INTERNATIONAL EDITION v2

The ANAESTHETIC CRISIS MANUAL

David C. Borshoff

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CRISIS PREVENTION

ENDORSED BY

European Society of Anaesthesiology ESA

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The Anaesthetic Crisis Manual (The ACM) is designed for use as a cognitive aid. It is not a substitute for experience, clinical acumen, or simulator visits, but it can provide immediately accessible guidance when used during a perioperative emergency.

In potentially stressful, time dependent conditions, even senior clinicians can forget important steps in treatment pathways. Inspired by the cockpit Quick Reference Handbook (QRH) concept of commercial aviation, the placement of The ACM in all anaesthetising locations provides welcome support for the team leader, reinforces crew resource management principles (CRM) and may ultimately improve patient outcome.

Embedding cognitive aids into crisis management culture continues to be a challenge, but incorporating the location of emergency manuals into the surgical safety checklist, delegating a reader early in a crisis event and teaching from the protocols will all contribute.

The ACM has proven to be a valuable resource when confronting the unexpected, and as more supportive evidence emerges for better outcomes, we anticipate its use becoming routine in anaesthetic crisis management.

David C. Borshoff

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The creation of this manual was inspired by Irma Brayley.

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CRISIS PREVENTION

CARDIAC ARREST SHOCKABLE

VF VT

Maintain CPR throughout. Aim to keep any pause < 5 seconds.

1. Check pulse and monitors to confirm diagnosis.
2. Start CPR 100-120 per minute and turn to 100% O_2.
3. Call for help, communicate and delegate.
4. Stop agent and secure airway while applying pads.
5. Shock and resume CPR without pausing for rhythm check.
6. Review 4Hs 4Ts and consider TTE at next changeover.
7. Shock at 2 minutes, resume CPR, give **1mg adrenaline IV**.
8. Shock at 4 minutes, resume CPR, give **300mg amiodarone IV**.
9. Shock every CPR cycle, and give epinephrine every 2nd cycle.
10. After the 5th cycle, give a further 150mg amiodarone.
11. Activate cardiac catheter lab or ECMO team as appropriate.

General principles: Organise the team so that pulse, rhythm checks and CPR changeovers are done at the end of each 2 minute CPR cycle. Counting out loud helps coordinate these activities.

CARDIAC ARREST SHOCKABLE

VF VT

1B

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CRISIS PREVENTION

Reversible Causes	4Hs 4Ts
Hypoxia	**Tension**
Hypovolaemia	**Tamponade**
Hypothermia	**Thrombosis**
Hypo/hyperkalaemia	**Toxins**

Use subxiphoid TTE or TOE to assist diagnosis.

Delegating staff members to chest compressions, crash trolley retrieval, documentation and airway management allows hands off leadership and helps situational awareness.

Always check airway placement, correct any hypoxia and ventilate at 10 normal tidal breaths/min. If intubating, perform quickly by the most experienced practitioner *after* starting compressions and preferably during the brief pause for rhythm checks.

Maintain minimally interrupted, high quality chest compressions and allow complete chest recoil. Don't lean on chest in between - it affects negative intrathoracic pressure and venous return.

Use $EtCO_2$ to confirm correct placement of airway device, check CPR effectiveness and monitor cardiac output. If there is a sudden spike in EtCO2 continue CPR until the end of the cycle and perform a brief rhythm check at the next changeover.

Aim for $EtCO_2$ > 20mmHg, diastolic pressure > 20mmHg and pauses < 5 - 10sec.
If $EtCO_2$ falls below 10mmHg, adjust technique and change CPR person each cycle. If using a manual defibrillator, continue CPR while machine charges to minimise 'pre-shock' pause in compressions. Use successive *stacked* shocks (up to 3) only for witnessed VF VT.

Shock energy Biphasic defibrillator: 120-200J first shock. Use same or greater for subsequent shocks. Monophasic defibrillator: 360J each shock.

All drugs should be administered via a peripheral or central venous line. If not possible, the tibial or humeral interosseous route is used.

Following return of spontaneous circulation (ROSC) implement targeted temperature management (TTM) 32-36. Avoid fever, hyperglycemia, hypercarbia and hyperoxemia.

Drug	IV Dose
Magnesium	1–2g over 3 minutes for Torsade de Pointes or hypomagnesemia.
Calcium chloride 10%	10mL for hyperkalaemia, hypocalcaemia or overdose of Calcium channel blockers. Repeat dose if indicated.
Sodium bicarbonate 8.4%	1-2mL/kg for hyperkalaemia and antidepressant overdose – not prolonged resuscitation.
Lignocaine	1mg/kg if amiodarone not available.

CARDIAC ARREST NON-SHOCKABLE

Asystole Pulseless Electrical Activity

Maintain CPR throughout. Aim to keep any pause < 5 seconds.

1. Check pulse, all monitors and confirm ECG rhythm.
2. Cease any vagal stimulus and turn to 100% O_2.
3. Call for help, communicate and delegate.
4. Start CPR 100-120/min and give **1mg adrenaline IV**.
5. Stop agent and consider intubation at next rhythm check.
6. Review 4Hs 4Ts and check ECG rhythm at CPR changeover.
7. Consider TTE during the brief interruption of CPR (< 5sec).
8. Give 1mg adrenaline IV every 2nd cycle.
9. If rhythm changes to VF VT, shock and go to **tab 01**.
10. In asystole, pace only for high degree heart block.
11. Activate cardiac cath lab or ECMO team as appropriate.

General principles: Organise the team so that pulse, rhythm checks and CPR changeovers are done at the end of each 2 minute CPR cycle. Counting out loud helps coordinate these activities.

CARDIAC ARREST NON-SHOCKABLE

Asystole Pulseless Electrical Activity

Reversible Causes	4Hs 4Ts
Hypoxia	**T**ension
Hypovolaemia	**T**amponade
Hypothermia	**T**hrombosis
Hypo/hyperkalaemia	**T**oxins

Use subxiphoid TTE or TOE to assist diagnosis.

Be aware of hypovolaemia, anaphylaxis or high spinal in the surgical setting. Maintain high index of suspicion for unrecognised haemorrhage, particularly with laparoscopic surgery.

Checking all monitors (ECG, ABP waveform, SpO_2 and $EtCO_2$) can rapidly distinguish artifact or transducer and calibration issues from asystole or PEA.

Always check airway placement, correct any hypoxia and ventilate at 10 normal tidal breaths/min. If intubating, perform quickly by the most experienced practitioner *after* starting compressions and preferably during the brief pause for rhythm checks.

Maintain minimally interrupted, high quality chest compressions and allow complete chest recoil. Don't lean on chest in between - it affects negative intrathoracic pressure and venous return.

Use $EtCO_2$ to confirm correct placement of airway device, check CPR effectiveness and monitor cardiac output. If there is a sudden spike in EtCO2 continue CPR until the end of the cycle and perform a brief rhythm check at the next changeover.

Aim for $EtCO_2$ > 20mmHg, diastolic pressure > 20mmHg and pauses < 5 - 10sec.
Adjust technique if necessary and change CPR compression person each cycle.

Correct electrolytes according to blood chemistry. See dosages tab 01.

Tamponade, tension pneumothorax and thromboembolic obstruction are all difficult to diagnose without significant knowledge of clinical history. Ultrasound imaging provides information that may assist diagnosis. A subziphoid view obtained during the brief rhythm check is recommended. Aim for normovolemia – in the absence of hypovolemia, excessive infusion of fluid should be avoided.

All drugs should be administered via a peripheral or central venous line. If this is not achievable, the tibial or humeral interosseous route is used until IV access established.

PAEDIATRIC ADVANCED LIFE SUPPORT

1. Check pulse, oxygenation and confirm ECG rhythm.*
2. Stop any vagal stimulus and turn to 100% O_2.
3. Call for help, communicate and delegate.
4. Start CPR at 100-120 minute.
5. If **VF VT** - shock (use 4 J/kg) and go to tab 01.
6. If **Asystole PEA** - give 10mcg/kg adrenaline IV.
7. Stop agent and consider intubation during rhythm check.
8. Review 4Hs4Ts. Check rhythm/output at CPR changeover.
9. Consider TTE during the brief CPR interruption (<5 secs).
10. Give adrenaline every 2nd cycle and keep reviewing 4Hs4Ts.
11. Activate extra-corporeal cardiopulmonary resuscitation when appropriate in hospitals with trained teams and protocols.

PAEDIATRIC ADVANCED LIFE SUPPORT

Reversible Causes	4Hs 4Ts
Hypoxia	**T**ension
Hypovolaemia	**T**amponade
Hypothermia	**T**hrombosis
Hypo/hyperkalaemia	**T**oxins

Use subxiphoid TTE to assist diagnosis.

Hypoxia, vagal stimulation and hypovolemia are the most frequent reversible causes in children. Excluding cardiac anesthesia, most arrests are asystole or PEA.

*Cardiac arrest in children is not usually from a primary cardiac cause. More commonly it is an asphyxial arrest from progressive respiratory failure or shock. Always check airway placement, immediately correct any hypoxia, then ventilate at 10 breaths per minute. Avoid simultaneous delivery by pausing slightly for each breath or inflating just after compression.

High quality compressions are: 1/3 AP diameter of chest with complete recoil, $EtCO_2$ > 20mmHg and diastolic pressure > 20mmHg. If EtCO2 falls below 10mmHg adjust technique or rotate CPR person.

Use $EtCO_2$ to confirm correct placement of airway device, check CPR effectiveness and monitor cardiac output. If there is a sudden spike in EtCO2 continue CPR until the end of the cycle and perform a brief rhythm check at the next changeover.

Paediatric doses

Adrenaline 10mcg/kg is given immediately in asystole or PEA and then every 3–5 minutes. Atropine is not recommended unless bradycardia associated with increased vagal tone (0.02mg/kg). For VF/VT, **adrenaline 10mcg/kg** and **amiodarone 5mg/kg** are both given after the 3rd and 5th shock via intravenous or intraosseous routes.

Lignocaine 1mg/kg bolus and 20-50mcg/kg/min infusion can be used as alternative to amiodarone. **Adrenaline 100mcg/kg** can be given via endotracheal tube only if unable to secure other routes.

Defibrillation

Use 4J/kg for each shock.
For AEDs, a **paediatric attenuated adult shock energy** should be used for those < 8yrs old.

INTRAOPERATIVE MYOCARDIAL ISCHAEMIA

1. Administer supplemental O_2 to maintain normal SpO_2.
2. Ensure adequate ventilation, anaesthesia and analgaesia.
3. Confirm ST changes with expanded monitor view or 12 Lead ECG.
4. If there is ST **elevation**, consult immediately with cardiologist and surgeon, and have defibrillator readily accessible.
5. If there is ST **depression**, aim to reduce O_2 demand while maximising O_2 supply.
6. If **hypotensive**, correct any volume loss and check Hb.
7. Adjust anaesthetic dose or titrate vasoconstrictor if vasodilated.
8. If **hypertensive**, titrate beta blocker and consider GTN infusion.
9. Control the heart rate aiming for 50-60 bpm.
10. When volume and BP are corrected, carefully titrate GTN.
11. Use invasive monitoring and echocardiography to assess RWMAs, contractility, and to optimise filling pressures.
12. If ischaemia persists, suspect non-ST elevation Acute Coronary Syndrome and seek urgent cardiology review.

