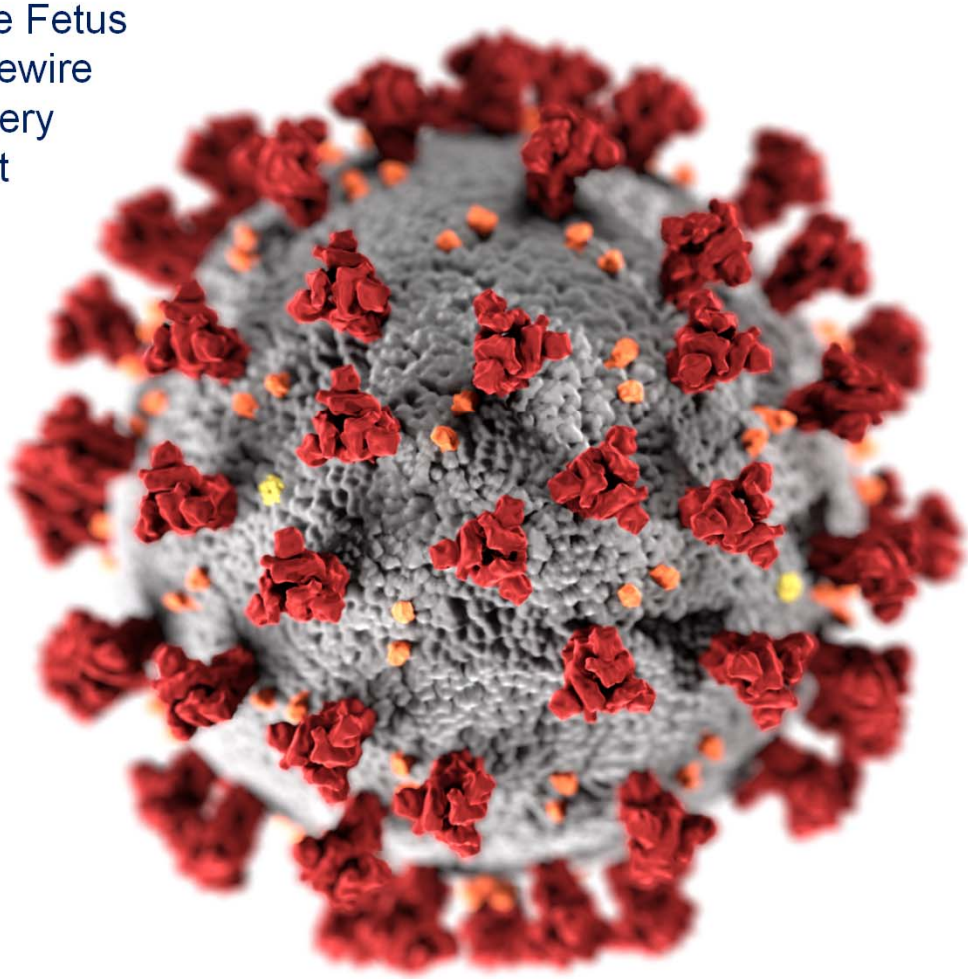
Volume 19 Issue 2 Summer 2020

The International Student Journal of Nurse Anesthesia

TOPICS IN THIS ISSUE



INTERNATIONAL STUDENT JOURNAL OF NURSE ANESTHESIA
Vol. 19 No. 2 SUMMER 2020

Front Cover:
This illustration, created at the Centers for Disease Control and Prevention (CDC), reveals ultrastructural morphology exhibited by coronaviruses. Note the spikes that adorn the outer surface of the virus, which impart the look of a corona surrounding the virion, when viewed electron microscopically. A novel coronavirus, named Severe Acute Respiratory Syndrome coronavirus 2 (SARS-CoV-2), was identified as the cause of an outbreak of respiratory illness first detected in Wuhan, China in 2019. The illness caused by this virus has been named coronavirus disease 2019 (COVID-19).
Photo Credit & Content Providers: Alissa Eckert, MSMI, & Dan Higgins, MAMS
https://phil.cdc.gov/Details.aspx?pid=23312

*The **opinions** contained in this journal are those of the authors and do not necessarily represent the opinions of the program or the University.*

***Disclaimer for all articles authored by military personnel**:*
The views expressed in this journal are those of the authors and do not necessarily reflect the official policy or position of their respective Military Department, Department of Defense, nor the U.S. Government. The work was prepared as part of the official duties of the military service member. Title 17 U.S.C. 105 provides that 'Copyright protection under this title is not available for any work of the United States Government'. Title 17 U.S.C. 101 defines a United States Government work as a work prepared by a military service member or employee of the United States Government as part of that person's official duties.

Publication Information:

The International Student Journal of Nurse Anesthesia (ISSN 2688-5263) is published three times a year in the spring, summer, and fall. Current and past issues, and the Guide for Authors of this free, open access, electronic journal can be found at:

www.aana.com - Member Resources → Students → International Student Journal
https://www.aana.com/studentjournal
https://ifna.site/international-publications/international-student-journal-for-nurse-anesthesia/

For additional information, please contact:

Vicki Callan Coopmans, PhD, CRNA
Webster University Department of Nurse Anesthesia
Browning Hall, ISB 107
8274 Big Bend Blvd.
St. Louis, MO 63119
314-246-5928; Intsjna@aol.com

Table of Contents

COVID-19: Protecting Anesthesia Practitioners During Airway Manipulation

Sarah Jean Kopriva, BSN
Wake Forest School of Medicine

Keywords: COVID-19, intubation, extubation, aerosol, protection

Severe acute respiratory syndrome-corona virus-2 (SARS-CoV-2), the culprit of coronavirus disease 2019 (COVID-19), is extremely contagious. The maximum concentration of SARS-CoV-2 resides within the sputum and secretions of the upper airway.[1-3] Anesthesia practitioners are at an increased risk of exposure during airway management. A lack of a clear consensus persists on how to effectively protect operating room staff when caring for a patient who tests positive for COVID-19. The following is an exploration of various innovations used to mitigate the risk of exposure to those on the frontlines.

Innovation Report

At this time, definitive modes of SARS-CoV-2 transmission include droplet and contact routes. There is an absence of conclusive evidence of airborne transmission; however, the virus can be spread during aerosol generating procedures, such as intubation of the trachea.[2,4] If transmission of COVID-19 were to occur during tracheal intubation or extubation, the severity of illness could be increased due to direct exposure to the high viral load in the sputum.[2] The SARS-CoV-2 pandemic has triggered clinical discussions regarding approaches to maintaining a safe environment for airway management. The Centers for Disease Control recommends that anesthesia practitioners wear personal protective equipment (PPE) consisting of a properly fitted N95 or a powered air purifying respirator (PAPR) during airway manipulation to yield the highest level of protection.[5] Healthcare innovators are adding further layers of protection for the amplified risk period of aerosol generating procedures.

Countries across the globe, including the United States, are implementing extra measures in an effort to minimize risk to anesthesia and hospital staff during heightened periods of exposure. At a large, tertiary care academic medical center, operating room staff leave the room while two anesthesia practitioners perform intubation and extubation of the trachea. If help is deemed necessary during these critical periods, supplementary anesthesia practitioners are called to the room, rather than depending on ancillary staff. Non-anesthesia personnel stay out of the operating room for a minimum of five minutes following intubation and extubation of the trachea, allowing for at least one air exchange.

In addition to PPE, a layer of plastic is laid over the patient during airway management. Experiments conducted by Matava et al. reviewed the efficacy of containing virus aerosolization with this measure.[4] A visual tool evaluating viral spread with ultraviolent light was applied to the oropharynx and mid-trachea of a mannequin. A cough was simulated by connecting a calibrated, medical air gun to the distal trachea. This simulated cough fired over 0.4 seconds, delivering cough peak expiratory flow rates of 150-180 liters per minute outward from the trachea. When a plastic drape was absent, a wide distribution of particles contaminating the surrounding area was demonstrated. A single layer clear plastic drape, applied directly over the head and endotracheal

tube, restricted droplet spread of particles when a cough was simulated during extubation of the trachea. The third trial employed a three drape clear plastic technique with one drape underneath the head of the patient, the second over the chest, and the third layer over the head and endotracheal tube. The three drape plastic approach reduced aerosolization even further. All methods successfully reduced contamination of the area immediately surrounding the patient, potentially providing an added layer of protection to the anesthesia practitioner. Operating room personnel were able to remove the drapes avoiding contamination by carefully rolling the drape toward the feet of the patient.[4]

Other facilities are implementing intubation boxes for use during airway manipulation. These intubation boxes are typically clear acrylic cubes with two circular holes through which the hands of the anesthesia practitioner can enter to perform airway management.[6] This design was originally developed by Dr. Hsien Yung Lai, an anesthesiologist in Taiwan.[7] The box is designed to cover the head of the patient while allowing the anesthesia practitioner clear access to the airway.[6,7] Canelli et al. investigated the efficacy of the intubation box by simulating a patient cough during airway management. Aerosolized droplet spread was represented by a small latex balloon filled with fluorescent dye placed into the hypopharynx of a mannequin. A crude cough was replicated by inflating the balloon with compressed oxygen until it burst. Fluorescent dye representing contamination traveled 2 meters and was found on the gown, gloves, face mask, eye shield, hair, neck and shoes of the anesthesia practitioner. Using the intubation box, the inside of the box, gloves, and forearms of the anesthesia practitioner sustained contamination during a simulated cough. This demonstrated a reduction in particle spread when an intubation box was implemented.[7]

Discussion

Reports of healthcare professionals contracting and resultantly dying of SARS-CoV-2 have been nothing short of tragic. Aerosol generating procedures, including intubation and extubation of the trachea, expose the anesthesia practitioner to additional risk.[2] Innovations used to supplement PPE, such as a plastic layer or intubation box, reduces the risk imposed on anesthesia practitioners during airway manipulation.

An intubation box is innovative, but has limitations. An increase in body habitus of the patient may not prove conducive to the utilization of an intubation box. The dimensions of the circular arm holes are likely not suitable for every anesthesia practitioner and may limit hand movement during airway management. Anesthesia practitioners will have to be trained in using the box and must remain open to abandoning it if intubation proves difficult.[6,7] Other identified barriers to the box include time for production and cost. The benefit of an intubation box is that it can be cleaned with 70% alcohol and safely used in subsequent cases.[6] Hospitals may want to consider ordering intubation boxes in a variety of dimensions to best fit all patients and practitioners. The use of a plastic layer or several plastic layers seems to provide a more practical and less expensive protective measure. Plastic sheeting limits aerosolization of droplet spray during airway manipulation.[4] A plastic layer allows for free movement of the hands of the anesthesia practitioner and is not limited by the body habitus of a patient. Additionally, specified dimensions are unnecessary and hospitals avoid production time. One perceived drawback is that

the plastic layer is a single use item; however, this approach is inexpensive, so this does not represent an appreciable hindrance to its usage.[4]

Aerosol generating procedures, such as intubation and extubation of the trachea, pose significant risk to the anesthesia practitioner performing airway manipulation in a COVID-19 patient.[1,2] The current recommendation is that the anesthesia practitioner don an appropriately fitted N95 or PAPR when airborne particles are generated.[5] However, many institutions have applied innovative practices, such as the application of a clear plastic drape or intubation box, over the patient. These efforts have been employed to provide an enhanced layer of protection to vulnerable practitioners who are serving others during these uncertain times.

References

1. Wang W, Xu Y, Gao R, et al. Detection of SARS-CoV-2 in different types of clinical specimens. *JAMA*. 2020;3. doi:10.1001/jama.2020.3786.
2. Cook T, El-Boghdadly K, McGuire B, McNarry A, Patel A, Higgs A. Consensus guidelines for managing the airway in patients with COVID-19: guidelines from the Difficult Airway Society, the Association of Anaesthetists the Intensive Care Society, the Faculty of Intensive Care Medicine and the Royal College of Anaesthetists. *Anaesthesia*. 2020;3. doi:10.1111/anae.15054.
3. Wu Z, McGoogan J. Characteristics of and important lessons from the Coronavirus disease 2019 (COVID-19) outbreak in China: summary of a report of 72314 cases from the Chinese Center for Disease Control and Prevention. *JAMA.* 2020;323(13):1239-1242. doi:10.1001/jama.2020.2648.
4. Matava C, Yu J, Denning S. Clear plastic drapes may be effective at limiting aerosolization and droplet spray during extubation: implications for COVID-19. *Can J Anaesth*. 2020;3. doi.org/10.1007/s12630-020-01649-w.
5. Centers for Disease Control and Prevention. Interim infection prevention and control recommendations for patients with suspected or confirmed Coronavirus disease 2019 (COVID-19) in healthcare settings. CDC. https://www.cdc.gov/coronavirus/2019-ncov/hcp/infection-control-recommendations.html. Updated April 13, 2020. Accessed April 15, 2020.
6. Canelli R, Connor C, Gonzalez M, Nozari A, Ortega R. Barrier enclosure during endotracheal intubation. *N Engl J Med*. 2020;4. doi: 10.1056/NEJMc2007589.
7. Girgis A, Aziz M, Gopesh T, et al. Novel Coronavirus disease 2019 (COVID-19) aerosolization box: design modifications for patient safety. *J Cardiothorac Vasc Anesth.* 2020. doi: 10.1053/j.jvca.2020.05.001.

Mentor: Kate Saftner, MSN, CRNA

Novel Technique for Nasotracheal Intubation during the COVID-19 Pandemic

Stephen Williams, BSN
Wake Forest School of Medicine

Key Words: pediatric, nasotracheal intubation, videolaryngoscopy, COVID-19, SARS-CoV-2

The virus SARS-CoV-2 and the associated disease known as COVID-19 has been declared a global pandemic by the World Health Organization (WHO). According to the WHO, there are over 24 million confirmed cases of COVID-19 as of September 6th, 2020.[1] Transmission occurs through aerosolization of large particles (droplets) in the air that eventually come into contact and contaminate a person or surface.[2] Healthcare workers are at an increased risk of contracting SARS-CoV-2 while caring for infected patients, especially during aerosolizing procedures such as endotracheal intubation and mask ventilation.[2] Additionally, this virus is known to have a long prodromal phase of up to 5 days during which the virus can be transmitted in the absence of symptoms.[3]

The American Association of Nurse Anesthetists (AANA) and the Anesthesia Patient Safety Foundation (APSF) have released recommendations on management of the patient with COVID-19. These recommendations are useful for the adult patient undergoing oral intubation for general anesthesia. However, there is currently no evidence or recommendations for pediatric patients undergoing inhalation induction and nasotracheal intubation. This case study discusses a technique for pediatric nasotracheal intubation while utilizing current AANA recommendations to prevent the spread of SARS-CoV-2.

Case Report

A 4-year-old 15 kg male arrived for full mouth dental rehabilitation due to severe dental decay and multiple abscessed teeth. Past medical history was significant for well controlled mild-intermittent asthma. No past surgical history or drug allergies were noted. Nasotracheal intubation was necessary for surgical exposure.

The patient was taken to the operating room and standard monitors were applied. Appropriate PPE was donned by anesthesia staff including N95 respirator masks, face shields, and double gloves. Additionally, a high efficiency hydrophobic filter was placed between the mask and the anesthesia circuit Y-piece. An inhalation induction was performed with sevoflurane 8% inspired concentration, N_2O 7 L/min and O_2 3 L/min. A clear surgical drape was placed over an ether screen creating a clear barrier between the patient and the anesthesia provider. A 22-gauge peripheral intravenous (IV) catheter was inserted into the right hand, and the patient received fentanyl 15 mcg and a propofol 40 mg bolus followed by a propofol infusion at 250 mcg/kg/min.

Figure 1: Clear Plastic Surgical Drape and Ether Screen Protective Barrier

While the patient remained under the clear surgical drape and ether screen tent, phenylephrine nasal spray was applied to each nare and a 4.0 Nasal RAE endotracheal tube (ETT) was advanced through the right nare until the tube passed through the nasopharynx and entered the laryngopharynx. A videolaryngoscope with a Mac 2 blade was inserted into the mouth and the ETT was noted to be just posterior the corniculate cartilage in the pyriform sinus. Downward pressure to the cricoid cartilage was initiated and the ETT pilot balloon was inflated, allowing the ETT to rise up into the glottic aperture. The ETT balloon was deflated and the ETT was easily passed through the vocal cords, then the balloon was re-inflated. Continuous $ETCO_2$ was noted and general anesthesia was maintained with a propofol infusion for total intravenous anesthesia (TIVA). The surgical procedure was uneventful and the patient received acetaminophen 324 mg per rectum, dexamethasone 4 mg IV, and ketorolac 9 mg IV.

During emergence, the clear surgical drape and ether screen created a barrier between the patient's head and the anesthesia provider. The patient was noted to be spontaneously ventilating with adequate tidal volumes, $ETCO_2$, and pulse oximetry. The ETT cuff was taken down with the ETT in place to assess response to laryngeal stimulation. Patient was noted to maintain regular respirations, the ETT was removed in a deep plane of anesthesia to minimize coughing, and the patient transported to the post anesthesia recovery unit (PACU). The patient was discharged home shortly afterward with no complications.

Discussion

Pediatric nasotracheal intubation during the COVID-19 pandemic creates a unique challenge to the anesthesia provider. During this case, we focused on three main recommendations set forth by the AANA to reduce the spread of SARS-CoV-2; utilizing proper PPE, placing a high efficiency hydrophobic filter between the mask and anesthesia circuit, and taking steps to minimize aerosolization of particles.[4]

The AANA recommends using full PPE during aerosolizing procedures. Although SARS-CoV-2 is predominantly spread by contact or droplet transmission of relatively large particles which travel on average 1 meter from the patient, these particles can become aerosolized during certain medical procedures such as intubation.[5] The aerosolization of particles is thought to occur when air accelerates across a fluid surface, and these particles stay in the air longer and can travel much further than larger droplets.[5] Therefore, it is recommended that all staff in the operating room utilize airborne precautions if present for intubation.[5]

Different types of masks carry various levels of protection for different precautions (droplet vs airborne). A standard surgical mask is estimated to provide droplet and contact protection within 1-2 meters from patient.[5] The N95 designation is obtained when the mask is tested and demonstrates blocking at least 95% of solid and liquid aerosol test particles.[5] Due to the highly contagious nature of this disease and frequent exposure of operating room staff to aerosolizing procedures, airborne precaution PPE is a necessary safety measure.

A high efficiency hydrophobic filter was placed between the patient mask and the anesthesia circuit and remained at the end of the ETT after successful intubation. The hydrophobic filter prevents moisture and contaminants from entering the anesthesia breathing circuit. This is essential during the COVID-19 pandemic due to viral colonization in the mucosa of the upper airway and respiratory tract.[3] Positive pressure mechanical ventilation creates a rapid airflow that has the potential to aerosolize the virus, contaminating the breathing circuit and operating room. A study by Rees, et. al. determined that on average contamination can be detected using bioluminescence on the machine side of a breathing circuit 8% of the time when hydrophobic filters are used.[6] However, the use of these filters is still recommended by the AANA to prevent environmental contamination and should be utilized during this pandemic.

During this case two main methods were instituted to reduce the aerosolization of particles during intubation: utilizing a clear surgical drape and an ether screen to create a barrier between the patient and anesthesia provider (Figure 1), and avoiding the use of McGill forceps during nasotracheal intubation.

An obvious method for reducing transmission of aerosolized particles is to establish a barrier between the patient and anesthesia provider that is transparent to allow visualization of airway structures. Canelli, et. al. describes the use of an “aerosol box” that serves as a protective barrier during intubation[7] that is similar to the clear surgical drape and ether screen described in this case report. It was determined that contamination of both the anesthesia provider and surrounding environment up to 2 meters was confirmed without the “aerosol box”, but contamination was contained when the “aerosol box” was used.[7] This suggests that the use of a transparent protective barrier between the patient and anesthesia provider would result in decreased spread of aerosolized particles in the operating room.

Another method for reducing aerosolization of the virus is to reduce the amount of instruments that contact the upper airway mucosa during intubation. This presents a challenge during nasotracheal intubation, as McGill forceps are often needed to advance the ETT tube from the hypopharynx into the glottic aperture. Utilizing McGill forceps creates another potential vector for the spread of viral particles. During this case report, videolaryngoscopy was performed once the ETT was advanced through the nasal turbinates and utilization of external cricoid pressure and pilot balloon inflation were utilized to move the ETT tip anteriorly out of the pyriform fossa and into the glottic aperture. The balloon was then deflated and the ETT easily advanced through the vocal cords. This method for nasotracheal intubation has been described by Goodine, et al. as the “alignment approach”.[8] The use of the alignment approach to nasotracheal intubation has the advantage of avoiding the use of McGill forceps, which has been associated with the rupture of the ETT balloon.[8] During the COVID-19 pandemic it is of great importance to limit potential

exposure to upper airway secretions and having one less instrument coming into contact with upper airway mucosa may reduce the environmental spread of the virus.

Through this case report we demonstrate a novel technique for pediatric nasotracheal intubation while adhering to the AANA recommendations for the COVID-19 patient. Emphasis was given to the utilization of proper PPE, using hydrophobic filters and taking measures to minimize aerosolization and thus environmental contamination with particles potentially containing SARS-CoV-2. More research is needed to determine the efficacy of these techniques in regards to preventing transmission of aerosolized SARS-CoV-2 particles and clinical guidelines must be developed to address the proper care for pediatric patients undergoing nasotracheal intubation during this pandemic for patient and provider safety.

References

1. Word Health Organization, 2020. *Coronavirus Disease (COVID-19) Weekly Epidemiological Update*. [online] Word Health Organization, p.1. Available at: <https://www.who.int/docs/default-source/coronaviruse/situation-reports/20200907-weekly-epi-update-4.pdf?sfvrsn=f5f607ee_2> [Accessed 8 September 2020].
2. Cook TM, El-Boghdadly K, McGuire B, McNarry AF, Patel A, Higgs A. Consensus guidelines for managing the airway in patients with COVID-19: Guidelines from the Difficult Airway Society, the Association of Anaesthetists the Intensive Care Society, the Faculty of Intensive Care Medicine and the Royal College of Anaesthetists. *Anaesthesia*. 2020;75(6):785-799. doi:10.1111/anae.15054
3. Ludwig S, Zarbock A. Coronaviruses and SARS-CoV-2: A Brief Overview. *Anesth Analg*. 2020;131(1):93-96. doi:10.1213/ANE.0000000000004845
4. Aana.com. 2020. *COVID-19 Faqs*. [online] Available at: <https://www.aana.com/aana-covid-19-resources/covid-19-faqs> [Accessed 8 September 2020].
5. Cook TM. Personal protective equipment during the coronavirus disease (COVID) 2019 pandemic - a narrative review. *Anaesthesia*. 2020;75(7):920-927. doi:10.1111/anae.15071
6. Rees LM, Sheraton TE, Modestini C, Wilkes AR, Hall JE. Assessing the efficacy of HME filters at preventing contamination of breathing systems. *Anaesthesia*. 2007;62(1):67-71. doi:10.1111/j.1365-2044.2006.04867.x
7. Canelli R, Connor CW, Gonzalez M, Nozari A, Ortega R. Barrier Enclosure during Endotracheal Intubation. *N Engl J Med*. 2020;382(20):1957-1958. doi:10.1056/NEJMc2007589
8. Goodine C, Sparrow K, Asselin M, Hung D, Hung O. The alignment approach to nasotracheal intubation. *Can J Anaesth*. 2016;63(8):991-992. doi:10.1007/s12630-016-0641-y

Mentor: Ken Thompson, DNP, CRNA

Artificial Intelligence to Determine Treatment of COVID-19

Katherine R. Bliss, BSN
Keiser University

Keywords: COVID-19, artificial intelligence, REMAP, treatment

When trying to determine a treatment plan for a pandemic, such as coronavirus disease 2019 (COVID-19), time is a limiting factor. The standard randomized control trial requires substantial time to yield adequate results. Artificial intelligence (AI) is being used more frequently in healthcare as a means to increase productivity, as well as more positive patient outcomes. Artificial intelligence systems synthesize treatment methods based on algorithms, which spares the clinician time and error in the decision-making process.[1] It is possible that AI can help determine an effective treatment plan for COVID-19.

Discussion

Developing a Treatment Plan

Ogbagaber et al. published an article in 2017 on the statistical data of the use of adaptive treatment strategies for treating complex, multi-stage diseases, such as AIDS, depression, or cancer.[2] Sequential multiple assignment randomized designs assess and compare the adaptive treatment strategies. This design suggests a beneficial method for determining multifaceted treatments for complex diseases. COVID-19 is different than AIDS, depression, or cancer; however, the strategies used to determine the treatment plans for such conditions may also provide beneficial insight on how to proceed with determining a treatment plan for COVID-19.[2]

Coronavirus Pathology

The pathology of COVID-19 is complex. The virus triggers leukocytes to attract immune mediators such as interleukin-6 (IL-6), which are pro-inflammatory and capable of creating an immense immune reaction. The virus is transmitted by respiratory droplets and direct contact.[3] While some infected individuals are asymptomatic, others are in critical condition. Common symptoms of COVID-19 include fever, loss of smell and taste, cough, and fatigue.[3] Less common symptoms include nausea, diarrhea, headaches, sore throat, and rhinorrhea. Patients diagnosed with COVID-19 who require critical care often have comorbidities, such as chronic obstructive pulmonary disease, asthma, diabetes, hypertension, and obesity.[3] In the more severe cases, patients require ventilatory support in the intensive care unit due to severe respiratory failure, and multiple organ dysfunction syndromes.[3] As of September 9, 2020, there have been 27,605,560 confirmed cases of COVID-19 worldwide, and 6,328,099 in the United States. Currently, the total death rate is 898,284 worldwide. [4]

Therapies

There are several therapies being implemented to treat COVID-19, but currently there is not a clear cut treatment plan for this virus. An initial treatment implemented was anti-malarial drugs, such as hydroxychloroquine, in combination with antibiotics, such as azithromycin.[5] Some providers have prescribed immuno-modulating therapies, including corticosteroids, to regulate the immune system while combating the virus. In some cases, symptom management has been

the focus. Some current studies focus on the use of convalescent plasma, which filters antibodies from recovered COVID-19 patients to transfuse into currently infected patients.[6]

The incubation period of COVID-19 is up to 14 days. People are initially unaware they have the virus, and expose many others throughout the incubation period. This leads to a significant number of people being unknowingly contagious. Lack of time to adequately perform randomized control trials to determine the most efficient treatment plan is a major rate-limiting factor in determining optimal management of the disease.

Randomized, Embedded Multifactorial Adaptive Platform (REMAP)
The University of Pittsburgh Medical Center (UPMC) is implementing an innovative design using AI to rapidly assess which medical management plans are working most efficiently and safely to treat patients positive for COVID-19. Dr. Derek Angus and Dr. Donald Yealy conducted a conference on April 9, 2020, and discussed how UPMC is making strides to determine an effective treatment for COVID-19 patients. The plan is to use REMAP to test multiple new therapies, as well as therapies currently in use, in a short amount of time. This platform works using reinforcement learning. It identifies the therapies with higher success and can assign those therapies more frequently.[7] UPMC uses an electronic medical record, which provides a subject pool for the trials. With Institutional Review Board approval, UPMC will recruit patients upon admission; those who consent to participate in the trial are immediately assigned a treatment therapy. As the electronic medical record is updated in real-time, information, such as the patient's clinical status, appears in the study, and efficacy of the treatment methods are evaluated.

The therapies used as treatment or management of COVID-19 are assigned to patients with severe COVID-19. The inclusion criteria are adult patients admitted to an ICU for severe COVID-19 associated pneumonia within 48 hours, and receiving ventilatory support and/or vasopressors. Exclusion criteria include healthcare-associated pneumonia, and imminent death in 24 hours.[7] Some of the treatment combinations assigned include hydroxychloroquine, steroids, and immunomodulators. New drugs can be rolled into the platform as needed. With more data in real-time, the REMAP uses an algorithm to assess which therapies are providing the most beneficial outcomes, and those therapies are assigned more frequently to COVID-19 positive patients.[7] This results in more patients receiving the most beneficial treatment and therapy without the length of time it would require to complete traditional randomized controlled trials.

Conclusion

Social-distancing, allowing six feet between others at all times, and quarantine, staying out of contact with other people for an extended amount of time, have been recommended by the Center for Disease Control (CDC) and enforced to decrease the rate of transmission of the COVID-19. These interventions prevent overwhelming hospitals and medical staff with a large volume of admissions.[7] As businesses re-open their doors to the public, and people increase their social interactions, COVID-19 rates will likely increase.[7] COVID-19 is not going to disappear, but until a vaccine is available, effective treatment plans are essential. UPMC is making strides to treat and manage COVID-19 effectively. Traditional randomized control trials require more time than using REMAP to determine an effective treatment plan. AI and REMAPs will

drastically aid in the management and treatment of those patients positive for COVID-19. A progressive outlook on the current devastating circumstance, demonstrates that science is launching forward with many innovative designs.

References

1. Habli I, Lawton T, Porter Z. Artificial intelligence in health care: accountability and safety. *Bull World Health Organ*. 2020; 98:251-256. http://dx.doi.org/10.2471/BLT.19.237487
2. Ogbagaber S B, Karp J, Wahed A S. Design of sequentially randomized trials for testing adaptive treatment strategies. *Stat Med*. 2016; 35:840-858. Wileyonlinelibrary.com DOI: 10.1002/sim.6747
3. Gennaro F D, Pizzol D, Marotta C, et al. Coronavirus diseases (COVID-19) current status and future perspectives: a narrative review. *Int J Environ Res Public Health*. 2020; 17(8): 2960. Https://doi.org/10.3390/ijerph17082690
4. Johns Hopkins University & Medicine, Coronavirus COVID-19 global cases by the Center for Systems Science and Engineering (CSSE). *Coronavirus Resource Center*. Https://coronavirus.jhu.edu/map.html. Published 2020. Updated September 9, 2020. Accessed September 9, 2020.
5. Reardon S. Antibiotic treatment for COVID-19 complications could fuel resistant bacteria. *Science Magazine*. 2020. https://www.sciencemag.org/news/2020/04/antibiotic-treatment-covid-19-complications-could-fuel-resistant-bacteria doi:10.1126/science.abc2995
6. Sparks D, Infographic: Convalescent plasma. *MayoClinic*. 2020 April 12. https://newsnetwork.mayoclinic.org/discussion/infographic-3/
7. Angus D, Yealy D. *UPMC and University of Pittsburgh COVID-19 press briefing* [Conference]. Pittsburgh, PA. 2020.

Mentor: Leonardo Campero, DNAP, CRNA

COVID-19 Pandemic N95 Decontamination for Reuse

Rose Marthe Esther Buteau, MSN
Keiser University

Keywords: N95 filtering facepiece respirator, COVID-19, hydrogen peroxide (VHP) decontamination

The coronavirus disease 2019 (COVID-19) is a highly contagious respiratory illness that is affecting individuals worldwide. The disease requires healthcare personnel to have specific types of personal protective equipment (PPE) such as respirator masks, gloves, and gowns as a standard of care to prevent contamination. However, because of the high volume of PPE needed worldwide, manufacturers are unable to keep up with global demand. The shortage poses a risk for healthcare occupational exposure to COVID-19. Resolving this issue will reduce the exposure risk of healthcare workers but also enable them to respond effectively to the pandemic.

Discussion

Recommendation for Extended and Limited Reuse

Due to the pandemic, healthcare facilities are experiencing a higher than average influx of patients, which results in a shortage of PPE. The deficiency of PPE specifically affects the single-use N95 filtering facepiece respirator mask, which is essential when caring for any airborne respiratory disease. This issue triggered a call to action by the government and many public health organizations. In response, the National Institute for Occupational Safety and Health (NIOSH), along with the Centers for Disease Control and Prevention (CDC), offered many strategies to optimize N95 respirators. These crisis strategies propose the reuse of the N95 respirators beyond the manufacture's recommended shelf life and the extended use or limited reuse of N95 respirators between patients with similar COVID-19 diagnosis.[1, 2]

The term *extended reuse* refers to the use of one N95 respirator by the same healthcare provider during encounters with different patients without replacing the mask in between patients. The practice of *limited reuse* involves the re-usage of the respirator in between patient encounters of the same diseases with a restriction on the number of times it is reused.[1] The CDC and the NIOSH had initially suggested these practices during previous outbreaks of respiratory diseases such as the influenza (H1N1) outbreak in 2009 and the severe scute respiratory syndrome (SARS) in 2004.[2] While the CDC recommended the extended use and limited reuse as a strategy, they also provide considerations and recommendations that healthcare providers should be mindful of during the re-usage of N95 respirators. Part of the consideration involved the continuous reassessment of the integrity of the N95 respirator, and the safe discard of the mask if it is visibly soiled.[1]

Manufacturers of the N95 respirator do not provide a safe method for decontamination of the device. As a result, the CDC stresses that the recommendation for extended use and reuse is strictly event-specific and pathogen-explicit, and should not become the standard of care.[1] The primary hazard associated with reusing the single-use N95 respirator is contact transmission and thus the potential for cross-contamination.[1] Only the manufacturers of face masks can provide guidance on how to decontaminate their specific products.[1] Decontamination methods have been investigated by NIOSH's National Personal Protective Technology Laboratory and other researchers. Their research is summarized below.

Suggested Types of Decontamination

The CDC states that a decontamination system is effective if it reduces the pathogenic particles without compromising the integrity of the respirator and does not introduce hazardous chemical residue into the respirator.[1] The types of decontamination systems recommended by the CDC and the NIOSH for potential means of sterilization of the N95 respirator include vaporous hydrogen peroxide (VHP), ultraviolet germicidal irradiation (UVGI), and moist heat.[1] These stated techniques are the only evaluated methods that might provide an optimal outcome of sterilization measures of the N95 respirator for reuse.

Ultraviolet germicidal irradiation

Studies of UVGI demonstrate a statistical reduction of tested virus particles on the N95 respirator.[4-6] While the UVGI method provides promising results, the multiple layers of the N95

respirator make this method unlikely to destroy all surrogate viruses on the mask, depending on the type of ultraviolet germicidal irradiation lamp used. Therefore, treatment must be adjusted depending on the type of ultraviolet lamp used.[1] Furthermore, UVGI also demonstrated some degree of degradation of the respirator structural material. Lindsey et al's investigation on the effect of UVGI on the N95 respirator structural integrity and performance showed a reduction in the strength of the structural integrity of the respirator, however little to no impact on the flow resistance and performance of the respirator was noted.[4] Viscusi et al. also evaluated the impact that different decontamination methods had on the N95 respirator.[5] The study suggested that UVGI has little to no effect on post decontamination fitting of the respirator. [5]

Moist Heat Method

The moist heat method also showed satisfactory results by inactivating viral pathogens of influenza A (H5N1) on N95 respirators in a study done by Lore and colleagues.[6] Per the CDC, moist heat showed very little degradation on the performance, and minimally affected the integrity of filtration on the respirator. However, the CDC also points out concerns that arise due to the limited research evaluating the effect of moist heat method on decontamination with different types of biological pathogens.[1]

Vaporous hydrogen peroxide method

The result of the VHP method showed minimal effect on structural material, performance, and fitting of the N95 respirator from a pilot study project conducted by the Battelle Memorial Institute.[1,8] The comprehensive pilot study evaluated the efficacy of the "Bioquell Clarus C" device for the decontamination of N95 respirators for reuse. The study demonstrated successful decontamination of N95 respirators up to 20 treatments per mask, without affecting the integrity of the N95 respirator, with total eradiation of bacteriophages and viruses, including the coronavirus (SARS-CoV-2).[1,8,9] However, the study reported that after 20 treatments the N95 respirator showed some strap degradation.[1,8,9] The report confirmed that the device can sterilize tens of thousands of N95 respirators with each cycle using vapor phase hydrogen peroxide concentrates. [8]. The system works through the exposure of the N95 respirator's biological contaminants at a calculated concentration level of vapor hydrogen peroxide over a specific length of time.[8] Other studies reported using the vaporous hydrogen peroxide method using different models and types of vaporous hydrogen peroxide generating devices; however, the results presented with adverse effects on the filtration performance or airflow resistance of the N95 respirator. Furthermore, these investigational studies also presented with structural degradation of the respirator after just a few treatment cycles.[1]

Emergency approval Decontamination system

The successful result of the Batelle Memorial Institute's pilot study on the sterilization of the single-use N95 respirator using the "Bioquell Clarus C" became an asset amid the shortage of PPE brought on by COVID-19. The success of the pilot study created an effective and efficient means of the decontamination of the N95 respirator. The US Food and Drug Administration (FDA), on March 29, 2020, issued an emergency use authorization for the decontamination system by Battelle Memorial Institute.[1,7] The FDA's approval of the critical care decontamination system (CCDS) of the Battelle Memorial Institute addressed the shortage of N95 respirator within the United States, for the removal of the SARS-CoV-2 on the respirator.[7-9] The implementation of the authorization is an emergency strategy to help optimize supplies of

the N95 respirator in response to the shortage due to the COVID19 pandemic. Therefore, it will help tackle the concerns of cross-contamination during the re-usage of the N95 respirator by healthcare workers. Also, it will help decrease the risk of spreading the disease in healthcare settings and reduce the risk of occupational hazards exposure to healthcare workers. Furthermore, this type of sterilization process will facilitate recycling implementation at the facility level of the single-use N95 respirator for decontamination within the United States.

