There is substantial variability in patients’ response to medications. The healthcare system is in the midst of a transformation to a targeted precision health approach in which disease treatment and prevention take into account individual genetic variability. This change is informed by studies, which show that genetic variations alter the structure and function of proteins such as drug transporters, drug-metabolizing enzymes, and receptors. Tailoring medication administration based on genetic makeup can minimize adverse effects and maximize efficacy. As a result, many healthcare centers have begun incorporating genomic information into healthcare decision making. Unfortunately, many anesthesia providers may be unfamiliar with the genetics concepts and principles underlying variability in patients’ response to medication. This article reviews genetic diversity in humans and the various ways in which this genetic variability may influence pharmacokinetics and pharmacodynamics of drugs. This knowledge will ensure that anesthesia providers can effectively tailor anesthesia care and postoperative pain management to improve outcomes.

Keywords: Pharmacogenetics, pharmacogenomics, postoperative pain, primer, review.
the same or may be different versions of the gene. The most common form is referred to as the major or wild-type allele, while the other forms are referred to as minor alleles. The frequency of the variant allele in the population is used as the basis for classifying variations as mutations and polymorphisms. By convention, any inherited genomic variant in a population with a frequency of greater than 1% is termed a polymorphism, and a variant with a population frequency of less than 1% is termed a mutation. The frequency at which the second most common (mutant) allele occurs in the given population is known as the minor allele frequency. Moreover, the allele frequency can vary from one population (e.g., race or ethnicity) to the next.

Human genomic variations can also be distinguished by the nature of the variant: quantitative (change of gene dose, defined below) and qualitative (affecting single nucleotides) variants. Regarding quantitative variants, humans inherit most genes as 23 pairs of chromosomes (46 total): 22 pairs of autosomes and 1 pair of sex chromosomes. The quantitative description of genetic material is referred to as the gene dose. Having the right number of genes is essential for normal function; lesser or greater than 46 chromosomes is associated with disease and disorders. For instance, about half of patients with Turner syndrome have only 1 sex chromosome (typically 1 X chromosome and no Y chromosome), which results in 45 total chromosomes. These patients have characteristic short webbed neck, kidney problems, and cardiac defects. On the other hand, most cases of Down syndrome result from having an extra copy of chromosome 21 (3 instead of 2 copies of chromosome 21, trisomy 21), which results in 47 total chromosomes. Similarly, lesser or greater dosages of standard gene copies are related to altered protein function. For instance, lack of a gene that codes for a drug-metabolizing enzyme may result in a decrease in the rate of metabolism of that drug, whereas having multiple copies of that gene may increase the rate of metabolism of that drug. On the other hand, in qualitative variations, individuals have the “normal” quantity of genetic material, but variations in the content of the genes result in differences in the structure and function of proteins (quality of the proteins). A single nucleotide variation such as substitution of one nucleotide for another on a gene is an example of a qualitative variation.

**Single Nucleotide Variations.** Single nucleotide variations that occur in more than 1% of the population are referred to as single nucleotide polymorphisms (SNPs, pronounced “snips”). SNPs are the most common type of genetic variations among humans. The human genome contains approximately 3 billion base pairs (nucleotide pairs), and each SNP represents a difference in a single nucleotide. For instance, a SNP may be the substitution of the nucleotide adenine (A) for thymine (T) on the DNA sequence. About 10 million such differences (SNPs) have been identified in the human genomes. Most SNPs have no clinical relevance because the difference in nucleotide does not result in a change in the amino acid sequence of the protein, or it occurs in regions that do not code for or regulate protein synthesis. A SNP that does not result in a change in the amino acid sequence is known as a synonymous polymorphism, whereas one that results in a change in the amino acid sequence is known as a nonsynonymous polymorphism.

Nonsynonymous SNPs change the amino acid sequence of proteins and may alter the structure and function of those proteins. For instance, substitution of guanine with adenine on the catechol-O-methyltransferase (COMT) gene results in the amino acid change from valine to methionine at codon 158 (Val158Met). This nonsynonymous change alters the structure and function of the COMT enzyme, which breaks down catecholamines such as dopamine in the brain. In this example, the wild-type allele, guanine (G), codes for the amino acid valine, whereas the minor allele (variant), adenine (A), codes for the amino acid methionine. In the postoperative period, having the minor allele (A) has been associated with decreased enzyme activity and decreased morphine consumption. This type of SNP that substitutes one amino acid for another is known as a missense mutation. A mutation resulting in the premature termination of the amino acid sequence (a shortened protein) is referred to as a nonsense mutation. Clinically significant SNPs affect the quality of the gene and resultant protein.

**Structural Variations.** Unlike SNPs that primarily affect gene quality, deletions, insertions, copy number variation, and tandem repeats affect the quantity of the gene. Deletion results in the removal of a segment of the DNA. The segment of the DNA deleted can vary from a few nucleotide pairs within a gene to entire genes or several genes on a chromosome. The absence of the deleted segment on the DNA alters the structure and function of the resultant protein or proteins. Just as deletion can result in loss of functions, insertion of extra copies of genetic material can alter the function of the protein. The genetic material inserted can include nucleotides, gene segments, genes, or chromosome segments. Insertions and deletions are collectively referred to as “indels” because the genetic material deleted from one segment may insert into another segment of a homologous chromosome. In some instances, indels cause genes to be copied an abnormal number of times, resulting in gene duplication or copy number variation. Duplication of the normal gene may result in increased activity of the resultant protein. For instance, individuals classified as ultrarapid metabolizers inherit more than 2 copies of the normal CYP2D6 gene. This gene codes for the enzyme that converts codeine into morphine. Duplication of the gene results in increased enzymatic activity and rapid
conversion of codeine into morphine, increasing the risk of morphine toxicity.³

Indels can result in a special kind of duplication in which a short DNA sequence that involves a repetitive unit of 2 to 6 base pairs is repeated a variable number of times end-to-end at a defined locus. Such repetitive DNA sequences, also called satellite DNA, are grouped into 2 main categories: microsatellites and minisatel-

lies. Microsatellites, also known as short tandem repeats (STR), are tandem repeats of 2, 3, or 4 nucleotide repeat units that occur in 5 to 25 copies. In STR, different alleles are the result of differing numbers of repeated nucleotide units. Minisatellites, on the other hand, are an array of 100 to more than 1,000 copies, in tandem, of 10 to 100 nucleotide repeats. Minisatellites, also known as variable number tandem repeats (VNTR), usually have many alleles due to the variation in the number of copies of tandem repeats. Tandem repeat polymorphisms are usually groups of indels because of their variation between individuals. These DNA segments are used by the Federal Bureau of Investigation for identifying indi-

viduals as suspects at a crime scene. Hence, they are referred to as DNA “fingerprints.” A typical DNA fingerprint includes more than a dozen VNTR.

Overview of Pharmacogenetics
The terms pharmacogenetics and pharmacogenomics are often used interchangeably. However, there is a subtle difference. Although pharmacogenetics refers to the effect of single genes on drug response, pharmacogenomics encompasses the relationship between the genomic varia-

tions and drug response. Pharmacogenomics uses a genomewide approach to investigate genomic relationships with drug response, with no a priori knowledge of the role of the genes. Rather, the entire genome is queried for asso-

ciation with a specific drug response. Pharmacogenetics, on the other hand, uses a candidate gene approach in which genes with known effects are investigated for association with drug response based on a hypothesis.

Pharmacogenomics and pharmacogenetics play 2 important roles in precision medicine. First, they guide the pharmaceutical industry in drug discovery and de-

velopment. Second, they guide healthcare providers in selecting the right drug for the right patient, at the right dose, and right frequency based on the patient’s genetic makeup, to maximize efficacy and minimize adverse effects. Genetic variability may affect pharmacokinet-

ics (drug transport proteins, and drug-metabolizing enzymes), pharmacodynamics (drug-receptor proteins), and associated downstream responses, which ultimately produce a therapeutic effect or adverse reaction. It is important to remember that nongenetic factors such as age, disease, environment (smoking, diet, alcohol), and drug interactions may also influence a patient’s re-

sponse to medication.

Pharmacokinetic Variability
Pharmacokinetics refers to the absorption, transport, metabolism, and excretion of drugs. Drug transporters regulate the movement of drugs across basal epithelial cells and cell membranes to reach their target receptors. They are localized in organs such as the small intestine, the liver, and the kidney, which are critical for absorption and elimination of drugs. In addition, they are found in specialized barriers such as the blood-brain barrier, where they regulate the concentration of drugs in the central nervous system. Besides drug transporters, metabolism also affects the concentration of drugs at the receptor site. Drug metabolism is frequently divided into phase 1 and phase 2 metabolism, which converts lipophilic (fat-soluble) drugs into hydrophilic (water-soluble) molecules for elimination. Phase 1 metabolism is characterized by oxidation, hydrolysis, and reduction processes, which start the detoxification of active drugs or conversion of prodrugs into active drugs. On the other hand, phase 2 metabolism is characterized by glucuronida-

tion, acetylation, and sulfation reactions (catalyzed mainly by transferases). Collectively, genetic variations that affect the structure and functions of drug transporters and drug-metabolizing enzymes may affect pharmacokinetics and effects of the drug. The next sections will discuss the genetic bases of variability in drug transport proteins and drug-metabolizing enzymes.

Drug Transport Proteins
Drug transport genes encode the production of mem-

brane proteins that regulate the movement of drugs into or out of cells or specialized junctions such as the blood-

brain barrier. The most common transporters belong to 2 superfamilies, ABC (ATP-binding cassette) and SLC (solute-linked carrier). Although SLC transporter is a gradient facilitator, ABC uses energy to transport drugs against a concentration gradient. Studies have shown that the ABC transporters control the efflux of drugs such as morphine across the blood-brain barrier, thereby affecting their pharmacokinetic properties.⁷ As a result, the ABC superfamily of drug transporters is a major target for variability in response to pain medications.

The ABC gene encodes the ABC drug transporters. This superfamily of transporters includes the ABC subfamily B member 1 (ABCB1), which has become one of the most widely studied and best-characterized members of the ABC superfamily. The ABCB1 gene, also known as the multidrug-resistant protein 1 (MDRI) gene, encodes the P-glycoprotein 1 (P-gp) that pumps foreign substances out of cells (cellular efflux transporter) against its concentra-

tion gradient.⁸ The ABCB1 gene is expressed in various tissues, including the liver, kidneys, intestine, lungs, placenta, and brain.⁹ Some studies have reported associations of polymorphisms of the ABCB1 gene with variability in response to opioids in the postoperative setting.⁷ 8 10 14
Drug-Metabolizing Enzymes

The cytochrome P450 (CYP) and UDP glucuronosyltransferase (UGT) superfamilies of enzymes metabolize most drugs. CYP enzymes catalyze the phase 1 metabolism of most analgesic medications, while UGT enzymes catalyze the phase 2 metabolism. It has been estimated that polymorphisms in drug-metabolizing enzymes account for 10-fold to 10,000-fold variations in drug activity. The variations can be divided into those CYP and UGT polymorphisms.

- **Cytochrome p450.** The CYP superfamily comprises about 57 enzymes, which are divided into families, subfamilies, isoenzymes, and alleles according to their share sequence homology. Enzymes in families 1, 2, and 3 are polymorphic and are responsible for 70% to 80% of phase 1 metabolism of clinically useful drugs. Polymorphisms in CYP genes can produce enzymes with abolished, reduced, normal, or increased enzyme activity. Results of genetic testing of CYP enzymes are frequently reported as star (*) alleles, and nomenclature of the CYP star alleles has previously been explained. Briefly, enzymes in the same family (CYP2) share 40% amino acid variants, whereas those in a subfamily (eg, CYP2B, CYP2D) share 55% amino acid variants. The number after the letter identifies the specific isoenzyme (eg, CYP2D6, CYP2C9, CYP2B6), and the *number is used to designate specific allele variants (eg, CYP2D6*1, CYP2D6*3).15

- **CYP2D6.** Variations in CYP enzyme activity was first reported for CYP2D6 gene, which is a highly polymorphic gene located on chromosome 22q13.1. Despite the fact that it accounts for only about 2% to 4% of hepatic metabolism, CYP2D6 is the most studied CYP enzyme consisting of more than 100 alleles. Given that CYP2D6 is subject to deletions and gene duplications and multiplications, functional polymorphisms in the CYP2D6 alleles are frequently classified by enzyme activity: poor metabolizers (PM), intermediate metabolizers (IM), extensive metabolizers (EM), and ultrarapid metabolizers (UM). The Table summarizes phenotypic classification, some functional alleles, incidence in the population, impact on CYP2D6 enzyme function and clinical considerations. Sufficient to mention that while PMs have no functional alleles, UMs have duplicate or multiduplicate copies of the normal CYP2D6 alleles. Unlike most CYP genes that are inherited as diplootypes, UMs can inherit 3 or more copies of the CYP2D6 gene. In fact, carriers of up to 13 copies of the functional allele have been identified. Many studies have explored the impact of CYP2D6 polymorphism on postoperative pain management. The Clinical Pharmacogenetics Implementation Consortium has published guidelines for genotype-guided clinical use of codeine.

- **CYP2C9.** In the human liver, CYP2C9 is one of the most abundant enzymes, accounting for about 20% of the total hepatic CYP content. It is located on chromosome 10q24 and metabolizes several clinically relevant drugs, including warfarin, antibiotics, antihypertensive agents (β-blockers), cannabinol, and nonsteroidal anti-inflammatory drugs (NSAIDs, including ibuprofen, naproxen, indomethacin, meloxicam, and diclofenac). To date, about 60 alleles have been identified in the human CYP2C9 gene, with CYP2C9*2 (R144C) and CYP2C9*3 (I359L) classified as variants with low enzyme activity (poor metabolizers). Several studies have reported that adverse effects of NSAIDs are associated with the presence of CYP2C9*2 and *3 alleles.

- **CYP2B6.** CYP2B6 has been mapped to chromosome 19q13.2 and is expressed in the liver, skin, brain, kidney, lung, and right heart ventricle. CYP2B6 is one of the most polymorphic genes containing over 100 SNPs, many haplotypes, a variety of ethnic variabilities and controversy regarding differences in liver expression related to gender. The enzymes oxidize steroids, fatty acids, and xenobiotics, such as the substrates including antidepressants (bupropion), anesthetics (propofol and ketamine), and synthetic opioids (methadone) to date, 37-star alleles of the CYP2B6 isoenzyme with distinct amino acid sequences have been identified. These include CYP2B6 *6A, *16, and *26, which have decreased activity, and CYP2B6*4A, which has increased enzyme activity. CYP3A4. The CYP3A4 gene is part of the CYP450 genes found on chromosome 7q21.1, and CYP3A4 enzymes metabolize approximately 50% of medications on the market today. Inhibition and ease of induction of CYP3A4 and CYP3A5 are quite common and may contribute to adverse effects of the medications that use these pathways, such as ketamine. Some inhibitions, as with grapefruit, may be related to the presence of intestinal CYP3A4. Studies have indicated that CYP3A4 substrates are metabolized more quickly in females compared with males. Many potent inhibitors that have been identified include clarithromycin, erythromycin, verapamil, and grapefruit. Some inducers of the CYP3A4/3A5 enzymes include medications such as phenobarbital, phenytoin, and glucocorticoids.

