Cardiac anaesthesia

APPROACH TO ANAESTHESIA FOR ADULT CARDIAC PATIENT

Preoperative evaluation, preparation and monitoring
Heart and vascular system:
Standard monitoring via the ECG involves using the five-lead electrode system (detecting of arrhythmia and myocardial ischaemia)

Arterial and central venous pressure monitoring:
Invasive arterial cannulation and monitoring are considered to be a standard care for cardiac surgical patients. Patient risk factors and comorbid conditions often necessitate real-time, beat-to-beat assessment of arterial perfusion pressure and arterial blood gases monitoring. During nonpulsatile cardiopulmonary bypass, noninvasive blood pressure recordings are not accurate.

Central venous pressure monitoring and central venous access are essential during cardiac surgery. Central venous catheters provide portals for volume replacement, pharmacologic therapy and insertion of other invasive monitors (such as pulmonary artery catheters) or pacing wire.

Pulmonary artery catheterization
Expert opinion does not support routine use of the pulmonary catheter in low-risk patients.

Echocardiography
Transoesophageal echocardiography (TEE) is extremely helpful both as a monitoring system and as a tool for the haemodynamic management of patients under general anaesthesia. TEE can directly visualize the cardiac structures but also it can measure blood flow velocity. TEE is routinely used to monitor for the presence of retained intracardiac air after open chamber procedures such as valvular surgery. TEE enables to show the thoracic aorta in real time – this is invaluable aid in correctly positioning intraaortic balloon pump.

Evaluation for diastolic dysfunction should be part of the routine echocardiographic examination of cardiac surgical patients. Patients with preexisting diastolic dysfunction are prone to post-cardiopulmonary bypass complications, including difficulty separating from cardiopulmonary bypass and the need for more vasoactive support in the intensive care unit (ICU).

CARDIOPULMONARY BYPASS
Cardiopulmonary bypass (CPB) is a form of extracorporeal circulation (ECC) (extra = “outside of”; corporeal = “the body”) in which the patient’s blood is rerouted outside the vascular system and the function of the heart, the lungs, and to a lesser extent the kidneys is temporarily assumed by surrogate technology.

The goal of CPB is to provide a motionless and bloodless field for the surgeon by rerouting blood away from the area of surgical interest.
Venous blood is intercepted as it returns to the right atrium and diverted through a venous line of a cardiopulmonary bypass circuit to a venous reservoir. The arterial pump functions as an artificial heart by withdrawing blood from the reservoir and by propelling it through a heat exchanger, an artificial lung (the oxygenator), and an arterial line filter before returning it through the arterial line to the patient’s arterial system. Additional pumps and components are used to assist in the operation to manage shed blood (the pump sucker), decompress the heart (vent) and deliver cardoplegia solution (for myocardial protection and interrupting myocardial electromechanical activity).
INDUCTION OF ANAESTHESIA AND THE PREBYPASS PERIOD

Premedication
Patients should receive their usual long-term medications on the day of the surgery, especially β-adrenergic blocking agents. ACEIs, if administered on the day of surgery, may increase the patient’s propensity for hypotension (due to peripheral vasodilation).

With respect to aspirin, it is well recognized that aspirin administration in the early postoperative period reduces the risk of ischaemic complications after CABG surgery. However, patients receiving aspirin immediately before surgery may have more mediastinal bleeding and greater transfusion requirements. Aspirin should be administered within 6 hours after CABG surgery, regardless of whether preoperative aspirin therapy is discontinued. Aspirin should be continued until the time of the surgery in patients at high risk for coronary thrombosis (i.e. those with unstable angina or recent myocardial infarction). In patients undergoing unquestionably elective cardiac surgery aspirin should be discontinued for 3 to 5 days beforehand because active platelet aggregation is less of a risk factor of ischaemia in these patients.
Clopidogrel is usually discontinued 5 to 7 days before elective cardiac surgery.

The prospect of undergoing cardiac surgery provokes anxiety in most patients. Insertion of intravenous and arterial catheters is painful and occurs before anaesthesia is induced. Anxiety and pain can lead to undesirable sympathetic stimulation resulting in tachycardia and hypertension. The first step in preventing this cycle is explaining the anticipated anaesthetic techniques and procedures to the patient.

Premedication with an anxiolytic or narcotic agent (or both) is usually indicated before the patient is transported to the operating room. Supplemental intravenous drugs (midazolam, fentanyl) can be added during radial artery cannulation before induction of anaesthesia.

