

Educating the 2018 Cohort of Student Registered Nurse Anesthetists on the Diagnosis and

Management of Perioperative Bronchospasm using a Simulation Scenario

Jenee Smith RN, BSN, SRNA & Joseph Przybylowicz RN, BSN, SRNA

Nurse Anesthesia Program, Adventist University of Health Sciences

Project Mentor: Ed Delgado, DO, Chief Anesthesiologist, USAP-JLR Medical Group

Project Chair: Manuel Tolosa, DNAP, CRNA, ADU-NAP Department Assistant Program

Administrator and USAP- JLR Medical Group

Nurse Anesthesia Program, Adventist University of Health Sciences

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Abstract

Anesthesia providers have the responsibility to minimize potential hazards and promptly address anesthetic emergencies in order to provide safe and competent care. A perioperative bronchospasm is an uncommon event and potential anesthetic emergency that can lead to catastrophic patient outcomes from hypoxia if not corrected in a timely manner. It arises from a sudden increase in bronchial smooth muscle tone and results in the closure of small airways. Successful management and timely recognition of this crisis may pose a challenge to the novice provider. Therefore, the intent of this capstone was to conduct a thorough literature review, condense it, and present our summary of the existing literature on this topic to our sample group. This capstone assessed the current knowledge and educated our convenience sample, the class of 2018 cohort of student registered nurse anesthetists (SRNA) at Adventist University of Health Sciences on the physiology, diagnosis, and management of perioperative bronchospasm through the use of a power point presentation and simulation scenario to augment the didactic course work. The project's efficacy was determined by comparing scores of identical pre- and post-tests as well as pre- and post-simulation scenarios.

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Problem

A bronchospasm arises from a sudden increase in bronchial smooth muscle tone and results in the closure of small airways. A perioperative bronchospasm is an uncommon event and potential anesthetic emergency that can lead to catastrophic patient outcomes from hypoxia if not corrected in a timely manner. Olsson (1987) reports the incidence of bronchospasm during anesthesia as 1.7 per 1000 patients after completing a retrospective study of 136,929 patients. However, the prevalence increases significantly with conditions that increase airway reactivity. According to the 2008 American Society of Anesthesiologist (ASA) Closed Claims database, adverse respiratory events constituted 23% of the anesthesia closed claims, of these adverse events, bronchospasm was only 1% (Posner & Caplan, 2015).

A bronchospasm has multiple etiologies dependent on its occurrence during the perioperative period as well as the patient's past medical history. It is also associated with multiple anesthetic techniques and presents with varying signs and symptoms. The pharmacologic management of a bronchospasm is a multi-modality approach guided by correcting the precipitating cause (Nagelhout & Plaus, 2014). Therefore, differential diagnosis from other respiratory complications, treatment, as well as the infrequent occurrence of a perioperative bronchospasm may pose a challenge for the novice provider.

The intent of this capstone was to conduct a thorough literature review, condense it, and present our summary of the existing literature on this topic to our sample group. This capstone assessed the current knowledge and educated the class of 2018 cohort of student registered nurse anesthetists (SRNA) at Adventist University of Health Sciences on the physiology, diagnosis, and management of perioperative bronchospasm through the use of a power point presentation and simulation scenario to augment the didactic course work.

Review of Literature

A case scenario on Bronchospasm during Anesthetic Induction was published as a continued medical education article in the American Society of Anesthesiologists and gave a thorough review on detection and treatment of perioperative bronchospasms (Dewachter, Mouton-Faivre, Emala, & Beloucif, 2011). The authors concluded that fatal consequences of bronchospasm can be caused by substandard care, inadequate practice, and system failures (Dewachter et al., 2011). They also reasoned that the use of simulators can help compensate for the low frequency of perioperative bronchospasms that will occur with the average practitioner (Dewachter et al., 2011).

Research by Green, Tariq and Green (2016) compared anesthesia to the aviation industry. In both industries, the patient entrusts their lives to both the pilot and the anesthesia provider and there is minimal risk for error. Improvement in the safety of the aviation industry is linked to the routine use of aviation simulators during pilot training (Green, Tariq & Green, 2016). Inherently, simulation training has now become an adjunct to classroom instruction and clinical experience in the anesthesia curriculum.

The use of simulation training dates back to the Medieval Times (Green, et al., 2016). Nowadays, patient simulators have realistic airways, allow vascular access with drug recognition software and can even display neurological and physiological symptoms such as seizures or tears (Green, et al., 2016). With the current advancements in this technology, there are always areas that need improvement. Green, Tariq and Green (2016) also compared the simulators to actual patients and found that endotracheal intubations were similar, while LMA placement resulted in no visible chest rise in about 30% of the cases using simulators.

Although engineers are continuing to make improvements to the simulators to make them more “human-like”, there are key benefits to incorporating this mode of training. Simulators offer the benefit of crisis scenario simulation and hands-on training of procedures in a completely risk-free environment (Green, et al., 2016). Not only does simulation training improve technical skills, but it also enhances nontechnical skills such as: leadership, team work abilities, situation awareness, and decision making (Green, et al., 2016).