Conclusion

Due to the global shortage of PPE created by the COVID-19 pandemic, the CDC and the NIOSH suggested extended use and limited reuse recommendations for the N95 respirators masks as a strategy to maintain supply.[1] The idea of reusing the N95 respirator raised practical concerns of cross-contamination and possible occupational hazard exposure of the disease within healthcare facilities. Many investigators evaluated various types of decontamination methods such as moist heat, UVGI, and VHP.[1] Several of the investigated decontamination methods showed promising results, while others generated concerns with structural degradation, decreased performance, and issues with filter flow resistance.[1,4-6] However, the study conducted by the Battelle Memorial Institute on the VHP decontamination method using the "Bioquell Clarus C" device showed effective destruction of various bacteriophages and viruses, including SARS-CoV-2 and did not affect the filter performance of the N95 respirator up to 20 treatments.[1,8-11] Therefore, the FDA issued the first approval on a decontamination system for the N95 respirator in an effort to eliminate concerns and health risks associated with the re-usage of the N95 respirator. This may assist with the PPE shortage created by the coronavirus respiratory disease faced by the United States and the world.

References

1. Centers for Disease Control and Prevention (CDC). Decontamination and reuse of filtering facepiece respirators. https://www.cdc.gov/coronavirus/2019-ncov/hcp/ppe-strategy/decontamination-reuse-respirators.html. Published March 2020. Accessed April 9, 2020.
2. Fisher EM, Shaffer RE. Considerations for recommending extended use and limited reuse of filtering facepiece respirators in health care settings. *J Occup Environ Hyg.* 2014;11(8):D115-D128. doi:10.1080/15459624.2014.902954
3. National Institute for Occupational Safety and Health. Recommended guidance for extended use and limited reuse of N95 filtering facepiece respirators in healthcare settings. https://www.cdc.gov/niosh/topics/hcwcontrols/recommendedguidanceextuse.html. Published March 27, 2020. Accessed April 9, 2020
4. Lindsley WG, Martin SB, Thewlis RE, et al. Effects of ultraviolet germicidal irradiation (UVGI) on N95 respirator filtration performance and structural integrity. *J Occup Environ Hyg*. 2015;12(8):509-517. doi:10.1080/15459624.2015.1018518.
5. Viscusi DJ, Bergman MS, Novak DA, et al. Impact of three biological decontamination methods on filtering facepiece respirator fit, odor, comfort, and donning ease. *J OccupEnviron Hyg*. 2011;8(7):426-436. doi:10.1080/15459624.2011.585927.

6. Lore MB, Heimbuch BK, Brown TL, Wander JD, Hinrichs SH. Effectiveness of Three Decontamination Treatments against influenza virus applied to filtering facepiece respirators. *Ann Occup Hyg*. 2012;56(1):92-101. doi:10.1093/annhyg/mer054.
7. Food and Drug Administration. Investigating decontamination and reuse of respirators in public health emergencies. https://www.fda.gov/emergency-preparedness-and-response/mcm-regulatory-science/investigating-decontamination-and-reuse-respirators-public-health-emergencies. Published April 10, 2020. Accessed April 10, 2020.
8. Food and Drug Administration: Final report for the bioquell hydrogen peroxide vapor (HPV) decontamination for reuse of N95 respirators. https://www.fda.gov/media/136386/download. Published April 10, 2020. Accessed April 13, 2020.
9. Battelle Memorial Institute. COVID-19: Deploying a critical new ppe decontamination system. https://inside.battelle.org/blog-details/covid-19-deploying-a-critical-new-ppe-decontamination-system. Published March 28, 2020. Accessed April 10, 2020.
10. Bergman MS, Zhuang Z, Hanson D, et al. Development of an Advanced Respirator Fit-Test Headform. *J Occup Environ Hyg*. 2014. 11(2): p. 117-125. doi:10.1080/15459624.2013.816434.
11. Kenney, Patrick, et al. Hydrogen peroxide vapor sterilization of N95 respirators for reuse. *medRxiv*. 2020.03.24.20041087. doi: https://doi.org/10.1101/2020.03.24.20041087

Mentor: Leonardo Campero, DNAP, CRNA

New Cloth Mask Technology during COVID-19

Porsha Neal, BSN
Keiser University

Keywords: Coronavirus, COVID-19, N95, cloth masks, Coronavirus transmission, safety, healthcare professionals.

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is the virus responsible for Coronavirus Disease 2019 (COVID-19). The COVID-19 pandemic has exposed weaknesses in healthcare systems around the world. Many needed medical devices such as ventilator machines, personal protective equipment (PPE) such as airborne filter masks, and protective gowns are not processed and shipped fast enough to keep up with the demand. Many recommendations have been established to buffer the shortage of PPE to try to prevent frontline workers from being infected. Further, concerns of the general population and frontline healthcare professionals continue to rise regarding recommendations on how to decrease spread of COVID-19. As the United States (US) began to feel the impact of the COVID-19 pandemic, health organizations began to create guidelines to prepare for the worsening crisis. With an influx of COVID-19 positive patients into healthcare facilities, N95 respirator supplies became even more scarce. In March 2020, the Cable News Network (CNN) reported that the Centers for Disease Control and Prevention (CDC) suggested “as a last resort . . . health care providers could consider using "homemade masks" -- such as bandanas or scarves -- to care for coronavirus patients, ideally in combination with a face shield.”[1] Current CDC guidelines do not include this language, but it is important to consider and explore new technologies in facemask protection.

Transmission of Coronavirus (COVID-19)

The COVID-19 pandemic began in Wuhan, China in 2019. Chinese officials and medical specialists reported that virus transmission occurred via droplets. Due to this pertinent information, the US official health and disease organizations informed the public, as well as the healthcare professionals, of the severity and health risks of COVID-19. As of March 2020, the WHO published that the transmission of COVID-19 has several modes depending upon the circumstances and condition of the infected patient.[2]

Transmissions of pulmonary infections are classified based on particle size released by an infected person. Particles 5-10 μm in diameter are classified as droplet transmission while particles less than 5 μm in diameter are classified as airborne transmission.[2] Droplet transmission of bacteria and viruses are transmitted from an infected person to noninfected person when droplets and individuals are in proximity. When the infected person coughs or sneezes, the noninfected person risks particles entering their body, leading to an infection. When compared to airborne transmission particles, droplet particles do not linger in the air for as long;[2] however, if a noninfected person touches the surface where particles land, this increases the chances of transmission.[2] Airborne transmission involves microbes with droplet nuclei that remain in the air for an extended period of time, which can be transmitted long distances. Current information from WHO states SARS-CoV-2 is transmitted via droplet, however, it can also be transmitted via droplet in situations such as intubation, nebulizer treatment, invasive positive pressure ventilation, cardiopulmonary resuscitation, disconnecting an infected patient from a ventilator, and open suctioning.[2]

N-95 Respirators for COVID-19

N-95 respirators, commonly referred to as N-95 masks, are designed to be efficient in the filtration of airborne particles. The masks contain four layers of an interwoven matrix designed to trap viral organisms, while still allowing air to filter through.[3]

Not only are these masks capable of highly efficient of filtration, but the coating on the mask is also designed to reduce and kill microorganisms. Though not approved by the Food and Drug Administration (FDA), some manufactures of N-95 respirators provide added protection via a hydrophilic plastic coating on the outer layer, and both outer and second layers are treated with compounds to inactive viruses.[3] The mask is designed to fit close to the face forming a tight seal around the nose and mouth in order to maintain filtration capabilities. N-95 respirators are used in the healthcare setting to protect healthcare professionals from airborne transmitted infections. N-95 respirators are the best option to prevent SARS-CoV-2transmission due to their filtration and special coating created to minimize transmission of small particles. The effectiveness of cloth masks of preventing the spread from a positive COVID-19 patient to the healthcare professionals caring for them depends upon the fabric's ability to capture airborne particles.[4] FDA strongly suggest not sharing or reusing N-95 respirators.[4]

Healthcare professionals should wear N-95 respirators when caring for a suspected or known positive COVID-19 patient. At the onset of COVID-19 pandemic, there was a shortage of N-95 respirators worldwide. If healthcare professionals need to resort to wearing homemade masks,

they should be used with caution and preferably worn with a faceshield.[5] Since this recommendation has been released, more information about the transmission of SARS-CoV-2 has been gathered. As of late, the CDC strongly recommends that healthcare professionals resume standard practices of PPE when as the facility's inventor allow.[5]

Lack of Protection from Cloth Masks

Cloth masks lack the filtration and unique coating of N-95 respirators. The lack of filtration generates uncertainty related to COVID-19 transmission especially in those who present as asymptomatic. Patients who are positive for COVID-19 and have no symptoms can spread the infection to others without either party being aware. Cloth masks are not considered protective equipment due to lack of information on their capabilities to filter microorganisms.[5] Many medical supply companies have become innovators to fill in the gaps in needed medical supplies.

Due to the CDC's recommendation for healthcare professionals to wear homemade face coverings in the absence of N-95 respirators, many homebased cloth mask companies are on the rise to ensure that enough masks are available to healthcare professionals and the public.

Emory Healthcare System (EHC) has initiated a "Mask Sewing Project" for seamstresses to donate homemade masks to the Emory hospitals and clinics. The goal of the "Mask Sewing Project" is to provide masks to their healthcare workers during personal protective equipment shortage. EHC encourages those interested in making cloth masks to shrink the fabric before sewing cloth into pre-approved patterns. The idea behind pre-shrinking fabric in hot water is to tighten the interwoven pattern of the fabric. EHC does not require the masks to have covered sides even though particles can enter the mouth and nose via openings in mask.[6]

In 2015, a randomized controlled trial was conducted in a Vietnamese hospital comparing cloth masks to surgical masks for protection against transmission of influenza. The trial found that hospital workers wearing cloth masks were 13 times more likely to become infected with the virus over those who wore disposable surgical masks.[7] Influenza is thought to be transmitted via droplet particles and can spread up to 6 feet.[8]

An Alternative to Consider: Antiviral Cloth Face Coverings

Following the H1N1 influenza outbreak in 2009, The University of Manchester created a research team led by Sabin Flitsch to discover a way to isolate viral strains responsible for seasonal influenza.[9] The University of Manchester partnered with a biotech company to develop and test protein coatings against influenza. In 2016, the team discovered that proteins could be anchored to substrates to be utilized against viral attacks acting as a protective role against the strain. The research team discovered specific glycoproteins that can mimic carbohydrate structures of the human esophagus and nasal passages using our natural proteins to capture pathogens.[9] Due to this discovery, the University of Manchester has begun prototype testing impregnating fabric. Further studies are required to test the efficacy of this product along with OSHA/FDA approval. If approved, the antiviral cloth coverings could be an alternative for use by healthcare professionals when N-95 respirators are unavailable.

Conclusion

Cloth masks alone do not offer the needed protection against COVID-19 for frontline healthcare professionals. Healthcare professionals and the general public understand that hospital representatives, government officials, and manufacturing companies are working hard to produce more N-95 respirators. The use of antiviral cloth coverings may help mitigate the shortage of N-95 respirators for healthcare professionals. An efficient, reusable cloth mask may help slow community spread of COVID-19 and reduce the demand on the healthcare system and restore supplies of needed protective medical equipment.

Reference List

1. Azad, A., Nedelman, M. Used facemasks and bandanas: How the CDC is warning hospitals to prepare for coronavirus shortages. CNN Health. https://www.cnn.com/2020/03/19/health/hospital-coronavirus-shortages-preparedness/index.html. Published March 19, 2020. Accessed August 30, 2020.
2. World Health Organization (WHO). Modes of transmission of virus causing COVID-19: Implications of IPC precaution recommendations. https://www.who.int/news-room/commentaries/detail/modes-of-transmission-of-virus-causing-covid-19-implications-for-ipc-precaution-recommendations. Published March 29, 2020. Accessed April 10, 2020.
3. Filligent (HK) Limited. 510(k) Summary: BioFriend/BioMask N95 Surgical Respirator. https://www.accessdata.fda.gov/cdrh_docs/pdf12/K122702.pdf. Published August 28, 2012. Accessed August 7, 2020.
4. Food and Drug Administration (FDA). N95 respirators and surgical masks (face masks). https://www.fda.gov/medical-devices/personal-protective-equipment-infection-control/n95-respirators-and-surgical-masks-face-masks. Published April 5, 2020. Updated June 7, 2020. Accessed April 11, 2020.
5. Center for Disease Control and Prevention (CDC). Strategies for optimizing the supply of facemasks. https://www.cdc.gov/coronavirus/2019-ncov/hcp/ppe-strategy/face-masks.html. Published March 17, 2020. Updated June 28, 2020. Accessed April 11, 2020.
6. Emory Healthcare (EHC). COVID-19 (Coronavirus): Mask sewing information. https://www.emoryhealthcare.org/covid/n95-mask.html. Accessed April 12, 2020.
7. MacIntyre, C. R., Seale, H., Dung, T. C., et. al. A cluster randomized trial of cloth masks compared with medical masks in healthcare workers. *BMJ Open*. 2015;5(4):1-10.
8. Center for Disease Control and Prevention (CDC). How flu spreads. https://www.cdc.gov/flu/about/disease/spread.htm. Updated August 27, 2018. Accessed April 12, 2020.
9. The University of Manchester. Pioneering germ trap technology moves forward with first commercial application. https://www.manchester.ac.uk/discover/news/pioneering-germ-trap-technology/. Published October 25, 2016. Accessed August 8, 2020.

The author has no conflicts of interest to report.

Mentor: Leonardo Campero, DNAP, CRNA

Hypercoagulability in COVID-19 Patients

James Ciaburri, BSN
Yale New Haven Hospital School of Nurse Anesthesia

Keywords: COVID-19, Hypercoagulability, Thromboembolism, Coagulation

The 2019 novel coronavirus (COVID-19) pandemic has spread rapidly, infecting millions of people in a matter of months.[1] While COVID-19 is commonly linked to severe pneumonia often progressing to acute respiratory distress syndrome (ARDS), a new feature of the disease has come to light.[2] A remarkably high number of COVID-19 patients are presenting with a deranged coagulation profile resulting in a hypercoagulable state.[1-10] The coagulation defect noted in this population has been linked to an increased risk of thrombotic complications as well as poor prognosis.[2,4,6,7] Proposed mechanisms behind the coagulopathy include severe inflammation, hypoxia, immobilization, and diffuse intravascular coagulation (DIC). However, there is much to be understood about the underlying disease process.[2,9] While a lack of knowledge exists regarding the nature and management of hypercoagulability in the COVID-19 population, new reports continue to develop. Recent research studies have surfaced offering new information about the pathophysiology, prevalence, treatment options, and guidelines for COVID-19 coagulopathy. A review of the most current findings on the coagulation defects caused by COVID-19 will provide new insight into treatment modalities of the disease.

Methods

Evidence Based Practice Model

An evidence-based practice model utilizing the problem, intervention, comparison, and outcome (PICO) method was used to complete this study.

Problem – There is a lack of information regarding the nature and management of hypercoagulability in COVID-19 patients. Obtaining and disseminating the most current information is essential to improving the treatment of those affected by the disease.

Intervention – Conducting a literature review with the goal of gathering and sharing the most current knowledge about this emerging issue.

Comparison – Comparing the findings of a variety of recent reports on hypercoagulability in COVID-19 patients.

Outcome – Readers will gain knowledge on the most current information regarding the prevalence, clinical features, and management recommendations of hypercoagulability in COVID-19 patients.

Search Methods

A thorough literature search was performed to gather the most current information about this recently discovered issue. The search was conducted using PubMed and UpToDate. Search terms utilized included COVID-19, hypercoagulability, thromboembolism, and coagulation. The

objective of this search was to locate any human trials or expert consensus guidelines that may aid in the management of the COVID-19 disease process.

Levels of Evidence
Due to the recent nature of the topic at hand, human studies regarding hypercoagulability in COVID-19 patients were scarce. Most of the studies reviewed include retrospective and prospective studies involving COVID-19 patients specific to a single hospital.

Literature Analysis

Coagulation studies
The pathophysiology behind COVID-19 coagulopathy remains poorly understood. New knowledge regarding hemostasis abnormalities in this population may be vital to managing the disease. Multiple studies have recently surfaced providing new insight into coagulation changes in COVID-19 patients.

In a study from China by Tang et al., coagulation tests were retrospectively reviewed in 183 consecutive patients infected with COVID-19.[4] Findings revealed that non-survivors of the disease had significantly higher D-dimer, fibrin degradation products (FDPs), and longer prothrombin times (PT) compared to those of survivors on admission.[4] The authors state that the use of these conventional coagulation tests may be beneficial in guiding treatment and that they may be directly related to patient prognosis.[4] Additional findings included significantly lower fibrinogen and antithrombin (AT) levels during late hospitalization.[4] It was also noted that 71.4% of non-survivors met criteria for overt disseminated intravascular coagulation (DIC), making it a common feature of severe disease in this cohort.[4]

Physicians at a hospital in Milan, Italy observed a number of patients developing deep vein thromboses (DVTs) and pulmonary embolisms (PEs), while lacking the presence of DIC as described by Tang et al.[4,6] Panigada et al. decided to create their own study utilizing a thromboelastography (TEG) point of care device as well as conventional coagulation testing.[6] The study included 24 intubated COVID-19 patients who were randomly selected from those admitted to the intensive care unit (ICU).[6] Collective TEG results revealed a significant state of hypercoagulability, which may provide explanation for the frequency of DVTs and PEs seen within their facility. Indicators of DIC as reported by Tang et al. such as prolonged PT, low platelet count, and low fibrinogen clotting activity were not found in this study.[4,6] Conventional blood tests revealed a normal platelet count with greatly increased levels of fibrinogen, von Willebrand factor, factor VIII, and D-dimer levels.[4] A similar study was conducted at Padua University Hospital in Italy that aimed to evaluate coagulation abnormalities in 22 ICU patients suffering from COVID-19 pneumonia. Coagulation was analyzed using traditional tests along with a ROTEM delta apparatus to measure whole blood thromboelastometry. Results revealed significantly higher fibrinogen and D-dimer levels in comparison to a healthy control group. Thromboelastometry profiles were similar to those observed by Panigada et al., as faster clot formation times and higher maximum clot firmness seen were consistent with a hypercoagulable state.[6,7] Spieza et al. mentions 23% of the patients in the study developed DVTs, even while being treated with prophylactic anticoagulation.[7] While neither of these studies support the notion of consumption coagulopathy as described by Tang et al., they do share the finding of

markedly high D-dimer levels, supporting the conclusion that elevated D-dimer is an independent risk factor for severe disease.[4,6,7] The hypercoagulability described in both reports may also support the use of antithrombotic drugs in this population as a method to prevent thromboembolic complications.[6,7]

Han et al. sought to evaluate the differences among blood coagulation parameters between COVID-19 patients and healthy controls in order to provide insight into their ability to predict progression of the disease.[10] The study involved 94 patients from a Wuhan China hospital with positive COVID-19 diagnosis and 40 healthy patients for the control group.[10] Compared to the control group, COVID-19 patients presented with significantly lower AT values and significantly higher FDP and D-dimer values.[10] After confirming findings from previous studies, the cohort of COVID-19 positive patients was split into three groups classified as ordinary, severe, or critical states of disease.[4,6,7,10] D-dimer and FDP values were found to be significantly higher in the severe disease group when compared to the mild disease group.[10] This finding highlights potential benefits of D-dimer and FDP monitoring in predicting the progression of COVID-19 disease.[10]

Fogarty et al. considers how ethnicity plays a significant role in coagulation, and that Chinese are 3-4 times less likely to experience venous thromboembolism (VTE) when compared to Caucasians.[2] In fact, VTE prophylaxis is far less prevalent in China for this reason.[2] Researchers designed a study to determine whether coagulopathic features differ between Chinese and Caucasian patients.[2] The cohort consisted of 83 COVID-19 positive patients recruited from St. James Hospital in Dublin, Ireland. Eighty-one percent of patients were Caucasian, 12% Asian, and 6% African.[2] Blood tests were performed on admission revealing significantly increased D-dimer, fibrinogen, and C-reactive protein levels.[2] The cohort was next divided into two groups based on patients who were discharged and those requiring ICU admission.[2] D-dimer, CRP, and fibrinogen levels on admission were found to be significantly higher in the groups requiring ICU level care.[2] Unlike Tang et al., DIC was not identified within the cohort and platelet levels remained within a normal range.[4,2] The lack of DIC development is hypothesized to be related to the use of pharmacologic thromboprophylaxis, which was utilized in all patients within this cohort.[10] Significant hypercoagulability related to severity of disease was, however, similar to that seen in Chinese cohorts.[2,4,10]

Incidence of Thromboembolism

The hypercoagulable state described in previous studies has been found to leave patients with COVID-19 infections at increased risk for thromboembolism. The following studies are aimed at identifying the incidence of this prevalent complication in order to guide prophylaxis and treatment of thromboembolic complications.

A research study by Klok et al. was created to evaluate the frequency of thrombotic complications in COVID-19 infected ICU patients from three Dutch hospitals.[9] The cohort consisted of 184 patients with COVID-19 pneumonia, receiving systemic thromboprophylaxis, who were followed for a total of 30 days.[9] The incidence of both venous and arterial thrombotic complications were evaluated, including PE, DVT, ischemic stroke, myocardial infarction, and arterial embolism.[9] Diagnostic evaluation was initiated only if patients showed signs of a thrombotic event.[9] The incidence of thrombotic complication within the cohort was 31%, with

PE being the most frequently recorded (81%).[9] Results exemplify the alarmingly high venous thromboembolism risk within this population and are suggestive of the need for a required increase in surveillance for these complications, as well as the need for thromboprophylaxis reevaluation. Limitations include the fact that most of the cohort remained in the ICU after 30 days, which may have resulted in thrombotic events that went unaccounted for.
In a retrospective study currently taking place at a French hospital, 100 COVID-19 positive patients exhibiting severe clinical features underwent contrast-enhanced computerized tomography scans.[8] The incidence of PE was recorded, revealing that 23% of the cohort suffered from acute PE.[8] Patients diagnosed with PE were found to be more likely to require mechanical ventilation, ICU admission, and be of the male gender.[8] The authors recommend the routine use of contrast-enhanced CT scans in this population, as patients with severe clinical features had a high incidence of PE.[8]

Benefits of Anticoagulation
In a second study by Tang et al., 28-day mortality between heparin users and non-users was retrospectively reviewed in order to analyze the efficacy of heparin in improving outcomes of COVID-19 patients.[5] The cohort consisted of 449 patients with a severe classification of COVID-19 disease.[5] Treatment with at least seven days of heparin prophylaxis was utilized in 22% of the cohort.[5] When comparing 28-day mortality between heparin users and non-users, no significant difference was present (30.3% vs. 29.7%, $P = 0.910$).[5] The cohort was further divided using a "sepsis-induced coagulopathy" (SIC) score, which signified an early phase of sepsis-associated DIC, as well as by D-dimer level.[5] Heparin prophylaxis was found to be associated with lower mortality in groups with elevated SIC scores as well as those with D-dimer levels over 3.0 ug/ml (40.0% vs. 64.2%, $P = 0.029$) and (32.8% vs. 52.4%, $P = 0.017$).[5] These results revealed a possible decreased mortality in patients exhibiting characteristics of severe disease who received heparin therapy.[5]

Professional Recommendations
In order to view the empirical evidence in the prevention and treatment of COVID-19 associated thromboembolism, a group of physicians and researchers from China and Europe worked together to develop a list of recommendations utilizing evidence and opinion-based guidelines.[1] The authors recommend employing frequent and dynamic risk analysis methods, considering the rapidly progressive nature of the disease.[1] In patients with mild to moderate cases, the use of venous thromboembolism (VTE) risk assessment scales are recommended to determine the need for VTE prophylaxis.[1] The researchers state that all critically ill COVID-19 patients are at high risk of thromboembolism, therefore VTE prophylaxis should be administered in the absence of contraindication.[1]

In all critically ill COVID-19 patients, subcutaneous low molecular weight heparin as first-line prophylaxis is recommended.[1] In the presence of heparin-induced thrombocytopenia, a non-heparin anticoagulant such as argatroban or bivalirudin is suggested.[1] Mechanical VTE prophylaxis utilizing intermittent pneumatic compression (IPC) is recommended as an alternative treatment in patients with high risk for bleeding.[1]
Hospitalized patients with mild to moderate COVID-19 infection should be graded using a VTE risk assessment scale.[1] Those with moderate to high VTE risk should be considered for LMWH therapy.[1] In low risk patients, adequate hydration and regular mobilization should be

encouraged.[1] Fever, diarrhea, and anorexia may occur with COVID-19 infection, resulting in dehydration requiring fluid administration.[1] Discharged patients with continued risk for VTE should be considered for LMWH therapy at home.[1] It is recommended that direct oral anticoagulants (DOACs) be used cautiously in this population as there may be an increased risk of bleeding when combined with antiviral medications used for treating COVID-19.[1]
Aggressive monitoring for symptoms of PE and DVT are encouraged.[1] Signs of DVT such as lower limb pain or swelling should be investigated using ultrasound.[1] Signs of worsening chest pain, hemoptysis, dyspnea, or hypoxemia warrant computed tomography pulmonary angiography.[1] Echocardiography is also recommended during suspicion of PE, as it may be more quickly available and practical for the quarantined patient.[1]
Authors acknowledge the use of D-dimer, fibrinogen, and FDP values as important indicators of disease progression as well as VTE risk.[1] In the case of rapidly increasing levels of these markers, it is recommended that bilateral lower extremity ultrasound be performed.[1]

Table 1. Synthesis of COVID-19-related Coagulopathy Literature

Author/Date	Level of Evidence	Population	Purpose	Key Findings
Tang et al.[5]	Single center retrospective cohort study	449 patients with severe COVID-19 infection	To validate the usefulness of SIC scores and coagulation parameters in screening patients who may benefit from anticoagulant therapy	-20% reduction in 28-day mortality with heparin therapy in patients with elevated D-dimer or meeting SIC criteria
Tang et al.[4]	Single center retrospective cohort study	183 patients with COVID-19 pneumonia	To analyze the coagulation features of patients with COVID-19 pneumonia	-Non-survivors had significantly higher D-dimer, FDP levels, and longer PT compared to survivors on admission - Fibrinogen and AT levels were significantly lower in non-survivors in late stages of the disease
Panigada et al.[6]	Single center randomized prospective cohort study	24 COVID-19 patients admitted to the ICU with acute respiratory failure	Hemostasis evaluation using TEG and plasma testing	-Cohort results consistent with hypercoagulability with severe inflammatory process -Inconsistent with acute DIC as described in previous studies

Spiezia et al.[7]	Single center prospective case-control study	22 COVID-19 patients admitted to the ICU with acute respiratory failure	Evaluation of coagulation abnormalities	-Results revealed severe hypercoagulability rather than consumptive coagulopathy -Significantly higher fibrinogen and D-dimer levels compared to healthy controls
Grillet et al.[8]	Single center retrospective cohort study	100 patients with severe COVID-19 infection examined with contrast enhanced CT scan	Evaluation of the association of COVID-19 and frequency of pulmonary embolus	-Revealed a high prevalence of pulmonary embolus in patients with severe COVID-19 (23%)
Klok et al.[9]	Multi-center retrospective cohort study	184 ICU patients with COVID-19 pneumonia on systemic thromboprophylaxis	Analyzing the incidence of venous thromboembolism and arterial thrombotic complications	-31% of patients experienced thrombotic complications -Pulmonary embolism was the most frequently observed complication -Age and coagulopathy were independent risk factors for thrombotic complication
Han et al.[10]	Prospective single center case-control study	94 patients admitted with COVID-2 infections compared to 40 healthy participants	To investigate the blood coagulation function of this population	-Antithrombin levels were significantly lower in the COVID-19 group -D-dimer, fibrin degradation products, and fibrinogen were significantly higher in the COVID-19 group -D-dimer and FDP levels significantly higher in the severe COVID-19 group compared to the mild group

Fogarty et al.[2]	Prospective single center cohort study	83 patients admitted with COVID-19 infections	To investigate COVID-19 coagulopathy in Caucasian patients	-Severe COVID-19 infection is associated with significant coagulopathy -Coagulopathy associated with marked increase in D-dimer and disease severity
Zhai et al.[1]	Evidence and opinion based expert consensus	N/A	To provide recommendations for providers in the prevention and treatment of VTE in COVID-19 patients	Offers a comprehensive guide including risk management, prophylaxis, and treatment strategies for VTE in COVID-19 patients

Conclusion

There is still much to be understood about the coagulopathic changes in the COVID-19 population; the studies reviewed offer further understanding. Findings confirm the presence of a hypercoagulable state places patients at increased risk for thrombotic complications.[1-10] Various coagulation tests such as D-dimer values may be useful in detecting disease severity and thromboembolic risk during early stages of the disease.[2,4,6,7,10] The potential benefit of heparin therapy in this population was also noted; however, further studies are needed as patients on prophylactic heparin continue to develop VTEs.[5,8,9] Most of the studies reviewed are single center studies with small cohorts, indicating that they hold a lower level of evidence. The lack of research available is due to the new and unfamiliar nature of the topic at hand. Further studies, including larger and more diverse populations are necessary to attain much needed information on this rapidly emerging issue. Nevertheless, being cognizant of the hypercoagulability and thrombotic risks present in this population is imperative in guiding treatment.

References

1. Zhai Z, Li C, Chen Y, et al. Prevention and treatment of venous thromboembolism associated with coronavirus disease 2019 infection: a consensus statement before Guidelines. *Thromb Haemost*. 2020; doi:10.1055/s-0040-1710019.
2. Fogarty H, Townsend L, Cheallaigh CN, et al. COVID-19 coagulopathy in caucasian patients. *Br J Haematol*. 2020; doi:10.1111/bjh.16749.
3. Giannis D, Ziogas A, Gianni P. Coagulation disorders in coronavirus infected patients: COVID-19, SARS-CoV1, MERS-CoV and lessons from the past. *J Clin Virol*. 2020;127:104362. doi:10.1016/j.jcv.2020.104362.

4. Tang N, Li D, Wang X, Sun Z. Abnormal coagulation parameters are associated with poor prognosis in patients with novel coronavirus pneumonia. *J Thromb Haemost*. 2020;18:844-847. doi:10.1111/jth.14768.
5. Tang N, Bai H, Chen X, Gong J, Li D, Sun Z. Anticoagulant treatment is associated with decreased mortality in severe coronavirus disease 2019 patients with coagulopathy. *J Thromb Haemost*. 2020; doi:10.1111/JTH.14817.
6. Panigada M, Bottino N, Tagliabue P, et al. Hypercoagulability of COVID-19 patients in intensive care unit. A report of thromboelastography findings and other parameters of hemostasis. *J Thromb Haemost*. 2020; doi:10.1111/JTH.14850.
7. Spiezia L, Boscolo A, Poletto F, et al. COVID-19-related severe hypercoagulability in patients admitted to intensive care unit for acute respiratory failure. *Thromb Haemost*. 2020; doi:10.1055/s-0040-1710018.
8. Grillet F, Behr J, Calame P, Aubry S, Delabrousse E. Acute pulmonary embolism associated with COVID-19 pneumonia detected by pulmonary CT angiography. *Radiology*. 2020; doi:10.1148/radiol.2020201544.
9. Klok FA, Kruip MJHA, Van Der Meer NJM, et al. Incidence of thrombotic complications in critically ill ICU patients with COVID-19. 2020; doi:10.1016/j.thromres.2020.04.013.
10. Han H, Yang L, Liu Rui, et al. Prominent changes in blood coagulation of patients with SARS-CoV-2 infection. *Clin Chem Lab Med*. 2020; doi:10.1515/cclm-2020-0188.

Mentor: Marianne S. Cosgrove, PhD, DNAP, CRNA, APRN

Management of the Vasoplegic Syndrome Patient undergoing Cardiac Surgery

Molly Edmundson, DNP
University of North Dakota

Key words: vasoplegic syndrome, methylene blue, hydroxocobalamin, cardiopulmonary bypass, hypotension

The importance of blood pressure management during cardio pulmonary bypass (CPB) is necessary for maintaining perfusion to vital organs. Hypotension can be resultant of the following: surgical failure, pump failure, ventricular dysfunction, vasoplegia, dysrhythmias, and outflow obstruction.[1] The incidence of vasoplegic syndrome (VS) is up to 25 percent in patients undergoing cardiac procedures requiring CPB.[3] Management of VS is imperative to reduce the morbidity and mortality associated with CPB. In addition to traditional methods, methylene blue and hydroxocobalamin are non-traditional options in the treatment of VS.

Case Report

A 72-year-old, 170 cm, 75.7 kg male presented for on-pump coronary artery bypass graft (CABG) with left internal mammary artery and saphenous endoscopic vein harvest. Pertinent medical history included hyperlipidemia, hypertension, congenital tracheoesophageal fistula, esophageal atresia, and chronic renal disease. Previous surgical history included partial nephrectomy, cystoscopy, and duodenal excision. The patient had allergies to simvastatin, sulfa

drugs, and latex. Home medications included lisinopril, omeprazole, tamsulosin, atorvastatin, isosorbide mononitrate, and aspirin. Events leading to admission included chest pain and subsequent diagnosis of non-ST elevation myocardial infarction (NSTEMI). Coronary angiogram was performed revealing severe three-vessel coronary artery disease (CAD) and the decision was made to proceed with CABG.

The airway evaluation was unremarkable. Baseline vital signs were as follows: BP 113/53 mm Hg, heart rate 64/min, respiratory rate 12/min, SpO_2 97% and temperature 36.5ºC. Midazolam 2 mg and fentanyl 50 mcg were administered for arterial line placement and an additional peripheral IV. Induction of general anesthesia was achieved with etomidate 8 mg, fentanyl 150 mcg, rocoronium 50 mg, phenylephrine 100 mcg, and lidocaine 40 mg. Atraumatic intubation of the trachea with an 8.0 mm endotracheal tube was achieved with a Macintosh 3 blade after obtaining a grade 1 view. The maintenance of anesthesia was achieved with 1.2% inspired isoflurane. The patient was mechanically ventilated with a volume assist mode. Blood pressure support was required after induction; this was achieved with multiple boluses of norepinephrine, phenylephrine, and ephedrine. Prior to sternal splitting, propofol 100 mg and fentanyl 150 mcg of were given. A tranexamic acid bolus and infusion was started. 30,000 units of heparin were given prior to initiating CPB. Additional medications administered included calcium 500 mg, protamine 260 mg, and mannitol 25 g.

The total pump time was approximately three hours and twenty minutes. During CPB, MAP ranged from 45 to 60 mm Hg. The CPB perfusionist supported blood pressure with phenylephrine, ephedrine, vasopressin, and norepinephrine. MAP values during CPB averaged 58.21 mm Hg in comparison to pre and post CPB MAP values, 72.78 and 60.08 mm Hg respectively. CPB sweep values ranged from 1 to 1.97 L/min. Blood pressure values were low at the beginning of bypass as well as during the process of coming of CPB. In addition to the perfusionists' management of blood pressure, the anesthesia team supported blood pressure with a phenylephrine infusion via the central line. CO values were 3.7 to 5.1 L/min, and SVR values ranged from 500 to 1150 dyn·seg/cm5·m2. Of note, SVR values were low coming off CPB and extending through the administration of protamine. Cerebral oximetry was maintained within 15 percent of the patient's baseline while on CPB. Urine output was maintained at 30 ml per hour and blood pH ranged from 7.15 to 7.34.

After the completion of four-vessel coronary artery revascularization, the patient was weaned off of bypass and was given protamine following the removal of CPB cannulas. A phenylephrine infusion was continued while coming off pump. Fluid administration throughout the case included 329 ml of cell saver and 1.5 L of crystalloid. The patient was then transported to the cardiovascular intensive care unit with plans to extubate. Post-operative day one was negative for stroke as well as, any other neurological deficits. Evaluation of renal function at this time indicated an increased creatinine of 1.5 mg/dl; this was a 26% increase from baseline. Nephrology was consulted and no further treatments were indicated at that time.

Discussion

Detrimental effects of hypotension during cardiac surgery results from inadequate perfusion to organs and tissues. These detrimental effects can extend up to five years from the initial

surgery.[4] The body requires necessary perfusion for the maintenance of oxygen and nutrient supply.[1-2] -While damage resulting from hypotension can be caused to any tissue or organ, literature focuses on its affects in the kidneys and brain.

