Anaesthesia and the Prone Position: A Case Report and Review of Literature

***Edubio MN[1], Eguma SA[2]**

ABSTRACT

The patient was a 14year old boy with a diagnosis of posterior cranial fossa tumour scheduled for suboccipital craniectomy and supratentorial craniotomy. Under general anaesthesia, with relaxant technique and mechanical ventilation, the tumour was excised in a prone head-up position. Intraoperative, the patient was closely monitored with a pulse oximeter, non-invasive blood pressure monitor, a 5-lead ECG, an end-tidal carbon dioxide monitor, and a urinary catheter. The patient was moderately hyperventilated to an end tidal carbon dioxide ($ETCO_2$) of between 3.7-4.2kPa throughout surgery and transfused 2units of whole blood. While no direct complication of the prone position was noted throughout the duration of this surgery, intraoperative complications included fluctuations in blood pressure and episodes of tachyarrhythmia that needed no direct interventions. Neuroanaesthesia in the prone position will continuously remain a challenge to the anaesthetist, not only because of this abnormal position, but also the physiological changes occasioned by this position.

Keywords: general anaesthesia, posterior cranial fossa tumour, prone position.

INTRODUCTION

The posterior fossa is a compact and rigid compartment with poor compliance. Small additional volume (e.g. tumour) within the space can result in significant elevation of the compartmental pressure resulting in life-threatening brainstem compression[1]. This fossa contains many important structures including the brainstem, cerebellum and lower cranial nerves. The cerebrospinal fluid (CSF) pathway is very narrow through the cerebral aqueduct and any obstruction can cause hydrocephalus, which can result in a significant increase in intracranial pressure (ICP)[1]. Tumours are the commonest pathology affecting the posterior fossa. They account for more than 60% of all brain tumours in children[2]. The prone position offers good access to structures in the midline, but care should be taken to avoid abdominal compression to minimize surgical bleeding. The prone position is associated with significant cardiovascular changes and problems with airway maintenance.This case report seeks to highlight the challenges of the prone position to the anaesthetist and principles of neuroanaesthesia.

Department of Anaesthesia, University of Uyo/University of Uyo Teaching Hospital, Uyo
Department of Anaesthesia, University of Calabar/University of Calabar Teaching Hospital, Calabar

*Corresponding author: markedubio@gmail.com

CASE REPORT

The patient, a 14-year old junior secondary school male student, presented with a five month history of increased size of the head and an inability to walk. He was well until about ten months prior to presentation when he was noticed to be sleeping excessively, walking with a staggering gait and complaining of blurred vision. About five months after the onset of symptoms, progressive enlargement of the head was noticed. There was associated throbbing headache and occasional projectile vomiting. There was no history of fever, neck stiffness or convulsion. Patient was initially incontinent of both urine and faeces for about 3months, but regained some control later. His appetite was poor with associated weight loss. There was no history of trauma to the head.

Examination revealed an afebrile but drowsy and aphasic patient, who was not in obvious respiratory distress, and was not pale. He was moderately dehydrated and wasted, with generalised café-au-lait spots. Occipito-frontal circumference (OFC) was 66cm with closed anterior frontanelle. Visual acuity of 2/6 bilaterally and fundoscopy revealed papilloedema. Other cranial nerves were grossly intact. Muscle power was 3/5 in the upper limbs, and 2/5 in both lower limbs. There was hypotonia and hyperreflexia in all four limbs with loss of muscle bulk. Sensory sensation was preserved. He weighed 32kg.

The pulse rate was 80 beats/minute, full and regular, blood pressure was 110/80mmHg, and heart sounds were S1 and S2 only. The respiratory rate was 20cycles/minute, breath sounds were vesicular and both lung fields were clinically clear. A Full Blood Count and Serum Urea and Electrolytes were within normal values. Skull x-ray showed an enlarged cranium with thin skull bones.

Magnetic resonance imaging showed triventricular dilatation with normal 4th ventricle. There was a large cystic lesion in the posterior fossa; infratentorially compressing the midbrain and pons anteriorly. Generalised cerebral oedema was clearly evident. These features were in keeping with haemangioblastoma of the posterior fossa. A diagnosis of hydrocephalus with quadriparaesis secondary to haemangioblastoma of the posterior cranial fossa was made. He was scheduled for ventriculo-peritoneal (V-P) shunt and posterior fossa craniectomy.

