

ANESTHESIA ABSTRACTS

www.AnesthesiaAbstracts.com

ISSN Number: 1938-7172

Providing the discerning anesthetist with up-to-date resources for evidence based practice.

January 31, 2010

Volume 4

Number 1

Editor:

Michael A. Fiedler, PhD, CRNA

Contributing Editors:

Penelope S. Benedik PhD, CRNA, RRT
Joseph F. Burkard, DNSc, CRNA
Mary A. Golinski, PhD, CRNA
Gerard T. Hogan, Jr., DNSc., CRNA
Alfred E. Lupien, PhD, CRNA
Dennis Spence, PhD, CRNA
Steven R. Wooden, MS, CRNA

Guest Editors:

A Publication of Lifelong Learning, LLC © Copyright 2010

New health information becomes available constantly. While we strive to provide accurate information, factual and typographical errors may occur. The authors, editors, publisher, and Lifelong Learning, LLC is/are not responsible for any errors or omissions in the information presented. We endeavor to provide accurate information helpful in your clinical practice. Remember, though, that there is a lot of information out there and we are only presenting some of it here. Also, the comments of contributors represent their personal views, colored by their knowledge, understanding, experience, and judgment which may differ from yours. Their comments are written without knowing details of the clinical situation in which you may apply the information. In the end, your clinical decisions should be based upon your best judgment for each specific patient situation. We do not accept responsibility for clinical decisions or outcomes.

TABLE OF CONTENTS

Editorial

- THIS MAY BE THE MOST IMPORTANT THING YOU READ TODAY

Cardiovascular

- ANESTHESIA FOR CAROTID ENDARTERECTOMY: THE THIRD OPTION. PATIENT COOPERATION DURING GENERAL ANESTHESIA

General

- UNEXPECTED CARDIOVASCULAR COLLAPSE FROM MASSIVE AIR EMBOLISM DURING ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY

Orthopedics

- CEREBRAL ISCHEMIA DURING SHOULDER SURGERY IN THE UPRIGHT POSITION: A CASE SERIES

- MATERNAL AND NEONATAL EFFECTS OF BOLUS ADMINISTRATION OF EPHEDRINE AND PHENYLEPHRINE DURING SPINAL ANAESTHESIA FOR CAESAREAN DELIVERY: A RANDOMISED STUDY

- EFFECT OF COMBINING DEXMEDETOMIDINE AND MORPHINE FOR INTRAVENOUS PATIENT-CONTROLLED ANALGESIA

Policy, Process, & Economics

- EFFECTS OF INTRAOPERATIVE READING ON VIGILANCE AND WORKLOAD DURING ANESTHESIA CARE IN AN ACADEMIC MEDICAL CENTER

Regional Anesthesia

- ADVERSE EVENTS ASSOCIATED WITH INTRAVENOUS REGIONAL ANESTHESIA (BIER BLOCK): A SYSTEMATIC REVIEW OF COMPLICATIONS

Pharmacology

-  • KETAMINE, BUT NOT PRIMING, IMPROVES INTUBATING CONDITIONS DURING A PROPOFOL-ROCURONIUM INDUCTION

 Indicates Continuing Education Credit is available for this abstract and comment during the CE approval period.

Continuing Education Credit is available to individual subscribers on the Anesthesia Abstracts web site at www.AnesthesiaAbstracts.com.

THIS MAY BE THE MOST IMPORTANT THING YOU READ TODAY

EDITORIAL

In advertising, a title like this is called a “hook”. A successful hook draws readers to the material and convinces the reader they are reading an invaluable source of information. Unfortunately, many scientific publications find themselves caught between providing scientifically relevant material and making it interesting enough to keep readers coming back, often leaning toward the interesting rather than the relevant. Last year, Anesthesia Abstracts included an abstract about the relevance of clinical data in “respected” anesthesia journals. The study concluded that less than 20% of articles published in such journals were clinically relevant (Lauritsen & Moller, 2004).

With our busy schedules and limited time to read we may find ourselves reading only the study titles and the short explanation of the content provided by some journals. Without dissecting the contents for appropriate study methods and clinical relevance, the title and limited explanation of a particular article might lead us to believe the information is appropriate for us to include in our clinical practices. A title such as “Anesthesiologist-associated risk factors for inadequate postoperative pain management” might peak our interest. Reading part of the abstracted conclusion may lead you to believe that a study has actually found an association between age, experience, educational background, the work environment of anesthesia providers and inadequate postoperative pain management. Those of us who read the entire article found little resemblance between the title, short conclusion, and the actual relevance of the so called study. One more example was another study abstracted by Anesthesia Abstracts, an article warning of the potential bleeding hazards from small doses of dexamethasone used to reduce the incidence of nausea and vomiting in tonsillectomy patients (Czarnetzki et al., 2008). The article warned of dire consequences, but when the study was reviewed there were more questions revealed than answers. The researchers had overlooked a plethora of variables within the study.

Don’t be complacent in developing your medical knowledge. As individual providers, we need to evaluate the body of evidence available on each subject (Evidenced Based Practice). The contributing editors of *Anesthesia Abstracts* carefully evaluate the clinical questions and study methods of each article they abstract. The contributors present both the advantages and the weaknesses of each article, while also adding insight based upon personal experience in our practices. One of our goals is to encourage you to question what you read in scientific journals and other health care sources, and continue to look for broader bases of information to improve your personal practice. As always, we encourage your contribution to *Anesthesia Abstracts* by requesting specific topics of interest.

Steven Wooden, MS, CRNA

Czarnetzki, C., Elia, N., Lysakowski, C., Dumont, L., Landis, B. N., Giger, R., et al.. Dexamethasone and risk of nausea and vomiting and postoperative bleeding after tonsillectomy in children: a randomized trial. *JAMA* 2008;300:2621-2630.

Lauritsen, J., & Moller, A. M. Publications in anesthesia journals: quality and clinical relevance. *Anesth Analg* 2004;99:1486-1491.

Mr. Wooden is a rural anesthetist in independent practice and a doctoral student. He has been an Anesthesia Abstracts contributing editor since 2007.

ANESTHESIA FOR CAROTID ENDARTERECTOMY: THE THIRD OPTION. PATIENT COOPERATION DURING GENERAL ANESTHESIA

Anesth Analg 2009;108:1929-36

Bevilacqua S, Romagnoli S, Ciappi F, Gelsomino S, Pratesi C, Gensini G

Abstract

Purpose The purpose of this study was to assess the feasibility, safety, patient, and surgeon satisfaction of a new anesthetic technique for carotid artery surgery. The technique was a modified total intravenous general endotracheal anesthetic, however, during carotid artery clamping, the hypnotic component of the anesthetic, a propofol infusion, was temporarily suspended. The patient remained narcotized via a remifentanil infusion but awake and able to follow commands. Intraoperative neurological monitoring ensued. The technique was called Cooperative Patient General Anesthesia (Co.PA.Ge.A.).

Background Carotid endarterectomy (CEA) is one the most common vascular surgery procedures performed for those at risk of embolic stroke from an atherosclerotic plaque at the carotid artery bifurcation. Perioperatively, stroke and major cardiac events such as myocardial infarction, are the most feared and common complications of CEA. The procedure can be done under regional anesthesia, typically superficial and deep cervical plexus block, or general endo- tracheal anesthesia, or a combination of the two.