Acute kidney injury (AKI), is an independent contributor to increased morbidity and mortality associated with cardiopulmonary bypass (CPB) surgery. Cardiac surgery is the second most common cause of AKI.[5] Hypotension, anemia, blood transfusion and decreased arterial oxygen content may be significant risk factors for AKI during cardiac surgery.[5] Hemodilution, bleeding, and systemic inflammatory response are some common causes responsible for decreased oxygen-carrying capacity.[5] Neurological injuries, such as cognitive decline, delirium, and stroke, are frequent occurrences following CPB.[4] Neurological injury after CPB is most likely due to particulate or microgaesous emboli that are created by CPB.[1] Additional cerebral hypoperfusion and systemic inflammatory response also increase the risk for neurological injury.[1]

Vasoplegic syndrome (VS) is loosely defined as hypotension that is refractory to most hypotension regimens. Etiologies for vasoplegia are thought to be similar to shock, and focus on the body's physiologic responses to CPB.[2,6] Further refinement from the previous VS definition includes severe hypotension (MAP less than 50 mmHg), low SVR, normal to elevated cardiac output, increased vasopressor requirements and fluid resuscitation.[2]

Treatment goals are focused on maintaining perfusion to tissues by means of maintaining a MAP value that is individualized for the patient.[7] Recent studies have suggested methods in identifying a patients' autoregulatory curve. For example, the cerebral oximetry index is a measurement obtained by doppler evaluation of cerebral flow, which can provide real-time values for cerebral perfusion.[7] Management of hypotension during CPB can be achieved through the modification of the anesthetic via pharmacological and/or non-pharmacological methods. Most commonly sympathomimetic agents, hypotension during CPB will be treated with sympathomimetic agents that exert their effects through action at adrenergic receptors. Examples of these agents include phenylephrine, norepinephrine, dopamine, and epinephrine. Vasopressin is a non-sympathomimetic agent that is used in the management of hypotension. In addition to vasopressors, volume resuscitation and calcium can also be utilized to improve MAP. Non-traditional medications, such as methylene blue (MB) and hydroxocobalamin, have also been included in the management of hypotension refractory to traditional methods.[2] A caveat to the use of any of these medications is an evaluation of the underlying etiology for hypotension. Attention must be paid to evaluate cardiac function and focus not just on increasing SVR.[6]

The patient exhibited the following factors associated with development of vasoplegic syndrome: intraoperative hypotension and decreased SVR. Additionally, the patient had been on an ACEi and was exhibiting signs of impaired LV function. During the case, it was noted that traditional methods used in the management of hypotension were being exhausted. However, the patient was able to be successfully weaned from CPB and maintained satisfactory blood pressures. While in the ICU, a decrease in renal function was noted. The patient's neurocognitive status was maintained at baseline. In this case, the use of MB or hydroxocobalamin may have improved MAP and decreased the overall need for vasopressors, potentially improving renal blood flow and preventing further insult to already impaired kidneys. In conclusion of this review of the management of VS in patients undergoing cardiovascular surgery, it has been made

evident that the anesthesia practitioner must perform a thorough preoperative evaluation and choose a VS treatment management plan which best supports the patients' hemodynamic needs.

References

1. Sarwar MF, Searles BE, Shore-Lesserson L, Stone ME, Shore-Lesserson L. Anesthesia for Cardiac Surgical Procedures. In Gropper MA, Cohen NH, Eriksson LI, Fleisher LA, Leslie K, Wiener-Kronish JP, eds. *Miller's Anesthesia* (9th ed.). Philadelphia, PA: Elsevier Churchill Livingstone; 2019: 1717-1814.
2. Liu H, Yu L, Yang L, Green M. Vasoplegic syndrome: An update on perioperative considerations. *J Clin Anesth*. 2017;40:63-71.doi: 10.1016/j.jclinane.2017.04.017.
3. Mazzeffi M, Hammer B, Chen E, Caridi-Scheible M, Ramsay J, Paciullo C. Methylene blue for postcardiopulmonary bypass vasoplegic syndrome: A cohort study. *Ann Card Anaesth.* 2017:20(2):178-181. doi: 10.4103/aca.ACA_237_16.
4. Cropsey C, Kennedy J, Han J, Pandharipande P. Cognitive dysfunction, delirium, and stroke in cardiac surgery patients. *Semin Cardiothorac Vasc Anesth*. 2015:19(4),309-317. doi: 10.1177/1089253215570062.
5. Haase M, Bellomo R, et al. Effect of mean arterial pressure, haemoglobin and blood transfusion during cardiopulmonary bypass on post-operative acute kidney injury. *Nephrol Dial Transplant*. 2012: 27 (1),53-160. doi: 10.1093/ndt/gfr275.
6. Shaefi S, Mittel A, et al. Vasoplegia after cardiovascular procedures: Pathophysiology and targeted therapy. *J Cardiothorac and Vasc Anesth*. 2018:32(2),1013-1022. doi: 10.1053/j.jvca.2017.10.032
7. Hessel E. What's new in cardiac surgery. *J Cardiothorac Vasc Anesth*. 2019:31(4),57-68. doi: 10.1053/j.jvca.2019.01.039.

Mentor: James Sperle, PhD, CRNA

Intrathecal Tetracaine for Inguinal Hernia Repair in a Preterm Infant

Adrianna J. Sutfin, DNP
Fairfield University & Bridgeport Hospital Nurse Anesthesia Program

Keywords: Premature infant, preterm infant, spinal or regional anesthesia, herniorrhaphy or hernia repair, apnea of prematurity, bronchopulmonary dysplasia

Premature birth occurs in 1 in 10 neonates.[1] Inguinal hernias are the most common surgical indication in premature infants, with an incidence of 38% and growing due to the advances in neonatal care.[2] Intraoperative management of premature infants is complex and challenging related to significant respiratory, cardiac and neurological comorbidities.[3] The use of general anesthetics (GA) in this population is associated with an increased incidence of postoperative adverse events including apnea and bradycardia, the potential for cerebral hypoperfusion, and concerns of anesthesia-related neurotoxic effects on the developing premature brain.[2-4] Intrathecal anesthesia allows for optimal surgical conditions while maintaining infant safety and stability.

Case Report

A 38-week, 5-day old infant, 3.2 kg and 46 cm tall, was scheduled for a left inguinal hernia repair with possible right inguinal exploration. He was born at 29-weeks, 2-days gestational age weighing 1420 g with a history significant for premature cesarean delivery following a motor vehicle accident, twin pregnancy, respiratory distress syndrome, sleep apnea, chronic lung disease of prematurity, suck-swallow incoordination, chronic anemia of prematurity, hyponatremia and suspected bilateral inguinal hernia.

After delivery, he was admitted to the neonatal intensive care unit (NICU) for aggressive respiratory management and monitoring. Despite continuous positive pressure ventilation (CPAP), he continued to have retractions, intermittent tachypnea, and transient decreases in his oxygen saturation as measured by pulse oximetry (SpO_2). His medical chart reports one episode requiring bag mask ventilation; however, intubation was deferred given improving arterial blood gas values and overall work of breathing. Initial chest x-ray reported diffuse nodular opacities in the lung bilaterally.

According to a note from the neonatologist the day before surgery, the infant no longer required CPAP or supplemental oxygen. The last documented episode of sleep apnea was 2 weeks prior with resolution to tactile stimulation. He was tolerating room air at rest, however continued desaturating with feedings, although self-resolving. Physical examination was significant for mild to moderate subcostal retractions; no upper or lower congestion was noted, and breath sounds were clear to auscultation, bilaterally. Other significant abnormal findings included mild pretibial edema and a prominent left inguinal hernia. Medications included caffeine for respiratory stimulation and diuretics, chlorothiazide 50mg BID and spironolactone 10mg daily. Most recent chest x-ray reported hyperinflation of the lungs with coarsened parenchymal markings suggestive of chronic lung disease of prematurity. Latest ultrasound of the head reported benign enlargement of the subarachnoid space. Preoperative vital signs were stable and lab work was unchanged from baseline. The infant was still hyponatremic, hypochloremic and hyperkalemic.

In preparation for the case, the operating room (OR) temperature was increased, heat lamps were utilized, and an underbody forced air blanket was placed on the table. The patient was transferred from the NICU to the OR per protocol in a transport crib, with standard monitors, emergency medications and airway equipment on hand. Once in the OR, the infant was placed and maintained in a seated position on the OR table, a pulse oximeter was placed on the lower extremity to monitor the oxygen saturation. Bilateral iliac crests were palpated to identify the L4 vertebrae, and the area was prepared and draped in a sterile fashion. A 22-gauge 1.5-inch cutting needle was inserted into the intrathecal space at L3-4. Placement of the needle was confirmed with aspiration of cerebrospinal fluid; subsequently, a single-shot injection was administered. The injection contained a combination of 1% tetracaine 0.22 ml, epinephrine 0.02 ml and 10% dextrose, 0.06 ml. After injection, the infant was placed supine with his legs flat against the bed to avoid any migration of the SA. Standard noninvasive monitoring was utilized. Soft bilateral upper arm restraints were secured and oral sucrose drops were placed on a pacifier for the infant to suck. No sedation was used.

Throughout the case, the infant exhibited several transient episodes of partial airway obstruction and mild oxygen desaturation as measured by SpO_2 to the mid-90s. These episodes were responsive to light jaw thrusts and high-flow O_2 10 L/min blow-by. He had no noted episodes of apnea or significant bradycardia. The postoperative NICU note indicated that after returning from the OR, the infant experienced oxygen desaturations to the 50s with feeding requiring repositioning, and self-resolving desaturations to the 80s while sleeping overnight. After the first postoperative night, there were no further episodes of desaturation and the patient was discharged home a few days later.

Discussion

Fifteen percent of infants born at 32 to 33 weeks gestation, 54% born at 30 to 31 weeks, and nearly all neonates born at <29 weeks exhibit apnea of prematurity (AOP), best defined by a pause of breathing >20 seconds, or <20 seconds accompanied by oxygen desaturation ($SpO_2 \leq 80\%$ for ≥4 seconds), bradycardia (HR<2/3 of baseline for ≥4seconds), cyanosis, pallor, or hypotonia in infants <37 weeks gestation.[2,5] This is secondary to immature respiratory systems, poorly developed central nervous system controls and pulmonary reflexes, and coexisting factors and disease states such as anemia, hypoglycemia and electrolyte imbalances.[5] They exhibit unstable, elastic rib cages and respiratory musculature prone to upper airway obstruction and lower airway collapse.[2] Even brief episodes of apnea can lead to oxygen desaturation, bradycardia, and hypotension and when prolonged, can lead to cerebral hypoperfusion and potential neurological deficits and developmental delays.[5]

While AOP is physiologic and can resolve with respiratory maturity, many premature neonates develop chronic lung disease of prematurity, also known as bronchopulmonary dysplasia (BPD).[6] This chronic disease is characterized by stunted lung growth, impaired vascular development, alveolar immaturity and abnormal pulmonary function persisting into adulthood.[6] With over 10,000 new cases in the US every year, BPD is the most common serious complication experienced by premature infants.[6]

With the administration of sedatives, narcotics, and volatile anesthetics, preterm infants are nearly twice as likely to develop adverse events from residual sedation, respiratory depression and muscle weakness compared to term infants.[3] This risk continues in former premature patients up to 23 years old.[3] Even in healthy premature infants undergoing herniorrhaphy with GA at a postmature age, 20 to 30% will have at least one apneic episode.[2] Research shows statistically significant decreases in the incidence of early apnea with SA as opposed to GA.[7] SA can reduce the risk of postoperative apnea by up to 47%.[2]

This technique remains underestimated and underutilized, due to practitioner unfamiliarity and fear of potential regional related adverse effects. In adults, high levels of a subarachnoid block can result in a preganglionic sympathetic blockade, leading to hemodynamic instability.[4] However, infants up to age one maintain cardiovascular stability after SA.[4] This may be related to a smaller venous capacitance and decreased pooling in the extremities; an immature sympathetic nervous system with a decreased dependence on sympathetic control of vascular tone; and decreased cardiac vagal activity due to a predominant effect on the parasympathetic nervous system.[4] Compared to infants receiving GA, infants receiving SA display higher systolic

blood pressures, slightly increased heart rates, and fewer hypotensive events requiring intervention.[7] SA is believed to modify the surgical stress response as evidenced by a decrease in epinephrine levels.[4]

The benefits of SA are limited in cases requiring supplementation with sedatives or conversion to GA due to unsuccessful placement or anesthetic failure. For this reason, infant preparation and practitioner technique is critical. Experienced centers report success rates close to 100%; lower rates have been attributed to poor access to the subarachnoid space, bloody taps, and blocks requiring supplementation.[8] All actions were taken to ensure successful subarachnoid placement, optimize the benefits of the anesthetic, and minimize the risk of failure and adverse events. Proper positioning enhanced the recognition of anatomical landmarks, which was critical due to the caudal extension of the spinal cord to L3 in infants compared to L1 in adults.[4] As per the literature, 1% tetracaine was utilized, mixed with 10% dextrose to increase baricity of the solution, and epinephrine which can prolong the duration of a block by 32%.[4] Infants exhibit faster drug distribution, uptake and elimination. These variations in pharmacokinetics are related to a larger volume of distribution, greater surface area of the spinal cord and nerve roots, increased cardiac output, and increased blood flow to the spinal cord, which explains why the motor level regression of a SA in an infant is approximately 5 times faster than that in an adult.[4]

A small 22-gauge spinal needle was used to prevent the well-known but extremely rare complication of postdural puncture headache; the use of a smaller-gauge, pencil-point needle is the most effective preventative measure.[4] We had to consider the possibility of the infant's inability to tolerate the wakefulness or the supine position, or the procedure outlasting the duration of the LA. The research supports the use of a pacifier dipped in 10% dextrose or oral sucrose to settle and soothe, maneuvers to prevent airway obstruction, oxygen by face mask or blow-by to maintain SpO_2, and the use of soft upper arm restraints if needed.[2,4]
SA is the safest technique for premature infants undergoing inguinal hernia repair, as it avoids the sedation, respiratory depression, hemodynamic instability, and residual muscle weakness associated with GA. SA decreases the risk of intraoperative and postoperative complications while providing analgesia and relaxation with rapid recovery and minimal physiologic changes, which is especially beneficial in this complex and high-risk population.

References

1. Division of Reproductive Health, National Center for Chronic Disease Prevention and Health Promotion. Premature Birth. *Centers for Disease Control and Prevention*. https://www.cdc.gov/reproductivehealth/maternalinfanthealth/pretermbirth.htm. Updated October 21, 2019. Accessed April 20, 2020.
2. Jones LJ, Craven PD, Lakkundi A, Foster JP, Badawi N. Regional (spinal, epidural, caudal) versus general anaesthesia in preterm infants undergoing inguinal herniorrhaphy in early infancy. *Cochrane Database Syst Rev.* June 2015. https://www.cochranelibrary.com/cdsr/doi/10.1002/14651858.CD003669.pub2/full
3. Havidich JE, Beach M, Dierdorf SF, Onega T, Suresh G, Cravero JP. Preterm Versus Term Children: Analysis of Sedation/Anesthesia Adverse Events and Longitudinal Risk. *Pediatr*. 2016;137(3):12. doi:10.1542/peds.2015-0463.

4. Libby A. Spinal anesthesia in preterm infants undergoing herniorrhaphy. *AANA J*. 2009;77(3):199-206. PMID: 19645169.
5. Zhao J, Gonzalez F, Mu D. Apnea of prematurity: from cause to treatment. *Eur J Pediatr*. 2011;170(9):1097–1105. doi:10.1007/s00431-011-1409-6.
6. Michael Z, Spyropoulos F, Ghanta S, Christou H. Bronchopulmonary Dysplasia: An Update of Current Pharmacologic Therapies and New Approaches. *Clin Med Insights Pediatr*. 2018;12. doi:10.1177/1179556518817322.
7. Davidson AJ, Morton NS, Arnup SJ, et al. Apnea after Awake Regional and General Anesthesia in Infants: The General Anesthesia Compared to Spinal Anesthesia Study--Comparing Apnea and Neurodevelopmental Outcomes, a Randomized Controlled Trial. *Anesthesiology*. 2015;123(1):38–54. doi:10.1097/ALN.0000000000000709.
8. Frawley G, Bell G, Disma N, et al. Predictors of Failure of Awake Regional Anesthesia for Neonatal Hernia Repair: Data from the General Anesthesia Compared to Spinal Anesthesia Study--Comparing Apnea and Neurodevelopmental Outcomes. *Anesthesiology*. 2015;123(1):55–65. doi:10.1097/ALN.0000000000000708.

Mentor: Steven L. Belmont, DNP, CRNA, APRN

Anesthetic Management of Failed Tracheostomy Insertion

Taylor Nix, MSN
Samford University

Keywords: failed tracheostomy, goiter, subglottic mass, difficult airway, tracheal malacia

Tracheostomy procedures are used to provide an airway for patients requiring long-term assisted ventilation or an alternative airway conduit.[1] Patients needing tracheostomy cannulation may present with multiple comorbidities along with poor respiratory function.[1] Potentially, one of the most devastating complications is cannulating a false passage outside of the trachea resulting in loss of the airway and potential damage to nearby structures.[1] The following case study describes failure to ventilate via a tracheostomy tube due to an unknown goiter causing tracheal compression.

Case Report

A 57-year-old, 170 cm, 69 kg female presented for open tracheostomy tube insertion. She was admitted with respiratory distress from her nursing home residence 6 days prior to the day of surgery. Her history included paraplegia resulting from a motor vehicle accident, previous tracheostomy, pneumonia, hypertension, atrial flutter, coronary artery disease, diabetes mellitus and anemia. On admission, she was hypotensive and receiving ceftriaxone for presumed pneumonia. She was intubated in the intensive care unit and sedated with a dexmedetomidine infusion. She failed weaning trials so tracheostomy tube placement was scheduled. Radiological findings included bilateral opacities with the right side more extensive than the left secondary to recent pneumonia. On the day of surgery evaluation, she was awake and following commands with stable vital signs and clear but diminished bilateral breath sounds.

In addition to the dexmedetomidine infusion, midazolam 2 mg and rocuronium 50 mg were administered intravenously (IV) for transport to the operating room (OR). The patient was induced with propofol 100 mg to mitigate hypertension with positioning and maintained with isoflurane 1.0% inspired concentration in O_2 0.5 L/min and air 1.5 L/min. As the procedure began, her SpO_2 was 100% and $EtCO_2$ was 32 mmHg. Low inspired O_2 concentrations were used to minimize fire risks. Bilateral breath sounds via the preexisting 7.5 mm endotracheal tube (ETT) were confirmed.

Approximately 45 minutes into the procedure, the surgeon requested that the ETT be withdrawn to his satisfaction as he inserted the tracheostomy cannula. The ETT was withdrawn to the level just proximal of the tracheostomy insertion site. The anesthesia circuit was passed through the drapes, attached to the tracheostomy adapter and attempts were made to ventilate. There was no $EtCO_2$ tracing seen and the SpO_2 rapidly dropped to 75%. The surgeon was immediately informed, and the attending anesthesiologist was called for assistance. The tracheostomy tube was withdrawn and the ETT successfully re-advanced into the trachea. Upon reinflation of the ETT cuff, a leak was noted, and a torn cuff was suspected. An $EtCO_2$ waveform was present and the SpO_2 recovered with manual ventilation of 100% O_2 at 2 L/min. This sequence was repeated multiple times as the surgeon would request withdrawing the ETT for placement of the tracheostomy tube, manual ventilation would fail, the tracheostomy tube would be removed and the ETT re-advanced into the trachea. Peak airway pressures increased from 26 to 32 cm H_2O and pink frothy secretions were noted in the ETT, presumed to be related to acute pulmonary edema. Furosemide 40 mg IV and albuterol through the ETT tube were administered.

A cardiothoracic surgeon was consulted and the decision was made to perform additional diagnostic studies before reattempting the tracheostomy procedure. A bougie stylet was used to exchange the ETT with the torn cuff for another 7.5 mm ETT. Dexamethasone 4 mg IV was administered to minimize the risk of airway edema. Routine postoperative chest radiograph (CXR) is typically performed following tracheostomy, especially in the setting of acute pulmonary edema with increased peak airway pressures.[1] The CXR confirmed a left pneumothorax and a pneumomediastinum, likely related to the trauma of multiple attempts at tracheal cannulation. With stable vital signs, the patient was transported to the interventional radiology suite for chest tube placement.

Follow up imaging revealed that the trachea was collapsed around the ETT. Computerized tomography (CT) demonstrated a prominent thyroid isthmus near the site of the attempted tracheostomy tube placement. It was determined that airway compression by the goiter was obstructing ventilation through the tracheostomy tube. The tracheostomy tube terminated proximal to the compression whereas the ETT could be advanced past it. This could not be visualized intraoperatively, even with the open tracheostomy approach. Following evaluation of the imaging studies, a thyroidectomy with tracheostomy tube placement was successfully performed.

Discussion

Tracheostomy tube placement is useful for improving comfort and quality of life for patients requiring prolonged assisted ventilation but is not without risk.[2] Complications are classified as

immediate, early or late.[2] Intraoperative complications most likely to influence anesthetic management include pneumothorax, hemorrhage, cannula misplacement, posterior tracheal perforation, thyroid injury, recurrent laryngeal nerve injury, subcutaneous emphysema, cardiopulmonary arrest and airway fire.[1] Pneumothorax is the most common adverse event, occurring in up to 17% of cases.[1]

Authors of a systematic review appraised 109 manuscripts published between 1990 and 2015 describing tracheostomy related deaths. Over 25,000 tracheostomy procedures were reviewed with 350 deaths reported. The most common causes of death were attributed to hemorrhage, airway loss and false passages occurring during the intraoperative and postoperative periods. These authors conclude with recommendations that tracheostomy related deaths can best be avoided by vigorous training and mentorship by experienced physicians, use of the World Health Organization's Surgical Safety Checklist and continuous, vigilant nursing care.[3]

Although cannula misplacement is a recognized complication of tracheostomy tube insertion, anesthesia practitioners have developed methods to minimize the risk of complete loss of the airway. In a published case report, practitioners described the use of a Cook airway exchange catheter (Cook Inc., Bloomington, IL) through the ETT with the help of a swivel adapter.[4] This allowed for O_2 insufflation while the ETT was retracted and the surgeon obtained visualization.[4] The airway exchange catheter remained in place until successful tracheostomy tube insertion with adequate ventilation was confirmed.[4]

In another published case report, anesthesia practitioners described the events of a failed tracheostomy insertion resulting in cardiac arrest. Following inability to ventilate via the tracheostomy tube, the anesthesia team placed an ETT through the tracheostomy incision yet there was still failure to adequately ventilate the patient.[5] Recommendations from this case study included use of a jet ventilation airway exchange catheter to prepare for a lost airway.[5] The catheter can be advanced through the ETT through a bronchoscope attachment prior to surgical entry of the trachea.[5] This allows for O_2 insufflation until the airway can be reestablished. In the case report related to airway obstruction by the goiter, a bougie stylet was used to secure the airway for exchange of the ETT. The stylet could have remained within the ETT each time it was withdrawn and re-advanced to increase the likelihood of successful reintubation.

Radiologic imaging is the standard preoperative assessment tool prior to tracheostomy procedures.[6] In some cases, a chest film may not be adequate to assess anatomy of the neck. There should be a low threshold for ordering preoperative CT scans.[6] In this case study, a preoperative CT scan was not done but could have assisted in minimizing the risk of failed surgical attempts and airway trauma that resulted in hypoxia and pulmonary edema.

Communication and responsiveness were primary factors in this patient's eventual successful outcome. Universal to any difficult airway or emergency algorithm is the need for the primary anesthesia practitioner to call for help.[7] In this case, the CRNA promptly recognized the patient's loss of airway with the first failed cannulation attempt and called for an additional anesthesia practitioner. Effective communication between the CRNA and surgeon was essential while the absence or presence of effective ventilation was being determined. According to statistics

reported by the Joint Commission, communication breakdown is the third leading root cause of sentinel events.[8]

This case study demonstrates a rare but life-threatening complication of tracheostomy tube placement. An unknown goiter obstructed ventilation through the tracheostomy tube and after multiple attempts with subsequent patient deterioration, the procedure was terminated. Although tracheostomy tube placement for this patient was not anticipated to be technically challenging, challenges did occur. There are suggested ways to minimize the risk of tragic outcomes related to tracheostomy cannulation including preoperative CT imaging, the intraoperative use of alternative airway devices and vigilance.

References

1. Stawicki S, Cipriano A, Mao M, et al. An overview of complications associated with open and percutaneous tracheostomy procedures. *Int Cri Illness Inj Sci*. 2015;5(3):179. doi:10.4103/2229-5151.164994
2. Cheung NH, Napolitano LM. Tracheostomy: epidemiology, indications, timing, technique, and outcomes. *Respir Care*. 2014;59(6):895-919. doi:10.4187/respcare.02971
3. Klemm E, Nowak AK. Tracheotomy-related deaths - a systematic review. *Dtsch Arztebl Int*. 2017;114: 273–279. doi:10.3238/arztebl.2017.0273
4. Gitman M, Mueller M. The use of an airway exchange catheter during a difficult tracheostomy. *J Neurosurg Anesthesiol.* 2012;24(1):78-79. doi:10.1097/ana.0b013e318237fa57
5. McGuire G, El-Beheiry H, Brown D. Loss of the airway during tracheostomy: Rescue oxygenation and re-establishment of the airway. *Can J Anesth*. 2001;48(7):697-700. doi:10.1007/bf03016207
6. Kanwalpreet, S. Anticipated difficult tracheostomy: Should CT scan be a pre-requisite. *J Emerg Intern Med.* 2017;1(2):14.
7. Apfelbaum JL, Hagberg CA, Caplan RA, et al. Practice guidelines for management of the difficult airway. *Anesthesiology*. 2013;118(2):251-270. doi:10.1097/aln.0b013e31827773b2
8. Schumacher P, ed. Patient safety: Sentinel event statistics released for 2014. *Joint Commission Online*. April 2015:1-2. https://www.jointcommission.org/assets/1/23/jconline April_29_15.pdf. Accessed June 23, 2019.

Mentor: Lisa Herbinger, DNP, CRNA

Epidermolysis Bullosa

Amy Laflamme, MS
Northeastern University

Keywords: epidermolysis bullosa, esophageal strictures, esophageal dilation

Epidermolysis bullosa (EB) is a rare group of genetic diseases characterized by bullae (blister) formation, particularly in the oropharynx and esophagus.[1] While there are more than 20 subtypes, the three main categorizations of EB are simplex, junctional, and dystrophic.[2] Bullae may form in this population spontaneously, or as a result of minimal trauma, leading to scar formation, esophageal strictures, microstomia, tissue contraction and pseudosyndactyly.[1,3] Epidermolysis bullosa poses unique challenges to the anesthesia practitioner with many aspects of care to consider when caring for this population.

Case Report:

A 9-year-old (21.1 kg, 125 cm) female presented with esophageal strictures secondary to recessive dystrophic EB for an esophagogastroduodenoscopy with dilation and fluoroscopy, . Significant past medical history included multiple esophageal stricture dilations, failure to thrive with g-tube dependence, constipation, dysphagia, right hand syndactyly, and superficial desquamation of oral mucosa, tongue and hard palate. Her current home medications included Miralax and daily dressing changes with dilute bleach baths followed by moisturization with hydrolatum to any open areas. She had received anesthesia in the past for multiple esophageal stricture dilations and dental extractions. These past procedures were complicated by oral hematomas and oral mucosal sloughing resulting in admission to the hospital. Notable exam findings included limited mouth opening, a Mallampati III airway classification, white discoloration of the tongue, and an oblong blister on her nose extending along the bridge.

The patient was premedicated with midazolam 10 mg orally in the preoperative holding area and brought to the procedure room with her mother. The patient moved herself to the procedure table at her own pace to prevent skin shearing. Due to skin sensitivity, adhesive was removed from infant electrocardiogram leads, lubricant was applied, and Mepitec tape (Mölnlycke Health Care, Peachtree Corners, GA) was used to secure the leads to her chest. The adhesive from the Sp02 pulse oximeter was removed and the sensor was secured with Mepitec tape. The patient's left leg was wrapped with Webril (Covidien/Medtronic, Minneapolis, MN), and the blood pressure cuff was placed on top of the Webril. Once standard noninvasive monitors were applied, the patient was preoxygenated with O_2 6 L/min via a child sized mask covered in Xeroform gauze (McKesson, Irving, TX) where the mask would come into contact with the patient's face. An inhalation induction was performed with sevoflurane 6.2% inspired concentration in O_2 2.5 L/min and N_2O 4 L/min.

Once anesthetized, gentle mask ventilation was performed with minimal manipulation, while a 24-gauge peripheral intravenous (IV) line was placed under ultrasound guidance with the provider's hand used as tourniquet over Webril. The peripheral IV was secured with Mepitec tape and Webril gauze was placed under the hub. Rocuronium 10 mg was given following line

placement. A video laryngoscope was used in order to minimize airway manipulation. A Mac 2 blade and 5.0 mm cuffed endotracheal tube (ETT) were both lubricated before instrumenting the airway. The video laryngoscope blade was gently used to obtain a IIa direct and indirect partial view of the glottis. The styletted ETT was then advanced atraumatically to 14.5 cm at the lip under video visualization. The ETT cuff pressure was checked with a manometer to ensure an appropriate pressure between 20 and 30 cm H_2O. The patient's lips were lubricated, and the ETT was held by hand with lubricated gloves throughout the case to avoid pressure on the corner of the mouth or tongue.

Sevoflurane was delivered at a 2.2% expired concentration with air 1 L/min and O_2 1L /min for maintenance of anesthesia throughout the 44-minute case. Acetaminophen 240 mg was administered intravenously for pain control, and 2 mg ondansetron was given for postoperative nausea and vomiting prophylaxis. Neuromuscular blockade was antagonized with glycopyrrolate 0.2 mg and neostigmine 1 mg. The patient's oropharynx was carefully suctioned with a soft catheter, and the patient was extubated to blow by O_2 after meeting extubation criteria. The challenge was that the patient needed to be awake enough to decrease the risk of an emergent re-intubation, but also comfortable enough that she would not be coughing and thrashing which could cause oral trauma. Given these challenges, the video laryngoscope was kept in the room for emergence. The patient was transported to the post anesthetic care unit where her mother was brought back to the bedside as soon as possible to minimize emergence delirium and agitation.

Discussion

Epidermolysis bullosa is a heterogeneous group of hereditary diseases resulting in exceptionally fragile mucous membranes and skin leading to the formation of blisters and ulcers.[2] Blisters may form spontaneously or after minor trauma. There are three main subtypes of EB categorized by blister depth of the skin layers: simplex involving epidermal cells, dystrophic involving type VII collagen and junctional involving the dermal-epidermal junction.[1] The patient in this case had dystrophic EB which is caused by mutations affecting collagen VII and leaves dystrophic scars after blister healing.[2] Over time, blister formation may create esophageal strictures eventually resulting in dysphagia. Repeated esophageal dilatations are necessary to avoid complications such as malnutrition and delayed wound healing.[5]

This disease poses many challenges for the anesthetic practitioner. Thorough preoperative assessment is crucial to determine current, active blisters or areas that may require additional care. In this case, the patient and family requested a mask induction with oral premedication. To minimize trauma with mask ventilation, xeroform was applied to the mask, which provided a gentler approach to inhalation induction. In addition, the xeroform also facilitated a good seal which further minimized airway manipulation. Further, patients with EB may have temporomandibular joint involvement contributing to a potentially difficult airway.[3] Despite a small oral opening, the patient's previous anesthesia records demonstrated successful intubation using a video laryngoscope. Given the short duration of the case, the decision was made to avoid securing the ETT with any form of adhesive. While certainly not always an option, it allowed for minimal trauma in this particular case.

Potential complications related to the application of monitors and securing IV access also posed a rare challenge. We ensured that all equipment that would potentially touch the patient was thoughtfully padded and any linen that the patient would be lying on had no creases.[1] This patient's disease also involved pseudosyndactyly, which occurs from repeated scarring caused by EB and leads to joint contractures and fusion of digits.[4] This presented a challenge when applying the pulse oximeter as well as when attempting to obtain IV access. Tourniquets may be used with caution, however, we used Webril with a hand acting as the tourniquet as this proved to be atraumatic for the patient in the past.[4] The patient had been scheduled in the main operating room due to increasing issues with her dilations, specifically her difficult IV access.

Emergence presents another set of risks in this fragile population. While we reversed muscle relaxant with neostigmine and glycopyrrolate, it would certainly be worth considering the use of sugammadex if there was any question of residual neuromuscular blockade. It is critical to avoid any sort of emergent re-intubation. In the case of a patient with EB undergoing a painful operation, a well thought out pain control plan will need to be formulated as a team prior to the patient arriving to the recovery area.[4] The prevention of postoperative pain and delirium is prudent in order to avoid potential new bullae formation.

Given the chronic nature of EB, it is common for the same patient to appear in the operating room repeatedly. For this reason, it is essential to be an expert record keeper. Well-kept anesthesia records allow future team members to confidently, and safely, deliver expert care. Caring for this rare patient population can be daunting, especially in the operating room. However, with collaboration, thoughtful planning and clear team communication, the patient can have a safe anesthetic experience.

References

1. Marschall K. Skin and musculoskeletal diseases. In Hines RL, Marschall K. *Stoelting's Anesthesia and Co-Existing Disease*. 7th ed. Saunders; 2018:507-538.
2. Araújo M, Brás R, Frada R, Guedes-Martins L, Lemos P. Caesarean delivery in a pregnant woman with epidermolysis bullosa: anaesthetic challenges. *Int J Obstet Anesth*. 2017;30:68-72.
3. Özkan AS, Kayhan GE, Akbaş S, Kaçmaz O, Durmuş M. Emergency difficult airway management in a patient with severe epidermolysis bullosa. *Turk J Anaesthesiol Reanim*. 2016;44(5):270-272.
4. Strupp KM, Zieg JA, Johnson B, Szolnoki JM. Anesthetic management of a patient with epidermolysis bullosa requiring major orthopedic surgery: A case report: A case report. *A A Case Rep*. 2017;9(3):73-76.
5. Gollu G, Ergun E, Ates U, Can OS, Dindar H. Balloon dilatation in esophageal strictures in epidermolysis bullosa and the role of anesthesia: Dilatation in esophageal strictures in epidermolysis bullosa. *Dis Esophagus*. 2017;30(3):1-6.
6. Narejo AS, Khan MU, Alotaibi WM, Khan MM. Anesthetic consideration in dystrophic epidermolysis bullosa. *Saudi J Anaesth*. 2016;10(1):110-112.

7. Al-Abadi A, Al-Azri SA, Bakathir A, Al-Riyami Y. Dental and anaesthetic challenges in a patient with dystrophic epidermolysis bullosa. *Sultan Qaboos Univ Med J*. 2016;16(4):e495-e499.

Mentor: Maria Van Pelt, PhD, CRNA

Airway Management for Pediatric Patients with Trisomy 21

Richie Gallegos, DNAP
University of Southern California

Keywords: Trisomy 21, Down syndrome, mask ventilation, airway management, pediatric

Trisomy 21 or Down Syndrome (DS) occurs in approximately 1 out of every 707 live births in the United States, estimating a rate of 5,568 births annually. When adjusted for advanced maternal age, this rate increases to 6,242 births annually, or approximately 1 out of every 635 live births.[1] Children with Trisomy 21 often have a higher incidence of genetic heart defects, autoimmune disorders, musculoskeletal anomalies, vision and hearing problems, and respiratory complications. Airway considerations contributing to anesthetic challenges and potential difficult airway management include macroglossia, midface hypoplasia, a narrow nasopharynx, laryngotracheal anomalies, and muscular hypotonia.[2] Therefore, it is critical for anesthesia providers to perform thorough preoperative physical assessments, understanding the physiological changes associated with the disorder.

Case Report

A 6-year-old male with Trisomy 21 presented to the ambulatory surgical center for an ear exam under anesthesia and bilateral myringotomy tube placement. Past medical history included evidence of a developmental delay, hearing loss, and frequent episodes of otitis media requiring antibiotics. The parents reported a "hole" in the child's heart that closed spontaneously at age one. No cardiology follow-up was necessary. Parents denied any history of heavy snoring, gasping or cessation of breathing at night. No other significant comorbidities were identified. The patient did not have any past surgical history.

Upon physical examination, the patient measured 18 kg and 108 cm, with a body mass index (BMI) of 15.4 kg/m2, placing him in the 52nd percentile for boys aged 6 years. Airway examination revealed a small mouth opening, large tongue, and short neck. Full neck range of motion was intact without cervical spine abnormalities. His lungs were clear to auscultation bilaterally, and his heart rate and rhythm were regular. No murmurs or abnormal heart sounds were present.

Preoperative sedation was established with oral midazolam 9 mg in preparation for an intraoperative mask induction. Preoperatively, the patient had a blood pressure of 100/59 mmHg, heart rate of 90/min, respiratory rate of 22/min, SpO_2 of 97%, and temperature of 36.4ºC.

In the operating room, the patient was placed on a mixture of O_2 3 L/min and N_2O 6 L/min via face mask. Standard noninvasive monitors were applied, and baseline vital signs were recorded. Sevoflurane was initiated and titrated to an inspired concentration of 8%. Nitrous oxide was discontinued when stage 3 of anesthesia was established. Spontaneous respirations were maintained during the induction phase, and supportive manual breaths were provided as needed. An oropharyngeal airway was placed when the patient demonstrated signs of upper airway obstruction. Once a sevoflurane minimum alveolar concentration (MAC) of 2 was reached, dexmedetomidine 20 mcg was administered intranasally. General anesthesia via mask was maintained at a MAC of 1.4 with a mixture of O_2 0.5 L/min and air 1.5 L/min. Peripheral intravascular access was not attempted. Spontaneous respirations continued throughout the case.

Intraoperatively, surgical visualization of the left and right ears required turning of the patient's head. Adequate mask seal and airway patency were maintained during the positioning of the patient's head and neck. At surgery end, sevoflurane was discontinued, and oxygen was administered at a rate of 10 L/min. Total surgical time was 10 minutes.

The patient was transported to the post anesthesia care unit with 6 L/min O2 via face mask in a side lying recovery position. The oropharyngeal airway was removed by the recovery nurse upon return of airway reflexes. Postoperatively, the patient had a blood pressure of 85/52 mmHg, heart rate of 81/min, respiratory rate of 19/min, SpO_2 of 99%, and temperature of 36.2 ºC. No postoperative complications were identified.