INTRAOPERATIVE MYOCARDIAL ISCHAEMIA

Perioperative Myocardial Ischemia is caused by an imbalance in myocardial oxygen supply and demand. Can be due to atherosclerotic plaque rupture with vessel occlusion or inadequate coronary perfusion pressure.

Treatment is based on reducing O_2 demand an increasing the O_2 supply.

Treatment goals to reduce O_2 demand:
Control HR and treat any arrhythmia according to guidelines tab 29
Reduce shivering and fever
Afterload reduction

Treatment goals to increase O_2 supply:
Maintain MAP >75mmHg
Correct any relative or absolute volume deficit
Treat anemia and use supplemental oxygen to normalise SpO_2

In hypotensive patients, check Hb early and correct volume loss with blood if indicated. Adjustment of anaesthetic agent and cautious titration of vasoconstrictor can help correct anaesthesia induced vasodilation.

Filling pressure
CPP = ADP – LVEDP*
With severe coronary artery obstruction **distal coronary pressure** may be very low so avoid elevated LVEDP.

GTN will both dilate coronaries and reduce LVEDP.

Drug doses for 70kg patient:

Metoprolol	2.5 mg boluses
Esmolol	0.5mg/kg bolus 50–200mcg/kg/min infusion
Phenylephrine	25–50mcg bolus
Metaraminol	0.5–1mg bolus
GTN	50mg in 50mL 0.9% saline Commence at 3mL–5mL/hr and titrate to response.

* LVEDP = Left Ventricular End Diastolic Pressure
CPP = Coronary Perfusion Pressure
DBP = Diastolic Blood Pressure

SEVERE INTRAOPERATIVE HAEMORRHAGE

Practice damage control resuscitation and give blood products early

1. Call for help, communicate and delegate.
2. Rapidly assess bleeding and consider early activation of Massive Transfusion Protocol.
3. Review airway, consider intubation and increase FiO_2 to maintain adequate SpO_2.
4. Insert 2 large bore cannulas and consider an 8.5F CV PA sheath.
5. Confirm surgical effort to control bleeding and insert art line.
6. Warm fluids, warm theatre and warm patient.
7. Contact transfusion services early to plan component therapy.
8. Utilise the rapid infusor and cell saver, but avoid fluid overload.
9. Tolerate permissive hypotension (aim for MAP ≥ 65)* using vasopressors only to maintain vital organ perfusion.
10. Consider antifibrinolytic agents early.
11. Carefully monitor Calcium levels and keep > 1.1mmol/L.
12. Establish bedside monitoring of urine output, temp, CVP, Hb and coagulation but follow up with laboratory testing.

* MAP ≥ 80 in head injury.

SEVERE INTRAOPERATIVE HAEMORRHAGE

Nominate a communicator for interdepartmental phone calls, and a runner for blood sample and blood product transportation.

If time does not permit crossmatched blood (severe anaemia with ongoing bleeding), use O neg or group specific.

Keep the surgical team informed of haemodynamic status and prompt them if necessary for surgical control to allow catch up. It may require compression, packing, direct pressure, arterial or aortic clamping, or a combination of these.

Massive intraoperative hemorrhage will usually require activation of an MTP and a more balanced transfusion ratio (1:1:1 or 2:1:1) of blood, plasma and platelets. Early infusion of FFP (15mL/kg) may prevent impending haemostatic failure and microvascular bleeding. If fibrinogen <1g/L, and PT or aPTT > 1.5 normal, there is established haemostatic failure and larger volumes will be needed.

Consider using TEG® or ROTEM® if available for more rapid assessment as well as routine laboratory coagulation profile.

Haemodiluted, hypocalcaemic, cold patients don't clot so manage aggressively.

Aim for:
T > 35
pH > 7.2
BE < -6
Platelets > $75x10^9$/L
INR < 1.5
PT APTT < 1.5
Fibrinogen > 1g/L
Ca^{2+} > 1.1mmol/L
Lactate < 4mmol/L

Therapy	Indication	Initial dose
FFP	PT, aPTT >1.5 normal, fibrinogen <1g/L	15mL/kg
Cryoprecipitate	Fibrinogen <1g/L	1 unit per 5-10kg
Fibrinogen Concentrate	Fibrinogen <1g/L	25-50mg/kg
Prothrombin Complex Concentrate	Massive hemorrhage unresponsive to conventional therapy, coumarin reversal, FFP unavailable.	25-50 IU/kg
Factor VIIa	As above	90mcg/kg
Tranexamic Acid	Fibrinolysis	1gm IV over 10 min then 1gm over 8 hrs
Platelets	Platelet count <$75x10^9$	15–20mL/kg

Massive transfusion or critical haemorrhage: > 1 blood volume / 24hrs; > 1/2 blood volume / 4hrs; > 150mL/min.

ANAPHYLAXIS

1. Call for help, communicate and delegate.

2. Remove any suspected triggering agents.

3. **If cardiac output not detectable:** check ECG rhythm, start CPR and go to arrest protocol tab 01 or tab 02.

4. **If cardiac output detectable:** Stop procedure, reduce agent, consider early intubation and turn to 100% O_2.

5. Assess severity and give IM or IV adrenaline - see table.

6. Confirm large bore peripheral IV access first and then consider early arterial line insertion for samples and monitoring.*

7. Infuse at least 20mL/kg IV fluid. Larger volumes and a second IV line may be necessary.

8. If signs persist, start an adrenaline infusion but also review 4Hs 4Ts for other possible missed diagnoses.

9. If still no response, consider using additional agents.

10. Take tryptase samples at 1, 4 and 24hrs, start adjunctive therapy once stable and organise referral to allergy clinic.

ANAPHYLAXIS

Common triggers: muscle relaxant, antibiotics, latex, chlorhexidine, IV contrast.
May require early intubation if there is airway edema or respiratory compromise.

Clinical Signs of Anaphylaxis	
Mild (Grade 1)	Generalised mucocutaneous signs: erythema, urticaria +/- angioedema
Moderate (Grade 2)	• hypotension, tachycardia • evidence of bronchospasm, cough, difficult ventilation • mucocutaneous signs
Life Threatening (Grade 3)	• severe hypotension • bradycardia or tachycardia, arrhythmias • severe bronchospasm, and/or airway oedema • cutaneous signs may be absent, or present only after correction of hypotension
Arrest (Grade 4)	Cardiopulmonary arrest

IV Adrenaline BOLUS Give dose below every 1-2 minutes prn			No IV Access† Give IM lateral thigh 5 minutely prn
	Grade 2	**Grade 3**	**Grade 2 & 3**
ADULT 1mg in 10mL (100mcg/mL)	20mcg (0.2mL)	100–200mcg (1-2mL)	>12yrs 500mcg
PAEDIATRIC 1mg in 50mL (20mcg/mL)	2mcg/kg (0.1mL/kg)	4–10mcg/kg (0.2-0.5mL/kg)	<6yrs 150mcg 6-12yrs 300mcg

Start adrenaline infusion 3mg/50mL saline at 3mL/hr (0.05–0.5mcg/kg/mi) and titrate.
† IM route also used if no haemodynamic monitoring. Scale up for 100/250mL infusion bags.

If poor response to conventional therapy

Noradrenaline	0.1mcg/kg/min
Vasopressin	2 IU/hr or 2 IU bolus
Glucagon	1-2mg over 5min for beta blocker reversal

For resistant bronchospasm review differential diagnosis **tab 14**.
For resistant hypotension review differential diagnosis **tab 28**.
When haemodynamically stable consider steroids and oral 2nd generation antihistamines (parenteral not recommended).

* A CVC may be indicated if peripheral IV access is difficult or ongoing infusions are required.

HAEMOLYTIC TRANSFUSION REACTION

1. Cease transfusion of the blood product and change IV tubing.
2. Call for help, communicate and delegate.
3. Titrate FiO_2 to maintain adequate SpO_2.
4. Treat hypotension with fluids and vasoactive agents.
5. Insert arterial line for ABGs and monitoring.
6. Once haemodynamically stable consider CVC and IDC insertion.
7. Maintain urine output – use diuretic therapy.
8. Treat any developing coagulopathy in consultation with transfusion services. Use the table in tab 05 for guidance.
9. Return all products to blood bank and take fresh blood and urine samples for analysis.
9. Prepare for ICU admission.

HAEMOLYTIC TRANSFUSION REACTION

Signs in the anaesthetised patient include:

Hypotension	Wheeze	Cola-coloured urine
Tachycardia	Tachypnoea	Bleeding (membranes, infusion sites)
Bronchospasm	Urticaria	Cardiovascular collapse
Hypoxia	Oedema	

Although rare, this carries significant mortality.
Staff should be informed immediately and the blood rechecked against the patient.
More blood should be taken for further testing.

Treatment is directed towards circulatory support, alleviating respiratory symptoms and anticipating and treating coagulopathy (see also Anaphylaxis **tab 06**, Major Haemorrhage **tab 05** and Bronchospasm **tab 14** protocols).

Diuretics and inotropic support should be commenced to maintain urine output of 0.5 to 1.5mL/kg/hr.

Treatment of any developing coagulopathy should be directed by the coagulation profile (see table on **tab 05** for guidance).

All products should be returned to transfusion for further analysis.

Drug doses

Mannitol 25%	0.5g–1g/kg IV
Frusemide	0.5mg/kg IV
Methylprednisolone	1–3mg/kg IV

Drug	Adult	Paediatric
Adrenaline	3mg/50mL saline (60mcg/mL)	0.05–0.5mcg/kg/min
Dobutamine	250mg/50mL saline (5mg/mL)	2–20mcg/kg/min
Noradrenaline	4mg/50mL saline (80mcg/mL)	0.02–1.0mcg/kg/min

In a 70kg adult, infusions can be commenced at 5mL/hr and then titrated to response.
Dilutions are given for syringe drivers. Scale up for 100mL or 250mL infusion bags.

VENOUS AIR EMBOLISM

1. Call for help, communicate and delegate.

2. Ventilate with 100% O_2. Avoid Nitrous Oxide.

3. Prevent further entrainment of gas - see opposite.

5. Place the patient in a head down, left lateral position.

6. Aspirate the CVC and start closed cardiac massage.

7. Increase IV fluid therapy and keep well hydrated.

8. Use **adrenaline** for haemodynamic support.

9. Consider **hyperbaric oxygen therapy** and ICU following successful resuscitation.

VENOUS AIR EMBOLISM

Signs during anaesthesia include:

↓ SpO_2
↓ $EtCO_2$
Hypotension
Pulmonary oedema
Elevated CVP
Tachycardia
Bronchospasm
Elevated PA pressure
Air on cardiac TTE or TOE
Cardiovascular collapse

Methods to stop entrainment:

Flood the field
Temporarily occlude vessel
Cease insufflation
Decompress pressurised systems
Use bone wax
Place operative site below heart

If there is significant hemodynamic compromise, intubate if ETT not already in place. Hyperventilate with 100% O_2 and consider PEEP only to support oxygenation. Initially thought to help prevent venous air embolism PEEP may increase the risk of paradoxical air embolism, impair venous return and its sudden cessation can enhance entrainment.

Aspirate if a CVC is in situ, but emergent placement is not evidence based.