Performing CEA under regional anesthesia allows for continuous neurologic monitoring and can guide the surgeon in deciding whether or not to shunt when clamping the common carotid artery. The disadvantage of regional anesthesia, is that it can be extremely stressful and uncomfortable for the patient. General endotracheal anesthesia has the advantage of protection of the airway, and since the patient is fully anesthetized, there is no anxiety or agitation experienced. However, it is difficult for the surgeon to make the clinical decision whether or not to shunt. There exists a plethora of monitoring devices to give quantitative information regarding intra-operative cerebral ischemia, however none of the technology is fully effective and reliable. This study proposed a technique of general endotracheal anesthesia using total intravenous anesthesia (no inhalation agents), which included remifentanil as the analgesic. The propofol or hypnotic component of the anesthetic was discontinued during the time when neurological assessment was necessary. The goal was to provide an acceptable level of analgesia while patients were awake and being assessed for neurologic changes. It was assumed that if neurologic changes occurred during carotid clamping, a shunt would be inserted and the neurologic outcomes favorable.

Methodology There were 181 patients enrolled in this study. Those with high grade carotid artery stenosis determined by carotid duplex testing met the inclusion criteria. In order to assess for the primary and secondary outcome variables, the following anesthesia protocol was followed:

Time 0	premedication with oral diazepam
Time 1	insertion of peripheral IVs and arterial line
Time 2	operating room 'in time'-standard monitoring initiated
Time 3	infusion of remifentanil begun to achieve 8 ng/mL plasma concentration (see notes)
Time 4	propofol induction and infusion initiated. Remifentanil on infusion pump to maintain the achieved Ce (see notes)
Time 5	surgeon infiltrates local anesthesia at incision site

Time 6	incision
Time 7	propofol ceased 20 minutes prior to cross clamping
Time 8	patient remains intubated, maintained on remifentanil infusion, neurologic monitoring conducted by asking patient to perform simple commands i.e. squeeze hands
Time 9	carotid artery clamp time. Neurologic continual assessments and insert shunt if necessary
Time 10	carotid reopening, re-initiate propofol infusion
Time 11	end of surgery, stop remifentanil and propofol infusion
Time 12	emergence

The duration of anesthesia and surgical time was recorded, as was carotid clamp time, shunt insertions, and hemodynamic variables during carotid clamping.

Upon arrival to the post anesthesia care unit, a comprehensive neurologic examination was completed by the anesthesia provider and repeated prior to discharge from the hospital by a neurologist. On postoperative day number 1, patients were asked to complete an extensive questionnaire regarding their awareness and satisfaction with the anesthetic technique. Components of the patient satisfaction questionnaire included asking about their perception during anesthesia in general, specific inquiries in to auditory awareness, visual awareness- stimuli experienced, tactile perception, whether or not they felt paralyzed (no paralytics were administered), pain perception during the procedure, feelings, thoughts and effects of the procedure. Additionally the surgeons were asked to rate their level of satisfaction with this technique.

Results In terms of the feasibility and safety of the technique, Co.Pa.Ge.A., was successfully carried out in all but two patients. During carotid clamping those two patients suffered anxiety and agitation. They were anesthetized with inhalation agent and the surgeon used a shunt. The median target remifentanil Ce at clamp time was 7.7 ng/mL; this corresponded to a median infusion dose of 0.24/mcg/kg/min. When the remifentanil infusion was initiated prior to induction, respiratory depression was observed but patients responded to commands to breathe. Target analgesic concentration was easily determined via pinches to the trapezius muscle while patients were assessed for response. There were an additional four conversions to general anesthesia with neuroprotective techniques utilized when a neurologic deficit noted during clamping and insertion of the shunt did not reverse the deficits. None of these patients showed persistent deficits upon awakening. The following safety outcomes were also noted:

- No neurologic complications within 30 days of surgery
- Two post-operative MIs; neither were fatal and both discharged in stable clinical condition
- Two patients experienced atrial fibrillation post operatively
- No fatalities

Secondary outcome variables:

- 172 auditory recalls
- 3 visual recalls
- 173 tactile recalls
- 0 perceptions of muscle paralysis
- 139 recalls of feeling vocal cord paralysis
- 1 recall of pain
- 2 recalls of bad feelings

Patient satisfaction scores:

- Very satisfied with the technique n = 112
- Satisfied with the technique n = 67
- Unsatisfied n = 2
- Very unsatisfied n = 0

Surgeon satisfaction scores:

- Very satisfied n = 170
- Satisfied n = 11
- Unsatisfied and very unsatisfied n = 0

Conclusion This technique using a remifentanyl infusion appeared to be safe and with a high degree of patients and surgeon satisfaction. It also had a very low rate of conversion to inhalation agents due to patients' anxiety, pain, or general discomfort. 'Awake' patients and clinical neurological monitoring during carotid clamping allowed for early detection of changes and subsequent surgical intervention to prevent stroke. Further studies are necessary, however, comparing this technique versus carotid endarterectomy under cervical plexus block and even general endotracheal anesthesia with inhalation agents.

Comment This technique appears quite complex and challenging relative to those techniques that are considered standard but are a bit antiquated. It takes a degree of 'work' that some may not feel is 'worth it.' It is absolutely necessary to explain in great detail to the patients, the benefits over the risk in using such a procedure for CEA, a surgical procedure where negative outcomes are *not* rare. Often times, we don't use drugs like remifentanyl or the technical devices such as these infusion pumps that do sophisticated calculations-- because of the cost. We need significant evidence that demonstrates patient outcomes *are* superior and complications *are* minimized, when cost has become a key decision making factor to consider. We must provide evidence that shows superior patient and surgeon satisfaction, an important consideration that also outweighs the cost of such a procedure. Growing our body of knowledge and achieving the evidence is not negotiable. These innovative techniques may be more costly in terms of the anesthetic—but we owe it to our patients to use the scientific method to discover better ways to ensure more favorable outcomes.

Mary A. Golinski, PhD, CRNA

Notes- Effector site concentration (C_e) is individualized for each patient. Using a computerized controlled infusion pump for intravenous anesthetic agents such as remifentanyl, the pumps are set to obtain a specific target concentration of medication at the specific receptor site. Using this form of drug delivery, the goal is to maintain constant concentration or level of drug in a specific pharmacological compartment. The pumps are controlled by mathematical models i.e. algorithms, based on the pharmacokinetic and pharmacodynamic properties of the specific agent. The remifentanyl in this study was initially set to a target plasma concentration of 8 ng/mL. The software calculates in real time, the actual remifentanyl C_e and continuously displays the C_e on the device monitor.

UNEXPECTED CARDIOVASCULAR COLLAPSE FROM MASSIVE AIR EMBOLISM DURING ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY

Acta Anaesthesiol Scand. Published Ahead of Print

Goins KM, May JM, Hucklenbruch C, Littlewood KE, Groves DS

Abstract

Purpose The purpose of this case report was to describe the occurrence of unexpected cardiac arrest secondary to massive venous air embolism (VAE) during endoscopic retrograde cholangiopancreatography (ERCP).

Background VAE is a rare but potentially fatal complication of ERCP. While it is typically associated with procedures above the level of the heart (i.e., sitting craniotomies), it can also occur during procedures involving insufflation of high-pressure air.

Methodology A 72-year old female presented for an outpatient ERCP for diagnostic intraductal endoscopy under general endotracheal anesthesia. Her past medical history was significant for unresectable cholangiocarcinoma. After induction of general anesthesia and intubation the procedure began with the patient breathing spontaneously with stable hemodynamics. Twenty minutes later during a technically difficult part of the case the end tidal carbon dioxide (EtCO₂) rapidly decreased from 50 mm Hg to 5 mm Hg with hypotension, hypoxia, and bradycardia followed by pulseless electrical activity. Peak airway pressures increased to approximately 40 mm Hg. The procedure was stopped and CPR and ACLS were initiated, and an arterial line placed. Based on the presentation a pulmonary embolism was suspected. A transesophageal echo probe was placed (TEE) which showed a large amount of trapped air within the right heart consistent with a massive VAE. A pulmonary artery catheter was placed and under TEE guidance 30 mL of air was aspirated from the right ventricle. Within five minutes the patient regained spontaneous circulation and was transported intubated to the Intensive Care Unit. The patient was weaned from the ventilator the following day.