Green, Tariq and Green (2016), also defined three features of a clinical scenario: the initial briefing, the actual experience and a debriefing. According to Overstreet, McCarver, Shields, and Patterson (2015), grading rubrics offer an invaluable tool for debriefing. Overstreet et al. (2015) developed a simulation rubric using Examsoft to evaluate students without subjectivity. Overstreet et al. (2015) conducted a study using the rubric and concluded that it provided immediate feedback and consistency among graders.

Prior to reviewing the current literature on bronchospasm, it would be beneficial to briefly discuss the bronchial physiology and physics of gas flow as they apply to airway resistance to better comprehend the diagnosis and management modalities for a perioperative bronchospasm. In general, there are excitatory and inhibitory signaling pathway in the central nervous system that regulate bronchial constrictive responses via cholinergic outflow. Bronchopulmonary sensory receptors are innervated by myelinated A-delta ($A\delta$) and non-myelinated C fibers. Excitatory signal arising from bronchopulmonary afferents activate second order neurons in the nucleus solitary tract of the brain stem by the glutamate-AMPA pathway causing an increase in cholinergic outflow to vagal neurons resulting in bronchoconstriction. The inhibitory pathway uses gamma-aminobutyric acid (GABA) as the signaling molecule that

decreases the excitability of vagal neurons by decreasing cholinergic outflow (Haxiu et al., 2005).

There are three subtypes of muscarinic receptors found in the airway. Acetylcholine binds muscarinic 1 (M1) and 3 (M3) receptors causing smooth muscle contraction and an increase in mucous production. Stimulation of M1 and M3 receptors by acetylcholine causes smooth muscle contraction by activating a G-protein (Gq) and increases cyclic guanosine monophosphate (cGMP). Gq stimulation activates the phospholipase C second messenger system producing inositol triphosphate (IP3) causing an increase in intracellular calcium and activating myosin light chain kinase inducing smooth muscle contraction (Flood, Rathmell, & Shafer, 2015).

The sympathetic nervous system is less dominant in maintaining bronchial tone than the parasympathetic nervous system and causes bronchial dilation via activation of Beta-2 adrenergic receptors. Bronchial beta-adrenergic receptors are coupled to stimulatory G-proteins and the enzyme adenylyl cyclase that produces the secondary messenger system cyclic adenosine monophosphate (cAMP). Cyclic adenosine monophosphate activates protein kinase A (PKA) which phosphorylates various intracellular or membrane proteins. The cAMP-PKA dependent process causes smooth muscle relaxation by preventing the activation of the calcium-calmodulin complex through phosphorylation of myosin light chain kinase and increases calcium reuptake into the sarcoplasmic reticulum via a PKA-dependent phosphorylation of phospholamban. Beta-adrenoceptor-mediated smooth muscle relaxation can also occur via non-cAMP processes. This includes decreasing sarcolemma calcium influx through L-type voltage-gated calcium channels therefore decreasing intracellular calcium levels and by opening potassium channels

causing an increase in potassium conductance and membrane hyperpolarization (Tanaka, Horinouchi, & Koike, 2005).

The literature well describes the applicability of Poiseuille's law as well as Reynolds number in airway resistance to gas flow associated with a bronchospasm. Poiseuille's law describes the process in which airway resistance increases significantly as the airway lumen narrows. As resistance to gas flow increases during a bronchospasm, this causes symptoms associated with bronchospasm such as wheezing, an increase in peak inspiratory pressures, as well as a decrease in the expiratory phase of the end-tidal carbon dioxide waveform (Looseley, 2011). As the diameter of a lumen narrows, gas flow changes from laminar to turbulent, therefore increasing resistance to gas flow. During laminar flow, resistance inversely corresponds to the gas flow rate, however, during turbulent flow, resistance increases in proportion to the flow rate (Barash et al., 2013). Turbulent flow (Reynolds number >2000) is associated with a bronchospasm and an increased resistance to gas flow (Linck, 2007).

In order to properly diagnose and treat a perioperative bronchospasm, the anesthesia provider must be well informed on the etiology and occurrence of the event during the perioperative period, and common signs and symptoms, as well as conditions that increase airway reactivity.

