SELECTED READINGS IN NEUROANESTHESIOLOGY

Compiled by Shaheen Shaikh, MD
Assistant Professor of Anesthesiology
Director of Neuroanesthesiology

UMass Medical School
UMass Memorial Medical Center

Table of Contents

I. Selected Chapters from "Complications in Anesthesia, 2nd Edition"

John L. Atlee, MD

Selected Readings in Neuroanesthesiology

I. Selected Chapters from "Complications in Anesthesia, 2nd Edition"
John L. Atlee, MD

Chapter 174 – Intracranial Hypertension by Rosemary Hickey

Chapter 175 – Venous Air Embolism by Jennifer E. Souders and Maurice S. Albin

Chapter 176 – Posterior Fossa Surgery by Donald S. Prough and Eric Bedell

Chapter 177 – Pituitary Tumors: Diabetes Insipidus by Melissa A. Laxton and Patricia H. Petrozza

Chapter 178 – Intracranial Aneurysms: Rebleeding by Philippa Newfield

Chapter 179 – Intracranial Aneurysms: Vasospasm and Other Issues by Philippa Newfield

Chapter 180 – Arteriovenous Malformation: Normal Perfusion Pressure Breakthrough by Shailendra Joshi and William L. Young

Chapter 181 – Pediatric Neurosurgery by Lynda Wells

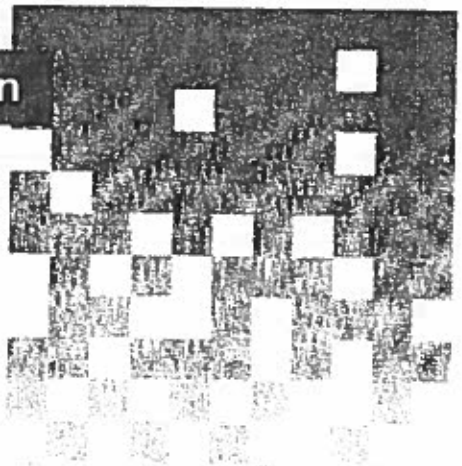
Chapter 182 – Head Injury by Arthur M. Lam and M. Sean Kincaid

Chapter 183 – Spinal Cord Injury by Tod B. Sloan

John L. Atlee, MD
Professor of Anesthesiology
Department of Anesthesiology
Medical College of Wisconsin
Milwaukee, Wisconsin

Complications in Anesthesia

2nd Edition



SAUNDERS
ELSEVIER

SAUNDERS
ELSEVIER

1600 John F. Kennedy Blvd.
Ste 1800
Philadelphia, PA 19103-2899

COMPLICATIONS IN ANESTHESIA, 2nd edition

ISBN-13: 978-1-4160-2215-2
ISBN-10: 1-4160-2215-5

Notice

Knowledge and best practice in this field are constantly changing. As new research and experience broaden our knowledge, changes in practice, treatment, and drug therapy may become necessary or appropriate. Readers are advised to check the most current information provided (i) on procedures featured or (ii) by the manufacturer of each product to be administered, to verify the recommended dose or formula, the method and duration of administration, and contraindications. It is the responsibility of the practitioner, relying on his or her own experience and knowledge of the patient, to make diagnoses, to determine dosages and the best treatment for each individual patient, and to take all appropriate safety precautions. To the fullest extent of the law, neither the Publisher nor the Editors assumes any liability for any injury and/or damage to persons or property arising out of or related to any use of the material contained in this book.

The Publisher

Library of Congress Cataloging-in-Publication Data
Complications in anesthesia / [edited by] John L. Atlee. -- 2nd ed.
p. ; cm.
Includes bibliographical references and index.
ISBN-13: 978-1-4160-2215-2 ISBN-10: 1-4160-2215-5
1. Anesthesia--Complications. I. Atlee, John L.
[DNLM: 1. Anesthesia--adverse effects. 2. Anesthetics--adverse effects. WO 245 C7369 2007]
RD82.5.C63 2007
617.9'6041--dc22 2006040549

ISBN-13: 978-1-4160-2215-2
ISBN-10: 1-4160-2215-5

Executive Publisher: Natasha Andjelkovic
Developmental Editor: Jean Nevius
Publishing Services Manager: Tina Rebane
Project Manager: Amy Norwitz
Marketing Manager: Dana Butler

Printed in the United States of America

Last digit is the print number: 9 8 7 6 5 4 3 2

Intracranial Hypertension

Rosemary Hickey

174

Case Synopsis

A 64-year-old man presents with progressive personality changes, memory disturbances, and urinary incontinence. The physical examination is remarkable for depressed consciousness and papilledema. The computed tomography scan reveals a large frontal mass consistent with a meningioma.

PROBLEM ANALYSIS

Definition

Intracranial hypertension exists when there is a sustained elevation in intracranial pressure (ICP) of more than 15 to 20 mm Hg. It results when the three intracranial components—blood, brain, and cerebrospinal fluid (CSF)—are no longer able to compensate for volume changes occurring within the cranium. CSF translocation from the head into the spinal subarachnoid space and its reabsorption via the arachnoid villi are the major compensatory means of accommodating intracranial volume increases. Spatial compensation can also be achieved through compression of the venous system and, ultimately, capillary collapse, leading to cerebral ischemia.

Changes in ICP that occur with changes in intracranial volume can be described by the intracranial elastance curve (Fig. 174-1). The shape of the curve may be influenced by the type of lesion causing the increase in volume; for example, slower-growing lesions may be better tolerated than rapidly growing ones.

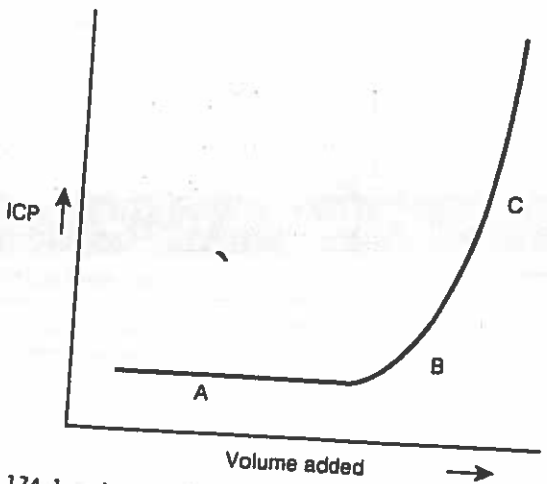


Figure 174–1 – Intracranial elastance curve. *A*, Normal elastance. Reduced elastance (small increase in intracranial pressure [ICP] with creasing intracranial volume). *C*, Poor elastance (large ICP increase th minimal increase in cerebral volume). (From Mahla ME: Neurologic gery. In Kirby RR, Gravenstein N [eds]: Clinical Anesthesia Practice. iladelphia, WB Saunders, 1994, pp 1283-1311.)

Recognition

The signs and symptoms most frequently associated with intracranial hypertension include headache, nausea, vomiting, papilledema, unilateral pupillary dilatation, and oculomotor or abducens nerve palsies. Changes in consciousness and irregular ventilatory patterns indicate advanced stages of intracranial hypertension.

Headache is typically present on awakening, or it may awaken the patient from sleep. It is related to traction and distortion of pain-sensitive cerebral blood vessels and the dura mater. Vomiting may be due to direct stimulation of the vomiting centers by local compression. Papilledema is the only reliable sign of an increase in ICP, although intracranial hypertension may be present without it. Oculomotor palsies are secondary to herniation or compression of the nerve, and abducens palsies result from stretching of the nerve as the brainstem is displaced inferiorly by the increased pressure. A general slowing in mentation occurs from continuously increased ICP and a diffuse decrease in cerebral blood flow. Further deterioration in the level of consciousness indicates progressive transtentorial herniation. Alterations in vital signs (bradycardia, hypertension, depression of respiration) also may occur from increased ICP and brainstem compression. Computed tomography scanning, magnetic resonance imaging, or angiography provides indirect evidence of elevated ICP. These studies may reveal a mass lesion accompanied by a midline shift of at least 0.5 cm, encroachment of the CSF cisterns by the expanding brain, or both.

Risk Assessment

The three major mechanisms of increased ICP are (1) increased intracranial volume due to an intracerebral mass lesion (e.g., tumor, massive infarction, trauma, hemorrhage, abscess), extracerebral mass lesion (e.g., tumor, hematoma, abscess), or acute brain swelling (e.g., anoxic states, acute hepatic failure, hypertensive encephalopathy, Reye's syndrome); (2) high venous pressure resulting from heart failure, superior mediastinal obstruction, or cerebral or jugular venous obstruction, which increases blood volume in the pial veins and dural sinuses and may interfere with CSF absorption; and (3) obstruction to the flow (hydrocephalus) or absorption (pseudotumor cerebri) of CSF.

Implications

The danger of intracranial hypertension lies in the potential for cerebral ischemia and herniation of brain tissue. If ICP, either locally or globally, reaches levels exceeding mean arterial pressure, cerebral ischemia will develop. Cerebral perfusion pressure is calculated as mean arterial pressure minus ICP. The likelihood of permanent tissue damage from cerebral ischemia depends on the severity and duration of the ischemia. If ICP is sufficiently high to obstruct venous outflow from the brain, arterial inflow also may be compromised.

Brain herniation can occur around any fixed structure in the skull. In open head trauma, injured brain may herniate through the fractured skull. In the intact skull, herniation sites include the falx cerebri, under which the cingulate gyrus of the frontal lobe can herniate; temporal lobe (uncal) herniation through the tentorium cerebri; and classic herniation of the cerebellum through the foramen magnum, compressing the medulla and resulting in cardiovascular and respiratory collapse.

MANAGEMENT

Therapeutic interventions to lower elevated ICP are categorized according to its intracranial determinant (Table 174-1). Parenchymal volume may be reduced in several ways. Mannitol results in an osmotic reduction of brain water content. It may also improve blood rheology and microcirculatory flow. Loop diuretics (furosemide) provide intracranial decompression through a diuresis-mediated brain dehydration, reduced CSF formation, and resolution of cerebral edema via improved cellular water transport. Corticosteroids reduce peritumoral edema but are not useful for treating intracranial hypertension secondary to head trauma. Surgical excision of mass lesions reduces the volume of the intracranial space occupied by parenchymal components and thus improves intracranial elastance. Techniques to reduce CSF volume include ventricular or lumbar puncture, drains, and shunts. Cerebral blood flow and volume and ICP are reduced by hyperventilation; however, such a reduction in cerebral blood flow may be poorly tolerated.

Jugular venous oxygen saturation monitoring is used to guide the level of hyperventilation in head trauma. Va greater than 75% indicate hyperemia, so induced vasoc striction associated with hyperventilation may be valuab values less than 50% indicate cerebral ischemia, so attem to induce further cerebral vasoconstriction may be harm Measurement of brain tissue oxygen tension can provide information about the safety of hyperventilati Some intravenous anesthetic drugs (e.g., lidocaine, thiope tal, etomidate, propofol) are beneficial for decreasing IC A continuous infusion of propofol combined with a low-d inhalational agent is another useful anesthetic techniq Venous drainage is maximized by keeping the head elevat 15 to 30 degrees, but without excessive rotation or flexion.

PREVENTION

Prevention of intracranial hypertension centers on avoidi factors that are known to increase ICP. Intravenous flu management is directed toward achieving a euvolemic sta Therapy should avoid the use of intravenous solutions th decrease plasma osmolality (5% dextrose in water, 0.45 sodium chloride, lactated Ringer's solution). The factor administered fluid that most affects brain edema is th osmolality. An acute drop in osmolality affects brain wat content and ICP more than an acute drop in oncot pressure. Glucose-containing solutions are avoided becau hyperglycemia may aggravate ischemic brain injury.

Other factors that increase ICP and should be avoide include compression of jugular veins by improper hea positioning, coughing and straining on the endotrache tube, seizure activity, hypercarbia, and hypoxia. Increase body temperature raises cerebral metabolic oxygen consump tion and should be avoided. Volatile anesthetic agents ma cause an increase in cerebral blood flow, cerebral bloo volume, and ICP. In the presence of intracranial hypertensio these agents should be used in moderation and in combina tion with hyperventilation and intravenous anesthetics wit favorable effects on ICP (e.g., thiopental, etomidate, pro pofol, fentanyl). If used at a minimum alveolar concentra tion (MAC) of 1.2 in combination with hyperventilatio

Table 174-1 • Determinants of Intracranial Pressure and Therapeutic Techniques to Lower It

Determinant	Therapeutic Intervention	Mechanism	Duration of Effe
Volume of parenchyma	Mannitol infusion	Osmotic reduction of brain water content	Hours to days
	Corticosteroids	Reduction of peritumoral or peri-inflammatory edema	Days to weeks
	Excision of mass	Volume reduction	Indefinite
	Craniectomy	Increased craniospinal compliance	Indefinite
Cerebrospinal fluid volume	Ventricular or lumbar puncture	Volume reduction	Hours
	Ventriculostomy or lumbar drain	Volume reduction	Days
	Ventricular or lumbar shunt	Volume reduction	Indefinite
Cerebral blood volume	Hyperventilation	Cerebral vasoconstriction due to decreased Pco_2	Hours
	Barbiturates	Cerebral vasoconstriction	Hours to days

Revised from Broaddus WC, Delashaw JB, Park TS: Anatomic, physiologic, and neurosurgical considerations in neuroanesthesia. In Sperry RJ, Stirt JA, Stone DJ (eds): Manual of Neuroanesthesia. Toronto, BC Decker, 1989.

desflurane and isoflurane have similar effects on cerebral perfusion pressure, mean arterial pressure, and lumbar CSF pressure. Nitrous oxide is cerebrostimulatory and increases cerebral blood flow and cerebral metabolic oxygen consumption, especially when combined with volatile anesthetics. Use of nitrous oxide should be avoided with pneumocephalus (e.g., recent craniotomy) because of its potential to diffuse into and expand intracranial and other air-containing spaces.

Further Reading

Adams RD, Victor M: Disturbances of cerebrospinal fluid circulation, including hydrocephalus and meningeal reactions. In Adams RD, Victor M (eds): Principles of Neurology. New York, McGraw-Hill, 1993, pp 539-553.

Bedell E, Prough DS: Anesthetic management of traumatic brain injury. Anesthesiol Clin North Am 20:417-439, 2002.

Bendo AA, Luba K: Recent changes in the management of intracranial hypertension. Int Anesthesiol Clin 38:69-85, 2000.

Coles JP, Minhas PS, Fryer TD, et al: Effect of hyperventilation on cerebral blood flow in traumatic head injury: Clinical relevance and monitoring correlates. Crit Care Med 30:1950-1959, 2002.

Diringer M: Hyperventilation in head injury: What we have learned in 43 years. Crit Care Med 30: 2142-2143, 2002.

Dutton R, McCunn M: Traumatic brain injury. Curr Opin Crit Care 9: 503-509, 2003.

Hickey R: Neurosurgical emergencies. Am Soc Anesthesiol Refresher Courses 24:97-110, 1996.

Imberti R, Bellinzona G, Langer M: Cerebral tissue Po_2 and $Sjvo_2$ changes during moderate hyperventilation in patients with severe traumatic brain injury. J Neurosurg 96: 97-102, 2002.

Kaye A, Kucera IJ, Heavner J, et al: The comparative effects of desflurane and isoflurane on lumbar cerebrospinal fluid pressure in patients undergoing craniotomy for supratentorial tumors. Anesth Analg 98:1127-1132, 2004.

Shapiro HM: Anesthesia and intracranial pressure. In Sperry RJ, Johnson JO, Stanley TH (eds): Anesthesia and the Central Nervous System. Dordrecht, Netherlands, Kluwer Academic Publishers, 1993, pp 119-138.

Venous Air Embolism

17

Jennifer E. Souders and Maurice S. Albin

Case Synopses

Gravitational Pressure Gradient of 7.5 cm H_2O

During a repeat lumbar laminectomy in the prone position with an orthopedic frame, a 55-year-old man suddenly develops severe hypotension, rapidly goes into electromechanical dissociation, has cardiac arrest, and cannot be resuscitated after 1 hour of effort.

Gravitational Pressure Gradient of 20 cm H_2O

A 42-year-old woman with acromegaly secondary to pituitary adenoma undergoes transsphenoidal resection of the tumor in the semisitting position (head elevated 30 degrees). Severe hypotension (60 mm Hg systolic) occurs when surgical manipulations are carried out in the area of the sella.

PROBLEM ANALYSIS

Definition

Air can enter the venous circulation when there is a negative gravitational gradient between the right atrium and the upper area of incision or the air's point of entrance. Albin and coworkers reported that a 5 cm H_2O gravitational gradient was sufficient to entrain air in a neurosurgical case. The entry of a bolus of 100 mL of air into the venous circulation can be fatal, and it has been calculated that this amount of air can pass through a 14-gauge needle with a gradient of 5 cm H_2O in a matter of seconds. Factors modifying air entrainment include body position, depth of ventilation, volume of air entering the vessel, rate of gaseous entry, and composition and concentration of gases in the inhaled anesthetic mixture. Animal studies and human cases have shown that the transpulmonary passage of air can occur without a patent foramen ovale. Reduced central venous pressure due to a contracted blood volume or hemorrhagic hypovolemia, or decreased intrathoracic pressure due to the use of a table or frame to reduce abdominal compression, can help increase the gravitational pressure gradient and enhance the entrainment of air.

The fate of entrained air is illustrated in Figure 175-1. In the first case synopsis, the gravitational gradient was probably less than 7.5 cm H_2O but was enhanced by blood loss and use of an orthopedic frame, which reduced abdominal pressure, allowing the development of negative intrathoracic pressure with expiration. Because 50% nitrous oxide (N_2O) was used, this increased the air bubble size by a factor of about two.[1] Autopsy revealed air in the coronary vessels, heart, spinal cord, and cerebral and mesenteric vessels, despite a non-probe-patent foramen ovale.

In the second case synopsis, more than 150 mL of air was aspirated from the central line after the hypotensive episode. The gravitational pressure gradient was at least 20 cm H_2O, and the air bubble volume was approximately doub because 50% N_2O again was used. Postoperatively, a te netium lung scan revealed a peripheral decortication pat in the posterosuperior portion of the right and left lung fi and an abrupt decrease in perfusion to the right middle l all due to the entrance of air into the pulmonary system.

These cases show that venous air embolism (VAE) occur in any position, as long as a pressure gradient all the ingress of air between the procedural area and the h (Table 175-1). Evidence has accumulated that VAE is from rare in patients undergoing procedures in the pr position, especially spinal procedures; there have been least 22 cases reported, with a total of 13 deaths, 10 of wh were in the pediatric age group. In addition to neurosurg VAE has been reported with virtually all surgeries endoscopy. It also occurs with catheterization for cardiac central vascular access, arteriovenous shunts, and int venous infusions and transfusion therapy.

Recognition

Physical signs and symptoms include gasping respiration spontaneously breathing patients, increased central veno and pulmonary artery pressures, cardiac arrhythmias, e trocardiographic (ECG) changes, hypotension, abnormal h sounds, changes in heart rate, decreased peripheral resista reduced cardiac output, cyanosis, a mill-wheel murmur, cardiac arrest. Increased pulmonary artery pressure is most prominent physical sign of VAE during controlled v tilation, irrespective of the volume or rate of air entrainm The more rapidly air enters the pulmonary circulation, more rapidly and severely the pulmonary artery pressure rise. If it rises dramatically over the systemic pressure, a rig to-left shunt can occur through a septal defect (i.e., pat foramen ovale) and cause paradoxical embolism of air i the left heart. ECG changes with air embolism are quite va able and include tachyarrhythmias, varying degrees of at oventricular block, right ventricular strain, and ST segme changes. Very large volumes of entrained air may cause su

[1]Increased gas bubble volume with N_2O is approximated as $100/(100 - FiN_2O) = 100/(100 - 50) = 2$.

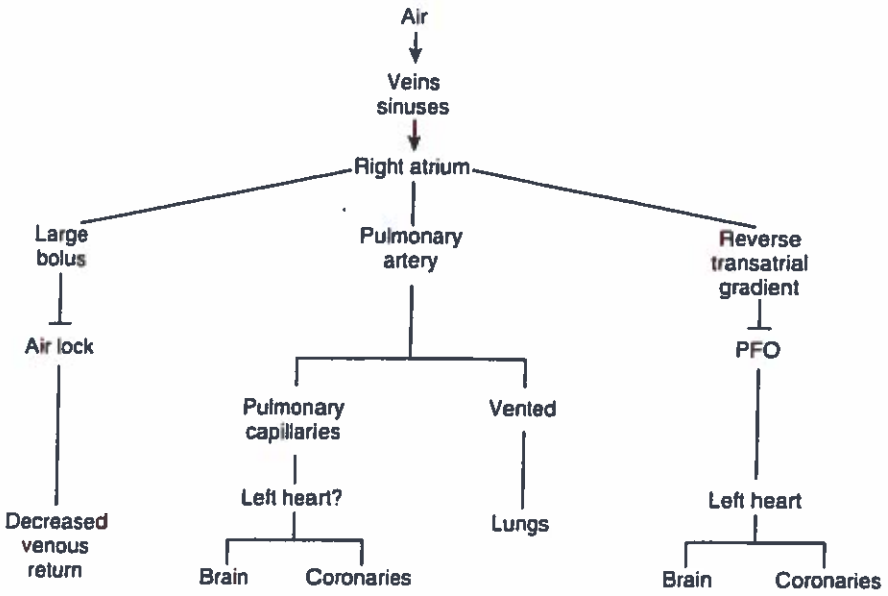


Figure 175–1 · Fate of entrained air after venous air embolism. PFO, patent foramen ovale.

severely increased right ventricular afterload that the right ventricle becomes ischemic and fails acutely. Right heart failure is the primary cause of acute hypotension, reduced cardiac output, and cardiac arrest after massive air embolism. A mill-wheel murmur indicates that a significant volume of air has entered the right heart chambers. If so, cardiac arrest may be imminent. Air causes this churning sound and is one of the last signs observed.

Besides physical signs and symptoms, the other methods for detecting intraoperative air embolism, in order of sensitivity, are transesophageal echocardiography (TEE), precordial Doppler ultrasonography, end-tidal carbon dioxide (CO_2), pulmonary artery catheter, pulse oximetry, and direct observation of the surgical site. TEE can detect both venous and paradoxical embolism consisting of as little as 0.02 mL/kg of air. However, it is expensive and may be inaccessible in some surgical locations; it has no audible alarms and may be difficult for solo practitioners to use when they are occupied with urgent patient care duties. A well-positioned precordial Doppler probe detects 0.05 mL/kg of intravascular air, is noninvasive, and alerts both the anesthesiologist and the surgeon simultaneously. As mentioned earlier, although pulmonary artery catheters can show early and prominent signs of air embolism, they are highly invasive and less sensitive than precordial Doppler.

A sudden reduction in end-tidal CO_2 concentration is the most convenient and widely used noninvasive method for detecting air embolism. The magnitude and duration of the decrease in end-tidal CO_2 correlate positively with the volume of air entrained, and detection is possible during any general anesthetic. In contrast, pulse oximetry is relatively insensitive, because decreases in arterial oxygen saturation often occur late with a decrease in arterial oxygen tension. Further, the surgical field is often overlooked. Especially in high-risk surgery, it may be easy to see whether there is a lack of venous oozing, indicating subatmospheric venous pressure. In high-risk procedures, combined precordial Doppler ultrasonography and end-tidal CO_2 monitoring should be used. Doppler tone activation and reduced end-tidal CO_2 signal air entrainment. VAE is confirmed if gas bubbles can be aspirated from a central line.

Risk Assessment

The incidence of VAE is uncertain, largely because the criteria for VAE vary. Nevertheless, we have a general idea about

Table 175-1 • Incidence of Air Embolism in Neurosurgery by Position

Position	No. of Patients	Detected Embolism		Air Aspirated (mL)	Gradient (cm H_2O)
		No.	%		
Sitting	400	100	25.0	2-500	20-65
Lateral	60	5*	8.3	3-200	5-18
Supine	48	7†	14.6	2-150	5-18
Prone	10	1‡	10.0	45	7.5
Total	518	113	21.8		

*Two cases of tic douloureux, two cases of hemifacial spasm, one case of tumor.
†Three cases of transsphenoidal hypophysectomy, three cases of intracranial tumor, one case of tic douloureux (air was detected after reapplication of the pinhead holder while the patient was in the supine position, before being put in the sitting position).
‡Ependymoma of the spinal cord.
From Albin MS, Carroll RG, Maroon JC: Clinical considerations concerning detection of air embolism. Neurosurgery 3:380-384, 1978.

the incidence of VAE and the associated morbidity and mortality rates for neurosurgical procedures performed with the patient in the sitting position. The overall incidence is about 25%, ranging from 2% to 60%. In 10 studies of more than 5000 patients, the mortality rate did not exceed 1% in any individual report. Morbidity data, even in neurosurgical sitting cases, are more difficult to ascertain. Albin and coworkers reported 100 cases of VAE in 400 patients operated on in the sitting position. These patients were considered to have VAE only if both Doppler activation and visual aspiration of air from a central line occurred. Under these conditions, 25 of the 100 patients with recognized VAE developed symptoms ranging from severe hypotension to cardiac arrest. Paradoxical air embolism (air entering the left side of the heart via a patent foramen ovale or transpulmonary passage) caused significant mortality in the small number of cases reported. Somewhat surprisingly, most VAE-related mortality appears to occur in non-neurosurgical cases, possibly because anesthetists fail to appreciate that it can occur in these cases and the patient is not monitored adequately for VAE. Adding to this lack of appreciation is the medicolegal "fear factor," which likely leads to underreporting of VAE in the medical literature. There is a significant risk of VAE in cesarean section, spinal surgery, and total hip arthroplasty.

Implications

Because of coalescence and filming of bubbles at the blood-bubble interface, the passage of air into the right atrium can impede or even halt venous return to the right side of the heart. The consequences are hypotension, arrhythmias, and even circulatory arrest, because cardiac output can be severely compromised. The occurrence of an "airlock" in the right ventricle has been postulated as the cause for hemodynamic collapse with massive VAE. However, more recent studies indicate that right ventricular dysfunction is more likely the result of an acute increase in afterload. Continuous entrainment and passage of large volumes of air may lead to the inability of the lungs to adequately vent air from the pulmonary circulation. This results in the liberation of vasoactive substances from the blood-air interface, leading to pulmonary perfusion deficits.

Ventilation-perfusion inhomogeneity is due to the redistribution of pulmonary perfusion. Areas of dead space and high ventilation-perfusion ratios reduce end-tidal CO_2 and increase arterial CO_2 tension. Hypoxia results from altered intrapulmonary shunt, mixed venous oxygen saturation, and redistribution of pulmonary blood flow to regions that are relatively overperfused and underventilated (low ventilation-perfusion ratio). These ventilation-perfusion defects can be variable, because the distribution of air in the pulmonary vessels is a function of both buoyancy and regional pulmonary perfusion. Although ventilation-perfusion inhomogeneities may resolve in as little as 30 minutes after VAE, they can also become progressively worse as a result of the inflammatory response to air in the vascular space. Continuous entrainment of large volumes of air can lead to progressive pulmonary compromise, pulmonary capillary leak, and acute respiratory distress syndrome. Such volumes of air may also reach or exceed the threshold for transpulmonary passage of air, so that it enters the left side of the heart and coronary sinuses and moves into the brain. This can lead to coronary occlusion and cardiac arrest as well as cerebral air embolization, with stroke and associated dysfunction.

MANAGEMENT AND PREVENTION

Given the severity of VAE sequelae, prevention and early detection are far preferable to management after the fact. The key to preventing VAE is a greater appreciation of risk factors. Patients who will undergo procedures in which a gravitational gradient will be present, blood loss may be significant, or the surgical site is in a highly vascular area are predisposed to air entrainment and VAE. Good examples from the literature include radical retropubic prostatectomy and repeat lumbar or thoracic laminectomies in the prone position.

Monitoring for VAE should include ECG, blood pressure, pulse oximeter, end-tidal CO_2, precordial Doppler, and a multiorificed catheter with its tip 1 to 2 cm past the junction of the right atrium and superior vena cava (Fig. 175-2). Although the last is important for treatment, the ability to aspirate air from this catheter leaves no doubt about the diagnosis. Further, the transducer of the right atrial catheter can be placed at the level of the surgical site to determine whether a negative pressure gradient exists. In patients thought to be at risk for VAE and in whom invasive monitoring is contemplated, the use of an indwelling catheter for arterial blood gas and pressure monitoring is also advised.

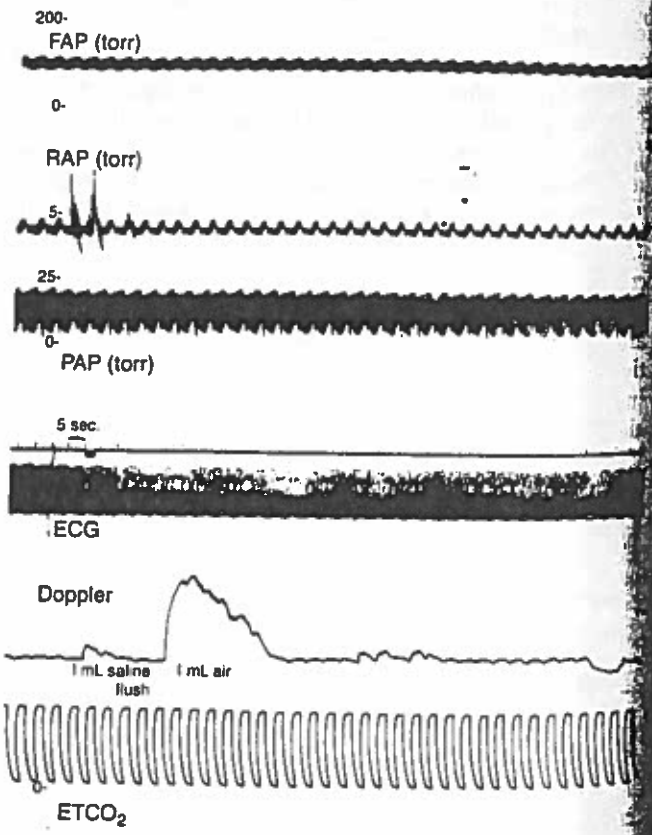


Figure 175–2 • Monitoring for venous air embolism. ECG, electrocardiogram; $ETCO_2$, end-tidal CO_2; FAP, femoral artery pressure; PAP, pulmonary artery pressure; RAP, right atrial pressure.

Preventive measures for VAE are few and may be contraindicated in certain patients. Hydration can be used to decrease the pressure gradient between the right heart and the surgical site, provided the patient can tolerate increased right ventricular preload. Many patients with intracranial pathology are not suitable candidates. Although the use of positive end-expiratory pressure to increase intrathoracic pressure has been proposed, it may increase right ventricular preload and is also controversial because it may increase the transatrial gradient and open a patent foramen ovale, thus allowing air to egress into the left heart and brain. For intracranial surgery, bilateral manual jugular venous compression temporarily elevates cerebral venous pressure, thereby preventing ongoing cerebral air embolism; it may also help localize the source. This maneuver is safe and effective, but only if applied gently and transiently in patients without preexisting carotid artery disease.

With Doppler activation, a decrease in end-tidal CO_2, or both, the central line must be aspirated *immediately* (using a 50-mL syringe attached to a stopcock). A delay of even a few seconds might allow the entrance of large volumes of air. At the same time, inspired N_2O or air should be replaced with 100% oxygen, and the surgeons should be notified to flood the field with water and look for any open veins. Any resulting hypotension or cardiac arrhythmias should be treated symptomatically with positive inotropes and vasopressors to improve contractility and support the circulation. Epinephrine is the drug of choice for resuscitation from massive VAE. If recovery to pre-VAE physiologic levels does not occur in a very short time, or if air continues to be aspirated, the patient should be returned to a position in which there is no gradient present.

In the event VAE is suspected and the patient remains comatose after surgery or has a neurologic deficit that is thought to be unrelated to the surgical procedure, neurology or neurosurgery consultation is in order, and magnetic resonance imaging should be performed to diagnose the presence of intra-axial air. If air is visualized, a course of hyperbaric oxygen therapy should be considered.

Further Reading

Adornato DC, Gildenberg PL, Ferrario CM, et al: Pathophysiology of intravenous air embolism in dogs. Anesthesiology 49:120-127, 1978.

Albin MS: Air embolism. In Albin M (ed): Textbook of Neuroanesthesia with Neurosurgical and Neuroscience Perspectives. New York, McGraw-Hill, 1997, pp 109-125.

Albin MS, Carroll RG, Maroon JC: Clinical considerations concerning detection of venous air embolism. Neurosurgery 3:380-384, 1978.

Albin MS, Wills J, Schwend RM: Patent foramen ovale and unexplained stroke. N Engl J Med 354:1753-1755 (Letter to the Editor), 2006.

Black S, Cucchiara RF, Nishimura RA, et al: Parameters affecting occurrence of paradoxical air embolism. Anesthesiology 71:235-241, 1989.

Chang JL, Albin MS, Bunegin L, et al: Analysis and comparison of venous air embolism detection methods. Neurosurgery 7:135-141, 1980.

Drummond JC, Prutow RJ, Scheller MS: A comparison of the sensitivity of pulmonary artery pressure, end-tidal carbon dioxide, and end-tidal nitrogen in the detection of venous air embolism in the dog. Anesth Analg 64:688-692, 1985.

Fong J, Gadalla R, Pierri MK, et al: Are Doppler-detected venous emboli during cesarean section air emboli? Anesth Analg 71:254-257, 1990.

Geissler MJ, Allen SJ, Mehlhorn U, et al: Effect of body repositioning after venous air embolism: An echocardiographic study. Anesthesiology 86:710-717, 1997.

Matjasko J, Petrozza P, Cohen M, et al: Anesthesia and surgery in the seated position: Analysis of 554 cases. Neurosurgery 17:695-702, 1985.

Newfield P, Albin MS, Chestnut JC, et al: Air embolism during transsphenoidal pituitary operations. Neurosurgery 2:39-42, 1978.

Souders JE: Pulmonary air embolism. J Clin Monit Comput 16:375-383, 2000.

Souders JE, Doshier JB, Polissar NL, Hlastala MP: Spatial distribution of venous gas emboli in the lungs. J Appl Physiol 87:1937-1947, 1999.

Toung T, Ngeow YK, Long DL, et al: Comparison of the effects of positive end-expiratory pressure during continuous venous air embolism in the dog. Anesthesiology 64:724-729, 1986.

Verstappen FT, Bernards JA, Kreuzer AF: Effects of pulmonary gas embolism on circulation and respiration in the dog. I. Effects on circulation. Pflugers Arch 386:89-96, 1977.

Verstappen FT, Bernards JA, Kreuzer AF: Effects of pulmonary gas embolism on circulation and respiration in the dog. II. Effects on respiration. Pflugers Arch 386:599-604, 1977.

Wills J, Schwend RM, Paterson A, Albin MS: Intraoperative visible bubbling may be the first sign of venous air embolism during posterior surgery for scoliosis. Spine 30:E629-E635, 2005.

Posterior Fossa Surgery 176

Donald S. Prough and Eric Bedell

Case Synopsis

A 28-year-old woman undergoes posterior fossa craniotomy for removal of a brainstem tumor. Preoperative symptoms included headache, facial asymmetry, and difficulty swallowing. The intraoperative course is complicated by periods of bradycardia sufficient to reduce blood pressure and a brief episode of asystole. At the conclusion of the case, the patient opens her eyes and follows simple commands, but she has no spontaneous respiratory efforts and only a weak cough and gag.

PROBLEM ANALYSIS

Definition

The most important aspect of posterior fossa surgery is location. A review of posterior fossa anatomy demonstrates that lesions, stimulation, or damage to small areas associated with the brainstem or cerebellum can profoundly influence the operative course and long-term outcome of neurosurgical patients.

Anatomically, the posterior fossa is defined posteriorly by the occipital bone; laterally by the occipital, temporal (petrous and mastoid portions), and parietal (posteroinferior angle) bones; anteriorly by the sphenoid (clivus), temporal (petrous), and occipital (clivus) bones; superiorly by the tentorium cerebelli; and inferiorly by the foramen magnum. Important structures located within the posterior fossa include the cerebellum, cerebral aqueduct, fourth ventricle, midbrain, pons, medulla, and proximal spinal cord. Located within these structures are the nuclei for all cranial nerves and important afferent and efferent tracts.

The oculomotor nerve (3rd cranial nerve) originates in the rostral midbrain, acquires parasympathetic fibers from the Edinger-Westphal nucleus, and courses ventrally through the midbrain. The trochlear nerve (4th cranial nerve) arises from the contralateral caudal midbrain and decussates before traveling ventrally. Other midbrain structures include the corticospinal and corticobulbar tracts, substantia nigra, red nuclei, and decussation of the superior cerebellar peduncles. The pons contains the nuclei for the trigeminal (5th), abducens (6th), facial (7th), and auditory (8th) cranial nerves. The medulla contains the remaining cranial nerves: glossopharyngeal (9th), vagus (10th), spinal accessory (11th), and hypoglossal (12th). The medulla also contains the decussation of the corticospinal tracts ventrally and the inferior cerebellar peduncles posteriorly.

From the perspective of intraoperative and postoperative management, one of the most important considerations is that critical respiratory and cardiovascular control centers reside in the brainstem. Involuntary respiratory control is a complex process involving multiple structures, including the pneumotaxic center (upper pons), which is involved in the transition from inspiration to exhalation; the apneustic center (lower pons), which is involved in the control of inspiration; and the medullary respiratory center (dorsal and ventral respiratory groups), which influences both inspiration and exhalation and coordinates those functions with extracranial nerve input. Vasomotor and cardiac centers, located predominantly in the medulla, powerfully influence resting vascular tone, blood pressure, and heart rate.

Lesions of the posterior fossa can generate diffuse or localized signs and symptoms, depending on the structures where the lesions are located or the structures compressed by mass lesions. A small lesion impinging on the cerebral aqueduct may result in obstructive hydrocephalus (producing symptoms such as headache and altered mental status). Similarly, a small lesion located in the lateral pons may result in isolated cranial nerve dysfunction. Therefore, important clinical data include the anatomic location of any posterior fossa lesion and the presence and magnitude of associated neurologic or systemic compromise.

Intraoperative stimulation, retraction, or damage to structures located within the posterior fossa may activate or inhibit nearby nuclei, leading to rapid and dramatic systemic responses. Intraoperative damage to adjacent structures may result in postoperative alterations in neuronal function (either activation or inhibition), leading to a wide array of clinical problems for postsurgical patients and for those providing postoperative care.

The typical presentation of complications related to postoperative edema or bleeding may differ in important respects from that seen after supratentorial surgery. In general, supratentorial lesions lead to a rostral-to-caudal progression of signs and symptoms. This progression may include headache, mental status changes, respiratory alterations, pupillary and oculomotor changes, hemodynamic changes, and, finally, motor abnormalities. In posterior fossa lesions, deterioration may be rapid, may fail to demonstrate a pattern of deterioration, and may present with localized cranial or brainstem deficits.

Finally, the surgical approach to the posterior fossa must be considered. The three general approaches are sitting, prone, and lateral (either routine or exaggerated, such as the three-quarter prone–park bench position). Each position has its own risks and benefits and will influence anesthetic management. Because of the significant risk of venous air entrainment (as high as 30%), posterior fossa craniotomies in the sitting position are being performed less frequently. However, even with horizontal positioning,

venous air embolism—with the attendant risk of cardiovascular collapse and paradoxical air embolism—is a possibility that should be considered in all posterior fossa surgery (also see Chapters 168 and 175).

Recognition

Special care is required when evaluating and managing patients with posterior fossa lesions. Mass lesions located within the posterior fossa or that compress posterior fossa structures can generate diffuse or localized signs and symptoms. A thorough preoperative evaluation, with attention to signs and symptoms produced by such lesions, is mandatory. Intraoperatively, vigilance for signs and symptoms of possible stimulation of or damage to critical portions of the brainstem and cerebellum is paramount. This extends to the postoperative period as well.

Owing to the risks of hydrocephalus, cranial nerve dysfunction, and alterations in respiratory function, extreme caution must be used when administering any form of sedative, hypnotic, or analgesic medications. Even small doses of benzodiazepines or narcotics may produce unacceptable respiratory depression. Therefore, they should be administered only when patients are directly monitored. For lesions involving the pons and medulla, airway maintenance and protective reflexes may be impaired by bulbar dysfunction, making aspiration and airway compromise a significant risk. Monitoring should include frequent evaluation of the level of consciousness, airway maintenance and protection, oxygenation (e.g., pulse oximetry), ventilation (capnography), heart rate, and blood pressure. The importance of frequent neurologic examination and assessment of ventilation and cardiovascular function cannot be overemphasized.

Intraoperative monitoring during posterior fossa surgery can be complex and must be tailored to the brain regions at highest risk during surgery. The cerebellum, though important for the patient's coordination and long-term function, has relatively little impact on intraoperative anesthetic management. Lesions in other areas may have more intraoperative impact. They often require other techniques, which can be roughly divided into (1) monitoring for nerve function and (2) monitoring for other dangerous conditions (e.g., hemodynamic instability, airway compromise, respiratory insufficiency).

Intraoperative monitoring for nerve integrity and function is often accomplished through provocative testing. Common techniques include somatosensory evoked potentials and facial nerve monitoring. In each case, specific monitoring modalities are used in an attempt to assess the integrity and function of the nerve or nerve pathways at risk. These techniques often have anesthetic implications (e.g., stable, low concentrations of potent inhalational agents to avoid excessive attenuation of somatosensory evoked potentials) and thus require appropriate anesthetic management to provide the best monitoring conditions. Failure to appreciate the specific monitoring needs for the proposed surgery may result in inadequate patient monitoring and suboptimal outcomes.

Intraoperative monitoring for hemodynamic instability and postoperative monitoring for neuronal dysfunction, hemodynamic instability, airway compromise, or respiratory insufficiency are important mandates for anesthesiologists. Stimulation of or damage to brainstem cardiac and vasomotor centers can lead to rapid and unpredictable hemodynamic changes. Extreme heart rate and blood pressure alterations are common with surgical manipulation, and rapid diagnosis and treatment are required.

Consideration of the manner of treatment is also important, for neurosurgeons often rely on hemodynamic changes to guide the extent of surgical exploration. Thus, prophylactic treatment of heart rate (i.e., with vagolytic agents) and blood pressure is generally discouraged. In practice, it is more important to recognize when a critical portion of the brainstem is stressed than to blunt all hemodynamic responses.

The risk of venous air embolism is also a consideration in all posterior fossa surgery, and there should be a plan in place for diagnosis and management. Finally, there are no adequate intraoperative monitors for a large number of important brainstem functions, such as airway maintenance and protection, swallowing, and respiratory control. Thus, anesthetic management must be planned and executed to provide rapid and clear emergence with tight hemodynamic control.

Close communication between the anesthesiologist and the neurosurgeon is vital. Specifically, hemodynamic parameters, expected neuronal or bulbar dysfunction, and anticipated alterations in airway protection and respiratory function should be discussed. Postoperative ventilatory support, intubation, or diagnostic studies (e.g., angiography, computed tomography, magnetic resonance imaging) must be discussed before emergence, and appropriate plans must be developed in light of those discussions.

Risk Assessment

Knowledge of the anatomic location of the lesion of interest, the planned surgical procedure, and the actual structures involved in the surgery is a critical element of posterior fossa surgery. Risk assessment is possible only after a review of the individual patient's history and physical examination, an evaluation of radiologic studies, and a discussion with the neurosurgeon. The greatest risks are associated with tumors directly involving the brainstem (e.g., pons and medulla), lesions with direct involvement of the cranial nerves required for airway maintenance and protection, lesions involving the facial nerve, and surgeries conducted with the patient in the sitting position. The actual events encountered during surgery are impossible to predict, which contributes to the challenge of providing anesthesia for neurosurgery in general and for posterior fossa surgery in particular. At a minimum, plans for the diagnosis and management of hemodynamic instability, respiratory dysfunction, alterations in cranial nerve function, and venous air embolism should be made before starting any posterior fossa surgery.

Implications

The risks to patients undergoing posterior fossa surgery can be divided into preoperative, intraoperative, and postoperative complications. Before surgery, patients must be carefully monitored, and sedative-hypnotic and analgesic drugs must be titrated with extreme care. Intraoperative risks are

predominantly hemodynamic instability and cardiovascular collapse. Especially with surgery involving the pons and medulla, extreme hemodynamic variability in heart rate and blood pressure may result in patient instability. This instability is usually limited to periods of direct surgical retraction and manipulation, but it can be clinically important. Hemodynamic collapse and cardiac arrest have resulted from venous air entrainment, and both are a constant risk during all posterior fossa (and skull base) surgery, even in patients who are horizontally positioned. Important postoperative risks include alterations in respiratory function, rapid development of increased posterior fossa pressure (e.g., from hematoma formation), development of hydrocephalus, and alterations in cranial nerve function. Because there are limited intraoperative methods of monitoring for these possibilities, rapid and clear emergence from anesthesia with limited respiratory depression and tight hemodynamic control is of primary importance and should be a major determinant in the choice of anesthetic technique.

MANAGEMENT

A full understanding of the patient's condition and anticipated surgical requirements represents an important part of management. Failure to understand the specific location and effect of the posterior fossa lesion severely limits the delivery of optimal therapy. Support and protection of oxygenation and ventilation should be the primary focus when managing complications associated with posterior fossa surgery. Hemodynamic monitoring and modification of heart rate and blood pressure through the use of vasoactive medications are common requirements during posterior fossa surgery. However, remember that prophylactic treatment of heart rate and blood pressure is not indicated, because the surgeon often depends on the development of hemodynamic alterations to guide ongoing surgery.

Postoperatively, to determine the need for ongoing monitoring and support, all cranial nerve and brainstem functions associated with the site of surgery should be specifically evaluated once the patient is awake. This requires that the anesthetic technique permit neurologic examination at the conclusion of surgery, preferably in the operating room before transport to the intensive care unit.

Black and coworkers reported their experience with 570 posterior fossa craniotomies performed in 1981 through 1984. During this period, the number of sitting position craniotomies performed at the Mayo Clinic markedly decreased, while the number of horizontal position craniotomies markedly increased. Overall, there were no significant differences in mortality or other postoperative outcomes between patients undergoing surgery in the two positions (Table 176-1). The incidence of important complications was substantial after surgery in either position.

The time course and presenting signs and symptoms of posterior fossa deterioration may be different from those associated with supratentorial surgery. With supratentorial lesions, deterioration (usually due to an expanding mass or hydrocephalus) generally progresses over time, so serial monitoring is appropriate. For posterior fossa surgery, rapid localized deterioration may occur, leading to a loss of bulbar function, respiratory arrest, or hemodynamic collapse. Thus vigilance and a high index of suspicion must be maintained into the postoperative period.

Table 176-1 ▪ Postoperative Outcomes Not Affected by Position

Outcome	Sitting (*N* = 333)		Horizontal (*N* = 246)	
	No.	*%*	*No.*	*%*
Mental status deteriorated	14	4	9	4
Mental status improved	7	2	12	5
Eye injury	8	2	12	5
Seizures	6	2	2	1
Motor deficit new or worse	17	5	16	6
Motor deficit improved	29	9	9	4
Sensory deficit new or worse	6	2	5	2
Sensory deficit improved	19	6	4	2
Complete loss of facial nerve function	23	7	26	11
Perioperative myocardial infarction	1	0.3	4	1.6
Respiratory complications	7	2	8	3
Coma (>1 wk)	6	2	3	1
Cerebrovascular accident	8	2	8	3
Congestive heart failure	1	0.3	0	
Hemodynamic instability	5	1.5	10	4
Pulmonary embolus	0		2	1
Re-exploration for bleeding	6	1.8	6	2.4
Re-exploration for infection	2	0.6	2	0.8
Acute mortality (within 30 days)	9	2.7	5	2
Quadriparesis	0		0	
Symptomatic pneumocephalus	0		0	
Peripheral nerve injury	0		0	
Laryngeal or lingual edema	0		0	

From Black S, Ockert DB, Oliver WC Jr, Cucchiara RF. Outcome following posterior fossa craniectomy in patients in the sitting or horizontal positions. Anesthesiology 69:49-56, 1988.

PREVENTION

Careful evaluation of the patient and discussion with the surgeon about location, impact, and proposed surgical approach are required for the optimal management of patients undergoing posterior fossa surgery. Anticipation of the more common severe complications, such as postoperative venous air embolism and airway or respiratory dysfunction, is a critical part of anesthetic management, as is recognition of the need for specialized monitoring techniques. Although serious complications associated with posterior fossa surgery are uncommon with current surgical procedures, a high index of suspicion and constant vigilance are the most important aspects of perioperative care.

Further Reading

Black S, Ockert DB, Oliver WC Jr, Cucchiara RF: Outcome following posterior fossa craniectomy in patients in the sitting or horizontal positions. Anesthesiology 69:49-56, 1988.

Endo T, Sato K, Takahashi T, Kato M: Acute hypotension and bradycardia by medulla oblongata compression in spinal surgery. J Neurosurg Anesth 13: 310-311, 2001.

Nolte J: The Human Brain: An Introduction to Its Functional Anatomy, 5th ed. St. Louis, Mosby–Year Book, 2002.

Plum F, Posner JB: The Diagnosis of Stupor and Coma, 3rd ed. Philadelphia, FA Davis, 1982.

Williams DL, Umedaly H, Martin IL, Boulton A: Chiari type I malformation and postoperative respiratory failure. Can J Anaesth 47:1220-1223, 2000.

Pituitary Tumors: Diabetes Insipidus

177

Melissa A. Laxton and Patricia H. Petrozza

Case Synopsis

A 58-year-old man undergoes transsphenoidal hypophysectomy for resection of a prolactin-secreting pituitary adenoma with suprasellar extension. Ten hours after surgery, urine output exceeds 3 L/hour, and the serum sodium level is 150 mEq/L.

PROBLEM ANALYSIS

Definition

Diabetes insipidus is a syndrome characterized by polyuria, thirst, and polydipsia triggered by plasma hyperosmolarity. *Neurogenic diabetes insipidus* results from insufficient antidiuretic hormone (ADH) secretion, secondary to damage to the hypothalamic-neurohypophysial axis. Loss of approximately 75% of ADH-secreting neurons is needed for the development of clinically relevant polyuria. In contrast, *nephrogenic diabetes insipidus* is characterized by renal resistance to the action of ADH.

An absolute deficiency of ADH results in impaired urine concentrating ability, polyuria, and a tendency toward dehydration. Most patients have incomplete neurogenic diabetes insipidus and retain a limited ability to concentrate urine and conserve free water. However, if access to water is impaired (e.g., unconsciousness, perioperative nothing-by-mouth status), hypertonic dehydration and hypernatremia may develop. Signs and symptoms of hypernatremia include psychomotor agitation, neuromuscular irritability, lethargy, coma, and seizures.

Recognition

Diabetes insipidus occurs in as many as 20% of adult patients after transsphenoidal pituitary surgery. The syndrome is usually transient in this setting, and perioperative glucocorticoid replacement may facilitate the development of polyuria. Often, polyuria appears on or before the first postoperative day. The polyuria of diabetes insipidus is characterized as follows:

- A 24-hour urine volume greater than 50 mL/kg
- Urine osmolarity greater than 300 mOsm/kg H_2O
- Urine specific gravity less than 1.010

Chronic polyuria causes the hypertonic renal medullary concentration gradient to be "washed out." Additional urine concentrating mechanisms become impaired, so that polyuria increases. Alternative causes of polyuria must be eliminated to make the diagnosis of primary neurogenic or nephrogenic diabetes insipidus with confidence (Table 177-1).

Risk Assessment

As noted earlier, transient diabetes insipidus occurs in up to 20% of patients after transsphenoidal hypophysectomy. However, it becomes permanent in about 2% of cases. A macroadenoma with suprasellar extension is associated with a higher risk for postoperative diabetes insipidus than is a lesion confined to the sella. Recent data suggest that an endoscopic transsphenoidal approach for resection of pituitary tumors may decrease both the short- and long-term incidence of diabetes insipidus compared with the traditional direct transsphenoidal approach. The secretory type of tumor appears to have no effect on the postoperative occurrence of diabetes insipidus.

Postoperative diabetes insipidus is usually recognized within 12 to 24 hours of the initial insult, but delays of days to weeks have been recorded. In approximately 50% to 60% of cases, diabetes insipidus is transient, lasting only 3 to 5 days. More rarely, it may last several weeks, followed by gradual resolution. This pattern is more common after resection of pituitary adenomas confined to the sella. After transcranial approaches to pituitary macroadenomas with suprasellar extension, or procedures in which proximal damage to the pituitary stalk is likely, both complete and partial diabetes insipidus have been observed; in some cases, it takes several years for this condition to improve or resolve.

A small group of patients (5% to 10%) exhibits a classic triphasic response to injury. This pattern most commonly

Table 177-1 • Causes of Polyuria Other Than Primary Neurogenic or Nephrogenic Diabetes Insipidus

Chemical diuresis
Mannitol
Urea
Radiocontrast agents
Hyperglycemia
Furosemide, thiazides, ethacrynic acid
Acute renal failure
Drug-induced nephrogenic diabetes insipidus (e.g., cisplatin, lithium)
Postobstructive diuresis
Postresuscitation diuresis

follows hypophysial stalk injury due to severe head trauma or the resection of extensive suprasellar tumors. The initial phase is characterized by an abrupt cessation of ADH release. This is followed by polyuria, which begins within 12 to 24 hours after injury and lasts for 4 to 8 days. An antidiuretic phase, lasting 5 to 6 days, follows. It is characterized by concentrated urine, with plasma hyposmolarity and hyponatremia as a result of free water reabsorption. Profound hyponatremia and its attendant complications may develop if there is a delay in recognizing this phase. Excessive release of stored ADH from degenerating neurohypophysial tissues is the likely explanation for this antidiuretic phase. Once this stored ADH release is complete, diabetes insipidus frequently recurs. Although usually persistent, sometimes it may improve or resolve.

Implications

A patient with diabetes insipidus is unable to concentrate urine and retain water. Without treatment, intravascular volume depletion results, cardiac stroke volume declines, and heart rate increases in an effort to maintain cardiac output. Hypoperfusion may be signaled by weak peripheral pulses; orthostatic hypotension; cold, clammy skin; rapid, shallow respirations; and a reduced level of consciousness. Hypernatremia may manifest as seizures and hyperreflexia.

MANAGEMENT

Owing to the predominantly transient nature of perioperative diabetes insipidus, some mild cases are managed with oral fluid replacement, especially if the patient is cooperative and the thirst mechanism is intact. However, if the patient is unable to cooperate, and there is associated hypokalemia and concern about "wash-out" of the renal medullary concentration gradient, more aggressive therapy may be warranted.

Exogenous replacement of ADH is with either desmopressin or aqueous vasopressin. After transsphenoidal resection, desmopressin is usually administered subcutaneously in a dosage of 1 to 2 μg every 8 to 12 hours. Desmopressin lacks the vasoconstrictor effects of vasopressin and is less likely to cause hypertension or abdominal cramping. For patients requiring long-term ADH replacement, both intranasal and oral preparations are available. However, the dose must be titrated individually.

Although desmopressin is clearly the drug of choice for the chronic treatment of diabetes insipidus, its duration of action is 12 to 18 hours. Some clinicians prefer aqueous vasopressin if diabetes insipidus is likely to be transient. Aqueous vasopressin is formulated as 20 pressor units/mL of solution. The peak effect occurs by 1 to 2 hours, and the duration of action is 4 to 8 hours. The usual starting dosage is 2 to 5 units subcutaneously or intramuscularly every 4 to 6 hours as needed.

Careful assessment of fluid intake; urine output, osmolarity, and specific gravity; plasma osmolarity; serum sodium concentration; and body weight should guide therapy with vasopressin or desmopressin. Clinicians must be alert to the possible development of an antidiuretic phase of hormonal dysfunction, complicated by water intoxication.

PREVENTION

Meticulous surgical resection is the best means of preventing perioperative diabetes insipidus. Anesthesiologists should maintain a high index of suspicion for the development of diabetes insipidus, especially when there is suprasellar extension of a pituitary tumor or other endocrine abnormalities in a neurosurgical patient.

Further Reading

Arafah BM, Nasrallah MP: Pituitary tumors: Pathophysiology, clinical manifestations and management. Endocr Relat Cancer 8:287-305, 2001.

Barker FG II, Klibanski A, Swearingen B: Transsphenoidal surgery for pituitary tumors in the United States, 1996-2000: Mortality, morbidity, and the effects of hospital and surgeon volume. J Clin Endocrinol Metab 88:4709-4719, 2003.

Carrau RL, Kassam AB, Snyderman CH: Pituitary surgery. Otolaryngol Clin North Am 34:1143-1155, 2001.

Fukuda I, Hizuka N, Takano K: Oral DDAVP is a good alternative therapy for patients with central diabetes insipidus: Experience of five-year treatment. Endocr J 50:437-443, 2003.

Rajaratnam S, Seshadri MS, Chandy MJ, et al: Hydrocortisone dose and postoperative diabetes insipidus in patients undergoing transsphenoidal pituitary surgery: A prospective randomized controlled study. Br J Neurosurg 17:437-442, 2003.

Singer PA, Sevilla LJ: Postoperative endocrine management of pituitary tumors. Neurosurg Clin N Am 14:123-138, 2003.

Vance ML: Perioperative management of patients undergoing pituitary surgery. Endocrinol Metab Clin North Am 32:355-365, 2003.

Verbalis JG: Management of disorders of water metabolism in patients with pituitary tumors. Pituitary 5:119-132, 2002.

Intracranial Aneurysms: Rebleeding

178

Philippa Newfield

Case Synopsis

A 64-year-old man undergoing craniotomy for clip-ligation of a right anterior communicating artery aneurysm 12 hours after initial subarachnoid hemorrhage becomes acutely hypertensive and experiences bradycardia during the induction of anesthesia.

PROBLEM ANALYSIS

Definition

Subarachnoid hemorrhage (SAH) from the rupture of an intracranial aneurysm (ICA) occurs with a frequency of 6 to 8 per 100,000 persons in most Western populations. Rates of ICA rupture are 0.05% to 6% per year, depending on the size and location of the aneurysm. The risk of rupture is 11 times greater in patients with previous SAH than in those with symptomatic aneurysms.

Rebleeding is the occurrence of further hemorrhage after the initial SAH. Such episodes can be catastrophic, with high mortality and chronic morbidity rates. If untreated, 50% of ruptured ICAs rebleed within 6 months of the initial SAH. The incidence of rebleeding is highest within 24 hours of SAH (4%); it then declines to 1% to 2% per day for the next 13 days. About 20% to 30% of ruptured ICAs rebleed within 30 days of the initial SAH. Another 10% to 15% of patients rebleed during the ensuing 5 months.

ICA rerupture produces neurologic deterioration by raising intracranial pressure (ICP) and impairing cerebral perfusion. Many complications may ensue (Table 178-1). Hydrocephalus can develop acutely, because sudden clot deposition throughout the subarachnoid space blocks the passage of cerebrospinal fluid (CSF) through the basal subarachnoid cisterns. Late-onset hydrocephalus is due to obstruction of CSF drainage pathways by organized subarachnoid clot.

Brain infarction also occurs due to direct, hematoma-induced brain destruction or shifts in the intracranial contents, along with vascular compromise. The larger the volume of subarachnoid blood and the greater the ICP, the more likely it is that cerebral blood flow (CBF) will be reduced and the patient's neurologic condition will worsen. SAH also impairs autoregulation, the ability of the brain to maintain CBF fairly constant over mean arterial pressures between 50 and 150 mm Hg, and it reduces the cerebral metabolic rate of oxygen (O_2) consumption.

The incidence of intraoperative ICA rupture ranges from 6% to 8%. It varies among institutions and depends on the size and location of the aneurysm. Causes of ICA rupture and rebleeding during surgery, in decreasing order of frequency, are dissection, brain retraction, hematoma evacuation, and opening of the dural and arachnoid membranes.

Recognition

Signs of rebleeding with reruptured ICAs are largely due to intracerebral hemorrhage. The risk of such bleeding is higher with subsequent episodes of ICA rupture. This is because adhesions from the prior SAH seal off the aneurysm from the subarachnoid space and deflect any new bleeding into the brain parenchyma. After ICA rebleeds, the level of consciousness deteriorates, and patients develop focal neurologic deficits (aphasia, hemiplegia), abnormal vital signs (hypertension, bradycardia, arrhythmias, irregular respirations), and temperature elevation. They also have fluid and electrolyte imbalance (especially hyponatremia), and retinal hemorrhage may be evident on ophthalmologic examination (Table 178-2).

If ICA rebleeding occurs during or immediately after the induction of anesthesia, the patient's blood pressure will increase, and the heart rate may or may not decrease. It is important to realize that the ICP will also increase. At the

Table 178–1 • Complications of Subarachnoid Hemorrhage

Early	Late
Hematoma, ↑ ICP, rebleeding, seizures, hydrocephalus	Rebleeding, hydrocephalus, vasospasm, infarction, epilepsy
Nerve palsy, hemiparesis, reduced LOC	Permanent hemiparesis, cognitive disabilities
Cardiac arrhythmias	Myocardial infarction, pneumonia, hepatic and renal dysfunction
Transient ↑ BP	Persistent ↑ BP
Impaired vision	Vitreous hemorrhage
Fluid and electrolyte imbalance	Neurologic deterioration, death

BP, blood pressure; ICP, intracranial pressure; LOC, level of consciousness.

Table 178–2 ▪ Effects of Aneurysmal Rebleeding

- Direct brain destruction
- Disturbance of CSF flow → hydrocephalus
- ↑ ICP from hematoma, intracerebral hemorrhage, intraventricular hemorrhage
- Cerebral infarction from ↓ CBF
- Fluid and electrolyte imbalance
- Cardiac arrhythmias, ↑ BP
- Respiratory impairment

BP, blood pressure; CBF, cerebral blood flow; CSF, cerebrospinal fluid; ICP, intracranial pressure.

puncture, ICA rupture is diagnosed by intracranial Doppler ultrasonography, and the efficacy of management is monitored thereafter. Intraoperative rupture of an ICA is readily apparent. Rebleeding after completion of the operation is signaled by failure to awaken from anesthesia or by further neurologic deterioration after awakening (e.g., decrease in level of consciousness, development of new focal neurologic deficits or aphasia).

Risk Assessment

ICA rerupture is one of the major causes of neurologic deterioration after initial SAH (Table 178-3). Risk of rebleeding begins immediately after the initial ICA hemorrhage and is the major threat early after SAH. The likelihood of rebleeding is directly related to the patient's systolic blood pressure in the post-SAH period. For patients who have already had multiple rebleeding episodes, the likelihood of further rupture and death is much greater. Other risk factors include poor neurologic status (owing to initial SAH parenchymal injury), shorter time since initial SAH, female gender (twice the incidence of rebleeding versus males), poor medical condition, older age, posterior ICA, higher rates of intracerebral or intraventricular hematoma, and abnormal clotting parameters. During pregnancy, the risk of rebleeding from an unsecured ICA is 33% to 50%. Although this is fatal in 50% to 68% of patients, there is no evidence that the rebleeding rate in pregnant patients is different from that in the general population.

