Acute renal failure

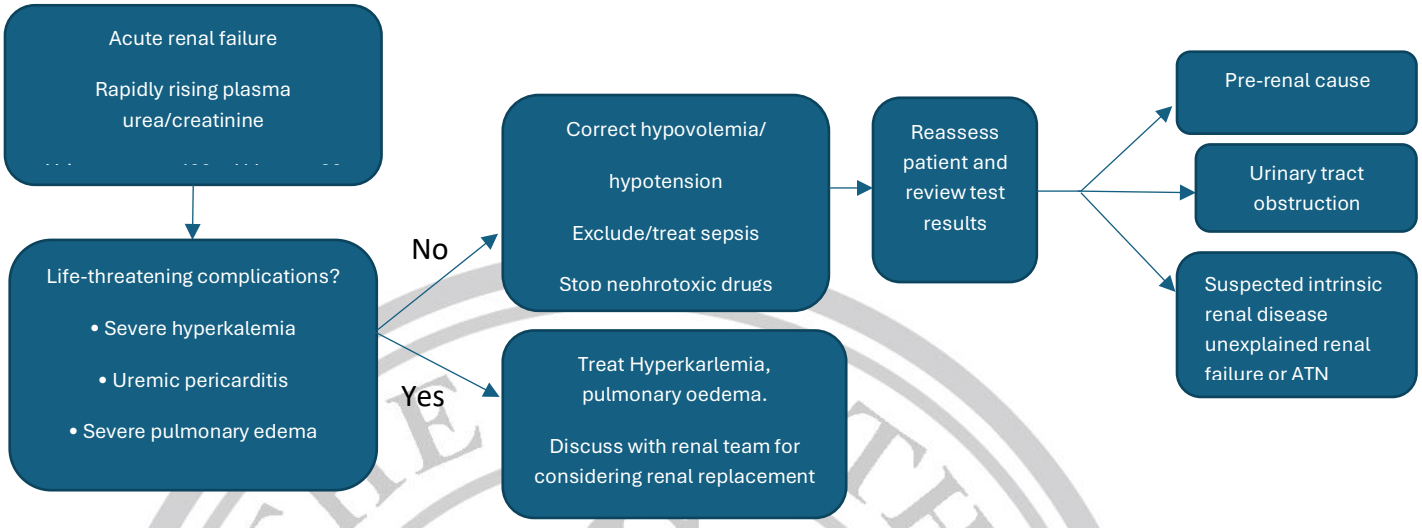



Figure 4.5. Acute management of Acute Kidney injury.

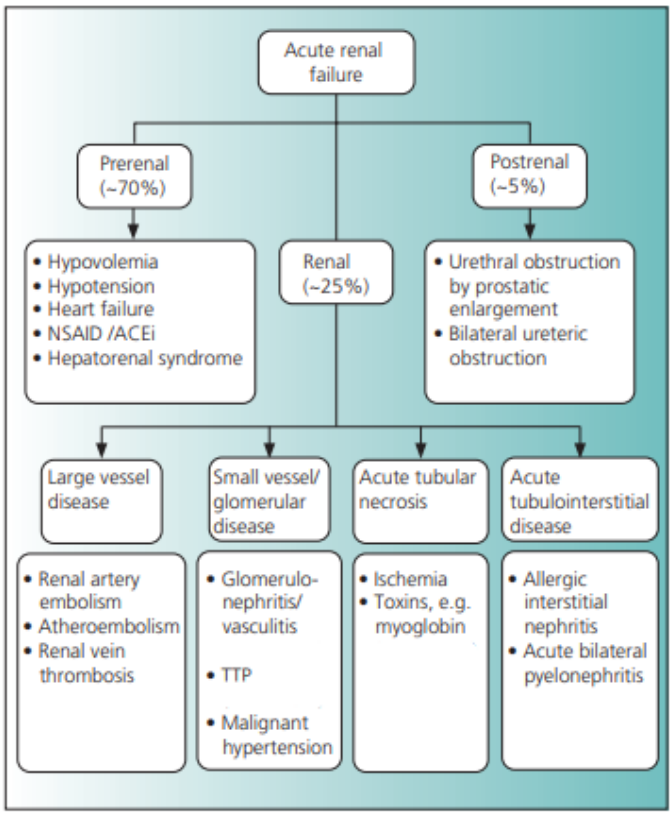



Figure 4.6. Causes of acute renal failure.

| Urine dip analysis and Microscopy |
| --- |
| **Red cells and cast, proteinuria**<br>Glomerulonephritis<br>Acute vasculitis |
| **Urine dip test positive for blood, No Blood cells on microscopy**<br>Rhabdomyolysis |
| **Tubular cell and granular casts**<br>Acute tubular necrosis |
| **White cells**<br>Urinary tract infection<br>Interstitial nephritis |
| **Normal**<br>Prerenal<br>Urinary tract obstruction<br>Acute tubular necrosis (in nephrotoxic/non-oliguric acute tubular necrosis)<br>Hypercalcemia<br>Tubular obstruction<br>Renal athero-embolism |

Table 4.6. Urine dip and Microscopy interpretation in acute kidney injury.

Section 5

# Neurology

## Acute management of stroke

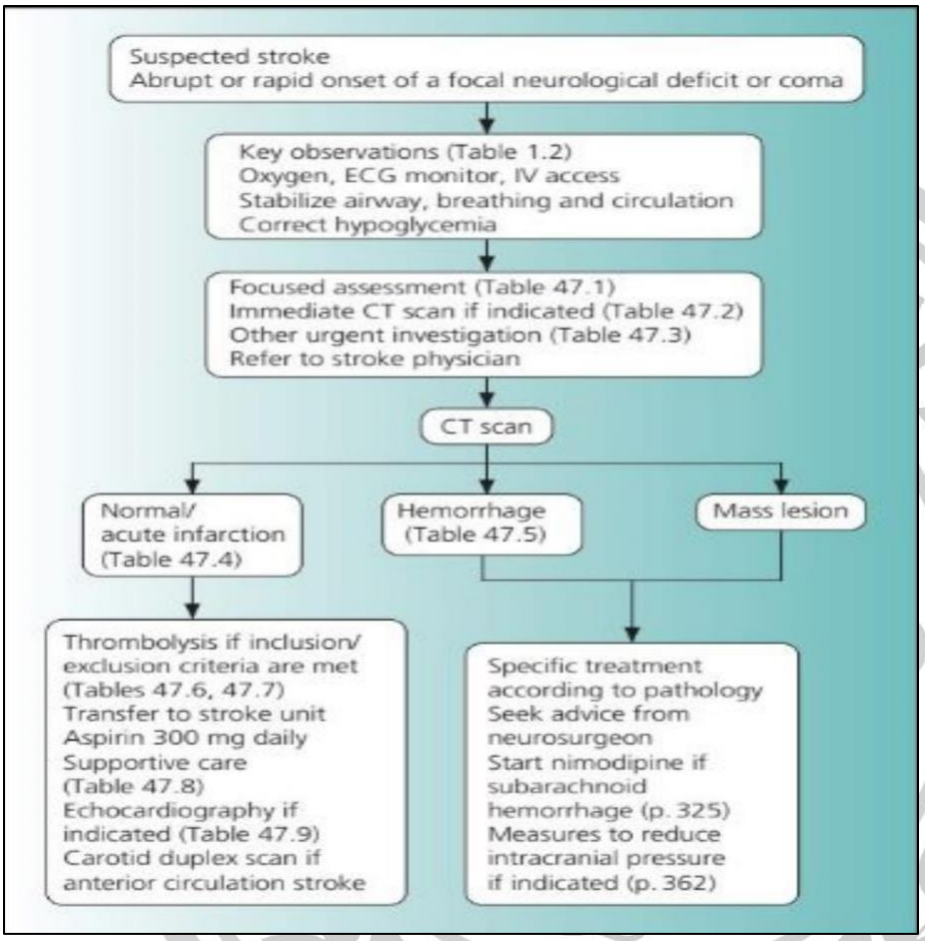



Figure 5.1. Acute management of stroke

| Focused approach for a suspected stroke: |
| --- |
| • Ensure stabilization of the airway, breathing, circulation, and blood glucose.<br>• Could this be a stroke, or is it another condition mimicking a stroke?<br>• History of trauma or alcohol abuse (consider extradural or subdural hematoma)?<br>• Gradual onset over several days (suggesting subdural hematoma or tumor)?<br>• Presence of fever (indicating brain abscess, meningitis, encephalitis, endocarditis, cerebral lupus, or cerebral malaria)?<br>• Neck stiffness (associated with meningitis or subarachnoid hemorrhage)?<br>• Severe hypertension (diastolic pressure >120 mmHg, with papilledema, retinal hemorrhages, and exudates, suggesting hypertensive encephalopathy)? |

Table 5.1. focused approach for a suspected approach

| Indications for Immediate CT Scan in Suspected Stroke: |
| --- |
| • Thrombolysis is being considered (to rule out hemorrhage).<br>• To exclude extradural or subdural hematoma.<br>• Patient on anticoagulation i.e warfarin with an increased bleeding tendency.<br>• Deteriorating consciousness level.<br>• Possible diagnoses of meningitis, encephalitis, subdural empyema, or brain abscess.<br>• Uncertainty regarding stroke diagnosis |

Table 5.2. Indications for immediate CT scan in a suspected stroke

| Urgent Investigations in Suspected Stroke: |
| --- |
| • Full blood count.<br>• ESR or C-reactive protein (consider vasculitis, endocarditis, myxoma if elevated).<br>• Coagulation screen (prothrombin time, INR if on warfarin, activated partial thromboplastin time).<br>• Sickle solubility test if possible<br>• Blood glucose<br>• Sodium, potassium, creatinine, and lipids<br>• Blood Culture (X2) if febrile or endocarditis/meningitis is suspected<br>• ECG (check for atrial fibrillation, left ventricular hypertrophy, myocardial infarction).<br>• Cranial CT (immediate if indicated, otherwise within 24 hours).<br>• Echocardiography if needed.<br>• Chest Xray |