In a study by Westhorpe et al. (2005) of 103 reports of perioperative bronchospasms, 21% were due to anaphylaxis or allergic in nature, while 79% were non-allergic. Among these 103 reported events 41% occurred during the induction of anesthesia, 43% occurred during the maintenance phase, 6% during emergence, and 10% during the recovery phase. During induction, bronchospasm was primarily related to airway irritation from laryngoscopy or intubation (55%), while the remaining causes were anaphylaxis (14%), endotracheal tube

misplacement (14%), aspiration (9%), pulmonary edema (5%), and unknown cause (3%). During the maintenance phase of anesthesia, bronchospasm was mainly related to anaphylaxis (34%), endotracheal tube malposition (i.e. endobronchial intubation) (23%), unknown cause (21%), aspiration (11%), drug induced (5%), pneumothorax (2%), profuse bronchial mucous (2%), and pulmonary edema (2%). During emergence, bronchospasm was most commonly associated with aspiration (29%) and pulmonary edema (29%), followed by unknown cause (14%), extubation spasm (14%), and inadvertent extubation (14%). Finally, during the recovery phase, unknown causes (60%), pulmonary edema (20%), anaphylaxis (10%), and aspiration (10%) were the most common causes of perioperative bronchospasm. The authors stated that bronchospasms caused by airway irritation during induction and maintenance phases of anesthesia, as well as bronchospasms caused by unknown causes during emergence and recovery occurred in patients who had predisposing factors for airway hyper-reactivity such as asthma, smoking or chronic obstructive pulmonary disease.

Fisher et al. (2009) performed a retrospective study on 183 patients referred to an allergy clinic following a bronchospasm during the induction of anesthesia. They reported that 57.3% (105/183) of the bronchospasms on induction had an allergic causative mechanism. However, a previous history of asthma was present in 52.8% of the non-allergic and 60.1% allergic bronchospasms. Therefore, reactive airway disease is frequently involved with both allergic and non- allergic mechanisms.

Although the incidence of bronchospasm during neuraxial anesthesia is significantly low due to the lack of airway manipulation, the literature does reveal case studies in which bronchospasms were triggered by spinal anesthesia. Rodilla-Fiz et al. (2016) report a case study and literature review on bronchospasm following spinal anesthesia. The proposed mechanism is

neuraxial blockade of thoracic sympathetic nerve fibers and unopposed parasympathetic stimulation of muscarinic receptors of bronchial smooth muscle. Again, a previous history of reactive airway disease, such as asthma was a risk factor and diagnosis was one of exclusion.

Looseley (2011) reports the etiology of a perioperative bronchospasm may be from an isolated event such as endotracheal intubation, pulmonary aspiration, drug induced bronchoconstriction, or inadequate anesthesia depth. A previous history of anaphylaxis, asthma, chronic obstructive pulmonary disease, upper respiratory infection (URI), smoking, and atopy increase the risk of airway reactivity and bronchospasm. Pharmacologic agents frequently given in OR implicated in bronchospasm are volatile anesthetic agents such as desflurane if administered rapidly, beta-blockers, NSAID's, and cholinesterase inhibitors. Drugs associated with histamine release may also induce a bronchospasm which include: sodium thiopental, atracurium, mivacurium, morphine, and d-tubocurarine (Looseley, 2011). The use of intravenous adenosine intraoperatively for narrow complex supraventricular tachycardia and during electrophysiology procedures such as ablations has been implicated in bronchospasms in patients with and without pre-existing reactive-airway disease (Salter, O'Donnell, Weiner, & Fischer, 2015).

Haxiu et al. (2005) report the proposed mechanism of airway hyper-reactivity causing bronchoconstriction following exposure to a noxious stimuli such as smoking or tracheal manipulation is an upregulation of excitatory glutamatergic and a downregulation of inhibitory GABAergic inhibitory influences on the airway, therefore enhancing vagal pre-ganglionic neurons susceptibility to excitatory inputs. This shifts the balance from inhibition to excitation and may cause reflex bronchoconstriction.

Parameswara (2015) describes how viral infections associated with a URI produce an immunologic and inflammatory response in the respiratory tract causing an increased production of IgE, pro-inflammatory cytokines, and antigen-antibody complexes, as well as vasoconstrictive mediators that include histamine, leukotrienes, and tachykinins. There is also a decrease in endopeptidases, the enzymes responsible for the degradation of tachykinins causing unopposed vasoconstriction from these mediators. The damage to respiratory tract epithelial cells results in a decrease of epithelial relaxing factors that include PGE2 and PGI2. Some viral infections may also cause a decrease in Beta-2 adrenergic receptors leading to increased parasympathetic afferent response of muscarinic bronchial smooth muscle receptors.

In a retrospective study conducted by Westhorpe et al. (2005), the first present sign associated with a bronchospasm due to anaphylaxis or allergy was rash (27%), increased inflation pressures (18%), wheeze (18%), hypotension (18%), decreased oxygen saturation (14%), and capnography change (5%). The first presenting sign with a non-allergic process was “bronchospasm”, however, the study did not indicate if this was wheezing or increased inflation pressures. It can be concluded from the data that “bronchospasm” signs included increased inflation pressures and wheezing (68%), a decreased oxygen saturation (24%), capnography change (3%), low tidal volume (3%), and hypotension (2%). Among the 41% of non-allergic or anaphylactic bronchospasms that occurred on induction of anesthesia, the most common presenting sign was increased inflation pressures and wheeze (86%), low oxygen saturation (8%), and capnography change (6%). Of the 43% of the non-allergic or anaphylactic bronchospasms during the maintenance phase of anesthesia, increased inflation pressures and wheezing (59%), low oxygen saturation (28%), low tidal volume (7%), capnography change (3%), and hypotension (3%). Of the 16% of the non-allergic or anaphylactic bronchospasms that

occurred during the emergence and recovery phase of anesthesia, the most common presenting signs were low oxygen saturation (50%), wheeze (44%), and hypotension (6%).

