

Risk of Harm Associated With Using Rapid Sequence Induction Intubation and Positive Pressure Ventilation in Patients With Hemorrhagic Shock

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ABSTRACT

Based on limited published evidence, physiological principles, clinical experience, and expertise, the author group has developed a consensus statement on the potential for iatrogenic harm with rapid sequence induction (RSI) intubation and positive-pressure ventilation (PPV) on patients in hemorrhagic shock.

“In hemorrhagic shock, or any low flow (central hypovolemic) state, it should be noted that RSI and PPV are likely to cause iatrogenic harm by decreasing cardiac output.”

The use of RSI and PPV leads to an increased burden of shock due to a decreased cardiac output (CO), which is one of the primary determinants of oxygen delivery (DO₂). The diminishing DO₂ creates a state of systemic hypoxia, the severity of which will determine the magnitude of the shock (shock dose) and a growing deficit of oxygen, referred to as oxygen debt. Rapid accumulation of critical levels of oxygen debt results in coagulopathy and organ dysfunction and failure. Spontaneous respiration induced negative intrathoracic pressure (ITP) provides the pressure differential driving venous return. PPV subsequently increases ITP and thus right atrial pressure. The loss in pressure differential directly decreases CO and DO₂ with a resultant increase in systemic hypoxia.^{1, 2} If RSI and PPV are deemed necessary, prior or parallel resuscitation with blood products is required to mitigate post intervention reduction of DO₂ and the potential for inducing cardiac arrest in the critically shocked patient.

Situational Guidance:

1. An important part of the clinical decision-making in a patient with hemorrhagic shock is awareness of the fact that RSI and PPV are likely to decrease CO and worsen the

shock state. It is not recommended that RSI and PPV are seen as a first-line standard of care for these patients.

2. The primary management focus in the hemorrhagic shocked patient should be hemorrhage control and resuscitation with blood products of which whole blood (WB) probably represents the best combination of effectiveness and convenience.³
3. If RSI and PPV are required, every attempt should be made to ensure that resuscitation has been effective enough to enable the patient to withstand the impact of the intervention. As a surrogate for this approach a systolic blood pressure (SBP) of > 100mmHg is recommended.⁴
4. Aggressive ventilation by “breath stacking,” high ventilatory pressures, positive end-expiratory pressure (PEEP), and high ventilatory rates is likely to further reduce the cardiac output.^{5, 6}

Takeaway Points:

- This recommendation reflects the position of the author group and is based on the interpretation of existing evidence applied to physiologic principles, in addition to clinical experience and expertise. It is not intended to be a replacement for clinical judgement in the management of individual patients.
- This opinion relates specifically to the resuscitation of patients with hemorrhagic shock and applies to clinical judgement and the balance of risk and benefit to the patient.
- In hemorrhage, blood products are recommended for the resuscitation of hemorrhagic shock with WB being considered optimal in the remote damage control resuscitation (RDCR) environment.⁷

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- The resuscitation of casualties suffering hemorrhagic shock should begin as soon as blood products and appropriately trained personnel are available. Ideally, resuscitation should not be delayed for more than 30 minutes from the time of injury.^{8,9}
- It should be noted that spontaneous respiration, inducing negative ITP, is beneficial to the shocked patient. Every effort should be made to retain this driver of venous return and hence CO by using postural airway positioning, intubation without ventilation using short-acting neuromuscular blocking agents (NMBAs) or surgical cricothyroidotomy.

KEYWORDS: rapid sequence induction (RSI); intermittent positive-pressure ventilation; positive-pressure respiration; hemorrhagic shock; iatrogenic disease

Introduction

In the treatment of a patient with critical hemorrhagic shock, a systematic approach is widely recommended. During the primary survey, an Airway - Breathing - Circulation - Disability - Environment (A-B-C-D-E)-based system or adaptations of this are most common. The airway is considered first, with the gold standard of airway management being the placement of a cuffed tube in the trachea. This protects the airway from obstruction and aspiration. In the emergent setting the procedure is RSI. To facilitate the passing of the tube through the glottis the responsive patient needs to be both sedated and paralyzed with NMBAs. The drugs used to accomplish this vary between systems and practitioners.