Avoid oversedation!
- in patients with low cardiac output secondary congestive heart failure oversedation may result in myocardial depression and hypotension
- in patients with significant pulmonary hypertension, oversedation and respiratory depression may lead to hypoxia or hypercapnia

Induction of anaesthesia
Anaesthetics and techniques for induction of anaesthesia should be selected with consideration of the patient’s cardiac pathophysiology and comorbid conditions. No single “recipe” can guarantee haemodynamic stability during anaesthetic induction.

Hypotension may result from:
- a relatively hypovolaemic state
- vasodilation secondary to a reduction of sympathetic tone induced by anaesthetics
- poor left ventricle function.

Hypertension may occur during induction due to preinduction anxiety or sympathetic stimulation caused by laryngoscopy or endotracheal intubation.

It is important to cannulate the radial artery or an alternative site before induction of anaesthesia to monitor arterial pressure on a beat-to-beat basis. Noninvasive blood pressure measurement can be used to check the accuracy of pressure readings.

Other basic monitors should be used or inserted during induction of anaesthesia: ECG, pulse oximeter, central venous catheters, urinary catheter, nasogastric tube, TEE probe, additional temperature monitors (a nasopharyngeal probe)

Choosing anaesthetic agents and doses during induction and maintenance:
Anaesthesia is most commonly induced with an opioid (fentanyl, sufentanil) and a sedative-hypnotic (etomidate, midazolam, propofol, or thiopental). All anaesthetics decrease blood pressure by decreasing sympathetic tone, decreasing systemic vascular resistance (SVR), inducing bradycardia, or directly depressing myocardial function.

Ketamine is the only exception. It has sympathomimetic effects. However, in patients with catecholamine depletion, sympathomimetic effects may not counterbalance its direct negative inotropic effects. Muscle relaxants are usually given early in the sequence of induction, particularly if relatively high doses of opioids are administered (to minimize chest wall rigidity).

Volatile agents are often chosen as the primary maintenance anaesthetic. The predominant effect of isoflurane, desflurane and sevoflurane is dose-dependent vasodilation with resultant decrease in SVR and blood pressure. The volatile agents (compared to propofol or midazolam) have advantage in inducing preconditioning, which is
particularly important in patients undergoing procedures in which myocardial insults are likely. Multiple cardioprotective effects of the volatile anaesthetics have been studied, including trigerring of the preconditioning cascade and mitigation of reperfusion injury.

Nitrous oxide is avoided by most cardiac anaesthetist because of its ability to increase gaseous bubble size and its undesirable effect on pulmonary vascular resistance (PVR).

**The pre-cardiopulmonary bypass period**

Several important details must be kept in mind during positioning the patient:
- avoid plexus brachial injury – by hyperextending the arms
- avoid ulnar nerve injury – by improperly padding the olecranon
- avoid radial nerve injury – by compressing the upper part of the arm against the sternal retractor support post
- avoid finger injury – by entrapping the finger against the metal edge of the surgical table
- the head should be patted and occasionally repositioned during the procedure to prevent occipital alopecia
- the eyes should be taped, possibly lubricated, and definitely free from pressure

Pressure-related injury to any soft tissue can be exacerbated by hypothermia and decreased perfusion during cardiopulmonary bypass.

Antibiotics must be administered (with documentation) within 1 hour of incision (vancomycin within 2 hours). Arterial blood gases and blood chemistry (electrolytes, glucose and calcium) should be measured shortly after anaesthesia is induced.

During prebypass period, the major goal is to maintain haemodynamic and metabolic stability of the patient while making preparations for cardiopulmonary bypass.

The degree of surgical stimulation varies markedly during this period:

<table>
<thead>
<tr>
<th>Minimal sympathetic stimulation (hypotension, bradycardia)</th>
<th>More intense surgical stimulation (hypertension, tachycardia and dysrhythmias)</th>
</tr>
</thead>
<tbody>
<tr>
<td>- patient positioning</td>
<td>- chest incision</td>
</tr>
<tr>
<td>- preparation of the skin</td>
<td>- sternal splitting</td>
</tr>
<tr>
<td>- harvesting of the saphenous veins</td>
<td>- harvesting of internal mammary artery</td>
</tr>
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</table>

Just before initiation of cardiopulmonary bypass, during cannulation of the great vessels, surgical stimulation is against minimal, and manipulation of the heart and great vessels may transiently decrease venous return and cause a dramatic decline in blood pressure.