Closed cardiac massage has been shown to break up large volumes of air in the cardiac chambers. As little as 0.5mL of air in the coronary arteries can precipitate ventricular fibrillation.

Hyperbaric oxygen up to 6 hours (possibly more) following the event should be considered in large **paradoxical** air embolism – a patent foramen ovale is present in 10–30% of the population.

Risk Factors for Venous Air Embolism

Surgical	Anaesthetic	Patient
Sitting craniotomy Posterior fossa surgery Spinal surgery Shoulder surgery Laparoscopic surgery (CO_2 embolism) Caesarean section Exteriorisation of the uterus	Central venous access Pressurised infusions Non-primed giving sets Unrecognised epidural vein cannulation	Trauma (blunt or penetrating) Hypovolaemia

Source: www.wfsahq.org

Prevention

Position to minimise pressure gradients, keep well hydrated, avoid N_2O and outline high risk surgical manoeuvres in pre-op surgical/anaesthetic team briefing.

The use of positive pressure ventilation, end tidal monitoring, central venous or pulmonary catheters, precordial doppler and transesophageal echo in high risk procedures can lead to early diagnosis and treatment.

DIFFICULT MASK VENTILATION

1. Check circuit integrity and use high flow 100% O_2.
2. Call for help, communicate and delegate.
3. Optimise ventilation attempts using actions listed opposite.
4. If ventilation is difficult or only partially achieved, stop and think through options including waking the patient.

If there is no improvement associated with good filling of reservoir bag, good facemask seal, high circuit pressure and difficulty emptying reservoir bag on attempted ventilation then consider **Laryngeal Spasm** tab 12, or other causes of **Elevated Airway Pressure** tab 13.

If there is no improvement associated with rapid deflation of reservoir bag, poor refilling, low circuit pressure and ongoing facemask leak, **go to step 5**.

5. Ensure anaesthesia deep enough to facilitate airway control.
6. Attempt up to 3 SGA insertions - try different size and type.
7. Revert to attempting FMV while preparing for intubation.
8. If FMV fails again mobilise CICO resources. ***READY***
9. Confirm good muscle relaxation and attempt intubation.
10. If unsuccessful, move to difficult intubation protocol tab 10 and have CICO rescue equipment open and accessible. ***SET***

This can be used as a realtime prompt or as a drill to regularly rehearse.

DIFFICULT MASK VENTILATION

This protocol is based on the assumption of a recently checked anaesthetic machine and an intact circuit with fresh gas flow – **confirmed by preoxygenating the patient and witnessing a visible $EtCO_2$ trace.** In practice, these conditions may not always be met. In difficult mask ventilation when machine or circuit is suspected, removing them and using a self-inflating resuscitator is the most time efficient method of excluding these possibilities.

The call for help is placed early in this sequence as it assists in hands off leadership and helps maintain situational awareness.

Time management and decision making during airway crises is vital to a positive outcome. It can be complicated by partial ventilation, borderline saturations, the arrival of another clinician, equipment delays, drug onset times and fixation error. Delegating a staff member the role of calling elapsed time intervals as well as monitoring SpO_2 and $EtCO_2$ may prevent prolonged hypoxia.

To maximise conditions for FMV:

• Head position	neck flexion head extension (sniffing).
• Two person technique	two handed jaw thrust while second person squeezes bag.
• Oral/nasal airway	opt for larger size.
• Mouth opening	ensure adequate anaesthetic depth/muscle relaxation.
• Perioral area	remove any moisturiser or airway lubricant to allow firm grip on face and mask.

Waking the patient may be an option if factored into the anaesthetic plan e.g. gaseous induction or gradual onset TIVA to test ventilation in suspected difficult airway. However, if difficulty was not anticipated and the patient received a **full anaesthetic induction dose**, the anaesthetist may be committed to securing the airway.

Ensuring adequate anaesthesia is a prerequisite for mask ventilation and LMA insertion. Preserved airway reflexes, insufficient anaesthetic depth and inadequate muscle relaxation will diminish the likelihood of success. Another attempted FMV is suggested prior to intubation as chances of success maybe greater after deepening of anaesthesia (not always established for initial FMV following induction).

Always preoxygenate the patient before induction if possible.

SGA = supraglottic airway
FMV = facemask ventilation
READY, SET, GO = The Vortex Approach to priming for CICO rescue

UNANTICIPATED DIFFICULT INTUBATION

If oxygenation is established at any stage in supraglottic rescue, stop and think through options, including waking the patient.

1. Call for help, communicate and delegate.
2. Revert to FMV, get difficult airway trolley, and consider options.
3. Ensure adequate anaesthetic depth and muscle relaxation.
4. Monitor $Sp0_2$, $EtCO_2$ and elapsed time until rescue complete.
5. Try the manoeuvres listed opposite to maximise laryngeal view and utilise adjuncts like bougie, introducer or stylet.
6. Allow up to 3 optimised intubation attempts and consider videolaryngoscopy +/– hyper-angulated/low profile blade.
7. If unsuccessful, mobilise resources for CICO rescue. ***READY***
8. Attempt up to 3 SGA insertions – try different size or type.
9. If unsuccessful, have CICO equipment open and ready. ***SET***
10. If following complete muscle relaxation and an FiO_2 of 1:
    - best effort at all 3 supraglottic rescue options fails
    - one final maximised FMV fails
    - SpO_2 is rapidly falling and $EtCO_2$ not detectable

    Optimise patient position and initiate CICO rescue tab 11. ***GO***

UNANTICIPATED DIFFICULT INTUBATION

There is overlap of the **Difficult Mask Ventilation** and **Difficult Intubation** protocols, as each technique uses the other as a rescue option. This is also highlighted in The Vortex Approach to Airway Management.

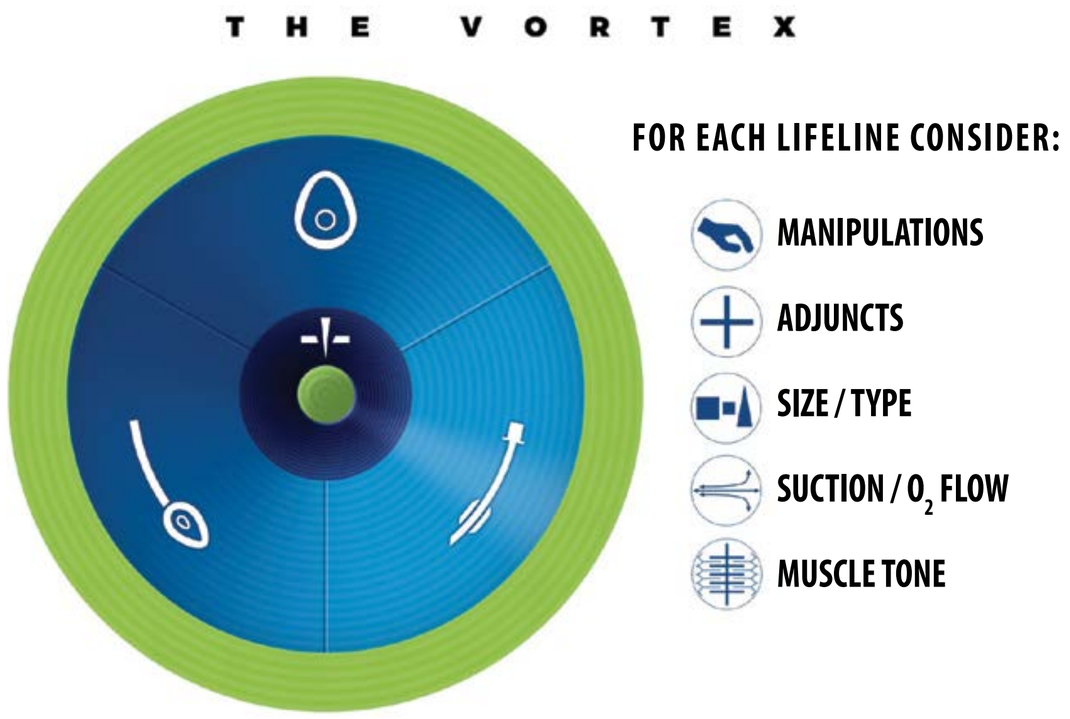
Rescue Options

Can't bag and mask

- Wake the patient if possible
- Use SGA
- Intubate

Can't intubate

- Wake the patient if possible
- Bag and mask
- Use SGA



To maximise laryngeal view:

- Neck flexion head extension
- Adjust cricoid pressure
- Use external manipulation
- Try long or straight blade
- Use video laryngoscope

Delegating someone to monitor SpO_2, $EtCO_2$ and elapsed time can prevent both profound hypoxia and the development of fixation error with multiple intubation attempts converting a can't intubate ***can*** oxygenate to a can't intubate ***can't*** oxygenate emergency (CICO).

Given the variability in difficult airway scenarios, familiarity with the principles underlying the decision making process is a prerequisite for safe practice. SGA insertion after muscle relaxation may improve rescue success rate.

Once facemask or SGA ventilation of the lungs is established, the airway can be secured by an increasing number of techniques available. The clinician should use that which is most familiar and likely to be successful in the particular clinical circumstance.

Depending on type and availability, many experienced clinicians would use videolaryngoscopy (VL) immediately. Hyper-angulated and low profile blades, combined with skillful bougie manipulation can significantly improve the chance of success.

SGA = supraglottic airway
FMV = facemask ventilation
READY, SET, GO = The Vortex Aproach to priming for CICO rescue

Chrimes N. The Vortex: a universal 'high-acuity implementation tool' for emergency airway management. *Br J Anaesth* 2016; **117 Suppl** 1: i20-i27

VortexApproach.org

CAN'T INTUBATE CAN'T OXYGENATE

Cricothyroidotomy | Cannula Technique

1. Palpate anatomy and use cricothyroid membrane if easily identified - if not, go lower down and aim for tracheal midline.

2. Puncture using syringe cannula combination and continuous aspiration.

3. Stabilise the trocar, slide cannula into trachea, confirm position by aspirating full length of syringe and release plunger to check for recoil.

4. Attach either a jet ventilator set to 1 Bar (14.5 psi) or a flow regulated insufflation device set to 15L/min.

5. Inflate for 4 secs and then wait 30 secs for SpO_2 response.

6. Use subsequent 2 second inflations titrated to SpO_2.

7. Observe chest expansion and confirm exhalation through upper airway, monitoring for autoPEEP.

8. If unsuccessful proceed to surgical cricothyroidotomy.

Recommendations:
Choose a 14kg kink resistant cannula.
Use a 5mL syringe with saline.
Be familiar with high pressure ventilation to reduce risk of barotrauma.
Attend advanced airway workshops for hands on experience.

This is not a checklist but a guideline to regularly rehearse.

CAN'T INTUBATE CAN'T OXYGENATE

Cricothyroidotomy | Scalpel Bougie or Surgical

Anatomy palpable – scalpel bougie technique

1. Make a transverse stab incision through the cricothyroid membrane using a scalpel with size 10 blade.
2. Rotate the scalpel 90° and apply sideways pressure.
3. Insert bougie alongside the scalpel blade to guide into trachea.
4. Railroad a 6mmID ETT or tracheostomy tube over bougie.*
5. Ventilate from standard source and confirm with $EtCO_2$.
6. If unsuccessful proceed to surgical cricothyroidotomy.