The second consideration is Breathing; as the patient is paralyzed, they are required to be ventilated. This may be accomplished using a bag-valve mask (BVM) or a mechanical ventilator. Both methods ventilate by positive pressure. The decision to RSI and PPV carries benefits: the airway is protected and secured if a patient deteriorates, surgical grade anesthesia is produced, ventilation can be controlled, and supplemental oxygen can be delivered. For these reasons, RSI and PPV have become a standard of care with critical patients in many emergency medical systems. Unfortunately, RSI and PPV also carry risk, of which the reduction in CO is the most harmful in the shocked patient.

The decision to perform an intervention when indicated is the process of critical clinical decision making and is a balance between risk and benefit. An example of the challenge in this case is reflected in the European guideline on management of major bleeding and coagulopathy following trauma, which states that RSI is “mandatory” in hemorrhagic shock; however, the next sentence states “the introduction of positive pressure (ventilation) can induce potentially life-threatening hypotension in hypovolemic patients.”^{10, 11}

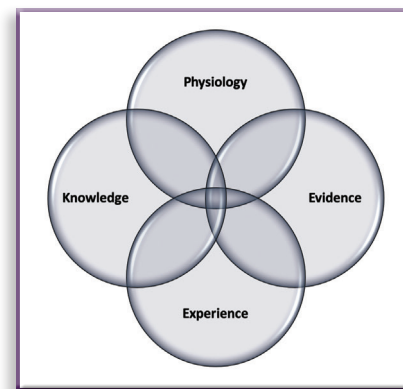
In situations where the procedure is highly likely to be helpful and has a low risk associated with the implementation, there is good reason to be aggressive in the delivery of that procedure. When the procedure is likely to be helpful, but the risk of clinical complications is higher, a more cautious approach is required. Last, when the procedure is not sure to help and the risk is high, a conservative approach is recommended. In the patient with hemorrhagic shock, it is the position of the author

group that RSI and PPV fall into the latter category; there is limited benefit from the PPV of hypoperfused lungs, even if this procedure delivers a greater percentage of oxygen. The PPV is likely to further decrease lung perfusion and increase V/Q mismatch. When considering the risk, the effect of PPV on CO may be disastrous for the patient in the low-flow state, often precipitating cardiac arrest.

A global picture of the patient’s pathophysiology is needed. The compensatory reserve and the proximity to physiological exhaustion should be noted. The impact of any intervention must also be carefully considered along with the potential for iatrogenic harm. The failure to consider iatrogenesis can be termed “iatrogenic blindness” contributing to poor outcomes. If the practitioner is not blind to the potential harm then steps can be taken to offset the impact of the intervention; for example, in severe hemorrhagic shock, the patient may be resuscitated with blood products to the point where RSI and PPV may be tolerated.

The rise of evidence-based medicine (EBM) has placed ever greater emphasis on the grade of evidence that exists for any treatment or procedure. Unfortunately, many a literature review concludes with a lamentation of the paucity of available evidence or the poor quality of the studies with high risk of bias. Where evidence exists, it must be carefully appraised and considered. In the absence of high-quality evidence, we must use our current understanding of physiology and pathophysiology to further guide our understanding. The power of empirical observation and clinical experience is well understood and one of the cornerstones of clinical judgment. The experienced clinician’s dynamic assessment of patient care and the effects of interventions, although not evidence based, are key requirements of clinical practice. One approach is to produce a synthesis of physiology, evidence, experience, and knowledge (PEEK) as a tool to aid clinical judgment (Figure 1).

FIGURE 1 *The PEEK clinical judgment tool.*



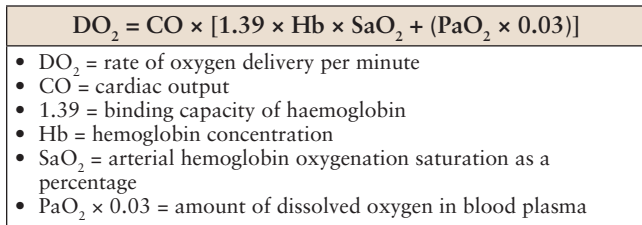
(Original diagram by P. Thompson, 2019.)

Physiology

Central hypovolemia is a primary feature of hemorrhagic shock and some other shocked states and is a feature of many disease processes. In hemorrhagic shock, the loss of circulating blood volume reduces venous return with a subsequent reduction in cardiac filling and thus end-diastolic volume (EDV). In accordance with Starling’s law of the heart, lower EDV translates into a decrease in stroke volume and CO. Any further reduction in CO will correspond to a reduction in DO_2 . If this reduction is beyond the capacity of the combined compensatory reserve mechanisms then the oxygen consumption will become

delivery dependent, at a point termed critical DO_2 (DO_{2crit}) (Figure 2).

