

Cardiac Surgery Complications

Cardiac Anesthesia Complications

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1. Introduction

Advances in technology have improved anesthesia care for patients undergoing cardiac surgery and progress in

pharmacology has led to the availability of anesthetic medications with low side-effect profiles and fewer effects on patient hemodynamics. Anesthesia machines are now more sophisticated and multi-functional, able to provide complex modes of ventilation such as synchronized intermittent mandatory ventilation (SIMV) and pressure-control. Gas analyzers closely follow the level of anesthetic delivered to and received from the patient, and the use of intraoperative transesophageal echocardiography is common in many cardiac surgery centers for diagnosis of preoperative and intraoperative problems. This progress has helped advance patient care; however, it has also presented a challenge to today's practicing anesthesiologist.

Cardiac anesthesiologists are faced with the task of mastering not only pharmacology, physiology, and the physiologic consequences of anesthetics, but they must also understand the operation, risks, and benefits of the equipment used for close patient monitoring. Vigilance is of utmost importance in caring for all patients undergoing anesthesia.

Cardiac anesthesia complications can arise during a number of different situations involving patients who are undergoing a variety of procedures including aortocoronary bypass grafting, valvuloplasty, valve replacement, aortic aneurysm resection, transplant, carotid endarterectomy, lobe resection, pneumonectomy, and peripheral vascular procedures. Many of these situations are unique to the cardiac surgery patient and quick recognition and efficient management of these perioperative complications can help to reduce patient morbidity and mortality.

2. Pharmacologic Effects

a. Inhalational Anesthetics: Volatile agents such as isoflurane, desflurane, sevoflurane, and halothane are supplied in liquid form. Each volatile agent has a temperature dependent

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vapor pressure, and vapor administration to patients is carefully controlled with a special delivery system usually involving a variable bypass vaporizer. When inhaled, these agents produce loss of consciousness with amnesia, hypnosis, analgesia, and muscle relaxation to varying degrees depending on the dose administered. Most volatile agents are irritant to the airway with the exception of sevoflurane and halothane, which are better tolerated and suitable for inhalation induction. Progressive administration can lead to ventilatory depression and hypotension secondary to systemic vasodilatation and myocardial depression.

Management of the unconscious patient with residual vapor on board is supportive until the remaining agent is exhaled and/or metabolized. This may include assisted or mechanical ventilation, replacement of volume deficits, and the use of vasopressors or inotropes to maintain adequate hemodynamics. Major sequelae include hypoxemia, hypercarbia, respiratory acidosis, hypotension, and increased intracranial pressure. Time to recovery depends on many factors including minute ventilation, the type of agent and dose administered, and any lingering effects of adjuvant anesthetics used such as narcotics or tranquilizers.

Nitrous oxide is an odorless inorganic anesthetic gas with a low blood/gas partition coefficient. It leads to an altered state of consciousness and is 35 times more soluble in blood than nitrogen. As a result, it diffuses into air-containing spaces faster than nitrogen is able to exit. Therefore, its use may lead to enlargement of gaseous emboli, and rapid expansion with the potential for hemodynamic compromise in patients with pneumothorax, pneumocephalus, and pneumomediastinum. Nitrous oxide must also be avoided in cases of intestinal obstruction due to the distension of closed loops of bowel.

b. Induction Agents: Intravenous agents such as sodium thiopental, propofol, and etomidate are commonly used during

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the induction phase of general anesthesia. When given as a bolus, these agents induce a rapid state of unconsciousness and hypnosis accompanied by apnea, amnesia, and attenuation or abolition of airway reflexes. After receiving a single bolus dose, patients will quickly return to a state of wakefulness in roughly 3-5 minutes secondary to the short alpha redistribution half times for these agents. Tools for providing ventilation and securing the patient's airway should be immediately available when administering these medications.

Thiopental is a rapid acting barbiturate with a long-standing safety record. Induction doses range from 3-5 mg/kg, and there is evidence that thiopental is neuroprotective in cases of focal cerebral ischemia. Pentothal induced hypotension is mainly secondary to vasodilatation; however, in higher doses myocardial depression may become a significant cause of hypotension, especially in the patient with marginal myocardial function.

Propofol (Diprivan ®) is provided as a 1% aqueous solution and is often recognized by its white color. It is an emulsion containing soybean oil, glycerol, egg lecithin, and EDTA. Propofol may induce a greater degree of hypotension secondary to vasodilatation (primarily arterial) relative to pentothal, and it creates an intense burning sensation when given intravenously. This burning sensation can be minimized with the concurrent administration of intravenous lidocaine. Myocardial depression may also occur during its use. The induction dose is 2-2.5 mg/kg with doses for sedation ranging from 50-200 mcg/kg/min. One advantage clinically, in addition to rapid awakening, is that propofol appears to have some anti-emetic properties which makes it useful in patients with a history of post-operative nausea and vomiting.