Anatomy not palpable – surgical cricothyroidotomy

1. Make a 6-8cm vertical neck incision from sternal notch.
2. Blunt finger dissect strap muscles and expose trachea.
3. Proceed with cannula or scalpel bougie technique.†

* Some airway groups suggest a ventilating bougie with appropriate adaptor allowing immediate oxygenation using standard circuit or self-inflating resuscitator.

† Anaesthesiology institutions differ in their support of one or both techniques.

LARYNGOSPASM

1. Turn to high flow **100% oxygen**.
2. Cease all stimulation.
3. Remove airway devices and suction.
4. Use jaw thrust, apply gentle CPAP and consider oral airway.

If spasm persists and desaturation continues,

5. Call for help, communicate and delegate.
6. Deepen anaesthesia.
7. Give **suxamethonium** and continue CPAP.
8. If SpO_2 does not improve, secure the airway with intubation.
9. Consider IV atropine for the treatment of associated bradycardia.

This is not a checklist but a guideline to regularly rehearse.

LARYNGOSPASM

Although stimulating airway devices contributing to spasm should be removed, the **Guedel (oral) airway** may be helpful in providing CPAP.

Call for assistance early. The situation deteriorates rapidly in children.

Delegate responsibilities clearly including ETT preparation and suxamethonium administration. Some practitioners have **pre-drawn suxamethonium** easily accessible to save time during desaturation (and may reduce drug error).

Deepening anaesthesia is an option in adult anaesthesia.
Rapid development of hypoxia in children usually precludes this.

Spasm will 'break' with sufficient hypoxia and time, but predisposes to bradycardia, cardiac arrest, regurgitation and pulmonary oedema. These can be prevented with early intervention.

Drug dose:	suxamethonium	0.1-1mg/kg IV
		2-4mg/kg IMI/IL/IO
	atropine	10-20mcg/kg IV

In complete obstruction, forced inflation attempts will add to obstruction (Fink ball/valve effect) and inflate the stomach.

Consider stomach deflation before emergence.

In a rapidly desaturating child, immediate intubation without relaxation may be the appropriate treatment.

IL = Intralingual
IO = Intraosseous

ELEVATED AIRWAY PRESSURE

1. Manually ventilate to confirm high pressure, examine the $EtCO_2$ waveform and check the airway for any obvious change.
2. Exclude light anaesthesia and inadequate muscle relaxation.
3. Perform a systematic circuit, valve and ventilator inspection.
4. If autoPEEP suspected, look for elevated end-expiratory alveolar pressure or persistent expiratory flow when next breath commences.
5. Replace circuit with a self inflating resuscitator connected **directly** to the airway device.
6. Check airway position and patency – suction the full length of the tube and use a bronchoscope to confirm.
7. If pressure remains high, examine patient's respiratory system and consider calling for assistance.
8. If any doubt, **replace the airway**. If ventilating with an LMA, consider replacing with an ETT.
9. Review checklist of most common patient causes opposite.

ELEVATED AIRWAY PRESSURE

Immediately assessing $EtCO_2$ trace may provide an early answer to the pressure rise e.g. muscle relaxation or bronchospasm. Checking muscle relaxation eliminates the most likely cause. In the unintubated patient this is commonly **laryngospasm**.

If eliminating inadequate relaxation does not correct the rise in pressure, systematically working through the steps should determine the problem.

While hand ventilating, check all tubes, valves, connections and filters. Check the machine position to exclude tube kinking or obstruction.

Once the circuit is removed and replaced with a self-inflating resuscitator, if the pressure remains high, the problem is isolated to the airway or patient.

The airway should be: checked for position and patency
suctioned down its full length
assessed with bronchoscope
replaced if the problem is unresolved

A chest examination should be performed before undertaking airway replacement.
If there is no change following these procedures (new airway and new circuit), the problem must then be with the patient.

Consider:

Laryngospasm	Pneumothorax
Bronchospasm	Haemothorax
Oedema	Chest wall rigidity

For a more comprehensive diagnostic checklist of Elevated Airway Pressure see **tab 33**.

Although calling for help is not first in this sequence, it could occur at any stage the practitioner feels appropriate.

Always consider the timing of the event.
Check for recent CVC insertion, drug administration, tube adjustment, position change, pneumoperitoneum or surgical intervention.

SEVERE BRONCHOSPASM

1. Call for help, communicate and delegate.
2. Review airway placement and patency.
3. Hand ventilate, switch to 100% O_2 and deepen anaesthesia.*
4. Utilise in-circuit salbutamol and ipratropium bromide.
5. Monitor progress with EtCO2 waveform and airway pressures.
6. Set I:E ratio for long expiratory phase, intermittently disconnect and use low pressure PEEP to reduce hyperinflation.
7. Consider IV fluids, arterial line and serial ABGs.
8. Give adrenaline (or salbutamol) as IV boluses but commence an infusion if spasm persists.
9. Use hydrocortisone (or methylprednisolone) and magnesium as adjunctive therapy.
10. Depending on the severity and the response to therapy, consider need for surgery to continue and contact ICU.

SEVERE BRONCHOSPASM

Hand ventilating allows for immediate assessment of compliance and severity of spasm.

The causes of bronchospasm include: exacerbation of asthma
anaphylaxis
airway malposition
foreign body
aspiration

Drug doses

Adrenaline bolus	0.1-1.0mcg/kg titrated to haemodynamics
Adrenaline infusion	0.1mcg/kg/min
	Note that with 3mg in 50mL, rate in mL/hr = mcg/min
	Therefore commence at 7mLs/hr for a 70kg man

Use an arterial line and serial ABGs to guide management.

Adult

Salbutamol IV bolus	250mcg over 5 min
Salbutamol infusion	200mcg load over 1 min then 5-25mcg/min

Children 2–12 years

Salbutamol infusion	Loading dose of 5 mcg/kg/minute (maximum 200 mcg/minute) for 1 hour and then infusion of 1–2 mcg/kg/minute (maximum 80 mcg/minute)

There is little evidence to support the use of salbutamol over adrenaline.
Dosages vary between guidelines.

Intermittent disconnection allows CO_2 escape and prevents hyperinflation and autoPEEP.
Hand ventilation with permissive hypercapnia may also be required to avoid the complications of high airway pressure ventilation.

Treatment can be assessed by haemodynamic parameters, airway pressure, ABGs and the CO_2 waveform. With resolution, the sloping upward trace of $EtCO_2$ returns to normal with the more horizontal alveolar plateau.

* Deepening anaesthesia contributes to smooth muscle relaxation of airways and is helpful in exacerbation of asthma. Delay using if anaphylaxis is not excluded or hemodynamics don't permit.

ASPIRATION

1. Call for help, communicate and delegate.
2. Place head down and consider left lateral position.
3. Remove the airway and suction the pharynx.
4. Intubate and suction the bronchial tree.
5. Ventilate with 100% O_2 then titrate to normal SpO_2.
6. If severe aspiration, proceed only with emergencies.
7. Empty stomach before emergence.
8. Consider ICU/HDU admission.

ASPIRATION

In The Royal College of Anaesthetists fourth National Audit Project (NAP4) over 50% of airway related deaths in anaesthesia were a consequence of aspiration.

How much assistance required depends on the severity and circumstances. Mild regurgitation in a fasted patient may be managed simply with suction only, but more significant aspiration needs immediate aggressive management.

Immediate communication with the surgeons and delegation of tasks (e.g. turning the patient) may limit the amount of aspiration. Positioning the patient will depend on the type of surgery and practical limitations.

Steps 1 to 4 should be achieved *before* step 5 if SpO_2 permits.

Cricoid pressure can be used during intubation but not during active vomiting or regurgitation.

Mild aspiration often resolves without specific treatment, and at 2 hours post aspiration if the patient is not symptomatic, the chest X-ray is clear and the SpO_2 is normal, ICU can be avoided.

However, if there is **particulate matter**, indicative of more severe aspiration, ICU will be required for post-op management.

Steroid and antibiotic therapy are not indicated in the short term management of aspiration. Antibiotics are used only if pneumonia develops. Steroids have shown no effect on outcome or mortality.

Some evidence suggests that residual neuromuscular block significantly reduces **upper oesophageal sphincter tone** for a significant time after emergence, increasing the risk of aspiration during the recovery phase.

Risk Factors		
Patient	**Surgery**	**Anaesthetic**
Inadequate fasting Delayed gastric emptying Incompetent Lower Oesophageal Sphincter (Emergency, Pregnancy, Diabetes, Obesity, Hiatus Hernia, GORD)	Upper GI tract Lithotomy Head down Laparoscopy Cholecystectomy	Light anaesthesia Supraglottic airways PPV Difficult airway Long surgery

TOTAL SPINAL

Treatment steps also apply to non pregnant patient.

1. Call for help, communicate and delegate.
2. Reassure the awake patient while working through steps.
3. Give 100% O_2 and provide gentle ventilatory support if needed.
3. If conscious state deteriorates, prepare to secure the airway.
4. Elevate legs, rapidly infuse IV fluids and apply LUD.
5. Use vasopressors to maintain blood pressure.
6. Give atropine +/- adrenaline for associated bradycardia.
7. If loss of cardiac output, start CPR, give 1mg of epinephrine IV and go to PEA protocol tab 02.
8. In the parturient, prepare and aim for delivery within 4 minutes if no ROSC.
9. Transfer to ICU for ongoing ventilation and management until block reverses and function returns.

TOTAL SPINAL

Call for assistance immediately. If unconscious, the pregnant woman will require many hands to manage during resuscitation. Delegate clearly and convey the sense of urgency, including the need to prepare for potential delivery.

Emphasis should be on vasopressors, fluids and ventilatory support.

With appropriate support and delegation, steps should be performed simultaneously rather than sequentially.

The diagnosis is usually apparent – rapidly ascending numbness and paralysis following spinal or epidural. If not witnessed or diagnosis uncertain, see Maternal Collapse **tab 18**.

Differential diagnosis includes:
Vasovagal
Haemorrhage
LA toxicity
IVC compression
Embolus

A pregnant woman is prone to reflux. Intubation is preferred but should not exclude any other form of airway management if conditions don't permit (cricoid pressure is recommended).

If the patient has lost consciousness, intubation can be accomplished without an induction agent or with relaxant only.

In profound spinal blockade without detectable cardiac output, CPR should be commenced until there is a response to fluids and vasopressors.

It should be performed in accordance with the maternal resuscitation guidelines (see Maternal Collapse **tab 18**) including emphasis on left uterine displacement and delivery of the baby. Lateral tilt no longer recommended.

Delivery should be considered 4 minutes after commencement of CPR.

Bolus drug dose:		
	Atropine	0.6–1.2mg
	Ephedrine	12–15mg
	Phenylephrine	50–100mcg
	Adrenaline	25–50mcg

ROSC = Return of Spotaneous Circulation
LUD = Left Uterine Displacement

POST PARTUM HAEMORRHAGE

1. Call for help, communicate and delegate.
2. Administer 100% oxygen.
3. Insert 2 large bore IV cannulae.
4. Use crystalloid resuscitation until blood available.
5. Perform uterine massage or use bimanual compression.
6. Call blood bank for crossmatch, component therapy, and activation of **massive transfusion protocol** if indicated.
7. Consider group specific or O negative blood.
8. Notify operating theatres for immediate transfer.
9. Use **oxytocics** for uterine atony.
10. Use vasopressors only to support vital organ perfusion.
11. For surgical control, induce general anaesthesia using RSI.
12. Continue with Severe Haemorrhage protocol **tab 05**.