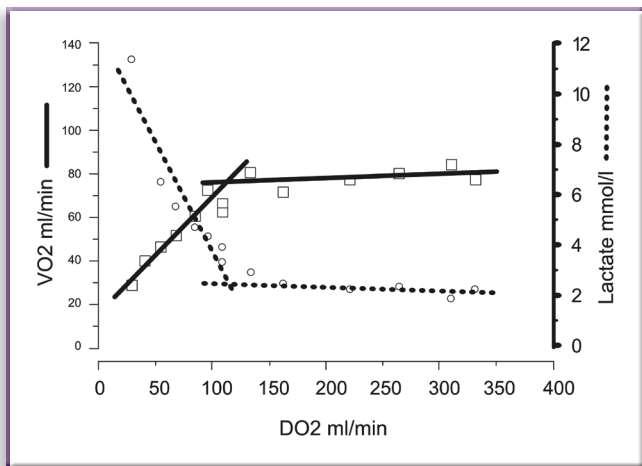
FIGURE 2 DO_2 equation. Based on Yartsev, A. *Deranged Physiology: The oxygen carrying capacity of whole blood* [Internet]. *DerangedPhysiology.com*; [updated 2020 July 25; cited 2020 August 30].



Original diagram by P. Thompson, 2020.

Further reductions in DO_2 will drive the VO_2 below this compensatory endpoint with cellular energy demand failing to be met by aerobic metabolism alone with the development of an ensuing oxygen debt. In the absence of adequate oxygen to meet cells' energy demand, the cells of the body must rely on anaerobic glycolysis to fill the energy "gap" left by inadequate DO_2 .¹² The end product of anaerobic glycolysis is lactic acid, the accumulation of which leads to a metabolic acidosis with elevated serum lactate. This metabolic acidosis is recognized as a surrogate for systemic hypoperfusion and is used as one of the triggers for massive transfusion protocols in many emergency medical systems (Figure 3).

FIGURE 3 Relationship between oxygen consumption (VO_2) and oxygen delivery (DO_2) when DO_2 is acutely reduced by tamponade or hemorrhage in anesthetized animals (data pooled from several studies). Note that blood lactate levels increase as soon as DO_2 falls below a critically low value (DO_{2crit}).

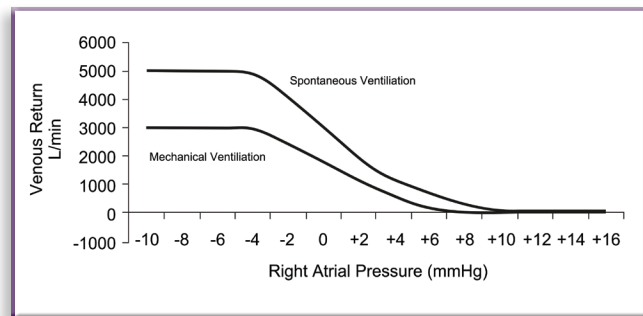


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The primary negative impact of PPV is an elevated intrathoracic pressure (ITP) that is associated with reduced venous return,¹⁴ lower cardiac filling,¹⁵ and subsequent decreases in CO and thus DO_2 . This has been well understood, demonstrated, and published since the work carried out by Guyton in the 1950s.¹⁶ Figure 4 illustrates waveforms showing the effect of PPV.

The drugs used in RSI are themselves not without risk. Many protocols advise reductions in dose of induction drugs in the shocked state in recognition of this fact. NMBAs and opiates induce vasodilation with a further reduction in venous return. Ketamine has been perceived as a relatively safe drug

FIGURE 4 Waveforms represent instantaneous measurements during a single cardiac cycle. Venous return is affected by the size of the vena cavae as well as by the right atrial pressure during cardiac filling. Positive-pressure ventilation reduces the maximum flow by its effect on cardiac output, and reduces the rate of atrial filling, because of intrathoracic caval compression. This functional obstruction causes atrial filling to cease at a lower atrial pressure.



