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PACSA SUPPLEMENT

The physiologically difficult airway

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Introduction

The difficult airway has traditionally referred to anatomical characteristics which made visualisation of the glottic opening or placement of the endotracheal tube through the vocal cords difficult. In this paper we discuss how physiological derangements of the patient may place them at increased risk of cardiorespiratory collapse from airway management.[1] There are several predictable anatomical and physiological differences in children which places them at greater risk during the intubation process. These are briefly summarised in Table I.

The **anatomically difficult airway** is defined as a clinical situation in which a conventionally trained anaesthesiologist experiences difficulties with facemask ventilation, tracheal intubation or both.[3] With the expansion of devices available for airway management, contextual factors such as operator experience, time pressures, the clinical setting, and the patient's underlying physiological alterations still often result in difficulty with optimisation of gas exchange, which is the primary goal. The **physiologically difficult airway** is one where pre-existing physiological derangements (e.g. cardiopulmonary pathology, anaemia, low cardiac output, VQ mismatch and hypermetabolic states) place the patient at higher risk of cardiovascular collapse with intubation and conversion to positive pressure ventilation.[1,4] These factors should be accounted for in the intubation plan.[1]

The high risk nature of these airways is accentuated by the fact that 'waking up' the patient is often not an option, and reversing the effects of drugs does not reverse the mechanical or physiological cause of airway difficulty. Anatomically normal airways become physiologically difficult due to rapid deterioration, decreased reserve and urgency.[5]

Pathophysiology

The physiologically difficult airway is not well described and there is very limited data available on management methods particularly in the paediatric population. Most of the published literature is from Emergency Medicine and Critical Care units with predominantly experience-based recommendations, and where available, evidence-based recommendations.[1,5-8] Four clinically relevant physiologically difficult airway scenarios that the anaesthesiologist may commonly encounter include hypoxaemia, hypotension, severe metabolic acidosis, and right ventricular dysfunction or failure.

Hypoxaemia

Hypoxaemic respiratory failure (Type 1) commonly occurs due to an aetiology that disrupts optimal alveolar-capillary gas exchange e.g. pneumonia, paediatric acute respiratory distress syndrome (PARDS), and cardiogenic or non-cardiogenic pulmonary oedema, resulting in a ventilation/perfusion (VQ) mismatch.

There is increased risk of atelectasis with apnoea e.g. increased abdominal load, decreased muscle tone with reduced functional residual capacity. These patients are at high risk for rapid desaturation, resulting in bradycardia, haemodynamic instability, cardiopulmonary arrest and hypoxic brain injury. It is thus necessary to identify these patients with limited reserve, and to utilise all techniques available to prolong time to desaturation or 'safe apnoea time'.

Table I. Anatomical and physiological differences in children and strategies to address them[2]

Anatomical/physiological differences	Strategy to address
Large occiput	Position patient appropriately to align external auditory meatus with sternal notch. Infants may need shoulder roll, toddlers and school age children usually well aligned without support, older children and adolescents frequently benefit from elevation of the head similar to adults
Anterior, cephalad airway	Videolaryngoscopy and/or fibre-optic bronchoscopy, rescue device being supraglottic device
Large floppy epiglottis	Straight blade, engaging hypo-epiglottic ligament
Elliptical shaped airway	Appropriate cuffed endotracheal tubes
Smaller lung volume	
↓FRC, ↑closing capacity →rapid desaturation ↑ metabolic rate, ↑O_2 consumption →↓reserve Short apnoea time	Preoxygenation Apnoeic oxygenation

Recommendations

1. Preoxygenation

Preoxygenation depends on spontaneously breathing 100% oxygen. This denitrogenates the functional residual capacity (FRC) of the lungs and hence increases the FRC oxygen store and delays the onset of arterial desaturation and hypoxaemia.[9] The current standard of oxygenation involves the use of a tight fitting face mask (that prevents air leak from the anaesthetic circuit) with tidal breathing for 3–5 minutes with the aim of achieving FeO2 > 90%.[10] Safe apnoeic time is prolonged with preoxygenation, but variable with device seal, patient agitation/movement, factors that change the rate of oxygen consumption or functional residual capacity.