- **Glucuronosyltransferase.** The uridine-diphosphate glucuronosyltransferases (UGT) catalyze the addition of glucuronic acid to lipophilic medications or their metabolites to form hydrophilic metabolites. The UGT superfamily of enzymes contains 2 families, UGT1 and UGT2, according to their primary amino acid sequence homology. The UGT1 enzymes catalyze the glucuronidation of endogenous compounds (eg, bilirubin) and drugs. The isoenzyme UGT1A3 contributes to the glucuronidation of many drugs, including hydromorphone. Variants of the UGT1A3 SNPs show variable enzyme activity. UGT2B7 catalyzes the glucuronidation of corticosteroids and important drugs such as codeine, hydromorphone, morphine, oxycodone, and oxymorphone. The UGT2B7
enzyme is polymorphic, and variants of the UGT2B7 show variable enzyme activity. For instance, patients, who are homozygous for UGT2B7 802C needed less morphine in the postoperative period for pain relief in one report.

Pharmacodynamic Variability

Pharmacodynamic variability refers to the variations in the interaction of drugs with receptors or intracellular signal transduction. Genetic variations of the drug-receptor proteins may affect the affinity of the drug for its receptor, with a resultant alteration in drug efficacy and drug toxicity. Drug-receptor genes of relevance to postoperative pain management include μ-opioid receptor (OPRM), COMT, and cyclooxygenase (COX) genes.

• Mu-Opioid Receptor Gene. The opioid receptor μ 1 (OPRM1) gene encodes the μ-opioid receptor, which is the primary site of action for endogenous and exogenous opioids such as β-endorphin and encephalin. The OPRM1 gene has more than 100 SNPs, with the most well-characterized variants being the A118G, which is located on chromosome 6q25.2. Several studies have shown that carriers of the minor allele (G) reported higher pain and required higher doses of opioids to achieve adequate pain management relief in the postoperative period. However, other studies have found that carriers of the A alleles reported lower pain scores and required less opioid for pain relief. A study of 196 women did not find any association between OPRM1 genetic variants and postoperative fentanyl requirements. Thus, even though the OPRM1 A118G variant appears to influence opioid analgesia, its role in postoperative pain management remains inconclusive.

• Catechol-O-Methyl Transferase Gene. The COMT gene encodes the COMT enzyme, which is one of the several enzymes that metabolize catecholamines such as dopamine, epinephrine, and norepinephrine. These neurotransmitters play an important role in modulating response to pain. SNPs in the COMT gene account for about 10% of the variability in sensitivity to pain. A study of 196 women did not find any association between OPRM1 genetic variants and postoperative fentanyl requirements. Thus, even though the OPRM1 A118G variant appears to influence opioid analgesia, its role in postoperative pain management remains inconclusive.

Table. Genotype and Description of CYP2D6 Alleles

<table>
<thead>
<tr>
<th>Phenotype</th>
<th>Rate of metabolism</th>
<th>Clinical effect</th>
<th>Clinical recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poor metabolizers (PM)</td>
<td>None</td>
<td>Unable to convert prodrug into active drug, resulting in lack of efficacy</td>
<td>Avoid prodrugs due to lack of response (eg, no conversion of codeine to morphine)</td>
</tr>
<tr>
<td>Intermediate metabolizers (IM)</td>
<td>Reduced</td>
<td>Lack of metabolism of active drug with resultant accumulation and toxicity in reduced efficiency</td>
<td>Avoid or reduce dose of active drug (eg, tramadol) to reduce risk of toxicity</td>
</tr>
<tr>
<td>Extensive metabolizers (EM)</td>
<td>Normal</td>
<td>Convert prodrug into active drug at a very slow rate, resulting in reduced efficacy</td>
<td>For active drug (eg, tramadol) monitor closely for side effects</td>
</tr>
<tr>
<td>Ultrarapid metabolizers (UM)</td>
<td>Rapid</td>
<td>Metabolize active drug at a very slow rate with higher risk of side effects</td>
<td>For active drug (eg, tramadol) monitor closely for side effects</td>
</tr>
</tbody>
</table>

Table. Genotype and Description of CYP2D6 Alleles

<table>
<thead>
<tr>
<th>Genotype (CYP2D6)*</th>
<th>Incidence (%)</th>
<th>Clinical effect</th>
<th>Clinical recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>*9</td>
<td>Whites: 51</td>
<td>Lack of metabolism of active drug with resultant accumulation and toxicity</td>
<td>Avoid or reduce dose of active drug (eg, tramadol) to reduce risk of toxicity</td>
</tr>
<tr>
<td>*10, *17, *29, *41</td>
<td>Whites: 1-2</td>
<td>Convert prodrug into active drug at a very slow rate, resulting in reduced efficacy</td>
<td>For active drug (eg, tramadol) monitor closely for side effects</td>
</tr>
<tr>
<td>*1</td>
<td>Whites: 71</td>
<td>Convert prodrug to active drug at normal rate</td>
<td>Administer the recommended dose</td>
</tr>
<tr>
<td>*2</td>
<td>Whites: 51</td>
<td>Convert prodrug into active drug at a very slow rate with higher risk of side effects</td>
<td>For active drug (eg, tramadol) monitor closely for side effects</td>
</tr>
<tr>
<td>*3, *29</td>
<td>Asians: 54</td>
<td>Metabolize active drug at a very slow rate with higher risk of side effects</td>
<td>Administer the recommended dose</td>
</tr>
<tr>
<td>*1 x N, *2 x N</td>
<td>Whites: &lt; 2-4 African Americans: 2-4</td>
<td>Fasting rate resulting in risk of toxicity</td>
<td>Avoid prodrug due to high risk of toxicity (eg, rapid conversion of codeine to morphine, may result in morphine toxicity)</td>
</tr>
</tbody>
</table>

Table. Genotype and Description of CYP2D6 Alleles

<table>
<thead>
<tr>
<th>Incidence (%)</th>
<th>Clinical effect</th>
<th>Clinical recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>*2</td>
<td>Normal</td>
<td>Administer the recommended dose</td>
</tr>
<tr>
<td>*4, *12, *39</td>
<td>Normal</td>
<td>Administer the recommended dose</td>
</tr>
<tr>
<td>*1 x N</td>
<td>Fasting rate resulting in risk of toxicity</td>
<td>Avoid prodrug due to high risk of toxicity (eg, rapid conversion of codeine to morphine, may result in morphine toxicity)</td>
</tr>
</tbody>
</table>
associated with more fentanyl consumption 24 hours after radical gastrectomy.\textsuperscript{30} 

- **Cyclooxygenase Gene.** The COX 1 and 2 receptors are encoded by the prostaglandin-endoperoxide synthase 1 (PTGS1) and prostaglandin-endoperoxide synthase 2 (PTGS2) genes, respectively. Genetic polymorphism in these genes would be expected to be associated with variability in response to NSAIDs. However, the functional effects of the SNPs in the PTGS1 and PTGS2 genes remain unknown.\textsuperscript{31}

**Conclusion**
Implementation of pharmacogenomics into clinical practice is rapidly progressing in many institutions across the United States. The FDA has approved genetic insert for more than 100 drugs that are currently in clinical use. These include frequently used medications such as codeine, tramadol, amitriptyline, lidocaine, and prilocaine. Genetic polymorphisms alter the efficacy and toxicity of these medications. As the cost of pharmacog- enomic testing decreases and as direct-to-consumer testing increases, correct interpretation and utilization of actionable genetic information may become a standard of care.

**REFERENCES**


29. Martis S, Mei H, Vizjelaar R, Edelmann L, Desnick RJ, Scott SA.
National and state legislative decisions have an impact on the care that Certified Registered Nurse Anesthetists (CRNA) provide their patients. Historically, professional advocacy resulted in CRNA title recognition and direct reimbursement for CRNA services and led to providing states an opt-out option for medical supervision when billing Medicare and Medicaid. It is critical that CRNAs continue to grow as advocacy leaders and, in preparing for this role, each CRNA must be provided with the knowledge and skills to be successful. The objective of this research project was to determine the impact that CRNA advocacy education in Pennsylvania nurse anesthesia educational programs has on students’ professional involvement. Two surveys were distributed to all 12 Pennsylvania nurse anesthesia educational programs to determine the quality and quantity of advocacy education incorporated into their program and how it affected the professional political involvement of senior student registered nurse anesthetists. Pearson r calculations were conducted to assess for correlations between variables. The results showed a strong positive correlation between advocacy education in nurse anesthesia educational programs and the impact it has on students’ professional advocacy involvement (r = 0.481, P = .001).

Keywords: Advocacy education, CRNA advocacy, CRNA advocacy education, nurse anesthesia, nurse anesthesia advocacy education.

Developing professional leadership skills, specifically, advocacy efforts for Certified Registered Nurse Anesthetists (CRNAs), early in a CRNA’s career is arguably more important now than ever before. It is a pivotal time in nurse anesthesia practice when legislators are making decisions that will affect our scope of practice and title recognition as advanced practice nurses.1 Political issues vary by state and are closely related to scope of practice restriction, reimbursement imparity, and professional encroachment, specifically by anesthesiologist assistants. In some regions of the United States, CRNAs are not permitted to use all the skills they are trained to perform, which impedes professional autonomy and constrains scope of practice.1,2 In Pennsylvania, the scope of practice for CRNAs is restricted by state and facility regulations.2

Pennsylvania is 1 of only 2 states that does not recognize the CRNA title under statute. Without an official CRNA designation, the credentialing process for CRNAs and student registered nurse anesthetists (SRNAs) can be delayed up to 6 months.2 Of major importance, this lack of designation impedes military CRNAs and CRNAs who volunteer for disaster relief efforts outside Pennsylvania.2 Reciprocally, CRNAs who wish to relocate to Pennsylvania may face similar issues. Therefore, it is critical for CRNAs to identify these challenges and maintain political vigilance to promote our profession.

A recent study demonstrated that nurses who receive advocacy education are more likely to have political skills that will enhance their ability to educate legislators on all aspects of clinical practice.3 However, lack of formal education on negotiating the political arena hinders CRNAs’ political astuteness.4 According to Mund,4 in 2012, out of 105 nurse anesthesia programs in the United States, only 25 programs required students to enroll in a health policy class. This lack of standardized advocacy education may lead to a lack of policy knowledge, resulting in weak political skills and reduced political interest.

Lack of professional advocacy may stem from 2 main factors: (1) the shortage of leaders to mentor colleagues and students and (2) limited exposure to advocacy concepts in nurse anesthesia educational programs.4 The Council on Accreditation of Nurse Anesthesia Educational Programs (COA) requires that each nurse anesthesia education program include 45 hours of professional aspects content in the curriculum.5 A professional aspects course in nurse anesthesia educational programs presents the SRNA with information about the profes-
sional discipline of nurse anesthesia, including ethical, legal, and policy issues. However, the content covered in these required courses is at the discretion of the program administrator. Some program administrators may choose to teach students about the ethical considerations of anesthesia, but place less emphasis on policy issues. As a result, many students may not be exposed to advocacy concepts during nurse anesthesia school.

The aim of this research project was to determine the impact that CRNA advocacy education in nurse anesthesia educational programs has on students’ professional involvement. The first research objective was to determine, using surveys, whether Pennsylvania nurse anesthesia educational program administrators incorporate professional advocacy concepts into the curriculum. The second objective was to determine whether Pennsylvania SRNAs have received advocacy education in their nurse anesthesia educational program and whether they have participated in advocacy events. The final objective was to correlate advocacy education provided by program administrators with SRNAs’ advocacy involvement.

Review of the Literature
Using EBSCO Host and PubMed, a literature review was performed on CRNA advocacy, advanced practice registered nurse (APRN) advocacy, and advocacy education. Search terms entered into EBSCO host were CRNA AND advocacy, CRNA AND politics, APRN AND advocacy, and CRNA advocacy AND anesthesia school. The search term entered into PubMed was CRNA advocacy in nurse anesthesia school. The search yielded 7 relevant articles. After a review of each article’s references, another 5 articles were considered relevant. A total of 12 articles were critically appraised using John Hopkins Nursing Evidence-Based Practice Research Evidence Appraisal Tool and were considered pertinent to the research topic. To gather data related specifically to Pennsylvania CRNA advocacy and nurse anesthesia educational programs’ education requirements, the authors accessed the Pennsylvania Association of Nurse Anesthetists website and the COA website.

Each article was evaluated using John Hopkins Nursing Evidence-Based Practice Level and Quality Guide. Of the 12 pertinent articles, none were graded Level I, 3 were Level II, 7 were Level III, none were Level IV, and 2 were Level V. Six of the articles were graded high quality, and 6 were graded good quality.

It is important to note that even though a Level V opinion article is less credible, in this case, it revealed relevant information on advocacy education pilots. For example, Barbara B. Little, DNP, MPH, a senior teaching faculty member from Florida State University Doctor of Nursing Practice (DNP) Program, implemented an advocacy program in 2015, named Advocacy Days. This
program is a 2-day event that not only exposes DNP students to their local legislators but also allows the students to observe health policy debates by watching a committee meeting. Thus far, the reported results are promising. Students testified that this program boosted their interest in advocacy, reporting that they would now feel less intimidated participating in professional activism.

The review of the literature also concluded that the role of a CRNA has evolved since the late 1800s. In 1956, successful professional advocacy resulted in CRNA title recognition, and professional advocacy resulted in direct reimbursement for CRNA services in 1986. Most recently, advocacy has led to giving states an opt-out option for medical supervision. The opt-out rule permits the governor of each state to determine if the requirement for a physician to supervise a CRNA should be lifted. To date, 17 states have opted out of this federal supervision requirement, allowing CRNAs to practice without physician supervision.

Research not only supports the importance of professional advocacy but also indicates that the nursing profession lacks formal leadership development in nursing programs. The material covered in nursing school may focus on management techniques, but not necessarily on leadership skills. Nurse anesthesia educational programs could set the educational framework necessary to support CRNA advocacy and leadership.

Materials and Methods

This study received institutional review board (IRB) approval from La Roche College, Pittsburgh, Pennsylvania. Two surveys were sent to all 12 Pennsylvania nurse anesthesia educational programs. To maintain anonymity of the nurse anesthesia educational programs, each program was randomly assigned a program number from 1 to 12.