Result After extubation the patient demonstrated short-term memory problems and deficits in fine and gross motor function. An MRI three days later revealed two small foci of acute infarct in the right superior frontal lobe consistent with a watershed infarct due to decreased cerebral perfusion during cardiac arrest. The patient fully recovered and was discharged without sequelae six days later. The patient returned twice for follow-up ERCP laser treatments. No complications occurred with either procedure.

In the discussion the authors provide a succinct review of the risk of VAE during ERCP procedures. Many of the cases described occurrence of the VAE during technically difficult points during the procedures when possible small vessel injury may have occurred. Though no bleeding was noted because of insufflation pressures, it is possible that venous air in this case could have been introduced into the bile ducts and into the portal venous system, followed by the inferior vena cava and right heart. X-ray images during the procedure demonstrated contrast initially filling the bile ducts followed by air in the bile ducts.

Conclusion VAE is a rare and many times unsuspected complication of ERCP. VAE can occur during technically challenging portions of the procedure, thus communication between the anesthesia provider and gastroenterologist is essential. Warning signs may be subtle, but decreases in EtCO₂, hypoxia, bradycardia, and hypotension may all be signs of impending cardiac arrest secondary to VAE. Appropriate management is essential to avoid fatal consequences.

Comment

I thought this was an important case report to present because anesthesia providers are being consulted more often to provide anesthesia for gastroenterology procedures. When I first read this report I must admit I had only recently heard that VAE was a complication associated with ERCP. The exact incidence of VAE is unknown, but a quick review of the literature found at least 14 cases, with four being fatal.¹ My guess is the incidence is a lot higher than this because I have heard of cases of VAE at other facilities that have not been reported in the literature.

VAE is a life threatening event that requires prompt recognition and early intervention to minimize morbidity and mortality. Mirski et al.² provide an excellent review on the identification, prevention and treatment of VAE. In their report they describe the rate and volume of air accumulation associated with VAE is dependent on vascular lumen size and the pressure gradient. The most sensitive detection method is TEE followed by precordial doppler.² Measures with moderate sensitivity include end tidal nitrogen and EtCO₂, with the latter being the most readily available. However for EtCO₂ to be sensitive to detect a VAE, a secure airway is most likely needed (LMA or endotracheal tube).

I have provided both sedation and general anesthesia with intubation for ERCP procedures, with the choice of anesthesia being dependent on the patient's comorbidities, positioning and local practice. The most common reason I have seen recently is for obstructive sleep apnea (OSA). These patients are very sensitive to sedatives and opioids, therefore the anesthesia provider must be very vigilant and cautious in administering sedation to these patients, especially for ERCP procedures where the position may be a modified swimmers position (prone position). The ASA practice guidelines recommend EtCO₂ monitoring in OSA patients requiring moderate or deep sedation, and that general anesthesia with a secured airway is preferable with this level of sedation in patients requiring upper endoscopy procedures.³ While these are only guidelines, not standards, providers need consider them when developing an anesthetic plan for a patient with OSA undergoing an ERCP. It may be prudent to consider performing an elective intubation with general anesthesia or TIVA given this provides a secure airway and allows for EtCO₂ monitoring, which may aid in identifying a VAE. Other considerations to think about should be the location (operating room vs. offsite location), personnel availability (i.e., extra anesthesia provider or technician), and equipment (i.e., anesthesia machine, precordial doppler or TEE) when providing anesthesia for an ERCP. I know I will be considering these the next time I am asked to provide anesthesia for an ERCP.

Dennis Spence PhD, CRNA

1. Bisceglia M, Simeone A, Forlano R, Andriulli A, Pilotto A. Fatal systemic air embolism during endoscopic retrograde cholangiopancreatography. *Adv Anat Pathol*. 2009; 16(4):255-62.
2. Mirski MA, Vijay Lele A, Fitzsimmons L, Toung TJK. Diagnosis and treatment of vascular air embolism. *Anesthesiology*. 2007; 106(1):164-77.
3. Gross JB, Bachenberg KL, Benumof JL, et al. Practice guidelines for the perioperative management of patients with obstructive sleep apnea. *Anesthesiology*. 2006; 104(5):1081-1093.

The views expressed in this article are those of the author and do not reflect official policy or position of the Department of the Navy, Department of Defense or the United States Government.



CEREBRAL ISCHEMIA DURING SHOULDER SURGERY IN THE UPRIGHT POSITION: A CASE SERIES

J Clin Anesth 2005;17:463-469

Pohl A, Cullen DJ

Abstract

Purpose The purpose of this case series was to report four cases of brain injury or death following general anesthesia for sitting shoulder surgery.

Background Perioperative stroke and brain injury is uncommon. When it does occur, the incidence of death is greater than when stroke occurs unassociated with anesthesia and surgery. Most of these complications occur in patients with known risk factors and/or following cardiovascular surgical procedures.

A significant number of surgical procedures on the shoulder are performed with the patient positioned in a sitting, or semi-sitting position. There have been anecdotal reports of brain injury or death in patients at low risk for cardiovascular events following general anesthesia for sitting shoulder surgery.

Blood pressure (BP) is not uniform throughout the body. In vessels of similar diameter, blood pressure is a function not only of cardiac output and vascular resistance, but also of hydrostatic pressure. Non-invasive BP machines measure blood pressure at the point where the BP cuff is placed on the patient.

A chief factor determining the difference in blood pressure between the point of measurement and any other point in the body is the vertical distance between the two points. This vertical distance, and the density of the fluid (whole blood) in the circulatory system, results in a hydrostatic “fluid column” pressure. Near the bottom of the fluid column, the pressure is greater because of the weight of the column of fluid above it. Near the top of the column the opposite is true. Mercury, the fluid whose density we reference when reporting blood pressure (mm Hg), is much more dense than whole blood.

1 cm H₂O = 0.7355 mm Hg.

Whole blood is 1.06 times more dense than water, so:

1 cm whole blood = 0.7796 mm Hg.

When the height of the fluid column is significant, cerebral perfusion pressure (CPP) may be inadequate, resulting in insufficient brain oxygen delivery.

Case #1 A 47 y/o female with a preoperative BP of 125/83 received 2% isoflurane and nitrous oxide. During the case her BP was reduced to 100/60. Subsequent systolic pressures were 80 to 90 “for the remainder of the case.” E_TCO₂ was in the “low 30’s” throughout. She was in the “barbershop” position. The location of her BP cuff was not described. She did not awaken from general anesthesia. Two weeks later her Glasgow Coma Scale was 3. (This is the lowest GCS possible, indicating “no response” to eye opening, motor response, or verbal response.)

Result In this case let us assume that the systolic BP was 90 for the bulk of the case, that the BP cuff was on the upper arm, that the vertical distance from the BP cuff to the circle of Willis in this small woman was only 20 cm, and that she was normocapnic. In this case the MAP at the BP cuff was likely about 70 mm Hg. Cerebral MAP would then be 16 mm Hg lower or 54 mm Hg. This MAP is slightly below the threshold of cerebral autoregulation in a healthy, normotensive individual, thus, the effects of the inhalation anesthetic on autoregulation are irrelevant. Under ideal circumstances cerebral perfusion pressure would have been minimally adequate with no margin of safety remaining. To the extent that her MAP was lower than 70 and the vertical distance between the BP cuff and her head was greater than 20 cm her cerebral perfusion pressure (CPP) would have been lower. If CPP was decreased in excess of the reduction in $CMRO_2$ caused by general anesthesia, ischemia would have resulted.