POST PARTUM HAEMORRHAGE

Major PPH is a blood loss >1000mL with shock or ongoing bleeding. Blood loss is frequently underestimated and **coagulopathy may be disproportionate to blood loss**.

Use warm crystalloid solutions (Normal Saline or Ringer's Lactate) for resuscitation, up to 3.5L while waiting for blood. There is no evidence to support the use of colloid.

Uterine massage or bimanual compression can slow and reduce blood loss. Although there is no evidence, it is still supported by professional consensus. A fibrinogen level of > 2g/L should be maintained. Cryprecipitate and fibrinogen concentrate are both options for fibrinogen replacement.

Senior clinicians anticipating the need for component therapy reduce the time lag due to thawing. With early infusion of FFP 15mL/kg, haemostatic failure may be prevented. Products should subsequently be titrated against coagulation results. Viscoelastic techniques (TEG®, ROTEM®) can provide guidance in a much shorter time than laboratory testing.

Life threatening haemodynamics may require the use of uncrossmatched (O negative) or group specific blood.

Drug Doses			
Oxytocics		**Vasopressor**	
Oxytocin	5 IU slow push IV 10 IU per hour infusion	Ephedrine	6–12mg IV
Ergometrine	500mcg IMI or slow IV	Metaraminol	0.5–1mg IV
Misprostol	400–1000mcg PR/SL	Phenylephrine	25–50mcg IV
Carboprost	250mcg IMI/intrauterine (15 minutely, max 8 doses)	Vasopressin	0.5–1.0 IU IV

Alternatively, ergometrine 250mcg can be given IM or slow IV 5 minutely up to a max of 1mg in the absence of contraindications.

Causes of PPH

Tone - uterine atony
Trauma - genital tract
Tissue - retained products
Thrombin - coagulopathy

Surgical treatment options

Balloon tamponade
Brace suturing
Ligation/occlusion/embolization
(Uterine/Iliac arteries)
Hysterectomy

MATERNAL COLLAPSE

1. Call for help, communicate and delegate.
2. Assess conscious state, breathing and circulation.
3. If no output, start CPR, use left uterine displacement (LUD) and prepare for possible imminent delivery.
4. Place monitor leads, confirm cardiac rhythm and select relevant arrest protocol tab 01 or tab 02.
5. Intubate early and ventilate with 100% O_2.
6. Establish IV access with two large bore cannulae.
7. Review 4Hs 4Ts targeting pregnancy events first.
8. Immediately treat any reversible cause.
9. If pregnancy > 24 weeks and no ROSC, deliver after 4 minutes.
10. Debrief and support resuscitation team.

MATERNAL COLLAPSE

Be aware of maternal resuscitation differences:
Uterine displacement, early intubation and potential delivery.

Early intubation reduces aspiration risk.

Maternal resuscitation requires many hands – delegation of key roles such as airway, compressions, LUD, IV insertion, timing and documentation is crucial. Lateral tilt no longer recommended.

Common Causes	Uncommon Causes
Delivery less likely	**Delivery more likely**
Vasovagal	Pulmonary embolus
High epidural/spinal block tab 01	Uterine rupture
LA toxicity tab 20	Amniotic fluid embolus
Haemorrhage tab 17	Cardiac event tab 01
Drug overdose	Cerebral event
Hypoglycaemia	Anaphylaxis tab 06
Hypertensive disease of pregnancy	

For hypertensive disease of pregnancy:

Commence magnesium sulphate therapy for seizures
Loading dose 4g over 15 minutes (1g = 4mmol Mg)
Infusion 1g/hr for 24 hours
Use 2g boluses if seizures persist

Maternal cardiopulmonary resuscitation teams should regularly rehearse.
A Perimortem delivery pack should be kept on the resuscitation trolley. The most experienced clinician performs the C-section with an incision allowing the quickest delivery.

Maternal resuscitation is traumatic for all concerned including staff and family. Debriefing and professional supportive counseling are recommended.

NEONATAL RESUSCITATION NEWBORN LIFE SUPPORT

1. Start the clock.
2. Dry, warm and cover the baby to maintain temperature.
3. Assess colour, tone, breathing and heart rate.
4. If floppy, gasping or not breathing, open the airway and give 5 inflation breaths using air.
5. If no improvement in heart rate or chest movement, call for help and delegate tasks (SpO_2 monitor +/- ECG).

60s

6. Reposition head, review airway maneuvers and repeat inflation breaths.
7. If still no response, visualise pharynx, look for obstruction, gently suction if indicated and intubate. Increase FiO_2.
8. Once ventilation established, if heart rate below 60/min start compressions – use a 3:1 compression ventilation ratio.
9. Aim for 120 events/min – 90 compressions 30 breaths.
10. Reassess every 30s and if no response give 10-30mcg adrenaline IV.
11. In the presence of hypovolaemia or known blood loss, 10mL/kg of isotonic crystalloid or appropriately treated blood can be given and repeated if indicated.
12. Depending on outcome, notify neonatal unit and prepare for team and family debriefing.

NEONATAL RESUSCITATION NEWBORN LIFE SUPPORT

An inflation breath is 2–3 seconds in duration and 15-30cm H_2O pressure. If the heart rate responds by increasing, continue to ventilate at a rate of 30–40/min until there is adequate spontaneous ventilation.

Begin resuscitation with room air, and supplement oxygen if failure to respond. Avoid hyperoxaemia.

If still no chest movement after 60s, try the following **airway maneuvres** to aerate effectively:
- Reposition head (neutral)
- Jaw thrust (may require assistance)
- Oropharyngeal airway (e.g. Guedel)
- Laryngoscopy +/- suction +/- intubation

Gentle oropharyngeal suctioning is preferred – nasopharyngeal suctioning has been associated with bradycardia.

Confirm lung aeration and ventilation before progressing to circulatory support. Use a compression ventilation ratio of 3:1. Avoid simultaneous delivery by pausing slightly for each breath.

Colour is not a reliable indicator of SpO_2 but may pallor may indicate acidosis or anemia. SpO_2 soon after birth should be 60% increasing to 90% at 10 minutes – see table.

Ventilation and chest compression fail to resuscitate less than 1 in 1000 babies.

Acceptable Pre-ductal $Sp0_2$	
2 min	60%
3 min	70%
4 min	80%
5 min	85%
10 min	90%

Adrenaline dose is 10mcg/kg but can be increased to **30mcg/kg** if the lower dose is not effective. Bicarbonate is not recommended.

Post resuscitation care is similar to adults: avoid hyperoxia, maintain normal $EtCO_2$ and blood sugar level.

Therapeutic hypothermia, commenced within 6 hours of birth is still recommended for those with asphyxial encephalopathy.

LOCAL ANAESTHETIC SYSTEMIC TOXICITY

1. Stop giving local anaesthetic.
2. Call for help, communicate and delegate. Ask for Intralipid.
3. Assess ECG rhythm and cardiac output - if circulatory arrest, start CPR and alert cardiopulmonary bypass team.
4. Review the airway, intubate if indicated and use 100%O_2.
5. Confirm or secure IV access and treat any seizures.
6. Give 1.5 mL/kg of 20% Intralipid and commence infusion.
7. If hypotension or arrhythmias, follow standard treatment. Be aware of altered doses and drugs to avoid.
8. Persist with CPR and consider cardiopulmonary bypass if failure to respond.

LOCAL ANAESTHETIC SYSTEMIC TOXICITY

If there is no circulatory arrest use conventional supportive measures to maintain haemodynamic stability. Amiodarone can be used for ventricular irritability but lignocaine and other class1B antiarrhythmic agents should be avoided. See tab 01 or tab 34 for dosage. **Avoid beta blockers, calcium channel blockers and vasopressin.** Be prepared for prolonged advanced life support because of myocardial binding duration of LA agent.

Drug Dosages			
Anticonvulsants	**Dose**	**70kg patient**	**20kg patient**
Midazolam	0.05–0.1mg/kg	2–5mg	1–2mg
Diazepam	0.1–0.2 mg/kg	2–5mg	1–2mg
Thiopentone	1mg/kg	50mg	20mg
Propofol*	0.5–2mg/kg	30–50mg	10–20mg

Repeat bolus dose if required. *Propofol should be avoided in cardiovascular instability.

Intralipid Regimen

Immediately 1.5mL/kg bolus over 1 minute (100mL in adult).
Commence infusion of 15mL/kg/hr (1000mL per hour in adults).

At 5 minutes Repeat the bolus dose and double the infusion rate if not responding.

Allow a total of three bolus doses 5 min apart. Maximum cumulative dose 8-12mg/kg.

Local anaesthetic	Max. dose without adrenaline (mg kg^{-1})	Max. dose with adrenaline (mg kg^{-1})
Lignocaine	3	7
Bupivacaine	2	2
Ropivacaine	3	3
Prilocaine	6	9
Mepivicaine	7	7

Maximum safe dose can vary according to source and is a guide only. Potential for toxicity depends on site, injection rate, vasoconstrictor use, absorption rate, type of LA and its process of elimination.

HYPERKALAEMIA

1. Establish cardiac monitoring and IV access.
2. Check for artifact and repeat the sample.
3. Stop any source of K^+ administration.
4. Review history, blood levels, ECG and clinical stability to determine if immediate treatment indicated.
5. If $K^+ > 6.5$, ECG changes or unstable, use drug therapy in table opposite to stabilise the myocardium and shift K^+ into cells.
6. Start nebulised salbutamol 10-20mg and hyperventilate if the patient is mechanically ventilated.
7. If passing urine, give normal saline with 20-80mg frusemide IV to increase elimination.
8. Check K^+ and blood glucose at 30-60 minute intervals up to 6 hrs.
9. If levels remain high at 60 minutes, repeat steps 5 through 7 and consider urgent haemodialysis.
10. Correct reversible precipitating factors listed opposite.

HYPERKALAEMIA

Exclude artifact by repeating sample from a new site, but do not wait for results to implement treatment if the level exceeds 6.5mmol/L (depending on the rate of K^+ rise), there are ECG changes or clinically unstable.

IV Therapy for Life Threatening Hyperkalemia		
Drug	**Adult**	**Paediatric**
Calcium chloride (central)	5–10mL 10%	0.2mL/kg 10% over 5 min
Calcium gluconate (peripheral)	15–30mL 10%	1mL/kg 10% over 3–5 mins
$NaHCO_3$ (only in acidosis)	50mL	1-2meq/kg
Dextrose	50% 25–50mL	25% 0.5g/kg (2ml/kg)
Insulin	10 units	0.1units/kg

Unless otherwise stated in the table, all drugs are given by slow IV push.

Calcium chloride is highly irritating to veins and usually given through central venous access. Calcium gluconate has approximately one third potency of the chloride but is suitable for peripheral administration. It acts by stabilising the myocardium.

Up to 200meq of $NaHCO_3$ can be given in the adult if there is associated acidosis.
It acts by promoting intracellular shift of K^+ but is not effective with a normal PH.