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in shocked patients. Ketamine increases sympathetic tone in most patients, this effect is not seen if presynaptic catecholamine stores are depleted. The result in these situations is a net myocardial depression. Miller et al observed that patients with a high shock index (SI) demonstrated a higher incidence of hypotension post RSI. These investigators stated "Ketamine is considered a stable induction agent for rapid sequence induction; however, hypotension rates up to 24% are reported."¹⁸ There are also case reports of cardiac arrest in critically ill patients following the use of ketamine for RSI.¹⁹

Evidence

In a review of the literature, there is little high-quality evidence. There are no meta-analyses of prospective, multicenter, randomized controlled trials (RCTs). The evidence that exists must be understood to be mostly retrospective, have a small sample size, or be confounded by heterogeneity and bias. It is for this reason that weight must be placed on the elements of the PEEK clinical judgment tool. Presented next is a sample of the available evidence.

Lockey et al. examined the survival of trauma patients who had prehospital tracheal intubation without anesthesia or muscle relaxants and found that the outcome was almost always fatal (99.8%).²⁰ Although this cohort are among the most severely injured, it may also give a clue to the catastrophic effect of PPV on the critically ill patient in the low-flow state.

Shafi et al. found an association with RSI and PPV and decreased survival in hypovolemic trauma patients, which was worse when the patients were intubated in the prehospital setting. "Patients intubated in the field were more likely to be hypotensive upon arrival in the Emergency Department (ED) (SBP < or = 90 mm Hg; ED 33%, pre-hospital 54%, $p < 0.001$), and had worse survival (ED 45% versus pre-hospital 24%, $p < 0.001$). Even after controlling for potential confounders, pre-hospital Endotracheal Intubation (ETI) was still an independent predictor of hypotension upon arrival in ED (OR 1.7, 95% CI 1.46–2.09, $p < 0.001$) and decreased survival (OR 0.51, 95% C.I. 0.43–0.62, $p < 0.001$)."²¹ The authors state in the conclusion, "This may be mediated by the effect of positive pressure ventilation during hypovolemic states."²¹

Heffner et al. found that 22% of normotensive patients experience hypotension after emergency intubation in the ED, suggesting that an elevated ITP caused by PPV alone may be sufficient to reduce CO in normotensive patients.²²

Kim et al. found that systolic hypotension prior to intubation, defined as a systolic blood pressure ≤ 90 mmHg, was independently associated with postintubation cardiac arrest (odds ratio [OR] 3.67, 95% CI: 1.58–8.55; $P = .01$).²³

In a retrospective analysis of trauma patients, Chou et al. found that field intubation may be associated with higher mortality in trauma patients with hemorrhagic shock requiring massive transfusion. Less invasive airway interventions and rapid transport might improve outcomes for these patients.²⁴

Fevang et al., in a systematic review and meta-analysis comparing mortality in prehospital tracheal intubation to ED intubation in trauma patients, found the median mortality rate in patients undergoing prehospital intubation was 48% (range 8–94%), compared with 29% (range 6–67%) in patients undergoing intubation in the ED. The authors stated that “Cardiovascular collapse is a known complication of TI in this patient group, and some centres deliberately postpone in-hospital TI in patients in shock until after initial stabilization.”²⁵

In a retrospective database review, Crewdson et al. found that their “results suggest an association between prehospital emergency anesthesia and in-hospital mortality in awake hypotensive trauma patients, which is strengthened when hypotension is due to hypovolemia.” Based on these findings, the authors suggested that delayed induction of anesthesia may be appropriate.²⁶

Schwaiger et al. demonstrated that postponing intubation in spontaneously breathing major trauma patients on ED admission does not impair outcome in a retrospective analysis. Although a relatively small sample size was analyzed, these results demonstrated the feasibility of delaying intubation.²⁷

Taghavi et al. investigated “permissive hypoventilation” in a swine model of hemorrhagic shock.²⁸ The authors concluded that retaining spontaneous ventilation “results in less hemodynamic suppression and better perfusion of vital organs.” They recommended that “In severely injured penetrating trauma patients, consideration should be given to immediate transportation without PPV.”

The perspective to limit RSI and PPV in clinical states of severe blood loss or hemorrhagic shock until intravascular volume is restored with blood products is particularly important to military medical operations. A preliminary analysis generated from the US Department of Defense Trauma Registry indicates that 59% of the 8,653 combat casualties from 2002 to 2019 who were intubated also suffered from hemorrhage, indicated by receiving at least 1 unit of WB. This means that a significant proportion of injured who receive airway management may be at increased risk of suffering from detrimental effects such as iatrogenic harm induced by RSI and PPV.