Case #2 A 57 y/o male underwent shoulder surgery following trauma that also resulted in a compressed cervical vertebrae with some motor weakness in his arms. His BP was 125/60 “early in the case.” He received 2.5% isoflurane and nitrous oxide. During the case BP was in the range of 100/55. The lowest reported BP was 90/55. E_TCO_2 was “normal” throughout. He was in the “beach chair” position. The location of his BP cuff was not described. He did not awaken from general anesthesia. Neurology suggested, “a posterior circulation infarct involving the midbrain and cortical thalamic region.”

Result In this case let us assume that the BP was 100/55 for the bulk of the case, that the BP cuff was on the upper arm, and that the vertical distance from the BP cuff to the circle of Willis was only 25 cm. In this case the calculated MAP at the BP cuff was 70 mm Hg. Cerebral MAP would then be 19 mm Hg lower or 51 mm Hg. This MAP is slightly below the threshold of cerebral autoregulation in a healthy, normotensive individual, thus, the effects of the inhalation anesthetic on autoregulation are irrelevant. So, under ideal circumstances cerebral perfusion pressure was slightly inadequate. To the extent that his MAP was lower than 70 and the vertical distance between the BP cuff and his head was greater than 25 cm his CPP would have been lower. If CPP was decreased in excess of the reduction in $CMRO_2$ caused by general anesthesia, ischemia would have resulted. With the information included in the case report, global cerebral hypotension appears to be the most likely cause of injury. Given the postoperative injury that resulted, a local reduction in blood flow (vertebral arteries) should also be considered as a primary or contributory cause. No information was reported on head position. His neck trauma introduced a number of unknowns that could be relevant but are not documented.

Case #3 A 53 y/o man with a history of hyperlipidemia and a family history of vascular disease had a preoperative BP of 130/70. During the case BP was decreased to 90/50. For the “last half hour” of the case his BP was about 80/50. The location of his BP cuff was not described. He received isoflurane and nitrous oxide. He was in the “beach chair” position with his upper body elevated to “almost 90°” from the horizontal. He did not awaken from general anesthesia. Neurology believed he had a left hemispheric watershed infarct.

Result This case reported a BP of 80/50 for at least 30 minutes. Let us assume that the BP cuff was on the upper arm, that the vertical distance from the BP cuff to the circle of Willis was 30 cm (upper body elevated “almost 90°”), and that he was normocapnic. The calculated MAP at the BP cuff was 60 mm Hg. Cerebral MAP would then be 23 mm Hg lower or 37 mm Hg for a sustained period. This MAP is inadequate. With the information included in the case report global cerebral hypotension appears to be the most likely cause of injury.

Case #4 A 54 y/o woman underwent shoulder surgery in the sitting position. She had no history of hypertension but no “baseline” BP was reported. She was in the “beach chair” position. The BP cuff was on her calf. She received 3.5% isoflurane and

nitrous oxide. Nitroglycerine and labetalol were used to reduce BP to between 85 and 100 systolic. BP reportedly ranged between 70/40 and 90/60. When the BP fell to 50/25 it was treated with phenylephrine. $E_T\text{CO}_2$ was in the “high 20’s” throughout. The case lasted for over three hours. She did not awaken from general anesthesia. The upper spinal cord and medulla were found to be infarcted at autopsy.

Result In this case let us assume that the BP was 90/60 for the bulk of the case, that the vertical distance from the BP cuff on her calf to the circle of Willis was only 70 cm, and that arterial CO_2 was 30 torr. The blood pressure assumption appears to be generous but will not affect the outcome of the example. In this case the calculated MAP at the BP cuff was 70 mm Hg. Cerebral MAP would then be 55 mm Hg lower or 15 mm Hg. This cerebral MAP is inadequate. Compounding the cerebral hypotension, the slightly decreased arterial CO_2 would have caused cerebral arterial constriction, further limiting blood flow. With the information included in the case report, global cerebral hypotension appears to be the inescapable cause of injury.

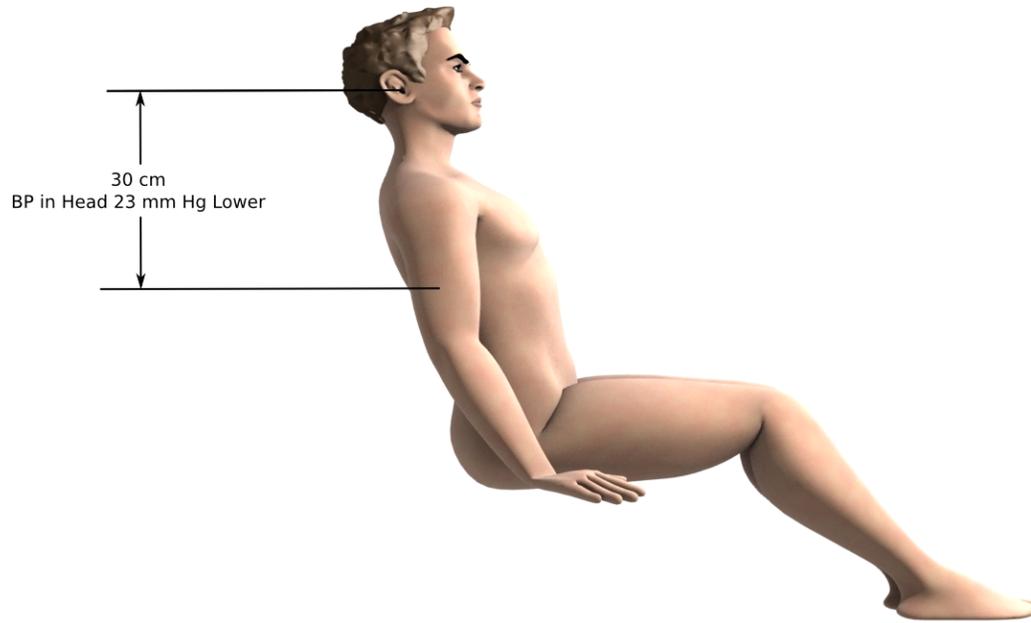
Conclusion Despite the lack of known cerebrovascular risk factors, these patients experienced CNS ischemia. Blood pressure cuff measurements do not accurately indicate brain blood pressure.

Comment

While Anesthesia Abstracts normally reviews *current* literature, there are occasions when it is appropriate to go back a bit further. Brain injury during sitting shoulder surgery and general anesthesia is a problem currently being talked about, but there is very little in the literature about it. During sitting shoulder surgery with general anesthesia the most likely primary cause of permanent CNS injury or death is cerebral hypotension.

During the preanesthetic evaluation, a minimum acceptable brain mean arterial pressure (MAP) should be identified and maintained throughout sitting shoulder surgery. This step is especially important if pathology is present that might result in the need for a higher than normal brain MAP to provide adequate perfusion. Using clinical judgment to choose this value preoperatively avoids the influence of the surgeon intraoperatively when the BP “needs to be just a little lower” to eliminate blood from the surgical view.

In a sitting patient, close monitoring of systemic blood pressure with an automated, oscillometric blood pressure machine is inadequate to insure sufficient cerebral blood pressure. Blood pressure in the brain can easily be 20 mm Hg lower than BP in the arm and this difference is often overlooked, especially by surgeons who often want the BP kept low enough to minimize bleeding into the field. (See figure on the next page.) If an automated BP cuff is used to monitor BP, the anesthetist should also measure the vertical distance between the BP cuff and the external auditory meatus. This measurement can be used to determine how much lower brain BP is than cuff BP.