The patient had no previous exposure to anaesthesia, no allergy to any drug, and was not a sickle cell disease patient, asthmatic or diabetic. He was on tablet acetazolamide 0.25mg twice daily. Airway assessment was Mallampati II with an American Society of Anesthesiologists (ASA) physical health status score of ASA III. An informed consent was obtained from the father and he was placed on nil per os (NPO) after dinner. He was told to take his drug by 6:00am on the day of surgery with a tablespoonful of water. Three pints of whole blood were ordered grouped and cross-matched. Intravenous dexamethasone 4mg 6hourly was prescribed. Intravenous fluid was limited to 30ml/kg/day.

In the theatre, the Drager anaesthetic machine was checked and drugs drawn and labelled. Patient was placed in the supine position on the operating table, and recorded vital signs were within normal limits. Non-invasive blood pressure was set to measure at 3-minute intervals. An intravenous line was set up on the left forearm with 18G cannulae and maintained with 500ml of normal saline. Oxygen saturation was 96% on room air and axillary temperature was 37^{0}C.

Patient was catheterized, preoxygenated and premedicated with atropine 0.4mg intravenously. This was followed by intravenous sodium thiopentone 200mg and application of cricoid pressure by an assistant as soon as consciousness was lost. Laryngoscopy and intubation, with a size 6.0mm low-pressure high-volume cuffed endotracheal tube (ETT), was facilitated with suxamethonium 50mg intravenously. The cuff was inflated and the ETT connected to the ventilator through a mainstream capnometer. The position of the ETT was verified by auscultation and by the capnograph before it was secured firmly with adhesive plaster.

The ventilatory mode was intermittent positive pressure ventilation (IPPV), at a rate of 14cycles/min, oxygen flow rate 5L/min and tidal volume at 250ml. The isoflurane vaporizer was turned on and adjusted based on vital signs. Atracurium 20mg and morphine 5mg were administered intravenously.

With assistance, patient was placed in the prone (ventral decubitus) position, with elevation of the head, after disconnecting the anaesthetic circuit and monitors for about 10 seconds (Fig 1). The anaesthetic circuit and monitors were then reconnected. The position and centrality of the endotracheal tube was rechecked. Pillows were placed under the chest and pelvis, with padding for the knees, ankles, elbows and wrists. The abdomen was left free. The elevated head of the patient was held between the pins of a Mayfield 3-point fixator. A 5-lead ECG was attached ventrally on the patient. Intravenous ceftriazone 750mg was given as antibiotic prophylaxis.

The patient was cleaned, draped and the scalp infiltrated with 10ml of 1% lidocaine with adrenaline 1:200,000. A tubed occipital ventriculostomy was performed to enable external ventricular drainage (EVD). The patient was moderately hyperventilated to an end tidal carbon dioxide ($ETCO_2$) of between 3.7-4.2kPa before the establishment of EVD. Through a right hockey stick incision, suboccipital craniectomy and supratentorial craniotomy were done, and the cystic tumour exposed and excised.

The blood pressure fluctuated between 130/100-90/60mmHg; mean arterial pressure was between 70-110mmHg. There were episodes of tachyarrhythmia for less than 60 seconds on the ECG tracing, with return of sinus rhythm without the use of an antiarrhythmic agent. The heart rate ranged between 70-160beats/min. In the course of the 6-hour operation, the patient was transfused with two pints of whole blood, and had 750ml of 0.9% saline. He lost approximately 950ml of blood and had made 580ml of clear urine. At the end of surgery, the patient was repositioned supine and residual muscle relaxant was reversed. The pharynx was suctioned and patient was extubated awake.

He was transferred to the ICU with oxygen via a facemask, from where he was moved to the neurosurgical ward after 72hours. Postoperatively, patient was placed on intravenous ceftriaxone 750mg 12hourly and gentamicin 40mg 8hurly. Intravenous fluid was changed to 5% dextrose saline 500ml 12hourly. Intramuscular morphine was given at 2.5mg 4hourly.

