Cardiopulmonary bypass: Management

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INTRODUCTION — Cardiopulmonary bypass (CPB) is a form of extracorporeal circulation in which the patient's blood is diverted from the heart and lungs and rerouted outside of the body. The normal physiologic functions of the heart and lungs, including circulation of blood, oxygenation, and ventilation, are temporarily taken over by the CPB machine. Typically, cardioplegia solution is administered to allow the cardiac surgeon to operate on a nonbeating heart in a field largely devoid of blood, while other end organs remain adequately oxygenated and perfused.

This topic will discuss management of CPB. Preparations for initiation of CPB are discussed separately. (See "Cardiopulmonary bypass: Preparations and initiation".)

The process of weaning from CPB and common problems encountered in the immediate postbypass period are addressed separately. (See "Weaning from cardiopulmonary bypass" and "Management of problems after cardiopulmonary bypass".)

GENERAL PRINCIPLES

Equipment and physiology — Components of the CPB machine include pumps, tubing, and gas (oxygenator) and heat exchange units (figure 1A) [1]. Modern CPB machines are also equipped with systems that continuously monitor line or circuit pressure, temperature, and blood parameters (eg, oxygen saturation, blood gases, hemoglobin [Hgb], potassium), as well as safety features such as air and fluid level detection systems and a blood filter in the arterial line.

During CPB, venous blood is drained from the right atrium (RA; or both superior and inferior vena cavae) and is diverted through the venous line of the CPB circuit into a venous reservoir (figure 1A-B). CPB machines are typically equipped with vacuum-assisted technology that facilitates drainage to maintain a bloodless surgical field and allow use of smaller venous cannulae and reduced CPB circuit volumes. The arterial pump functions as an artificial heart by withdrawing blood from this reservoir and propelling it through a heat exchanger, an artificial lung (oxygenator or gas exchanger), and finally an arterial line filter. The blood is then returned to the patient via an arterial cannula positioned in the ascending aorta or other major artery. Additional CPB circuit pumps or other components are employed as needed to suction blood from the surgical field, deliver cardioplegia solution to

Contact of blood with nonendothelial surfaces of the CPB circuit induces an intense inflammatory response [1,2]. This results in platelet activation, initiation of the coagulation cascade, and decreased levels of circulating coagulation factors. Endothelial cells and leukocytes are activated, releasing mediators that may contribute to capillary leakage and tissue edema. Many of the challenges encountered during weaning from CPB and the postbypass period (eg, myocardial dysfunction, vasodilation, bleeding) are thought to be consequences of this inflammatory sequence [3-5]. Also, the priming solution for the CPB circuit (typically 1 to 2 L of a balanced crystalloid solution) results in hemodilution with temporary or persistent anemia and coagulopathy.

Protocol — Surgical procedures requiring CPB follow a predictable sequence of events that includes priming and testing of the CPB circuit, anticoagulation, vascular cannulation, initiation and maintenance of CPB, myocardial arrest with myocardial protection, myocardial reperfusion, and finally weaning and termination of CPB.

Established protocols for management of CPB use parameters approximating normal physiology (table 1) [6,7]:

- In adults, the target flow rate during CPB is 2.2 to 2.4 L/min/m^2 in normothermic patients to approximate a normal cardiac index; cardiac index is appropriately decreased if hypothermia is induced [6].
- Mean arterial pressure (MAP) is generally targeted at ≥65 mmHg, but the target may be higher in older patients and those with cerebrovascular disease [6-9]. MAP should not exceed 100 mmHg.
- Adequacy of end-organ perfusion is determined by arterial blood gas analysis [6,7] and the mixed venous oxygen saturation (SvO_2), which is continuously monitored and maintained ≥75 percent throughout CPB. Arterial blood gases, base deficit, and lactate levels are intermittently checked (approximately every 30 minutes).
- Preparations for weaning from CPB and checklists to ensure readiness for weaning (table 2) are described separately. (See "Weaning from cardiopulmonary bypass", section on 'Preparation for weaning' and "Weaning from cardiopulmonary bypass", section on 'The weaning process'.)

MANAGEMENT OF CARDIOPULMONARY BYPASS — Goals during CPB include maintenance of general anesthesia, anticoagulation, and parameters that approximate normal physiology for optimal end-organ function (table 1) [6,7].

Oxygenation, ventilation, and arterial blood gases — Arterial pO_2 is maintained at 150 to 250 mmHg during CPB [6,7]. A continuous arterial blood parameter monitoring system is located in the arterial line of the CPB circuit, and a continuous venous oximeter is located in the venous return line. Arterial blood gas values are checked in the laboratory or by point-of-care testing approximately every 30 minutes, which also allows intermittent recalibration of the continuous blood gas monitor in the arterial line.