Michael Fiedler, PhD, CRNA

© Copyright 2010 Anesthesia Abstracts · Volume 4 Number 1, January 31, 2010



KETAMINE, BUT NOT PRIMING, IMPROVES INTUBATING CONDITIONS DURING A PROPOFOL-ROCURONIUM INDUCTION

Can J Anesth 2010;57:113-119

Topcuoglu PT, Uzun S, Canbay O, Pamuk G, Ozgen S

Abstract

Purpose The purpose of this study was to test the hypothesis that adding ketamine to an induction dose of propofol would improve the speed of onset of rocuronium and improve intubating conditions.

Background Rocuronium's speed of onset is dose-dependent and onset speed is crucial during rapid sequence intubation. Rocuronium has been reported to produce less than optimal rapid sequence intubating conditions in up to 25% of patients. Priming with a small dose of muscle relaxant has been shown to shorten the onset of paralysis in some studies, but not uniformly so with rocuronium. But priming has been shown to cause muscle weakness, trouble swallowing, blurred vision, and respiratory failure in some patients while still awake.

Previous studies have shown a faster onset of nondepolarizing muscle relaxants when co-administered with ephedrine. Ultimately, the speed of onset of a muscle relaxant depends upon how quickly the drug is delivered to the neuromuscular junction. How quickly the muscle relaxant reaches the neuromuscular junction is inversely proportional to cardiac output. Induction drugs that preserve cardiac output have been previously shown to be associated with faster onset of muscle relaxation.

Methodology This prospective, randomized, double-blind study included ASA physical status I & II patients scheduled for elective nasal surgery with general anesthesia. Those at risk for pulmonary aspiration or difficult intubation were excluded.

Patients were divided into four groups of 30 patients each. The groups were as follows: Control (no ketamine or priming), Priming (no ketamine), Ketamine (no priming), and Ketamine-Priming. When included, ketamine 0.5 mg/kg was added to the induction dose of propofol. Priming doses of rocuronium were 0.06 mg/kg administered 2 minutes before the main intubating dose. Twitch responses were monitored every 10 seconds after induction at the adductor pollicis with an S/5 neuromuscular transmission module (Datex-Ohmeda, Madison, WI). All patients received an induction dose of 2.5 mg/kg propofol and a total of 0.6 mg/kg rocuronium, including the priming dose, if used. Laryngoscopy and intubation was performed one minute after induction by someone unaware of which group the patient was in. Statistical analysis was conservative.

Result The four study groups included a total of 120 patients. Demographics were similar in all groups.

Overall intubating conditions were better in both groups that received ketamine (Ketamine group and Ketamine-Priming group) ($P = 0.001$). A priming dose of rocuronium did not improve overall intubating conditions ($P = 0.35$). The time between administration of

the main rocuronium dose and complete disappearance of all four twitches in the train-of-four was shorter in both groups that received ketamine ($P = 0.001$). Average time to no detectable twitch was 3.6 min and 3.5 min in the Control and Priming groups compared to 2.7 min and 2.8 min in the Ketamine and Ketamine-Priming groups. Ketamine was associated with faster elimination of the twitch response ($P = 0.001$) but rocuronium was not ($P = 0.94$). In the groups that received ketamine, the drop in BP was smaller after induction ($P = 0.001$) but post induction heart rates were not statistically significantly different ($P = 0.095$).

Conclusion The addition of 0.5 mg/kg ketamine to a propofol / rocuronium induction improved jaw relaxation and vocal cord abduction, reduced movement in response to intubation, and speeded the onset of muscle relaxation one minute after induction of general anesthesia. Priming with 0.06 mg/kg rocuronium did not.

Comment

Ketamine is an underappreciated drug. I think much of the fear of ketamine use is rooted in high dose ketamine techniques used years ago. All drugs have side effects, and ketamine's side effects are highly dose dependent.

For me, this was one of those “why didn't I think of that” studies. Of course, muscle relaxants can't work until they get to the neuromuscular junction and they get their through the circulation. So it makes sense that if the circulation is moving faster the relaxant will get there sooner. What is most interesting is that this is *clinically* significantly true. I suspect that this study actually *understated* the effect. After all, 2.5 mg/kg of propofol is a full induction dose and 0.5 mg/kg ketamine is another half an induction dose. We know that propofol is a potent myocardial depressant, so cardiac output likely goes down after induction, slowing the movement of the muscle relaxant to its site of action. Since the ketamine dose is half an induction dose, it would be interesting to know if the onset of rocuronium was even faster with a smaller propofol dose, say about 1.5 mg/kg in combination with the ketamine.

The 2.5 to 3.5 minute onset times for rocuronium may look long to some. Clearly an intubating dose of rocuronium works faster than that. The reason these times look long is that they are the times to 100% paralysis. We don't normally wait that long to attempt intubation. The key finding is that, with or without priming, adding ketamine to the propofol increased the speed of onset of rocuronium by about 30%.

I have long added 50 mg of ketamine to each 200 mg of propofol for induction of anesthesia. I've done so primarily for the preemptive analgesic effect, rock solid amnesia, and the moderating effect it has on propofol hypotension and occasional bradycardia. Now I have one more reason to continue the practice, by maintaining or increasing cardiac output, ketamine makes the muscle relaxant work faster too. Cool!

Michael Fiedler, PhD, CRNA



MATERNAL AND NEONATAL EFFECTS OF BOLUS ADMINISTRATION OF EPHEDRINE AND PHENYLEPHRINE DURING SPINAL ANAESTHESIA FOR CAESAREAN DELIVERY: A RANDOMISED STUDY

Int J Obstet Anesth 2010;19:24-30

Prakash S, Pramanik V, Chellani H, Salhan S, Gogia AR

Abstract

Purpose The purpose of this study was to compare the neonatal effects of ephedrine and phenylephrine administered as an intermittent bolus to treat maternal hypotension following subarachnoid block for cesarean section.

Background Hypotension is a frequent complication of subarachnoid block for cesarean section. It can have adverse effects on both mother and fetus and vasoactive drugs are routinely needed to prevent or treat it. Some current evidence suggests that phenylephrine may be a better choice for treatment of maternal hypotension than ephedrine. Clinical trials with large total doses of ephedrine used to prevent hypotension have shown a reduction in fetal pH and base excess. Prophylactic administration often results in the patient receiving a larger total dose. The effect on fetal pH with smaller, intermittent doses of ephedrine are less certain. Multiple studies comparing bolus doses of phenylephrine and ephedrine for subarachnoid block induced hypotension found no difference in the incidence of fetal acidosis. This was true both in women having elective and non-elective cesarean sections.

Phenylephrine 100 µg is equivalent to 8.1 mg ephedrine as a vasopressor.

Methodology This randomized, double-blind study included ASA I women scheduled for elective cesarean section with spinal anesthesia. Exclusion criteria included hypertension, diabetes, cardiovascular or cerebrovascular disease and fetal abnormalities. All women received ranitidine and metoclopramide preoperatively. Baseline blood pressure (BP) was determined by the average of three BP readings taken after five minutes resting in the supine position with left uterine displacement. All women received a 10 mL/kg bolus of Ringer's lactate over 15 minutes before induction of subarachnoid block with 10 mg of hyperbaric bupivacaine. A vasopressor was administered after the block if maternal BP fell below 80% of baseline. Women received either 100 µg phenylephrine or 6 mg ephedrine. Additional doses were administered if needed until BP was at least 80% of baseline. The statistical analysis was fairly conservative.

Result A total of 30 women each were included in the analysis from the phenylephrine and ephedrine groups. The two groups were similar demographically. Specifically, block height, induction to delivery times, uterine incision to delivery times, and infant birth weights were not statistically significantly different.