Discussion

Trisomy 21 is associated with physical characteristics that may lead to medical conditions requiring surgical intervention. For example, chronic otitis media with effusion typically develops due to short Eustachian tubes and hypotonia of the external auditory meatus and tracheobronchial muscles. Accumulation of mucous and fluid in the middle ear can lead to recurrent ear infections and hearing loss. Surgical intervention is required to relieve excess fluid or pressure. Compared to pediatric patients without the disorder, children with Trisomy 21 are 13 times more likely to require pressure equalization tubes to correct hearing loss.[3] In anticipation of this population's increased surgical and anesthetic need, it is important for anesthesia providers to be prepared to handle the challenges that accompany these patients with craniofacial abnormalities.

Obstructive Sleep Apnea (OSA) is a respiratory issue frequently encountered in patients with Trisomy 21, with rates occurring in as high as 79% of subjects being studied.[4] OSA may be the result of various factors, including adenotonsillar hypertrophy, macroglossia, and obesity. In a study by Chamseddin et al, 106 children with Trisomy 21 were examined to identify any risk factors predictive of severe OSA. Obesity was the most significant risk factor, demonstrating the importance of counseling parents on weight monitoring and weight loss management for their children.[5] The child described in this case report had a BMI in the 52nd percentile for males his age. Although his weight did not place him at high risk of severe OSA, macroglossia was noted upon physical assessment.

To prevent worsening symptoms of OSA during general anesthesia, dexmedetomidine can be used as an alternative to medications that depress airway reflexes. In a study by Mahmoud et al, dexmedetomidine was used to mimic non-rapid eye movement (NREM) sleep in Trisomy 21 patients being assessed for pharyngeal collapsibility. The study determined that patients with OSA under a dexmedetomidine-induced sleep were capable of maintaining their airway responses as if they were experiencing natural sleep.[6] Intranasal dexmedetomidine was used safely as a sedative while preserving a patent airway. In this case study, potential airway complications were avoided with intranasal dexmedetomidine and an inhalation agent delivered by mask.

If general anesthesia by mask is not possible, an alternative approach is laryngeal mask airway (LMA). If direct laryngoscopy with endotracheal tube placement is necessary, anesthetic considerations should recognize the airway anatomy unique to Trisomy 21. Using magnetic resonance imaging (MRI), Shott evaluated the airways of 42 children with Trisomy 21 and compared them with the airways of 32 control subjects. The study concluded that the subglottises and tracheal diameters of children with Trisomy 21 were significantly smaller than those in the control group. Therefore, when intubating a child with Trisomy 21, the endotracheal tube size should be at least two sizes smaller than what would normally be considered standard for his or her age. Furthermore, a leak test should be a routine part of the intubation process to confirm the proper fit of the endotracheal tube.[7]

Whether administering general anesthesia to a patient with Trisomy 21 by mask, LMA, or endotracheal tube, caution must be taken when positioning the patient's head and neck. Cervical instability may be the result of ligamentous laxity or osseous abnormalities, and over half of children with Trisomy 21 may present with either atlanto-occipital or atlanto-axial instability. However, only about 1% to 2% of patients are symptomatic, and few reports of injury have actually been reported. Nevertheless, common ear, nose, and throat procedures should be performed with the head in neutral position and with neck rotation limited to 60 degrees. If possible, rolling the operating table is preferable to rotating the patient's head.[8] The parents of the patient in this case report did not report any issues with cervical instability, nor were there clinical signs of neck range of motion limitations. However, cervical instability could not be ruled out, as no x-rays or radiology reports were charted. Despite this, the patient tolerated minor neck rotations during the procedure.

Summary and Conclusion

When determining the anesthetic approach for a pediatric patient with Trisomy 21, it is important to consider not only the type and duration of the procedure, but also the ability of the patient to tolerate the approach. Although general anesthesia under routine mask management may be appropriate for children with normal airways undergoing short procedures, it may be challenging for those with airway anomalies. The case report illustrates successful mask management of a patient with Trisomy 21; however, anesthesia providers must use skilled judgment when choosing alternative airways in high-risk patients. An oropharyngeal airway was used to alleviate tongue obstruction during this case. Otherwise, airway complications were minimal. Anticipated problems were prevented with a thorough preoperative physical exam and vigilant perioperative monitoring.

References

1. Mai CT, Isenburg JL, Canfield MA, et al. National population-based estimates for major birth defects, 2010-2014. *Birth Defects Res*. 2019;111(18):1420-1435. doi: 10.1002/bdr2.1589.
2. Watts R, Vyas H. An overview of respiratory problems in children with Down's syndrome. *Arch Dis Child*. 2013;98(10):812-817. doi: 10.1136/archdischild-2013-304611.
3. Bernardi GF, Pires CTF, Oliveira NP, Nisihara R. Prevalence of pressure equalization tube placement and hearing loss in children with Down syndrome. *Int J Pediatr Otorhinolaryngol*. 2017;98:48-52. doi: 10.1016/j.ijporl.2017.04.041.
4. Subramanyam R, Fleck R, McAuliffe J, et al. Upper airway morphology in Down Syndrome patients under dexmedetomidine sedation. *Rev Bras Anestesiol*. 2016;66(4):388-394. doi: 10.1016/j.bjane.2014.11.019.
5. Chamseddin BH, Johnson RF, Mitchell RB. Obstructive sleep apnea in children with Down syndrome: Demographic, clinical, and polysomnographic features. *Otolaryngol Head Neck Surg*. 2019;160(1):150-157. doi: 10.1177/0194599818797308.
6. Mahmoud M, Ishman SL, McConnell K, et al. Upper airway reflexes are preserved during dexmedetomidine sedation in children with down syndrome and obstructive sleep apnea. *J Clin Sleep Med*. 2017;13(5):721-727. doi: 10.5664/jcsm.6592.
7. Shott SR. Down syndrome: Analysis of airway size and a guide for appropriate intubation. *Laryngoscope*. 2000;110(4):585-592.
8. Bertolizio G, Saint-Martin C, Ingelmo P. Cervical instability in patients with Trisomy 21: The eternal gamble. *Paediatr Anaesth*. 2018;28(10):830-833. doi: 10.1111/pan.13481.

Mentor: Judy Koempel, CRNA

ROTEM Analysis to Guide Blood Product Replacement

Michelle Allen, MSNA
Westminster College

Keywords: Rotational thromboelastography (ROTEM), blood replacement, blood products, coagulopathy, trauma

Rotational thromboelastography (ROTEM) is a method of testing efficacy of blood coagulation. ROTEM is often used interchangeably with the term thromboelastography (TEG). ROTEM can be used to measure the strength and speed of a blood clot formation, specifically by measuring the activity of plasma coagulation pathways, platelet function and the breakdown of the clot, called fibrinolysis. It is useful in anesthesia to guide blood product replacement during surgery when there is a large volume of blood loss such as in a trauma, organ transplant or any surgical procedure where blood loss is in excess.

Case Report

A 53-year-old male was brought in to the emergency department by air transport in the early morning hours following trauma alert activation. According to air transport report he was involved in a high-speed, head-on motor vehicle accident. Upon arrival in the emergency department he was alert, oriented but somewhat repetitive in his words. He was able to verbalize that he was diabetic, and drinks alcohol. The patient reported being short of breath and having chest pain.

On exam by the emergency department trauma team, he was found to be hypotensive with decreased breath sounds bilaterally, and had a rigid and tender abdomen. A baseline complete blood count (CBC) and basic metabolic panel (BMP) were drawn and significant for a blood glucose of 463 mg/dL, base excess of -13 mEq/L, and an arterial blood gas (ABG) revealed a pH of 7.23 that indicated significant metabolic acidosis. At this point a focused assessment with sonography (FAST) was performed which was positive for hemoperitoneum. The FAST exam also revealed fluid collection, likely blood, in the Morrisons pouch, a potential space between the liver and right kidney, and fluid collection in the splenorenal junction. There was no fluid found in the pelvis or in the pericardium.

The patient was immediately taken to the operating room (OR) for possible perforation of viscous tissue or a solid organ injury. He was moved to the OR table and midazolam 5 mg was administered. Loss of consciousness was confirmed, and lidocaine 100 mg and rocuronium 50 mg were given, followed by oral endotracheal intubation. Following placement of the endotracheal tube (ETT) the patient received ampicillin/sulbactam 3 g and low expired sevoflurane anesthetic gas concentrations between 0.5% and 2.0%. After placement of a right internal jugular cordis and triple lumen central line, an arterial line was placed for continuous monitoring of blood pressure. Immediately after opening the peritoneum the surgeon evacuated 500 mL of accumulated blood. Two units of packed red blood cells (PRBC) were given through the peripheral IV and central line on the rapid infuser. Shortly after, the surgeon was asked by anesthesia to hold manual pressure on the aorta while an additional four units of blood were rapidly infused due to hypotension and hypovolemia. The blood pressure was maintained around 120/70 mm Hg after the transfusion was completed.

After infusion of six units of PRBCs an arterial sample was obtained and sent to the lab for ROTEM analysis. The A10 INTEM value came back critically low at 36 and INTEM clot formation time (CFT) 186. At that time, fresh frozen plasma (FFP) and platelets were transfused, followed by cryoprecipitate. In total, six units of PRBCs, five units FFP, one pack platelets and two units cryoprecipitate were transfused. Following evacuation of free blood from the abdomen and closure of bleeding vessels near the splenorenal junction, the abdomen was then packed and left open, with applied suction for a staged closure at a later time. The patient remained intubated and sedated postoperatively in the intensive care unit.

Discussion

There are many goals when caring for a trauma patient, including: replacing blood products, optimizing fluid blood volume, managing coagulopathies, preventing death by hemorrhage, and

later on, exsanguination. It is the anesthesia provider's role to effectively manage these complex patients with the goal of having positive postoperative outcomes. This is done by providing focused and time-sensitive life saving measures with emphasis on resuscitation after trauma based on the most up-to-date literature and practice management.[1]

The mortality rates for patients who have received a massive transfusion, commonly defined as more than 10 units of blood products in 24 hours, range between 20% to 50%.[2] Many of these patients exist in a hypocoagulable state known as coagulopathy of trauma. The coagulopathy of trauma may be due to; dilution of hemostatic factors from crystalloid administration, consumptive coagulopathy due to widespread trauma. Coagulopathy can be exacerbated by metabolic acidosis, hypothermia, hypocalcemia, and excessive break down of clots, hyperfibrinolysis.[2] Hypothermia, acidosis, and coagulopathy is termed the lethal triad of trauma. The correction of such coagulopathy should therefore begin early on in the perioperative stage to prevent what is called a "bloody vicious cycle".[3] The vicious cycle is named due to its self-perpetuating nature. The cycle is often initiated by an outside factor, like trauma or surgery, causing hemorrhage which leads to acidosis and hypothermia, both of which inhibit proper function of platelets and the coagulation cascade.[3] Coagulopathies then develop and continue to worsen as the patient continues to bleed, and thus the cycle ensues. The cycle will need to be interrupted with replacement of coagulation factors, blood products and correction of acidosis and hypothermia in order to stop the positive feedback.

Rotational thromboelastography can be useful in guiding blood and coagulation factor replacement by measuring the speed and strength of clot formation. This measures activity of plasma coagulation pathways, estimates platelet function, and fibrinolysis speed. When looking at TEG and ROTEM results, it is important to note: R time, K Time, Alpha angle, Maximum amplitude (MA) and Maximum lysis (ML).[4] R time represents the time it takes to begin a clot formation, and when prolonged, is treated with FFP. K time, or clot formation time, shows the time it took for the clot to achieve a fixed strength, and is corrected with cryoprecipitate. An alpha angle shows the speed of fibrin formation, and when an acute alpha angle is noted, it should also be corrected with cryoprecipitate. An A10 time shows the amplitude, or strength of the blood clot 10 minutes after clotting time, and a MA is usually measured 20 minutes after clotting time. A low amplitude or decreased clot strength is best treated with platelets.[5]

The interpretation of the ROTEM values during this case is what led anesthesia to decide which blood products to transfuse following replacement of PRBCs to correct blood volume. After a blood sample was sent to the lab showing a prolonged K time or prolonged clot formation time cryoprecipitate was then transfused. Likewise, when the A10 time was reported to be critically low at 36 the patient the received a platelet transfusion. Treatment goals of platelets and cryoprecipitate will help the patient to begin faster and stronger clot formation, to decrease bleeding, interrupt the vicious cycle of coagulopathy, acidosis, and hypothermia.

In this case, the patient remained sedated and was transferred to intensive care after a computed tomography (CT) scan of the head. His hemoglobin and hematocrit values were monitored postoperatively, but unfortunately, a repeat ROTEM sample was not obtained before transferring the patient to the intensive care to evaluate the impact of giving platelets, FFP and cryoprecipitate. His stay in the hospital included two more staged laparotomies and eventual

closure of the abdomen. He was eventually able to be transferred out of intensive care to a step-down care unit.

References

1. McCunn M, Dutton R, Dagal A, et al. Trauma, Critical Care, and Emergency Care Anesthesiology: A New Paradigm for the "Acute Care" Anesthesiologist? *Anesth Analg*. 2015;121(6):1668-1673. doi:10.1213/ANE.0000000000000782.
2. Borgman MA, Spinella PC, et al. The Ratio of Blood Products Transfused Affects Mortality in Patients Receiving Massive Transfusions at a Combat Support Hospital. *J Trauma*. 10AD;63(4):805-813. doi:10.1097/TA.0b013e3181271ba3.
3. Holcomb JB, Jenkins D, et al. Damage Control Resuscitation: Directly Addressing the Early Coagulopathy of Trauma. *J Trauma.* 2007;62(2):307-310. doi:10.1097/TA.0b013e3180324124.
4. Thiruvenkatarajan V, Pruett A, Adhikary SD. Coagulation in the perioperative Period. *Indian J Anaesth*. 2014;58(5):565-572. doi: 10.4103/0019-5049.144657.
5. Whiting D, DiNardo JA. TEG and ROTEM: Technology and clinical applications. *Am J Hematol.* 2014;89(2):228-232 doi: 10.1002/ajh.23599.

Mentor: James Stimpson, DNP, CRNA

Anesthetic Management of the Vasculopathic Patient in the Prone Position

Brad Goll, MS
Westminster College

Keywords: Hemodynamic collapse, vasculopathy, METS <4, mitral regurgitation, prone position

Although spine surgery is a common occurrence in operating rooms across the nation, accompanying pathology can complicate this procedure. Patients with vasculopathy can be challenging to manage from an anesthesia perspective. Orthopedic spinal surgery places patients at an intermediate or 1-5% risk of having a perioperative myocardial infarction (MI).[1] Vasculopathic patients often have significant hemodynamic instability perioperatively. Spinal surgery requires prone positioning, which can exacerbate instability in these patients. In this case report, mitral regurgitation further increased the risk of perioperative morbidity and mortality and may have contributed to severe intraoperative hypotension that ultimately led to case cancellation.

Case Report

A 65-year-old, 89 kg, 157 cm female presented for a lumbar 4-5 decompression and posterior spinal fusion. Past medical history included: gastroesophageal reflux disease, type 2 diabetes, chronic obstructive pulmonary disease, asthma, 42-year history of tobacco smoking, chronic renal insufficiency, hypertension, coronary artery disease (CAD), multiple myocardial

infarctions (MI), and peripheral artery disease (PAD). The transthoracic echocardiogram (TTE) from four months prior, showed an estimated ejection fraction of 55% with moderate mitral regurgitation. The patient experiences shortness of breath when climbing any amount of stairs and her which estimates her metabolic equivalent of tasks (MET) as less than four. An electrocardiogram (ECG) showed normal sinus rhythm. Relevant past surgical history included: multiple percutaneous coronary interventions with stent placement, 4-vessel coronary artery bypass grafting in 2017, and iliac artery stenting.

The patient was transported to the operating room (OR) where standard noninvasive monitors were applied, and O_2 10 L/min was administered via a face mask. Pre-induction vital signs were blood pressure 110/61 mmHg, heart rate 92 beats/min, and SpO_2 99%. After pre-oxygenation, general anesthesia was induced on the patient's stretcher with intravenous (IV) sufentanil 2 mcg, lidocaine 100 mg, propofol 150 mg, and rocuronium 50 mg. The trachea was atraumatically intubated with a 7.0 mm cuffed endotracheal tube (ETT), and a mechanical ventilator delivered ventilation. General anesthesia was maintained with sevoflurane set to 2% inspired concentration in a mixture of air 1 L/min and O_2 1 L/min. A left radial arterial line was placed in a sterile fashion. The patient was maintained within 15% of baseline vital signs up to this point.

Upon moving the patient to the prone position on the OR table, their blood pressure decreased abruptly to 66/45 mm Hg per the arterial line, and the heart rate increased to 115 beats/min. The end-tidal sevoflurane concentration was 0.9% at the time and was discontinued. Boluses totallinging 700 mcg of phenylephrine, 6 units of vasopressin, and 1 liter of lactated ringers were administered IV over the next 10 10 minutes with minimal increase in blood pressure. The patient's skin became progressively mottled, and the decision to cancel surgery at this point in the resuscitation was determined. The patient was promptly returned to the supine position, and neuromuscular blockade was antagonized with sugammadex 200 mg IV.

With the patient in the supine position, the patient's hemodynamics stabilized with blood pressure 110/80 mm Hg and heart rate 85 beats/min. The patient emerged from anesthesia. Once extubation criteria were met, the ETT was removed. The patient recovered in the post-anesthesia recovery unit without further incident.

Discussion

Vasculopathy is an umbrella term for diseases affecting the blood vessels. These vascular abnormalities may be caused by genetic, degenerative, metabolic, or inflammatory conditions. Patients with a systemic vascular disease such as PAD often have concomitant heart disease, because the cardiac vessels are also compromised.[1] Those with PAD should be assumed to have CAD as well, and perioperative hemodynamic instability should be anticipated. The causes of PAD, the most common vasculopathy in the United States, include smoking, diabetes, hypertension, obstructive sleep apnea, and autoimmune diseases. PAD and other vascular conditions most commonly involve the aortic, coronary, cerebral, femoral, and iliac arteries. The consequences of severe vascular disease include stroke, MI, renal failure, and pulmonary emboli. The preoperative evaluation of the vasculopathic patient should include both the causes and consequences of their disease process.[2] This patient's vascular disease was extensive, as

evidenced by previous myocardial infarctions , iliac artery stenting, and a four4-vessel coronary bypass operation.

When interviewing patients preoperatively, it is advisable to estimate the patient's MET. Patients that are unable to climb a flight of stairs without developing signs of cardiac ischemia are considered to have a MET <4. MET<4 puts the patient at a significantly higher risk of perioperative morbidity and mortality[3]. An assessment of MET <4 should prompt further cardiac workup in patients undergoing non-cardiac surgery.[4] Adam et al[4] conducted a study comparing MET estimated by anesthesia providers to actual quantitative stress test MET. The study found that anesthesia providers underestimate MET 91% of the time. Estimating MET to be lower than it is can be beneficial as it triggers a more extensive cardiac workup with greater frequency. In this case, the patient had a TTE 4 months prior, 12 lead EKG within 6six weeks, as well as a visit with her cardiologist to determine preoperative optimization.

Moderate mitral regurgitation was assessed in this patient during preoperative TTE. Hemodynamic goals for a patient with mitral regurgitation are to maintain forward blood flow through the heart chambers. Forward flow is achieved by maintaining a normal preload, a normal to relatively tachycardic heart rate, and a reduced afterload. These interventions prevent overdistension of the ventricles and maintain effective ejection of blood.[5] It is essential to keep the patient euvolemic, as excessive preload will raise pulmonary artery pressures quickly in the setting of mitral regurgitation. In already vasculopathic patients, a rise in pulmonary artery pressures may result in pulmonary edema and progressive heart failure.

The prone position puts abnormal pressure on anterior structures of the body. Pressure on the abdomen raises intra-abdominal pressure, thereby limiting venous return to the heart and increasing intrathoracic pressure during ventilation. The increased intrathoracic pressure decreases ventricular compliance, which limits ventricular stroke volume and, consequently, cardiac output due to the Frank-Starling law.[6] These pressure changes and reduced cardiac output are responsible for the hypotension that may be seen when a patient is moved from supine to prone position. Combined with a pathologic cardiovascular system, this can quickly lead to cardiovascular collapse.

The combination of multiple pathologies and positioning can synergize to create a very unstable patient under general anesthesia. Vasculopathy, mitral regurgitation, MET <4, and prone positioning are all likely to cause intraoperative hypotension. Whenever multiple morbidities are likely to produce the same effect, in this case,, hypotension, the anesthesia provider must remain vigilant and take extra precautions. These precautions include preoperative optimization of the patient's cardiovascular system, placement of invasive monitors to identify and treat the anticipated hypotension quickly, as well as vasopressors prepared ahead of time. In this case, the combination created a nearly catastrophic hemodynamic collapse. While even with proper preparation, the patient could not tolerate the procedure, they were safely emerged from anesthesia without suffering major perioperative complications.

References

1. Nagelhout J. Autonomic and cardiac pharmacology. In: Nagelhout J, Sass E, eds. N*urse Anesthesia*. 6th ed. St. Louis, MI: Elsevier; 2018:182-188.
2. Neves S. Anesthesia for patients with peripheral vascular disease and cardiac dysfunction. *Anesthesiol Clin*. 2016;34(4):775-795. doi:10.1016/j.anclin.2016.06.011
3. Fleisher L, Fleischmann K, Auerbach A et al. 2014 ACC/AHA Guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery. *Circulation.* 2014;130(24). doi:10.1161/cir.0000000000000106
4. Weinstein A, Sigurdsson M, Bader A. Comparison of preoperative assessment of patient's metabolic equivalents (METs) estimated from history versus measured by exercise cardiac stress testing. *Anesthesiol Res Pract*. 2018;2018:1-5. doi:10.1155/2018/5912726
5. Paul A, Das S. Valvular heart disease and anaesthesia. *Indian J Anaesth*. 2017;61(9):721–727. doi:10.4103/ija.IJA_378_17
6. Kwee MM, Ho YH, Rozen WM. The prone position during surgery and its complications: a systematic review and evidence-based guidelines. *Int Surg*. 2015;100(2):292–303. doi:10.9738/INTSURG-D-13-00256.1

Mentor: James Stimpson, DNP, CRNA

An Unusual Case of Delayed Emergence after Bronchoscopy

Rebecca Tuthill, DNAP
Yale New Haven Hospital School of Nurse Anesthesia

Keywords: delayed emergence, delayed awakening, VAE, PRES, postictal

Delayed emergence from anesthesia is defined as the failure to regain consciousness within 30-60 minutes after the surgical procedure.[1] Failure to awaken after general anesthesia may indicate several life-threatening disease states. Therefore, the potential for patient harm necessitates rapid diagnosis and treatment to avoid complications. This case report describes an incident of delayed emergence of unknown etiology after bronchoscopy in a patient with numerous comorbidities. Vascular air embolism (VAE), posterior reversible encephalopathy syndrome (PRES), and postictal state are considered as the differential diagnoses.

Case Report

A 59-year-old woman presented as an outpatient to the bronchoscopy suite after several months of moderate shortness of breath and wheezing. Her chest x-ray demonstrated extensive mediastinal adenopathy, right lower lobe ground-glass opacity, and hilar lymphadenopathy, concerning for sarcoidosis. She underwent bronchoscopy with bronchoalveolar lavage, transbronchial biopsies, and lymph node fine-needle aspirates (FNA). Her medical history included end-stage renal disease (ESRD) secondary to lupus nephritis and requiring hemodialysis (last dialysis was the day prior), epilepsy with status epilepticus, recurrent intraparenchymal hemorrhage (IPH) secondary to cerebral amyloid angiopathy with residual

right hemiparesis, hypercholesterolemia, hypertensive emergency, pulmonary hypertension, diabetes mellitus, anemia, renal artery stenosis, poorly controlled gastroesophageal reflux disease (GERD), hypothyroidism, and interstitial lung disease. Home medications included levetiracetam, lacosamide , and pregabalin. Preoperative physical examination was significant for lethargy and memory impairment. The patient was a poor historian and unable to recall when her anti-epileptic drugs (AEDs) were last taken. Her vital signs were stable with an SpO_2 of 93% on room air. Electrocardiogram (ECG) demonstrated normal sinus rhythm with a right bundle branch block (RBBB). The patient's laboratory results were unremarkable.

After the application of standard monitors, O_2 10 L/min was administered via face mask for 3 minutes prior to induction. Rapid sequence induction was achieved with midazolam 1 mg, fentanyl 50 mcg, and propofol 100 mg, followed by succinylcholine 100 mg intravenous (IV). The patient was intubated atraumatically with an 8.0 mm endotracheal tube (ETT); placement was confirmed via sustained capnography and auscultation. General anesthesia was maintained with propofol 150 mcg/kg/min, fentanyl 50 mcg, intermittent boluses of succinylcholine (75 mg total). The procedure was complicated by a brief period of bleeding during excision of a right upper lobe lesion causing a small mucosal tear. At this time, the patient experienced a 3-minute period of hypotension and hypoxia (MAP 50-60 mm Hg; SpO_2 79-92%. During this period of hypotension and hypoxia, the patient developed transient ST elevation and had a short, self-limited run of supraventricular tachycardia. After the patient demonstrated spontaneous respiration, adequate tidal volumes, and opened eyes to vocal commands, the ETT was removed and she was transferred to the post-anesthesia care unit (PACU).

While in the PACU, the patient's systolic blood pressure increased to 205 mm Hg and she became unarousable. Hydralazine 10 mg IV was administered and cardiology was consulted. An ECG revealed a submillimeter depression in lead V2 and a known RBBB. The patient was admitted for observation to trend troponins for suspected demand ischemia. The patient was noted to have a deteriorating neurologic exam: eyes open to noxious stimuli, inability to follow commands, roving eye movement with no apparent gaze deviation, absent motor response in the upper extremities and spontaneous movement of lower extremities. In an effort to rule out midazolam administration as the cause of delayed emergence, flumazenil 0.2 mg IV was administered with no change in condition. Since the prolonged sedative effect of midazolam was ruled out as a differential diagnosis and given the concern for the presence of non-convulsive seizures, lorazepam 2 mg IV was administered which also had no effect. Neurology was consulted due to the patient's prior history of hemorrhagic cerebrovascular accident (CVA) and epilepsy. A head computed tomography angiography (CTA) was performed and intracranial hemorrhage was ruled out. The patient was transferred to the emergency department (ED) to be triaged to the appropriate inpatient setting.

In the ED, the patient's mental status continued to deteriorate. Levetiracetam 3 g was administered and the patient was reintubated for poor mental status and airway protection. She was taken for a repeat CTA and admitted to the neurological intensive care unit for continuous electroencephalogram (cEEG). Her cEEG demonstrated abundant periods of high amplitude, very sharp theta/delta activity that suggested cortical hyperexcitability. These symptoms improved with lacosamide and levetiracetam doses; however, no seizures were noted. Her neurological exam returned to baseline by postoperative day (POD) 2. She was extubated,

transitioned to room air and transferred to the medical floor on POD 3. Serial chest x-rays were performed over her 3-day admission, demonstrating diffuse infiltrates consistent with persistent pulmonary edema.

Discussion

This case report describes a patient who underwent general anesthesia for bronchoscopy and developed delayed emergence which deteriorated to a comatose state. The patient received an appropriate depth of anesthesia, and opioids were not administered in excessive doses. Though midazolam may potentiate prolonged sedation in patients with ESRD, pharmacologic reversal with flumazenil did not result in emergence.[2] Therefore, the most probable etiology of delayed emergence was postictal state, PRES, or VAE. The initial precipitating events causing hypoxemia, hypotension, tachyarrhythmia, and ST-segment changes intraoperatively could have been the result of a VAE.

Air embolus is a rare but potential complication in fine-needle aspiration (FNA) during bronchoscopy. There is a wide spectrum of issues associated with VAE, including numerous neurologic symptoms such as confusion, visual disturbances, and delayed emergence from anesthesia.[3] The literature suggests that air may enter the pulmonary system through the needle.[3] If atmospheric pressure is higher than pulmonary venous pressure, air emboli can transverse down the pressure gradient and enter the pulmonary veins.[3] This is more likely to occur if the patient were to inhale deeply during the procedure.[3] Second, if the internal airway pressure distal to the bronchoscope was to rise, the risk of embolism may increase.[2] This may occur with coughing and straining during the procedure, which creates a sudden pressure increase distal to the needle.[3] Therefore, increasing the depth of anesthesia in patients showing signs of airway resistance (e.g. coughing, deep breathing) may reduce the risk of embolus.[3]

Though diagnosis of an air embolism is primarily based on clinical presentation, a definitive diagnosis is accomplished by brain CT scan demonstrating air bubbles in the cerebral vessels, aorta, pulmonary veins, and left atrium and ventricle.[3] However, an air embolism less than 1.3 cm may not be detected by CT or CTA and will only be seen on magnetic resonance imaging (MRI).[3] The patient received several CTAs to rule out hemorrhage but no VAE was noted on imaging. However, the patient did not undergo an MRI which may have helped to definitively rule out or diagnose VAE as the causative factor. Therefore, if a patient has clinical symptoms that allude to CVA, it is imperative to obtain definitive exclusion criteria by obtaining a CT or CTA to detect air embolism, followed by an MRI if no air embolism is noted on the CTA.[3]

Anesthetic care for a patient with a history of epilepsy should focus on avoiding anesthetics with proconvulsant effects (e.g. nitrous oxide, methohexital, ketamine, etomidate, and meperidine).[4] Fortunately, none of these anesthetics were administered. One risk factor for developing an intraoperative seizure was that the patient may not have taken her AEDs on the morning of surgery. Patients must continue these medications preoperatively because it is essential to avoid disruption of antiepileptic medication perioperatively.[4] Recommendations for missed dosing are described below in Table 1.

Table 1. Management of Differential Diagnoses for Delayed Emergence After Bronchoscopy

Differential Diagnosis	Presentation	Prevention and Treatment
VAE	Neurologic: confusion, personality change, dizziness, visual disturbance, paresthesia, focal neurologic deficits, delayed recovery from anesthesia[3] CV: cardiovascular instability[3]	● Detection: TEE or esophageal doppler[3] ● Hyperbaric oxygen therapy with 100% oxygen[3] ● Left lateral decubitus position[3] ● Trendelenberg[3] ● CT or CTA to rule out VAE; MRI if no VAE noted on CT or CTA[3]
PRES	CV: abrupt hypertension Neurologic: seizures, acute neurologic deterioration, delayed emergence from anesthesia[5] Risk Factors: History of autoimmune disease with immunosuppressant use and ESRD Secondary Complications: status epilepticus, intracranial hemorrhag massive ischemic infarction[5]	● Gradual blood pressure control[5] ● CT to observe for presence of vasogenic edema (most commonly in the parieto-occipital lobes)[5]
Non-convulsive Seizures	Neurologic: Impaired consciousness[4] Risk Factors: history of epilepsy, structural brain abnormality[4]	● **Prevention:** Avoid seizure provoking anesthetics[4] ● If single dose of AEDs missed, administer as soon as possible after surgery[4] ● If multiple doses of AEDs missed, administer parenterally[4] ● If multiple doses of AEDs missed and AEDs not available in parenteral form, consult neurology[4] ● **Detection:** Continuous electroencephalography (cEEG)[3] ● **Treatment:** Rapidly acting AEDs such as diazepam[4]

Another consideration in the differential diagnosis of delayed emergence from anesthesia is PRES. Posterior reversible encephalopathy syndrome refers to a disorder of reversible subcortical vasogenic brain edema in patients with acute neurological symptoms in the setting of renal failure, blood pressure fluctuation, and autoimmune disorders.[5] It is often, associated with acute onset hypertension.[5] Brain imaging usually reveals vasogenic edema predominantly involving the bilateral parieto-occipital regions.[5] Patients typically present with encephalopathy, seizures, headache, visual disturbances, focal neurological deficits, and status epilepticus.[5] The treatment for PRES is supportive, but the disorder is usually reversible with rapid control of the triggering cause (e.g. hypertension).[5,6] While PRES was considered due to the patient's acute neurologic deterioration, history of ESRD, lupus, and pronounced blood pressure fluctuations, she was never conclusively diagnosed based on the absence of vasogenic edema on her CTA.

Lastly, air embolism should be included in the differential diagnosis of patients with delayed emergence after bronchoscopy, particularly for those who underwent FNA.[3] Hyperbaric oxygen therapy is considered the gold standard for treatment of systemic air embolism because it increases the ambient pressure, thereby compressing the air embolism and decreasing its size.[3] Patients suspected of having a VAE should be placed in the left lateral decubitus position with trendelenburg position.[3] Other interventions include positive pressure ventilation, administration of FiO_2 1.0 and cardiovascular support until the embolus dissipates.[3]

The etiology of delayed emergence can be difficult to diagnose as it may be multifactorial and stem from one or a combination of patient comorbidities, anesthetic agents, pharmacologic interventions, and intraoperative complications. Though PRES and postictal state were considered in the differential diagnosis of this patient, VAE was not excluded and a definitive diagnosis was never determined.

References

1. Maeda S, Tomoyasu Y, Higuchi H, Ishii-Maruhama M, Egusa M, Miyawaki T. Independent predictors of delay in emergence from general anesthesia. *Anesth Prog.* 2015;62(1):8-13.
2. Campbell JP, Cousins JM. Anesthesia for dialysis patients. Post TW, ed. *UpToDate*. Waltham, MA: UpToDate Inc. https://www.uptodate.com (Accessed July 29, 2019).
3. Almas ET, Casserly B. Air embolism following bronchoscopy with fine needle aspiration: An unexpected complication. *Resp Med Case Rep*. 2018; 25:228–232. doi:10.1016/j.rmcr.2018.09.012
4. Perks A, Cheema S, Mohanraj R. Anaesthesia and epilepsy, *BJA*. 2012; 108(4):562–571.
5. Fugate JE, Rabinstein AA. Posterior reversible encephalopathy syndrome: clinical and radiological manifestations, pathophysiology, and outstanding questions. *Lancet Neurol.* 2015;14:914.
6. Hobson, E, Craven, I, and Catrin, S. Posterior reversible encephalopathy syndrome: a truly treatable neurologic illness. *Perit Dial Int*. 2012; 32:590–594. doi: 10.3747/pdi.2012.00152
7. Benoist G, Dossier C, Elmaleh M, et al. Posterior reversible encephalopathy syndrome revealing renal artery stenosis in a child. *BMJ Case Rep* Published online: 2013 Aug 23. doi:10.1136/ bcr-2013-010110
8. Kanchustambham V, Saladi S, Mehta K, Mwangi J, Jamkhana Z, Patolia S. Vascular Air Embolism During Bronchoscopy Procedures- Incidence, Pathophysiology, Diagnosis, Management and Outcomes. *Cureus*. 2017;9(3):e1087. Published 2017 Mar 9. doi:10.7759/cureus.1087

Mentor: Marianne S. Cosgrove, PhD, DNAP, CRNA

Methadone for Postoperative Analgesia in Complex Spinal Surgery

Brittney Bucholtz, DNAP
University of Southern California

Keywords: methadone, chronic pain management, anesthesia, spine surgery, spinal fusion

Severe pain is frequently experienced by the post-operative spinal fusion surgery patient within the first seventy-two hours.[1] Methadone, a synthetic opioid used for the treatment of chronic pain and detoxification of opioid addiction, is well-known for its long duration of action and effectiveness in complex spinal surgeries. Methadone helps to minimize intraoperative and postoperative opioid requirements. A clear understanding of the pathophysiology, in addition to the adjuvant treatment options of methadone, can guide the anesthesia practitioner to develop a successful treatment regimen and improve patient outcomes.

Case Report

A 70-year-old, 70kg, 170cm female presented for a complex, multilevel spinal fusion surgery with a diagnosis of cervical kyphosis, pseudoarthritis, and spondylolisthesis. Past medical history included opioid dependence/chronic pain syndrome and Sjogren's syndrome. The patient's home medications consisted of fentanyl 25 mcg/hr transdermal patch for 72 hours, metaxalone 800 mg twice daily, gabapentin 600 mg every four hours prn, and oxycodone/acetaminophen 5/325 mg every six hours prn. Based on this home medication regimen, a morphine equivalent dose (MED) of 90 was generated from the hospital's electronic health record. Per institutional policy, MED scores are used to translate patient doses and routes of opioids consumed within twenty-four hours to reduce the risk of overdose.

Upon initial consult in the preoperative setting, the patient presented anxious, tearful, and reported 10/10 back pain. The patient had not taken her home medications, except for a fentanyl transdermal patch, which was placed 24 hours before surgery. After performing preoperative anesthesia assessment and obtaining anesthesia consent, gabapentin 600 mg and acetaminophen 1000 mg were administered orally. The patient was then premedicated with intravenous methadone 10 mg, which decreased her pain score to 7/10. The patient was given an additional intravenous methadone 10 mg bolus 20 minutes later, which further decreased her pain to a reported tolerable level of 4/10. The transdermal fentanyl patch was removed, and the patient was transferred to the operating room.