Precipitating factors include:
trauma
burns
suxamethonium (burns, spinal injury, neurological disease)
malignant hyperthermia
acidosis
acute renal failure
organ reperfusion following clamp release
haemolyis/massive transfusion
medications

Avoid: suxamethonium
respiratory acidosis
Hartmann's solution

MALIGNANT HYPERTHERMIA (MH)

1. Call for help, communicate and delegate. Ask for MH Box.
2. Stop any volatile agent and remove the vaporiser.
3. Allocate task cards from box.
4. **Give dantrolene until crisis is over.**
5. Hyperventilate with 100% high flow O_2 (>10L/min).
6. Do not waste time with circuit change but use activated charcoal filters (if available) on both limbs of circuit.*
7. Maintain anaesthesia with TIVA.
8. Insert arterial line and consider CVC.
9. Actively cool the patient if Temp > 38.5.
10. Treat associated hyperkalemia, acidosis and arrhythmias.
11. Maintain urine output > 2mL/min.
12. Monitor with ABGs, electrolytes and temp, and prepare for ICU.

MALIGNANT HYPERTHERMIA (MH)

Signs of MH		
Early	**Developing**	**Late**
Elevated $EtCO_2$ Masseter spasm Tachycardia Arrhythmias	Acidosis Temperature rise CVS instability Hyperkalaemia	Cola-coloured urine ↑↑ CK Coagulopathy ↓ SpO_2 Cardiac Arrest

Give 2.5mg/kg dantrolene IV, repeat every 10-15 minutes until crisis is over.
Dantrolene requires dedicated staff to prepare. Mix each 20mg vial with 60mL sterile water. Mobilise additional dantrolene as each dose may require 8-10 vials. Alternatively use Ryanodex (new dantrolene) 250mg reconstituted with 5mL of water.

Hyperkalaemia is treated according to protocol **tab 21**.
Maintain urine output at >2mL/kg/hr.
Each vile of dantrolene contains 3g of mannitol. Ryanodex (new dantrolene) contains 125mg of mannitol.
Consider the treatment of acidosis with **8.4% $NaHCO_3$** only if there is associated hyperkalaemia.

Arrhythmias may require:

Amiodarone 3-4mg/kg slow IV
Lignocaine 1-2mg/kg IV
Metoprolol 1-2mg IV prn

Dantrolene may interact with calcium channel blockers leading to cardiovascular collapse associated with marked hyperkalaemia.

Active cooling includes:

Intra-abdominal lavage (saline at 4C)
Cool IV fluids
Cool sponging and ice packs
Lowering operating room temperature

Blood chemistry includes FBC, electrolytes, ABG's, CK, clotting profile and myoglobin levels.

* UK guidelines recommend circuit change and paralysing patient with non depolarising relaxant.

TERMINAL EVENT CHECKLIST – THE 10 Ts

1. **T**ubes - circuit or airway placement, patency, integrity.
2. **T**orrential haemorrhage.
3. **T**ryptase - anaphylaxis induced cardiac arrest.
4. **T**erminal rhythm - primary cardiac disease.
5. **T**amponade - traumatic or surgical.
6. **T**ension Pneumothorax.
7. **T**hrombus - cardiac, pulmonary, amniotic fluid, air or fat embolus.
8. **T**oxic - drugs, electrolytes, metabolic derangement.
9. **T**otal spinal.
10. **T**umour - Intracranial swelling and ↑ ICP.

The 10Ts is a perioperative, anaesthetic oriented checklist for clinicians managing an anaesthetic crisis when the diagnosis has not been established.

CRISIS AFTERMATH

1. Contact senior colleague or duty clinician for assistance.
2. Establish appropriate ongoing patient care post crisis.
3. If there is a death, fulfil legal obligations and notify coroner.
4. Quarantine drugs and equipment used.
5. When the patient is harmed, inform them as soon as conscious state permits.
6. **In all cases notify next of kin promptly and accurately, with well-planned team approach using clearly delineated roles, expressions of regret or apologies when appropriate.**
7. Establish communication channels for patient/family support.
8. Conduct a debriefing session for all staff immediately after the event and repeat in the following days if needed.
9. Complete records and all documentation for the case.
10. The anaesthetist should also write a personal account.
11. The anaesthetist and his/her family should be well supported and closely monitored post crisis.
12. File reports to relevant critical incident and insurance agencies at both local and national level.
13. Consider ≥ 24hr abstention from duties for anaesthetist involved.
14. Perform root cause analysis and implement recommendations.

THE IMPAIRED COLLEAGUE

The impaired practitioner experiencing mental health issues, cognitive decline, relationship breakdown or grief reaction may be successfully encouraged to seek help through an informal approach by a concerned colleague. However, for instances of substance use, dependence or professional misconduct, following the protocol below may be more appropriate.

1. Request help to manage the initial meeting. Carefully consider who is most suited to make the approach.
2. Have all supportive evidence documented beforehand.
3. Consider all legal and medical board obligations.
4. Includes risk assessment in pre-meeting preparations.
5. Show compassion and do not react, judge or try to diagnose.
6. Ask the subject to listen to all information before responding.
7. Use language carefully, keep sentences simple, objectively present the facts, but be prepared for defensive denial.
8. Acknowledge the discomfort but emphasise a desire to help.
9. Instil a sense of hope, reassuring them of the availability and provision of appropriate treatment.
10. Have professional resources in place, making sure a support person is present immediately following the meeting.

CRISIS PREVENTION

Contents

25 INDEX CRISIS PREVENTION

DESATURATION

Diagnostic Checklist

↓ Delivery of O_2 to lungs

Check O_2 supply - follow lines from wall outlet or cylinders to patient.

- Oxygen supply failure
- Circuit obstruction/disconnect
- Low or inappropriate FGF
- Ventilator malfunction/setting
- Airway position/obstruction/trauma
- Endobronchial intubation
- Sputum plug/foreign body
- Laryngospasm/bronchospasm
- Apnoea/hypoventilation

↓ Delivery of blood to lungs

- Cardiac arrest
- Cardiac failure
- Anaphylaxis
- Pulmonary embolism
- Congenital heart disease
- Right to left cardiac shunt

Impaired oxygen exchange or ↑ pulmonary shunt

- One lung ventilation
- Pulmonary oedema
- Aspiration
- Contusion
- Atelectasis
- Pneumothorax
- Pneumoperitoneum
- Pneumonia/abscess
- Sepsis/ARDS
- Interstitial lung disease
- COPD

Artifact

- Hypothermia
- Poor peripheral circulation
- Probe displacement

Most common

- **Probe displacement**
- **Apnoea/hypoventilation**
- **Tube position**
- **Laryngospasm**

DESATURATION

Diagnostic Pathway

- Check oxygen is turned on, confirm FiO_2, adjust to 100% and systematically exclude the most common causes (bottom left).

- Simultaneously visualise patient colour, while checking peripheral circulation, temperature, nails and probe position to assess validity of reading.

- Scan the SpO_2 and the $EtCO_2$ waveforms while hand ventilating to test circuit integrity and lung compliance.

If the CO_2 waveform is absent or altered

- Exclude oesophageal intubation, disconnect, or poor cardiac output.

- Confirm the airway is positioned appropriately and the patient is ventilating. Look for bag movement or the correct ventilator mode and setting.

- Check for laryngospasm, severe bronchospasm, kinked tube, sputum plug or secretions - suction the airway and consider bronchoscopy.

If the CO_2 waveform is unchanged

- Check O_2 supply - follow lines from wall outlet or cylinders to patient.

- Confirm appropriate fresh gas flow.

- Exclude endobronchial intubation.

- Examine the chest, neck veins and review the CXR if available. Consider pulmonary shunt and its causes listed opposite.

- If appropriate and surgery permits, position the patient head up to assist respiratory mechanics while working through the diagnostic checklist.

- If desaturation persists, call for help and review steps together.

HYPERTENSION
Diagnostic Checklist

Anaesthesia	Intubation/emergence
	Inadequate depth
	Inadequate analgesia
	Hypoxia
	Hypercarbia
	Malignant hyperthermia
	Drug effect
	Incorrect drug dose
	Drug interaction
	Transducer height/calibration
Surgery	Tourniquet application
	Aortic clamping
	Carotid endarterectomy
	Baroreceptor stimulation
Patient	Essential hypertension
	Full bladder
	Pre-eclampsia
	Renal disease
	Phaeochromocytoma
	Thyroid storm
	Raised intracranial pressure
Most common	**Intubation/emergence**
	Inadequate anaesthesia/analgesia
	Drugs
	Essential hypertension

HYPERTENSION
Diagnostic Pathway

- Establish the validity of the reading – check equipment, including transducer height and zeroing.

- Perform a BOAC check of monitors: **B**P/HR, **O**xygen, **A**gent, **C**O_2/RR.
  This helps confirm an oxygenated, ventilated, anaesthetised patient.

- Determine if there is stimulating surgical activity.
  Use additional narcotic if appropriate.

- Check for inadvertent vasopressor administration: LA with adrenaline, vasopressor infusions or drug error.

- Check tourniquet time and bladder volume.

- After systematically working through diagnostic checklist and treating reversible causes, consider IV pharmacotherapy outlined below.

Category	Drug		
Beta blockers	Metoprolol	Atenolol	Esmolol
Alpha blockers	Phentolamine	Labetalol*	
Alpha agonist	Clonidine		
Vasodilators	GTN	Hydralazine	Nitroprusside
Calcium antagonist	Nicardpine	Diltiazem	Clevidipine
Ace inhibitors	Enalaprilat		
D_1 receptor agonist	Fenoldapam		

Beta blockers should only be considered for BP control in toxidrome of catecholamines (e.g. pheochromocytoma) after alpha blockade is established, since in the absence of beta 2 mediated vasodilation, profound unopposed alpha mediated vasoconstriction may lead to hypertensive crisis or pulmonary oedema.

*Labetalol is a mixed alpha and beta receptor blocker.

For drug dosage tab 34.

HYPOTENSION

Diagnostic Checklist

↓ Preload	Blood loss
	Obstructed venous return
	Pneumoperitoneum
	Dehydration
	Capillary leak
	Elevated intrathoracic pressure
	Tamponade
	Embolism
	Patient position
	Pregnancy (IVC compression)
↓ Contractility	Drugs (including volatile agents)
	Ischemic heart disease
	Cardiomyopathy
	Myocarditis
	Arrhythmia
	Valvular heart disease
	Sudden increased afterload
↓ Systemic vascular resistance	
	Volatile agents / narcotics / vasodilators
	Anti hypertensive drugs (ACE inhibitors, ARBs)
	Regional blockade
	Anaphylaxis
	Sepsis / manipulation of infected tissue
	Post cardiopulmonary bypass
	Neuropathy
	Tourniquet / clamp release
	Addison's disease
	Thyroid disease
	Bone cement
Most common	**Anaesthetic agent**
	Narcotics
	Regional blockade
	Hypovolemia, bleeding
	Elevated intrathoracic pressure

HYPOTENSION

Diagnostic Pathway

- Look at the patient, check ECG rhythm, validate BP reading and scan the CO_2 waveform to assess the severity of the pressure drop.
- If no cardiac output detected, commence cardiac arrest protocol **tab 01** or **tab 02**. Consider anaphylaxis, massive haemorrhage or cardiac event first.
- In any minimally invasive or 'scope' surgery, always suspect canula related trauma and haemorrhage - check Hb early.
- For mild to moderate hypotension, consider head down or leg elevation, check airway pressure, review FiO_2, and correct common causes (bottom left).
- For persistent, unexpected or severe hypotension, consider placement of invasive monitoring and echocardiography to assist diagnosis.
- Use monitor information (below) to establish the preload, contractility or SVR contribution to hypotension. Systematically work through the diagnostic checklist for each category.