A survey was sent to program administrators to determine the quantity and quality of advocacy education presently incorporated into their program (Figure 1). A different survey was sent to all senior students (those who had completed a minimum of 1 year of anesthesia school) to determine their perception of advocacy training in their nurse anesthesia education program and whether that training has affected their professional political involvement (Figure 2). To ensure survey validity, the authors used a 7-step process established by the Association for Medical Education in Europe (AMEE) for the survey design.

The goal of the research was to analyze program
administrators’ and senior students’ responses independently and then together to demonstrate a correlation between nurse anesthesia educational programs that implemented advocacy education and SRNAs’ self-reporting of professional involvement.

The barriers to implementing this study included gaining program administrators’ cooperation. Another barrier was procuring student involvement in the survey considering their already heavy workloads. Adding more information to a program’s curriculum may not be feasible because of time constraints. However, nurse anesthesia educational programs are required to offer a doctorate entry-level program beginning in 2022. This requirement will increase the total length of nurse anesthesia educational programs to a minimum of 36 months and will subsequently increase the number of credit hours required for graduation. Therefore, this study is well-timed because it may coincide with the addition of new advocacy curriculum to fulfill a percentage of these credit hours.

Data collection for the project started on October 31, 2016. An initial email was distributed to all nurse anesthesia program administrators advising them of the study and informing them that within the following 2 weeks they would receive an email with 2 survey links: 1 for the program administrator and the second for their SRNAs. The surveys were created in SurveyMonkey and distributed via email to each program administrator. The email requested that each program administrator complete the program administrator survey and forward the student survey to all his or her current SRNAs. Responses were anonymous and confidential. Return of the voluntary survey was considered implied participant consent.

Four program administrators completed the survey, and 72 SRNAs completed the survey after the first request. A second email was sent as a reminder to each administrator. On January 23, 2017, data collection ended after a total of 6 program administrators and 94 SRNAs completed the survey. Surveys were analyzed in their original format. Pearson *r* calculations were conducted to look for correlations between survey response variables using statistical analysis software (IBM SPSS Statistics 21).

**Results**

Of 94 responses collected from students, 54 participants from 8 nurse anesthesia educational programs self-identified as seniors who had completed a minimum of 1 year of anesthesia school. Figure 3 shows the percentage of qualifying participants by nurse anesthesia educational program. As outlined in the project design, only data for senior students were analyzed.

During the analyses, some variables were combined to create new variables that better encompass the participants’ experiences within their nurse anesthesia educational programs. Questions that allowed participants to “select all that apply” were tallied to provide a total number of that event experienced. For example, one survey question asked student participants to select all the instances in which they participated in professional advocacy. If a student selected all 5 options, he or she received a score of 5 for a new variable called “number of advocacy events involved.”

Analyses found that the number of ways in which administrators incorporated an advocacy lecture into the professional aspects class was significantly correlated with an increase in the ways their programs attempted to encourage advocacy in the students (*r* = 0.333, *P* = .027, *n* = 44). Administrators who incorporated an advocacy lecture in more ways were also more likely to rank higher the importance of CRNAs who politically advocate for their profession (*r* = 0.746, *P* = .088, *n* = 6) and to rate higher the importance of teaching advocacy to SRNAs (*r* = 0.746, *P* = .088, *n* = 6).

Finally, students reported increased involvement in advocacy events as administrators reported a greater variety of incorporating advocacy into program lectures (*r* = 0.481, *P* = .001, *n* = 47). As administrators reported participating in more advocacy events, students reported participating in more advocacy events (*r* = 0.744, *P* < .0001, *n* = 47). The correlation between the administrators’ ratings of the importance of political advocacy and the importance of teaching advocacy to students was significant and strong (*r* = 1.000, *P* < .0001, *n* = 6).

Regarding the students’ experiences, analyses showed significant correlations between the students’ ratings of their knowledge of current policy issues facing their profession and their experiences of advocacy in general (Figure 4). Students who rated their knowledge as higher tended to report participating in professional advocacy events with more frequency since beginning school (*r* = 0.453, *P* = .001, *n* = 54). Students who rated their knowledge as higher also reported fewer reasons that they would feel uncomfortable in meeting with legislators to discuss policies related to their profession (*r* = -0.284, *P* = .037, *n* = 54).

Students’ rating of their knowledge of current policy
issues also significantly correlated with their experiences in the nurse anesthesia education program. Students were more likely to rate their knowledge as higher when their administrator reported participating in more advocacy events themselves ($r = 0.429, P = .003, n = 47$) and when their administrator reported more ways in which they incorporated an advocacy lecture into the professional aspects class ($r = 0.289, P = .048, n = 47$).

The number of advocacy events in which students report participating since starting school was significantly correlated with their experiences in their nurse anesthesia educational programs as well. Students who reported a greater number of advocacy topics presented in their classes reported taking part in more advocacy events themselves ($r = 0.309, P = .023, n = 54$). The number of advocacy events in which a student was involved was also correlated with fewer reported reasons the student would feel uncomfortable meeting with a legislator ($r = -0.309, P = .023, n = 54$). Greater student participation in advocacy events was also associated with how highly they rated the influence of their program in their perception of the importance of advocacy ($r = 0.281, P = .040, n = 54$).

A greater number of ways in which students reported advocacy in their class lectures was significantly correlated with how important the student thought it was to discuss advocacy in school ($r = 0.410, P = .002, n = 54$) and with how influential the students thought their program was in affecting their perception of advocacy ($r = 0.669, P < .0001, n = 54$). Students who reported more forms of advocacy lectures in their classes were more likely to report fewer reasons to feel uncomfortable in talking with legislators regarding the profession ($r = -0.355, P = .008, n = 54$). The reported number of ways in which administrators and students saw advocacy as being presented in class were significantly correlated ($r = 0.373, P = .010, n = 47$).

Students who rated higher the importance of advocacy education in their nurse anesthesia educational programs also tended to rate higher both the importance of CRNAs politically advocating for their profession ($r = 0.516, P < .0001, n = 54$) and the influence of their program on their perceptions of the value of advocating ($r = 0.285, P = .037, n = 54$). Student ratings of the importance of CRNAs as political advocates significantly correlated with their administrators’ perceptions of that importance ($r = 0.303, P = .038, n = 47$) and their administrators’ perceptions of the importance of teaching advocacy in general ($r = 0.303, P = .038, n = 47$).

**Discussion**

Advocacy by CRNAs continues to be a critical component in the survival and growth of our profession. The shortage of political advocacy mentors and the lack of formal education on professional advocacy will hinder a CRNA’s political astuteness and effectiveness.

Studies are limited on nurse anesthesia education and the impact it has on advocacy; however, research has shown that nurses who receive advocacy education are more likely to have strong political skills. The Institute of Medicine’s report *The Future of Nursing* recommends that nursing education include health policy concepts, and due to the new education standards requiring doctorate-level-entry nurse anesthesia educational programs, the curricula...
could be developed to support this recommendation.  

Program administrators should consider a 2-pronged approach to building a leadership foundation within their nurse anesthesia education program. First, integrating health policy concepts and advocacy activities into the curriculum will lay the groundwork for students to assume these leadership roles. The political process of state and federal governments, historic legislative efforts that affected the CRNA profession, and current political affairs should be introduced to SRNAs. Assigning students to participate in Legislative Days, attend legislation committee hearings, or meet with legislators to discuss legislative issues are activities that will empower continued participation. Second, program administrators should partner with state and national CRNA leaders to establish a mentoring program. Exposing SRNAs to advocacy experts will provide them with unique perspectives that will position them for success and inspire them to engage in health policy.  

This study has some limitations. A power analysis was not performed because the recommended sample size was not easily obtainable due to the limited number of anesthesia schools in this study. The sample size for the SRNAs was indeterminable because of the inability to ascertain how many students received the survey. The surveys used for data collection were developed for this study; a previously validated survey was not available. By using the AMEE process, the validity and reliability of the surveys was increased.

In addition, this study included nurse anesthesia programs in Pennsylvania, resulting in a small sample size of program administrators and SRNAs. It is important to note that the relatively small sample size likely inflates the variables and the results. Last, the content expert used during the AMEE process was also a study participant, and this may have resulted in skewed results. More research should be done to evaluate the effects and correlations on a larger scale.

In the time since this study concluded, the COA revised the Standards for Accreditation of Nurse Anesthesia Programs Practice Doctorate. Advocacy education and professional development activities are incorporated in the professional role standard: to inform the public of the role and practice of the CRNA, evaluate how public policy-making strategies have an impact on the financing and delivery of healthcare, advocate for health policy change to improve patient care, and advocate for health policy change to advance the specialty of nurse anesthesia. The results of this study support these curricular changes for nurse anesthesia educational programs.

REFERENCES


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Laryngospasm is an exaggeration of a protective reflex that prevents aspiration of foreign objects into the lower airway (e.g., during swallowing). This results in complete or partial closure of the glottis, and impedance or total obstruction of airflow to the trachea and lungs. Often, the resulting hypoxia will by itself break a laryngospasm; however, if the spasm continues without relief, it can lead to pulmonary edema, cardiac dysrhythmias, cardiac arrest, and ultimately death. This evidence-based literature review explores the pathophysiology of laryngospasm and covers mechanical and pharmacologic prevention and treatment modalities in pediatric patients.

**Keywords:** Laryngospasm, pediatric, prevention, protective reflex, treatment.

Pediatric laryngospasm is a life-threatening event that results in complete or partial blockage of the airway. This blockage can lead to hypoxemia, negative-pressure pulmonary edema, pulmonary aspiration, and cardiac arrest. The treatment of laryngospasm has traditionally been succinylcholine, a short-acting, depolarizing neuromuscular blocking agent that relaxes muscle tension and breaks the laryngospasm. Succinylcholine, although very effective at treating laryngospasm, comes with potential serious side effects such as bradycardia and arrhythmias. In recent years, articles have been published that suggest different treatment modalities for pediatric laryngospasm, but providers have been slow to incorporate them into practice. Pharmacologic interventions such as the use of lidocaine, midazolam, or propofol have emerged as potentially useful treatments for breaking or preventing laryngospasm.

This literature review revisits principles in anesthesia regarding pathophysiology of laryngospasm, followed by methods for its prevention and treatment. The goal is to compile newer studies regarding pediatric laryngospasm and to present data and interventions that the anesthetists can incorporate in their practice, with the ultimate goal of improving patient outcomes.

**Methods**

This systematic review of the literature began with a search for articles pertinent to this topic. Multiple literary search engines were used to find these articles including: Academic Search Complete, CINAHL Complete, MEDLINE Complete, EBSCO host, Environment Complete, SPORTDiscus, Education Source, Business Source Complete, Communication & Mass Media Complete, SocINDEX, Military & Government Collection, PsycINFO, and PsycARTICLES. Article search criteria were limited to articles in English, published in the last 15 years, and with available full text. Certain articles published before 2002 were necessary to include for the literature review as they contained foundational knowledge pertinent to our topic. Searches were conducted using keywords, phrases, and specific subject headings, including pediatric, laryngospasm, treatment, break, pharmacology, prevention, incidence, propofol, lidocaine, magnesium, succinylcholine, extubation, maintenance, induction, risk factors, surgery, LMA [laryngeal mask airway], ETT [endotracheal tube], pathophysiology, airway, and maneuvers. The initial search yielded 3,275 results, 32 of which were included in this literature review once our inclusion criteria—pediatric focus, anesthesia related, full text form, peer-reviewed journals published in English—were applied. The evidence was evaluated using the method described by Melnyk and Fineout-Overholt. This review consists of 1 meta-analysis, 9 randomized controlled trials (RCTs), 1 nonrandomized controlled study, 7 prospective cohort studies, 7 retrospective cohort studies, 8 literature reviews, and 1 animal study.

**Results**

- **Pathophysiology of Laryngospasm.** Laryngospasm is an exaggeration of a protective reflex that prevents aspiration of foreign objects into the lower airway (e.g., during swallowing). This results in complete or partial closure of the glottis, and impedance or total obstruction of airflow to the trachea and lungs. Often, the resulting hypoxia will by itself break a laryngospasm; however, if the spasm continues without relief it can lead to pulmonary edema, cardiac...
dysrhythmias, cardiac arrest, and ultimately death.\(^6\)

The laryngospasm reflex is controlled by the extrinsic and intrinsic muscles of the larynx, and innervated by the internal branch of the superior laryngeal nerve, a branch of the vagus nerve.\(^6\) Abnormal excitation of this pathway occurs most commonly during lightened anesthesia (ie, stage 2) and thus poses the greatest threat during induction and emergence.\(^7,8\)

Furthermore, there are 3 mechanisms to laryngospasm—expiratory stridor, inspiratory stridor, and ball-valve obstruction.\(^9\) Expiratory stridor involves the intrinsic muscles of the larynx, and results in adduction of the vocal cords. Inspiratory stridor, also controlled by the intrinsic muscles, results from failure of the abductor muscles. Ball-valve obstruction is controlled by the extrinsic muscles, and involves closure of the false and true vocal cords, as well as collapse of soft tissue above the glottis.\(^9\)

- **Prevention of Pediatric Laryngospasm.** Agents used to prevent laryngospasm in pediatric patients include magnesium, lidocaine, and intermediate-acting muscle relaxants, such as rocuronium.
  - **Magnesium.** Magnesium is an intracellular cation and smooth muscle inhibitor.\(^10\) A study by Gulhas et al\(^10\) was performed on the efficacy of magnesium in the prevention of pediatric laryngospasm in patients undergoing adenotonsillectomy. This was a double-blind study in 40 patients between the ages of 3 and 12 years. Twenty participants in the magnesium group were given 15 mg/kg of magnesium sulfate 2 minutes after intubation. The other 20 patients were given 30 mL of normal saline. The results concluded there was no incidence of laryngospasm observed in the group that received magnesium, whereas the placebo group had a 25% rate (\(P < .05\)). The authors believe the mechanism of action of breaking laryngospasm is by deepening the anesthetic and enhancing muscle relaxation. This study had a small sample population and used intravenous (IV) lidocaine (1 mg/kg) on induction, which could be a confounding factor on magnesium’s prevention efficacy.\(^11\)

  Savran-Karadeniz et al\(^11\) conducted a similar study in 2016 that eliminated lidocaine from the anesthetic and used higher preventive magnesium doses (30 mg/kg). The surgical procedure studied was esophageal dilation in children between 2 and 12 years of age. The findings of this study showed that the incidence of laryngospasm in the group that received magnesium vs the control group was 10% and 33.3%, respectively (\(P = .057\)). This study’s findings corroborated the earlier study by Gulhas et al,\(^10\) but more studies are needed.

