Document heading doi: 10.21276/apjhs.2018.5.4.27 **Case report**

Anaesthesia for anterior chamber wash-out andcorneo-scleral repair in a patient with hypertensive heart disease: A case report

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Abstract

AO, a 48-year-old police officer with poorly controlled hypertension and previous myocardial infarction was referred from a peripheral hospital to University of Port Harcourt Teaching Hospital (UPTH), Port Harcourt, Nigeria on account of gunshot injury to the right eye. His blood pressure was 180/120 mmHg. The electrocardiogram (ECG) showed evidence of previous inferior myocardial infarction (MI) and 1st degree right bundle branch block. Emergency anterior chamber wash out and repair of corneo-scleral laceration was deferred for twenty-four hours to allow successful control of hypertension. The surgical procedure was finally performed under general anaesthesia with endotracheal intubation and muscle relaxation. Intraoperatively, patient developed few ventricular ectopic beats which resolved spontaneously. He had uneventful postoperative recovery and was discharged home on the 12th postoperative day. This report discusses anaesthetic challenges associated with hypertensive heart disease patients requiring emergency surgery in a resource limited setting.

Keywords: Anaesthesia, corneo-sclera, repair, hypertensive, heart-disease.

Introduction

Corneo-scleral laceration connotes a penetrative eye injury. Ocular trauma is a major cause of visual loss and blindness[1]. It is often associated with road traffic accidents, conflicts, corporal punishments and industrial accidents[2]. It is an ophthalmic emergency requiring early repair. There is the risk of extrusion of ocular contents, and measures should be taken to minimize increases in intra-ocular pressure (IOP).

Hypertensive heart disease is defined as heart disease secondary to hypertension. It ranges from left ventricular hypertrophy, myocardial infarction, unstable angina, and life-threatening arrhythmias, to frank heart failure[3]. Patients with cardiac conditions may present for cardiac surgery but more often, they present for non-cardiac surgery. The adrenergic response to surgery and the circulatory effects of anaesthetic agents, endotracheal intubation, positive pressure ventilation, blood loss, fluid shifts and alterations in body temperature impose additional burdens on an already compromised cardiovascular system[4,5]

Silent myocardial infarction is more frequent in hypertensives in the perioperative period than in normotensive patients[5,6]. A previous myocardial infarction (MI) predisposes the patient to re-infarction in the perioperative period. Hypertension, especially in association with tachycardia, can precipitate or exacerbate myocardial ischemia, ventricular dysfunction or both[7].The same principles used in treating these diseases preoperatively should be applied intraoperatively for a favourable outcome. A meticulous pre-operative assessment and peri-operative monitoring are essential to avoid morbidity and mortality[8].

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Case Presentation

AO, a 48-year-old police officer was referred from a peripheral hospital to UPTH on account of gunshot injury to the right eye by armed robbers while on duty. There was associated haemorrhage and loss of vision on the same eye. Heinitially presented to a nearby hospital from where he was referred to UPTH. He volunteered a history of intermittent severe palpitations, chest pain and dizziness which started six earlier prior this incident and he had planned to see a doctor for these symptoms. He had been diagnosed with hypertension 5 years prior to presentation but was not compliant with treatment nor follow-up due to financial reasons. He was not a known diabetic. He admits a previous history of chronicconsumption of alcohol which he stopped 5 months previously on self-counsel because of his health. He did not smoke cigarettes.

Physical examination revealed a fully conscious but anxious man with oozing of blood from the right eye. There were multiple injury points to the left arm, finger and left thigh. He was moderately pale, anicteric and afebrile. Cardiovascular examination revealed a thickened arterial wall with a pulse rate of 110 beats per minute that was regular and of moderate volume. The blood pressure was 180/120 mmHg. Heart sounds were normal and there was no murmur. The jugular venous pressure was not raised, apex beat was at the 6^{th} left intercostal space lateral to the mid clavicular line. Peripheral pulses were all palpable. Respiratory rate was 18 cycles per minute and air entry was equal bilaterally. Examination of the right eye revealed oedematous eye lids. The conjuctiva was markedly injected and was bleeding. There was also chemosis, total hyphaema, corneo-scleral laceration with uveal prolapse. The left eye was essentially normal. A diagnosis of open eye injury in a patient with hypertensive heart disease was made.

