

Case Study:  
Non Opioid Craniotomy

By

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### **Case Study Introduction:**

Opioid use during induction and maintenance of anesthesia for craniotomy has been practiced successfully in many operating theatres. The need for neurological assessment in a patient with a recent history of cerebral vascular accident with a planned surgical bypass of the right superior temporal artery to the middle cerebral artery (MCA) necessitated a different anesthetic strategy. In this case study the anesthetic plan without opioid use is discussed. The neurological protective qualities of each of the medications used during the planned anesthetic are reviewed. Finally, the use of opioids in craniotomy is discussed.

### **Case Summary:**

Robert is a pleasant 59 year old African American male who has had two previous cerebral vascular accidents (CVA). He was admitted to a major medical center following a near syncope episode and fall. Robert recently suffered a right caudate stroke and in addition last year he sustained an acute right frontal lobe infarct.

His past medical history is significant for hypertension and two prior strokes as mentioned. He is a current smoker with a 60 pack year history, peripheral vascular disease, claudication, right internal carotid artery occlusion and a history of a left femoral artery bypass surgery.

A review of systems provides additional information about Robert's overall readiness for surgery. He currently has weakness on his left side and increased tone on his right upper and lower extremities. He denies any respiratory complications such as shortness of breath, history of asthma or tuberculosis. There is no history of chest pain, prior myocardial infarction, angina or exercise intolerance.

Laboratory data collected immediately before the scheduled craniotomy for bypass graft surgery revealed sodium of 140, potassium of 4.1, chloride of 105, CO<sub>2</sub> of 25, BUN 16 and creatinine of 1.0. Robert's white cell count was 6.3, hemoglobin of 12.5, hematocrit of 38.0, and platelets 278,000. His coagulation profile was normal. An MRI of the brain revealed a new ischemic infarct which involved the right anterior superior frontal lobe. A brain Spect Scan the day before surgery revealed a large area of activity absence in the right frontal cortex and areas of severely decreased activity in the right

parietal cortex. A subsequent vascular reserve study revealed poor collateral supply to the infarcted areas.

A cardiac consult was obtained due to Robert's extensive peripheral vascular disease. An adenosine myocardial perfusion study revealed small areas of ischemia in the anterior myocardium as well as the inferoseptal wall. His electroencephalogram showed normal sinus rhythm. Subsequently he was considered medically optimized on his current medication regimen of Zocor 20 milligrams daily and prevacid 30 milligrams daily. Robert has been noncompliant with taking the coumadin that had been prescribed for him in the past.

A physical examination of Robert revealed a pleasant African American without any acute distress. His blood pressure was 140/80, a pulse of 72 and respiratory rate of 12. He was afibrile. An examination of his chest revealed clear lung fields and heart tones that are regular without an appreciable murmurs, gallops or rubs. He does exhibit marked left sided weakness with strength 4/10 in both upper and lower extremities. Vascular access was impossible to obtain in a peripheral vein due to his extensive peripheral vascular disease. Two skilled anesthesia providers were unable to acquire vascular access before surgery.

It was decided to not attempt central venous access in his groin due to his prior femoral bypass graft surgical procedures that he has undergone. Additionally it was felt that neck access was another poor choice as vascular complication in this area might worsen supply or drainage from his brain. The decision was made to perform a mask induction with Sevoflurane and then to obtain vascular access once he became more peripherally dilated.