  - **Lidocaine.** Lidocaine has been a controversial and highly studied pharmacologic agent in the prevention of pediatric laryngospasm.\(^6\) Because of the uncertainty concerning the benefit of lidocaine, in 2014 Mihara et al\(^12\) conducted a systematic literature review and meta-analysis of the efficacy of lidocaine to prevent laryngospasm in children. Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) was adhered to throughout the data collection process, and Grading of Recommendations Assessment, Development and Evaluation (GRADE) was used to evaluate the quality of evidence for each study. The meta-analysis combined 9 different studies with a total of 787 patients. Studies examining both IV and topical lidocaine routes of administration were included.

  The results demonstrated a statistically significant reduction in the incidence of laryngospasm for both the IV and topical lidocaine routes. The authors postulated that the most efficacious time to administer IV lidocaine is within 5 minutes of tracheal extubation. Gharaei et al\(^12\) specifically compared IV vs topical lidocaine and found the difference in the prevention of pediatric laryngospasm to not be statistically significant.

  - **Intermediate-Acting Muscle Relaxants.** There has been minimal research done evaluating the use of intermediate-acting muscle relaxants in the prevention of pediatric laryngospasm. A study done by Martin-Flores et al\(^13\) evaluated the use of rocuronium on prevention of laryngospasm in cats. The researchers gave 8 cats anesthesia 4 separate times and used IV rocuronium at doses of 0.1 mg/kg, 0.2 mg/kg, 0.3 mg/kg, and 0.6 mg/kg. A videoendoscope was inserted through an LMA and recorded the laryngeal response to a sterile water spray. A response was recorded at baseline without rocuronium, and then after the rocuronium was given. The results showed a significant decrease in the completeness and duration of laryngeal responses with the rocuronium doses of 0.3 mg/kg and 0.6 mg/kg.

  Although this study was done in cats and not pediatric patients, another study’s authors believed that long-acting muscle relaxants could facilitate better intubating conditions and lower laryngospasm rates following a failed laryngoscopy. A 2016 study by Spaeth et al\(^14\) focused on reducing serious airway events and airway cardiac arrests during pediatric anesthesia. The authors used 3 main quality indicators: nondepolarizing muscle relaxants in children younger than 2 years of age, for cases lasting less than 30 minutes, having succinylcholine and atropine out and available, and assessing ventilation after extubation by auscultation or end-tidal carbon dioxide. The finding of the study showed a 44% reduction in serious airway events and a 59% reduction in airway cardiac arrests. This study did not specifically look at the reduction of laryngospasm, but it did use long-acting muscle relaxants to decrease airway events such as laryngospasm.

  - **Subhypnotic Dose of Propofol.** Propofol is known to inhibit airway reflexes and deepen anesthesia.\(^15\) Batra et al\(^15\) researched propofol’s ability to be prevent pediatric laryngospasm. This RCT had 120 patients randomly assigned to receive either propofol, 0.5 mg/kg, before extubation or a control dose of the equivalent amount of saline. The presence of laryngospasm was evaluated by
Pharmacologic Treatment of Pediatric Laryngospasm.

Treatment of laryngospasm in pediatric patients is with pharmacologic or physical techniques.

• Propofol. Salah and Azzazi suggested that a subhypnotic dose of IV propofol at 0.5 mg/kg was effective in treating pediatric laryngospasm. In a group of 20 patients experiencing laryngospasm, Salah and Azzazi found that 15 of those patients responded favorably to propofol. Propofol inhibits airway reflexes and relaxes tissues in the upper airway. Advantages of propofol include rapid onset (30-45 seconds), rapid clearance, and avoidance of side effects such as bradycardia and myalgias associated with succinylcholine. However, the provider should be aware of side effects of propofol, including hypotension and transient apnea. Propofol is also useful in patients in whom succinylcholine is contraindicated (eg, burn victims, muscular dystrophies, cholinesterase deficiency).

• Midazolam. In the same study, Salah and Azzazi found that IV midazolam at 0.03 mg/kg effectively treated postextubation laryngospasm. In a group of 20 patients experiencing laryngospasm, it was noted that 17 of the subjects responded favorably to midazolam. In similar fashion to propofol, midazolam decreases upper airway reflexes. Salah and Azzazi also mentioned that midazolam was effective in managing recurrent postoperative laryngospasm in anxious children, a condition referred to as hysterical stridor.

• Succinylcholine. Succinylcholine has long been a preferred pharmacologic agent for treating laryngospasm because of its rapid onset and short duration of action. Intravenous succinylcholine is the gold standard in the treatment of pediatric laryngospasm, with a dose of 1 to 2 mg/kg. Succinylcholine is given along with atropine (0.02 mg/kg) to prevent bradycardia.

When IV access is unavailable, succinylcholine may be administered intramuscularly at a dose of 4 mg/kg. The disadvantage to this, however, is an onset time of 3 to 4 minutes for maximal twitch depression, although it has been suggested that relaxation of airway tissue occurs within 1 minute. Furthermore, at a dose of 4 mg/kg, succinylcholine may last upward of 20 minutes. Although sublingual and intraosseous routes also are acceptable, Walker and Sutton suggest that intramuscular succinylcholine is probably the most reliable agent to break laryngospasm when IV access is unavailable.

• Physical Techniques to Treat Pediatric Laryngospasm. Different physical techniques can be attempted to break laryngospasm before pharmacologic intervention.

• Positive-Pressure Ventilation. The most common physical method described by clinical providers is the immediate application of positive-pressure ventilation and administration of 100% oxygen. Gavel and Walker described that if a laryngospasm is suspected and there is soft-tissue compression of the larynx, application of positive pressure may relieve the obstruction. The initiation of positive-pressure ventilation could also relieve a supraglottic obstruction or partial laryngospasm.

• Larson Maneuver. After positive-pressure ventilation has failed, the Larson maneuver is another technique that can be beneficial to help break a pediatric laryngospasm. Abelson explains that this maneuver, also called laryngospasm notch pressure, is the application of firm and inward pressure at the laryngospasm notch. This notch is located slightly cephalad to the earlobe and between the mastoid process (posterior) and mandibular condyle (anterior). Application of pressure here while simultaneously performing a jaw thrust may resolve a pediatric laryngospasm before the administration of pharmacologic agents. Abelson further postulates that the anesthetist should not wait until desaturation to administer paralytic agents and that there are insufficient data to systematically evaluate the Larson maneuver’s efficacy.

• Gentle Chest Compression. Gentle chest compressions have been described in the literature in the past, but no clinical trials have examined the method’s effectiveness. The mechanism for the relief of pediatric laryngospasm is unknown, but the thought is that chest compression pushes air cephalad from the lungs against the vocal cords to relieve the spasm. Al-Metwalli et al conducted an RCT for 4 years in pediatric patients undergoing tonsillectomy procedures. A total of 632 patients were delegated to the standard-practice group, which treated patients in whom a laryngospasm developed with 100% oxygen and positive-pressure ventilation. A total of 594 patients were in the chest compression group, in which patients who experienced laryngospasm were managed with 100% oxygen and gentle chest compressions at a rate of 20 to 25 compressions per minute. In both groups, succinylcholine was given if the first interventions did not break the spasm. Results of the study showed that 73.9% of patients with laryngospasm were effectively treated by gentle chest compressions without the need to use succinylcholine vs the standard-practice group, which had only 38.4% with successful treatment of laryngospasm (P < .001). Gastric distention developed in 86.5% of patients in the standard-practice group compared with none in the chest compression group (P < .0001). This is the first known RCT evaluating gentle chest compressions for treatment of pediatric laryngospasm, and more studies are needed to determine the optimal rate and force of compressions.

Discussion

Pediatric laryngospasm incidence has historically been 1.74%, but more recent data suggest that incidence is decreasing to 0.53%. Although the incidence may be...
decreasing, there is still a need to further lower the incidence to achieve the best possible outcomes for pediatric patients undergoing anesthesia. Randomized controlled trials can help identify the most efficacious treatment options for laryngospasm, but algorithms help combine the RCTs’ findings into a tool that can help the practitioner make the best treatment decisions. There are a variety of algorithms for pediatric laryngospasm, but we want to build one that uses the most current RCTs and supporting evidence to provide the most up-to-date information on risk factors, prevention, and treatment options.

Many algorithms focus on treatment of laryngospasm, and not the prevention aspect. A focus on prevention is important because it can help eliminate the negative outcomes associated with pediatric laryngospasm such as oxygen desaturation, negative-pressure pulmonary edema, and death.\(^3\) There are many different pharmacologic therapies (eg, lidocaine, midazolam, propofol) that can be used to lower the incidence of laryngospasm. We created an algorithm (Figure) that has both the prevention and treatment options to provide the best outcomes for our pediatric patients. The rationale for this algorithm is explained here.

- **Risk Factors.** Pediatric laryngospasm that is unanticipated can be difficult to treat.\(^23\) Knowing and identifying risk factors for increased incidence of laryngospasm (see Figure) in the pediatric population must be of paramount importance. In our literature review, we identified several prominent risk factors that were prevalent in recent studies. Age of the pediatric patient is a major risk factor, with younger children more susceptible to laryngospasm.\(^21,24\) Another risk factor identified was obesity. Children at or above the 85th percentile of BMI and with a diagnosis of sleep-disordered breathing were found to have a significantly increased incidence of laryngospasm.\(^25\) Environmental tobacco smoke exposure and recent upper respiratory tract infections (within past 30 days) both were shown to increase rates of laryngospasm in the pediatric population.\(^21,22,24,26,27\) Finally, the last risk factor identified was the type of procedure that the pediatric patient was undergoing. Several types of procedures with increased incidence of laryngospasm include appendectomy, otolaryngology (especially adenotonsillectomy), plastic surgery, hypospadias repair, and esophageal endoscopy.\(^8,10,15,21\)

- **Prevention.** A study by Lee et al\(^28\) saw the adverse events and laryngospasm incidence increase with an increased number of attempts of pediatric laryngoscopy. This can vary greatly depending on provider experience and competency level.\(^28\) We recommend that intubation attempts be limited to the least possible number of attempts.

Several RCTs compared how ideal the intubating conditions were, with or without the use of muscle relaxants.\(^16,29\) The studies showed that intubating conditions were ideal, but more importantly there was no incidence of laryngospasm in either group. We did not recommend the use of muscle relaxants during induction to prevent laryngospasm in our algorithm (see Figure).

The risk of using an LMA vs ETT is controversial, with different studies showing different results on which method has a higher incidence of pediatric laryngospasm.\(^7,22,24,30\) There are no definitive studies that consistently show one is superior to the other in terms of laryngospasm in the pediatric population. Because of the lack of consensus among the studies, we elected to not use that in our prevention algorithm.

Magnesium was shown to be effective in 2 different RCTs.\(^10,11\) Magnesium is believed to help deepen the anesthetic and enhance muscle relaxation.\(^11\) Both studies used magnesium doses of 15 mg/kg and 30 mg/kg.\(^10,11\) We used the range of 15 to 30 mg/kg of magnesium before induction.

Intermediate-acting muscle relaxants for procedures have not been studied enough to include in a prevention
algorithm, but this drug class was shown to be effective in a randomized controlled trial in felines.\textsuperscript{13} There have been some attempts to use intermediate-acting neuromuscular blocking agents in different algorithms,\textsuperscript{14} but further studies are needed before we recommend them in our algorithm.

Lidocaine has been shown by a large meta-analysis to help prevent pediatric laryngospasm.\textsuperscript{3} This analysis included studies that used doses of 1.0 to 2.0 mg/kg of lidocaine intravenously and should be given within 5 minutes of tracheal extubation. Mihara et al\textsuperscript{3} also concluded that topically administered lidocaine lowers the incidence of laryngospasm, and the time of administration was either before intubation or during the airway device insertion. Mihara et al\textsuperscript{3} did not have a clear recommendation for the dose of topically applied lidocaine. (See Table.) We elected to use 1.0 to 2.0 mg/kg of IV lidocaine for our recommendation.

Propofol was shown to be highly effective in the prevention of pediatric laryngospasm.\textsuperscript{15} The study by Batra et al\textsuperscript{15} administered 0.5 mg/kg of propofol 60 seconds before extubation, which decreased the incidence of laryngospasm. This dose helps deepen the anesthetic and inhibit airway reflexes, preventing laryngospasms.\textsuperscript{15}

There was no clear evidence that extubating a patient who is awake vs under deep anesthesia had any advantage in preventing laryngospasm.\textsuperscript{31,32} There were no differences in laryngospasm rates in the studies that we included in our study.\textsuperscript{32} Therefore, we did not indicate an extubation preference for our prevention algorithm.

\textbf{Treatment}. The positive-pressure ventilation or continuous positive airway pressure (CPAP) with the administration of 100% oxygen is the most common and earliest method to treat laryngospasm.\textsuperscript{18} This treatment is recommended if there is a soft-tissue blockage that is compressing the larynx.

Larson maneuver is another quick early treatment option that has been used by providers. This technique uses the application of pressure slightly cephalad to the earlobe to facilitate patency of the upper airway. Larson maneuver should be used before pharmacologic agents, but a provider should not wait until desaturation of the patient to use other forms of treatment.\textsuperscript{19}

Gentle chest compressions is a treatment option for laryngospasm that has been studied in the literature. This treatment is thought to push air from the lungs against the vocal cords.\textsuperscript{20} The results of the study showed that gentle chest compressions could be an alternative and effective treatment compared with traditional methods to break laryngospasm. More studies are needed to determine the rate and force of the compressions, so we do not recommend this treatment in our algorithm yet.

