INTRODUCTION

Physicians, journalists, scientists and the lay public prefer a plausible narrative (particularly if it includes mechanistic details) to being forced to acknowledge that “I don’t know” or “the data are suggestive but inconclusive.” The inevitable result is that unproven hypotheses, repeatedly endlessly in lectures and textbooks and assumed to be facts, become part of the canon of the specialty. Whether these misapprehensions be “lies, damned lies, and statistics,” using a turn of phrase that Mark Twain attributed to Benjamin Disraeli (erroneously) in Twain’s Chapters from My Autobiography, or whether they be “anesthesia myths” I will leave to the reader/listener to decide.1

We will consider a representative subset of unproven (and, in some cases, disproven) hypotheses during the course of this brief presentation. For convenience, I have divided the topics into two classes: those related to general anesthesia and those related to regional anesthesia.

GENERAL ANESTHESIA
Cricoid pressure improves patient safety

Cricoid pressure was introduced to anesthesia by Brian Sellick article in 1961.2 In 26 patients considered at risk for aspiration, no regurgitation occurred before or after application of cricoid pressure in 23. In 3 patients, regurgitation occurred after cricoid pressure was relieved following tracheal intubation. The assumption was that cricoid pressure prevented regurgitation from occurring prior to and during intubation in these 3 patients. But, Sellick did not provide details of induction drugs, ventilation, patient body habitus, or other relevant factors that might also explain differences between the two groups.2 Sellick assumed that the cricoid cartilage, esophagus, and anterior surface of the vertebral body would be in consistent anterior to posterior alignment. He presumed that his maneuver would fully occlude the esophagus, would prevent gastric contents from refluxing past the cricoid, and thus would reduce the incidence of pulmonary aspiration associated with “full stomach” conditions. Finally, he assumed that cricoid pressure had no adverse consequences. Current data using CT and MR imaging techniques confirm that the cricoid, esophagus, and vertebral body are not in consistent alignment and cricoid pressure does not consistently occlude the esophagus. Small studies in animals and cadavers demonstrate that cricoid pressure prevents reflux of water injected at increased pressure into the esophagus, but there are no human studies on this phenomenon.4 There are no outcome studies showing a reduced incidence of aspiration with use of cricoid pressure, but such studies would not be feasible given rates of aspiration during emergency surgery of 1 per 1000 or less. As for adverse effects of cricoid pressure, multiple studies have shown that it can worsen the clinician’s view of the airway during direct laryngoscopy.5 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 D could be assigned.6 Curiously, cricoid pressure is regarded as standard of care by many.

Invasive monitoring yields a more hemodynamically stable induction

Many books and many clinicians emphasize the importance of placing hemodynamic monitors before induction of general anesthesia. But is there any evidence that having information from a pulmonary artery catheter improves 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.6 Moreover, placement of an introducer sheath and 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 improves outcomes.7

A slow, careful cardiac induction is preferable

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 intravenous fluid boluses than a slower (2 min) opioid-relaxant induction or a very slow, careful (5-10 min) opioid-relaxant induction.8,9

REGIONAL ANESTHESIA
pKa predicts speed of onset of regional anesthesia

All local anesthetic compounds (with the exception of 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.10 It has long been assumed that when
two local anesthetic compounds are compared for speed of onset, the compound with the reduced pKa will be the faster one, because after injection, a greater 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 than bupivacaine and lidocaine has a faster onset. But, chloroprocaine has the largest pKa of all and it has the fastest onset of all, 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 procaine or chloroprocaine, but has by far the slowest onset of these three agents.

Methemoglobinemia and prilocaine

Methemoglobinemia has long been linked to prilocaine, the only local anesthetic that is metabolized to o-toluidine. According to textbooks, prilocaine will reliably produce medically important degrees of methemoglobinemia when doses >600 mg are administered. Recent work by Vasters and colleagues suggests that serious degrees of methemoglobinemia can be associated with prilocaine doses as small as 400 mg in fit adult patients. Interestingly, in another recent study, the local anesthetic most commonly associated with serious degrees of 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 suggestion was made (and reinforced in an ASRA 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? In fact, large series of interscalene blocks performed in patients receiving general anesthesia report an incidence of adverse neurologic events no different from that reported in large series of interscalene blocks performed without general anesthesia. In the absence of evidence, and based only case reports, is it reasonable to issue a practice guideline that, in effect, labels the use of deep sedation or general anesthesia before interscalene block as malpractice?

CONCLUSIONS

There are a great many accepted practices and published statements in anesthesia that are not supported by strong data sets. In some cases, the available data contradict the prevailing opinion. Although there is little evidence that out and out lies are being promulgated knowingly, it is clear that myths and unproven hypotheses continue to masquerade as received knowledge in our specialty.

SELECTED REFERENCES