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Anesthesiologists' Duty of Care and Questions of Conscience

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The conflict between an individual physician's duty to treat versus their personal issues of conscience and moral integrity has become more complex in recent years (Clinical Ethics: A Practical Approach to Ethical Decisions in Clinical Medicine, 8th edition, 2015). The loss of physician autonomy due to recent societal pressures, changes in practice models, and ever-changing legal landscapes has

potentially placed additional strain on the relationship between one's professional duty to care and one's conscience, values, and beliefs. Respect for a patient's right of self-determination is in accordance with Principles I, II, IV, and VIII of the American Medical Association (AMA) Principles of Medical Ethics, as well as sections I.1. and I.2 of the ASA Guidelines for the Ethical Practice of Anesthesiology (asamonitor.pub/3XKmV6D; asamonitor.

pub/4eiWSca; *J Healthc Leadersh* 2023;15:153-60). The physician's duty to treat is not absolute, except in cases of emergency. The above documents emphasize the importance of physicians acting in accordance with their conscience while balancing professional responsibilities to patients. They stress the need for physicians to uphold ethical standards, provide care in emergencies, respect patient autonomy, and not

discriminate. They also address the duty to inform patients of services that cannot be provided due to personal beliefs before establishing a patient-physician relationship. Additionally, the documents discuss moral complicity, factors influencing it, the duty to inform patients about treatment options, and the duty to refer patients to other providers when necessary.

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Shared Perspectives: How ABA Supports ASA's Anesthesia Education Efforts

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ASA and the American Board of Anesthesiology (ABA) are separate organizations with separate missions. Together, our work is important to the practice of anesthesia and the profession of anesthesiology. The education, assessment, and certification of anesthesiologists

are upheld and delivered through the efforts of these organizations. This edition of Shared Perspectives examines how ABA supports ASA's efforts to educate, demonstrate how ASA and ABA collaborate, and consider what the future may bring.

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AIRS Case 2024-12: Managing Anomalies

A 3-month-old infant with an unspecified mitochondrial/metabolic disorder underwent general anesthesia for cardiac catheterization for VSD. The anesthetic administered was 200 mcg/kg/min of propofol for three hours. The child subsequently developed severe metabolic acidosis, circulatory collapse, myoglobinuria, and renal failure concerning for propofol infusion syndrome (PRIS) vs. metabolic crisis. Unfortunately, he progressed to multiorgan failure and death.

Dealing with rare diseases in the OR or the interventional and imaging suite is perhaps a more common occurrence

in pediatric anesthesia, as these children often require anesthesia for multiple procedures, including those for which adults can tolerate sedation, local anesthesia, or being awake. Even the most experienced pediatric anesthesiologist cannot be intimately familiar with the thousands of conditions that potentially affect the conduct of an anesthetic. Mitochondrial disorders, often thought to be exceedingly rare, may, especially in their less profound expressions, be more common than generally suspected. These alternations in cellular energetic pathways have profound

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SPECIAL SECTION

When Bad Things Happen to Good Anesthesiologists 13-21

Guest Editor: Muhammad B. Rafique, MD, FASA

AIRS Case 2024-12: Managing Anomalies*Continued from page 1*

implications for anesthetic pharmacology (*Paediatr Anaesth* 2013;23:785-93). In this brief report, we will examine an adverse event from the database from two perspectives: first, to discuss this case itself, and second, to describe specific strategies and resources for the anesthesiologist to use when confronted with these patients.

Propofol is, of course, one of the most ubiquitously used drugs in anesthetic practice and is often used as a continuous infusion for anesthesia in the cardiac catheterization laboratory. However, there are several effects of the drug on cellular metabolism that, under rare circumstances, can cause metabolic decompensation that can be irreversible in some patients. Propofol has two effects that may destabilize cellular metabolism, resulting in a crisis of cellular energetics. First, it interferes with the transport of long fatty acids into the cell, starving the cell of substrate (*Lancet* 2001;357:606-7). Second, it disrupts electron transport in the mitochondria, specifically at complexes I and IV, and possibly at complex II, leading to a breakdown in the critical mechanisms of energy production of the cell and metabolic failure (*Paediatr Anaesth* 2013;23:785-93). Thus, it is prudent to avoid propofol in patients with mitochondrial disease, although an induction dose alone (without subsequent infusion) might be safe in some patients. Total intravenous anesthesia with remifentanyl and dexmedetomidine has been used successfully in these patients. It is also important to provide a dextrose infusion during surgery, both to provide an energy substrate and to suppress fat metabolism. Minimizing fasting times is similarly important.