Table 178–3 ▪ Causes of Neurologic Deterioration after Subarachnoid Hemorrhage

- Rebleeding—intracranial hypertension
- Hematoma
- Hydrocephalus
- Cerebral edema
- Seizures
- Meningitis
- Disordered autoregulation
- Disordered carbon dioxide responsiveness
- Acid-base disturbances
- Fluid and electrolyte disturbances
- Vasospasm
- Delayed ischemic deficit
- Cerebral infarction—secondary cerebral insults
  - Hypotension
  - Hypoxemia
  - Hyperglycemia
  - Intracranial hypertension (beyond initial hemorrhage)

Once the ICA has bled, the risk of rebleeding is greatest within the first 24 hours (4%); this is because clot sealing the aneurysmal rent is tenuous, and systemic blood pressure is usually at its highest. The cumulative rebleed rate for ruptured ICAs is 19% at 14 days and about 40% at 179 days. Patients whose ruptured ICAs remain untreated continue to rebleed at a rate of 3% per year for up to 15 years. Late rebleeding is fatal in 67% of cases.

The International Subarachnoid Aneurysm Trial compared operative aneurysmal clip-ligation with endovascular coiling in 2143 patients with ICA-related SAH. At 1-year follow-up, results of the randomized study showed a low risk of rebleeding in both groups (2.4% for coil versus 1.0% for clip repair). However, even after accounting for effects of rebleeding, the relative risk for death or significant disability was 22.6% lower for endovascular versus surgical repair, an absolute risk reduction of 6.9%. Most of these patients were in good condition after SAH (World Federation of Neurosurgical Societies grades I and II) and had small anterior ICAs (92% <11 mm in size). For such ICAs, endovascular and surgical repairs are considered equivalent therapies.

Implications

Pathophysiologic sequelae and complications of rebleeding after initial aneurysmal SAH are considerable. Because a recurrent hemorrhage is usually more severe than the initial one, mortality with recurrent hemorrhage doubles to 80%, with significant associated morbidity in the surviving patients. The size of the hematoma is the most critical factor in determining outcome (Table 178-4). Patients with large subdural hematomas and more of a midline shift on computed tomography scanning have a poorer prognosis, as do those with associated intracerebral or intraventricular hemorrhage.

Because the majority of rebleeding takes place within the first 6 to 24 hours after the initial SAH, early intervention to secure the aneurysm (whether by surgical clipping or endovascular coiling) has become the mainstay of treatment for rebleeding. Thus, diagnosis and treatment of rebleeding must be accomplished quickly and efficiently. Further, because increased experience with SAH, its sequelae, and its treatment improves patient care, collaborative relationships between community hospitals and centers specializing in the surgical and endovascular treatment of ICAs are mandatory.

Table 178–4 ▪ Predictors of Mortality after Acute Subarachnoid Hemorrhage

- Poor clinical status or grade on admission—directly related to size of hematoma
- Decreased level of consciousness
- Elevated blood pressure
- Rebleeding
- Delayed ischemic deficit (vasospasm)
- Thickness of subarachnoid clot on initial computed tomography scan
- Basilar aneurysm
- Older age
- Preexisting medical illness

MANAGEMENT

Therapy for rebleeding after an initial SAH is designed to maintain cerebral perfusion, reduce intracranial hypertension and volume, control systemic blood pressure, and decrease transmural pressure (mean arterial pressure minus ICP) across the aneurysm wall. Within this context, optimization of brain O_2 delivery depends on total arterial O_2 content and necessitates the maintenance of normal hemoglobin concentrations and arterial O_2 saturations.

Specific therapy varies according to the stage of ICA therapy at which rerupture occurs (Table 178-5). If the aneurysm bleeds before, during, or after the induction of anesthesia, the patient is hyperventilated with 100% O_2. Thiopental, which also affords some amount of cerebral protection, or intravenous sodium nitroprusside or nicardipine[1] will lower blood pressure, although excessive lowering of blood pressure at this juncture can be detrimental if it interferes with cerebral perfusion. Nitroprusside also causes cerebral vasodilatation, which may further raise ICP and impair cerebral perfusion, thereby increasing the ischemic penumbra.[2] Immediate craniotomy for "rescue clipping" after ICA rupture during induction has been successful.

Intraoperative rupture of an ICA mandates rapid achievement of surgical control. The mean arterial pressure may be reduced briefly to 50 mm Hg to facilitate temporary proximal and distal occlusion of the parent vessel in preparation for clip-ligation of the aneurysmal neck. Once the parent vessel is occluded, blood pressure is increased to normal to enhance collateral circulation during the period of temporary occlusion. This may be superior to the use of controlled hypotension after rupture. Alternatively, the ipsilateral carotid artery can be manually compressed for 3 minutes to produce a bloodless field. Also, if the bleeding is sufficient to cause hypovolemia, induced hypotension may not be an option. Any blood loss is replaced immediately with whole blood, blood products, colloid, or crystalloid. It is essential to maintain normal blood volume while the blood pressure is lowered.

Although barbiturates and etomidate have been advocated to protect against focal brain ischemia, the clinical efficacy is unproved. Also, with hypovolemia, the associated hypotension can be detrimental. Stable patients can receive thiopental or etomidate before temporary occlusion.

For all patients, temperature is maintained in the low-normal range (34°C to 35°C). Even moderate hypothermia confers some cerebral protection by reducing the release of excitatory neurotransmitters and the cerebral metabolic rate of O_2 consumption (by 7% to 8% per 1°C). However, results of the recent International Hypothermia for Aneurysm Surgery Trial suggest that intraoperative hypothermia (33°C) does not improve neurologic outcomes compared with maintaining normothermia (target temperature 36.5°C). Any increase in temperature above normal should be promptly reduced.

[1]The latter may be more effective for reducing associated vasospasm.
[2]Zone of ischemic brain surrounding nonviable brain tissue.

Table 178-5 • Aneurysmal Rupture: Management Priorities

During or After Induction
Hyperventilation
100% oxygen
Blood pressure control
Barbiturates
During Dissection
Induced hypotension
Proximal vascular or carotid occlusion with high normal blood pressure
100% oxygen
Pharmacologic metabolic suppression
Volume resuscitation

Patients who do not awaken as expected following the operation, or who awaken and then deteriorate neurologically, require timely diagnosis of the cause. Emergent computed tomography scans can help differentiate ICA rebleed, rupture of another ICA, postsurgical bleeding, pneumocephalus, acute hydrocephalus, and acute cerebral infarction as the cause of deterioration.

If there is intracranial hypertension postoperatively, the patient requires intracranial volume-reducing measures such as hyperventilation with 100% O_2, mannitol, cerebral vasoconstricting drugs (e.g., thiopental, propofol), and augmentation of cerebral perfusion through maintenance of systemic blood pressure in the patient's high-normal range. Emergency reoperation may be necessary for rescue clipping of the ruptured ICA, evacuation of hematoma, control of bleeding, or ventricular drainage. In an emergency, an external ventricular drain may be inserted in the postanesthesia care area or intensive care unit to decompress the ventricular system.

PREVENTION

The only definitive measure to prevent ICA rebleeding is early surgical clip-ligation or endovascular obliteration of the aneurysm. Once the ICA has been secured, the risk of rebleeding is reduced to practically zero, with late rebleeding occurring more often after endovascular than neurosurgical intervention. After securing the ICA, the patient can receive prophylaxis against or treatment for cerebral vasospasm such as hypertensive hypervolemic hemodilution ("triple H therapy"), without fear of ICA rerupture.

Short of securing the ICA by mechanical means, preoperative measures to prevent rebleeds include maintenance of blood pressure in the patient's normal range, maintenance of euvolemia (Table 178-6), and avoidance of seizures (which may be associated with hypertension). Blood pressure control is achieved with analgesics and short-acting antihypertensive drugs (e.g., labetalol) that do not affect the cerebral vasculature. Lowering blood pressure has not been shown to reduce the risk of rebleeding in any controlled trial, but prospective studies have correlated rebleeding with higher systolic blood pressures. Beyond the first few days after initial SAH, the risk of lowering the blood pressure increases

Table 178–6 ▪ Prevention of Aneurysmal Rebleeding

Preoperative
- BP control: sedatives, short-acting antihypertensive drugs
- Maintain adequate cerebral perfusion pressure (70 to 80 mm Hg)
- Analgesic drugs
- Cautious HHH therapy for vasospasm
- Early aneurysmal clip-ligation or endovascular obliteration

Intraoperative

Induction
- Maintain normal BP
- Maintain direct BP monitoring
- Avoid surges in systolic BP
- Ensure adequate depth of anesthesia
- Provide optimal oxygenation
- Maintain normocapnia

Craniotomy
- Osmotic diuretic with craniotomy
- CSF drainage after craniotomy

Aneurysmal manipulation
- Proximal temporary occlusion
  - High-normal BP
- Hypotension
- Osmotic diuretics
- CSF drainage
- Hyperventilation
- Venous drainage
- Normoglycemia
- Hypothermia

Adequate analgesia

Maintain normovolemia

Monitor central venous pressure, urine output, blood loss

Emergence

Avoid surges in systolic BP

Adequate analgesia

Postoperative

Avoid surges in systolic BP

Maintain intravascular volume

Avoid hypotension

Adequate analgesia

BP, blood pressure; CSF, cerebrospinal fluid; HHH, hypertensive hypervolemic hemodilution; ICP, intracranial pressure.

because the patient is now susceptible to vasospasm. At this point, it is best to let the patient's blood pressure self-adjust, although pain should be treated appropriately to prevent associated increases in blood pressure.

If the patient deteriorates neurologically from cerebral vasospasm before the ICA is secured, triple H therapy (see earlier) must be instituted with caution. To avoid rebleeding, the systolic pressure is increased modestly from 120 to 150 mm Hg, central venous pressure from 10 to 12 mm Hg, and pulmonary capillary wedge pressure from 12 to 16 mm Hg.

Avoidance of lumbar puncture and rapid ventricular drainage before ICA clip-ligation may also protect against rebleeding. However, these measures are sometimes used to lower ICP (as a calculated risk) when cerebral perfusion is seriously compromised by intracranial hypertension.

The antifibrinolytics ε-aminocaproic acid and tranexamic acid can reduce the likelihood of ICA rebleeding. However, associated cerebral vasospasm limits their usefulness and may double mortality rates due to delayed ischemia. Thus, there is little if any indication for the use of these drugs after SAH.

Table 178–7 ▪ Induction of Anesthesia: Aneurysmal Clip-Ligation

- Optimal head position
- Deep level of anesthesia
  - Propofol (1-2 mg/kg)
  - Thiopental (3-5 mg/kg)
  - Fentanyl (3-5 μg/kg)
  - Sufentanil (0.5-1 μg/kg)
  - Vecuronium (0.1 mg/kg)
  - Low-dose inhalation anesthetic
- Controlled ventilation
  - 100% O_2
  - Normal $Paco_2$ (35-40 mm Hg)
- Before laryngoscopy
  - Lidocaine (1.5 mg/kg)
  - Thiopental (2-3 mg/kg)
  - Propofol (0.5 mg/kg)
- Brief, gentle laryngoscopy
- Intubation

During the induction of anesthesia for craniotomy for ICA clip-ligation, it is essential to maintain transmural pressure across the ICA wall in the patient's preoperative range by the judicious use of drugs and meticulous technique (Table 178-7). Certainly, one must prevent sudden increases in systemic blood pressure and decreases in ICP. Direct blood pressure monitoring provides beat-to-beat information about the immediate effects of anesthetic or neurosurgical interventions (e.g., laryngoscopy, application of pin headholders). Anticipation of a blood pressure increase with these maneuvers can facilitate the timely use of drugs such as propofol and thiopental to deepen anesthesia.

Avoiding sudden increases in transmural pressure from a decrease in ICP before the bone flap is turned is also important. Ventilation is adjusted to maintain normocapnia (arterial carbon dioxide tension 35 to 40 mm Hg) and intracranial volume until the dura is opened. However, if the patient has a large subdural hematoma, hyperventilation and other maneuvers to improve intracranial compliance are indicated during induction. The volume-reducing effect of mannitol also may decrease ICP before the skull is opened. To avoid consequent increased transmural pressure and the potential for ICA rerupture, mannitol is not administered until after the craniotomy has been performed, when the intracranial contents are at atmospheric pressure. Lumbar drainage of CSF also facilitates ICA access by relaxing the brain, but this too increases transmural pressure by reducing ICP if it is performed before the cranium has been opened.

Interventions to prevent rebleeding are also necessary during ICA manipulation for clip-ligation. Temporary proximal occlusion of the parent vessel is used to decrease the turgor of the ICA sac, and the blood pressure is maintained in the patient's high-normal range to enhance distal and collateral perfusion. Of course, if the temporary clip is removed before the aneurysm has been secured, blood pressure must be quickly returned to the patient's low-normal range to prevent aneurysmal rupture.

Hypotension with isoflurane or nitroprusside to a mean arterial pressure of 50 mm Hg in normotensives and 60 mm Hg or higher in hypertensives was once used to increase the safety of aneurysmal manipulation. This is no longer done,

however, because hypotension to lower CBF may adversely affect patients with or in the process of developing cerebral vasospasm.

Although there are no controlled human studies of the protective effects of intravenous drugs during ICA surgery, the ability to quickly institute prophylactic protective measures before the onset of ischemia is desirable. A number of intravenous drugs, alone and in combination, have been administered to extend the safe duration of temporary vascular occlusion. High-dose mannitol (2 g/kg) enhances the microcirculation and increases regional CBF in areas of ischemia. Because the production of free radicals may contribute to neuronal damage from ischemia, vitamin E and dexamethasone are used to augment mannitol's effects in some protocols.

To the regimen of normotension, normovolemia, and mannitol, some neurosurgeons have added electroencephalographic burst suppression (with etomidate or barbiturates), with reported benefit. Propofol, if administered to provide burst suppression before temporary ICA occlusion, may also confer cerebral protection. Normoglycemia and relative hypothermia to 35°C may also reduce the ischemic risk with temporary occlusion of cerebral vessels.

Control of blood pressure is essential during emergence from anesthesia, because patients are at risk for rebleeding during this time as well. This may be due to multiple ICAs, whether diagnosed or not. If one has been clipped, another unsecured one may bleed on emergence. Hypertension with emergence also threatens surgical hemostasis and may produce intracranial hemorrhage. Finally, wrapping the ICA (versus clipping) does not necessarily protect against rebleeding during emergence from anesthesia.

Further Reading

Chang HS, Hongo K, Nakagawa H: Adverse effects of limited hypotensive anesthesia on the outcome of patients with subarachnoid hemorrhage. J Neurosurg 92:971-975, 2000.

Cross DT, Tirschwell DL, Clark MA, et al: Mortality rates after subarachnoid hemorrhage: Variations according to hospital case volume in 18 states. J Neurosurg 99:810-817, 2003.

Egge A, Waterloo K, Sjoholm H, et al: Prophylactic hyperdynamic postoperative fluid therapy after aneurysmal subarachnoid hemorrhage: A clinical, prospective, randomized, controlled study. Neuro 49:593-606, 2001.

Eng CC, Lam AM: Cerebral aneurysms: Anesthetic considera Cottrell JE, Smith DS (eds): Anesthesia and Neurosurgery, St. Louis, Mosby–Year Book, 1994, pp 376-405.

Fridriksson S, Saveland H, Jakobsson KE, et al: Intraoperative compli in aneurysm surgery: A prospective national study. J Ne 96:515-522, 2002.

Gianotta SL, Oppenheimer JH, Levy ML, et al: Management of intra tive rupture of aneurysms without hypotension. Neuro 28:531-536, 1991.

Haley EC Jr, Kassell NF, Torner JC, et al: The International Coop Study on Timing of Aneurysm Surgery: The North American e ence. Stroke 23:205-214, 1992.

Kett-White R, Hutchinson PJ, Al-Rawi PG, et al: Adverse cerebral detected after subarachnoid hemorrhage using brain oxyge microdialysis probes. Neurosurgery 50:1213-1222, 2002.

LeRoux P, Winn HR: Management of the ruptured aneurysm. In LeR Winn HR, Newell DW (eds): Management of Cerebral Aneur Philadelphia, WB Saunders, 2004, pp 303-333.

Molyneux A, Kerr R, Stratton I, et al: International Subarachnoid Ane Trial (ISAT) of neurosurgical clipping versus endovascular coil 2143 patients with ruptured intracranial aneurysms: A rando trial. Lancet 360:1267-1274, 2002.

Murayama Y, Song JK, Uda K, et al: Combined endovascular treatment both intracranial aneurysm and symptomatic vasospasm. AJNR A Neuroradiol 24:133-139, 2003.

Newfield P: Anesthetic management of intracranial aneurysms. In Newfi Cottrell JE (eds): Handbook of Neuroanesthesia, 3rd ed. Philadel JB Lippincott-Williams & Wilkins, 1999, pp 175-194.

Qureshi AI, Suri MF, Yahia AM, et al: Risk factors for subarachnoid he rhage. Neurosurgery 49:607-613, 2001.

Rabinstein AA, Pichelmann MA, Friedman JA, et al: Sympto vasospasm and outcomes following aneurysmal subarachnoid he rhage: A comparison between surgical repair and endovascular occlusion. J Neurosurg 98:319-325, 2003.

Sluzewski M, Bosch JA, van Rooij WJ, et al: Rupture of intracr aneurysms during treatment with Guglielmi detachable c Incidence, outcome, and risk factors. J Neurosurg 94:238-240, 20

Smith MJ, Le Roux PD, Elliott JP, Winn HR: Blood transfusion and incre risk of vasospasm and poor outcome after subarachnoid hemorrha J Neurosurg 101:1-7, 2004.

Solenski NJ, Haley EC Jr, Kassell NF, et al: Medical complications aneurysmal subarachnoid hemorrhage: A report of the Multice Cooperative Aneurysm Study. Crit Care Med 23:1007-1017, 1995.

Todd MM, Hindman BJ, Clarke WR, et al: Mild intraoperative hypother during surgery for intracranial aneurysm. N Engl J Med 352:135-1 2005.

Treggiari-Venzi MM, Suter PM, Romand JA: Review of medical preventi of vasospasm after subarachnoid hemorrhage: A problem of ne intensive care. Neurosurgery 48:249-261, 2001.

Intracranial Aneurysms: Vasospasm and Other Issues

Philippa Newfield

Case Synopsis

A 47-year-old woman underwent craniotomy for clip-ligation of a middle cerebral artery aneurysm. The procedure was successful, and the patient was alert and neurologically intact until postoperative day 4, when her level of consciousness decreased and she developed a new hemiparesis.

PROBLEM ANALYSIS

Definition

Vasospasm is the transient, self-limited narrowing of intradural subarachnoid arteries that occurs several days after subarachnoid hemorrhage (SAH). It is a result of sustained contraction of arterial smooth muscle. The subsequent delayed ischemic deficit and infarction caused by cerebral vasospasm are a major cause of disability and death after SAH, accounting for 30% of SAH-induced morbidity and mortality. Cerebral vasospasm is associated with a deterioration in clinical status in 30% of patients after SAH. Up to 10% of patients die, and another 10% have permanent neurologic deficits. This reactive narrowing of the subarachnoid arteries occurs after rupture of an intracranial aneurysm because these vessels are bathed by spasmogenic breakdown products of red blood cells (especially hemoglobin) released into the cerebrospinal fluid.

Recognition

Angiographic vasospasm begins 3 to 5 days after SAH. The narrowing is maximal at 6 to 8 days and gradually resolves 12 to 14 days after a single episode of SAH. Angiographically severe vasospasm is defined as a decrease of 50% or greater in arterial diameter. The diagnosis of cerebral vasospasm (Table 179-1) is based on clinical signs of progressive impairment in mental status and level of consciousness or the appearance of new focal neurologic deficits more than 3 days after the initial SAH that cannot not attributed to any other structural or metabolic cause. The onset of SAH may be sudden or insidious and is often accompanied by increased headache, meningismus, and fever. Although some evidence of vasospasm is apparent on angiography in 70% to 90% of cases, only one third of patients develop full clinical expression. It is important to rule out other causes of neurologic deterioration with suspected SAH, including rebleeding, intracerebral hemorrhage, hydrocephalus, subdural hematoma, cerebral infarction, cerebral edema, meningitis, seizures, electrolyte and acid-base disturbances, and adverse drug reactions.

Cerebral angiography is the most reliable test for diagnosing and evaluating vasospasm. On angiography, vasospasm may be focal, diffuse, or segmental. Clinical signs and symptoms of decreased cerebral blood flow (CBF) usually develop when there is greater than 50% reduction in the diameter of the arterial lumen. Angiography is indicated for patients suspected of having cerebral vasospasm who do not improve after the administration of intravenous fluids and induced hypertension. It is also used for those who cannot tolerate the aforementioned therapy to rule out vasospasm as a cause of deterioration.

Computed tomography (CT)–angiography can detect severe or no cerebral vasospasm in proximal cerebral arteries. It is less reliable for assessing cerebral vasospasm in more distal arteries and intermediate degrees of vasospasm. Methodologies for measuring CBF are positron emission tomography (PET), single photon emission computed tomography (SPECT), and xenon-enhanced CT. PET studies have revealed a fall in the cerebral metabolic rate for oxygen following SAH. Angiographic vasospasm, delayed ischemic deficits, and increased transcranial Doppler velocities are associated with regions of cerebral hypoperfusion on SPECT. Xenon-enhanced CT is a fairly inexpensive technique and can reveal and quantify reductions in regional CBF in patients with clinical vasospasm; it can also be

Table 179-1 • Diagnosis of Cerebral Vasospasm

Clinical appearance of new neurologic signs and symptoms
Decrease in level of consciousness
Focal weakness
Angiography
Positron emission tomography (PET)
Single photon emission computed tomography (SPECT)
Computed tomography (CT)–angiography
Xenon cerebral blood flow measurement
Transcranial Doppler (TCD)

repeated within 20 minutes. Further, it is possible to fuse regional CBF data with conventional CT anatomy and distinguish ischemia from other causes of neurologic deterioration after SAH.

Transcranial Doppler (TCD) ultrasonography is also used to diagnose cerebral vasospasm. Either a sharp increase (e.g., middle cerebral artery velocity >120 cm/second) or a rapid rise in TCD blood flow velocity (e.g., >50 cm/second in 24 hours) is indicative of a reduction in the caliber of the vessels. Peak TCD flow velocity of 140 to 200 cm/second is associated with moderate cerebral vasospasm; values greater than 200 cm/second indicate severe vasospasm. CBF velocities become maximal 7 to 20 days after SAH. Critical TCD blood flow velocities (>120 cm/second) correlate strongly with vasospasm on angiography. As such, TCD is a better corroborative tool than a predictive one. Either a reduction in TCD velocity or a return to normal often indicates abatement of vasospasm and can be used to determine the efficacy and duration of treatment. Because TCD is operator dependent and involves other technical factors (e.g., intracranial pressure [ICP], cardiac output, the artery being assessed), it is important to correlate any TCD results with sequential neurologic examinations and other monitoring modalities, including ICP, blood pressure, and cardiac output.

Jugular bulb venous oximetry detects changes in cerebral oxygen extraction. In one study, patients who developed clinical vasospasm were noted to have a significant rise in cerebral oxygen extraction approximately 1 day before the onset of signs of neurologic deficits. When these patients were treated with hypertensive hypervolemic hemodilution, their deficits resolved, and there was a significant improvement in cerebral oxygen extraction. There was no increase in cerebral oxygen extraction in patients who did not have clinical vasospasm; therefore, increases in this parameter may be predictive of the impending onset of clinical vasospasm.

Risk Assessment

After clip-ligation of cerebral aneurysms, and regardless of clinical status, all patients have a 50-50 chance of developing cerebral vasospasm. Vasospasm is directly related to the severity of the hemorrhage from aneurysmal rupture, which correlates well with the location and volume of blood noted on the initial posthemorrhage CT scan. The risk is increased by the presence of cerebral dysautoregulation and abnormal carbon dioxide responsiveness after SAH. Elderly patients may be at less risk for developing vasospasm, but they do not tolerate ischemia as well as younger ones do and therefore develop cerebral infarction more frequently. The timing of surgery has no effect on the subsequent development of angiographic cerebral vasospasm, nor does surgical versus endovascular occlusion have an effect. Other indicators of increased risk for the development of vasospasm include an admission Glasgow Coma Scale score less than 14 (see Table 182-1), an early increase in mean middle cerebral artery flow velocity on TCD, and anterior cerebral or internal carotid artery aneurysms.

Angiographic vasospasm (>30% reduction in cerebral vessel diameter) is a significant risk factor for the development of infarction. Death from vasospastic infarction occurs in 5% to 17% of patients after SAH. Modifiable risk factors that affect the progression from ischemia to infarction include a premorbid history of hypertension and smoking.

Transfusion of packed red blood cells intraoperatively is a risk factor for poor outcome. Also, postoperative transfusion is correlated with the development of angiographically proven cerebral vasospasm. The mechanism may involve depletion or inactivation of nitric oxide, an endogenous vasodilator that transfused red blood cells appear to lack. Transfused cells may also have proinflammatory effects or may induce immune system dysfunction. If so, before transfusion, one must determine whether SAH patients are symptomatic from any associated anemia.

Implications

Cerebral vasospasm appreciably worsens patient outcomes after SAH. It is believed to be the cause of 28% and 39% of all associated deaths and disability, respectively. Thus, it is responsible for extensive utilization of limited health care resources. Owing to the high mortality, and because survivors of SAH with vasospasm are more likely to have serious permanent neurologic deficits, considerable research efforts and dollars are being expended to identify pharmacologic and other measures to prevent, ameliorate, or eradicate the devastating sequelae of SAH-related cerebral vasospasm.

The presence of cerebral vasospasm has implications for anesthetic management as well. Cerebral perfusion pressure is maintained at higher-than-normal levels to enhance cerebral perfusion. Hypotension, including controlled hypotension during aneurysmal dissection, should be avoided. Because autoregulation and carbon dioxide responsiveness are impaired to varying degrees with cerebral vasospasm, blood pressure stability and normocapnia are maintained.

MANAGEMENT

Pharmacologic and other modalities used to treat cerebral vasospasm after SAH are listed in Table 179-2. Early operation for clip-ligation of the ruptured aneurysm after SAH secures the aneurysm and permits the removal of fresh clot by irrigation and suction. The surgeon may also apply tissue plasminogen activator (tPA) directly into the subarachnoid space to dissolve remaining clot. Although this fibrinolytic drug can reduce vasospasm, it also has the potential to cause rebleeding by dissolving normal clot. Thus, only patients at high risk for clinically significant vasospasm are candidates for tPA treatment. Early obliteration of the aneurysm by endovascular coils also facilitates the subsequent treatment of vasospasm.

Table 179-2 ▪ Pharmacologic and Other Modalities Used to Treat Cerebral Vasospasm

Hypertensive hypervolemic hemodilution
Volume expansion with crystalloids and colloids
Vasopressors (e.g., dopamine, dobutamine, phenylephrine)
Transluminal balloon angioplasty

Both hypervolemia and hypertension are used to increase cardiac output and augment cerebral perfusion in vasospastic areas of the brain with impaired autoregulation. Early institution of these measures can mitigate or avoid the progression of vasospasm-induced ischemia to infarction. Hemodilution alone is unlikely to be beneficial and may reduce cerebral oxygen delivery. However, a hematocrit of 30% to 35% is likely adequate. Complications of induced hypervolemia and hypertension include rebleeding, hemorrhagic infarct transformation, cerebral edema, hypertensive encephalopathy, intracranial hypertension, myocardial infarction, heart failure, pulmonary edema, coagulopathy, and dilutional hyponatremia, as well as complications related to central vascular catheterization.

Expansion of intravascular volume is necessary because total circulating blood and red blood cell volumes are reduced in most patients after SAH. This is secondary to supine diuresis, peripheral pooling, negative nitrogen balance, reduced erythropoiesis, iatrogenic blood loss, and increased natriuresis. Limits for crystalloid and colloid volume expansion are central venous and pulmonary capillary wedge pressures of 10 to 12 and 12 to 16 mm Hg, respectively. There is a suggestion that albumin may improve the clinical outcome at 3 months and reduce hospital costs when normal saline alone has failed to increase the central venous pressure to at least 8 mm Hg.

Vagal and diuretic responses to intravascular volume augmentation might dictate the need for a drug such as vasopressin to reduce urine output to less than 200 mL/hour. Hydrocortisone has also been used to attenuate excessive natriuresis and hyponatremia in patients with SAH, as well as to prevent the associated decrease in total blood volume. It appears to have no serious side effects.

Vasopressors, including dopamine, dobutamine, and phenylephrine, might also be required to increase blood pressure and augment cardiac output. Invasive hemodynamic monitoring (e.g., direct arterial, central venous, or pulmonary artery pressure; cardiac output) is required for patients with induced hypertension. Before the aneurysm is secured, systolic blood pressure is maintained between 120 and 150 mm Hg. Once secured, it can be increased to 160 to 180 mm Hg.

Transluminal balloon angioplasty is also used to relieve cerebral vasospasm. The inflatable intravascular balloon mechanically dilates the segmental zone of vasospastic narrowing. This may improve the patient's level of consciousness by relieving focal ischemic deficits. However, early intervention is critical. Another treatment is serial papaverine angioplasty. It improves cerebral circulation times, but serial infusions are required for recurring cerebral vasospasm.

PREVENTION

Cerebral Vasospasm

The prevention of cerebral vasospasm requires a high level of vigilance and care, maintenance of normovolemia, careful monitoring, and prevention of secondary cerebral insults and medical complications (Table 179-3). Early occlusion of the aneurysm facilitates subsequent efforts to prevent and treat vasospasm. Monitoring in an intensive care unit or a transitional area is indicated until after the peak time for the development of vasospasm has passed. The purpose of such care is to avoid hypovolemia, hyponatremia with inappropriate diuresis, arrhythmias, hyperthermia, pulmonary edema, hypoxia, hypercarbia, and intracranial hypertension. Any of these has the potential to exacerbate cerebral vasospasm.

Table 179-3 ▪ Pharmacologic and Other Modalities Used to Prevent Cerebral Vasospasm after Subarachnoid Hemorrhage

Administer nicardipine (IV)
Administer nimodipine (orally or via gastric feeding tube)
Maintain normal electrolyte balance
Provide adequate analgesia
Maintain normovolemia
Maintain normothermia
Maintain normotension

After SAH, adults need 3 to 4 L of fluid a day to maintain normovolemia. Hypotonic solutions (e.g., lactated Ringer's) are avoided. Hyponatremia is treated with either normal or hypertonic saline as necessary. However, Egge and colleagues showed that prophylactic hypertensive hypervolemic hemodilution after aneurysmal SAH neither prevents vasospasm nor improves outcomes when compared with controls treated with normovolemia. In addition, costs were higher and complications were more frequent in patients receiving hyperdynamic therapy. In the International Subarachnoid Aneurysm Trial, patients with better clinical grades (World Federation of Neurosurgical Societies grades I to III on admission) whose aneurysms were occluded with endovascular coils rather than surgical clipping were less likely to have symptomatic vasospasm. However, there was no difference in clinical outcome between the groups at the end of the follow-up period.

Although blood pressure is controlled before the aneurysm is secured, it is not treated thereafter, unless elevations are critically high. ICP is maintained in the normal range with mannitol, ventricular drainage, and mild ventilation. The goal is to keep cerebral perfusion pressure above 60 to 70 mm Hg.

Use of the dihydropyridine calcium channel blocker[1] nimodipine within 96 hours of SAH in good- and poor-grade patients has been shown to reduce the morbidity and mortality associated with aneurysmal cerebral vasospasm. It is now a standard of care after SAH. Nimodipine improves the poor outcome associated with vasospasm in all grades of patients, improves the chance of a good to fair outcome, and reduces the chance of infarction after SAH. However, the incidence of symptomatic vasospasm is not affected by nimodipine. Because it has a limited effect on the angiographic caliber of vessels, it is postulated that nimodipine

[1]Dihydropyridine calcium channel blockers are selective for vascular smooth muscle versus cardiac muscle, in contrast to non-dihydropyridines such as verapamil and diltiazem. Intravenous nicardipine, a dihydropyridine calcium channel blocker, is increasingly used for the treatment of vasospasm in aneurysmal SAH, although long-term outcomes are not yet known.

confers cerebral protection by reducing the influx of calcium in marginally ischemic neurons. Alternatively, it may increase CBF by dilating pial collateral vessels not seen on angiography. Nimodipine also reduces blood pressure; however, it does so by reducing systemic vascular resistance, not preload.

Treatment with subcutaneous low-molecular-weight heparin (enoxaparin 20 mg/day) for 3 weeks after SAH also appears to improve overall outcomes at 1 year. Apparently, this is due to a reduction in delayed ischemic deficits and cerebral infarction. Patients who received enoxaparin also had fewer intracranial bleeding events and a lower incidence of severe (i.e., shunt-dependent) hydrocephalus.

Other drugs have been investigated for the prevention of vasospasm. Tirilazad, an antioxidant and free radical scavenger, showed mixed clinical results. Nicaraven, a free radical scavenger, showed a trend toward improved survival, good outcome, and smaller infarct size at 3 months. Ebselen, an antioxidant and anti-inflammatory drug, has neuroprotective properties and appears to be effective in acute ischemic stroke. Intra-arterial fasudil, a kinase inhibitor, has been used to treat clinical vasospasm. However, there was no difference in neurologic outcome versus placebo, and patients treated with fasudil had more pneumonia and hypotensive episodes. Owing to increased endothelin (an endothelial-derived vasoconstrictor peptide) with cerebral vasospasm, an endothelin antagonist has also been investigated. Intracisternal tPA prevents vasospasm but does not improve outcome because of increased bleeding associated with its use. Finally, although antifibrinolytics reduce rebleeding, they increase delayed cerebral ischemia and therefore are rarely used.

Hydrocephalus

Chronic hydrocephalus occurs in 10% of patients after SAH. It is due to obstructed pathways for cerebrospinal fluid drainage (i.e., subarachnoid venous granulations). Development of arachnoid adhesions also prevents the reabsorption of cerebrospinal fluid. If the blockage is incomplete, the problem persists only for several weeks. Hydrocephalus that either causes intracranial hypertension or reduces CBF can adversely affect the outcome following SAH. Whether the aneurysm is occluded using surgical or endovascular techniques does not affect the subsequent risk for hydrocephalus.

Acute hydrocephalus is associated with a poor clinical grade and thickened subarachnoid or intraventricular hemorrhage on admission CT scans. It occurs in 15% to 20% of SAH patients. Other associations are alcoholism, female sex, older age, larger aneurysms, pneumonia, meningitis, and hypertension. It is recognized by the onset of lethargy and coma within 24 hours of SAH.

Development of acute ventricular dilatation soon after SAH is a cause of sudden deterioration in neurologic status and may require external ventricular drainage to normalize ICP. External ventricular drainage is used only when the patient's level of consciousness becomes depressed. Good results have been achieved when this is done along with early aneurysm occlusion. Ventricular drainage should be used with caution, however, to avoid changes in the transmural pressure that may precipitate aneurysmal rebleeding. Because acute hydrocephalus is often associated with vasospasm, early aneurysm occlusion allows the u hyperdynamic therapy and angioplasty.

Half of patients who develop acute hydrocephalus n a ventriculoperitoneal shunt, but the need for a perm shunt is reduced by external ventricular drainage. Predic the need for permanent shunting include poor gra admission, rebleeding, and intraventricular hemorrhage.

Chronic hydrocephalus, seen in 25% of patients *survive* SAH, is an important cause of the subsequent physical decline of patients who were originally in good dition. Symptoms include an increasingly impaired le consciousness and the development of dementia, gait di bances, and incontinence. A CT scan is indicated with month after SAH to ascertain ventricular size.

Abnormalities of Cerebral Autoregulation

The central nervous system is directly affected by SAH and resultant hematoma, vascular disruption, and edema. interferes with cerebral autoregulation, which is the ability the cerebral vasculature to maintain normal (uncha CBF over a wide range of cerebral perfusion pressures (m arterial pressure minus ICP), from 50 to 150 mm Importantly, this range is higher (shifts to the right) patients with chronic hypertension. Intracranial aneury (especially giant aneurysms) and SAH-induced hemat and cerebral edema can cause intracranial hypertension, a consequent decrease in the patient's level of consciousn and the potential for brainstem herniation and death. Pati with intracranial hypertension also have reduced CBF cerebral metabolic rate for oxygen. The extent of such imp ment correlates with the patient's clinical grade. The respo of the cerebral vasculature to changes in arterial carb dioxide tension is preserved after SAH. A decline in carb dioxide reactivity usually does not occur until there is ext sive disruption of cerebral homeostasis.

Seizures

The seizure incidence after SAH is from 3% to 26%. E seizures occur in 1.5% to 5% of patients, and late ones in 3 Seizures are detrimental after SAH because they increase C and cerebral metabolic rate for oxygen and also may ca rebleeding, owing to increased blood pressure. Patients highest risk for seizures have either thick cisternal blood on scan or lobar intracerebral hemorrhage. Other risk factors rebleeding, vasospasm with delayed ischemic neurolog deficits, middle cerebral artery aneurysm location, subdu hematoma, and chronic central nervous system impairment

Use of prophylactic antiepileptics is controversi because most seizures occur within the first 24 hours af SAH, often before hospitalization. Therefore, neurosurgeo use seizure prophylaxis (e.g., phenytoin, fosphenytoin, le tiracetam) for only 1 to 2 weeks after SAH. Patients with o or more intracerebral hemorrhages or early seizures rec anticonvulsants for at least 6 months.

Cardiac Disturbances

Electrocardiographic changes occur in 27% to 100% patients with SAH. Most common are T-wave inversion or

segment depression. Others are new U or Q waves and Q-T interval prolongation. Rhythm disturbances occur in 30% to 80% of patients and include premature ventricular beats (most common), sinus bradycardia and tachycardia, lower escape rhythms, atrial fibrillation, and tachyarrhythmias (atrial or ventricular in origin). Arrhythmias commonly occur within 7 days of SAH, with the peak occurrence between the second and third days.

The extent of myocardial dysfunction correlates with the severity of neurologic injury after SAH. The cause of this dysfunction is believed to be related to hypothalamic injury, with consequent autonomic imbalance and release of catecholamines, causing myocardial ischemia and infarction. Increased adrenergic tone may persist for the first week after SAH. These SAH-related cardiac abnormalities are similar to those seen with acute coronary syndromes (myocardial ischemia, infarction, and reperfusion injury) and may predispose patients to life-threatening arrhythmias. Associated Q-T interval prolongation makes patients more vulnerable to ventricular tachyarrhythmias (see Chapter 81). This risk is increased with low serum potassium or magnesium levels and with drugs that prolong the Q-T interval. The routine measurement of Q-T intervals may identify patients at risk for potentially lethal arrhythmias.

Often, the question for the neurosurgeon and anesthesiologist is whether to proceed with surgical or endovascular intervention to secure an aneurysm emergently, even if a delay might put the patient at increased risk for rebleeding and compromise the treatment for vasospasm. Serial cardiac isozymes and ventricular function assessment by echocardiography may indicate the magnitude of ischemia. Use of a pulmonary artery catheter to measure pulmonary capillary wedge pressure and cardiac output can both facilitate the management of cardiac dysfunction and monitor the response to hyperdynamic therapy for the treatment of cerebral vasospasm. The presence of severe arrhythmias (about 5% of patients with arrhythmias) or significant cardiogenic pulmonary edema may necessitate postponing surgical or endovascular intervention until treatment has begun. Prophylactic β-adrenergic blockade can improve the cardiac outcome in some patients.

Further Reading

Chang HS, Hongo K, Nakagawa H: Adverse effects of limited hypotensive anesthesia on the outcome of patients with subarachnoid hemorrhage. J Neurosurg 92:971-975, 2000.

Cross DT, Tirschwell DL, Clark MA, et al: Mortality rates after subarachnoid hemorrhage: Variations according to hospital case volume in 18 states. J Neurosurg 99:810-817, 2003.

Egge A, Waterloo K, Sjoholm H, et al: Prophylactic hyperdynamic postoperative fluid therapy after aneurysmal subarachnoid hemorrhage: A clinical, prospective, randomized, controlled study. Neurosurgery 49:593-606, 2001.

Eng CC, Lam AM: Cerebral aneurysms: Anesthetic considerations. In Cottrell JE, Smith DS (eds): Anesthesia and Neurosurgery, 3rd ed. St. Louis, Mosby-Year Book, 1994, pp 376-405.

Fridriksson S, Saveland H, Jakobsson KE, et al: Intraoperative complications in aneurysm surgery: A prospective national study. J Neurosurg 96:515-522, 2002.

Gianotta SL, Oppenheimer JH, Levy ML, et al: Management of intraoperative rupture of aneurysms without hypotension. Neurosurgery 28:531-536, 1991.

Haley EC Jr, Kassell NF, Torner JC, et al: The International Cooperative Study on Timing of Aneurysm Surgery: The North American experience. Stroke 23:205-214, 1992.

Kett-White R, Hutchinson PJ, Al-Rawi PG, et al: Adverse cerebral events detected after subarachnoid hemorrhage using brain oxygen and microdialysis probes. Neurosurgery 50:1213-1222, 2002.

Le Roux P, Winn HR: Management of the ruptured aneurysm. In Le Roux P, Winn HR, Newell DW (eds): Management of Cerebral Aneurysms. Philadelphia, Elsevier Science, 2004, pp 303-333.

Molyneux A, Kerr R, Stratton I, et al: International Subarachnoid Aneurysm Trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: A randomized trial. Lancet 360:1267-1274, 2002.

Murayama Y, Song JK, Uda K, et al: Combined endovascular treatment for both intracranial aneurysm and symptomatic vasospasm. AJNR Am J Neuroradiol 24:133-139, 2003.

Newfield P: Anesthetic management of intracranial aneurysms. In Newfield P, Cottrell JE (eds): Handbook of Neuroanesthesia, 3rd ed. Philadelphia, JB Lippincott-Williams & Wilkins, 1999, pp 175-194.

Qureshi AI, Suri MF, Yahia AM, et al: Risk factors for subarachnoid hemorrhage. Neurosurgery 49:607-613, 2001.

Rabinstein AA, Pichelmann MA, Friedman JA, et al: Symptomatic vasospasm and outcomes following aneurysmal subarachnoid hemorrhage: A comparison between surgical repair and endovascular coil occlusion. J Neurosurg 98:319-325, 2003.

Sluzewski M, Bosch JA, van Rooij WJ, et al: Rupture of intracranial aneurysms during treatment with Guglielmi detachable coils: Incidence, outcome, and risk factors. J Neurosurg 94:238-240, 2001.

Smith JS, Le Roux PD, Elliott JP, et al: Blood transfusion and increased risk of vasospasm and poor outcome after subarachnoid hemorrhage. J Neurosurg 101:1-7, 2004.

Solenski NJ, Haley EC Jr, Kassell NF, et al: Medical complications of aneurysmal subarachnoid hemorrhage: A report of the Multicenter Cooperative Aneurysm Study. Crit Care Med 23:1007-1017, 1995.

Treggiari-Venzi MM, Suter PM, Romand JA: Review of medical prevention of vasospasm after subarachnoid hemorrhage: A problem of neurointensive care. Neurosurgery 48:249-261, 2001.

Arteriovenous Malformation: Normal Perfusion Pressure Breakthrough

180

Shailendra Joshi and William L. Young

Case Synopsis

A 39-year-old woman is given general anesthesia for resection of a right superior temporal gyrus arteriovenous malformation (AVM) measuring 3 by 3 by 2 cm (Figs. 180-1 and 180-2). After surgery, her mean arterial pressure increases to 100 mm Hg when phenylephrine is used to confirm surgical homeostasis (Fig. 180-3). The patient emerges from anesthesia without neurologic deficits. Six hours later, she complains of a severe headache, vomits, and becomes lethargic. The right pupil is dilated. Immediate computed tomography scan reveals a large hemorrhage into the operative site and a midline brain shift (Fig. 180-4). After surgery to evacuate the clot, there is no residual AVM, the feeding artery is thrombosed, the surrounding brain is lax, and a vessel on the anterior rim of the AVM bed is identified as the source of bleeding. Postoperative neurologic examination reveals an appropriate response to painful stimuli and recovery of pupillary reaction. Four hours later, the patient's intracranial pressure suddenly increases from 10 to 80 mm Hg and her pupils become fixed and dilated. Immediate repeat exploration reveals the source of hemorrhage to be an arterial vessel on the posterior rim of the AVM bed. The brain is edematous and adheres to the dura. The postoperative neurologic evaluation shows no improvement. Subsequent examination shows no evidence of brainstem function, and serial electroencephalograms are isoelectric. The patient dies. At autopsy, there is no residual AVM.

PROBLEM ANALYSIS

Definition

Normal perfusion pressure breakthrough (NPPB) after AVM resection is a catch-all term that describes unexplained intraoperative brain swelling or diffuse bleeding from the AVM bed or unexplained postoperative brain swelling or intracranial hemorrhage (ICH). NPPB is a diagnosis of exclusion. Although much has been written about NPPB, the lack of a rigorous definition makes interpretation of the existing literature difficult.

The proposed pathophysiology of NPPB is as follows: High blood flow through the arteriovenous fistula creates a region of chronic cerebral hypotension in the neighboring vascular territories. Chronic cerebral hypotension may lead to a state of near-maximal vasodilatation and vasoparalysis that impairs the vessels' ability to constrict or even dilate effectively. Excision of the low-resistance AVM shunt restores perfusion in the formerly hypotensive regions of brain. However, owing to the inability of these beds to effectively vasoconstrict, normalization of cerebral perfusion pressure results in cerebral hyperemia ("luxury perfusion"), with the potential for cerebral edema formation and ICH. Although this is an attractive hypothesis, the pathophysiology has not been proved. Abnormal vascular reactivity, such as an impaired vasodilator response to acetazolamide, has been observed in regions adjacent to cerebral AVMs that show marked hyperperfusion after resection. Possibly, NPPB shares certain similarities to cerebral hyperemia after carotid endarterectomy or transluminal angioplasty and stenting of extracranial cervical arteries.

Some observations argue against a "hydraulic hypothesis" to explain the pathophysiology of NPPB. First, hypotensive vascular beds in proximity to the AVM retain the ability to vasoconstrict. Also, pressure autoregulation can be shown in these hypotensive beds, although the cerebral autoregulation curve is shifted to the left. Second, severe cerebral hypotension (feeding artery pressure <50% of systemic blood pressure) in normal, functional vascular beds is often seen in proximity to an AVM (approximately half of cases), although NPPB is a rare complication of AVM surgery. Third, NPPB hyperemia is not limited to hypotensive areas near the AVM; it appears to be global.

Alternative mechanisms for unexplained hemorrhage or swelling have been suggested, such as (1) unrecognized

Figure 180-1 Lateral arteriogram showing a moderate-sized arteriovenous malformation (AVM), with a large arterial supply (*closed arrows*) and abundant venous drainage (*open arrows*). These are indicative of very large, high-flow AVM shunts. (From Young WL, Prohovnik I, Ornstein E, et al: The effect of arteriovenous malformation resection on cerebrovascular reactivity to carbon dioxide. Neurosurgery 27:257–267, 1990.)

Figure 180-3 Operative photograph after surgical resection of the arteriovenous malformation (AVM). This depicts a dry surgical bed and surrounding dilated arteries (*arrows*). These became enlarged after interruption of the AVM. (From Young WL, Prohovnik I, Ornstein E, et al: The effect of arteriovenous malformation resection on cerebrovascular reactivity to carbon dioxide. Neurosurgery 27:257–267, 1990.)

technical complications at the time of surgery; (2) vascular disturbances due to abnormal autonomic activity, resulting in the release of vasoactive peptides from innervated cerebral vessels; (3) hemorrhage from a structurally deficient capillary vessel bed adjacent to the AVM, perhaps secondary to overexpression of angiogenic factors such as vascular endothelial growth factor or angiopoietin-2; and (4) venous occlusion after resection of the AVM. With regard to the third hypothesis, Sato and colleagues recently described markedly dilated capillary networks in the perinidal AVM region. Vessel diameters were 10 to 25 times those of normal capillaries and vascular connections to the nidus, including feeding arteries and arterioles, drainage veins and venules, and the normal capillary network. With regard to the fourth hypothesis, severe global hyperemia (i.e., increased cerebral blood flow) that occurs immediately after AVM resection appears to be associated with NPPB later in the postoperative course. In the case synopsis, cerebral blood flow significantly increased immediately after AVM resection, although ICH or cerebral edema and ICH did not occur until several hours later.

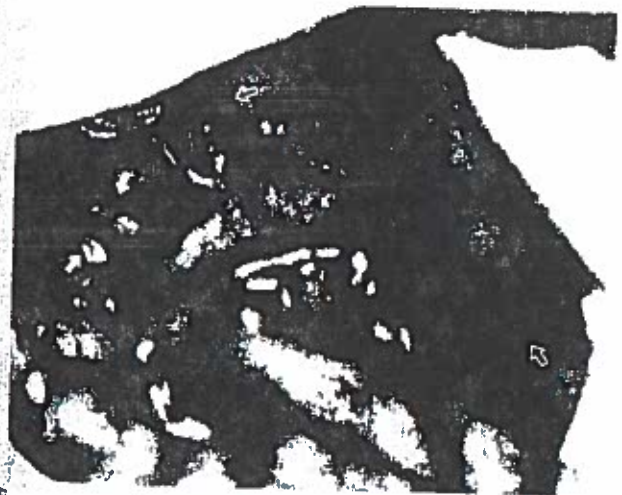


Figure 180-2 Operative photograph showing the surface of the temporal lobe, with prominent arterial supply (*closed arrows*) and venous drainage (*open arrows*). (From Young WL, Prohovnik I, Ornstein E, et al: The effect of arteriovenous malformation resection on cerebrovascular reactivity to carbon dioxide. Neurosurgery 27:257–267, 1990.)

Figure 180-4 Computed tomography scan taken 6 hours postoperatively showing massive hemorrhage into the arteriovenous malformation (AVM) bed. The hemorrhage was under tension, with a mass shift of intracranial structures. It was evacuated, but the patient died after further hemorrhages. (From Young WL, Prohovnik I, Ornstein E, et al: The effect of arteriovenous malformation resection on cerebrovascular reactivity to carbon dioxide. Neurosurgery 27:257–267, 1990.)

Recognition

NPPB is a controversial entity, and the diagnosis carries a certain degree of subjectivity. The incidence of NPPB after AVM resection is about 2.5%. NPPB is a diagnosis of exclusion, after more common causes of cerebral edema or hemorrhage have been ruled out. Causes of cerebral edema after AVM resection include hypoxia, increased venous pressure, decreased serum osmolality, systemic hypertension, and surgical trauma. After AVM resection, ICH may be due to the presence of residual AVM, poor control of systemic blood pressure, or uncorrected coagulopathy.

Risk Assessment

Predictors of NPPB after AVM ablation remain controversial. Some have proposed that large (≥4 cm) AVMs with high blood flow through the shunt and evidence of decreased perfusion in the neighboring regions may predict an increased likelihood for NPPB. Intraoperative monitoring of cerebral blood flow with laser Doppler or near-infrared spectroscopy may also reveal patients at risk of developing postoperative hyperemia. A sudden increase in laser Doppler blood flow in cortical regions adjacent to the AVM, after temporary clipping of the feeding arteries, is often seen in patients at risk for developing NPPB. Intraoperative near-infrared spectroscopy permits measurements of tissue oxygen saturation and blood volume. An increase in pre- to post-resection oxygen saturation and a blood volume ratio greater than 2 might indicate an increased risk for NPPB. Postoperative blood flow mapping by positron emission tomography (PET) or single photon emission computed tomography (SPECT) may help predict NPPB. There is no evidence that the choice of anesthetic agent influences the development of NPPB.

Implications

Although NPPB represents a class of complications without a clearly defined cause, it has been suggested that staged surgical resection or endovascular embolization could reduce the likelihood of NPPB. Staged resection or embolization permits vessels to adapt to increased perfusion pressure by gradually normalizing cerebral perfusion pressure (or it may permit adaptation to as yet unidentified pathophysiologic changes). Changes in cerebral blood flow after ablation of the AVM, however, may not be related to the preintervention feeding artery pressure. Despite the lack of precise pathophysiologic information, preoperative endovascular embolization may serve other useful purposes, such as facilitating surgery by minimizing intraoperative blood loss or by defining the location and extent of the AVM. Embolization may also reduce the size of the AVM, making it more amenable to surgery or radiosurgery. It might also alleviate neurologic symptoms by decreasing the AVM mass effect and reducing tissue perfusion in adjacent areas.

Recent evidence suggests that another technique used for AVM removal might affect the incidence of subsequent rebleeding, at least for small (< 3 cm) AVMs located in critical or eloquent areas of the brain (e.g., sensorimotor, language, or visual cortex; hypothalamus or thalamus; interna sule; brainstem; cerebellar peduncles; deep ce nuclei), where rebleeding often results in disablin logic defects. Stereotactic (gamma knife) radiosu often used to remove such small AVMs and provid ographic evidence of AVM "cure" (obliteration) in 95% of patients after a latency period of 3 to 5 years was how bleeding during the latency period would with bleeding in patients with similar but untreate It was found that the risk of hemorrhage from sma was significantly reduced after radiosurgery (bu angiographic obliteration) and was even lower afte graphic obliteration. Whether radiosurgery for large would reduce the incidence of rebleeding compar surgical resection is unknown, but it might be tested cal AVM resection is recommended for less strat located, larger AVMs amenable to surgery, but some p choose radiosurgery instead because it seems less inv

MANAGEMENT

Unexplained cerebral edema or ICH after AVM resec managed using standard cerebral resuscitative guid Treatment of cerebral edema requires careful mana of fluid and electrolyte imbalances, judicious use of o and loop diuretics, and attention to cerebral perfusio sure. Severe symptomatic swelling may necessitat trolled ventilation and, rarely, barbiturate coma. If N suspected, blood pressure is empirically maintained 10% of the baseline. Cerebral outflow pressure (i.e., venous or intracranial pressure) must be maintai levels consistent with adequate cerebral perfusion an diac preload. If deliberate systemic hypotension i assessment should include whether it is necessary to tain collateral perfusion in any cerebral territories tha have their primary feeding supplies interrupted during resection. Surgical intervention may be required for re of intracranial blood clot or for institution of intrac pressure monitoring.

PREVENTION

In the absence of a clearly defined explanation of NP empirical strategy is to prevent cerebral edema and rhage after AVM resection by careful control of blood pressure to avoid hypertension. The use of int ative embolization of the AVM nidus via the ligated arteries while the patient is under general anesth been noted to prevent NPPB in high-risk cases. M temic hypertension is sometimes used to test surgical ostasis before dural closure. Once this has been a however, the systemic blood pressure must be maint close to the patient's baseline blood pressure as feasi

After resection of an AVM, strict maintenance motension may serve two purposes. First, prevent blood pressure increases may be important for the p tion of postoperative hematoma. This could be cau

rupture of cauterized stumps of dysplastic feeding vessels to the AVM or an unidentified residual nidus. Second, avoidance of hypertension prevents the cerebral hyperemia and edema that result from exceeding the upper limit of the flow-pressure autoregulation curve. This can be explained as follows: Functionally normal but chronically hypotensive cerebral beds in proximity to the AVM show a leftward shift in the cerebral autoregulatory curve. It is generally believed that in intact human cerebral circulation, cerebral hyperemia and edema occur whenever cerebral perfusion pressure increases beyond the upper limit of autoregulation. If the cerebral autoregulation curve shifts to the left, it may be that the upper limit of pressure autoregulation also shifts to a lower pressure. Ablation of the AVM shunt increases the regional perfusion pressure in the hypotensive areas, even at normal systemic arterial pressures. The magnitude of increase in the regional perfusion pressure is difficult to predict. In the absence of means to monitor regional cerebral perfusion in the perioperative period, it is reasonable to maintain strict normotension. In selected high-risk cases, mild systemic hypotension might minimize the chances of post-resection hyperemia and edema. Decreasing systemic perfusion pressure, however, might jeopardize brain regions that depend on collateral pathways for the maintenance of perfusion. Therefore, induced systemic hypotension to any significant degree should be considered carefully within the context of the patient's overall circulatory status.

Further Reading

Brown AP, Spetzler RF: Intracranial arteriovenous malformations. In Batjer HH, Caplan L, Friberg L, et al (eds): Cerebrovascular Hemodynamics, Cerebrovascular Disease. Philadelphia, Lippincott-Raven, 1997, pp 833-842.

Hashimoto T, Lam T, Boudreau NJ, et al: Abnormal balance in the angiopoietin-2 system in human brain arteriovenous malformations. Circ Res 89:111-113 2001.

Joshi S, Ornstein E, Young WL: Cerebral and spinal cord blood flow. In Cottrell JE, Smith DS (eds): Anesthesia and Neurosurgery, 4th ed. St. Louis, Mosby, 2001, pp 19-68.

Kader A, Young WL: The effects of intracranial arteriovenous malformations on cerebral hemodynamics. Neurosurg Clin North Am 7:767-781, 1996.

Kader A, Young WL: Arteriovenous malformations: Considerations for perioperative critical care monitoring. In Batjer HH, Caplan LR, Friberg L, et al (eds): Cerebrovascular Disease. Philadelphia, Lippincott-Raven, 1997, pp 857-869.

Ko N, Achrol A, Gupta D, et al: Cerebral blood flow changes after endovascular treatment of cerebrovascular stenoses. AJNR Am J Neuroradiol 26:538-542, 2005.

Sato S, Kodama N, Sasaki T, et al: Perinidal dilated capillary networks in cerebral arteriovenous malformations. Neurosurgery 54:163-170, 2004.

Young WL, Ornstein E, Baker KZ, et al: The Columbia University AVM Project: Cerebral hyperemia after AVM resection is related to "breakthrough" complications but not to feeding artery pressure. Anesth Analg 80:S573, 1995.

Young WL, Ornstein E, Baker KZ, et al: Neuroanesthesia considerations for surgical and endovascular therapy of arteriovenous malformations. In Batjer HH, Caplan LR, Friberg L, et al (eds): Cerebrovascular Disease. Philadelphia, Lippincott-Raven, 1997, pp 843-855.

Young WL, Prohovnik I, Ornstein E, et al: The effect of arteriovenous malformation resection on cerebrovascular reactivity to carbon dioxide. Neurosurgery 27:257-267, 1990.

Pediatric Neurosurgery 181

Lynda Wells

Case Synopsis

A previously healthy 14-month-old child is admitted to the emergency department following a motor vehicle accident in which he sustained a closed head injury associated with loss of consciousness and a large scalp laceration. A grand mal–type seizure occurs on arrival at the hospital. Physical examination reveals a lethargic, tachypneic, hypotensive, and tachycardic child. His pupils are equal and reactive, and there is no evidence of papilledema. Computed tomography scan of the head reveals diffuse cerebral swelling and subdural hematoma. He undergoes anesthesia for a craniotomy to evacuate the subdural hematoma, repair the scalp laceration, and place an intracranial pressure (ICP) monitor.

PROBLEM ANALYSIS

Definition

Surgical procedures in children with central nervous system (CNS) pathology are performed to correct pathologic entities (e.g., evacuation of hematoma, excision of tumors or seizure foci, closure of meningomyelocele) and to facilitate monitoring (e.g., ICP monitoring). Brain tumors are the most common solid tumors in children and are the second most common malignancy after the leukemias. Trauma is the leading cause of death in children older than 1 year, and traumatic brain injury (TBI) is the major cause of morbidity and mortality. Outcome is determined by the extent of primary and secondary brain injury. The former is the biomechanical injury that occurs with trauma; it is irreversible. Management must focus on preventing the sequelae of primary brain injury, termed secondary brain injury (Table 181-1); these management goals include reducing cerebral edema, preventing cerebral hypoxia, maintaining cerebral perfusion, avoiding increases in the cerebral metabolic rate for oxygen, and avoiding damage to neuronal membranes. Similarly, prevention of secondary brain injury is the focus of treatment for nontraumatic CNS lesions.

Recognition

The most reliable signs of TBI severity are degree of change in level of consciousness and impaired CNS function. The Glasgow Coma Scale score (see Chapter 182 and Table 182-1) adapted for pediatric patients provides a tool to assess the severity of primary and secondary brain injury and trends. The major cause of secondary brain injury involves failure of perfusion, leading to tissue hypoxia and brain edema. Associated brain swelling impairs tissue perfusion, leading to further CNS functional deterioration. The failure of cerebral oxygenation arises from hypoxemia, hypotension, hypovolemia, hyperemia, and acidosis.

When the pathologic process evolves slowly (e.g., expansion of solid tumors), physiologic compensation may occur. However, in the event of TBI, cerebral edema evolves quickly, and any compensatory mechanisms are easily overcome. The intracranial contents in children are less compliant than in adults. Thus, comparable increases in ICP are more likely to produce ischemia and herniation in children than in adults. Although hyperemia and increased cerebral blood flow in response to TBI were once considered common in children, recent data suggest that hyperemia may not be so common. Open fontanelles do not automatically exclude brain injury from increased ICP.

Table 181-1 ▪ Prevention of Secondary Brain Injury

Maneuver	Expected Effect
30-degree head-up tilt (waist up)	Increases cerebral venous drainage while maintaining CPP
Mechanical ventilation	Maintains normocapnia to slight hypocapnia to prevent cerebral vasodilatation and ↑ ICP
Systemic steroids	Improves outcome with spinal cord injury; reduces vasogenic cerebral edema with tumors; stabilizes neuronal membrane; may act as free radical scavengers
Muscle paralysis	Avoids coughing, straining, or other movement that might increase ICP
Ventricular drainage	Reduces ICP
Antihypertensive drugs*	Prevents further cerebral edema or hemorrhage leading to further ischemia, especially when due to cerebral vasospasm
Anticonvulsants	Prevents seizures and associated increase in ICP and $CMRO_2$
Mild hypothermia	Reduces $CMRO_2$ and cerebral glucose consumption
Barbiturate coma	Reduces CBF and $CMRO_2$ and may have membrane-stabilizing effect

*For example, dihydropyridine calcium channel blockers
$CMRO_2$, cerebral metabolic rate for oxygen; CBF, cerebral blood flow; CPP, cerebral perfusion pressure; ICP, intracranial pressure

Table 181–2 ▪ Neurophysiologic Effects of Commonly Used Anesthetic Agents

Agent	MAP	CBF	CPP	ICP	$CMRO_2$	CSF (Synthesis)	CSF (Absorption)	SEP (Amplitude)	SEP (Latency)
Nitrous oxide	0 or ↓	↑ or ↑↑	↓	↑ or ↑↑	↓ or ↑	↑ or ↓	↑ or ↓	↓	↑ or 0
Halothane	↓↓	↑↑↑	↑↑	↑↑	↓↓	↑ or ↓	0 or ↓	↓	↑
Enflurane	↓↓	↑↑	↑↑	↑↑	↓↓	↑	↓	↓	↑
Isoflurane	↓↓	↑	↑↑	↑	↓↓↓	↓ or ↑	↑	↓	↑
Sevoflurane	↓↓	↑	↑	↑	↓↓↓	?	?	↓	↑
Desflurane	↓↓	↑	↑	↑	↓	↓	↓	↓	↑
Thiopental	↓↓	↓↓↓	↑↑↑	↓↓↓	↓↓↓	↑ or ↓	↑	↓	↑
Propofol	↓↓↓	↓↓↓	↑↑	↓↓	↓↓↓	?	?	↑	↑
Etomidate	0 or ↓	↓↓↓	↑↑	↓↓↓	↓↓↓	↑ or ↓	↑	↑	↑
Ketamine	↑↑	↑↑↑	↓	↑↑↑	↑	↑ or ↓	↓	↑	0
Benzodiazepines	0 or ↓	↓↓	↑	0 or ↓	↓↓	↑ or ↓	↑	↑	0 or ↑
Opiates	0 or ↓	↓	↑ or ↓	0 or ↓	↓	↑ or ↓	↑	↓	↑
Droperidol	↓↓	↓	↑	↓	0 or ↓	↑ or ↓	?	?	?

CBF, cerebral blood flow; $CMRO_2$, cerebral metabolic rate for oxygen; CPP, cerebral perfusion pressure; CSF, cerebrospinal fluid; ICP, intracranial pressure; MAP, mean arterial pressure; SEP, somatosensory evoked potential.