Propofol has been studied and recommended as a preventive pharmacologic agent used for pediatric laryngospasm, but an RCT in 2014 found that it can be an effective treatment once a laryngospasm is present.\textsuperscript{4} This study used a subhypnotic dose of IV propofol (0.5 mg/kg) and found that it was effective in 75% of pediatric patients who had laryngospasm.\textsuperscript{4}

The same study that found that midazolam was also effective at treating postextubation laryngospasm. Benzodiazepines have been previously shown in the literature to decrease upper airway reflexes.\textsuperscript{4} Of 20 patients who were experiencing laryngospasm, 17 responded fa-

<table>
<thead>
<tr>
<th>Source</th>
<th>Study design</th>
<th>Sample size</th>
<th>Results and conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oofuvong et al,\textsuperscript{22} 2014</td>
<td>Retrospective cohort study</td>
<td>N = 14,153 pediatric patients over 6-year period</td>
<td>• Laryngospasm incidence 0.53% for children ≤ 15 years • URI increased the incidence of laryngospasm 1.7 times (95% CI = 0.78-3.7) • LMA with assisted ventilation carried twice the risk of laryngospasm (95% CI = 1.2-3.3, (P &lt; .001))</td>
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<td>Drake-Brockman et al,\textsuperscript{30} 2017</td>
<td>RCT</td>
<td>LMA group: n = 85 ETT group: n = 95</td>
<td>• 3.82 times the incidence of laryngospasm in ETT vs LMA (95% CI = 1.13-12.96, (P = .02))</td>
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<tr>
<td>Gulhas et al,\textsuperscript{10} 2003</td>
<td>Double-blind RCT</td>
<td>IV magnesium before induction: n = 20 Normal saline before induction: n = 20</td>
<td>• Group that received IV magnesium had no laryngospasms, whereas group that received normal saline had a 25% incidence of laryngospasm ((P &lt; .06))</td>
</tr>
<tr>
<td>Mihara et al,\textsuperscript{3} 2014</td>
<td>Meta-analysis</td>
<td>Combined 9 studies: N = 787</td>
<td>• Demonstrated that IV or topical lidocaine is an effective medication in preventing pediatric laryngospasm</td>
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<tr>
<td>Batra et al,\textsuperscript{15} 2005</td>
<td>RCT</td>
<td>Propofol group: n = 60 Control group: n = 60</td>
<td>• Propofol group had a 6.6% incidence of laryngospasm vs a 20% incidence of laryngospasm in control group ((P &lt; .05))</td>
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<tr>
<td>Al-Metwalli et al,\textsuperscript{20} 2010</td>
<td>Nonrandomized controlled study</td>
<td>Gentle chest compression: n = 594 Standard-practice group: n = 632</td>
<td>• Gentle chest compressions effectively treated 73.9% of laryngospasms that developed ((P = .0005)), but the standard-practice group treated only 38.4% ((P &lt; .001))</td>
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Succinylcholine has long been the classic treatment of a patient having a laryngospasm. Because of the adverse side effects (eg, bradycardia, arrhythmias), it is usually the last option, but the most reliable pharmacologic agent to break a laryngospasm. The recommended dose of succinylcholine is 1.0 to 2.0 mg/kg intravenously or 4 mg/kg for the intramuscular route.

**Conclusion**

Laryngospasm is still a potentially life-threatening event that occurs in the pediatric population. Although many studies have looked at the prevention and treatment of laryngospasm, researchers need to continue studying the issue until laryngospasm is eliminated. We believe that our algorithm combines evidence from the most recent studies to prevent and treat pediatric laryngospasm in the most effective manner. There has been a vast improvement in the incidence of pediatric laryngospasm and its treatment options with the advancements of surgical techniques, pharmacologic options, and a better understanding of the phenomenon. We believe that as more funding is obtained and studies are conducted on pediatric laryngospasm, the incidence will continue to decline.

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21. Bajal RG, Bidani SA, Minard CG, Watcha MF. Perioperative respira-


AUTHORS
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Assessing Frailty and Its Implications on Anesthesia Care and Postoperative Outcomes in Surgical Patients

Timothy J. Donoghue, DNAP, CRNA

Objectives
At the completion of this activity, the learner will be able to:
1. Define frailty.
2. List the 5 traits of the frailty phenotype.
3. Describe the Accumulated Deficits Theory of Frailty.
4. Discuss modified frailty indexes and their potential impact on anesthesia care.
5. Examine potential anesthetic implications of frailty screening and its impact on intraoperative case management and postoperative outcomes.

Introduction
Poor outcomes resulting from postoperative complications create a costly burden on the healthcare system. Currently there is no standard accepted clinical method for identifying patients who may have a higher chance of poor outcomes following surgery. If these patients could be identified early, perhaps preoperative optimization, medication management, social support, and anticipated rescue resources could be mobilized earlier in an effort to prevent complications. Patients 65 years and older represent one population that may benefit from a standardized method of preoperative risk stratification. As of July 2016, there were almost 50 million people aged 65 or older in the United States. Furthermore, the number of people older than 65 years worldwide is expected to increase from 461 million to 2 billion people by 2050. The fastest growing segment of the population, people 85 years and older, is expected to triple in the next 40 years. As the elderly population continues to grow, the number of surgical procedures performed in these patients will likely increase, and outcomes will become a focus for improving quality of care. With increasing surgeries on this vulnerable patient population, and as reimbursement payments become more closely tied to patient outcomes, hospitals have a tremendous interest in improving care and preventing unexpected complications.

The elderly patient population is particularly vulnerable to postoperative complications because of diminished physiologic reserve and reduced tolerance of stressful events such as surgery that accompany aging. Age-associated declines in physiological reserve and tolerance of stressful events contribute to greater difficulty in recovery from surgery, regaining independence, and

Keywords: Accumulated deficits of frailty, frailty, modified frailty index, phenotype of frailty, postsurgical outcomes.

Identification of elderly patients at high risk of poor outcomes following surgery remains difficult. Clinicians currently lack a tool to consistently aid them in this process. For instance, the ASA physical status score (ASA-PS) is one commonly used tool to identify high-risk surgical patients using comorbidities. However, this scoring system is too subjective to yield consistent results. According to retrospective research, the concept of frailty is a valid construct with the potential to create a generalizable method for improving poor healthcare outcomes by risk stratification. Research has shown frail patients have higher rates of morbidity, mortality, and postoperative complications. This article aims to explore the Phenotype Theory of Frailty and the Accumulated Deficits Theory of Frailty and possible anesthetic implications of incorporating frailty screening into patient care. Use of a modified frailty index as a tool in a high-quality preoperative evaluation may help practitioners risk-stratify patients. This can allow for earlier mobilization of care resources before surgery to improve outcomes.

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returning home in the elderly population. Of patients 65 years or older undergoing surgical procedures, 45% will require continued medical care in a skilled nursing facility, inpatient rehabilitation center, or home health services. Additionally, postsurgery discharge to a postacute care facility such as a skilled nursing facility is associated with a fourfold increase in mortality in elderly patients. Growth in surgical procedures among the elderly population, postoperative complications, and anesthesia implications are important issues that require addressing.

Anesthesia Implications
Postoperative complications are a critical part of the discussion of surgical care of elderly patients because with postoperative complications come increased morbidity, mortality, and cost. Surgical site infection, myocardial infarction, cerebrovascular accident, pneumonia, delirium, and falls represent some potential complications affecting elderly patients who undergo surgery. Intraoperative factors affecting postoperative outcomes include patient hypothermia, blood loss, morbid cardiac events, abnormally high and low blood glucose levels, atelectasis, pulmonary edema, and shivering. Surgical site infections lead to an increased length in hospital stay by 7 to 10 days, costing $10 billion annually. Preoperative assessment of elderly surgical patients is critical because it helps identify comorbidities, cardiac, neurologic, pulmonary, and physical baseline levels for patients before surgical interventions. A thorough preoperative evaluation of surgical patients is an important tool that may be used to tailor care with the goal of minimizing poor outcomes. As a first step toward this goal, Chow et al developed general guidelines for preoperative assessment of elderly patients. Some of their recommendations include assess-

### Table 1. Optimal Preoperative Assessment of the Elderly Surgical Patient

<table>
<thead>
<tr>
<th>Class</th>
<th>Definitions and examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Normal healthy patient who does not smoke and drinks alcohol minimally</td>
</tr>
<tr>
<td>2</td>
<td>Patient with mild systemic disease. Mild diseases only without substantive functional limitations. Examples include (but not limited to) current smoker, social alcohol drinker, pregnancy, obesity (BMI 30 to &lt; 40 kg/m²), well-controlled diabetes or hypertension, mild lung disease.</td>
</tr>
<tr>
<td>3</td>
<td>Patient with severe systemic disease that is not a constant threat to life, but poses functional limitations. Examples include (but not limited to) uncontrolled DM, hypertension, COPD, alcohol dependence, BMI &gt; 40 kg/m², pacemaker implantation, &gt; 3-month history of MI, IVA, or CAD with stents.</td>
</tr>
<tr>
<td>4</td>
<td>Patient with severe systemic disease that is a constant threat to life. Examples include (but not limited to) recent (&lt; 3 months) MI, IVA, TIA, or CAD with stents, ongoing cardiac ischemia or severe valve dysfunction, severe reduction of ejection fraction, sepsis, DIC, ARD, or ESRD not undergoing regular dialysis.</td>
</tr>
<tr>
<td>5</td>
<td>A moribund patient who is not expected to survive without surgery. Examples include (but not limited to) ruptured abdominal/thoracic aneurysm, massive trauma, intracranial bleed with mass effect, ischemic bowel in the face of significant cardiac pathology or multiple organ/system dysfunction.</td>
</tr>
<tr>
<td>6</td>
<td>A patient declared brain dead whose organs are being removed for donation</td>
</tr>
</tbody>
</table>

### Table 2. ASA Physical Status Classification System

<table>
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</tr>
<tr>
<td>6</td>
<td>A patient declared brain dead whose organs are being removed for donation</td>
</tr>
</tbody>
</table>

Abbreviations: ARD, acute respiratory distress; BMI, body mass index; CAD, coronary artery disease; COPD, chronic obstructive pulmonary disease; CVA, cerebral vascular accident; DIC, disseminated intravascular coagulation; DM, diabetes mellitus; ESRD, end-stage renal disease; MI, myocardial infarction; TIA, transient ischemic attack.
ing the patient's cognitive ability, screening for depression, identifying risk factors for postoperative delirium, current alcohol intake, and substance use. Additionally, cardiac and pulmonary evaluation along with assessing patient's functional status, history of falls, social support, and nutrition status are recommended for the preoperative assessment of geriatric patients (Table 1). Through completion of these evaluations, there is the potential to identify patients with modifiable risk factors. Earlier mobilization of rescue resources might result from a thorough evaluation because changes in baseline patient status would be detected earlier. In this lies the potential for improved postoperative outcomes by optimizing high-risk patients before surgery.

Currently, there is no standard, comprehensive tool for identifying patients at highest risk of poor outcomes following surgery. The ASA physical status (ASA-PS) scale represents an attempt at evaluating each patient's health status. The ASA-PS is designed to measure a patient's preoperative health status using a grading scale based on comorbidities (Table 2). An interviewing anesthesia provider decides on a patient's physical status, and this subjectivity leads to variability in scores. Healthcare providers have attempted to use the ASA-PS as a predictor of poor postoperative outcomes, but because of its subjectivity, results have been mixed. Although a higher ASA-PS is associated with postoperative complications, this phenomenon is not consistently replicable because it was meant to evaluate preoperative health status, not to predict postoperative complications.

If the healthcare team, including anesthesia providers, could identify high-risk patients, they could mobilize hospital resources earlier, establish multidisciplinary care teams, and provide targeted therapy, thereby potentially reducing poor outcomes. Geriatricians could more effectively manage patient medications, treat depression, and address social support issues before surgery. Frailty represents a novel concept to surgeons and anesthesia providers that may help stratify complication risk among surgical patients. Numerous studies found frailty assessments are valid and reliable predictors of poor outcomes in high-risk patients.

Frailty

A broad definition of frailty addresses physiological, emotional, cognitive, and social parameters. Fried et al. posited “the multidimensional nature of frailty … [as] age associated decline in physiologic reserve and function across multiple organ systems, resulting in diminished strength and endurance, increased vulnerability to stressors, risk of falls, disability, hospitalization and mortality has been accepted.” Despite a generally accepted

Table 3. Five Traits Compiling the Frailty Phenotype

1. Unintentional weight loss of ≥ 4.5 kg (10 lb) in the previous year, or a > 5% loss of body weight at follow-up
2. Strength as measured by grip strength
3. Endurance as measured by self-reports of exhaustion
4. Slowness as measured by a timed 4.5-m (15-ft) walk
5. Low physical activity level as measured by kilocalorie expenditure per week
description of frailty, quantifying it remains difficult. A major obstacle to implementing interventions has been the lack of a standardized frailty assessment. As a concept, frailty is multifactorial involving physiologic, cognitive, emotional, and social age-related decline. This leads to impaired responses to stressors and is distinguishable from disability. A possible definition of frailty is a biologic syndrome of decreased reserve and resistance to stressors, resulting from cumulative declines across physiologic systems. Although a gradual age-related decline in physiologic reserve occurs, frailty accelerates these declines, leading to homeostatic mechanism failure. Although a gradual age-related decline in physiologic reserve occurs, frailty accelerates these declines, leading to homeostatic mechanism failure. Decreased response to stressors makes patients vulnerable to poor outcomes. Frailty is a well-established measure of outcomes in surgical patients. It is superior to age in predicting short-term outcomes and major adverse health events. Frailty represents an independent predictor of postoperative complications and length of hospital stay for elderly patients. Despite proven validity, measurement of frailty has yet to routinely occur in the clinical setting. The frailty phenotype by Fried et al and the Accumulated Deficits of Frailty Theory by Rockwood et al are accepted theories attempting to define and quantify this construct.