Standard noninvasive monitors were applied, and the patient was preoxygenated with O_2 10L/min for 5 minutes. General anesthesia was induced with intravenous propofol 140 mg, ketamine 50 mg, rocuronium 50 mg, and esmolol 40 mg. Successful endotracheal intubation with a 7.0 mm endotracheal tube was achieved by elective fiberoptic bronchoscopy and confirmed by positive $ETCO_2$ and bilateral breath sounds. Volume control ventilation was initiated with a rate of 12/min, tidal volume 480 mL, and positive end-expiratory pressure 5 cm H2O. Sterile arterial cannulation was performed on the right radial artery, and an additional 16 gauge IV was placed in the right forearm. A bispectral index monitor was applied and the patient was turned prone. Somatosensory monitoring was initiated and indicated neuromuscular blockade had resolved; no

additional neuromuscular blocking agents were administered throughout the remainder of the case. General anesthesia was maintained with sevoflurane 0.5% inspired concentration in a mixture of O_2 1 L/min and air 1 L/min, and a propofol infusion was started at 50 mcg/kg/min and titrated to 200 mcg/kg/min as needed. Methadone 5 mg IV was administered prior to incision, and additional 5 mg doses were given in 30-45 minute increments to total 40 mg. The patient received Robaxin 500 mg IV 30 minutes prior to extubation. Neuromuscular blockade was antagonized with sugammadex 140 mg IV, evidenced by four twitches in response to neuromuscular stimulation and sustained tetany greater than five seconds. The patient's trachea was extubated when awake and she was then transferred to the post-anesthesia care unit on O_2 6L/min via simple face mask. She denied pain or nausea, and vital signs were stable and within preoperative parameters. The case duration totaled three and a half hours.

Discussion

Severe pain following spinal fusion surgery remains a common yet overlooked problem within the first three postoperative days.[1] Despite advances in surgical techniques and multimodal approaches to anesthetic pain management, existing chronic neuropathic pain and opioid dependence add complexity to these challenging cases.[1,2] Inappropriate pain management for spinal operations poses consequences such as increased morbidity risk and hospital length of stay, pulmonary and cardiovascular complications, and development of chronic and severe pain.[3,4] Anesthesia practitioners must be well-informed about the chronic effects of opiates such as receptor desensitization and downregulation, and development of tolerance and physical dependence after long-term exposure to opiates.[4]

Methadone was first discovered in 1946 and has gained popularity in the perioperative setting due to its favorable properties compared with other opioids.[5] Methadone is a potent mu-opioid receptor agonist that has the longest half-life of all opioids, lasting 24 hours in opioid-dependent patients and approximately 55 hours in opioid naive patients.[5,6] N-methyl-D-aspartate (NMDA) receptors are responsible for the development of opioid tolerance, hyperalgesia and chronic pain.[1] Methadone inhibits NMDA receptors and contains antihyperalgesic and antiallodynic properties that prevent the development of tolerance, and has been useful in the management of chronic, neuropathic pain.[1] Additionally, methadone acts on the central nervous system to decrease serotonin and norepinephrine reuptake which may contribute to perceptual dimensions of pain processing.[1] Possible side effects of methadone may include nausea, vomiting, dizziness, lethargy, bradycardia and respiratory depression.[6] Respiratory depression is the most concerning side effect for the perioperative patient.[6]

Innovative studies that pioneered the use of methadone in perioperative settings demonstrated methadone 20 mg IV following induction of anesthesia for orthopedic and general surgeries significantly reduced postoperative pain. Of the patients observed, none experienced postoperative respiratory depression, defined by a rate less than 10 breaths/min, or postoperative nausea/vomiting.[3] In 2017, Murphy et al. conducted a randomized, double-blinded, controlled trial that studied 115 patients undergoing spinal fusion surgery.[1] The authors aimed to determine whether methadone 0.2 mg/kg at the start of surgery or hydromorphone 2 mg upon surgical closure provided better pain relief through postoperative day three.[1] The patients receiving intravenous methadone required less intravenous and oral opioid requirements, and reported less postoperative pain and improved satisfaction.[1] No differences among opioid related adverse

effects were found when comparing hydromorphone and methadone; methadone study groups were not found to be at increased risk of respiratory depression.[1] Additionally, Gottschalk et al. conducted a prospective randomized single-blinded study to explore the efficacy of a single dose of methadone prior to surgical incision in twenty-nine patients presenting for multilevel complex spinal surgery.[2] The authors concluded significant reduction in post-surgical pain and opioid requirements up to 72 hours in patients receiving methadone 20-30 mg.[2]

Future anesthetic recommendations for dosing methadone may be better understood by the term "equianalgesia," which refers to varying doses among different analgesics that provide similar pain relief.[3,4] Although limited recent studies exist that guide methadone dosing, equianalgesic tables may serve as useful guidelines. Equianalgesic tables convert oral and/or transdermal opioid doses to corresponding intravenous doses and calculate a patient's cumulative opioid consumption within 24 hours.[7] For example, fentanyl 25 mcg/hr transdermal patch is equivalent to morphine 20 mg IV, and guidelines from 2001 highlight fentanyl 25 mcg/hr transdermal patch is equivalent to methadone 9-14 mg IV.[4,8] Of note, recommendations suggest fentanyl patches be removed prior to major surgeries and equianalgesic doses of opioid be given.[4] Calculation of opioid doses based solely on equianalgesic tables is an oversimplification of pain management that poses an increased risk for adverse consequences if not closely monitored. Equianalgesic tables should not outweigh interpatient variability, clinical experience, and practitioner opinion. Based upon the patient's preoperative history, MED score, and practitioner experience, 40 mg methadone IV was administered perioperatively. A 24-hour post-anesthesia follow-up revealed the patient reported minimal pain levels without respiratory depression.

Without appropriate and meticulous management, complex spinal fusion surgery may lead to profound pain that has been shown to be most severe within the first three postoperative days.[1] By understanding and employing aggressive adjunct treatment regimens, the anesthesia practitioner may better manage postoperative pain for this surgical patient population. Methadone, when used judiciously, may improve patient satisfaction, minimize further opioid requirements, and provide adequate analgesia up to 72 hours postoperatively.

References

1. Murphy GS, Szokol JW, Avram MJ, et al. Clinical effectiveness and safety of intraoperative methadone in patients undergoing posterior spinal fusion surgery: a randomized, double-blinded, controlled Trial. *Anesthesiology*. 2017;126(5):822-833. doi:10.1097/aln.0000000000001609.
2. Gottschalk A, Durieux ME, Nemergut EC. Intraoperative methadone improves postoperative pain control in patients undergoing complex spine surgery. *Anesth Analg*. 2011;112(1):218-223. doi:10.1213/ANE.0b013e3181d8a095.
3. Kharasch ED. Intraoperative methadone: rediscovery, reappraisal, and reinvigoration?. *Anesth Analg*. 2011;112(1):13-16. doi:10.1213/ANE.0b013e3181fec9a3.
4. Coluzzi F, Bifulco F, Cuomo A, et al. The challenge of perioperative pain management in opioid-tolerant patients. *Ther Clin Risk Manag*. 2017;2017(13):1163-1173. doi:10.2147/TCRM.S141332.
5. Murphy, G, & Szokol, JW. Use of Methadone in the Perioperative Period. *The Official Journal of the Anesthesia Patient Safety Foundation*. 2018;32(3):81-82.

https://www.apsf.org/article/use-of-methadone-in-the-perioperative-period. Accessed August 19, 2020.
6. Grissinger M. Keeping patients safe from methadone overdoses. *P T*. 2011;36(8):462-466. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC317182. Accessed August 19, 2020.
7. Knotkova H, Fine PG, Portenoy RK. Opioid rotation: the science and the limitations of the equianalgesic dose table. *J Pain Symptom Manage*. 2009;38(3):426-439. doi:10.1016/j.jpainsymman.2009.06.001.
8. Pereira J, Lawlor P, Vigano A, Dorgan M, Bruera E. Equianalgesic dose ratios for opioids. a critical review and proposals for long-term dosing. *J Pain Symptom Manage*. 2001;22(2):672-687. doi:10.1016/s0885-3924(01)00294-9.

Mentor: Robert A. Olson, DNAP, CRNA

Anesthetic Considerations for a Patient with Brugada Syndrome

Hai Ly, MSNA
Westminster College

Keywords: Brugada syndrome, sudden cardiac death, cardiac conduction abnormality, arrhythmias, anesthetic management, dysrhythmia

Brugada syndrome (BS) is an arrhythmogenic disease that is associated with persistent ST segment elevation, right bundle branch block, and sudden cardiac death due to ventricular arrhythmias.[1] This disease is one of the leading causes of cardiac death in patients under the age of 40 years.[2] The administration of general anesthesia may precipitate a malignant arrhythmia due to several factors.[3] This case report discusses the anesthetic management of a patient with known Brugada syndrome for laparoscopic inguinal hernia repair.

Case Report

A 63-year-old, 178 cm, 73 kg Caucasian male presented for a laparoscopic inguinal hernia repair. His past surgical history included a left total knee replacement in 2015 under general anesthesia, with no reported complications. His past medical history included BS and gastroesophageal reflux disease, which was well-controlled with famotidine. During the preoperative interview, the patient stated that he was diagnosed with BS in the year 2002 during a routine physical that included a 12-lead EKG, which was required for him to obtain life insurance through his employer. The patient stated that he had one episode of syncope when he was in his 30's, but did not follow-up with a healthcare provider about it because he just thought he over-exerted himself while exercising. Other than this one episode of syncope, the patient denied ever having problems with dizziness, seizures or any other syncopal episodes.

Preoperative vital signs were stable with a heart rate of 71 beats/min, blood pressure 132/82 mm Hg, respiratory rate 10/min, temperature 37°C, and SpO_2 100% on room air. A baseline 12-lead EKG was obtained the day of surgery which demonstrated a right bundle branch block and ST segment elevation in leads V1 to V3. Upon collaboration with other anesthesia practitioners, the

decision was made to continue with surgery with a specific anesthetic plan aimed at avoiding medications that could cause arrhythmias.

Upon arrival to the operating room, non-invasive blood pressure, continuous 5-lead EKG, and SpO_2 monitors were applied to the patient. Additionally, external defibrillator pads were applied to the patient, with a cardiac defibrillator readily available. General anesthesia was induced with midazolam 2 mg, fentanyl 100 mcg, and propofol 150 mg. After successful bag-mask ventilation, rocuronium 50 mg was administered. The patient was intubated with a 7.5 mm oral endotracheal tube (ETT) using a Miller 2 blade. After verification of the ETT placement using capnography and auscultation of bilateral breath sounds, the mechanical ventilator was set to pressure-controlled ventilation. Fresh gas flows were adjusted to a mixture of O_2 at 1 L/min and air at 1 L/min, with sevoflurane 2.4% expired concentration. Before initiation of the pneumoperitoneum, the surgeon was reminded to apply insufflation slowly to minimize the risk of arrhythmias and hemodynamic instability.

Following completion of the surgical laparoscopic inguinal hernia repair, sugammadex 150 mg was administered to reverse paralysis. The peripheral nerve stimulator elicited a 4/4 train-of-four response with sustained tetany after the administration of sugammadex, the patient was spontaneously breathing and obeyed commands. The ETT was removed and O_2 at 6 L/min via simple face mask was applied upon transfer to the post-anesthesia care unit (PACU). In PACU, his vital signs were stable and he was alert and oriented with no complaints of shortness of breath, pain, or palpitations.

Discussion

Brugada syndrome (BS) is a genetic disease that affects the cardiac conduction of the heart, putting those with BS at increased risk of arrhythmias and sudden cardiac death.[5] BS wasn't clinically described until 1992, when cardiologist brothers from Spain, Pedro and Josep Brugada, first introduced their findings.[4] The prevalence of BS is rare, occurring in about five to 20 out of every 10,000 people worldwide.[2] This syndrome is seen more frequently in males and in populations from Asian and Southeast Asian countries, especially Thailand, Philippines, and Japan, where BS is considered endemic.[4] The onset of arrhythmic events is usually observed around 40 years of age, however there are reports of BS being initially observed in patients as young as two years old and as old as 77 years old.[3]

The diagnosis of BS is made based on both characteristic EKG findings and clinical criteria.[4] Looking at precordial leads V1 to V3, a coved-shaped ST segment elevation followed by a negative T-wave is one of the indications of BS.[4] Additionally, the diagnosis of BS requires at least one of the following criteria: a family history of BS, arrhythmia-related symptoms (syncope or seizures), documented ventricular arrhythmias, or inducibility of ventricular tachycardia with programmed electrical stimulation.[4]

Currently, the only proven effective treatment of BS is an implantable cardioverter defibrillator (ICD). [4] Placement of ICDs are typically reserved for patients who are symptomatic and at higher risk of sudden cardiac death.[4] For all BS patients, whether symptomatic or not, lifestyle changes are recommended including avoiding use of medications or drugs that may induce ventricular

arrhythmias, immediate treatment of fevers, and avoiding excessive alcohol and consumption of heavy meals.[1] Quinidine, an oral antiarrhythmic drug, may be an alternative treatment for patients who refuse or have a contraindication for ICD implantation.[1]

In patients with BS, there is a mutation in the cardiac sodium channel gene (SCN5A), and the greatest concern for anesthesia practitioners is the routine administration of drugs that interact with cardiac ion channels which can potentially trigger the development of malignant arrhythmias.[5] Arrhythmic episodes can be induced during anesthesia by several conditions such as sinus bradycardia, thermal variations, hyperkalemia, hypokalemia, and hypercalcemia.[4] Arrhythmias may also be triggered by certain pharmacologic agents commonly used during anesthesia including vagotonic agents, beta-receptor antagonists, alpha-receptor agonists, lidocaine, and propofol.[4] Additionally, neostigmine may also accentuate the ST segment elevation.[3] In regards to this particular case, the administration of lidocaine on induction and neostigmine on emergence were purposely avoided.

Before induction of anesthesia, external defibrillation pads must be applied to patients with BS.[2] During the intraoperative period, the anesthesia practitioner should continuously monitor EKG, temperature, degree of neuromuscular blockade, bispectral index (BIS) (Medtronic, Minneapolis, MN), and blood pressure.[2] Continuous BIS monitoring of anesthetic depth not only aids in maintaining adequate anesthesia, but also aids in preventing very deep anesthesia. Deep anesthesia should be avoided because ventricular arrhythmias in BS patients usually occur during periods of bradycardia and increased vagal tone.[2]

General anesthesia can be safely administered in patients with BS.[2] However, because this disorder is rare, there is a lack of large prospective studies that are necessary to define the risks and hence the ability to formulate specific guidelines for anesthesia in this patient population.[1] Therefore, anesthesia management for patients with BS includes careful consideration of each drug being administered, avoiding factors that are known to potentially induce arrhythmias, and maintaining the utmost vigilance in monitoring.

In the aforementioned case, the patient underwent general anesthesia for a laparoscopic inguinal hernia repair with no complications. The patient lacked a thorough cardiology work-up, which ideally should have been completed prior to surgery. Vigilant monitoring, and avoiding factors and conditions that may lead to the development of ventricular arrhythmias are the most imperative factors in the anesthetic management of patients with BS. Additionally, effective communication with the surgeon is important, especially during key events such as the initiation of pneumoperitoneum insufflation.

References

1. Letsas KP, Asvestas D, Baranchuk A, Liu T, Georgopoulos S, Efremidis M, et al. Prognosis, risk stratification, and management of asymptomatic individuals with Brugada syndrome: A systematic review. *Pacing Clin Electrophysiol*. 2017;40(12):1332-1345. doi:10.1111/pace.13214.

2. Dendramis G, Paleologo C, Sgarito G, Giordano U, Verlato R, Baranchuk A, et al. Anesthetic and Perioperative Management of Patients With Brugada Syndrome. *Am J Cardiol*. 2017;120(6):1031-1036. doi:10.1016/j.amjcard.2017.06.034.
3. Raval C, Saeed K. Anaesthetic management of a patient of Brugada syndrome for an emergency appendicectomy. *Anesth Essays Res*. 2012;6(1):101-104. doi:10.4103/0259-1162.103390.
4. Dendramis G. Brugada Syndrome: Diagnosis, Clinical Manifestations, Risk Stratification and Treatment. New York: Nova Science Publishers, Inc; 2015. http://search.ebscohost.com.ezproxy.westminstercollege.edu/login.aspx?direct=true&db=nlebk&AN=1100288&site=eds-live. Accessed July 17, 2019.
5. Kloesel B, Ackerman MJ, Sprung J, Narr BJ, Weingarten TN. Anesthetic management of patients with Brugada syndrome: a case series and literature review. *Can J Of Anaesth*. 2011;58(9):824-836. doi:10.1007/s12630-011-9546-y.

Mentor: James Stimpson, DNP, CRNA

Autoimmune Anti-N-Methyl-D-Aspartate Receptor Encephalitis

Steven B. Clifford, MSNA
Westminster College

Keywords: Autoimmune, Anti NMDA, Encephalitis, Anesthesia, Neurologic

Anti-N-Methyl-D-Aspartate (NMDA) receptor encephalitis is an autoimmune neurologic disease where antibodies are created against the NMDA receptors in the brain, causing normal brain signals to be disrupted. Initial clinical symptoms include hallucinations, seizures, confusion, and memory loss.[1] Subsequently, patients develop movement disorders, altered temperature perception, and variations in blood pressure and heart rate.[1] It can affect both men and women; however, it is more common among women, especially those with ovarian teratoma. The NMDA receptor is the target of numerous drugs used every day in anesthesia practice. An important patient safety consideration while preparing any anesthetic plan is understanding the pharmacologic interactions of these anesthetics. Or more precisely, actions of these drugs to altered receptor site anatomy and physiology.

Case Report

An 18-year-old, 68 kg, 183 cm male presented for a port-a-cath exchange in the catheterization lab. The patient's medical history included anti-NMDA receptor encephalitis, epilepsy, asthma, sleep disturbances, hallucinations, and agitation, all of which are related to anti-NMDA receptor encephalitis. His surgical history included a prior port-a-cath placement and revision. Medication allergies included fosphenytoin, penicillins, corticosteroids, fentanyl, latex, and the benzodiazepines lorazepam and midazolam; food allergies were to banana, kiwi, and avocado. The patient was unaware of allergic response to specific medications; he was told by clinicians he exhibited a sensitivity during prior procedures The sensitivity was self-reported by the patient

as “high heart rate, low blood pressure, and difficulty waking up”. Preoperative laboratory testing included a CBC and BMP, with all results within normal limits.

During the pre-anesthetic assessment, the patient’s mother stated, “he does well with propofol.” Preoperative vital signs were within normal limits, and the patient demonstrated normal cognitive function. The airway assessment revealed a Mallampati score of two and a thyromental distance greater than seven centimeters. The patient was not premedicated and self-ambulated into the operating room suite. The patient was preoxygenated with O_2 10 L/min for 3 minutes via a standard anesthesia mask. An intravenous (IV) induction commenced with lidocaine 60 mg and propofol 200 mg. A size four laryngeal mask airway (LMA) was placed without difficulty and the patient resumed spontaneous ventilation throughout the case. Clindamycin 600 mg IV was infused prior to incision while the patient was prepped and draped. The decision of general anesthesia was based on the ability to control the patient’s hemodynamics, respiratory status, and limited access to the patient’s airway.

Anesthesia was maintained with a propofol infusion started at 150 mcg/kg/min. The propofol infusion was titrated to the patient’s Bispectral Index (BIS) (Aspect Medical Systems, Norwood, MA) monitor to maintain a value of 45–50, which at times required up to 350 mcg/kg/min to maintain adequate anesthesia for the procedure. A total of 2,200 mg of propofol was administered throughout the 67-minute case. Standard monitoring was utilized with vital signs remaining stable throughout the case. Mean arterial pressure (MAP) was maintained between 60–75 mm Hg, heart rate ranged from 61–78/min, and respiratory rate was noted at 16/min pre-induction and 12–14 breaths/min throughout the case. The patient’s SpO_2 remained >98%, and $EtCO_2$ remained 36–44 mm Hg for the duration of the case with spontaneous ventilation. The patient did not experience any hemodynamic instability during the procedure that may present in anti-NMDA receptor encephalitis.

An incision was made at the left mid-clavicular line in the 4th intercostal space to gain access to the current nonfunctioning port-a-cath. The device was removed, and the skin was closed using standard sutures. The patient was placed in a 15-degree Trendelenburg position with a rolled towel placed longitudinally between the scapulae under the thoracic spine. A contralateral incision was made at the right mid-clavicular line in the 4th intercostal space gaining access to the right subclavian vein. The catheter was inserted into the superior vena cava (SVC) and the port-a-cath was placed under the skin on the right side. The propofol infusion was discontinued while the surgeon sutured the epidermis. The LMA was removed and the patient was transported to the post anesthesia care unit (PACU), alert and following commands with zero pain endorsed.

Discussion

Anti-NMDA receptor encephalitis is an immune-mediated disease described more than a decade ago by Dr. Josep Dalmau and colleagues. This is a rare disease with an estimated incidence of 1.5 per million population per year, with female predominance (a female-to-male ratio of approximately 8:2) and median age of 21 years at time of discovery.[2]

In anti-NMDA receptor encephalitis, antibodies are generated against NMDA receptors, specifically to the GluN1 subunit. The body’s immune system attacks these receptors where they

are found in the highest concentrations and induces glutaminergic transmission impairment.[3] Clinical presentation consists of bizarre and disturbing behaviors. Patients may have visual hallucinations, develop strange beliefs or appear agitated, and demonstrate a movement disorder consistent with continuous writhing and twitching of face and limbs.[1]

Currently the treatment for anti-NMDA receptor encephalitis involves escalation of immunosuppression therapy. First-line treatment begins with steroids, intravenous immunoglobulins, or plasma exchange. If a more aggressive therapy is warranted, transitioning to second-line therapies of rituximab or cyclophosphamide is common.[2]
The anesthetic considerations are related to autonomic instability, including hyperthermia, hypertension, and tachycardia or bradycardia.[1] General anesthesia using nondepolarizing neuromuscular blocking agents with benzodiazepines and opioids as adjuncts are the preferred anesthesia technique in patients with anti-NMDA receptor encephalitis. For the patient discussed here, an LMA was used without neuromuscular blockers and all benzodiazepines and fentanyl were held due to the patient's aforementioned allergies to these medications. An axillary temperature probe was used to monitor the potential variations in body temperature, and a forced air warming/cooling device was in place. Vasopressors, beta blockers, antihypertensives, and anticholinergics were readily available so that any autonomic instability could be corrected in a timely fashion.[4]

Inhaled anesthetics inhibit NMDA receptors in a reversible, concentration-dependent, noncompetitive manner. It is possible that inhaled anesthetics are unsafe in patients with this disease, although there are several reports of inhaled anesthetics being used successfully in critically ill patients.[5] Specifically, nitrous oxide can inhibit NMDA receptors potentially worsening the patient's symptoms of hallucinations, seizures, confusion, and memory loss.[3] Sevoflurane has a highly fluorinated methyl isopropyl ether molecular structure which enhances GABAergic transmission. Its effect on the NMDA pathway causes inhibition of NMDA-gated currents and NMDA-induced mitochondrial membrane depolarization.[6] Until further research is conducted, the use of the direct acting NMDA receptor antagonists ketamine, nitrous oxide, methadone, and dextromethorphan should be used with caution to avoid exacerbating symptoms and potentially creating hemodynamic instability.

Taking into consideration the effects inhaled anesthetics have on NMDA receptors, all inhaled anesthetics were avoided for this patient. With the potential worsening of symptoms including neurologic disturbances and hemodynamic variability. All direct acting NMDA receptor antagonists were also avoided for this patient. With the patient's history, allergic profile, and ability to tolerate propofol the decision was to proceed with total intravenous anesthetic. Prior to the infusion of propofol lidocaine was administered intravenously to decrease propofol induced pain at the site of injection. The surgical sites were infiltrated with local anesthetic by the surgeon for pain control post operatively. Overall, the anesthetic plan proved beneficial and safe for the patient; he maintained stable vital signs, spontaneous respirations with an LMA in place, and experienced prompt emergence with the ability to self-report no pain.

References

1. Sarosh I, John R. Anti-NMDAR encephalitis. *Curr Neurol Neurosci Rep.* 2011;11(3):298-304.
2. Josep D, Thais A, Jesús P, Marija R, Francesco M, Frank L, Christian G, Eric L, Maarten T, Myrna R, Francesc G. An update on anti-NMDA receptor encephalitis for neurologists and psychiatrists: mechanisms and models. *Lancet Neurol.* 2019;18(11):1045-1057. doi.10.1016/S147-442(19)30244-3
3. Shaikh MA, Dhansura T, Gandhi S, Shaikh T. Anesthetic management of a patient with anti-NMDA receptor encephalitis. *Indian J Anaesth*. 2015;59(4):248–250. doi:10.4103/0019-5049.155005
4. Pryzbylkowski PG, Dunkman WJ, Liu R, Chen L. Anti-N-Methyl-D-Aspartate Receptor Encephalitis and Its Anesthetic Implications. *Anesth Analg*. 2011;113(5):1188-1191. doi: 10.1213/ANE.0b013e31822b8a7d
5. Senbruna B, Lerman J. Anesthesia Management for a Boy with Anti-N-Methyl-D-Aspartate Receptor Encephalitis. *A A Case Reports*. 2015;5(10):182–184. doi: 10.1213/XAA.0000000000000225
6. Criswell HE, Ming Z, Pleasant N, Griffith BL, Mueller RA, Breese GR: Macrokinetic analysis of blockade of NMDA-gated currents by substituted alcohols, alkanes and ethers. *Brain Research.* 2004; 1015(1–2):107–113.

Mentor: Art Shimata, DNAP, CRNA

Anesthesia Management for Emergent Pediatric Craniotomy

Suzanne E. Tremblay, MSNA
Westminster College

Keywords: pediatric fluid resuscitation, intracranial pressure monitoring, craniotomy, pediatric hypovolemia, blood glucose management

Trauma is the most significant cause of morbidity and mortality in the pediatric population in the United States.[1] The goal of resuscitation after neurotrauma is to limit secondary brain injury, maximize cerebral perfusion, improve oxygen delivery and minimize intracranial pressure increase. Fluid resuscitation and blood glucose management after trauma and continued intraoperatively are essential in optimizing outcomes.

Case report

A 2-year-old, 16 kg male was admitted to the emergency room after a pedestrian versus motor vehicle collision. Upon arrival, the patient had a pediatric Glasgow Coma Score of 13 with a deficit of 3 out of 5 for best verbal response as evidenced by persistent cries and screams. Traumatic injuries included communicated left frontal, parietal and temporal bone fractures with significant underlying parenchymal contusion, right 8th rib fracture with associated right upper abdomen contusion, and small bilateral pneumothoraces. Rapid sequence intubation was

performed by emergency room personnel and a propofol infusion was initiated. Computed tomography scan with contrast of the head, neck, chest, abdomen, and pelvis were performed. The patient was transported directly to the operating room for repair of complex open skull fracture, dural lacerations, and evacuation of epidural hematoma.

The patient had no medical or surgical history. He had no home medications or known drug allergies. Preoperative blood pressure, heart rate, respiratory rate and SpO_2 were 76/46 mm Hg, 172/min, 24/min, and 99% SpO_2 on 90% FiO_2 via endotracheal tube, respectively. A complete blood count with metabolic panel, urinalysis, and coagulation tests were performed on admission. Abnormal laboratory values were as follows: venous pH: 7.25, venous bicarbonate: 19 mmol/L; base excess: -8; potassium: 3.1 mmol/L; glucose: 159 mg/dL; creatinine: 0.4 mg/dL; platelets: 502 k/uL; alanine aminotransferase: 61 U/L; aspartate aminotransferase: 160 U/L; prothrombin time: 15.1 seconds.

The patient was transported to the operating room. Upon arrival to the operating room, the 4.5 mm oral endotracheal tube that had been placed previously in the emergency room, was connected to the anesthesia circuit. The propofol infusion was discontinued, Sevoflurane was initiated and titrated throughout the case. Dextrose 5% with sodium chloride 0.9% was infused upon arrival to the operating room. This infusion was stopped and sodium chloride 0.9% was initiated. A fluid bolus of 160 mL was administered. Weight based cefazolin 640 mg was administered and intermittent doses of rocuronium and fentanyl were administered via peripheral IV access for paralysis and analgesia. Central venous and arterial access were obtained via the right femoral vein and artery, respectively. Two units of packed red blood cells totaling 300 mL, platelets 54 mL and mannitol 8 gm were administered throughout the case.

Point of care blood analysis was assessed three times during the procedure using i-STAT (i-STAT Handheld, Abbott, Princeton, NJ). Sodium bicarbonate and calcium chloride were replaced based on the i-STAT results. Vital sign trends revealed heart rate changes from 180/minute to 120/minute and the blood pressure from 76/48 mm Hg to 110/72 mm Hg throughout the case.

At the conclusion of the surgical procedure, a right frontal Camino Intraparenchymal Pressure Monitor (Integra, Plainsboro, NJ) was placed with baseline intracranial pressure of 12-13 mm Hg. Transfer of care was established in the operating room to the medical flight team for transport to a tertiary care center. The patient remained intubated and the propofol infusion was resumed for transfer. The patient was seen by the admitting physician two weeks postoperatively - healthy, healing, and with no neurologic deficits.

Discussion

Intraoperative fluid management in the pediatric trauma patient begins with the knowledge of age related normal vital signs. Tachycardia is an early sign of hypovolemia related to an increased sympathetic tone resulting in increased heart rate.[2] This sign has high sensitivity but low specificity as pain, light anesthesia, or other factors may affect heart rate.[1] Additional signs of hypovolemia are delayed capillary refill, weak pulses, and mottling.[1] Hypotension is a late and ominous indicator of hypovolemia. Pediatric patients may maintain a normal blood pressure until

substantial blood volume is lost due to increases in systemic vascular resistance due to stimulation in sympathetic tone.[1]

Significant factors to consider regarding intraoperative fluid management include assessments of intravascular volume, estimated fluid deficit, ongoing loss, and third space. Ongoing loss is a result of direct whole blood loss in the surgical field, capillary leak, and surgical trauma resulting in extravasation of isotonic fluid into non-functioning compartments or third spacing. Indirect loss can be related to anesthetic induced vasodilation and direct evaporation.

Estimated blood volume for infants is 80 mL/kg. The circulating blood volume in this patient was calculated to be 1280 mL. Blood loss from initial trauma was unknown, but deviations from known normal values in heart rate and blood pressure indicated significant hypovolemia. Intraoperative estimated blood loss was 200 mL. After initial fluid resuscitation in the emergency room, one bolus of sodium chloride 0.9% at 10 mL/kg was administered intraoperatively, followed by packed red blood cells and platelets. The decision for intraoperative packed red blood cell administration was based on hemodynamic instability and inadequate tissue oxygenation demonstrated by metabolic acidosis on point of care analysis. Once the patient's blood pressure was stabilized, mannitol was administered.

Sodium chloride 0.9%, at an osmolality of 308 mOsm/L, is the most common fluid for patients at risk for cerebral edema. Isotonic saline and the avoidance of hypo-osmolar fluids or dextrose containing fluids is well accepted and supported by the American Heart Association.[3] This is due to the osmotic effect of hypo-osmolar fluids leading to cell swelling which may contribute to cerebral edema. Plasma-lyte A is a fluid option with an osmolality of 294 mOsm/L and is more physiologic than normal saline. The use of Plasma-lyte A is increasing in neurosurgery, although its use has a lack of formal researched data to support it.[4] It is well known that lactated ringers is a poor choice in patients with an increased risk of intracranial pressure as it could contribute to cerebral edema. This is due to lactated ringer's hypo-osmolarity (273 mOsm/L) when compared to plasma with an accepted range of 280-300 mOsm/L.

It is well understood and should be noted that surgical stress frequently induces hyperglycemia as the body experiences a significant sympathetic nervous system response. This "fight or flight" response induces proinflammatory cytokines, elevated concentrations of catecholamines, growth hormone, glucagon, and glucocorticoids. This causes increased gluconeogenesis, depressed glycogenesis, induced glucose intolerance and insulin resistance leading to hyperglycemia. Hyperglycemia can be detrimental to brain tissue as it compromises microcirculatory blood flow, increased blood-brain barrier permeability, promotes inflammation and is associated with an increase in post-operative infections.[2] Additionally, hyperglycemia is associated with increased tissue edema in patients with cerebral ischemia, this may lead to an increased risk cerebral bleeding.[3] Both the intensity of hyperglycemia and the duration of time that the condition persists is associated with an increased duration of stay in intensive care units in the pediatric population.[5] Pre-operative blood glucose showed a stress increase of 159 mg/dL, peaking on the first i-STAT assessment at 266 mg/dL and trending downward to 205 mg/dL just prior to transfer of care. Variations in published literature exist regarding optimal perioperative blood glucose values, but suggested values trend near 140 and 180 mg/dL for blood glucose levels.[6] Continued monitoring of serum blood glucose in the postoperative period is necessary and insulin therapy

may be considered. Anesthesia professionals who may manage similar cases should familiarize themselves with up to date management strategies and continue to follow current literature.

References

1. Hobson M. Pediatric hypovolemic shock. *The Open Pediatric Medicine Journal*. 2013;7:10-15. https://openpediatricmedicinejournal.com/contents/volumes/V7/TOPEDJ-7-10/TOPEDJ-7-10.pdf. Published January 2nd, 2013. Accessed August 9th, 2020
2. Nagelhout, JJ. Neuroanatomy, neurophysiology and neuroanesthesia; pediatric anesthesia. In: Nagelhout JJ, Elisha S. *Nurse Anesthesia.* 6th ed. St. Louis, MO. Elsevier, Inc. 2018; 645-681, 1117- 1135.
3. Wijdicks EFM, Sheth KN, Carter BS, et al. Recommendations for the management of cerebral and cerebellar infarction with swelling: a statement for healthcare professionals from the american heart association/american stroke association. *Stroke*. 2014;45(4):1222-1238. doi: 10.1161/01.str.0000441965.15164.d6
4. Nair S, Joseph M. Is buffered crystalloid safer than normal saline in neurosurgery? *Neurol India.* 2018;66(5):1301-1302. doi:10.4103/0028-3886.241386
5. Piastra M, Pizza A, Tosi F, et al. Validation of the glycemic stress index in pediatric neurosurgical intensive care. *Neurocritical Care*. 2016;26(3):388-392. doi:10.1007/s12028-016-0344-9
6. Godoy DA, Di Napoli M, Biestro A, Lenhardt R. Perioperative glucose control in neurosurgical patients. *Anesthesiol Res Pract.* 2012;2012:690362. doi:10.1155/2012/690362

Mentor: James Stimpson, DNP, CRNA

Anesthetic Management of a Parturient with Dilated Cardiomyopathy

Courtney Miniter, DNAP
Yale New Haven Hospital School of Nurse Anesthesia

Keywords: Dilated cardiomyopathy, cesarean section, epidural anesthesia, pregnancy, anesthetic management

Dilated cardiomyopathy (DCM) is characterized by a dilated left ventricular (LV) chamber and systolic dysfunction that commonly results in congestive heart failure (CHF).[1,2] Anesthetic management of a pregnant patient with pre-existing DCM undergoing cesarean section (CS) is challenging and associated with high mortality.[3,4] In the United States, cardiovascular disease is the leading cause of death in pregnant women, accounting for 4.23 deaths per 100,000 live births.[4] Hemodynamic challenges of pregnancy, labor, and delivery pose unique risks which can result in clinical decompensation with overt heart failure.[5] This case report describes the anesthetic management of a parturient with DCM undergoing CS.

Case Report

A 31-year-old, gravida 3, para 2, 194.6 kg female at 39 weeks of gestation with a known case of idiopathic DCM was admitted for scheduled repeat CS. Her past obstetric history was significant for emergent CS at term in 2017 due to labor dystocia, which was further complicated by a failed epidural block resulting in general endotracheal anesthetic (GETA). She was subsequently admitted to the intensive care unit (ICU) with a diagnosis of superimposed pre-eclampsia with severe symptomatology and was treated with magnesium sulfate. The earliest report of DCM occurred in 2015, when medical records revealed an echocardiogram with a left ventricular ejection fraction (LVEF) of 16%. The patient was followed by cardiology and optimized with implanted automatic implantable cardioverter-defibrillator (AICD) in 2017 and a medication regimen of carvedilol 25 mg and enalapril 5 mg QD with routine evaluation. Other pertinent medical history included Class III obesity with a body mass index (BMI) of 63 kg/m^2, obstructive sleep apnea, hypertension, and CHF.

Pre-anesthetic examination revealed stable vital signs. Hemoglobin, hematocrit and platelets were within normal limits. An echocardiogram revealed global hypokinesia of the left ventricle with an LVEF of 47%. In the preoperative holding area, routine monitors were attached, intravenous (IV) access was established, and the patient was preloaded with 500 mL of Ringer's lactate solution. An epidural block was placed in the sitting position at the L2-L3 intervertebral space using ultrasound guidance. After negative aspiration of the catheter, a test dose was administered using lidocaine 1.5% 3 mL with epinephrine 15 mcg with no change in vital signs.

In the operating room, the patient was attached to standard monitors and prepped using strict aseptic precautions. A left radial arterial line was placed for continuous BP monitoring. The AICD was deactivated with attachment of a magnet to the left pectoral region, and the external defibrillator pads were applied and connected to the defibrillator. Epidural anesthesia was achieved with lidocaine 2% with epinephrine 1:200,000 15 mL in 5 mL increments and fentanyl 25 mcg. Before surgical incision, a positive Allis test verified inadequate anesthesia and the epidural was re-dosed with an additional 5 mL of lidocaine. After a negative Allis test, the obstetric team was given permission to proceed; however, the patient reported pressure and discomfort, and was transitioned to GETA. A rapid sequence induction was achieved with midazolam 2 mg, propofol 200 mg, and succinylcholine 160 mg IV. Laryngoscopy and intubation were performed using the GlideScope (Verathon Inc., Bothell, WA). Surgical GA was maintained with a propofol infusion at 75 mcg/kg/min and rocuronium 50 mg IV. Peri-operative analgesics administered were epidural-dosed fentanyl 75 mcg, morphine 1.5 mg IV, and acetaminophen 1 g IV. A healthy male neonate was delivered after 20 minutes with APGAR scores of 9^1 and 10^5.