APGAR scores at 1, 5, and 10 minutes and time to sustained respirations were no different between groups. No umbilical artery pH values were below 7.2 (acidosis). Both umbilical venous and umbilical arterial pH were significantly lower in the Ephedrine group (P

= 0.002 and P = 0.01). Umbilical venous and umbilical arterial base deficit were greater in the Ephedrine group as well (P = 0.001 and P < 0.001). Neurobehavioral scores at 2-4 hours, 24 hours, and 48 hours post-delivery were no different in babies born to mothers in the Ephedrine and Phenylephrine groups.

Conclusion Phenylephrine 100 µg was as effective as Ephedrine 6 mg for treatment of maternal hypotension during subarachnoid block. Although there were statistically significant differences in umbilical arterial and venous pH and base deficit between groups, the differences were small and all values were within normal limits. Early neonatal neurobehavioral scores were no different between groups.

Comment

This is one of those studies that, at first, looks like it has something profound to say but when you think about it for a little while it's not clear that it is really telling you anything helpful. Worse yet, the methods and analysis used were pretty clean, so any findings of dubious clinical importance can't be chalked up to poor methods. The "first" message is that Ephedrine was associated with lower umbilical arterial and venous pH and a greater base deficit. That sounds bad. But when you look more closely, they are only "lower" and "greater" within the normal range. The average values in both groups were perfectly normal. What's more, the average values were better than the average normal values published just 15 years ago. So, really, ephedrine didn't do anything bad, it just produced normal values that were closer to the bad side of normal than phenylephrine did. What do I do with that clinically?

Years ago I was taught that ephedrine was the only vasopressor that was safe to use in a pregnant woman. All alpha agonists constricted uterine arteries causing fetal hypoxia. Then, we started seeing evidence that low doses of phenylephrine were OK to use and didn't produce fetal hypoxia. More recently it has been suggested that ephedrine causes fetal acidosis and that phenylephrine is the "good drug" for pregnant women – a complete reversal of what we "knew" 20 years ago.

Here is what I think this study has to teach us. In healthy women, both ephedrine and phenylephrine in clinically useful bolus doses increase maternal BP without harm to the fetus. The idea that ephedrine is "bad" is not supported by this study. While umbilical blood gases were perhaps slightly "less good" in the ephedrine group, they were normal and 4, 24, and 48 hours later a thorough neurobehavioral assessment couldn't tell the neonates apart.

This is why Evidence Based Practice is important and why it is important to keep up-to-date with the latest anesthesia knowledge! What we "know" changes. The world is no longer flat.

Michael Fiedler, PhD, CRNA



EFFECT OF COMBINING DEXMEDETOMIDINE AND MORPHINE FOR INTRAVENOUS PATIENT-CONTROLLED ANALGESIA

Br J Anaesth 2009;102:117-22

Lin T, Yeh Y, Lin F, Wang Y, Lin C, Sun W, Fan S

Abstract

Purpose The purpose of this study was to investigate the efficacy, in terms of postoperative pain management and side effects, of adding dexmedetomidine to patient-controlled analgesia (PCA) morphine for females undergoing total abdominal hysterectomy.

Background The benefits of maximizing pain relief post abdominal surgery are well known. Several multimodal approaches exist, some which have been trialed and/or studied, but research regarding the efficacy of combining the alpha-2 adrenoreceptor agonist dexmedetomidine with PCA morphine, has not been carried out. Dexmedetomidine possesses several beneficial attributes; most significant are its analgesic, anxiolysis and sedative properties, sympatholysis properties, and a notable absence of any respiratory depression. Opioids like morphine can produce adequate analgesic effects, however they are not without problematic effects, including but not limited to, respiratory depression and severe nausea and vomiting. By adding dexmedetomidine to PCA morphine, the researchers hypothesized that less morphine may be required to achieve effective analgesia, the patient would benefit by the additional attributes of dexmedetomidine, and therefore the side effects of PCA morphine would be minimized.

Methodology This was a randomized, double-blind study. After IRB approval and patient consent, 100 ASA I and II patients scheduled for total abdominal hysterectomy under general anesthesia, aged 18 years to 65 years old, were randomized to one of two treatment arms:

- Group M received PCA morphine 100 mg /100 mL of normal saline with the first dose administered in the recovery room if the first pain score, using the verbal analogue scale 0-10, was > 5 .
 - A bolus was given initially (2 mL at 5 minute intervals) until the pain score achieved was < 4 , then the patient was instructed to self administer 1 mL boluses with a 5 minute lockout
- Group D received PCA morphine 100 mg PLUS 500 mcg dexmedetomidine in 100 mL of normal saline also with the first dose administered in the recovery room if the first pain score was > 5
 - The bolus for group D was administered in the same manner as in group M

Demographic data was collected preoperatively for analysis post procedure. Intra-anesthetic data was collected specific to hemodynamic variables and parameters for each individual patient, as well as doses of all anesthetic agents used. Each general anesthetic was given in a standardized format. Fentanyl was the intra- anesthetic opioid of choice. Patients were assessed upon

arrival to the recovery room and at 1, 2, 4 and 24 hour intervals post surgery for pain scores both at rest and with movement. Additionally, data was collected during the same time intervals on deviation of vital signs from the preoperative state, nausea and vomiting, pruritis, sedation, cumulative PCA requirements, rescue medications needed and general patient satisfaction with postoperative pain management. A power analysis was conducted to determine the sample size needed in order to detect a 25% reduction in morphine requirements in the first 24 hours postoperatively.

Result A total of 98 patients were recruited to the study; 2 had to be removed, 1 for requiring re-operation and one for anemia related symptoms. There were no significant differences in demographics between groups. The cumulative PCA morphine use during the first 24 hours postoperatively was significantly less in group D ($P < 0.01$). From the 2nd postoperative hour onward, pain scores were consistently lower in Group D versus group M, both at rest ($P < 0.05$) and with movement ($P < 0.01$). Two patients in Group M did not achieve acceptable pain relief and had to be treated with adjunct medications. This did not occur in Group D. No patient in either group experienced hemodynamic alterations requiring any type of intervention. In terms of nausea and vomiting, the overall incidence was not significantly different between the two groups however, the incidence of nausea during the 4-24 hour time frame was significantly lower in Group D ($P < 0.05$). There were no differences seen between the two groups regarding degree of pruritis or sedation scores. More patients were satisfied with their pain management in general in Group D although this did not achieve statistical significance.

Conclusion This study demonstrated that adding dexmedetomidine to morphine, using a PCA methodology, provided acceptable pain relief for those undergoing hysterectomy. Due to the properties of dexmedetomidine, including enhancing the analgesic effect of morphine, the side effects of the larger doses of morphine were minimized. Patients receiving dexmedetomidine mixed with morphine were more satisfied with their pain management method than those who received plain morphine.

Comment

Dexmedetomidine is a unique therapeutic modality approved by the US Food and Drug Administration (FDA) in December 1999. Its original approval was for sedation in patients hospitalized in intensive care settings. Because of its properties and established margin of safety, its efficacy has allowed it to emerge outside of the intensive care unit setting and into the anesthesia care setting. There are many published research works regarding dexmedetomidine in the anesthesia/surgical setting (see notes below) and the reader is encouraged to explore those for its potential use in their own setting. The only constraint of the alpha-2 agonist, which is a very critical constraint, is its cost. However, as with any newer, more costly, novel agent, if the benefits related to superb quality care and enhanced patient safety are easily recognized, justification for use and therefore a more costly anesthetic, are appropriately rationalized.

Mary A. Golinski, PhD, CRNA

- **Dexmedetomidine** is an alpha-2 agonist that possess both sedative and analgesic properties. The ratio of alpha-2 to alpha-1 receptor activity is 1600:1. It was developed as an alpha-2 adrenoreceptor agonist to stimulate receptors in the CNS to produce analgesia, anxiolysis, and sedation.