Another approach to the examination of evidence against the use of PPV in patients with severe blood loss or hemorrhagic shock is to extract data from the literature that alter the paradigm of PPV by examining the effect of creating more negative

ITP. Contrary to the impact of PPV on lowering EDV, SV, CO, and DO_2 , multiple human experiments have demonstrated the effect of enhancing cardiac filling, CO, and tissue perfusion by reducing ITP,²⁹ increasing tolerance to experimentally progressive reductions in central blood volume similar to hemorrhage,³⁰ and promoting higher perfusion (arterial) pressures in patients with various etiologies of hypotension³¹ and hemorrhage.³² These consistent observations that link lower ITP with enhanced hemodynamic responses opposite to those induced by PPV also provide inferential evidence to avoid the use of intubation in hypovolemic patients when possible.

Experience

The experience of every clinician may vary, but there are many well-known risks of RSI and PPV besides the reductions in CO.

- After RSI, the potential Can't Intubate Can't Oxygenate (CICO) situation
- Unrecognized esophageal intubation
- Loss of situational awareness
- Complex decision-making, thereby increasing cognitive load and loss of bandwidth
- Fixation error during the process of RSI Intubation.
- Prolonged on scene time, and thus time to surgical control of bleeding, which is known to be an important factor in patient survival²⁷
- Resource intensive both in equipment and personnel
- In the prehospital or RDCR environment, if a difficult or failed intubation is encountered, there is often no recourse to specialist assistance.
- Hyperventilation both in the preoxygenation phase before RSI and postintubation has the effect of further decreasing venous return.
- In the patient with penetrating injury or poly trauma, the move to PPV raises the incidence of tension pneumothorax, which in turn necessitates further advanced procedures like thoracostomies.
- Hypoxia is a well-documented complication of RSI both from lack of preoxygenation and prolonged, or successive, attempts to pass the tube.³³
- Prehospital anesthesia is associated with hypothermia, which has been shown to have a negative impact on outcomes in trauma patients.³⁴
- RSI and PPV contribute to the lethal triad of acidosis and coagulopathy, by lowered DO_2 and hypothermia by anesthesia.

The net outcome of the effects described here has led to the often-described sequence of events of RSI – PPV – CPR.

Knowledge

Any practitioner should have knowledge of the indications, contraindications, complications, managing or preventing the complications, recognizing a successful intervention, and recognizing the physiological impact of the intervention, whether positive or negative, before commencing with RSI and or PPV.

Indications

The primary need for advanced airway management remains the failure of basic procedures, especially patient positioning. It is also recognized that RSI and PPV are required for damage control surgery. Other indications include severe facial

trauma, swelling, and airway burns. In the case of RSI and PPV for the hypovolemic, shocked trauma patient, the author group has concerns that in some systems the procedures have become a standard of care, prior to resuscitation, which worsens the shock causing iatrogenic harm.

Contraindications

It may be reasonable to argue that central hypovolemia from hemorrhagic shock could be seen as a relative contraindication to RSI and PPV.

Complications

Because the prevention of complications is an important part of the knowledge of the provider, the main complication is the effect of RSI and PPV on CO as discussed; it is the critical clinical decision of the care provider to decide if the patient's condition is such that these procedures can be tolerated. The drugs associated with RSI may also exacerbate the patient's condition.

Mitigation

Resuscitation with blood products to an SBP of 100mmHg is the primary mitigation strategy; this restores circulating blood volume and the patient's ability to withstand the drop in CO due to the interventions, while simultaneously treating the shock state and repaying critical oxygen debt. It may also be possible to maintain spontaneous respiration while providing the protection by surgical cricothyroidotomy. Novel approaches to intubation while maintaining spontaneous respiration are ketamine-only breathing intubation or using short-acting NMBAs to facilitate tube placement allowing spontaneous respiration to return while maintaining sedation. The relatively recent concept of delayed sequence induction³⁵ is using procedural sedation, to ensure preoxygenation, and resuscitation with blood products, after which the patient can be paralyzed and intubated. If PPV is finally required, then PPV limit excessive ventilatory pressures and high tidal volumes.