Peri-operatively, the patient desaturated to the low 90s on an FiO_2 of 50%. Auscultated lung sounds were distant bilaterally, and chest x-ray (CXR) demonstrated white out of the left lung, suggesting CHF and possible aspiration. In addition, the endotracheal tube (ETT) was positioned at the carina. The anesthesia practitioner withdrew the ETT 1 cm and performed Valsalva recruitment maneuvers. Furosemide 40 mg IV was given and maintenance fluids were limited. Arterial blood gas values were as follows: pH 7.39, pCO_2 36 mmHg, pO_2 227 mmHg, HCO_3 21.3 mmol/L. Following the procedure, the patient remained intubated and was admitted to the CCU.

A repeat CXR demonstrated improvement in pulmonary edema following diuretic therapy, and antibiotics were administered for pulmonary aspiration prophylaxis. The patient rested on the ventilator overnight and was extubated post-operative day one with no further sequelae.

Discussion

DCM is a progressive myocardial disease associated with a decrease in forward blood flow that results in an increase in ventricular end-diastolic volume and ventricular filling pressures. This eventually leads to ventricular enlargement to maintain cardiac output (CO).[6,7] DCM is often accompanied by arrhythmias, heart failure, mitral or tricuspid regurgitation, and sudden death.[1-3] The clinical manifestations of DCM vary in severity, from cardiomegaly with minimal cardiovascular symptoms to CHF.[5] The predictors for poor prognosis are an EF of $< 20\%$ on echocardiography, left ventricular end-diastolic dilation, and hypokinetic left ventricle.[8,9]

The perioperative anesthetic management of a pregnant patient with DCM undergoing CS poses a challenge for the anesthesia practitioner. Parturients with preexisting DCM have an approximate 25% to 40% risk of developing major adverse cardiovascular events and recurrent heart failure during pregnancy.[1,3] Pregnancy provides a natural stress test because the cardiovascular system undergoes structural and hemodynamic adaptations to sustain a high-volume load.[5-7] Normal physiological changes during pregnancy threaten the functional adaptability of the cardiovascular system and may exacerbate pre-existing cardiomyopathy. The maternal circulation is commonly referred to as high-flow, low-resistance, owing to major increases up to 50% in CO, and a decrease of 20% in maternal systemic vascular resistance (SVR).[7] Pregnancy is associated with physiologic changes that prompt an increased production of progesterone and estrogen, and activation of the renin-angiotensin-aldosterone system, resulting in an increase in plasma volume by approximately 50%.[8] Marked increases in circulating blood volume are met with an increase in stroke volume and HR. However, patients with an underlying DCM are often unable to compensate for this pronounced increase in CO in the setting of an expanded intravascular blood volume, which results in decompensated heart failure.[7] Pregnancy and labor can have detrimental effects on patients with pre-existing DCM and is associated with high mortality. Therefore, careful anesthetic planning and management is essential.[8]

The primary goals of anesthetic management in patients with DCM are (1) avoidance of drug-induced myocardial depression, (2) maintenance of normovolemia, (3) prevention of increased ventricular afterload, (4) avoidance of aortocaval compression, (5) prevention of pain, hypoxemia, hypercarbia, and acidosis, all of which may increase pulmonary vascular resistance.[6,7] The optimum anesthetic technique for patients undergoing cesarean delivery with DCM is controversial, and use of both GETA and regional techniques have been described.

Epidural anesthesia can effectively be used with carefully titrated doses of local anesthetics and hemodynamic monitoring in patients with DCM undergoing a CS. The changes in preload and afterload produced by epidural anesthesia mimic the pharmacological goals and help to maintain forward flow from the left ventricle.[4] Slow administration and titration of a low dose of local anesthetic has been recommended to avoid rapid and extensive sympathetic nerve block.[3,4] In addition, the accompanying vasodilation reduces venous return, which might be favorable to

accommodate the sudden increase in intravascular volume that occurs due to autotransfusion and aortocaval decompression after delivery.[4]

Clinical scenarios in which neuraxial anesthesia is contraindicated include patients with a high-risk cardiac profile, on anticoagulation maintenance therapy, or unable to tolerate a reduction in SVR caused by the epidural blockade. IV anesthetics chosen should have minimal inhibition on cardiovascular function. Induction drugs such as etomidate (0.2 to 0.3 mg/kg IV) or ketamine (1 to 1.5 mg/kg IV), are supplemented with fentanyl (1 to 2 mcg/kg IV) to avoid a sudden decrease in SVR and subsequent hemodynamic collapse.[4] Propofol can reduce preload and afterload, induce myocardial depression, and impair early-diastolic left ventricular filling, but this effect may be reversed by inotropic drugs.[5]

In patients with severe LV systolic dysfunction and low CO syndrome, it may be necessary to administer inotropic agents such as milrinone or dobutamine.[8] However, during GA, milrinone infusion may result in excessive hypotension, necessitating administration of additional inotropes or vasopressors such as norepinephrine, epinephrine, or dopamine to maintain an acceptable BP.[7]

Appropriate cardiac monitoring during labor can predict and may prevent maternal cardiac or obstetric events. Insertion of an intra-arterial catheter is recommended for continuous BP monitoring prior to initiating any analgesic or anesthetic technique, enabling early identification and treatment of hemodynamic changes.[8] Insertion of a central line allows for central venous pressure (CVP) monitoring and provides access for administering inotropes and vasoconstrictors. Non-invasive methods to estimate CO and function can be utilized, such as Pulse Index Continuous Cardiac Output monitoring (PiCCO), stroke volume (SV), and stroke volume variation (SVV).[4,6,7] Trans-esophageal echocardiography (TEE) can be useful as it identifies causes of hypotension, response to inotropes or fluid loading, estimates preload, diastolic dysfunction, CO, regional wall motion and valve function.[8]

In women with underlying cardiomyopathy, changes in intravascular volume, CO, and peripheral vascular resistance coupled with an impaired ventricular reserve pose unique challenges in the management of pregnancy and labor.[8] A multidisciplinary, collaborative approach is key for the parturient at high risk for cardiovascular complications.[8]

References

1. Kaur H, Khetarpal R, Aggarwal S. Dilated cardiomyopathy: an anaesthetic challenge. *J Clin Diagn Res.* 2013;7(6):1174–1176. doi:10.7860/JCDR/2013/5390.3069.
2. Singh T, Taxak S, Hasija N, Anand A. Anaesthetic management of a parturient with dilated cardiomyopathy: a case report. *Egypt J Anesth.* 2014;30(4):439-441. doi:10.1016/j.egja.2014.03.010.
3. Chen CQ, Wang X, Zhang J, Zhu SM. Anesthetic management of patients with dilated cardiomyopathy for non-cardiac surgery. *Eur Rev for Med and Pharmacol Sci.* 2017;21(3):627-634.
4. Nallam SR, Kosinepalle S, Jyothirmai I, Ratnamaiah A, Reddy KS. Perioperative anaesthetic management of a case of severe dilated cardiomyopathy undergoing elective lower segment caesarean section under epidural anaesthesia. *Int J Sci Stud.* 2014;2(7):225-227.

5. Malik R, Sharma P. Successful outcome in a patient with idiopathic dilated cardiomyopathy with bad obstetric history with no live issue. *Int J Reprod Contracept Obstet Gynecol*. 2016;5(4):1225-1227. doi:10.18203/2320-1770.ijrcog20160693.
6. Stergiopoulos K, Shiang E, Bench T. Pregnancy in patients with pre-existing cardiomyopathies. *J Am Coll Cardiol.* 2011;58(4):337-350. doi:10.1016/j.jacc.2011.04.014.
7. Juneja R, Namibiar PM. Cardiomyopathies and anesthesia. *Indian J Anaesth.* 2017;61(9):728-735. doi:10.4103/ija.IJA_385_17.
8. Pregnancy and heart disease. ACOG Practice Bulletin No. 212. American College of Obstetricians and Gynecologists. *Obstet Gynecol.* 2019;133(5):e320–e356. doi: 10.1097/AOG.0000000000003243.

Mentor: Marianne S. Cosgrove, PhD, DNAP, CRNA

Anesthetic Considerations for Transoral Zenker's Diverticulectomy

Brittany Petersen, DNP
University of Wisconsin Oshkosh

Keywords: Zenker's diverticulectomy, transoral, aspiration risk, cricoid pressure, airway fire risk

Zenker's diverticulum is a posterior herniation of esophageal mucosa typically above the cricopharyngeus muscle and below the inferior pharyngeal constrictor muscle. This pathologic outpouching, which causes dysphagia, has anesthetic implications, most considerably for aspiration.[1] Typically, it develops in the 6th to 9th decades of life, and anesthetic implications for geriatric patients must be considered.[1] The incidence rate is 1:800 in upper gastrointestinal barium studies.[1] There are a few different surgical approaches for correction. This case report discusses the anesthetic implications of Zenker's diverticulectomy (ZD) by the transoral endoscopic laser method.

Case Report

An 88-year-old, 149 cm, 52 kg female with a body mass index (BMI) of 23.4 kg/m^2 presented for transoral ZD. The patient's past medical history included diabetes mellitus type II, hypertension, hyperlipidemia, arthritis, anxiety, and dysphagia. Recent medical history consisted of a spontaneous cerebellar hemorrhagic stroke with subsequent abnormal gait and worsened dysphagia requiring rehabilitation. The workup for dysphagia included a barium video swallow study, which identified an esophageal outpouching consistent with Zenker's diverticulum. The patient was on a liquid diet the week prior to surgery and nothing by mouth eight hours prior to surgery. Past surgical history included hysterectomy and esophagogastroduodenoscopy, both without anesthetic complications. Current medications included amlodipine, atorvastatin, carvedilol, escitalopram, gabapentin, lisinopril, and tramadol.

The patient was transported to the operating room (OR) and transferred to the procedure table. Standard noninvasive monitors were applied. Cefazolin 2 g was administered intravenously (IV).

The head of the OR table was elevated to 20 degrees. The patient was preoxygenated with O_2 10 L/min for 5 minutes via anesthesia mask. General anesthesia was induced with fentanyl 100 mcg, lidocaine 70 mg, propofol 60 mg, and rocuronium 20 mg IV. A 6.0 mm internal diameter laser resistant endotracheal tube (ETT) was placed to secure the airway. Saline was used to inflate the cuff. Placement of the ETT was confirmed with three breaths at 30 cm H_2O via end-tidal carbon dioxide (EtCO2) waveform and auscultation of bilateral breath sounds. Mechanical ventilation was initiated. The patient's eyes were protected with tape and plastic goggles. The pillow was exchanged for a foam head holder. Bispectral index (BIS) monitoring was applied to the patient's forehead. A nasopharyngeal temperature probe and lower body forced-air patient warming blanket were placed. Dexamethasone 10 mg IV was administered prior to the start of the procedure.

General anesthesia was maintained with sevoflurane 2.3% inspired concentration in a mixture of O_2 0.2 L/min and air 1.8 L/min, and BIS monitoring values were maintained between 40-60. The ventilator mode was set to synchronized intermittent mandatory ventilation – pressure control (SIMV – PCV). Minute ventilation was titrated by respiratory rate to keep $EtCO_2$ between 35 to 45 mm Hg. Peak inspiratory pressure remained less than 20 cm H_2O. Ephedrine was titrated in 5 to 10 mg doses for transient hypotension with bradycardia. During the maintenance phase, hydromorphone was administered in 0.5 mg doses for a total of 1 mg. Laser protective goggles were placed over the patient's eyes and provided to OR staff. The surgeon verified that the fraction of inspired oxygen (FiO_2) was less than 0.3 prior to the start of laser use.

After laser use was completed, gas flow rates were changed to O_2 2 L/min, and the use of air was discontinued. Ondansetron 4 mg IV was given prior to the completion of the procedure. After surgical completion, neuromuscular blockade was assessed by train-of-four (TOF) for ¾ twitches and was antagonized with neostigmine 3 mg IV and glycopyrrolate 0.6 mg IV. Sevoflurane in O_2 2 L/min was continued for deep extubation. TOF count was reassessed with 4/4 twitches and sustained tetany. The ventilator was turned off, and spontaneous ventilation was assessed. With adequate tidal volume and respiratory rate, the pilot balloon on the ETT was deflated, and removed. Ventilation was assisted via oral airway and anesthesia mask. Sevoflurane was discontinued, and O_2 10 L/min applied. The anesthesia mask was replaced with a simple facemask and continued O_2 delivery at 6 L/min. A total of 700 mL of IV crystalloid was administered for the 85-minute case.

The patient was transported to the post anesthesia care unit (PACU). In the PACU, the patient had stable vital signs, was arousable to touch, answered questions appropriately, and denied any discomfort.

Discussion

For patients undergoing surgical repair of Zenker's diverticulum, preoperative evaluation and preparation are of utmost importance to mitigate complications and uphold patient safety. This procedure is considered elective and therefore, reversible preoperative problems must be addressed.[1]

Anesthetic implications for the geriatric population must be considered, most notably is the need for coronary artery disease screening. The transoral method for Zenker's diverticulectomy requires hyperextension of the neck.[2] Patients should be assessed for neurological changes with this positioning. Carotid auscultation can assess for bruit, indicating turbulent flow through a stenotic lesion.

Diagnostic imaging will include an esophageal barium swallow study and typically reveal a pouch at or slightly above the level of the cricoid cartilage.[3] Aspiration is a common risk factor. Preoperative fasting per current recommendations is important. However, this does not ensure emptying of the pouch.[1, 4] Oral premedications, including alkalinizing agents and H_2-receptor antagonists, must be avoided to prevent collection within the pouch, which would increase the risk for aspiration. Due to the absence of gastric secretions, pouch contents are already alkaline, negating the need for previously mentioned premedications.[1]

Malnutrition is a common finding as 80 to 90% of symptomatic patients will experience dysphagia.[3] Laboratory studies, including biochemical markers indicative of nutritional status and organ functioning, should be completed preoperatively with correction of values if able. Optimization is important to improve postoperative surgical outcomes.[1]

The risk of aspiration during induction is of major concern. ZD is usually performed under general anesthesia.[1] Awake fiberoptic intubation is the safest method for securing the airway. Literature supports rapid-sequence induction (RSI), but with the modification of not using cricoid pressure and elevating the head 20 degrees.[4] Application of cricoid pressure could potentially increase pressure within the pouch leading to regurgitation of pouch contents into the hypopharynx and increasing the probability of pulmonary aspiration.[1]

The use of CO_2 laser is common with the transoral approach for ZD. Implications of laser use must be known and include a high risk of airway fire.[4] An airway fire requires three components: an ignition source, fuel, and oxidizer. The laser is considered the ignition source. Endotracheal tubes are considered fuel and therefore validates the importance of using a laser resistant ETT. It is recommended to instill saline, with or without blue dye, within the cuff to identify cuff perforation from the laser. In this case, saline without dye was utilized. Oxygen is a known oxidizer; to decrease the risk of airway fire, an FiO_2 less than 0.3 should be used.[4] Other precautions of laser use include protective goggles for OR staff and the patient, wet sponges and saline present in the room, and warning signs of laser use outside the OR doors.

Perforation of the diverticulum can occur with blind placement of an orogastric (OG) or nasogastric (NG) tube.[1, 4] These tubes should not be placed until access to the pouch is established and the surgeon can directly visualize the tube appropriately passing the pouch. In this case, the surgeon placed an OG at the end of the procedure under direct visualization past the pouch. Gastric contents were suctioned, and the surgeon removed the OG tube.

A smooth emergence from anesthesia is important to prevent coughing and straining with subsequent neck hematoma formation and airway compromise.[1, 4] This smooth emergence process can be facilitated with several techniques, including opioid loading and deep extubation. Opioid loading can be achieved with adequate doses of a long acting opioid, such as

hydromorphone.[1] In this case, hydromorphone 1 mg IV was titrated to the patient's respiratory rate and blood pressure tolerance. Deep extubation was performed to prevent coughing and straining during emergence. It should be noted that aspiration risk from pouch contents is no longer contributory since surgical correction occurred. The review of literature lacked discussion on use of corticosteroids. Corticosteroids prevent release of inflammatory mediators and reduce swelling of injured tissue. In this case, dexamethasone 0.2 mg/kg IV was given to decrease airway tissue inflammation.

Overall, the anesthetic discussed in this case report went without complications. The most interesting lesson learned was that RSI is controversial. Current literature supports avoidance of cricoid pressure since it can be more harmful to the patient as it can subsequently lead to aspiration.

References

1. Abdelmalak B, Khanna A. Anesthesia for Zenkers Diverticulectomy. *J Anesth Perioper Med*. 2017;4(1):17-22. doi:10.24015/japm.2017.0003.
2. Dhaliwal HS, Sinha SK, Kochhar R. Endoscopic management of Zenker's diverticulum. *J Dig Endosc*. 2015;06(02):045-054. doi:10.4103/0976-5042.159235.
3. Nuño-Guzmán CM, García-Carrasco D, Haro M, Arróniz-Jáuregui J, Corona JL, Salcido M. Zenkers Diverticulum: Diagnostic Approach and Surgical Management. *Case Rep Gastroenterol*. 2014;8(3):346-352. doi:10.1159/000369130.
4. Miller RD, Cohen NH. *Miller's Anesthesia:* Philadelphia, PA: Elsevier, Saunders; 2015.
5. Plzák J, Zábrodský M, Lukeš P. Zenkers Diverticulum: Carbon Dioxide Laser Endoscopic Surgery. *BioMed Res Int*. 2014;2014:1-5. doi:10.1155/2014/516231.
6. Verhaegen VJO, Feuth T, Frank J. A. Van Den Hoogen, Marres HAM, Takes RP. Endoscopic carbon dioxide laser diverticulostomy versus endoscopic staple-assisted diverticulostomy to treat Zenkers diverticulum. *Head & Neck*. 2011;33(2):154-159. doi:10.1002/hed.21413.

Mentor: LTC Peter Strube, DNAP, CRNA MSNA, MBA, APRN, APNP, ARNP, ANC

Acute Adrenal Insufficiency

LT Shaun Dunston, BSN
Uniformed Services University of the Health Sciences

Keywords: adrenal insufficiency, adrenal crisis, hypotension, anesthesia, etomidate

Acute adrenal insufficiency is a life-threatening postoperative condition that affects people who have adrenal suppression with an occurrence of 0.04% of cases per year, worldwide.[1] This clinical condition is brought on by a stressful event, such as surgery, that is a manifestation of decreased production or action of glucocorticoids.[2] Clinical symptoms include weakness, fatigue, anorexia, weight loss, abdominal pain, and refractory hypotension.[2,3] The following case report

describes a patient with postoperative hypotension that improved following exogenous glucocorticoid administration.

Case Report

A 60-year-old, 150 cm, 70.5 kg, BMI 30 kg/m^2, female with stage III breast cancer scheduled for unilateral mastectomy and contralateral subclavian port placement. No chemo- or radiation therapy had been initiated prior to surgery. The patient's past medical history included seasonal rhinitis and insomnia. The patient reported walking and biking weekly with no functional limitations. The patient's list of home medications included aspirin, zolpidem, pilocarpine drops daily, and doxylamine as needed. The patient did not take any medications day of surgery.

Intravenous (IV) midazolam 2 mg was administered in the preoperative area. The patient then received a left-sided erector spinae plane (ESP) block at the T5 level, which consisted of 15 mL bupivacaine 0.25% and 15 mL liposomal bupivacaine 1.33%. The patient was transported to the operating room (OR) and induction of anesthesia was given, including IV lidocaine 50 mg, propofol 200 mg, succinylcholine 120 mg, and dexamethasone 4 mg.

General anesthesia was maintained with an IV infusion of propofol 200 mcg/kg/min. A total of 125 mcg of fentanyl was administered intraoperatively. Intermittent hypotension towards the end of the case was treated with two phenylephrine IV boluses of 100 mcg each. The propofol infusion was discontinued 10 minutes prior to the end of surgery. Ondansetron 4 mg and ketorolac 30 mg IV were administered at the end of the case. A total of 900 mL of crystalloids were administered during the 112-minute case.

In the recovery room, phenylephrine 100 mcg IV was given to treat systolic blood pressure (SBP) in the 70s mm Hg. SBP remained in the upper 80s to low 90s mm Hg throughout the recovery phase following initial phenylephrine administration. After an 80-minute recovery room stay, she was transferred to the inpatient unit where her wound drain output increased to 80 mL/hr. Concurrently, her SBP decreased to 70 mm Hg after sitting upright. SBP was responsive to a 500 mL crystalloid bolus initially. Upon removal of the bandage wrap, an increase in serosanguinous fluid under the surgical flap was observed and hypotension became persistent. The patient was transferred to the intensive care unit (ICU). In the ICU, the patient was administered one unit of packed red blood cells (PRBCs). Her hemoglobin and hematocrit post-transfusion was 9.6 g/dL and 28.9%

The patient was transferred back to the OR for a wound revision within 6 hours after her initial procedure. The patient was administered IV midazolam 3 mg, etomidate 16 mg, and succinylcholine 80 mg. Six aliquots of IV phenylephrine 100 mcg, 2000 mL crystalloids, and 2 units of PRBCs were given for persistent hypotension. An estimated blood loss of 700 mL for the 55-minute case. After an uneventful emergence, the patient was transported back to the ICU with standard monitors and O_2 10 L/min via face mask.

Postoperatively, there was no further evidence of bleeding with minimal output from the wound drain. The hemoglobin and hematocrit were 11.1 g/dL and 34.5% after a total of 3 units of PRBCs and 3L of crystalloids. The patient was alert but hypotensive with SBP of 70 to 80 mm

Hg, accompanied by symptoms of dizziness, minimal urine output, and nausea. An echocardiogram performed on the patient was unremarkable with an ejection fraction of 60% to 65%. Serial troponin levels were negative, ruling out myocardial ischemia. A phenylephrine infusion initiated at 33 mcg/min and titrated to achieve an SBP greater than 90 mm Hg.

The patient's cortisol level the morning after surgery was 0.188 ug/dL. Computed tomography (CT) of her adrenal glands were unremarkable for any abnormalities. Serum electrolytes were unremarkable. Six doses of hydrocortisone 100 mg IV were administered over the next 24 hours. The patient's blood pressure remained stable without requiring phenylephrine infusion, and she was discharged on postoperative day 3.

Discussion

Hypotension was seen shortly after resuscitation of intravascular volume with PRBCs and crystalloids. Dysrhythmias should be considered as a sign of myocardial ischemia in cases of uncontrolled hypertension. However, dysrhythmias were not present in this case, per the 5-lead intraoperative monitors.[4]

Local anesthetic systemic toxicity (LAST) was considered since the patient had received a unilateral ESP block prior to the initial surgery. LAST may simultaneously cause myocardial depression, dysrhythmias, and reduced systemic vascular resistance resulting in hypotension.[5] Bupivacaine 37.5 mg and liposomal bupivacaine 200 mg were within the accepted maximum dosage for this 70 kg patient. No other cardiac or neurologic signs were present. The patient's syncope resolved when her blood pressure was corrected. Therefore, LAST would have been unlikely.

Primary AI occurs when the adrenal glands are unable to produce enough glucocorticoid, mineralocorticoid, and androgen hormones due to bilateral adrenal destruction.[3] Symptoms of primary AI include: fatigue, weakness, anorexia, nausea and vomiting, cutaneous and mucosal hyperpigmentation, hypovolemia, hyponatremia, and hyperkalemia.[3] Diagnosis of primary AI includes the constitutional symptoms listed above as well as visible destruction of the adrenal glands. The unremarkable adrenal CT scan ruled out primary adrenal insufficiency.

Secondary AI occurs when there is hypothalamic-pituitary axis suppression resulting in decreased cortisol, corticotropin-releasing hormone, and adrenocorticotropic hormone (ACTH).[3] The initial cortisol level of 0.188 ug/dL was notably below the normal morning cortisol threshold of 6-18 ug/dL.[6] Cortisol modulates beta-receptor synthesis, which supports cardiac output, contractility, and vascular tone.[3] Cortisol is integral in metabolism, electrolyte and water balance, and the anti-inflammatory response.[3] Adrenal suppression is typically a result of chronic exogenous glucocorticoid use, but it can also be precipitated by stress from surgery or by medications.[6] Etomidate is known to cause adrenal suppression by inhibition of 11-beta hydroxylase, which is the final enzyme in the production of cortisol, with effects for up to 8 hours from a single dose.[7] Catecholamine receptor sensitivity is coupled with the binding of cortisol, and hypotension is often the observed clinical effect.[6]

The patient's hypotension in this case was determined to be a result of adrenal insufficiency, which was treated successfully with exogenous cortisol. The patient was prescribed a 5-day course of steroids and a consultation with nephrology upon discharge. Steroid dosing strategies are particularly useful for patients who are taking an equivalent of prednisone greater than 5 mg/day.[3] In the absence of other clear causes, acute AI should always be on the differential diagnosis list in patients with refractory hypotension.

References

1. Prete A, Yan Q, Al-Tarrah K, et al. The cortisol stress response induced by surgery: A systematic review and meta-analysis. *Clin Endocrinol (Oxf)*. 2018;89(5):554-567. doi:10.1111/cen.13820
2. Charmandari E, Nicolaides NC, Chrousos GP. Adrenal insufficiency. *Lancet*. 2014;383(9935):2152-2167. doi:10.1016/S0140-6736(13)61684-0
3. Wall, RT. Endocrine Disease. In: Hines RL, Marschall KE, eds. *Stoelting's Anesthesia and Co-Existing Disease*. 7th ed. Philadelphia, PA: Elsevier; 2018:468-470.
4. Sinnenberg L, Givertz MM. Acute heart failure. *Trends Cardiovasc Med*. 2019:S1050173819300441. doi:10.1016/j.tcm.2019.03.007
5. El-Boghdadly K, Chin KJ. Local anesthetic systemic toxicity: Continuing professional development. *Can J Anesth Can Anesth*. 2016;63(3):330-349. doi:10.1007/s12630-015-0564-z
6. Puar THK, Stikkelbroeck NMML, Smans LCCJ, Zelissen PMJ, Hermus AdRMM. Adrenal crisis: Still a deadly event in the 21st century. *Am J Med*. 2016;129(3):339.e1-339.e9. doi:10.1016/j.amjmed.2015.08.021
7. Thompson Bastin ML, Baker SN, Weant KA. Effects of etomidate on adrenal suppression: A review of intubated septic patients. *Hosp Pharm*. 2014;49(2):177-183. doi:10.1310/hpj4902-177

Mentor: CDR Chad Moore, DNP, CRNA, CHSE, NC, USN

Hypoglossal Nerve Stimulator Implantation for Obstructive Sleep Apnea

Julia Camp, MSA, BSN, LT, NC, USN
Uniformed Services University

Keywords: Hypoglossal nerve stimulator, Obstructive sleep apnea, OSA

Continuous Positive Airway Pressure (CPAP) devices are frequently prescribed to patients with Obstructive Sleep Apnea (OSA). However, some patients are unable to tolerate CPAP and seek surgical treatment. A hypoglossal nerve stimulator is an emerging surgical option. Stimulation of the hypoglossal nerve dilates the posterior oropharynx and hypopharynx and reduces apneic and hypopneic events, improving sleep-related quality of life with minimal adverse effects.[1] This report details the first hypoglossal nerve stimulator implantation performed at our large academic military treatment facility (MTF).

Case Report

A 50-year-old, 175 cm, 70 kg male, with a body mass index (BMI) of 25 kg/m^2, presented to surgery for a hypoglossal nerve stimulator implant procedure. Pertinent medical history included OSA with an apnea-hypopnea index (AHI) of 58, hypertension, hyperlipidemia, gastroesophageal reflux disease, chronic neck pain, depression, anxiety, migraines, and insomnia. The patient had a 35 pack-year history of smoking. His prior surgeries included a drug-induced sleep endoscopy (DISE) in 2019, an anterior cervical discectomy and fusion in 2015 and a uvulectomy in 2004. He had no reported issues with general anesthesia. He endorsed no known allergies. His scheduled medications included levomilnacipran, naltrexone, gabapentin, sumatriptan, zolpidem, armodafinil, lamotrigine, bupropion, spironolactone, amlodipine, lisinopril, omeprazole, and rosuvastatin. His airway assessment showed a Mallampati score of 3, a thyromental distance and mouth opening of greater than three fingerbreadths, unremarkable neck, normal heart sounds, and lungs clear to auscultation.

In the preoperative area, the patient received midazolam 5 mg IV. Once in the operating room, standard monitors were applied, and the patient was induced with lidocaine 100 mg, remifentanil 30 mcg, propofol 180 mg, and a defasciculating dose of rocuronium 10 mg IV. Mask ventilation required two persons. Succinylcholine 140 mg IV was administered. A video laryngoscope was used to place a 7.0 neural integrity monitor (NIM) electromyogram endotracheal tube, with placement of the NIM tube also verified by the surgeons. General anesthesia was maintained with sevoflurane 1.2% expired concentration in a mixture of O_2 1 L/min and air 1 L/min along with a remifentanil infusion of 0.15-0.2 mcg/kg/min. The ventilator settings were volume control/synchronized intermittent mandatory ventilation/auto flow with tidal volumes of 7 ml/kg with a PEEP of 3 and an initial respiratory rate of 12 and later 14 breaths per minute at the end of the procedure. Acetaminophen 1000 mg IV and dexamethasone 8 mg IV were administered.

After the patient was prepped, draped, and the NIM monitor was deemed to be working correctly, the surgeon made an incision in the right neck down through the platysmal layer to dissect out the hypoglossal nerve. Each branch of the nerve was tested with the NIM stimulating probe to determine if that branch should be incorporated in the stimulator. This testing allowed the surgeon to identify the functional breakpoint and to discover the ideal placement for the nerve cuff from the stimulation lead. The stimulation lead was placed, and a second incision was made in the upper chest between the sternum and the deltopectoral groove. The impulse generator was implanted superficially to the pectoralis major. Lastly, a thoracic incision was made just inferior to the pectoralis major muscle. The surgeon dissected to the external oblique muscle, creating a space within the interfascial plane between the external and internal intercostal muscles. A pocket was created deep to the internal oblique muscles and a sensing lead placed. A tunneling tool was then used to connect the stimulating, and sensing leads to the impulse generator. Several tests were completed to ensure the sensing and stimulating leads were working correctly, and once complete, the incisions were closed.

The intraoperative anesthetic course was uneventful. The patient remained hemodynamically stable with minimal changes. During the final closure, the patient received ketorolac 30 mg IV and ondansetron 4 mg IV. Remifentanil was discontinued at the end of the surgery. Emergence was smooth, and the trachea was extubated without complications. The patient was transferred to

the PACU in stable condition. The patient remained stable with his pain controlled and was discharged home that day.

Discussion

Obstructive Sleep Apnea is a disorder caused by repetitive pharyngeal collapse.[2] The upper airway both collapses and dilates. The pharyngeal dilating muscle tone and longitudinal traction on the upper airway dilate the airway passage, while intraluminal negative inspiratory pressure and extraluminal positive pressure collapse the airway.[3] A functional collapse occurs when the collapsing forces overcome the dilating forces. Apnea occurs with a complete collapse, while a partial collapse leads to hypopnea. However, both types can cause changes in gas exchange leading to oxygen desaturation, hypercapnia, and sleep disturbances.[2] Past OSA research implicates cardiovascular, metabolic, and neurologic sequelae.[2] The prevalence of OSA in patients between the ages of 30-49 years is estimated at 36.1% of men and 11.4% of women. For those between 50-70 years of age, the estimates increase to approximately 60.6% of men, and 36.9% of women.[4]

Treatment for OSA include non-invasive management options, such as weight loss or the use of a CPAP. Surgical options include uvulopalatopharyngoplasty (UPPP), glossectomy, genioglossal advancement, hyoid suspension, maxillomandibular advancement, tracheostomy, and hypoglossal nerve stimulator implantation.[1] However, the improvement of OSA symptoms for UPPP are roughly 50%, and rates for tongue-based procedures range from 20 to greater than 60% but are associated with increased morbidity.[1] In the past, our patient received a uvulectomy, which did not resolve his symptoms. A CPAP machine was prescribed, but he did not tolerate the device while sleeping so he had not been using it.

The goal of upper airway nerve stimulation is to increase the upper airway diameter without removing soft or skeletal tissue by stimulating the hypoglossal nerve and contracting the protrusor muscle.[1] Upper airway stimulation uses a lead that senses respiration and applies a small stimulus directly to the hypoglossal nerve which increases airway patency as a result of contraction of the protrusor muscles of the tongue, mimicking muscle behavior that occurs during wakefulness.[1]

A five-year prospective outcome study found that patients with the implantation of a hypoglossal nerve stimulator had clinically meaningful and statistically significant improvements in polysomnographic measures of OSA, snoring, daytime sleepiness, and sleep-related quality of life, and a subsequent low incidence of device-related adverse outcomes.[5] These conclusions suggest the hypoglossal nerve stimulator is a viable treatment option for patients weighing surgical treatment.

Initial criteria for the hypoglossal nerve stimulator include: age older than 22 years, AHI between 15 and 65 with no more than 25% central apnea, and a BMI less than 35 kg/m^2.[1] In this case study, the patient met the criteria and was advanced to the next step. Once a patient meets the initial criteria, they undergo a drug-induced sleep endoscopy (DISE) to determine whether the closure that occurs in the pharynx during sleep is concentric or if the anterior-posterior or lateral walls collapse in isolation. The hypoglossal nerve stimulator is most effective with an

anterior-posterior collapse. During the DISE procedure, using a standardized protocol, the patient is sedated with propofol the point of airway obstruction, indicated by audible snoring. An endoscope is then placed nasally into the pharynx to visualize the functional anatomy of the obstruction. A jaw thrust is also performed to visualize the changes that occur. If the closure is concentric, nerve stimulator implantation may not be a viable option. The patient's DISE determined that the closure was anterior-posterior, and so he became a candidate for the hypoglossal nerve stimulator procedure. Following the stimulator implantation, the patient must wait one or two months to allow for healing before the stimulator can be turned on. Discomfort from the electrical stimulation is the most frequently reported adverse event, which can usually be resolved by adjusting the programming and overall, complications are relatively rare. [5]

Obstructive sleep apnea is a debilitating condition that can have physical as well as mental health consequences. Treatment for OSA ranges from weight loss or a CPAP to a variety of surgical options; however, results from surgeries for OSA have had limited success and often come with increased morbidity.[1] Implantation of a hypoglossal nerve stimulator is a novel approach to reducing the consequences of OSA and improving sleep-related quality of life with minimal complications. Anesthesia practitioners need to understand the options for OSA treatment, as patients may not always be compliant in the use of their CPAP, and there are health implications for non-compliance as well as the anesthetic considerations such as neuromonitoring and a surgical site in proximity of the airway.

References

1. Maurer JT, Heyning PVD, Lin H-S, et al. Operative technique of upper airway stimulation: an implantable treatment of obstructive sleep apnea. *Oper Tech Otolayngol Head Neck Surg*. 2012;23(3):227-233. doi:10.1016/j.otot.2012.07.002.
2. Jordan AS, McSharry DG, Malhotra A. Adult obstructive sleep apnoea. *Lancet*. 2014;383(9918):736-747. doi:10.1016/s0140-6736(13)60734-5.
3. Charchaflieh, JG. Sleep-Related Breathing Disorders. In Hines RL, Marschall KE. *Stoeltings Anesthesia and Co-Existing Disease*. Philadelphia, PA: Elsevier; 2018:5.
4. Paskhover B. An introduction to obstructive sleep apnea. *Otolaryngol Clin North Am.* 2016;49(6):1303-1306. doi: 10.1016/j.otc.2016.07.007
5. Woodson BT, Strohl KP, Soose RJ, et al. Upper Airway Stimulation for Obstructive Sleep Apnea: 5-Year Outcomes. *Otolaryngol Head Neck Surg.* 2018;159(1): 194–202. doi.org/10.1177/0194599818762383

Mentor: LCDR Lauren Suszan, DNP, MSN, CRNA, NC, USN

Tumescent Fluid in Plastic Surgery: Anesthetic Considerations

Andrea Szarkowicz, DNAP
Yale New Haven Hospital School of Nurse Anesthesia

Keywords: anesthesia, tumescent fluid, plastic surgery, liposuction

Tumescent fluid is commonly infiltrated by plastic surgeons during liposuction procedures. Tumescent fluid is a dilute combination of epinephrine and lidocaine in normal saline or Ringer's lactate solution. It is used therapeutically to reduce surgical field bleeding and provide local anesthesia.[1] This technique has a very low incidence of adverse outcomes when strict adherence to liposuction guidelines are followed, but these strategies may not be common knowledge amongst novice anesthesia providers.[1] Guidelines offer dose ranges for both the epinephrine and lidocaine used in tumescent fluid, as well as monitoring parameters for fluid volume status and patient temperature.[2]

Case Report

A 55-year-old female with a body mass index of 44 and a past surgical history significant only for a laparoscopic Nissen fundoplication, after which she lost 60 pounds, presented for a bilateral mastopexy and brachioplasty. Her estimated left ventricular ejection fraction based on transthoracic echocardiogram was 60% and her electrolytes were all within normal limits. General endotracheal anesthesia was selected for this patient due to a projected surgical time of ten hours. The patient's vital signs were stable before and during induction of general anesthesia with rocuronium and propofol and intubation with a 6.5 internal diameter endotracheal tube. A urinary catheter was placed after induction. The patient was maintained under general anesthesia with 1.5% end-tidal sevoflurane in combination with a dexmedetomidine infusion at 0.3 mcg/kg/hr. A sub-anesthetic infusion of propofol at 25 mcg/kg/hr was also used for antiemetic purposes. The surgical plan was to perform the mastopexy first, followed by each brachioplasty.

