# Anesthetic Management For Surgical Excision Of A Cerebellar Infarct In A Patient With Acute Myocardial Infarction

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**Citation**

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**Abstract**

A forty nine year old male who had sustained acute myocardial infarction two days earlier, experienced respiratory arrest and complete heart block and was admitted with a diagnosis of left cerebellar infarct and obstructive hydrocephalus. On examination he was responding to deep painful stimuli with right hemiparesis. ECG indicated acute inferolateral myocardial infarction. Echocardiogram showed an ejection fraction of 63%. Arterial blood gases revealed severe respiratory alkalosis and arterial desaturation. The lungs were ventilated and inotropic support was started to ensure a stable hemodynamic status. Evacuation of the cerebellar infarct was planned as an emergency procedure. Anesthesia was maintained using $N_2O$ — $O_2$ — narcotic — neuromuscular blocking drugs sequence. Continuous monitoring of ECG, ST segment, invasive arterial blood pressure, CVP, $ETCO_2$, $SaO_2$ and temperature were instituted. The intraoperative period was uneventful. Elective postoperative ventilation was continued for three days. He was then transferred to the step down intermediate care unit for definitive therapy.

## INTRODUCTION

The average incidence of myocardial infarction (MI) is 1 – 2% in unselected patients over forty years of age undergoing major non-cardiac surgery. Pre-existing coronary artery disease and in particular evidence of a previous MI result in a higher risk. Mortality in patients who suffer a perioperative MI may be as high as 60%. Preoperative evidence of uncontrolled hypertension, cardiac failure, arrhythmias or aortic stenosis also increases the risk of perioperative MI. In patients with ischaemic heart disease, postoperative MI is more likely if there is evidence of ischaemic changes on ECG during surgery. Such changes are associated more commonly with episodes of intraoperative hypotension, hypertension or tachycardia. The drugs used and the manners in which the anesthetist employs them influence the incidence of both intraoperative and post operative MI [1].

Five percent of patients over thirty-five years of age have asymptomatic heart disease. Patients who have had MI are at greater risk for reinfarction in the perioperative period than others [2]. A number of factors, which may be detected during preoperative assessment, are known to increase the likelihood of perioperative myocardial infarction. The most important of these is the time interval between surgery and a previous myocardial infarction. The rate of perioperative myocardial infarction decreases to 15% at three to six months and to 5% there after [1]. With intense perioperative monitoring, much lower rates of re-infarction can be achieved in three to six months and less than three months. Mortality from postoperative infarction is 40 – 60%[1]. The incidence of perioperative myocardial infarction is also related to intraoperative and postoperative factors. The magnitude of surgery is an important determinant and in patients with history of myocardial infarction the risk associated with major vascular surgery is considerably higher when surgery is performed out side the thorax and abdomen [1].

Given the increased risk of morbidity in patients with recent myocardial infarction, traditional anesthetic practice dictates that elective surgery be postponed until a six months interval has passed. Vascular and neurosurgery are often not elective. Urgent surgery is often required for patients experiencing transient ischaemic attacks or in patients with cerebrovascular accident where evacuation of hematoma is indicated as an emergency procedure as in this case.

## CASE REPORT

A forty nine year old male was referred to our critical care unit with a diagnosis of left cerebellar infarct and obstructive

hydrocephalus. He had sustained acute myocardial infarction with respiratory arrest and complete heart block two days earlier. He was thrombolysed for the same and a temporary pacemaker was inserted for heart block. The trachea was intubated and started on ventilatory support and inotropes. Subsequently he was weaned off the ventilator, extubated and he developed his own cardiac rhythm. However, the next day he became unconscious and developed hemiparesis on the right side. CT scan of the brain revealed a massive infarct on the left side of the cerebellum obstructing the fourth ventricle causing hydrocephalus. On examination he was unconscious and responding to deep painful stimuli with hemiparesis on the right side. He was tachypnoeic with a respiratory rate of forty-six per minute and oxygen saturation ($SaO_2$) 80-85%. However his cardiac parameters were stable with heart rate of eighty per minute, not pace maker dependent, and blood pressure 150/110 mm Hg. Auscultation of the chest revealed bilateral extensive coarse crepitations. Electrocardiogram (ECG) showed acute inferolateral myocardial infarction. The echocardiogram revealed an ejection fraction of 63%. Arterial blood gases revealed severe respiratory alkalosis. Since he was unconscious and airway compromised with severe respiratory alkalosis, he was immediately intubated and started on ventilatory support. The left subclavian vein was cannulated and nitroglycerine infusion started. Central venous pressure (CVP) was 10 cm of $H_2O$. A propofol infusion was started at 70mg per hour and fentanyl at 25 µg per hour.