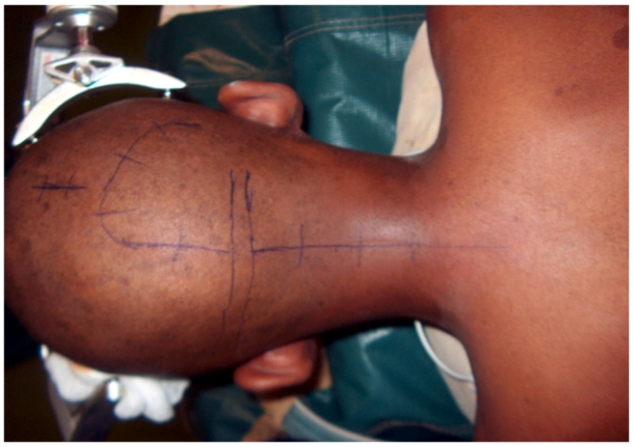

Fig 1: Patient in the Prone Head-up Position with a May field 3-point fixator

DISCUSSION

Posterior fossa tumours are more likely to be primary brain tumours, and are more common in children than in adult [3]. They also tend to be more common in females than in males (M: F ratio of 1:1.8)[3]. Surgery involving the posterior fossa can result in direct cranial nerve injury or in oedema that can compress cranial nerves. Bilateral vocal cord paralysis leading to post-obstructive pulmonary oedema has been described[4].

In the preanaesthetic evaluation of patients, assessment of the level of consciousness is important as it reveals the severity of the disease condition, and possible impairment of airway reflexes. The presence of bulbar palsy may lead to silent aspiration. Our patient was drowsy but conscious and was not in respiratory distress, he was able to swallow and chest findings were within normal limits.

The presence of raised intracranial pressure (ICP) in this patient was evident clinically by headache, vomiting, papilloedema and hydrocephalus. Magnetic resonance imaging (MRI)of the cranium also revealed generalised cerebral oedema. While the normal ICP is 5-12mmHg, changes in ICP reflect changes in the volume of intracranial contents held within the confines of the cranium. Compensatory mechanisms initially reduce the effect of an intracranial space-occupying lesion on ICP. These mechanisms involve displacement of cerebrospinal fluid (CSF) into the spinal subarachnoid space, increased absorption of CSF, and a reduction in intracranial blood volume. Eventually these mechanisms become overwhelmed and further small increases in intracranial volume result in a steep rise in intracranial pressure[5].

When ICP exceeds 30mmHg, cerebral blood flow (CBF) progressively decreases and a vicious cycle is established: ischaemia causes brain oedema, which in turn increase ICP, resulting in more ischaemia. If left unchecked, this cycle continues until the patient dies of progressive neurological damage or catastrophic herniation[5].

Vomiting in the patient could have led to depletion of intravascular fluid volume with the possibility of hypotension during induction. However, correction of fluid deficit must be done with caution to avoid increasing ICP. Patient was commenced on an oral diuretic tablet, acetazolamide, 48hours before surgery. Vomiting, with the use of a diuretic, can readily lead to electrolyte imbalance. Electrolyte check was however within normal limits. The only obvious neurological involvements were the reduced visual acuity and quadriparaesis. These were considered important as patient was to be placed in the prone position.

Our patient was commenced on intravenous dexamethasone 24 hours before surgery to reduce ICP. Corticosteroids reduce oedema surrounding tumours and abscess but have no role in head injury. They however take several hours to work[5]. The choice of intravenous 0.9% saline is important to maintain intravascular volume and cerebral perfusion pressure (CPP). Hypotonic solutions such as 5% dextrose should be avoided as fluid flux across the blood-brain barrier is determined by plasma osmolality and not oncotic pressure. Maintenance of a high normal osmolality is therefore essential.

Avoiding factors that can lead to an increase in cerebral blood flow (CBF) will prevent a rise in ICP. These factors include hypercarbia, hypoxia, hypertension and hypothermia. Intermittent positive pressure ventilation was used in this patient to control

$PaCO_2$, to between 38-42mmHg, and ensure good oxygenation. Provision of adequate analgesia, using intravenous morphine, was ensured. Opioid analgesics have little effects on CBF and ICP if an increase in $PaCO_2$ is avoided[5]. Mannitol, in doses of 0.25-1.0g/kg given over 15min, is especially effective in rapidly decreasing ICP. This can be given alone or with the loop diuretic furosemide 0.25-1.0mg/kg.