Synthesis

In critical illness, clinical decision-making may be difficult and time short. This may be compounded by environmental difficulties. In these situations, clinicians often follow highly protocolized guidelines. Many guidelines provide a list of procedures but not the clinical judgment required to accurately weigh the risks and benefits, which is recognized by the frequent inscription of "This document is a guideline only and not a substitute for clinical judgment." The path to experienced clinical judgment is not a short one. Simplicity and speed to definitive care with greater resources are often seen as key steps to patient survival. The discussion here may be synthesized into guidelines, facilitating a reduction in time at the scene, limiting difficult decision-making, focusing on the greatest pathophysiological risk to the patient, treating the problem, and avoiding iatrogenic harm. Reassessment remains key; if airway positioning is failing, then advanced airway management is required. However, even here there are options to maintain spontaneous breathing.

Conclusion

In hemorrhagic shock, the primary aim is to control the bleeding and limit time to definitive (surgical) care. The patient should be positioned to maintain airway patency. Once IV/IO access has been obtained, the patients should be resuscitated

with blood products to achieve an SBP above 100mmHg. Pain and/or combative patients can be treated with ketamine.³⁶ RSI and PPV may be delayed until the patient is resuscitated or damage control surgery is imminent. If advanced airway procedures are necessary, every effort should be made to resuscitate the patient first. In hemorrhagic shock with central hypovolemia the impact of RSI and PPV must be recognized as a contributing factor to the pathophysiological burden, which is harming the patient, potentially catastrophically. This procedure should not be attempted as part of a standard of care. In the risk benefit analysis of patients in hemorrhagic shock, the golden principle of "first do no harm" should be appropriately weighted due to the potential for iatrogenesis.

Author Contributions

PT wrote the first draft. GS, AH, VC, MD, CB, EG, AC, and PS contributed edits and references to the final draft, and all authors reviewed the final manuscript and suggested references.

Disclosures

The authors have nothing to disclose.

References

1. Cournand A, Motley HL. Physiological studies of the effects of intermittent positive pressure breathing on cardiac output in man. *Am J Physiol*. 1948;152(1):162–174.
2. Herff H, Paal P, von Goedecke A, et al. Influence of ventilation strategies on survival in severe controlled hemorrhagic shock. *Crit Care Med*. 2008;36(9):2613–2620.
3. Butler FK, Holcomb JB, Schreiber MA, et al. Fluid resuscitation for hemorrhagic shock in Tactical Combat Casualty Care: TCCC guidelines change 14-01--2 June 2014. *J Spec Oper Med*. 2014;14(3):13–38.
4. Woolley T, Thompson P, Kirkman E, et al. Trauma Hemostasis and Oxygenation Research Network position paper on the role of hypotensive resuscitation as part of remote damage control resuscitation. *J Trauma Acute Care Surg*. 2018;84(6S suppl 1):S3–S13.
5. Pepe PE, Lurie KG, Wigginton JG, et al. Detrimental hemodynamic effects of assisted ventilation in hemorrhagic states. *Crit Care Med*. 2004;32(9 Suppl):S414–S420.
6. Pepe PE, Roppolo LP, Fowler RL. The detrimental effects of ventilation during low-blood-flow states. *Curr Opin Crit Care*. 2005;11(3):212–218.
7. Jenkins DH, Rappold JF, Badloe JF, et al. Trauma hemostasis and oxygenation research position paper on remote damage control resuscitation: definitions, current practice, and knowledge gaps. *Shock*. 2014;41(suppl 1):3–12.
8. Alarhayem AQ, Myers JG, Dent D, et al. Time is the enemy: Mortality in trauma patients with hemorrhage from torso injury occurs long before the "golden hour." *Am J Surg*. 2016;212(6):1101–1105.
9. Shackelford SA, Del Junco DJ, Powell-Dunford N, et al. Association of prehospital blood product transfusion during medical evacuation of combat casualties in Afghanistan with acute and 30-day survival. *JAMA*. 2017;318(16):1581–15891.
10. Spahn DR, Bouillon B, Cerny V, et al. The European guideline on management of major bleeding and coagulopathy following trauma: fifth edition. *Crit Care*. 2019;23(1):98.
11. Schwartz DE, Matthay MA, Cohen NH. Death and other complications of emergency airway management in critically ill adults. A prospective investigation of 297 tracheal intubations. *Anesthesiology*. 1995;82(2):3673–3676.
12. Convertino VA. Mechanisms of inspiration that modulate cardiovascular control: the other side of breathing. *J Appl Physiol* (1985). 2019;127(5):1187–1196.
13. Vincent JL, De Backer D. Oxygen transport: the oxygen delivery controversy. *Intensive Care Med*. 2004;30(11):1990–1996.
14. Moreno AH, Burchell AR, Van der Woude R, et al. Respiratory regulation of splanchnic and systemic venous return. *Am J Physiol*. 1967;213(2):455–465.