In preparation for cardiopulmonary bypass, anticoagulation must be achieved. Heparin is the standard agent. It is administered through a central venous catheter at initial dose of 300 U/kg. Onset is almost immediate, but the drug is allowed to circulate for 3 to 5 minutes before its effect is measured.

Activated clotting time (ACT) is the mainstay of anticoagulation monitoring. Normal range is 80-120 seconds. Heparin dosing for extracorporal circulation is targeted at maintaining ACT values greater than 480 seconds. After heparinization the next major step in the prebypass phase is vascular cannulation. One or more large veins or the right atrium is cannulated so that all systemic blood is diverted to the pump oxygenator. A large artery, usually the ascending aorta is cannulated so that oxygenated blood is delivered back to the arterial circulation.

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1 Whole blood is added to a tube containing a contact-phase activator (celite or kaolin) and a small iron cylinder. The sample is warmed to 37°C, and the tube is rotated. Clot formation is detected by retracting the iron cylinder, which disrupts a magnetic field. Several clinical variables can affect the ACT. Platelet lysis and surgical stress shorten the ACT. Haemodilution, hypothermia, thrombocytopenia, platelet inhibitors prolong the ACT.
circulation. Heparin is always administered before cannulation. Arterial cannulation is established before venous cannulation to allow rapid volume or blood resuscitation if necessary.

<table>
<thead>
<tr>
<th>Complications of aortic cannulation</th>
<th>Complications of venous cannulation</th>
</tr>
</thead>
<tbody>
<tr>
<td>-arterial dissection, haemorrhage and resultant hypotension</td>
<td>-hypotension from blood loss</td>
</tr>
<tr>
<td>-inadvertent cannulation of the aortic arch vessels</td>
<td>-dysrhythmias</td>
</tr>
<tr>
<td>-embolic phenomena caused by dislodged atherosclerotic plaque or by air introduced into or entrained around the aortic cannula.</td>
<td>-surgical mechanical compression of the heart or great vessels</td>
</tr>
</tbody>
</table>

**Onset of cardiopulmonary bypass**

With the onset of cardiopulmonary bypass the perfusionist checks the aortic inflow line and seek for signs of inadequate venous return, while the anaesthetist checks for persistently low arterial pressure, unilateral blanching of the face, or any swelling in the neck veins, face or conjunctiva (risk for for malposition of the aortic or venous cannula).

Once full bypass is established and aortic ejection by the heart has ceased, ventilation and volatile agents can be discontinued. If pulmonary catheter is present, it is pulled back 3 to 5 cm to minimize the risk of pulmonary perforation as the pulmonary arteries collapse. The TEE probe may be used to watch for haemodynamic problems with the onset of cardiopulmonary bypass. Once cardiopulmonary bypass is established, the probe is left in neutral position until the cardiac chambers are de-aired and the patient is weaned from cardiopulmonary bypass.

To ensure adequate anaesthetic depth, supplemental intravenous sedative-hypnotics are administered, or a volatile agent is administered via a vaporizer connected to the oxygenator gas inlet of the cardiopulmonary bypass circuit. Administration of muscle relaxant is continued to prevent spontaneous ventilation, movement, or shivering during hypothermia and rewarming.

**WEANING FROM CARDIOPULMONARY BYPASS**

**Preparation for weaning from cardiopulmonary bypass: the “CVP” mnemonic**

For most patients separation from cardiopulmonary bypass is a relatively uneventful process. Several criteria should be met in all cardiac surgical cases before attempting weaning from cardiopulmonary bypass. Romanoff and Royster suggest a mnemonic “CVP” to help the clinician remember the main tasks necessary for successful termination of cardiopulmonary bypass.