Table 5.3 Urgent investigations in a suspected stroke

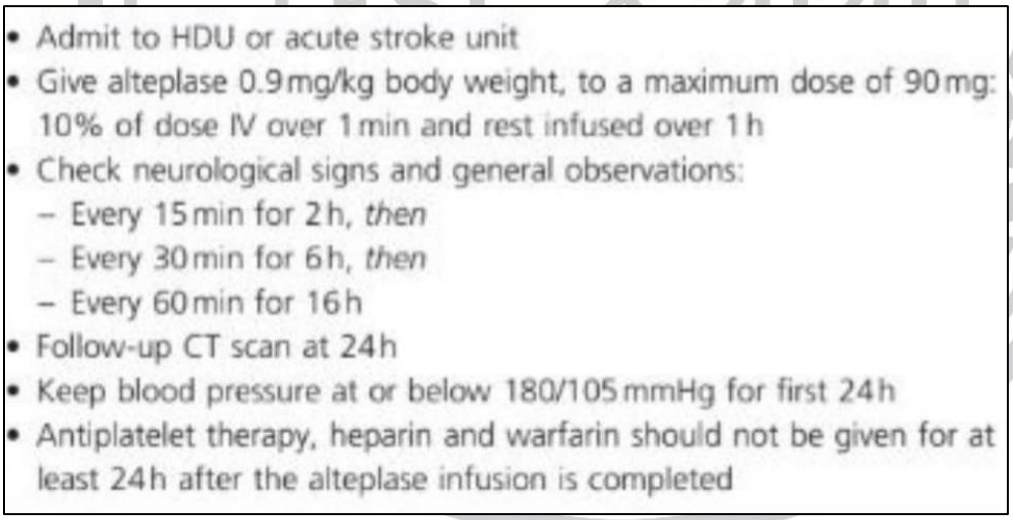



Figure 5.2: Thrombolytic therapy for acute ischemic stroke

| Inclusion Criteria for thrombolysis |
| --- |
| • Clinical diagnosis of ischemic stroke with measurable neurological deficit.<br>• Age: 18-80 years.<br>• Symptom onset <3 hours before treatment begins.<br>• CT scan shows no hemorrhage. |

Table 5.4 Inclusion criteria for thrombolysis

| Exclusion Criteria for thrombolysis: |
| --- |
| • Intracranial hemorrhage on CT.<br>• Symptoms suggest subarachnoid hemorrhage (even with normal CT).<br>• CT shows multi-lobar infarction (hypodensity > one-third of cerebral hemisphere).<br>• History of intracranial hemorrhage, neoplasm, arteriovenous malformation, or aneurysm.<br>• Uncontrolled hypertension (Systolic BP >185 mmHg, Diastolic BP >110 mmHg).<br>• Witnessed seizure at stroke onset.<br>• Active internal bleeding, recent trauma, or surgery. |

Table 5.5 Exclusion criteria for thrombolysis

## Transient Ischemic Attack

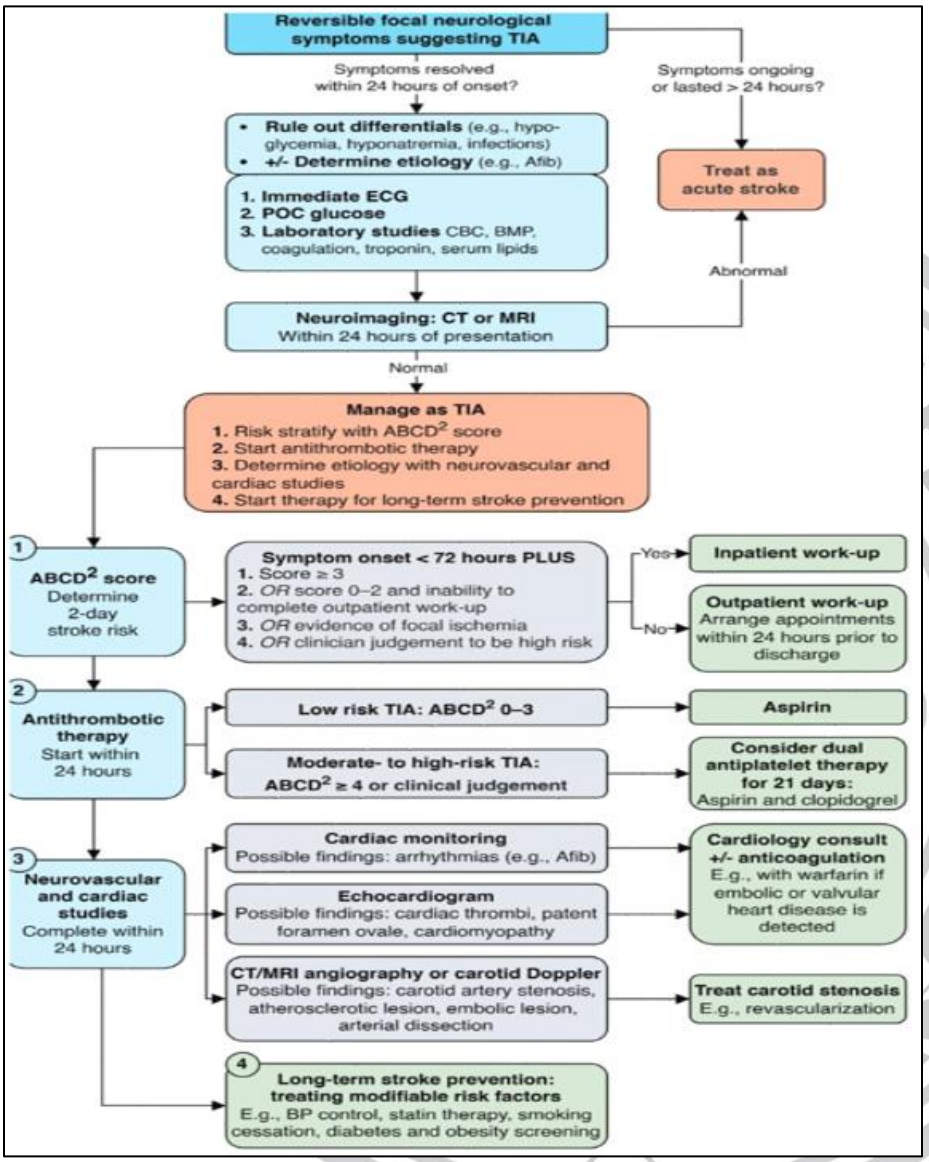



Figure 5.3. Acute management of TIA

According to the **NICE guidelines**, symptoms of a Transient Ischemic Attack (TIA) can be summarized as follows:

- Sudden weakness or numbness on one side of the body (face, arm, or leg).
- Sudden speech difficulties, such as slurred speech or inability to speak.
- Sudden vision loss or blurring in one or both eyes.
- Sudden dizziness, loss of balance, or coordination issues.

- Sudden confusion or difficulty understanding others.
- These symptoms are typically short-lasting but require urgent medical attention

| Symptom | Carotid TIA | Vertebrobasilar TIA |
|---|---|---|
| **Dysphasia** | Yes | No |
| **Loss of vision in one eye** | Yes | No |
| **Loss of vision in both eyes** | No | Yes |
| **Hemianopia** | Yes | Yes |
| **Diplopia** | No | Yes |
| **Dysarthria** | Yes | Yes |
| **Loss of balance** | Yes | Yes |
| **Unilateral motor loss** | Yes | Yes |
| **Unilateral sensory loss** | Yes | Yes |

Figure 5.6. Symptoms of carotid & vertebrobasilar TIA

**ABCD 2 SCORE**

| | | |
|---|---|---|
| Age 60 or older | - | 1 point |
| Blood pressure ≥140/90 | - | 1 point |
| Clinical: | | |
| Unilateral weakness | - | 2 points |
| Speech impairment | - | 1 point |
| Duration: | | |
| 60 minutes or more | - | 2 points |
| <60 minutes | - | 1 point |
| Diabetes mellitus | - | 1 point |

Figure 5.7 Criteria for Admitting patient with TIA for further workup

# Acute Bacterial Meningitis

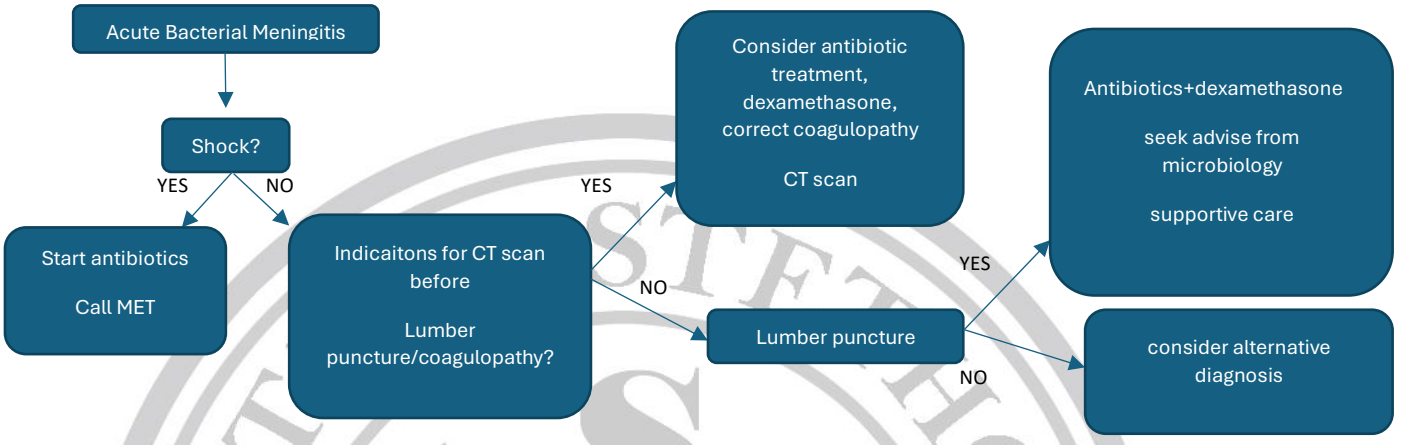



Figure 5.8. Acute management of bacterial meningitis

| Urgent investigations in suspected meningitis |
|---|
| Blood cultures X2 |
| Throat Swab |
| Lumber puncture after the CT head is done if indicated |
| FBC |
| Coagulation screen |
| CRP |
| Blood Glucose |
| Renal profile |
| ABGs or VBG |
| CXR |

Table 5.6. Urgent investigations in suspected meningitis

| Management of Acute Bacterial Meningitis |
| --- |
| **In-Hospital Antibiotic Treatment:**<br>**Infants (<3 months):**<br>**Intravenous cefotaxime** with **amoxicillin or ampicillin** (to cover *Listeria monocytogenes*).<br>**Children (3 months to 18 years) and adults:**<br>**Intravenous ceftriaxone** or **cefotaxime**.<br>**If *Listeria* is suspected** (age >50 years, immunocompromised):<br>Add **amoxicillin or ampicillin** to the antibiotic regimen.<br>**Penicillin allergy:**<br>Use **chloramphenicol** or **meropenem** (if severe allergy).<br>**Corticosteroids:**<br>**Dexamethasone** should be given immediately, ideally before or with the first dose of antibiotics:<br>**Adults and children (>3 months):** 0.15 mg/kg every 6 hours for 4 days (to reduce neurological complications).<br>**Supportive Care:**<br>**Oxygen therapy** if required.<br>**Intravenous fluids** to maintain hydration.<br>**Management of raised intracranial pressure** (e.g., positioning, possible diuretics).<br>**Antipyretics** for fever control.<br>**Prophylaxis for Contacts:**<br>**Antibiotic prophylaxis (e.g., rifampicin, ciprofloxacin, or ceftriaxone)** for close contacts in cases of meningococcal meningitis.<br>**Public Health Measures:**<br>Notify **public health authorities** of confirmed cases of meningococcal or Haemophilus influenzae type b (Hib) meningitis.<br>**Vaccination:**<br>**Meningococcal vaccines** should be considered for close contacts and outbreak management. |

Table 5.7. Management of Acute bacterial meningitis according to NICE guidelines

# Gullian Barre Syndrome

## Signs and symptoms / Presentation of patient with GBS

**Initial Symptoms:**

- **Tingling or numbness** (paraesthesia) in the hands and feet is often the earliest sign.
- **Muscle weakness** usually starts in the legs and may extend to the arms and face.
- **Aching or pain** in the lower back is common.