He was commenced on guttae atropine, chloramphenicol, oral ciprofloxacin, Cataflam and intramuscular tetanus toxoid and was scheduled for emergency corneo-scleral repair with anterior chamber wash out. Results of investigation carried out were as follows: Urinalysis- no abnormality detected, urea/ electrolytes- within normal limits, haemoglobin 90g/L (13-17g/L), fasting blood sugar 4.4mmol/L (normal). Electrocardiogram- Sinus rhythm, isoelectric S-T segment, Q-wave in inferior leads, Small R-waves in V_5 V_6, left axis deviation, right bundle branch block; findings were consistent with inferior myocardial infarction.Chest x-ray-Clear lung fields, cardiomegaly, unfolding of the aorta. Skull X-Ray- An oval opacity of metallic density overlying the right orbital cavity. There was no skull fracture.

He was reviewed by the physicians and was commenced on atenolol 100mg daily, nifedipine 20mg daily, Vasoprin 75mg daily, captopril 25mg 12hourly and moduretic 1tablet daily. There was a consensus by the anaesthetic and ophthalmology teams to reschedule the surgery to the next day to allow better control of the blood pressure.

Anaesthetic Management

At the pre-anaesthetic visit, the patient was reassured. The history and investigations were as already documented. Physical examination revealed a conscious but depressed, middle aged man in no obvious distress. He was moderately pale, anicteric and afebrile. The right eye was padded with sterile gauze and there were mild sero-sanginous staining of wound dressings over the left arm, finger and left thigh. He was able to climb a flight of stairs with minimal fatigue.

Cardiovascular system examination revealed a pulse rate of 98 beats per minute; blood pressure was 170/110 mmHg, and heart sounds were normal. Respiratory rate was 24 cycles per minute.Airway assessment showed a freely mobile temporomandibular joint and thyromental distance of 7 cm and categorised as Mallampati class 2. His physical status according to the American Society of Anesthesiologists (ASA) criteria was class 3. Nil per oral (NPO) was instituted from mid-night following the case discussion between Anaesthetic and Opthalmology teams. He was premedicated with oral diazepam 10 mg at night and on the morning of the surgery with a sip of water. He was also advised to take only atenolol and moduretic in the morning of the surgery.

Prior to the arrival of the patient in the theatre, routine checks were carried out on anaesthetic machine, monitors, and suction machine. Drugs for anaesthesia and other ancillary medications like hydralazine, adrenaline and lidocaine were drawn up in labelled syringes. On patient's arrival in the theatre, he was positioned supine with a 15-degrees head up tilt. Monitors were attached to the patient and base line vital signs were: blood pressure 150/90 mmHg, pulse rate 96 beats per minute, respiratory rate 22 cycles per minute, temperature 37 degrees Centigrade and arterial oxygen saturation while breathing room air was 100%. An intravenous line was set up on the dorsum of the left hand through which 5% dextrose in 0.9% saline

was infused.

The patient was preoxygenated for 5 minutes using a face mask with care not to compress the injured eye. Anaesthesia was induced with slow intravenous administration of 400 mg of sodium thiopentone. At the loss of eye lash reflex, he was test ventilated while on 0.75% halothane in oxygen and ability to ventilate with a face mask was confirmed before 30 mg of atracurium was administered intravenously. With close observation of the reservoir bag, patient and monitor (Dash 4000), spontaneous ventilation using a mixture of halothane and oxygen was allowed for 3 minutes until the patient stopped spontaneous breathing. Then gentle intermittent positive ventilation was administered via a face mask for another 5 minutes. Having ensured adequate muscle relaxation, swift laryngoscopy and tracheal intubation was carried out with a size 8.0 cuffed orotracheal tube. Correct placement of the tube was confirmed by equal chest expansion during lung ventilation, auscultation of the chest and capnograph before the orotracheal tube was secured to the angle of the mouth with adhesive tapes. The lungs were ventilated manually with oxygen using the Bain's breathing system at a fresh gas flow rate of 6 litres per minute, and the concentration of halothane was reduced to 0.5%. Atracurium, 10 mg was repeated intraoperatively. Intraoperative analgesia was with 30 mg of intravenous pentazocine.