<table>
<thead>
<tr>
<th>C</th>
<th>V</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cold</td>
<td>Ventilation</td>
<td>Predictors</td>
</tr>
<tr>
<td>Conduction</td>
<td>Visualization</td>
<td>Pressure</td>
</tr>
<tr>
<td>Cardiac output (contractility)</td>
<td>Vaporiser</td>
<td>Pressors</td>
</tr>
<tr>
<td>Cells</td>
<td>Volume expanders</td>
<td>Pacer</td>
</tr>
<tr>
<td>Calcium</td>
<td>Potassium</td>
<td></td>
</tr>
<tr>
<td>Coagulation</td>
<td>Protamine</td>
<td></td>
</tr>
</tbody>
</table>

C:  
1. **COLD** – refers to the patient’s temperature at the time of waning from cardiopulmonary bypass, which should be **36-37°C**; hyperthermia may increase the risk for the postoperative neurological complications  
2. **CONDUCTION** – refers to cardiac rate and rhythm;  
   a) heart rate of **80-100 beats/min** is usually desirable;
bradycardia is treated with epicardial pacing wires and with \( \beta \)-adrenergic agents that have chronotropic, dromotropic and inotropic properties; tachycardia (HR>120 beats/min) is also undesirable; sinus tachycardia may be due to hypovolaemia, anaemia, “light” anaesthesia or the administration of chronotropic agents; rhythm is also important factor in optimizing cardiac output; third-degree atrioventricular block requires pacing; sinus rhythm is preferable, particularly in patients with poor left ventricle compliance, who are especially dependent on “atrial kick” to achieve adequate filling; if supraventricular tachycardia is present, direct synchronized cardioversion is warranted; pharmacologic therapy with amiodarone, esmolol, verapamil, or adenosine may be used in the initial treatment or to prevent the reoccurrence of supraventricular tachycardia

3. CONTRACTILITY – may be estimated from TEE and CARDIAC OUTPUT can be measured with a pulmonary artery catheter (if available)

4. CELLS – refers to red blood cells. The patient’s haemoglobin concentration should be 7 to 8 g/dL or slightly higher before weaning from cardiopulmonary bypass; if the haemoglobin concentration is less than 6.5 g/dL when rewarming commences, the perfusionist and anaesthetist can consider haemoconcentration or transfusion of a unit of packed red blood cells

5. CALCIUM – the patient’s ionized Ca\(^{2+}\) level should be checked

6. COAGULATION – in patient at high risk for coagulation abnormalities, the prothrombin time, partial thromboplastin time, and platelet count should be measured a few minutes after administration of protamine

Risk factors for coagulation abnormalities: long cardiopulmonary bypass time, extreme hypothermia, elective circulatory arrest, chronic renal failure

V:

1. VENTILATION of the lungs
Pulmonary ventilation and oxygenation must be reestablished to allow the lungs to again become the site of gas exchange. The lungs are initially reinflated “by hand” with a few sustained inflations to a peak pressure of about 30 cm H\(_2\)O. The surgeon should remove any fluid or blood from the pleural spaces and ensure that any pneumothorax is treated with a chest tube.

2. VISUALIZATION of the heart, both directly in the surgical field and on TEE, to estimate global and regional contractility and degree of chamber filling (hypovolaemic, euvoalaemic, or distended)

3. VAPORIZER – if volatile agents were used to ensure lack of awareness or to control blood pressure during anaesthesia, the clinician should consider reinstituting a low dose immediately after waning; because all of the volatile agents decrease contractility and blood pressure, these effects can confuse the differential diagnosis of hypotension and dysfunction during weaning

4. VOLUME EXPANDERS
When all products from the pump have been exhausted and if blood transfusion is not indicated, crystalloid and albumin or hetastarch/gelatine should be readily available to rapidly increase preload if necessary.

P:

1. PREDICTORS of adverse cardiovascular outcome
Preoperative low ejection fraction and prolonged duration of cardiopulmonary bypass often predict difficulties in weaning the patient from cardiopulmonary bypass and the need for inotropic support. Emergency surgery in patients with acute coronary syndrome may lead to myocardial stunning. Inadequate surgical repair (e.g. incomplete coronary revascularization) may result in ongoing ischaemia.

2. PRESSURE – calibration, rezeroing the transducers should be accomplished, discrepancy between distal (usually radial) arterial and aortic pressure should be recognized; sometimes the surgeon may
need to insert a temporary aortic root cannula or a longer-lasting femoral arterial cannula to accurately monitor systemic blood pressure during and after termination of cardiopulmonary bypass

3. PRESSORS – vasopressors, inotropic agents (and vasodilators) should be immediately available

4. PACER – external pacemaker should be readily available for all patients; Pacing is often needed to treat bradycardia. In patients with heart block, ideally atroventricular sequential pacemaker is used to maintain the atrial “kick”.