Etomidate (Amidate ®) is an imidazole induction agent recognized for its minimal effects on the cardiovascular system. It is generally cardiac stable when used for induction in the

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dose of 0.3 mg/kg. However, several of its effects should be recognized. Etomidate may cause adreno-cortical suppression leading to hemodynamic compromise in critically ill patients. Its use is also associated with myoclonus and post-operative nausea and vomiting.

Ketamine (Ketalar \hat{a}) is a unique anesthetic agent that provides a dissociative state of anesthesia while minimally affecting respiratory drive. A derivative of phencyclidine (PCP), it is a potent analgesic. The intravenous induction dose is 1-2 mg/kg, and in contrast to the previously mentioned intravenous agents, ketamine leads to an increase in arterial blood pressure, heart rate, and cardiac output secondary to central stimulation of the sympathetic nervous system. However, in high enough doses and in marginal patients, it can also act as a significant myocardial depressant. Patients with coronary disease may have an increased risk for ischemia when receiving ketamine due to its sympathomimetic effects.

c. Opioids :Opioids are frequently used in cardiac anesthesia to provide potent analgesia in combination with other agents to provide hypnosis and amnesia. Fentanyl, sufentanil, alfentanil, morphine, and meperidine are common choices.

The administration of opioids not only provides profound analgesia, but it also markedly increases the apneic threshold and decreases hypoxic drive. Unmonitored and sedated patients are at risk of developing hypoxemia, hypercarbia, and aspiration secondary to obtunded airway reflexes. Opioids also reduce peristalsis that can slow gastric emptying. Patients receiving some of the more potent synthetic narcotics (e.g. fentanyl, sufentanil, alfentanil, and remifentanil) may experience severe chest wall rigidity impairing ventilation and oxygenation. The risk of rigidity can be minimized by decreasing the rate of opioid administration and by deepening

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the level of anesthesia with other intravenous or volatile agents.

With the exception of morphine and meperidine, opioids have very little effect on myocardial function. A mild decrease in blood pressure may be seen during their use due to bradycardia and some degree of vasodilatation. High dose morphine is rarely used due to its prolonged respiratory depressant effects. Meperidine must also be used with caution since patients may develop tachycardia due to its structural similarity to atropine.

It is imperative to closely monitor patients receiving opioids to avoid complications from hypoxemia and hypercarbia.

D. Muscle Relaxants: Muscle relaxants are commonly used during the delivery of anesthesia to facilitate tracheal intubation and to improve surgical exposure and operating conditions. There are two classifications of muscle relaxants: depolarizing and non-depolarizing.

Depolarizing Agents: The only depolarizer in clinical use is succinylcholine which is recognized for its rapid rate of onset (30 seconds) and rapid rate of offset (3-5 minutes). However, because of its depolarizing nature, its use must be selective. Fasciculations are commonly observed in doses of 1-2 mg/kg IV, and complications arising from succinylcholine administration include trismus, myalgias, increased intracranial/intragastric/intraocular pressures, and hyperkalemia with cardiac arrest, especially in pediatric patients with an undiagnosed myopathy. Patients with a history of a recent burn, crush injury, progressive myopathy or denervation injury should also be evaluated for an alternative agent due to the significant risk of hyperkalemic dysrhythmias and the potential for cardiac arrest.

Nondepolarizing Agents: The nondepolarizers do not create fasciculations during their administration. These agents

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work by competitive antagonism and the degree of muscle relaxation is often titrated with the use of a nerve stimulator. In general, these agents have a slower onset and longer duration of action when compared to succinylcholine. Some commonly used nondepolarizers with their induction doses are vecuronium (0.1 mg/kg), pancuronium (0.1 mg/kg), rocuronium (0.6-1.2 mg/kg), rapacuronium (1.5 mg/kg), cis-atracurium (0.15-0.2 mg/kg), mivacurium (0.15-0.2 mg/kg), and d-tubocurarine (0.5-0.6 mg/kg). Depending on the degree of blockade, the effects of these agents may be reversed by the administration of neostigmine (.04-.07 mg/kg IV) in combination with an anticholinergic such as glycopyrrolate (10 mcg/kg IV). Edrophonium (1 mg/kg) and atropine (.014 mg/kg) may also be used together to reverse the action of non-depolarizers. Anticholinergics should be administered concomitantly with anticholinesterases to help minimize the cholinergic side effects associated with neuromuscular blockade reversal.