Table 4. Examples of Energy Expenditure

<table>
<thead>
<tr>
<th>Activity</th>
<th>Kilocalories burned per hour for someone with a weight of 58.5 kg (130 lb)</th>
<th>74.3 kg (165 lb)</th>
<th>85.5 kg (190 lb)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Playing basketball</td>
<td>472</td>
<td>563</td>
<td>690</td>
</tr>
<tr>
<td>Raking a lawn</td>
<td>236</td>
<td>281</td>
<td>345</td>
</tr>
<tr>
<td>Walking 3 mph</td>
<td>207</td>
<td>246</td>
<td>302</td>
</tr>
<tr>
<td>Stationary bike</td>
<td>295</td>
<td>352</td>
<td>431</td>
</tr>
</tbody>
</table>

Table 5. Sample Items From 70-item Frailty Index Created in CSHA Study

<table>
<thead>
<tr>
<th>Item</th>
<th>Abbreviations: CSHA, Canadian Study of Health and Aging; COPD, chronic obstructive pulmonary disease.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Functional status</td>
<td>Changes in everyday activities, problems getting dressed, bathing, toileting, grooming, or eating</td>
</tr>
<tr>
<td>Neurological and cognitive status</td>
<td>History of depression, depressed mood, sleep changes, memory changes, clouding, delirium, seizures, tremors, cerebrovascular accident, syncope, headaches, changes in mental functioning, Parkinson’s or Alzheimer’s disease</td>
</tr>
<tr>
<td>Cardiac status</td>
<td>Hypertension, myocardial infarction, coronary artery disease, congestive heart failure, arrhythmias, peripheral vascular disease, ability to walk upstairs or complete house work</td>
</tr>
<tr>
<td>Pulmonary status</td>
<td>COPD, emphysema, asthma, smoking history</td>
</tr>
<tr>
<td>Gastrointestinal status</td>
<td>Bowel habits, any abdominal problems</td>
</tr>
<tr>
<td>Other</td>
<td>Diabetes mellitus, thyroid problems, alcohol or substance abuse, kidney problems, current medications, changes in health status, and energy intake</td>
</tr>
</tbody>
</table>

Table 6. NSQIP Variables That Constitute the 11 Modified Frailty Index Variables

<table>
<thead>
<tr>
<th>Modified Frailty Index Variables</th>
<th>Abbreviation: NSQIP, National Surgical Quality Improvement Program.</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of diabetes mellitus</td>
<td></td>
</tr>
<tr>
<td>Functional status of least 2</td>
<td></td>
</tr>
<tr>
<td>History of chronic obstructive pulmonary disease or pneumonia</td>
<td></td>
</tr>
<tr>
<td>History of congestive heart failure</td>
<td></td>
</tr>
<tr>
<td>History of myocardial infarction</td>
<td></td>
</tr>
<tr>
<td>High blood pressure requiring medication</td>
<td></td>
</tr>
<tr>
<td>History of peripheral vascular disease or ischemic chest pain</td>
<td></td>
</tr>
<tr>
<td>History of impaired sensorium</td>
<td></td>
</tr>
<tr>
<td>History of transient ischemic attack or cerebrovascular accident</td>
<td></td>
</tr>
<tr>
<td>History of percutaneous coronary intervention, stenting, or angina</td>
<td></td>
</tr>
</tbody>
</table>

common characteristics of frailty in this model include 5 traits. An unintentional weight loss of greater than or equal to 4.5 kg (10 lb) in the previous year, or a greater than 5% loss of body weight at follow-up represent 1 trait. Second, strength is measured as an indicator of frailty, specifically grip strength within the lowest 20 percentile at baseline for gender. Poor endurance and slowness represent the third and fourth elements of
fifty and final element to the Fried frailty phenotype is low physical activity level as determined by a weighted score of kilocalories expended per week (Table 3). For instance, male individuals expending less than 383 kcal/wk would be considered in the lowest 20th percentile (20,21,26) (Table 4). The presence of 3 or more of these 5 elements is required for a diagnosis of frailty (20). Patients with 1 or 2 elements present can be classified as prefrail.

To evaluate the effectiveness of the frailty theory, Fried et al. (20) used data from patients who were community dwelling and found the overall prevalence in frailty for this population to be 6.9%. The study indicated frailty was more strongly associated with being African American, lower educational level, poorer income and health, and higher rates of comorbidity. (20) Assessing the 5 elements of the frailty cycle allowed Fried and colleagues to identify patients at most risk of poor healthcare outcomes. Although this 5-point assessment method serves as an invaluable tool for assessing frailty, the practicality of using the frailty phenotype to identify inpatients at risk of complications in the hospital is limited. Often, hospital patients are bedridden and most likely unable to perform the elements of Fried’s frailty phenotype tests. A separate theory posited by Rockwood et al. (25,27) involves accumulated deficits. This theory represents a more feasible tool for frailty measurement in the clinical arena (25,27).

**Table 7. Summary of Frailty Instruments**

Abbreviation: NSQIP, National Surgical Quality Improvement Program.

<table>
<thead>
<tr>
<th>Author, year</th>
<th>Frailty instrument</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fried et al., 2001</td>
<td>Frailty phenotype</td>
<td>Patients with ≥ 3 frailty criteria are diagnosed as frail. Found frailty not synonymous with comorbidity or disability, but rather comorbidity is a risk factor for and disability is an outcome of frailty.</td>
</tr>
<tr>
<td>Rockwood et al., 2001</td>
<td>Canadian Study of Health and Aging Frailty Index (CSHA-FI)</td>
<td>Used 70 metrics to diagnose frailty</td>
</tr>
<tr>
<td>Saxton &amp; Velanovich, 2011</td>
<td>CSHA-FI and modified frailty index (mFI)</td>
<td>Used CSHA-FI to retrospectively measure frailty in elective complex surgical procedures. Found that measurement of preoperative functional status using mFI helped identify patients at high risk of postoperative complications.</td>
</tr>
<tr>
<td>Tsiouris et al., 2013</td>
<td>mFI</td>
<td>Used mFI of 11 variables from mapping of CSHA-FI to NSQIP comorbidities in patients undergoing thoracic lobectomies. Found mFI may help identify patients at higher risk of postoperative complications following lobectomy.</td>
</tr>
<tr>
<td>Velanovich et al., 2013</td>
<td>mFI and accumulating deficits model of frailty</td>
<td>Used mFI of 11 variables created by mapping CSHA-FI to NSQIP database for cardiac, general, gynecologic, neurosurgical, orthopedic, plastic, thoracic, urologic, and vascular surgical procedures. Found a simplified frailty index correlated with morbidity and mortality for all surgical specialties in the study.</td>
</tr>
<tr>
<td>Cooper et al., 2016</td>
<td>Frailty phenotype and CSHA-FI</td>
<td>Used a 42-variable frailty index to prospectively compare the frailty phenotype with CSHA-FI. Found similar predictability for poor postoperative outcomes in orthopedic patients.</td>
</tr>
<tr>
<td>McIsaac et al., 2016, 2017</td>
<td>Johns Hopkins Adjusted Clinical Groups Frailty-defining diagnoses indicator</td>
<td>Found frailty was associated with increased risk of 1-year mortality following surgery</td>
</tr>
<tr>
<td>Wahl et al., 2017</td>
<td>mFI</td>
<td>Used an 11-variable mFI to diagnose frailty in orthopedic, general, and vascular surgical patients. Found an increased mFI score was associated with poor surgical outcomes, and improving patient functional status could be an area of focus to reduce readmission and complication rates.</td>
</tr>
<tr>
<td>Partridge et al., 2017</td>
<td>Comprehensive Geriatric Assessment (CGA)</td>
<td>Studied use of CGA combined with preoperative patient optimization in patients undergoing abdominal aortic aneurysm repair or lower limb arterial surgery. Found patients who received CGA and optimization had decreased length of stay, delirium, and complications.</td>
</tr>
<tr>
<td>Joseph et al., 2017</td>
<td>Trauma-Specific Frailty Index (TS-FI)</td>
<td>Created and validated a 50-variable frailty index called TS-FI. Found geriatric frail patients to be 3 times more likely to be diagnosed with failure to thrive.</td>
</tr>
<tr>
<td>Hall et al., 2017</td>
<td>Risk Adjusted Index (RAI)</td>
<td>Created and validated RAI to screen for frailty in surgical patients. RAI-C is a questionnaire that can be used prospectively to identify frailty, and RAI-A is a retrospective measurement tool comparing patient data and NSQIP data. Found that implementing a frailty screening initiative was associated with decreased mortality.</td>
</tr>
</tbody>
</table>

**Theory of Accumulated Deficits**

In the Canadian Study of Health and Aging (CSHA), Rockwood et al. (27) studied the theory of accumulated deficits in relation to frailty. During the first stage of...
the CSHA, Rockwood’s group enrolled 10,263 people 65 years and older into a 5-year prospective cohort study, where deficits were considered in aggregate, and a rules-based definition of frailty was used. In the study, a 70-item CSHA Frailty Index (CSHA-FI) was created and found to have strong predictive value with regard to poor health outcomes in elderly patients (Table 5). The deficit accumulation approach was cross-validated by counting deficits in the standardized Comprehensive Geriatric Assessment.19 Rockwood et al20 established scales for function and overall clinical frailty, with the main objective of creating a tool for risk stratification of vulnerability in the elderly population.20 The frailty index was created to identify patients at greatest risk of increased morbidity and mortality.

Frailty scores were found by dividing the number of deficits by the total number of 70 possible metrics. The closer the ratio came to 1, the frailer a patient. Specifically, the resulting ratio was used to identify someone as robust (ratio of 0-0.12), prefrail (ratio of 0.13-0.43), or frail (ratio of ≥ 0.44).29,30 These stratifications were found to be predictive of increased morbidity and mortality.10,16,27,31,32 Rockwood et al19 and others22,25 compared the frailty index to Fried’s phenotype and found it comparable for predicting adverse health outcomes. Despite its usefulness in predicting adverse outcomes, the 70-item scale is time-consuming to perform in a clinical environment. Additional retrospective research projects using smaller frailty indexes have shown that variations of the CSHA frailty index may be useful in identifying vulnerable patients.2,14,16,17,33-35

**Modified Frailty Index**

Research has validated use of a modified frailty index (mFI) for identifying patients at high risk of poor health outcomes following surgery. Various modified frailty indexes have used 5 to 42 variables from the CSHA frailty index with similar predictability of poor outcomes.14,16,34

For example, Velanovich et al14 matched 11 items from the National Surgical Quality Improvement Program (NSQIP) to the 70-item CSHA frailty index and found a stepwise increase in mortality and morbidity for each unit increase in frailty index across surgical specialties (Table 6). Additional research from Saxton and Velanovich36 created an mFI by matching 15 variables from the NSQIP to 11 variables from the CSHA-FI.

Hall et al37 developed a similar mFI, the Risk Analysis Index (RAI). The RAI represents a 14-item tool for measuring frailty in surgical patients. It can be used prospectively to identify frail patients by using a clinical questionnaire (RAI-C) or retrospectively using variables from the NSQIP (RAI-A).37 The RAI is based on adaptations from the Minimum Data Set Mortality Risk Index-Revised, in which 12 variables that consistently predicted mortality were selected. From these variables, a 14-point survey was created for measurement purposes.37 The easily administered survey relies on patient reports. In their research, Hall et al38 validated the RAI-C and the RAI-A as effective tools for measuring frailty compared with other measures. The RAI has similar predictive ability regarding frailty as the CSHA-FI and the mFI created by Saxton and Velanovich, and moderate correlation between these measures has been noted.37 The research by Hall and colleagues38 demonstrated that a large frailty screening initiative is feasible and can allow the directing of resources to patients who need them most in an effort to improve outcomes. Table 7 summarizes the frailty measures discussed in this article, which are rooted in the gerontology literature.20 Frailty has been validated in retrospective research.19-21,42

The lack of a clear, concise assessment of frailty has led to the creation of a multitude of instruments to measure it. The frailty phenotype and various frailty indexes have been validated through retrospective research. However, high-quality prospective studies are currently lacking. Despite the usefulness of frailty measurements, tremendous difficulty operationalizing them clinically remains. The creation of a standardized modified frailty index using the theory of accumulated deficits may help address the use of frailty as a predictor of outcomes following surgical procedures.

A standardized mFI applied during the preoperative setting may help anesthesia providers stratify patient risk of complications and direct more resources to the patients with higher frailty scores. Potential resources include cardiac, pulmonary, neurologic, and geriatric consults in an effort to establish patient baseline status and create a tailored care plan to improve outcomes.3,38,39 During the intraoperative setting, anesthesia plans for these high-risk patients could be modified. For instance, maintaining normothermia and normal blood glucose levels, avoiding benzodiazepines and anticholinergic medicines, minimizing narcotic administration, and use of nonopioid pain medications could mitigate potential postoperative delirium. Perhaps, types of anesthesia techniques other than general endotracheal anesthesia such as regional anesthesia could also reduce intravenous narcotic administration, thereby avoiding side effects such as somnolence and nausea.3 During the postoperative setting, avoiding patient shivering, maintaining blood pressure as close to baseline as possible, normal blood glucose levels, and monitoring for delirium and falls risk could reduce the potential for poor outcomes following surgery.3,40,41

**Conclusion**

Frailty has been validated in retrospective research.19-21,42 There is benefit to the use of a high-quality multidisciplinary preoperative assessment, including an mFI as a means to identify surgical patients at high-risk of poor

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**Table 7**

<table>
<thead>
<tr>
<th>Frailty Index</th>
<th>Description</th>
</tr>
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<tr>
<td>CSHA-FI</td>
<td>70-item scale for frailty in the elderly population</td>
</tr>
<tr>
<td>RAI</td>
<td>14-item tool for measuring frailty in surgical patients</td>
</tr>
<tr>
<td>mFI</td>
<td>Modified Frailty Index</td>
</tr>
<tr>
<td>RAI-C</td>
<td>Retrospectively using variables from the NSQIP</td>
</tr>
<tr>
<td>RAI-A</td>
<td>Prospectively to identify frail patients</td>
</tr>
</tbody>
</table>

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surgical outcomes. As the number of surgical procedures performed annually on this patient population increases, improving postoperative outcomes will gain increased importance. Fried's theory regarding the phenotype of frailty and Rockwood's Accumulated Deficits Theory of Frailty represent foundations on which to base future frailty studies. Using frailty scores may improve postoperative outcomes in surgical patients because high-risk patients can be identify early thereby leading to earlier mobilization of care resources in an effort to optimize these patients.

REFERENCES


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April 1, 2019 - December 31, 2021, Michigan; Institute for Post-Graduate Education, Inc. - 50 CEC.

April 1, 2019 - January 1, 2022, Tennessee; Healthy Visions-MyCEcredit - 20 CEC.


April 1, 2019 - April 28, 2019, Ohio; The Cleveland Clinic Educational Foundation - 51.5 CEC. “Comprehensive Anesthesiology Review.” InterContinental Hotel and Bank of America Conference Center, Cleveland, OH. Darcy Lange, 1950 Richmond Road, TR204, Lyndhurst, OH 44124; (216) 448-8707; fax (216) 445-9406; email, langed@ccf.org; www.ccfcmce.org/GoAesReview.

April 26, 2019 - April 28, 2019, Nebraska; Nebraska Association of Nurse Anesthetists - 20 CEC. “2019 Nebraska Association of Nurse Anesthetists Spring Conference.” Embassy Suites Omaha - LaVista and Courtyard by Marriott, La Vista, NE. Emily Wilcox, 1633 Normandy Court, Suite A, Lincoln, NE 68512; (402) 476-3852; email, ewilcox@assocoffice.net; http://neaan.org.

May 4, 2019 - May 5, 2019, Virginia; Nurse Anesthesia Faculty Associate - 15 CEC. “Nurse Anesthesia Faculty and Administrator Workshop.” VCU Nurse Anesthesia Department, Richmond, VA. Suzanne Wright, Box 980226, Richmond, VA 23298; (804) 828-6734; email, nafa@vcu.edu; www.nafa-va.org.


May 6, 2019 - May 11, 2019, South Carolina; Anesthesia Business Seminars - 36 CEC. “Myrtle Beach a la carte: Business of Anesthesia & Beyond.” Island Vista Resort, Myrtle Beach, SC. Laura Moritz, 1080 Ayers Road, Moneta, VA 24121; (336) 577-8450; email, laura@associationmeetingplanners.com; www.AnesthesiaBusinessSeminars.com.