**Progression:**

- **Weakness that ascends**: It generally starts in the legs and spreads upward, eventually affecting the arms, trunk, and face.
- **Reduced or absent reflexes**: Reflexes, such as the knee jerk, often diminish or disappear early in the condition.

**Facial and Bulbar Involvement:**

- **Trouble with facial movements**, including difficulties with speaking, chewing, or swallowing.
- **Paralysis on both sides of the face** may occur in some cases.

**Respiratory Involvement:**

- **Breathing difficulties** due to muscle weakness can develop in more severe cases.
- In extreme cases, **respiratory failure** may occur, requiring mechanical ventilation.

**Autonomic Dysfunction (in some cases):**

- **Irregular heart rate** (can be either too fast or too slow).
- **Blood pressure fluctuations**, such as high or low blood pressure.
- **Bladder or bowel issues**, like difficulty urinating.

**Pain:**

- **Intense nerve pain**, particularly in the lower back and legs, can occur.

**Speed of Onset:**

- The condition usually develops **over days to weeks**, with the peak weakness occurring within two to four weeks after the first symptom.



Table 5.8. Signs and symptoms to look for in GBS

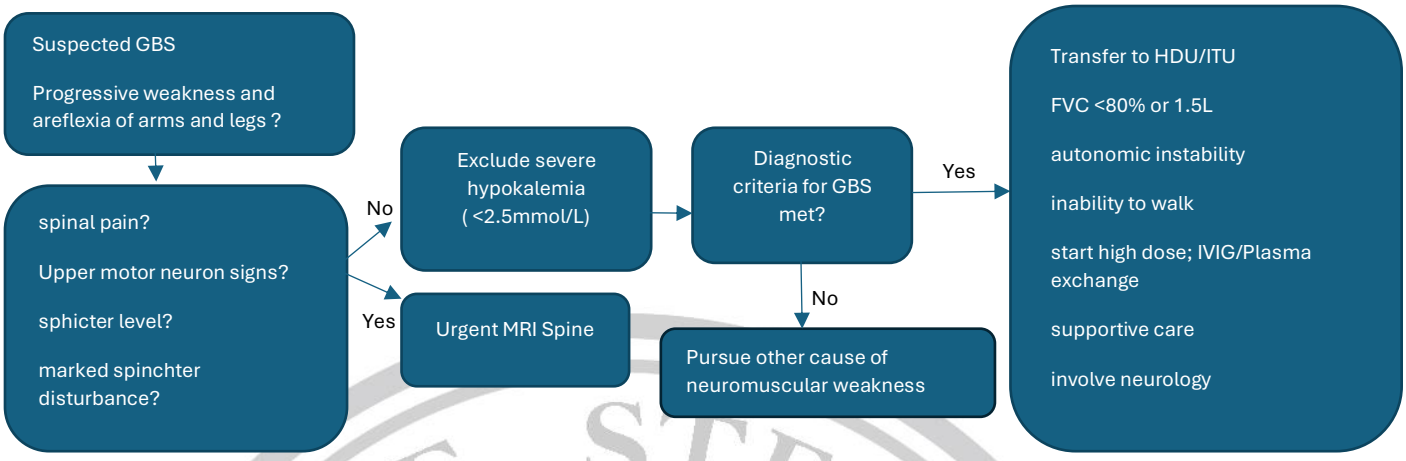



Figure 5.9. Acute management of Gullian Bare syndrome.

| Signs and symptoms suggestive of a different diagnosis |
| --- |
| • Recent diphtheria infection<br>• Diagnosis of other neurological problems i.e Myasthenia, Poliomyelitis<br>• Sensory deficit without weakness |

Table 5.9. Signs and symptoms suggestive of another diagnosis

| Investigations |
| --- |
| Electromyography (EMG) and Nerve Conduction Studies (NCS):<br>Lumbar Puncture – to check for elevated protein levels (albuminocytologic dissociation) with normal white cell counts, which is indicative of GBS.<br>Magnetic Resonance Imaging (MRI) – rule out other conditions (e.g., spinal cord lesions) and assess for nerve root swelling<br>Complete blood count (CBC), inflammatory markers, and tests for infectious agents (e.g., Campylobacter jejuni, cytomegalovirus, or Epstein-Barr virus) that may be associated with GBS.<br>Measurement of vital capacity with spirometer - To evaluate respiratory muscle involvement and monitor for signs of respiratory failure.<br>ECG<br>CXR |

Table 5.10. Investigations for GBS

| Management and Supportive care for GBS in the hospital |
| --- |
| **Continuous respiratory monitoring:**<br>Assess for early signs of respiratory failure, such as increased work of breathing, reduced tidal volume, or declining oxygen saturation.<br>**Transfer to ITU** if there are indications of respiratory distress or the need for mechanical ventilation.<br>**Regular evaluations of limb strength:**<br>Monitor muscle strength to determine the progression of the condition and adjust treatment plans as needed.<br>**Cardiovascular monitoring:**<br>Track heart rate and blood pressure, managing any autonomic instability or fluctuations.<br>Consider ITU transfer for severe autonomic dysfunction, such as significant bradycardia or hypotension that requires advanced monitoring and intervention.<br>**Effective pain management:**<br>Use analgesics or neuropathic pain medications to manage discomfort.<br>**Nutritional support:**<br>Provide enteral feeding or intravenous nutrition if the patient has swallowing difficulties.<br>**Preventive measures for immobility-related complications:**<br>Implement strategies such as compression stockings, regular repositioning, and physical therapy to minimize the risk of deep vein thrombosis (DVT) and pressure ulcers.<br>**Rehabilitation services:**<br>Facilitate recovery and improve functional mobility as strength returns. |

Table 5.11. Management of GBS

| Indications for transfer to ITU |
| --- |
| 1. Respiratory failure requiring mechanical ventilation.<br>2. Severe autonomic instability necessitating close monitoring and intervention.<br>3. Significant muscle weakness affecting vital functions, leading to potential complications.<br>4. Any other condition that may require advanced supportive care beyond what can be provided in a general ward setting. |

Table 5.12. Indications for transfer to ITU

## Spinal Cord Compression

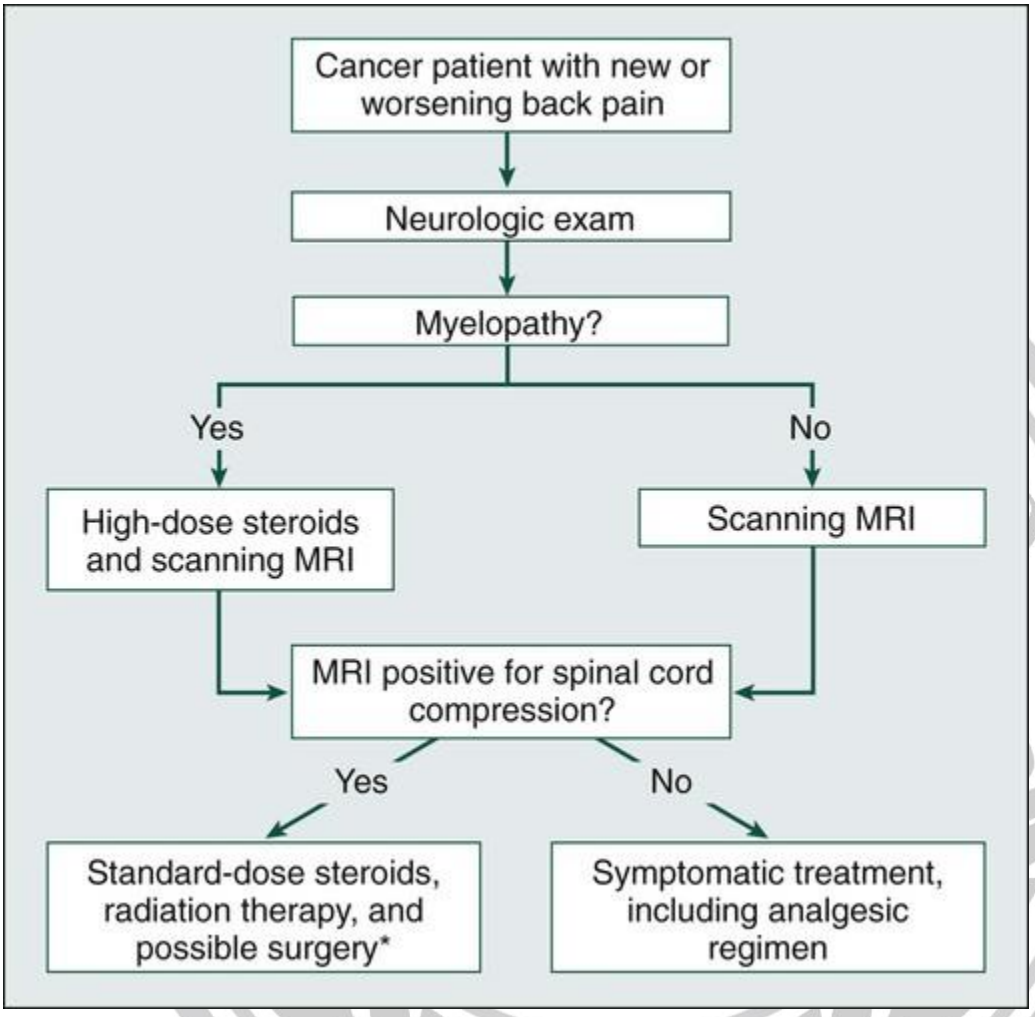



Figure 5.10. Acute management of Spinal cord compression

**Cord Compression**: Symptoms and management vary based on the level of compression. Immediate intervention may be required, especially for cervical and thoracic compression, where neurological functions can be significantly affected.