Non-invasive positive pressure ventilation (NIPPV) has been shown to improve oxygenation beyond usual preoxygenation methods, particularly in patients with obesity and shunt physiology. NIPPV increases mean airway pressure with the benefit of alveolar recruitment, temporarily decreasing the shunt fraction and improving oxygenation.[1] Nasal NIPPV has been shown to be well tolerated amongst the paediatric population. Positive pressure can be applied via standalone disposable continuous positive airway pressure (CPAP) masks connected to non-invasive machines or standard ventilators; or by closing the adjustable pressure limiting (APL) valve on the anaesthetic circuit and maintaining a tight seal as the patient breathes spontaneously or using a positive end-expiratory pressure (PEEP) valve on a standard bag mask.[11] NIPPV settings of inspiratory pressure 5–15 cm H_20, PEEP 5 cm H_20 and target tidal volumes of 6–8 ml/kg are usually used.

When NIPPV is deemed inadequate due to anatomic characteristics that make obtaining and maintaining an adequate mask seal difficult, supraglottic airways may be an option for preoxygenation.[1] Supraglottic airway devices have been inserted successfully in awake children after adequate local anaesthesia for the mouth and pharynx (e.g. LMA insertion in awake neonate with Pierre Robin syndrome after application of 2% lignocaine gel to airway).[12] Once inserted, it can be used as a conduit for tracheal intubation with a fibre-optic scope.[13]

Pharmacological assistance to decrease anxiety or induce sedation, without compromising airway tone, may be useful in improving patient tolerance during these manoeuvres. **Delayed sequence intubation (DSI)** is a technique in which ketamine (0.5–2 mg/kg) will dissociate the patient but allow them to maintain their protective airway reflexes while preoxygenating.[5,13,14]

Dexmedetomidine is an alternative.[7] This can be thought of as procedural sedation in which the procedure being performed is preoxygenation.

2. Apnoeic oxygenation

Apnoeic oxygenation is the passive flow of oxygen into the alveoli during apnoea. This can be achieved by oxygen insufflation through the nasopharynx (e.g. nasal prong oxygen, high-flow positive pressure humidified nasal prong oxygenation[15] or modified Trumpet manoeuvre[16]) or oropharyngeal passages, or through a needle inserted in the cricothyroid membrane,[10] or while intubating with a fibre-optic bronchoscope (FOB) through a supraglottic airway device[17] or even via an endotracheal tube attached to a laryngoscope e.g. Truview PCD videolaryngoscope.[18]

Transnasal humidified rapid insufflation ventilatory exchange (THRIVE) which is the same as high-flow positive pressure humidified nasal prong oxygenation ($HFNPO_2$), combines the benefits of apnoeic oxygenation and CPAP with some reduction in CO_2 levels through gaseous mixing and flushing of the dead space.[1,10] This can be used in preoxygenation and during apnoeic oxygenation.[15] Recommendations on flowrates are noted in Table II.

On average $PaCO_2$ increases 8 to 16 mm Hg in the first minute of apnoea, and then approximately 3–4.2 mm Hg per minute.[11,19] It is rare that the $PaCO_2$ increase and pH decrease are clinically significant. An exception is in profound metabolic acidosis (patients compensate for acidosis via tachypnoea and hyperpnoea) in patients with elevated intracranial pressure (CO_2 can lead to cerebral vasodilation) and pulmonary hypertension (where the hypercarbia and acidosis can precipitate a pulmonary hypertensive crisis).[11] Under these

Table II. Nasal cannula and $HFNPO_2$ flow rates

Proposed recommendations for nasal cannula flow rates during apnoeic oxygenation in children[2]			
	Adjusted per year of age	**Stepwise approach**	**Infant/child versus adolescent**
General Recommendations	1–2 L/min per year of age (max 15 L/min)	< 3 y: 2 L/min 3–8 y: 4 L/min > 8 y: 6 L/min	Infants/children: 5 L/min Adolescent: 15 L/min
Applying each recommendation across sample ages			
1-y-old	1–2 L/min	2 L/min	5 L/min
5-y-old	5–10 L/min	4 L/min	5 L/min
16-y-old	15 L/min	6 L/min	15 L/min
Recommendations for high flow humidified nasal prongs oxygen flow rates			
≤ 12 kg	2 L/kg/min		
> 12 kg	2 L/kg/min (for the first 12 kg) + 0.5 L/kg/min for each kg thereafter (max flow of 50 L/min)		

circumstances the patient should be actively hyperventilated prior to any airway intervention.