May 9, 2019 - May 12, 2019, Illinois; Northwest Anesthesia Seminars - 20 CEC. “Topics in Anesthesia.” Hyatt Chicago Magnificent Mile, Chicago, IL. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@northwestseminars.com; www.northwestseminars.com.

May 9, 2019 - May 12, 2019, Massachusetts; Boston Children's Hospital - 35 CEC. “Principles of Pediatric Anesthesia and Critical Care.” Fairmont Copley Plaza, Boston, MA. Amanda Buckley, 300 Longwood Ave, Bader 376 Anesthesia, Boston, MA 02115; (617) 355-7737; email, anesthesiadepartment@childrens.harvard.edu; www.PediatricAnesthesiaConference.com.

May 9, 2019 - May 16, 2019, Spain; Northwest Anesthesia Seminars - 20 CEC. “Clinical Concerns in Anesthesia.” 11-Night Western Mediterranean Cruise, aboard Celebrity Infinity. Roundtrip from Barcelona, Spain. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/europe/19mwc.html.

May 13, 2019 - May 16, 2019, New York; Northwest Anesthesia Seminars - 20 CEC. “Topics in Anesthesia.” Marriott New York, Brooklyn Bridge, Brooklyn, NY. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/newyork/19my.html.

May 13, 2019 - May 17, 2019, Massachusetts; Harvard Medical School - 50.75 CEC. “Anesthesiology Update 2019.” Fairmont Copley Plaza Hotel, Boston, MA. Ronald Mayes, 330 Brookline Avenue, Yaminis 219, Boston, MA 02215; (617) 667-5039; fax (617) 667-5031; email, rmayes@bdmc.harvard.edu; https://anesthesiology.hmscme.com.

May 16, 2019 - May 18, 2019, Ohio; American Association of Nurse Anesthetists - 23.5 CEC. “Spring 2019 Spinal Epidural with Obstetric Essentials Workshop.” University of Cincinnati, College of Nursing, Cincinnati, OH. (847) 655-8797; email, meetings@aana.com; www.aana.com/meetings.

May 16, 2019 - May 19, 2019, Florida; Northwest Seminars - 24 CEC. “Critical Care: The Team Approach.” Gaylord Palms Resort & Convention Center, Kissimmee (Orlando), FL. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@northwestseminars.com; www.northwestseminars.com/kissimmee19cmki.html.

May 17, 2019 - May 19, 2019, Florida; Airway Management Education Center - 17.75 CEC. “The Difficult Airway Course: Anesthesia.” Hyatt Regency, Orlando, FL. Registration Office, 333 South State Street, Suite V-324, Lake Oswego, OR 97034; (866) 924-7929; fax (404) 795-0711; email, registrations@theairwaysite.com; www.theairwaysite.com.

May 20, 2019 - May 23, 2019, California; Northwest Anesthesia Seminars - 24 CEC. “Topics in Anesthesia.” Hyatt Regency Indian Wells Resort & Spa, Indian Wells, CA. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/indianwells/19miw.html.


May 20, 2019 - May 24, 2019, Puerto Rico; Northwest Seminars - 20 CEC. “Topics in Emergency Medicine.” Condado Vanderbilt Hotel, San Juan, Puerto Rico. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@northwestseminars.com; www.northwestseminars.com/puertorico/19rpmir.html.

May 21, 2019 - May 24, 2019, Texas; Northwest Anesthesia Seminars - 24 CEC. “Current Topics in Anesthesia.” Fairmont Austin, Austin, TX. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com.


May 27, 2019 - May 31, 2019, Iceland; Northwest Anesthesia Seminars - 20 CEC. “Current Anesthesia Practice.” Hilton Reykjavik Nordica, Reykjavik, Iceland. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/iceland/19mi.html.

May 29, 2019 - June 1, 2019, South Carolina; Nurse Anesthesiology Faculty Associates - 22 CEC. “33rd Annual Anesthesia Seminar.” Hilton Head Marriott Resort and Spa, Hilton Head Island, SC. Suzanne Wright, Box 980226, Richmond, VA 23298; (804) 828-6734; fax (804) 828-0581; email, nafa@vcu.edu; www.nafa_va.org.

June 1, 2019 - June 1, 2019, Kentucky; University of Louisville - 7.5 CEC. “‘Mastering the Difficult Airway.” University of Louisville - School of Medicine - B bldg, Louisville, KY. Amy Reid, 530 S Jackson Street, RM C2A01, Louisville, KY 40202; (502) 852-1735; email, amreid@louisville.edu; http://bit.ly/airway19.

June 1, 2019 - June 20, 2019, North Carolina; Wake Forest School of Medicine Nurse Anesthesia Program - 100 CEC. “Comprehensive Review of Anesthesia.” Wake Forest Nurse Anesthesia Program, Winston-Salem, NC. Kristin Henderson, 525 Vine St, Ste 230, Winston-Salem, NC 27101; (336) 716-1411; fax (336) 716-1412; email, tnainfo@wakehealth.edu; http://www.wakehealth.edu/Nurse-Anesthesia-Program.
June 3, 2019 - June 6, 2019, South Carolina; Encore Symposiums - 23 CEC. “Charleston Harbor-Front Sanctuary 2019 Encore Symposium.” Beach Club at Charleston Harbor Resort & Marina, Mt Pleasant, SC. Nancy LaBrile, 1907 Loch Lombod Ct, Winston-Salem, NC 27106; (336) 768-9095; fax (336) 768-9055; email, nancy@escrnas.com; www.escrnas.com.

June 3, 2019 - June 6, 2019, North Carolina; Northwest Anesthesia Seminars - 20 CEC. “Topics in Anesthesia.” The Sanderling, Duck, NC. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/ducknc/19jnc.html.

June 5, 2019 - June 8, 2019, Idaho; Northwest Anesthesia Seminars - 20 CEC. “Relevant Topics in Anesthesia.” Hyatt Place Boise Downtown, Boise, ID. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/boise/19jbd.html.

June 5, 2019 - June 12, 2019, Ireland; Northwest Anesthesia Seminars - 20 CEC. “Topics in Anesthesia.” 11-Night Ireland and Iceland Cruise, aboard Celebrity Reflection, Roundtrip from Dublin, Ireland. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/europe/19jrc.html.

June 6, 2019 - June 13, 2019, Georgia; Northwest Anesthesia Seminars - 20 CEC. “Topics in Anesthesia.” The Ritz-Carlton Reynolds, Lake Oconee, Greensboro, GA. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/lakeoconee/19jgg.html.

June 10, 2019 - June 14, 2019, California; Northwest Anesthesia Seminars - 20 CEC. “Anesthesia Update.” Tenaya Lodge, Fish Camp (Yosemite National Park), CA. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/yosemite/19jyf.html.

June 13, 2019 - June 16, 2019, Florida; Northwest Anesthesia Seminars - 20 CEC. “Topics in Anesthesia.” Margaritaville Key West Resort, Key West, FL. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/keywest/19kw.html.

June 17, 2019 - June 21, 2019, Grand Cayman; Northwest Anesthesia Seminars - 20 CEC. “Anesthesia Update.” Grand Cayman Marriott Beach Resort, Grand Cayman, Cayman Islands. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/grandcayman/19jgg.html.

June 17, 2019 - June 21, 2019, Wyoming; Northwest Anesthesia Seminars - 20 CEC. “Relevant Topics in Anesthesia.” Hotel Terra, Jackson Hole, WY. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/teton/19jwy.html.

June 20, 2019 - June 23, 2019, Tennessee; Northwest Seminars - 24 CEC. “Critical Care: The Team Approach.” Renaissance Nashville Hotel, Nashville, TN. NWS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@northwestseminars.com; www.northwestseminars.com/nashville/19jcn.htm.

June 21, 2019 - June 23, 2019, Alabama; Lower Alabama Continuing Education Seminars, Inc. - 20 CEC. “2019 Orange Beach Sun and Fun Summer Seminar.” Perdido Beach Resort, Orange Beach, AL. Laura Lesley, 6218 Fox Br, Trussville, AL 35173; (205) 642-8052; email, Laura@lacesinc.com; https://lacesinc.com/2019dates/orangebeachsunandfunseminar.

June 22, 2019 - June 22, 2019, Texas; SLAM Airway Training Institute - 10 CEC. “SLAM I-Day Course on Emergency and Difficult Adult Airway.” SLAM Airway Training Center, Rowlett, TX. James Rich, 3250 Hwy 78, Suite 730453, Sachse, TX 75048; (972) 369-2098; fax (210) 910-6242; email, slamairwayinfo@gmail.com; www.slamairway.com.

June 24, 2019 - June 27, 2019, Nevada; Northwest Anesthesia Seminars - 24 CEC. “Anesthesia Jackpot.” Encore at Wynn Las Vegas, Las Vegas, NV. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/vegas/19jnv.html.

June 25, 2019 - June 28, 2019, Georgia; Northwest Seminars - 20 CEC. “Current Topics in Pediatric Emergency Medicine.” Hyatt Regency Savannah, Savannah, GA. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@northwestseminars.com; www.northwestseminars.com/savannah/19jemga.html.

June 26, 2019 - June 29, 2019, District of Columbia; Northwest Anesthesia Seminars - 20 CEC. “Relevant Topics in Anesthesia.” The Westin Georgetown, Washington, DC. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/dc/19jdc.html.

July 1, 2019 - July 12, 2019, Hawaii; Northwest Anesthesia Seminars - 20 CEC. “Current Anesthesia Practice.” The Kahala Hotel & Resort, Honolulu, HI. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/hawaii/19jho.html.

July 8, 2019 - July 12, 2019, Montana; Northwest Anesthesia Seminars - 20 CEC. “Current Anesthesia Practice.” The Lodge at Whitefish Lake, Whitefish (Glacier National Park), MT. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/whitefish/19jwl.html.

July 11, 2019 - July 14, 2019, Florida; Northwest Seminars - 20 CEC. “Topics in Emergency Medicine.” Tradewinds Island Grand Resort, St Pete Beach, FL. NWS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@northwestseminars.com; www.northwestseminars.com/stpetebeach/19jemfp.html.


July 15, 2019 - July 19, 2019, Florida; Northwest Seminars - 20 CEC. “Current Topics in Emergency Medicine.” 7-Night Eastern Caribbean Cruise, aboard Allure of the Seas, Roundtrip from Fort Lauderdale, FL. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@northwestseminars.com; www.northwestseminars.com/caribbean/19emjcc.html.

July 15, 2019 - July 19, 2019, Massachusetts; Northwest Anesthesia Seminars - 20 CEC. “Topics in Anesthesia.” Sea Crest Beach Hotel, Falmouth (Cape Cod), MA. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/capecod/19jma.html.

July 15, 2019 - July 20, 2019, New Jersey; Northwest Anesthesia Seminars - 20 CEC. “Current Anesthesia Practice.” 7-Day Bermuda Cruise, aboard Celebrity Summit, Roundtrip from Cape Liberty, NJ. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/bermuda/19jbeb.html.

July 15, 2019 - July 20, 2019, Washington; Northwest Anesthesia Seminars - 20 CEC. “Topics in Emergency Medicine.” Alaskan Explorer via Hubbard Glacier Cruise, aboard ms Noordam, Vancouver, British Columbia, Canada to Seward, Alaska. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/alaska/19emaakl.html.

July 18, 2019 - July 22, 2019, California; Northwest Anesthesia Seminars - 20 CEC. “Current Topics in Trauma Anesthesia.” Marriott Resort & Spa at Grande Dunes, Myrtle Beach, SC. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/myrtlebeach/19jmb.html.


August 1, 2019 - August 4, 2019, California; Northwest Anesthesia Seminars - 24 CEC. “Anesthesia Update.” Hotel Solamar, San Diego, CA. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/sandiego/19asd.html.

August 1, 2019 - August 4, 2019, California; Northwest Anesthesia Seminars - 20 CEC. “Current Topics in Emergency Medicine.” Paséa Hotel & Spa, Huntington Beach, CA. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/huntingtonbeach/19emahb.html.

August 5, 2019 - August 9, 2019, Canada; Northwest Anesthesia Seminars - 20 CEC. “Clinical Topics in Anesthesia.” The Rimrock Resort Hotel, Banff, Alberta, Canada. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/banff/19aca.html.

August 5, 2019 - August 10, 2019, Alaskan Cruise; Northwest Anesthesia Seminars - 20 CEC. “Topics in Anesthesia.” Vanderwood toeward via the Inside Passage Cruise, aboard ms Noordam, Vancouver, British Columbia, Canada to Seward, Alaska. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/alaska/19akac.html.

August 9, 2019 - August 9, 2019, Illinois; American Association of Nurse Anesthetists - 3.5 CEC. “2019 Fundamentals in Perioperative Transesophageal Echocardiogram Workshop - MORNING.” Hyatt Regency, Chicago, IL; (847) 655-8797; email, meetings@aana.com; www.aana.com/meetings.

August 9, 2019 - August 9, 2019, Illinois; American Association of Nurse Anesthetists - 7.25 CEC. “2019 Obstetric Regional Anesthesia - A Focused Review and Clinical Applications.” Hyatt Regency, Chicago, IL; (847) 655-8797; email, meetings@aana.com; www.aana.com/meetings.

August 9, 2019 - August 9, 2019, Illinois; American Association of Nurse Anesthetists - 4 CEC. “2019 Ultrasound-Guided Peripheral Nerve Blocks - A Focused Review and Clinical Applications.” Hyatt Regency, Chicago, IL; (847) 655-8797; email, meetings@aana.com; www.aana.com/meetings.

August 9, 2019 - August 13, 2019, Illinois; American Association of Nurse Anesthetists. “Nurse Anesthesia Annual Congress.” Hyatt Regency Hotel, Chicago, IL; (847) 655-8797; email, meetings@aana.com; www.aana.com/meetings.

August 12, 2019 - August 16, 2019, Montana; Northwest Seminars - 20 CEC. “Topics in Emergency Medicine.” The Lodge at Whitefish Lake, Whitefish (Glacier National Park), MT. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@northwestseminars.com; www.northwestseminars.com/whitefish/19emawl.html.

August 19, 2019 - August 23, 2019, Alaska; Northwest Seminars - 20 CEC. “Current Topics in Emergency Medicine.” The Lakefront Anchorage, Anchorage, AK. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@northwestseminars.com; www.northwestseminars.com/alaska/19emaakl.html.

August 20, 2019 - August 28, 2019, Italy; Northwest Anesthesia Seminars - 20 CEC. “Clinical Concerns in Anesthesia.” 11-Night Amalfi Coast & Greek Isles Cruise, aboard Celebrity Edge. Roundtrip from Rome (Civitavecchia), Italy. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/europe/19jaacg.html.
August 21, 2019 - August 24, 2019, Massachusetts; Northwest Anesthesia Seminars - 24 CEC. “Anesthesia Update.” Royal Sonesta Boston, Boston, MA. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/boston/19abt.html.