The presence of cervical spine trauma should always be assumed in children with TBI. Infants and young children are more likely to experience cervical spine trauma than are older children, owing to their large heads and relatively weak necks. Ligamentous injury is common in this age group. In contrast, bony injury is extremely rare. Therefore, unless there is radiologic evidence of odontoid displacement or spinal cord edema, cervical spine injury is diagnosed based solely on the clinical examination. Because this is often not possible when TBI presents, cervical spine trauma is presumed to exist. Also, neurologic signs from spinal cord injury may be absent initially.

Risk Assessment

Risk assessment relates to the likelihood of death or permanent CNS functional impairment. TBI that involves or is immediately adjacent to vital structure is more likely to be compounded by the need for surgical intervention; thus, it is associated with higher morbidity. Evidence of primary cortical brain injury (e.g., intracranial hematoma, seizures) and the presence of risk factors for secondary brain injury (e.g., hypovolemia, impaired ventilation) indicate more severe TBI as well as increased morbidity and mortality.

Classic signs of intracranial hypertension seen in adults (e.g., papilledema, pupillary dilatation, cranial nerve palsies, headache on awakening, vomiting) may be absent in children, even when ICP approaches fatal levels. The presence of intracranial hematomas with acute TBI indicates a significant force of impact. Seizures after TBI also indicate significant parenchymal injury. Spinal cord injury is assumed to be present in all cases of head trauma, at least until a definitive diagnosis can be made.

Implications

The danger of intracranial pathology is that expansion in an enclosed space leads to brain compression, causing ischemia, swelling, and loss of function that can be permanent and possibly fatal. Seizures greatly increase the cerebral metabolic rate for oxygen. They are also associated with regional ischemia that can lead to cell death and loss of cognitive and functional abilities. Compromised integrity of the membranes covering the CNS (e.g., meningomyelocele) presents a significant risk for infection, as well as cerebrospinal fluid loss and hypothermia.

Many children who present for surgical removal of tumors are malnourished and debilitated due to nausea, vomiting, and neurologic dysfunction with increased ICP. Acid-base, electrolyte, and endocrine abnormalities may be present. Patients with paralysis of an extremity of greater than 24 hours' duration are at risk for an exaggerated hyperkalemic response to succinylcholine. Obtunded patients are at increased risk for aspiration, airway obstruction, and hypoventilation.

Anesthetic management can influence the outcome and long-term prognosis in pediatric neurosurgical patients (Table 181-2). Therefore, conducting a thorough preoperative assessment, with indicated laboratory and radiographic studies; maintaining a stable intraoperative course (e.g., preserving cerebral perfusion while preventing increased ICP); and providing this same level of care throughout the postoperative period are critical.

MANAGEMENT

In TBI, immediate attention is directed to establishing the airway, ventilation, and circulation. Supplemental oxygen, a secure airway, and intravenous (IV) cannulation are required. A Glasgow Coma Scale score of 9 or less is an indication for tracheal intubation, because the patient will be unable to protect his or her airway. A history is taken and a comprehensive physical examination is performed as soon as possible to evaluate medical comorbidities and the extent and severity of other physical injuries. Spinal cord injury precautions are taken. Any obvious bleeding should be controlled. Blood should be sent for complete blood count, coagulation studies, clinical chemistry, and type and crossmatch. Radiographic investigation includes computed tomography scans of the

head, neck, and chest. Other investigations are performed based on the history and clinical findings.

Anesthetic management is geared toward preventing further increases in ICP and maintaining cerebral perfusion pressure. Anxiolytic premedication is often unnecessary in neurologically compromised children. If the child is crying and agitated, however, small doses of IV midazolam or rectal barbiturates may be given, provided airway patency and adequacy of ventilation are ensured.

After preoxygenation, anesthesia is usually induced with an IV induction agent (e.g., sodium thiopental). Ketamine and methohexital are generally contraindicated; the former increases ICP, and the latter lowers the seizure threshold. Rapid-sequence induction is indicated in patients who have not fasted or in whom there is an aspiration risk. If inhalation induction is desired, moderate hyperventilation is used to counter any vasodilatory effects of volatile anesthetics on the cerebral vasculature. Once effective mask ventilation is established, generous doses of opiates are given to obtund the sympathetic response to laryngoscopy and tracheal intubation. IV lidocaine also blunts the stimulus of laryngoscopy and tracheal intubation.

Muscle relaxation with succinylcholine and atropine is used to facilitate endotracheal intubation. If succinylcholine is contraindicated, a large dose of a nondepolarizing drug (e.g., rocuronium) is used. The airway should be secured as efficiently as possible to ensure optimal ventilation and to avoid hypoxia and hypercarbia. The necessary equipment to deal with a difficult airway should be on hand in the event of unanticipated difficult intubation. If there is doubt about the ability to secure the airway in a timely fashion, tracheostomy should be considered. In-line neck traction with direct laryngoscopy and fiberoptic-guided intubation are equally effective at minimizing cervical spine injury associated with intubation. The former is the more usual approach in small children, but practitioners should use the technique with which they are most facile. Moderate hyperventilation (arterial carbon dioxide tension 30 to 35 mm Hg) is indicated to prevent cerebrovascular vasodilatation and the subsequent increase in cerebral blood flow and edema formation. Hyperoxia is unnecessary, and hypoxia must be avoided.

Anesthesia is maintained with opioids and IV infusions of barbiturates or propofol, or with volatile anesthetic agents. Nitrous oxide is contraindicated in the presence of pneumocephalus, which can be present up to 3 weeks after previous craniotomy. Muscle relaxation is maintained to facilitate mechanical ventilation, prevent involuntary patient movement (e.g., coughing, bucking), and avoid increases in ICP. The drugs used for anesthetic induction and maintenance are chosen based on their effects on cerebral perfusion pressure and the patient's overall condition (see Table 181-2).

Hemodynamic stability is maintained using blood, crystalloid infusions, and vasopressors, as required. Osmotic pressure gradients are more important in avoiding cerebral edema than are oncotic pressure gradients. Thus, crystalloid rather than colloid infusions are the mainstay of fluid therapy. Hypertonic solutions (e.g., 3% saline) are reserved for refractory increased ICP. They are not advised in the perioperative period. Fluid maintenance is usually with 0.9% saline or balanced salt solutions with a physiologic osmolality (285 to 290 mOsm/L). Because 0.9% sali slightly hypertonic (306 mOsm/L), it can be given w relatively hypotonic salt solution if large volumes of are required. However, infused volumes are limited t replacement of deficits and surgical losses, and the maintained to avoid the exacerbation of coexisting cere edema. Blood should be given early in cases associated hemorrhage to prevent anemia, which can increase morbidity. Glucose-containing solutions should be only to maintain serum glucose in the normal range.

Patient monitoring includes the following: pulse oxi try, capnography, electrocardiography, invasive blood press central venous pressure, urine output, temperature, precor Doppler, and ICP monitoring if available. Cannulation of femoral vein may be preferable to use of the internal jug vein to avoid accidental neck trauma, which may aggra already increased ICP. Hyperthermia must be avoided, this increases the cerebral metabolic rate for oxygen. Norm body temperature or mild hypothermia is desira However, deep hypothermia should be avoided, because is associated with disorders of coagulation and gluco control as well as arrhythmias. Also, shivering on awaken increases the cerebral metabolic rate for oxygen and shou be avoided. If surgery involves or is proximate to the senso or motor cortex, sensory and motor evoked potentials c be measured. However, motor evoked potentials cannot monitored in denervated limbs or in the presence of neur muscular blocking drugs. The electroencephalogram is mon tored in patients undergoing surgery for seizures and so neurovascular lesions.

Careful positioning to avoid injury to soft tissues (e. eyes, nose, ears, joints, peripheral nerves) is required. Hea up tilt (15 to 30 degrees) improves cerebral venous drain but increases the risk for venous air embolism.

Smooth emergence and extubation are important t prevent increases in ICP due to cerebral venous congestio This is facilitated by sufficient analgesia and antieme Ondansetron is a popular choice because of its lack sedation. Except after certain neurovascular procedures, t patient should be awake before tracheal extubatio and should exhibit good muscle strength and ventilator drive. Consequently, muscle relaxation should always reversed. When there is any doubt whether the patient w maintain adequate spontaneous ventilation, he or s should be sedated and left intubated and ventilated. Such patients are cared for in the pediatric intensive care uni postoperatively.

Postoperative complications include impaired ventil tion in earlier extubated patients and intracranial bleedin Diabetes insipidus may also occur. Intracranial bleeding usually signaled by a diminishing level of consciousness o increasing ICP in an unconscious patient. Emergent hea computed tomography is indicated to confirm the diagnos and guide further management.

Diabetes insipidus is characterized by the passage o copious volumes of dilute urine, with increased seru sodium concentrations and osmolality. It often occurs afte surgery for hypothalamic tumors and TBI. Treatment consists of (1) replacing urine volume with dilute crystalloi (2) infusing aqueous vasopressin (1 to 10 mU/kg per hour and (3) monitoring serial serum electrolyte concentrations

Diabetes insipidus is often transient. Rebound volume overload and water intoxication can occur if vasopressin is not stopped and the fluid regimen is not adjusted when diabetes insipidus resolves.

Finally, antibiotic and anticonvulsant therapy is continued through the perioperative period, both for prophylaxis and for treatment.

PREVENTION

Prevention of primary TBI is best achieved through sociopolitical interventions and public education (e.g., use of appropriate child restraints in motor vehicles, obeying speed limits). Secondary brain injury is prevented by meticulous management of brain-injured patients, both in the field and in health care facilities. Aggressive resuscitation to maintain adequate oxygenation, ventilation, stable hemodynamics, and cerebral perfusion pressure, while minimizing intracranial hypertension, is the mainstay of therapy. Other therapeutic or prophylactic interventions are instituted after initial resuscitation and stabilization. New technologies (e.g., stereotactic-guided excision of intracranial tumors) have helped reduce the adverse impact of iatrogenic brain injury in pediatric neurosurgery.

Further Reading

Adelson PD, Bratton SL, Carney NA, et al: Guidelines for the acute medical management of severe traumatic brain injury in infants, children and adolescents: Use of hyperosmolar therapy in the management of severe pediatric traumatic brain injury. Pediatr Crit Care Med 4(3 Suppl): S40-S44, 2003.

Alderson P, Roberts I: Corticosteroids in acute traumatic brain injury: Systematic review of randomized controlled trials. BMJ 314:1855-1859, 1997.

Berger S, Schwarz M, Huth R: Hypertonic saline solution and decompressive craniectomy for treatment of intracranial hypertension in pediatric severe traumatic brain injury. J Trauma Injury Infect Crit Care 53:558-563, 2002.

Bullock R, Chesnut RM, Clifton G, et al: Guidelines for the management of severe head injury: Brain Trauma Foundation, American Association of Neurosurgical Surgeons, Joint Section on Neurotrauma and Critical Care. Eur J Emerg Med 3:109-127, 1996.

Carli P, Orliaguet G: Severe traumatic brain injury in children. Lancet 363:584-585, 2004.

Hammer GB, Krane EJ: Perioperative care of the neurosurgical pediatric patient. Int Anesthesiol Clin 34:55-71, 1996.

Johnson JO, Jimenez DF, Tobias JD: Anaesthetic care during minimally invasive neurosurgical procedures in infants and children. Paediatr Anaesth 12:478-488, 2002.

Rekate HL: Head injuries: Management of primary injuries and prevention of secondary damage. A consensus conference on pediatric neurosurgery. Childs Nerv Syst 17:632-634, 2001.

Vavilala MS, Lam AM: Perioperative considerations in pediatric traumatic brain injury. Int Anesthesiol Clin 40:69-87, 2002.

Head Injury

182

Arthur M. Lam and M. Sean Kincaid

Case Synopsis

A 22-year-old previously healthy man sustained a head injury and an open right femur fracture in a motorcycle accident. His initial Glasgow Coma Scale score was 9, and his right pupil was dilated and unreactive. Tracheal intubation was performed at the scene, and he was transported to the trauma center. A computed tomography scan revealed a large right epidural hematoma with a midline shift. Initial hematocrit was 32 after the administration of 2 L of crystalloid. His blood pressure was 130/80 mm Hg, and his heart rate was 120 beats per minute. He was scheduled for emergent evacuation of the epidural hematoma, followed by open reduction and internal fixation of the femur.

PROBLEM ANALYSIS

Definition

Head injury is a common problem, with an annual incidence of approximately 200 per 100,000 persons in the United States. Many of these injuries are minor, with few sequelae, but some are devastating. Car and motorcycle crashes are the most common cause of traumatic brain injury (TBI), followed by injuries from firearms, falls, and sports.

Severe TBI is defined as any injury that results in a Glasgow Coma Scale (GCS) score of 8 or less after adequate cardiopulmonary resuscitation. Damage to neural tissue directly related to trauma is considered the primary injury and includes cerebral contusion, diffuse axonal injury, hemorrhage into the epidural or subdural space, and intraparenchymal hemorrhage. Secondary injury is any insult to the brain occurring after the initial TBI that causes further neuronal damage. Although cerebral ischemia or hypoxia is the ultimate cause of secondary brain injury after TBI, systemic or local insults often contribute to such injury. Among these are elevated intracranial pressure (ICP), systemic hypotension, and hypoxemia.

Neuronal death is likely mediated by complex biochemical processes involving the release of excitatory amino acids (e.g., glutamate) and the cellular influx of calcium. Actual cell death may be necrotic or apoptotic in nature. Preventing or reducing secondary brain injury is the focus of most medical management of TBI in both the intensive care unit (ICU) and the operating room.

TBI is often associated with other injuries (as illustrated in the case synopsis). Thus, anesthesiologists may care for a patient during surgical intervention for TBI (e.g., evacuation of subdural hematoma, decompressive craniectomy) and for laparotomy or fracture fixation, as well as in the ICU.

Recognition

Primary Traumatic Brain Injury

Clinical Signs. TBI is suspected when head trauma is associated with mental status changes. Severity of TBI is commonly assessed by the GCS, which assigns a score to the patient's best motor, verbal, and eye-opening abilities (Table 182-1). A total score of 8 or less indicates severe TBI. Use of the GCS to evaluate patients with TBI reduces interobserver variability and allows for the comparison of serial examinations to evaluate disease resolution or progression. However, use of the GCS as a prognostic indicator is controversial. Further, assignment of a GCS score is appropriate only after adequate cardiopulmonary resuscitation, especially if severe hypotension or hypoxia is present.

Along with the GCS, pupil evaluation is important. TBI may manifest as alterations in pupil size, symmetry, and reactivity to light. With acute unilateral mass lesions, an ipsilateral dilated and unreactive pupil suggests uncal herniation. In contrast, bilateral fixed and dilated pupils suggest severe intracranial hypertension (ICH) that may result in brain herniation.

Vital signs may reflect the patient's overall clinical status aside from any TBI. For example, hypotension and tachycardia may be due to concealed hemorrhage with a femur fracture, and hypertension may be due to pain. Vital signs also

Table 182-1 ▪ Glasgow Coma Scale Score

Eye Opening	
Spontaneous	4
To speech	3
To pain	2
None	1
Verbal Response	
Oriented	5
Confused	4
Inappropriate	3
Incomprehensible	2
None	1
Motor Response	
Obeys commands	6
Localizes to pain	5
Withdraws to pain	4
Flexes to pain	3
Extends to pain	2
None	1

provide significant insight into the nature of TBI. Severe hypertension may be compensatory (i.e., to preserve cerebral perfusion pressure [CPP] in severe ICH; CPP is mean arterial pressure [MAP] minus ICP). Severe systemic hypertension with bradycardia is an ominous sign (Cushing's reflex). It signifies impending brain herniation and requires immediate therapeutic intervention.

Computed Tomography Findings. Cranial computed tomography (CT) is highly sensitive for detecting intracranial hemorrhage and acute mass lesions. CT findings that support a significantly elevated ICP include the following:

- Mass lesion greater than 25 mL
- Midline shift of 5 mm or more
- Compression of the basal cisterns or lateral ventricles
- Medial displacement of the uncus

Secondary Brain Injury

Secondary brain injury is due to systemic or cerebral factors (Table 182-2). Among these, hypoxia and ischemia are most likely to have an adverse impact on TBI outcome. However, the neurologic defects of primary TBI may obscure the signs of secondary injury due to cerebral hypoxia or ischemia. Although the calculation of CPP (which requires an arterial line and ICP monitor) is useful with abnormal head CT findings, even a normal CPP does not preclude secondary ischemia or cerebral hypoxia.

Other monitors are used to assess cerebral blood flow (CBF) and brain perfusion. A jugular venous bulb oximetric catheter (JBC) continuously measures brain venous oxygen saturation ($Sjvo_2$). Low brain perfusion increases oxygen extraction, causing a drop in $Sjvo_2$, while nonfunctioning brain extracts little oxygen to cause high $Sjvo_2$ values. $Sjvo_2$ less than 55% or greater than 75% is associated with a poor prognosis. $Sjvo_2$ catheters are especially useful to monitor cerebral metabolic rate (CMR) when deliberate hyperventilation is used in TBI to reduce global CBF. JBC lactate concentrations may also reveal anaerobic brain metabolism if they are higher than simultaneously drawn arterial lactate concentrations. A limitation of JBC is that it monitors only global CBF-CMR balance. $Sjvo_2$ values can be normal despite small regional areas of ischemia.

Two other devices may provide greater sensitivity for monitoring regional brain ischemia than the JBC: brain tissue oxygen tension ($P_{br}O_2$) monitors and microdialysis catheters. Neither is as widely used as the JBC, but the $P_{br}O_2$ monitor is readily available for clinical application. It provides a continuous measurement of brain parenchymal oxygen tension. This reflects the balance between local brain supply and demand for oxygen. Doppenberg and coworkers showed close correlation between $P_{br}O_2$ and CBF. A $P_{br}O_2$ of 26 mm Hg was about equivalent to a CBF of 18 mL/100 g per minute (i.e., ischemic threshold). Also, a $P_{br}O_2$ of approximately 39 mm Hg is correlated with a good outcome, whereas one of 19 mm Hg correlates with a bad outcome, thus offering some guidance for therapeutic intervention. The normal $P_{br}O_2$ is greater than 20 mm Hg.

Microdialysis catheters are placed in brain parenchyma, where they continuously perfuse the brain with a perfusate and sample small volumes of fluid (the dialysate), which is tested for lactate and pyruvate concentrations to estimate the balance between anaerobic and aerobic metabolism. In addition, glutamate, glucose, and glycerol can be measured. However, a fairly long lag time is needed to analyze samples, which hinders real-time clinical decision making. Thus, microdialysis catheters are predominantly a research tool in their present form.

Risk Assessment and Implications

Hypoxia and Hypercapnia. TBI patients are at increased risk for airway obstruction and hypoventilation. These lead to hypoxia and hypercapnia, which cause cerebral vasodilatation. The latter may aggravate any ICH.

Elevated Intracranial Pressure. An acute mass lesion increases ICP and reduces CPP. Increased ICP can lead to brain herniation, with catastrophic consequences.

Systemic Hypotension and Hypovolemia. Adults usually do not become hypovolemic and hypotensive as a result of blood loss from TBI alone. In contrast, small children can lose enough blood with TBI to become hypotensive. Other injuries (e.g., splenic rupture, femur fracture) can make TBI patients hypotensive and further compromise CPP in those with increased ICP. Compensatory hypertension and bradycardia (Cushing's reflex) with elevated ICP may further complicate the clinical picture. Thus, in patients with TBI, normotension and tachycardia can still be compatible with severe hypovolemia, with the latter "concealed" by increased systemic vascular resistance (Cushing's reflex). Thus, ample blood pressure may give clinicians a false sense of security

Table 182–2 • Risk Factors for Secondary Brain Injury

Cerebral Factors	Systemic Factors
Increased intracranial pressure	Hypotension
Expanding mass lesions	Hypoxemia
Hypercapnia	Anemia
Hypoxemia	Hypovolemia
Venous obstruction (cervical collar, poor positioning)	Hyperglycemia
Systemic hypotension (compensatory cerebral vasodilatation)	Hyponatremia
Excessive hyperventilation	Hypo-osmolar state
Post-traumatic vasospasm (patient with traumatic subarachnoid hemorrhage)	Coagulopathy
Seizures	

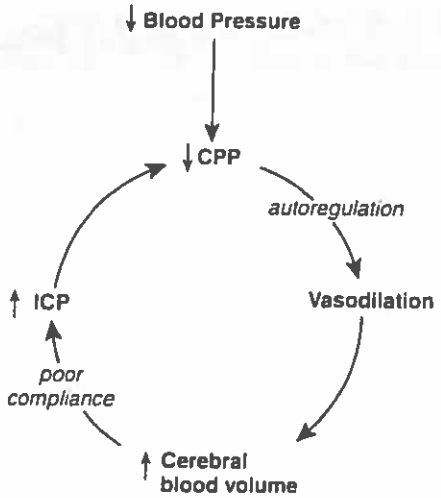


Figure 182–1 Vasodilator cascade, showing the potential interaction between systemic hypotension and intracranial hemodynamics when autoregulation is intact. A cascade in the opposite direction also occurs when blood pressure is increased. CPP, cerebral perfusion pressure; ICP, intracranial pressure.

regarding the progress of resuscitation. Should ICH be relieved by decompressive craniectomy or evacuation of an intracranial hematoma, profound hypotension or cardiac arrest may occur.

Of all the factors associated with secondary brain injury, systemic hypotension is likely the most significant. With impaired cerebral autoregulation, it invariably leads to reduced CPP. Patients with intact autoregulation but reduced intracranial compliance are also at risk for impaired CPP with hypotension. Reduced MAP dilates cerebral vasculature to increase cerebral blood volume and ICP. This increase in ICP further compromises CPP, leading to further compensatory cerebral vasodilatation. This vicious circle is referred to as the vasodilator cascade (Fig. 182-1).

Impaired Autoregulation. Cerebral autoregulation is a homeostatic mechanism that maintains near-constant perfusion of the brain over a wide range of MAPs. In normal adults, this range is 60 to 160 mm Hg. Autoregulation may be impaired in patients with TBI, and although the frequency of impaired autoregulation is higher in patients with severe TBI, it is clinically impossible to predict which patients will be affected. Even minor TBI may impair autoregulation. If so, CBF becomes directly proportional to blood pressure. Loss of cerebral autoregulation is associated with worse outcomes with TBI.

Coagulopathy. Severe TBI liberates enough thromboplastin from damaged neurons to cause coagulopathies, which may be mild to severe. They can increase surgical morbidity and mortality, can preclude or delay extracranial surgical procedures, and are associated with poorer outcomes.

Pyrexia. Fever raises the CMR, increasing the risk for ischemia and neural injury, especially when cerebral perfusion is marginal. Cerebral blood volume increases with pyrexia owing to flow-metabolism coupling, exacerbating any ICH. Although human studies do not conclusively link body temperature to outcome in TBI, both animal and human studies have linked brain infarct size and fever to ischemic brain injury.

Hyperglycemia. Hyperglycemia in TBI and stroke is associated with a poor prognosis, although a cause-effect relationship has not been clearly established. In experimental cerebral ischemia, detrimental effects of hyperglycemia have consistently been shown. Further, in one prospective trial, van den Berghe and colleagues found that patients with lax glucose control had worse outcomes than those with tight control.

Fluid and Electrolyte Abnormalities. Acute fluid and electrolyte disturbances occur in TBI patients, often due to inappropriate fluid administration. They can also be caused by diabetes insipidus. Hyponatremia and excessive free water may worsen cerebral edema, thereby increasing ICP.

Associated Injuries. As many as 10% of patients with TBIs also have spine injuries. Spinal evaluation is often delayed if the patient requires emergent neurosurgical intervention (e.g., evacuation of epidural or subdural hematoma). For this reason, spine precautions should be taken when moving or positioning patients before the completion of the spine injury workup. TBI patients may also have undiagnosed extremity injuries.

MANAGEMENT

Secure the Airway

Immediate tracheal intubation is necessary for severely head-injured patients, particularly those with GCS scores of 8 or less and without protective airway reflexes. Both propofol and thiopental are used as induction agents because they decrease CMR and lower ICP. However, either may cause hypotension, especially in inadequately fluid-resuscitated TBI victims, which negates their benefit. Because of a lower risk of untoward hypotension in TBI patients, etomidate may be a better choice. Ketamine is avoided because it increases ICP. A short-acting muscle relaxant should be used. Succinylcholine is preferred, and rocuronium is used when succinylcholine is contraindicated.

Maintain Adequate Cerebral Perfusion Pressure

The updated Brain Trauma Foundation guidelines (2003) advise keeping CPP between 60 and 70 mm Hg (in patients without cerebral ischemia); the trend today is to maintain CPP above 60 mm Hg. To maintain CPP, there must be good intravenous access, and fluid resuscitation must replete intravascular volume as needed. Fear of worsening cerebral edema should never dissuade one from providing adequate fluid resuscitation. Hypotonic fluids should be avoided, however (Table 182-3). Hypertonic fluids (e.g., 3% saline) may be used, although evidence is lacking to justify their routine use. Vasopressors and inotropes are often used along with fluid resuscitation to maintain CPP. However, they should be used with caution, because they may increase the risk for acute respiratory distress syndrome. Without ICP monitoring, MAP should be maintained at greater than 70 mm Hg.

Table 182–3 ▪ Intravenous Fluids

Fluids	Osmolality (mOsm/kg)	Oncotic Pressure (mm Hg)	Na^+ (mEq/L)	Cl^- (mEq/L)	K^+ (mEq/L)	Ca^{2+}/Mg^{2+} (mEq/L)	Gluco (g/L)
Plasma	289	21	141	103	4-5	5/2	
Crystalloid							
0.9% NS	308	0	154	154			
0.45% NS	154	0	77	77			
3% NS	1030	0	515	515			
7.5% NS	2400	0	1200	1200			
LR	273	0	130	109	4	3/0	
D_5LR	527	0	130	109	4	3/0	50
D_5W*	252	0					50
D_5 NS*	586	0	154	154			50
$D_5$0.45% NS*	406	0	77	77			50
Normosol	295	0	140	98	5	0/3	
Mannitol (20%)	1098	0					
Colloid							
Hetastarch (6%)	310	31	154	154			
Albumin (5%)	290	19					
Plasmanate	270-300	?	145	100	0.25		

*The osmolality of these dextrose solutions decreases as glucose enters the cells.
D5W, 5% dextrose in water; LR, lactated Ringer's; NS, normal saline.

Ischemia is likely the final pathway in secondary brain injury. Therefore, the hematocrit is kept at 30% to provide adequate oxygen delivery. If Cushing's reflex is present in patients with acute subdural or epidural hematoma, blood pressure may decline precipitously with surgical decompression. This is anticipated based on clinical findings (e.g., low GCS score, significant midline shift on CT, abnormal pupils), and preemptive intravenous fluid resuscitation should be undertaken. Prompt treatment of hypotension after surgical decompression with intravenous fluids and vasopressors or inotropes is essential.

Reduce Intracranial Pressure

To optimize CPP, one must try to reduce ICP. Recent data suggest that better physiologic parameters are maintained (e.g., $SivO_2$, arterial-venous difference in oxygen saturation) in patients with ICP of 20 mm Hg or less. CPP is maintained in the 60 to 70 mm Hg range. Mannitol (0.25 to 1 g/kg) is useful for reducing brain bulk and may decrease the production of cerebrospinal fluid; both these effects reduce ICP. Mannitol is given after volume repletion. Patients refractory to mannitol may respond to hypertonic saline (3% or 7.5%). Cerebral blood volume is reduced with acute hyperventilation, and CBF decreases by about 3% for each 1 mm Hg decline in arterial carbon dioxide tension. There is the potential for cerebral ischemia with excessive hyperventilation. Arterial carbon dioxide tension is not decreased to less than 30 mm Hg, except for brief periods (e.g., impending herniation). Otherwise, normocapnia or slight hypocapnia (35 to 40 mm Hg) is desirable when ICP is less than 20 mm Hg.

Barbiturates or propofol given to suppress CMR can reduce ICP. Effects are maximal with electroencephalogram burst suppression or an isoelectric electroencephalogram. Vasopressors may be required to support blood pressure with maximal CMR suppression. Low-dose propofol is often used in TBI, because it allows effective ICP control while permitting prompt neurologic evaluation. However, metabolic syndromes characterized by myocardial dysfunction and lactic acidosis have been observed after prolonged propofol infusions, especially in children.

Other techniques to reduce ICP are slight head-up and neutral neck positions. Both facilitate venous drainage. Many TBI patients have cervical collars in place, and it important to inspect the collar to ensure that it does impede venous drainage; a collar that is too tight can increase ICP. Circumferential endotracheal tube ties should be avoided for the same reason. In patients with increased ICP that is refractory to medical management, a decompressive craniectomy is indicated.

Correct Coagulopathies

Coagulopathies increase the morbidity associated with any surgery in TBI patients. Coagulation should be followed closely, and any deficient factors should be replaced aggressively. Some surgeons advocate early replacement of platelets and clotting factors based solely on clinical observations.

Treat Hyperglycemia

Dextrose-containing intravenous solutions are avoided during fluid resuscitation. They may cause hyperglycemia and worsen cerebral ischemic injury. Current ICU guidelines advise keeping blood glucose at 80 to 110 mg/dL. Some may be uncomfortable with such tight glucose control in anesthetized patients, because signs of hypoglycemia may be masked. Insulin infusions are titrated by frequent glucose determinations to keep glucose levels at less than 120 mg/dL.

Restore Normothermia

Clearly, hyperthermia is harmful to patients at risk f ischemic brain injury. Any beneficial effects of hypothermia

II. Selections from "Essentials of Neuroanesthesia and Neurointensive Care

Arun K. Gupta, MBBS, MA, PhD, FRCA

Adrian W. Gelb, MBChB, DA, FRCPC

II. Selections from "Essentials of Neuroanesthesia and Neurointensive Care
Arun K. Gupta, MBBS, MA, PhD, FRCA
Adrian W. Gelb, MBChB, DA, FRCPC

1. Supratentorial lesion
2. Subarachnoid Hemorrhage
3. Head Trauma
4. Cervical Spine Injury

Essentials of Neuroanesthesia and Neurointensive Care

Arun K. Gupta, MBBS, MA, PhD, FRCA
Consultant in Anaesthesia and Neurointensive Care
Director of Postgraduate Medical Education
Addenbrooke's Hospital
Cambridge University Hospitals
NHS Foundation Trust
Associate Lecturer
University of Cambridge
Cambridge, United Kingdom

Adrian W. Gelb, MBChB, DA, FRCPC
Professor
Department of Anesthesia and Perioperative Care
University of California–San Francisco
San Francisco, California

SAUNDERS
ELSEVIER

SAUNDERS
ELSEVIER

1600 John F. Kennedy Blvd., Suite 1800
Philadelphia, PA 19103-2899

Essentials of Neuroanesthesia and Neurointensive Care ISBN: 978-1-4160-4653-0

Library of Congress Cataloging-in-Publication Data
Essentials of neuroanesthesia and neurointensive care / [edited by] Arun K. Gupta, Adrian W. Gelb. – 1st ed.
p. ; cm. – (Essentials of anesthesia and critical care series)
Includes bibliographical references.
ISBN 978-1-4160-4653-0
1. Anesthesia in neurology. 2. Neurological intensive care. 3. Nervous system–Surgery. I. Gupta, Arun K. II. Gelb, Adrian W. III. Series.
[DNLM: 1. Anesthesia–methods. 2. Neurosurgical Procedures. 3. Brain–drug effects. 4. Intensive Care. 5. Perioperative Care. WL 368 E775 2008]

RD87.3.N47E87 2008
617.9'6748–dc22 2007038047

Executive Publisher: Natasha Andjelkovic
Developmental Editor: Isabel Trudeau
Publishing Services Manager: Joan Sinclair
Project Manager: Lawrence Shanmugaraj
Text Designer: Karen O'Keefe Owens

Printed in China
Last digit is the print number: 9 8 7 6 5 4 3 2 1

Clinical Cases in Neuro Anesthesia (Ref- Essentials of Neuroanesthesia and Neurointensive care- Authors Gelb and Gupta)

Supratentorial lesion

Case Summary

A 43-yr-old, otherwise healthy woman was evaluated for tonic-clonic seizures. Computed Tomography (CT) showed a large enhancing lesion in the right frontal lobe suggestive of glioma with a 2-mm shift. She had no focal neurological signs but had a headache, which improved. Four days after the seizure she underwent craniotomy for biopsy and probable resection. Her medications were phenytoin, 300 mg once daily before sleep, and dexamethasone, 4 mg once every 6 hours. Her weight was 81 kg, she had no history of allergies, and airway management suggested that there will not be any problem with tracheal intubation.

Anesthesia was conducted as follows:premedication with midazolam, 2 mg intravenously in the preoperative area; application of standard American Society of Anesthesiologists(ASA) monitors; induction of anesthesia with remifentanil, 2 mcg/kg, and propofol, 2 mg/kg; intubation facilitated with rocuronium , 50 mg; and insertion of a 7.0-mm cuffed endotracheal tube without difficulty. The patient was positioned facing the anesthesia team with a bolster under her right side, and her head was secured to the table with a Mayfield frame. Anesthesia was maintained with infusions of remifentanil 0.1 to 0.25 mcg/kg/min, and propofol, 50 to 100 mcg/kg/min and inhalation of sevoflurane, 0.5 end-tidal minimal alveolar concentration (MAC). A radial artery catheter and second intravenous line were inserted. Neuromuscular blockade was maintained throughout with rocuronium. The surgeon requested ceftriaxone, 1 gm, mannitol, 1 gm/kg, and dexamethasone, 10 mg.

Discussion

- Problem list
- New onset seizures
- Intraparenchymal brain tumor
- Increased intracranial pressure (ICP) symptomatically improved with dexamethasone

Clinical Findings

New onset seizure is a common manifestation of brain tumors, and these lesions often occur in the middle decades of life. In most patients, symptoms are controlled preoperatively with anticonvulsants and dexamethasone. Lesions in the frontal lobe may have no focal symptoms, but tumors in other areas or when ICP is raised can be manifested as weakness; vision, speech, or balance difficulty; headache; or nausea and

vomiting. Acute neurological deterioration may occur if there is a sudden increase in swelling around the tumor, bleeding, or necrosis.

Management

Preoperative preparation for a patient in stable neurological condition involves a complete history and physical and, if necessary, optimization of co morbid conditions. Preoperative administration of a benzodiazepine is acceptable because these patients are understandably very anxious. Caution is advised inasmuch as the sedative effect of the benzodiazepine is occasionally greater than the anticipated and the patient can become somnolent with increased PaCO2 and ICP. Patients who are already neurologically obtunded should not be premedicated. Many possible combinations of anesthetic drugs could be used for this type of surgery. There is no evidence from clinical trials to support better outcomes with any particular combination of drugs. Suitable alternatives include total intravenous anesthesia with propofol and remifentanil or fentanyl or a low-dose vapor (< 1 MAC) with opiod. In this case a combination of remifentanil with low-dose propofol and low-dose sevoflurane was used in the hope of obtaining some benefit from all three. Neuromuscular blockade is indicated for most straightforward craniotomy procedures such as this, and intermediate-acting drugs should be the first choice. Patients taking anticonvulsants (particularly phenytoin) for a week or more may have a very high requirement for muscle relaxant drugs. This is most marked for rocuronium, vecuronium, or pancuronium but is also significant for atracurium and cisatracurium.

More important than the choice of anesthetic drugs is understanding the goals of anesthetic management, which are cardiovascular stability to maintain cerebral perfusion, manipulation of brain volume to facilitate the surgical approach, and rapid emergence from anesthesia to facilitate early neurological examination. In the absence of indications to the contrary, blood pressure should be kept within the patient's normal range and isotonic fluids administered as needed. If there is significant blood loss, a hematocrit of 30% is a reasonable transfusion trigger.

The presence of a space-occupying lesion usually means that measures to decrease brain volume are indicated to facilitate surgical exposure, for example, to relieve pressure under the dura before it is incised. Standard interventions are mild to moderate hyperventilation, administration, and reverse Trendelenburg positioning. These interventions should always be performed after communication with the neurosurgical team. If these interventions are not effective, the surgeon may elect to drain cerebrospinal fluid directly. Finally, whatever drugs are used for anesthesia, the intent should be to have the patient emerge from anesthesia in a rapid and peaceful manner. Coughing on the endotracheal tube or its effects should be minimized; for example, with a head up position, opioid, or lidocaine. Supplementary doses of antiemetics may be required intraoperatively, especially if the procedure is prolonged.

Specialized Procedures

Tumors in areas of the brain with specific motor or speech functions will require modification of anesthetic management. Identification of motor areas by stimulation of the motor cortex specifically requires that the patient not be paralyzed during the procedure. Tumors in or near the speech areas will require speech mapping, which necessitates that the patient be awake for at least a significant portion of the procedure.

Clinical Cases in Neuro Anesthesia(ref- Essentials of Neuroanesthesia and Neurointensive care Authors –Gelb and Gupta)

Subarachnoid Hemorrhage

Case Summary

A 56-yr-old woman with a history of untreated hypertension woke up with a severe headache the morning after being at a party where she consumed a large amount of alcohol. She became very drowsy and confused but not hemi paretic and was taken to the Emergency Department by her family. A CT scan showed a grade 3 subarachnoid hemorrhage (SAH) in the area of her anterior communicating artery.

She was admitted to the ICU with a GCS score of 10 to 11 and given labetolol, morphine, and nimodipine. The next morning she underwent cerebral angiography under anesthesia, and an anterior communicating artery aneurysm was coiled under anesthesia by the neuron-interventional radiologist. Three days later she experienced deteriorating neurologic function, and severe vasospasm was treated by repeat angiography, angioplasty of the affected vessels, and intra-arterial injection of nicardipine. Hydrocephalus developed and was initially treated with an external ventricular drain, which was then converted to a ventriculo-peritoneal (VP) shunt 2 weeks after the initial bleeding. She was discharged home a week after the VP shunt was placed.

Discussion.

Problem List

- Subarachnoid Hemorrhage
- Vasospasm
- Hydrocephalus

Overview

Risk factors for SAH include female gender, hypertension, and binge drinking. Other putative risk factors are a smoking history and non white ethnicity. Her clinical state classified her as grade 3 under Hunt & Hess classification. Early intervention is important because the risk for rebleeding is 4%in the first 24 hours and greater than 1% per day for the first 2 weeks.

Initial Management

She received very conservative management in the ICU. It would have been preferable to have placed an intra-arterial catheter, possibly a central venous line, and urinary catheter, plus administer an H2 blocker. Her neurologic status did not warrant tracheal intubation.

Treatment Options

Although this patient had her anterior circulation aneurysm treated by endovascular coiling, the other main option would have been to have the aneurysm clipped surgically. There is now good evidence that 2-yr outcomes are superior with endovascular coiling versus surgical clipping, although longer-term data are not complete.

Anesthetic Management for Angiography

If the patient were just going to undergo diagnostic angiography without treatment, careful sedation might be sufficient but it is often difficult in confused patients. However, the objective was to treat the aneurysm, which required general endotracheal anesthesia. This guarantees no patient movement at critical moments and apnea for improved image quality. Specific management principles focus on tight control of hemodynamics. Severe hypertension might provoke aneurysmal rebleeding. Severe hypotension will worsen the cerebral ischemia that frequently accompanies SAH. Preinduction intra-arterial pressure monitoring is recommended.

Vasospasm

The patient's neurologic deterioration at 3 days was very suggestive of vasospasm and this was confirmed by four-vessel angiography. Transcranial Doppler ultrasonography may have been useful, although it is more predictive of vasospasm in the middle cerebral artery territory. Other potential diagnostic modalities are CT angiography, magnetic resonance imaging (MRI) and radio-nuclide imaging, but definitive treatment requires angiography.

Hemodynamic augmentation (hypertension, hypervolemia, hemodilution) or triple-H therapy was the only therapy for a long time. Although this modality in its various forms can reverse the symptoms of ischemia, it is associated with significant complications such as pulmonary edema and myocardial infarction. Intraluminal balloon angioplasty is effective for isolated spasm of proximal vessels in the circle of Willis, and intra-arterial infusion of vaso-dilators (such as nicardipine) can be used for more distal or diffuse spasm.

Final Disposition

This patient's course was fairly typical. She avoided possible outcomes that can accompany SAH, such as seizures, myocardial ischemia, congestive heart failure, acute lung injury, gastric erosions and cerebral salt wasting.

Clinical Cases in Neuro Anesthesia (Ref-Essentials of Neuroanesthesia and Neurointensive Care Authors Gelb and Gupta)

Head Trauma

Case Summary

An 18-yr-old man was struck on the head with a baseball bat and brought to the emergency department by ambulance. On admission his Glasgow Coma Scale (GCS) score was 8, his oxygen saturation was 89% on room air, and he was wearing a hard plastic neck collar. Immediate anesthesia consultation was sought, and the patient's airway was controlled with oxygenation, neuromuscular blockade, tracheal intubation, ventilation to normoxia, and PaCO2 in the low normal range.

CT showed severe contusions in the right hemisphere and no skull fracture or cervical spine injury. Once the patient was transferred to the neurosurgical intensive care unit (ICU), a ventriculostomy catheter was placed and a radial artery catheter inserted. His ICP was 25 mm Hg, so 20% mannitol, 2 ml/kg intravenously was infused, which reduced ICP to 20 mmHg.

Discussion

Problem List

- Comatose patient
- Elevated ICP
- Intracranial mass

Principles of Care

When faced with head-injured patients in a critical situation such as this, the guiding principle is to treat the greatest threat to life (airway, breathing, circulation [ABCS]) to avoid doing further harm, such as by inducing severe hypotension or further injuring the cervical spine. This patient has an isolated head injury, but many will be seen with multiple trauma. In such patients, clinical suspicion of and examination for concomitant injuries such as a ruptured spleen and bone fractures must be performed. In patients with multiple trauma, aggressive resuscitation and maintenance of hemodynamics and pulmonary function are of paramount importance.

Controlling the Airway

In this patient both the hypoxia and GCS of 8 are indications for immediate control of the airway with tracheal intubation, followed by mechanical ventilation. It is imperative that the airway be secured as swiftly as possible, usually by direct laryngoscopy. The nasal

route should be avoided if a basal skull fracture is suspected. The use of short-acting neuromuscular blocker succinylcholine is usually required to optimize intubating conditions. The transient rise in ICP as a result of muscle fasciculations has not been shown to be detrimental to outcome and the risks associated with a difficult intubation outweigh those of a depolarizing muscle relaxant.

Concomitant cervical spine injury may be present in patients with a severe head injury (GCS score </= 8), and the tracheal intubation technique must take account of this possibility. To facilitate laryngoscopy and intubation, the anterior part of the neck collar should be removed. Manual-in-line immobilization should be performed before the collar is removed. The procedure involves an assistant (from the side or behind) holding the mastoid processes and occiput to minimize neck movement during laryngoscopy. The aim is to stabilize the neck by counteracting the forces of laryngoscopy; active traction should not be applied.

Mainstays of Management

After tracheal intubation, initial goals of ventilation are normoxia and PaCO2 in the low normal range (30 to 35 mm Hg). Hypoxia with a saturation of less than 90% is associated with a worsened outcome. Hyperventilation to 30 mm Hg is indicated only if focal neurological signs (e.g. dilating pupil) are evident or ICP is sustained above 25 mm Hg because it can exacerbate cerebral ischemia. This patient's ICP of 25 mm Hg responded to mannitol and, without signs of herniation, did not justify hyperventilation.

Hypotension (systolic blood pressure <90 mm Hg) is independent predictor of outcome in brain injury, and vigorous maintenance of blood pressure of is indicated; isotonic fluids with the addition of a vasopressor if required should be instituted. The Brain Trauma Foundation guidelines suggest maintaining cerebral perfusion pressure around 60 mm Hg.

Evidence suggests that outcome is worse in brain injury with hyperglycemia, so blood sugar should be maintained within a normal range. Hyperthermia increases cerebral metabolic rate and blood flow and exacerbates cerebral ischemia. Elevated temperature should be returned to normal levels by surface cooling.

Other Therapeutic Modalities

The simplest is elevation of the head to decrease venous pressure. Angles of elevation of 10 to 30 degrees have been proposed. Seizures are very detrimental and should be actively suppressed, although there is no evidence of benefit of prophylactic anticonvulsant therapy. Sedation and neuromuscular blockade are used when ICP remains elevated, and barbiturate coma may be induced if ICP is refractory to all other medical therapies. Surgical decompression craniectomy with or without lobectomy or excision of contusions is reserved for situations in which brain swelling cannot be otherwise controlled.

Fluids

Hypertonic saline has several potential beneficial effects; it is useful as an expander of intravascular volume and as an osmotic agent. Consequently, it can decrease cerebral edema and improve regional cerebral blood flow. There is ongoing debate regarding colloid versus crystalloid as the preferred fluid, with no clear data in favor of either. Hypotonic fluids should be avoided.

Clinical Cases in Neuro Anesthesia (ref-Essentials of Neuroanesthesia and Neurointensive Care- Authors Gelb and Gupta)

Cervical Spine Injury

Case Summary

A 62-yr-old man arrived in the emergency department after a motor vehicle accident while on his way from dinner at a restaurant. He had multiple injuries, including a femoral fracture, and had a rapidly expanding abdomen, blood pressure of 80/50 mm Hg and HR of 121. He complained of neck pain. His neck was immobilized with a hard collar, sandbags, and tape across his forehead.

A cross table lateral radiograph showed no cervical spine injury. Because of the urgent need to proceed to exploratory laparotomy, there was no time for a further radiologic work-up. He weighed 97 kg, and his airway examination was MP2, with a four-fingerbreadth thyromental distance.

He underwent rapid-sequence induction of anesthesia with tracheal intubation via direct laryngoscopy and cricoid pressure. Only the front of the collar was removed, and manual-in-line stabilization was performed. At laparotomy he had a ruptured spleen and a liver laceration.

Discussion

Problem List

- Hypotension
- Ruptured abdominal organ
- Fractured femur
- Potential cervical spine injury
- Potential difficult airway

Risk of Significant Cervical Spine Injury and Radiology Work-up

This patient is at significant risk for having a neck injury in that he was involved in an apparently high speed auto accident and is complaining of neck pain. In the ideal world, a significant and potentially unstable neck injury would be ruled out before the patient's airway was manipulated. The regimen recommended for diagnosing and defining a neck injury is a three-view spine series (lateral, anteroposterior, and odontoid) with supplemental high-resolution CT for poorly visualized or suspicious areas. With this regimen, the risk of a false-negative result is very low. There is little place for MRI in the initial management of cervical spine injury. In this emergency clinical scenario, such an extensive work-up was not possible.

Airway Management

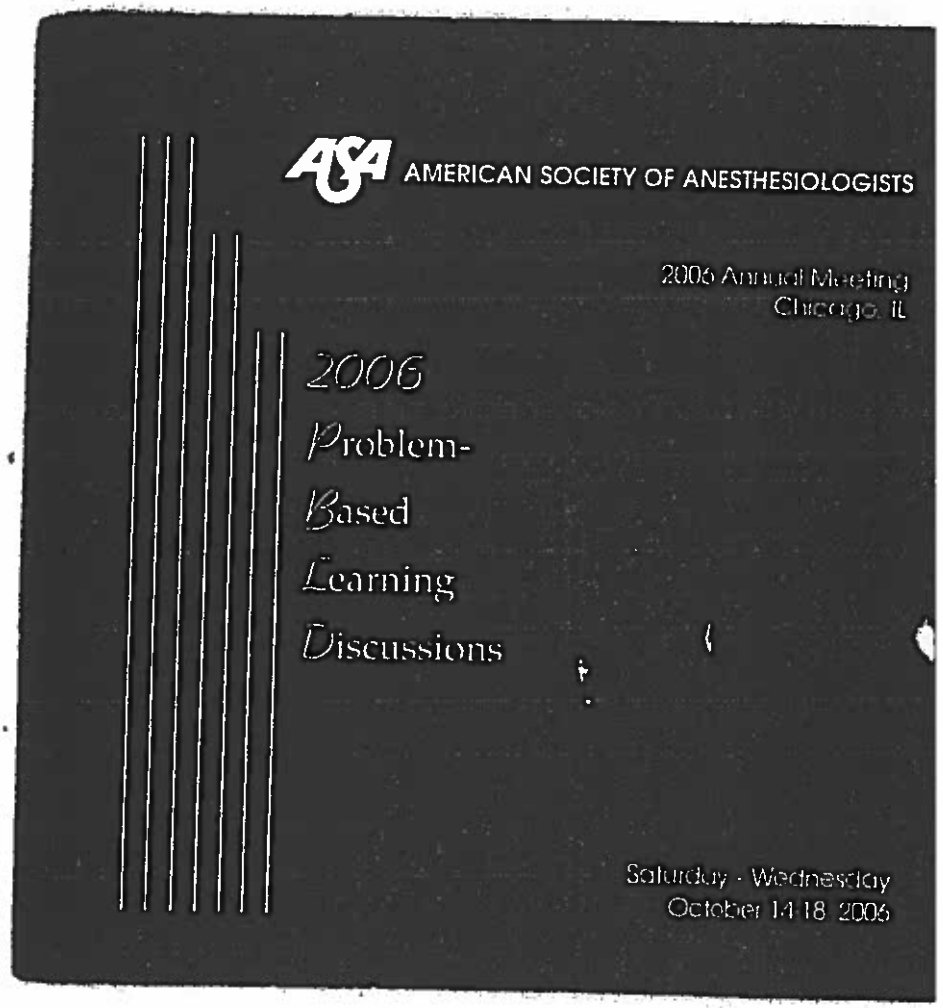
This patient's spine was immobilized in the ideal manner with a hard collar, sandbags and forehead tape. For airway management, only the front of the collar can be removed, and other immobilization measures must be maintained. The degree to which the cervical spine moves is greatest with maneuvers such as chin lift and jaw thrust and is less during laryngoscopy and intubation.

No technique for intubation is superior to or recommended over any other. Most clinicians, when asked, are of the opinion that awake fibreoptic intubation is the method of choice, but interestingly, most also state they are inexperienced with the technique, especially in emergency situations. The consensus is to use the technique most appropriate for the urgency of airway control and with which the clinician is most skilled. Techniques both reported and approved include direct laryngoscopy, rigid intubating laryngoscopes such as Bullard, intubating laryngeal mask airways, Combitube, lightwand, fibreoptic bronchoscopy and cricothyrotomy.

Secondary Injury

Secondary injury can occur after the initial trauma and may be associated with clinical interventions. The greatest risk factor is failure to suspect an underlying neck injury and not providing adequate immobilization. The spine should be immobilized in a neutral position for that patient. For example, with preexisting spinal deformity, the neutral position is the chronic position of the deformity, and this should be the position of immobilization.

There have been no reports of secondary injury attributable to airway management and tracheal intubation in which neck immobilization and manual-in-line stabilization were used. Hypotension may exacerbate a cord injury already present and should be treated aggressively. Other causes of secondary injury are vertebral artery injury and an ascending myelopathy, which may be produced by cord edema and inflammation or an apoptotic process resulting from the initial injury.


AMERICAN SOCIETY OF ANESTHESIOLOGISTS
2006 Annual Meeting
Chicago, IL
2006
Problem-
Based
Learning
Discussions
Saturday - Wednesday
October 14-18, 2006

III. Case studies from "2006 Problem-Based Learning Discussions

American Society of Anesthesiologists
2006 Annual Meeting, Chicago, IL

III. Case studies from "2006 Problem-Based Learning Discussions"
American Society of Anesthesiologists 2006 Annual Meeting, Chicago, IL

1. Subarachnoid Hemorrhage (SAH) and Critical Care Management – Not Just Your Patient's Headache by Martin L. De Ruyter, MD – Kansas City, Kansas
2. Anesthetic Challenges in Seizure Surgery and the Fancy Devices Used for Control by Ramachandran Ramani, MBBS, MD – Easton, Connecticut
3. Acute Stroke Following Cardiac Catheterization by Michelle Lotto, MD – Cleveland Ohio
4. Emergency Surgical Fixation of Unstable Cervical Spine by Ramesh Ramaiah, MD, FRCA, FCARCSI – Seattle, Washington

Subarachnoid Hemorrhage (SAH) and Critical Care Management – Not Just Your Patient's Headache

Martin L. De Ruyter, M.D. Kansas City, Kansas

OBJECTIVES

Review the incidence of subarachnoid hemorrhage, aneurysmal location, and associated mortality and morbidity.

Identify the signs and symptoms of SAH and discuss its initial evaluation

Discuss critical care management of SAH, focus on the role of a multidisciplinary approach in the acute interventions and strategies aimed at delayed ischemic injury

Discuss newer therapeutic approaches

Discuss definitive aneurysmal treatment.

STEM CASE - KEY QUESTIONS

A 56 year-old woman with a past medical history significant for mild hypertension and 35 pack-years of smoking presented to the emergency room confused, disorientated, and unable to follow commands. Eight hours prior, she had complained to her spouse the sudden onset of a severe headache, which she described as the "worst headache of my life". The review of symptoms revealed that one week prior to admission, the patient complained of a minor headache which was relieved with ibuprofen. She was a social alcohol user and physically active. Her medications included hydrochlorothiazide and daily vitamins. The patient had no known drug allergies. Her family history was significant for a sister who died suddenly at the age of 52 years after a fall.

On exam, the patient's blood pressure was 160/90 mmHg, pulse 80 beats/min, respiratory rate 20 breaths/min and core temperature 37^0 Celsius. Lungs fields were clear to auscultation and cardiac exam revealed a regular rate and rhythm with no gallops, clicks, or murmurs. Neurological exam revealed a patient who localized to the left upper extremity in response to stimulus. She spontaneously moved all four extremities, with the left side moving greater than the right side. In response to painful stimuli, the patient withdrew from pain on the left side and from deep pain on the right side. Motor strength was grossly intact on the left side, but decreased on the right side.

1. *What is the incidence of SAH in the United States and what are the common presentations? What are the risk factors for SAH? What is the distribution of intracerebral aneurysms?*

Following initial evaluation in the emergency room, the patient received a non-contrast head CT. The study revealed blood in the basal cisterns and in the fourth ventricle. Chest roentgenogram, electrocardiogram, and laboratory specimens were collected. Subarachnoid hemorrhage was diagnosed with her Glasgow coma score (GCS) = 12 and the World Federation of Neurological Surgeons (WFNS) grade = IV. The patient was admitted to the intensive care unit for further management and consultations by neurosurgery and neuroradiology.

2. *What are the components of the Glasgow Coma Scale, the World Federation of Neurological Surgeons grading scale, and the Modified Hunt-Hess grading scale? Do they prognosticate outcome?*

3. *What is the sequela of a missed diagnosis? What is a sentinel bleed?*

Upon arrival to the ICU, the patient was noted to be less responsive. The electrocardiogram revealed sinus tachycardia with large T wave inversions across the precordial leads. The patient's blood pressure was 180/95, she remained tachypneic and her GCS declined to 10. An arterial line and central venous line were placed for monitoring. Isotonic saline was administered intravenously and a urinary Foley catheter was placed to record urine output. Blood pressure was titrated to less than 160 mmHg systolic (mean < 100 mmHg) with labetalol. The patient underwent cerebral angiography, which revealed a ruptured aneurysm in the left middle cerebral artery trifurcation. Nimodipine 60 mg every four hours was initiated. Neurosurgical consultation recommended medical optimization to wait and observe if the patient's clinical condition would improve (i.e. WFNS score better than IV). The following day the patient's exam slightly worsened as she only opened eyes to pain, had incomprehensible speech and withdrewl to pain. A noncontrast head CT was obtained which revealed marked dilation of the ventricles and blood outlining the dependent ventricular cavity. Neurosurgery placed an external ventricular drain at the bedside. The patients WFNS grade remained IV and neuroradiology agreed to take the patient for endovascular coiling. Prior to travel to the radiology suite, it was decided to intubate the patient for airway protection and initiate mechanical ventilation. This proceeded in an uncomplicated manner. Neuroradiology was able to successfully coil the aneurysm and the patient returned to the ICU. Frequent neuro checks recorded the patient's progress. In the morning of hospital day #3, the patient was more responsive, opening eyes to commands and following simple commands. The patient was started on a ventilator weaning trial.

4. *What are the initial aims of SAH therapy?*

5. *Is outcome affected by early definitive aneurysm intervention?*

6. *Surgical intervention versus endovascular coiling – What is the desired approach?*

7. *What are the early complications of SAH*

8. *What practical approaches are undertaken in securing this patient's airway?*

9. *What would be a reasonable ventilator mode? What would be one's weaning protocol and extubation criteria?*

Laboratory exam revealed serum sodium of 129 mEq/l. The patient's blood pressure was 120/60, pulse 80 beats/min, and CVP = 5. Transcranial Doppler revealed peak velocities 110 meter/sec. Later that afternoon the patient's condition deteriorated. The patient was not following commands, unable to respond to verbal stimuli, blood pressure = 98/42, pulse = 99 beat/min, and CVP = 3.

10. What are the later complications of SAH?

11 What is vasospasm? How is it monitored? What is prophylactic therapy?

12. Why is the patient hyponatremic? What is the appropriate therapeutic action?

Initial therapy included infusion of normal saline with intermittent boluses of 5% albumin in an effort to increase the intravascular volume of the patient. Phenylephrine was added to augment the blood pressure after the CVP was greater than 10 to augment the blood pressure. The patient's condition improved transiently for a few hours then again deteriorated. A pulmonary artery catheter was placed and hyperdynamic therapy was begun with the addition of dobutamine, in an effort to increase cardiac index to >3.5 l/m. Phenylephrine was continued to maintain a mean arterial pressure >120 torr. Urine output was monitored to maintain an hourly output of 200 ml/hr.

13. What is hypervolemic therapy? What is "Triple-H" therapy? What are the end-points to therapy?

Despite these measures, the patient's condition failed to significantly improve. On hospital day #4 the patient returned to the radiology suite for balloon angioplasty and intra-arterial papaverine. After successful intervention, the patient returned to the ICU, serial neuro checks and TCDs recorded the patient's progress. The following morning the patient showed signs of improvement. The patient would raise two fingers to command. TCDs were less than 100 m/s. Triple-H therapy was continued for the next two days. Hospital day #7, the infusions were weaned off, hypervolemic therapy was continued and weaning from the ventilator resumed. Hospital day #9, the EVD was clamped, monitored and eventually discontinued.

14. What are the non-neurological morbidities associated with SAH, i.e. cardiac, pulmonary and other organ systems?

The patient continued to slowly recover. Nutritional needs were replaced via an enteral feeding tube and continued until the patient successfully passed a swallow test. Physical and occupational therapy proceeded to assist the patient. The patient was dismissed to a rehabilitation facility three weeks after initial presentation.

PROBLEM BASED LEARNING DISCUSSION

Model Discussion

Subarachnoid hemorrhage is not an uncommon, yet often a devastating neurological insult. Approximately 1:10,000 Americans are affected annually, resulting in a national occurrence of approximately 30,000 patients. 12% of patients die prior to receiving medical attention, 40% of those who receive care die within one month. Overall, 65% of patients succumb or are severely disabled. Less than a third of patients return to their premorbid status.

Risk factors that have been identified with SAH include history of SAH in first degree relatives, hypertension, tobacco use and moderate alcohol consumption. Aneurysms are generally confined to the Circle of Willis (95%), anterior communicating artery (40%), internal carotid artery (30%), middle cerebral artery (20%)and in multiple locations (20%).
The grading system of SAH has evolved over time. Hunt and Hess initially graded the severity of SAH based on clinical exam, and their grading system has been modified (Fig 1). The Glasgow Coma Score is an established assessment of neurological status but not specific for SAH (Fig 2).

The World Federation of Neurological Surgeons introduced a grading system that incorporates features of the GCS as well as the presence or absence of motor deficits (Fig 3).

Misdiagnosis of symptomatic cerebral aneurysms has been reported to be as high as 25% and is associated with disastrous consequences. SAH will often rebleed, and those patients suffer worse outcomes. Risk of rebleeding in the first 24 hours is 6-8%, with 1%-1.5% per day for the initial 14 days resulting in a 25% risk at 2 weeks, and 50% risk within one month. Overall mortality with rebleeding is estimated at 70%. If a patient is suspected of SAH, a non-contrast head CT must be obtained. If negative and clinical impression remains SAH, then a lumbar puncture should be performed and examined for xanthchromia. Cerebral angiography remains the definitive test for the diagnosis of cerebral aneurysms. Many patients (between 15-40%) have warning headaches ("sentinel bleed"), occurring days to weeks before the index episode of bleeding. This is thought to be due to a limited leakage of blood from an aneurysm.

The initial aim of SAH therapy is immediate stabilization and accurate evaluation of the patient. Overall the process should encompass five goals: 1) Treat the primary brain injury and its associated physiological derangements. 2) Prevent immediate, early neurological complications; 3) Prevent and treat delayed ischemic neurological deficits (DIND) and 4) Treat the aneurysm in a definitive manner and 5) Prevent and treat other non-neurological comorbidities..

Despite numerous advances, outcomes and prognosis remains disappointing. Presenting with higher grades of injury (WFNS = IV,V) is associated with the worst prognosis. Historically a debate existed regarding early surgical intervention versus delayed, but recent studies have confirmed the advantages of early definitive aneurysm therapy, particularly in reducing vasospasm, delayed ischemic events and hydrocephalus.

The International Subarachnoid Aneurysm Trial (ISAT) was a large, multicentered, randomized, prospective trial comparing surgical clipping versus endovascular coiling in ruptured aneurysms. It revealed a modest decrease in morbidity in the coiled patient group. The one-year risk of dependence or death was reduced by an absolute risk reduction of 6.9% in the coil group. In today's practice the two approaches may compliment one another. Surgical clipping of basilar aneurysms is associated with high morbidity rates, whereas endovascular coiling at this site is rather straightforward. Likewise, endovascular coiling of aneurysms in the MCA distribution are technically difficult, but more readily amenable to surgical clipping.

Early neurological complications of SAH include rebleeding as previously mentioned, but also acute hydrocephalus (25%), seizure (10-20%), intracerebral hematoma (4.5%) and cerebral ischemia due to mass effect.

Generally patients do not require endotracheal intubation and mechanical ventilation until GCS falls below 8. This is an arbitrary designation and the intensivist's impression of a rapidly deteriorating clinical status, associated neurogenic pulmonary edema or as in this case, transporting a patient to a distant hospital location, may prompt earlier intubation. Usual preparation and procedure are followed for intubation. Titrating induction agents to minimize hypotension is desired. Avoiding ketamine is recommended in neuro patients. Propofol, sodium pentothal and etomidate are acceptable agents. Decision on choice of muscle relaxant is balanced between concerns of increased intracranial pressure versus risk of aspiration. Ventilator modes

such as assist controlled, synchronized intermittent mandatory ventilation (SIMV) and pressure support are acceptable choices. Usual precautions against increased peak airway pressures, auto-PEEP and patient-ventilator dyssynchrony need to be taken. Weaning will likely depend on the course of the patient's illness. Beyond the usual criteria, other factors such as "ability to protect the airway" must be considered before extubation. We have found that SIMV with pressure support is a satisfactory approach in a majority of the patients.