A perioperative bronchospasm may present in a variety of ways that may mirror other respiratory conditions. Looseley (2011) reports differential diagnosis for wheezing under general anesthesia including a partial endotracheal tube obstruction, endobronchial intubation, pulmonary edema, aspiration, pulmonary embolism, and tension pneumothorax. An increase in pulmonary peak pressures during mechanical ventilation may also be caused by anesthesia related equipment failure such as excessive tidal volumes, high inspiratory flow rates, small endotracheal tube diameter, endobronchial intubation, kinked or obstructed endotracheal tube, as well as patient factors such as obesity, Trendelenburg position, pneumoperitoneum, or pneumothorax. Therefore a systematic approach is required to diagnosis this problem.

The literature indicates that the initial management of a suspected bronchospasm during general anesthesia is to stop stimulation and or surgery, hand ventilate with 100% oxygen, observe chest excursion, reverse desaturation or cyanosis, and treat the development of a cutaneous rash. Immediate priorities include preventing hypoxia and reversing bronchoconstriction. The anesthetic depth should be assessed and deepened if necessary (Looseley, 2011). The endotracheal tube should be assessed for proper placement and patency along with breath sounds for wheezing or absent breath sounds with severe obstruction. The patient's medical history should be considered, especially that which relates to reactive airway disease, recent URI, or smoking. The anesthetic drugs, antibiotics, and blood products should be reviewed for the possibility of an allergic reaction. The onset of a rash, wheezing, increased pulmonary pressures, and hypotension strongly suggests anaphylaxis. The phase of anesthesia (i.e. induction, maintenance, emergence & recovery) should be considered in the assessment of a

bronchospasm, as the etiologies of a bronchospasm vary in each phase (Westhorpe et al., 2005). Aspiration should always be considered in an awake patient or with the use of a laryngeal mask airway (LMA) with signs of a bronchospasm. Alternate diagnoses such as pneumothorax and pulmonary edema should be considered after ruling out a bronchospasm and more common conditions that resemble a bronchospasm.

Pharmacologic treatment of a perioperative bronchospasm should be focused on five categories: underlying cause, autonomic nervous system, inflammation, anesthetic agents to reduce bronchial smooth muscle contraction, and adjunctive agents. The literature suggests that beta-adrenergic agonists, specifically inhaled Beta-2 adrenergic receptor agonists are the first line or primary therapy for bronchospasm and acute airway obstruction (Flood et al., 2015). Beta-receptor agonists can also be administered via inhalation or systemically and induce bronchodilation via cAMP-PKA dependent and non-cAMP processes. Inhaled selective short acting Beta-2 agonists Albuterol, Levalbuterol, Metoproteronol, and Pirbuterol are used for rapid relief of airway obstruction associated with a bronchospasm. Systemic beta-adrenergic are also reported in the use of acute bronchoconstriction if inhalation therapy is not readily available. This includes terbutaline given subcutaneously (SQ) or intravenously (IV), Epinephrine (SQ or IV), and Albuterol (IV). Anticholinergic medications are described in the treatment of bronchoconstriction, however, are generally not used in acute conditions. Bronchodilation occurs by antagonizing M1 and M3 airway receptors and decreasing cGMP and intracellular calcium. They may also be administered via inhalation (i.e. Ipratropium, Tiotropium) and systemically (i.e. intravenous atropine, glycopyrrolate, scopolamine). The literature reports that the clinical use of systemic anticholinergics for bronchodilation is limited due to side effects (i.e. blurred vision, tachycardia, dry mouth) (Flood et al., 2015).

The use of pharmacologic agents that decrease airway inflammation and bronchoconstriction is primarily used in the management of chronic airway diseases such as asthma and chronic obstructive airway disease (Flood et al., 2015). These include corticosteroids, leukotriene modifiers, mast cell stabilizers, and methylxanthines. However, Looseley 2011 describes the use of IV corticosteroids (i.e. hydrocortisone) and methylxanthines (i.e. aminophylline) in the second line treatment of an intraoperative bronchospasm. The mechanism of corticosteroids is in the nucleus of the cell and can induce or suppress gene expression such as the ones responsible for the production of pro-inflammatory mediators. Methylxanthines cause relaxation of smooth muscle relaxation by a couple of mechanisms that include nonselective inhibition of phosphodiesterase, therefore, increasing levels of cAMP and cGMP. Also antagonizing adenosine receptors inhibiting the release of histamine and leukotrienes (Flood et al., 2015).