Alpha-stat management of arterial blood gases without temperature correction is employed to maintain a normal range for pCO_2 (35 to 45 mmHg [4.7 to 6 kPa]) and pH (7.35 to 7.45) [6,7,10]. Maintaining $PaCO_2$ and pH within this physiologic range during CPB is important to preserve cerebral autoregulation because hypocarbia decreases cerebral blood flow. (See "Anesthesia for aortic surgery requiring deep hypothermia", section on 'Acid–base management'.)

Ventilation of the lungs during CPB has not been demonstrated to improve pulmonary function and may increase technical difficulty for the surgeon [11-13]. Some clinicians use continuous positive airway pressure (CPAP)

Pump flow and mixed venous oxygen saturation — CPB flow rates are set at 2.2 to 2.4 L/min per m^2 in a normothermic patient to provide adequate blood flow for optimal perfusion of the brain and other end organs. These rates may be slightly decreased if hypothermia is employed.

Acute decreases in mean arterial pressure (MAP) or increases in central venous pressure (CVP) may indicate acute reduction in venous return due to the surgeon lifting the heart (causing a reduction in flow), a malpositioned or kinked arterial or venous cannula, or obstruction to blood flow by an air lock. With severe reduction in venous return, the perfusionist may need to administer volume into the CPB reservoir or to reduce arterial flow. Persistent reductions in arterial line flow and/or venous return must be urgently addressed by identifying and correcting the cause.

Mixed venous oxygen saturation (SvO_2) is maintained ≥75 percent throughout CPB as a monitor of adequacy of peripheral perfusion. Persistent SvO_2 values <75 percent may indicate inadequate oxygen delivery and are associated with worse outcomes including postoperative delirium and decreased long-term survival [14,15]. Also, lactate values and base deficit are measured when arterial blood gases are obtained approximately every 30 minutes. Although absolute lactate values are multifactorial, rising levels during CPB represent anaerobic metabolism at the cellular level due to inadequate tissue oxygen delivery, and may reflect hypoperfusion during CPB, particularly if sustained or associated with low mixed venous oxygen saturation values (SvO_2 <70%) [16-18].

Treatment for SvO_2 <75 percent, base deficit lower than -5, or lactate level >4 mEq/L is directed at increasing the CPB flow rate, as well as assuring that arterial blood gases and hemoglobin (Hgb) levels are adequate. It is common practice to administer sodium bicarbonate for base deficit lower than -5, or lactate level >4 mEq/L, but excessive sodium bicarbonate administration can cause postoperative hypernatremia [18,19]. Although a rising lactate level during CPB is most often the result of inadequate tissue perfusion, persistent lactic acidosis in the postoperative period may occur due to other factors (eg, the beta adrenergic metabolic effects of epinephrine infusion) [20].

Mean arterial pressure — In the context of acceptable CPB flow rates, MAP is generally targeted at ≥65 mmHg; a higher target may be selected in older patients and those with cerebrovascular disease [6-9]. MAP should not exceed 100 mmHg. In most patients, this MAP range permits autoregulation of cerebral and other end-organ circulation throughout CPB, although it is not possible to determine a precise autoregulatory range for each individual.

Hypotension

- **Moderate hypotension** – In the context of acceptable CPB flow rates, if MAP falls below the target range, the perfusionist may increase the pump flow (equivalent to increasing cardiac output), particularly if it is <2.4 L/minute/m^2. If hypotension persists after increasing pump flow, a vasopressor can be administered as an intravenous (IV) bolus or via continuous infusion. In many institutions, small bolus doses of phenylephrine (eg, 40 to 100 mcg) are administered directly into the CPB reservoir to treat hypotension. Infusions of phenylephrine at 10 to 200 mcg/minute, vasopressin at 0.04 units/minute, or norepinephrine at 0.02 to 0.06 mcg/kg/minute are also commonly employed (table 3). However, there is no evidence that administration of vasopressor therapy during CPB in an attempt to increase MAP to near physiological values can affect the volume or number of new cerebral infarcts [21,22]. During attempts to increase MAP, clear communication is necessary between the anesthesiologist (who may be adjusting the patient's systemic vascular resistance

vascular resistance (SVR) and low MAP during and after CPB, occurs in 5 to 25 percent of patients undergoing cardiac surgery [23-26]. Risk factors include preoperative use of agents such as angiotensin-converting enzyme (ACE) inhibitors, heparin, or calcium channel blockers, as well as prebypass hemodynamic instability [23,24,26,27]. (See "Postoperative complications among patients undergoing cardiac surgery", section on 'Vasodilatory shock' and "Management of problems after cardiopulmonary bypass", section on 'Vasoplegia'.)

Prior to treating low blood pressure near the end of the period on CPB, it is important to verify that the radial arterial pressure is not markedly underestimating the central aortic pressure [28-30]. A significant central to peripheral pressure gradient associated with rewarming at the end of CPB is often present during cardiac surgery (figure 2). Connection of a pressure transducer to the side port of the aortic cannula after termination of CPB, or use of a femoral intra-arterial catheter inserted by the surgeon on the field will typically provide an accurate estimate of the true central aortic pressure.