Commencing around day #4, patients may develop late complications of SAH, specifically vasospasm and delayed ischemic deficits, stroke, hydrocephalus, cerebral edema and death. Vasospasm, although unclear in etiology, is thought to be associated with the breakdown of blood from the SAH and perhaps some unidentified metabolic product of oxyhemoglobin. Clinically, this produces pathological narrowing of the cerebral arteries, which in turn result in further ischemic injury to the brain. It is a self-limited process, beginning around day #4 and lasting approximately two weeks. Complications can be significant and further stroke or death may affect up to 1/3 of the patients. Monitoring vasospasm is achieved via frequent neuro checks by trained personnel. Transcranial Doppler (TCD) is a bedside device that allows determination of flow velocities in cerebral arteries. It may be technically difficult and windows may be small and challenging to find. For all practical purposes, the best windows are obtained for measuring velocities of the vessels in the MCA distribution. Therefore it may have little benefit in assessing vasospasm in other distributions of the brain, i.e. posterior basilar aneurysms. TCD recording of 120 meter/sec in the MCA window is associated with a 50% narrowing of the vessel. A recent study demonstrated that velocities slower than 120 m/sec and greater than 200 m/sec have strong negative and positive predictors for determining which patients will develop ischemic deficits.

Integral to treating SAH and in an effort to prevent the development of delayed ischemic deficits and vasospasm, the standard of care is nimodipine 60 mg orally every 4 hours. Studies and meta-analysis have supported its therapeutic utility. On angiography it does not affect vessel narrowing, so its benefit is of undetermined etiology.

Hyponatremia in the setting of SAH is not uncommon and the intensivist must determine its etiology. Historically this was thought to be the syndrome of inappropriate antidiuretic hormone (SIADH). It is recognized that with SAH, a catecholamine surge occurs along with the release of vasopressin. Patients who developed hyponatremia were presumed to have SIADH and early practitioners treated the hyponatremia with fluid restriction. However, following SIADH patients are not euvolemic and unfortunately the early practice of fluid restriction only worsened neurological outcome. In the setting of SAH, the kidney fails to resorb sodium in the distal tubules and thus "dumps" excessive sodium, hence the term "cerebral salt wasting". The treatment is opposite of SIADH, patients are treated with intravenous saline, and often hypertonic saline.

The mainstay for management of SAH in an effort to prophylaxis against developing delayed ischemic neurological deficits (DIND) is to maintain patients in at least an euvolemic to hypervolemic state in terms of intravascular volume status. Figure 4 and 5 are algorithmic approaches to treating patients with SAH who are either asymptomatic or symptomatic. Despite a Cochrane database systematic review which concluded there was no strong support for hypervolemic therapy to improve outcomes following SAH, this remains a primary practice in neuro intensive care units. The therapy of "Triple-H" is a goal directed therapy to produce

hypervolemia, hyperdynamic cardiovascular performance and hemodilution (HHH) in effort to correct symptomatic vasospasm. This therapy has not been formally tested in a prospective randomized trial.

Additionally, SAH is not confined to the brain. Other non-neurological morbidities are associated with SAH. The intensivist needs to be attentive to a patient's cardiac and pulmonary status. A myriad of electrocardiogram changes can be seen, but classically large T wave inversions have been described ("cerebral T waves"). Neurogenic etiology of cardiac failure, myocardial injury and infarction have been reported. The patient's pulmonary status may be jeopardized and patients may develop non-cardiogenic, neurogenic pulmonary edema. These patients may need to be intubated, ventilated and supported with positive end-expiratory pressure. GI ulcer prophylaxis should be employed, as well as prophylaxis against deep vein thrombosis.

In summary, SAH is a profound, sudden neurological insult with often devastating consequences. The intensivist needs to be aware of the parameters that SAH encompasses not only on a single organ, i.e. the brain, but impacts on multiple organ systems. The approach to patient care is multidisciplinary, involving critical care, neurosurgery, neuroradiology, neurology, and perhaps several internal medicine subspecialties. The goals for managing SAH remain: 1) Treat the primary brain injury and its associated physiological derangements. 2) Prevent immediate, early neurological complications; 3) Prevent and treat delayed ischemic neurological deficits; 4) Treat the aneurysm in a definitive manner and 5) Prevent and treat other non-neurological comorbidities.

References

Wijdicks EF. Kallmes DF. Manno EM. Fulgham JR. Piepgras DG., Subarachnoid hemorrhage: neurointensive care and aneurysm repair. Mayo Clinic Proceedings. 80(4):550-9, 2005 Apr.

Manno EM., Subarachnoid hemorrhage. Neurologic Clinics. 22(2):347-66, 2004

Molyneux A, Kerr R, Straton I, et al., International Subarachnoid Aneurysm Trial (ISAT) Collaborative Group. International Subarachnoid Aneurysm Trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: a randomized trail. Lancet 2002;360:1267-1274.

Rinkel GJ. Feigin VL. Algra A. van Gijn J. Circulatory volume expansion therapy for aneurysmal subarachnoid haemorrhage. Cochrane Database of Systematic Reviews. (4):CD000483, 2004.

Berkow LC, Mirski M, Kirsch JR, Subarachnoid Hemorrhage and Cerebrovascular Accident, in Critical Care Medicine - Perioperative Management, 2nd ed. Ed: Murray MJ et al. Lippincott Williams & Wilkins (Philadelphia), 2002. pp264-74.

SELECTIVE REFERENCES

Wijdicks EF. Kallmes DF. Manno EM. Fulgham JR. Piepgras DG., Subarachnoid hemorrhage: neurointensive care and aneurysm repair. Mayo Clinic Proceedings. 80(4):550-9, 2005 Apr.

Manno EM., Subarachnoid hemorrhage. Neurologic Clinics. 22(2):347-66, 2004

Molyneux A, Kerr R, Straton I, et al., International Subarachnoid Aneurysm Trial (ISAT) Collaborative Group. International Subarachnoid Aneurysm Trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: a randomized trail. Lancet 2002;360:1267-1274.

Fig 1 Modified Hunt-Hess Grading Scale

Grade	Clinical Findings	Hospital Mortality 1968 (%)	Hospital Mortality 1997 (%)
I	Asymptomatic or mild headache	11	1
II	Moderate to Severe headache or oculomotor palsy	26	5
III	Confused, drowsy, or mild focal signs	37	19
IV	Stupor (localizes to pain)	71	42
V	Coma (posturing or no motor response to pain)	35	18

Modified from Tamargo et al. New Horiz 1997, 5:364-375

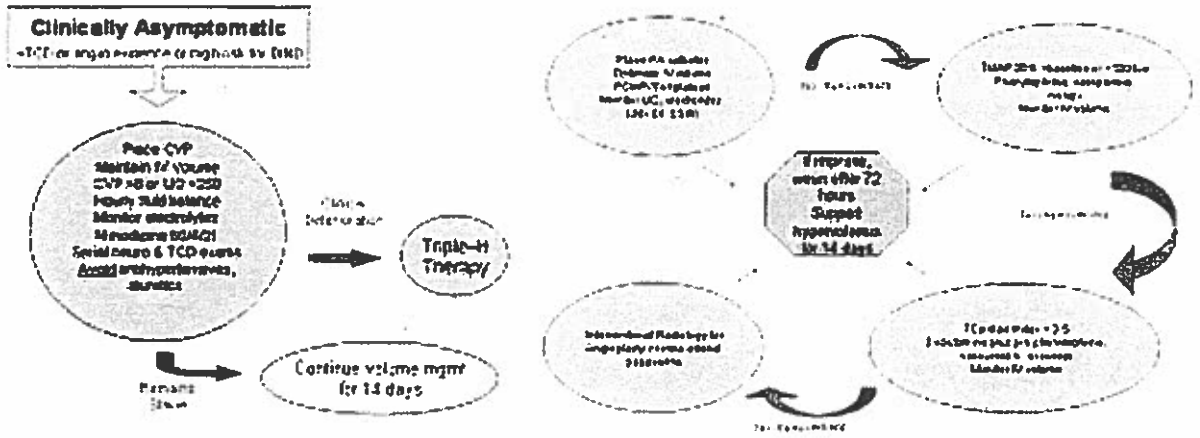
Fig 2. Glasgow Coma Score

Item	Response	Score
Verbal Response	Oriented	5
	Confused	4
	Inappropriate words	3
	Incomprehensible	2
	None	1
Eye Openning	spontaneous	4

	to speech	3
	to pain	2
	None	1
Motor	Obeys Commands	6
	Localizes Pain	5
	Withdraws	4
	Abnormal Flexion	3
	Abnormal extension	2
	None	1

Fig 3. World Federation of Neurological Surgeons (WFNS) Grading Scale

Grade	Glasgow Coma Score	Motor Deficit
I	15	Absent
II	13 or 14	Absent
III	13 or 14	Present
IV	7-12	Present or Absent
V	3-6	Present or Absent



LEARNING SUMMARY

Subarachnoid hemorrhage is not an uncommon, yet devastating neurological event. Inprovement in outcomes are largely dependent on a multidiciplinary approach with the critical care intensivist coordinating the patient's care and directing therapy. This session focuses on the presentation of SAH, its critical care management, and therapuetic approaches.

Anesthetic Challenges in Seizure Surgery and the Fancy Devices Used for Seizure Control

Ramachandran Ramani, M.B.B.S., M.D. Easton, Connecticut

OBJECTIVES

1) Seizure surgery: Participants will get a brief overview of seizure surgery and its anesthetic implications
2) Anesthetic considerations in "awake" craniotomy – seizure surgery is one of the indications for awake craniotomy. A detailed review of "awake" craniotomy and its anesthetic management will be presented in discussion format.
3) Functional Localization: Considering the critical importance of functional localization in seizure surgery, this will be one of the focuses of the PBLD.
4) Vagus nerve stimulator: Vagus nerve stimulation therapy will be discussed (with focus on what an anesthesiologist should be aware of) as more than 32,000 devices have been implanted worldwide (Cyberonics website). A brief description of "neuropace" also will be included.

STEM CASE - KEY QUESTIONS

A 29 years old male with history of uncontrolled seizures (partial complex) since the age of 3 years is scheduled for a resection of seizure focus (left side partial hippocampectomy). Surgeon is debating between doing it awake or under general anesthesia, because of the location of the gliotic focus close to speech area (Broca's area). Patient gives a history of seizures once every alternate day. Following the seizure he has difficulty in talking for a few hours and then complete neurological recovery after that.

Medications: Patient is on Dilantin and lamotrigine. Plasma level of both drugs is therapeutic.

Past medical history: Except for the fact that he has frequent absent attacks, falls down and gets hurt, there is nothing significant in the past history.
Past surgical history: No surgery in the past. Patient had a MRI (magnetic resonance imaging) and WADA test (named after Dr. Juhn Wada – a neurologist) under MAC (monitored anesthesia care).

Labs: hematocrit, electrolytes are within normal limits.
MRI: MRI shows a gliotic lesion 7 mm in size in the left hippocampus.
WADA Test: Wada test was done to check which side is dominant for memory. On injecting Amylobarbitol in the left middle CBL artery patient had no memory impairment.
Besides this patient also had a phase I and phase II work up for epilepsy. As a part of phase III he was advised resection of the gliotic focus.

Physical: Patient weighs 80kgs and is 71 inches tall. Vital signs were normal. Cardiovascular and chest exam also was normal. Airway: Mallampatti 2, good mouth opening, thyromental distance of more than 6 cms and no restriction of neck movements.

Patient also has been told that he has a reasonably good chance of getting seizure control with the surgery. However in the event of recurrence of seizures later in life the only available FDA

approved device is a vagus nerve stimulator (VNS) therapy. However he was told not to worry about the VNS now as there is a 90% chance that he will be seizure free after this surgery.

Key Questions:
1) Epilepsy: What is a medically resistant seizure?
2) What is the magnitude of this problem?
3) How effective is the response to medical treatment in a newly diagnosed case of seizures?
4) What are the treatment options available in such cases?3

Anesthetic challenges in Seizure surgery:
1) How do seizure medications influence anesthetic management?
2) Surgeon informs us that he is going to be using the neuro navigation system. Does it influence our management in any way?
3) Could the patient have an intraoperative seizure? How do we manage an intraoperative seizure?
4) How important is brain relaxation in a case where neuro navigation is being used for locating a 7 mm lesion?
5) At the end of the case, should we wake up this patient or leave him on ventilator?

Awake Craniotomy:
1) What is the indication for awake craniotomy in this case?
2) If this is to be done awake how do we manage the asleep – awake – asleep anesthesia?
3) Surgeon complains that brain is bulging while doing the craniotomy – how do we manage?
4) Any specific precautions during the case (antiemetics, analgesics) ?

Functional Localization:
1) How important is functional localization and why?
2) What is a Wada test?
3) What are the other techniques for preoperative functional localization?
4) How is intraoperative functional mapping carried out in awake craniotomy?

Vagus Nerve Stimulator:
1) What is a Vagus nerve stimulator?
2) How does it work?
3) How is it connected?
4) How effective is it? What are the side effects?

PROBLEM BASED LEARNING DISCUSSION

Model Discussion:

1) Epilepsy:A While the experts differ in what is a medically refractory seizure, one of the accepted definitions is – more than 6 seizures in a month and inter seizure interval of not more than 2-3 weeks. Epilepsy is the second most common neurological disorder. The incidence of seizure in the US population is in the range of 0.5 to 2%. This translates to 2 million patients with seizure. Of these one third of them are refractory to medical treatment and half of those with refractory seizures have partial onset seizure. Patients with partial onset seizures (~ 300,000) are the one's who can be worked up for possible surgical therapy.

Patients with generalized seizure are not candidates for surgery. Only 10-30% of these patients with partial onset seizures (30,000 to 100,000) are candidates for surgery. The treatment options available are focal resection of gliosis, partial temporal lobectomy, subpial disconnection etc.

2) Anesthetic challenges in seizure surgery:
Medications: These patients are on antiseizure medications for a longtime which can influence our anesthetic management. Phenytoin augments the liver enzymes resulting in enhanced metabolism of several drugs including muscle relaxants. Lamotrigine and Carbamazepine can cause hyponatremia which could by itself cause a seizure. Carbamazepine and sodium valproate can cause liver dysfunction and LFT needs to be monitored periodically.

Multiple surgeries: These patients come for several surgeries and sometime within a short period of time. These surgeries include grid / strip placement, removal of grids, resection of focus etc. Blood loss is one of the issues. In patients who have had a temporal craniotomy in the past because of sclerosis of the temporalis muscle, mouth opening could be inadequate causing difficulty in laryngoscopy during subsequent surgeries.

Associated medical problems like mental retardation, cerebral palsy etc.

Neuronavigation: Precise localization of the lesion is and has always been of paramount importance in neurosurgery. Neuronavigation systems have greatly enhanced the precision of localizing pathological lesions in the brain. These imaging systems (like brain lab) give us a reconstructed tomographic image of the brain in relation to the patients' anatomical landmarks on the scalp. Since it is not a real time image it is imperative that once the head is fixed on the Mayfield clamps there should be no movement of the head. Any movement (after the initial registration) can alter the landmarks. This is important from the point of view of brain relaxation also. While brain relaxation is always important in intracranial approach, undue relaxation as a result of hyperventilation, mannitol etc, alter the relationship between the brain lab reconstructed picture (based on preoperative MRI) and the realtime image during surgery.

Emergence: Temporal lobe is the location for majority of the partial seizures. Hence these surgical resections are very close to speech area, motor cortex, memory etc. Hence post operative neurological exam is important, in order to confirm these functions are intact following the surgery. Anesthetic medications should be titrated to ensure that patient wakes up awake and alert, able to follow commands.

Awake Craniotomy:
Indications: Awake craniotomy is indicated when the neurological lesion being operated upon is very close to important functional regions of the brain like motor cortex and speech area (Broca's – motor or Wernicke's – sensory area). In order to map the location of these regions during surgery and define its relationship to the lesion being operated, patient has to be awake during part of the surgery (described in the next section).
Asleep-awake-asleep procedure:1, 2 It is critical to position the patient properly when he is awake. Once the IV and monitors are in place patient is sedated and local infiltration is carried

out. Craniotomy is done with the patient sedated. Once the dura is open, patient is allowed to wake up for the mapping. Once the mapping is carried out and decision about the region to be resected has been taken, patient is sedated again. The challenge during sedation is three fold – adequate sedation and analgesia (combination of local infiltration and narcotics), maintaining the airway and ventilation and at the same time ensure an immobile patient. Short acting agents like propofol and remifentanil are preferred.C In a spontaneously breathing subject, as low as 0.05µg/kg/minute of remifentanil can cause apnea. Nasal trumpet can be used for airway patency. Some centers use LMA (laryngeal mask airway) also. Sedation should be titrated on and off slowly.

Intraoperative seizure can occur during an awake craniotomy. It is more likely during the stage of cortical activation. Hence it is important to check prior to surgery the plasma level of the antiseizure medications and administer additional doses if required. In spite of this, if a patient does get a seizure, management protocol would be like any other seizure. It is preferable to use short acting drugs because of the transient nature of the cortical stimulation.

Prophylactic antiemetic therapy is advisable to prevent any gagging (and rise in ICP) during the procedure.

Discussion pertaining to navigation and brain relaxation hold good for awake craniotomies as well.

Functional Localization:4 Various localization procedures are available which could be used either preoperatively and could be used intraoperatively also. As the predictive accuracy of the preop procedures improve it is predicted that in the future we might be able to rely on these procedures entirely and eliminate the intraoperative mapping procedure, as is commonly done during awake craniotomies. The techniques commonly used are:

1) Wada test: This test is done preoperatively with the patient awake. Left middle cerebral artery is selectively cannulated and 5mg of amylobarbitone is injected to suppress the function of the hippocampus on the ipsilateral side and a memory check is carried out. This determines which side memory function is localized in the patient.
2) Functional MRI and PET (positron emission tomogram) scan: With functional MRI and PET scan, the change in metabolic activity during a functional activity (like motor movement, speech, memory activation etc) can be imaged. When this image is overlaid on the structural MR image, the location of the pathological lesion relative to the functional regions of interest can be accurately assessed.
3) Intraoperative mapping as described earlier. This could be combined with electrocorticogram also.

Vagus nerve therapy (VNS):B,5

VNS was approved by FDA in 1997, as an adjunct therapy for patients over 12 years of age with partial onset seizures. Over 32000 devices have been implanted worldwide. The electrodes are implanted over the mid cervical portion of the left vagus nerve, while the device is placed in the infra clavicular region (like a pacemaker). The device works by stimulating the locus coeruleus (afferent pathway of the vagus nerve) and inducing release of norepinephrine. The efficacy of the

VNS is defined based on the 50% decrease in seizure frequency. Efficacy increases over the years. As per the VNS registry more 50% patients (out of 2229 patients) had a 50% reduction in frequency at 12 months. This increased to 59% at 24 months. In a 12 year follow up 60% patients responded to VNS. Transient side effects are reported like hoarseness, constriction in the pharynx, paresthesia, dyspnea and cough in the first year. Subsequently they tend to resolve. In addition to decrease in seizure frequency, patients are less depressed and more active mentally. This improvement in depression is out of proportion to the decrease in seizure frequency.

The next step in seizure control is use of "Neuropace". This device is described by many as the AICD equivalent for the CNS. Electrodes are directly implanted over the seizure focus. The generator is located on the scalp (in place of a piece of bone flap). In response to seizure activity the generator gives a counter stimulus to neutralize the seizure activity. This device is at present undergoing a phase II trial.

REFERENCES

1) Vitaz TD. Comparison of conscious sedation and general anesthesia for motor mapping and resection of tumors near motor cortex. Neurosurgical Focus 2003; 15: E8
2) Huncke. The asleep-awake-asleep technique for intra operative language mapping. Neurosurgery 1998; 42: 1312-1317
3) McKhann. Novel surgical treatment for epilepsy. Current neurology neuroscience reports 2004; 4: 335-339
4) Majos. Cortical mapping by functional MRI in patients with brain tumors. European Radiology 2005; 15: 1148-1158
5) Groves. Vagus nerve stimulation-a review of its applications and potential mechanism that mediates its effects. Neuroscience and Behavioral Review 2005; 29: 493-500.

SELECTIVE REFERENCES

1) Cascino DG: Surgical treatment for epilepsy. Epilepsy Research 2004; 60: 179-186
2) Ben Menachem: Vagus nerve stimulation for treatment of epilepsy. Lancet Neurology 2002; 1: 477-482
3) Keifer John C: A retrospective analysis of remifentanil / propofol anesthesia for craniotomy before awake functional brain mapping. Anesthesia Analgesia 2005; 101: 502-508.

LEARNING SUMMARY

Participants will get a brief overview of surgical therapy for seizure followed by a discussion on the anesthetic issues in seizure surgery and awake cranitomy. Various localizations techniques in use (considering that this is fast becoming a routine practice in all cranitomies) will be discussed. Finally a brief review of vagus nerve stimulation therapy will be presented.

Acute Stroke Following Cardiac Catheterizaiton

Michelle Lotto, MD

Cleveland, OH

OBJECTIVES

Be aware of endovascular therapy for stroke and the emerging role of the anesthesiologist in caring for these patients during intra-arterial thrombolysis.

Understand the mechanisms of ischemic brain injury, and develop an approach to caring for the acute stroke patient including avoidance of potential secondary injury.

Understand the benefits, risks and limitations of new therapies for treatment of acute stroke

Prepare for the treatment of complications following lytic therapy in stroke.

STEM CASE - KEY QUESTIONS

You are called on a Saturday night by the neuroradiologist to provide anesthesia for arterial thrombolysis in an acute stroke. The patient is an 80 year old gentleman transferred by med flight from a local hospital for left sided weakness and left facial drop that began during a cardiac catheterization today. The patient has an expressive aphasia, but can answer questions yes and no by shaking his head. The onset of symptoms occurred 3 hours earlier. The patient's neurologic examination has not changed since that time. The cardiologist has left the catheter in place.

The emergency room records indicate a past medical history of CVA, HTN, and CHF. The patient is unable to elaborate on his history due to his aphasia.

Med List from the transfer notes:

Digoxin 125 mcg

Insulin

Heparin IV infusion

Famotidine

Atorvastatin

VS: BP: 178/99 HR: 88 RR:16 SaO2: 97% 2L NC T: 36.8.

Questions:

What is the urgency of this case?

What specific preoperative lab tests or additional information would you require for management of this patient?

What are the current indications and time window for use of intra-arterial t-PA in patients with acute stroke, for IV thrombolysis?

Does this patient's recent heart cath contraindicate thrombolytic therapy?

Would you treat the patient's blood pressure, if so what medications would you use?

Labs obtained in the ED show:

Na+ 135 K+5.2 Cl 103 HCO3 21, BUN 30 Cr 1.4 Glucose 232

WBC 10.01 HGB 13.5 HCT 41.6 PLT 200 INR 1.1 APTT 32.6

EKG: Shows a sinus rhythm with a rate of 75 BPM with a left bundle branch block morphology.

CT: reveals diffuse atrophy, it is negative for hemorrhagic conversion of the stroke.
The neuroradiologist is pacing back and forth and would like to proceed immediately to the neuroendovascular suite.

Would the laboratory evidence change your anesthetic plan or cause any concerns?

What would be your approach management in this patient, what type of anesthetic would you choose?

What monitors would you require for the case?

The patient is transferred to the neuroendovascular suite. An angiogram reveals thrombotic occlusion of the right MCA artery. A catheter is placed past the thrombus and Tissue plasminogen activator is infused. At the end of the procedure the vessel shows 60% recanalization. The patient has remained stable throughout the procedure. The blood pressure is 165/88. The neuroradiologist is requesting the pressure be lowered.

What would be a tolerable range for post-procedural blood pressures in this patient?

What agents would you use to treat the blood pressure and why?

What would you plan for disposition for this patient, the neurosurgical unit or the cardiac care unit?

What are the pros and cons of postoperative intubation or extubation of this patient?

Will you extubate the patient in the radiology suite or the intensive care unit?

Postoperatively the neurologic examination demonstrates the patient to be following commands. The left leg weakness is improved and left hand grip is only mildly improved. The next morning the patient becomes lethargic and unresponsive.

What is the differential for the patient's change in mental status?

Emergent CT scan demonstrates a large parietal lobe hemorrhage. Despite discussions of a poor prognosis for functional recovery, the family wants everything done, including surgery.

Are there ethical concerns that would affect the decision to proceed with surgery or not to proceed with this patient?

If surgery is chosen how would you prepare for this case?

PROBLEM BASED LEARNING DISCUSSION

Thrombolytic Therapy and Stroke

Prior to the implementation of lytic therapy for ischemic stroke, the treatment of stroke was limited to supportive care and expectant management. Investigation of thrombotic therapy for the treatment of ischemic stroke began in the early to mid 90's, and has shown promise in multiple prospective randomized trials. The goal of therapy is early restoration of blood flow to the ischemic brain tissue to minimize cellular metabolic events that will eventually lead to cell death and apoptosis. Time to therapy is a critical factor for preservation of brain function, therefore initiation of treatment should be considered emergent. Success of treatment with thrombolysis is dependent upon the route of delivery, agent used for thrombolysis, and the time to treatment from onset. Currently the only FDA approved lytic stroke therapy is tissue plasminogen activator (tPA) administered intravenously within 3 hours of symptom onset. Meta-analysis of studies using IV tPA within 3 hours of symptom onset suggest 140 more patients with independent survival per 1000 treated. The risk of death (22%) is not significantly different from the placebo treated group. Another large prospective trial (National Institute of Neurologic Disorders and Stroke NINDS) demonstrated 11 to 13% improvement in excellent outcomes in tPA treated patients over placebo controls. Evidence does not seem to support use of IV thrombolysis at >3 to 6 hours.

Recommended Inclusion criteria for Intravenous tPA

Age > 18
Clinical DX of stroke with neurologic deficits
Clear onset <180 Minutes before treatment
CT scan demonstrating no intracranial hemorrhage

Recommended Exclusion Criteria for Intravenous tPA

Rapidly Improving symptoms
Major surgery within past 2 weeks
Head injury within 3 weeks
GI tract injury within 3 weeks
Lumbar puncture within 1 week
SBP>185mmHG DBP>100mmHg
Platelet count <100,000

Current use of oral anticoagulants INR>1.7
Pregnant or lactating women

Endovascular Therapy for Stroke
Intra-arterial (IA) administration of thrombolytics using angiography and endovascular catheters to access the thrombosed artery have higher rates of target vessel canalization. Randomized trials have demonstrated improved recannulation rate over controls (66% vs 18%) when treatment was initialized within 6 hours of symptom onset. The PROACT II trial randomized patients with acute stroke of the middle cerebral artery (MCA) territory to receive IA prourokinase (pro-UK) + IV heparin vs IV heparin alone. The primary endpoint was the percentage of patients achieving independent survival at 90 days. The final analysis demonstrated 40% of the intra-arterial lysis patients vs 25% of the controls achieved survival with minimal or no deficit. Although, 90 day mortality was similar in both groups at 25% for pro-UK patients and 27% for controls, symptomatic intracranial hemorrhage was significantly greater in the pro-UK treated patients (10% vs 2% of controls.) Intra- arterial thrombolysis is not yet FDA approved in the United States, but is supported by the AANS as a beneficial treatment for acute large vessel stroke.

The use of IA thrombolysis for postoperative stroke has been reported in a small series of patients. The risk of bleeding from the surgical site was 25% with 8% having fatal bleeding (2 after craniotomy.)

Brain Ischemia
Although neuronal cells will die within minutes of loss of perfusion, areas around the stroke foci may experience a more tolerable level of ischemia, and may not convert to infarction if flow is restored within a critical time period. Experiments in primates have shown that ischemic tolerance of the brain is dependent upon both the severity of blood flow reduction and the duration of ischemia. The Normal blood flow to the brain is 50 ml/100mg/min When cerebral blood flow is decreased to 20ml/100g/min loss of consciousness and a slowing of the EEG is seen, however, no permanent structural consequences occur. This level of ischemia may be reversible even after 3 hours of ischemic time. As cerebral flow decreases to 18 ml/100g/minute metabolic activities are jeopardized and ischemic tolerance time is reduced. At 10 ml/100g/minute the integrity of cellular function is lost and the ischemic tolerance time is on the order of minutes.

Managing the Acute Stroke Patient

The goals of management of the patient with brain ischemia include maintaining cerebral perfusion pressure and avoiding mechanisms of secondary injury that can worsen outcomes such as hypoxia, fever and hyperglycemia.

<u>Hemodynamic management</u>
Hypertension is a very common finding in acute thrombotic stroke, and is thought to be a reactive mechanism to improve perfusion to the ischemic areas of the brain. The autoregulation of the ischemic brain has been demonstrated to be impaired, and decreases in systolic blood pressure can further compromise the ischemic penumbra and convert viable brain to stroke. Traditional recommendations for treatment of hypertension in stroke suggest no intervention should be initiated unless the DBP>120 or the SBP>220. However, analysis of the patients with

intracranial hemorrhage following IV TPA suggests a significant increase in the risk of intracranial hemorrhage when SBP exceeds 185 or DBP exceeds 110. Eligibility for any type of thrombolytic therapy requires treatment of hypertension to maintain SBP<185 and DBP<110. The American stroke council currently recommends treatment with labetalol or nitro paste.

Glucose Management

Hyperglycemia has been demonstrated to increase ischemic stroke volume in animal models following occlusion of large cerebral vessel with subsequent reperfusion. Retrospective studies in human stroke victims have associated hyperglycemia with larger stroke volumes and worse outcomes, however, it is unknown whether hyperglycemia is a cause or a sequela of a large stroke. There are no compelling prospective human trials investigating the benefits of glycemic control in stroke patients. The GIST trial prospectively randomized acute stroke patients to insulin infusions vs saline for strict glucose control post ischemic stroke, to demonstrate feasibility of glucose control, although the study was not powered to evaluate outcomes, there was no difference in neurologic outcomes between the study and control group. Hyperglycemia has been associated with hemorrhage conversions of cerebral infarcts in the past. A recent analysis of intracranial hemorrhage in the PROCT study found a glucose >200 mg/dl to be a univariate predictor of intracranial hemorrhage following IA lysis.

Temperature Management

Fever has been associated with worse neurologic outcomes in the setting of acute stroke. Mild hypothermia has been demonstrated to protect against cerebral infarction during focal ischemia in animal models. Small clinical trials have demonstrated the feasibility of long term cooling of acute stroke patients with surface cooling and endovascular devices following thrombolysis. However, no human studies have been performed to evaluate the effect of hypothermia on neurologic outcomes following ischemic stroke.

Ventilation

Normocarbia should be the goal in patients with flow related ischemic stroke. The cerebral vessels in the ischemic region will be maximally vasodilated and will lose autoregulation due to the downstream occlusion in the vessel. Flow in the maximally dilated vessel becomes dependent upon the mean arterial pressure. Hypotension may convert ischemic penumbra into stroke. Changes in cerebrovascular tone related to changes in pCO_2 can also affect the regional blood flow. Hypoventilation and hypercarbia will lead to global cerebral vasodilation which may theoretically cause shunting of blood away from the maximally dilated, flow dependent vessels of the ischemic brain region. Hyperventilation will decrease global cerebral blood flow and may worsen cerebral perfusion deficits.

Intracranial Hemorrhage following IA Thrombolysis

Intracranial hemorrhage with associated neurological deterioration occurs in 10% of patients treated with IA thrombolysis. In the PROACT study, ICH occurred at a mean of 10 hours after initiation of thrombolytic therapy. The mortality for symptomatic ICH following IA lysis was 83%. Treatment of a patient with ICH following should include reversal of the TPA and neurosurgery consultation for possible craniotomy for removal of the hemorrhage mass. The American heart association recommends the administration of 6 to 8 units of platelets and 10 units of cryoprecipitate for reversal of the hemolytic effects of TPA.

REFERENCES

Bibliography

Jones TH, Moriwetz RB, Crowell RM et al. Thresholds of focal cerebral ischemia in awake monkeys. *J Neurosurg* 1981;54:773-782.

Bruno, A. Williams, L. Kent, T. How important is hyperglycemia during acute brain infarction? *Neurolog.* 2004; 10: 195-200.

Powers, W. Acute hypertension afer stroke: the scientific basis for treatment decisions. *Neurology* 1993;43:461-467.

Furlan, A. et al. Intra-arteral Prourokinase for acute ischemic Stroke. The PROACT II study. A Randomized Controlled Trial. JAMA 1999.282:2003-2011

The National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. Tissu plasminogen activator for acute ischemic stroke. *N Engl J Med* 1995;333:1581-1587

Kase. CS et al. Cerebral Hemorrhage after Intra-arterial thrombolysis for ischemic stroke. The PROACT II trial.

Yanamoto H, Nagata I, Niitsu Y, Zhang Z. et al. Prolonged Mild hypothermia therapy protects the brain against permanent focal ischemia. *Stroke.* 2001. 32:232-239

Krieger D, De Georgia M, Abou-Chebl A. et al. Cooling for Acute Ischemic Brain Damage (COOL AID). An Open Pilot Study of Induced Hypthermia in Acute Ischemic Stroke. Stroke.2001; 32:1847-1854.

Chalela J, Katzan I, Liebeskind D. Rasmussen P. et al. Safety of intra-arterial thrombolysis in the postoperative period. *Stroke* 2001;32:1365-

SELECTIVE REFERENCES

Readings

Adams, H. et al. Guidelines for the early management of Patients with Ischemic Stroke. A scientific Statement from the strokecouncil of the American Stroke Association. *Stroke.*2003. 34:1056-1083.

Albers, GW. Amarenco P. Easton DJ, Sacco R, and Teal P. Antithrombotic and Thrombolystic therapy for Ischemic Stroke. Chest. The seventh ACCP conference on antithrombotic and thrombolyitic therapy. Chest 2004;126: 483S-512S.

Kase. CS et al. Cerebral Hemorrhage after Intra-arterial thrombolysis for ischemic stroke. The PROACT II trial.

LEARNING SUMMARY

Be aware of endovascular therapy for stroke and the emerging role of the anesthesiologist in caring for these patients during intra-arterial thrombolysis.

Develop an approach to caring for the acute stroke patient including avoidance of potential secondary injury.

Prepare for the treatment of complications following lytic therapy in stroke.

Emergency Surgical Fixation of Unstable Cervical Spine

Ramesh Ramaiah, M.B., F.R.C.A., F.C.A.R.C.S.I. Seattle, Washington

OBJECTIVES

1) What is the range of motion in cervical spine in healthy adults normally and during laryngoscopy

2) What are stable and unstable cervical spine fractures?

3) Discuss the different options to secure airway in a patient with C-spine injury, including local anesthesia and sedation for awake fiberoptic intubation.

4) Discuss the diagnosis and management of intraoperative air embolism.

STEM CASE - KEY QUESTIONS

A 26 year old male is involved in a motor vehicle accident as unrestrained passenger in the front seat. He sustained left radius and ulna fracture with absent distal pulses and unstable compression fracture of C3 and C4 vertebrae with bone fragments impinging on the spinal cord. Surgeons sceduled him to OR for exploration of left forearm and posterior stabilization of the spine and instrumentation. He is seen in the ER by anesthesiology resident. His neck is in rigid C-collar, he is awake oriented and able to move all four limbs. He has no other medical back ground. He is a smoker and uses alcohol occassionally. His blood work is within normal limits.

What other injuries you want to rule out before surgery?

What is stable and unstable C-spine?

CT head, thorax, abdomen and pelvis are normal, other limb x-rays are also normal. His blood work is within normal range with hematocrit of 38. He has 18g and 16g IVs.

What options are available to secure airway? is awake fiberoptic intubation the only choice for securing airway in all unstable c-spines injuries?

Awake fiberoptic intubation was done to secure the air way under sedation.

What are the different methods that can be used to anesthetize upper airway?

What are the sedative drugs used for awake fiberoptic intubation, in what way dexmedetomidine is better sedative than conventional sedatives?

Following brief neurological assessment, which was unchanged anesthesia was induced with propofol and maintained with TIVA using propofol and remifentanyl infusions, which were titrated to maintain BIS around 40-55.

His left forearm was explored and ORIF of both ulna and radius was done with regaining vascular flow to the distal limb. Before patient was positioned prone, neurophysiologist connected SSEP and MEP monitors and base line recordings were obtained before incision.

What are the different methods to monitor spinal cord function and does SSEP and MEP monitoring impact your anesthetic plan?

Surgery was going well and half way through the surgery patient attempted to breathe and he was allowed to breathe spontaneously. About 45 minutes later, suddenly patient's heart rate dropped to 30/min along with drop in the end tidal CO2 to 15 mmHg, spo2 to 82% and BP to 60/30 mmHg. Surgeon was notified and requested to pack the wound and turn the patient supine immediately .

What is the differential diagnosis? How will you confirm or rule out your diagnosis?
What is your immediate management?
Could spontaneous breathing have contributed to this?

What is the volume of air that is lethal in patient with air embolism, is TEE most sensitive to diagnose air embolism?, what other methods are available to detect air embolism and how sensitive and specific are they?

After initial resuscitation patient was stable, he was transferred to ICU with a plan to bring him back to OR once stabilized. Following day the patient was stable, he was taken to OR for completion of the surgery.

What precautions will you take to prevent air embolism during the surgery?

PROBLEM BASED LEARNING DISCUSSION

This patient has been involved in a major trauma. Before taking him to surgery, he needs to have CT scan of the head, thorax, abdomen and pelvis to rule out any occult injuries. Cervical Spine Injury (CSI) occurs in up to 2% of all blunt trauma patients. The incidence increases to 10% in patients with significant head injury (GCS <8 or focal neurological deficit). Immobilization of the spine after injury is advocated as standard of care. A three view x-ray series supplemented with CT imaging is an effective imaging strategy to rule out CSI. Flexion-extension in the upper cervical spine occurs at both atlanto-occipital and atlanto-axial articulation and a combined 24° of motion may be achieved. A further 66° of flexion-extension may be achieved in the lower cervical spine with C5-C7 segments contributing the largest component. During laryngoscopy the primary force is an upward lift which can be as high as 50-70N. Greater force may be required in difficult airway. This results in extension of the occiput on C1 combined with flexion at lower vertebrae. Data collected in large series (cinefluroscopy) during laryngoscopy indicate that direct laryngoscopy with Mac 3 blade results in near maximal extension at atlanto-occipital

joint with flexion below C2-C3. There are only minimal differences with the use of straight or curved blades.

Spinal instability usually results from vertebral displacement. White and Panjabi concluded that a normal adult spine would not permit horizontal motion greater than 2.7mm between vertebrae. On x-ray if you see the horizontal displacement more than 3.5mm or 20% of the vertebral body width, it is abnormal and the spine is considered unstable. The upper limit of physiological angular displacement of a vertebral body compared with adjacent vertebrae is 11°. If there is a greater angulation demonstrated on imaging studies, the spine is considered unstable at the site of excessively rotated vertebrae. The National Emergency X-Radiography Utilization Study (NEXUS) group identified the following injuries as not clinically significant: spinous process fracture, wedge compression fractures with loss of 25% or less of body height, isolated avulsion fracture without ligament injury, type-1 odontoid fractures, end-plate fractures and isolated transverse fractures. When there is no radiographic evidence of CSI, the possibility of a neurologically significant cervical cord injury still exists. A special entity called spinal cord injury with out radiographic abnormality (SCIWORA) syndrome occurs when the elastic ligaments of the neck are stretched during trauma resulting in neurological injury. This syndrome may account for 70% of spinal cord injuries in children less than 8 years.

A patient about to undergo surgical reduction and fixation of a spinal column fracture poses a number of challenges for the anesthesiologists. First and foremost is the need for intubation of the trachea in a patient with CSI. The most appropriate airway management of the patient with CSI continues to be debated. All airway maneuvers will result in some degree of spine movement, however the available data and clinical experience support that these interventions are performed with reasonable care are unlikely to cause any neurological injuries. Direct laryngoscopy with manual in line immobilization is appropriate in the emergency settings and in the unconscious, combative or hypoxic patients, when the status of the spine is not known. The resulting neurologic outcome compare favorably to similar patient population undergoing awake intubation and patients who did not require any airway interventions after CSI. In the OR an awake, alert and cooperative patient can be intubated by a number of different methods known to produce less displacement of the cervical spine and presumably less risk of worsening an unstable CSI. The most common technique in current clinical practice is awake fiberoptic intubation. Recent survey indicates that majority of American anesthesiologists prefer awake fiberoptic intubation in at-risk patients. Blind nasal, transillumination with lighted stylet, the use of intubating LMA, or Bullard laryngoscope are all acceptable. There are no clinical out come data that suggest better neurologic out comes with any particular technique. The clinician is advised to use the equipment and technique that are most familiar.

Adequate airway anesthesia is important in order to be successful in intubating trachea. Several techniques are used in clinical practice; they include topically anesthetizing the airway by using sprays and direct application of local anesthetics to the respiratory mucosa, as well as a variety

of nerve blocks. Topical anesthesia can be achieved by commercially available aerosol cans of benzocaine and usually 3 applications to the oropharynx with duration of approximately 1 second each is sufficient, however its use is associated with the risk of methemoglobinemia. Lidocaine 4% is frequently sprayed on the mucosa using an atomizer device. 4 ml of 4% lidocaine can be used to anesthetize nasopharyngeal and laryngeal mucosa via nebulization. If nasal intubation is planned, use cotton tipped swabs soaked in either lidocaine or cocaine. The swabs are placed superiorly and posteriorly in the nasopharynx and left for several minutes to block the branches of ethmoidal and trigeminal nerves. The other method involves coating a nasopharyngeal airway with viscous lidocaine mixed with a vasoconstrictor and gently inserting the airway into the nares. For oral intubation, gargling of several mls of 4% lidocaine provides anesthesia to oral and pharyngeal tissues. The fiberoptic broncoscope itself can be used to apply local anesthetic to the target mucosa through the working channel of the scope.

Nerve blocks are also often used to provide anesthesia for awake fiberoptic intubation. Three nerve blocks are used to provide anesthesia to the upper airway; glassopharyngeal(oropharynx), superior laryngeal(larynx above the cords), and translaryngeal(larynx and trachea below the cords). Superior laryngeal nerve can be blocked useing topical application of local anesthetic in the pyriform fossa useing Jackson or Krause forceps.

Providing adequate sedation is important and advantageous in both anesthetizing the airway as well as during the intubation itself. Spontaneous ventilation with patient cooperation should be maintained through out the procedure. Benzodiazepines(commonly midazolam) provide sedation and amnesia and can help prevent seizure activity in the event of local anesthetic toxicity. However, they are more likely to produce unconsciousness and thus uncooperative patient. Opioids(commonly fentanyl) are also useful for providing analgesia and can help blunt airway reflexes, especially coughing. However, they do not provide amnesia and can cause significant respiratory depression. Case reports suggest that dexmedetomidine provides moderate level of sedation without causing respiratory depression or hemodynamic instability. Additionally it decreases salivary secretions, through sympatholytic and vagomimetic effects, which is desirable when fiberoptic intubation is performed. However, it does not provide amnesia and avoided or used with caution in patients with shock .

Spinal cord monitoring: During spine surgery the spinal cord is at risk of injury. Following surgery to correct scoliosis, the incidence of motor deficit or paraplegia with out spinal cord monitoring is between 3.7% to 6.9%. This figure may be reduced to 0.5% by intraoperative spinal cord monitoring. It is now considered mandatory to monitor spinal cord function where there is a significant risk of injury to the nervous system. Knowledge of the methods used to monitor intraoperative spinal cord function is essential to the anesthesiologist as the anesthetic techniques have profound effect on the ability to monitor spinal cord function. There are four main methods of intraoperative spinal cord function monitoring; Ankle clonus test, the stagnara wake-up test, SSEP, and MEP.

Ankle Clonus test; Historically, this was the first method to be used. This is usually performed during emergence, either at the end of surgery or during wake-up test. This test is easy to perform and has high level of sensitivity and specificity. It can only be performed intermittently and the absence of clonus could not only be due to spinal cord damage, but also due to an inadequate or deep level of anesthesia.

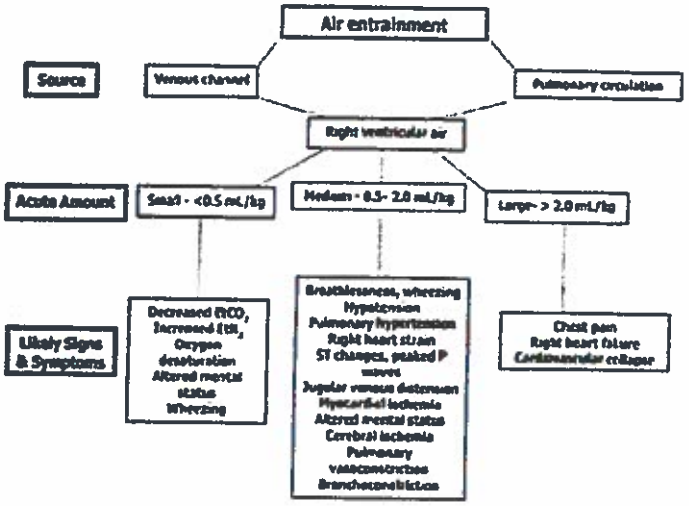
Stagnara wake-up test; This was first described in 1973. Preoperatively, the need for this test is explained to the patient and it will involve specific motor responses usually of lower limbs to verbal commands during surgery. It has number of disadvantages; it requires patient cooperation, it poses risk to the patient of moving or falling from the operating table, inadvertent tracheal extubation and dislodgement of vascular access.

SSEPs(Somatosensory Evoked Potential) are elicited by electrical stimulation of mixed nerves peripherally and recordings are made at a distant site cephalad to the level at which surgery is performed. The functional integrity of SSEP is determined by comparing change in the amplitude and latency obtained during surgery to the baseline values. A 50% reduction in amplitude and a 10% increase in the latency are generally considered significant. Inhalational agents and nitrous oxide cause a dose dependent reduction in amplitude and increase in latency. 60% Nitrous oxide with 0.5 MAC of isoflurane or 0.5% of enflurane is compatible with effective SSEP mointoring. TIVA has minimal effects on SSEP monitoring and the combination of propofol with remifentanyl or midazolam/low dose ketamine/narcotic has been successfully used. In a large retrospective multicenter study of over 51 000 procedures, SSEP monitoring was found to have sensitivity of 92% and specificity of 98.9% in detecting spinal cord integrity. The false negative rate was 1 in 787 procedures and false positive rate was 1 in 67 procedures.

MEP(Motor Evoked Potential); Following reports of post operative paralysis despite apparently normal intraoperative SSEPs, led to the efforts to monitor motor tract of the spinal cords using MEPs. Inhalational agents are powerful suppressant of cortical evoked MEPs. IV propofol with fentanyl or remifentanyl has been advocated and provides for adequate MEP recordings.

The clinical picture and laboratory evidence suggest that this patient had developed intraoperative pulmonary embolism. Embolism may result from intravasular gas (air, carbon dioxide or oxygen), thrombus, amniotic fluid, fat, bone marrow, aggregated blood products and a variety of foreign bodies(example cannula fragments). In this patient it was air embolism which was confirmed by TEE. Cervical surgical procedures are not commonly associated with air embolism but it is well known. Vascular air embolism (VAE) is the entrainment of air from the operative field or other communications with the environment into the venous or arterial system with the systemic manifestation (mainly pulmonary,cardiovascular and central nervous system). The true incidence of VAE may never be known, many of them are sub clinical with out any untoward effects and may go unreported. From the case reports of accidental intravascular delivery of air, the adult lethal volume has been described between 200 to 300 ml or 3-5 ml/kg.

Clinical presentation may include cardiovascular, pulmonary, and neurological sequelae, the spectrum of the adverse events dependent on rate and volume of air entrained and whether patient is spontaneously breathing, creating negative intrathoracic pressure facilitating of air entrainment or patient is on controlled positive pressure ventilation. Following figure illustrates clinical presentation of acute air embolism.



In table 1 relative risk of air embolism in commonly performed procedure and in table 2 comparing different methods of detecting air embolism are shown. Transesophageal echocardiography is the most sensitive monitoring device for detecting air embolism. It can detect as little as 0.02 ml/kg of air administered by bolus injection.

Table 1; Relative Risk of Air/Gas Embolism

Common Procedures	Relative Risk
Sitting position craniotomy	High
Posterior fossa/neck surgery	High
Laproscopic procedures	High
Total hip arthroplasty	High
Cesarean delivery	High
Central venous access-placement/removal	High
Craniosynostosis repair	High
Spinal fusion	Medium
Cervical laminectomy	Medium
Prostatectomy	Medium
Gastrointestinal endoscopy	Medium
Contrast radiography	Medium
Blood cell infusion	Medium
Coronary surgery	Medium
Peripheral nerve procedures	Low
Anterior neck surgery	Low
Burr hole neurosurgery	Low
Vaginal procedures	Low
Hepatic surgery	Low

Approximate expected reported incidences: high,>25%; medium, 5-25%, low < 5%

Table 2; Comparison of Methods of Detection of Vascular Air Embolism

Method of Detection	Sensitivity(ml/kg)	Availability	Invasiveness	Limitations
TEE	High(0.02)	Low	High	Experts required, expensive
Precordial Doppler	High(0.05)	Moderate	None	Obese patients
PA catheter	High(0.25)	Moderate	High	Fixed distance small orifice
TCD	High	Moderate	None	Expertise required
ET N2	Moderate(0.5)	Low	None	N2O, hypotension
ETCO2	Moderate(0.5)	Moderate	None	Pulmonary disease
Oxygen saturation	Low	High	None	Late changes
Direct visualization	Low	High	None	No physiologic data
Esophageal stethescope	Low(1.5)	High	Low	Late changes
Electrocardiogram	Low(1.25)	High	Low	Late changes

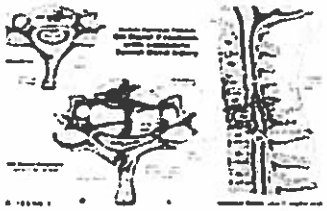
ETCO2=end-tidal carbon dioxide; ETN2= end-tidal nitrogen; N2O= ntrous oxide; PA = pulmonary catheter; TCD= transcranial doppler; TEE= transesophageal echo

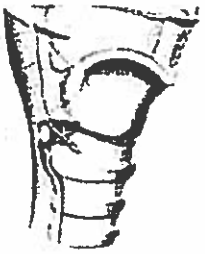
Management of air embolism; Newer monitoring devices have enabled us to diagnose and treat the air embolism early before the life threatening events occur. When there is high index of suspicion inform the surgeon, prevent further entrainment/infusion of air, flood the surgical field with saline/saline soaked dressings, institute 100% oxygen with hand ventilation, turn off nitrous oxide and volatile agent if hypotensive. It may be possible to relieve air-lock in the right heart by placing the patient in partial left lateral decubitus position(Durant maneuver), aspirate central venous line if already in situ. Do not hesitate to treat as a cardiac arrest- institute external cardiac compressions, iv epinephrine 0.001mg/kg bolus and if necessary start infusion at 0.00015mg/kg/min along with volume expansion with crystalloid 10ml/kg. Early hyperbaric oxygen has been show in case series to have beneficial effects. There have been benefits observed in animal models with fluorocarbon derivatives in the management of VAE, especially cerebral ischemia, but human data are lacking.

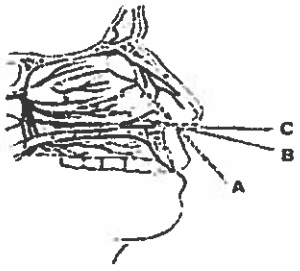
Prevention of air embolism is accomplished by avoiding patient position such as prone or park bench or by decreasing the gradient between the open surgical site veins and right atrium via leg elevation and using the flex option on the operating table control. Adequate hydration reduces VAE risk by preventing wide gradients between the right atrium and the entraining veins. The use of military antishock trousers have been show to reduce the VAE risk by increasing right atrial pressure, however its routine use in high risk patients can not be justified. Prophylactic use of PEEP has been controversial and it should be used with caution and used to improve oxygenation rather than as a means to minimize VAE.

Learning Summary;

Participants in this problem based learnig discussion will discuss the airway management of patients with CSI,different methods to monitor spinal cord function during spine surgery and diagnosis and management of intraoperative air embolism.







REFERENCES

Edward T. Crosby: Airway management in adults after cervical spine injury. Anesthesiology 2006; 104: 1293-318

Marek A. Mirski, M.D., PhD, Abhijit Vijay Lele, M.D, Lunei Fitzsimmons, M.D., Thomas J.K. Toung M.D: Diagnosis and treatment of vascular air embolism, Anesthesiology 2007; 106: 164-77

Stuart A. Grant, MB, ChB, Dora S. Breslin, MB, David B. MacLeod, MBBS, David Gleason, CRNA, Gavin Martin, MB, ChB; Dexmedetomidine infusion for sedation during fiberoptic intubation: A report of three cases. Journal of clinical anesthesia 16:124-126,2004

Shawn T. Simmons, M.D and Arno R. Schleich, M,D. Airway regional anesthesia for awake fiberoptic intubation: Regional anesthesia and pain medicine, vol 27, no 2 (march-april) 2002 pp 180-192.

Stiell IG, Wells GA, Vandemheen KL, Clement CM, Lesiuk H, De Maio VJ, Laupacis A, Schull M, McKnight RD, Verbeek R, Brison R, Cass D, Dreyer J, Eisenhauer MA, Greenberg GH, MacPhail I, Morrison L, Reardon M, Worthington J; The canadian C-spine rule for radiography in alert and stable trauma patients. JAMA 2001; 286:1841-8

White AA III, Punjabi MM: Clinical Biomechanics of the spine,2nd edition.Philadelphia, JB Lippincott,1990, pp 314-7.

SELECTIVE REFERENCES

Edward T. Crosby: Airway management in adults after cervical spine injury. Anesthesiology 2006; 104: 1293-318

Marek A. Mirski, M.D., PhD, Abhijit Vijay Lele, M.D, Lunei Fitzsimmons, M.D., Thomas J.K. Toung M.D: Diagnosis and treatment of vascular air embolism, Anesthesiology 2007; 106: 164-77

Stuart A. Grant, MB, ChB, Dora S. Breslin, MB, David B. MacLeod, MBBS, David Gleason, CRNA, Gavin Martin, MB, ChB; Dexmedetomidine infusion for sedation during fiberoptic intubation: A report of three cases. Journal of clinical anesthesia 16:124-126,2004.

LEARNING SUMMARY

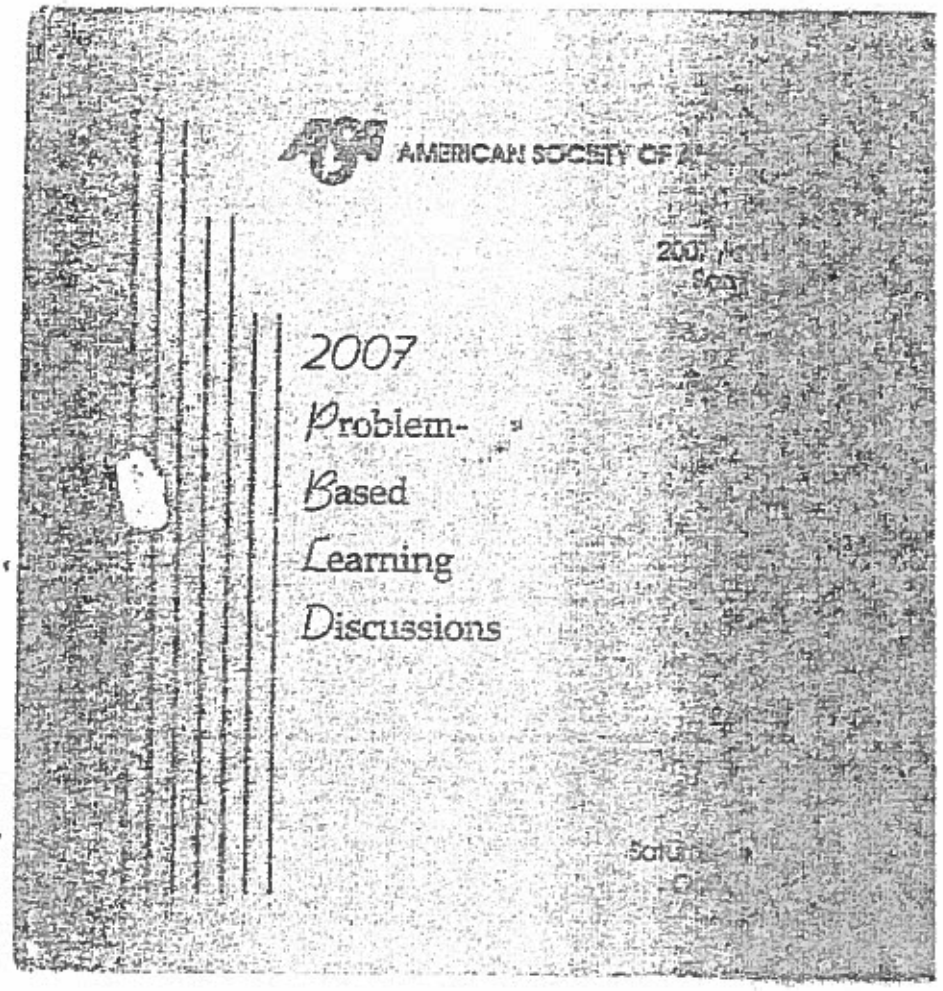
Emergency management of Unstable Cervical Spine Injury.

IV. Case studies from "2007 Problem-Based Learning Discussions

American Society of Anesthesiologists
2007 Annual Meeting, San Francisco, CA

IV Case studies from "2007 Problem-Based Learning Discussions"
American Society of Anesthesiologists 2007 Annual Meeting, San Francisco, CA

1. A Young Man Presents for Awake Craniotomy and Seizure Focus Excision by Heidi M. Koenig, MD – Louisville, Kentucky
2. Perioperative Visual Loss: 31-Year-Old Female Scheduled for a Redo Spinal Fusion with Instrumentation by Lorri A. Lee, MD - Department of Anesthesiology, University of Washington, Seattle, Washington
3. Head Injury in Adults by Martin Smith, MD – Director of Neurosurgical Critical Care, The National Hospital for Neurology and Neurosurgery, University College London, Queen Square, London, UK
4. Somatosensory Evoked Potential (SSEP) / Motor Evoked Potential (MEP) Monitoring in Scoliosis Surgery by Deborah A. Rusy, MD – Madison, Wisconsin
5. The Disappearing Waveforms: Cervical Spine Surgery with Evoked Potential Monitoring by Julia I. Metzner, MD – Seattle, Washington
6. Carotid Endarterectomy – I Thought That Was Replaced by Carotid Stenting – Not yet!!!!!! by Anthony N. Passannante, MD – Chapel Hill, North Carolina
7. The Acromegalic for Transsphenoidal Resection of a Pituitary Adenoma by Edward C. Nemergut, MD – Charlottesville, Virginia
8. The Neurosurgeon Asks You to Place Your Patient in the Sitting Position. Now What? By James R. Munis, MD, PhD – Rochester, Minnesota
9. Neurogenic Pulmonary Edema and Cardiac Failure Following Severe Traumatic Brain Injury by Maureen McCunn, MD, MIPP – Baltimore, Maryland
10. Spine Surgery in Adolescents: What Can Go Wrong? By Mohamed A. Mahmoud, MD – Cincinnati, Ohio
11. Awake Craniotomy and Seizure focus Excision in a Young Man by Heidi M. Koenig, MD – Louisville, Kentucky
12. He Can't Be Bind: It Was Only Back Surgery by Elizabeth A. Frost, MD – New York, New York
13. Only a MAC Case in Interventional Neuroradiology. The Patient for Vertebroplasty and Kyphoplasty by Elizabeth A. Frost, MD – New York, New York
14. Anesthesia for Selective Intra-Arterial Nicardipine Injection To Relieve Intracranial Vasospasm by Rafi Avitsian, MD – Cleveland, Ohio


AMERICAN SOCIETY OF
2007
Problem-
Based
Learning
Discussions

A Young Man Presents for Awake Craniotomy and Seizure Focus Excision

Heidi M. Koenig, M.D. Louisville, Kentucky

OBJECTIVES

The participant will be able to:

1) Understand the indications, contraindications, and preoperative preparation of the patient and the medical team for awake craniotomy.

2) Understand that the patient must be deeply sedated to tolerate certain parts of the procedure, but wide awake, yet comfortable for the testing and resection of seizure focus.

3) Understand the major intraoperative difficulties that arise at some point during many of these procedures.

4) Understand the medications that may not be used and those which are useful and when to give them during the procedure.

STEM CASE - KEY QUESTIONS

A 24-year-old man is scheduled for left temporal craniotomy and seizure focus excision during sedative anesthetic. He suffered a seemingly minor blunt head injury several years ago and now suffers almost daily seizures. The MRI shows a tiny scar-type lesion on or near his dominant (left) motor strip. The plan is to excise this with intraoperative electrocorticography (ECoG) and motor testing.

Have you ever seen or performed an awake craniotomy before? What are the indications for such a procedure? Are the concerns the same for awake craniotomy and for deep brain stimulators? How about trigeminal neuralgia treatments? Are there any contraindications to such an intervention?

The patient is otherwise healthy and has undergone several uncomplicated anesthetics for orthopedic injuries. The nurse has performed the preoperative evaluation. She told the patient that she couldn't imagine that anyone would do a craniotomy awake. He assures her this is what the neurosurgeon plans and that he is quite concerned about being awake during the whole process.

Do you need to meet with this patient or have a conversation with the neurosurgeon regarding this case?

You set up a meeting with the neurosurgeon, the neurologist, the patient, and his parents in order to go through the requirements for the patient, the monitoring, and for the surgical conditions. The patient thinks he can tolerate this if he listens to special music during the procedure; unfortunately, you happen to know that the neurosurgeon wants absolute silence in the OR.

How can you please both?

The next day the patient arrives in the holding area. The neurosurgeon requests sedation for the application of the halo frame. You look at it and see where he wants to put it. It will be quite close to his nose and just above his mouth.

Do you tell the surgeon you are uncomfortable with the position of the frame? What drugs can you give? Not give? Is there anything else that you could suggest to decrease the pain of the local anesthetic injection?

Now the patient needs to go to MRI for stereotactic scan with the halo on so that the computerized planning of the craniotomy can continue.

Sometimes the mapping is quite difficult and takes a long time. Are you and your team prepared to go with the patient? Could you give agents that would allow the patient to travel to get the MRI without anesthesia personnel? Should you give any special instructions to the OR personnel regarding this case?

You insist on recovering the patient for 20 minutes until the effects of your sedation have worn off and so you do not need to accompany him. He goes to the scanner and mapping takes place. He returns to the holding area in two hours. The neurosurgeon gives you the go ahead to take him to the room and initiate sedation for the procedure. You proceed. The first phase is a preparatory one. Monitors, an arterial catheter, a central venous catheter, and a urinary catheter must be placed; the head must be shaved; and the scalp block performed.

Should you do the scalp block? How deep should the sedation be at this point? What medications should you be using? What local anesthetics should you use? What about benzodiazepines or sodium thiopental?

The halo frame is secured to the operating room table and the patient is put in a slight head up and right tilt position with the use of the mechanical operating room table. You rouse him to be certain he is comfortable. You pad and secure his legs and tuck the left arm. The right arm must be free to allow careful testing during the excision. Next, the surgical prep of the head is done. The patient startles at the wet and cool stimulus. You are concerned that he may move in the head pins and hurt himself.

Are there any special considerations as you position the table? Should you use soft restraints on the patient's right arm to prevent him from interfering with the surgical process?

The process moves forward and the patient is once again quiet on the table. The procedure starts. At the time when the temporalis muscle is being taken down the patient complains of pain and starts to squirm. You deepen the level of sedation. Suddenly you notice that there is no end tidal CO_2 tracing and the O_2 saturation is decreasing. The patient is obstructing his airway, but is still making respiratory efforts.

Now what do you do? Should you just tell the surgeon to forget it? Should insert an LMA?
The situation resolves. You continue. Again there is some arousal and squirming at the time of the drill opening the craniotomy. Now the patient is complaining of nausea. He looks unwell.

What can you do?