Laryngoscopy and intubation, if improperly performed, can severely compromise intracranial dynamics and increase morbidity. Every patient with raised ICP has an increased risk of aspiration and almost always requires positive pressure respiration; both of which are limitations of the LMA[6].

The patient was given lidocaine 30mg intravenously before laryngoscopy. Alternatively, the short acting selective beta-blocker esmolol (0.25-0.5mg/kg i.v.) or an additional small dose of thiopentone or propofol can be administered to maintained mean arterial pressure in the desired range.

With the exception of ketamine, all intravenous anaesthetic induction agents decrease cerebral metabolism, CBF and ICP. Ketamine increases ICP and should be avoided. Autoregulation of the cerebral circulation is well maintained during propofol/thiopentone anaesthesia. Suxamethonium, which was used to facilitate intubation in this patient, causes a marginal and transient rise in ICP and increases venous pressure. If there are contraindications to the use of suxamethonium, rocuronium (0.6-1.2mg/kg) provides excellent intubating conditions in 50-60sec[7].

Vecuronium is more cardiostable than pancuronium and atracurium, and would have been preferred but was not available. Pancuronium increases heart rate and blood pressure due to vagolysis and sympathetic stimulation. Complete neuromuscular blockade should be verified with a nerve stimulator before laryngoscopy to prevent coughing and associated increase in ICP.

Anaesthesia in this patient was maintained with isoflurane. Volatile anaesthetic agents uncouple metabolism and blood flow; reducing cerebral metabolism while increasing CBF and ICP. They abolish autoregulation in sufficient doses. Intracranial pressure is not affected by concentrations of less than one minimum alveolar concentration (MAC) of isoflurane, sevoflurane and desflurane[5]. Enflurane may cause seizures, particularly with hypocapnia, and has no place in neuroanaesthesia. Isoflurane increases ICP the least and halothane the most.

The application of the pins of a Mayfield 3-point fixator can elicit severe haemodynamic responses. Costello *et al.*[8] found oral clonidine premedication to be effective in reducing the mean arterial blood pressure increase resulting from this application. A combination of adrenaline and lidocaine was used to infiltrate the scalp prior to skin incision. This attenuates the hypertension adrenaline alone would have caused, but can result in a biphasic hypotensive response[9]. Adrenaline also prolongs the action of lidocaine because of its vasoconstrictive effect.

In the prone position, the chest and pelvis should be adequately supported, as was done in this case, to avoid abdominal compression, which will result in increased cerebral venous pressure, splitting of the diaphragm and attendant respiratory compromise. Also, the circulatory dynamics vary according to the postural modification used. In the case presented, there was a slight head-up rotation of the chassis of the table. In this position, pooling of venous blood in distensible dependant vessels is likely to occur. The mean vascular pressures are decreased according to the distance above the heart. Air entrainment in open veins is possible and conjunctival oedema is less evident or absent.
Backofen *et al*[16]. found that even the carefully established and supported prone position caused a significant fall in stroke volume and cardiac index, despite the development of increased vascular resistance in both systemic and pulmonary circulation. However, no significant changes were detected in mean arterial pressure, right atrial pressure, or pulmonary artery occlusion pressure.

Proper positioning can retain more nearly normal pulmonary compliance by minimizing the cephalad shift of the diaphragm caused by compressed abdominal viscera. If a patient is positioned so that the abdomen hangs free, the loss of functional residual capacity is less in the prone position than in either the supine or the lateral positions.

The prone position, and any position that requires extreme neck flexion or twisting, can be associated with endotracheal tube obstruction and venous congestion. Biting of the endotracheal

tube by a patient who is not sufficiently anaesthetized may also cause obstruction of the endotracheal tube[11]. A short bite block that does not extend to the back of the tongue may be inserted and secured to prevent this. If there is any doubt as to whether the ETT is obstructed, a suction catheter can be passed into the endotracheal tube and any difficult or impossible passage will confirm partial or complete obstruction[12]. None of these precautions were required in the management of the index case.