**Cauda Equina Syndrome**: Represents a surgical emergency due to potential permanent damage. Immediate diagnosis and decompression are critical to prevent lasting impairments.

| Level of Cord Compression | Signs and Symptoms | Management |
|---|---|---|
| Cervical Compression (C1-C8) | - Neck pain<br>- Arm weakness or numbness<br>- Changes in grip strength<br>- Difficulty walking<br>- Loss of bowel/bladder control | - Immediate imaging (MRI/CT)<br>- Surgical intervention if severe<br>- Pain management<br>- Physical therapy |
| Thoracic Compression (T1-T12) | - Upper back pain<br>- Weakness in legs<br>- Sensory loss below the level of compression<br>- Difficulty with balance<br>- Bowel and bladder dysfunction | - Imaging studies to assess compression<br>- Surgical decompression<br>- Pain management<br>- Rehabilitation |
| Lumbar Compression (L1-L5) | - Low back pain<br>- Weakness in legs<br>- Sensory changes in the legs or feet<br>- Reflex changes (e.g., diminished ankle reflex) | - Imaging to evaluate compression<br>- Consider surgical intervention<br>- Pain relief measures<br>- Physical therapy |
| Cauda Equina syndrome (Typically occurs at the L2 level or below, involving lumber and sacral nerve roots (L2 -L5)) | - Severe lower back pain<br>- Sciatica (radiating leg pain)<br>- Saddle anesthesia (numbness in the buttocks/perineal area)<br>- Urinary retention or incontinence<br>- Bowel dysfunction<br>- Lower extremity weakness | - Urgent MRI to confirm diagnosis<br>- Immediate surgical decompression (ideally within 24-48 hours)<br>- Pain management<br>- Supportive care and rehabilitation |

Table 5.13. Spinal cord compression and QES

## Status Epilepticus

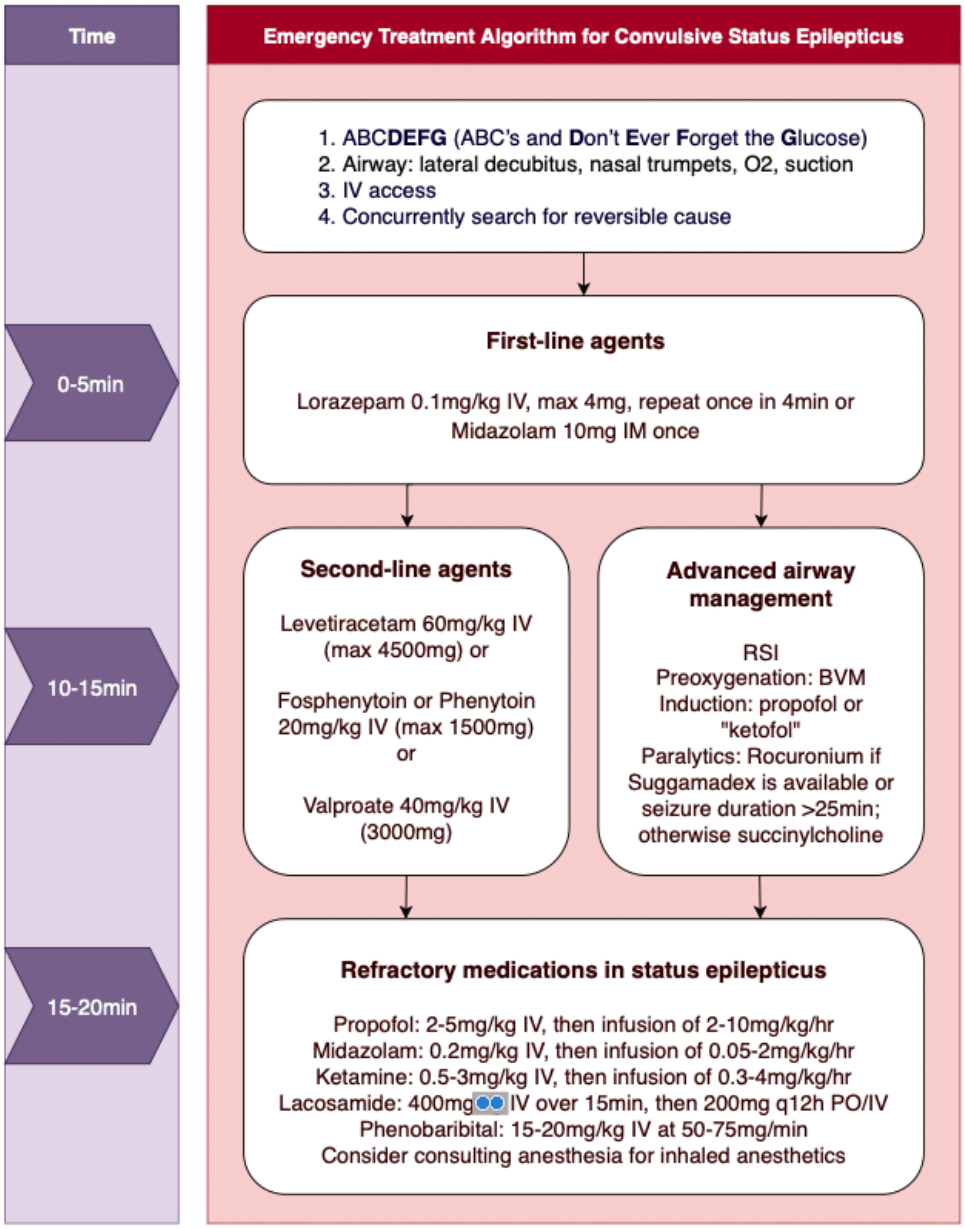



Figure 5.11. Acute management of status epilepticus

| Treatment line | Medication | Dosage |
|---|---|---|
| First line | Lorazepam | - Adults: 4 mg IV over 2 minutes (may repeat after 10-15 minutes if seizures persist)<br>- Children: 0.1 mg/kg (max 4 mg) IV over 2 minutes (may repeat after 10-15 minutes) |
| | Diazepam (alternate) | - Adults: 10-20 mg IV (may repeat every 10-15 minutes, max 30 mg)<br>- Children: 0.1-0.3 mg/kg IV (max 10 mg) (may repeat every 10-15 minutes) |
| Second Line | Phenytoin (or Fosphenytoin) | - Phenytoin: 15-20 mg/kg IV (load at 50 mg/min)<br>- Fosphenytoin: 20 mg PE/kg IV (load at 3 mg PE/kg/min) |
| Third Line | Valproate (or Levetiracetam) | - Valproate: 20-40 mg/kg IV (loading dose, max 3000 mg)<br>- Levetiracetam: 1 g IV (may repeat after 10-15 minutes) |
| Fourth Line | Midazolam (or Thiopental) | - Midazolam: Continuous IV infusion starting at 0.1-0.2 mg/kg/hour (titrate as needed)<br>- Thiopental: Loading dose of 3-5 mg/kg IV, followed by continuous infusion |

Table 5.14. Medications used in Status Epilepticus and their dosage

| Features of non-epileptic attack disorder aka Pseudo seizures |
| --- |
| • Non-rhythmic, often jerky or thrashing movements.<br>• Movements may lack the organized pattern seen in epileptic seizures.<br>• Incontinence and tongue biting is rare<br>• Normal reflexes<br>• Minimal or no postictal confusion<br>• No epileptiform activity observed during EEG monitoring during attacks |

Table 5.15. Features of pseudo seizures

| Investigations |
| --- |
| • CBC, Blood glucose, RLU, Blood gas<br>• Anticonvulsant levels if patient is already taking any.<br>• CXR<br>• Blood Culture<br>• LP – if cerebral infection is suspected<br>• CT head – (persistent headache, on anticoagulation, persistently confused, Focal neurological deficit)<br>• ECG |

Table 5.16. Investigations

Section 6

# Endocrine and Metabolic

# Hypoglycemia

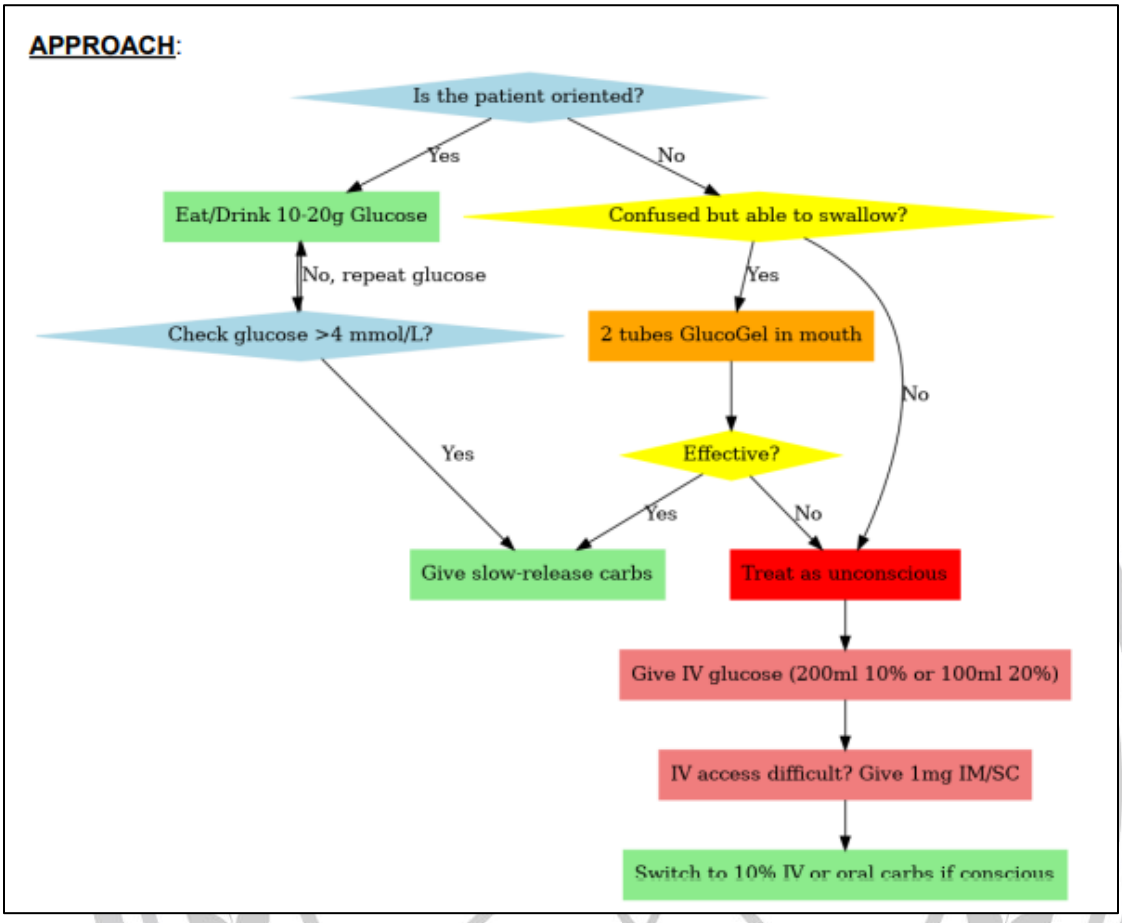



Figure 6.1. Acute management of Hypoglycemia

**DEFINITION**

Glucose <3 mmol/L with or without symptoms.