If hypotension due to vasoplegia (low SVR) is confirmed, it is usually necessary to administer a continuous vasopressor infusion. Vasopressin at 0.04 units/minute IV is often effective since low circulating levels of circulating vasopressin are associated with vasoplegia [25,31]. Alternatives or adjuncts to vasopressin include norepinephrine at 0.02 to 0.06 mcg/kg/minute or phenylephrine at 10 to 200 mcg/minute (table 3). Although no optimal vasopressor approach has been established, observations in the setting of distributive shock suggest that combining vasopressin with catecholamines is associated with lower rates of atrial fibrillation compared to catecholamines alone [32]. If these agents are ineffective, methylene blue 1 to 2 mg/kg IV over 20 minutes may be administered to reduce resistance vessel responsiveness to nitric oxide. Notably, methylene blue administration should be avoided for patients receiving chronic serotonergic therapy (eg, fluoxetine) due to the risk of serotonin syndrome [33], and may interfere with monitors that employ oximetry to measure oxygen saturation (eg, pulse oximetry and cerebral oximetry) [24,34].

Hypertension — If MAP increases to >90 mmHg during CPB, treatment includes increasing volatile anesthetic concentration administered via the CPB circuit and/or administering additional IV anesthetic. Occasionally, administration of a vasodilator may be necessary (table 3). For brief periods, pump flow may be reduced while these pharmacologic interventions take effect.

Maintenance of anticoagulation — Adequacy of heparin anticoagulation is measured with point-of-care tests such as activated whole blood clotting time (ACT) every 30 minutes to maintain a targeted value throughout CPB (typically above 480 seconds) [35]. If available, plasma heparin concentrations may also be determined by point-of care assays such as the Hepcon, with target heparin concentration ≥4 units/mL [35,36]. Protocols in some institutions emphasize treatment of heparin concentrations <4 units/mL, even if ACT values are adequate.

Anticoagulation in patients with heparin-induced thrombocytopenia (HIT) is discussed separately. (See "Cardiopulmonary bypass: Preparations and initiation", section on 'Heparin-induced thrombocytopenia (HIT)'.)

Maintenance of anesthesia and neuromuscular blockade

Anesthetic agents — Inadequate anesthetic depth is treated by increasing the volatile anesthetic concentration administered via the CPB circuit or by administering additional IV anesthetic agents [37].

Deliberate hypothermia during CPB decreases anesthetic requirement [38-40]. During rewarming, the dose of volatile anesthetic agent should be increased or additional IV agents should be administered to avoid inadequate anesthesia (see 'Temperature' below). Use of a benzodiazepine may reduce risk of awareness during rewarming,

It is reasonable to monitor processed electroencephalogram (EEG) indices (eg, bispectral index) or the unprocessed EEG to provide data that may detect inadequate anesthesia during CPB [1,38,39,42-45]. Some institutions measure concentration of the volatile anesthetic agent in the expiratory gas of the oxygenator [1,38,39]. Despite use of these monitoring techniques, anesthesia awareness may still occur during cardiac surgery [46]. (See "Awareness with recall following general anesthesia", section on 'Monitoring'.)

Neuromuscular blocking agents — Decreased dosing of the selected neuromuscular blocking agent (NMBA) may be adequate during the hypothermic period of CPB because hypothermia directly reduces muscle strength (up to 10 percent per degree Celsius) and enhances NMBA action [1,47]. However, additional NMBA is typically required during rewarming.

Neuromuscular function can be assessed with a peripheral nerve stimulator (PNS) throughout CPB to maintain an appropriate degree of neuromuscular blockade [1]. The PNS electrodes are placed along the course of facial nerve supplying the orbicularis oculi muscle. Since movement of the diaphragm or other slight movement provides an indication of inadequate anesthetic depth, complete paralysis during CPB is avoided in some institutions. Complete neuromuscular blockade with absence of twitches on the PNS may increase risk of awareness. (See "Awareness with recall following general anesthesia", section on 'Neuromuscular blockade'.)

Temperature — Mild (32 to 35°C), moderate (28 to 32°C), or deep (<28°C) hypothermia is used as a protective strategy for the brain and vital organs during CPB for many cardiac surgical procedures [48-50]. The cerebral metabolic rate of oxygen consumption ($CMRO_2$) decreases approximately 7 percent per degree Celsius reduction in temperature [51]. Mild hypothermia (approximately 34°C) is typically selected for coronary artery bypass grafting (CABG) surgery [52]. Moderate hypothermic temperatures may be selected for cardiac valve repair or replacement surgery due to the length and complexity of these procedure. Moderate reductions in temperature confer the same neuroprotective benefits as deeper levels of hypothermia during focal ischemia [50,53]. For procedures requiring a temporary period of elective circulatory arrest (eg, repair of portions of the ascending aorta or aortic arch), a deep hypothermic temperature may be selected to achieve EEG isoelectricity. (See "Anesthesia for aortic surgery requiring deep hypothermia", section on 'Cooling and deep hypothermia'.)

