Lies, Damned Lies and Anesthesia Myths

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INTRODUCTION
Physicians, scientists, journalists, and the lay public prefer a plausible explanation (particularly if accompanied by a molecular mechanism) to an admission of ignorance. As a result, unproven hypotheses, opinions and plausible guesses are repeated in lectures and textbooks and become embedded in the canon of our specialty.

We will consider a representative subset of unproven (and, in some cases, disproven) hypotheses and assertions during the course of this brief presentation. The reader should judge whether these old chestnuts arise from the scheming of “liars, damned liars, or scientific experts,” (using a description of unreliable witnesses attributed to Robert Giffen), and should decide whether they now deserve to be called out as anesthesia myths.1

RESUSCITATION TOPICS
Is Normal Saline “Normal” or Beneficial? Intravenous fluid therapy arose in the 1800s as a means of combating dehydration from cholera, then became part of routine care for surgical patients in the 1900s. At present, the IV fluids of choice for adults in most surgical suites are either 0.9% (normal) saline or a “balanced” salt solution (Normosol, Plasma-lyte, or lactated Ringer’s (Hartmann’s) solution). The sad truth is that multiple lines of evidence demonstrate that use of 0.9% saline leads predictably to an increased incidence of hyperchloremia, a condition associated with worse outcomes (including longer lengths of stay and a greater likelihood of death).3-5 In the absence of hyperchloremic metabolic alkalosis there are sparse indications for large volumes of 0.9% saline, and no good reasons to use 0.9% saline as a routine maintenance solution.5

Cricoid pressure Improves patient safety during emergency intubations Cricoid pressure was introduced to medicine by Brian Sellick in 1961.7 In 26 patients considered at risk for aspiration, no regurgitation occurred during or after application of cricoid pressure in 23. In 3 patients, regurgitation occurred only after cricoid pressure was relieved following tracheal intubation. Sellick surmised that cricoid pressure had prevented regurgitation from occurring before and during intubation in these 3 patients. Nevertheless, Sellick provided no details regarding induction drugs, ventilation, patient body habitus, or other relevant factors that might also explain his findings.7

Sellick made several key assumptions.

1. That the cricoid cartilage, esophagus, and anterior surface of the vertebral body would be in constant alignment;

2. That his maneuver would fully occlude the esophagus and would prevent gastric contents from refluxing past the cricoid;

3. That his maneuver would reduce the incidence of pulmonary aspiration associated with “full stomach” conditions;

4. That cricoid pressure had no adverse consequences.

Current data using computed tomography and magnetic resonance imaging techniques show that assumptions 1 and 2 are false.7 There are no outcome studies supporting assumption 3, but such studies likely would not be feasible given rates of medically consequential aspiration during emergency surgery of ≤1 per 1000. As for assumption 4, multiple studies have shown that cricoid pressure can worsen the clinician’s view of the airway during direct laryngoscopy.8 If one were to grade the quality of the evidence supporting the use of cricoid pressure using standards of the Oxford Centre for Evidence Based Medicine, a grade no better than C could be assigned!9 In a recent survey, only 30% of Swiss and 52% of Austrian anesthesiologists use cricoid pressure as part of rapid sequence induction.9 Nevertheless, some regard cricoid pressure both standard care and standard of care.

GENERAL ANESTHETIC TOPICS
Invasive monitoring increases hemodynamic stability during induction Many books and oral examination candidates emphasize the value of invasive hemodynamic monitoring during induction of general anesthesia for “sick” patients. But is there any evidence that having information from a central line or a pulmonary artery catheter increases hemodynamic stability during induction? In a randomized comparison, inductions conducted without benefit of pulmonary artery catheter data required no more interventions to maintain stable hemodynamics than inductions “guided” by data from the pulmonary artery catheter.10 Moreover, placement of the pulmonary artery catheter after induction of general anesthesia took less time than when performed before induction. Finally, there are no convincing data showing that pulmonary artery catheterization reduces the likelihood of mortality in this or any other circumstance.11

A slow induction increases hemodynamic stability Many clinicians recommend a “slow, careful induction” in cardiac and other sick patients. But, is there evidence that a slow induction results in fewer hemodynamic perturbations than a well-conducted rapid sequence induction? In patients scheduled for coronary artery surgery, rapid sequence induction with sufentanil and succinylcholine produced similar hemodynamics and necessitated no more interventions with vasoactive drugs or IV fluid boluses than a slower (2 min) opioid-relaxant induction or a very slow, careful (5–10 min) opioid-relaxant induction.12-14

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REGIONAL ANESTHETIC TOPICS

PKa predicts speed of onset of regional anesthesia

All local anesthetic compounds (save for benzocaine) in widespread clinical use have a tertiary amine nitrogen, the protonation of which is influence by the pH of its environment. The charged (protonated) form of the local anesthetic is less membrane permeable than the uncharged (neutral base) form of the compound. It has long been assumed that when two local anesthetic compounds are compared for speed of onset, the compound with the reduced pKa will have the faster onset of action, because after injection, a larger fraction of this compound will be in the neutral form as compared to the compound with the larger pKa. The only problem with this “truism” is that it is incorrect. It is true that lidocaine has a smaller pKa and a faster onset than bupivacaine. But, chloroprocaine has the largest pKa of all and it has the fastest onset of all local anesthetics, even faster than lidocaine, disproving the “rule.” Moreover, the pKa rule fails even when used to compare structurally similar compounds given that tetracaine has a smaller pKa than bupivacaine, but has by far the slowest onset of these three drugs.

Methemoglobinemia and prilocaine

Methemoglobinemia has long been associated with prilocaine, the only local anesthetic that is metabolized to o-toluidine. According to many textbooks, prilocaine will reliably produce medically important degrees of methemoglobinemia when doses >600 mg are administered. Vasters et al. found that serious degrees of methemoglobinemia can arise after prilocaine doses as small as 400 mg in fit adult patients. Interestingly, in a North American study, the local anesthetic most commonly associated with dangerous methemoglobinemia was benzocaine.

Interscalene blocks and general anesthesia

In 2000 a report appeared in Anesthesiology describing 4 patients who experienced disastrous neurological complications after undergoing interscalene blocks while anesthetized. The author suggested (and the suggestion was repeated in an American Society of Regional Anesthesia guideline) that “Interscalene blocks should not be performed in anesthetized or heavily sedated adult or pediatric patients.” But, does the evidence show that anesthetized or heavily sedated patients are more likely to have neurologic damage? There are case reports of nerve damage after interscalene blocks performed in awake patients. Children routinely undergo nerve blocks (including interscalene blocks) while anesthetized and infrequently experience nerve damage. Moreover, large series of interscalene blocks performed in patients receiving general anesthesia report an incidence of adverse neurologic events no more frequent than that reported after interscalene blocks performed without general anesthesia.

Intraneural injections and nerve damage

William S. Halsted, the first physician to perform brachial plexus blocks in North America, injected cocaine into nerves under direct vision. Yet most modern textbooks indicate that intraneural injections must be avoided because they will consistently result in persisting deficits. Recent articles tend to emphasize the differences between intraneural injections that disrupt nerve structure and those that do not. They also emphasize the fact that unintended intraneural injections commonly take place despite use of either ultrasound guidance, motor nerve stimulation, or both, and that awake patients most often will not report symptoms during these injections.

CONCLUSIONS

There are many long-accepted practices and published guidelines in anesthesia that either are not supported or are contradicted by the available data. Myths and unproven hypotheses continue to masquerade as received knowledge in our specialty.

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