The anesthetic was explained to Robert and it was decided to proceed with this semi-emergent surgery using an inhalational induction. His ischemic stroke was 48 hours old. All ASA monitors were employed and the patient was pre-oxygenated for several minutes before induction. The inhalational induction with Sevoflurane was accomplished with preloading the anesthesia circuit with 3 percent volatile agent. Within several breaths Robert had lost his eye lid reflex. Taking over his respirations with a hand mask ventilation technique, the depth of anesthesia was increased. Succinylcholine 120 mg was given intramuscularly and within 100 seconds his airway was secured with an

endotracheal tube. Succinylcholine has traditionally been thought to increase intracranial pressure (ICP). The mechanism of this increased ICP is has now been shown to be a result of muscle fasciculation which can be blunted with a defasciculating dose of a non-depolarizing muscle relaxant (Warner, 2002). The volatile agent was switched to Desflurane at 3 percent and mechanical ventilation was initiated. At this time vascular access was able to be performed. A propofol infusion was then started at 80 mcg/kg/min. An arterial line was placed in his left radial artery to monitor beat to beat blood pressure and facilitate arterial blood gas measurement and serial hemoglobin levels. A foley catheter was also inserted to measure urine output. An intravascular infusion of mannitol 0.5 gr/kg was instituted to provide an osmotic diuresis and provide brain relaxation to facilitate surgical exploration. Dexamethasone 10 mg was also given to decrease cerebral edema by decreasing the number and activity of inflammatory cells within the brain (Omoigui, 2004). An antibiotic was also administered prior to skin incision.

Maintenance of anesthesia was accomplished with a constant percent of the volatile agent Desflurane at 3 percent and an infusion of propofol at 100 to 160 mcg/kg/min titrated to surgical stimulation. A mild hypothermia was allowed to a core temperature of 35 degrees centigrade realizing that hypothermia provides brain protection (Leslie, 2002). Cerebral blood flow is lowered 5-7% per 1° C decrease in core temperature. Mild hypothermia lowers both cerebral blood flow and cerebral metabolic rate (Morgan, 2006). The vasoactive medications ephedrine, neosynephrine as well as nitroglycerine were at hand if the hemodynamic profile of the patient during the operative time required these interventions.

Moderate hyperventilation was employed during the surgical procedure to end-tidal CO<sub>2</sub> values of 30 to 35 which were confirmed with arterial blood gas samples. This technique will lower intracranial pressure. Additionally lowering the CO<sub>2</sub> values to below 30 have not been shown to be of benefit and may cause focal ischemia (Morgan, 2006). Intravenous fluid replacement was limited to 2 liters of normal saline during the 4 hour operation. Dextrose is relatively contraindicated in patients with neurologic ischemic injury. This patient population tends to have high glucose levels due to steroid use at baseline. Additional glucose load has been implicated in increased ischemic brain injury (Morgan, 2006). Additional surgical tools for brain assessment included

somatosensory evoked potential (SSEP) and motor evoked potentials (MEP). These modalities required that no muscle relaxation be employed. Intraoperative wake up for neurological assessment has been replaced in many settings with the use of SSEP and MEP monitoring which was used in this case.

Emergence was uneventful with a very quick wake up after the volatile agent was discontinued and the propofol infusion was stopped. Several minutes prior to tracheal extubation, lidocaine 100 mg was given intravenously to assist with suppressing laryngeal reflexes before extubation. The patient opened his eyes on command and the endotracheal tube was removed. There was no evidence of pain or discomfort, respiratory rate was 14 with ventilatory tidal volumes of 6 to 8 ml/kg. The patient asked if the surgery was over.

### **Discussion:**

An opioid based anesthetic for craniotomy has been well documented in the literature. A search was done using Ovid using the terms 'opioid analgesics' and 'craniotomy'. A total of 16,785 journal articles appeared as a result of the search. Further narrowing this pool of articles to non-opioid based anesthetic was difficult. Apparently there have been no studies evaluating a non-opioid technique using propofol and low dose volatile agent for craniotomy.

The competing demands of hemodynamic stability during intense surgical stimulation, brain protection and a rapid postoperative wake up and neurological evaluation all seem to require different anesthetic techniques. Remifentanyl infusion with a propofol infusion has been used successfully to accomplish these anesthetic demands of stable intraoperative conditions and quick wake up for neurologic tests (Coles, 2000). Comparisons between remifentanyl and fentanyl for craniotomy with space occupying lesions favor the use of remifentanyl for its rapid metabolism through plasma and tissue nonspecific esterase's making it an acceptable choice for patients with abnormal plasma cholinesterase (Balakrishnan, 2000). These studies reinforce the belief that an opioid based combined anesthetic is the best choice for craniotomy.