**CAUSES OF HYPOGLYCAEMIA**

Patients with diabetes:

● Overuse of insulin ● Excessive intake of sulfonylurea ● Renal failure resulting in reduced clearance of insulin and sulfonylurea ● Coexisting endocrine disorders, such as adrenal insufficiency, hypothyroidism, or hypopituitarism ● Gastroparesis or poor absorption in the gastrointestinal system

Patients with or without diabetes:

● Alcohol binge, which inhibits liver gluconeogenesis ● Prolonged fasting or malnutrition ● Severe liver disease ● Sepsis ● Salicylate toxicity ● Adrenal insufficiency

## Hyperosmolar Hyperglycemic state:

| Clinical features (all the below) | | Aims of therapy | Criteria for resolution of HHS: Holistic assessment of the following: |
|---|---|---|---|
| 1) Marked hypovolaemia | **A mixed picture of HHS and DKA occurs relatively frequently** | 1) Improvement in clinical status and replacement of all estimated fluid losses by 24 hours | 1) Clinical and cognitive status is back to the pre-morbid state |
| 2) Osmolality ≥320 mOsm/kg | | 2) Gradual decline in osmolality: drop of 3-8 mOsm/kg/hr | 2) Osmolality <300 mOsm/kg |
| 3) Marked hyperglycaemia (≥30 mmol/L) | | 3) Blood glucose: aim to keep to 10-15 mmol/L in the first 24 hours | 3) Hypovolaemia has been corrected (urine output ≥0.5 ml/kg/hr) |
| 4) Without significant ketonaemia (≤3.0 mmol/L) | | 4) Avoid hypoglycaemia and hypokalaemia | 4) Blood glucose <15 mmol/L |
| 5) Without significant acidosis (pH ≥7.3) and bicarbonate ≥15 mmol/L | | 5) Prevent harm: VTE, osmotic demyelination, fluid overload, foot ulceration | |

| Theme / Time | 0-60 minutes | 60 minutes - 6 hours | 6-12 hours | 12-24 hours | 24-72 hours |
|---|---|---|---|---|---|
| **Clinical assessment and monitoring** | | | | | |
| **Clinical status / NEWS** | ABCDE approach, History/Examination, NEWS, cardiac monitoring, urine output<br>Establish adequate intravenous lines (preferably 2 large bore IV cannulas)<br>Discuss with outreach/ICU team early if there are markers of high severity **(see Table 1 overleaf)** | | | Check for continuing improvement | Expect steady recovery, patient eating and drinking, and biochemistry as it was prior to HHS<br>Ongoing management of the precipitating cause(s)<br>Replacement of all estimated fluid losses by 24 hours<br>Individual BG target 6-10 mmol/L |
| **Precipitating cause(s)** | Assess for precipitating cause(s):<br>sepsis, diabetic foot infection, treatment omissions, vulnerable adult, vascular event (myocardial infarction, stroke) | | | Ongoing management of the precipitating cause(s) | |
| **Osmolality (VBG/blood)**<br>**Measure/calculate (2xNa⁺) + Glucose + Urea**<br>**Aim for gradual decline of 3-8 mOsm/kg/hr** | Check every hour for 6 hours<br>Until the urea is available, calculate using (2 x $Na^+$ + glucose). Recalculate osmolality once urea is available, and then use (2 x $Na^+$ + glucose + urea) | | Check every 2 hours | Check every 4 hours (if no clinical improvement then check every 2 hours) | |
| **How to interpret osmolality results** | **Check Figure 1 overleaf** | **Check Figure 1 overleaf** | **Check Figure 1 overleaf** | **Check Figure 1 overleaf** | |
| **Blood glucose (BG)**<br>**(Aim for 10-15 mmol/L in the first 24 hours)** | Check every hour<br>Fall in BG should be up to 5.0 mmol/L per hour<br>**(check Figure 2 overleaf for details)** | | **Check every hour (check Figure 2 overleaf for details)** | **Check every hour (check Figure 2 overleaf for details)** | |
| **Interventions** | | | | | |
| **Intravenous fluid (0.9% saline)**<br>**(In IV line 1)**<br>**(caution in HF/CKD/BW <50 kg)** | 1 litre over 1 hour<br>(caution in HF/CKD/BW <50 kg) | Aim for 2-3 litres positive balance by 6 hours | Aim for up to 6 litres positive balance by 12 hours | Reassess fluid balance to plan fluids replacement for the next 12 hours | Can be stopped if patient is eating and drinking |
| **Insulin infusion**<br>**(FRIII 0.05 units/kg/hr using Actrapid®)**<br>**(In IV line 2)** | Use DKA guidelines if ketonaemia (>3.0 mmol/L) or ketonuria (≥2+)<br>Start FRIII if ketonaemia (>1.0 - ≤3.0 mmol/L) or ketonuria (<2+) | Only commence if positive fluid balance and BG plateaued on repeated measurements (>2 occasions) | | Rate may need adjustment by another 1 unit/hr to achieve BG target 10-15 mmol/L | VRIII if not eating and drinking<br>Otherwise convert to subcutaneous insulin |
| **Glucose infusion: 5% or 10% @ 125ml/hr**<br>**(In IV line 2)** | Not required at this stage | Only initiate if BG <14 mmol/L | | Continue infusion at 125 ml/hr | Can be stopped if patient is eating and drinking |
| **Potassium (avoid hypokalaemia)** | Senior review / ICU outreach if potassium <3.5 or >6.0 mmol/L | **Check Table 2 overleaf for potassium replacement guidelines** | **Check Table 2 overleaf for potassium replacement guidelines** | **Check Table 2 overleaf for potassium replacement guidelines** | Check U&Es daily |
| **Assessments and prevention** | | | | | |
| **Prevent harm** | VTE prophylaxis (low molecular weight heparin)<br>Assess for complications e.g. fluid overload, cerebral oedema, osmotic demyelination (deteriorating conscious level) | | | | VTE prophylaxis until discharge<br>Daily feet checks |
| **Prevent hypoglycaemia** | Glucose 5% or 10% at 125 ml/hr if BG <14 mmol/L | | | | Target BG 6-10 mmol/L |
| **Prevent foot ulceration** | Daily foot checks | | | | Daily foot checks |
| **Refer to the inpatient diabetes team early.**<br>**Escalate management if there is clinical deterioration.** | | | | | Review by inpatient diabetes team before discharge |

Figure 6.2. Hyperosmolar Hyperglycemic state care pathway.

- Osmolality >350 mOsm/kg
- Sodium >160 mmol/L
- Venous/arterial pH <7.1
- Hypokalaemia (<3.5 mmol/L) or hyperkalaemia (≥6 mmol/L) on admission
- Glasgow Coma Scale (GCS) <12 or abnormal AVPU (Alert, Voice, Pain, Unresponsive) scale
- Oxygen saturation <92% on air (assuming normal baseline respiratory function)
- Systolic blood pressure <90 mmHg
- Pulse >100 or <60 beats per minute
- Urine output <0.5 ml/kg/hour
- Serum creatinine >200 µmol/L and/or Acute kidney injury
- Hypothermia
- Macrovascular event such as myocardial infarction or stroke
- Other serious co-morbidity

Figure 6.3. ITU referral criteria

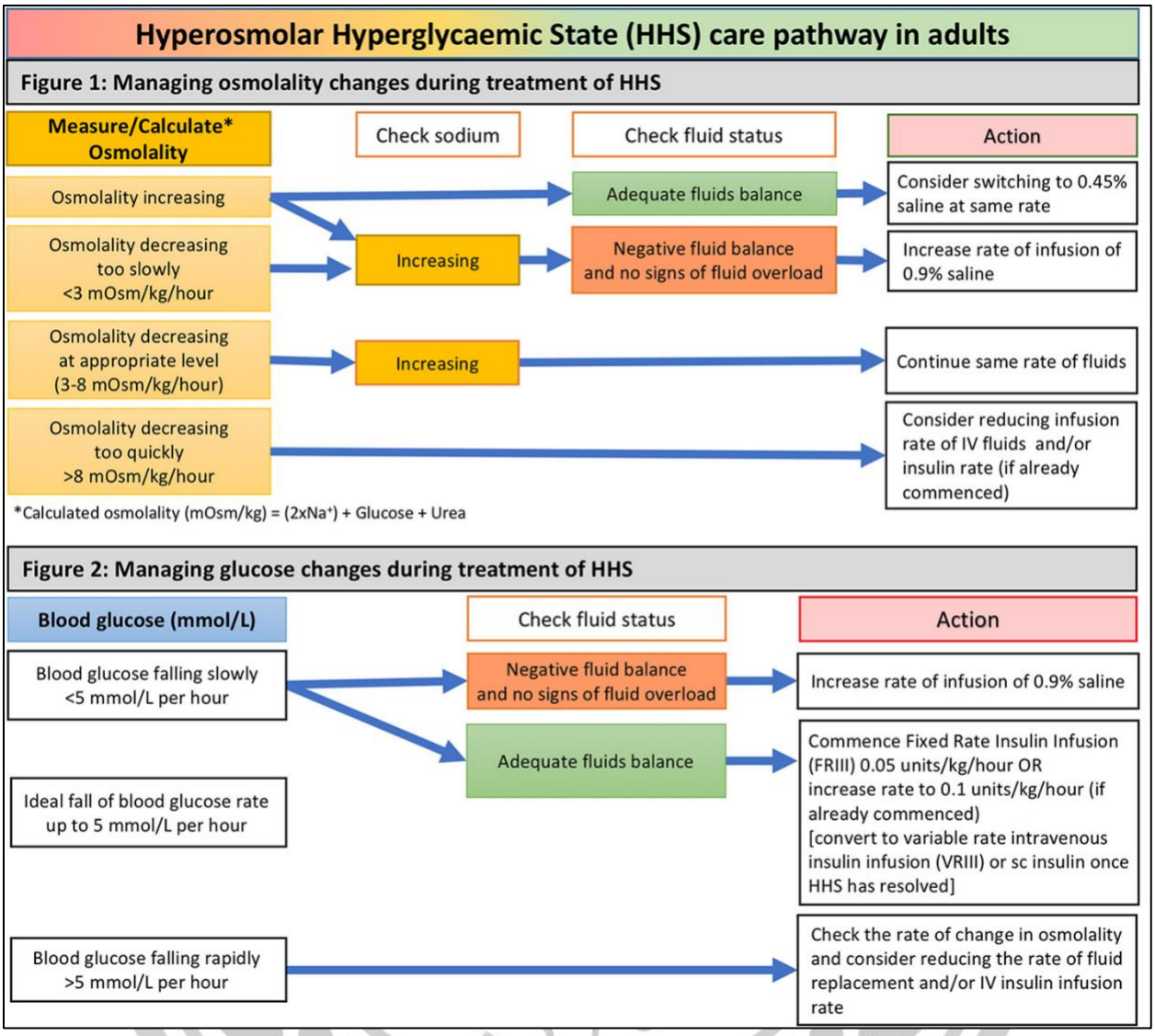



Figure 6.4. Acute management of HHS

| Potassium level in first 24 hours (mmol/L) | Potassium replacement in infusion solution |
|---|---|
| ≥6.0 | Senior review ICU/outreach |
| 5.5-5.9 | Nil |
| 3.5-5.5 | 40 mmol/L |
| <3.5 | Senior review. Additional potassium is required (via central line in high dependency unit). |