Intraoperatively, the pulse rate ranged between 88 and 110 beats per minute while the blood pressure was in the range of 90-159mmHg systolic and 70-90 mmHg diastolic.The ECG showed few ventricular ectopic beats and the arterial oxygen saturation was between 97 and 100%. The axillary temperature ranged between 36.9 and 37.2^{0}C. Estimated blood loss was less than 100mls and he received a total of 1litre of 5% dextrose in 0.9% saline during the operation.At the end of surgerywhich lasted 45 minutes, halothane was turned off. The hypopharynx was cleared of secretions with sterile catheter before the patient regained consciousness. He was continued on 100% oxygen until he was finally extubated awake. He was transferred to the recovery room where he was monitored for 40 minutes before discharge to the eye ward.

Surgery and Findings

The patient was placed in the supine position with 15 degrees head up tilt. Routine cleaning and draping were done. The eye lids were retracted with a lid retractor and corneoscleral laceration was seen at 10 O' clock with uveal tissue prolapsing. The uveal tissue wasreduced and the cornea was sutured with nylon (8.0). Sublid gentamycin, dexamethasone, guttae mexitol and chloramphenicol were administered. The eye was padded with sterile gauze.

Postoperative Management

The patient received 5% dextrose in 0.9% saline 1 litre 8 hourly for 24 hours. Oral feeding was commenced within 24 hours and it was well tolerated by the patient. Oral ciprofloxacin 500 mg twice daily, metronidazole 400 mg 3 times daily and his antihypertensive drugs were commenced along with oral feeds. Postoperative analgesia was achieved with oral ibuprofen 100 mg 12 hourly for 5 days. He was discharged on the 12th postoperative day.

Discussion

Arterial hypertension is linked with an increase in the cardiovascular morbidity and mortality associated with anaesthesia and surgery[6,8,9]. In patients diagnosed as hypertensive and receiving anti- hypertensive medication, treatment of hypertension should be maintained throughout the perioperative period.Where hypertension is poorly controlled, management of the patients should follow the principles applicable to untreated hypertension. In untreated patients, mild hypertension (Stage 1: 140-159/90- 99mmHg), does not constitute a major threat. Moderate hypertension (Stage 2: 160-179/100-109mmHg), constitutes a threat especially where it is associated with target organ involvement (coronary, cerebrovascular or renal disease). Inthissituation treatment prior to elective surgery is recommended. Severe hypertension (Stage 3: 180-209/110-119) and marked left ventricular hypertrophy (on ECG and/or chest X-ray) is associated with increased risk of complications. Such patients should be treated before surgery.This also applies to patients with malignant hypertension (Stage 4: BP > 210/>120)[10].

Chronic hypertension may be associated with base line increase in cardiac output and systemic vascular resistance. With time the cardiac output returns to normal but systemic vascular resistance remains abnormally high leading to a chronic increase in after-load, which eventually results in concentric left ventricular hypertrophy as seen in this patient.Myocardial ischaemia and infarction are significantperioperative complications which are associated with poor patientoutcome. Attending anaesthetist should fully assess patients and the risks involved, adopting measures to prevent cardiac and

other incidents and to accurately detect such occurrence and provide rapid, effective management. Myocardial infarction that occurred less than three months before surgery could be associated with a high risk of reinfarction[10]. The American College of Cardiologists and American Heart association (ACC/AHA) recommend a delay of at least six weeks after uncomplicated myocardial infarction before administration of anaesthesia and surgery[10]. Irrespective of the delay between infarction and surgery, cardiac risks remain high in patients presenting for major abdominal or thoracic or vascular surgery. This is also applicable in patients who have suffered acute left ventricular failure at the time of their infarction.Morbidity and mortality are high in patients who exhibit poor left ventricular function and those who experience on-going angina. Evaluation of left ventricular function is therefore essential as there is an inverse relationship between left ventricular function and adverse cardiac outcome[11].

Goldman et al. defined cardiac risk of intraoperative and postoperative fatality or life threatening cardiac complications in surgeries[9]. Their report shows that the most important preoperative risk factors are a history of recent myocardial infarction and evidence of congestive cardiac failure. Two risk factors in this patient were previous myocardial infarction and ventricular ectopic beats These accounted for 12 of 53 possible points on the Goldman's cardiac risk index. This had placed him in class 2 and the risk of cardiac mortality and severe cardiovascular complications when subjected to anaesthesia and surgery were 4% and 17% respectively. Identifying patients at greatest risk allows appropriate measures to be taken to reduce morbidity and mortality. Generally accepted contraindications to elective non-cardiac surgery include myocardial infarction less than 1 month prior to surgery, uncompensated heart failure and severe aortic or mitral stenosis[12].