Progressive administration of muscle relaxants will lead to bulbar muscle weakness, airway obstruction, diplopia, ineffective ventilation and oxygenation, and eventual paralysis. Residual neuromuscular blockade must be recognized quickly to avoid significant hypoxemia. Patients will appear weak and tachypneic, unable to lift their head or extremities to any great extent. Their appearance is likened to that of a "floppy fish". The treatment for residual neuromuscular blockade (in addition to adequate reversal) is mechanical ventilation with sedatives to provide amnesia. Great attention must be taken to ensure amnesia in a patient that is receiving relaxants and lorazepam, diazepam, midazolam, and propofol are drugs commonly used to achieve this goal.

E. Cardiovascular Recovery Considerations:

Recognizing the effects of anesthetic agents in the cardiovascular recovery setting is vital to proper patient management. There should be little to no hemodynamic

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consequence from inhalational agents; however, residual muscle relaxants may often be present and steps should be taken to ensure patient amnesia until their effects subside. Patients undergoing major cardiac surgery will often remain analgesic throughout the immediate post-operative period secondary to large doses of intra-operative narcotics, but upon emergence, post-operative pain control can become a very serious issue.

Post-operative pain may be managed in a variety of ways ranging from parenteral narcotic administration to regional anesthetic techniques. Depending on the type of surgical procedure, regional anesthesia may be an appropriate choice. For example, patients undergoing thoracotomy with thoracic epidural anesthesia are often candidates for early extubation, and the analgesia provided by the epidural can be used intra-operatively to blunt the stress response to surgery. Furthermore, thoracic epidural anesthesia may encourage deep breathing, coughing, and early ambulation post-operatively. For peripheral vascular procedures involving the lower extremities, lumbar epidurals not only provide pain relief, but they may also improve graft flow. Intrathecal administration of preservative-free preparations of morphine, fentanyl, and sufentanil may also be used for pain relief. Furthermore, peripheral and selective nerve blocks (e.g., rib blocks) are also appropriate choices for analgesia.

The intravenous or intramuscular use of narcotics is an effective and proven method of pain management. Patient controlled analgesia (PCA) is also popular in many institutions. When compared to intravenous dosing, intramuscular narcotic administration allows an increase in the dosing interval with a more sustained release of medication from the IM depot. Non-narcotic analgesics such as ketorolac (Toradol ®) may also be used in addition to a variety of NSAIDs and cyclooxygenase-II inhibitors.

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Complications arising from regional techniques include spinal and nerve root injury. Epidural hematoma formation with neurologic compromise can be a devastating consequence, and alternatives to regional should be considered in patients who will be heparinized to any degree peri-operatively. Patients may also experience severe hypotension and bradycardia as a result of sympathetic blockade created by the regional anesthetic. Vasoconstrictors and volume doses can be used to alleviate this effect. Intrathecal narcotic administration may lead to delayed emergence and prolonged respiratory depression, and patients may also experience adverse reactions to the local anesthetics and narcotics such as neuromuscular weakness, itching, and nausea. Effective pain management not only achieves patient comfort, but it also provides the basis for a stable patient hemodynamic profile.

3. Procedural Complications

a. Arterial Catheters: Arterial lines are a common form of invasive blood pressure monitoring used during cardiac surgery and anesthesia. Many different types of catheters may be used including standard IV angiocaths, and special catheters designed by Arrow[®], Cook[®], and Medi-Cut[®].

Sites for cannulation include the radial, ulnar, brachial, axillary, femoral, dorsalis pedis, and temporal arteries. The most common site for invasive blood pressure monitoring is at the radial artery, which offers easy accessibility and good collateral circulation.

The risk to benefit ratio of arterial cannulation is extremely low. Most complications are due to emboli, and thrombosis of the radial artery is usually a benign event due to arterial recanalization. If a limb containing an invasive line appears to be poorly perfused, the patient should be evaluated for signs of generalized vasoconstriction. If after this, the line remains suspect, papaverine 3-5 mg given slowly through the arterial

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line will help to counteract any vasospasm. If there is no improvement after papaverine administration, the line may be removed.

Complications from arterial cannulation include vessel dissection, vasospasm, emboli (air, plaque, debris), hematoma, pain, bleeding, nervous injury, and infection. Depending on the site of cannulation, partial or complete occlusion of arterial flow can potentiate limb and digit ischemia. However, Slogoff and Keats demonstrated that radial artery cannulation is a low-risk, high benefit monitoring technique (see suggested readings).

b. Central Venous Catheters: Central access is vital to caring for the cardiac patient in the operating suite. Central lines allow the monitoring of intracardiac filling pressures and provide a means for the rapid administration of volume and blood products. They also provide a site for frequent blood draws.