During the first 2 hours of surgery, the patient's total urine output was 20 mL. At this time, 1 liter of warmed Ringer's lactate solution was infused. Although her systolic blood pressure and heart rate remained between 105-135 and 60-85 respectively, due to her low urine output, the patient was assumed to be hypovolemic. An additional liter of IV fluid was infused over the next hour along with albumin 25 g. The patient's urine output did not improve significantly, only increasing to 15 mL in the third hour of surgery. An additional 500 mL of IV fluid was given and by the fifth hour, the patient's urine output began to increase to an appropriate level.

At this point in the case, the surgeon completed the mastopexy and was beginning to use liposuction for the bilateral brachioplasty. One liter of tumescent fluid was irrigated during the mastopexy. A single 3L bag of tumescent fluid containing 500mg 0.025% lidocaine and 0.5mg 1:1,000,000 epinephrine/liter of 0.9% saline was used for the entire case. A total of 800 mL of subcutaneous fat was ultimately removed from both of the patient's arms via liposuction, along with another 1 L of tumescent fluid used as irrigation. By the end of the surgery, the patient had received a total of 2850 mL of Ringer's lactate solution, albumin 25 g and 2 L of tumescent fluid

irrigation. The patient's total urine output was 1900 mL. The patient's temperature was monitored and she remained normothermic throughout the case.

The patient was extubated at the end of the case and proceeded to the post-anesthesia care unit (PACU). Her post-operative laboratory results, specifically hemoglobin, hematocrit and electrolytes, were all within normal limits. The patient spent one day on the surgical inpatient unit upon which her urinary catheter was removed and she and was discharged home.

Discussion

Tumescent fluid is a mixture of either 0.9% saline solution or Ringer's lactate solution with 1:1,000,000 epinephrine and 0.025-0.12% lidocaine. Current literature establishes a clear benefit to using tumescent fluid during various liposuction, vein stripping and cosmetic procedures; the combination of lidocaine and epinephrine serve to provide significant analgesia and effectively reduce blood loss.[1-3] As a solution, the amount of tumescent fluid used by surgeons for different procedures varies. However, up to 4 mL of tumescent fluid for every 1mL of fat aspirated during liposuction can be administered.[2] Because such large volumes can be used, it is important for the anesthesia provider to monitor the patient's fluid volume status, electrolytes, and core temperature as tumescent fluid is not typically warmed.[2]

Multiple studies have been conducted to establish maximum doses for both the lidocaine and epinephrine used in tumescent fluid. Two landmark studies done in the 1990s provide a maximum dose range for lidocaine in tumescent fluid of 35-55 mg/kg.[5, 6] This is considerably more than the recommended 4.5 mg/kg maximum dosage. A more recent study from the department of anesthesiology at Yale School of Medicine acknowledged this maximum dose range as safe but highlights another important point: the variability of absorption rates of lidocaine after tumescent fluid is used. Serum lidocaine concentrations peak 12-16 hours after irrigation with tumescent fluid containing both lidocaine and epinephrine.[3] The risk of local anesthetic systemic toxicity (LAST) is decreased with slower administration of more dilute concentrations of lidocaine, but lipid emulsion and appropriate resuscitation equipment should always be available.[3,4] Tumescent fluid is also widely utilized in the office-based setting because it allows for a lighter plane of anesthesia to be used and patients require few narcotics to tolerate their procedures. In both the acute care setting and office-based cases, patients and caregivers should be made aware of the signs of LAST because many of these symptoms may not appear until long after discharge.[2] Total doses of epinephrine in tumescent fluid should be limited to less than 0.07mg/kg to decrease the risk of cardiovascular side effects.[2] It is vital for the anesthesia provider to inquire about the exact concentration of both lidocaine and epinephrine in tumescent fluid for each individual case.

The American Society of Plastic Surgeons recommends the total volume aspirant, which includes supernatant fat and fluid, should not exceed 5L if liposuction is performed in isolation, or 2L if liposuction is performed in conjunction with another aesthetic procedure.[2] The risk of dilution of serum electrolytes and subsequent volume overload resulting in peripheral and pulmonary edema should be kept in mind when large volumes of tumescent fluid are used.[2] It is the responsibility of the anesthesia provider to monitor the volume of tumescent fluid used and closely monitor urine output as well as appropriate administration of parenteral fluids.

During liposuction, this patient received a total of 2 L of tumescent fluid containing approximately 1,000 mg of lidocaine and 1 mg of epinephrine. These doses were both well within the recommended safe ranges. The patient's persistently low urine output, despite receiving maintenance IV fluid, was treated with additional IV fluid and 25g of IV albumin. Oliguria, defined as urine output of less than 0.5 mL per kilogram per hour, was evident. The patient weighed 78 kg; therefore, her urine output should have been at least 39 mL per hour. Approximately 3L of Ringer's lactate solution was infused parenterally throughout the nearly 10-hour case in addition to 2 L of tumescent fluid instillation to the surgical sites. Shortly after administration of the albumin, the patient's urine output increased significantly and proceeded to approximate her parenteral input volume by the end of the operative day.

In retrospect, the patient should have received IV albumin earlier in response to her low urine output. As a colloid solution, albumin remains in the intravascular space and can help to restore oncotic pressure and blood volume, thereby facilitating filtration via the kidneys. This patient was allowed to remain oliguric for 3-4 hours before albumin was administered and her urine output then responded as expected to the relatively large volume of fluids being administered. Fortunately, the patient tolerated the procedure well with no apparent sequelae and on post-operative day 1, a basic metabolic panel revealed serum electrolytes all within normal limits.

References

1. Nagelhout JJ. Local Anesthetics. In Nagelhout JJ, Elisha S, eds. *Nurse Anesthesia*. 6th ed. St. Louis, MO: Elsevier; 2018:110-127.
2. Shapiro, FE, Osman, BM. Office-based anesthesia. *UpToDate*. https://www.uptodate.com/contents/office-based-anesthesia. Updated Feb 18, 2020. Accessed July 7, 2020.
3. Holt, N. (2017). Tumescent anaesthesia: its applications and well tolerated use in the out-of-operating room setting. *Curr Opin Anaesthesiol*. 30(4):518-524.
4. Klein, J. and Jeske, D. (2016). Estimated Maximal Safe Dosages of Tumescent Lidocaine. *Anesth Analg*. 60(6):263-264.
5. Coleman, W. (1996). Tumescent Anesthesia with a Lidocaine Dose of 55 mg/kg Is Safe for Liposuction. *Dermatol Surg*. 22(11):919.
6. Klein, J. (1990). Tumescent Technique for Regional Anesthesia Permits Lidocaine Doses of 35 mg/kg for Liposuction. *J Dermatol Surg Oncol*. 16(3):248-263.

Mentor: Marianne S. Cosgrove, PhD, DNAP, CRNA

Caesarean Section for Trisomy 18 in a Non-Viable Fetus

George B. Higgins, DNAP
University of Southern California

Keywords: Trisomy 18, ethics, pregnancy, labor, epidural, Caesarean section

Trisomy 18 syndrome is the second most-common aneuploidy chromosomal abnormality following Trisomy 21. It is associated with myriad systemic abnormalities including cardiovascular (VSD, patent ductus arteriosus, auricular septal defect, coarctation of aorta, pulmonic stenosis), skeletal (craniofacial, hand/foot, thorax abnormalities), renal (hydronephrosis, polycystic kidney), and central nervous system (cerebellar hypoplasia, agenesis of corpus callosum, hydrocephalus). Prevalence is wide-ranging and disparate, between 1:3000 and 1:10000. Pathogenesis lies within errors in maternal meiosis. Life expectancy anticipates 50% of newborns will perish prior to 1 week, and over 90% will not survive beyond 1 year.[1]

Case Report

A 32-year-old female presented at 34 weeks, 2 days for antenatal steroids and medical induction of labor. The parturient was a gravida 2 para 1001. Pregnancy had been complicated by gestational hypertension and suspicion for fetal Trisomy 18. Non-invasive prenatal tests showed the presence of multiple fetal abnormalities. The patient's health history and medications were unremarkable. Prognosis for the fetus was poor, with anticipated viability limited to a few minutes. Notable was the patient's goal of care: to deliver her baby alive to allow for skin-to-skin time before the neonate's anticipated imminent death.

Upon admission, labor was medically induced per her primary obstetrics team. A lumbar epidural was placed, but no infusion was initiated. The epidural was performed so in the likelihood of an emergent CS, general anesthesia could be avoided. The needle was placed in the L3-L4 interspace without issue, the catheter was threaded, and a test dose was administered without evidence of intrathecal or intravascular injection. Over the course of labor, fetal heart tracings worsened to a Category II pattern with increased frequency and duration of variable/late decelerations.[2] The decision was made to proceed to emergent CS due to a prolonged deceleration of 16 minutes.

The epidural was dosed prior to operating room (OR) arrival with 3% 2-Chloroprocaine 5mL followed by an additional 15mL over 10 minutes during transport and arrival to the OR. Oxygen was administered via facemask at 10L/min. The patient was positioned supine with left uterine displacement. The level of epidural blockade was assessed with cold and pin prick, with a T10-L4 dermatomal level noted prior to surgical preparation. Cefazolin 2g was administered, as well as ranitidine 50mg, metoclopramide 10mg, and ondansetron 4mg.[3] After surgical draping, the level of epidural blockade was noted to be inadequate for surgery. The patient refused general anesthesia, thus 2% lidocaine with epinephrine 1:200000 with sodium bicarbonate was administered in 5mL increments over 5 minutes to total 15mL. An adequate surgical level (T4-S2 dermatome) was achieved and the CS proceeded in a rapid fashion.

Uterine incision was made 15 minutes after room entry and delivery of neonate was 1 minute later. The infant's Apgar score was 1 due to blue color; slow/irregular respirations; and absence of pulse, activity, and reflex irritability. The neonate was brought immediately to the mother for skin-to-skin bonding. Life-sustaining interventions, such as intubation and cardiovascular support, were not indicated per family's wishes. An intravenous (IV) bolus of oxytocin 3 units was administered, followed by an infusion at 125 milliunits/min. Phenylephrine was administered in 100mcg increments totaling 300mcg over 5 minutes to maintain maternal blood pressure. The remainder of the surgery was uneventful. Interventions rendered by the anesthesia practitioner focused on maximizing maternal-neonate bonding time. This included active warming of the dyad, repositioning, music therapy, and cleansing of the infant. Preservative-free morphine 3mg via the epidural catheter and IV ketorolac 30mg were administered. After surgical closure, the neonate was assessed by pediatrics and pronounced deceased at 48 minutes post-delivery.

Discussion

Nagase et al reviewed 123 cases at a single perinatal center between 1993 and 2009, examining outcomes of fetuses with Trisomy 18.[4] The study's facility policies recommended against invasive procedures for mother and infant, though accommodations were made for parents that were adamant in their wishes for CS delivery. Planned lifesaving measures for the infant were minimized. Palliative care options focused on maximizing time spent between mother and neonate, which mirrors the support decisions made in the context of the presented case. The study's recommendations reiterate the need for individualized treatment and clear delineation of the risks and benefits of CS, vaginal delivery, and palliative care. Referrals were made to other facilities for families desiring more invasive management of the neonate postpartum. The poor prognosis for this disease process leads to questions about the ethics of providing treatment to maximize life (e.g., CS and surgical/invasive interventions for the neonate) versus vaginal delivery and/or palliative care.

In a 2013 *Pediatrics* case study examining a prenatal ethics consultation for a trisomy 18 patient, the parents desired to exclude the genetic diagnosis from medical/surgical decision-making and to make all life-preserving therapies available to their neonate.[5] The ethics committee recognized the conflict as a dichotomy between respecting the parents' autonomy versus the need for a practitioner to act in alignment with their conscience, especially regarding futility of care and resource utilization. Further confounding is the difficulty anticipating fetal viability and the outcomes of many high-risk life-prolonging surgical interventions. This is salient to the anesthesia practitioner who may be placed in a situation counter to their own ethics when responding to an emergent CS or when asked to participate in other interventions, especially when the sole practitioner.

With this unique disease process, there is no available literature for the anesthesia practitioner to refer to with regards to patient management, likely as there are a paucity of these cases performed worldwide. Patients undergoing CS are awake, fearful, and unempowered, and relies upon the anesthesia practitioner to manage their physiological as well as psychosocial needs in this trying time. It is impossible to predict the outcome, and one must be prepared to shepherd the patient through a fetal demise while the CS is completed. Preservation of patient autonomy in

some ways rivals preservation of life. Non-medical interventions, such as music therapy; therapeutic presence; and controlling the tone, noise, and atmosphere of the operating room during this somber time are paramount and may fall to the anesthesia practitioner.

In the case presented, the patient's desire to be awake and participate in the CS delivery obviated the possibility for a general anesthetic, which was nearly required after inadequate initial epidural blockade. If an anesthesia professional places an epidural catheter yet leaves it inactive for a significant period of time, the catheter's ability to provide a surgical level of anesthesia remains unproven. The situation was worsened by the non-reassuring prolonged deceleration that persisted unmonitored after beginning skin preparation. A balance needed to be struck between risking inadequate surgical coverage, contravening the patient's desires in order to preserve maternal safety, and contending with the dwindling viability of the decompensating fetus. Interventions to augment the block, such as ketamine, opioids, or benzodiazepines, would have negatively impacted the mother's ability to be present for the limited bonding time she desired.

Considering advances in life-preserving interventions, anesthesia practitioners will find themselves in increasingly unprecedented situations that evoke competing ethical priorities. Diagnoses that were once terminal now have become nebulous, and prenatal testing allows for more anticipatory planning for postpartum management. Critical to navigating these experiences is having a clear discussion of goals of care involving all potential participants along each phase of care. Future research should be focused on caring for the CS patient in the setting of delivering a non-viable fetus. This case's outcome was the best possible given the dismal diagnosis, and the family expressed a sense of relief and satisfaction for the time they were able to spend with their infant.

References

1. Imataka G, Suzumura H, Arisaka O. Clinical features and survival in individuals with trisomy 18: a retrospective one-center study of 44 patients who received intensive care treatments. *Mol Med Rep.* 2016;13:2457-2466. doi:10.3892/mmr.2016.4806.
2. Ayres-de-Campos D, Spong CY, Chandraharan E. FIGO consensus guidelines on intrapartum fetal monitoring: cardiotocography. *Int J Gynaecol Obstet.* 2015;131:13-24. doi:10.1016/j.ijgo.2015.06.020.
3. Turnbell, J, Bell R. Obstetric anaesthesia and peripartum management. *Best Pract Res Clin Obstet Gynaecol.* 2014;593-605. doi:10.1016/j.bpobgyn.2014.03.008.
4. Nagase H, Ishikawa H, Toyoshima K, Itani Y, Furuya N, Kurosawa K, Hirahara F, Yamanaka M. Fetal outcome of trisomy 18 diagnosed after 22 weeks of gestation: experience of 123 cases at a single perinatal center. *Congenit Anom (Kyoto).* 2016;56:35-40. doi:10.1111/cga.12118.
5. Boss RD, Holmes KW, Althaus J, Rushton CH, McNee, H, McNee T. Trisomy 18 and complex congenital heart disease: seeking the threshold benefit. *Pediatrics.* 2013;132:161-165. doi:10.1542/peds.2012-3643.

Mentor: Erica McCall, MSN, MPH, CRNA

Bent Multi-lumen Access Catheter and Guidewire

Elizabeth Doty, DNAP
University of Southern California

Keywords: central venous catheter, guidewire, insertion, complications

Ultrasound guidance is recommended for placement of central venous catheters (CVCs) to improve patient safety by reducing potential complications, such as, pneumothorax, vascular injury, and failure to identify the vein. The American Society of Anesthesiologists (ASA) recommends the use of ultrasound rather than anatomic landmarks for the insertion of CVCs and to verify placement of the catheter, guidewire, and needle.[1] The following report provides a description of an ultrasound-guided multi-lumen access catheter (MAC) insertion complicated by the inability to remove the guidewire after the catheter was thought to be in the right internal jugular vein (IJV).

Case Report

An 81-year-old, 86 kg, 177 cm, male with a past medical history of coronary artery disease, hypertension, hyperlipidemia, bilateral carotid artery disease, intermittent symptomatic claudication of bilateral extremities, 25 pack-year smoking history, idiopathic pulmonary fibrosis, and restrictive lung disease presented for coronary artery bypass grafting. Past surgical history included inguinal hernia repair and hip replacement. Current medications were metoprolol, lisinopril, aspirin, atorvastatin, gabapentin, nitroglycerin, and isosorbide mononitrate. The patient had no known drug allergies.

In the preoperative holding area, the patient was interviewed, assessed, and consent for anesthesia was obtained. An 18-gauge intravenous catheter and a right radial arterial line were placed without difficulty.

In the operating room standard monitors were applied, in addition to electroencephalography and cerebral oximetry monitors. The patient was preoxygenated with O_2 10 L/min followed by an intravenous induction consisting of lidocaine 100 mg, incremental doses of propofol totaling 200 mg, esmolol 50 mg, and nitroglycerin 50 mcg. An endotracheal tube was successfully inserted into the trachea utilizing direct laryngoscopy and proper tracheal placement was confirmed with bilateral chest rise, bilateral lung sounds, and capnography. Following securement of the endotracheal tube, the stomach was suctioned with an orogastric tube and a transesophageal echocardiography (TEE) probe was placed uneventfully in the esophagus.

The patient was subsequently placed in a 10° Trendelenburg position with his head turned to the left to facilitate insertion of a CVC into the right IJV. The insertion site and procedure field were prepared in sterile fashion. Under ultrasound guidance, the IJV was identified and punctured using an introducer needle while maintaining negative pressure in the syringe. Blood was aspirated, and the guidewire was threaded through the introducer needle. The guidewire was passed smoothly and easily while monitoring telemetry for arrhythmias. Location of the guidewire within the venous system was confirmed by visualization of the distal end in the right

atrium via TEE. The introducer needle was removed, and the MAC combined with a vessel dilator was advanced over the guidewire. The guidewire was withdrawn until the end of the wire was visible through the proximal end of the MAC. Using a scalpel, a controlled stab incision at the entry site was made to facilitate advancement of the MAC and dilator into the vein. The dilator and MAC were advanced together while intermittently checking the guidewire to ensure it could move back and forth with ease. Two "pops" were felt, once when the dilator was thought to have entered the IJV, and the second when the distal end of the MAC was considered to have entered the IJV.

The MAC was then advanced, while the dilator and guidewire were held in place. Upon attempting to remove the dilator and guidewire in tandem, the dilator was easily withdrawn while the guidewire was unable to be removed. Gentle attempts to move the guidewire back and forth were unsuccessful. An attempt to aspirate the side ports was made and neither port returned blood. After discussion with the cardiac surgeon, a decision was made to remove both the MAC and guidewire. This was done without difficulty or resistance, and the site was manually compressed until hemostasis was achieved. Upon inspection of the withdrawn catheter, the distal end of both the MAC and the guidewire were significantly bent. A second attempt at cannulating the right IJV was made, with no difficulty encountered on this attempt.

Discussion

The Seldinger technique is frequently used during insertion of CVCs.[2] This technique involves advancing a guidewire through a hollow needle, removing the needle while keeping the guidewire in place, and advancing a catheter over the guidewire into the desired vessel.[3] After the guidewire is inserted, subcutaneous dilation techniques vary depending on the size of CVC being used. When smaller catheters are used, the dilator is advanced and removed independently of the catheter, which is threaded over the guidewire and advanced into the vessel after the tissue has been dilated. However, for large-bore catheters, the dilator and catheter are threaded over the guidewire together, with the dilator extending beyond the length of the catheter. The reason for this difference in technique is due to changes in catheter compliance that occur with changes in size; the dilator helps stabilize the large compliant catheter.[4] A larger, more pliable catheter has a greater tendency to fold in on itself during advancement due to the compressive forces of the surrounding tissues.[4] The guidewire and dilator are meant to be removed in unison, leaving just the catheter behind in the blood vessel.

Complications encountered during CVC placement may be associated with both dilator and guidewire use. Mechanical complications associated with dilator use include: hemothorax, cardiac injury, and arteriovenous fistula.[5] Excessive guidewire insertion length contributes to many guidewire-related complications.[2] Guidewire-specific complications include: cardiac dysrhythmias or conduction abnormalities; vessel or cardiac chamber perforation; kinking, looping, or knotting of the wire; entanglement of previously placed intravascular devices; embolization of a broken-off distal tip of the guidewire; and complete loss of the guidewire within the vascular system.[2] Recommendations for guidewire insertion lengths to prevent complications have been reported in the literature. The upper-limit length recommendation for guidewire insertion at all access sites in adults is approximately 18 cm.[6] It is suggested that an average distance for right IJV insertion into the junction of the superior vena cava and right

atrium is approximately 16 cm.[6] Gender-based estimations to achieve accurate positioning have also been made: cannulation of the IJV for males is 13-14 cm and for females is 12-13 cm.[7] Additionally, height-based formulas can correctly estimate the length required for insertion; the formula for the right IJV is the patient's height (in cm) divided by 10.[8]

In the case presented, it is likely that the dilator and guidewire were not held in place while the catheter was being advanced, and that the dilator was inadvertently withdrawn to a point outside the IJV. Since the tip of the dilator was no longer in the vessel, the catheter was advanced outside the vessel and into the surrounding tissue, which caused it to bend as the external forces of the subcutaneous tissue compressed the soft catheter. This explained the inability to aspirate blood, the difficulty in removing the wire, and bending of the catheter and wire. During the second attempt at cannulation, care was taken to fix the dilator and guidewire in place and the catheter was placed uneventfully.

When placing CVCs, the provider should be familiar with practice guidelines and recommended techniques to reduce complications and improve patient safety. The ASA recommends using ultrasound-guidance for vessel location and needle insertion into the IJV and ultrasound verification of the guidewire within the IJV.[1] Consideration should also be given to the site-specific and height-based recommendations for guidewire insertion length to ensure appropriate catheter location.[6,7,8] Intermittently moving the guidewire in and out as the dilator and catheter duo are advanced can also help prevent and detect guidewire-related complications.[5] Although all of these interventions were employed in this case, they did not prevent inadvertent withdrawal of the dilator during catheter advancement, which resulted in advancement of the catheter tip into the subcutaneous tissue. To avoid similar complications, one must be vigilant to ensure the wire and dilator are held firmly in place when advancing the catheter.

References

1. American Society of Anesthesiologists Task Force on Central Venous Access. Practice guidelines for central venous access. *Anesthesiology*. 2012; 116(3):539-573. doi:10.1097/aln.0b013e31823c9569.
2. Khasawneh FA, Smalligan RD. Guidewire-related complications during central venous catheter placement: A case report and review of the literature. *Case Rep Crit Care*. 2011; 2011:1-4. doi:10.1155/2011/287261.
3. Higgs Z, Macafee D, Braithwaite B, Maxwell-Armstrong C. The Seldinger technique: 50 years on. *The Lancet*. 2005; 366(9494):1407-1409. doi:10.1016/s0140-6736(05)66878-x.
4. Wetzel LR, Patel PR, Pesa NL. Central venous catheter placement in the left internal jugular vein complicated by perforation of the left brachiocephalic vein and massive hemothorax. *A A Case Rep*. 2017; 9(1):16-19. doi:10.1213/xaa.0000000000000511.
5. Scott WL, Collier P. The vessel dilator for central venous catheter placement: forerunner for success or vascular misadventure? *J Intensive Care Med*. 2001; 16(6):263-269. doi:10.1177/088506660101600602.
6. Andrews RT, Bova DA, Venbrux AC. How much guidewire is too much? Direct measurement of the distance from subclavian and internal jugular vein access sites to the superior vena cava-atrial junction during central venous catheter placement. *Crit Care Med*. 2000; 28(1):138-142. doi:10.1097/00003246-200001000-00023.

7. Rao S, Rao SM, Mrinal M. How correct is the correct length for central venous catheter insertion. *Indian J Crit Care Med*. 2009;13(3):159-162. doi:10.4103/0972-5229.58543.
8. Czepizak CA, O'Callaghan JM, Venus B. Evaluation of formulas for optimal positioning of central venous catheters. *Chest*. 1995; 107(6):1662-1664. doi:10.1378/chest.107.6.1662.

Mentor: Ramana Duvvuri, MD

Sniffing Position versus Simple Head Extension Position

Duong Hoang, DNAP
Bryan College of Health Sciences

Keywords: sniffing position, simple head extension, percentage of glottic opening, intubation difficulty scale, Cormack and Lehane grade

Introduction

Traditionally the sniffing position (SP) is considered the ideal for intubation. However, this was challenged with radiographic evidence suggesting the three axis alignments do not exist and the SP is only beneficial in obese patients and patients with limited head extension.[1,2] Adnet et al. were criticized for not using muscle relaxants, an important component in facilitating intubation. Even though widely accepted, the clinical effectiveness of SP versus simple head extension (SHE) has not been established through evidence. The purpose of this evidence-based practice analysis is to compare the SP to SHE for optimization of glottic view and ease of intubation.

Methods

A PICO format guided the clinical question. In adult patients undergoing general anesthesia with endotracheal intubation (P), does SP (I) compared to SHE (C) provide greater glottic view and ease of intubation (O)?

An electronic database search was conducted using EBSCO, PubMed, Google Scholar, Cochrane Database, Scopus, and CINAHL. Peer-reviewed journals published in English were searched. Keywords used individually or in combination to search included sniffing, sniff, head extension, posture, patient positioning, intubation, intubated, and laryngoscopy. All relevant studies written in English and published between 2011 and 2019 were evaluated. Studies were included if SP was compared to SHE in regard to glottic view. Ten studies met the inclusion criteria. Two were excluded due to the use of videoscope and fiberoptic intubation; the remaining eight studies were analyzed. Five were randomized controlled trials (RCT) and three were randomized crossover studies, which were all categorized as 1.c on the Joanna Briggs Institute hierarchy of evidence.

Literature Analysis

All studies included adults scheduled for elective surgery requiring endotracheal intubation. The participants ranged from 1 to 3 on the American Society of Anesthesiologists physical status

(ASA PS) classification and less than 35 kg/m^2 on the body mass index (BMI) scale. Participants with known or predicted difficult intubation were eliminated. All participants were preoxygenated with O_2 100% for at least 3 minutes, followed by induction of anesthesia with IV propofol and an IV muscle relaxant. DL was performed by experienced laryngoscopists using a Macintosh laryngoscope blade size 3 or 4.

In these studies the SP was achieved by using a rigid head cushion of 6 cm to 8 cm for neck flexion and head extension during DL. SHE was done by removal of the rigid head cushion and placing the head in extension on a flat surface. A first success intubation attempt is defined as successful placement of the endotracheal tube without any supplementary attempts, additional laryngoscopists, or other alternative intubation techniques. The Cormack and Lehane Grade (CL grade) was used in six studies, and two used the Percentage of Glottic Opening (POGO) score to evaluate the view of the glottis. CL grade I was a full view of vocal cords. CL grade II was a partial view of the glottis or arytenoids. CL grade III was only the epiglottis visible, and CL grade IV was neither glottis nor epiglottis visible. A POGO score of 100 % was achieved when the entire glottic opening from the anterior commissure of the vocal cords to the interarytenoid notch was viewed. A POGO score of 0 corresponded with no visualization of the glottic opening. The Intubation Difficulty Scale (IDS) was used in four of the studies to evaluate the ease of intubation. An IDS score of 0 correlated with an easy intubation, a score of 1 to 5 was mildly difficulty, and a score of greater than 5 corresponded to severe difficulty.

Akhtar et al.[3] conducted an RCT with 500 participants between the ages of 42 to 65. Participant with an abnormal airway was excluded from the study. SP was achieved by an incompressible head cushion of 7 cm and with maximal head extension at the atlanto-occipital joint. SHE was performed by placing the head flat on the table with maximal head extension. All participants received fentanyl 2 mcg/kg, propofol 2 mg/kg, and atracurium 0.5 mg/kg for induction of anesthesia. CL grade I was seen in 169 of 250 (67.6%) with SP and 171 of 250 (68.4%) with SHE. CL grade II was seen in 54 of 250 (21.6%) with SP and 49 of 250 (19.6%) with SHE. CL grade III and IV was seen in 27 of 250 (10.8%) with SP and 30 of 250 (12%) with SHE. No significant difference was found in CL grades ($p=0.852$). The IDS was used to rate ease of intubation with 58.8% of the SP subjects and 40% of the SHE subjects rated as easy ($p<0.001$). The first attempt success rate with SP was 226 of 250 (90%) and 213 of 250 (85%) with SHE ($p=0.101$).

An RCT done by Bahattarai et al.[4] included 400 participants between 20 to 60 years of age. Subjects with a BMI > 30 kg/m^2, or with a risk of regurgitation or aspiration were excluded. Induction of anesthesia was facilitated by IV midazolam 0.03 mg/kg, fentanyl 2 mcg/kg, propofol titrated to loss of response to verbal command, and vecuronium 0.1 mg/kg. SP was achieved with an 8 cm head cushion and maximal extension on the atlanto-occipital joint. SHE was only maximal head extension on the atlanto-occipital joint. CL grade I was seen in 133 of 200 (66.5%) with SP and 119 of 200 (59.5%) with SHE. CL grade II was seen in 62 of 200 (31%) with SP and 64 of 200 (32%) with SHE. CL Grade III and IV was found in 5 of 200 (2.5%) with SP and 17 of 200 (8.5%) with SHE. The CL grades were not significantly different between the groups ($p>0.05$). An IDS rating of easy intubation occurred in 58% with SP group and 41% with SHE group which was a significant difference ($p<0.05$). A significantly higher number in the SHE group were rated with mild difficulty (57%) compared to the SP group (41%)

($p<0.05$). First successful attempt was achieved in 90% of participants in the SP group compared to 78% in the SHE group ($p<0.05$).

A prospective randomized crossover comparison study by El-Orbany et al.[5] included 167 participants between the ages of 18 to 87 years. Any participant with poor dentition, gastroesophageal reflux disease, risk of aspiration or a BMI > 35 kg/m^2 was excluded. Induction of anesthesia was done with IV propofol 2 mg/kg and rocuronium 0.6 mg/kg. SP was achieved by 6 cm head cushion with slight head extension, and SHE with only slight head extension. CL grade I was seen in 58 of 167 (34.7%) with SP and 24 of 167 (14.3%) with SHE ($p<0.001$). CL grade II was seen in 105 of 167 (62.9%) with SP and 129 of 167 (77.2%) with SHE ($p=0.006$). CL grade III was seen in 4 of 167 (2.4%) with SP and 14 of 167 (8.4%) with SHE ($p=0.027$).

Hafizhoh and Choy[6] conducted a prospective randomized single-blinded clinical trial of 378 participants between the ages of 18 to 75 years. Participants with a BMI > 35 kg/m^2 or requiring rapid sequence intubation was excluded. Induction of anesthesia was done with IV fentanyl 2 mcg/kg and propofol 2 mg/kg titrated until loss of verbal response. Muscle relaxant was achieved by IV rocuronium 0.6 mg/kg. SP was obtained by a 7 cm head cushion with slight head extension, and SHE with only slight head extension. CL grade I was seen in 283 of 378 (74.8%) with SP and 107 of 378 (28.3%) with SHE. CL grade II was seen in 87 of 378 (23%) with SP and 236 of 378 (62.4%) with SHE. CL grade III was seen in 8 of 378 (2.11%) with SP and 34 of 378 (6.35%) with SHE. The CL grades were significantly different between the groups ($p<0.001$). First successful attempt was 83.5% for SP and 64% for SHE ($p<0.05$).

Kim et al.[7] completed a small randomized cross-over trial of 18 elderly edentulous participants with a mean age of 75 years. Excluded were those with anatomical abnormalities in the neck, larynx, or pharynx; risk of aspiration; or a BMI > 30 kg/m^2. Induction of anesthesia was done by IV propofol 1.5 mg/kg, fentanyl 1.5 - 2.0 mcg/kg, and rocuronium 0.6 mg/kg. The SP was achieved by a 7 cm rigid head cushion with head extension, and SHE was achieved by head extension only. The mean POGO score for SP was 78.9% and 53.8% for SHE ($p=0.001$).

A larger randomized cross-over trial included 200 adult participants, 100 male and 100 female.[8] Participants with deformities of the face, neck, or upper airway were excluded. Induction of anesthesia was accomplished with IV propofol 1.5 mg/kg and IV rocuronium 0.6 mg/kg. SP was achieved with a 7 cm rigid head cushion and head extension. SHE was achieved by head extension only. The mean POGO Score for males was 58% for SP and 42% for SHE ($p<0.05$). Mean POGO score for females for SP was 45% and 47% for SHE ($p>0.05$).

The Prakash et al.[9] RCT included 546 participants with ages ranging from 22 to 51years. Participants with malformations of the neck or face, unstable cervical spine, or requiring rapid sequence intubation were excluded. Induction of anesthesia was done with IV fentanyl 2 mcg/kg, propofol 2-2.5 mg/kg, and vecuronium 0.1 mg/kg. SP was achieved by 7 cm head cushion and extension of the head on the neck. SHE was done with only simple head extension. CL grade I was seen in 171 of 275 (62.2%) with SP and 145 of 271 (53.5%) with SHE. CL grade II was seen in 82 of 275 (29.8%) with SP and 101 of 271 (37.3%) with SHE. CL grade III and IV was seen in 22 of 275 (8.0%) with SP and 25 of 271 (9.2%) with SHE. The CL grades were not significantly different between SP and SHE ($p=0.144$). An easy IDS rating was seen in 60.4% of

SP and 47.6% of SHE (p=0.005). First attempt success was seen in 253 of 275 (92%) with SP and 236 of 271 (87%) with SHE (p>0.05).

Another RCT with 100 participants ranging from 18 to 65 years of age was conducted by Vikasgupta et al.[10] Participants with a BMI > 30 kg/m^2 were excluded. Induction of anesthesia was done with IV propofol titrated to loss of response to verbal commands and succinylcholine 2 mg/kg for muscle relaxant. SP was achieved by an 8 cm rigid head cushion and maximal extension on atlanto-occipital joint. SHE was done with maximal extension on atlanto-occipital joint. CL grade I was seen in 19 of 50 (38%) with SP and 30 of 50 (60%) with SHE. CL grade II was seen in 26 of 50 (52%) with SP and 18 of 50 (18%) with SHE. CL grade III was seen in 5 of 50 (10%) with SP and 2 of 50 (4%) with SHE. The CL grades were significantly different between the groups (p=0.04). An easy IDS rating occurred in 36% with SP and 60% with SHE (p=0.02). First attempt success rate with SP was 45 of 50 (90%) and 47 of 50 (94%) with SHE (p> 0.05).

In all the studies, the CL grade I incidence ranged from 34.7% to 74.5% with SP and 14.3% to 68.4% with SHE. The mean POGO score with the SP was 45% to 78.9% and with SHE was 42% to 47%. An IDS rating of easy occurred in 36% to 58.8% with SP and 40% to 60% with SHE. The first attempt success rate in these studies ranged from 83.5% to 92% with SP and 64% to 94% with SHE.