Individual actions of the α_2 receptor:

- Mediates synaptic transmission in pre- and postsynaptic nerve terminals.
 - Decrease release of acetylcholine
 - Decrease release of norepinephrine
 - Inhibit norepinephrine system in brain

- Inhibition of lipolysis in adipose tissue
- Inhibition of insulin release in pancreas
- Induction of glucagon release from pancreas
- Platelet aggregation
- Contraction of sphincters of the gastrointestinal tract
- ↓ secretion from salivary gland
- Relax gastrointestinal tract(presynaptic effect)

Epinephrine has higher affinity for the alpha-2 receptor than has norepinephrine, which, in turn, has much higher affinity than has isoprenaline.

Other agonists include:

- Clonidine
- **Dexmedetomidine**
- Lofexidine (antihypertensive)
- Tizanidine (in spasms, cramping)
- Guanfacine (antihypertensive)

For further reading on the various uses of Dexmedetomidine as a sedative/analgesic agent for anesthesia, see: Mil Med. 2009;174:100-102. Awake thyroidectomy under local anesthesia and dexmedetomidine infusion. Plunkett AR, Shields C, Stojadinovic A, Buckenmaier CC

EFFECTS OF INTRAOPERATIVE READING ON VIGILANCE AND WORKLOAD DURING ANESTHESIA CARE IN AN ACADEMIC MEDICAL CENTER

Anesthesiology 2009;110:275-283

Slagle JM, Weinger MB

Abstract

Purpose The purpose of this study was to measure the effects of reading on vigilance, workload, and task distribution during general anesthesia via a behavioral task analysis.

Background Whether or not reading during slow periods of a general anesthetic is acceptable behavior is controversial and can produce fervent debate. Talking to others in the operating room about topics unrelated to patient care is more widely accepted. Many believe reading reduces the anesthesia provider's vigilance and, thus, reduces the quality of care being provided. Even if this is not the case, anesthesia providers who read during a case present the appearance of being inattentive.

Previous observations have revealed that, in the absence of reading during general anesthesia, many anesthesia providers "keep themselves busy" during low workload periods of an anesthetic by engaging in non-essential activities. These activities include rechecking and organizing the anesthesia workspace, talking to others in person or on the phone, and listening to music.

Studies have reported that up to 40% of routine anesthetics may be low workload "idle" periods. The longer the period of idle time, the more likely boredom, inattentiveness, and sleepiness are to occur. Boredom has been shown to contribute to human error in other industries, negatively affecting the performance of pilots during long duration flights. Secondary tasks may contribute to maintenance of alertness.

There is some evidence that reading may improve vigilance in some circumstances by keeping the anesthesia provider mentally stimulated. Boredom, and the resulting inhibition of mental function, may be a greater threat to vigilance than is reading. Evidence provided by studies of monitoring tasks outside of the anesthesia environment shows that adding a secondary task during periods of low mental stimulation can improve vigilance and overall task performance. No scientific evidence is known to exist in this area that is specific to anesthesia practice.

Methodology This study included ASA physical status I-III patients undergoing routine cases with general anesthesia for between 0.75 hours and 6 hours during the daytime. Data were collected at two institutions, a university teaching hospital and a Veteran's Affairs hospital. Inclusion criteria were met by 172 cases. Some reading occurred in 60 cases (Reading group). No reading occurred in 112 cases and 78 of these were selected as the No-Reading group based upon their similarity to cases in the reading group. Areas of similarity considered included: the duration of the case, the anesthesia provider's level of experience, patient ASA physical status, case difficulty, and the type of surgical procedure.

Anesthesia providers were physician residents, nurse anesthetists, fellows, and attending anesthesiologists. Ongoing research was common at both institutions so being involved in data collection was not a novel experience for the anesthesia providers. Participants were told the research was examining "task patterns, vigilance, and workload" during general anesthesia. Data was collected by a trained, non-anesthesia, observer who sat in the OR throughout each case. Observers met performance standards for the specific data being collected in this study prior to beginning data collection.

In the Reading group, data from periods of intraoperative reading were first compared with non-reading periods during the *same case*. Subsequently, the entire anesthesia maintenance period from all 78 No-Reading group cases was compared with three different aspects of the Reading group:

1. The entire anesthesia maintenance period of the Reading group (including periods of reading and periods where no reading occurred)
2. Periods of reading during anesthesia maintenance in the Reading group (non-reading periods not included)
3. Non-reading periods during anesthesia maintenance in the Reading group (periods of reading not included)

Vigilance was measured as the time it took the anesthesia provider to indicate to the observer, verbally or with a hand signal, that they had noticed that a red “alarm” light had turned on. The red light was positioned within the cluster of anesthesia monitors. It was programmed to turn on at random intervals every 7 to 15 minutes. This method of measuring vigilance is well validated. Psychological Workload was measured at random intervals with the Borg Workload scale by both the observer and the anesthesia provider.

No attempts were made to compare adverse events or clinical outcomes between the Reading and No-Reading groups.

Result Intraoperative reading occurred in 35% of cases meeting the inclusion criteria (n=60). The Reading and No-Reading groups were demographically similar. The anesthesia providers were 32% first year residents, 28% second year residents, 23% third year residents, 10% CRNAs, and 7% fellows or attending anesthesiologists. Overall, 83% of the providers were physician residents.

In the Reading group, anesthesia providers read an average of 25% ($\pm 20\%$) of the maintenance phase of the general anesthetic. This amounted to an average of 29 min (± 7 min) per case. Although not part of the study data collection, it appeared to investigators that those who read most commonly read anesthesia related materials. Reading was accompanied by the simultaneous performance of other tasks during 44% ($\pm 24\%$) of the total time spent reading.

While reading, anesthesia providers spent significantly less time on charting and patient care tasks ($P < 0.01$). Specifically, during periods of reading, anesthesia providers spent significantly less time preparing medications for the anesthetic they were providing (average 5 min vs. 16 min, $P < 0.05$) or for their next anesthetic (average 3 min vs. 26 min, $P < 0.05$). Tasks were also shorter in duration during reading. Significantly less time was also spent talking to others during periods of reading ($P < 0.05$).

Workload was significantly lower during periods of reading compared to both periods of non-reading during the same case and during No-Reading cases. Also, when comparing the groups in their entirety, overall workload was significantly lower in Reading than in No-Reading cases. Vigilance was no different during periods of reading or non-reading *within* the Reading group, nor was vigilance different *between* the Reading and No-Reading groups. Mean time to respond to the red “alarm” light was 28, 29, and 27 seconds respectively.

Conclusion These results suggest that anesthesia providers use judgment when deciding whether or not, when, and how long to read during a general anesthetic and that this “selective” reading may not impair vigilance.

Comment

This is one of the most interesting and well executed studies I have read in quite a while. Before I comment on it, I must tell you that I have a long held view that reading unrelated to clinical care during an anesthetic is unacceptable behavior. This study *begins* to

challenge my viewpoint.

If our concern is vigilance in the conduct of an anesthetic, then being critical of reading is probably too narrow. We should, instead, be critical of all forms of distraction from anesthesia care. We can be distracted by things other than reading; loud music, the conversations of others in the room, conversations we are having *with* others in the room, a surgeon's angry demands, or the "ding" of incoming text messages on our cell phone, as examples. I've worked in rooms where the music was so loud I couldn't hear my monitors. While I'm still biased against reading, logically it makes no sense to say that reading is never acceptable but that non-patient care related conversations, loud music, and many other sorts of distractions are always acceptable.