A reinforced endotracheal tube or a standard oral airway may reduce the likelihood of endotracheal tube obstruction related to head position and can be used electively if the probability of obstruction is high[12]. Even with all these measures, care should still be taken to avoid flexion of the neck that can potentially cause spinal cord injuries, or contribute to swelling of the head, tongue and neck.

Inadvertent extubation may occur secondary to positioning and repositioning, surgical manipulation, head movement, coughing and loosening of the adhesive tape. The management of inadvertent extubation depends on the position of the patient and accessibility of the airway. Mask ventilation should always be tried as the first step in re-establishing the airway.

Even in the prone position, direct laryngoscopy is a possibility. If not surgically contraindicated, turning the patient's head to the right side facilitates reintubation if required, since it allows for laryngoscopy from the right side of the mouth. In an emergency, the patient may have to be positioned supine and the head pins or stereotactic head frame removed to facilitate intubation.

If direct laryngoscopy is impossible, an LMA may be used to re-establish oxygenation and ventilation. Fibreoptic endotracheal intubation may be facilitated through the LMA. If an airway cannot be secured quickly with the LMA, a Combitube may provide an alternative option[13]. Tracheal jet ventilation with a 14 or 16-gauge needle as described by Benumof *et al*.[14] can provide effective oxygenation in an emergency situation but would appear to be severely limited in the prone position.

Taylor *et al.*[15] reported a successful CPR, with defibrillation, in the prone position after cardiac arrest. In their opinion, prone resuscitation should always be considered as a first response in the event of cardiac arrest during prone surgery.

Although no complications, as a result of the prone position, was seen in this patient, other complications of the prone position to which there should be constant attention are retinal ischaemia and blindness from orbital compression. This problem may be compounded by low arterial pressure, low haematocrit level, and poor cerebral venous drainage[16].

Air embolization is a potential lethal complication of the prone position. Venous air embolism occurs in the presence of a pressure differential at two different sites of the venous system, which in turn causes a negative pressure or sub-atmospheric gradient between the right atrium and the cranial venous sinuses. When the venous system of the central nervous system is exposed to environmental pressure and there is a difference of at least 5 cm of H_2O between the two sites, there will be air inflow. In humans, the lethal dose of air embolism is 3-4 mL/kg[2]. The volume of air in the venous system that may trigger clinical manifestations is around 100 mL for the adult[17].

Our patient was monitored using a non-invasive blood pressure monitor, a pulse oximeter, an axillary temperature probe, an $ETCO_2$ monitor, a urinary catheter, and a 5-lead ECG. Rapid blood pressure changes occurring during induction, hyperventilation, intubation, positioning, surgical manipulation, and emergence necessitate continuous blood (direct intra-arterial) pressure monitoring. This facility was not immediately available. Electrophysiological monitor can be used to detect nerve injury depending upon the neural pathway at risk during the procedure. Spontaneous of evoked electromyographic activity, somatosensory evoked potentials, or brainstem auditory evoked potentials are frequently employed.

Arterial blood gas measurements are necessary to closely regulate $PaCO_2$, as measurement of $ETCO_2$ alone cannot be relied upon for precise regulation of ventilation. The arterial to end-tidal CO_2 gradient should be determined. The $ETCO_2$ does not provide a stable reflection of $PaCO_2$ in many patients undergoing craniotomies[18] since $ETCO_2$ is lower than $PaCO_2$[5].

The timing and technique of extubation of

extubation in the neurological patient are just as critical as those of intubation. Ideally, the ETT should be removed before the patient is aware of its uncomfortable presence, as this can trigger physiological responses detrimental to cerebral perfusion[12]. Though some anaesthetists favour "deep extubation" over "awake extubation" to avoid such complications, studies comparing the incidence of coughing and bucking after deep and awake extubation do not support the belief that deep extubation decreases the incidence of such events[19,20]. Our patient was extubated awake to properly assess his reflexes. As a result of the many surgical and non-surgical issues that make evaluation of the anaesthetized patient's readiness for extubation difficult, extubating awake seems appropriate for most surgical patients[12].