Management during cooling and hypothermia

- **Management during cooling** – During cooling, the temperature gradient between the venous inflow and arterial outlet on the oxygenator is maintained at <10°C, similar to the recommendations of the Society of Thoracic Surgeons (STS), Society of Cardiovascular Anesthesiologists (SCA), and American Society of ExtraCorporeal Technology (AmSECT) [48,49]. Any peripheral body warming devices (eg, forced-air warming blankets, insulation water mattresses, devices for warming IV fluids) are turned off during cooling and hypothermia.

- **Management during hypothermia** – During hypothermic CPB, the oxygenator arterial outlet temperature should be used as the best measure of cerebral temperature [49]. However, other temperature sites that are also monitored include the oxygenator venous inlet and specific sites in the patient's body (eg, nasopharyngeal, bladder [or the rectal site for patients who do not make urine], pulmonary arterial blood temperature if a pulmonary artery catheter [PAC] is in place). Predictable discrepancies between temperatures measured at each site reflect differences in perfusion. For example, the arterial inlet temperature decreases first during active cooling, followed by the nasopharyngeal temperature, while bladder temperature is usually the slowest to change.

gradient between the venous inflow and arterial outlet on the oxygenator is maintained at ≤4°C. This slow rewarming may require 60 to 90 minutes or more in a moderately or deeply hypothermic patient [49,54,55]. (See "Anesthesia for aortic surgery requiring deep hypothermia", section on 'Rewarming strategies'.)

The final target temperature for separation for CPB is 37°C at the nasopharyngeal site, and hyperthermia is to be avoided. The nasopharyngeal temperature site serves as the in vivo monitor of brain temperature and is typically higher than other sites due to the proximity of the aortic cannula to the great vessels and head. However, all monitored temperature sites may underestimate the true cerebral temperature during rewarming [56]. To minimize risk of cerebral and systemic hyperthermia, which is associated with worsened neurologic and neurocognitive outcomes [48,49,54,55,57], acute kidney injury (AKI) [58], and mediastinitis [59], the arterial outlet temperature should never exceed 37°C.

The oxygenator venous blood inflow temperature typically lags behind the arterial outlet and nasopharyngeal temperatures. Target temperature will be achieved more rapidly at highly perfused sites (eg, nasopharyngeal tissue) that receive the majority of the systemic blood flow throughout rewarming. The peripheral ("core" or "shell") sites required a considerably longer period to equilibrate. For example, the bladder temperature site (which is used to estimate "core temperature" in most cases) will only be approximately 35.5°C when nasopharyngeal temperature is stable at 37°C.

- **Management during weaning from CPB** – The nasopharyngeal and/or PAC sites are used for temperature monitoring during weaning from CPB and in the immediate postbypass period. The temperature gradient between these highly perfused sites and the periphery (eg, the bladder site) produces volume and heat redistribution. Thus, mild systemic hypothermia may develop before the patient is admitted to the intensive care unit (ICU). Depending on the urine output (UO), bladder temperature may remain a poor indicator of core temperature until complete equilibration of tissue temperature has occurred, typically several hours after CPB [60].

Urine output — Maintenance of renal blood flow is achieved by maintaining adequate CPB pump flow throughout CPB to minimize the risk of AKI (see 'Renal insufficiency or failure' below). Although mannitol 1 gram/kg is often added into the CPB circuit priming solution, there are no data supporting the efficacy of pharmacologic agents (eg, mannitol, furosemide, or low "renal dose" dopamine infusion) for prevention of AKI after CPB [61].

If oliguria develops during CPB (ie, urine output <0.5 mL/kg per hour), we check the bladder catheter for kinking or disconnection, as well as checking the bladder itself by palpation or ultrasound performed on the surgical field. Aortic dissection as a possible cause of oliguria is ruled out with transesophageal echocardiography (TEE). Adequacy of pump flow, MAP, SvO_2, and arterial blood gases are checked and closely monitored (table 1).

Hemoglobin/hematocrit — For Hgb <7.5 g/dL (or hematocrit [Hct] <22 percent), initial treatment during CPB is removal of fluid by ultrafiltration (hemoconcentration) when possible [62]. Transfusion of packed red blood cells (RBCs) is reasonable if Hgb remains <7.5 g/dL when hemoconcentration is not possible or is ineffective [63]. However, transfusion decisions are individualized, taking into account patient-related factors (eg, age, severity of illness, cardiac function, risk for critical end-organ ischemia), the clinical setting (massive or active blood loss, the operation being performed), and clinical or laboratory parameters indicating hypoperfusion (eg, metabolic acidosis, lactate acidosis, SvO_2 <60 percent) [64,65]. When RBC transfusion is necessary, leukocyte-reduced blood is preferred [66]. (See "Indications and hemoglobin thresholds for red blood cell transfusion in the adult", section on 'Cardiac surgery'.)

cardiac surgical patients at moderate-to-high risk for death, no benefit was demonstrated for a composite outcome of death, myocardial infarction, stroke, or new-onset renal failure with a liberal transfusion strategy in the operating room and postoperative intensive care unit (Hgb trigger set at <9.5 g/dL), compared with a restrictive transfusion strategy (Hgb trigger set at <7.5 g/dL) [63]. In the restrictive group, fewer patients received any RBC transfusion (52 versus 73 percent; odds ratio [OR] 0.41, 95% CI 0.37-0.47), and fewer RBC units were transfused (two versus three units; rate ratio [RR] 0.85, 95% CI 0.82-0.88). Similar results have been noted in other trials [72,73].