Figure 6.5. Potassium management

## Diabetic ketoacidosis:

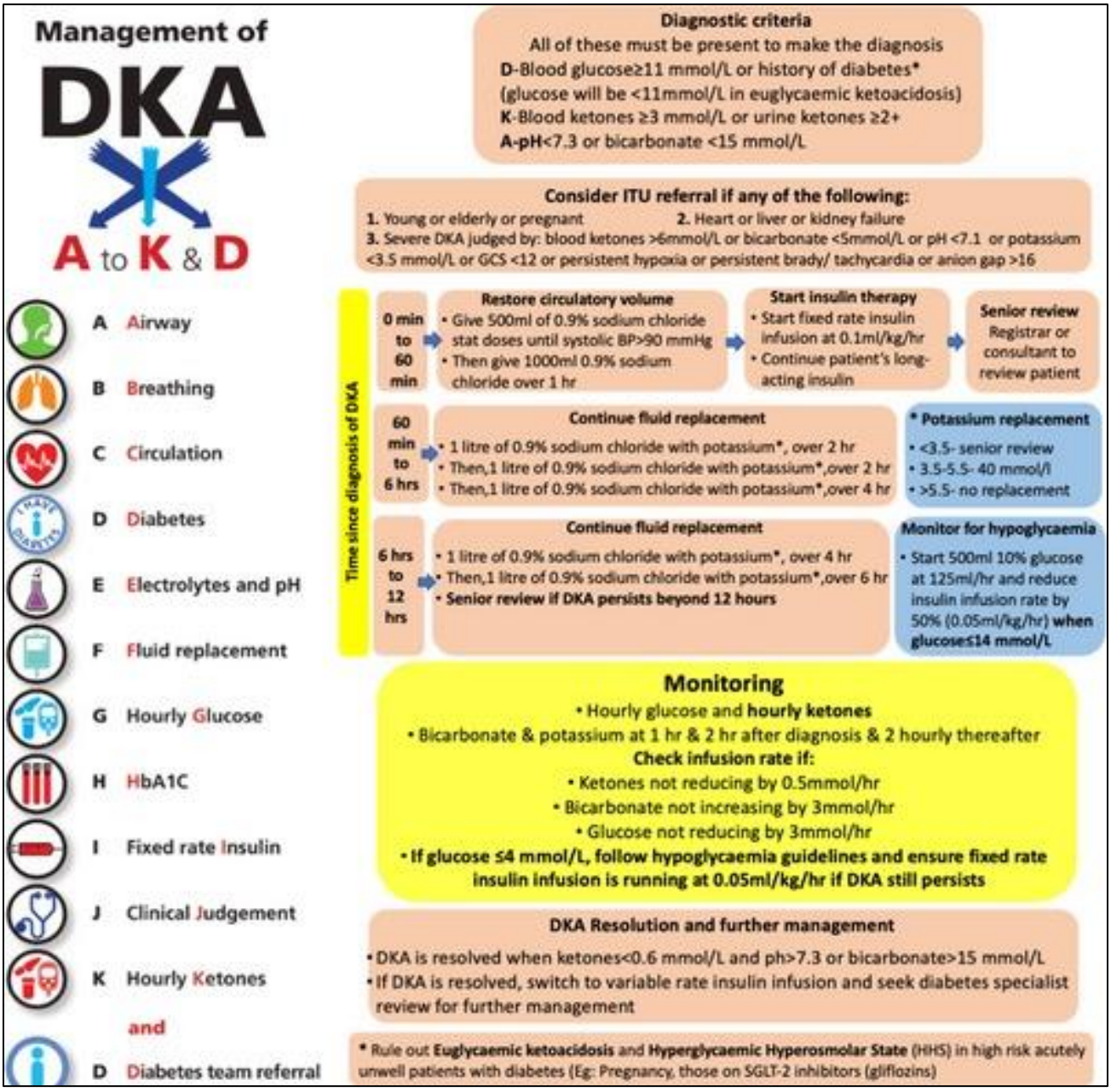



Figure 6.6. acute management of Diabetic ketoacidosis

# Thyrotoxicosis

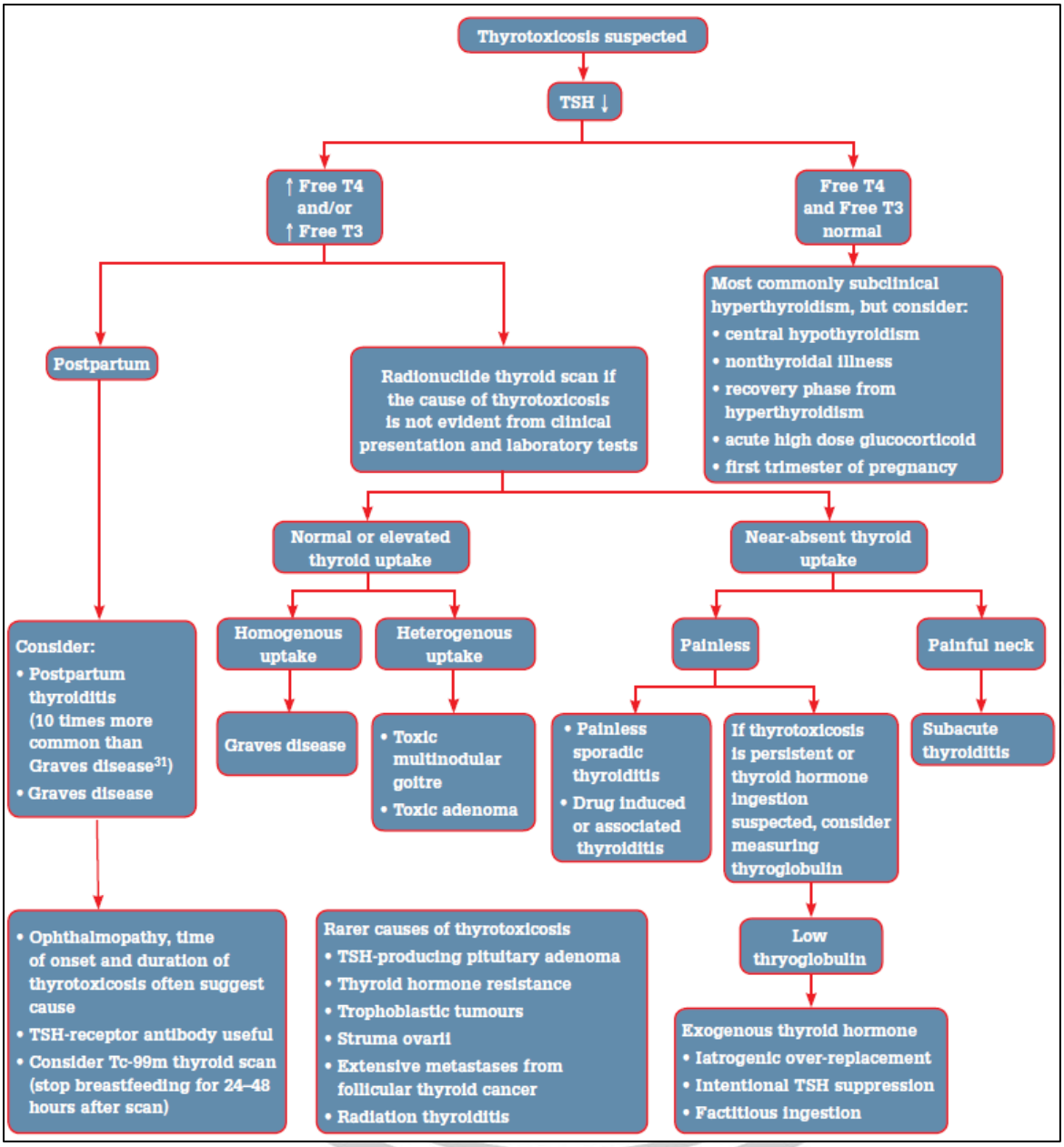



Figure 6.7. Acute Causes of thyrotoxicosis

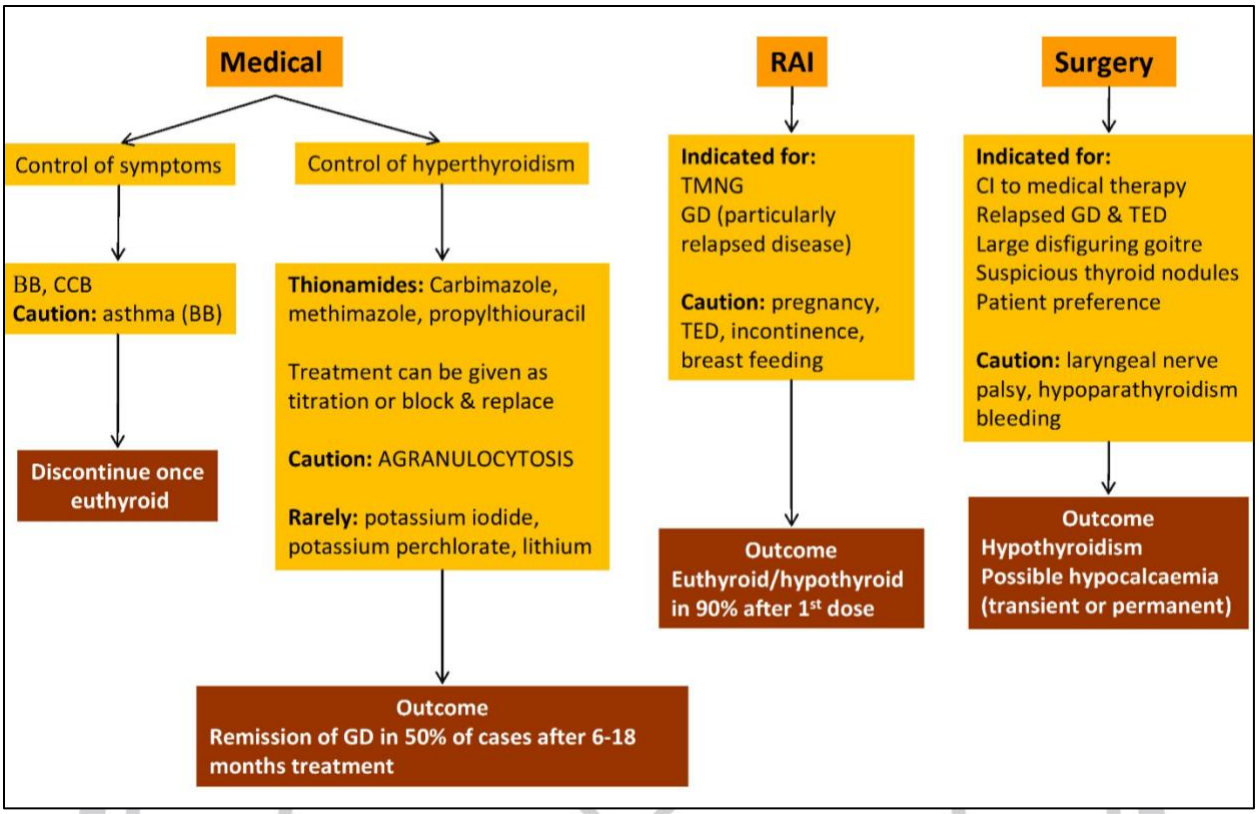



Figure 6.8. Treatment options for thyrotoxicosis

## Myxedema coma

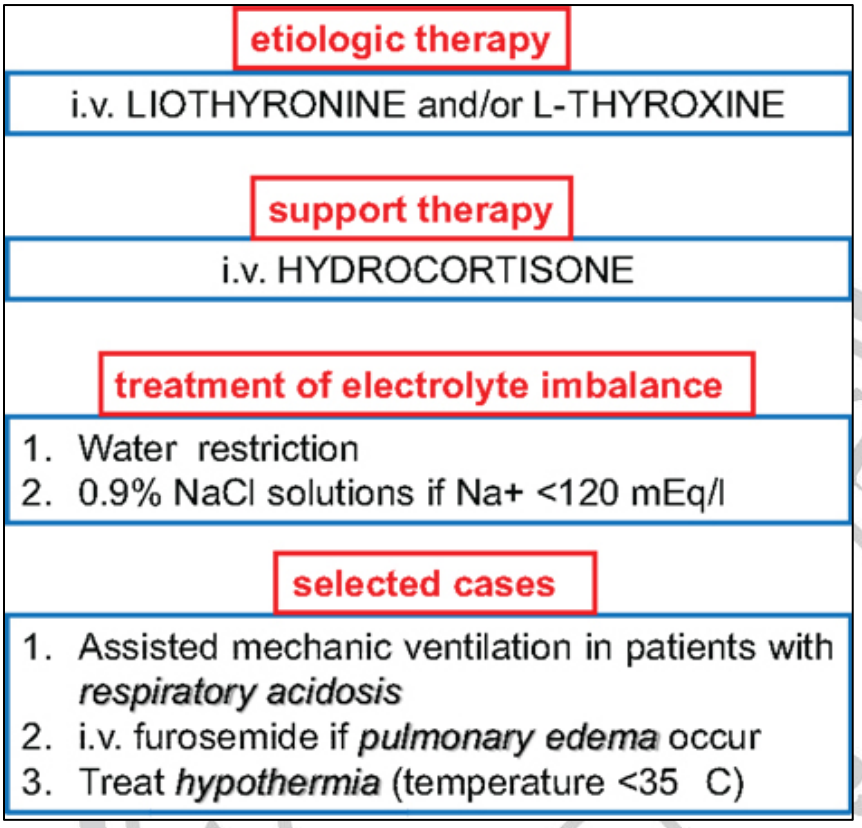



Figure 6.9. Acute management of Myxedema coma

# Adrenal Crisis

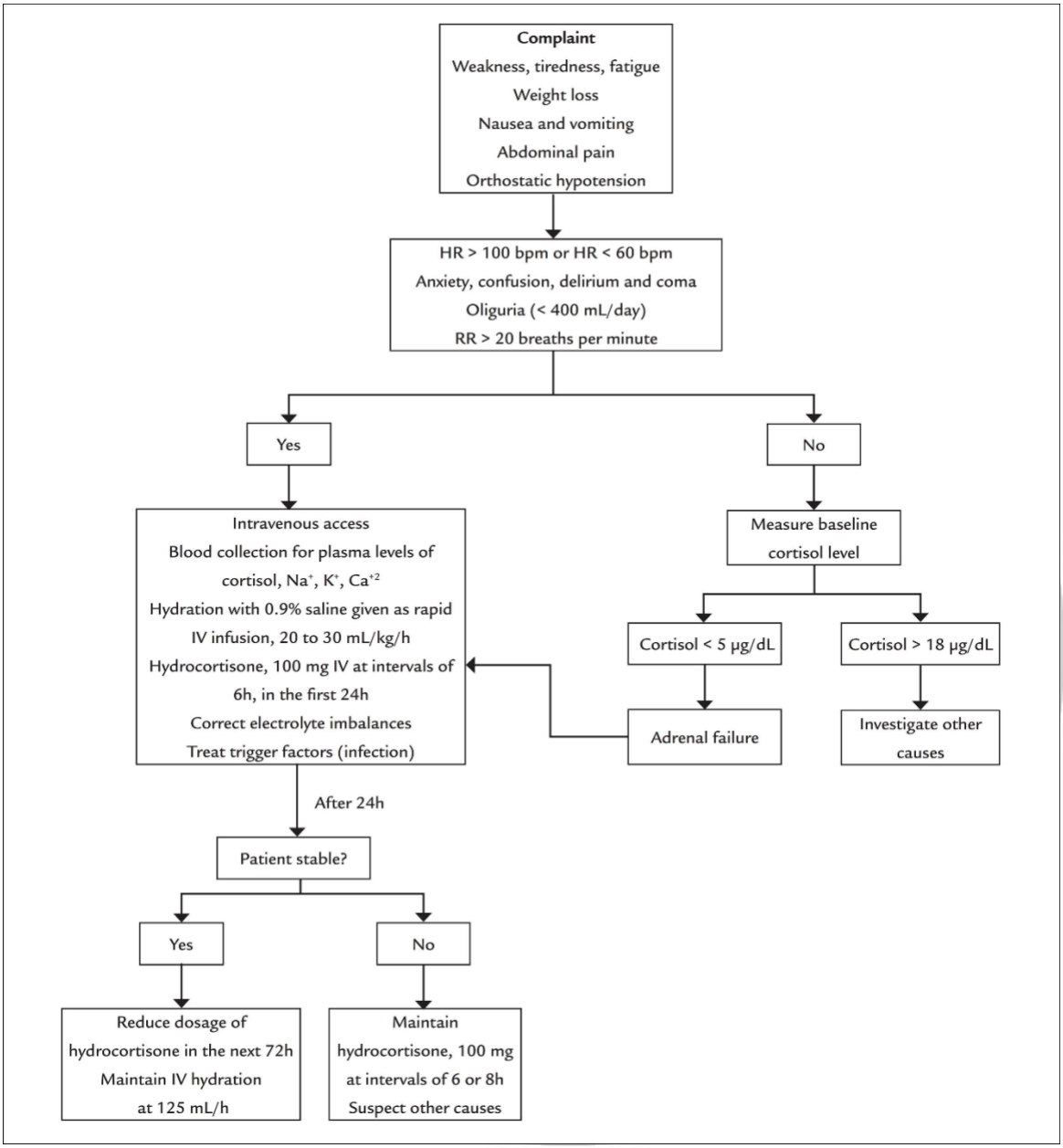



Figure 6.10. Acute management of adrenal crisis

Section 7

# Hematology and Oncology

# Neutropenic sepsis

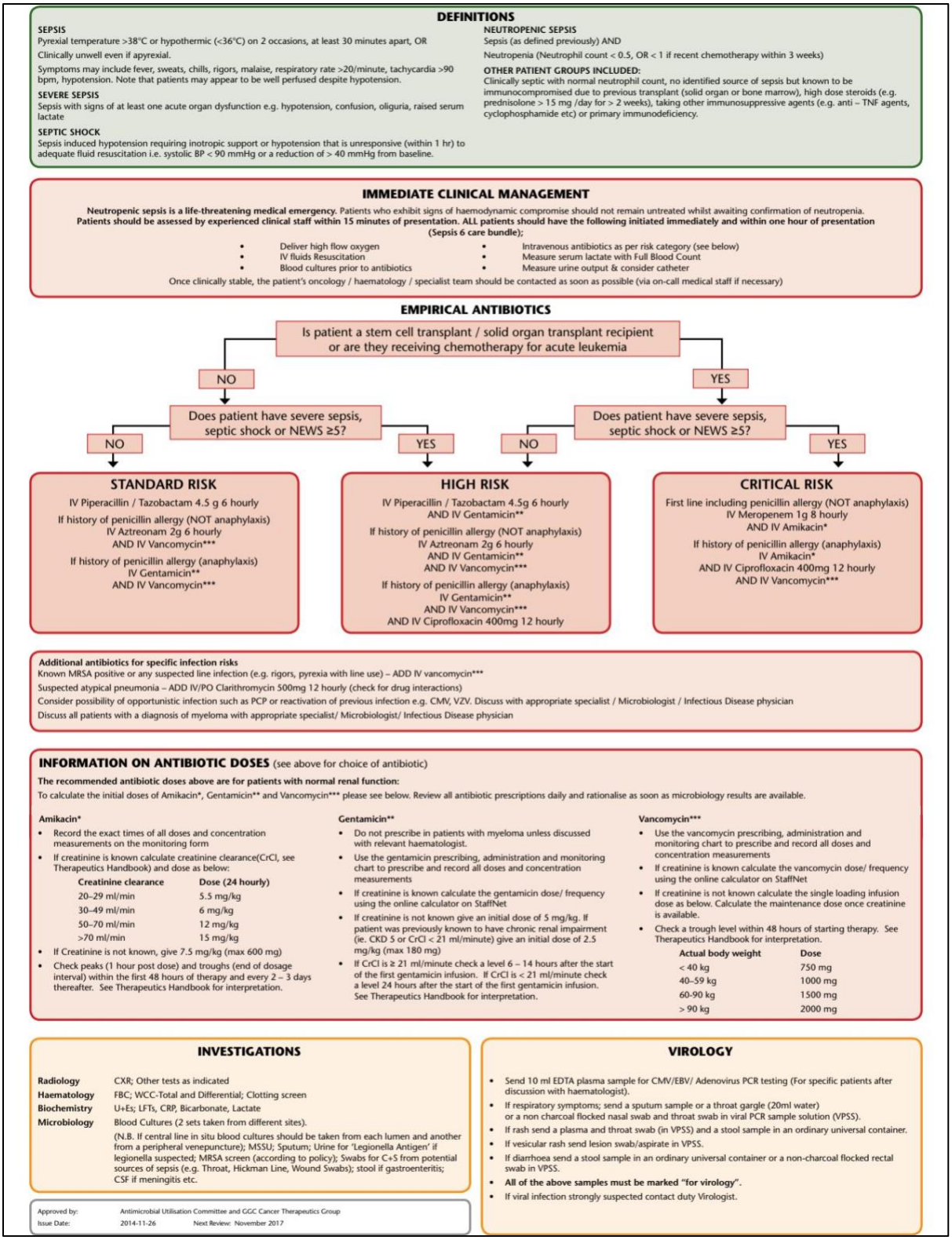



Figure 7.1. Acute management of Neutropenic sepsis

## Sickle cell crisis

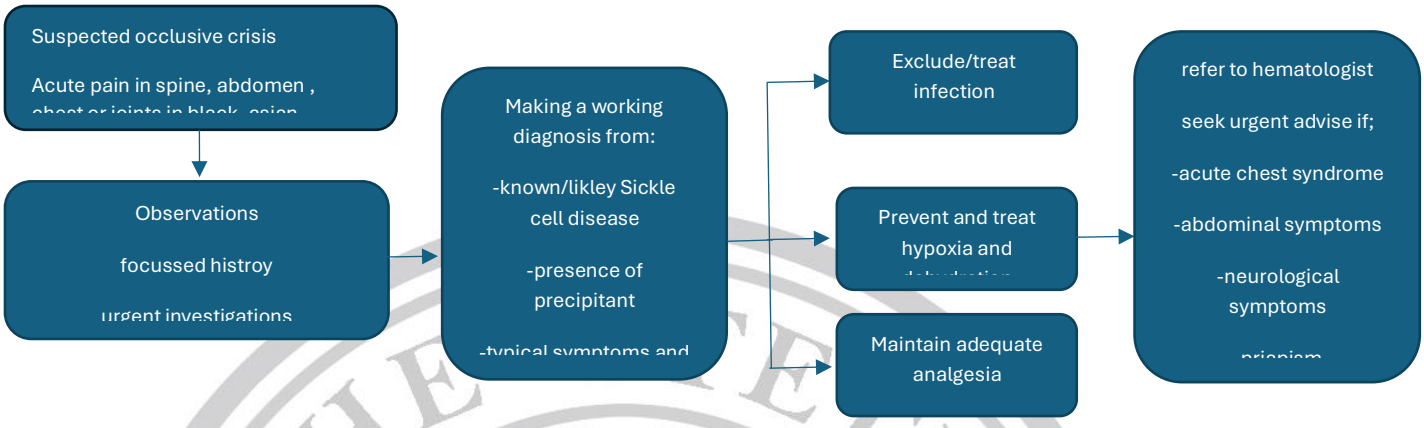



Figure 7.2. Acute management of sickle cell crisis

| Management | Actions | Details |
|---|---|---|
| **Initial Assessment** | - Access pain, monitor vitals | - Document pain, check for hypoxia, fever, and complications (acute chest syndrome, stroke). |
| **Pain Management** | - Rapid opioid administration | - Administer opioids (e.g., morphine) within 30 minutes. Use PCA for severe pain. |
| | - Paracetamol/NSAIDs as adjuncts | - Use paracetamol or NSAIDs with opioids, unless contraindicated. |
| | - Reassess pain regularly | - Adjust analgesia based on pain response. |