Sites for placement include the internal and external jugular veins, the subclavian vein, and the femoral vein. Peripherally inserted central catheters (PICC lines) may also be used. Most commonly, for cardiac surgery, the right internal jugular vein is cannulated because of ease of access and decreased incidence of complications.

The left internal jugular vein may also be used, but large bore lines should be avoided due to the risk of ino-inate vein perforation. Subclavian access is a good choice for patient comfort, and the left subclavian allows easier access to the superior vena cava and the right atrium when compared to the right subclavian. When placing a pulmonary artery catheter, the right internal jugular vein and left subclavian vein are the two most common sites. Femoral lines can provide good central access; however, great care must be taken to keep the site clean and dry. Furthermore, flexing and movement of the lower extremities can severely alter catheter function.

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Complications from central venous cannulation include pneumothorax, hemothorax, hematoma, vascular or nervous injury, pain, bleeding, infection, and cardiac dysrhythmias. Inexperience lends to a higher complication rate during central venous line placement. Therefore, the combination of an experienced operator and a cooperative patient can significantly reduce the rate of adverse events.

c. Pulmonary Artery Catheters: PA catheters are used perioperatively to help manage critically ill patients with conditions such as severe ventricular dysfunction and pulmonary hypertension. PA catheters allow continuous monitoring of the CVP, PA pressures, and cardiac output via thermodilution or continuous cardiac output (CCO) monitoring. Mixed venous blood may also be sampled from the distal port allowing the calculation of intrapulmonary shunt and the assessment of global adequacy of cardiac output based on mixed venous oxygen tension and saturation values.

The placement of pulmonary artery catheters can carry significant risk. In addition to the complications associated with placing a central line, patients may experience dysrhythmias including complete heart block in patients with a preexisting LBBB, ventricular perforation, pulmonary artery rupture, and knotting with entrapment of the catheter itself. The practitioner must also be aware of catheter movement and migration during sternotomy and cardiopulmonary bypass which may increase the risk of over-wedging and PA rupture. PA catheters are also not associated with improved outcome, but they may be useful for trending in chronic patients.

Transesophageal echocardiography (TEE) is a better tool at detecting cardiac ischemia and regional wall motion abnormalities. In our practice at The Texas Heart Institute, PA catheters are placed when trying to manipulate the PA pressures such as in patients with pulmonary hypertension or those undergoing orthotopic heart transplantation. When

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compared to pulmonary artery catheterization, TEE is more commonly used as a diagnostic tool and peri-operative monitor in our patients.

d. Intubation: Securing a patient's airway may be performed in a variety of ways including both orotracheal and nasotracheal approaches. Nasotracheal intubations are usually not performed in cardiac patients due to the risk of bacteremia and sinusitis. It is important to remember that adequate ventilation and oxygenation are the top priority, and these goals may be reached with an ambu bag and a skilled practitioner providing a mask airway while intubation supplies are prepared.

Orotracheal intubation with direct laryngoscopy is common to most general anesthetics. A laryngoscope is used to sweep the tongue and expose the larynx for placement of the endotracheal tube. Miller (straight) and MacIntosh (curved) are the two most prevalent blades in use today. Oral approaches may also include the use of laryngeal mask airways, fiberoptic bronchoscopes, and other devices such as Bullardâ laryngoscopes, light wands, and esophageal combi-tubes.

Nasotracheal intubation is often accomplished with the use of a fiberoptic bronchoscope or a blind technique, such as a blind nasal intubation using a Beck's whistle to audibly follow the patient's respirations. A nasal intubation may be more difficult when compared to an oral intubation; however, a nasal approach allows placement of the ETT from the nasopharynx during spontaneous ventilation without interference from the teeth of a combative or struggling patient. Nasotracheal tubes are generally better tolerated by patients, but the majority of patients receiving one will develop a maxillary sinusitis.

Complications from tracheal intubation are numerous and can have serious consequences. An unrecognized esophageal intubation can quickly become a catastrophe. The use of end-tidal capnometry, auscultation, radiography, and fiberoptic

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confirmation all help to minimize this complication. Mucosal tears with bleeding, esophageal perforation, damage to the teeth/lips/gums, trauma to the epiglottis, larynx, and trachea can also occur. Dental damage may even occur post-op when patients with fragile teeth bite down on the endotracheal tube while awakening. This is difficult to avoid. Trauma to the airway may result in significant edema leading to airway obstruction after extubation, but this risk may be minimized through the use of racemic epinephrine (0.3 cc of a 2.25% solution nebulized), steroids (e.g. dexamethasone 4-8 mg IV), and increasing the humidification of inhaled gases.