The 2007 American Heart Association (AHA) and the American College of Cardiology (ACC) guidelines for perioperative cardiovascular evaluation for non-cardiac surgery assesses various levels of risk using certain clinical markers, functional capacity and the type of surgery[13]. The major clinical markers include recent myocardial infarction, unstable angina, untreated heart failure, significant arrhythmias and severe valvular heart disease. Intermediate clinical markers include mild angina, history of angina, treated heart failure and diabetes mellitus. Abnormal ECG, non-sinus rhythm, and uncontrolled hypertension such as observed in the patient were grouped under minor clinical markers. A simple but reliable clinical method of assessing a patient's cardio-pulmonary reserve is the ability to climb a flight of stairs with minimal fatigue or dyspnoea[6]. This patient was able to perform this test with minimal fatigue.

In recent myocardial infarction, ECG reveals depression of S-T segment and there may be T-wave inversion, with the presence of pathologic Q-waves. S-T segment returns to iso-electric line in old infarcts. The diagnosis of old inferior myocardial infarction in this patient was based on the presence of pathologic Q-wave in lead III and avF (inferior leads), inverted T-wave in lead III and iso-electric S-T segment. The ECG may be normal at rest unless a previous myocardial infarction had occurred. Abnormalities of ECG usually develop with exercise but there may be false positives and false negatives[14]. Presence of hypertension and left ventricular hypertrophy in a patient is an important predictor of cardiovascular morbidity and mortality and it is associated with increased ventricular ectopic beats, impaired coronary reserve and death[14]. Preoperative chest radiograph may show evidence of left ventricular hypertrophy and unfolding of the aorta as was observed in this patient and was consistent with his hypertensive state. Long standing uncontrolled hypertension predisposes to glomerulosclerosis which may impair renal function and diuretics if used in the treatment of hypertension may cause electrolyte derangement. Electrolyte imbalance may also cause arrhythmias. Patient's drug history should be reviewed, and serum electrolyte, urea and creatinine estimated. This patient's result were mostly within normal range.

The use of echocardiography is popular because it allows the evaluation of regional and global functions of the heart and may reveal the presence of hitherto unrecognized valvular defect. It is also regarded as a gold standard for detecting myocardial ischaemia[15]. Dobutamine stress echocardiography is a sensitive and specific test for ischaemic heart disease[15]. Radionuclide ventriculography if available, gives detailed information on regional wall motion, synchrony of contractions and global function of the myocardium.

Other investigations which may be necessary to exclude secondary causes of hypertension include 24-hour urinary creatinine clearance, urinary catecholamines (if phaeochromocytoma is suspected), plasma renin activity, renal angiography, intravenous urography, test for Cushing's and Conn's syndromes and abdominal ultrasound. They were not done because

of cost implication and their benefit in the anaesthetic management of the patient were considered in significant. Perioperative treatment of hypertension is necessary because it reduces the frequency of haemodynamic fluctuations, that could be related to morbidity[4. 14]. Prys-Roberts suggested that in untreated patients, postponement of surgery is unnecessary unless the diastolic pressure exceeds 120mmHg[16]. For treated hypertension, cancellation in order to improve treatment may be justified if the diastolic pressure exceeds 110mmHg. Similarly, the AHA/ACC guidelines suggest that patients with a diastolic blood pressure above 110mmHg should be treated before surgery[13].Medication for ischaemic heart disease should be continued throughout the perioperative period. The American College of Physicians guideline for assessing and managing the perioperative risk from ischaemic heart disease associated with major non-cardiac surgery highlights the efficacy of beta-blockersin patients with ischaemic heart disease[13]. A study by Poldermans et al.on the effects of perioperative beta-blockade in patients with coronary artery disease concluded that atenolol given one week perioperatively resulted in lower mortality[17]. In order to achieve better control of blood pressure and improve the cardiovascular condition in this patient, surgery was postponed overnight and the patient received the morning dose of atenolol on the day of surgery. In emergency situation parenteral anti- hypertensives such as hydralazine, sodium nitroprusside, nitroglycerine, propranolol and esmolol may be used.