5. POTASSIUM
   Hypokalaemia may contribute to dysrhythmias, and hyperkalaemia may result in conduction abnormalities.

6. PROTAMINE
   The protamine dose required to reverse heparin is 1 to 1.3 mg of protamine for every 100 units of heparin. Protamine should be administered slowly, over a period of 5 minutes or longer.

**Termination from cardiopulmonary bypass**

After ventilation has been reestablished, venous return to the pump is reduced by clamping venous line. The patient’s intravascular volume is carefully increased by continued inflow via the aortic cannula. Ventricular distension should be avoided because it increases wall tension and myocardial oxygen consumption. When loading conditions are optimal and contractility appears adequate, the aortic inflow line may be clamped to separate the patient from cardiopulmonary bypass. At this point global and regional function of both ventricles is determined by the anaesthetist and surgeon. Afterload can also be optimized at this point. If the patient is haemodynamically unstable and additional time is needed to administer initial or additional inotropes or vasoconstrictors, cardiopulmonary bypass can be reinstituted by unclamping venous outflow line and directing all flow to the oxygenator again.

When protamine is administered, the venous and arterial cannulas are removed.

**The postbypass period**

**Maintaining anaesthesia**

Consideration should be given to continuing to administer a volatile agent once pulmonary ventilation is reestablished and to administering additional sedative/hypnotic doses, a narcotic agent or both. Some clinicians begin infusing an agent, such as propofol or dexmedetomidine shortly after weaning the patient from cardiopulmonary bypass and plan to continue it during and after transport to the ICU or cardiac recovery area.

Another decision to be made is whether additional muscle relaxant is needed during and after weaning from cardiovascular bypass. Patient movements can be extremely dangerous if it results in dislodgement of the aortic or venous cannulas. Muscle relaxant also prevents shivering after cardiopulmonary bypass.

**Cardiovascular decompensation**

Causes:

1. Ventricular flutter or fibrillation must be treated immediately with defibrillation. Internal paddles are applied do the heart to deliver 10-20 J of electricity. If ventricular arrhythmias persist or occur, an antiarrhythmic drug (lidocaine, amiodarone) is infused.

2. Failure of right or left ventricle
   Such failure may result in part from preexisting chronic ventricular dysfunction exacerbated by the ischaemia-reperfusion injury that occurs after cardiopulmonary bypass with cardioplegia-induced cardiac arrest. Heart failure after cardiopulmonary bypass may also occur because of inadequate myocardial protection or inadequate revascularization with resultant ischaemia or infarction.

3. Vasodilation with low systemic vascular resistance (hypotension associated with protamine, chronic use of medications such as ACEIs, severe anaemia, acid-base disturbance and infection)
**Metabolic disturbances**
Hypokalaemia, hyperkalaemia, hypocalcaemia, hypomagnesaemia, hyperglycaemia

**Chest closure**
Haemodynamic deterioration may occur during chest closure because of cardiac tamponade in patients with ongoing bleeding. Other reasons for severe hypotension during chest closure include: hypovolaemia, ischaemia secondary to kinking of an arterial or venous coronary graft, and impairment of right ventricle contractility and venous return in patients with significant myocardial oedema.
Occasionally the patient’s sternum cannot be closed because of haemodynamic instability. In such cases only skin closure is attempted, and plans are made to return to the operating theatre for sternal wiring after a period of myocardial recovery in the ICU.

**Transport to intensive care unit**
After all incisions are closed and dressings are applied, provided that patient is haemodynamically stable, the next step is transport to the ICU.

**Problems in the postoperative period:**

*Low Cardiac Output Syndrome (LCOS)*
Criteria used to define LCOS include:
- \( CI < 2.4 \text{ L/min/m}^2 \)
- elevated lactate levels
- urine output < 0.5 mL/hr for more than 1 hour
$S_{P_{O_2}}$ is also a good indicator of LCOS – $S_{P_{O_2}}$ reflects the balance between cardiac output and systemic oxygen demand.