The authors make some important points about the negative impact of boredom on vigilance. I suspect we've all experienced times during a long anesthetic that is going well in a darkened operating room when our minds have crept towards a trance-like state – accompanied by a blank stare, looking at nothing in particular and seeing the same. Certainly, that state has a negative effect on anesthesia vigilance because, at that point, we are not monitoring the patient, not monitoring the conduct of the anesthetic, and not thinking about either one.

All this brings us back to Evidence Based Practice. There is a reason that evidence based practice is important. It is important because valid, relevant evidence is a way to move our thinking away from sometimes strongly held beliefs that may be based on not much of anything. I still see non-patient care related reading during an anesthetic as unacceptable. But these investigators have presented some pretty good evidence that such reading may not reduce vigilance and may even improve it in some circumstances. If I'm wise, I'll carefully consider the evidence they present and alter my beliefs to include the evidence they offer. That can be harder to do than it sounds.

The investigators comment only briefly on how an anesthesia provider reading looks to others. Appearance *is* important. How surgeons, circulating nurses, and non-professional personnel perceive my skill, ability, and professionalism affects our ability to work as a team. Teamwork is essential, especially when things are going badly. It may be that no matter how strong the evidence that reading does not reduce vigilance, the appearance of reading strikes others so negatively that they can never trust the ability or judgment of the anesthesia provider.

There are a couple limitations to this very well done study that are worth considering. First, the red light used to simulate an alarm may be well validated as a test of vigilance but it is not subtle. I cannot help thinking that vigilance is not an all or nothing phenomenon. I see vigilance as a continuum. Perhaps distracters, like reading, impair vigilance for more subtle findings first and the red light was simply too big to miss. Second, although the anesthesia providers in this study were used to data being collected during cases they did, they still knew they were in a study of their "task patterns, vigilance, and workload." It may be that they concentrated more on their vigilance because they knew they were in a study and this incentive was enough to compensate for the reduced attention to vigilance that the distraction of reading created.

In general, I prefer to hold professionals accountable for outcomes and expect them to act professionally rather than dictating specific behaviors with policy. Some say there should be a policy that you can't have a cell phone in the OR because you might talk on it or take a picture of something. I say my smart phone has a comprehensive drug database, 5- minute clinical consult, and the direct phone number for every OR in it and I am grown-up enough not to be using my phone for things I shouldn't use it for. By believing that reading is never acceptable behavior during an anesthetic, I may have violated one of my own rules. Either way, this study offers us valid evidence. If we are to practice anesthesia based upon evidence we must give the information it provides due consideration – even if we *want* to believe something else.

ADVERSE EVENTS ASSOCIATED WITH INTRAVENOUS REGIONAL ANESTHESIA (BIER BLOCK): A SYSTEMATIC REVIEW OF COMPLICATIONS

J Clin Anesth 2009;21:585-594

Guay J

Abstract

Purpose The purpose of this review was to catalogue the reported complications associated with intravenous regional anesthesia.

Background Intravenous Regional Anesthesia (IVRA), or Bier block, is a commonly used anesthetic technique for hand and distal forearm surgery. The ASA closed claims study identified three cases of death or brain damage associated with IVRA between 1980 and 1999.

Methodology Two biomedical databases were searched on the terms, "Bier block," "intravenous regional anesthesia," and "complications." At least partial information was located for 64 cases between 1964 and 2005. Forty were adults, 19 female, 17 male, and 4 adults with gender unspecified. Nine cases were children. The remaining 15 cases did not specify age.

Result Complications were related to local anesthetic toxicity in 39 cases (61%). Nine of these patients were judged to be preictal. One patient experienced partial loss of "muscle power." One patient was unable to speak for 20 hours. One patient was "temporarily" blind. Of the entire group of local anesthetic toxicities, there were 24 seizures. In 12 cases the seizure occurred while the tourniquet was inflated, in 9 after the tourniquet was deflated, and in 3 the state of the tourniquet was not reported. Seizures followed tourniquet times of up to 60 minutes and were delayed by as long as 10 minutes after deflation.

Three different local anesthetics were used in blocks with reported local anesthetic complications; lidocaine (n=16), prilocaine (n=2), and bupivacaine (n=20). The lowest dose of local anesthetic injected for the IVRA in patients who had a seizure was 1.4 mg/kg, 4 mg/kg, and 1.3 mg/kg respectively.

Death and/or Cardiac Arrest occurred in 13 patients who received either lidocaine (lowest dose 2.5 mg/kg) or bupivacaine (lowest dose 1.6 mg/kg). Lidocaine was used in 3 of these cases, only one of which resulted in death. Bupivacaine was used in 10 cases, 8 of which resulted in death.

"Nerve damage" occurred in 2 patients and compartment syndrome in 10. Thrombophlebitis occurred in 8 patients; 2-chloroprocaine in 5 and lidocaine containing chlor-cresol in 3.

Conclusion IVRA is associated with a low incidence of complications.

Comment

Since there is no organized and comprehensive tracking of Bier block complications, this review does the next best thing; it tries to catalogue all reports of complications in the literature. Of course, the down side is that we know neither how many complications actually occurred (reporting is not required), nor the total number of Bier blocks performed. Thus, we don't know the incidence of

complications. Nevertheless, this gives us some clues and teaches a little history along the way. The author located only 64 Bier block complications over a 41 year period. Given how commonly used Bier blocks are, both in anesthesia and by orthopedic surgeons outside the OR, it is likely that Bier blocks are an incredibly safe anesthetic. All the more reason that we should do them really well and use them whenever appropriate.

At first, I was caught off guard by some of the complications. They described things I'd never seen with my blocks or heard of from colleagues. But the author wisely included available details of every complication in a table spanning six pages. Examination of the details made many of the complications understandable.

I typically use 50 mL of 0.5% lidocaine (250 mg total) for a Bier block in an average sized individual. In a 70 kg patient that is about 3.5 mg/kg. I never see CNS side effects while the tourniquet is inflated, and almost never see CNS side effects after deflation in as little as 10 minutes. I was puzzled that the author found 39 complications related to local anesthetic toxicity. I was perplexed that 16 involved lidocaine. But it made more sense when I saw the mg/kg dose of lidocaine that was used in blocks that had complications. In 11 of the 16 lidocaine blocks (69%) the dose of lidocaine was 4.3, 4.6, 6.3, 6.3, 7.1, 7.3, 7.6, 8, 11.4, 12, & 20 mg/kg; up to 5.7 times the dose I use.

I was also surprised to see local anesthetic complications long after tourniquet deflation. The few times I've seen local anesthetic toxicity after tourniquet deflation it has been almost immediate. But with 20 mg/kg of lidocaine in an arm or bupivacaine (highly lipid soluble) absorbed into the arm and then released slowly back into the circulation, a delayed onset makes more sense.

I was surprised again to see local anesthetic toxicity reported while the tourniquet was inflated. But in some cases tourniquets were identified as "leaking" after toxicity was discovered, in some cases the tourniquet was inflated to a specified pressure above a previously measured baseline blood pressure rather than current BP, and in yet other cases local anesthetic was injected quickly or at an antecubital vein. Both techniques shown to result in local anesthetic escaping past the tourniquet.

I was fortunate to be taught a Bier block technique that has produced high quality blocks and, so far, not a single incident of clinically significant local anesthetic toxicity in over 25 years. The key aspects that were drilled into me were:

- *Careful exsanguination*
- *Double Tourniquet*
- *Tourniquet 300 torr, if BP is too high, treat the BP*
- *Most distal injection possible*
- *Injection over at least 2 minutes*

Once one removes complications most likely related to bupivacaine, single tourniquets, tourniquet failures, and large doses of lidocaine from this report one is left with 7 reported local anesthetic complications and 14 non-local anesthetic complications over 41 years. I'll take that.

Michael Fiedler, PhD, CRNA