4. Special Circumstances

a. Coagulopathy: Patients undergoing cardiac surgery are at an increased risk of bleeding and hemorrhage. Patients presenting for cardiac surgery are often on medications which have antithrombotic/antihemostatic activity such as heparin, low molecular weight heparin, and aspirin. Antiplatelet agents such as clopidogrel (Plavixâ), eptifibatide (Integrelinâ), and abciximab (Reoproâ) have also become more prevalent. Although these agents may help prevent pre-operative morbidity, they can also potentiate significant perioperative bleeding.

In addition to specific medications, cardiopulmonary bypass (CPB) can also create a coagulopathic state for many reasons. Placing a patient on CPB involves a certain pump priming volume which varies from institution to institution. At The Texas Heart Institute, our standard crystalloid prime consists of 20 cc/kg of 5% Dextrose in Lactated Ringers. Priming volumes instantly alter a patient's effective blood volume and volume of distribution. Dilutional thrombocytopenia and dilution of circulating coagulation factors occurs when instituting CPB. However, during routine CPB runs, the dilution of coagulation factors is often not clinically significant, and

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depending on the specific factor, only 20-30% needs to be present to have an intact coagulation cascade.

An important reason for coagulopathy related to CPB is platelet dysfunction. CPB partially activates platelets, attenuating their granule release and decreasing their ability to aggregate and respond to vascular injury. Post pump, when attempts at hemostasis are made after protamine administration, patients are often thrombocytopenic with dysfunctional, partially activated platelets, and longer pump runs increase the risk for platelet dysfunction. If significant enough, the remedy to this situation in the absence of a correctable surgical cause is fresh platelet transfusion, and the administration of DDAVP (0.3 ug/kg IV/SQ) to increase circulating vWF to aid in platelet aggregation. In diagnosing this problem, it is important to understand the difference between absolute platelet count and platelet function. Platelet function can be measured in the laboratory by a special calcium/ADP titrated assay based on the transmission of light. The result is reported as a percentage (e.g., 50% platelet function).

Unfortunately, a completely non-thrombogenic CPB circuit does not exist. Despite clinically adequate doses of heparin, there still exists evidence of fibrin formation within the CPB circuit, but this is not clinically significant in pump runs under 120 minutes. Likewise, CPB incites fibrinolysis which may also potentiate bleeding. However, fibrinolysis secondary to CPB is usually self-limited and not a prominent cause of bleeding in the perioperative period. The effects of fibrinolysis can also be minimized with the use of specific antifibrinolytic agents such as epsilon aminocaproic acid (150 mg/kg load followed by 15 mg/kg/hr) and tranexamic acid (15 mg/kg load followed by 1.5 mg/kg/hr). Aprotinin is associated with a significant decrease in bleeding, and it is often used in redo-patients. However, there is a risk of graft thrombosis associated with its use. For aprotinin, regimen A (Hammersmith High Dose) consists of a 1 cc test dose followed by 2 million KIU load, 2 million KIU to the

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CPB prime, and 500,000 KIU/hr infusion. Regimen B (Hammersmith Low Dose) is exactly one-half of the regimen A dosing. Pulmonary microemboli have also been associated with the use of aprotinin, which can lead to pulmonary vascular obstruction, increases in PVR, and acute precipitation of right heart failure, although the complete etiology is unknown.

Hypothermia is often employed during runs of cardiopulmonary bypass to globally decrease oxygen consumption and create a margin of safety in the event of cerebral or cardiac ischemia. Hypothermia provides neuroprotection, and completely normal temperature is associated with an increase in neurologic complications. Hyperthermia post-operatively may also exacerbate neurologic injury. Hypothermia aids in preserving and protecting organ function during low-flow and embolic states (e.g., involving air, plaque, and debris). Furthermore, it allows a lower flow state to help with a quiet and bloodless surgical field. However, it may also potentiate a coagulopathic state by inducing splanchnic sequestration of platelets leading to thrombocytopenia. Other deleterious effects of hypothermia include decreased coagulation enzymatic cascade function, cardiac dysrhythmias, delayed wound healing, increased peripheral vascular resistance, increased blood viscosity, and left-shifting of the hemoglobin-oxygen dissociation curve. Shivering in the non-relaxed patient may also cause a problem by increasing oxygen consumption in non-essential areas (e.g., limb muscles), thus placing vital organs such as the newly revascularized heart at an increased risk for ischemia.