You treat the nausea and the procedure goes on. After dural opening, you reduce the level of sedation and prepare for testing. As the neurosurgeon approaches the area of interest, the patient's face, then his arm, begins to twitch.

What's going on? What should one do?

The neurosurgeon asks you not to give midazolam or barbiturates. He does something and the twitching activity stops. He wants the patient to move his thumb and his great toe intermittently. You relay the requests to the patient and he does the desired activity. Suddenly after about 15 minutes of testing, he is unable to move his thumb. You inform the surgeon.

The surgeon mumbles and does something. Then he requests the patient move his thumb again. It moves again.

What just happened?

Now the patient is complaining of thirst and an ache in his neck. You reassure him and ask him to not move. He complies for a little while, but soon is asking for something to drink again. The surgeon is not done with the testing and resection yet.

What do you do now?

The mapping goes on and the patient starts to complain of urinary urgency. You assure him there is a catheter in place and that he will not embarrass himself. Now he complains of an itch under his left knee, then of thirst and urinary urgency. The surgeon is getting impatient with the situation.

What can you do to get through the final 20 minutes of mapping and resection?

Finally, the surgeon announces that the mapping and resection are complete. The patient believes him and reminds you every minute or so. You ask the surgeon if you can deepen the sedation. He allows this and asks you to give the patient his usual anticonvulsants. The neurosurgeon then scrubs out. He leaves his assistant to assure hemostasis and to close the craniotomy.

You administer additional propofol and midazolam. The patient goes to sleep. You call for someone to give you a break now that the critical part of the procedure is done. You go to the break area and are immediately paged back to the OR! The neurosurgeon has decided to resect one more section of the brain where there might be some additional scar tissue. You turn off the propofol and offer to give flumazenil. You try to rouse the patient, but he's too sleepy to cooperate. When you are able to get a response out of him he startles. You are afraid he'll injure himself since his head is fixed in a frame.

Now what should you do? Should you give flumazenil or insist that the team wait until the patient can cooperate again?

The surgeon doesn't want to wait. He resects the additional area without testing. The assistant closes and you take the sleepy patient to the ICU.

The neurosurgeon and the neurologist are looking at the functional MRI and the intra- and extra-operative ECoG materials. As the patient awakens you are eagerly testing his motor function on his dominant side. He has a mild deficit of the thumb. He is grateful for your efforts and somewhat apologetic for his neediness during the procedure. You feel awful.

Should you tell him that you prevented the team from testing him for the last bit of the resection and put him at risk? Should you assure him the strength will return? What should you do if the neurosurgical team indicates his deficit is due to a poorly performed sedative anesthetic?

PROBLEM BASED LEARNING DISCUSSION

A 24-year-old man is scheduled for left temporal craniotomy and seizure focus excision during a sedative anesthetic. He suffered a seemingly minor blunt head injury several years ago and now suffers almost daily seizures. He hates his medication regime, or that he cannot drive or have even an occasional drink of alcohol. The MRI / functional MRI shows a tiny scar type lesion on or near his dominant (left) motor strip. The surgeon doesn't want to disable the man, so the plan is to excise this with intraoperative electrocorticography (ECoG) and motor testing during a sedative anesthetic.

Have you ever seen or performed an awake craniotomy before?

In preparation for a procedures that is unfamiliar to the team, there must be conversations which review every detail and expectation of everyone involved---in this case everyone from the patient and neurosurgeon to the nurses and neurologists.

This is not a procedure that should be undertaken without a large amount of preparation of the patient, the family, and the whole perioperative management team. Everyone in the room needs to be aware of the fact that the patient is awake and keep their conversations to a minimum and respectable. This will facilitate communication between the neurosurgeon, the monitoring team, the patient, the anesthesiologist, and the nursing personnel.

What are the indications for such a procedure?

A common indication is the need for intraoperative assessment and preservation of neurologic function when the procedure is in an eloquent area of the brain, as in temporal lobe surgery for seizure focus excision. The other indication is the need to verify that the procedure has indeed controlled the problem, such as the movement disorder of Parkinson's disease. Other awake neurosurgical procedures include tumor and AVM resections. Rarely awake cranial procedures are performed for chronic pain syndromes. Basically, neurosurgeons are performing more and more invasive procedures that require patient cooperation intraoperatively. Deep brain stimulators for patients with Parkinson's disease, AVMs, tumors in eloquent areas of the brain, and stimulator for pain procedures are just a few of them that require a wide awake and cooperative patient during key portions of the procedure.

Are the concerns the same for awake craniotomy and for deep brain stimulators?

No. Actually in many centers neurosurgeons perform the deep brain stimulators without any anesthesia personnel present. In the United States, anesthesiologists are usually present to monitor the patient and tend to any emergencies that might arise. When performing sedation for deep brain stimulators, one is much less limited in the choice of medications. All sorts of interesting things can occur during these minimally invasive procedures, but they are usually related to the underlying disorer. The patients must refrain from taking their medications prior to surgery for movement disorders. This so that the surgeons can be certain the abnormal movements are controlled during the intraoperative trial of the stimulator leads.

In one case, a Parkinsonian patient was experiencing such vigorous movements and sweating that it was difficult to carry on the procedure and monitoring. She was also being treated for mild nausea, which she related to having her head in a fixed position when her body was moving so much. Eventually the anesthesiologist realized the patient was experiencing diaphoresis and hypoglycemia from all the energy expenditure! A little D5 IV and she were able to go on with the testing. Those procedures are amazing! The patients can usually drink water from a cup without spilling at the time when the deep brain stimulator is activated.

A good sedative to use for these procedures is dexmedetomidine (either as an infusion or as carefully titrated bolus [10mcg] doses) as it doesn't interfere with the respiratory drive and or increase secretions. Most patients are uncannily able to awaken themselves and cooperate when coached. They feel as if they've had a very pleasant experience afterward.

What about trigeminal neuralgia treatments?

These are extremely difficult, because, once again, the lesion is permanent. The patient must be absolutely lucid and cooperative and then deeply anesthetized several times in sequence for the ablations and testing sessions. It is important to have all the x-ray and radiofrequency ablation equipment in the room and ready to go when the patient arrives for the procedure to decrease the commotion and the patient's anxiety.

Are there any contraindications to such an intervention?

YES! 1) A patient who is almost continuously seizing or is unable to cooperate or to lie still for many hours. 2) There are cases that are ill advised to try to do under awake conditions. The neurosurgeons, family, and neurologists all need to be realistic about what the anesthesiologists can accomplish. 3) We're good, but we cannot perform this type of procedure in someone who does not speak the same language we do.

Do you need to meet with the patient or have a conversation with the neurosurgeon regarding this case?

You would need to set up a meeting with the neurosurgeon, the neurologist, the patient, and his parents. At the meeting you assure the patient that he'll be comfortable and detail what the plan is with regard to the halo, the urinary catheter, the lines, and the monitors. In addition you warn him about the potential for some noise in the operating room during the procedure and that you will be with him throughout the entire time.

You go through the requirements for the patient during the mapping and resection. Take this time to build rapport with the patient. If he wants to listen to special music during the procedure, it must be interrupted during the testing phase. Also, if you can reach the ears, ear buds or headphones can be positioned and kept at a low volume so the surgical team is not disturbed.

The next day when the patient arrives in the holding area, what drugs can you give for the application of the head frame?

One possibility is to give small boluses of dexmedetomidine (10 mcg increments) and remifentanyl (6.25 mcg increments). These are very potent drugs and should not be given by inexperienced personnel. The combination, if given quickly, can lead to extreme bradycardia, so wait for the full effect of each dose before giving more. Also, if you have never administered them by bolus before, this is not the situation in which to try something new. These meds allow the anesthesiologist to momentarily give the patient excellent analgesia and sedation for administration of the local anesthetic and application of the frame. Also, after 5 minutes the sedation wears off and the patient can travel to MRI or CT for mapping without an anesthesiologist accompanying them. Midazolam and barbiturates should be avoided. You can recommend that the surgeon add sodium bicarbonate the local anesthetic (0.1 meq NaBicarb/ml of local anesthetic) and thus decrease the pain of application. Pain can be further reduced by jet injection of the local anesthetic.

Are there any special considerations as you position the table? Should the anesthesiologist use soft restraints on the patient's right arm to prevent him from interfering with the surgical process?

When the patient returns from the scans and mapping, you can initiate the sedation and proceed into the OR. Some anesthesiologists do the scalp block. As long as there is excellent sedation and monitoring, this is not a problem.

The halo frame is secured to the operating room table and the patient is put in a slight head up and right tilt position with the use of the mechanical operating room table. You rouse the patient to be certain he is comfortable. You pad and secure his legs and tuck his left arm. The areas of interest during the testing must be free enough to allow testing during the excision. Throughout the process, you must rely on your rapport with the patient to sustain much of the calming necessary to keep him on the table.

What can you do when the patient requires more analgesia and obstructs his airway? Should you just tell the surgeon to forget it? Should you insert an LMA?

The best thing to do is to gently perform a jaw thrust without moving the surgical field. If that doesn't help resolve the situation, you can try inserting a nasal airway. This is usually too stimulating for the patient to tolerate and the patient breathes well enough not to require it any more. Of course, the patient may also move and strain during the process.

What can you do when the patient freaks over the drilling and becomes nauseated?

Temporarily deepen the sedation carefully. Treat the nausea with antiemetics. Many anesthesiologists give prophylactic antiemetics, such as dexamethosone and a $5HT_3$ antagonist, at the beginning of the sedation. Again, rely on the rapport you developed with the patient.

As the neurosurgeon approaches the area of interest, the patient's face and then his arm begin to twitch. What's going on? What should you do?

This is a seizure. The neurosurgeon can irrigate the area with iced saline and interrupt it temporarily. This also interrupts the electrocorticography temporarily, but it is reversible. If you give intravenous anticonvulsants, you will have the effect of the medications for a long time and you may not be able to get adequate monitoring again. If the patient goes into a persistent seizure you must protect the airway and administer medications to stop it.

The patient is moving his thumb and great toe at the neurosurgeon's request intermittently during testing. Suddenly after about 15 minutes of testing, he is unable to move his thumb. You inform the surgeon. He mumbles and does something. Then he requests the patient move his thumb again. It moves again.

What just happened?

The surgeon was probably stimulating an area of seizure focus that demonstrated motor function on the fMRI. He will not resect it if it controls the dominant opposing digit.

When the patient becomes uncomfortable and complains of thirst and aches and such what do you do?

Depend on the rapport you developed with him earlier. Sometimes a little ice or a damp cloth on the lips is helpful. Reassure him that you are working as fast as you can.

What can you do to get through the final 20 minutes of mapping and resection when the patient is becoming anxious and complaining of urinary urgency, itchiness, thirst, and aches?

This is common and you must reassure the patient over and over.

When the procedure is truly completed, you should administer the patient's anticonvulsant drugs and deepen the sedation. Many anesthesiologists wait until the dura is closed to initiate benzodiazepine therapy and anticonvulsant therapy to avoid the scenario described above.

If the surgeon does return to resect one more focus, flumazenil and naloxone will awaken the patient, but these agents are somewhat unpredictable and you may end up with a dangerously awake and frustrated patient who is possibly having a seizure! There is no right answer. If possible, allow a bit of time to pass, and then when the patient is arouseable again, retest and complete the resection.

If the patient does end up with a deficit postoperatively, should you tell him that, as his anesthesiologist, you prevented the team from testing him for the last bit of the resection

and put him at risk? Should you assure him that the strength will return? What should you do if the neurosurgical team indicates his deficit is due to a poorly performed sedative anesthetic?

These are extremely difficult situations to deal with and each individual has to come up with his or her own answer. One thing is certain; you must always assure the patient that no matter what the outcome, you truly did the best that you were able. And if there is a deficit postoperatively you should not accept blame or suffer guilt over the whole situation if you followed the best medical practice. If something occurs in the OR or ICU, everyone should be honest and not imply that another practitioner performed poorly. You may want to invite the neurosurgeon to discuss the intraoperative events and the possible reasons for the adverse outcome with the patient and family to help them understand the dilemmas and ward off bad feelings.

One way to perform these cases is as follows: Use dexmedetomidine, 10 mcg boluses —with or without an infusion — to keep the patient comfortably awake without respiratory depression, supplement with small amounts of narcotics and propofol during the most stimulating portions of the procedure, and add a little benzodiazepine at the end of the case. Most of all, keep good rapport with the patient and the surgeon.

Many practitioners put the patient to sleep for the initial part of the procedure, wake and extubate them for the testing, and then replace the ETT or LMA for the closure. It depends on the comfort level of the neurosurgeon and the anesthesiologist as well as the patient with all the airway manipulation. If in a situation that requires airway support, if at all possible place an LMA using the thumb to guide it into the pharynx without removing drapes or frame. Usually, the positioning allows for enough oral opening for the LMA to slip into place. If this doesn't work, the anesthesiologist can place a mask on the patients until the sedation wears off enough for them to maintain their own respiration again. If need be, the anesthesiologist can request the surgeon to cover the field and remove all or part of the frame to secure the airway again. Remember it is not necessarily a failure of skill to change to a sleeping technique, but it is in the patient's best interest if he can't tolerate the process or has a seizure or surgical issue, such as a swollen brain, which requires more control of the airway.

Is possible, continue a technique of anesthesia that allows for continued monitoring of the electrical activity of the brain and the guided resection can be completed

These really challenging cases are becoming more common. Review the procedure and expectations with everyone involved in advance of OR entry. What ever you do, choose medications and airway management techniques that you are comfortable with. Keep in constant communication with the patient and the surgeon and the cases will go quite well and everyone will be satisfied in the end.

References

1) Bekker AY, Kaufman B, Samir H, Doyle w. The use of dexmedetomidine infusion for awake craniotomy. Anesthesia and Analgesia 2001;29(15)1251-1253.

2) Craen RA, Herrick IA, Seizure surgery: general considerations and specific problems associated with awake craniotomy. Anesth Clin of NA 1997;15(3): 655-672.

3) Gumnit RJ. Selection of adult patients for surgical treatment of epilepsy. ACTA Neurol Scand Suppl 1988;117:42-46.

4) Koffke WA, Tempelhoff R, Dashliff RM. Anesthetic implications of epilepsy: Status epilepticus and neurosurgery. J Neuorsurgical Anesthesia 1997;9(4):349-72.

5) Maze M, Scarfini C, Cavaliere F. New agents for sedation in the Intersive Care Unit. Crit Care Clin 2001;17(4):881-97.

6) Sarang A Dinsmore J. Anaesthesia for awake craniotomy---evolution of a technique that facilitates awake neurological testing. British J Anaesthesia 2003;90(2)161-165.

7) Skukas AP, Artru A. Anesthetic complications of awake craniotomies for epilepsy surgery. Anesthesia and Analgesia 2006;102:882-7.

8) Soriano SG, Eldredge EA, Wang Ek, Kull L, Madsen JR, Black PMcL, Rivello JJ, Rockoff M. The effect of propofol on interaoperative electrocorticography and cortical stimulation during awake craniotomy in children. Paediatric Anaesthessia 2000;10:29-34.

9) Suhjpaul RI. Awake Craniotomy: controversies, indications and techniques in the surgical treatment of temporal lobe epilepsy. Can J Neurol Sci 2000;27 Suppl 1:S55-63 and S92-6.

10) Venkatraghavan L, Manninen P, Mak P, Lukitto K, Hodaie M, Lozano A. Anethesia for functional neurosurgery: a review of complications. J Neurosurg Anesthesiol 2006;18(1):64-7.

Selective References

1) Venkatraghavan L, Manninen P, Mak P, Lukitto K, Hodaie M, Lozano A. Anethesia for functional neurosurgery: a review of complications. J Neurosurg Anesthesiol 2006;18(1):64-7.

2) Bekker AY, Kaufman B, Samir H, Doyle w. The use of dexmedetomidine infusion for awake craniotomy. Anesthesia and Analgesia 2001;29(15)1251-1253.

3) Sarang A Dinsmore J. Anaesthesia for awake craniotomy---evolution of a technique that facilitates awake neurological testing. British J Anaesthesia 2003;90(2)161-165.

LEARNING SUMMARY

The participant will be able to:

1) Understand the indications, contraindications, and preoperative preparation of the patient and the medical team for awake craniotomy.
2) Understand that the patient must be deeply sedated to tolerate certain parts of the procedure,but wide awake, yet comfortable for the testing and resection of seizure focus.
3) Understand the major intraoperative difficulties that arise at some point during many of these procedures.
4) Understand the medications that may not be used and those which are useful and when to give them during the procedure.

Perioperative Visual Loss:
31-Year-Old Female Scheduled for a Redo Spinal Fusion with Instrumentation

Lorri A. Lee, M.D., Department of Anesthesiology, University of Washington, Seattle, WA

Objectives:
After preparing for and discussing this case the participants will:

1. Discuss the preoperative workup and consent for a patient undergoing major spinal surgery.
2. Understand the physiology and potential complications of prolonged surgery in the prone position.
3. Discuss the monitoring and anesthetic management for major spinal surgery, including the role of deliberate hypotension and fluid management.
4. Understand the different etiologies that have been suggested to contribute to perioperative visual loss.
5. Discuss what precautions have been suggested to decrease the risk of perioperative visual loss in the prone position, and what documentation is recommended.
6. Discuss the best ophthalmologic exam needed to aid diagnosis of perioperative visual loss.
7. Understand the differential diagnosis and diagnostic workup of perioperative visual loss, and the association with particular procedures.
8. Discuss the possible treatments for perioperative visual loss and the potential for improvement.

Case: 31 yo female is scheduled for a redo spinal fusion with instrumentation. She is S/P Harrington rod fixation at 15 years of age.

Preoperative Evaluation:

1. What are this patient's risk factors for perioperative visual loss?
2. Does her relatively young age protect her from this complication?
3. Should your consent cover the risks of perioperative visual loss?
4. What monitors would you use for this case?

Anesthetic Management Issues:

1. Because this is a multi-level redo spine operation with instrumentation, the surgeons have requested that you manage the anesthetic with deliberate hypotension – what do you tell them?
2. The surgeons want to put the head in Mayfield tongs "so the patient won't go blind." What is your response?
3. Are there any monitors available to monitor optic nerve ischemia?
4. Will you change your management in any way with the intent to decrease the risk of perioperative visual loss?

Intraoperative Course:

After approximately 6 hours of operating with an estimated blood loss (EBL) of 1500ml, transfusion of packed red blood cells (PRBCs) and cell saver has resulted in a hematocrit of 29% and a CVP of 17 mm Hg. However, the blood pressure has gradually decreased to a mean arterial pressure (MAP) of approximately 60 mm Hg. Her baseline MAP is 100 mm Hg. You attempted to reduce the anesthetic depth, but this resulted in inadequate anesthesia and interference with the SSEP signals.

1. Is a MAP of 60 mm Hg is sufficient for this 31yo patient in the prone position?
2. Would you try to increase her MAP with volume? Colloid vs. crystalloid?
3. If volume does not improve her MAP, would you start a phenylephrine infusion to increase her blood pressure? Does phenylephrine affect the blood flow to the optic nerve?
4. Would you encourage the surgeons to close soon and stage the procedure?

Postoperative Course:

The operation was not staged and lasted 10 hours. Total EBL was 3200ml and ending hematocrit was 28%. The patient was turned supine and her face was very edematous. You elected to keep her intubated overnight until the head and neck edema subsided.

1. Are there any special precautions you will take with the intent of decreasing her risk of perioperative visual loss while she remains intubated?
2. If she could count fingers in each eye, would the possibility of visual loss be excluded? What is the best test for perioperative visual loss?

Approximately 2 hours after she is extubated on POD #1, she complains of inability to see from her left eye.

1. What is the most likely diagnosis of her visual loss? What is the differential diagnosis?
2. What tests besides the ophthalmologic consult would be helpful to diagnose her lesion?
3. What treatment options have been tried for perioperative ischemic optic neuropathy (ION)?
4. What are her chances of vision recovery?
5. If the surgeons had staged her procedure originally, how long would you make them wait before returning to the operating room?

Perioperative Visual Loss

Perioperative visual loss has received increased attention over the last decade from surgeons, anesthesiologists and ophthalmologists. The perceived increase in the incidence of perioperative visual loss after spine surgery in the prone position is probably a result of 3 changes: 1) increased awareness of the disease; 2) a dramatic increase (300 – 400%) in the use of instrumentation since 1996 when interbody cages were FDA approved, which results in longer operative times and increased blood loss; and 3) an aging population who require more spine operations for degenerative conditions. Despite this increased attention, little progress has been made because of the low incidence of this complication (highest estimates at 1 in 500 for spine operations [1]) and the inability to monitor the optic nerve function intraoperatively. Although many factors such as hypotension, anemia, long duration in the prone position, large blood loss, fluid management, adverse drug effects, and unique anatomical variations in optic nerve blood supply have been proposed as contributing to this complication, none have been causally linked in randomized controlled trials or animal models.[2]

The most common operations associated with perioperative visual loss are cardiac bypass procedures, spine surgery in the prone position, and head and neck operations.[3] The incidence, presumed etiology, and type of lesion may vary depending on the type of procedure. However, regardless of the procedure, the differential diagnosis is the same. The primary ophthalmologic lesions to consider are 1) central retinal artery occlusion (CRAO); 2) anterior ischemic optic neuropathy (AION); 3) posterior ischemic optic neuropathy (PION); and 4) cortical blindness. All of these lesions can occur in patients undergoing spine surgery in the prone position, but the most common lesion associated with this procedure is ischemic optic neuropathy (ION), particulary PION. Preliminary results from the ASA Postoperative Visual Loss Registry demonstrated that 81% of 53 spine cases in the database were diagnosed with either AION or PION.[4] Only 13% of cases were diagnosed with CRAO.

Central Retinal Artery Occlusion (CRAO)

CRAO displays an absent pupillary reflex or relative afferent pupillary defect. It is almost always unilateral, usually involves total loss of vision, and rarely recovers. Fundoscopic exam reveals a pathognomonic cherry red spot at the macula, narrowed retinal arterioles, and a pale and edematous retina. Etiologies proposed for CRAO with respect to spine surgery include external globe compression, emboli, hypotension, venous congestion, or vasculitis. Although most postoperative visual deficits after prone spine surgery are caused by ION not associated with external globe pressure, the rare cases that are diagnosed with CRAO in the prone position are frequently associated with globe compression. Associated ipsilateral features such as extraocular muscle paresis, bruising, periorbital edema, or proptosis implicate external globe compression as the causative factor in CRAO. Blood loss and duration of surgery for patients diagnosed with CRAO are usually significantly less compared to patients diagnosed with ION.[4] Many of the case reports with CRAO occurred with the use of the horseshoe headrest, though it can occur with soft foam cushions as well. In fact, the original 8 cases of CRAO published in 1954 that occurred in the prone position in association with pressure on the globe, all used the horseshoe headrest.[5] Because CRAO still occurs secondary to external globe compression in the prone position, careful eye checks every 15 to 30 minutes in the prone position with documentation is recommended. Mayfield tongs are an alternative to the horseshoe headrest when the eyes cannot be adequately

protected or frequently checked during the procedure. However, tongs will not prevent the occurrence of ischemic optic neuropathy (ION).

Ischemic Optic Neuropathy: AION and PION

ION is the most common diagnosis for perioperative visual loss associated with spine surgery in the prone position. AION and PION may be difficult to distinguish from one another because of subtleties of the fundoscopic exam, and the fact that both lesions are identical several days to weeks after the event. Early fundoscopic exam will demonstrate disc swelling / edema in AION, whereas the fundus is entirely normal in appearance with PION. Several days to weeks later when disc edema has usually resolved, the fundoscopic exam reveals optic nerve pallor in both AION and PION, and they are indistinguishable at this point. It is currently unclear if these 2 lesions represent a continuum of the same disease or not when associated with spine surgery. Pupillary light reflexes in ION are either absent or delayed. Unlike CRAO, both eyes are affected in just over half of the ION cases in the ASA Postoperative Visual Loss Registry.[4] Loss of vision can include total blindness or altitudinal field cuts. Some recovery occurs in approximately 40 to 45% of patients, but vision rarely returns to baseline. High dose steroid and / or hyperbaric oxygen treatment have been utilized for treatment of ION with variable success. Restoration of the MAP to baseline and treatment of significant anemia is recommended by most consultants. Proposed etiologies for ION include hypotension, anemia, venous congestion, edema, adverse drug effects, and individual patient variation in anatomy and physiology of the optic nerve blood flow. Embolic causation is less likely with this lesion because the retina lacks these findings. This lesion is not associated with external pressure on the globe, and 8 of 43 spine cases with ION from the ASA Postoperative Visual Loss Registry had their heads in Mayfield tongs with their eyes free of any pressure.[4] Spine surgery patients with ION from this database had an associated large operative blood loss (median 2.3 liters), long duration in the prone position (median 8 hours), some degree of hypotension (median lowest MAP 37% decrease below baseline), and moderate anemia (median 25.5%).

Cortical Blindness

Cortical blindness demonstrates normal pupillary light reflexes with a normal fundoscopic exam. Peripheral vision is frequently affected, but total blindness may occur as well. Both eyes are usually affected, and approximately 2/3 of cases may get some improvement in vision.[3] Etiologies for cortical blindness include hypotension and emboli. Treatment is directed at restoration of baseline MAP. This lesion is rarely diagnosed after spine surgery in the prone position unless some intraoperative catastrophic event occurs leading to profound hypotension.

Diagnostic Studies for Perioperative Vision Loss

Ophthalmologic exam is the first and only essential test that should be ordered for any patient with perioperative visual loss. Visual acuity, intraocular pressures, color testing, gross visual fields, pupillary reflexes and fundoscopy with pupillary dilation should be performed. The ability to read letters and numbers or count fingers does not preclude the possibility of scotoma or visual field cuts as occurs in ION. Formal visual fields should be assessed in the ophthalmologist's office prior to discharge when there is a complaint of visual loss. Some

defects are so subtle that patients do not notice them until they go to read a book several weeks after their operation.

CT and MRI should demonstrate occipital cortical strokes consistent with cortical blindness. CT is not useful for the diagnosis of ION. MRI is not particularly sensitive for detecting early ischemia of the optic nerves, but may occasionally show swelling or abnormal signal intensity. Electroretinograms (ERG) are sensitive detectors of retinal ischemia and should be useful for detecting CRAO, though fundoscopic exam should be sufficient for diagnosing CRAO with a finding of the pathonogmonic cherry red spot. Visual evoked potentials (VEPs) detect abnormalities of the optic nerve and its projections to the occipital cortex. VEPs are useful in the diagnosis of ION before optic nerve pallor has emerged, particularly when visual acuity is unable to be assessed. Currently, VEPs are unreliable when used in the presence of general anesthesia, volatile or total intravenous based, and therefore cannot be utilized as an intraoperative monitor of optic nerve function. [6]

Perioperative Visual Loss Associated with Spine Surgery in the Prone Position

The majority of ION cases associated with spine surgery in the prone position are diagnosed as PION. This is also the most commonly diagnosed ophthalmologic lesion that occurs after radical neck procedures in which both internal jugular veins are ligated.[7] Therefore, venous congestion is thought to be a significant etiologic factor in the development of this disease. Both intraocular pressure (IOP) and intracranial pressure (ICP) are known to increase in the Trendelenburg and prone positions, presumably from venous engorgement, as central venous pressure also increases.[8-10] Because maintenance anesthesia is often associated with a 10% to 30% decline in MAP from baseline, the perfusion pressure of the optic nerve may significantly decrease during prone spine surgery or during operations in which the venous drainage from the head and neck is compromised. Further reduction of inflow, as with deliberate hypotension, or increase in venous congestion from lowering the head position may cause a critical reduction in perfusion pressure to the optic nerve in susceptible individuals. This rationale has served as the basis for the recommendation to maintain the MAP within 20% of baseline for spine surgery in the prone position.

There are currently no adequate randomized controlled trials (RCTs) demonstrating the benefits of deliberate hypotension in spine surgery in the prone position. One small study with only 24 patients demonstrated reduced blood loss, but operating time was not shortened. Moreover, five of these patients had SSEP changes requiring reversal of the hypotension and wake-up tests.[11] It is unclear whether or not reducing the arterial blood pressure would decrease bleedingin spine surgery to the same degree as in hip surgery, without affecting spinal cord blood flow. Although the benefits of deliberate hypotension were demonstrated in a RCT for hip surgery,[12] the venous congestion in spine surgery in the prone position is likely to be significantly greater than in hip surgery where the leg is frequently elevated.

Other factors that may also contribute to the development of ION are significant anemia with reduced oxygen delivery. Many ION case reports after spine surgery are associated with large blood loss and anemia, as well as hypotension. It is unclear if the anemia is an incidental finding or contributory factor. Although many studies have demonstrated increased blood flow with anemia, they have not been performed under conditions of high venous "back pressure" for prolonged periods of time. Therefore, it is unclear what the transfusion threshold should be in these cases. Blood loss is increased with increased number

of levels of instrumentation, redo spine operations, and certain diseases (e.g., Charcot joints, infection, and non-embolized tumors).

Edema formation around the optic nerve is another proposed etiologic factor. One of the leading theories about ION associated with surgery in the prone position is the development of a compartment syndrome caused by edema formation at either the lamina cribrosa or optic canal. This theory has led to speculation that perhaps colloids should be used over crystalloids in an attempt to reduce edema formation. The role of pharmacological interventions to elevate the blood pressure such as phenylephrine has also been proposed as a possible contributory factor since the ophthalmic and ciliary arteries which supply the optic nerve have alpha-1 receptors.[13] Unfortunately, the inability to study the blood flow to the posterior optic nerve in humans makes in vivo data on drug effects impossible to study currently.

The occurrence of this complication in children (3 pediatric cases in the ASA Postoperative Visual Loss Registry associated with the prone position), and in relatively healthy young adults suggests that this particular ophthalmologic lesion in these procedures is brought about by "normal" physiologic changes, rather than a result of disease-induced changes such as atherosclerosis. Moreover, the etiology appears to be multi-factorial because there are many ION case reports which lack hypotension, anemia, operative time > 5 hours, or use of vasopressors. Patient-specific factors that are not readily detectable such as anatomic and physiologic variations in the optic nerve blood supply are also likely to contribute to ION. These inter-patient differences may account for similar anesthetic management in patients resulting in different outcomes.

Potential Preventative Measures

Unfortunately, definitive preventative measures for ION associated with spine surgery in the prone position cannot be put forth without having RCTs. Therefore, the following interventions that have been suggested are merely speculative, and their risk:benefit ratio is unknown.

Intervention	Benefit	Potential Harm
1.Elevating head	Improved venous drainage.	Increased venous pressure and potentially increased bleeding at low thoraco-lumbar operative site; may decrease MAP which may require increased fluids or vasoactive agents.
2.Keep head neutral	Improved venous drainage.	None.
3.Avoid constrictive ties around neck	Improved venous drainage.	None.
4.Increased colloid	Potentially decreased edema.	Increased expense.

5.Maintenance of MAP within 20% of baseline	Improved perfusion pressure of optic nerve.	Potentially increased volume administration and / or use of vasoactive agents.
6.Anti-fibrinolytics	Decreased blood loss.	Unknown effect on optic nerve vasculature / blood flow; increased expense.
7.Staging Procedure	Decreased time in prone position.	Increased length of hospital stay; potential for increased risk of infection
***Not recommended**		
Restrict fluids*	Decreased edema of head.	**Many**: hypotension which may lead to decreased perfusion pressure of optic nerve; inadequate tissue perfusion; metabolic acidosis; rhabdomyolysis; ATN; tachycardia with increased myocardial demand.

Medico-legal Issues

Many patients who develop perioperative visual loss file claims against the anesthesiologist and / or surgeon. Not only is this perioperative complication frequently disabling, but it is difficult for the lay person to understand how their vision can be damaged when the operation was on their back. Physician negligence is frequently their initial conclusion. In fact, for many years, most surgeons, anesthesiologists, and ophthalmologists believed that all injuries to the visual system after spine surgery in the prone position were caused by external compression of the globe.

The major aspects of anesthetic management that plaintiffs' lawyers tend to focus on are: 1) Lack of documentation of frequent eye checks, even if the diagnosis is ION. (Pressure on the globe is the most simple explanation for a jury to believe.) 2) Inadequate blood pressure maintenance, particularly when deliberate hypotension was used. 3) Anemia, hematocrit < 30%. 4) Lack of informed consent regarding the risks of visual loss. Though these issues make defense of cases more difficult, successful defense verdicts have been obtained in the presence of these issues.[14]

References

1. Stevens W, Maj M, Glazer P, et al. Ophthalmic complications after spinal surgery. Spine 1997; 22(12):1319-1324.
2. Lee L, Lam A. Postoperative Visual Loss. Advances in Anesthesia 2003; 21:81-108.
3. Roth S, Gillesberg I. Injuries to the Visual System and Other Sense Organs. *In* Benumof J, Saidman L, eds. Anesthesia and Perioperative Complications. St. Louis: Mosby, 1999. pp. 377-408.

4. Lee L. ASA Postoperative Visual Loss Registry: Preliminary Analysis of Factors Associated with Spine Operations. ASA Newsletter 2003; 67(6).
5. Hollenhorst R, Svien H, Benoit C. Unilateral blindness occurring during anesthesia for neurosurgical operations. A.M.A. Arch Ophth 1954; 52:819-830.
6. Weidemayer H, Fauser B, Armbruster W, et al. Visual evoked potentials for intraoperative neurophysiologic monitoring using total intravenous anesthesia. J Neurosurg Anesthesiol 2003; 15(1):19-24.
7. Pazos G, Leonard D, Blice J, Thompson D. Blindness after bilateral neck dissection: case report and review. Am J Otolaryngol 1999; 20(5):340-5.
8. Lam A, Douthwaite W. Does the change of anterior chamber depth or / and episcleral venous pressure cause intraocular pressure change in postural vaariation? Optom Vis Sci 1997; 74(8):664-667.
9. Cheng M, Todorov A, Tempelhoff R, et al. The effect of prone positioning on intraocular pressure in anesthetized patients. Anesthesiology 2001; 95(6):1351-5.
10. Lee L, Vavilala M, Sires B, et al. Intraocular pressures during prone spine surgery do not predict visual deficits. American Society of Anesthesiologists 2001 Annual Meeting 2001.
11. Grundy B, Nash CJ, Brown R. Deliberate hypotension for spinal fusion: prospective randomized study with evoked potential monitoring. Can Anaesth Soc J 1982; 29(5):452-62.
12. Thompson G, Miller R, Stevens W, Murray W. Hypotensive anesthesia for total hip arthroplasty: a study of blood loss and organ function (brain, heart, liver, and kidney). Anesthesiology 1978; 48(2):91-6.
13. Ohkubo H, Chiba S. Vascular reactivities of simian ophthalmic and ciliary arteries. Curr Eye Res 1987; 6(10):1197-203.
14. Preferred Physicians Medical Risk Retention Group I. POVL: Michigan Defense Verdict. Anesthesia and the Law Newsletter 2004(14).

Head Injury in Adults

Martin Smith, M.D.,
Director of Neurosurgical Critical Care
The National Hospital for Neurology and Neurosurgery, University College London, Queen Square, London, UK

The Case

A twenty four year old male involved in a road traffic accident was unconscious at the scene. He was intubated and ventilated prior to transfer to the emergency department where a cranial CT scan revealed an acute subdural hematoma. Secondary survey confirmed that there were no other significant injuries. The patient underwent craniotomy for evacuation of the hematoma and was transferred to the neurosurgical ICU for postoperative monitoring and management. Over the next few days he developed intracranial hypertension and severe respiratory and cardiovascular instability.

Objectives:

After discussing this case, the participants will:

1. Understand the important issues during resuscitation of a patient with a severe traumatic brain injury.
2. Be aware of the monitoring techniques that may assist clinicians in managing patients with severe traumatic brain injury on the ICU.
3. Understand the causes of elevated intracranial pressure in a patient following evacuation of an intracranial hematoma and be aware of the treatment options.
4. Understand the relative merits of cerebral perfusion and intracranial pressure guided therapy and be aware of the potential disadvantages of each.
5. Be aware of the potential causes of systemic complications after severe traumatic brain injury and understand the potential management challenges.

Resuscitation

Resuscitation after traumatic brain injury (TBI) should begin in the pre-hospital phase and is a key stage at which mortality and morbidity can be influenced. The primary goal of management is the prevention, recognition and treatment of conditions known to cause secondary brain injury, including identification and evacuation of surgically remedial compressive lesions and prevention of secondary systemic insults. The importance of cardiopulmonary stabilization cannot be overemphasized because the risk of secondary brain injury begins and continues from the moment of trauma. Consensus guidelines for the management of TBI have been issued [1,2].

Because of the well-established association between hypoxemia and worsened outcome after severe TBI, all patients should be intubated to allow controlled ventilation. During intubation the cervical spine must be protected with manual in-line immobilization because of the associated risk of cervical injury. Oral endotracheal intubation should be carried out following administration of an intravenous anaesthetic agent (thiopental, propofol or etomidate are appropriate choices), in a dose calculated to minimize the intracranial pressure (ICP) response to laryngoscopy whilst maintaining cardiovascular stability. A full stomach should be assumed and a rapid sequence induction is mandatory with placement of an oro-gastric tube post-intubation to decompress the stomach and prevent acute gastric dilatation. The use of succinylcholine in TBI

remains controversial, although the benefits of rapid and full relaxation to facilitate expeditious airway control are likely to outweigh the potential disadvantages of a small and transient increase in ICP. Once the airway has been secured, mechanical ventilation should be commenced to maintain $PaO_2 > 13.5$ kPa and $PaCO_2$ between 4.0 - 4.5 kPa. Hypotension should be rapidly treated with volume replacement to maintain mean arterial blood pressure > 90mmHg [3]. Standard resuscitation fluids include iso-osmolar crystalloid and/or colloid solutions and blood products. The superiority of colloid or crystalloid for fluid replacement has not been resolved, although there is no advantage of one fluid over another in experimental models of head injury [4]. However, glucose containing solutions should be avoided to minimize the risk of hyperglycemia [5]. Volume resuscitation using hypertonic saline (HS) has been associated with lower ICP than resuscitation with crystalloid or colloid, but a clear benefit on outcome has not yet been demonstrated in clinical studies [6]. Following adequate volume resuscitation, the hypotensive effect of sedative agents may require additional support with modest doses of vasopressors or inotropes. In adults, hypotension is rarely caused by closed head injury alone and another injury site should be sought by a detailed secondary survey. Life-threatening extracranial injuries should be treated prior to definitive neurosurgical treatment but it is sufficient simply to stabilize non-life threatening injuries. Intracranial hypertension is present in around 50% of comatose head-injured patients and, prior to placement of an ICP monitor, it must be assumed that ICP is elevated. This may be temporarily controlled in the acute phase using standard methods such as moderate head-up position, sedation and controlled ventilation. Mannitol (0.5-1.0 g/kg) is also indicated if there are signs of herniation or other neurological deterioration.

Monitoring on the ICU

The aim of the intensive care management of head-injured patients is to anticipate, prevent and treat secondary physiological insults. Specialist neurocritical care, with ICP and cerebral perfusion pressure (CPP) guided therapy as part of a multi-faceted strategy of neuroprotection, is likely to improve outcome after TBI [7].

The measurement of ICP is the cornerstone of modern neurocritical care and the indications for ICP monitoring after TBI are well established [8]. ICP monitoring allows measurement of CPP and detection of abnormal ICP waveforms that occur due to phasic increases in ICP triggered by cerebral vasodilatation in response to a reduction in CPP. Transcranial Doppler ultrasonography (TCD) measures blood flow velocity in the middle cerebral artery and may be used to assess cerebral autoregulation and provide a non-invasive assessment of CPP [9].

Because knowledge of actual CPP does not confirm its adequacy in a particular patient, the simultaneous measurement of cerebral oxygenation is valuable in the management of patients with severe TBI. Regional and global changes in cerebral hemodynamics and oxygenation occur frequently after head injury and often go undetected by standard monitoring techniques [10]. Positron emission tomography and magnetic resonance imaging/spectroscopy offer 'gold standard' measurement of both focal and global ischemic burdens but are not available routinely in many units. Furthermore, they are neither continuous nor bedside techniques. Jugular venous oxygen saturation (SjO_2) monitoring is a bedside measure of the balance between cerebral oxygen supply and demand and may be used to guide therapy. Jugular venous desaturation is a useful indicator of inadequate CPP and $SjO_2 < 55\%$ is associated with worsened neurologic outcome [11]. Because SjO_2 is a 'flow-weighted' global measure, normal or supranormal values cannot exclude regional ischemia. Brain tissue oxygen tension can be measured with miniature sensors inserted directly into the brain. Critical reductions in brain tissue PO_2 have been related to

reduction in cerebral perfusion and associated with adverse outcome but brain tissue PO_2 measurements are hyper-focal and the position of the probe is crucial [12]. Near infrared spectroscopy is a continuous technique that can measure cerebral oxygenation and hemodynamics non-invasively. The contribution of extracranial structures to NIRS measurements is a potential difficulty but modern instrumentation using spatially resolved spectroscopy can make absolute measurements of cerebral oxygen saturation with a high degree of sensitivity and specificity [13]. Cerebral microdialysis is a well-established laboratory tool that is increasingly being used as a bedside monitor to provide on-line analysis of brain extracellular fluid biochemical markers of ischemia [14].

Individual monitoring techniques provide information about specific aspects of cerebral physiology and well-being but all have disadvantages and most suffer from significant artifact. Monitoring of several variables simultaneously (multimodality monitoring) allows cross validation between monitors, artifact rejection and greater confidence to make treatment decisions.

Intracranial hypertension

Intracranial hypertension occurs in 50-70% of patients after evacuation of an intracranial hematoma [15]. Surgically remedial causes of elevations in ICP should be excluded by prompt CT scanning and include post-operative hematoma, progressive focal contusions or hydrocephalus. Other causes of intracranial hypertension include diffuse brain swelling, seizures or systemic complications. Although there are no class 1 data, several clinical studies demonstrate that treatment of ICP > 20 mmHg reduces mortality and improves outcome [16].

Sedation is an essential part of the management of severe TBI and propofol is widely used because its favorable pharmacological profile allows easy titration of sedation levels and rapid wake-up. Propofol, in common with other iv anesthetic agents except ketamine, which should be avoided, causes a dose-dependant reduction in cerebral metabolic rate, CBF and ICP. Although the routine use of barbiturates in unselected patients does not reduce morbidity, they do have a place in the management of refractory intracranial hypertension so long as treatment-related hypotension is avoided [17]. Neuromuscular blocking drugs have no direct effect on ICP but may prevent rises that are caused by coughing or straining on an endotracheal tube. Prolonged use is associated with an increased risk of pulmonary complications and increased length of ICU stay after TBI [18].

Hyperventilation was once the cornerstone of ICP control after TBI but empirical and excessive hyperventilation is associated with adverse neurological outcome [19]. Moderate hyperventilation to $PaCO_2 < 4.0$ kPa may be used to reduce elevated ICP in selected patients but should be guided by simultaneous monitoring of cerebral oxygenation to minimize the risk of secondary ischemic damage [20].

Moderate hypothermia (32-34°C) has been shown to ameliorate neuronal damage in animal models of TBI and several small clinical studies over the last decade have also demonstrated benefit from early and late therapeutic hypothermia [21]. However, a recent randomized multi-center study found no difference in neurological outcomes in matched patients with severe TBI treated within 48 hours of moderate hypothermia compared with those maintained at normothermia but did suggest that rapid re-warming of patients who present with hypothermia may be deleterious [22]. Despite the disappointing results of clinical outcome studies, moderate

hypothermia remains an effective adjunct to controlling elevated ICP. Pyrexia worsens injury in experimental models of brain trauma and fever is common in the neuro-ICU and an independent predictor of poor outcome. The effect of fever control on clinical outcome has not yet been tested, in part because conventional methods of cooling may be ineffective, but treating elevated temperature remains an important part of TBI management on the ICU [23].

Mannitol remains the recommended pharmacologic agent for treating intracranial hypertension but a recent Cochrane review concluded that it was beneficial only in the pre-operative management of patients with acute intracranial hematoma with little evidence to justify its empirical and regular use in patients with raised ICP due to diffuse brain swelling [24]. Recent studies investigating the use of HS solutions have been promising and, in addition to the osmotic properties, HS has hemodynamic, vasoregulatory, immunologic and neurochemical effects [6].

Drainage of CSF via an EVD is an effective means of reducing ICP and decompressive craniectomy is becoming more widely used as another therapeutic option for refractory intracranial hypertension.

ICP vs CPP directed therapy

Over the last decade there has been a shift of emphasis from primary control of ICP to a multi-faceted approach of maintenance of CPP and brain protection. Maintenance of an adequate CPP, either by a reduction ICP or an increase in mean arterial pressure, is the primary focus of treatment after TBI, although the CPP target remains the subject of debate [25]. Aggressive fluid replacement and cardiovascular support with vasopressors and inotropes to augment mean arterial pressure and maintain CPP > 70 mmHg reduces mortality and improves outcome after severe TBI [26]. Although this strategy is associated with a reduction in secondary cerebral ischemia, the mortality and morbidity is similar to other treatment options because of the high incidence of systemic (particularly respiratory) complications [27]. There is now a substantial body of data to justify a CPP target of > 60 mmHg in most cases [25], but optimal CPP should be determined individually for each patient and situation [28]. Both ICP and CPP-directed strategies have a role if applied appropriately and multi-modality monitoring guides tailored therapy.

Systemic complications

Acute lung injury (ALI) is common after TBI and occurs secondary to direct pulmonary injury, aspiration injury, neurogenic pulmonary edema [29] and as a complication of treatment [27]. Pneumonia occurs in over 40% of patients and usually presents some days after injury. Significant cardiovascular complications may also occur and include cardiac arrhythmias, neurogenic hypertension and myocardial ischemia. Supraventricular tachycardia, sinus bradycardia, heart block and repolarization abnormalities, such as T-wave inversion and alterations in the S-T segment, are common features. Myocardial ischemia occurs in about 50% of patients after severe TBI and is likely to be related to hyperstimulation of the sympathetic nervous system [30]. This acute, usually reversible, cardiac injury ranges from hypokinesis with normal cardiac index to low output cardiac failure. Endocrine disturbances, related to both anterior and posterior pituitary insufficiency, may contribute to cardiovascular instability in some patients.

Management of systemic complications presents a significant challenge because established ventilatory strategies for the management of ALI may be inappropriate in some brain-injured

patients. The prone position and permissive hypercapnea are both contraindicated in the presence of intracranial hypertension and fluid restriction to reduce alveolar edema is in conflict with the requirement to maintain cerebral perfusion. The classic teaching of no or low level positive end expiratory pressure (PEEP) to prevent rises in ICP is no longer appropriate because ventilation without PEEP often fails to correct hypoxemia. With adequate volume resuscitation PEEP $<10cmH_20$ does not increase ICP and may in fact result in a decrease because of improved cerebral oxygenation [31]. Hemodynamic instability is a common feature after severe TBI and the initial hyperdynamic phase is often followed by hypotension refractory to fluid resuscitation and catecholamine vasopressors. Such patients may respond to low-dose vasopressin infusion. Good neurologic outcome depends upon the prevention of hypoxemia and hypotension at all stages of management and invasive monitoring, full investigation and appropriate intervention are required early to optimize cardiorespiratory function and minimize the risk of secondary ischemic injury after TBI.

References

1. The Brain Trauma Foundation. Guidelines for the management of severe head injury. American Association of Neurological Surgeons, Joint Section on Neurotrauma and Critical Care. *J Neurotrauma* 1996;13:641-734
2. Maas AI, Dearden M, Teasdale GM, et al. European Brain Injury Consortium – Guidelines for management of severe head injury in adults. *Acta Neurochir (Wien)* 1997;139:286-294
3. Chesnut RM. Avoidance of hypotension: condito sine qua non of successful severe head-injury management. *J Trauma* 1997;42:S4-S9
4. Zornow MH, Prough DS. Fluid management in patients with traumatic brain injury. *New Horiz* 1995;3:488-498
5. Lam AM, Winn HR, Cullen BF, et al. Hyperglycaemia and neurological outcome in patients with head injury. *J Neurosurg* 1991;75:545-551
6. Bhardwaj A, Ulatowski JA. Hypertonic saline solutions in brain injury. *Curr Opin Crit Care* 2004;10:126-131
7. Patel HC, Menon DK, Tebbs S, et al. Specialist neurocritical care and outcome from head injury. *Intensive Care Med* 2002;28:547-553
8. The Brain Trauma Foundation. The American Association of Neurological Surgeons. The Joint Section of Neurotrauma and Critical Care. Indications for intracranial pressure monitoring. *J Neurotrama* 2000;17:479-491
9. Lang EW, Lagopoulos J, Griffith J, et al. Noninvasive cerebrovascular autoregulation assessment in traumatic brain injury: validation and utility. *J Neurotrauma* 2003;20:69-75
10. Coles JP. Regional ischemia after head injury. *Curr Opin Crit Care* 2004;10:120-125
11. Schoon P, Mori LB, Orlandi G, et al. Incidence of intracranial hypertension related to jugular bulb oxygen saturation disturbances in severe traumatic brain injury patients. *Acta Neurochir* 2002;81:285-287
12. Gupta AK, Hutchinson PJ, Al-Rawi P, et al. Measuring brain tissue oxygenation compared with jugular venous oxygen saturation for monitoring cerebral oxygenation after traumatic brain injury. *Anesth Analg* 1999;88:549-553
13. Al-Rawi PG, Smielewski P, Kirkpatrick PJ. Evaluation of a near-infrared spectrometer (NIRO 300) for the detection of intracranial oxygenation changes in the adult head. *Stroke* 2001:32:2492-2500
14. Hutchinson PJ, al-Rawi PG, O'Connell MT, et al. On-line monitoring of substrate delivery and brain metabolism in head injury. *Acta Neurochir Suppl* 2000;76:431-435

15. Miller JD, Becker DP, Ward JD, et al. Significance of intracranial hypertension in severe head injury. *J Neurosurg* 1977;47:503-516
16. Lang EW, Chesnut RM. Intracranial pressure and cerebral perfusion pressure in severe head injury. *New Horiz* 1995;3:400-409
17. Cormio M, Gopinath SP, Valadka A, Robertson CS. Cerebral hemodynamic effects of pentobarbital coma in head-injured patients. *J Neurotrauma* 1999;16:927-936
18. Hsiang J, Chesnut RM, Crisp CB, et al. Early, routine paralysis for ICP control in severe head injury: is it necessary? *Crit Care Med* 1994;22:1471-1476
19. Muizelaar JP, Marmarou A, Ward JD, et al. Adverse effects of prolonged hyperventilation in patients with severe head injury. A randomized clinical trial. *J Neurosurg* 1991;75:731-739
20. Cruz J. The first decade of continuous monitoring of jugular bulb oxyhemoglobin saturation: management strategies and clinical outcome. *Crit Care Med* 1998;26:344-351
21. Marion DW, Penrod LE, Kelsey SF, et al. Treatment of traumatic brain injury with moderate hypothermia. *N Engl J Med* 1997;336:540-546
22. Clifton GL, Miller ER, Choi SC, et al. Lack of effect of induction of hypothermia after acute brain injury. *N Engl J Med* 2001;344:566-563
23. Diringer MN. Treatment of fever in the neurological intensive care unit with a catheter-based heat exchange system. *Crit Care Med* 2004;32:559-564
24. Roberts I, Schierhout G, Wakai A. Mannitol for acute traumatic brain injury. *Cochrane Database Syst Rev 2* 2003:CD001049
25. Robertson CS. Management of cerebral perfusion pressure after traumatic brain injury. *Anesthesiol* 2001;95:1513-1517
26. Rosner MJ, Rosner SD, Johnson AH. Cerebral perfusion pressure: management protocol and clinical results. *J Neurosurg* 1995;83:949-962
27. Robertson CS, Valadka AB, Hannay HJ,et al. Prevention of secondary ischemic insults after severe head injury. *Crit Care Med* 1999;27:2086-2095
28. Hlatky R, Valadka AB, Robertson CS. Intracranial hypertension and cerebral ischemia after severe traumatic brain injury. *Neurosurg Focus* 2003;14:1-6
29. Bratton SL, Davis RL. Acute lung injury in isolated traumatic brain injury. *Neurosurgery* 1997;40:707-712
30. Clifton GL, Robertson CS, Grossman RG. Cardiovascular and metabolic responses to severe head injury. *Neurosurg Rev* 1989;12 (Suppl 1):465-473
31. Huynh T, Messer M, Sing RF, et al. Postive end-expiratory pressure alters intracranial and cerebral perfusion pressure in severe traumatic brain injury. *J Trauma* 2002;53:488-492

Somatosensory Evoked Potential (SSEP) / Motor Evoked Potential (MEP) Monitoring in Scoliosis Surgery

Deborah A. Rusy, M.D. Madison, Wisconsin

OBJECTIVES

After preparing and discussing the case, the PBLD participant will:

1. Understand the basic neurophysiologic principles of Somatosensory Evoked Potential (SSEP) and Motor Evoked Potential (MEP) monitoring.

2. Formulate an anesthetic plan for a patient undergoing scoliosis surgery with SSEP and MEP monitoring.

3. Understand the effects of anesthetics, physiologic changes, and neurologic injury on SSEPs and MEPs.

4. Develop strategies to trouble-shoot and attempt to reverse adverse SSEP and MEP amplitude and latency changes that may occur during routine scoliosis surgery.

STEM CASE - KEY QUESTIONS

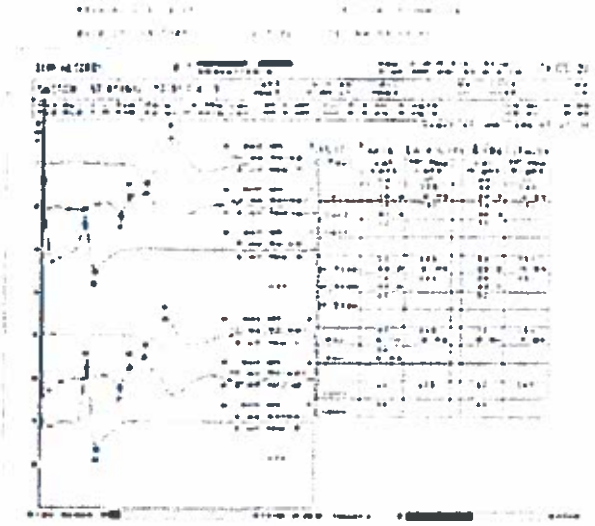
A 12 year-old 50 kg male patient presents for T4-S1 Anterior Spinal Fusion/Posterior Spinal Fusion with Instrumentation and Somatosensory Evoked Potential and Motor Evoked Potential Monitoring.
The patient has a history of mild asthma treated with an Albuterol inhaler on a prn basis, otherwise is in good health.

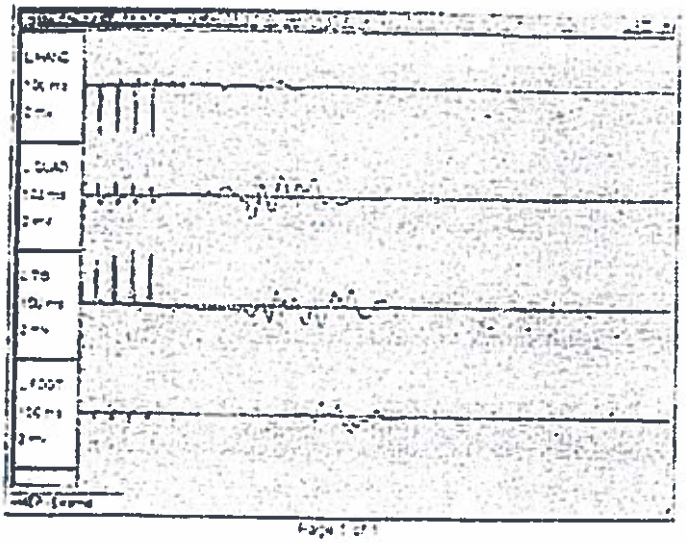
-What are the basic principles of Somatosensory Evoked Potential Monitoring?
-What other options for spinal cord monitoring are available?
-What are the advantages and limitations of SSEP monitoring in comparison to other types of monitoring?
-What are the different classifications of scoliosis, and how might they affect baseline SSEP recordings.
-What are the basic principles of Motor Evoked Potential Monitoring?
-What are the advantages and limitations of MEP monitoring in comparison to other types of monitoring?

The anesthetic plan, SSEP and MEP monitoring, benefits and risks are discussed with the boy's parents prior to surgery.

-What is the justification for performing SSEP monitoring during scoliosis surgery? MEP Monitoring?
-How well does SSEP monitoring detect an injury that may cause paralysis?
-What type of an anesthetic will you use during the case?
-What are the effects of the different anesthetics on SSEPs?
-What are the effects of the different anesthetics on MEPs? Other pharmacological concerns when performing MEP Monitoring?

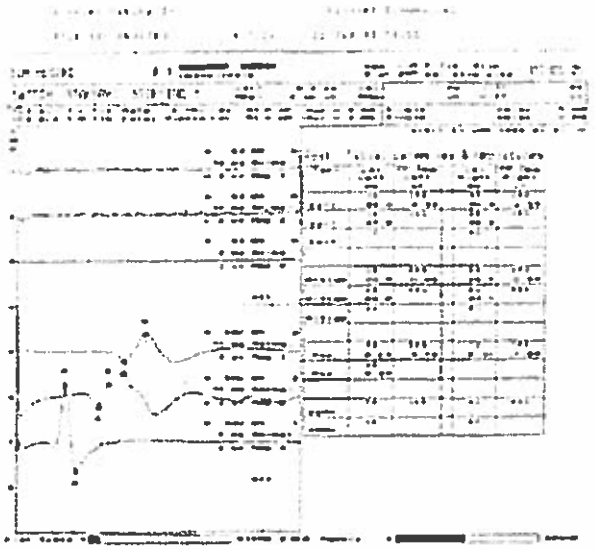
The patient is brought to the operating room and monitors are placed. Induction and intubation are uneventful. A second peripheral I.V. and an arterial line are placed.
SSEP monitors are placed, bilateral baseline SSEP waveforms are collected, and the latencies and amplitudes are recorded.





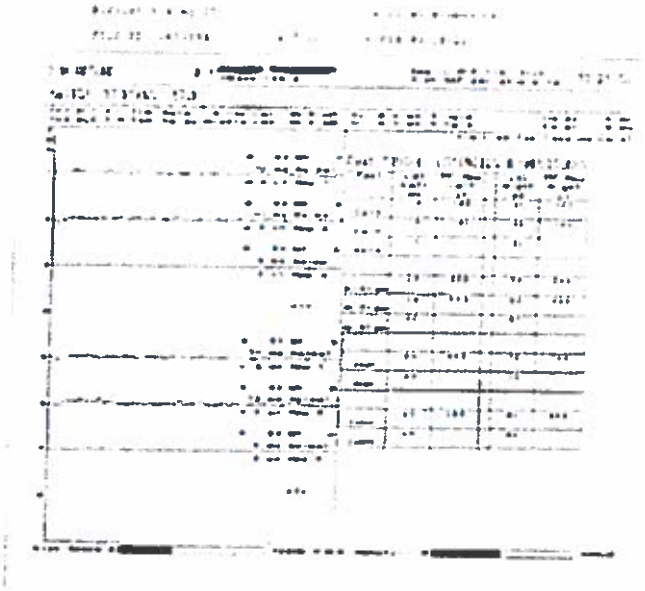
-Where are SSEP stimulating and recording electrodes placed?
-How are SSEP waveforms generated?
-What SSEP waveforms are recorded?
-What are normal SSEP waveform amplitudes and latencies?
-Where are MEP stimulating and recording electrodes placed?
-How do MEPs differ from SSEPs?

Midway during the first part (Anterior Spinal Fusion) of the case, there is a sudden change in the popliteal, brainstem and cortical SSEP recordings on the left.



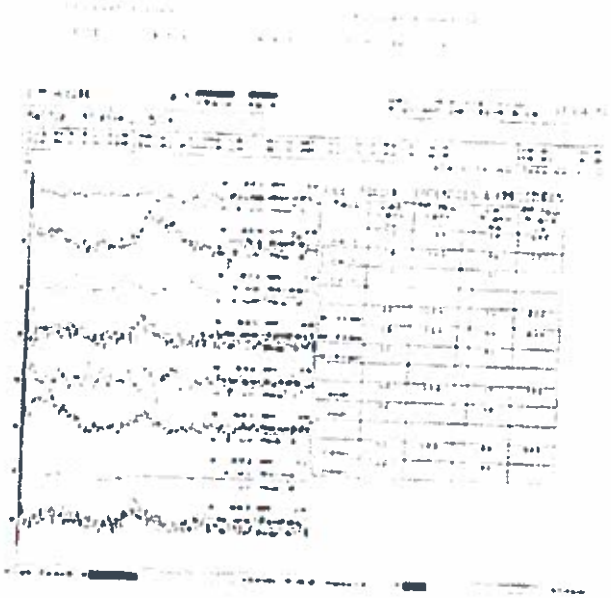
-What are the possible causes for these sudden changes, and how could you correct them.
-What is considered a significant change in latency and in amplitude?

The problem was quickly detected and corrected, with restoration of the baseline SSEP waveforms. The surgeons completed the anterior spinal fusion portion of the case, all monitors and lines except the pulse oximetry probe and a PIV were disconnected, and the patient was then turned to the prone position on a different operating room table.
Pressure points were padded, breath sounds were equal, and monitors and lines were reattached. Baseline SSEP recordings in the prone position were attempted, however all tracings were flat.



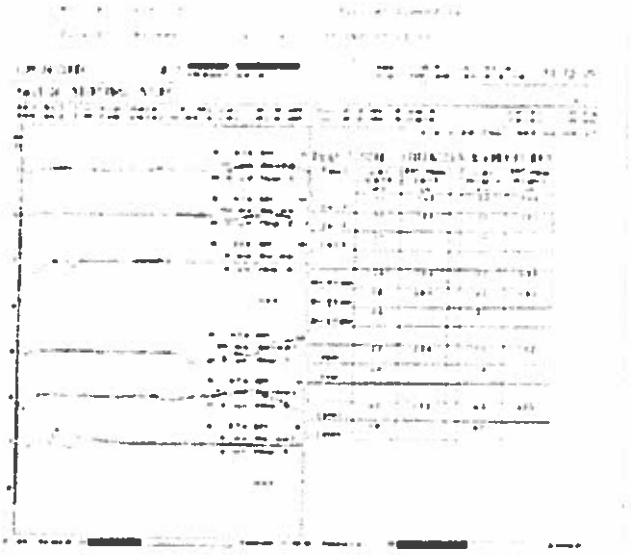
-What are the possible causes of these SSEP changes, and how could you correct them?

Again the reason for the changes was found and corrected, and baseline amplitudes and latencies were restored. However after about 10 minutes the SSEP waveforms suddenly became very jagged, and difficult to read.



-What could be the causes of this?

This artifact lasted for 15 minutes, then spontaneously resolved. Midway through the posterior spinal fusion, again there was a change noted in the SSEP.

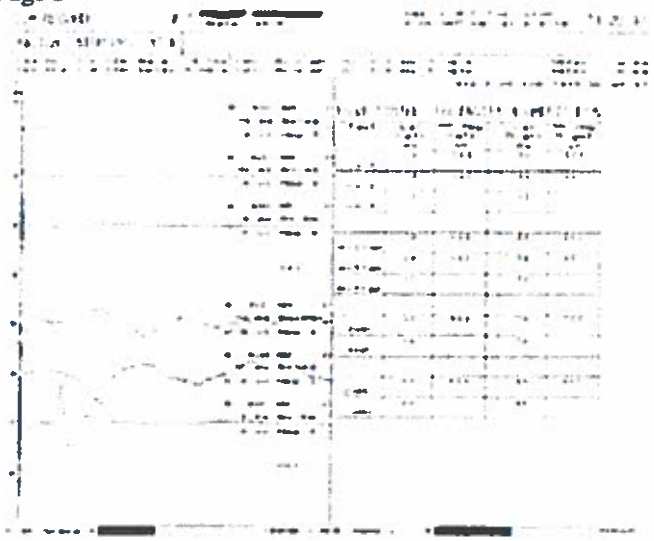


-Now what do you suspect potential causes may be?