Glucose — We agree with the STS guidelines, which recommend maintaining blood glucose levels <180 mg/dL (10 mmol/L) during CPB and the postbypass period, with a single dose or intermittent doses of insulin if effective or with a continuous insulin drip if glucose levels are persistently elevated (>180 mg/dL [10 mmol/L]) [7,74]. Blood glucose and potassium are monitored frequently (approximately every 30 minutes) to prevent hypoglycemia and hypokalemia in response to treatment. (See "Glycemic control and intensive insulin therapy in critical illness", section on 'Surgical patients'.)

Electrolytes — Metabolic abnormalities (eg, hyperglycemia, hypocalcemia, hyperkalemia, hypokalemia, hypomagnesemia) are common during CPB. Frequent laboratory testing during CPB (approximately every 30 minutes) is helpful for recognition and treatment. Management of persistent abnormalities during the postbypass period is addressed separately. (See "Management of problems after cardiopulmonary bypass", section on 'Metabolic abnormalities'.)

Hypocalcemia — Hypocalcemia (measured as the ionized fraction of total calcium concentration) is common during CPB and is typically corrected (eg, with administration of calcium chloride 5 to 10 mg/kg IV). Administration of calcium is avoided while the aortic cross-clamp is in place and for at least 10 to 15 minutes after removal of the aortic crossclamp, thus allowing a period of myocardial reperfusion [75]. (See "Management of problems after cardiopulmonary bypass", section on 'Hypocalcemia'.)

Hyperkalemia — Transient hyperkalemia is common during CPB, particularly just after administration of high-potassium cardioplegia solution (ie, before systemic redistribution of cardioplegia solution has occurred).

Persistent hyperkalemia during CPB may be managed by administration of combinations of glucose and insulin, or the perfusionist may employ zero-balance ultrafiltration (Z-BUF), which allows removal of potassium from the blood [76,77]. As plasma water is removed, an equal amount of buffered potassium-free solution is added. Normal saline solution is typically used, but monitoring to avoid hypernatremia or hyperchloremia is necessary. Alternative treatments include administration of furosemide to eliminate potassium via diuresis or administration of calcium chloride (which is more typically administered to treat hyperkalemia during the postbypass period). (See "Treatment and prevention of hyperkalemia in adults", section on 'Patients with a hyperkalemic emergency'.)

Hypokalemia — Hypokalemia is uncommon during CPB but may be treated if present after cross-clamp removal. Typically, potassium chloride is administered in increments of 10 to 20 mEq IV by slow infusion over 30 to 60 minutes. Further management after weaning from CPB is discussed separately. (See "Management of problems after cardiopulmonary bypass", section on 'Hypokalemia'.)

Hypomagnesemia — Studies have demonstrated that hypomagnesemia is common during and after CPB due to diuresis and hemodilution with magnesium-free fluids during CPB [78]. Postoperative hypomagnesemia is associated with dysrhythmias, myocardial ischemia, and ventricular dysfunction [78,79]. Thus, magnesium

block, ventricular fibrillation, junctional rhythm), although normal sinus rhythm is eventually restored in most patients who had a normal preoperative rhythm. A lidocaine bolus of 100 mg is often administered immediately prior to aortic cross-clamp removal to decrease the risk of ventricular fibrillation [80-82]. Temporary external cardiac pacing may be necessary for patients with heart block, intraventricular conduction delay, or bradycardia until recovery to normal sinus rhythm.

Defibrillation with internal paddles applied directly to the heart using 10 to 20 joules is usually effective for the treatment of ventricular fibrillation immediately after aortic cross-clamp removal if blood temperature is >30°C, MAP is adequate, and there are no significant electrolyte abnormalities such as hyperkalemia. For persistent or recurrent ventricular fibrillation, it is important to identify and treat potential underlying causes (eg, hypokalemia, hypomagnesemia, compromised coronary graft anastomosis with inadequate coronary flow, air embolism into a coronary artery, left ventricular [LV] distention, hypothermia). After correcting potential causes, persistent ventricular arrhythmias are typically treated with boluses of antiarrhythmic drugs such as lidocaine (100 mg x 2) or amiodarone (300 mg). In some cases, a continuous infusion of amiodarone may be necessary, as described separately. (See "Amiodarone: Clinical uses", section on 'Clinical uses of amiodarone for ventricular arrhythmias'.)