b. Pressure-Flow Uncoupling: The relationship between pressure and flow is greatly altered during cardiopulmonary bypass. Classically, pressure (P) is directly proportional to flow (Q) times resistance (R).

$$P = Q \times R$$

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Translating into clinical terms using perfusion pressure (PP), cardiac output (CO), and total peripheral resistance (TPR), the following relationship applies:

$$PP = CO \times TPR$$

Furthermore, total peripheral resistance is dependent on the systemic vascular resistance (SVR) and viscosity (V), leading to the following equation:

$$PP = CO \times (SVR \times V)$$

Changes in blood viscosity secondary to acute hemodilution are largely responsible for the uncoupling of the pressure-flow relationship, especially with crystalloid primes. Hemodilution decreases blood viscosity and enhances microcirculatory perfusion, even though perfusion pressure falls. Changes in viscosity secondary to hemodilution allow adequate organ perfusion during CPB to occur at significantly lower pressures, so pressure during CPB is no longer an adequate indicator of flow and is not associated with organ hypoperfusion.

c. Hemodilution

Hemodilution during CPB has several beneficial effects in addition to improving microcirculatory flow. The use of crystalloid primes was a great advancement in cardiac surgery which helped to conserve scarce blood bank resources. Crystalloid primes also made it possible for Jehovah's Witness patients to undergo cardiopulmonary bypass for cardiac surgery.

Hemodilution decreases the interaction between formed blood elements, thus decreasing the formation of cellular rouleaux which leads to enhanced perfusion through changes of decreased viscosity. Since CPB often involves some degree of hypothermia (18-32°C), patients often have increased blood viscosity and increased peripheral resistance which may act to decrease cardiac output and perfusion; however, these changes are counteracted by the effects of hemodilution.

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In addition to cardiac output, oxygen delivery depends largely on the amount of oxygen carried by hemoglobin and less on the amount of dissolved oxygen present in blood. Therefore, creating excessive hemodilution may be disadvantageous, overwhelming the effects of decreased viscosity and increased microcirculatory perfusion. Clinically, hematocrits as low as 15% are well tolerated, but acceptable limits vary from institution to institution.

Generally, the hematocrit is kept less than 30% if the patient's temperature will be kept at less than 30°C. For greater degrees of hypothermia (temperatures less than 25°C), the hematocrit is usually kept less than 25%. Separation from CPB usually takes place with hematocrits in the 20% range with transfusion of pump volume to the patient post-bypass to help correct anemia and hypovolemia.

d. Neuropsychiatric Changes

It is known that the majority of neurologic complications associated with CPB and cardiac surgery are related to embolic events. These emboli may be composed of air, atheromatous debris, calcium, or intracardiac and intramural thrombi. Dislodgement of these emboli may occur at any time, but especially during cannulation and clamping/declamping of the aorta, and patients are at an increased risk of experiencing sequelae from emboli during open-heart procedures such as valve replacements and the implantation of ventricular assist devices (VADs). Meticulous cannulation of the aorta with the use of aortic palpation and perhaps epiaortic scanning, in addition to adequate de-airing of the heart at the end of CPB are important in helping to minimize neurologic complications.

The brain has the ability to autoregulate, maintaining a consistent level of cerebral blood flow (50 ml/min/100g tissue) over a range of mean pressures. Furthermore, there exists a direct relationship between CMRO₂ (cerebral metabolic oxygen consumption) and CBF (cerebral blood flow) known as flow-

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metabolism coupling. However, both variables in this relationship can be altered during cardiac anesthesia and cardiopulmonary bypass. During hypothermic CPB, CMRO₂ is decreased and when utilizing pH-stat acid/base management, there exists a condition of "luxury perfusion" to the brain related to cerebral vasodilatation and increased CBF as a result of higher partial pressures of carbon dioxide in arterial blood as opposed to patients being managed with alpha-stat acid/base strategies. The improved cerebral blood flow with pH-stat management is accompanied by a theoretically increased risk of cerebral emboli and the potential for intracerebral steal, but this has never been adequately tested. During pH-stat management, the relationship between flow and metabolism is uncoupled. Alpha-stat supporters argue that maintaining the physiologic relationship between CBF and CMRO₂ is more homeostatic and advantageous over "luxury perfusion"; however, the debate continues.