The surgeon announces that there is increased bleeding and the blood pressure has fallen to 50/20. The patient is initially very tachycardic, then begins to get bradycardic. A Hematocrit at this time is 12. The patient is quickly resuscitated with fluids (packed red blood cells and fresh frozen plasma), phenylephrine and epinephrine boluses. A normal hematocrit and blood pressure are restored, and 20 minutes later, the SSEP waveform latencies and waveforms return to insignificant changes from the baseline.

-What do you suspect happened to the MEP at the time the patient's Hematocrit was 12?

The surgeon now performs the instrumentation, and while placing a laminar wire there is again a SSEP change.



-Suspected causes?

The surgeon was notified and the laminar wire was removed, however left cortical and brainstem amplitudes and latencies remained significantly changed throughout the remainder of the case. The patient awoke postoperatively with left sided lower extremity paralysis.

PROBLEM BASED LEARNING DISCUSSION

Basic Principles of Somatosensory Evoked Potential Monitoring:

Definition: Electrophysiological Responses of the nervous system to a peripheral motor or sensory stimulus.

There are many different types of Evoked Potentials (EP's), which are distinguished by the various stimulus and recording methods used.

Stimulus Modality and Type:
1. Somatosensory Evoked Potentials (SSEPs)
Assesses the sensory neural axis from peripheral nerve, through the brainstem to the cortex.

Subdivided by location of stimulus:
posterior tibial nerve
median nerve
ulnar nerve
etc.
Subdivided by location of response:
cortical
brainstem
popliteal
Erb's point

Responses to stimuli are carried by the ipsilateral dorsal column, ipsilateral dorsal spinocerebellar tract, and contralateral ventrolateral tracts.

Principle of Averaging:
Recording is dependent on the summation or averaging of responses to a repeated stimulus. Averaging increases the signal-to-noise ratio, allowing one to distinguish the evoked potential signal from background noise such as spontaneous EEG activity, ECG activity, or artifact. Averaging relies on the principle that the electrophysiological response is time-locked to a stimulus, while background noise occurs randomly.

General Characteristics of Evoked Potentials:
The average evoked potential is displayed as a plot of voltage over time.
Latency: the time from stimulus onset to the point of maximum amplitude of a positive or negative peak.
Amplitude: Maximum height of a positive or negative peak.
Waveshape:
Positive or negative polarity:

Stimulus Methods: Electric shocks to a peripheral nerve delivered through surface or needle electrodes.
Stimulus intensity: usually set at 2.5-3.0 times the sensory threshold.
Stimulus duration: usually 200 micro-sec.
Stimulus Rate: 4-7/sec.
responses averaged: 500-2,000.

Recording Methods: Recording electrode surface must achieve a low-impedance connection that can not interact with skin electrolytes. Gold, tin or platinum electrodes are sufficient.
Various Electrode Types: Needle, EEG cup electrode, Adhesive patch electrodes

Electrode Placement: Upper Extremity/Cortical: C3, C4, FPz, Ground
Lower Extremity/Cortical: Cz, FPz, Ground

Other options for spinal cord monitoring:
1. Wake-up Test
2. Motor Evoked Potentials (MEPs)

Advantages and limitations of SSEP monitoring in comparison to other types of monitoring:
Advantages: Provides continuous monitoring of the sensory neurologic pathways during surgery. Decreases the risk for possible accidental extubation, patient awareness, or embolism that may occur with wake-up test.

Disadvantages: Does not have perfect correlation with the motor neurologic pathways.
Expensive equipment.
Need for trained personnel to perform neurophysiologic monitoring.
Needles must be placed into the patient (invasive procedure).
Cortical recordings may be attenuated by certain anesthetics.

Classifications of scoliosis and their effects on baseline SSEPs
Patients with Idiopathic or congenital scoliosis generally have intact CNS pathways with easily achievable spinal, brainstem and cortical SSEPs. In patients with scoliosis secondary to neuromuscular disorders, hereditary degenerative neurological disorders or preoperative paraplegia it may be impossible to obtain any baseline evoked potential. Patients with cerebral palsy or static encephalopathy may have normal spinal or brainstem evoked potentials, however unobtainable cortical waveforms. Prior to surgical incision it is essential to determine that an easily interpreted, reproducible SSEP waveform is present that will allow accurate detection of intraoperative somatosensory changes.

Justification for performing SSEP monitoring during scoliosis surgery:
Studies have shown that SSEP monitoring is predictive of neurologic outcome in spine surgery. The Scoliosis
Research Society performed a multicenter survey in 1995 in which they reviewed over 51,000 surgical spine cases. They found that patients with experienced SSEP neuromonitoring teams had 50% fewer postoperative neurological deficits than patients with inexperienced teams, confirming the clinical efficacy of SSEP recording during Scoliosis surgery. (Nuwer 1995 p. 6-11). Other human studies such as Meyer et al (Meyer 1988), and Epstein et al. (Epstein 1993) also support this conclusion.
Should the surgeon decide prior to the procedure that they will not vary from the operative plan, despite changes seen in intraoperative SSEP recordings, there is no clinical reason to perform intraoperative monitoring.

How well does SSEP monitoring detect an injury that may cause paralysis?
Several animal studies have shown a good correlation of simultaneous changes in both sensory and motor pathways with mechanical cord changes from surgical manipulation.
The Scoliosis Research Society has reviewed over 51,000 human surgical spine cases with SSEP monitoring and found that the occurrence of a motor deficit without SSEP warning ("false negative") was 0.63%. A SSEP change was seen in all other patients who experienced a post-surgical neuro deficit.

Anesthetic Effects on SSEPs:
Drug Amplitude Latency
Thiopental Small/None Increased
Etomidate Increased Increased
Fentanyl Small/None Modest or No Increase
Diazepam Decreased Increased
Midazolam Decreased Increased
Ketamine Increased Increased
Propofol None Increased
Nitrous Oxide Decreased No Change

Volatile Anesthetic Decreased Increased

Other factors effecting SSEPs:
Patient primary disease and physiologic state
Temperature
Nerve Ischemia
Hypoxia
Hypotension
Anemia
Background electrical interference (60 Hz artifact, cautery,)

Placement of SSEP stimulating and recording electrodes:

Normal waveform amplitudes and latencies:

What is significant change in latency and in amplitude?
It is generally felt that when decreases in amplitude of greater than 50%, or increases in latency of greater than 10-15% are seen, and technical, electrical, physiologic and anesthetic causes are ruled out, the surgeon should be immediately notified for possible consideration that spinal cord function has been compromised due to instrumentation.
Nuwer et. al. has found that if evoked potential amplitudes were attenuated greater than 50%, the patient had a high risk for postoperative neurological impairment. (Nuwer 1986 p.81)

Possible causes for sudden SSEP changes:
Surgical
Physiologic
Technical

Motor Evoked Potentials (MEPs)
• Responses of muscle to stimulation of the spinal cord or motor cortex (TcMEP).
• Stimulation to the cortex or cord can be electrical or magnetic.
• Responses are myogenic: achieved with recording electrodes placed over distal peripheral muscles (mMEP), or neurogenic: from distal spinal cord or peripheral nerve (nMEP).
• High amplitude response (mV), which does not require signal averaging.

Transcranial electrical Motor Evoked Potentials (TceMEPs)
• High voltage (up to 750 volts), short duration stimulus
• Depolarization of pyramidal neurons of the motor strip leads to activation of corticospinal tract, alpha motor neurons, peripheral nerve and muscle
• A compound muscle action potential (CMAP) is recorded.
• Disadvantages:
– Very susceptible to anesthetic agents.
– Current can cause neuronal injury
– May see kindling or production of a self sustaining seizure focus.

Transcranial magnetic Motor Evoked Potentials (TcmMEPs)
• A high current (approx. 4,00 amps) is discharged into a coil, which produces a magnetic field.

• This pulsed magnetic field (1.5-2.0 tesla) is applied over the motor cortex.
• This leads to activation of cortical and subcortical neurons.
• Disadvantages:
– Good signals are very difficult to obtain in the OR
– Coil device application may be difficult.
– More suppression by anesthetics than TceMEP.

Anesthetic Effects on MEPs:
• Profound decreases in amplitude, and increases in latency seen with nitrous oxide and volatile anesthetics.
• Little/no change seen with fentanyl and etomidate.
• New studies have suggested Propofol at 150mcg/kg/min or less produces little/no change.

Effects of Anesthetic Drugs on Motor Evoked Potentials
Drug Amplitude Latency
Thiopental Profound Decrease Profound Increase
Etomidate Little or no change Little or no change
Fentanyl Little or no change Little or no change
Diazepam Decreased Increased
Midazolam Decreased Increased
Ketamine Little or no change Little or no change
Propofol None (<150 mcg/kg/min) None (<150 mcg/kg/min)
Nitrous Oxide Profound Decrease Profound Increase
Volatile Anesthetic Profound Decrease Profound Increase

MEP Neurogenic responses:
Recordings from spinal cord (D wave) or a peripheral nerve
Single stimulus (0.5 ms duration, intensity up to 200mamp)
D wave is resistant to anesthetics, temperature
Muscle artifact can distort the signal from a peripheral nerve
Muscle relaxants may improve the quality of the signal
Preservation of 50% amplitude of D wave is predictive of good motor outcome.

MEP Myogenic responses:
Recordings from peripheral muscle
Filters set at 10 and 5000 Hz
Stimulus is constant voltage: usually 150-500V
Multi pulse stimuli (train of 3-9)
Multi-pulse stimulation with a rate of 4.7 Hz, duration of 0.3 msec
Accumulated activity allows alpha motor neurons to reach firing threshold
Suppressed by Volatile anesthetics and Nitrous oxide
Compatible Anesthetic regimen: Propofol (up to 150 mcg/kg/min) and narcotic infusion
Neuromuscular blockade can eliminate the recordable compound muscle action potential (Remifentanil, Fentanyl)
Sensitivity usually between 50-200microV
No averaging needed

Contraindications for MEPs:
Calvarial defects
Epilepsy
Implantable metal near stimulus site

Devices that may be affected by electrical stimulation:
Cardiac Pacemakers
Implanted drug pumps
Dorsal column stimulators

MEP Complications:
Electrical neural injury
Kindling.

REFERENCES

Padberg AM, Thuet ED, Intraoperative electrophysiological monitoring: considerations for complex spinal surgery. Neurosurg Clin N Am.2006 July

Epstein NE, Danto J, Nardi D. Evaluation of intraoperative somatosensory-evoked potential monitoring during 100 cervical operations. Spine 1993;18.

Zouridakis G, Papanicolaou AC, A Concise Guide to Intraoperative Monitoring. 2001, CRC Press, New York, NY

Deletis V, Shil JL, Neurophysiology in Neurosurgery. A Modern Intraoperative Approach. 2002, Academic Press Elsevier Science, New York, NY

Misulis KE, Spehlmann's Evoked Potential Primer. Visual Auditory and Somatosensory Evoked Potentials in Clinical Diagnosis. Second Ed. 1994, Butterworth-Heinmann, Newton, MA.

Nuwer MR, Dawson EG, Carlson LG, Kanim LE, Sherman JE, Somatosensory evoked potential spinal cord monitoring reduces neurologic deficits after scoliosis surgery: results of a large multicenter survey. Electroenceph. clin. Neurophysiol. 1995;96.

Sloan TB, Evoked Potential Monitoring. International Anesthesiology Clinics, 1996, 34;3.

SELECTIVE REFERENCES

Padberg AM, Thuet ED, Intraoperative electrophysiological monitoring: considerations for complex spinal surgery. Neurosurg Clin N Am.2006 July

Zouridakis G, Papanicolaou AC, A Concise Guide to Intraoperative Monitoring. 2001, CRC Press, New York, NY

Deletis V, Shil JL, Neurophysiology in Neurosurgery. A Modern Intraoperative Approach. 2002, Academic Press Elsevier Science, New York, NY.

LEARNING SUMMARY

After preparing and discussing the case, the PBLD participant will:

1. Understand the basic neurophysiologic principles of Somatosensory Evoked Potential (SSEP) and Motor Evoked Potential (MEP) monitoring.

2. Formulate an anesthetic plan for a patient undergoing scoliosis surgery with SSEP and MEP monitoring.

3. Understand the effects of anesthetics, physiologic changes, and neurologic injury on SSEPs and MEPs.

4. Develop strategies to trouble-shoot and attempt to reverse adverse SSEP and MEP amplitude and latency changes that may occur during routine scoliosis surgery.

The Disappearing Waveforms: Cervical Spine Surgery with Evoked Potential Monitoring

Julia I. Metzner, M.D. Seattle, Washington

OBJECTIVES

1) Delineate the basic principles and importance of neuromonitoring during spine surgery.
2) Discuss the impact of anesthetic drugs on the accuracy of neuromonitoring.
3) Discuss airway management options for the cervical spine patient.
4) Understand and manage acute intraoperative evoked potential changes.

STEM CASE - KEY QUESTIONS

A 64-year-old male presents with complaints of progressive loss of coordination in the lower extremities and numbness in the fingers of both hands. Work-up identifies severe cervical spondylotic myelopathy at the level of C4-5, as well as significant cervical stenosis at C5-6 with cord compression. The patient is scheduled for a C4-7 anterior cervical discectomy, instrumentation and fusion with intraoperative SSEP monitoring. His past medical history is relevant only for HTN (BP 150/85) controlled with medication.

1. What are SSEPs, how are they generated and what do they monitor?

2. Which intraoperative SSEP changes should be considered "alarming"?

3. Does the choice of SSEP monitoring influence your anesthetic plan?

On the morning of surgery, after you have just completed your set-up, the surgeon mentions that additional MEP monitoring is planned.

4. What are MEPs and what information do they add over SSEPs?

5. Which anesthetic technique would you choose in this situation? Is one anesthetic technique or drug combination better than all others in facilitating MEP recording?

6. What additional information would you want before proceeding? How would you manage the airway, if the preoperative airway evaluation revealed normal anatomy, but severe neck pain and numbness of both hands on minimal neck extension? Does the approach to intubation affect the extent of vertebral movement?

7. How would you monitor this patient? Would you consider BIS monitoring? Does BIS monitoring have any specific advantages during a TIVA technique as compared to inhalation agents?

After an uneventful anesthetic induction and intubation, the patient is positioned supine with his head placed in a Mayfield head holder. Baseline SSEP and MEP recordings are obtained prior to skin incision. Anesthesia is maintained with the technique discussed and selected during the PBLD session.

The procedure starts and you are on a short break when the surgeon requests you back to the room. You are surprised to hear that during cervical muscle dissection there was a sudden loss of the right upper and lower limb MEPs, accompanied by a decrease in the amplitudes on the left side. The surgeon thinks that something is "wrong" with the anesthesia. The colleague that relieved you previously, informs you that boluses of 100 mg of propofol and 100mcg of fentanyl were given because the patient appeared to be "light" (BIS 62-66).

8. Do you agree with the surgeon? If yes, please explain.

The waveforms return back to baseline over the next 15 minutes and surgery continues uneventfully. Upon bone graft placement the MEP amplitudes from the right hand and foot decrease more than 50%, followed by a decrease in SSEP amplitudes and an increase in latency. At this point your monitors show: MAP 75mmHg, End-tidal CO2 37mmHg, SpO2 98%, T 35.7°C, BIS 48 on continuous infusion of propofol 140 mcg/kg/min/ and remifentanil 0.3 mcg/kg/min.

9. What is the significance of these changes? What are the management options?

10. What physiologic and/or anesthetic causes should be quickly ruled out when EP changes occur?

11. Would it be necessary to perform a wake-up test? If yes, how will you manage the anesthetic?

The patient responded favorably to the corrective interventions. The EPs returned to baseline values and remained stable throughout the rest of the procedure. The patient awoke without any neurological deficit. However, hoarseness was noted immediately after surgery that lasted over the next days and weeks.

12. What is the most likely diagnosis of the patient's hoarseness in this scenario?

13. Are there any known interventions to avoid this complication?

PROBLEM BASED LEARNING DISCUSSION

It is well recognized that surgery on the spine poses a small, but significant risk of injury to the spinal cord. The incidence of severe motor deficit after scoliosis surgery in the absence of spinal cord monitoring has been estimated to be between 4-7%. With the introduction of multimodality monitoring techniques using somatosensory(SSEP) and motor evoked potentials (MEP), this number has decreased to 0.5%.[1] In a review of over 30,000 anterior cervical discectomy procedures, Flynn reported a 0.3% incidence of new neurologic deficits. In the presence of advanced monitoring modalities, this figure may be reduced to 0.1%. [2] The most commonly utilized EPs are those produced by stimulation of the sensory pathways. Somatosensory evoked potentials (SSEPs) are the electrophysiological responses of the nervous system to electrical stimulation. Stimulation of a sensory nerve initiates a chain of electrical events culminating in the SSEP waveform. Typically a peripheral nerve, e.g. the posterior tibial, peroneal, ulnar or median is electrically stimulated, and the impulse is carried through the dorsal columns to the brainstem and the sensory cortex. The response is recorded along several sites of this pathway,

e.g. popliteal fossa, cervical spine, sensory cortex, and after amplification, produces reproducible waveforms that have a characteristic amplitude and latency. During surgery these characteristics are continuously checked and compared to the baseline measurements obtained at the start of the case. No universally accepted criteria exists for what constitutes a significant waveform change, but decreases in amplitude of greater than 50%, or increases in latency of greater than 10%, or both, it is generally accepted to be indicative for ongoing neurological insult and risk for postoperative neurologic deficit.[3]

Most anesthetic agents influence in a dose-dependent manner the sensitivity and reliability of SSEPs in predicting reversible neurologic injury. False positive responses (depressed SSEP with no postop neurologic deficit) were found in 1/ 67 procedures.[1] There is evidence that high dose volatile anesthetics markedly depress the SSEP waveform, therefore the use of more than 1 MAC iso-, sevo, or desflurane is not recommended. [1,3,4] Nitrous oxide acts synergistically with the halogenated agents, consequently is better to avoid it, or reduce the other volatile anesthetics to 0.5 MAC or less. TIVA (total intravenous) techniques have minimal effect on SSEP recordings. Combinations of propofol/opioid or midazolam/low dose ketamine/opioid has been used successfully in many cases. Moreover many studies demonstrated that etomidate and ketamine increase the amplitude of SSEPs and could facilitate monitoring in patients with otherwise unrecordable waveforms.[3,4] Etomidate infusions however have been shown to cause significant adrenal suppression and ketamine may cause psychomimetic effects at higher doses. Neuromuscular blocking agents do not interfere with SSEP recording, in fact it has been suggested that with the reduction of artifacts, recording can be improved.

Since ascending sensory signals are carried mainly in the posterior spinal cord, while descending motor control is carried mainly in the anterior spinal cord, it is possible to have no detectable change in SSEPs and still have a significant motor injury. Because of this possibility, motor evoked potentials (MEPs) are increasingly used concomitantly with SSEPs to assess the integrity of the neural pathways during procedures such as removal of spinal cord tumors, correction of scoliosis, aortic aneurysm repair, and cervical spine surgery. Several MEP monitoring techniques have been developed in the past 10 years. The method most commonly used in the US consists of transcranially stimulating axons of the motor cortex with high frequency electrical pulses, and recording the compound muscle action potentials (CMAPs) at targeted muscles in the hand or foot. The advantage of this technique is that it monitors the entire motor tract from the cortex down to the neuromuscular junction, including the ischemia-sensitive anterior horn. The major disadvantage is the intense inhibitory effect of many anesthetics on MEP recordings, limiting anesthetic choices and making it difficult to choose standard warning criteria for amplitude and latency changes.

MEP recordings are extremely inhibited by the most commonly used anesthetic drugs. Volatile agents in doses less than 0.5 MAC may render the MEPs unrecordable. Even multi- pulse stimulation cannot overcome this anesthetic suppression, therefore some authors recommend excluding these agents when MEP recording is planned. Nitrous oxide appears to be less suppressive, particularly when supplemented solely with opioids. Although some studies reported that propofol in a dose of 75-100 mcg/kg/min produces CMAP depression in a range from 33% to 83%, many centers (including ours) achieve good MEP signals using TIVA with propofol/opioid combination. Midazolam, and certainly ketamine, and etomidate have less effect on MEPs, but the well known side effects limit their use in clinical practice. Although not

contraindicated, the use of neuromuscular blockade (NMB) needs careful titration and monitoring. Most authors recommend continuous NMB infusion, titrated to correspond with one or two twitches in a TOF.[4] In summary, maintenance of a stable anesthetic technique with low dose propofol/opioid supplemented with low dose ketamine may provide the best results for MEP recording.[5,6]

In addition to routine monitors, many anesthesiologists will use an arterial line for accurate MAP control, essential in maintaining adequate spinal cord perfusion. Because no major fluid shift is anticipated during this surgery, use of a central venous line is not needed.

During inhalational anesthesia end-tidal inhaled agent (MAC) monitoring may ensure adequate depth of anesthesia, whereas IV anesthetic concentration cannot be measured in real time. The BIS index is an empirically derived measure of hypnotic drug effect on the CNS and has been shown to reflect the anesthetic depth particularly during propofol induced hypnosis. BIS values of 40-60 may be effective in reducing the potential of intraoperative awareness. Furthermore, several studies and a recent meta-analysis demonstrated that judicious, BIS guided titration of anesthetics can reduce drug overdose and facilitate early emergence, a major advantage in spine surgery if a wake-up test would be required. [7]

Optimal airway management of the patient with severe cervical spine disease is a topic of ongoing discussion in the literature. In general, if there is no spinal cord compression or cervical spine instability, then no special airway techniques are indicated. In the presence of severe cord compromise by acute or chronic conditions, the optimal intubation technique, e.g. awake vs. asleep, conventional laryngoscopy vs. fiberoptic (FOB) scope is controversial. Crosby recently reviewed the literature in an attempt to identify the safest techniques for airway management in patients at risk for C-spine injury. [8] Based on studies evaluating cervical spinal motion and different intubation characteristics with different intubation tools, he concluded: all airway maneuvers cause some degree of cervical spine movement, the greatest motion of the intact and also injured spine occurs at the craniocervical junction; indirect laryngoscopes, such as the Bullard or light wand causes less motion compared with direct laryngoscopy (DL). However, evidence supports that even with DL, these movements are minimal and unlikely to induce neurologic injury. Therefore, use of DL with manual in-line immobilization is acceptable in uncooperative patients, or whenever emergent airway management is necessary. Although, there is no data that intubation with a FOB results in better neurologic outcomes, Crosby's review reveals that the majority of American anesthesiologists would prefer this option when intubating elective patients at risk for cervical spine injury. Both awake intubation and intubation after induction of general anesthesia have been used safely. Advantages of awake intubation include: maintenance of normal muscle tone, easy access in patients wearing halo vests, and most importantly confirmation of intact neurologic status after intubation and/or positioning. Disadvantages are: time consuming airway topicalization, and the need of patient cooperation throughout the procedure. In summary, no single best method exists to secure the airway in cervical spine disease patients. Judicious planning and attention to minimize cervical spine motion during airway maneuvers are more important to success than choice of a particular technique.

Once loss of intraoperative EP occurs, it is important to correct any contributing factors as soon as possible. Surgical maneuvers, such as distraction, discectomy, plate, and graft placement are

the most common causes of surgery related alerts. It seems unlikely that abrupt changes would occur with muscle dissection. On the other hand, there is evidence that bolus administration of IV anesthetics can cause long-lasting (15-20 min) reduction or loss of MEP responses and should be avoided. [5] For that reason, it should not be surprising if the depicted changes were indeed the result of bolus dosing. There is not much data available in the literature regarding how to deal with such cases. A good way to deepen anesthesia but avoid waveform suppression would be to give a small dose of midazolam or ketamine, drugs not associated with MEP depression.

The loss in amplitude of more than 50% should be considered as a sign of evolving spinal cord injury, a situation that demands prompt response before the damage becomes permanent. (in animal studies irreversible changes occurred in less than 10 min!). Experience and many studies have shown that intraoperative EP changes may be the consequence of surgical trauma, spinal cord ischemia, hypotension, anesthetic drugs, and other variables. Teamwork and communication between surgeon, monitoring staff and anesthesiologist is essential to identify and correct the precipitating event. If surgical and technical factors have been ruled out, it is the anesthesiologist's responsibility to rule out hypotension, hypothermia, low Hgb, severe hypoxemia, and anesthetic effects as causative factors. Control of the blood pressure is critical to maintain the perfusion pressure of the spinal cord, especially during surgical maneuvers such as decompression or grafting. Many authors agree that reliable EPs should be recorded at MAP of 60-70mmHg. [1,5] But what to do in the situation when the MAP is over the magic 60-70 mmHg? There is no definitive answer in the literature. However recent studies showed improvement, even reversal in amplitude depression, when BP was increased by 20-30% above the value at which the initial changes occurred. [9] For this reason, the patient's BP should be increased to his pre-op values (150/85mmHg). Another option is to change the anesthetic regimen to one less suppressive such as etomidate/opioid or ketamine/low dose propofol. Although controversial, some centers administer steroids for spinal cord protection.

If all these interventions failed and there is no improvement in amplitudes, one should decide whether to perform a wake-up test. The test consists of lightening the anesthetic state to a point at which the patient can follow commands. A good method is: stop propofol or inhalational agent but keep remifentanil on a minimum infusion rate to avoid pain with awakening .Hold the endotracheal tube and head securely, and be ready to reinduce anesthesia as soon as limb movement is confirmed. With this technique awakening should occur in less than 10 min. One recent study reported an awakening time of less than 6 min with the use of desflurane /remifentanil regimen. [10] The wake-up test has many potential hazards, such as: recall, accidental extubation, or loss of IV lines. Moreover, it does not pinpoint the time of onset of neurologic injury and at best it shows that damage has occurred- not that is occurring. Ultimately, to perform or not the wake-up test will depend on how stable the spine is and on the comfort level of the surgical team.

At our institution we have developed a clinical protocol for the occurrence of intraoperative EP changes:

1) Rule out surgical factors: stop surgical maneuvers

2) Rule out technical causes (loose, dislodged electrodes); increase stimulation intensity

3) Rule out physiologic causes: low MAP, hypoxemia, hypothermia, and anemia

MOST IMPORTANT: increase MAP 20-30% above baseline (even when normal)

4) Stop volatile agents, switch to TIVA, and consider etomidate or ketamine

5) Consider wake-up test

6) Consider steroid infusion

Hoarseness is attributable to transient vocal cord palsy, a relatively common complication following anterior cervical spine procedures. It is speculated to be the result of intraoperative recurrent laryngeal nerve (RLN) injury, the precise mechanism of which is debated. The most likely explanation is pressure on the RLN by the tracheal tube following surgical retractor placement. In his study, Apfelbaum demonstrated that deflating the cuff after retractor placement with subsequent reinflation to just-seal pressure, reduces the incidence of transient vocal cord palsy from 6.4% to 1.7%. [11] In most references, RLN palsy is reported as being transient with resolution of the symptoms within weeks or months. The management is conservative and consists in follow-up and speech therapy.

References

1. Raw DA, Beattie JK, Hunter JM. Anesthesia for spinal surgery in adults. BJA 2003; 91: 886-904.
2. Lee JY, Hilibrand AS, Vaccaro AR,Albert TJ. Characterization of neurophysiologic alerts during anterior cervical spine surgery. Spine 2006; 31(17): 1916-21
3. Banoub M,Tetzlaff JE, Schubert A. Pharmacologic and physiologic influences affecting sensory evoked potentials: implications for perioperative monitoring. Anesthesiology 2003; 99(3):716-737.
4. Sloan TB, Heyer EJ. Anesthesia for intraoperative neurophysiologic monitoring of the spinal cord. J Clin Neurophysiol. 2002; 19(5) 430-443.
5. Lotto ML, Banoub M, Schubert A. Effects of anesthetic agents and physiologic changes on intraoperative motor evoked potentials. J Neurosurg Anesthesiol.2004; 16(1):32-42.
6. Kawaguchi M, SakamotoT, Takanori I. Low dose propofol as a supplement to ketamine-based anesthesia during intraoperative monitoring of motor evoked potentials. Spine 2000; 25(8): 974-79
7. Liu SS. Effects of Bispectral index monitoring on ambulatory anesthesia: a meta-analysis of randomized controlled trials and a cost analysis. Anesthesiology 2004; 101:311-315.
8. Crosby ET. Airway management in adults after cervical spine trauma. Anesthesiology 2006; 104:1293-318.
9. Hilibrand AS, Schwartz DM, Sethuraman V, Vaccaro AR, Albert TJ.Comparison of transcranial electric motor and somatosensory evoked potential monitoring during cervical spine surgery. J Bone Joint Surg Am. 2004; 86(6):1248-53.
10. Grottke O, Dietrich PJ, Wappler F. Intraoperative wake-up test and postoperative emergence in patients undergoing spinal surgery. Anesth Analg. 2004; 99(5):1521-7
11. Apfelbaum RI, Kriskovich MD, Haller JR. On the incidence, cause, and prevention of recurrent laryngeal nerve palsies during anterior cervical spine surgery. Spine 2000; 25:2906-12.

SELECTIVE REFERENCES

1. Banoub M,Tetzlaff JE, Schubert A. Pharmacologic and physiologic influences affecting sensory evoked potentials: implications for perioperative monitoring. Anesthesiology 2003; 99(3):716-737.
2. Raw DA, Beattie JK, Hunter JM. Anesthesia for spinal surgery in adults. BJA 2003; 91: 886-904.
3. Lotto ML, Banoub M, Schubert A. Effects of anesthetic agents and physiologic changes on intraoperative motor evoked potentials. J Neurosurg Anesthesiol. 2004; 16(1):32-42.

Drug advisory for spine surgery with neuromonitoring at our institution

DRUGS	SSEP	MEP	OBSERVATION
INHALATIONAL:			
ISO, SEVO, DES	*≤1 MAC	NC	
Nitrous Oxide	NC	NC	
INTRAVENOUS:			
PROPOFOL	YES: I+M	YES: I+ MM< 150mcg/kg/min: *TIVA: basic component	
BARBITURATE	YES: I	YES: I	
BENZODIAZEPINES	PREOP	PREOP	
ETOMIDATE	YES: I+M	YES: I+M	Limited: adrenocortical supression
KETAMINE	YES: I+M	YES: I+M	Limited: dissociative effect
OPIOIDS	YES	YES	* TIVA: basic component
NMB (RELAXANTS)	YES: I+M	YES: I	Short acting drugs
DEXMEDETOMIDINE	ADJUVANT to TIVA : 0.2-0.4 mcg/kg/min		

NC: non-compatible; (I): induction; (M): maintenance; MAC: minimal alveolar concentration

LEARNING SUMMARY

1) Delineate the basic principles and importance of neuromonitoring during spine surgery.
2) Discuss the impact of anesthetic drugs on the accuracy of neuromonitoring.
3) Discuss airway management options for the cervical spine patient.
4) Understand and manage acute intraoperative evoked potential changes.

Carotid Endarterectomy – I Thought That Was Replaced by Carotid Stenting – Not yet!!!!!!!

Anthony N. Passannante, M.D. Chapel Hill, North Carolina

OBJECTIVES

1. Learn the indications for carotid endarterectomy in 2007
2. Learn the indications for carotid stent placement in 2007
3. Review anesthetic issues relevant to both endarterectomy and stenting
4. Discuss regional and general anesthesia for carotid endarterectomy.

STEM CASE - KEY QUESTIONS

You are interviewing a 65-year-old man in preoperative clinic. He has hypertension, COPD (100 pack-year smoking history), non-insulin dependent diabetes, and a myocardial infarction one year prior to admission treated with a drug-eluting stent. His past surgical history is notable for a C6-C7 cervical fusion five years ago. He presents now with symptomatic TIA's, a 70% occlusion of his right internal carotid artery, and a complete occlusion of his left internal carotid artery. Physical examination reveals a 5'9" 100kg male with limited neck mobility and diffusely decreased breath sounds, frequent bouts of coughing, but otherwise a normal physical examination. His current medications include glyburide, hydrochlorothiazide, lovastatin, clopidogrel, and an albuterol inhaler prn. He is scheduled to undergo carotid endarterectomy, but he is very frightened of anesthesia. He asks you about the risks of anesthesia for this procedure, and he asks your advice about traveling to another institution to have a carotid stent placed instead of undergoing an open surgical procedure.

Key Questions:

1. Is further preoperative evaluation necessary?
2. Does preoperative evaluation differ for general versus regional anesthesia?
3. Is Carotid Endarterectomy (CEA) or Carotid Stenting (CAS) indicated?
4. What surgical and medical factors determine the appropriate type of revascularization procedure (CEA vs CAS)
5. Are cardiovascular and neurologic outcomes different between the two procedures?
6. What are distal embolization protection devices and are they necessary for CAS?

As it turns out he has a TIA while leaving the clinic and your vascular surgeon insists that CEA be performed the next morning. The patient has recovered from his TIA (amaurosis fujax) and he strongly prefers general anesthesia. The surgeon strongly prefers regional anesthesia. The three of you agree on a general anesthetic, and an arterial line is inserted before induction, revealing a blood pressure of 165/94mmHg. Anesthesia is induced with thiopental, lidocaine, fentanyl, and vecuronium. Blood pressure spikes to 210/117mmHg with intubation, and drops to 110/55mmHg 10 minutes after intubation. Dissection of the carotid artery proceeds without incident. The surgeon asks you to measure a stump pressure and it is 55mmHg. Surgery proceeds without the placement of a shunt and a patch is sewn in place after the endarterectomy. Clamp time is 45 minutes. After removal of the clamp blood pressure decreases to 90/60mmHg. He coughs vigorously on emergence and a moderate amount of blood is noted seeping through the surgical dressing. He is taken to recovery room where blood pressure remains in the 90's systolic and he complains of moderate postoperative pain.

Key Questions (continued):

5. Is regional anesthesia safer than general anesthesia for CEA?
6. If regional anesthesia was chosen, what block would you perform?
7. Does his smoking history lead you towards regional or general anesthesia?
8. How would you manage his blood pressure if you performed general anesthesia?
9. How would you manage his blood pressure if you performed regional anesthesia?
10. What management issues frequently arise when CEA is performed under general anesthesia?
11. What management issues frequently arise when CEA is performed under regional anesthesia?

PROBLEM BASED LEARNING DISCUSSION

The question of whether or not additional preoperative evaluation is necessary is a complicated one. It is unlikely that additional preoperative workup would uncover a condition amenable to intervention that would be of higher risk to this patient than his symptomatic carotid disease, and he does not appear to have cardiovascular issues severe enough (acute coronary syndrome, decompensated congestive heart failure, significant valvular disease, or significant arrhythmia) to suggest that cardiology consultation/intervention is necessary before his carotid disease is addressed. Certainly his diabetes and severe lung disease should be aggressively treated, and proper perioperative management of his coronary disease includes perioperative sympatholysis (he may be an individual to treat with alpha-agonists rather than beta-blockers due to his lung disease), continuation of his statins, and probable continuation of his clopidogrel due to his drug-eluting stent. Preoperative evaluation does not differ based on whether regional or general anesthesia is planned.

The question regarding the type of surgical intervention that is most appropriate is one that is not typically posed to anesthesiologists, and it is a difficult question to answer even if you are an expert in vascular or neuroanesthesia. We are certainly obligated to be honest with our patients, and your institution may have outstanding expertise in vascular surgery and not much in interventional neuroradiology, or vice-versa, which would influence your response. Clinical practice in the area of carotid artery disease is evolving extremely rapidly, and is being driven both by both reimbursement patterns and improving clinical evidence. An added complexity in determining best currently available therapy is the rapidity of evolution in technology available in this area, and the slowness with which RCT (randomized controlled trial) data will emerge. The patient in the stem has symptomatic carotid disease, and is at high surgical risk based on the contralateral carotid occlusion, the severity of his coronary disease with the presence of a drug-eluting stent, his limited neck mobility, and perhaps the severity of his pulmonary disease. Clopidogrel treatment, which will be difficult to interrupt due to his active TIA's and coronary stent, may increase bleeding complications from CEA. Current AHA (American Heart Association) guidelines suggest revascularization in this patient based on his recent TIA and ipsilateral high-grade carotid stenosis. While practice patterns certainly differ, many would suggest that in 2007 either CAS or CEA would be a reasonable choice for this patient, with many leading towards CAS due to his anatomic and surgical risk factors. In most institutions anesthesiology involvement in carotid artery stent placement is minimal or absent, and if stent placement were planned this patient would most likely be taken care of by an interventional radiologist, a vascular surgeon, or a cardiologist credentialed to place carotid stents. In this circumstance, minimal sedation would be administered as per unit protocol, and the procedure

would typically be performed via percutaneous access to the circulation through a femoral artery. While hemodynamic instability has been reported during carotid stenting, usually mediated by bradycardia, adverse outcomes from such transient instability have been infrequent.

The surgical and medical factors that determine the most appropriate type of intervention (CEA vs CAS) are numerous. Anatomic considerations that predict high-risk from CEA include limited neck mobility, very high or very low carotid stenosis, previous radiation therapy or radical surgery to the neck, prior ipsilateral CEA, contralateral carotid occlusion, contralateral laryngeal nerve palsy, and a tracheostomy. Medical factors which predict high risk from CEA include age 80 or older, Class III/IV CHF, Class III/IV angina pectoris, left main coronary disease, need for urgent heart surgery, LVEF 30% or less, recent MI, severe lung disease, and severe renal disease. It would be reasonable to suggest CAS in patients with one or more of the above risk factors, and especially in patients with both anatomic and medical issues that confer a high degree of risk.

It is difficult to be dogmatic about outcome until more data from RCT's are available. Data from registries is frequently contradicted by later RCT data, and if CAS is performed on patients with low risk lesions, adverse events should be low. The most recent widely cited data comparing CEA with CAS is the EVA-3S non-inferiority trial from France, conducted in patients with severe carotid stenosis, which was stopped prematurely due to increased adverse events in the CAS group (6-month stroke and death combined 6.1% from CEA vs 11.7% from CAS). To be fair to CAS enthusiasts, embolic protection devices were not routinely used in this study until about halfway through the study period. The SAPPHIRE RCT results supported CAS in patients at high risk for CEA, with composite one month MI, stroke, death rate 4.8% after CAS versus 9.8% in CEA (p=0.09). Six RCT's are currently ongoing, and will provide additional guidance as results are reported over the next several years. As our knowledge base improves, and equipment continues to evolve, the indications for CAS placement may well increase, but it now appears that this will be an evidence driven process.

Regional versus general anesthesia for CEA has stimulated much debate over the years. There is little doubt that an awake patient is the best neurologic monitor available, but there is also little doubt that not every patient is a suitable candidate for CEA under regional anesthesia, and that not every surgeon enjoys performing CEA on awake patients. With an appropriate patient and a cooperative or better surgeon, neurologic outcome may be marginally better with a regional technique, as it allows for the placement of fewer shunts and unsurpassed neurologic monitoring. The patient in this case has a cough and severe pulmonary disease, which will complicate management regardless of the type of anesthetic chosen. Some patients with severe pulmonary disease have great difficulty lying flat and still for the length of time necessary for CEA, which would specifically complicate regional anesthesia. On the other hand regional anesthesia offers the opportunity to perform a CEA without airway instrumentation, which certainly has the potential for minimizing coughing. Current evidence supports utilization of a superficial cervical plexus block to obtain adequate surgical operating conditions for CEA, as the risks of this block are fewer than deep cervical plexus block, and operating conditions are equivalent. The question of sedation during carotid endarterectomy is somewhat controversial. The level of sedation needs to be light enough to permit the patient to function as an unsurpassed neurologic monitor, and this can be accomplished with a number of different sedation techniques. Important questions to consider when planning carotid endarterectomy under regional anesthesia include backup plans

for airway management if the patient can no longer tolerate the procedure or if cerebral ischemia makes the patient uncooperative, how long the procedure will last, how anxious the patient is preoperatively, and whether or not the patient suffers from claustrophobia. Success with a regional technique is much easier to achieve if surgery proceeds expeditiously, if the surgeons are gentle and accustomed to working under regional anesthesia, if patient anxiety is minimal, and if the patient does not suffer from claustrophobia. While this may seem like a long list of preconditions, many patients are excellent candidates for regional anesthesia for CEA, and many centers utilize regional for CEA extensively. Management issues that arise only in patients under regional anesthesia include plans for a failed or inadequate block, how you will handle anticoagulant therapy, how you will maintain patient comfort (patients often feel hot), how you will manage adverse reactions from sedation, and management of the rare immediate adverse neurologic event.

Blood pressure will need to be managed regardless of the type of anesthetic administered. When regional anesthesia is chosen the awake patient provides evidence of adequate cerebral perfusion after carotid cross-clamping, so there may be no need for elevation of blood pressure during clamping to increase collateral perfusion. With general anesthesia hypotension must be very carefully avoided around the time of carotid clamping, for the reason listed above. With either anesthetic hypertension after clamp release should be avoided, and it is usually easier to avoid with regional anesthesia.

Overall general anesthesia is used more frequently for CEA and there is no doubt that excellent results can be obtained with carefully performed general anesthesia. When considering GA for CEA consideration must be paid to maintenance of hemodynamic stability (which will most likely require some work), management of blood pressure during carotid clamping, and how the adequacy of cerebral perfusion is going to be monitored or ensured during carotid clamping. Surgical practice with regards to indication for carotid shunt placement varies, with many surgeons currently utilizing some measure of adequacy of collateral perfusion such as measurement of carotid stump pressure to guide placement of shunts. If this is done reasonable evidence supports using 40mmHg stump pressure as a threshold below which a shunt will be inserted when general anesthesia is utilized. All modern volatile anesthetics reduce cerebral metabolic rate, but many would chose a rapidly eliminated drug to allow early neurologic examination. It is unlikely that the choice of general anesthetic agent would affect outcome from CEA. When CEA is performed under GA, a wide variety of strategies can be employed to guide assessment of the adequacy of cerebral perfusion and whether or not cerebral ischemia is occurring. There is not enough evidence to declare a single method or strategy standard of practice, but an understanding of the relevant issues is mandatory. It is reasonable to expect collateral cerebral perfusion to be improved with higher stump pressures, as it is reasonable to expect global cerebral oxygenation to be better with reassuring EEG, processed EEG, or cerebral oximetry readings. Unfortunately, none of these techniques can reliably identify of prevent embolic events, which are responsible for significant morbidity after CEA, and may even be increased by some techniques to increase cerebral perfusion, such as carotid shunt placement. Blood pressure management after removal of the carotid clamp may be problematic. There is no longer a need to increase blood pressure to improve collateral perfusion, and increased blood pressure may well cause increased surgical bleeding. Some patients will become franky hypotensive, which is probably mediated by stimulation of recently uncovered carotid baroreceptors, and it is not unheard of for an occasional patient to require phenylephrine infusion

to maintain adequate blood pressure in the recovery room. Postoperative pain is not typically a major problem after CEA, but superficial cervical plexus block can improve pain control and reduce narcotic analgesic requirements, which may improve the sensitivity of early neurologic examination. Management issues that arise only under general anesthesia include more frequent interventions to stabilize blood pressure, airway management issues around intubation and again around extubation, a strategy to minimize coughing and hemodynamic instability around the time of emergence, and a strategy for cerebral perfusion during carotid clamping that does not use an awake patient as a neurologic monitor.

CAS is a new procedure (FDA approval only granted in 2004), and expertise with CAS is rapidly increasing, and the equipment available for CAS is improving at a rapid rate. Many of the initial studies of CAS utilized unprotected devices which were not able to minimize the spread of atheromatous debris created by treatment of the carotid stenosis. Experience with the technique and the obviousness of the potential problem has led to the development and widespread utilization of embolic protection devices. These devices are designed to trap plaque debris, rather than allowing it to spread into the cerebral circulation and perhaps cause an embolic stroke. While there is no Class 1 evidence to support the use of embolic protection devices (EPD) during CAS placement, their use makes intuitive sense, debris is usually caught in them, and everyone is using them. EPD's do not eliminate the possibility of embolism, they do require the placement of a larger sheath, and they add complexity and can potentially lead to stent dislodgement, but all current RCT's involving CAS utilize EPD's, so the forthcoming data we have to analyze will have them as a defacto standard.

The last comment to make is that the Center for Medicare Services (CMS) has published credentialing requirements for Institutions to meet if Medicare reimbursement is expected for the placement of CAS. CMS will currently reimburse for CAS placement if a patient has a symptomatic carotid stenosis (70% or greater) and is at high risk for CEA. For CAS placement in symptomatic patients with lower grade (50-69%) stenosis, reimbursement is only available if the patient is enrolled in an FDA approved registry or clinical trial. CMS reimbursement is not available for CAS placement in asymptomatic patients. Since the most common indication for carotid artery revascularization is not symptomatic TIA's but is rather asymptomatic carotid stenosis (this leads to 80-90% of carotid interventions) and reimbursement for CAS is restricted by CMS for this group of patients, CEA will be around for several years at least. It is likely that aggressive treatment of atherosclerosis with statins and widespread implementation of perioperative sympatholysis has reduced peri-procedural cardiovascular morbidity/ mortality, leading to improved outcome overall from CEA, which will mean that CAS will have to improve significantly to gain widespread acceptance as a treatment for those at low risk from CEA.

REFERENCES

1. Narins CR, et al: Patient selection for carotid stenting versus endarterectomy: A systematic review. J Vasc Surg 44:661-672, 2006.
2. Bates ER et al. ACCF/SCAI/SVMB/SIR/ASITN 2007 Clinical Expert Consensus Document on Carotid Stenting. JACC Vol 49(1):126-70, 2007.
3. Iihara K, et al: Outcome of carotid endarterectomy and stent insertion based on grading of carotid endarterectomy risk: A 7-year prospective study. J Neurosurg 105:546-554, 2006.

4. The SPACE Collaborative Group: 30 day results from the SPACE trial of stent-protected angioplasty versus carotid endarterectomy in symptomatic patients: A randomized non-inferiority trial. Lancet 368:1239-1247, 2006.
5. Mas J-L, et al: Endarterectomy versus stenting in patients with symptomatic severe carotid stenosis. NEJM 355 (16):1660-1671.
6. Park B, et al: Clinical outcomes and cost comparison of carotid artery angioplasty with stenting versus carotid endarterectomy. J Vasc Surg 44:270-276, 2006.
7. Kastrup A, et al: Comparison of angioplasty and stenting with cerebral protection versus endarterectomy for treatment of internal carotid artery stenosis in elderly patients. J Vasc Surg 40:945-951, 2004.
8. Bush, RL, et al: A comparison of carotid artery stenting with neuroprotection versus carotid endarterectomy under local anesthesia. Amer J Surg 190:696-700, 2005.
9. Yadav JS, Wholey MH, Kuntz RE et al. Protected carotid-artery stenting versus endarterectomy in high-risk patients. N Engl J Med 351:1493-1501, 2004.
10. Calligaro KD, Dougherty MJ. Correlation of carotid artery stump pressure and neurologic changes during 474 carotid endarterectomies performed in awake patients. J Vasc Surg 42(4): 684-9, 2005.
11. Bellosta R, Luzzani L, Carugati C et al. Routine shunting is a safe and reliable method of cerebral protection during carotid endarterectomy. Ann Vasc Surg 20(4):482-7, 2006.
12. Pandit JJ, Brees S, Dillon P et al. A comparison of superficial versus combined (superficial and deep) cervical plexus block for carotid endarterectomy: a prospective, randomized study. Anesth Analg 91(4): 781-86, 2000.
13. Messner M, Albrecht S, Lang W et al. The superficial cervical plexus block for postoperative pain therapy in carotid artery surgery. A prospective randomized controlled trial. Eur J Vasc Endovasc Surg 33(1): 50-4, 2007.
14. Watts K, Lin PH, Bush RL et al. The impact of anesthetic modality on the outcome of carotid endarterectomy. Am J Surg 188(6):741-7, 2004.

SELECTIVE REFERENCES

1. Narins CR, et al: Patient selection for carotid stenting versus endarterectomy: A systematic review. J Vasc Surg 44:661-672, 2006.
2. Bates ER et al. ACCF/SCAI/SVMB/SIR/ASITN 2007 Clinical Expert Consensus Document on Carotid Stenting. JACC Vol 49(1):126-70, 2007.
3. Mas J-L, et al: Endarterectomy versus stenting in patients with symptomatic severe carotid stenosis. NEJM 355 (16):1660-1671.

LEARNING SUMMARY

Participants will become familiar with the indications for both carotid endarterectomy and carotid stenting. Recent controversies regarding carotid stenting will be discussed. Risk factors predictive of surgical difficulty with carotid endarterectomy will be stressed, and options for anesthetic management will be discussed.

The Acromegalic for Transsphenoidal Resection of a Pituitary Adenoma

Edward C. Nemergut, M.D. Charlottesville, Virginia

OBJECTIVES

Attendees, after preparation and participation will be able to:

1. Understand the anesthetic considerations of transsphenoidal surgery.
2. Manage the unique anesthetic implications of acromegaly.
3. Discuss airway management options for the acromegalic patient.
4. Make rational decisions regarding the intraoperative monitoring and anesthetic options for acromegalic patients undergoing surgery.
5. Manage the unique postoperative challenges after pituitary surgery.

STEM CASE - KEY QUESTIONS

Your patient is a 50 year-old right-handed man who reports to your hospital's preoperative area for transsphenoidal resection of a growth hormone secreting pituitary adenoma macroadenoma.

Key Questions

- What is the epidemiology of pituitary tumors?
- How can pituitary tumors be surgically approached?
- What are the common presenting complaints of acromegalic patients?
- Is surgery the only treatment option? How effective is surgery?

The patient's past medical history is significant hypertension for which he takes enalapril. Two years ago, the patient had a carpal tunnel release with local anesthesia and conscious sedation. In addition, the patient underwent knee arthroscopy ten years ago under general anesthesia with no apparent anesthetic complications. The patient works as a pulmonologist and does not smoke or consume alcohol. Finally, the patient, who had been an avid jogger completing his first marathon at age 32, indicates markedly reduced endurance such that although he still jogs two miles most mornings, he reports increased dyspnea and now stops to walk up all hills. He denies chest pain or dyspnea at rest.

Key Questions

- Is carpal tunnel syndrome more common in acromegaly? Is this important?
- What are the cardiac manifestations of acromegaly?
- What would you expect this patient's ECG to reveal? Echocardiogram?
- Do you feel this patient requires additional cardiac evaluation prior to surgery?

Physical examination reveals a 95 kg, 72 inch tall, adult man with BP 162/90, pulse 78 bpm, and respiratory rate 14 bpm. The patient presents with coarse facial features and slight frontal bossing. The patient's chest is clear with regular heart sounds. Neurologic exam reveals the patient to be alert and oriented times four with intact sensation and normal motor function. The patient has no drift or finger-to-nose dysmetria. Pupils are equally round and reactive to light.

Visual fields are full with intact extraocular movements. Closer examination of the patient's airway reveals a Mallampati III opening and full dentition. The patient has been recently diagnosed with obstructive sleep apnea (OSA).

Key Questions

- What changes in airway anatomy accompany acromegaly?
- Is OSA more common in acromegaly?
- Is airway management more likely to be difficult in acromegalic patients?
- How will you intubate this patient?
- If this patient were noticeably hoarse, would this alter your approach?

After a thorough discussion of your anesthetic plan, the patient is brought back to the operating room and standard monitors are placed.

Key Questions

- What, if any, additional monitors should be used in this patient? Why?
- Is neurophysiologic monitoring indicated for this patient?
- How much blood loss is expected for this procedure?

After the induction of anesthesia, the patient is intubated successfully and the invasive monitors discussed and selected above are placed without difficulty. A lumbar intrathecal catheter ("lumbar drain") is placed by the neurosurgeon. The patient is positioned and surgery begins.

Key Questions

- Is the patient undergoing transsphenoidal surgery at risk for venous air embolism (VAE)?
- Would you monitor for VAE? If so, how would you monitor?
- Is nitrous oxide contraindicated in this patient?
- What is the purpose of the lumbar intrathecal catheter?

Surgery continues uneventfully with minimal blood loss. The nose is packed and the head of the bed is returned to you. The patient's neuromuscular blockade is reversed and the patient is breathing spontaneously.

Key Questions

- What is your plan for extubation?
- How do you plan to treat the patient's pain? Is ketorolac contraindicated?
- Will you provide the patient with an empiric anti-emetic?
- Does vomiting pose a specific risk in this patient?

The patient is taken to the appropriate post-operative area. The patient's pain is well controlled, his visual fields are full, and he does not report any nausea; however, the patient has produced 1300 ml of urine over the past 3 hours and complains of extreme thirst.

Key questions

- Is urine output of concern? If so, what are the likely causes?
- How would you determine the etiology of the diuresis?
- At what point, if any should this be treated? How?

PROBLEM BASED LEARNING DISCUSSION

What is the epidemiology of pituitary tumors?

Patients with tumors of the pituitary gland are commonly encountered, representing approximately 10% of diagnosed brain neoplasms. Although the overall incidence of pituitary tumors remains stable, autopsy series suggest that as many as 20% of people may have a pituitary tumor on post-mortem examination. Pituitary adenomas are normally found in adults with a peak incidence during the fourth to the sixth decade of life. Tumors larger than 10 mm in any dimension are classified as "macroadenomas" while tumors smaller than 10 mm are classified as "microadenomas." Pituitary adenomas may be further classified as "functioning" or "non-functioning." Approximately 75% of pituitary tumors are "functioning" and produce a single, predominant hormone. In general, pituitary tumors can present in three discrete ways: 1) hormonal hypersecretion; 2) local mass effects; or, 3) tumors may be discovered incidentally during cranial imaging for an unrelated condition. Patients with functioning adenomas typically present with the symptoms of anterior pituitary hormone excess.

How can pituitary tumors be surgically approached?

Neurosurgeons can approach a pituitary tumor in two discrete ways. The first method is to perform a bifrontal craniotomy and approach the pituitary gland by following the optic nerves down to the sella. This method allows for greater "exposure" and permits for direct observation of the optic nerves. The second, more common approach is to approach the gland transsphenoidally. As a bifrontal craniotomy is associated with significantly greater patient morbidity and mortality, it is rarely utilized.

The transsphenoidal approach may be carried out using two separate techniques: endonasal and sublabial. Pituitary tumors are most commonly approached endonasally. The sublabial transseptal approach is only necessary in patients with extremely large tumors and in young children where endonasal exposure may prove inadequate. Traditionally, resection has been guided by the use of intraoperative fluoroscopy; however, computer-guided frameless stereotaxy may also be utilized. These neuronavigational techniques may best employed when the normal anatomy is distorted and landmarks are difficult to determine with fluoroscopy.

What are the common presenting complaints of acromegalic patients?

Despite the multi-system nature of acromegalic disease, the most common initial complaint of any patient with a pituitary tumor is headache. In patients with a macroadenoma, visual loss (classically temporal or bitemporal hemianopsia) from optic nerve compression frequently accompanies headache as an initial complaint. After patients seek medical attention and acromegaly is eventually diagnosed, patients "retrospectively" note other complaints that were initially thought to be unrelated (see below).

Is surgery the only treatment option? How effective is surgery?

Non-surgical options currently available include radiotherapy and medical therapy with the dopamine agonists and somatostatin analogues. Transsphenoidal surgery is the most effective treatment option and is the only therapy that offers a potential cure. The success of surgery is highly dependent upon the size of the tumor and the experience of the surgeon. Surgery is curative in 80-90% of acromegalic patients with a microadenoma and 50-60% of patients with a macroadenoma. Radiotherapy seems most efficacious when used in combination with transsphenoidal surgery, especially when directed at residual tumor. Medical therapy can reduce many of the signs and symptoms of systemic disease; however, it does not offer a cure and fails to reduce tumor size in most patients.

Is carpal tunnel syndrome more common in acromegaly? Is this important?

Carpal tunnel syndrome resulting from the soft tissue overgrowth is commonly observed. Often, the diagnosis of carpal tunnel syndrome precedes the diagnosis of acromegaly by a year or more. The same soft-tissue overgrowth that causes carpal tunnel syndrome may result in a reduction of ulnar arterial flow. Indeed, blood flow through the ulnar artery may be compromised in up to 50% of acromegalic patients. In these patients, placement of a radial artery catheter may potentially result in hand ischemia.

What are the cardiac manifestations of acromegaly?

Cardiac disease is the most important cause of morbidity and mortality in acromegalic patients. Indeed, the most frequent cause of death in untreated acromegaly is cardiovascular with 50% of patients dying before the age of 50. Older reviews suggest that as many of 80% of patients died from cardiovascular complications before the age of 60. A recent series suggested that as many as 10% of newly diagnosed patients may have overt heart failure upon initial diagnosis. The most prominent feature of acromegalic cardiac disease is myocardial hypertrophy. Left ventricular hypertrophy (LVH) can occur in the presence of systemic hypertension, but also occurs in at least 50% of normotensive acromegalic patients. Overall, two-thirds of patients will have LVH at the time of diagnosis. The prevalence of LVH increases with patient age and is greater than 90% in elderly patients with longer disease duration.

What would you expect this patient's ECG to reveal? Echocardiogram?

Echocardiography reveals increases in left ventricular mass, stroke volume, cardiac output, and isovolumic relaxation time. These changes occur independently from systemic hypertension.

EKG changes such as S-T segment depression, T-wave abnormalities, and increased QRS voltage are typical of LVH and are frequently observed. Classically, it had been thought the incidence of supraventricular and ventricular ectopy was not increased in resting acromegalics; however, given the poor exercise tolerance of acromegalic patients and a lower threshold to what may be considered "exercise," cardiac arrhythmias are frequently observed.

Do you feel this patient requires additional cardiac evaluation prior to surgery?

There is no right or wrong answer to this question. In the opinion of the author, the patient does not require additional cardiac evaluation. Although the patient has noted a decrease in exercise tolerance, the patient still has excellent functional capacity. Nevertheless, many anesthesiologists may prefer to have some information regarding the degree of cardiac disease. As a transthoracic echocardiogram is a simple and non-invasive test, it may be a reasonable option. Medical therapy with somatostatin should be considered in patients with severe cardiomyopathy and significant functional limitation before surgery to reduce cardiac risk.

What changes in airway anatomy accompany acromegaly?

Hypertrophy of the facial bones, especially the mandible, and coarsening of facial features lead to significant changes in patient appearance. Soft tissues of the nose, mouth, tongue, and lips become thicker and help give acromegalic patients their characteristic facade. In addition to the easily observed external changes, there is thickening of the laryngeal and pharyngeal soft tissues. Hypertrophy of the periepiglottic folds, calcinosis of the larynx, and recurrent laryngeal nerve injury can all contribute to airway obstruction and respiratory disease. Indeed, hypertrophy can cause significant reduction in the size of glottic opening. Laryngeal stenosis and abnormal vocal cord function may be present and patients may report hoarseness or changes in vocal tone, quality, or strength.

Is obstructive sleep apnea more common in acromegaly?

Obstructive sleep apnea (OSA) can affect up to 70% of acromegalic patients. Airway obstruction is three-fold more common among male acromegalics than female acromegalics.

Is airway management more likely to be difficult in acromegalic patients?

Successful endotracheal intubation and management of the acromegalic airway can be extremely difficult. As might be expected, difficult laryngoscopy and poor laryngeal view has been associated with Mallampati class 3 and 4 airway examinations; however, 20% of acromegalic patients assessed as Mallampati class 1 and 2 have been noted to be difficult to intubate. As such, difficult endotracheal intubation may be unpredictable in acromegalic patients. Indeed, routine tracheostomy had been historically advocated for management of the acromegalic airway; however, this is rarely necessary. The anesthesiologist should approach any acromegalic airway, regardless of anticipated difficulty, with extreme caution.

How will you intubate this patient?

The selection of an intubation technique should always be based upon preoperative patient airway assessment and the anesthesiologist's ability and comfort level. Nevertheless, certain techniques may be more or less useful in the acromegalic patient. For example, the intubating Laryngeal Mask Airway has been associated with a low (52.6%) first-attempt success rate in unparalyzed acromegalic patients. In addition, flexible fiberoptic laryngoscopy can be more difficult secondary to abundant pharyngeal soft tissue. As always, awake techniques offer the greatest margin of safety.

If this patient were noticeably hoarse, would this alter your approach?

As noted above, hoarseness may occur secondary to calcinosis of the larynx or recurrent laryngeal nerve injury. Stenosis and calcinosis of larynx is more common and any acromegalic patient that complains of significant hoarseness should be considered at risk. In such a patient, a smaller than predicted endotracheal tube may be necessary.
The Parker Flex-Tip endotracheal tube is associated with higher success rates during fiberoptic tracheal intubation compared to standard endotracheal tubes and may be particularly useful in acromegalic patients with laryngeal stenosis.

What, if any, additional monitors should be used in this patient? Why?

The placement of invasive monitoring should always be based on each patient's preoperative assessment. In addition to standard monitors, many anesthesiologists employ invasive arterial monitoring during transsphenoidal surgery. Acromegalics may have a significant cardiomyopathy as noted above, and close monitoring with intraarterial catheter may be useful. In addition, transsphenoidal surgery can be associated with significant intraoperative hemodynamic changes. Indeed, an arterial catheter may allow for earlier diagnosis and treatment of both hypo- and hypertension. Nevertheless, there are no data to suggest that excessive hemodynamic instability accompanies acromegaly in the absence of specific cardiovascular disease.

Generally speaking, central venous pressure (CVP) or pulmonary artery pressure (PAP) monitoring is not necessary during transsphenoidal surgery. In any acromegalic patient with cardiovascular disease significant enough to necessitate CVP or PAP monitoring, medical therapy to abrogate cardiovascular disease should be initiated until the patient is a better candidate for elective surgery. Should the patient require surgery emergently (for increased ICP, pituitary apoplexy, etc.), it should be noted that in patients with a cardiomyopathy a pulmonary artery catheter (PAC) may be a better monitor of left ventricular preload.

Is neurophysiologic monitoring indicated for this patient?

As opposed to a bifrontal craniotomy, the transsphenoidal approach does not allow for direct observation of the optic nerves. As visual impairment is a feared complication of transsphenoidal surgery, some practitioners have utilized visual evoked potential (VEP) monitoring surgery. During VEP monitoring, goggles are placed over the patient's eyes and a bright flash of light periodically stimulates the eyes. The bright flash evokes an electrical response in the occipital

cortex. The electric response is recorded by scalp electrodes and is monitored over the course of the procedure. As such, VEP's continually monitor the integrity of visual pathway during surgery. Although theoretically useful, it is extremely difficult to use VEP monitoring in practice. VEP's are exquisitely sensitive to the effects of anesthetic agents. Indeed, even narcotic-induced papillary constriction can interfere with appropriate stimulation of the retina. As there is little data to support the routine use of VEP monitoring, most pituitary centers do not employ this technique.

Should the patient have blood available for transfusion prior to the induction of anesthesia?

Transsphenoidal surgery is normally associated with minimal blood loss; however, the pituitary gland sits in close approximation to the carotid arteries. Indeed, arterial injury is a feared complication of the transsphenoidal approach and is associated with significant perioperative morbidity and mortality. In addition, growth hormone secreting tumors can be associated with impressive dilatation of the intracranial arteries. Large bore intravenous access and the ready availability of blood should be considered prior to the surgical incision.

Is the patient undergoing transsphenoidal surgery at risk for venous air embolism (VAE)? Would you monitor for VAE? If so, how would you monitor?

