The Physiologically Difficult Airway

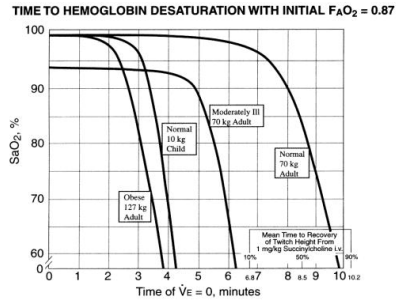
- The physiologically difficult airway is one in which physiologic derangements place the patient at higher risk of cardiovascular collapse with intubation and conversion to positive pressure ventilation.
- Hypoxemic respiratory failure patients are at high risk for rapid desaturation during intubation, which may result in hemodynamic instability, hypoxic brain injury, and potentially cardiopulmonary arrest.

Respiratory failure

- Acute hypoxemic respiratory failure (Type I) is failure to maintain adequate arterial oxygenation, most commonly due to any etiology that disrupts optimal alveolar-capillary gas exchange, such as pneumonia, acute respiratory distress syndrome (ARDS), and cardiogenic or noncardiogenic pulmonary edema.
- Hypercapneic respiratory failure (Type II), is due to decreased alveolar ventilation or an increase in dead space. Hypoxemia from Type II respiratory failure is relatively easily corrected with supplemental oxygen or an increase in minute ventilation.

Safe apnoea time

- Safe apnea time is prolonged with pre-oxygenation, but variable depending on factors that change the rate of oxygen consumption or functional residual capacity (FRC) such as critical illness and obesity.



Potentially hypoxaemic patient

- Preoxygenation and apneic oxygenation should be performed in all critically ill patients.
- A low-cost, low risk application of apneic oxygenation is via standard or wide-bore nasal prongs at 10–15L/pm. This flow rate is well tolerated, provides near 100% FiO2 to the nasopharynx during the apneic period and may prevent desaturation in some patients.
- The rigid mask of the NRB does not create an adequate seal and thus ambient air is entrained around the mask and decreases the effective FiO2 to much less than 100%. The higher the minute ventilation, the more this ambient air dilutes the FiO2 by admixing with the oxygen reservoir from the NRB.
- In patients with shunt physiology due to atelectasis or alveolar filling from pneumonia, ARDS or pulmonary edema, NIPPV can improve alveolar recruitment and oxygenation.
- When hypoxemic patients preoxygenated with NIPPV are removed from positive pressure for the intubation procedure, there is a risk of derecruitment of alveolii

causing rapid desaturation. Nasal continuous positive airway pressure with a nasal mask may be useful to maintain alveolar recruitment during intubation in patients at high risk.

- In select patients, supraglottic airways may be considered when higher pressures are needed or a mask seal with NIPPV cannot be achieved.
- For patients who cannot tolerate the NIPPV mask (e.g. delirium), analgesia, anxiolysis, or delayed sequence intubation (DSI) may be considered to optimize preoxygenation.
- One must be prepared to intubate at the onset of DSI, even with ketamine, due risk of cardiac arrest, laryngospasm and apnea, which have all been reported with ketamine.

Potentially hypotensive patient

- Approximately 25% of patients develop transient hypotension after emergent intubation and transition to positive pressure ventilation.
- Peri-intubation hypotension is a major risk factor for adverse events, including cardiopulmonary arrest related to airway management, longer intensive care unit stays and increased hospital mortality.
- Transition to positive pressure ventilation increases intrathoracic pressure and thus right atrial pressure, decreasing the pressure differential driving venous return.
- Common causes of shock such as volume depletion, capillary leak, or a loss of systemic vascular resistance will decrease the mean systemic pressure and venous return making these patients particularly susceptible to positive pressure ventilation induced hypotension.
- Fluid resuscitation is important in critically ill patients, as an 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 a "volume responder" and cardiac output will increase.
- Rapid evaluation of volume responsiveness is easily performed at the bedside by a number of techniques evaluating cardiopulmonary interactions, such as respiratory changes in inferior vena cava diameter, arterial waveform analysis, or Doppler assessment of aortic flow velocities
- Not all patients will be volume responsive, in which case vasopressors may be helpful for maintaining vascular tone and perfusion pressure and norepinephrine is preferred vasopressor in critically ill patients.
- Pure vasoconstrictors such as phenylephrine will increase vascular resistance and blood pressure, but will depress the cardiac output and decrease venous return. In patients who are in shock, or under-resuscitated, this decrease in venous return and depressed cardiac output may actually worsen hemodynamics despite improved blood pressure.
- In patients with transient hypotension during intubation from vasodilation or a positive pressure induced decrease in venous return, peripherally administered vasopressors may be useful for maintaining adequate end-organ perfusion pressure until adequate fluid resuscitation is achieved.
- Diluted metaraminol boluses may be useful for ameliorating the decrease in vascular tone induced by anesthetic agents and maintain systemic vascular resistance and diastolic perfusion of the coronary arteries until the transient hypotension resolves

or fluid resuscitation can be optimized. When given for a short duration, peripherally administered vasopressors have been shown to be low risk.

- Benzodiazepines and propofol have a sympatholytic effect, leading to myocardial depression and a decrease in vascular tone.
- Ketamine is also an attractive choice for an induction agent given its sympathomimetic properties, although there have been reports of cardiac arrest after ketamine administration.
- Strongly consider using lower doses of induction agents for patients at risk of hypotension.