Neuropsychiatric changes post CPB are quite common. Patients may experience short-term memory loss, exhibit episodes of confusion, and demonstrate a decreased ability to perform analytical tasks. A classic example is the patient who is unable to complete the New York Times crossword puzzle as efficiently as he could pre-operatively after undergoing coronary bypass surgery with CPB. Fortunately, the rate of severe neurologic complications related to cardiac surgery and CPB is around 1%, with the overall rate of neuropsychiatric changes being 6-10%.

e. Renal Dysfunction

Renal failure related to cardiac surgery and CPB can greatly impact a patient's hospital course. There exists a high association between renal failure and hospital mortality. In the days of early CPB, renal function on bypass was correlated with urine output. However, with modern CPB and hemodilution providing increased flow to the kidneys, the relationship

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between post-op renal function and urine output on CPB no longer exists.

Probably the greatest predictor of post-op renal dysfunction is the presence of pre-op renal dysfunction and low cardiac output syndrome requiring inotropic and vasopressor support. Several maneuvers can be used intra-operatively in an attempt to minimize the risk of developing renal dysfunction and failure.

Mannitol can be administered in a dose of 0.5-1.0 g/kg to act as an osmotic diuretic. It can be added to the pump prime, and it helps to maintain an osmotic diuresis during CPB. There also is evidence that mannitol acts as a free radical scavenger which may help to preserve renal function and lessen injury during times of low flow and ischemia. However, renal protective effects have never been shown in human clinical studies.

Glucose primes are another method of effecting an osmotic diuresis. Hyperglycemia creates hyperosmolarity helping to maintain flow to the kidneys. The benefits of a glucose-containing prime such as 5% Dextrose in Lactated Ringer's has been well described (Metz and Keats). Patients receiving glucose primes also retain less fluid perioperatively due to less third-spacing of crystalloid.

The use of glucose primes has been challenged. There exists a supposed association between hyperglycemia and insult to neurologic injury; however, our outcomes and data at The Texas Heart Institute do not support this theory. 5% Dextrose in Lactated Ringer's is our standard pump prime.

Diuretics such as furosemide and bumex can help to maintain non-homeostatic urine output; however, their use is not proven. Theoretically, using a loop diuretic like furosemide will decrease oxygen consumption at the level of the renal medulla by inhibiting ion exchange at the thick ascending limb and possibly lessen the risk of renal ischemia and damage during periods of low flow.

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Renal dose dopamine (3-5 mcg/kg/min) is yet another strategy to help maintain urine output and hopefully reduce the incidence of renal dysfunction.

g. Dysrhythmias

Dysrhythmias are quite common intraoperatively during cardiac surgery and are usually due to surgical manipulation. Sinus bradycardia and ventricular fibrillation (VF) are commonly seen during CPB while cooling just prior to the placement of the aortic cross clamp. Magnesium may be used intra-operatively to help decrease the incidence of supraventricular dysrhythmias.

The advanced cardiac life support protocols for dysrhythmia management apply in the operating room. However, the OR setting does provide some advantages. For example, while on CPB the cardiac output is maintained regardless of the type or severity of dysrhythmia. Furthermore, the surgeon and anesthesiologist have access to the myocardium allowing improved cardioversion through direct electroversion (usually 5-10 joules).

Atrioventricular pacing wires can easily be placed to help control rhythm disturbances. The use of intravenous amiodarone has also helped to improve dysrhythmia treatment. Amiodarone in doses up to 5 mg/kg can be used to control most rhythm problems, atrial and ventricular, while minimizing side effects from single IV dosing. Caution must be used, however, with respect to the development of complete heart block from excessive amiodarone acutely which may require pacing. Other complications from amiodarone administration include impaired sinus node function, profound vasodilatation, and myocardial depression.

5. Hemodynamic changes

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a. Blood Pressure Control: Evaluating and managing a patient's blood pressure in the perioperative setting needs to be systematic and efficient. As previously stated, the components of perfusion pressure (PP) are cardiac output (CO) and total peripheral resistance (TPR).

$$PP = CO \times TPR$$

Cardiac output is further composed of heart rate (HR) and stroke volume (SV), thus, leading to the following relationship:

$$PP = [HR \times SV] \times TPR$$

The stroke volume is also based on the difference between the end-diastolic volume (EDV) and the end-systolic volume (ESV). This leads to the following equation:

$PP = [HR \times (EDV - ESV)] \times TPR$ By individually evaluating each of the above parameters, causes of hypertension and hypotension can be recognized and treated.

b. Heart Rate and Rhythm Disturbances: Sinus rhythm is the rhythm of choice with respect to cardiac performance and ventricular filling. Electroversion or pharmacologic intervention with pacing may often times be necessary to maintain NSR. Bradycardia can be treated with pacing and the administration of anticholinergics or catecholamines. Atropine (10-40 mcg/kg) and glycopyrrolate (10-20 mcg/kg) are two popular anticholinergics, but they are usually not effective post bypass. Catecholamines such as epinephrine, norepinephrine, isoproterenol, and ephedrine may also be used to treat symptomatic bradycardia. Sinus bradycardia with an adequate stroke volume may respond to anticholinergics; however, bradycardic patients with depressed ventricular performance should likely receive a catecholamine such as epinephrine. More serious bradycardias (e.g., ventricular escape or idioventricular rhythms) will require more aggressive therapy.