He was posted for craniotomy and evacuation of cerebellar infarct as an emergency procedure and was taken-up for surgery under ASA IV. Continuous monitoring of ECG, ST segment, invasive arterial blood pressure, CVP, end-tidal carbon dioxide ($ETCO_2$), $SaO_2$ and temperature were instituted. A pulse generator was connected to the pace maker and kept ready just incase he developed bradycardia and cardiovascular collapse. Isoprenaline and dopamine were loaded and kept ready for emergency infusion. Anesthesia was maintained using $N_2O$ – $O_2$ – fentanyl – propofol and vecuronium. After excision of the infarct brain was pulsatile, the cerebrospinal fluid (CSF) drained from the cisterna magna and tonsillar herniation was alleviated. Normocarbia and normothermia were maintained throughout the intraoperative period. The intraoperative period was uneventful. There was no hypotension, hypoxaemia or change in heart rate. Postoperatively he was shifted to intensive care unit (ICU) for intensive therapy where he was electively ventilated for three days following which the ventilation was changed to weaning mode and subsequently extubated. He was conscious and vital signs were stable. He was then transferred to the step down intermediate care unit for definitive therapy and management.

## DISCUSSION

The main indication for emergency intracranial surgery is bleeding as a result of trauma or cerebrovascular accident (CVA), which may be exacerbated in patients on anticoagulants especially aspirin. Intracranial hematoma may arise either epidurally, subdurally or intracerebrally and may either accumulate rapidly or slowly. Many patients who present for anesthesia are unconscious or semiconscious and irritable as a result of raised intracranial pressure and cerebral compression. Virtually all patients with head injury or CVA have had an emergency CT scan as part of their initial management. Many have undergone tracheal intubations and ventilation of the lung for this procedure and are subsequently kept anaesthetized and taken straight to the operating theatre for surgery to decompress the brain. With an expanding intracranial hemotoma, speed is of essence if cerebral damage is to be minimized or avoided. While adequate anesthetic time may be taken to ensure safety, excessive delay may seriously affect the overall result of decompression and make the difference between a good and merely a moderate recovery [2].

The intraoperative techniques utilized should permit modulation of sympathetic nervous system responses and prompt control of hemodynamic variables [3]. The basic challenge during induction and maintenance of anesthesia for patients with ischaemic heart disease is to prevent further myocardial ischaemia. This goal is logically achieved by maintaining the balance between myocardial oxygen delivery and consumption and is more important than the specific anesthetic techniques or drugs selected to produce anesthesia and neuromuscular blockade [4,5,6]. Intraoperative events associated with persistent tachycardia, systolic hypertension, sympathetic nerve system stimulation, arterial hypoxaemia or diastolic hypotension can adversely affect this delicate balance. It is important to avoid persistent and excessive changes in heart rate and systemic blood pressure. A common recommendation is to maintain the patients heart rate within 20% of normal awake value. Iatrogenic hyperventilation, which greatly reduces $PaCO_2$, is avoided, as hypocapnia may evoke coronary vasoconstriction. Normocapnia should be maintained because hypereapnia

may evoke arrhythmias while hypocapnia causes peripheral and coronary vasoconstriction and shifts the oxygen dissociation curve to the left [7].

The anesthetic techniques for emergency intracranial surgery can be performed by a short acting intravenous opioid, neuromuscular blockade with vecuronium or rocuronium and intermittent positive pressure ventilation (IPPV) to a $PaCO_2$ of 4 kPa. Patients who are likely to remain intubated postoperatively, an anesthetic regimen based primarily on a narcotic such as fentanyl and a neuromuscular blocking agent usually serves well [8].

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