In order to prevent persistent hypertension from anxiety, the patient was reassured during the preanaesthetic visit as this has been shown to reduce anxiety[18]. Supplementation of psychotherapy with oral diazepam is necessary. In this way, the patient was made to arrive in the operating room calm and sedated. Adequate premedication in hypertensive patients diminishes the likelihood of violent alteration in either direction of the blood pressure during the intraoperative period[4]. Oral anticoagulation of a patient with preoperative arrhythmias but no mechanical valve, can be discontinued for up to 1 week before surgery[14]. Low dose aspirin may have little or no effect on platelet function or coagulation and therefore was discontinued the night before surgery. Treatment with angiotensin converting enzyme inhibitor (ACE) is associated with the risk of refractory hypotension after induction of anaesthesia and during surgery[19]. Omission of the morning dose of ACE inhibitors is often indicated.

The anaesthetic technique for a patient with hypertensive heart disease presenting for ophthalmic surgery should include control of IOP, provision of akinesia, identification of intraoperative manifestation and treatment of oculo-cardiac reflex. It must also ensure adequate oxygenation of the myocardium, adequate maintenance of cardiac output and adequate systemic arterial pressure to maintain major organ perfusion such as brain, heart, kidneys and the liver. It should also ensure maintenance of a balance between the oxygen supply and demand of the myocardium to minimize the risk of perioperative ischaemia and infarction.

Available anaesthesia options for eye surgery include regional block, general anaesthesia with endotracheal intubation or laryngeal mask airway (LMA). Regional anaesthesia has the advantage of rapid onset of action, rapid recovery and avoidance of risk of general anaesthesia. However, this technique is inappropriate for open eye injury as it may lead to an increase in IOP and result in extrusion of ocular contents. LMA insertion is easily applied and the problems of coughing, straining and laryngospasm are minimized but it does ensure adequate protection of the airway. General anaesthesia with endotracheal intubation was chosen because airway protection was guaranteed and it offered the surgeon unlimited access to the operation site.

Disturbances of cardiac rhythm during the intraoperative period are more common in patients with heart disease and pre-existing dysrhythmias[4]. Predisposing factors include: increased plasma catecholamine levels due to anxiety, laryngoscopy and intubation, inadequate analgesia, hypoxia and hypercarbia. These factors may also increase IOP. Application of the anaesthetic technique that may avoid all these factors should be considered. Adequate ventilation and oxygenation of the patient in the perioperative period is mandatory to prevent hypoxia and hypercabia[20]. Hypoxia from apnoea following induction of anaesthesia should be prevented by preoxygenation for 3-5 minutes with high flow rate of 100% oxygen (more than 6 litre/minute)[21]. The face mask should be applied with caution during preoxygenation since pressure on the eye with penetrating injury can result in extrusion of ocular content. Pressure on the eye or traction on the medial rectus as well as other orbital structures triggers oculo-cardiac reflex. The most common manifestation is bradycardia, a wide spectrum of dysrrhythmia may occur including junctional rhythm, trigeminy and asystole[12].

Induction of anaesthesia and endotracheal intubation are often associated with haemodynamic instability in hypertensive patients. Regardless of the level of preoperative blood pressure control, many patients with hypertension display an accentuated hypotensive response to induction of anaesthesia followed by an exaggerated hypertensive response to intubation[4]. The hypotensive response at induction may reflect the additive circulatory depressive effects of anaesthetic agents and antihypertensive agents which are vasodilators, cardiac depressants or both. The superiority of any induction agent over another has not been clearly established but the manner in which it is given is important. Most induction agents decrease IOP and therefore can be successfully used with careful titration and monitoring[4,20]. Ketamine is contraindicated, because it causes sympathetic stimulation which can precipitate marked hypertension and increase in IOP[4,20] which is undesirable in this patient. Thiopentone is associated with hypotension, often with compensatory tachycardia[4]. It should therefore be administered cautiously as was done here. Etomidate has less effect on the pulse and blood pressure than thiopentone and can be used[22]. Propofol causes hypotension and bradycardia[24]. Mild bradycardia can be beneficial by allowing greater diastolic filling provided the blood pressure is maintained. The hypotensive effect of propofol can be reduced by slow induction.