The effect of succinylcholine in patients with mitochondrial myopathies has not been thoroughly investigated,

This entry was written by David Polaner, MD, FAAP, on behalf of the AIRS Committee.

and although malignant hyperthermia and mitochondrial myopathies are genetically and biochemically distinct disorders, there are at least two reports of them occurring in the same patient. It is probably best to avoid succinylcholine in any of these patients.

Propofol-related infusion syndrome (PRIS) was first described in children in the early 1990s but has since been reported in adults. It is characterized by otherwise unexplained metabolic acidosis, hyperlipidemia, rhabdomyolysis (which in turn results in acute renal failure), hepatomegaly, dysrhythmias, and rapidly progressive cardiac failure. Without early recognition and institution of supportive care, it is often fatal. Prolonged use of propofol (>48 h at infusion rates >65 mcg/kg/min) has been identified as a risk factor, although it is suspected (but unproven) that undiagnosed mitochondrial cytopathies, even mild ones, might be predisposing factors (*Crit Care Med* 2018;46:e91-4). Indeed, it is unknown whether patients who developed PRIS during much shorter timeframes, such that occur with propofol-based anesthetics, might have subclinical underlying mitochondrial defects. There are indications that even in “normal” children and adults without overt PRIS, evidence can be detected for subtle effects on both mechanisms of disrupted energetics (*Crit Care Med* 2018;46:e206-12; *Anesthesiology* 2007;106:1134-8). Certainly, the defining line between these two conditions in some situations is blurred, yet the physiological and clinical courses coalesce as full-blown metabolic crisis ensues. It also must be noted that in some mitochondrial disorders, exquisite sensitivity to volatile anesthetics can be present, and deep levels of anesthesia can be seen with very low end-tidal concentrations of sevoflurane (*Anesth Analg* 2021;133:924-32). The use of a processed EEG monitor can be very helpful in avoiding relative anesthetic “overdose” in these patients.

In this tragic report to the AIRS database, both diagnoses were correctly considered in the differential diagnosis after metabolic decompensation and organ failure developed. Although we cannot make a definitive diagnosis based on the limited information that the anesthesiologist reported, the existence of an uncharacterized mitochondrial defect suggests that this patient was likely at some increased risk for metabolic decompensation and failure.

When confronted with a patient with an unusual genetic or metabolic condition, where should one turn to for easily accessible information? There are three textbooks and two websites that anesthesiologists might find most helpful. The classic text in the field, “Smith’s Recognizable Patterns of Human Malformation,” is focused on anatomic conditions, but many of these have physiological consequences as well (Smith’s Recognizable Patterns of Human Malformation. 8th ed, 2021). The virtues of this book include its many photographs and an outline format notable for both brevity and ease of reading. There are two indispensable anesthesia-specific texts. The first, by Baum and O’Flaherty, “Anesthesia for Genetic, Metabolic and Dysmorphic Syndromes of Childhood,” is organized alphabetically, with concise and clear entries (*Anesthesia for Genetic, Metabolic and Dysmorphic Syndromes of Childhood*. 3rd ed, 2015). The second, “Syndromes: Rapid Recognition and Perioperative Implications,” by Bissonnette, Luginbuehl, and Engelhardt, is more encyclopedic and expansive (*Syndromes: Rapid Recognition and*

Perioperative Implications. 2nd ed, 2019). Because online resources are easily accessible, there are two web-based databases that anesthesiologists should add to the favorites tab of their browsers. Frances Veyckemans and Jean-Louis Scholtes have detailed the anesthetic implications of thousands of syndromes and congenital conditions (asamonitor.pub/3U1zliP). It is up to date and referenced, although the references are not hyperlinked. The Mendelian Inheritance in Man compendium began as an encyclopedic textbook by McKusick but is now available online – and it is the most comprehensive information site about syndromes and congenital disorders (omim.org). It contains links to every paper ever indexed and published on each condition, and you can search by findings as well as the syndrome’s name. Click on “clinical synopsis” for a concise outline of the critical aspects of the syndrome. Although extraordinarily complete and continually updated, it offers less anesthesia-specific content.

Rare metabolic diseases require special care and management; however, even “normal” patients might have subclinical variations in the function of the respiratory chain enzymes that place them at risk for increased sensitivity to anesthetics. The more we learn about the biochemical effects of the agents we use daily, and about the many genetic polymorphisms that affect cellular energetics, we can expect that we will uncover a multitude of subtleties in the cellular responses to anesthetics that can have sometimes profound effects on their outcomes. ■

Each month, the AQI-AIRS Steering Committee abstracts a patient history submitted to AIRS and authors a discussion of the safety and human factors challenges involved. Absence of commentary should not be construed as agreement with the clinical decisions described. Reader feedback can be sent to airs@asahq.org. Report incidents at aqiairs.org.

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