Findings	Diagnoses	Treatment
LVEDV low **PPV SVV**† > 12% **CVP*** trend down	Volume loss Decreased preload	Check Hb, give crystalloid or blood Treat underlying cause
LVEDV Low **PPV SVV** > 12%** **CVP** trend high, SvO_2↓ **Volume unresponsive**	Isolated RHF Embolus • Tension • Tamponade Infarct • Post bypass • Lung disease	Inotropes, Pulmonary vasodilators Thrombolysis, Thrombectomy Surgery ± mechanical support Treat underlying cause
LVEDV normal **PPV SVV** < 12% **LV f^{n}** normal/hyperdynamic	Low SVR Early sepsis	Vasopressors Treat underlying cause
LVEDV normal to high, **PPV SVV** < 12% **LV f^{n}** decreased	Cardiac pathology	Inotropes Stents, IABP Mechanical support Treat underlying cause
LVEDV normal **PPV SVV** < 12% **LV f^{n}** normal/decreased **SVR** low	Post bypass Severe or late sepsis	Inotropes, IABP, LVAD Vasopressors Treat underlying cause

* *Although still widely used, studies have shown that CVP is not a reliable indicator of volume status.*
** *May be >15% with tension pneumothorax or tamponade.*
† *PPV SVV calculation based on ventilated patient using tidal volumes of 8-10mL/kg ideal body weight.*

LVEDV = left ventricular end-diastolic volume
PPV = pulse pressure variation
SVV = stroke volume variation
CVP = central venous pressure
SVR = systemic vascular resistance
LV f^{n} = left ventricular function

TACHYCARDIA

Diagnostic Checklist

Primary causes

Ischaemic heart disease
Post cardiac surgery
Cardiomyopathy
Accessory conduction pathways
Sick sinus syndrome
Congestive heart failure
Myocarditis
Pericarditis
Valvular disease
Congenital heart disease

Secondary causes

Hypovolaemia
Anaesthetic depth
Drugs including local anaesthetic with adrenaline
Anxiety
Anaphylaxis
Electrolyte abnormalities
Tamponade
Pneumothorax / AutoPEEP
Sepsis
Throtoxicosis
Lung disease
Malignant hyperthermia

Most common

Anxiety
Intubation
Anaesthetic depth
Drugs
Hypovolaemia

TACHYCARDIA

Diagnostic Pathway

- Perform a BOAC check of monitors: **B**P/HR, **O**xygen, **A**gent and **C**O_2/RR. This helps confirm an oxygenated, ventilated, anaesthetised patient and may also indicate the severity of haemodynamic change.

- Give 100% O2 until tachycardia resolved and patient is stable.

- **If there is diagnostic uncertainty and compromised perfusion (rate usually > 150), synchronised DC shock is indicated.**
  Call for expert assistance.

- Use ECG width, regularity and rate to determine if it is a complex tachyarrythmia or sinus tachycardia associated with anaesthesia and surgical intervention.

- If it is sinus tachycardia, review the most common causes first (bottom left). Check for surgeon administered local anaesthetic with adrenaline.

- If it's a complex tachyarrhymia and haemodynamics permit, follow resuscitation guidelines:

ECG	Narrow Complex	Wide Complex
Regular	Vagal maneuvres Adenosine **Alternatives** Beta blockers Diltiazem Verapamil	**Ventricular Tachycardia** Amiodarone Procainamide Sotalol Lignocaine*
		SVT with aberrancy Treat as for narrow complex
Irregular	**Rate Control** Beta blocker Diltiazem Verapamil Digoxin **Rhythm Control** Amiodarone Flecainide Procainamide	**Torsade de Pointes** Magnesium
		AF with pre excitation Amiodarone Avoid A-V blocking drugs
		AF with aberrancy Treat as for narrow complex

Treat electrolyte imbalance, stop drugs that prolonge QT interval, screen for poisons.
Avoid AV blocking drugs in wide complex tachycardia if diagnosis uncertain.
*Lignocaine less effective. Use only if others unavailable.
Avoid verapamil with beta blockers.

For drug dosage tab 34.

BRADYCARDIA

Diagnostic Checklist

Primary causes

Ischaemic heart disease
Sick sinus syndrome
Degeneration of conduction system
Valvular disease
Myocarditis
Cardiomyopathy
Post cardiac surgery
Hereditary conduction disorders
Physiological fitness

Secondary causes

Electrolyte abnormalities
Antiarrhythmic medication
Anaesthesia
Hypothyroidism
Hypothermia
Vasovagal syndrome
Increased intracranial pressure

Anaesthetic causes

Hypoxia
Volatile agent
Muscle relaxant
Narcotic
Anticholinesterase
High spinal/epidural
Vasopressor reflex

Most common

Drug related
Vasovagal
Spinal anaesthesia
Fitness

BRADYCARDIA

Diagnostic Pathway

- Perform an anaesthetic BOAC monitor check: **B**P/HR, **O**xygen, **A**gent, **C**O_2/RR.
- Exclude hypoxia, vagal stimulation and consider increasing FiO_2.
- Use the table below to determine normal values for age group and fitness before treatment decision.
- If the patient is normotensive and well perfused, no immediate treatment initiative is required until diagnosis is made.
- Look for common causes first and then systematically check off possibilities from the diagnostic checklist.
- If the patient is symptomatic from hypotension and poor perfusion or the HR falls below the low limit for age and fitness, consider the following:

Atropine 20mcg/kg
Glycopyrrolate 5mcg/kg

Glucagon 2-4mg IV
(For beta blocker reversal)

Adrenaline 2-10mcg/min
Dopamine 2-20mcg/kg/min
Isoprenaline 5mcg/min

Calcium chloride 1g IV
(For calcium blocker toxicity)

Transcutaneous or transvenous pacing

- If there is no immediate response and $EtCO_2$ falls below 20mmHg start CPR and follow PEA arrest protocol **tab 02**.

Normal values for heart rate (beats/minute)

Age	Resting	Awake
Newborns 0 - 1 month old	90 - 160	100 - 205
Infants 1 - 11 months old	90 - 160	100 - 190
Children 1 - 2 years old	80 - 120	100 - 140
Children 3 - 5 years old	65 - 100	80 - 120
Children 6 - 11 years old	60 - 90	75 - 120
Children 12+	50 - 90	60 - 100
Athletes	40 - 60	N/A

Note: Ranges vary significantly according to populations and sources.

INCREASED END-TIDAL CO_2

Diagnostic Checklist

↑ Production	**Endogenous**	Sepsis
		Malignant hyperthermia
		Thyroid storm
		NLMS
		Reperfusion
		Shivering
	Exogenous	Bicarbonate administration
		CO_2 insufflation
		TPN
		CO_2 in fresh gas flow
		Exhausted soda lime
↓ Elimination	**Circuit**	Airway obstruction
		Inadequate fresh gas flow
		Valve malfunction in circuit
		Incorrect ventilator settings
	Lungs	Spontaneous hypoventilation
		Bronchospasm
		Chronic airways disease
Most common		**Spontaneous hypoventilation**
		Exhausted soda lime
		Fresh gas flow setting
		Ventilator setting

INCREASED END-TIDAL CO_2

Diagnostic Pathway

31B

INDEX

CRISIS PREVENTION

- Perform a BOAC monitor check: **B**P/HR, **O**xygenation, **A**gent, **C**O_2/RR. Analyse the CO_2 waveform.
- Review drug doses and anaesthetic depth.
- Check soda lime and patient temperature.
- Conform correct fresh gas flow setting for the circuit type as well as the size and condition of the patient.
- Check the ventilator mode is appropriate and settings are correct.
- The previous steps will eliminate the most common causes. If increased CO_2 persists systematically work through the diagnostic checklist.

BRADYCARDIA

Diagnostic Pathway

- Perform an anaesthetic BOAC monitor check: **B**P/HR, **O**xygen, **A**gent, **C**O_2/RR.
- Exclude hypoxia, vagal stimulation and consider increasing FiO_2.
- Use the table below to determine normal values for age group and fitness before treatment decision.
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Children 12+	50 - 90	60 - 100
Athletes	40 - 60	N/A

Note: Ranges vary significantly according to populations and sources.

INCREASED END-TIDAL CO_2

Diagnostic Checklist

↑ Production	**Endogenous**	Sepsis
		Malignant hyperthermia
		Thyroid storm
		NLMS
		Reperfusion
		Shivering
	Exogenous	Bicarbonate administration
		CO_2 insufflation
		TPN
		CO_2 in fresh gas flow
		Exhausted soda lime
↓ Elimination	**Circuit**	Airway obstruction
		Inadequate fresh gas flow
		Valve malfunction in circuit
		Incorrect ventilator settings
	Lungs	Spontaneous hypoventilation
		Bronchospasm
		Chronic airways disease
Most common		**Spontaneous hypoventilation**
		Exhausted soda lime
		Fresh gas flow setting
		Ventilator setting

INCREASED END-TIDAL CO_2

Diagnostic Pathway

31B

INDEX

CRISIS PREVENTION

- Perform a BOAC monitor check: **B**P/HR, **O**xygenation, **A**gent, **C**O_2/RR. Analyse the CO_2 waveform.
- Review drug doses and anaesthetic depth.
- Check soda lime and patient temperature.
- Conform correct fresh gas flow setting for the circuit type as well as the size and condition of the patient.
- Check the ventilator mode is appropriate and settings are correct.
- The previous steps will eliminate the most common causes. If increased CO_2 persists systematically work through the diagnostic checklist.

DECREASED END-TIDAL CO_2

Diagnostic Checklist

↓ Production	Hypothermia
	Hypothyroid
↑ Elimination	Spontaneous hyperventilation
	Inappropriate ventilator setting
↓ Alveolar ventilation	ETT obstruction
	Incorrect airway placement
	Laryngospasm
	Severe bronchospasm
	Dead space
↓ Pulmonary blood flow	Severe hypotension
	Anaphylaxis
	Cardiac arrest
	Cardiac tamponade
	Pulmonary or Air embolus
	Tension pneumothorax
Sampling dilution	Entrainment
	Inappropriate sampler placement
	High fresh gas flows
No $EtCO_2$ waveform	Oesophageal intubation
	Disconnect
	No ventilation
	No sampling
Most common	**Hyperventilation**
	Inadequate tidal volume
	Laryngospasm
	Incorrect airway placement
	Hypotension

DECREASED END-TIDAL CO_2

Diagnostic Checklist

- Perform a BOAC monitor check: **B**P/HR, **O**xygen, **A**gent and **C**O_2/RR.
  This will help confirm the level of oxygenation, anaesthesia and analgaesia, and indicate if there is significant hypotension.