| **Hydration** | IV fluids if needed, avoid overhydration | - Use 0.9% saline if dehydrated or unable to drink. Avoid fluid overload. |
| **Oxygen Therapy** | - Oxygen if hypoxic (SpO2 < 95%) | - Administer supplemental oxygen if respiratory |

| | | |
|---|---|---|
| | | symptoms or hypoxia (SpO2 <95%) |
| **Monitoring** | - Frequent monitoring of vitals, watch for complications | - Monitor SpO2, HR, BP, RR every 2 hours initially. Look for signs of acute chest syndrome, stroke. |
| **Infection control** | - Investigate and treat infection | - Send cultures, start broad-spectrum antibiotics (e.g., ceftriaxone). |
| **Blood transfusion** | - Simple or exchange transfusion for severe cases | - Consider transfusion for patients with severe anemia (Hb <5g/dL), worsening ACS, stroke, or multiorgan failure. |
| **Anticoagulation** | - VTE prophylaxis | - Use low-molecular-weight heparin in hospitalized patients. |
| **Complication Management** | - **Acute Chest Syndrome (ACS)** | - Treat promptly with oxygen, antibiotics, and possibly transfusion. |
| | - **Stroke or neurological symptoms** | - Immediate neuroimaging (CT or MRI) and urgent exchange transfusion. |
| | - **Priapism** | - Treat urgently with hydration, analgesia, and urological consultation. Consider aspiration if persistent. |