The literature indicates during the acute phase of a perioperative bronchospasm the goal of treatment is to correct hypoxia and reverse smooth constriction (Looseley, 2011). All volatile inhaled anesthetics with the exception of desflurane cause bronchodilation. The mechanism of bronchodilation is thought to occur by increasing intracellular cAMP and decreasing sensitivity of calcium controlled by protein kinase C, therefore, decreasing intracellular calcium. Propofol, a GABA receptor agonist, is reported to reduce bronchial tone via decreasing cholinergic output to vagal pre-ganglionic neurons as well as direct effects on muscarinic receptors interfering with cellular signaling and calcium mobilization. However, newer formulas that contain a metabisulfite preservative have been noted to induce bronchoconstriction, especially in patients with reactive airway disease (Flood et al., 2015). Local anesthetics are reported to be used to blunt airway reactivity due to tracheal manipulation or intubation, however, doses required to

relax bronchial smooth muscles will likely cause toxicity and are not used in the clinical setting.

Ketamine is an N-Methyl-D-Aspartate receptor (NMDA) antagonist and is reported in the treatment of an intraoperative bronchospasm. It's mechanism for bronchodilation is thought to be due to increasing circulating catecholamine's, inhibition of catecholamine uptake, blockade of voltage-sensitive calcium channels, and inhibiting post-synaptic muscarinic receptors (Flood et al., 2015).

Adjunctive therapy described in the literature is used in conjunction with conventional therapy or when conjunctive therapy fails. Linck (2007) reports a case study in the use of heliox (80%/20% helium-oxygen mixture) in the treatment of an intraoperative bronchospasm. The lower density of this gas mixture in comparison with air-oxygen mixture decreases airway resistance by producing laminar gas flow. The use of IV magnesium sulfate is described as adjunctive therapy for bronchoconstriction. Magnesium sulfate is reported to cause bronchodilation through inhibiting calcium-mediated smooth muscle contraction by decreasing acetylcholine release (Parameswara, 2015).

Project Description

Prior to the implementation of this Capstone Project, the researchers sought SRC and IRB approval from ADU research office. As addressed in the problem statement, a bronchospasm is a potential perioperative anesthetic emergency and must be detected and managed within seconds to avoid disastrous effects. Once the patient is intubated, a bronchospasm can be as subtle as just a change in the ETCO₂ waveform or it can be drastic with a complete loss of ETCO₂ and dangerously low oxygen saturations. The latter can have many different possibilities for the origin making the correct detection nearly impossible for a novice anesthesia provider. The goal of this Capstone Project was to enhance the students' knowledge and equip them with vital information on bronchospasms so that they may readily identify it and be able to treat it in the unlikely event that it should occur.

After performing an extensive comprehensive review of literature on the subject of detection and management of perioperative bronchospasm, a PowerPoint module was created to serve as an educational tool for a sample size of twenty-five SRNA students. Prior to the PowerPoint Presentation, a simulation scenario was held in the Anesthesia OR Simulation Lab followed by a pre-test. Six students volunteered and were assigned randomly to act out various Operating Room Personnel (Surgeon, Surgical Scrub Tech, Circulator Nurse, two Nurse Anesthetists and Anesthesiologist) while the remaining students watched live in the classroom. Each student was given a script to start the scenario. For example, the surgeon impatiently hurried the staff to proceed with a Laparoscopic Cholecystectomy. The student chosen to be the second nurse anesthetist entered the case after the "time-out" to relieve the first nurse anesthetist. The patient was already draped. Prior to incision, the surgeon reported that the patient was moving and was "light". The monitors reflected pre-recorded states that led to the signs,

symptoms, and decompensation associated with a bronchospasm. Based on the clinical situation, the SRNA's were asked to accurately identify and manage the bronchospasm.

Evaluation

The goal of this project was to enhance the 2018 cohort of student registered nurse anesthetist (SRNA) students' knowledge of bronchospasms so that they will have prompt detection and management if they are faced with this event in future practice. During the simulation scenario, the lab model displayed signs and symptoms of an intraoperative bronchospasm. Based on the clinical situation, the SRNA's were asked to accurately identify and manage the bronchospasm. The identification and management of the bronchospasm was graded according to a rubric devised from Overstreet et al. (2015) as referenced earlier in the literature review. The rubric consisted of a grading system based on the number of signs and symptoms identified and the amount of interventions performed to treat the bronchospasm. Next, the students returned to the classroom for a pre-test and PowerPoint presentation on the topic addressing the physiology, recognition and treatment of bronchospasms. Finally, the same six students returned to the simulation lab to complete the scenario for a second time and were graded with the same rubric as before. There was also a post-test given to assess the student's knowledge after the lecture and second scenario was completed.