MANAGEMENT OF SPECIAL POPULATIONS — Specific management strategies are employed during CPB for patients with aortic insufficiency, cerebrovascular disease, renal insufficiency, or vasoplegia and for those undergoing a period of elective deep hypothermic circulatory arrest (DHCA).

Aortic regurgitation — Preexisting aortic regurgitation (AR) can be diagnosed and its severity characterized with intraoperative transesophageal echocardiography (TEE) examination in the prebypass period (movie 1 and image 1 and image 2 and image 3). During CPB, the presence of AR may limit the effectiveness of antegrade delivery of cardioplegia solution into the coronary artery ostia after the ascending aorta is cross-clamped (figure 1B). Much of the cardioplegia solution will regurgitate back through the incompetent aortic valve into the left ventricle (LV). The severity of AR may be influenced by the increased aortic root pressure that occurs during attempted delivery of antegrade cardioplegia and surgical manipulations that further distort the normal geometry of the aortic root and valve. (See "Cardiopulmonary bypass: Preparations and initiation", section on 'Aortic cross-clamping and antegrade cardioplegia administration'.)

Also, the cardioplegia solution flowing back across the incompetent aortic valve can cause LV distention once the ventricle is not ejecting regularly due to bradycardia, asystole, or ventricular fibrillation. Distention causes increased LV wall tension. In combination with inadequate coronary delivery of antegrade cardioplegia solution, this may result in inadequate myocardial protection and severe postbypass LV dysfunction. In this situation, an LV vent is placed by the surgeon to maintain the ventricle in a decompressed state (figure 1B). Correct placement of the vent and effective decompression of the LV are then confirmed with TEE examination. Subsequently, continuous monitoring of the TEE and pulmonary artery pressure (PAP) supplement surgical detection of a dislodged LV vent or recurrence of LV distention. (See "Cardiopulmonary bypass: Preparations and initiation", section on 'Left ventricular vent placement'.)

If antegrade cardioplegia delivery is inadequate due to AR, the cardiac surgeon typically minimizes attempted delivery by this route, opting instead for retrograde cardioplegia delivered through the coronary sinus (see "Cardiopulmonary bypass: Preparations and initiation", section on 'Retrograde cardioplegia administration'). In selected aortic valve or aortic root procedures, it may be necessary to deliver cardioplegia solution directly into the coronary ostia after cross-clamping the aorta and opening the aortic root.

directly to the heart to deliver 10 to 20 joules of electricity. Subsequently, if LV distention recurs after the aortic cross-clamp has been applied, an LV vent may be inserted. (See "Cardiopulmonary bypass: Preparations and initiation", section on 'Left ventricular vent placement'.)

During and after weaning from CPB, ventricular pacing may be necessary to achieve a faster heart rate in a patient who has residual AR. The faster rate minimizes regurgitant blood flow though the incompetent aortic valve. (See "Weaning from cardiopulmonary bypass", section on 'Maintenance of optimal pacemaker function'.)

Cerebrovascular disease — Considerations for patients with known cerebrovascular disease and/or evidence of severe aortic atherosclerosis include maintaining a higher mean arterial pressure than patients without these comorbidities, with fastidious attention to maintenance of the hemoglobin (Hgb) and hematocrit (Hct) levels, and avoidance of cerebral hyperthermia. The use of intraoperative near-infrared spectroscopy (NIRS) cerebral oximetry monitoring and maintenance of regional cerebral oxygen saturation (rSO_2) within 20 percent of baseline has been advocated for patients at high risk for adverse neurologic outcomes, including those with known cerebrovascular disease [83-90].

Other considerations for cardiac surgery in a patient with severe cerebrovascular disease are discussed separately. (See "Coronary artery bypass grafting in patients with cerebrovascular disease".)

- **Mean arterial pressure** – It is reasonable to maintain a mean arterial pressure (MAP) during CPB that is slightly higher than the target MAP (≥65 mmHg) for patients without likely cerebrovascular disease (table 1) [6,7,91,92]. However, the optimal target for MAP during CPB that may decrease risk of neurologic complications is unknown in patients with or without known cerebrovascular disease [21]. (See 'Mean arterial pressure' above.)

- **Hemoglobin/hematocrit** – It is reasonable to maintain Hgb ≥7.5 g/dL or Hct ≥22 percent, based on studies indicating that lower nadir Hgb or Hct values during CPB are associated with increased risk of stroke, particularly in patients with cerebrovascular disease [68,69,93]. However, red blood cell (RBC) transfusion is avoided when possible since transfusion is also associated with stroke after cardiac surgery [93]. (See 'Hemoglobin/hematocrit' above.)

- **Temperature** – Hyperthermia (brain temperature >37°C) during rewarming on CPB should be avoided because it may increase the risk for brain injury or worsen the severity of brain injury as a consequence of CPB. (See 'Temperature' above.)