3. Recruitment

Anaesthesia and intubation attempts worsen pulmonary mechanics and gas exchange in the critically ill. Provided haemodynamic stability is maintained, recruitment manoeuvres are beneficial in hypoxaemic patients following intubation. Various techniques have been suggested, one of which is using inspiratory pressure of 30–40 cm H_20 for 25–30 s to increase lung volume and oxygenation, and decrease atelectasis without adverse effects.[5]

Hypotension

When managing the airway of a sick child, there needs to be an awareness of normal cardiopulmonary interactions. The reduced left ventricular afterload that results from positive pressure ventilation (PPV) may be beneficial when myocardial contractility is poor. However, securing the airway of these patients carries risk. Positive pressure ventilation reduces preload, potentially reducing cardiac output. Small infants can have augmented vagal response to PPV, leading to bradycardia and further reduction in cardiac output. These changes with PPV can be exacerbated by hypovolaemia, sepsis, acidosis, reduced cardiac contractility, attenuation of catecholamine surge with resolution of hypoxia and hypercarbia, vasodilatory and myocardial depressing effects of induction agents.[20] It is thus necessary to predict or mitigate peri-intubation hypotension.

Recommendations

1. Fluid resuscitation

Increase in circulating volume will increase mean systemic pressure and venous return. If the right heart can accommodate the increased venous return, the patient will be volume responsive and the cardiac output will increase. Volume responsive is typically defined as an increase in cardiac output by > 15% in response to a fluid challenge.[1] Those patients who are not fluid responsive will require inotropic support. See Figure 1.

2. Inotropic support

Inotropic support may need to be considered before commencing with airway management. Adrenalin and/or dobutamine infusions may be helpful in maintaining vascular tone and perfusion pressure. They are useful for ameliorating the decrease in vascular tone induced by anaesthetic agents and maintaining systemic vascular resistance and diastolic perfusion of the coronary arteries until the transient hypotension resolves or fluid resuscitation is optimised. When given for a short period of time, diluted peripherally administered vasopressors have been shown to be low risk.[9]

3. Haemodynamically neutral induction agents

There needs to be careful consideration and selection of induction agents as they are often associated with haemodynamic effects. No drug is risk free. Ketamine is often an attractive choice due to its sympathomimetic properties. Etomidate maintains blood pressure during intubation but has potential adrenal suppressive effects, especially in septic patients; these effects can worsen haemodynamic instability.

Severe metabolic acidosis

When acidemia develops from metabolic acidosis, mechanism of acid base homeostasis depends on a compensatory respiratory alkalosis from alveolar hyperventilation.[1] When hypocapnia is already present due to respiratory alkalosis, further hyperventilation results in incrementally smaller decreases in $PaCO_2$ and eventually reaches a plateau at which point there is no effect of further increasing alveolar ventilation (Figure 2). Thus in severe metabolic acidosis as occurs with salicylate toxicity or severe lactic acidosis, the acid production demands on alveolar