Perioperative tachycardia responds well to electroversion and pharmacologic therapy. Post-electroversion pacing can also help to control dysrhythmias. In the absence of electrolyte

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disturbances, new onset atrial flutter and atrial fibrillation are receptive to synchronized cardioversion. Beta-blockers (e.g. metoprolol and propranolol), calcium channel blockers (e.g. verapamil and diltiazem), and digoxin can also be used to help control the rate. Ventricular tachycardia can be treated with magnesium, lidocaine, bretylium, procainamide, and direct-current cardioversion. The treatment of ventricular fibrillation is immediate asynchronous cardioversion.

Often times after initial rhythm correction maneuvers, intravenous medications such as lidocaine (1-3 mg/kg), procainamide (250-500 mg load then 15-60 mcg/kg/min), bretylium (5-10 mg/kg), and amiodarone (1-5 mg/kg) may need to be started to help suppress further rhythm disturbances, and infusions of these medications are often necessary.

c. Stroke Volume: The stroke volume portion of blood pressure analysis involves assessing ventricular performance and in the open chest, the left ventricle is difficult to visualize. Methods of evaluating its function include the use of TEE and pulmonary artery catheters with continuous or thermodilution cardiac output monitoring. Low cardiac output syndrome (LCOS) secondary to stroke volume problems can be remedied in several ways. Since $SV = EDV - ESV$, one can improve on the EDV side of the equation with a fluid challenge using crystalloid or colloid. If the patient is anemic or coagulopathic, blood component therapy may be used. The practitioner can also have the patient eject to a lower ESV by improving the patient's myocardial contractility through the use of an inotrope. Inotropic support may include the use of epinephrine (.01-0.5 mcg/kg/min), norepinephrine (.01-0.5 mcg/kg/min), dobutamine (5-10 mcg/kg/min), dopamine (3-20 mcg/kg/min), and milrinone (0.3-0.75 mcg/kg/min). The chosen inotropes depend on the type of ventricular dysfunction and practitioner preferences. The early use of intra-aortic balloon counterpulsation may also be of great benefit.

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d. Total Peripheral Resistance: Multiple changes in the TPR occur during CPB and cardiac surgery. Initial cooling during CPB causes an increase in the TPR, while rewarming causes vasodilatation and a drop in the TPR. Hemodilution also causes a decrease in the TPR secondary to changes in viscosity.

It is important to remember that patients are often vasodilated from rewarming when it is time to separate from cardiopulmonary bypass. Thus, upon separation, the perceived cardiac output may be normal whereas the mean blood pressure may be low. To counteract these changes in SVR, calcium chloride can be given (10 mg/kg) to help increase SVR through vasoconstriction. Calcium may also improve myocardial contractility, especially in pediatric CV patients, during states of ionized hypocalcemia, and its administration helps to protect myocardial function from the effects of hyperkalemia from residual cardioplegia. Other alpha-agonists such as phenylephrine may be used in doses of 0.25-0.75 mcg/kg/min to help maintain SVR. However, caution must be used to make sure that detrimental increases in SVR do not significantly decrease cardiac output because of increased afterload changes. Vasopressor therapy must be closely monitored since mean arterial pressure may appear acceptable as the cardiac output progressively declines.

6. Conclusion: Caring for today's cardiac surgical patient is not only safer than before, but it is also more complex. Medical and surgical advancements have led to new medications and monitors to help care for our patients. The cardiac anesthesiologist must stay focused on the priority of patient care while balancing the input of all surrounding devices and equipment.

Many complications experienced by cardiac patients are a result of cardiopulmonary bypass. It is imperative to understand the physiology of CPB to help aid in-patient care. Cardiac

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patients are subjected to a myriad of procedures such as invasive lines, vascular cannulations, and aortic clamping and declamping that are not routinely experienced by the general surgery patient population. Therefore, the caregiver must be prepared to handle these specific complications in a variety of situations.

An attentive anesthesiologist is an integral part of the cardiac surgical team responsible for taking patients through today's complex cardiac surgeries, and further advancements in research and patient care will hopefully continue to improve patient outcome.