When considering the relative value of each component of the anesthetic for craniotomy several factors immerge that seem important. Hemodynamic stability, brain protection and quick wake up are three issues that are most important in this patient

population. An opioid has traditionally been added to provide hemodynamic stability by blunting the sympathetic outflow from surgical stimulation. Increases in blood pressure and heart rate have classically been attributed to nociceptor stimulation. During a craniotomy the greatest amount of pain receptors are in the skin. The dura on the other hand is abundantly supplied with sympathetic fibers which when stimulated cause a catecholamine outflow increasing heart rate and blood pressure. This is not a pain response. Blunting the sympathetic outflow with beta-blockers may be an alternative to an opioid in this situation to maintain hemodynamic stability. The question of whether sympathetic blockade produces analgesia has been disputed. However it has been demonstrated that somatic nerve fibers do travel with sympathetic nerves and may be affected by neural blockade of 'sympathetic trunks' which may produce an analgesic effect (Ellis, 2004).

Brain protection during craniotomy is of paramount importance. Evaluating each agent used in the armamentarium for producing a good outcome during open skull surgeries necessitates this close scrutiny. Opioids unfortunately do not produce a decrease in cerebral metabolic rate (CMR) nor do they decrease cerebral blood flow (CBF). These two factors have traditionally been associated with 'brain protection' (Morgan, 2006). Propofol is a hypnotic and reduces CBF as well as CMR in a similar fashion to the barbiturates both of which are considered brain protective medications. Additionally, propofol has anticonvulsant activity (Morgan, 2006). The draw back to propofol use is two fold; first, it does not have analgesic properties; and second, it may lower blood pressure excessively in those patients with hemodynamic instability. At lower rates of infusion, propofol 100 to 150 mcg/kg/min, the hemodynamic effects of propofol do not seem to be excessive. These qualities make propofol a better agent as part of a strategy for maintenance of anesthesia in the craniotomy patient population than an opioid based anesthetic. A propofol infusion combined with low dose volatile agent provides brain protection and ideal operating conditions. Beta-blocker therapy intraoperatively may be used to blunt sympathetic responses.

Wake up is especially important in this patient group. Quick neurologic assessment in the operating room at the end of the surgical case will indicate if there is an improvement in neurologic function or an obvious post-surgical deficit that may be

amenable to re-exploration. The use of an opioid technique may lead to a continued somnolence and bring the etiology of the neurologic status into question. In this case no opioid had been given which precluded an opioid induced deficit as a possibility. The greatest benefit for not using an opioid technique in this craniotomy population is the rapidity of the wake up and the clear mentation immediately after emergence from general anesthesia. Obviously it is not to be advocated that pain be ignored. In this case there was no evidence of postoperative pain which brings into question the routine use of opioids for craniotomy without demonstratable need.

**Conclusion:**

This case of a gentleman suffering from ischemic stroke and surgical bypass of the right superior temporal artery to the middle cerebral artery demonstrates that a non-opioid anesthetic technique may be used with success in craniotomy. Further study of a non-opioid technique in craniotomy seems warranted. In the course of preparing for this case, 2 dozen non-opioid craniotomy surgical procedures were performed with this anesthetic technique. It would be productive to study this further with a randomized trial comparing an opioid based technique with the non-opioid group. Outcome data including postoperative pain scores, rapidity of wake up and neurologic function immediately following craniotomy seem logical measurement points. It was my great pleasure to participate in the anesthetic for this gentleman. My special thanks to Dr. Robert Naruse for his assistance and mentorship during this anesthetic.

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