- **Cerebral oximetry monitoring and interventions** – We try to mitigate acute decreases in regional cerebral oxygen saturation (rSO_2) when near-infrared spectroscopy (NIRS) is employed. Baseline preoperative rSO_2 varies considerably, with an overall pooled mean and standard deviation of 66.4 percent ± 7.8 percent according to a meta-analysis that included 953 patients undergoing cardiac surgery [90].

  During CPB, interventions are employed to treat acute decreases in rSO_2, when the decrease is more than 20 percent below the baseline value. Interventions include (algorithm 1) [83,85] (see "Anesthesia for aortic surgery requiring deep hypothermia", section on 'Cerebral oximetry'):

  - Increasing cardiac output by increasing pump flow
  - Increasing MAP by administering a vasopressor

- Decreasing the cerebral metabolic rate of oxygen consumption ($CMRO_2$) by deepening anesthesia
- Increasing blood oxygen-carrying capacity with RBC transfusion (particularly if Hgb is <7.5 g/dL)

While no studies have demonstrated that monitoring rSO_2 values can prevent stroke by increasing or restoring low rSO_2 values in patients with or without significant cerebrovascular disease [94-96], this technique may improve other neurologic outcomes. A meta-analysis of randomized trials noted that use of intraoperative NIRS in 314 patients undergoing cardiac surgery resulted in less postoperative cognitive dysfunction at one week compared with 297 patients who did not receive NIRS-guided interventions (risk ratio [RR] 0.55; 95% CI 0.36-0.86; four trials) [97].

Renal insufficiency or failure — Patients with dialysis-dependent end-stage renal disease (ESRD) typically undergo routine scheduled dialysis prior to elective cardiac surgery. For emergency surgery or if additional fluid removal is needed, hemoconcentration by ultrafiltration during CPB may be performed if necessary, or zero-balance ultrafiltration (Z-BUF) may be employed if toxin or drug removal is needed [98-104].

Other considerations for patients with renal insufficiency undergoing CPB include:

- **Antifibrinolytic administration** – The risk for seizures may be greater after administration of the antifibrinolytic agent tranexamic acid (TXA) in patients with ESRD or moderate to severe renal insufficiency, particularly if bolus and infusion dosing is in the upper range [105]. Thus, the lower range for TXA dosing should be used. (See "Cardiopulmonary bypass: Preparations and initiation", section on 'Antifibrinolytic administration'.)

- **Balanced salt solutions** – We do not use normal saline instead of a balanced salt solution (eg, lactated Ringer's, Normosol-R, or Plasma-Lyte A) in the pump prime for patients with ESRD because the amount of potassium in these balanced salt solutions is unlikely to contribute to hyperkalemia. Also, the hyperchloremia that may occur after administration of a large volume of normal saline has been associated with increased mortality and risk of renal dysfunction after noncardiac surgery [106,107].

- **Avoidance of hydroxyethyl starch** – Use of hydroxyethyl starch solutions is avoided due to concerns regarding acute kidney injury (AKI) [61,108,109]. Also, risk of bleeding may be increased with use of HES for pump prime and/or intraoperative fluid therapy compared with use of balanced salt solutions [109-111].

- **Pump flow** – In patients who have preexisting renal insufficiency who are not dialysis dependent, maintaining renal perfusion during CPB with adequate pump flow is the most effective method to prevent AKI (table 1) [112,113].

- **Temperature** – Hyperthermia (body temperature >37°C) is avoided during and after CPB because it may increase the risk of end-organ injury. In one retrospective study, both cumulative duration of arterial outlet temperature >37°C as well as elevated temperature upon arrival in the intensive care unit (ICU) were independent predictors of AKI [58]. (See 'Temperature' above.)

- **Hemoglobin/hematocrit** – It is reasonable to maintain Hgb ≥7.5 g/dL or Hct ≥22 percent in these patients because lower nadir Hgb or Hct values during CPB have been associated with increased risk of AKI [70,114-120]. However, RBC transfusion has also been associated with AKI after cardiac surgery [117,120,121]. Leukocyte filtration of transfused RBCs resulted in a fivefold reduction in the incidence of worsening postoperative renal function (from 7.5 to 1.1 percent) in a meta-analysis of six trials that included 374

Interventions with uncertain efficacy in preventing AKI include:

- **Mean arterial pressure** – In the setting of adequate pump flow, reductions in MAP below an individual patient's limit of cerebral autoregulation defined with NIRS cerebral oximetry monitoring during CPB were independently associated with AKI, although there was no association with reduction below any absolute MAP value [124]. Thus, it is reasonable to avoid hypotension in patients at risk for AKI. Regarding absolute MAP values, in one prospective study in patients with known risk factors for AKI, maintaining MAP ≥75 mmHg during CPB had no benefit on the incidence of postoperative AKI compared with a control group with MAP maintained at 50 to 60 mmHg [125]. Similarly, a retrospective study noted no increase in AKI incidence when low MAP and anemia were concurrent during CPB compared with anemia alone during CPB [114]. Other retrospective studies have found no association between AKI and low MAP during CPB (<50 mmHg) [115,126-129]. Notably, in all the above mentioned studies, perfusion targets during CPB were assumed to have been achieved (ie, hypotension was not a surrogate for low pump flow rates).