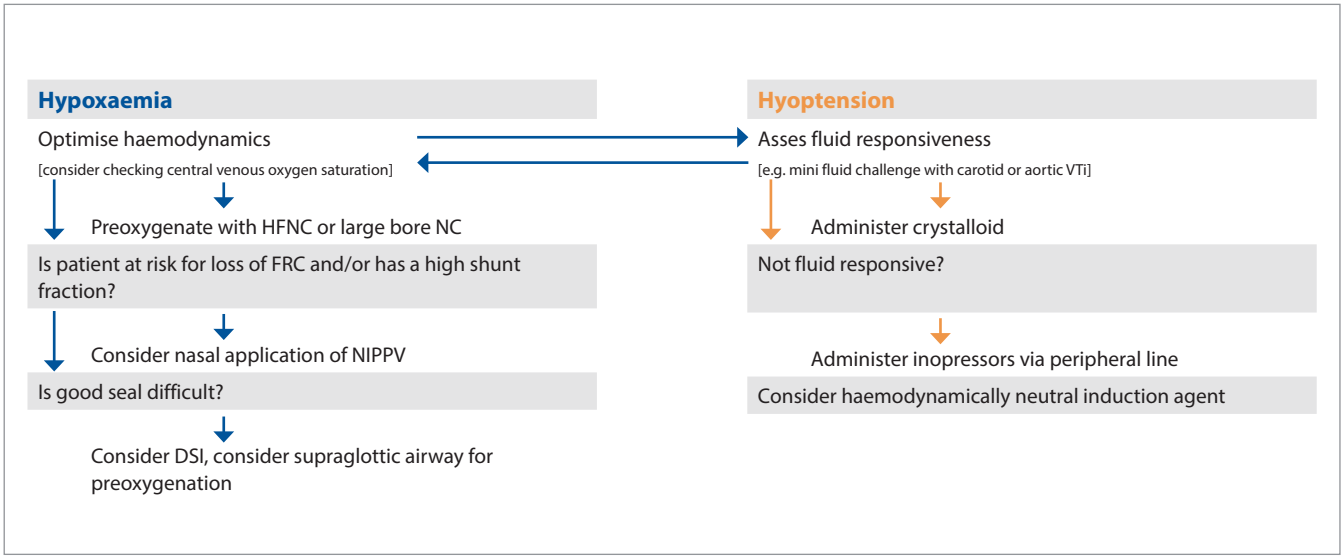


Figure 1. Summary of the management strategies in hypoxaemia and hypotension

NIPPV - non-invasive positive pressure ventilation, DSI - delayed sequence intubation, HFNC - high flow nasal cannula, VTi - velocity time integral, FRC functional residual capacity[6]

ventilation cannot be met and subsequently a profound acidemia develops. In this event should the patient require intubation, even a brief period of apnoea can lead to a precipitous drop in the pH. Furthermore, the pre-intubation ventilation cannot be matched by the mechanical ventilator. Consequently lung protective strategies may need to be abandoned.[1,21]

Recommendations

1. Intubation should be avoided if possible; instead the underlying metabolic derangement should be corrected.
2. Where intubation is necessary, use short-acting agents, so that there is rapid return of spontaneous respiratory drive. This will allow the patient to maintain their own high minute ventilation.
3. Once intubated, choose ventilation mode that allows better patient synchrony and comfort in order to maintain their respiratory compensation e.g. pressure support or pressure control modes.

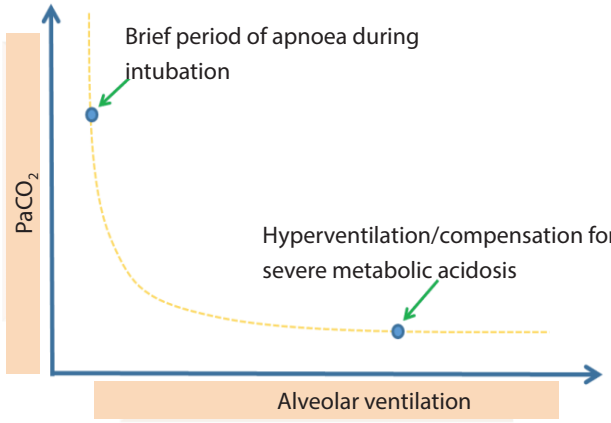


Figure 2. The relationship between alveolar ventilation and $PaCO_2$. A brief fall in alveolar ventilation (e.g. with intubation) can result in dramatic increases in $PaCO_2$ and acidemia which may be fatal in pulmonary hypertension[21]

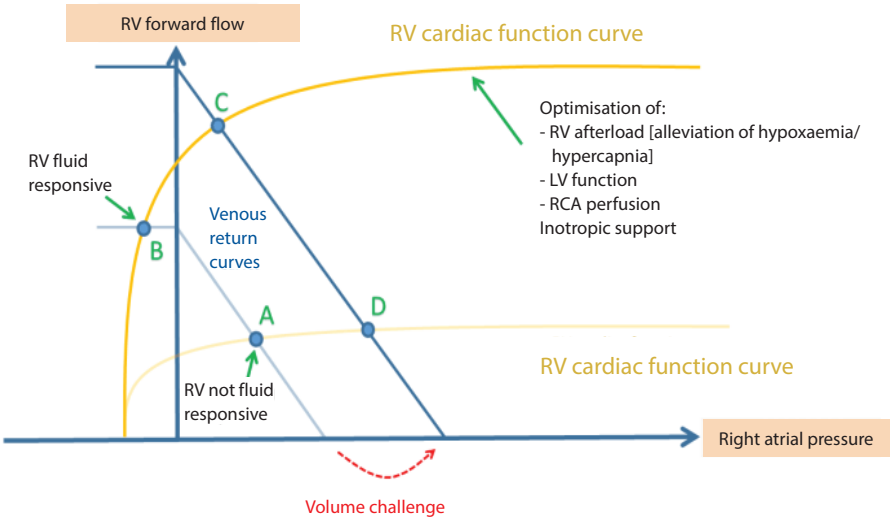


Figure 3. Optimising RV function first will move the operating point from A to B. Provision of fluids at point B will shift the venous return curve and RV forward flow will rise from point Bb to C. If volume is given prior to optimisation of RV function, the operating point will move from A to D. This will raise right atrial pressure without augmenting flow.