The measurable outcomes in this project were the students' enhanced intellectual growth and change in behavior as it relates to detection and management of an intraoperative bronchospasm. The researchers of this project graded each pre and post-test and transcribed the results into a Microsoft Excel Spreadsheet. The results were submitted to Dr. Roy Lukman, the Chair of the Scientific Review Committee (SRC). Dr. Lukman conducted a simple t-test for paired samples, which established a correlation between the pre and post-tests. This ultimately served as the tool for the evaluation of the lecture along with percentage values from rubric scores of each simulation scenario.

Results and Conclusion

Two identical tests (Appendix C) and simulation scenario were administered pre and post-PowerPoint presentation. A total of 25 SRNA's were able to participate for testing and 6 SRNA's for the simulation. A t-test for paired samples was conducted to investigate for any significant difference between pre-test and post-test average scores. The obtained t value was -11.290 which is associated with a p value that is less than the .05 level of confidence. Data analysis indicated that statistical significance has been achieved. The mean test scores significantly increased from the pre-test (55.1) to post-test (88.1). Therefore, it can be concluded that there was a significant increase between the pre-test and post-test average scores. Mean scores on the simulation scenario rubric scores similarly improved from pre-presentation (70) to post-presentation (100). Refer to appendix D for complete statistical results.

The statistical analysis demonstrated that there was a significant increase from the pre and post-test evaluations as well as pre and post-simulation performance. Moreover, it can be implied that the method of teaching, which was a PowerPoint presentation was an effective tool in educating the 2018 cohort of SRNA's at Adventist University of Health Sciences on the diagnosis and management of a perioperative bronchospasm. The researchers were able to attain their previously projected objectives of a 20% increase on the post assessment and post-scenario rubric scores. Additionally, we anticipate it will have a lasting influence their future anesthesia practice by guiding their decisions and actions when presented with a perioperative bronchospasm.

Although we far exceeded the anticipated outcome, there were a few limitations with this project. The first scenario rubric results could have been affected because the students had to initially sign the informed consent before proceeding. The title of our capstone project was in the informed consent, therefore the students could have prematurely known that the scenario encompassed treating a bronchospasm. We had also administered the pre-tests before the simulation. Knowing this

information prior to the simulation scenario can lead to quicker identification of the bronchospasm rather than having to actually go through and perform a differential diagnosis.

Another limitation to this project dealt with the actual simulator. Although the simulator in the lab emitted all the signs and symptoms that we planned, there was one big factor missing. Since there was no actual volatile agent in the anesthesia machine, there were no end-tidal reading for sevoflurane. This posed a challenge for CRNA#2 during differential diagnosis because the bronchospasm was due to “light anesthesia.” Although we had scripted in advance for the surgeon to complain about the patient’s movement, not knowing the end-tidal of the volatile agent posed as a slight distraction during the process of differential diagnosis. Perhaps, the researchers should have altered the scenario by creating a scenario using total intravenous anesthesia (TIVA) so that an end-tidal reading was not necessary.

The last limitation in this project involved the drug recognition software in the simulator. The barcode that scanned medications did not scan all the medications properly. Drugs used in the treatment of a bronchospasm such as ketamine and dexamethasone weren’t available in the anesthesia cart. With those limitations, we had to rely on the volunteers to state aloud the drugs they were giving along with the doses. The lab attendant running the simulator modified the simulation based on what he heard. The scenario was recorded and although the lab attendant was in the back viewing room he had to rely on us to reiterate various interventions and medications. This process could have led to numerous errors and could have affected the sequence of the simulation.

Although the researchers attained the goals set for this capstone, a prominent lesson was learned that we would like to share to future students that choose to incorporate a simulation in their capstone. The method of assessing students should either be with simulation or pre/post-tests. For example, the results of the initial simulation will be skewed if you administer the pre-test first and vice-versa if you switch the order. Choosing one form of assessment will minimize unfavorable results.

In conclusion, as verified by the literature review a perioperative bronchospasm is rare anesthetic emergency causing potentially detrimental patient outcomes. The timely diagnosis and management often poses a challenge to both novice and expert practitioners. This is partly because a bronchospasm may have an ambiguous clinical presentation. It can mirror a multitude of other respiratory complications making differential diagnosis challenging. A bronchospasm has multiple etiologies dependent on its occurrence during the perioperative period, varying anesthetic techniques, medications, as well as surgical and patient related factors. Bringing awareness to future nurse anesthetists on this perioperative event will enable the betterment of clinical practice, patient outcomes, and exceed the high standards expected of SRNA's and Nurse Anesthetists.

The results of this project will be presented to students and faculty that attend Capstone Poster Presentations in April 2017. The findings from this project can be further studied by future students and may be used in future Capstone Projects. The researchers are hopeful that future students may want to present more simulation scenarios on adverse events in the operating room.