Intubation of the trachea stimulates laryngeal receptors, resulting in marked increase in secretion of sympathomimetic amines. This sympathetic stimulation results in tachycardia and a rise in blood pressure and IOP which is undesirable in this instance. Such rise in blood pressure and tachycardia occurs about 14 seconds after the start of laryngoscopy and becomes marked after 30 seconds to 45 seconds of direct laryngoscopy. Different agents have been employed to attenuate sympathetic response to laryngoscopy and tracheal intubation[23-25]. Ventilation with a potent volatile agent was used and found effective. Administration of bolus dose of a narcotic (fentanyl 2.5ug/kg, alfentanil 15-25ug/kg or sufentanil 0.25-0.5ug/kg) is recommended[4]. Lignocaine at a dose of 1.5mg/kg given intravenously or intratracheally can obtund the pressor response to laryngoscopy. Other methods include intravenous B-adrenergic blockers e.g. Esmolol 0.3-1.5mg/kg, propranolol 1-5mg/kg or labetalol 10-15mg/kg. Second-dose thiopentone, sodium nitroprusside or nitroglycerine can also help to blunt a rise in blood pressure and heart rate following laryngoscopy and intubation. Nicardipine and verapamil are effective in attenuating pressor response to laryngoscopy and intubation but do not control trachycardia[25] and topical local anaesthetic to the airway is also promising. Premedication with intravenous clonidine ($alpha_2$ agonist) has a proven to be efficacy as well as use of magnesium sulphate[26].

The most important factor that minimizes exaggerated blood pressure at intubation is limitationof duration of laryngoscopy to 15 seconds or less[27]. Deepening anaesthesia with halothane has been employed to achieve same purpose.Combination of these 2 methods satisfactorily attenuated sympathetic response to tracheal intubation in this patient. There was no significant haemodynamic change after intubation.Suxamethonium is traditionally the muscle relaxant of choice with rapid onset of action that facilitates rapid sequence induction and tracheal intubation but was avoided here because the patient did not have a full stomach and its use carrieda risk of rise in intraocular and intracranial pressures[28]. The choice of atracurium to facilitate tracheal intubation and maintain muscle paralysis was based on its blood pressure lowering effect which is beneficial in hypertensive heart disease[28]. Vecuronium causes less haemodynamic changes and is therefore a good alternative[28]. Pancuronium induces vagal blockade and neural release of catecholamines which can exacerbate hypertension in poorly controlled patients[28].

Anaesthesia may be safely continued with volatile agents (alone or with nitrous oxide), a balanced technique (narcotic + nitrous oxide + muscle relaxant), high doses of opioids or under totally intravenous techniques. The vasodilation and relatively rapid and reversible myocardial depression afforded by volatile agents allows titration of their effects against arterial pressure. They also have additional advantage of reduction of IOP. Isoflurane might cause less haemodynamic changes but has been implicated in pre-stenotic vasodilation which diverts blood away from already ischaemic areas of the myocardium, a phenomenon referred to as coronary steal especially in patients receiving nifedipine[29]. Its use was therefore contraindicated in this patient who was on nifedipine preoperatively. Halothanecauses arrhythmias especially in the presence of increased circulating catecholamines[12]. Avoidance of factors that may predispose the patient to raised catecholamines such as hypoxia, hypercapnia is often necessary. The depressant effects of halothane on the cardiac output are augumented in the presence of beta-blockade; itmay be advantageous in patients with coronary artery

disease because of the reduced oxygen demand caused by a low heart rate and decrease contractility[12]. A low concentration of halothane was used and few ventricular ectopic beats observed, resolved spontaneously.

Central venous pressure (CVP) has a direct relationship with intra- ocular pressure[4,20]. It is increased by coughing, breath-holding and obstruction to respiration which were avoided by muscle paralysis and manual ventilation. A head up tilt of 15 degrees produces a significant reduction in CVP and IOP due to enhanced venous drainage of the head and neck. High inspired oxygen concentration is essential in patients with hypertensive heart disease in order to maintain adequate tissue oxygenation. Monitoring of arterial oxygen saturation as was done here is mandatory. Oxygen delivery to the tissues also depends on the oxygen carrying capacity of the blood which in turn depends on the haemoglobin concentration of the blood. Blood loss should therefore be closely monitored, and significant blood loss replaced. The relationship between carbon dioxide and IOP is almost linear, hyperventilation to arterial carbon dioxide of between 3.5 – 4.0 kPa has been found to produce a significant reduction in IOP[20].