Transsphenoidal operations are generally performed with the patient supine with some degree of head-up position (normally 20-40 degrees). Any time the operative field is above the right atrium, venous air embolism (VAE) is a theoretical risk. Although a 10% risk of VAE in the semi-seated position has been reported, a clinically significant VAE associated with significant morbidity or mortality has not been reported. At our institution, we have anesthetized nearly 5000 patients for transsphenoidal surgery and have not had a clinically significant VAE. Consequently, routine monitoring with capnography seems adequate. Nevertheless, echocardiography, precordial Doppler, and end-tidal N2 monitoring may be considered.

Is nitrous oxide contraindicated in this patient?

Despite the potential risk of a VAE, nitrous oxide is not contraindicated. As noted above, a clinically significant VAE associated with significant morbidity or mortality has not been reported.

What is the purpose of the lumbar intrathecal catheter?

A lumbar intrathecal catheter (lumbar drain) is used to assist in visualization of the tumor. The catheter can be used to manipulate cerebrospinal fluid pressure (CSF) pressure by the injection of saline or aspiration of CSF. In patients with large macroadenomas with significant suprasellar extension, some pituitary surgeons will inject intrathecal air. The air serves to increase CSF pressure and may "push" the suprasellar portion of the tumor into the operative field. The injected air may also serve to outline a tumor, allowing for fluoroscopic visualization. Obviously, strict asepsis should be utilized when anything is injected into the CSF. If air is injected into the CSF, nitrous oxide should be discontinued.

What is your plan for extubation?

All patients are at an increased risk of airway obstruction after transsphenoidal surgery and loss of airway patentcy can be associated with obvious morbidity and mortality. To tamponade mucosal bleeding, many neurosurgeons will leave nasal packs in place for a variable period of time after surgery. As any patient with nasal packs in place is an obligate mouth breather, care should be taken to assure oropharyngeal airway patentcy. In acromegaly, enlargement of the tongue and upper airway as noted above can result in easy occlusion of the oral airway. Finally, blood and surgical debris in the naso- and oropharynx may further serve to occlude the airway.

How do you plan to treat the patient's pain? Is ketorolac contraindicated?

Transsphenoidal surgery is normally associated with only mild patient discomfort. Indeed, a recent review suggests that the median consumption of morphine in the PACU was only 4 mg. Interestingly, increased morphine requirements were associated with the appearance of post-operative diabetes insipidus (DI) and the use of a lumbar drain or a CSF leak was associated with a decreased need for postoperative narcotic analgesia. The use of bilateral infraorbital nerve blocks for postoperative analgesia has been reported. Nevertheless, narcotics such as morphine and fentanyl should be used with great care in any acromegalic with a history OSA. Ketorolac is efficacious after transsphenoidal surgery. It is not contraindicated and has not been associated with an increase in the incidence of bleeding.

Will you provide the patient with an empiric anti-emetic?
Does vomiting pose a specific risk in this patient?

Nausea and vomiting are very common postoperative complications in patients undergoing neurosurgical procedures, with nearly 40% of patients reporting either complaint; however, a recent review of the perioperative records of 877 patients undergoing transsphenoidal surgery by the same surgeon suggest the incidence was much lower. After transsphenoidal surgery, the overall incidence of postoperative emesis was 7.5%, significantly lower than most studies of neurosurgical patients. An intraoperative CSF-leak and subsequent fat grafting, the use of lumbar intrathecal catheter, and patients presenting for the resection of a craniopharyngiomas all had a significantly increased incidence of postoperative emesis (11.4%, 17.1%, and 18%, respectively). Interestingly, antiemetic prophylaxis did not decrease the risk of vomiting overall or in any cohort of patients; however, both droperidol and ondansetron decreased the incidence of nausea in the PACU. Given the high risk for vomiting in patients after transsphenoidal surgery and the detrimental effects of vomiting on ICP, routine pharmacologic prophylaxis seems reasonable, despite its apparent lack of efficacy in retrospective review. Given the potential effects of steroids on the hypothalamus-pituitary-adrenal axis, we tend to avoid dexamethasone as an antiemetic.

Is urine output of concern? If so, what are the likely causes?
How would you determine the etiology of the diuresis?

A variety of factors could be responsible for polyuria after transsphenoidal surgery. Overzealous perioperative fluid administration may result in immediate postoperative polyuria. Osmotic diuresis, which could be due to mannitol administration, steroid administration, and hyperglycemia, can also result in polyuria and polydipsia. Indeed, glucose intolerance or frank

diabetes mellitus may be associated with acromegaly. Patients with acromegaly can also demonstrate a robust physiological diuresis following successful tumor resection. Finally, disorders of water balance resulting from perturbations in secretion of anti-diuretic hormone (ADH) are some of the most frequently encountered acute perioperative complications of transsphenoidal surgery. The relative or absolute deficiency of ADH results in DI.

DI is characterized by polyuria and polydipsia in the setting of dilute urine. If water excretion exceeds intake, hypovolemia, hypotension and elevated serum osmolarity and sodium result. Serum sodium and osmolarity may remain normal if intake matches output (typically the case in awake and alert adults with intact thirst mechanisms). Diabetes insipidus is a common early perioperative complication and fortunately is often transient. The overall reported incidence of transient DI varies widely affecting 4 to 80% of patients following surgery, while permanent diabetes insipidus is seen in 0.5 to 2%. Postsurgical DI typically manifests within 24 to 48 hours postoperatively and should be suspected when there is a sudden onset of voluminous polyuria. If the patient is awake and alert, thirst will accompany the polyuria. For early detection of DI, it is recommended that urine output and specific gravity be measured routinely after pituitary surgery. Diagnostic features of DI are hypotonic urine (< 300 mOsm or specific gravity of < 1.005) and high urine output (as much as 4-18 L/day).

At what point, if any should this be treated? How?

As the majority of patients in the post-operative period following transsphenoidal surgery are awake and alert with intact thirst mechanisms, the development of significant volume depletion and hyperosmolarity is relatively uncommon if patients have adequate access to fluid. Conservative management coupled with close electrolyte and urine monitoring is the most appropriate management strategy.

When patients are unable to keep up with their fluid requirements or unrelenting urination is present (often interfering with sleep), specific pharmacologic treatment should be instituted. Desmopressin acetate (DDAVP) is a synthetic analogue of ADH is the pharmacologic agent of choice. The oral formulation is effective and should be considered first line treatment. An initial dose of 0.1 mg DDAVP can be administered orally and usually is effective in controlling postoperative DI. Alternatively, if patient is unable to take oral medications, 1mcg of DDAVP can be administered subcutaneously.

As noted above, DI is transient and self-limited in the overwhelming majority of cases; however, treatment with DDAVP may result in "overshoot" hyponatremia that can be associated with significant morbidity such as confusion and seizures. As such, it is imperative that careful electrolyte monitoring is continued during treatment with DDAVP.

References

Flynn, B.C. and Nemergut, E.C. "Postoperative Nausea and Vomiting and Pain after Transsphenoidal Surgery: A Review of 877 Patients" Anesthesia & Analgesia 2006; 103(1): 162-167.

Nemergut, E.C., Zuo, Z.: "Airway Management in Pituitary Disease: A Review of 746 Patients" Journal of Neurosurgical Anesthesiology 2006; 18(1): 73-77.

Nemergut, E.C., Dumont, A.S., Barry, U.T., and Laws, E.R.: "Perioperative Management of Patients Undergoing Transsphenoidal Pituitary Surgery" Anesthesia & Analgesia 2005; 101(4): 1170-81.

Nemergut, E.C., Zuo, Z., Jane, JA, Laws, ER: "Diabetes Insipidus after Transsphenoidal Surgery: A Review of 881 Patients" Journal of Neurosurgery 2005; 103(3): 448-454.

Jane JA, Jr., Thapar K, Kaptain GJ, Maartens N, Laws ER, Jr. Pituitary surgery: transsphenoidal approach. Neurosurgery. Aug 2002;51(2):435-442; discussion 442-434.

Schmitt H, Buchfelder M, Radespiel-Troger M, Fahlbusch R. Difficult intubation in acromegalic patients: incidence and predictability. Anesthesiology. Jul 2000;93(1):110-114.

SELECTIVE REFERENCES

Nemergut, E.C., Dumont, A.S., Barry, U.T., and Laws, E.R.: "Perioperative Management of Patients Undergoing Transsphenoidal Pituitary Surgery" Anesthesia & Analgesia 2005; 101(4): 1170-81.

Jane JA, Jr., Thapar K, Kaptain GJ, Maartens N, Laws ER, Jr. Pituitary surgery: transsphenoidal approach. Neurosurgery. Aug 2002;51(2):435-442; discussion 442-434.

Schmitt H, Buchfelder M, Radespiel-Troger M, Fahlbusch R. Difficult intubation in acromegalic patients: incidence and predictability. Anesthesiology. Jul 2000;93(1):110-114.

LEARNING SUMMARY

Attendees, after preparation and participation will be able to:

1. Understand the anesthetic considerations of transsphenoidal surgery.
2. Manage the unique anesthetic implications of acromegaly.
3. Discuss airway management options for the acromegalic patient.
4. Make rational decisions regarding the intraoperative monitoring and anesthetic options for acromegalic patients undergoing surgery.
5. Manage the unique postoperative challenges after pituitary surgery.

The Neurosurgeon Asks You To Place Your Patient in the Sitting Position. Now What?

James R. Munis, M.D., Ph.D. Rochester, Minnesota

OBJECTIVES

To understand the preoperative considerations to address before agreeing to place a patient in the sitting position.

To understand the mechanical pitfalls and perils associated with positioning in the sitting position.

To understand the basic considerations of venous air embolism (VAE): its mechanism, detection, and treatment.

To understand both the advantages and disadvantages of TEE monitoring and antecubital CVP placement for cases in the sitting position.

STEM CASE - KEY QUESTIONS

A 36 year old woman presents with recurrent occipital headaches and new onset gait disturbance. Imaging studies, including an MRI, reveal a 3 cm midline mass in the cerebellum. Subsequent MRA/MRV and angiography reveal that the mass is an arterial-venous malformation (AVM). Her neurosurgeon plans to resect the lesion in the sitting position.

[Preoperative Issues]

What else would you like to know about this patient before bringing her to the OR; and before agreeing to do the case in the sitting position? What should you tell the surgeon are the pros and cons of the sitting position from an anesthetic point of view?

Your hospital performs very few sitting cases. Who will you discuss the logistics of the case with? What special equipment will you need for safe positioning?

The patient's height is 160 cm, and her weight is 110 kg. She has no prior history of general anesthesia. Her mouth opening is small, she has full dentition, including capped upper and lower incisors, and a Mallampati III airway exam. Her neck extension is slightly limited, but she has normal rotation and flexion. Are you worried about her airway exam? What is your plan for managing her airway? What is your backup plan? How might your airway management influence intracranial pressure?

Preoperative vital signs are: HR 72 (regular); BP 110/64; Resp 16; Temp 36.8° C; Room air O_2 sat 94%. Heart exam reveals a regular rate and rhythm, with a 2/6 non-radiating systolic murmur best heard at the right sternal border. Chest exam is normal. Laboratory results are unremarkable. Hgb = 13.0 gm/dl; plt = 235,000; Na = 142 mEq/L; K = 4.2 mEq/L; Cr = 1.0

Are you worried about her cardiac exam? Would you like additional preoperative cardiac history? If you are worried, do you think she should have a preoperative echocardiogram? How would this change your management?

If no echocardiogram or additional cardiac workup is performed, what, if anything, will you do to rule out a PFO in the operating room?

What will you do to monitor for venous air embolism (VAE) during the case?

[Intraoperative Management]

After successful intubation, an arterial line is placed, as well as an antecubital CVP catheter. Also, a precordial Doppler is placed after the patient is positioned upright.

What mechanical positioning issues need to be addressed and checked before allowing the surgery to proceed?

What are the advantages and disadvantages of an antecubital CVP for this case?... of a TEE?

How can you assure proper positioning of the antecubital CVP? What are the relative sensitivities of the different options for detecting VAE?

Where do you place the arterial pressure transducer in the sitting position? Why? How is cerebral perfusion pressure calculated in the sitting position? Related to these questions, what is the actual mechanism of VAE in a mechanically ventilated patient?

Should you use nitrous oxide? Why or why not? Does it depend on what type of monitoring you have?

During the case, the patient becomes suddenly hypotensive (BP decreases from 110/72 to 74/40). Oxygen saturation falls from 99% to 74%.

What additional information would you like? If you suspect or confirm VAE, what do you do now?

The patient stabilizes, the surgery is completed, and the TEE probe is removed. Prior to extubation, you notice that the tongue is slightly swollen.

Do you extubate now?

PROBLEM BASED LEARNING DISCUSSION

The sitting position provides several advantages to the neurosurgeon, including brain decompression, drainage of blood and irrigation away from the operative site; and in select cases, superior visual access to the operative site. It also presents several challenges to the anesthesiologist and can be particularly frustrating if it is unfamiliar or only infrequently used. This PBLD is aimed at the anesthesiologist who may be asked to manage sitting cases, but not on a day-to-day basis, and who would like an interactive overview of strategies for dealing with

the major dangers of the position. It is also aimed at more experienced neuroanesthesiologists who are already familiar with the sitting position, but who would enjoy a dynamic exchange of ideas about management strategies and controversies.

We start with the practical question of agreeing to do the case in this position in the first place. Because the sitting position courts unique positioning, hemodynamic, and venous air embolism (VAE) dangers, a frank assessment is required to ensure that appropriate OR and anesthesia equipment are available. OR nursing and surgical staff should be prepared ahead of time to accommodate the position with a specialized pinion head frame holder that clamps to the OR table around the patient's upright torso. If the patient is morbidly obese, the frame needs to be large enough to encompass the patient without placing pressure on her arms. The OR table needs to flex appropriately at both hips and knees. All involved OR staff (nursing, surgical, and anesthesia) should have the same defined goal of providing a stable position without arm compression, head movement, overflexion of the neck, or allowing "hanging" from the pinions because of a lack of lumbar and hip support. Importantly, each of these pieces of equipment should be checked ahead of time to ensure that they work as planned. During this initial discussion, the participants will be pushed to take a stance that it is their responsibility to make sure that these issues are addressed before agreeing with the surgeon do place the patient in this position. The participants will also be asked to construct a relevant and cogent "pro-con" discussion with the surgeon. Can the surgeon do the case in the lateral decubitus or prone position? Does the surgeon know what special risks and logistical considerations attend the sitting position? Is he or she willing to accommodate them?

Next, the discussion moves to the patient herself. She presents with a daunting airway exam. As with all patients, the options for dealing with a potentially difficult airway need to be considered and planned for ahead of time. What's emphasized here, though, is the context of intracranial pressure (ICP). The concept of competing concerns is addressed: the most "conservative" management strategy for securing her airway (awake fiberoptic intubation) may not be the most conservative strategy for her ICP (coughing and straining). The additional risks that hypercapnia and hypoxia present to her elevated ICP are also broached. The participants are also asked to consider the effects of prolonged mask ventilation on dead space, and consequently, on arterial CO2 and ICP. A reasonable compromise between these competing concerns is reached (I favor awake fiberoptic intubation with attention to airway topicalization and avoidance of over-sedation and hypoventilation, but that is not the only reasonable choice), and a backup plan is resolved.

The next preoperative issue is the patient's cardiac exam. Her history is unimpressive, but she does have a murmur, and a PFO cannot be excluded. The participants will be asked to consider a thoughtful and efficient algorithm for addressing that concern. There is no right answer to pre-operative echocardiogram versus intraoperative TEE to rule out a PFO. The discussion will encompass the use of a TEE bubble-test for PFO, as well as a plan for using the information. Given the patient's risk of VAE, a positive PFO test should provoke a discussion with the surgeon about alternative positions. For this case, the PFO test is negative. The participants will be asked to consider the important difference in relative risk for VAE between this case and a more common sitting case (cervical laminectomy).

We then move on to decisions about intraoperative monitoring for VAE. This is properly a part of the preoperative discussion because the relevant equipment is not readily available in most OR's. A nod is given to the "textbook" discussion of the relative sensitivities of various monitors to VAE. The participants will be pushed to place that information in a more practical context, however. For example, how much air contrast visible in the right atrium on TEE constitutes a significant "VAE"? In spite of the sensitivity of TEE, is it realistic to expect the first detection of VAE to be a TEE observation, rather than precordial Doppler, since the probe is not always turned on, and the screen is not always watched? The risks of TEE placement and use are explored, as well as the risks and benefits of antecubital CVP catheters. The participants will also discuss whether or not an alternative central access should be employed if an antecubital CVP is not successfully placed.

The intraoperative phase begins. Following successful awake fiberoptic intubation, an arterial line is placed, as well as an antecubital CVP catheter and a TEE. The logistics of passing an antecubital CVP are discussed. The bubble test is negative for PFO. Now the patient is sat up. Participants are asked what to check for after the final sitting position is assumed. In particular, the dangers of overflexion of the neck and "hanging" from the pinions because of inadequate lumbar and hip support are discussed.

A controverial question is asked: should nitrous oxide be used? The particpants are led to consider both pros and cons of nitrous oxide (for example, the potential expansion of air emboli versus enhancement of mass-spec detection of elevated nitrogen above a zero baseline).

We now come to another simple, but loaded question: where should the arterial line transducer be positioned? The textbook answer is at head (Circle of Willis) level. In addressing this question, the participants are asked a related but often neglected question: what is the mechanism of VAE in mechanically ventilated patients? ... and why does elevation of the surgical site above the heart increase the risk of VAE? This may very well be the first time that participants have thought about this simple mechanistic question because it has been neglected in textbooks in favor of more familiar discussions about the detection and treatment of VAE, not its actual mechanism. A thorough consideration of how to think about cerebral perfusion pressure in the sitting position depends on the answer to this mechanistic question, however, because the phenomenon of VAE suggests the operation of a siphon effect in the cerebral circulation. Only a small amount of time is spent on this issue, but it will provoke an interesting an unusual application of basic physics and physiology to a practical case management question.

An intraoperative event occurs. VAE is detected, and it is large enough to have hemodynamic significance. The discussion will include the variety of VAE diagnostic signs: hypotension, rhythm disturbances, desaturation, an abrupt fall in end-tidal CO_2, the appearance of end-tidal N_2 on mass-spectrometry, a mechanical sound on precordial Doppler; and finally, visual confirmation of VAE from TEE. Now that significant VAE is detected, its management and treatment are discussed. The surgeon is notified and places a saline-soaked packing over the likely source of air entry; F_IO_2 is changed to 100% O_2, air is removed form the antecubital CVP, lateral decubitus position is discussed but not employed because the patient is fixed in pinions and still has cardiac function, hemodynamic support is given, TEE confirmation is obtained that no air has crossed into the left heart. The episode resolves with surgical closure of the site of air entry, and the AVM resection is completed.

After apparent resolution of the danger, the patient is noted to have modest tongue swelling after removal of the TEE probe, but before extubation. A discussion is held about the risks of neck flexion in the sitting position, and of TEE placement. The participants are pressed to consider the timing of extubation. This is a rare but potentially serious consequence of the sitting position, and the participants should be equipped to be vigilant for it.

REFERENCES

Senn N. An experimental and clinical study of air-embolism. *Ann Surg* 2: 22-50, 1885 and 1: 517-549, 1885.

Albin MA. The sights and sounds of air. *Anesthesiology* 58: 113-114, 1983.

Michenfelder JD, Martin JT, Altenburg BM, Rehder K. Air embolism during neurosurgery: An evaluation of right-atrial catheters for diagnosis and treatment. *JAMA* 208:1353-1358, 1969.

Michenfelder JD, Miller RH, Gronert GA. Evaluation of an ultrasonic device (Doppler) for the diagnosis of venous air embolism. *Anesthesiology* 36: 164-167, 1972.

Cucchiara RF, Seward JB, Nishimura RA, Nugent M, Faust RJ. Identification of patent foramen ovale during sitting position craniotomy by transesophageal echocardiography with positive airway pressure. *Anesthesiology* 63: 107-109, 1985.

Cucchiara RF, Nugent M, Seward JB, Messick JM. Air embolism in upright neurosurgical patients: Detection and localization by two-dimensional transesophageal echocardiography. *Anesthesiology* 60: 353-355, 1984.

Matjasko J, Petrozza P, Cohen M, and Steinberg P. Anesthesia and surgery in the seated position: analysis of 554 cases. *Neurosurgery* 17: 695–702, 1985.

S. Black, D.B. Ockert, W.C. Oliver Jr. and R.F. Cucchiara. Outcome following posterior fossa craniectomy in patients in the sitting or horizontal positions, *Anesthesiology* **69**: 49–56, 1988.

Engelhardt M, et al. Neurosurgical operations with the patient in sitting position: analysis of risk factors using transcranial Doppler sonography. *Br J Anaesth.* 96: 467-72, 2006.

Leslie K, Hue R, Kaye AH. Venous air embolism and the sitting position: a case series. *J Clin Neurosci.* 13: 419-22, 2006.

Munis JR and Lozada LJ. Giraffes, siphons, and starling resistors. Cerebral perfusion pressure revisited. *J Neurosurg Anesthesiol* 12: 290–296, 2000.

Hicks JW, Munis JR. The siphon controversy counterpoint: the brain need not be "baffling". *Am J Physiol* 289: R629-632, 2005.

Mirski MA, Lele AV, Fitzsimmons L, Toung TJK. Diagnosis and treatment of vascular air embolism. *Anesthesiology* 106:164-167, 2007.

SELECTIVE REFERENCES

Matjasko J, Petrozza P, Cohen M, and Steinberg P. Anesthesia and surgery in the seated position: analysis of 554 cases. *Neurosurgery* 17: 695–702, 1985.

S. Black, D.B. Ockert, W.C. Oliver Jr. and R.F. Cucchiara. Outcome following posterior fossa craniectomy in patients in the sitting or horizontal positions, *Anesthesiology* **69**: 49–56, 1988.

Engelhardt M, et al. Neurosurgical operations with the patient in sitting position: analysis of risk factors using transcranial Doppler sonography. *Br J Anaesth.* 96: 467-72, 2006.

LEARNING SUMMARY

1. To understand the preoperative considerations to address before agreeing to place a patient in the sitting position.
2. To understand the mechanical pitfalls and perils associated with positioning in the sitting position.
3. To understand the basic considerations of venous air embolism (VAE): its mechanism, detection, and treatment.
4. To understand both the advantages and disadvantages of TEE monitoring and antecubital CVP placement for cases in the sitting position.

Neurogenic Pulmonary Edema and Cardiac Failure Following Severe Traumatic Brain Injury

Maureen McCunn, M.D., M.I.P.P. Baltimore, Maryland

OBJECTIVES

- Understand the pathophysiology of non-neurologic organ system failure
- Be aware of alternative therapies used in the management of critically injured patients
- List the indications and contraindications of extracorporeal support modalities following traumatic injury.

STEM CASE - KEY QUESTIONS

A 16 year old female sustains a fall, hitting her head. At the scene of the injury she is unresponsive with no respiratory effort. She is intubated in the field by paramedics and is transported by helicopter to your trauma center. Her initial neurologic exam on admission shows a Glasgow Coma Score (GCS) of 7T (E1M5V1t). She moves all four extremities equally and has reactive pupils. Blood pressure is 126/72, heart rate 101, and O2 saturation 100% on FiO2 1.0.

KEY QUESTION #1: What are the initial management priorities in a patient with severe (defined as GCS < 8) traumatic brain injury (TBI)?

KEY QUESTION #2: What are your goals for cerebral perfusion, CO2 and oxygenation?

Initial head computed tomography (CT) shows diffuse cerebral edema with effacement of the third ventricle, a small left epidural hematoma and small rim subdural on the right side with minimal amount of shift, as well as bi-frontal contusions with subarachnoid hemorrhage. Admission chest x-ray demonstrates only left lower lobe atelectasis. The remainder of her trauma work-up is negative. An intraventricular catheter and a cerebral oximetry monitor are placed. Initial intracranial pressures are 8-15 mmHg and PBRO2 is 15-20 mmHg. Two hours later the patient has sustained elevation of ICP that does not respond to medical therapy (sedation, paralysis, mannitol, hypertonic saline, paCO2 ~ 34). Repeat head CT shows increasing size of the epidural hematoma with increasing shift. The neurosurgeons post the case for an emergent craniotomy and evacuation of epidural hematoma.

KEY QUESTION #3: What are the intravenous access, monitoring and blood product needs for a craniotomy following trauma?

During emergent evacuation of her epidural hematoma, the patient becomes hemodynamically unstable and does not respond to volume loading. ST-segment depression is noted on the monitor; a pulmonary artery catheter is placed, with an initial cardiac index of 2.0 $L/m/m^2$. Norepinephrine is begun in order to maintain cerebral perfusion pressure. New onset pulmonary edema is suggestive of congestive heart failure and serum troponins and CPK-MB fractions are elevated. The neurosurgeons complete the case and the patient is admitted to the ICU.

Post-operatively, ICP and cerebral perfusion pressure (CPP) normalize. Later that night the patient has worsening oxygenation and rising CO2. She is transitioned to airway pressure release ventilation (APRV) with minimal improvement. Emergent transesophageal echocardiography (TEE) is performed and is significant for severe global biventricular failure with an estimated ejection fraction of 20% and 4+ mitral regurgitation (MR). No evidence of systolic anterior motion is present. The patient is becoming more and more difficult to ventilate. Her ICPs continue to rise in spite of therapy.

KEY QUESTION #4: What pharmacologic treatments are effective in lowering ICP following TBI?

KEY QUESTION #5: What therapies are effective to treat hypoxia and ARDS in patients with elevated ICP?

On ICU day #3 she develops cyanotic digits and systemic acidosis.

KEY QUESTION #6: Could this be propofol infusion syndrome?

Elevated ICP prevents intermittent prone positioning therapy and the patient is placed on a hydraulic "tilt-table" in the vertical position in an attempt to improve oxygenation. Any attempt to lower the head of the bed results in severe intracranial hypertension (ICP > 40 mmHg) and hypoxia (02 saturation ~ 30%).

A decompressive laparotomy is performed which transiently lowers her intracranial pressure from 30 to 15 mm Hg … but again the ICP increases. On hospital day #4, the patient is placed on extracorporeal life support (ECLS). Veno-venous cannulation (right femoral vein and right internal jugular vein) is performed while the patient is in a fully upright position on the tilt table. Systemic heparinization is avoided through the use of Carmeda-coated heparin-bonded circuitry

for the first 24 hours of therapy and thereafter activated clotting time (ACT) is maintained ~ 180 seconds. The driving pressure on the ventilator is radically reduced. Her CO2 normalizes, followed shortly thereafter by her ICP.

KEY QUESTION #7: What are the indications and contra-indications for ECLS in patients following trauma?

PROBLEM BASED LEARNING DISCUSSION

Neurogenic pulmonary edema (NPE) and cardiac dysfunction have been reported with subarachnoid hemorrhage (SAH) and with isolated traumatic brain injury (TBI). [5,21,22] The patho-physiology is poorly understood; [3] nevertheless, the consequences of these conditions: hypoxia, hypotension and/or hypercapnia, may increase intracranial pressures and worsen outcome. [8] It is now recognized that patients with traumatic brain injury (TBI) develop non-neurologic organ dysfunction and that this organ failure is associated with a worse neurologic outcome.[28]

The first report of a connection between intracranial hemorrhage and cardiac dysfunction was made by Cushing in 1903. He noted alterations in blood pressure and cardiac rhythm in patients with intracranial hemorrhage.[4] With the introduction of the EKG, an abnormality, referred to as a cerebral T waves, was described in patients with intracranial bleeding. The presence of extra-axial blood within the cranial vault has been shown to induce arrhythmias, such as torsade de pointes, in patients with SAH.

More recently, the direct effects of intracranial bleeding upon organ systems have been identified. Neurogenic pulmonary edema (NPE) and reversible myocardial contraction abnormalities have been described in patients with subarachnoid hemorrhage[12] and patients with no angiographic evidence of coronary artery disease or vasospasm develop left ventricular wall motion abnormalities. The occurrence of symptoms varies from several hours to several days after the initial injury.

Neurogenic pulmonary edema and myocardial dysfunction have been reported in both SAH and isolated TBI. One theory suggests NPE is related to a catecholamine surge that results in severe systemic and pulmonary hypertension and a resultant increase in venous return. This leads to translocation of blood from the systemic to the pulmonary circuit and favors the development of isolated pulmonary edema. In the case of TBI the catecholamine release is probably due to hypothalamic stress. [2] It is also been shown that catecholamines act directly upon the myocardium and result in direct myocyte injury.[27] Elevated catecholamine levels decrease the viability of myocytes through cyclic AMP-mediated calcium overload. As catecholamine levels return to normal, cardiac recovery begins. Return of normal wall motion is seen in as many as 73% of affected patients. Another theory postulates that there is a neurally-induced increase in capillary permeability. The resulting respiratory and cardiac failure occurs along a spectrum of severity, with the case presented here representing the extreme. [13]

Multiple organ failure (MOF) is a major cause of death in critically ill patients. Approximately half of the patients admitted to the ICU develop MOF, and 40% of these patients die as a result of the disease. In an observational cohort study of 209 consecutive patients with severe TBI, 89% developed dysfunction of at least one non-neurologic system. Most importantly, non-neurologic organ dysfunction is independently associated with a worse neurologic outcome.

Therapies to support organ dysfunction may impact other organ systems. For instance, intravascular volume therapy to support cardiovascular function can produce increased interstitial edema and pulmonary function. Nursing patients with the head of the bed elevated to treat intracranial hypertension limits the ability to do chest physical therapy. Development in pulmonary dysfunction in both of these may require increased driving pressures on the ventilator, limiting venous return and worsening cardiac function. Propofol reduces ICP and is a reversible sedative agent but also drops blood pressure, worsening cerebral perfusion. Propofol infusion syndrome may lead to or exacerbate myocardial failure. Each of these can produce a self perpetuating cycle termed multiple compartment syndrome. All of these lead to organ failure.

Extracorporeal life support (ECLS) modalities following trauma are well described both as a rescue therapy for severe respiratory therapy as well as an adjunct to resuscitation during hemorrhage control. ECLS has also been used as a therapy for patients in profound cardiogenic shock following cardiac arrest. Central nervous system injury is a relative contraindication to ECLS as it often requires systemic anticoagulation, but has been successfully performed, with good neurologic outcome.

References

1. Anderson HL 3rd SM, Delius RE, Steimle CN, Chapman RA, Bartlett RH: Extracorpreal life support for respiratory failure after multiple trauma. **J Trauma 37**:266-272, 1994

2. Atkinson JL: The neglected prehospital phase of head injury: apnea and catecholamine surge. **Mayo Clin Proc 75**:37-47, 2000

3. Bratton SL, Davis RL: Acute lung injury in isolated traumatic brain injury. **Neurosurgery 40**:707-712; discussion 712, 1997

4. Cushing H: The blood pressure reaction of acute cerebral compression illustrated by cases of intracranial hemorrhage. **Am J Med Sci 125**:1017-1044, 1903

5. Di Pasquale G, Andreoli A, Lusa AM, et al: Cardiologic complications of subarachnoid hemorrhage. **J Neurosurg Sci 42**:33-36, 1998

6. Di Russo GB MG: Extracorporeal membrane oxygenation for cardiac disease: no longer a mistaken diagnosis. **Semin Thorac Cardiovasc Surg Pediatr Card Surg Annu.**:34-40, 2005

7. Fontes RB, Aguiar PH, Zanetti MV, et al: Acute neurogenic pulmonary edema: case reports and literature review. **J Neurosurg Anesthesiol 15**:144-150, 2003

8. Friedman JA, Pichelmann MA, Piepgras DG, et al: Pulmonary complications of aneurysmal subarachnoid hemorrhage. **Neurosurgery 52:**1025-1031; discussion 1031-1022, 2003

9. Heard S FM: Multiple organ failure syndrome – part I: Epidemiology, prognosis and pathophysiology. **J Intensive Care Med 6:**4-11, 1991

10. Joseph DK DR, Aarabi B, Scalea TM: decompressive laparatomy to treat intractable intracranial hypertension after traumatic brain injury. **J Trauma 57:**687-693, 2004

11. Kirkpatrick AW GN, Brone DR, Nash D, Ng A, Lawless B, Cunningham J, Chun R, Simons RK: Use of a centrifugal blood pump and heparin-bonded circuit for extracorporeal rewarming of severe hypothermia in acutely injured and coagulopathic patients. **J Trauma 55:**407-412, 2003

12. Kono T, Morita H, Kuroiwa T, et al: Left ventricular wall motion abnormalities in patients with subarachnoid hemorrhage: neurogenic stunned myocardium. **J Am Coll Cardiol 24:**636-640, 1994

13. Maron MB: A canine model of neurogenic pulmonary edema. **J Appl Physiol 59:**1019-1025, 1985

14. Marshall J: Both the disposition and the means of cure: "Severe SIRS," "sterile shock," and the ongoing challenge of description. **Crit Care Med 25:**1765-1766, 1997

15. Massetti M TM, LePage O, Deredec R, Babatasi G, Buklas D, Thuaudet S, Charbonneau P, Hamon M, Grollier G, Gerard JL, Khayat A: Back from irreversibility: extracorporeal life support for prolonged cardiac arrest. **Ann Thorac Surg:**178-184, 2005

16. McCunn M RH, Cottingham CA, Scalea TM, Habashi NM: Extracorporeal support in an adult with severe carbon monoxide poisoning and shock following smoke inhalation: a case report. **Perfusion 15:**169-173, 2000

17. Michaels AJ SR, Kolla S, Awad SS, Rich PB, Reickert C, Younger J, Hirschl RB, Bartlett RH: Extracorporeal life support in pulmonary failure after trauma. **J Trauma 46:**638-645, 1999

18. Perchinsky MJ LW, Hill JG, Parsons JA, Bennett JB: Extracorporeal cardiopulmonary life support with heparin-bonded circuitry in the resuscitation of massively injured trauma patients. **Am J Surg 169:**488-491, 1995

19. Pyeron AM: Respiratory failure in the neurological patient: the diagnosis of neurogenic pulmonary edema. **J Neurosci Nurs 33:**203-207, 2001

20. Reynolds HN CC, McCunn M, Habashi NM, Scalea TM: Extracorporeal lung support in a patient with traumatic brain injury: the benefit of heparin-bonded circuitry. **Perfusion.:**489-493, 1999

21. Rogers FB, Shackford SR, Trevisani GT, et al: Neurogenic pulmonary edema in fatal and nonfatal head injuries. **J Trauma 39:**860-866; discussion 866-868, 1995

22. Simmons RL MA, Heisterkamp CA, Ducker TB.: Respiratory insufficiency in combat casualties: II-Pulmonary edema following head injury. **Ann Surg 170:**39-44, 1969

23. Theodore J, Robin ED: Speculations on neurogenic pulmonary edema (NPE). **Am Rev Respir Dis 113:**405-411, 1976

24. Weinberg SJ, Fuster JM: Electrocardiographic changes produced by localized hypothalamic stimulations. **Ann Intern Med 53:**332-341, 1960

25. William J MR: The inflammatory response. **J Intensive Care Med 7:**53-66, 1992

26. Wittebole X, Hantson P, Laterre PF, et al: Electrocardiographic changes after head trauma. **J Electrocardiol 38:**77-81, 2005

27. Wittenstein B, Ng C, Ravn H, et al: Recombinant factor VII for severe bleeding during extracorporeal membrane oxygenation following open heart surgery. **Pediatr Crit Care Med 6:**473-476, 2005

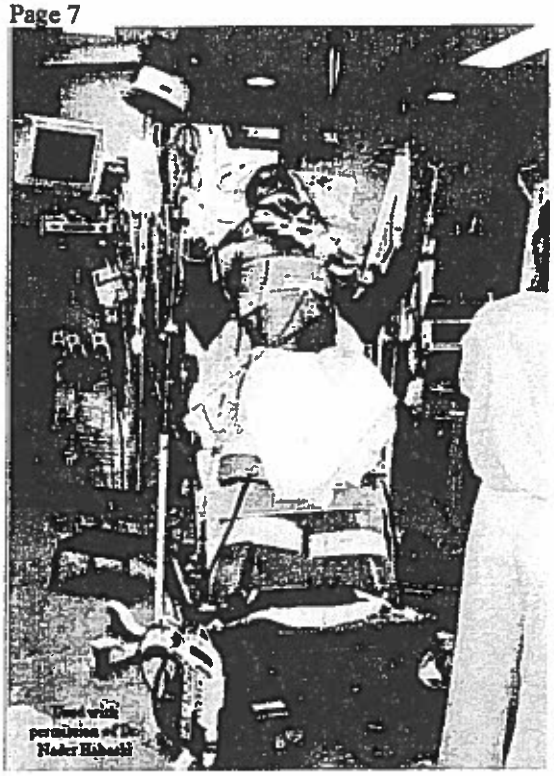
28. Zygun DA KJ, Fick GH, Laupland KB, Doig CJ: Non-neurologic organ dysfunction in severe traumatic brain injury. **Crit Care Med 33:**654-660, 2005.

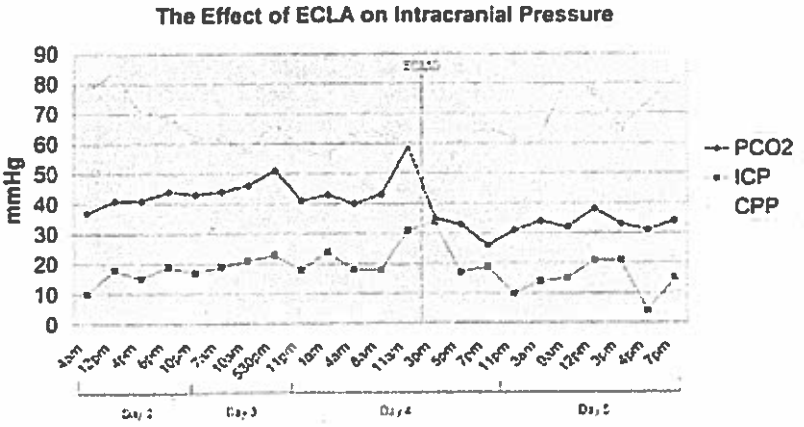
SELECTIVE REFERENCES

1. Zygun DA KJ, Fick GH, Laupland KB, Doig CJ: Non-neurologic organ dysfunction in severe traumatic brain injury. **Crit Care Med 33:**654-660, 2005

2. Michaels AJ SR, Kolla S, Awad SS, Rich PB, Reickert C, Younger J, Hirschl RB, Bartlett RH: Extracorporeal life support in pulmonary failure after trauma. **J Trauma 46:**638-645, 1999

3. Joseph DK, Aarabi B, Dutton RP, Scalea TM: Decompressive laparatomy to treat intractable intracranial hypertension after traumatic brain injury. **J Trauma 57:**687-693, 2004.





LEARNING SUMMARY

- Understand the pathophysiology of non-neurologic organ system failure
- Be aware of alternative therapies used in the management of critically injured patients
- List the indications and contraindications of extracorporeal support modalities following traumatic injury.

Spine Surgery in Adolescents: What Can Go Wrong?

Mohamed A. Mahmoud, M.D. Cincinnati, Ohio

OBJECTIVES

Present anesthetic plan for an adolescent who is scheduled for extensive spine surgery requiring one lung ventilation

Be familiar with the effects of different anesthetic agents on different modalities of intraoperative neurophysiologic monitoring

Devise a strategy for anesthetic management when the neurophysiologic signals are lost intraoperatively

Be familiar with physiologic and potential pathologic consequences of patient positioning for combined anterior and posterior spine surgery

Describe appropriate postoperative consultation and care for the patient with nerve injury related to positioning during surgery.

STEM CASE - KEY QUESTIONS

A 15- yr-old female with kyphoscoliosis presented for anterior thoracic vertebrectomy, posterior spine decompression with instrumentation fusion with autologus bone graft. Past medical history was significant for obesity and back pain. No previous surgeries. Laboratory tests are within normal limits .She weighs 98 kg, height 165 cm .Preoperative HR 80 bpm and BP 125/72 mm Hg. She donated 2 units of autologous blood. Last hemoglobin 4 days before surgery was 11.2 g/dL. At the preanesthetic visit, the patient and her parents are asking you what can go wrong during this procedure.

Key Questions

1. What risks and possible complications should be disclosed to the patient and her parents?
2. The parents are asking about options for post operative pain control. Is continuous epidural an option? What about single shot intrathecal morphine?
3. The surgeon did not order an electrocardiogram before surgery. Would you proceed without a base line EKG?

4. Does this patient require any further additional studies such as echocardiogram and pulmonary function tests?

5. Is her obesity a significant concern?

6. The surgeon indicates that the blood loss will be significant and is asking about all options that can minimize blood loss.

7. The surgeon told the parents about using cell saver during surgery and they are asking you not to give any homologous blood transfusion. How do you respond?

8. A new neurophysiologist is asking you not to use any inhalational anesthetic agent, nitrous oxide or muscle relaxant because they will affect his neurophysiologic monitoring of the patient. How do you handle this request?

9. You decide to use total intravenous anesthesia (TIVA) for anesthesia maintenance. What anesthetic agents will you choose?

10. The neurophysiologist is asking what agents you chose for TIVA?. What are the effects of high doses of propofol, remifentanil, morphine, and alpha 2 agonists on both SSEP and MEP? How do you answer this question?

11. What monitors would you use intraoperatively?

12. The orthopedic surgeon is notifying you preoperatively to get prepared for an intraoperative wake up test in addition to utilizing neuromonitoring. Does this impact your selection of anesthetic technique? How do you prepare the patient psychologically for this test?

13. The surgeon came back and told you that he forgot to mention that the last anesthesiologist he worked with was not able to give good lung isolation; and he is happy that he is working with you today. What are the options for one lung ventilation in this patient?

14. Can the position of the patient be monitored throughout the case? How can you utilize the neurophysiologist, who is asking you many questions, to help you to provide proper positioning by monitoring nerves and plexus?

The patient is turned to prone position, one orthopedic surgeon is working on the anterior decompression part and the other orthopedic surgeon is working simultaneously on the posterior fusion part of the procedure. The surgeons are satisfied with an excellent access to surgical field because of your perfect lung isolation. The estimated blood loss now is 3.5 liters. You have replaced it with the 2 autologous units, one unit of homologous blood, 1L of 5 % albumin and 3200 ml of LR. Her blood pressure is 92/68 mm Hg and her hemoglobin is 8.7 gm/dl. The surgeon is getting upset because of blood loss and is asking you to help him with controlled hypotension.

Key Questions

15. Would you agree with him? Is controlled hypotension appropriate in this setting?

16. If you do, what agents will you use to decrease blood pressure and why?

17. In spine surgery with increased difficulty with hemostasis, do you have to recheck patient position? What would you look for?

18. You gave more blood and colloid but the patient is oliguric now. Would you give more fluids to increase urine output?

19. What is the likely diagnosis?

Blood loss is controlled now and the surgeon started to smile again and telling jokes. After the surgeon placed all the screws, the neurophysiologist tells you that there is moderate attenuation of the transcranial motor evoked potential amplitude. The surgeon loosened all the screws, but the neurophysiologist is still not happy as the motor evoked potential signal is still attenuated.

Key Questions

19. The surgeon and neurophysiologist are asking if any anesthetic agent you are using could be the reason for this problem?

20. What you should do once you have any problem with neuromonitoring in spine surgery?

21. The surgeon is telling you that it could be a false positive test and he wants you to do an intraoperative wake up test.

23. Would you give steroids now or wait for the wake up test response?

You increased the mean arterial pressure and did the wake up test and patient did not have any motor deficit and the surgeon is satisfied with the response. At the end of the procedure, the

patient recovers but has a very swollen face. In the intensive care unit after you signed off to the ICU team, the patient tells that she is not able to feel her right hand and it is numb.

Key Questions

24. Why her face is so swollen?

25. How do you approach post operative nerve injury problem?

26. What would you tell the parents about the numbness?

27. When do you call risk management?

PROBLEM BASED LEARNING DISCUSSION

The anesthesiologist must anticipate all potential problems in anesthetizing a patient for complex spinal surgery and be prepared to manage all issues and complications. The use of both anterior endoscopic release and fusion combined with posterior instrumentation and fusion continues to evolve. Indications for the endoscopic fusion are large curve magnitude, skeletal immaturity, and/or thoracic hyperkyphosis. Theoretically, the advantages of anterior instrumentation include prevention of lumbar curve decompensation by shortening the convexity of the thoracic curve. In addition, by removing the disc, better correction of thoracic hypokyphosis could be obtained.

the safety of staged vs. continuous anterior and posterior spinal fusion has been studied and the results show that 1) a continuous procedure is faster than the staged procedure; 2) there is less blood loss; 3) fewer days are spent in the hospital; and 4) better correction of the spinal deformity is achieved. Also, the complications were less frequent and less severe with the continuous procedure. It was concluded that the continuous procedure is safe and efficacious and has several advantages over the staged procedure.

The primary aim of preoperative evaluation of patients with scoliosis is to detect the presence and the extent of cardiac or pulmonary compromise. Respiratory reserve can be assessed by exercise tolerance and pulmonary function tests mainly vital capacity. Cardiac studies can be performed as indicated.

The patient and her parents should be advised about all possible complications including massive blood loss, paralysis, peripheral nerve injury, and awareness during surgery, post operative vision loss, prolonged postoperative intubation, deep venous thrombosis, pulmonary embolism.

Several recently developed analgesic techniques effectively control pain after major orthopedic surgery. Neuraxial analgesia provided by epidural and spinal administration of local anesthetics and opioids provides the highest level of pain control; however, such therapy is highly invasive and labor intensive. Although intrathecal morphine is used as an analgesic in a variety of medical and surgical conditions, very little has been published on its use after posterior spine fusion . Intrathecal morphine in doses of 2 and 5 μg/kg provided potent analgesia in the first 24 h after spinal fusion in children, as evidenced by low pain scores and low additional PCA morphine consumption. Several studies have already outlined the interest of intrathecal morphine for postoperative analgesia after spinal surgery Because the thecal sac is readily available during these procedures, the addition of a single injection of morphine before wound closure can be done with technical ease if the orthopedic surgeon is willing to offer this option.Intrathecal morphine can be safe and efficacious as an early postoperative analgesic after lumbar fusion when respiratory monitoring is used Clinical advantage of epidural opiate/local anesthetic analgesia over systemic opiate by patient-controlled analgesia for spinal fusion patients still controversial. However, possible technical limitations (namely, the low dosage of bupivacaine and placement of the catheter tip) may have prevented adequate delivery of anesthetic to the involved segments. Although the incidence of side effects is similar, cost factors and a high incidence of epidural catheter dislodgment might favor use of patient-controlled analgesia.

Scoliosis surgery is an extensive operative procedure that is often associated with substantial blood loss. Significant oozing may occur from large area of exposed cancellous bone, the amount of blood loss is related to the length of time required for instrumentation. The vertebral venous system provides channels into which blood may be delivered from the lower part of the body if the inferior vena cava becomes obstructed in prone position; any rise in intraabdominal pressure is transmitted to IVC. Consequently blood is diverted into vertebral venous plexus, causing excessive blood loss. The patient's body must be well protected. There should be no abdominal compression and eyes must be padded and observed frequently and recorded in the anesthesia chart. Operative timing should be as short as possible and staging of procedures may be considered.

Infusion of tremendous amounts of crystalloids should be avoided because in prolonged spinal surgery, in the prone position especially in a degree of Trendelenberg tilt, fluids will gravitate to dependent soft tissue in the face and around the eyes causing edema and increase venous pressure, also the intraabdominal pressure will be increased due to intestinal wall edema that leads to decreased urine output and again leading to engorgement of vertebral venous plexus and excessive blood loss. Infusion of colloid is a good alternative to crystalloids. Blood replacement should not be delayed till the end of surgery even if the patient is stable, especially if the patient predonated, blood should be replaced as it is lost.

Moderate induced hypotension (reduction of systolic blood pressure 20 mm Hg from baseline or lowering mean arterial pressure to 65 mm Hg in the normotensive patient) has been shown to decrease blood loss and reduce transfusion requirements. However, induced hypotension is not

without risk and has been reported to cause cord ischemia and neurologic deficit including permanent loss of vision. Factors associated with increased risk for spinal cord injury include intraoperative mean arterial pressure less than 60 mm Hg, rapid decrease in blood pressure and anemia. So, invasive monitoring of blood pressure is advised to allow accurate assessment of blood pressure and frequent sampling to test hemoglobin.

Excessive bleeding often occurs during pediatric scoliosis surgery and is attributed to numerous factors; including accelerated fibrinolysis.Drugs that can modulate the coagulation cascade can be used to decrease blood loss during major spine surgery. These drugs include tranexamic acid, aprotenin and aminocaproic acid. Tranexamic acid is a synthetic antifibrinolytic drug that reduces transfusion requirements especially in patients with neuromuscular scoliosis. The efficacy of prophylactic Tranexamic acid to reduce perioperative blood transfusion requirement has been studied in forty patients, 9-18 yr of age, who were randomized to either tranexamic acid (initial dose of 10 mg/kg and infusion of 1 mg. $kg^{-1}.h^{-1}$ or placebo (isotonic saline). Perioperative management was standardized with a uniform transfusion threshold for noncell saved red blood cells at 7.0 g/dL. In another study included forty-four patients scheduled to undergo elective spinal fusion were randomly assigned to receive either 100 mg/kg tranexamic acid before incision followed by an infusion of 10 mg /kg/hour during surgery (tranexamic acid group) or 0.9% saline (placebo group) .The total amount of blood transfused in the perioperative period was significantly reduced in the Tranexamic group in both studies.

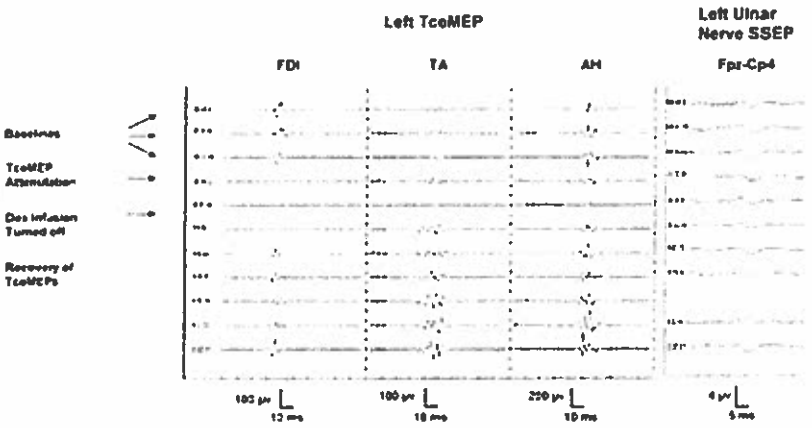
Aprotinin reduces intraoperative bleeding and transfusion requirements in cardiac surgery, liver resection and orthopedic surgery . The mechanism of this blood-sparing effect is unclear, although a protective effect on platelet membrane binding function and the inhibition of intraoperative fibrinolysis have been suggested

Intraoperative neurophysiologic monitoring using transcranial electric motor evoked potentials (TceMEP) has been increasingly utilized to reduce the risk of spinal cord injury during corrective spine surgery. Since inhalational anesthetic agents considerably depress TceMEP amplitude in a dose dependent manner, total intravenous anesthesia (TIVA) techniques with propofol as a central component have been advocated to optimize TceMEP monitoring during spine surgery.

Volatile anesthetic, nitrous oxide increases the latency and decrease the amplitude of somatosensory-evoked potentials and decrease the amplitude of motor-evoked potentials . Because spinal cord injuries also elicit the same signal changes in SSEP and TcMEP, certain anesthetics compromise the ability of the neurophysiologist to identify spinal cord injury. Therefore volatile agents and nitrous oxide as well as neuromuscular blocking drugs are avoided whenever TcMEPs are utilized

Most commonly used anesthetic drugs produce dose related changes in the amplitude and latency of TceMEP. As a result, TIVA has been increasingly used during intraoperative neurophysiologic monitoring to provide adequate anesthesia with minimal interference of neurophysiological signals.

Of the intravenous anesthetic medications, propofol has become a popular agent and as a central component of balanced TIVA. A mild depression of TceMEP amplitude will occur with high infusion rates propofol. The effects of opioid analgesics (remifentanil, fentanyl, sufentanil) on SSEP and TceMEP are less than any other anesthetic agents, making opioids as important components of TIVA for neurophysiological monitoring. Ketamine and etomidate are unpredictable in that they can increase as well as decrease evoked potentials.



Non anesthetic patient factors that affect intraoperative monitoring include spinal cord blood flow, hematocrit level, minute ventilation and body temperature.

1. Decrease in mean arterial pressure below the auto regulatory threshold will decrease SSEP and TceMEP.

2. PaCO2 less than 20 mmHg will lead to decrease in SSEP amplitude

3. Moderate systemic hypothermia will decrease amplitude and increase latency of SSEP

So the anesthesiologist must pay attention to all of these factors in case of loss or attenuation of neurophysiologic signals.

Nerve injury associated with anesthesia is a significant source of morbidity for patients and it is a liability for anesthesiologists. Improper anesthetic care and malpositioning have been implicated as causative factors in the development of ulnar neuropathy. Proper positioning and intra-operative monitoring of brachial plexus by the neurophysiologists should help to prevent and

identify early injuries. There are no new strategies for prevention of nerve damage because the mechanisms(s) for most injuries, particularly those of the ulnar nerve, are not apparent.

If the EMG is performed immediately after the onset of symptoms, it is possible to determine whether or not the neuropathy was present preoperatively because the signs of denervation resulting from acute injury appear 18-21 days after the event.

There is no reliable treatment of ulnar nerve palsy. It is difficult to predict which patient will show significant postoperative significant postoperative improvement .Usually, treatment is limited to physiotherapy to prevent excessive muscle atrophy while the nerve regenerates with reappearance of motor and sensory activity.

REFERENCES

1. Sabina DiCindino, DO, Danial Schwartz, PhD. Anesthetic management for Pediatric spinal fusion: Implications of advances in spinal cord monitoring. Anesthesiology Clin N Am 23(2005) 765-787

2. Bai-Han Li, J Lohmann, H Gregg Schuler, a Cronin. Preservation of the cortical somatosensory – evoked potential during Dexmedetomidine in Rats. Anesth Analg.96:1155-60, 2003

3. M Bloom, A Beric and A Bekker: Dexmedetomidine infusion and somatosensory-evoked potentials. J Neurosurgical Anesth. 13: 320-322, October 2001

4. KM Scheufler, J Zentner. Total intravenous anesthesia for intraoperative monitoring of the motor pathways: an integral view combining clinical and experimental data. J. Neurosurg.96:571-579, 2002

5. *Sethna, Navi. Ch.B. Zurakowski, Davi.; Brustowicz, Robert M; Bacsik, Julianne. Sullivan, Lorna; Shapiro, Frederic.* Tranexamic Acid Reduces Intraoperative Blood Loss in Pediatric Patients Undergoing Scoliosis Surgery. Anesthesiology. 102(4):727-732, April 2005

6. David T. Neilipovitz, Kimmo Murto, Leslie Hall, Nicholas J. Barrowman, William M. Splinter .A Randomized Trial of Tranexamic Acid to Reduce Blood Transfusion for Scoliosis Surgery. Anesth Analg 2001; 93:82-87

7. *Cole, Jennifer W.; Murray, David J; Snider, Rebecca J.; Bassett, George S.; Bridwell, Keith H.; Lenke, Lawrence G.*Aprotinin Reduces Blood Loss During Spinal Surgery in Children. Spine. 28(21):2482-2485, November 1, 2003.

8. *Urban, Michael K.; Beckman, James; Gordon, Michael; Urquhart, Barbara; Boachie-Adjei* The Efficacy of Antifibrinolytics in the Reduction of Blood Loss During Complex Adult Reconstructive Spine Surgery. Spine. 2001; 26(10):1152-1156,

9. *Florentino-Pineda, Ivan; Blakemore, Thompson, George H.; Poe-Kochert, Connie; Adler, Patricia; Tripi, Paul* The Effect of ε-Aminocaproic Acid on Perioperative Blood Loss in Patients With Idiopathic Scoliosis Undergoing Posterior Spinal Fusion: Spine. 26(10):1147-1151, May 15, 2001.

10. *Cheney, Frederick W. MD; Domino, Karen B. MD; Caplan, Robert A. MD; Posner, Karen L. PhD* Nerve Injury Associated with Anesthesia: A Closed Claims Analysis. Anesthesiology. 90(4):1062-1069

11. Sucato Daniel, Elerson Emily.A comparison between the prone and lateral position for performing a thoracoscopic anterior release and fusion for pediatric spinal deformity. Spine. 2003; 28 2176-2180

12. Shufflebarger Hl, Grimm JO, Bui V, Thomson JD.Anterior and posterior spinal fusion. Staged versus same-day surgery. Spine 1991 Aug; 16(8):930-3

13. Warner Me, Warner MA, Garrity JA ET al .The frequency of perioperative vision loss.Anes Analg.2001; 93:855-9

14. Stevens WR, Glazer PA, Kelley SD ET al.Opthalmic complications after spinal surgery. Spine .1997; 22:1319-24

15. Sawyer, R. J. 1; Richmond, M. N. 2; Hickey, J. D. 3; Jarrratt, J. Peripheral nerve injuries associated with anaesthesia. Anaesthesia. 55(10):980-991, October 2000.

16. L Perreault, P Drolet and J Farny. Ulnar nerve palsy at the elbow after general anaesthesia Canadian Journal of Anesthesia, 1992 Vol 39, 499-503.

SELECTIVE REFERENCES

1. Sabina DiCindino, DO, Danial Schwartz, PhD. Anesthetic management for Pediatric spinal fusion: Implications of advances in spinal cord monitoring. Anesthesiology Clin N Am 23(2005) 765-787

2. *Sethna, Navil F.; Zurakowski, Brustowicz, Robert.; Bacsik, Julianne. Sullivan, Lorna J.; Shapiro, Frederic.* Tranexamic Acid Reduces Intraoperative Blood Loss in Pediatric Patients Undergoing Scoliosis Surgery. Anesthesiology. 102(4):727-732, April 2005

3. *Cheney, Frederick W; Domino, Karen; Caplan, Robert A; Posner, Karen L.*Nerve Injury Associated with Anesthesia: A Closed Claims Analysis. Anesthesiology. 90(4):1062-1069, April 1999.

LEARNING SUMMARY

Present anesthetic plan for an adolescent who is scheduled for extensive spine surgery requiring one lung ventilation.

Be familiar with the effects of different anesthetic agents on different modalities of intraoperative neurophysiologic monitoring.

Devise a strategy for anesthetic management when the neurophysiologic signals are lost intraoperatively.

Be familiar with physiologic and potential pathologic consequences of patient positioning for combined anterior and posterior spine surgery.

Describe appropriate postoperative consultation and care for the patient with nerve injury related to positioning during surgery.

Awake Craniotomy and Seizure Focus Excision in a Young Man

Heidi M. Koenig, M.D. Louisville, Kentucky

Objectives

The participant will:

1) Understand the indications, contraindications, and preoperative preparation of the patient and the medical team for awake craniotomy.

2) Understand the three general approaches to management of the awake craniotomy.

3) Understand that the patient must be deeply sedated to tolerate certain parts of the procedure, but wide awake, yet comfortable, for the testing and resection of seizure focus.

4) Understand the major intraoperative difficulties that arise at some point during many of these procedures.

5) Understand the medications that may not be used and those that are useful and when to give more or less during the procedure.

Stem Case - Key Questions

A 24-year-old man is scheduled for left temporal craniotomy and seizure focus excision during sedative anesthetic. He sustained a seemingly minor blunt head injury several years ago and now suffers seizures almost daily. The MRI shows a tiny scar-type lesion on or near his dominant (left) motor strip. The plan is to excise this with intraoperative electrocorticography (ECoG) and motor testing.

1) Have you ever seen or performed an awake craniotomy before? What are the indications for such a procedure? Are the concerns the same for awake craniotomy and for deep brain stimulators? How about trigeminal neuralgia treatments? Are there any contraindications to such an intervention?

2) How do you talk to the patient and the team to prepare them all for this procedure without frightening them?

3) What are your options for anesthetic management? Should the patient be awake for any portion of the procedure? Should you secure the patient's airway at any point in the procedure? What medications should be utilized? Are any commonly used anesthetics contraindicated?

4) What are the five most stimulating parts of the procedure? What special anesthetic considerations are associated with each one? For example: How will you manage the pain / sedation for the head frame application? Are you and your team prepared to go with the patient to the MRI suite? Should you do the scalp block? How deep should the sedation be at

this point? What medications should you be using? What local anesthetics should you use? What medications are contraindicated?

5) What are the potential neurologic, cardiovascular, respiratory or other complications associated with an awake seizure focus excision?

PROBLEM BASED LEARNING DISCUSSION

A 24-year-old man is scheduled for left temporal craniotomy and seizure focus excision during a sedative anesthetic. He suffered a seemingly minor blunt head injury several years ago and now suffers almost daily seizures. He hates his medication regime and that he cannot drive or have even an occasional drink of alcohol. The MRI / functional MRI (fMRI) shows a tiny scar type lesion on or near his dominant (left) motor strip. The surgeon doesn't want to disable the man, so the plan is to excise this with intraoperative electrocorticography (ECoG) and motor testing during a sedative anesthetic.

Have you ever seen or performed an awake craniotomy before?

In preparation for a procedure that is unfamiliar to the team, there must be conversations which review every detail and expectation of everyone involved---in this case everyone from the patient and neurosurgeon to the nurses, psychologists and neurologists.

This is not a procedure that should be undertaken without a large amount of preparation of the patient, the family, and the whole perioperative management team. Everyone in the room needs to be aware of the fact that the patient is awake and keep their conversations to a minimum and respectable. This will facilitate communication between the neurosurgeon, the monitoring team, the patient, the anesthesiologist, and the nursing personnel.

What are the indications for such awake intracranial procedures?

A common indication is the need for intraoperative assessment and preservation of neurologic function when the procedure is in an eloquent area of the brain, as in temporal lobe surgery for seizure focus excision. The other indication is the need to verify that the procedure has indeed controlled the problem, such as the movement disorder of Parkinson's disease. Other awake neurosurgical procedures include tumor and AVM resections. Rarely, awake cranial procedures are performed for chronic pain syndromes. Basically, neurosurgeons are performing more and more invasive procedures that require patient cooperation intraoperatively. Deep brain stimulators for patients with Parkinson's disease, AVMs, tumors in eloquent areas of the brain, and stimulator for pain procedures are just a few of the procedures that require a wide awake and cooperative patient at key decision- (to resect) making points.

Are the concerns the same for deep brain stimulators as for awake craniotomy?

No. Actually in many centers neurosurgeons perform the deep brain stimulators without any anesthesia personnel present. In the United States, anesthesiologists are usually present to monitor the patient and tend to any emergencies that might arise. When performing sedation for deep brain stimulators, one is much less limited in the choice of medications. All sorts of interesting things can occur during these minimally invasive procedures, but they are usually

related to the underlying disorder. The patients must refrain from taking their medications prior to surgery for movement disorders. This so that the surgeons can be certain the abnormal movements are controlled during the intraoperative trial of the stimulator leads.

In one case, a Parkinsonian patient was experiencing such vigorous movements and profuse perspiration that it was difficult to carry out the procedure and monitoring. She was also being treated for mild nausea, which she related to having her head in a fixed position when her body was moving so much. Eventually the anesthesiologist realized the patient was experiencing diaphoresis and hypoglycemia from all the energy expenditure! A small amount of intravenous dextrose was given, the symptoms abated and she was able to go on with the testing. Those procedures are amazing! Patients, who cannot hold a cup preoperatively, can drink without spilling when the properly positioned deep brain stimulators are activated.