<table>
<tr><th>Study</th><th>Sample/Design</th><th>Laryngeal View</th><th>Intubation Results</th><th>Conclusion</th></tr>
<tr>
<td>Akhtar et al., 2017[3]</td>
<td>RCT

N = 500
Age 42 – 65
MMC 1-4
Laryngoscopist = 1</td>
<td>
<table>
<tr><td>CL</td><td>SP
n= 250</td><td>SHE
n= 250</td></tr>
<tr><td>I</td><td>67.6%
n= 169</td><td>68.4%
n= 171</td></tr>
<tr><td>II</td><td>21.6%
n= 54</td><td>19.6%
n= 49</td></tr>
<tr><td>III</td><td>9.6%
n= 24</td><td>11.2%
n= 28</td></tr>
<tr><td>IV</td><td>1.2%
n= 3</td><td>0.8%
n= 2</td></tr>
</table>
p = 0.852
</td>
<td>
<table>
<tr><td>IDS</td><td>SP</td><td>SHE</td></tr>
<tr><td>Easy</td><td>58.8%</td><td>40%</td></tr>
<tr><td>Mild</td><td>38.4%</td><td>60%</td></tr>
<tr><td>Severe</td><td>2.8%</td><td>0%</td></tr>
</table>
p <0.001

<table>
<tr><td></td><td>SP</td><td>SHE</td></tr>
<tr><td>1st attempt</td><td>90%
n=226</td><td>85%
n=213</td></tr>
</table>
p = 0.101
</td>
<td>No significant difference in CL grade (p= 0.852)

Significantly easier to intubate with SP (p <0.001)

No significant difference in first attempt success (p = 0.101)</td>
</tr>
<tr>
<td>Bahattarai et al., 2011[4]</td>
<td>RCT

N=400
Age 20 – 60
MMC 1- 4
Laryngoscopist = 1</td>
<td>
<table>
<tr><td>CL</td><td>SP
n=200</td><td>SHE
n=200</td></tr>
<tr><td>I</td><td>66.5%
n=133</td><td>59.5%
n=119</td></tr>
<tr><td>II</td><td>31%
n=62</td><td>32%
n=64</td></tr>
<tr><td>III</td><td>2%
n= 4</td><td>8.5%
n= 17</td></tr>
<tr><td>IV</td><td>0.5%
n= 1</td><td>0%
n= 0</td></tr>
</table>
p > 0.05
</td>
<td>
<table>
<tr><td>IDS</td><td>SP</td><td>SHE</td></tr>
<tr><td>Easy</td><td>58%</td><td>41%</td></tr>
<tr><td>Mild</td><td>41%</td><td>57%</td></tr>
<tr><td>Severe</td><td>0.01%</td><td>0.02%</td></tr>
</table>
Easy p < 0.05
Mild p < 0.05
Severe p > 0.05

<table>
<tr><td></td><td>SP</td><td>SHE</td></tr>
<tr><td>1st attempt</td><td>90%</td><td>78%</td></tr>
</table>
</td>
<td>No significant difference in CL grade (p > 0.05)

Significantly easier to intubate with SP (p<0.05)
Significantly higher first attempt success with SP (p <0.05)</td>
</tr>
</table>

			p <0.05	
El-Orbany et al., 2014[5]	Prospective, randomized crossover comparison study N = 167 Age 18 – 87 MMC 1-3 Laryngoscopist = 3	CL: SP n=167, SHE n=167 I: 34.7% n=58, 14.3% n=24 II: 62.9% n=105, 77.2% n=129 III: 2.4% n= 4, 8.4 % n=14 IV: 0%, 0% CL I: p <0.001 CL II: p = 0.006 CL III: p =0.027		CL grade significantly improved with SP (p<0.001) Significantly higher incidence of no glottic view with SHE (p = 0.027)
Hafizhoh & Choy., 2014[6]	Prospective randomized single-blinded clinical trial N = 378 Age 18-75 MMC 1-2 Laryngoscopist= 3	CL: SP n=378, SHE n=378 I: 74.8% n=283, 28.3% n=107 II: 23% n=87, 62.4% n=236 III: 2.11% n=8, 6.35% n=34 IV: 0%, 0% p < 0.001	1st attempt: SP 83.5%, SHE 64% p < 0.05	CL grade significantly improved with SP (p<0.001) SP provided higher first attempt success rate (p < 0.05)
Kim et al., 2016[7]	Randomized cross-over trial N = 18 Mean age = 75 Edentulous MMC 1 & 2 Laryngoscopist = 1	POGO: SP, SHE Mean: 78.9%, 53.8% p = 0.001		SP provided significantly greater glottic view (p = 0.001)
Park et al. 2014[8]	Randomized cross-over trial N = 200 Age 18 – 75 MMC 1-4 Laryngoscopist = 3	POGO (mean): SP n=200, SHE n=200 Male n=100: 58% n=58, 42% n=42 Female n=100: 45% n=45, 47% n=47 Male: p < 0.05 Female: p > 0.05		In males, SP provided significantly greater glottic view (p <0.05) In females, no significant difference in glottic view (p >0.05)
Prakash et al., 2011[9]	RCT	CL: SP n=275, SHE n=271	IDS: SP, SHE Easy: 60.4%, 47.6%	No significant difference in CL

<table>
<tr>
<td></td>
<td>N = 546
Age 22 – 51
Mallampati 1- 4
Laryngoscopist = 3</td>
<td>
<table>
<tr><td>I</td><td>62.2% n=171</td><td>53.5% n=145</td></tr>
<tr><td>II</td><td>29.8% n=82</td><td>37.3% n=101</td></tr>
<tr><td>III</td><td>7.6 % n=21</td><td>9.2% n=25</td></tr>
<tr><td>IV</td><td>0.4% n=1</td><td>0%</td></tr>
</table>
p = 0.144
</td>
<td>
<table>
<tr><td>Mild</td><td>38.2%</td><td>52.4%</td></tr>
<tr><td>Severe</td><td>1.5%</td><td>0%</td></tr>
</table>
p = 0.005

<table>
<tr><td></td><td>SP</td><td>SHE</td></tr>
<tr><td>1st attempt</td><td>92% n=253</td><td>87% n=236</td></tr>
</table>
p > 0.05
</td>
<td>grade (p = 0.144)

Intubation significantly easier with SP (p = 0.005)

No difference in first attempt success (p > 0.05)</td>
</tr>
<tr>
<td>Vikasgupta et al., 2018[10]</td>
<td>RCT

N = 100
Age 18-65
laryngoscopist = 1</td>
<td>
<table>
<tr><td>CL</td><td>SP n=50</td><td>SHE n=50</td></tr>
<tr><td>I</td><td>38% n=19</td><td>60% n=30</td></tr>
<tr><td>II</td><td>52% n=26</td><td>36% n=18</td></tr>
<tr><td>III</td><td>10% n=5</td><td>4% n=2</td></tr>
<tr><td>IV</td><td>0%</td><td>0%</td></tr>
</table>
p = 0.04
</td>
<td>
<table>
<tr><td>IDS</td><td>SP</td><td>SHE</td></tr>
<tr><td>Easy</td><td>36%</td><td>60%</td></tr>
<tr><td>Mild</td><td>64%</td><td>40%</td></tr>
<tr><td>Severe</td><td>0%</td><td>0%</td></tr>
</table>
p = 0.02

<table>
<tr><td></td><td>SP</td><td>SHE</td></tr>
<tr><td>1st attempt</td><td>90% n=45</td><td>94% n=47</td></tr>
</table>
p > 0.05
</td>
<td>SHE provided significantly greater glottic view (p = 0.04)

Intubation significantly easier with SHE (p = 0.02)

No significant difference in first attempt success (p > 0.05)</td>
</tr>
</table>

Conclusion

Eight studies were evaluated to compare SP to SHE for glottic visualization and ease of intubation. Four of the eight found that SP resulted in greater glottic visualization than SHE.[5–8] Three found no difference in glottic view, although intubation was easier with SP compared to SHE in these studies.[3,4,9] Only Vikasgupta et al.[10] found that SHE resulted in greater glottic visualization and ease of intubation than SP. However, the first attempt success rate was the same with the two positions. First attempt success rate was only found to be significant with SP.[4,6] In the four studies in which SP result in greater glottic visualization, SP was achieved by head elevation and slight head extension. In studies where SP found no difference in glottic view, SP was achieved by head elevation and maximal extension at the atlanto-occipital joint. Prakash et al.[9] did not support this trend therefore future studies should determine the exact degree of head extension for SP to have optimal results.

Additional intubation positions such as the elevated sniffing positions (ESP) and ramped position were not evaluated. El-Orbany et al. [5] and Kim et al.[7] compared the SP with ESP and SHE. El-Orbany et al. found that ESP provided a lower incidence of CL grade III and IV than both SP and SHE. Kim et al. found that ESP and SP both provide greater glottic view than SHE. Even though ramped position was not included in any of the studies, some suggested that fewer CL grade III and IVs, and failed intubations, occur in patients with high difficult intubation scores in the ramped position compared to SP.[11]

All studies excluded participants with a history of known difficult intubation, a population for which the best intubation position needs to be established. A recent study found that SHE provides a higher POGO score than SP when using a fiberoptic bronchoscope to intubate. However, the ease of intubation was similar between the two positions.[12] The videoscopes are often used to rescue a difficult intubation encounter, and no significant difference in ease of intubation was found between SP and SHE with the use of videoscopes.[13]

This evidence-based practice analysis supports the SP as the ideal intubation position for DL with a Macintosh blade. The glottic view was optimized and ease of intubation facilitated nonobese patients and patients with no known or predicted difficult intubation. Further research is needed to evaluate the effectiveness of SP in other populations and with additional intubation techniques. A limitation of this analysis includes a lack of available data for patients with a BMI > 35 kg/m^2, which suggests further research is needed for this demographic. Also the Macintosh blade was used for all DLs, while further studies are needed to analyze the use of the Miller blade and videoscope.

References

1. Adnet F, Borron SW, Dumas JL, Lapostolle F, Cupa M, Lapandry C. Study of the "sniffing position" by magnetic resonance imaging. *Anesthesiology*. 2001;94(1):83-86. doi:10.1097/00000542-200101000-00017
2. Adnet F, Lefebvre L, Lapostolle F. Randomized Study Comparing the "Sniffing Position" with Simple Head Extension for Laryngoscopic View in Elective Surgery Patients. *Anesthesiology*. 2001;95(4):6.
3. Akhtar M, Ali Z, Hassan N, Mehdi S, Wani GM, Mir AH. A Randomized Study Comparing the Sniffing Position with Simple Head Extension for Glottis Visualization and Difficulty in Intubation during Direct Laryngoscopy. *Anesth Essays Res*. 2017;11(3):762-766. doi:10.4103/0259-1162.204206
4. Bhattarai B, Shrestha SK, Kandel S. Comparison of sniffing position and simple head extension for visualization of glottis during direct laryngoscopy. *Kathmandu Univ Med J KUMJ*. 2011;9(33):58-63.
5. El-Orbany MI, Getachew YB, Joseph NJ, Salem MR, Friedman M. Head elevation improves laryngeal exposure with direct laryngoscopy. *J Clin Anesth*. 2015;27(2):153-158. doi:10.1016/j.jclinane.2014.09.012
6. Nur Hafiizhoh AH, Choy CY. Comparison of the "sniffing the morning air" position and simple head extension for glottic visualization during direct laryngoscopy. *Middle East J Anaesthesiol*. 2014;22(4):399-405.
7. Kim H, Chang J-E, Min S-W, Lee J-M, Ji S, Hwang J-Y. A comparison of direct laryngoscopic views in different head and neck positions in edentulous patients. *Am J Emerg Med*. 2016;34(9):1855-1858. doi:10.1016/j.ajem.2016.06.011
8. Park S-W, Lee K-W, Jang M-S, Jung J-Y, Lee B-J, Kang J-M. Age and gender are important considerations in choosing the sniffing position for laryngoscopic view. *Int J Med Sci*. 2014;11(12):1258-1261. doi:10.7150/ijms.8585
9. Prakash S, Rapsang AG, Mahajan S, Bhattacharjee S, Singh R, Gogia AR. Comparative evaluation of the sniffing position with simple head extension for laryngoscopic view and

intubation difficulty in adults undergoing elective surgery. *Anesthesiol Res Pract*. 2011;2011:297913. doi:10.1155/2011/297913
10. Vikasgupta, Ruchisharma, Shrutisharma. Comparison of Sniffing Position and Head-Neck Position for Direct Laryngoscopy. *IOSR J Dent Med Sci*. 2019;18(4):43-46. doi:10.9790/0853-1804144346
11. Lee J-H, Jung H-C, Shim J-H, Lee C. Comparison of the rate of successful endotracheal intubation between the "sniffing" and "ramped" positions in patients with an expected difficult intubation: a prospective randomized study. *Korean J Anesthesiol*. 2015;68(2):116. doi:10.4097/kjae.2015.68.2.116
12. Park S, Lee HG, Choi JI, et al. Comparison of vocal cord view between neutral and sniffing position during orotracheal intubation using fiberoptic bronchoscope: a prospective, randomized cross over study. *BMC Anesthesiol*. 2019;19(1):3. doi:10.1186/s12871-018-0671-9
13. Mendonca C, Ungureanu N, Nowicka A, Kumar P. A randomised clinical trial comparing the "sniffing" and neutral position using channelled (KingVision®) and non-channelled (C-MAC®) videolaryngoscopes. *Anaesthesia*. 2018;73(7):847-855. doi:10.1111/anae.14289

Mentor: Sharon Hadenfeldt, PhD, CRNA

Editorial

Do you remember what you were doing in December 2019? It was during that time that the Coronavirus disease (COVID-19) outbreak was first made public across the globe.
Do you remember your New Year's resolutions and your plans for 2020? Whatever they were, they were drastically changed as the World Health Organization (WHO) declared the COVID-19 outbreak a Public Health Emergency of International Concern in January 2020 and then classified it as a pandemic in March 2020. The focus and conduct of our lives transformed to revolve around what needed to be done considering the ongoing pandemic.
This issue of the Journal includes the usual array of topics in addition to several articles that include practice considerations that we likely were unaware of prior to the current world situation.

During the pandemic we have quickly had to assimilate volumes of information and opinions barraging us every day. While the volume and pace of new information is not what it was at first, we still must stay attuned to what is new. We must continue to evaluate and incorporate the latest recommendations.

We transitioned from the focus of our care being primarily on the safety of our patients to include the safety of ourselves, our co-workers, our families, and the community. Hand washing and use of personal protective equipment (PPE) went from something we expected to hear from the Infection Control department to something we heard on the nightly news.
Our reliance on evidence-based practice was substituted by care based on the best guess of what we could gather from the latest recommendations and what was available for use (masks, gowns, video laryngoscopes, intubation boxes, COViD-19 tests with variable reliability…) as we cared for patients.

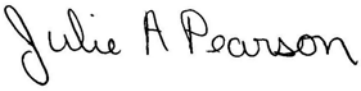
Some in anesthesia and throughout healthcare faced reduced hours, furloughs, and loss of employment. The challenges faced by nurse anesthesia programs and students included clinical restrictions, limitations of simulation experiences, and more remote learning. Those students who were nearing graduation were challenged to complete their studies, especially completion of some clinical experiences.

Luckily, most OR schedules are back to near normal volume. Many require pre-operative COVID-19 testing. Our new "normal" continues to evolve. In general nurse anesthetists are back to work and students are back to class and clinical, but there is an underlying concern that circumstances could change at any moment.

We have progressed to phased re-openings of businesses. We have had our temperatures taken more times than we can count. Many things that we once took for granted, like hair appointments or gathering with friends, are things we genuinely appreciate.

As we continue to progress through the trials of this pandemic, we hope that all are staying safe and making the best of the current situation. Our lives have changed dramatically this year. We look forward to a resolution of this pandemic with a renewed sense of purpose and appreciation for what we have learned during this challenging time.

Sincerely,



Julie A. Pearson, PhD, CRNA
Associate Editor

Vicki Callan Coopmans, PhD, CRNA, CHSE
Editor

"***The International Student Journal of Nurse Anesthesia*** **is produced exclusively for publishing the work of nurse anesthesia students. It is intended to be basic and introductory in its content. Its goal is to introduce the student to the world of writing for publication; to improve the practice of nurse anesthesia and the safety of the patients entrusted to our care.**"

INTERNATIONAL STUDENT JOURNAL OF NURSE ANESTHESIA
GUIDE FOR AUTHORS

MISSION STATEMENT

The International Student Journal of Nurse Anesthesia (ISJNA) is produced exclusively for publishing the work of nurse anesthesia students. It is intended to be basic and introductory in its content. Its goal is to introduce the student to the world of writing for publication; to improve the practice of nurse anesthesia and the safety of the patients entrusted to our care.

ITEM PREPARATION & SUBMISSION

Case reports, research abstracts, evidence-based practice (EBP) analysis reports, evidence-based practice project abstracts, and letters to the editor may be submitted. These items must be authored by a student under the guidance of an anesthesia practitioner mentor (CRNA or physician). Case and EBP analysis reports must be single-authored, while abstracts may have multiple authors. Submissions may list only one mentor. **Mentors should take an active role** in reviewing the item to ensure appropriate content, writing style, and format prior to submission. The mentor must submit the item for the student and serve as the contact person during the review process. Items submitted to this journal should not be under consideration with another journal. Authors and mentors should critically evaluate the topic and quality of the writing – multiple reviews of the item by the mentor, faculty, and peers (fellow graduate students) prior to submission is recommended. If the topic and written presentation are beyond the introductory publication level we strongly suggest that the article be submitted to a more prestigious publication such as the *AANA Journal*.

The journal is committed to publishing the work of nurse anesthesia students. The review process is always initiated with the following rare exceptions. We are conservative in accepting reports where the patient has expired, realizing that you can do everything right and still have a negative outcome. Submissions that report a case demonstrating failure to meet the standard of care (by any practitioner involved in the case) will not be accepted. Unfortunately, while the experiences in these cases can offer valuable insight, these submissions will not be accepted for review due to potential legal risks to the author, journal, and anyone else involved in evaluating the report.

It is the intent of this journal to publish items while the author is still a student. In order to consistently meet this goal, all submissions must be received by the editor at least **3 months prior** (4-6 months recommended) to the author's date of graduation. Manuscripts must be submitted by the mentor of the student author via e-mail to **INTSJNA@aol.com** as an attachment. The subject line of the e-mail should use the following format: ISJNA Submission_submission type_author last name_mentor last name. The item should be saved in the following format – two-three word descriptor of the article_author's last name_school abbreviation_mentor's last name_date (e.g. PedsPain_Smyth_GU_Pearson_5.19.09)

REVIEW PROCESS

Items submitted for publication are initially reviewed by the chief editor. If the chief editor does not acknowledge receipt of the item within two weeks, please inquire to ensure receipt. Upon receipt, the chief editor will review the submission for compliance with the Guide to Authors. If proper format has not been followed, the item will be returned to the mentor for correction. This is very important as all reviewers serve on a volunteer basis. Their time should be spent ensuring appropriate content, not making format corrections. It is the mentor's responsibility to ensure formatting guidelines have been followed prior to submission.

All accepted submissions undergo a formal process of blind review by at least two reviewers. After review, items may be accepted without revision, accepted with revision, or rejected with comments. Once the item has been accepted for review the chief editor will send a blinded copy to an editor, who will then coordinate a blinded review by two reviewers who are not affiliated with the originating program. The editor will return the item to the chief editor, who will return it to the mentor for appropriate action. Every effort is made to complete the process in an efficient, timely matter. Again, the goal is for all articles submitted by students to be published while the author is still a student. If an item is not ready for publication within 6 months after the student author has graduated it will no longer be eligible for publication. Mentors will be listed as contributing editors for the issue in which the item is published.

PHOTOS

Photos of students for the front cover of the Journal are welcome. Please contact the chief editor at intsjna@aol.com to submit photos for consideration. Only digital photos of high quality will be accepted. If the photo is accepted, consent forms must be completed and returned by all identifiable individuals in the photo, and the individual who took the photo.

ACADEMIC INTEGRITY

Issues of academic integrity are the responsibility of the author and mentor. Accurate and appropriate acknowledgement of sources is expected. The two most common breaches of academic integrity that have been identified in submissions to this journal are (AMA 10th ed., p. 158):

1. Direct plagiarism: verbatim lifting of passages without enclosing the borrowed material in quotation marks and crediting the original author.
2. Paraphrase: restating a phrase or passage, providing the same meaning but in a different form without attribution to the original author.

Please note that changing one or two words in a reference source passage (e.g. 'of' for 'in', or 'classified' for 'categorized') and then citing it as a paraphrase or summary is also not appropriate, and still falls within the definition of direct plagiarism. If plagiarism in any form is identified, review of the item will be suspended and it will be returned to the mentor. Repeated instances of plagiarism will result in rejection of the item.

Plagiarism detection software (TurnItIn, PlagScan, SafeAssign, etc . . .) can be used to analyze the document prior to submission to ensure proper citation and referencing, but is not required.

"Plagiarism is the presentation of someone else's ideas, writings, or statements as one's own. Plagiarism is a serious breach of academic integrity, and anyone who is found to have committed plagiarism will be subject to disciplinary action.
Paraphrase is the act of putting someone else's ideas into one's own words. The use of paraphrase can be an acceptable practice under some circumstances if it is used sparingly and if the original text is properly acknowledged. Unacknowledged paraphrase, like plagiarism, is a serious breach of academic integrity. Any improper use of sources may constitute plagiarism. Every quotation from another source, whether written, spoken, or electronic, must be bound by quotation marks and be properly cited. Mere citation alone is not sufficient when a scholar has used another person's words. Similarly, every paraphrase or summary (a more concise restatement of another's ideas) must be properly cited."
https://sites.google.com/a/georgetown.edu/gsas-graduate-bulletin/vi-academic-integrity-policies-procedures

GENERAL GUIDELINES

Items for publication **must adhere to the *American Medical Association Manual of Style*** (AMA 10th ed., the same guide utilized by the *AANA Journal* and such prominent textbooks as *Nurse Anesthesia* by Nagelhout and Plaus). Page numbers are provided for easy reference in the AMA Manual of Style throughout this document. The review process will not be initiated on items submitted with incorrect formatting and will be returned to the mentor for revision. Please note the following:

1. Use complete sentences.
2. Acronyms/Initialisms (p. 379) - spell out with first use, do not capitalize the words from which the acronym/initialism is derived unless it is a proper noun or official name. If you are using the phrase only once, do not list the acronym/initialism at all. Avoid beginning sentences with acronym/initialisms.
3. Abbreviations (p. 441)
4. Use *Index Medicus* journal title abbreviations (p. 472, http://www.ncbi.nlm.nih.gov/nlmcatalog/journals)
5. Always provide units of measure (p. 521 & 795). In most cases The International System of Units (SI) is used. Abbreviations for units of measure do not need to be spelled out with first use. Report height in cm, weight in kg, temperature in ºC, pressure in mm Hg or cm H_2O. Report heart and respiratory rate as X/min (e.g. the patient's heart rate increased to 145/min).
6. In general, first use of pulmonary/respiratory abbreviations should be expanded, with the following exceptions: O_2, CO_2, PCO_2, $PaCO_2$, PO_2, PaO_2, $EtCO_2$, N_2O. Please use SpO_2 for oxygen saturation as measured by pulse oximetry.
7. Use the nonproprietary (generic) name of drugs (p. 568) - avoid proprietary (brand) names. Type generic names in lowercase. When discussing dosages state the name of the drug, *then* the dosage (midazolam 2 mg).

8. Use of descriptive terms for equipment and devices is preferred. If the use of a proprietary name is necessary (for clarity, or if more than one type is being discussed), give the name followed by the manufacturer and location in parenthesis (p. 583, e.g. a GlideScope (Verathon Inc., Bothell, WA) was used) Please note, TM and ® symbols are not used per the AMA manual.
9. Infusion rates and gas flow rates:
    a. Use mcg/kg/min or mg/kg/min for infusion rates. In some cases it may be appropriate to report dose or quantity/hr (i.e. insulin, hyperalimentation). If a mixture of drugs is being infused give the concentration of each drug and report the infusion rate in ml/min.
    b. Report gas flow of O_2, N_2O and Air in L/min (not %) and volatile agents in % as inspired or expired concentration (e.g. General anesthesia was maintained with sevoflurane 3% inspired concentration in a mixture of O_2 1 L/min and air 1 L/min.)
10. Only Microsoft Word file formats will be accepted with the following criteria:
    a. Font - 12 point, Times New Roman
    b. Single-spacing (except where indicated), paragraphs separated with a double space (do not indent)
    c. One-inch margins
    d. End the sentence with the period before placing the superscript number for the reference.
    e. Do not use columns, bolds (except where indicated), or unconventional lettering styles or fonts.
    f. Do not use endnote/footnote formats.
11. Do not use Endnotes or similar referencing software – any embedded formatting must be removed prior to submission.
12. Remove all hyperlinks within the text.
13. Avoid jargon and slang terms. Use professional, scholarly, scientific language.
    a. *'The patient was reversed'* - Did you physically turn the patient around and point him in the opposite direction? "Neuromuscular blockade was antagonized."
    b. *The patient was put on oxygen.* "Oxygen 2 L/min was administered via face mask."
    c. *The patient was intubated and put on a ventilator*. "The trachea was intubated and mechanical ventilation was initiated.
    d. *An IV drip was started.* "An intravenous infusion was initiated."
    e. Avoid the term "MAC" when referring to a sedation technique - the term sedation (light, moderate, heavy, unconscious) may be used. Since all anesthesia administration is monitored, pharmacologic, rather than reimbursement, terminology should be used.
14. Direct quotes are discouraged for reports of this length – please express in your own words.
15. Use the words "anesthesia professionals" or "anesthesia practitioners" when discussing all persons who administer anesthesia (avoid the reimbursement term "anesthesia providers").
16. Do not include ASA Physical Status unless it is germane to the report.
17. Do not use the phrase "ASA standard monitors were applied". Instead, "standard noninvasive monitors" is acceptable – additional monitoring can be detailed as needed.
18. References
    a. The **AMA Manual of Style must be adhered to** for reference formatting.
    b. All sources should be published within the past 8 years. Seminal works essential to the topic being presented will be considered.
    c. Primary sources are preferred.
    d. **A maximum of one textbook (must be most recent edition available) may be used as reference for case report submissions only**.
    e. All items cited must be from peer-reviewed sources – use of sources found on the internet must be carefully considered in this regard. URLs must be current and take the reader directly to the referenced source.

Heading – for all submission types (Case Report, Abstract, EBPA Report) use the following format.

1. **Title** is bolded, centered, 70 characters (including spaces) or less
2. Author name (academic credentials only) and NAP are centered, normal font,.
3. *Graduation date and email address* are centered, italicized, and will be removed prior to publication)
4. **Keywords** is left-justified, bolded – list keywords that can be used to identify the report in an internet search

Title

Author Name

Name of Nurse Anesthesia Program
Anticipated date of graduation
E-mail address

Keywords: keyword one, keyword two, etc . . .

Case Reports - The student author must have had a significant role in the conduct of the case. The total word count should be between 1200 – 1400 words (references not counted). Case reports with greater than 1400 words will be returned to the mentor for revision prior to initiation of the review process. The following template demonstrates the required format for case report submission.

Heading (see above)

A brief introductory paragraph of less than 100 words to focus the reader's attention and interest them to continue reading. This may include historical background, demographics or epidemiology (with appropriate references) of the problem about to be discussed. It is written in the *present tense*. Although it is introductory, the heading word '***Introduction'*** is not used. Be certain to cite references in this section, especially statistics and demographics.
[space]
Case Report (bold, 400-600 words)
[space]
This portion discusses the case performed and is written in the *past tense*. Do not justify actions or behaviors in this section; simply report the events as they unfolded. Present the case in an orderly sequence. Some aspects need considerable elaboration and others only a cursory mention. Under most circumstances if findings/actions are normal or not contributory to the case then they should not be described. Events significant to the focus of the report should be discussed in greater detail. The purpose of the case report is to set the stage (and 'hook' the reader) for the heart of your paper which is the discussion and teaching/learning derived from the case.

- Give dosage and schedule only if that information is pertinent to the consequences of the case.
- **Significant** laboratory values, x-rays or other diagnostic testing pertinent to the case. Give the units of measure after the values (eg. Mmol/L or mg/dL).
- Physical examination/pre-anesthesia evaluation - **significant** findings only.
- Anesthetic management (patient preparation, induction, maintenance, emergence, post-operative recovery).

[space]
Discussion (bold, 600-800 words)
[space]
Describe the ***anesthesia*** implications of the focus of the case report citing current literature. Describe the rationale for your actions and risk/benefits of any options you may have had. This section is not merely a pathophysiology review that can be found in textbooks. *Relate the anesthesia literature with the conduct of your case noting how and why your case was the same or different from what is known in the literature.* Photographs are discouraged unless they are essential to the article. Photos with identifiable persons must have a signed consent by the person photographed forwarded to the editor via first class mail. Diagrams must have permission from original author. This is the most important part of the article. In terms of space and word count this should be longer than the case presentation. End the discussion with a summary lesson you learned from the case, perhaps what you would do differently if you had it to do over again.
[space]
References (bold)
[space]
A minimum of 5 references is recommended, with a maximum of 8 allowed. One textbook may be used as a reference – it must be the most recent edition. All references should be no older than 8 years, except for seminal works essential to the topic. This is also an exercise in searching for and evaluating current literature.
[space]
Mentor: (bold, followed by mentor name and credentials in normal text)
E-mail address: (normal text, will be removed prior to publication)

EBP Analysis Reports - Evidence-based practice analysis reports are limited to 3000 words. Please do not include an abstract. The report should provide a critical evaluation of a practice pattern in the form of a clinical question about a specific intervention, population, and outcome. The manuscript should:

1. Articulate the practice issue and generate a concise question for evidence-based analysis. A focused foreground question following either the PICO or SPICE format should be used.
2. Describe the methods of inquiry used in compiling the data.
3. Critically analyze the quality of research reviewed and applicability to different practice settings.
4. Draw logical conclusions regarding appropriate translation of research into practice.

The same general format guidelines apply with the exception of the section headings as below. Textbooks and non-peer reviewed internet sources may not be used, and sources of reference should be less than 8 years old unless they are seminal works specifically related to your topic of inquiry. A maximum of 16 references is allowed.

Heading

Introduction (bold)
[space]
Briefly introduce the reader to the practice issue or controversy, describe the scope or significance or problem, and identify the purpose of your analysis. Describe the theoretical, conceptual, or scientific framework that supports your inquiry.
[space]
Methods (bold)
[space]
Include the format used for formulating the specific question you seek to answer, search terms and methods used, and levels of evidence.
[space]
Literature Analysis (bold)
[space]
Analyze and critique the literature relevant to your question, determining scientific credibility and limitations of studies reviewed. Your synthesis table is included in this section. Your review and discussion of the literature should logically lead to support a practice recommendation. Subheadings may be used if desired.
[space]
Conclusions (bold)
[space]
Summarize the salient points that support the practice recommendation and make research-supported recommendations that should improve the practice issue, while also acknowledging any limitations or weaknesses
[space]
References (bold, 16 maximum)
[space]
Mentor: (bold, followed by mentor name and credentials in normal text)
E-mail address: (normal text, will be removed prior to publication)

Evidence Based Practice Project Abstracts - Evidence-based practice project abstracts are limited to 600 words. References do not impact the word count - a maximum of 5 are allowed. Note that the abstract is different from a project proposal. The following format should be used:

Heading

Introduction (bold)
[space]
A brief introductory paragraph including purpose (what change is intended) and rationale (why change is needed/evidence to support the change) here.
[space]
Design and Methods (bold)
[space]
Include population, intervention, and measures
[space]
Outcome (bold)

[space]
Present results from statistical analysis – do not justify or discuss here.
[space]
Conclusion (bold)
[space]
Discuss results (implications). Optionally include limitations, suggestions for future projects/research.
[space]
References (bold, 5 maximum)
[space]
Mentor: (bold, followed by mentor name and credentials in normal text)
E-mail address: (normal text, will be removed prior to publication)

Research Abstracts - Research abstracts are limited to 600 words. References do not impact the word count - a maximum of 5 are allowed. Note that the abstract is different from a research proposal. The following format should be used:

Heading

Introduction (bold)
[space]
A brief introductory paragraph including purpose and hypotheses.
[space]
Methods (bold)
[space]
Include sample and research design
[space]
Results (bold)
[space]
Present results from statistical analysis – do not justify or discuss here.
[space]
Discussion (bold)
[space]
Discuss results (implications, limitations, suggestions for future research)
[space]
References (bold, 5 maximum)
[space]
Mentor: (bold, followed by mentor name and credentials in normal text)
E-mail address: (normal text, will be removed prior to publication)

Letters to the Editor - Students may write letters to the editor topics of interest to other students. Topics may include comments on previously published articles in this journal. Personally offensive, degrading or insulting letters will not be accepted. Suggested alternative approaches to anesthesia management and constructive criticisms are welcome.
The length of the letters should not exceed 100 words and must identify the student author and anesthesia program.

AMA MANUAL OF STYLE
The following is brief introduction to the *AMA Manual of Style* reference format along with some links to basic, helpful guides on the internet. The website for the text is http://www.amamanualofstyle.com/oso/public/index.html. It is likely your institution's library has a copy on reserve. Some helpful websites are listed below:
https://guides.nyu.edu/amastyle
https://owl.english.purdue.edu/owl/resource/1017/01/

Journal names should be in *italics* and abbreviated according to the listing in the PubMed Journals Database. The first URL below provides a tutorial on looking up correct abbreviations for journal titles; the second is a link to the PubMed where you can perform a search.
http://www.nlm.nih.gov/bsd/viewlet/search/journal/journal.html
http://www.ncbi.nlm.nih.gov/pubmed

The International Student Journal of Nurse Anesthesia (ISJNA) is not listed in the PubMed Database. For the purpose of citing the ISJNA *in this Journal* use "**Int Student J Nurse Anesth**" as the abbreviation.

Journals - A comma is placed after the first initials until the last author, which has a period. If there are six or less authors **cite all six**. If there are more than six authors **cite only the first three** followed by "et al." Only the first word of the title of the article is capitalized. The first letters of the major words of the journal title are capitalized. There is no space between the year, volume number, issue number, and page numbers. If there is no volume or issue number, use the month. If there is an issue number but no volume number use only the issue number (in parentheses). Page numbers are inclusive - **do not omit digits** (note - some online journals do not use page numbers). Some journals may be available both as hard copies and online. When referencing a journal that has been accessed online, the DOI (digital object identifier) or PMID (PubMed identification number) should be included (see example below).

Journal, 6 or fewer authors:
Han B, Liu Y, Zhang X, Wang J. Three-dimensional printing as an aid to airway evaluation after tracheotomy in a patient with laryngeal carcinoma. BMC Anesthesiol. 2016;16(6). doi:10.1186/s12871-015-0170-1.

Journal, more than 6 authors:
Chen C, Nguyen MD, Bar-Meir E, et al. Effects of vasopressor administration on the outcomes of microsurgical breast reconstruction. *Ann Plast Surg*. 2010;65(1):28-31. PMID: 20548236.

Elayi CS, Biasse L, Bai R, et al. Administration of isoproterenol and adenosine to guide supplemental ablation after pulmonary vein antrum isolation. *J Cardiovasc Electrophysiol*. 2013;24(11):1199-1206. doi: 10.1111/jce.12252.

Electronic references - Only established, peer-reviewed sources may be referenced. Please do not reference brochures, fact sheets, or informational websites where a peer-review process cannot be confirmed. The URL must be functional and take the reader directly to the source of the information cited. The accessed date may be the only date available.

Author (or if no author, the name of the organization responsible for the site). Title. *Name of Website*. Year;vol(issue no.):inclusive pages. URL. Published [date]. Updated [date]. Accessed [date].

Examples:
Kamangar N, McDonnell MS. Pulmonary embolism. *eMedicine*. http://www.emedicine.com/med/topic1958.htm. Updated August 25, 2009. Accessed September 9, 2009

Howlader N, Noone AM, Krapcho M, Garshell J, Miller D, et al. SEER Cancer statistics review, 1975-2012. National Cancer Institute. http://seer.cancer.gov/csr/1975_2012/. Published April 2015. Updated November 18, 2015. Accessed February 29, 2016.

Textbooks - There are two types of books – 1) those that are fully authored by one or more individuals, and 2) those that are edited by one or more individuals, with chapters authored by different individuals. Edited textbooks give primary credit to the chapter authors, who are listed first, and the inclusive page numbers of the entire chapter are provided at the end. Textbooks that are authored do not have different chapter authors and the chapter titles are not listed, but the inclusive page numbers where the information was found are provided, unless the entire book is cited.

Authored text:
Shubert D, Leyba J, Niemann S. *Chemistry and Physics for Nurse Anesthesia*. 3rd ed. New York, NY: Springer; 2017:405-430.

Chapter from an edited text:
Pellegrini JE. Regional anesthesia. In Nagelhout JJ, Elisha S, eds. *Nurse Anesthesia*. 6th ed. St. Louis:Elsevier; 2017:1015-1041.

SUBMISSION CHECK LIST

___ **Adheres to AMA Manual of Style and all other format instructions**
___ Total word count not exceeded (1400 for case report, 600 for abstracts, 3000 for EBPA report)
___ The item is one continuous Word document without artificially created page breaks
___ All matters that are not common knowledge to the author are referenced appropriately
___ Generic names for drugs and products are used throughout and spelled correctly in lower-case
___ Units are designated for all dosages, physical findings, and laboratory results
___ Endnotes, footnotes not used
___ Jargon/slang is absent

Heading
___ Concise title less than 70 characters long
___ Author name, credentials, nurse anesthesia program, graduation date and email are included
___ Three to five **Keywords** are provided

Case Report
___ Introduction is less than 100 words.
___ Case Report section states only those facts vital to the account (no opinions or rationale)
___ Case report section is 400-600 words and not longer than the discussion
___ Discussion section is 600-800 words
___ Discussion of the case management is based on a review of current literature
___ Discussion concludes with lessons learned and how the case might be better managed in the future

Abstracts
___ The 600 word count maximum is not exceeded
___ Appropriate format used depending on type of abstract (research vs. EBP project)

EBPA Report
___ The 3000 word count maximum is not exceeded
___ A critical evaluation of a practice pattern in the form of a precise clinical question about a specific intervention, population, and outcome is presented
___ A focused foreground question following either the PICO or SPICE format is used
___ Includes Introduction, Methodology, Literature Analysis (with synthesis table), and Conclusion sections

References
___ Adheres to AMA Style format
___ Reference numbers are sequenced beginning with 1 and superscripted
___ References are from anesthesia and other current (within past 8 years) primary source literature
___ Journal titles are abbreviated as they appear in the PubMed Journals Database
___ Number of references adheres to specific item guidelines (1 textbook allowed for case reports only)
___ Internet sources are currently accessible, reputable, and peer reviewed

Transmission
___ The article is sent as a attachment to **INTSJNA@AOL.COM**
___ The file name is correctly formatted (e.g. PedsPain_Smyth_GU_Pearson_5.19.09)
___ Item is submitted by the mentor
___ Subject heading format - ISJNA Submission_submission type_author last name_mentor last name