September 2, 2019 - September 2, 2019, Nevada; Northwest Anesthesia Seminars - 8 CEC. “Business Concepts in Healthcare.” The Palms Casino Resort, Las Vegas, NV. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/businessconcepts/19bUs.html.

September 3, 2019 - September 6, 2019, Nevada; Northwest Anesthesia Seminars - 20 CEC. “Topics in Anesthesia.” The Palms Casino Resort, Las Vegas, NV. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/vegas/19shv.html.

September 5, 2019 - September 8, 2019, New Mexico; New Mexico Association of Nurse Anesthetists - 24 CEC. “Emergency Medicine.” Hotel Santa Fe, Santa Fe, NM. Laura Moritz, 1080 Ayers Rd, Moneta, VA 24121; (336) 577-8450; email, Laura@AssociationMeetingPlanners.com; http://www.nmana.org/.

September 9, 2019 - September 12, 2019, Nevada; Northwest Seminars - 20 CEC. “Topics in Anesthesia.” The Palms Casino Resort, Las Vegas, NV. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/vegas/19shv.html.

September 9, 2019 - September 12, 2019, Tennessee; Med City Anesthesia Seminars - 20 CEC. “Current Topics In Anesthesia.” Hilton Nashville Downtown, Nashville, TN. Karissa Goodrich, PO Box 711, Saint Charles, MN 55972; (800) 538-0217; email, mail@medcityanesthesiaseminars.com; www.medcityanesthesiaseminars.com.

September 9, 2019 - September 13, 2019, Hungary; Northwest Anesthesia Seminars - 20 CEC. “Current Topics in Anesthesia.” The Ritz-Carlton, Budapest, Hungary. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/budapest/19sbh.html.

September 12, 2019 - September 15, 2019, Alabama; Northwest Anesthesia Seminars - 24 CEC. “Relevant Topics in Anesthesia.” The Lodge at Gulf State Park, a Hilton Hotel, Gulf Shores, AL. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/gulfshores/19sal.html.

September 13, 2019 - September 15 2019, Washington; Airway Management Education Center - 17.75 CEC. “The Difficult Airway Course: Anesthesia.” Hyatt Regency, Seattle, WA. Registration Office, 333 South State Street, Suite V-324, Lake Oswego, OR 97034; (866) 924-7929; fax (804) 795-0711; email, registrations@theairwaysite.com; www.theairwaysite.com.

September 15, 2019 - September 15, 2019, Maine; Encore Symposiums - 8 CEC. “Encore Symposiums Pharmacology CPC Review Course.” Cliff House, Cape Neddick, ME. Nancy LaBrie, 1907 Loch Lomond Ct, Winston-Salem, NC 27106; (336) 768-9095; fax (336) 768-9055; email, nancy@escrnas.com; www.escrnas.com.

September 16, 2019 - September 18, 2019, Tennessee; Healthy Visions - American School of Clinical Hypnosis - 20 CEC. “Pain Management With Hypnosis.” Healthy Visions - American School of Clinical Hypnosis, Clinton, TN. Ron Eslinger, 351 Market Street, Clinton, TN 37716; (865) 269-4616; email, ron@eslinger.net; www.healthyvisions.com.

September 16, 2019 - September 19, 2019, Maine; Encore Symposiums - 23 CEC. “New England at the Cliff House Encore Symposium.” Cliff House, Cape Neddick, ME. Nancy LaBrie, 1907 Loch Lomond Ct, Winston-Salem, NC 27106; (336) 768-9095; fax (336) 768-9055; email, nancy@escrnas.com; www.escrnas.com.

September 16, 2019 - September 19, 2019, Maine; Med City Anesthesia Seminars - 20 CEC. “Current Topics In Anesthesia.” Tenaya Lodge, Fish Camp (Yosemite National Park), CA. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/yosemite/19sy.html.

September 16, 2019 - September 26, 2019, Europe - Iberian Adventure Cruise; Northwest Anesthesia Seminars - 20 CEC. “Current Topics in Anesthesia.” 12-Night Iberian Adventure Cruise, aboard ms Nieuw Statendam, from Amsterdam, the Netherlands to Civitavecchia (Rome), Italy. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/europe/19asic.html.

September 18, 2019 - September 21, 2019, Arizona; Northwest Anesthesia Seminars - 20 CEC. “Current Anesthesia Practice.” Hilton Sedona Resort at Bell Rock, Sedona, AZ. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/sedona/19saz.html.

September 19, 2019 - September 22, 2019, South Carolina; Northwest Anesthesia Seminars - 20 CEC. “Relevant Topics in Anesthesia.” The Westin Hilton Head Island Resort & Spa, Hilton Head Island, SC. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/hiltonhead/19shh.html.

September 23, 2019 - September 26, 2019, Arizona; Northwest Seminars - 20 CEC. “Pediatric Emergency Medicine.” Hilton Sedona Resort at Bell Rock, Sedona, AZ. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/sedona/19msaz.html.

September 23, 2019 - September 26, 2019, California; Cornerstone Anesthesia Conferences - 21 CEC. “Anesthesia Update in Wine Country.” Fairmont Sonoma Mission Inn & Spa, Sonoma, CA. Jayne Reuter, PO Box 7214, Houston, TX 77248; (281) 836-0777; email, info@CornerstoneAnesthesiaConferences.com; www.CornerstoneAnesthesiaConferences.com.

September 23, 2019 - September 26, 2019, Maine; Summit Anesthesia Seminars, LLC - 20 CEC. “Fall Foliage in Bar Harbor.” Bar Harbor Inn & Spa, Bar Harbor, ME. Rebecca Sullivan, PO Box 215, New Stanton, PA 15672; (888) 676- CRNA; email, summitanesthesiaseminars@gmail.com; www.summitanesthesiaseminars.com.

September 23, 2019 - September 27, 2019, Wyoming; Northwest Anesthesia Seminars - 20 CEC. “Clinical Anesthesia Update.” Hotel Terra, Tetons Village, WY. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/tetons/19wy.html.

September 30, 2019 - October 3, 2019, Florida; Northwest Anesthesia Seminars - 20 CEC. “Anesthesia Update.” Ritz Carlton Amelia Island, Amelia Island, FL. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/ameliaisland/19ai.html.
October 3, 2019 - October 6, 2019, Tennessee; Northwest Anesthesia Seminars - 20 CEC. “Anesthesia Spectrum.” The Park Vista, a Doubletree Hotel, Gatlinburg, TN. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/gatlinburg/19oga.html.


October 4, 2019 - October 6, 2019, Georgia; Georgia Association of Nurse Anesthetists - 16 CEC. “Georgia Association Symposium, GAS2019.” The Westin Buckhead Atlanta, Atlanta, GA. Laura Moritz, 1080 Ayers Rd, Moneta, VA 24121; (336) 377-8450; email, laura@AssociationMeetingPlanners.com; www.GANA.org.

October 4, 2019 - October 6, 2019, Illinois; Airway Management Education Center - 17.75 CEC. “The Difficult Airway Course: Anesthesia.” Hyatt Regency McCormick Place, Chicago, IL. Registration Office, 333 South State Street, Suite V-324, Lake Oswego, OR 97034; (866) 924-7929; fax (404) 795-0711; email, registrations@theairwaysite.com; www.theairwaysite.com.


October 6, 2019 - October 11, 2019, Mexico; Northwest Anesthesia Seminars - 20 CEC. “Current Topics in Anesthesia.” Hyatt Ziva Los Cabos, Los Cabos, Mexico. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/cabo/19omx.html.

October 7, 2019 - October 10, 2019, Rhode Island; Northwest Anesthesia Seminars - 20 CEC. “Current Topics in Anesthesia.” Gurney’s Newport Resort & Marina, Newport, RI. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/newport/19onp.html.

October 7, 2019 - October 11, 2019, Utah; Northwest Seminars - 20 CEC. “Topics in Emergency Medicine.” SpringHill Suites Springdale Zion National Park, Springdale, UT. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@northwestseminars.com; www.northwestseminars.com/zion/19emout.html.


October 13, 2019 - October 13, 2019, Massachusetts; Encore Symposiums - 8 CEC. “Encore Symposiums Pharmacology CPC Review Course.” Chatham Bars Inn, Chatham, MA. Nancy LaBrie, 1907 Loch Lomond Ct, Winston-Salem, NC 27106; (336) 768-9095; fax (336) 768-9055; email, nancy@escrnas.com; www.escrnas.com.

October 14, 2019 - October 17, 2019, Missouri; Northwest Anesthesia Seminars - 20 CEC. “Current Topics in Anesthesia.” Kansas City Marriott Downtown, Kansas City, MO. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/kansascity/19okc.html.

October 14, 2019 - October 18, 2019, Hawaii; Northwest Seminars - 20 CEC. “Topics in Emergency Medicine.” Hyatt Regency Maui, Lahaina, Maui, HI. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@northwestseminars.com; www.northwestseminars.com/maui/19emohi.html.

October 28, 2019 - October 31, 2019, Virginia; Northwest Anesthesia Seminars - 20 CEC. “Current Topics in Anesthesia.” Salamander Resort & Spa, Middleburg, VA. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/virginia/19ova.html.


November 4, 2019 - November 7, 2019, California; Northwest Seminars - 20 CEC. “Topics in Emergency Medicine.” The Ritz-Carlton, Half Moon Bay, Half Moon Bay, CA. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@northwestseminars.com; www.northwestseminars.com/halfmoonbay/19emhm.html.

November 7, 2019 - November 10, 2019, Florida; Northwest Anesthesia Seminars - 20 CEC. “Keys in Anesthesia.” Margaritaville Key West Resort, Key West, FL. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/keywest/19nkw.html.

November 10, 2019 - November 15, 2019, Hawaii; Northwest Anesthesia Seminars - 20 CEC. “Relevant Topics in Anesthesia.” Hyatt Regency Maui, Lahaina, Maui, HI. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/mau/i19nhf.html.

November 14, 2019 - November 17, 2019, Florida; Northwest Seminars - 20 CEC. “Topics in Emergency Medicine.” Margaritaville Resort & Marina Key West, Key West, FL. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@northwestseminars.com; www.northwestseminars.com/keywest/19emkmw.html.

November 17, 2019 - November 21, 2019, Florida; Northwest Anesthesia Seminars - 20 CEC. “Topics in Anesthesia.” 7-Night Eastern Caribbean Cruise, aboard ms Nieuw Amsterdam, Roundtrip from Fort Lauderdale, FL. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/caribbean/19necc.html.
November 17, 2019 - November 22, 2019, Turks & Caicos Islands; Northwest Anesthesia Seminars - 20 CEC. “Current Topics in Anesthesia.” Beaches Turks & Caicos Resort Villages & Spa, Providenciales, Turks and Caicos Islands. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/turks19ntc.html.

November 21, 2019 - November 24, 2019, New Mexico; Northwest Anesthesia Seminars - 24 CEC. “Current Challenges in Pain Management.” Hotel Albuquerque at Old Town, Albuquerque, NM. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/albuquerque19nmnm.html.

December 3, 2019 - December 6, 2019, California; Northwest Seminars - 20 CEC. “Emergency Medicine Update.” Sofitel Los Angeles at Beverly Hills, Los Angeles, CA. NWS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nws.com; www.nws.com/losangeles/19emdla.html.

December 3, 2019 - December 6, 2019, Georgia; Northwest Anesthesia Seminars - 24 CEC. “Current Topics in Anesthesia.” Hyatt Regency Savannah, Savannah, GA. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/savannah/19dga.html.

December 5, 2019 - December 8, 2019, Texas; Northwest Anesthesia Seminars - 24 CEC. “Current Challenges in Pediatric Anesthesia.” Hyatt Regency San Antonio Riverwalk, San Antonio, TX. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/sanantonio/19dtx.html.

December 5, 2019 - December 8, 2019, Virginia; Northwest Anesthesia Seminars - 20 CEC. “Topics in Anesthesia.” The Greenbrier Resort, White Sulphur Springs, WV. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/whitesulphursprings/19dvwv.html.

December 8, 2019 - December 13, 2019, Aruba; Northwest Anesthesia Seminars - 24 CEC. “Topics in Anesthesia.” The Hyatt Regency Aruba Resort, Palm Beach, Aruba. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/aruba/19dar.html.

December 9, 2019 - December 9, 2019, Nevada; Northwest Anesthesia Seminars - 8 CEC. “Business Concepts in Healthcare.” Encore at Wynn Las Vegas, Las Vegas, NV. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/businessconcepts/19BUS.html.

December 10, 2019 - December 13, 2019, Nevada; Northwest Anesthesia Seminars - 20 CEC. “Cardiothoracic and Vascular Anesthesia Update.” Encore at Wynn Las Vegas, Las Vegas, NV. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/vegas/19dvlv.html.

December 12, 2019 - December 15, 2019, Florida; Northwest Anesthesia Seminars - 20 CEC. “Topics in Emergency Medicine.” The Palms Hotel & Spa, Miami (South Beach), FL. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/savannah/19dsmh.html.

December 16, 2019 - December 19, 2019, Florida; Northwest Anesthesia Seminars - 20 CEC. “Topics in Anesthesia.” The Palms Hotel & Spa, Miami (South Beach), FL. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/miami/19dshb.html.

December 30, 2019 - January 4, 2020, Florida; Northwest Anesthesia Seminars - 20 CEC. “Current Anesthesia Practice.” 7-Night Eastern Caribbean/New Years Cruise, aboard Celebrity Edge, Roundtrip from Fort Lauderdale, FL. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/caribbean/19dec.html.

January 9, 2020 - January 12, 2020, Florida; Northwest Anesthesia Seminars - 24 CEC. “Current Topics in Anesthesia.” Key Largo Bay Marriott Beach Resort, Key Largo, FL. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com/keylargo/20jfl.html.

January 16, 2020 - January 24, 2020, Australia; Northwest Anesthesia Seminars - 24 CEC. “Clinical Concerns in Anesthesia.” 11-Night Great Barrier Reef Cruise, aboard Celebrity Solstice, Roundtrip from Sydney, Australia. NWAS, 1412 N 5th Ave, Pasco, WA 99301; (800) 222-6927; fax (509) 547-1265; email, info@nwas.com; www.nwas.com.
Index for Advertisers

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AANA Membership Inside front cover, 159, 160
AANA Publishing 144
AANA Thanks Its Corporate Partners 151
Core Concepts 160
Havel’s 89
Merck Back cover
Texas Christian University 114

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Baylor, Scott & White Health 123
Mednax Services, Inc. 93
Middle Tennessee School of Anesthesia 104
Midwestern University 168
United Anesthesia Associates 168

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