Intraoperative hypothermia can result in shivering which leads to increased oxygen demand by the myocardium. Significant heat is lost during humidification of dry anaesthetic gases, and infusion of cold fluids. Efforts should be made to reduce heat loss and monitoring of body temperature is mandatory. ECG allows early detection of perioperative myocardial ischaemia[12,24]. In high risk patients, invasive monitoring, including monitoring of the pulmonary occlusion pressure (pulmonary capillary wedge pressure) is useful for major surgery. Transoesophageal echocardiography (TOE) may be useful for the detection of ischaemia and the assessment of ventricular filling[14]. This should be done where facilities are available.

Controlled ventilation is associated with increased intra thoracic pressure, and hypotension with reduced coronary blood flow which may result from positive end-expiratory pressure (PEEP) ventilation. PEEP ventilation should therefore be avoided. Intraoperative increase in pulse rate and blood pressure can be caused by pain and light anaesthesia, thus adequate analgesia should be ensured. The monitoring of blood pressure is therefore mandatory in patients with hypertensive heart disease. Intraoperative hypertension that does not respond to an increase in anaesthetic depth is common in hypertensive patients. Readily reversible causes such as hypoxia or hypercapnia should always be excluded before initiating antihypertensive agent[4]. Selection of a hypotensive agent depends on the severity, acuteness, the cause of hypertension and also the heart rate. Nitroprusside if available remains the most rapid and effective agent for the intraoperative treatment of moderate to severe hypertension[4]. Nitroglycerine may be less effective but is also useful in treating or preventing myocardial ischaemia. A B-adrenergic blocker is a good choice for patients with good ventricular function and an elevated heart rate but is contraindicated in the presence of hyperactive airway[4,22]. Hydralazine produces sustained blood pressure control but has a delayed onset and is often associated with reflex tachycardia. The later is not seen with labetalol because of combined alpha- and beta-adrenergic blockade effect. Other agents that can be used include nicardipine, methyldopa, phentolamine, trimethaphan, esmolol, diazoxide and enalaprilat[4].

Treatment of intraoperative dysrrhythmia depends on the nature of the abnormal rhythm and its effect on cardiac output. Single supraventricular or ventricular ectopic beats and slow supraventricular rhythms do not require treatment unless cardiac output is compromised[7]. Supraventricular tachycardia may be treated with intravenous verapamil or beta-adrenergic receptor blockers. Verapamil is a calcium channel blocker which acts predominantly on the atrioventricular (AV) node to increase its block. Cardioversion may be performed if these drugs are ineffective. Ventricular tachycardia (VT) responds to intravenous lignocaine but if unsuccessful bretylium and amiodarone may be tried. Direct current (DC) shock should be employed early particularly if VT is associated with decompensation[12]. The antiarrhythmic drugs made available during the intraoperative period were not used as only occasional ventricular ectopic beats were noticed and the patient was in stable haemodynamic condition throughout the intraoperative period.

Oropharyngeal suctioning and extubation also stimulate sympathetic response. Extubation when anaesthesia is deep predisposes to airway obstruction and hypoventilation. This may lead to hypoxaemia and hypercabia predisposing the patient to myocardial ischaemia and increase in IOP. This was minimized by extubating the patient fully awake.

Marked sustained elevations in blood pressure can contribute to the formation of wound haematoma, disruption of vascular suture lines, myocardial

ischaemia and congestive heart failure in the early postoperative period. Hypertension in the recovery room is often multifactorial and enhanced by respiratory abnormalities, pain and volume overload or bladder distention. Close blood pressure monitoring, adequate analgesia and meticulous fluid management are necessary postoperatively. Hypertensionin the immediate post-operative period should be treated with sublingual nifedipine[4].

Eye surgeries are associated with postoperative nausea and vomiting (PNOV). It forms a limb of the oculo-medullary reflex tripod that has oculo-respiratory reflex, oculo-cardiac reflex and oculo-emetic reflex. PONV may be caused by pain. Perioperative provision of analgesia is essential (also employed here) and this patient did not experience PONV.Deep vein thrombosis and pulmonary embolism are common in patients with hypertensive heart disease[30]. Early ambulation should be encouraged and subcutaneous low molecular weight heparin is often indicated.

Conclusion

Optimal anaesthetic management of the patient with hypertensive heart disease requires a thorough knowledge of normal cardiac physiology and the circulatory effects of various anaesthetic agents. The same principles used in the management of this disease preoperatively also apply during the perioperative period. Anaesthetic technique should ensure adequate oxygen delivery without increasing myocardial oxygen demand.

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