15. Videbaek R, Norsk P. Atrial distension in humans during microgravity induced by parabolic flights. *J Appl Physiol* (1985). 1997;83(6):1862–1866.
16. Guyton AC. Determination of cardiac output by equating venous return curves with cardiac response curves. *Physiol Rev*. 1955;35(1):123–129.
17. Murphy BA, Durbin CG Jr. Using ventilator and cardiovascular graphics in the patient who is hemodynamically unstable. *Respir Care*. 2005;50(2):262–274; discussion 74.
18. Miller M, Kruit N, Heldreich C, et al. Hemodynamic response after rapid sequence induction with ketamine in out-of-hospital patients at risk of shock as defined by the shock index. *Ann Emerg Med*. 2016;68(2):181–8.e2.
19. Dewhirst E, Frazier WJ, Leder M, et al. Cardiac arrest following ketamine administration for rapid sequence intubation. *J Intensive Care Med*. 2013;28(6):375–379.
20. Lockey D, Davies G, Coats T. Survival of trauma patients who have prehospital tracheal intubation without anaesthesia or muscle relaxants: observational study. *BMJ*. 2001;323(7305):141.
21. Shafi S, Gentilello L. Pre-hospital endotracheal intubation and positive pressure ventilation is associated with hypotension and decreased survival in hypovolemic trauma patients: an analysis of the National Trauma Data Bank. *J Trauma*. 2005;59(5):1140–1145; discussion 5–7.
22. Heffner AC, Swords DS, Nussbaum ML, et al. Predictors of the complication of postintubation hypotension during emergency airway management. *J Crit Care*. 2012;27(6):587–593.
23. Kim WY, Kwak MK, Ko BS, et al. Factors associated with the occurrence of cardiac arrest after emergency tracheal intubation in the emergency department. *PLoS One*. 2014;9(11):e112779.
24. Chou D, Harada MY, Barmparas G, et al. Field intubation in civilian patients with hemorrhagic shock is associated with higher mortality. *J Trauma Acute Care Surg*. 2016;80(2):278–282.
25. Fevang E, Perkins Z, Lockey D, et al. A systematic review and meta-analysis comparing mortality in pre-hospital tracheal intubation to emergency department intubation in trauma patients. *Crit Care*. 2017;21(1):192.
26. Crewdson K, Rehn M, Brohi K, et al. Pre-hospital emergency anaesthesia in awake hypotensive trauma patients: beneficial or detrimental? *Acta Anaesthesiol Scand*. 2018;62(4):504–514.
27. Schwaiger P, Schochl H, Oberladstätter D, et al. Postponing intubation in spontaneously breathing major trauma patients upon emergency room admission does not impair outcome. *Scand J Trauma Resusc Emerg Med*. 2019;27(1):80.
28. Taghavi S, Jayarajan SN, Ferrer LM, et al. “Permissive hypoventilation” in a swine model of hemorrhagic shock. *J Trauma Acute Care Surg*. 2014;77(1):14–19.
29. Convertino VA, Lye KR, Koons NJ, et al. Physiological comparison of hemorrhagic shock and V O₂max: a conceptual framework for defining the limitation of oxygen delivery. *Exp Biol Med (Maywood)*. 2019;244(8):690–701.
30. Convertino VA, Ryan KL, Rickards CA, et al. Inspiratory resistance maintains arterial pressure during central hypovolemia: implications for treatment of patients with severe hemorrhage. *Crit Care Med*. 2007;35(4):1145–1152.
31. Convertino VA, Parquette B, Zeihr J, et al. Use of respiratory impedance in prehospital care of hypotensive patients associated with hemorrhage and trauma: a case series. *J Trauma Acute Care Surg*. 2012;73(2 suppl 1):S54–S59.
32. Convertino VA, Parquette BA, Wampler DA, et al. Use of intrathoracic pressure regulation therapy in breathing patients for the treatment of hypotension secondary to trauma. *Scand J Trauma Resusc Emerg Med*. 2017;25(1):105.
33. Davis DP, Hoyt DB, Ochs M, et al. The effect of paramedic rapid sequence intubation on outcome in patients with severe traumatic brain injury. *J Trauma*. 2003;54(3):444–453.
34. Langhelle A, Lockey D, Harris T, et al. Body temperature of trauma patients on admission to hospital: a comparison of anaesthetised and non-anaesthetised patients. *Emerg Med J*. 2012;29(3):239–242.
35. Weingart SD, Trueger NS, Wong N, et al. Delayed sequence intubation: a prospective observational study. *Ann Emerg Med*. 2015;65(4):349–55.35.
36. Merelman AH, Perlmutter MC, Strayer RJ. Alternatives to rapid sequence intubation: contemporary airway management with ketamine. *West J Emerg Med*. 2019;20(3):466–471.