RV - right ventricle, LV - left ventricle, RCA - right coronary artery[21]

Right ventricular failure

The right ventricle (RV) is a low-pressure, high-compliance, flow-based chamber geared to propel venous blood returning to the heart into the pulmonary circulation.[1] It is important to differentiate RV dysfunction (some reserve retained) from RV failure, where there is an inability to meet increased demands with ensuing RV dilatation, retrograde flow and ultimately systemic hypotension and cardiovascular collapse.[1] Positive pressure ventilation which causes an increase in intrathoracic pressure, increased RV afterload and decreased preload often leads to cardiovascular collapse in patients with pre-existing RV pathology. Elevations in pulmonary vascular resistance e.g. hypoxaemia, hypercarbia, acidosis and sympathetic stimulation such as intubation, also have the potential to further increase RV afterload.

Recommendations

1. Bedside echocardiographic assessment of RV function should be performed to differentiate RV dysfunction from RV failure. If there is still some contractile reserve (RV dysfunction), cautious fluid resuscitation is possible.[1]
   Preoxygenation and apnoeic oxygenation should be performed.
2. Haemodynamically stable drugs should be used.
3. Continous dobutamine infusion should be started prior to induction in the hypotensive patient with the goal of increasing mean arterial pressure higher than pulmonary artery pressure.
4. Goals of ventilation should be to keep low mean airway pressure to prevent excessive right ventricle overload, hyperventilation with high FiO_2 and moderate PEEP with the aim of decreasing RV afterload.

General management principles

It is imperative to have appropriate airway assessment, planning and preparation for the difficult intubation. Rescue techniques including front of neck access should be readily available. The double approach is encouraged. Furthermore, key factors in making problem solving and crisis management successful are teamwork and communication to ensure a shared mental model, and situational monitoring.[5,22]

Standard **monitoring** must include pulse-oximetry, waveform capnography, blood pressure, electrocardiogram and, where available, end-tidal oxygen concentration.[5] If clinically indicated, a preintubation focused echocardiography to assess RV function can be performed. After haemodynamic assessment of the

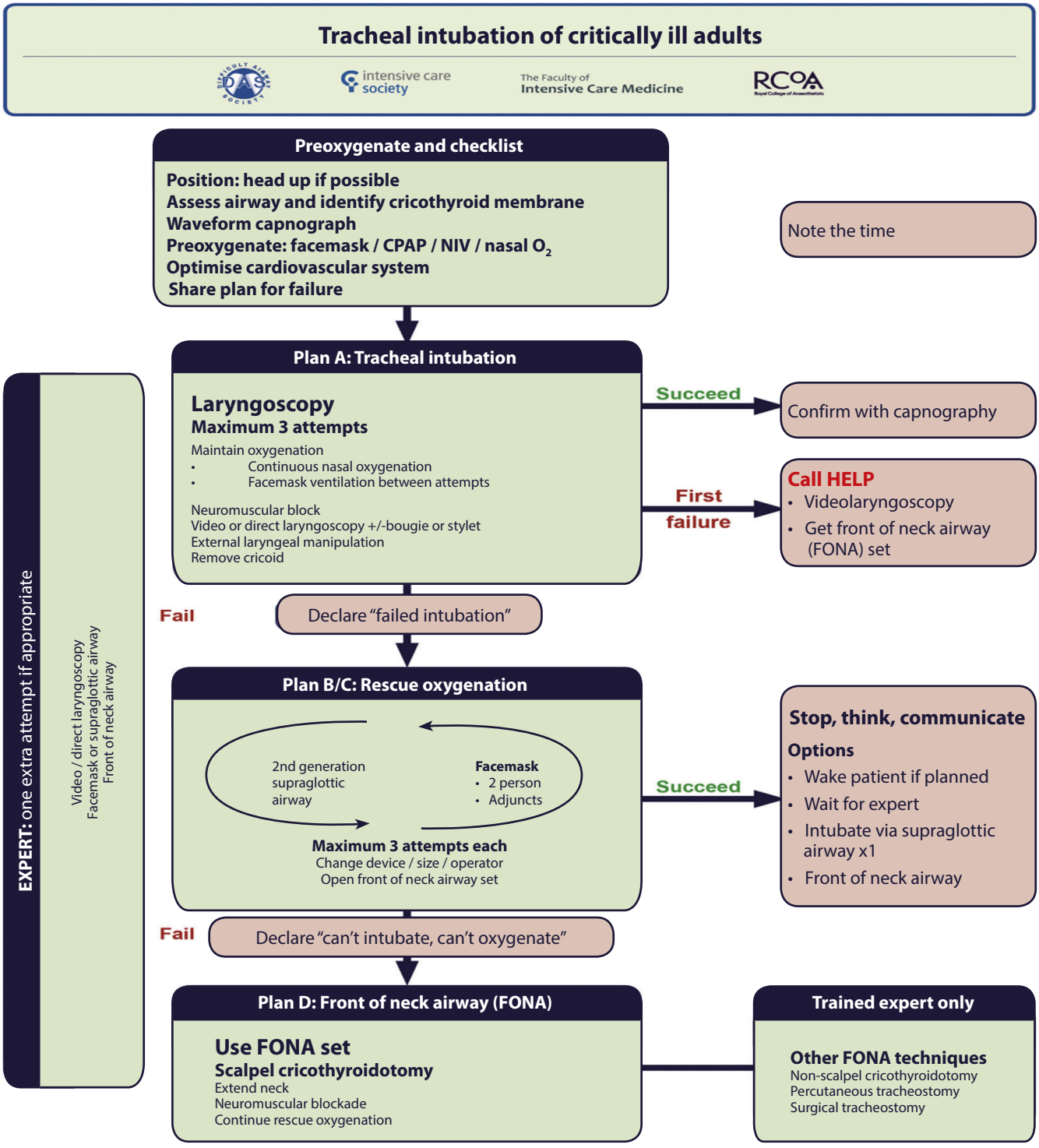
patient, consideration of fluid resuscitation and/or vasoactive agents takes place. Thereafter haemodynamically neutral induction agents may be used.