Patients with severe metabolic acidosis

- In severe metabolic acidosis from diseases such as diabetic ketoacidosis (DKA), salicylate toxicity, and even severe lactic acidosis, the organic acid production demands an alveolar ventilation requirement that sometimes cannot be met and patients can subsequently develop profound acidemia.
- Unlike the rapid decrease in PaCO2 possible during hypoventilatory states, when hypocapnoea is already present due to a compensatory respiratory alkalosis, further hyperventilation results in incrementally smaller decreases in PaCO2 and eventually reaches a plateau at which point there is no effect of further increasing alveolar ventilation.
- In the event that patients with severe acidemia require intubation, even a brief apneic period can lead to a precipitous drop in pH given the loss of the already inadequate respiratory compensation.
- The pre-intubation alveolar ventilation sometimes cannot be matched by the mechanical ventilator, which has physical limits on the volume and rate that can be delivered.
- Even if lung protective ventilation strategies are abandoned, the maximal attainable minute ventilation may be less than the pre-intubation minute ventilation, leading to a precipitous drop in pH and a high risk of hemodynamic deterioration after intubation.
- Patients with extremely high minute ventilation requirements are at high risk of developing relative hypoventilation, flow starvation, patient-ventilator dysynchrony and worsened acidosis.
- Intubation should be avoided, if possible, in patients with severe metabolic acidosis who have a minute ventilation requirement not likely to be met by the mechanical ventilator, despite a low pH.
- A short trial of NIPPV may adequately support the respiratory work of breathing until correction of the underlying metabolic acidosis can occur and will provide an estimate of the patient's intrinsic minute ventilation by measuring the patient's respiratory rate and tidal volume delivered with each breath.
- Rapid sequence intubation should be avoided if possible, and if one is deemed necessary, a short-acting neuromuscular blocker such as succinylcholine should be used.
- If intubation is necessary, maintaining spontaneous respiration becomes the critical action both during intubation and with mechanical ventilation. This will allow the patient to maintain their own high minute ventilation and includes using sedative agents that are less likely to reduce the patient's respiratory drive.

- A pressure-targeted ventilator mode such as pressure support ventilation or pressure control mode will allow the patient to set the rate and tidal volume received.
- Special care should be taken to monitor for air trapping given the high rates and tidal volumes reached as well as monitor for respiratory muscle fatigue, which will result in a loss of compensation.

Patients with right ventricular failure

- Under normal circumstances, the right ventricle is a low-pressure, high-compliance, flow-based chamber geared to propel venous blood returning to the heart into the pulmonary circulation.
- Any process that increases right ventricular (RV) afterload, such as chronic pulmonary hypertension from lung or left ventricular disease, pulmonary arterial hypertension, or acute pulmonary embolism strains the RV, which adapts by increasing both contractility and preload.
- The critical action for the emergency physician is to determine if the patient has RV dysfunction, where the RV has some reserve and is able to perform some of its pumping function, or overt RV failure, in which the RV is unable to meet increased demands leading to RV dilation, retrograde flow, decreased coronary perfusion, and ultimately systemic hypotension and cardiovascular collapse.
- Intrathoracic pressure changes with respiration have an exaggerated effect on hemodynamics in the patient with RV failure, worsening cardiopulmonary interactions and making intubation extremely risky.
- Unlike left ventricular function, which improves with positive pressure ventilation, RV function worsens with the increase in intrathoracic pressure induced by positive pressure ventilation. This occurs because the intrathoracic pressure is transmitted to the alveolar capillary bed, leading to collapse of these small vessels and increases the pulmonary vascular resistance against which the RV must pump.
- When patients with RV failure require intubation, the increased RV afterload and decreased preload associated with invasive mechanical ventilation can often lead to cardiovascular collapse.
- When possible, work of breathing and gas exchange should be supported with medications, oxygen, and if positive pressure ventilation is needed, then NIPPV and low positive end-expiratory pressure with the goals of decreasing work of breathing, limiting atelectasis, and reducing hypoxic vasoconstriction. These methods of support allow the patient to breathe spontaneously, resulting in a smaller rise in intrathoracic pressure than control modes.
- Patients with increased RV afterload often present with varying degrees of RV strain on bedside echocardiography, including a dilated RV and inferior vena cava, septal flattening during systole in pressure overloaded states, and septal flattening during diastole in volume overloaded states.
- The tricuspid valve regurgitation jet velocity, tricuspid annular plane systolic excursion (TAPSE), tricuspid annular peak velocity or isovolumetric contraction velocity (IVV) and RV outflow tract velocity-time integral are easy to perform and useful methods of determining the degree of RV strain, volume responsiveness, and contractile reserve on bedside echocardiography.

- While patients with RV dysfunction may respond to small fluid challenges or an inotropic agent, further increasing preload with a fluid challenge in patients with RV failure is unlikely to be fruitful, and may be deleterious as volume overloading a pressure overloaded RV increases diastolic wall tension and left ventricular diastolic dysfunction, directly worsening left ventricular filling and stroke volume.
- Bedside echocardiographic assessment of RV function should be performed to assess RV dysfunction versus RV failure. If the patient has some contractile reserve (RV dysfunction), cautious fluid resuscitation should be performed.
- Preoxygenation is essential despite the difficulties resulting from intracardiac shunt and ventilation-perfusion (V/Q) mismatch, which commonly occur in right heart failure.
- Apneic oxygenation should be performed given the potential for benefit
- Induction agents should be considered carefully.
- Intravenous fentanyl premedication may be useful to blunt the hypertensive response to laryngoscopy.
- Continuous norepinephrine infusion should be started prior to induction in hypotensive patients with the goal of increasing mean arterial pressure higher than pulmonary artery pressure, which can be determined by bedside echocardiography.
- For patients without hypotension, norepinephrine should be primed and "in-line" in the event of post intubation or sedative induced hypotension.
- The goals of mechanical ventilation include maintenance of a low mean airway pressure and avoidance of hypoxemia, atelectasis, and hypercapnea, which increase RV afterload.

Reference

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