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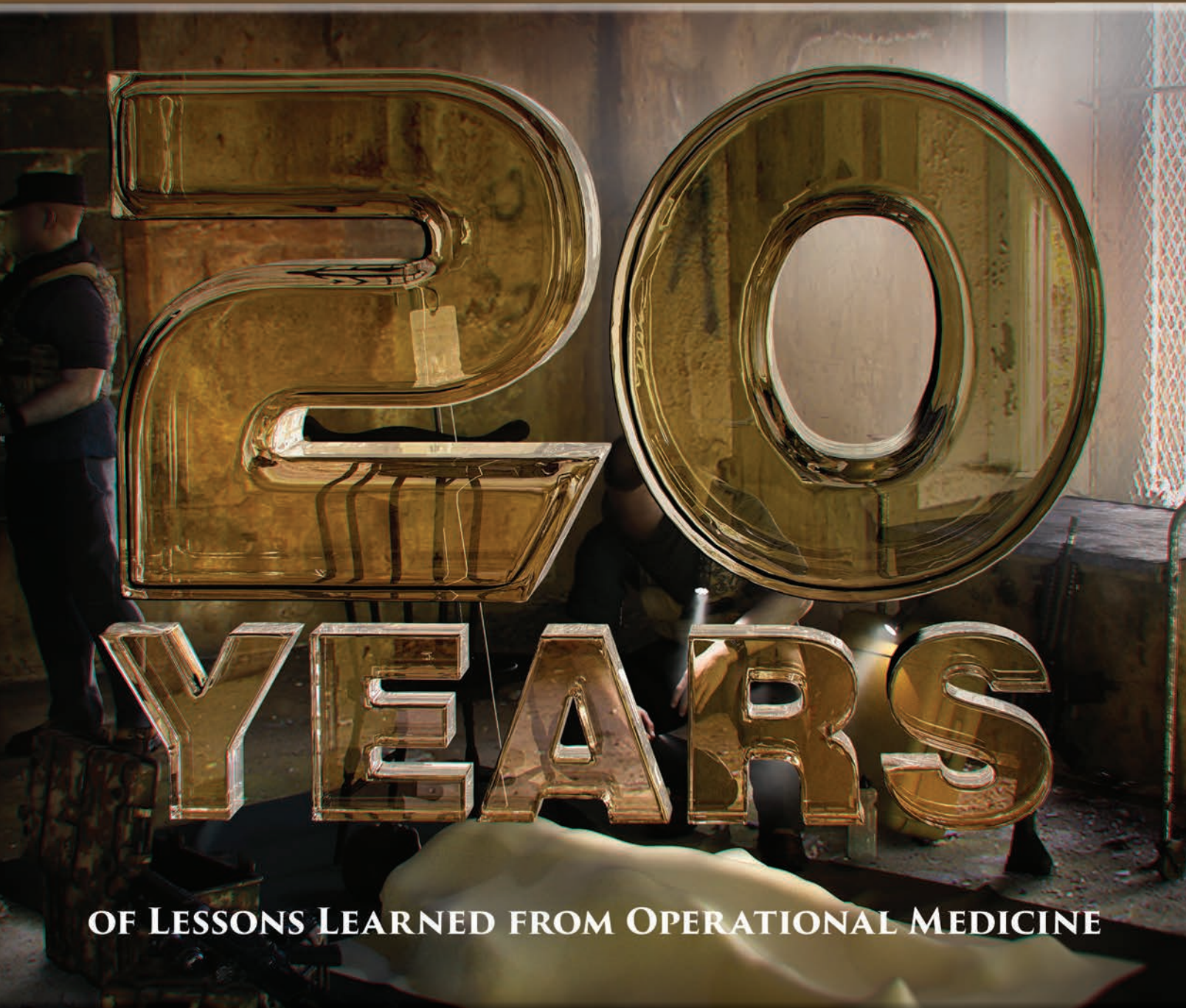
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