- **Remote ischemic preconditioning** – Remote ischemic preconditioning (using a cuff tourniquet to induce limb ischemia for several minutes) may reduce the incidence of AKI after cardiac surgery with CPB [130]. However, meta-analyses of randomized trials have not demonstrated important clinical benefits [131,132].

- **Anti-inflammatory strategies** – No specific agents that reduce inflammation (eg, steroids, statins), ischemia-reperfusion injury (eg, cyclosporine), or technical interventions for CPB circuits (eg, miniaturized circuits) have been found to be effective in reducing the incidence or severity of postoperative AKI [61,66,133,134].

Deep hypothermic circulatory arrest — Some surgical procedures require temporary interruption of cerebral and systemic blood flow (eg, repair of portions of the ascending aorta or aortic arch). Elective circulatory arrest is accomplished during a period of deep hypothermic circulatory arrest (DHCA) after cooling with the aid of CPB, typically to 16 to 18°C. Anesthetic management during and after DHCA is discussed separately. (See "Anesthesia for aortic surgery requiring deep hypothermia", section on 'Cardiopulmonary bypass with deep hypothermic circulatory arrest'.)

SUMMARY AND RECOMMENDATIONS

- Cardiopulmonary bypass (CPB) is a form of extracorporeal circulation in which the patient's blood is diverted from the heart and lungs and rerouted outside of the body. The normal physiologic functions of the heart and lungs, including circulation of blood, oxygenation, and ventilation, are temporarily taken over by the CPB machine. (See 'General principles' above.)

- Typical parameters during CPB in adults include flow rate controlled at 2.2 to 2.4 L/min/m^2, maintenance of mean arterial pressure (MAP) ≥65 mmHg, and mixed venous oxygen saturation ≥75 percent (table 1). Maintenance of renal blood flow is achieved by maintaining adequate CPB pump flow throughout CPB to minimize the risk of acute kidney injury (AKI). (See 'Pump flow and mixed venous oxygen saturation' above and 'Mean arterial pressure' above.)

- Arterial pO_2 is maintained at 150 to 250 mmHg during CPB alpha-stat management of arterial blood gases without temperature correction to maintain a normal range for pCO_2 (35 to 45 mmHg [4.7 to 6 kPa]) and pH (7.35 to 7.45). (See 'Oxygenation, ventilation, and arterial blood gases' above.)

- Deliberate hypothermia is employed in selected patients undergoing CPB. During cooling, the temperature gradient between the venous inflow and arterial outlet on the oxygenator is maintained at <10°C. Rewarming is gradual (≤0.5°C/minute), and the temperature gradient between the venous inflow and the oxygenator arterial outlet is maintained at ≤4°C. To minimize risk of cerebral hyperthermia, the arterial outlet and nasopharyngeal temperatures are maintained ≤37°C. (See 'Temperature' above.)

- Inadequate anesthetic depth is treated by increasing the volatile anesthetic concentration administered via the CPB circuit or by administering additional intravenous (IV) anesthetic agents. It is reasonable to monitor processed electroencephalogram (EEG) indices (eg, bispectral index) or the unprocessed EEG to provide data that may detect inadequate anesthesia during CPB. Decreased dosing of the selected neuromuscular blocking agent (NMBA) may be adequate during the hypothermic period of CPB; however, additional NMBA is typically required during rewarming. (See 'Maintenance of anesthesia and neuromuscular blockade' above.)

- For Hgb <7.5 g/dL (or hematocrit [Hct] <22 percent), initial treatment during CPB is removal of fluid by ultrafiltration (hemoconcentration) when possible [62]. Transfusion of packed red blood cells (RBCs) is reasonable if Hgb remains <7.5 g/dL when hemoconcentration is not possible or is ineffective. (See 'Hemoglobin/hematocrit' above.)

- Blood glucose levels <180 mg/dL (10 mmol/L) during CPB and the postbypass period, with a single dose or intermittent doses of insulin if effective or with a continuous insulin drip if glucose levels are persistently elevated (>180 mg/dL [10 mmol/L]. (See 'Glucose' above.)

- Metabolic abnormalities (eg, hyperglycemia, hypocalcemia, hyperkalemia, hypokalemia, hypomagnesemia) are common during CPB. Frequent laboratory testing during CPB (approximately every 30 minutes) is helpful for recognition and treatment. (See 'Electrolytes' above.)

- After removal of the aortic cross-clamp, management of cardiac dysrhythmias (eg, heart block, ventricular fibrillation, junctional rhythm) is often necessary. (See 'Arrhythmias' above.)

- Specific management strategies are employed during CPB in patients with aortic insufficiency, cerebrovascular disease, renal insufficiency, and for those undergoing elective deep hypothermic circulatory arrest (DHCA). (See 'Management of special populations' above.)

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Contributor Disclosures

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