A good sedative to use for these procedures is dexmedetomidine (either as an infusion or as carefully (check heart rate before each additional dose) titrated bolus [10mcg] doses), as it doesn't interfere with the testing or respiratory drive, or increase secretions. Anticholinergic pretreatment is essential to prevent bradycardia. Patients who are sedated with dexmedetomidine are uncannily able to awaken themselves and cooperate when coached. They feel as if they've had a very pleasant experience afterward.

What about trigeminal neuralgia treatments?

Sedation for these procedures is extremely difficult, because, once again, the lesion is permanent. The patient must be absolutely lucid and cooperative and then deeply anesthetized several times in sequence for the ablations and testing sessions. It is important to have all the x-ray and radiofrequency ablation equipment in the room and ready to go when the patient arrives for the procedure to decrease the commotion and the patient's anxiety.

Are there any contraindications to awake intracranial procedures?

YES! A patient who is seizing almost continuously or is unable to cooperate or to lie still for many hours is not a good candidate. There are cases that are ill advised to try to do under awake conditions. The neurosurgeons, family, and neurologists all need to be realistic about what the anesthesiologist can accomplish. We're good, but we cannot perform this type of procedure in someone who does not speak the same language we do.

Do you need to meet with the patient or have a conversation with the neurosurgeon regarding this case?

You would need to set up a meeting with the neurosurgeon, the neurologist, the patient, and his parents. At the meeting you assure the patient that he'll be comfortable and detail what the plan is with regard to the halo, the urinary catheter, the lines, and the monitors. In addition, you warn him about the potential for some noise in the operating room during the procedure and that you will be with him the entire time.

You go through the requirements for the patient during the mapping and resection. Take this time to build rapport with the patient. If he wants to listen to special music during the procedure,

it must be interrupted during the testing phase. Also, if you can reach the ears, ear buds or headphones can be positioned and kept at a low volume so the surgical team is not disturbed.

When the patient arrives in the holding area, what medications can you give for the application of the head frame?

One possibility is to give small boluses of dexmedetomidine (10 mcg increments) and remifentanil (6.25 mcg increments). These are very potent drugs and should not be given by inexperienced personnel. The combination, if given quickly, can lead to extreme bradycardia, so wait for the full effect of each dose before giving more. Premedication with glycopyrrolate or atropine is necessary. Dexmedetomidine is recommended by the manufacturer to be given as an infusion of 10 micograms per kilo over 10-15 minutes. This dose at this rate can lead to bradycardia and a longer period of sedation than is necessary for head frame application. In my hands, premedication with an anticholinergic and giving tiny doses at a time and checking the heart rate before each incremental dose prevents this. Also, if you have never administered these medications by bolus before, this is not the situation in which to try something new. Remifentanil and dexmedetomidine are medications that allow the anesthesiologist to momentarily give the patient excellent analgesia and sedation for administration of the local anesthetic and application of the frame. After a few minutes the sedation wears off and the patient can travel to MRI or CT for mapping without fear of respiratory embarrassment during the process. Avoid midazolam and barbiturates as this might interfere with testing during the resection.

To decrease the pain of local anesthetic administration, you can recommend that the surgeon warm the local anesthetic and add sodium bicarbonate (0.1 meq NaBicarb/mL of local anesthetic). Giving local anesthetic by jet injection can further reduce pain.

What are your options for anesthetic management? Should the patient be awake for any portion of the procedure? Should you secure the patient's airway at any point in the procedure? What medications should be utilized? Are there any commonly used anesthetics that are contraindicated?

Textbooks and the literature describe "Awake-Asleep-Awake," "Monitored Anesthetic Care" and "Sedative Anesthetic" as options. There are many and varied combinations of medications that have been successfully utilized in a variety of combinations for this type of anesthetic. The list includes, but is not limited to, dexmedetomidine, propofol, fentanyl, remifentanil, and droperidol. In general, a fairly short acting agent (propofol, remifentanil, or dexmedetomidine) is either infused continuously or given in small frequently repeated boluses to keep the patient comfortable, yet breathing during the most stimulating parts of the process — pinning the head, scalp block, elevation of the temporalis muscle, drilling of the skull, and closure. It is also advisable to have the surgeon give additional local anesthetic into the periostial attachment of the temporalis muscle, as the scalp block does not adequately numb this.

If the patient is quite anxious and has a good airway, you may consider securing the airway whether it is with endotracheal intubation or one of the many supraglottic airways. Whatever you chose to use, it should be a technique you are facile with. In addition, if you are considering the asleep-awake-asleep technique, you must use agents that are completely reversible or quickly eliminated to allow for awake testing after an initial deep sedation or general anesthetic phase.

Are there any special considerations as you position the table? Should the anesthesiologist use soft restraints on the patient's right arm to prevent him from interfering with the surgical process?

When the patient returns from the scans and mapping, you can initiate the sedation and proceed into the operating room. Some anesthesiologists perform the scalp block. As long as there is excellent sedation and monitoring, this is not a problem.

The halo frame is secured to the operating room table and the patient is put in a slight head up and right tilt position with the use of the mechanical operating room table. You rouse the patient to be certain he is comfortable. You pad and secure his legs and tuck his left arm. The areas of interest during the testing must be free enough to allow testing during the excision. Throughout the process, you must rely on your rapport with the patient to sustain much of the calming necessary to keep him on the table.

What can you do when the patient requires more analgesia and obstructs his airway? Should you just tell the surgeon to forget it? Should you insert an LMA?

The best thing to do is to gently perform a jaw thrust without moving the surgical field. If that doesn't help resolve the situation, you can try inserting a nasal airway. This is usually too stimulating for the patient to tolerate and the patient breathes well enough not to require it any more. Of course, the patient may also move and strain during the process. **Note:** Some practitioners place a nasal airway at the beginning of the sedation and leave it in place throughout the procedure to prevent this situation. Some practitioners even topically anesthetize the airway and leave a tube exchanger in place throughout the awake portion of the procedure to facilitate rapid reintubation!

What can you do when the patient experiences excessive anxiety at the time of the drilling and becomes nauseated?

Temporarily deepen the sedation carefully. Treat the nausea with antiemetics. Many anesthesiologists give prophylactic antiemetics, such as dexamethasone and a $5HT_3$ antagonist, at the beginning of the sedation. Again, rely on the rapport you developed with the patient.

As the neurosurgeon approaches the area of interest, the patient's face and then his arm begin to twitch. What's going on? What should you do?

This is a seizure. The neurosurgeon can irrigate the area with iced saline and interrupt it temporarily. This also interrupts the electrocorticography temporarily, but it is reversible. If you give intravenous anticonvulsants, you will have the effect of the medications for a long time and

you may not be able to get adequate monitoring again. If the patient goes into a persistent seizure you must protect the airway and administer medications to stop it.

The patient is moving his thumb and great toe at the neurosurgeon's request intermittently during testing. Suddenly after about 15 minutes of testing, he is unable to move his thumb. You inform the surgeon. He mumbles and does something. Then he requests the patient move his thumb again. It moves again.

What just happened?

The surgeon was probably stimulating an area of seizure focus that demonstrated motor function on the fMRI. He will not resect it if it controls the dominant opposing digit.

When the patient becomes uncomfortable and complains of thirst and aches and such what do you do?

Depend on the rapport you developed with him earlier. Sometimes a little ice or a damp cloth on the lips is helpful. Reassure him that the team is working as quickly as is safely possible.

What can you do to get through the final twenty minutes of mapping and resection when the patient is becoming anxious and complaining of urinary urgency, itchiness, thirst, and aches?

This is common and you must reassure the patient over and over.

When the procedure is truly completed, you should administer the patient's anticonvulsant drugs and deepen the sedation. Many anesthesiologists wait until the dura is closed to initiate benzodiazepine therapy and anticonvulsant therapy.

If the surgeon does return to resect one more focus, flumazenil and naloxone will awaken the patient, but these agents are somewhat unpredictable and you may end up with a dangerously awake and frustrated patient who is possibly having a seizure! There is no right answer. If possible, allow a bit of time to pass, and then when the patient is arouseable again, retest and complete the resection.

Closing Comments

Awake intracranial procedures, which are really challenging cases for anesthesiologists, are becoming more common. Review of the procedure and expectations with everyone involved in advance of operating room entry cannot be overemphasized. Likewise, building rapport with patients is critically important. Use medications and airway management techniques with which you are familiar and comfortable. Keep in constant communication with the patient and the surgeon and the cases will go quite well and everyone will be satisfied. This is not a procedure that should be undertaken without adequate preparation of the patient, the family, and the whole perioperative management team. Prevention of perioperative problems is key to this process as it is to all anesthetics. Many management strategies are practiced at various institutions. Use medications and techniques that you are familiar with. The techniques described here and in the

references should be considered as starting points from which to tailor optimal management for individual cases.

REFERENCES

1) Bekker AY, Kaufman B, Samir H, Doyle W. The use of dexmedetomidine infusion for awake craniotomy. Anesthesia and Analgesia 2001;29(15)1251-1253.

2) Cannestra AF, Pouratian N, Forage J, et al. Functional magnetic resonance imaging and optical imaging for dominant-hemisphere perisylvian arteriovenous malformations. Neurosurgery 2004;55:804-12 [discussion:812-4]

3) Craen RA, Herrick IA, Seizure surgery: general considerations and specific problems associated with awake craniotomy. Anesth Clin of NA 1997;15(3): 655-672.

4) Gumnit RJ. Selection of adult patients for surgical treatment of epilepsy. ACTA Neurol Scand Suppl 1988;117:42-46.

5) Koenig HM. Anesthesia for Awake Intracranial Procedures. Advances in Anesthesia 2006;24:127-145.

6) Koffke WA, Tempelhoff R, Dashliff RM. Anesthetic implications of epilepsy: Status epilepticus and neurosurgery. J Neuorsurgical Anesthesia 1997;9(4):349-72.

7) Mack PF, Perrine K, Kobylarz E et al. Dexmedetomidine and Neurocognitive testing in awake craniotomy. Journal of Neurosurgical Anesthesiology 2004;16:20-25.

8) Manninen PH, Balki M, Lukitto K et al. Patient satisfaction with awake craniotomy performed for tumor surgery: a comparison of remifentanil and Fentanyl in conjunction with propofol. Anesth Analg 2006;102:237-42.

9) Maze M, Scarfini C, Cavaliere F. New agents for sedation in the Intersive Care Unit. Crit Care Clin 2001;17(4):881-97.

10) Sarang A Dinsmore J. Anaesthesia for awake craniotomy---evolution of a technique that facilitates awake neurological testing. British J Anaesthesia 2003;90(2)161-165.

11) Shreeve J. Beyond the Brain. Natl Geogr Mag. 2005;207:2-31.

12) Skukas AP, Artru A. Anesthetic complications of awake craniotomies for epilepsy surgery. Anesthesia and Analgesia 2006;102:882-7.

13) Soriano SG, Eldredge EA, Wang Ek, Kull L, Madsen JR, Black PMcL, Rivello JJ, Rockoff M. The effect of propofol on interaoperative electrocorticography and cortical stimulation during awake craniotomy in children. Paediatric Anaesthessia 2000;10:29-34.

14) Sahjpaul RL. Awake Craniotomy: controversies, indications and techniques in the surgical treatment of temporal lobe epilepsy. Can J Neurol Sci 2000;27 Suppl 1:S55-63 and S92-6.

15) Venkatraghavan L, Manninen P, Mak P, Lukitto K, Hodaie M, Lozano A. Anesthesia for functional neurosurgery: a review of complications. J Neurosurg Anesthesiol 2006;18(1):64-7.

SELECTIVE REFERENCES

1) Koenig HM. Anesthesia for Awake Intracranial Procedures. Advances in Anesthesia 2006;24:127-145.

2) Mack PF, Perrine K, Kobylarz E et al. Dexmedetomidine and Neurocognitive testing in awake craniotomy. J Neurosurg Anesthesiol 2004;16:20-25.

3) Sarang A Dinsmore J. Anaesthesia for awake craniotomy---evolution of a technique that facilitates awake neurological testing. British J Anaesthesia 2003;90(2)161-165.

LEARNING SUMMARY

The participant will understand 1) patient selection, evaluation and preparation; 2) three general approaches to anesthetic management; 3) special pharmacologic considerations; and 4) potential intraoperative challenges associated with anesthetic care for awake craniotomy and seizure focus excision.

He Can't Be Blind: It Was Only Back Surgery

Elizabeth A. Frost, M.D. New York, New York

OBJECTIVES

Identify risk factors for postoperative visual problems

Be familiar with the ASA Postoperative Visual Loss Registry

Present an anesthetic plan for the patient who must undergo extensive back surgery

Devise a strategy to minimize risk of blindness

Describe appropriate postoperative consultation and care for the patient with visual loss.

STEM CASE - KEY QUESTIONS

A 56 year old bus driver is scheduled to undergo multilevel laminectomies with instrumentation, performed jointly by a neurosurgeon and orthopedist. Results of previous back surgeries, epidural steroid injections and many analgesics and even narcotic drug patches have been disappointing. Repeat MRI at this time indicates severe spinal stenosis and osteophytes. He gives a past history of heavy smoking although recently he has tried to decrease his intake. Previous surgeries have been without anesthetic complications. Laboratory tests are within normal limits except for a blood sugar level of 156mg/dl. He weighs 264lbs, height 5'10". BP is recorded at 160/95, heart rate 52. EKG shows non specific changes. Medications include metoprolol and amlodipine which he did not take today. He gives a history of recent kidney stones, treated by lithotrypsy. He requires glasses for reading. He donated 2 units of autologous blood 10 and 3 days before surgery. Immediately preoperatively, his Hb is 13.8g. At the preanesthetic assessment on the day of surgery, the patient asks the anesthesiologist what, if any risks are associated with anesthesia for this procedure

Does this patient require any further tests?

Does this level of hypertension increase risk?

What would be the optimal intraoperative level for blood pressure?

What are the effects of hyperglycemia?

Should this level of hyperglycemia be treated?

At what level of Hb should he be transfused?

Should any other risks be disclosed with the patient and family?

Is his history of multiple drug ingestions including narcotics and steroids of importance?

Both surgeons indicate that blood may be required. The surgery is scheduled for 7-9 hours.

Does the surgical opinion impact your management?

Is his obesity of concern?

Is continuous epidural anesthesia an option?

What monitors would you select?

Would blood conservation techniques be appropriate?

The neurosurgeon wants to monitor evoked potentials; does this impact your selection of anesthetic technique?

After induction of general endotracheal anesthesia, the patient is placed in a prone position. His head is placed on a foam pillow and all attempts are made to ensure that the eyes are not compressed. The face position is checked frequently throughout the procedure. A Wilson frame is used.

What types of tables are suitable?

How should the head be positioned?

How should the rest of the body be positioned?

Should the position be monitored throughout the case?

The case lasts for 9 hours.Blood loss is estimated at 3,500ml, replaced with 10 liters lactated Ringer's solution, 2 units of colloid, 2 units of autologous blood given between 6-7 hours, 1750ml cell saver blood returned after 7 hours, 1 unit of banked blood in the last hour and 1 liter of normal saline. Urine output is 535ml despite bolus infusions of crystalloids. Vital signs are fairly stable, with the blood pressure in the range of 110/80 to 95/70 thoughout the procedure. The final Hb is 9.6g.The patient awakes promptly at the conclusion of surgery but when he is turned his face is noted to be very swollen. In the PACU, he complains that his vision is blurred even when he pushes his eyes open and puts his glasses on. He cannot see the clock on the wall. The orthopedic surgeon is called and he, in turn, calls the anesthesiologist, questioning excessive pressure on the eyes, hypotension or anemia as possible causes.

Was the fluid replacement optimal?

Was blood pressure control appropriate?

Were the eyes adequately protected?

Why is the face swollen?

Is the patient oliguric?

What tests, consultations and/or therapy should be done now?

What is the likely diagnosis?

PROBLEM BASED LEARNING DISCUSSION

The most common eye injury after surgery is corneal abrasion. Blindness is very rare. Older studies have reported incidences of postoperative visual loss as approximately 1:61,000 cases. Since these earlier reports there has been further awareness in the anesthetic community that this devastating complication can occur after several different types of surgery: 0.11% after cardiac surgery and 0.08% after spine surgery. The incidence may be even higher as many cases may not be reported for medico legal reasons. Many reasons have been suggested for the increase. It may be that with the improvement in safety of anesthesia, rare problems assume greater visibility. Or it may be that with more aggressive surgery, procedures are longer and more complex, often combining hypotensive techniques and performed on older patients with more co morbidities who are at greater risk for postoperative problems. With the risk, small as it is, of disease transmission with blood transfusion, patients and their doctors are often hesitant to use banked blood. In the past, ophthalmologists have opined that the occurrence of visual defects after anesthesia is common and thus unremarkable. Also anesthesiologists have been unwilling to come forward with descriptions of adverse outcomes for fear of litigation. Data collection to define risk factors has been slow and difficult although several recent reviews have gone a long way to identify common elements and define better strategies.

After identification of postoperative blindness as a major concern by practicing anesthesiologists, the American Society of Anesthesiologists (ASA) Committee on Professional Liability established the ASA Postoperative Visual Loss (POVL) Registry to collect detailed information on these cases in 1999. The goal of the Registry was to identify intraoperative risk factors and patient characteristics by analyses of all cases received. (Using models developed through the ASA Closed Claims Project, the Committee has posted a detailed case report form on the ASA web site with instructions for anonymous case submission (www.asaclosedclaims.org). Completion of the reports requires access to preoperative, intraoperative (including all anesthesia charts) and postoperative (PACU and ophthalmologic examination) records. Although the case form is designed to be used by anesthesiologists, other health care professionals are encouraged to assist). Over 100 cases have been collected (which was the initial goal) and reported on in 2006. Additionally in 2005, the ASA appointed a task

force of 12 members to develop a practice advisory for perioperative visual loss associated with spine surgery

From the earliest reports it was clear that POVL was not a single entity. In a few cases, cause could be identified, as, for example, when foreign bodies entered the eyes causing corneal abrasions or pressure directly on the orbits had been long and excessive resulting in central retinal arterial or venous thrombosis. Also, some cases resolved completely, and others did not, probably due to the area of the nerve damaged and its blood supply.

Injury due to an ischemic event in the visual pathway that results in postoperative visual loss may be associated with several causes of decreased oxygen delivery.

Classification is based on the site of injury.

Ischemic injury to the optic nerve is divided to anterior ischemic optic neuropathy (AION) and posterior ischemic optic neuropathy (PION).In AION, optic disc swelling may be seen and in PION, fundal examination is normal initially but disc pallor becomes apparent.

Cortical blindness results from emboli, shock, or cardiac arrest and is caused by damage to the occipital cortex. Blindness, normal fundal examination and retention of light response are seen

Central retinal arterial occlusion occurs after embolic or thrombotic events or is associated with excessive extraocular pressure. A characteristic "cherry red" spot is seen on the retina.

Central retinal venous occlusion is diagnosed by finding retinal hemorrhages in all 4 quadrants, cotton wool spots and dilated tortuous retinal veins

Posterior ischemic optic neuropathy (PION), the most likely cause in this case, presents as acute loss of vision and is likely due to decreased oxygen delivery to the posterior part of the optic nerve. Although AION and PION are manifestations of vascular insufficiency to the optic nerve, they represent two subtly different pathophysiologic entities, a difference related mainly to the very different blood supplies.

Identification of risk factors for POVL has been the subject of several recent reviews. Prone position and long surgery are readily associated with POVL. Time honored speculation, mainly by surgeons and ophthalmologists has identified pressure on the eyes and, intraoperative hypotensive hypovolemia, combined with perioperative anemia as obvious risk factors. Review of cases from the registry indicates that direct eye pressure is probably not a factor but even mild hypotension sustained for hours may be partially causative, especially in a patient previously documented as hypertensive. To decrease the need for replacement with banked blood, crystalloid infusions are often given and the Hct decreased, resulting in hemodilution. Postoperative facial swelling is usually marked. Altered ocular venous pressure may also be a factor. Avoidance of Trendelenberg position and minimization of intravenous fluids are appropriate steps to avoid the complication. Intraocular pressure is increased in the prone position and thus ocular perfusion pressure decreases. Urine output is often markedly decreased or even absent. A table frame such as the Jackson frame allows better accommodation of an increased girth and may improve renal flow by maintaining renal perfusion pressure. Also, if

intraabdominal pressure is maintained close to normal, pressure is not increased in epidural veins and thus bleeding is lessened. Anatomical variation in the blood supply to the optic nerve is undetectable to the anesthesiologist but might explain why only a few patients sustain the complication. Or it may be that the optic head variation, combined with major shifts in fluid balance and in blood pressure may be responsible.

The POVL Registry has identified some common findings.:

1. The cause of postoperative blindness appears to be ischemic optic neuropathy in about 90% of cases. In only 6% (n=3) was central retinal artery occlusion diagnosed (i.e. POVL is rarely due to pressure on the eyes).

2. The prone position places patients at risk. The incidence dramatically increases for prone times between 5 and 9 hours. But the Committee cautions that as the Registry does not contain denominator data of all cases in the prone position, definitive conclusions regarding risk and duration of the prone position cannot be made.

3. Younger age does not appear to be protective as many patients are under 60 years. The high occurrence in younger, healthier individuals suggests that intraoperative physiologic variables such as edema formation and venous congestion in the prone position as well as "normal" physiologic variation in ocular hemodynamics may be important etiologic factors.

4. Measurement of intraocular pressure (IOP) over time in the prone position indicates about 100% increase over 6 hours and uniform increases from baseline of 20+/- 7mmHg to 29 +/- 9mmHg in the initial prone position to 41 +/- 10mmHg at the end of surgery. Given this increase in IOP, decreased mean arterial blood pressure could markedly reduce ocular perfusion pressure. Other authors have cited high blood sugar levels postoperatively and suggest that tight control during the perioperative period is essential to increase neuronal survival.

5. In all cases there was considerable blood loss and replacement with large volumes of crystalloid solutions.

Sifting through the available evidence it appears that certain patients are at risk of developing POVL. Although presence of any single factor listed below may not place the patient at increased risk, the combination of several circumstances should be considered as potentially problematic. Identified factors are as follows:

1. Repeat spinal surgery and the prone position. The patient may be a chronic pain patient who has had many previous surgeries and now presents for a potentially long procedure, which requires extensive instrumentation. Considerable blood loss may be anticipated and the patient may have predonated blood, thus reducing his/her hematocrit preoperatively.

2. Disc disease is often associated with smoking, obesity and sedentary life style. Obesity was identified in many of the registry patients. Hypoxia and/or bronchospastic disease may occur during anesthesia.

3. Hypertensive patients are often unstable intraoperatively and given the decrease of IOP associated with the prone position, ocular perfusion pressure may be seriously decreased if any period of hypotension occurs or if it is prolonged.

4. Diabetes and increased perioperative glucose levels have been associated with poor neurologic outcome as hypoxic or ischemic tissue is unable to metabolize sugar through normal pathways and the size of infracted areas is increased. Patients undergoing spinal surgery are often treated prophylactically with steroids to decrease edema formation, which further increases blood glucose levels. Stress also contributes to hyperglycemia. Recent studies have emphasized the need for tight perioperative glycemic control.

5. Hemodilution and predonation therapy may result in anemia. Earlier guidelines for care of the young trauma victim suggested that blood could be replaced with crystalloid in the amount of 1 to 3ml. However, patients for prolonged spinal surgery are usually not healthy. Crystalloids stay in the circulation for about an hour before leaking to other tissues. In the prone position, especially if there is a degree of Trendelenberg tilt, fluid will gravitate to dependent soft tissues in the face and around the eyes causing edema and increasing venous pressure. Excess fluid also fills the intestinal wall, further increasing intraabdominal pressure which decreases renal output (which may intern be treated by increased fluid boluses) and increases bleeding from epidural venous plexuses. Current guidelines advocate replacement of blood as necessary to maintain adequate oxygen delivery.

From the preanesthetic interview, the anesthesiologist must consider the management of a patient for complex spinal surgery in all its aspects. The patient should be advised that there is a risk for POVL. Several other perioperative precautions should be taken:

1. Preanesthetic assessment should investigate any history of vascular disease or diabetes and ensure that the patient is in optimal condition. A history of previous visual problems should be sought and documented.

2. The patient's body must be well protected. There should be no abdominal compression and the eyes should be padded and observed frequently. Notation must also be made at regular intervals on the record that the patient has been checked .Use of a Jackson frame may be indicated as it allows the abdomen to hang free and thus is less likely to cause increased intraabdominal pressure and decreased renal perfusion.

3. The head should be positioned at or above the level of the heart. If a Wilson frame is used, flexing the spine frame, allows the legs to be lowered, thus improving gravitational blood flow away from the operative site. However, it is important to note that there are different sizes of Wilson frames and using one that is too small may not prevent increased intraabdominal pressure or ensure that the head is positioned above the operative site.

4. Invasive monitoring of blood pressure is advised to allow accurate assessment of blood pressure and sampling of blood sugar levels. Elevated blood sugar levels should be promptly treated.

5. Blood pressure should be maintained at normal levels or at least within around 20% of baseline levels. Identifying a baseline value should be made on a review of previous readings. Knowledge that the patient has been diagnosed as hypertensive for a period of time should be taken into consideration. Antihypertensive medications may decrease blood pressure but do not cure the underlying disease. Should hypotension occur intraoperatively, the anesthetic depth should be reviewed and corrected if excessive. It may be necessary to change the anesthetic technique. Blood and colloid replacement should be prompt and occur as blood is lost. Volume status should be assessed frequently. Finally, neosynephrine may be given in bolus or low dose infusion.

6. Fluid balance should be maintained. Careful measurement of fluid input and output must be maintained (placement of a Foley catheter is necessary) and hemodilution should be minimized.

7. Infusion of colloid is an alternative to multiple liters of crystalloid.

8. Urinary output should be maintained at about 1ml/kg/hour, using small doses of furosemide (5-8mg) if necessary rather than resorting to large fluid challenges, especially in otherwise healthy individuals.

9. Blood replacement should be timely. Frequently anesthesiologists delay replacing blood until the end of the case in an otherwise stable patient, reasoning that it is preferable that the patient lose less of the high Hb replacement blood. Especially if the patient has predonated, and recognizing that replacement will be necessary, blood should be replaced as it is lost. Although base line and periodic Hct levels are customarily measured, red blood cell transfusions should not be dictated by a single hemoglobin "trigger". Also, shortly after predonation, the Hb level may be abnormally low.

10. Operative time should be kept as short as possible. Staging a procedure may be an alternative.

11. Accurate charting and recording of as much intraoperative information as possible is essential. The use of electronic record keeping is advised. Some anesthesiologists have argued that swings in blood pressure are common intraoperatively and although there are usually no postoperative consequences, these aberrations might only provide fodder for a plaintiff's lawyer. A defense expert can more easily persuade a jury that care and attention was given to the patient if much legible information is available. However, should the blood pressure be recorded as severely depressed for hours and no action was taken, then the defending anesthesiologist may well experience difficulties in the face of an adverse outcome.

12. Follow up of the patient through the Postoperative Care Unit with documentation is important. POVL may not be realized for several hours after emergence from anesthesia, especially if the eyes are swollen shut or if the patient does not have access to his glasses or his trachea has remained intubated Attempts to assess vision should be made and recorded as soon as possible. Also, if facial swelling is apparent, the patient should be placed in reverse Trendelenberg position, diuretics given to increase urinary output and promote fluid shifts

from the tissues, blood replaced to restore Hb to preoperative levels, normoglycemia assured and hemodynamic and respiratory stability maintained. Appropriate consults should be obtained

While adherence to these recommendations will decrease the risk of POVL, there remain unanswered questions. For example, although studies have confirmed that intraocular pressure increases approximately 100% in the prone position during prolonged operations, this measurement alone does not predict patients who will develop POVL. In a study of 23 cases, all of whom showed some increase in intraocular pressure, only one patient, who had only moderate increases in pressure, developed POVL. Placing the head in Mayfield tongs does not alter intraocular pressure increases or prevent POVL. It has been suggested that anatomic abnormalities that cannot be recognized preoperatively by the anesthesiologist may be a causative factor, especially when combined with some of the other factors identified.

The anesthesiologist must respond promptly to the consult. It is most important to accurately record the events of the case and to avoid "finger pointing". Other causes of visual difficulties such as corneal abrasions or excessive residual eye ointment should be excluded. The combination of red painful eye and visual loss may also be due to acute angle glaucoma, which has been described after many types of non ocular surgery. The patient may or may not have a previous history of eye disease but is usually over 50 years. Headache often accompanies the visual loss. While some authors have recommended visual testing postoperatively for early detection of ION, simple tests such as pupil reactivity or gross visual field testing may not be adequate. Because in its early course, PION may be reversible, aggressive treatment is essential. Ophthalmologic consult should be requested immediately. Therapy should be directed at improving ocular perfusion pressure to restore and improve circulation in the short posterior ciliary arteries. Mannitol, acetazolamide, furosemide and/or topical timolol may be used to lower intraocular pressure. Fluid replacement is recommended in the patient who is hypovolemic and/or hypotensive postoperatively. Anemia should be promptly corrected. Use of pressors that act by peripheral vasoconstriction is not indicated. Steroids have been used although no large studies have proven their efficacy (except in cases of AION due to arteritis). Moreover, any increase in glucose levels may further damage neural tissue. In patients with a history of glaucoma, the pressure should be measured and appropriately controlled. In most instances, resolution or at least improvement is to be expected. Nevertheless, the anesthesiologist is urged to contact risk management of the hospital, be aware of all consultations and communicate with colleagues, the patient and family.

REFERENCES

1. Torossian A, Schmidt J, Schaffartzik W, Wulf H. Loss of vision after non-ophthalmic surgery. Systematic review of the literature on incidence, pathogenesis, treatment and prevention. Anaesthetist, 2006; 55(4): 457-64

2. Kamming D, Clarke S. Postoperative visual loss following prone spinal surgery Br J Anaesth 2005. 95(5): 257-60

3. Rupp-Montpetit K, Moody ML Visual loss as a complication of non-ophthalmic surgery: a review of the literature. Insight, 2005; 30(1): 10-17

4. Gill B, Heavner JE Postoperative visual loss associated with spine surgery Eur Spine J 2006; 15(4): 479-84

5. Katz DA, Karlin LI Visual field defect after posterior spine fusion Spine. 2005 30(3): E 83-5

6. Hunt K, Bajekal R, Calder I, Meacher R, Eliahoo J, Acheson JF. Changes in intraocular pressure in anesthetized prone patients J Neurosurg Anesthesiol 2004; 16(4): 287-90

7. Ho VT, Newman NJ, Song S, Ksiazek S, Roth S. Ischemic optic neuropathy following spine surgery J Neurosurg Anesthesiol 2005; 17(1); 38-44

8. Lauer KK. Visual loss after spine surgery J Neurosurg Anesthesiol 2004; 1691); 77-9

9. Brown RH, Schauble JF, Miller NR. Anemia and hypotension as contributors to perioperative loss of vision. Anesthesiology. 1994; 80: 222-6

10. Posner KL. Committee on professional liability forms a new registry to investigate postoperative blindness. ASA Newsl 1999;63:25

11. Buono LM, Foroozan R Perioperative posterior ischemic optic neuropathy: review of the literature. Surv Ophthalmol. 2005; 50(1): 15-26

12. Cheng MA, Todorov A, Tempelhoff R. The effect of prone positioning on intraocular pressure in anesthetized patients Anesthesiology. 2001; 95: 1351-5

13. Practice guidelines for Blood Component Therapy; A Report by the American Society of Anesthesiologists Task Force on Blood Component Therapy. Anesthesiology. 1996; 84:732-47.

SELECTIVE REFERENCES

1. Kawasaki A, Purvin V recovery of postoperative visual loss following treatment of severe anaemia Clin Experiment Ophthalmol 2006; 34(5): 497-9

2. American Society of Anesthesiologists Task Force on Perioperative Blindness. Practice advisory for perioperative visual loss associated with spine surgery. Anesthesiology 2006; 104(6): 1319-28

3. Lee LA, Roth S, Posner KL, Cheney FW, Caplan RA, Newman NJ, Domino KB. The American Society of Anesthesiologists Postoperative Visual Loss Registry: analysis of 93 spine surgery cases with postoperative visual loss. Anesthesiology 2006; 105(4): 652-9

Further information

www.asahq.org

www.apsf.org

LEARNING SUMMARY

1. Identify risk factors for postoperative visual problems
2. Be familiar with the ASA Postoperative Visual Loss Registry
3. Present an anesthetic plan for the patient who must undergo extensive back surgery
4. Devise a strategy to minimize risk of blindness
5. Describe appropriate postoperative consultation and care for the patient with visual loss.

Only a MAC Case in Interventional Neuroradiology. The Patient for Vertebroplasty and Kyphoplasty

Elizabeth A. Frost, M.D. New York, New York

OBJECTIVES

At the end of the lesson, the reader will be able to:

Cite the incidence of osteoporosis

Identify the differences between vertebroplasty and kyphoplasty

Understand the special needs of providing anesthesia in the radiology suite

Anticipate problems and complications of the procedures

Outline a plan for moderate sedation/analgesia for kyphoplasty.

Identify changes in lung dynamics caused by vertebral fractures and position changes.

STEM CASE - KEY QUESTIONS

An 83 year old Chinese woman with limited communication ability in English with marked osteoporosis and several lumbar and thoracic vertebral fractures presents to the hospital with severe pain. She has been followed in the pain clinic for some years without significant improvement, despite epidural steroid injections and several courses of opioid patches. At this point, she is bed ridden and has had several bouts of aspiration pneumonia and repeated urinary tract infections. Hb level is 9.9gm She is unable to lie flat because of pain. She has a past history of hypertension treated with hydrochlorothiazide and metoprolol. Other medications include oxycodone, acetaminophen, omeprazole, albuterol inhaler, and sertraline Cardiac function was normal by echocardiography. Multilevel vertebroplasty and kyphoplasty has been planned in the neuroradiology suite. The neuroradiologist asks for conscious sedation with the presence of an anesthesiologist. He notes that he has done many such cases without the need for general anesthesia.

Does this patient require any further tests

What are the considerations for anesthesia in remote locations?

What are the special requirements for sedation/analgesia for vertebroplasty and kyphoplasty

How do vertebral fractures alter lung dynamics?

Is the opioid or steroid administration of significance?

Is she a suitable candidate for conscious sedation/moderate sedation/analgesia?

What is the impact of the co-morbidities, severe pain and possible language barrier in the selection of an anesthetic technique?

What risks should be disclosed?

The patient is brought into the neuroradiology suite. She is semi sitting in bed, breathing oxygen, 2l/min. Respiratory rate is 30/minute; blood pressure 165/90; pulse rate 64/minute. She is very apprehensive, gripping the bed sheets. The neuroradiologist says that the patient must be positioned prone and he will require about one to two hours as he is trying out some new equipment. He plans to insert balloons on both sides of the L1 vertebral body and to inject cement at two other thoracic levels. However, he requires that the patient lie completely still to achieve best imaging. At the time of balloon insertion and injection, he advises that there may be considerable pain.

How can analgesia best be achieved?

What alternatives exist if the patient cannot tolerate the prone position?

What are the alternative anesthetic techniques?

If general endotracheal anesthesia is elected, what are the plans for emergence?

The patient is moved to the imaging table and attempts made to position her prone. However, she cries out in pain. Standard ASA monitors are applied. She is medicated with midazolam 0.5mg and fentanyl 50ug. SPO2 is 96% on supplemental oxygen. She is turned to a semi prone position. She is moaning and cannot lie still. Pulse rate is 66 and BP has increased to 190/95. SPO2 is 95%. The surgeon is anxious to start.

How should we proceed?

Should alternative strategies be considered?

What if the patient refuses intubation?

The surgeon points out that if he could only insert the balloons which should not take longer than 20 minutes, the pain will be greatly relieved. Also, her medical doctor gave her clearance and he is uncertain how her condition can be improved.

Does that statement influence your strategy?

Should the case be postponed at this point?

Could anything be done to improve the patient's condition?

The patient was returned to the stretcher and an interpreter was contacted. The patient indicated that she needed to urinate and was concerned that her daoughter might not be able to locate her in the basement. She was reassured and the procedure was explained more precisely to her, including the need for her cooperation. She agreed to continue and asked for some more pain medication.

Problem Based Learning Discussion

Osteoporosis, or porous bone, is a disease characterized by low bone mass and structural deterioration of bone tissue. The resulting bone fragility leads to increased fractures of all bones, especially the hip, spine, and wrist. According to the National Institutes of Health on Osteoporosis, 10 million Americans suffer from osteoporosis and an additional 34 million are classified as having low bone mass. One in every two women and one in four men over 50 will have an osteoporosis related fracture in their lifetime. Osteoporosis is responsible for an estimated 1.5 million fractures, including >700,000 vertebral compression fractures annually, costing approximately $17 billion in direct national expenditure. As the population ages, costs are predicted to rise to an estimated $60 billion by 2030 or $164 million/day The demographics of this population are thus predominantly Caucasian and females of Asian descent, and with the co morbidities of the geriatric group. Other risk factors include poor nutritional status, a history of a primary relative with bone fragility, inactive lifestyle, early menopause, smoking, steroid use and alcohol. Diagnosis depends on a bone mineral density test and a duel-energy x-ray absorptiometry (DEXA) test.

Percutaneous vertebroplasty (PV) is a minimally invasive therapy that involves the injection of polymethylmethacrylate (PMMA, liquid or powder) into a collapsed vertebral body to stabilize it. Kyphoplasty is a more advanced technique that includes the insertion of a balloon which is then filled with cement. The filling materials should have good biocompatibility, good biomechanical strength and stiffness and radio opacity for the fluoroscopy guided procedures. Synthetic bone substitutes such as resin materials, calcium phosphate or sulfate cements and several formulations of PMMA are available.

Vertebroplasty was initially considered an outpatient procedure that did not require the involvement of an anesthesiologist. As the ability of the procedure to dramatically decrease pain has been repeatedly demonstrated, it has been applied to many more and sicker patients. Considerable flexibility is required by the anesthesiologist to present an analgesic plan in a distant location, depending on the unique circumstances of the patient.

Percutaneous vertebroplasty was first described by Herve Deramond in 1984 for the treatment of painful vertebral angiomas. Now it is used as a palliative treatment for osteoporotic and malignant vertebral lesions which weaken vertebrae and cause chronic pain. Over 70 studies involving >4000 patients have now been published. The technique has been shown to improve mobility and lessen pain almost immediately with a low complication rate. Improvement in pain

scores is dramatic. One recent series of 204 patients indicated a reduction of pain score from 8 at baseline to 1 at the end point (p= 0.0001) with 61% giving up brace support, and 62% no longer requiring any drug therapy. Beneficial results appear to be long standing.

Selection criteria are:

New fracture (1-7 months)

Pain refractory to medical management (bed rest, analgesics, calcitonin, or external bracing).

Respiratory compromise

Potential for worsening of disease.

Exclusion criteria include:

Vertebral body height loss of 100%,

Posterior wall involvement,

Involvement of the spinal cord,

Osteolytic metastatic lesions,

Bleeding diathesis

Inability to undergo emergency decompressive surgery.

Most of the exclusion criteria relate to the potential for complications.

Percutaneous vertebroplasty is a fluoroscopic guided injection of bone cement into a compressed vertebral body. Four steps are involved:

Vertebral puncture (to access the surgical site),

Spinal biopsy (to rule out metastasis and in some cases to aid in diagnosis; avoid if angioma is suspected)

Vertebral venography (to identify perivertebral drainage and extravasation of cement)

Injection of PMMA

Complications are associated with each step of the procedure. Vertebral puncture can enter surrounding structures: veins, arteries, and pleura. The needle can disrupt the internal cortex of the pedicle, increasing the risk of PMMA leakage. Risks of extravasation are increased by the

presence of osteoporotic and osteolytic lesions. Leakage of PMMA into perivertebral veins occurs in 30-67%, and can cause radiculopathy or embolization. Rarely there may be interference with pulse oxymetry readings. Radicular pain has occurred with PMMA leakage into the neural foramina via cortical fractures, micro fractures, or destruction of venous channels. Pain has also occurred without an identifiable leak. Treatment with non steroidal anti-inflammatory agents is generally successful, suggesting an inflammatory reaction to the cement. The most severe complication of PMMA leakage is spinal cord compression and requires immediate surgical decompression. Other complications particular to PMMA are hypotension, hypoxemia, and cardiac arrhythmias. Only transient hypotension has been reported with PV, due to the minimal amount of PMMA injected. A review of 117 patients who underwent PV identified 8 cases with complications. Six were local problems (puncture site hematoma, radiculopathy), and 2 had pulmonary embolism from cement migration. The risk of cement leakage is increased with vertebroplasty, 41% versus 4% with kyphoplasty. Thus complications are more common with vertebroplasty.

Kyphoplasty is a procedure that not only stops further compression but fixes the spinal deformity. Unlike PV which only prevents further vertebral destruction, kyphoplasty (Kyphon Corporation®, Santa Clara, CA), fixes spinal deformity. It is also more suitable for patients with metastatic lesions as a better structure is created. It involves the same steps as vertebroplasty except tamponading balloons are inserted into both sides of the body of the fractured vertebrae. The balloons are inflated under direct visualization to compact the cancellous bone and reexpand the vertebral body. PMMA is then inserted into the cavity. If done within three months of the onset of pain, kyphosis can be decreased by 50%. Complications are less than after PV because of greater containment of cement but have included epidural hematoma associated with postoperative heparin administration, lower extremity motor loss from cement extravasation into the spinal column, and anterior cord syndrome.

Anesthetic assessment is critical in these patients. Some of the considerations include:

Cardiac disease

Pulmonary compromise

Urinary tract infection

Multiple medications

Metastatic disease

Poor nutritional status

Narcotic dependency

Limited mobility

As age increases so do cardiovascular and pulmonary co morbidities. The decrease in pulmonary function associated with osteoporotic vertebral fracture may be clinically significant in a patient

with already reduced pulmonary and cardiovascular reserve. Previously pulmonary function in the osteoporotic patient was described as normal perhaps because height at age 25 years and not current height was used in pulmonary function test calculations. By adjusting for this change, a statistically significant decrease in vital capacity and FEV1 suggesting restrictive lung pattern may be identified. Also, the mortality rate from pulmonary disease (not lung cancer) is increased with osteoporotic vertebral fractures. Many vertebral fractures are caused by metastatic lesions. The primary source may or may not have been identified. In addition to the effects upon the pulmonary system, vertebral fractures also affect the gastrointestinal system. Loss of vertebral height decreases abdominal space and compromises gastrointestinal function. Long term opioid use causes constipation and decreases nutrient absorption. Psychological well being is adversely affected by insomnia and depressive effects of chronic pain.

Positioning the patient suffering from osteoporotic and/or vertebral fractures may cause excruciating pain. Anesthetic induction on the gurney or administration of adequate analgesia prior to movement to the operating table is essential. Also, these patients have limited range of motion and greater chance for fracture. If possible, the range of motion should be assessed pre-operatively. Radiolucent bolsters and or air mattress should be considered. Positioning also involves ensuring that the vertebrae are in line, (allowing for mechanical limitation of movement), avoiding stretch on the brachial plexus and limb girdle, and placing the hands and feet in anatomically neutral positions. The mechanics of the interventional radiology suite may also pose a challenge for the anesthesiologist as there are 4 viewing screens for the radiologist placed between the anesthetic machine and the patient. The anesthesiologist must acquaint him/herself with the arrangement and plan ahead to allow sufficient length of tubing and wires for oxygen and monitoring equipment.

Although small doses of midazolam (1-2mg, fentanyl (25-50ug) and occasional boluses of propofol often suffice, such a technique may not provide adequate pain control and appropriate conditions for the neuroradiologist. Low dose propofol (50-100ug/kg/min), remifentanil (0.25ug/kg/min) or dexmedetomidine (0.1-0.2ug/kg/min) may be supplemental or even provide an adequate sedation technique Supraglottic devices have been used successfully, and should always be immediately available. While the procedure may be done on an outpatient basis, the preference is usually for the patient to be observed for 24 hours.On rare occasions, general endotracheal anesthesia with inhal;ation agents is required. Generally, patients can be successfully extubated as pain is often dramatically relieved by the insertion of the cement.

Anesthesia in remote locations means that the anesthesiologist is far from home base. It is essential to ensure that all equipment is immediately available and that there is the ability to communicate with the control desk.In these procedures, there is frequent movement of the X-ray equipment and viewing screens over the patient. Thus all connection tubing must have extra length and the anesthesiologist must be flexible enough to accommodate to small and ever changing space size.

References

National Institutes of Health Osteoporosis and Related Bone Diseases – National Resource Center (www.osteo.org)

Togawa D, Kovacic JJ, Bauer TW et al. Radiographic and histologic findings of vertebral augmentation using polymethylmethacrylate in the primate spine: percutaneous vertebroplasty versus kyphoplasty. Spine, 2006 1;31 (1): E 4-10

Hochmuth K, Proschek D, Schwarz W et al. Percutaneous vertebroplasty in the therapy of osteoporotic vertebral compression fractures: a critical review. Eur Radiol. 2006; Jan 5;:1-7

Lieberman IH, Togawa D, Kayanja MM. Vertebroplasty and kyphoplasty: filler materials. Spine J. 2005; 5(6 supple): 305S-316S

Barragan-Campos HM, Vallee JN, Lo D et al. Percutaneous vertebroplasty for spinal metastases: complications Radiology. 2006 Jan; 238 (1): 354-62

Anselmetti GC, Corrao G, Monica PD etal. Pain relief following percutaneous vertebroplasty: Results of a series of 283 consecutive patients treated in a single institution. Cardiovasc Intervent Radiol 2007 Jan2nd. (Epub ahead of print)

Kaufmann TJ, Trout AT, Kallmes DF The effects of cement volume on clinical outcomes of percutaneous vertebroplasty. Am J Neuroradiol. 2006; 27(9): 1933-7

Liliang PC, Lu K, Liang CL et al. Dyspnoea and chest pain associated with pulmonary polymethylmethacrylate embolism after percutaneous vertebroplasty Injury 2006 (E pub ahead of print)

Shindle MK, Tyler W, Edobor-Osula F et al. Unsuspected lymphoma diagnosed with use of biopsy during kyphoplasty. J Bone Joint Surg Am. 2006; 88(12): 2721-4

Majd ME, Farley S, Holt RT Preliminary outcomes and efficacy of the first 360 consecutive kyphoplasties for the treatment of painful osteoporotic vertebral compression fractures. Spine J. 2005; 5(3): 244-55

Rodriguez- Catarino M, Blimark C, Willen J et al. Percutaneous vertebroplasty at C2: a case report of a patient with multiple myeloma and a literature review. Eur Spine J. 2006 (Epub ahead of print).

Cahana A, Seium Y, Diby M et al. Percutaneous vertebroplasty in octogenarians: redults and follow up. Pain Pract. 2005; 5(4): 316-23

Schlaich C, Minne HW, Wagner G et al, Reduced pulmonary function in patients with spinal osteoporotic fractures. Osteoporosis International. 1998(8):261-267.

SELECTIVE REFERENCES

Hulme PA, Krebs J, Ferguson SJ et al. Vertebroplasty and kyphoplasty: a systematic review of 69 clinical studies. Spine 2006; 31(17): 1983-2001.

Taylor RS, Taylor RJ, Fritzell P. Balloon kyphoplasty and vertebroplasty for vertebral compression fractures: a comparative systematic review of efficacy and safety. Spine. 2006; 31(23): 2747-55

Shen MS, Kim YH Vertebroplasty and kyphoplasty-treatment techniques for managing osteoporotic vertebral compression fractures. Bull Hosp Jt Dis 2006; 64 (3-4): 106-13.

LEARNING SUMMARY

Understand the differences between vertebroplasty and kyphoplasty
List the indications for the 2 procedures
Plan an appropriate anesthetic technique in the neuroradiology suite.

Anesthesia for Selective Intra-Arterial Nicardipine Injection To Relieve Intracranial Vasospasm

Rafi Avitsian, M.D. Cleveland, Ohio

OBJECTIVES

At the end of this PBLD the participants should be able to:

Know the incidence, etiology, clinical picture and complications of subarachnoid hemorrhage (SAH)

Be able to define the Hunt Hess classification

Identify medical and surgical treatments of SAH

Know the important anesthetic considerations of transporting critically ill neurosurgical patients and remote anesthesia

Recognize hemodynamic complications with intra-arterial injection of Nicardipine.

STEM CASE - KEY QUESTIONS

Case:

You are called by the neurosurgeon on Saturday morning for a case in the neuroradiology suite. The patient is a 34-year-old female with a history of subarachnoid hemorrhage (SAH) secondary to a ruptured right middle cerebral artery aneurysm, which occurred three days prior. On arrival, she was Class 2 on the Hunt Hess classification. There was no motor deficit and she complained of the worst headache she had ever experienced. She also complained of photophobia and was drowsy. An aneurysm coiling procedure was performed 48-hours ago. Last night she started to develop changes in her mental status and was transferred to the Neurosurgical intensive care unit (NICU).

Questions:

What is the Hunt Hess Classification?

What is the probable cause of this mental status change?

Describe ways to confirm the diagnosis?

What is the medical treatment in this situation?

Case:

You inquire about the planned procedure and the surgeon replies, "We will do an angio first, but we might perform angioplasty or injection."

As you ready the angiography suite, you send the resident to see the patient in the NICU.

Questions:

What are the major and important issues that are specific to her problem and should be considered in your pre-operative evaluation?

What laboratory or radiologic study will help you?

Would a call to the NICU physician help? What would you discuss?

Case:

Her past medical history shows dysmenorrhea and two Cesarean sections, but no other medical problems. She has no allergies and has been taking Ibuprophen occasionally before this hospitalization. She smokes and, on occasion, drinks wine. Her mother died from a stroke in her fifties.

Her recent aneurysm coiling was under general anesthesia with no complications. She is 64 kg, 162cm and her vital signs at present are:

HR= 81 BP= 168/98 RR=16 SpO2=99% with a nasal cannula

She hardly responds to verbal stimulation when called and mumbles.

The NICU team has started an arterial line, as well as a central venous catheter and a pulmonary artery catheter.

She is NPO, getting IV fluids at a rate of 100ml and currently receiving a 500 ml of albumin bolus.

Questions:

What is your plan for transporting this patient to the angiography suite?

How would you induce and maintain anesthesia in this patient?

What are the hemodynamic goals?

What is the ideal PCO2 in this situation?

Case:

The surgeon performs an angiography and mentions that there is vasospasm in the right middle cerebral artery. His plan is to dilate it with Nicardipine injection. He starts the Nicardipine injection and you notice a decrease in the blood pressure.

Questions:

What is Nicardipine?

What important side effects are you concerned with in this patient?

Would you prepare any drips for hemodynamic support?

You notice some ST changes in the EKG, what can this be related to?

What would you advise to the surgeon at this point?

Case:

The surgeon repeats the Nicardipine injection and after performing an angiography is satisfied with arterial dilation.

Questions:

Would you attempt to emerge this patient?

What is the destination and what is your plan for transport?

PROBLEM BASED LEARNING DISCUSSION

Subarachnoid hemorrhage (SAH) is a devastating disease with high mortality and morbidity accounting for 25% of cerebrovascular deaths.[1] Nearly half of the affected patients die, some even before reaching hospital. About one-third of survivors end up debilitated.[2]

The most common cause of SAH is ruptured intracranial aneurysm (85%), although 10% are from perimesencephalic hemorrhage and 5% are from other causes.[2] About 1-5% of adults have intracranial aneurysm and 1:10000 North Americans suffer a SAH because of a ruptured aneurysm. In addition to surgical management of SAH with intravascular coiling or surgical clipping, medical therapy focuses on maintenance of adequate volume, monitoring for cerebral vasospasm, and initiation of maneuvers to improve vessel patency and cerebral blood flow (CBF).[3] Cerebral vasospasm can occur after a SAH, decreasing cerebral blood flow and worsening the clinical picture. Although 70% of patients with SAH show angiographic signs of cerebral vasospasm, a clinical picture of focal ischemia can be seen in about 30%.[4] Even though this is a self-limiting complication that usually starts within hours of the bleed (peaking at 4-10 days[5] and lasting up to 4 weeks), it can cause cerebral ischemia, stroke and even death in these patients. Despite treatments, about 13% of patients with SAH die or develop permanent disability because of vasospasm.[6] Triple H therapy—induced *H*ypertension, *H*emodylution and *H*ypervolemia, is said to decrease the ischemic effects of vasospasm.[7,8] Hypertensive hypervolemic therapy of cerebral vasospasm is not free of morbidity and mortality and can cause pulmonary edema and myocardial ischemia.[9,10] Thus timing and monitoring is very important in this type of treatment with patients usually monitored in an Intensive Care setting with trained nurses.[6] Nimodipine, a calcium channel blocker, has also been used for prophylaxis as well as treatment of vasospasm after SAH.[8,11,12]

Recently, there have been case reports of selective intra-arterial injection of Nicardipine, another calcium channel blocker, to relieve vasospasm during angiography.[13] Selective injection of Papaverine and balloon angioplasty have also been used to relieve vasospasm after SAH.[14,15] Selective intra-arterial injection of Nicardipine can cause significant hemodynamic effects and would need supportive management by the anesthesiologist.

When faced with a patient scheduled for a procedure outside the operating room, there are certain precautions that need to be considered.[16] In cases where any change in the hemodynamics can cause a drop in the cerebral blood flow, there should be closer monitoring during transportation. The following section will discuss important issues in the preoperative, intraoperative and postoperative periods when facing these patients.

Preoperative: Almost all cases of intracranial vasospasm are after a SAH. Most occur following the rupture of an intracranial aneurysm. These aneurysms may or may not have undergone a surgical clipping or an intravascular coiling. Patients are usually in the Neurosurgical Intensive Care Unit (NICU).[17] A typical case involves mental status changes or deterioration in neurologic examination, as well as Trans-cranial Doppler (TCD), which can show the vasospasm.[5]

Selective intra-arterial injection of nicardipine should be performed with selective angiography in a bi-plane radiology facility, not in an operating room. The anesthesiologist should be familiar with the procedure and its potential complications[18, 19]. Such cases are usually urgent, which leaves the anesthesiologist little time for setting up the anesthesia machine in the neuroradiology suite and visiting the patient. If the patient has had a recent procedure (angiography, intravascular coiling, or craniotomy with clipping of aneurysm) then the previous anesthesia record will have invaluable information regarding his/her airway and hemodynamic responses. There might also be information about induced hypotension or hypertension and the patient's hemodynamic response to the used pharmacologic agents. Most patients would probably be under triple H therapy and have invasive monitoring (e.g. arterial, line central line, pulmonary artery (PA) catheter, ventriculostomy). In our practice we transport patients under full hemodynamic monitoring in order to treat any hemodynamic changes which could worsen cerebral blood flow. Some patients are already intubated, but since there might be a need for frequent neurological examination, they might be under sedation with a short-acting sedative such as propofol infusion. During transport if the patient is stimulated or an interruption of propofol infusion occurs, the patient might get agitated and extubation might occur. Thus we prefer to transport intubated patients sedated and paralyzed. Any drop in blood pressure secondary to sedation should be treated. Inotropic agents as well as medications and equipment for emergency reintubation should be available during transport in case accidental extubations occur. In situations where there is ventriculostomy, it is preferable to close the ventriculostomy drain if the intracranial pressure (ICP) is not elevated in order to avoid removal of large amounts of cerebrospinal fluid (CSF) during transportation.

Intraoperative: Transportation to the neuro-radiology table should be smooth to avoid any hemodynamic changes. Reattachments of monitoring should not cause a prolonged delay in continuous patient monitoring. It is preferable to have these patients under general anesthesia for the procedure. The interventionalist often requires the patient to be completely motionless during the procedure. Airway management in the middle of the case can be disastrous if complications arise and the patient becomes hemodynamically unstable.[19] In cases when emergency craniotomy or ventriculostomy becomes necessary, having an intubated patient could save vital time. In patients who are not intubated, induction should be smooth in order to avoid a sudden drop in blood pressure and a decrease in cerebral perfusion pressure (CPP). In some situations where there is a prolonged time gap between inducation and surgical incision, since there is no surgical stimulation it might be difficult to maintain a desirable blood pressure and there might be a need for induced hypertension with an inotropic agent like phenylephrine. During the angiographic stage of the surgery, additional extensions might be needed for IV lines as well as ventilatory circuits which can increase the chance of accidental extubation or line extraction. Bolus medications should be followed by flushes in order to ensure rapid delivery to the patient

through these extension lines. Any patient movement should be avoided after angiographic mapping. Some patients are on anti-seizure medications which could increase the non-depolarizing muscle relaxant metabolic rate. An atracurium infusion might be a better choice in these situations. During multiple angiographic runs with fluoroscopy, the interventionist might require apneic periods. This can increase the chance of hypoxemia and hypoventilation as well as inadequate anesthesia level due to decreased volatile agent delivery. It is desirable to use higher FiO2 and avoid nitrous oxide to decrease the chance of dilution hypoxia during the apnea episodes. In cases where prolonged apnea is needed, addition of an intravenous (IV) anesthetic method could be desirable. Although hypoventilation should be avoided, especially in cases with high ICP, hyperventilation could also be deleterious since it can cause vasoconstriction of collateral vessels that perfuse the ischemic areas of the brain.

The challenge, thus, would be to continue the induced hypertension after the addition of nicardipine infusion. A timely start of phenylephrine boluses, along with a continuous infusion will decrease the chance of hypotension during nicardipine infusion.

The most important key to success in maintaining a stable hemodynamic state is communication between the interventionalist and the anesthesiologist regarding the start and end of nicardipine infusion and its dose. Depending on blood pressure changes in response to nicardipine infusion, there might be a need for large doses of vasopressors. We have noticed myocardial strain pattern with electrocardiographic non-specific ST and T changes with high doses of phenylephrine. The interventionalist should be informed to stop or decrease the infusion rate in order to reduce the rate of intra-operative infusion. In cases of severe bradycardia following large doses of phenylephrine, glycopyrrolate or the addition of a dopamine infusion might be necessary.

Postoperative: Even though there is an instant decrease in vasospasm of the intracranial vessels after injection of nicardipine, there might be recurrence of vasospasm. It is desirable to keep the patient intubated, with any decisions on extubation made after neurologic examination in the NICU. In the immediate postoperative period, there might still be some hemodynamic instability following inotropic agents administered to keep a higher SBP.

Before transportation from the angiography suite, the anesthesia provider should call and report the course of surgery as well as the patient's hemodynamic status, ventilatory settings and ongoing therapeutic measures to the NICU nurse accepting the patient. The patients are transported to the NICU intubated, sedated and under full monitoring to ensure adequate cerebral perfusion. Special care should be given to decrease stimulation during transport. Accidental extubation and line removal during transport remains a major threat. It is advisable to have either a physician from the interventional team or the anesthesiologist accompany the anesthesia provider to the NICU. During transportation it is necessary to have the equipment and medications for treatment of hemodynamic changes as well as emergent reintubation in case of accidental extubations.

Conclusion:

Selective intra-arterial injection of Nicardipine for treatment of vasospasm can cause major hemodynamic changes. In patients with intracranial vasoconstriction who have impaired brain perfusion, a decrease in SBP can further decrease perfusion and worsen ischemia. Close hemodynamic monitoring and the addition of inotropic agent(s) is important during transportation to the neuroradiology suite, the procedure and afterwards so as to ensure adequate perfusion of the brain.

REFERENCES

1. Wardlaw JM, White PM. The detection and management of unruptured intracranial aneurysms. Brain 2000;123 (Pt 2):205-21.

2. van Gijn J, Rinkel GJ. Subarachnoid haemorrhage: diagnosis, causes and management. Brain 2001;124:249-78.

3. Manno EM. Subarachnoid hemorrhage. Neurol Clin 2004;22:347-66.

4. Bendo AA. Intracranial vascular surgery. Anesthesiol Clin North America 2002;20:377-88.

5. Janjua N, Mayer SA. Cerebral vasospasm after subarachnoid hemorrhage. Curr Opin Crit Care 2003;9:113-9.

6. Oyama K, Criddle L. Vasospasm after aneurysmal subarachnoid hemorrhage. Crit Care Nurse 2004;24:58-7.

7. Sen J, Belli A, Albon H, Morgan L, Petzold A, Kitchen N. Triple-H therapy in the management of aneurysmal subarachnoid haemorrhage. Lancet Neurol 2003;2:614-21.

8. Dorsch NW. Therapeutic approaches to vasospasm in subarachnoid hemorrhage. Curr Opin Crit Care 2002;8:128-33.

9. Miller JA, Dacey RG, Jr., Diringer MN. Safety of hypertensive hypervolemic therapy with phenylephrine in the treatment of delayed ischemic deficits after subarachnoid hemorrhage. Stroke 1995;26:2260-6.

10. Solenski NJ, Haley EC, Jr., Kassell NF, Kongable G, Germanson T, Truskowski L, Torner JC. Medical complications of aneurysmal subarachnoid hemorrhage: a report of the multicenter, cooperative aneurysm study. Participants of the Multicenter Cooperative Aneurysm Study. Crit Care Med 1995;23:1007-17.

11. Barker FG, Ogilvy CS. Efficacy of prophylactic nimodipine for delayed ischemic deficit after subarachnoid hemorrhage: a metaanalysis. J Neurosurg 1996;84:405-14.

12. Wilson SR, Hirsch NP, Appleby I. Management of subarachnoid haemorrhage in a non-neurosurgical centre. Anaesthesia 2005;60:470-85.

13. Biondi A, Ricciardi GK, Puybasset L, Abdennour L, Longo M, Chiras J, Van Effenterre R. Intra-arterial nimodipine for the treatment of symptomatic cerebral vasospasm after aneurysmal subarachnoid hemorrhage: preliminary results. AJNR Am J Neuroradiol 2004;25:1067-76.

14. Smith TP, Enterline DS. Endovascular treatment of cerebral vasospasm. J Vasc Interv Radiol 2000;11:547-59.

15. Schuknecht B, Fandino J, Yuksel C, Yonekawa Y, Valavanis A. Endovascular treatment of cerebral vasospasm: assessment of treatment effect by cerebral angiography and transcranial colour Doppler sonography. Neuroradiology 1999;41:453-62.

16. Stensrud PE. Anesthesia At Remote Locations. In: Miller RD, editor. Miller's Anesthesia, Sixth Edition. Elsevier, 2005, pp 2637-63.

17. Kraus J, Metzler M, Coplin W. Critical care issues in stroke and subarachnoid hemorrhage. Neurol Res 2002;24 Suppl:S47-57.

18. Hashimoto T, Gupta D, Young W. Interventional neuroradiology anesthetic considerations. Anesthesiol Clin North America. 2002;20:347-59.

19. Osborn I. Anesthetic considerations for interventional neuroradiology, Int Anesthesiol Clin 2003;41:69-77.

SELECTIVE REFERENCES

Miller JA, Dacey RG, Jr., Diringer MN. Safety of hypertensive hypervolemic therapy with phenylephrine in the treatment of delayed ischemic deficits after subarachnoid hemorrhage. Stroke 1995;26:2260-6.

Manno EM. Subarachnoid hemorrhage. Neurol Clin 2004;22:347-66

Hashimoto T, Gupta D, Young W. Interventional neuroradiology anesthetic considerations. Anesthesiol Clin North America. 2002;20:347-59.

LEARNING SUMMARY

1. Identify the medical and interventional treatments of intracranial vasospasm following subarachnoid hemorrhage
2. Know the important anesthetic considerations of transporting neurosurgical critically ill patients and remote anesthesia
3. Recognize hemodynamic complications with intra-arterial injection of Nicardipine.