The cardiovascular and intracranial effects of extubation in the awake patient can be effectively blunted with the intravenous administration of 1.5-2.0mg/kg of plain lidocaine or a small dose (2-5mg) of esmolol one to two minutes before the planned extubation.

CONCLUSION

Neuroanaesthesia in the prone position will continuously remain a challenge to the anaesthetist, not only because of this abnormal position, but also the physiological changes occasioned by this position. A detailed knowledge of the issues involved and a care conduct of anaesthesia in this position is a must for a successful outcome.

REFERENCES

1) Jagannathan S, Krowidi H. Anaesthetic consideration for posterior fossa surgery. Continuing Education in Anaesthesia, Critical Care & Pain 2003;1-5
2) Krane EJ, Phillip BM, Yeh KK, Domino KB. Anaesthesia for paediatric neurosurgery. In: Smith RM, Mototyama EK, Davies PJ, eds. *Smith's Anaesthesia for Infants and Children, 7th Edn.* Philadelphia: Mosby, 2006;651-84
3) Wanyoike PK. Posterior cranial fossa tumours in children at Kenyatta National Hospital Nairobi. East African Medical Journal 2004;258-260
4) Dohi S, Okudo N, KondoY. Pulmonary oedema after airway obstruction due to bilateral vocal cord paralysis. Can J Anaesth1991;38:492-5
5) McQuillan PM. Neurosurgery. In: Allman KG, Wilson IH, eds. *Oxford American Handbook of Anesthesia, 1st Ed.* Oxford University Press 2008;402-439
6) Benumof JL. Laryngeal mask airway: Indications and Contraindications (Editorial). Anesthesiology 1992;77:843-6
7) Magorian T, Flannery KB, Miller RD. Comparison of rocuronium, succinylcholine and Vecuronium for rapid-sequence induction of anaesthesia in adult patients. Anesthesiology 1993;79:913-8
8) Costello TG, Cormack JR. clonidine Premedication Decreases Hemodynamic Response to Pin Head-Holder Application During Craniotomy. AnesthAnalg 1998;86:1001-4
9) Murthy HS, Raw GS, Umamaheswara J. Cardiovascular Responses to Scalp Infiltration with Different Concentrations of Epinephrine with or without Lidocaine during Craniotomy. AnesthAnalg 2001;92:1516-19
10) Backofen JE, Schauble JR. Haemodynamic changes with prone positioning during general anaesthesia. AnesthAnalg 1985; 64:194
11) Korn S, Schubert A, Barnett G. Endotracheal tube obstruction during stereostactic craniotomy. NeurosurgAnesth 1993;5:272-5
12) Spiekmann BF, Stone DJ, Bogdonoff DL, Yemen TA. Airway management in neuroanaesthesia. Can J Anaesth 1996;43:820-34
13) R. Postoperative ischaemic optic neuropathy. AnesthAnalg 1995; 80:1081Green KS, Berger TH. Proper use of the combitube (Letter). Anesthesiology 1994;81: 513
14) Benumof JL, Schuller MS. The importance of transtracheal jet ventilation in the management of the difficult airway. Anesthesiology 1998;71:769-78
15) Taylor JCL, Buchanan CCR, Rumball MT. Cardiac Arrest During Craniotomy in Prone Position. Trends in Anaesthesia and Critical Care 2013;3:224-6
16) William EL, Hart WM, Tempelhoff R.

Postoperative ischaemic optic neuropathy. AnesthAnalg 1995;80:1018
17) Pahnon SC, Toung T. Special Article SP Venous Air Embolism: A Review. 1997; 8180:97
18) Russell G, Graybeal JM. The Arterial to End-Tidal Carbon Dioxide Difference in Neurosurgical Patient During Craniotomy. AnesthAnalg 1995;81:806-10
19) Pounder DR, Blackstock D, Steward DJ. Tracheal extubation in children: halothane versus isoflurene, anesthetized versus awake. Anesthesiology 1991;74: 653-5
20) Patel RI, Hannallah RS, Norden J, Casey WF, Verghese ST. Emergence airway complications in children: a comparison of tracheal extubation in awake and deeply anesthetized patients. AnesthAnalg 1991;73:266-70