In the **preoxygenation** period the following should take place:

- Position patient in the ear to sternal notch position.
- Risk categorise patient and apply appropriate preoxygenation assisted by DSI where necessary
- Low risk identified by SpO_2 96–100% receive a tight-fitting face mask, high risk identified by SpO_2 9–95% should get a tight-fitting face mask or CPAP or bag mask device with PEEP 5–15 cm.
- Aim to achieve SpO_2 > 98%.
- Consider slight hyperventilation in patients with PHT, ↑ICP and severe metabolic acidosis.

During the apnoeic period:

- Remove face mask and apply nasal cannulae at high flow, THRIVE or nasal CPAP (or other alternatives of **apnoeic oxygenation**) throughout the intubation attempt.



This flowchart forms part of the DAS, ICS, FICM, RCoA guideline for tracheal intubation in critically ill adults and should be used in conjunction with the text.

Figure 4. Algorithm for tracheal intubation of critically ill adults[5]

- Limit lengths of laryngoscopic attempts until oxygen saturations falls lower than a predetermined level (often 90–92%) or length of time (e.g. 30–45 seconds) to decrease the frequency of hypoxia without affecting success rates.

If visualisation of the glottis is poor, the provider should reassess the patient's position to improve the alignment of the oral-pharyngeal-laryngeal axes. **Optimal external laryngeal manipulation** (BURP – backwards, upward and rightward pressure on the larynx[23]) can be used to bring the larynx into view. Direct or indirect laryngoscopy techniques performed by an experienced provider should be used, failing which the provider should consider the usage of a supraglottic airway device and/or fibre-optic device. The double setup approach, a strategy of preparing for two airway approaches simultaneously in patients with anticipated difficult airway, is advocated.[7] The cricothyroid membrane may be identified by ultrasound or clinically before inducing the patient.

Every attempt should be made to establish institutionally relevant modifications of the **difficult paediatric airway guidelines.** Figure 4 represents an algorithm suggested by the Difficult Airway Society in 2017 for tracheal intubation in the critically ill adult. This encompasses the principles of having a maximum of 3 laryngoscopy attempts, with rapid progression to simple techniques of oxygenation and ventilation, failing which a surgical airway is recommended.[24-26]

Conclusion

There is no substitute for good clinical judgement, and first attempt success is the goal for airway management in patients with physiological derangements. Clinicians should thus optimise all critically ill patients prior to intubation as the physiological airway is a high-risk procedure requiring significant expertise in airway handling as well as understanding of pathophysiology of the disease process. There is minimal margin of error. Many of the recommendations are based on clinical experience and physiological principles in the adult patient, thus presenting an opportunity for formal investigation in the paediatric patient.

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