| | - **Splenic sequestration** | - Urgent blood transfusion for splenic sequestration causing hypovolemic shock. |

Table 7.1. Management of sickle cell crises

## Anaphylaxis

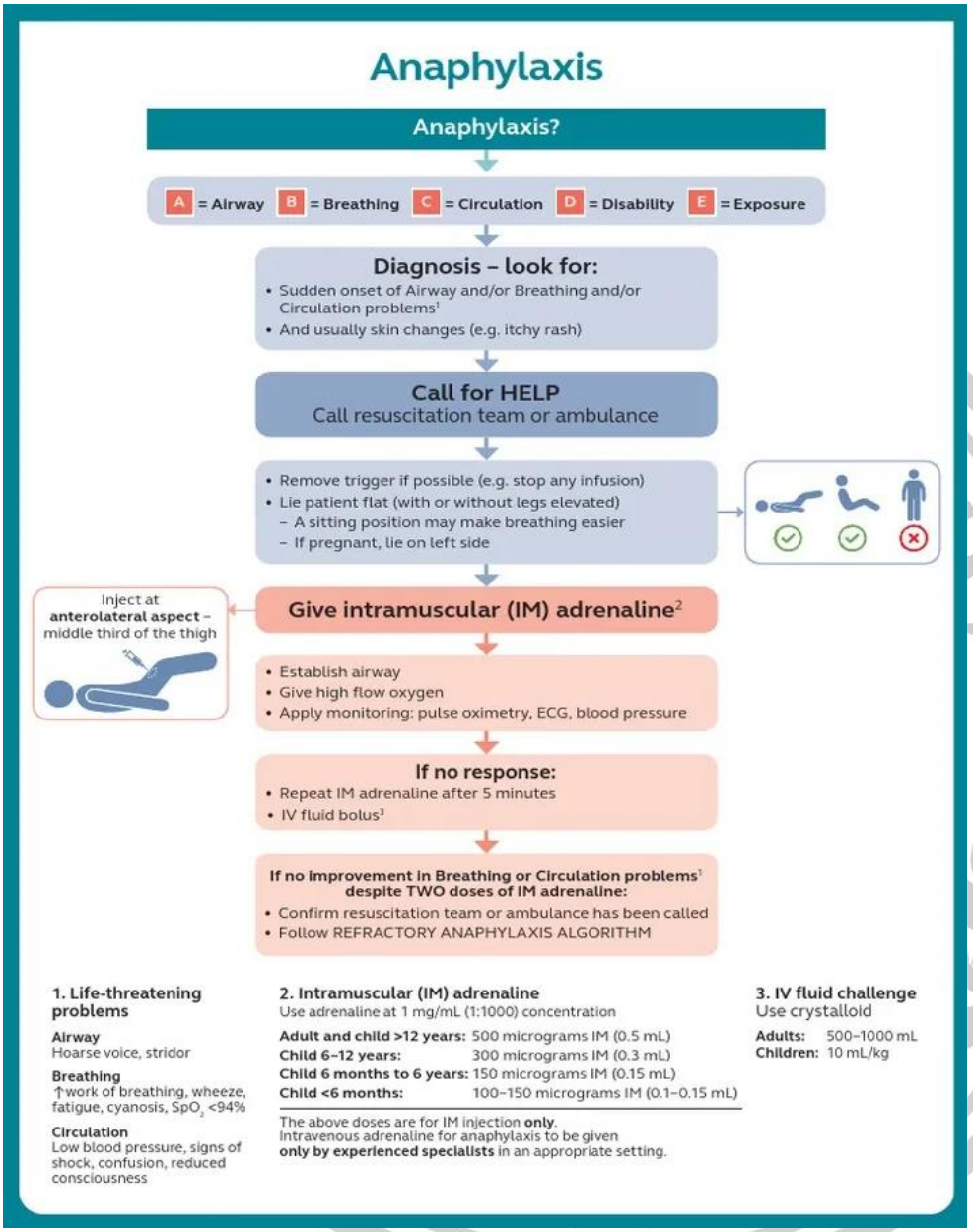



Figure 7.3. Resus UK- Acute management of Anaphylaxis

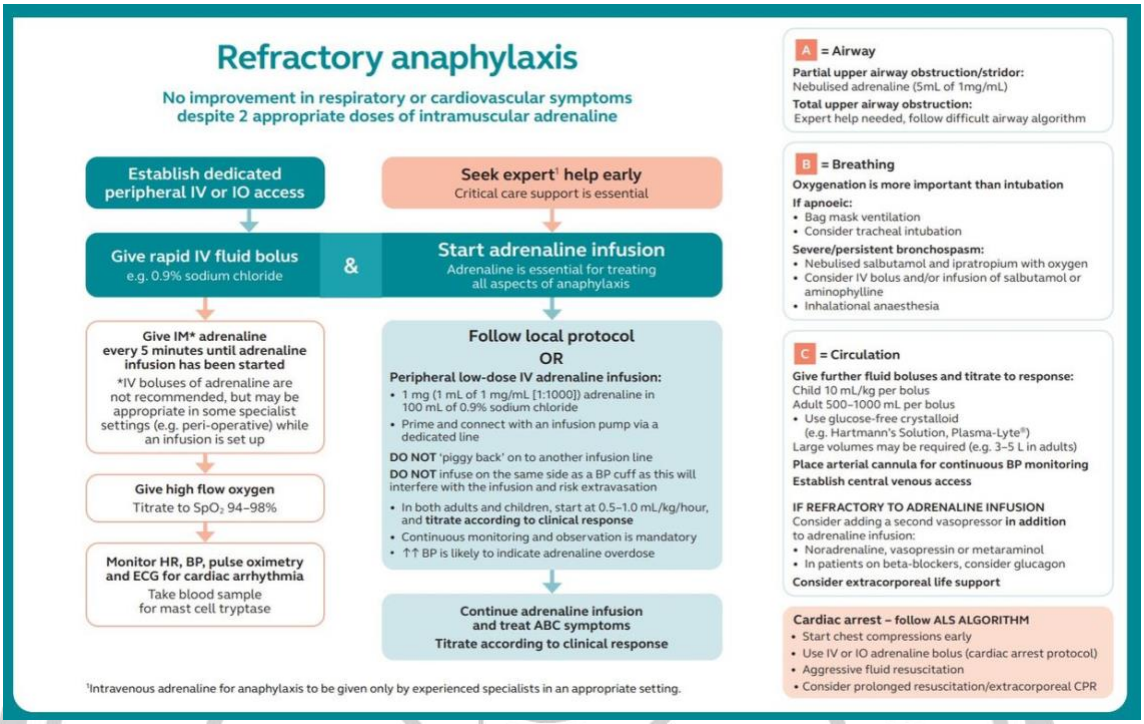



Figure 7.4. Resus UK- Acute management of Refactory Anaphylaxis

- Anaphylaxis is associated with the release of histamine and tryptase from the mast cells. Check the serum tryptase level within 6 hours of the attack for confirmation.

| Causes of Anaphylaxis |
|---|
| • **Foods**: Peanuts, tree nuts, shellfish, milk, eggs, soy, wheat.<br>• **Medications**: Penicillin, cephalosporins, NSAIDs, contrast dyes, biologics.<br>• **Insect stings**: Bees, wasps, hornets, fire ants.<br>• **Latex**: Gloves, medical equipment, balloons.<br>• **Exercise-induced anaphylaxis**.<br>• Unknown causes (idiopathic anaphylaxis).<br>• Blood products<br>• Vitamin K, chemotherapy agents |

Table 7.2. Causes of anaphylaxis

# Bibliography


- Acute Medicine- a practical guide to the management of medical emergencies, 4 ed; David sprignings, John B chambers.
- Advanced life support. Resuscitation Council, UK.
- National Institute for Health and Care Sciences Guidelines.
- Joint British Diabetes Societies guidelines
- British Thoracic Society Guidelines