- **Examine the EtCO_2. If there is NO waveform act immediately to exclude esophageal intubation, disconnect or failure to ventilate.**

- Check sampling line is connected and patent.

- Examine the patient. Check airway position and ventilatory pattern to exclude laryngospasm, airway displacement or poor tidal volumes.

- These steps eliminate the most common causes. If decreased EtCO_2 persists systematically work through the diagnostic checklist.

ELEVATED AIRWAY PRESSURE

Diagnostic Checklist

Circuit	Ventilator bag switch Ventilator setting Obstructed or kinked circuit Filter blockage Circle valve malfunction APL valve closed or stuck O_2 flush malfunction
Airway	Laryngospasm Tube position Tube size Tube obstruction
Patient	Bronchospasm AutoPEEP Tracheal pathology Respiratory tract tumours Foreign body Pneumothorax / Pneumoperitoneum Abdominal distention Trendelenberg position Chest wall rigidity Obesity Chest compression Alveolar pathology: *Oedema* *Fibrosis* *Contusion* *Infection* *ARDS*
Most common	**Inadequate muscle relaxant** **Airway position** **Laryngospasm** **Bag/ventilator settings**

ELEVATED AIRWAY PRESSURE

Diagnostic Pathway

- Exclude light anaesthesia and/or inadequate muscle relaxation.
- Manually ventilate to confirm high pressure, examine the $EtCO_2$ wave form and check the airway for any obvious change.
- Perform a systematic circuit, valve and ventilator inspection.
- If autoPEEP suspected, occlude expiratory limb at end expiration and look for pressure rise or examine ventilator display for persistent expiratory flow when next breath commences.
- If unresolved, replace the circuit with a self inflating resuscitator connected directly to the airway device. If pressure remains high, it is not the circuit.
- Check the position and patency of the airway by suctioning the full length of tube and using a bronchoscope if available.
- Examine the patient's respiratory system and consider calling for assistance.
- If any doubt, **replace the airway**. If ventilating with an LMA, consider replacing with an ETT.
- Review checklist of patient causes on opposite page.
  Also see explanatory notes **tab 13**.

Always consider the timing of the event.
Check for recent CVC insertion, drug administration, tube adjustment, position change, pneumoperitoneum or surgical intervention.

DRUG DOSAGES

Adenosine	6mg IV push – Rpt 12mg x 2
Amiodarone	300mg IV over 20min – Follow with 900mg over 24hrs
Atenolol	1-2mg IV rpt up to 10mg for arrhythmias or hypertension
Calcium Chloride	For hypocalcaemia: 500-1000mg IV over 5-10 min. Repeat as necessary For hyperkalaemia, hypermagnesemia: 500-1000mg IV over 5-10 min. Repeat PRN Calcium blocker overdose 1-2g IV over 10-20 min. Repeat 20min PRN
Clevidipine	Intermittent IV 1-2mg/hr Titrate to BP by doubling every 90 sec until toxicity or max dose of 32mg/hr
Clonidine	150mcg IV in divided doses for hypertension
Digoxin	8-12mcg/kg load IV – 50% over 5min, 25% at 4 & 8hrs
Diltiazem	15-20mg IV over 2min (0.25mg/kg) 20-25mg IV over 15min (0.35mg/kg) 5-15mg/hr maintenance
Enalaprilat	1.25mg IV over 5min. q6hr (up to 5mg/dose)
Esmolol	0.5mg/kg load. 0.05-0.3mg/kg/min infusion or 5-10mg increments titrated to BP or arrhythmia
Fenoldapam	Commence infusion at 0.05-0.1mcg/kg/min, then titrate up by 0.1/kg/min q15min until max dose 1.6mcg/kg/min or toxicity
Flecainide	2mg/kg IV up to 150mg over 30 minutes. Expert opinion for further dosage
Hydralazine	10mg IV repeat 20min, max 20mg/6hrs
Labetalol	20mg IV bolus then 20-80mg, 10 minutely (max 300mg). 0.5-2mg/min as infusion
Lignocaine	1-1.5mg/kg repeat 0.5-1mg 10 minutely until 3mg/kg. Infusion 1-4mg/min
Metoprolol	2-5mg IV over 1-2min. Repeat up to 15mg/3-6hrs
Magnesium	1-2g IV over 5-10min. Follow with 0.5-1g/hr infusion prn
Nicardipine	5mg/hr infusion, up to 15mg/hr titrate to BP
Nitroglycerin (GTN)	50mg in 50mL saline, titrate to BP. Start at 3-5mL/hr or 5mcg/min up to 100mcg/min
Nitroprusside	Infusion 0.25-0.5mcg/kg/min. Titrate up to 10mcg/kg/min or toxicity
Propranalol	0.5-1mg bolus over 1min. Repeat up to max of 0.1mg/kg. Infusion 3mg/hr
Phentolamine	5-10mg IV every 5-15min for hypertensive emergencies
Procainamide	20-50mg/min IV up to 17mg/kg. Maintenance infusion 1-4mg/min
Sotalol	1.5mg/kg IV over 5min. Expert opinion for further dosage
Verapamil	2.5-5mg IV over 2min, repeat with 5-10mg 15 minutely to max of 30mg

IV INFUSIONS

Adrenaline	3mg in 50mL NS = 60mcg/mL 0.05 - 0.5 mcg/kg/min (3-35mL/hr) in adult 0.1-1.0mcg/kg/min paediatric dose
Albuterol	5mg in 50mL of NS (100mcg/mL) 1 - 2 mcg/kg/min = 42-84 mL/hr (70kg adult)
Amiodarone	3mL (150mg) in 100mL 5% DW over 10 mintes 18mL (900mg) in 500mL 5% DW at 60mL per hour for 6 hours, then at 30mL per hour for 18 hours
Aminophylline	Load adult with 4-5mg/kg over 30minutes Then maintain with 0.2 to 0.8mg/kg/hr and titrate to serum concentration
Dobutamine	1000mg in 250mL NS or D5W = 5mg/mL 2.5-20 mcg/kg/minute. Maximum: 40 mcg/kg/min (34mL/hr) *Smaller dilution volumes e.g. 250mg/50mL, may be required depending on clinical circumstances*
Esmolol	2.5g in 250mL or 5g in 500mL (10mg/mL) Load with 1mg/kg (5-10 mL in adult) over 30 seconds Then 0.15 to 0.3 mg/kg/min (1.2 -2.4 mL/minute for 80kg patient)
Glucagon	5mg in 50mL NS 3-5mg bolus IV for Calcium Channel Blocker or Beta Blocker toxicity Repeat bolus up to 10mg Then 1-5 mg/hr infusion (10-50mL/hr)
Insulin	I unit/mL concentration e.g 100units soluble (novorapid) insulin in 100mLs Saline Also 50 units in 50 mL saline in syringe driver for use in the operating theatre
Magnesium	8 g in 100mL NS dilution 4 g in 15 min (200mL/hr) then 1 g each hour (12.5 mL/hr) *For eclampsia and seizures*
Metaraminol	10mg in 50mL Saline - titrate to effect Can also increase concentration to 1mg/mL e.g. 50mg in 50mL saline
Noradrenaline	4mg in 50mL NS = 80mcg/mL 0.1 to 2mcg/kg/min (5 - 100 mLs/hr) *Volume infused can be reduced by using 8 or 16mg of Noradrenaline.*
Nitroglycerin	50 mg in 250 mL (200 mcg/mL) D5W or NS 5-20mcg/min in adult (3 to 6mL/hr) and titrate to response (maximum dose: 400 mcg/minute)
Oxytocin	10 to 40 units in 1000mL Saline and titrate
Phenylephrine	40mg in 250mL NS (160mcg/mL) 40-60 mcg/min (15 - 25mL/hr); 0.1-0.5 mcg/kg/min paediatric dose
Tranexamic Acid	15mg/kg (1g or10mL in adult) undiluted over 10 minutes (maximum rate 100mg/min) Then 1gm in 100mL saline at 12.5mL hr
Vasopressin	Aim for 1u/mL concentration e.g. 50u in 50mL saline Titrate up to 0.04u/min (2u/hr)

15 POINT ANAESTHETIC MACHINE CHECK

Steps 1-15 should be performed at the beginning of each operating list.

1. Check the machine is switched on, uncluttered and positioned appropriately for the operating session.

2. Check service date and be aware of any notifications on the machine.

3. For modern electronic machines initiate self-testing.

4. Check monitors are on with appropriate limits, cycling times and sampling lines connected – confirm oxygen analyzer, pulse oximeter and capnograph are functioning.

5. 'Tug test' the pipeline connections.

6. Check the oxygen is connected and there is an adequate reserve supply.

7. Check other gas supplies are adequate and all pipeline pressures are between 400–500kpa.

8. Check flow meters are operating smoothly throughout the flow range.

9. Check the antihypoxia device and O_2 bypass are correctly functioning.

15 POINT ANAESTHETIC MACHINE CHECK

10 Check vaporisers are filled, seated, locked, filling port sealed, not leaking and then switched off.

11 Check the breathing circuit

- configuration
- connections
- valves
- leaks
- filters
- CO_2 absorber
- **two-bag test**

12 Check the ventilator

- tube connections
- pressure relief valve
- disconnect alarm
- high pressure alarm
- settings

13 Check the scavenging system is

- connected
- correctly configured
- functioning

14 Check the airway trolley – make sure all equipment needed for the anaesthetic plan (and contingency plan) is present and functioning.

15 Check suction, bed tilt, and **confirm an alternative means of ventilation** (eg. self inflating resuscitator).

PRE-INDUCTION CHECK

1. Check the machine, equipment and monitors.
2. Know the assistant's qualifications and experience.
3. Outline the anaesthetic plan, consider using a pre-intubation checklist, ask for feedback and encourage good communication.
4. Have a contingency plan – consult colleagues if uncertain.
5. Know the location of dantrolene, difficult airway trolley and resuscitation trolley.
6. Confirm correct patient, procedure and side... **Stop before you block!**
7. Be aware of any drug allergy.
8. Review airway and fasting status.
9. Check drug label and syringe... **Stop › Check › Inject**
10. Preoxygenate – check O_2 is on and confirm $EtCO_2$ trace.
11. Perform a BOAC post induction monitor check.

    B lood pressure / heart rate
    O xygenation
    A naesthetic agent
    C arbon dioxide / respiratory rate

12. In crisis management **call for help early**.

The ANAESTHETIC CRISIS MANUAL

INTERNATIONAL EDITION v2

The Anaesthetic Crisis Manual is a practical quick-reference handbook in Aviation Checklist format, giving step-by-step guidance for the management of the most common anaesthetic crises encountered in the OR.

- 32 crisis management protocols cover the major scenarios requiring immediate therapeutic intervention to prevent a catastrophic outcome. These include life-threatening cardiac, circulatory, airway, respiratory, metabolic and drug induced events.
- A crisis prevention section includes a 15-point machine check, adverse parameter checklists and diagnostic pathways to help rapid diagnosis of deteriorating events.
- Colour-coded, bulleted and numbered lists enhance memory recall in a stressful situation.
- The tabbed layout enables quick and easy navigation for use in the midst of a crisis.

A must for every anaesthesiologist, trainee and operating room.

David C. Borshoff is a cardiac anaesthesiologist, private pilot and advocate of immersive and simulation-based learning. He works in Perth, Western Australia.

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