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004457

Category		Scoring					
		<i>5pts</i>	<i>4 pts</i>	<i>3 pts</i>	<i>2 pts</i>	<i>1 pt</i>	<i>0 pts</i>
Identification of signs and symptoms	Student acknowledges: 1. Increased Peak Pressures 2. Desaturation 3. Audible Wheezes 4. Loss/ Decrease of ETCO ₂ 5. Reduction in Tidal Volumes	Student acknowledges 5/5 of the signs and symptoms.	Student acknowledges 4/5 of the signs and symptoms.	Student acknowledges 3/5 of the signs and symptoms.	Student acknowledges 2/5 of the signs and symptoms.	Student acknowledges 1/5 of the signs and symptoms.	Student fails to acknowledge any of the signs and symptoms.
Management	Student : 1. Administers 100% Oxygen and attempts to hand ventilate 2. Deepens Anesthetic (i.e. Propofol or Seveoflurane) 3. Notifies Surgeon & Anesthesiologist 4. Administers Beta Agonist (i.e.	Student performs 5/5 tasks.	Student performs 4/5 tasks.	Student performs 3/5 tasks.	Student performs 2/5 tasks.	Student performs 1/5 tasks.	Student fails to perform any of the tasks.

	Epinephrine or Albuterol) 5. Administers Steroids or additional medications						
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Appendix A

Appendix B

ADU-NAP CAPSTONE PROJECT INFORMED CONSENT FORM

My name is Jenee Smith and my colleague's name is Joseph Przybylowicz and we are Senior SRNA students in the Nurse Anesthesia Program at Adventist University of Health Sciences. Our Capstone Project is titled, "*Educating the 2018 Cohort of Student Registered Nurse Anesthetists on the Diagnosis and Management of Perioperative Bronchospasm using a Simulation Scenario*" and we are working under the direction of Dr. Manuel Tolosa. We are hopeful that you participate with us on this project to enhance your knowledge and improve your vigilance in the operating room. In order to maintain compliance with the Internal Review board we have to provide you with information so that you can make a decision as to whether or not to participate.

WHAT IS THIS PROJECT ABOUT?

The purpose of this project is to inform SRNA students about perioperative bronchospasms so that they will have prompt detection and management in the clinical field.

WHAT DOES PARTICIPATION IN THIS PROJECT INVOLVE?

If you choose to participate in this project, you will be asked to complete an anonymous pretest, attend a presentation on the subject, participate in classroom discussion on the topic and complete an anonymous posttest assessment. Several volunteer students will be asked to participate in a simulation scenario in the Anesthesia OR Simulation Lab while the other students watch live in the classroom.

WHY ARE YOU BEING ASKED TO PARTICIPATE?

The aim of this project is to sample the class of 2018 cohort of SRNA Students at Adventist University of Health Sciences. Participation in this project is strictly on a volunteer basis. You may decline participation at any time prior to or during this project.

WHAT ARE THE RISKS INVOLVED IN THIS PROJECT?

As with any project, there may be risks involved. But we do not anticipate any risks by participating in this project.

ARE THERE ANY BENEFITS TO PARTICIPATION?

The benefits to participation in this project are expanded knowledge of the subject area and improved intraoperative vigilance skills.

HOW WILL THE INVESTIGATORS PROTECT PARTICIPANTS' CONFIDENTIALITY?

All participants' information will remain confidential throughout this project. Pretest and posttests will be given by numerical assignment to ensure anonymity. There will be no permanent record of any video recordings from the simulation lab. For the simulation scenario, students will be assigned roles and graded by rubric scoring. All numerical results will be submitted to a research statistics expert for data analysis. Results of this project will be published but all identifiable information will remain confidential.

WILL IT COST ANYTHING OR WILL I GET PAID TO PARTICIPATE IN THE PROJECT?

There are no monetary gifts awarded for participation in this project. There also is no cost to participate in this project.

VOLUNTARY CONSENT

By signing this form, you are confirming that you have read this form in its entirety, understand the risks and benefits of participation, and agree to full participation with this project. If you have any further questions, you may contact Jenee Smith at jenee.smith@my.adu.edu or Joseph Przybylowicz at joseph.przybylowicz@my.adu.edu. If you have any additional questions or concerns you may contact the Nurse Anesthesia Program Office at (407) 303-9331.

Participant Signature

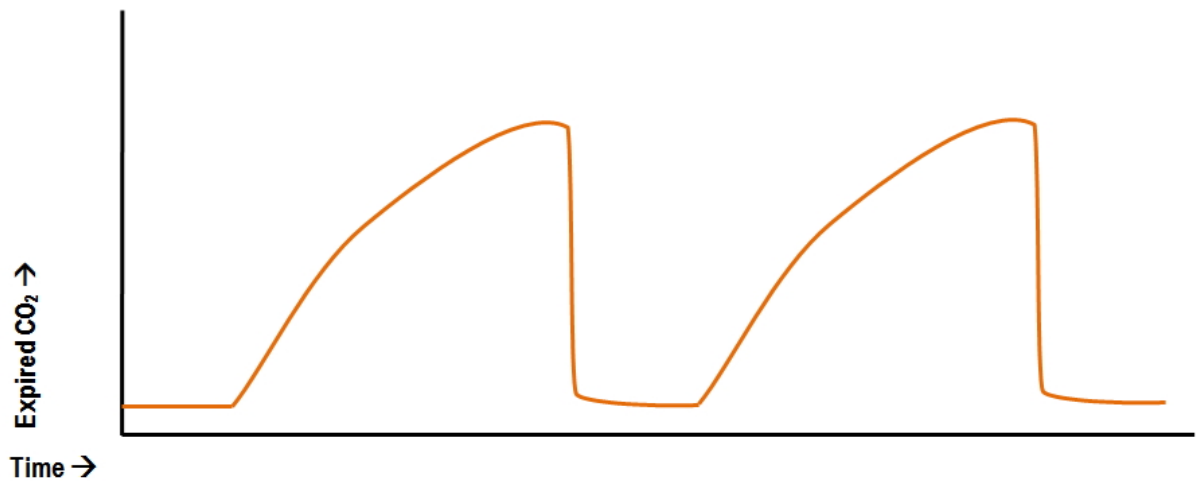
Date

Participant Name (PRINTED LEGIBLY)

Appendix C

Pre-Test/Post-Test

1. All volatile anesthetics reduce bronchomotor tone and produce bronchodilation EXCEPT:
A) Isoflurane
B) Desflurane
C) Sevoflurane
D) Halothane
2. Beta-adrenergic receptor agonists produce bronchial smooth muscle relaxations by what mechanism? (choose 2)
A) cAMP-PKA dependent process
B) Increase in intracellular calcium
C) Decreasing potassium (K) efflux
D) Prevent activation of calcium-calmodulin complex
3. True/False: Conditions such as asthma, upper respiratory tract infections (URI), and chronic obstruction pulmonary disease may increase airway reactivity?
4. Which law describes the process in which airway resistance increases significantly as the airway lumen narrows?
A) Boyle's Law
B) Fick's Law
C) Poiseuille's Law
D) Graham's Law
5. True/False: As the diameter of a lumen narrows, gas flow changes from laminar to turbulent?



6. The capnography waveform shown is most indicative of what event?
 - A) Bronchospasm**
 - B) Normal
 - C) Cardiac Oscillations
 - D) Curare Cleft

7. Which intravenous (IV) anesthetics produce favorable influence on bronchomotor tone?
 - A) Propofol
 - B) Sodium thiopental**
 - C) Ketamine
 - D) A&C**
 - E) All the above

8. Which muscarinic receptors found in the human airway are responsible for bronchoconstriction and mucus production when activated? (Choose 2)
 - A) M1**
 - B) M2
 - C) M3**
 - D) M4

9. All of the following are inhaled adrenergic agonists EXCEPT:
 - A) Albuterol
 - B) Metaproterenol
 - C) Ipratropium**
 - D) Formoterol

10. True/**False**: Acetylcholine binding to muscarinic receptors in the airway causes bronchodilation?

11. Which signs and symptoms of a patient under general anesthesia reflects resistance to airflow leading to a possible diagnosis of bronchospasm?
- A) Wheezing
 - B) Increase in peak airway pressure
 - C) Complete loss of capnography waveform
 - D) Delayed rise in capnography waveform “shark-fin” appearance
 - E) All the above
12. All the following pharmacologic agents may be used in treating a bronchospasm EXCEPT:
- A) Inhaled or Systemic Beta-adrenergic agonists
 - B) Inhaled Anticholinergics
 - C) Methylxanthines
 - D) Intravenous (IV) Magnesium Sulfate
 - E) Heliox
 - F) All the above are correct
13. The onset of rash, peak airway pressures, wheezing, and hypotension during the maintenance of general anesthesia is most commonly caused by which event?
- A) Anaphylaxis or Allergic reaction
 - B) Pulmonary Aspiration
 - C) Pulmonary Edema
 - D) Pneumothorax
14. True/False: Bronchospasm has been reported in anesthesia literature to occur during spinal anesthesia?
15. Immediate treatment of suspected intra-operative bronchospasm to prevent hypoxia and reverse bronchoconstriction includes all of the following interventions EXCEPT:
- A) Notify Surgeon/ stop stimulation
 - B) Ventilate by hand with 100% O₂
 - C) Request & review Chest X-Ray
 - D) Assess patient & ETT patency
 - E) Deepen anesthetic depth

Appendix D

Paired Samples Statistics					
		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Pre-Test	55.1200	25	12.15291	2.43058
	Post-Test	88.1600	25	8.59108	1.71822

Paired Samples Test								
		Paired Differences				t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference			
					Lower Upper			
Pair 1	Pre-Test - Post-Test	-33.04000	14.63295	2.92659	-39.08019 -26.99981	-11.290	24	.000

Simulation Grading Rubric	Mean Rubric Scores
Simulation Pre-Presentation #1	70
Simulation Post-Presentation #2	100