Risk factors of LCOS:

<table>
<thead>
<tr>
<th>Preoperative risk factors</th>
<th>Intraoperative risk factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>- poor LV function</td>
<td>- myocardial ischaemia during cross-clamping</td>
</tr>
<tr>
<td>- reoperation</td>
<td>- reperfusion injury</td>
</tr>
<tr>
<td>- urgency of the operation</td>
<td>- cardioplegia-induced myocardial dysfunction</td>
</tr>
<tr>
<td>- female gender</td>
<td>- activation of the inflammatory and coagulation cascades</td>
</tr>
<tr>
<td>- diabetes mellitus</td>
<td></td>
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<tr>
<td>- advanced age</td>
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<tr>
<td>- left main coronary artery disease</td>
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<tr>
<td>- myocardial infarction within the previous 30 days</td>
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<tr>
<td>- triple-vessel disease</td>
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<tr>
<td>- hypertension</td>
<td></td>
</tr>
<tr>
<td>- peripheral vascular disease</td>
<td></td>
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<tr>
<td>- renal disfunction</td>
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</table>

Preexisting diastolic dysfunction is associated with an increased risk of difficulty in separating patient from cardiopulmonary bypass and need for vasoactive support in the ICU. Evaluation of diastolic function should be part of the routine echocardiographic assessment of patients undergoing cardiac surgery!

Postoperative management of patients at high risk for LCOS requires a physiological approach:
- **Reducing afterload and optimizing preload** help maximize cardiac function
- tachycardia should be avoided (it increases the risk for ischaemia and postoperative myocardial infarction)
- shivering must be prevented (it raises the heart rate by increasing of oxygen demand)

Postoperative deep sedation and muscle relaxation are often used to reduce myocardial workload by reducing the body’s overall metabolic demand by 25-30%.

Pharmacological support is sometimes needed to improve contractility as the patient is weaned from cardiopulmonary bypass and recovering in the ICU. **Catecholamines** and **phosphodiesterase inhibitors** are the main classes of pharmacologic agents used for this purpose.

<table>
<thead>
<tr>
<th>Agent</th>
<th>Significant findings</th>
</tr>
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<tbody>
<tr>
<td>Epinephrine</td>
<td>Increases CI with a biphasic effect on SVR index. Produces a rise in serum lactate.</td>
</tr>
<tr>
<td>Dopamine</td>
<td>Increases the SVR index at doses above 5 µg/kg/min. Less clinical efficacy than with dobutamine, dopexamine, amrinone, or enoximone. Higher incidence of adverse cardiac events than with dopexamine.</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>Better efficacy than dopamine and epinephrine. Decreased SVR index. Tachycardia and tachyarrhythmia (especially atrial fibrillation) associated with its use. More ischaemic complications than with amrinone.</td>
</tr>
<tr>
<td>Dopexamine</td>
<td>Greater tachycardia than with dobutamine. More efficacious and fewer adverse events than with dopamine.</td>
</tr>
<tr>
<td>Amrinone</td>
<td>Improved weaning from cardiopulmonary bypass. Improves CI and decreases the SVR index with minimal effects on heart rate. Fewer ischaemic complications than with dobutamine. Reports of thrombocytopenia associated with use.</td>
</tr>
<tr>
<td>Enoximone</td>
<td>Significant increase in CI without tachycardia. Decreases the SVR index. As effective as dobutamine.</td>
</tr>
</tbody>
</table>
### Milrinone
- Significant increase in CI without tachycardia.
- Decreases the SVR index.
- As effective as dobutamine but less atrial fibrillation.
- Lusitropic.
- Improves internal mammary artery graft flow.
- As effective as 20 ppm NO in pulmonary hypertension.

### Levosimendan
- This calcium sensitizer exhibits potent inodilatoratory properties.
- Enhances calcium sensitivity in cardiac myocytes, thereby increasing stroke volume and CI while decreasing pulmonary artery occlusion pressure.
- Does not increase myocardial oxygen demand.

### Arrhythmias
Postoperative arrhythmias are seen in 10-40% of patients who undergo cardiac surgery.

Atrial arrhythmias occur in up to 50% of patients after valve surgery and in 17-33% of patients after CABG. The most common arrhythmia after cardiac surgery is atrial fibrillation. Patients are at the highest risk for new-onset atrial fibrillation in the first few days after cardiac surgery. New-onset atrial fibrillation can cause haemodynamic compromise, and it increases the risk for thromboembolic complications.

Treatment of atrial fibrillation has been achieved by pharmacological agents (β-blockers and amiodarone) and electrical means (synchronised cardioversion).

Ventricular arrhythmias occur after cardiac surgery, but sustained ventricular arrhythmias are relatively uncommon.

Associated factors of ventricular arrhythmias: haemodynamic instability, electrolyte abnormalities, hypoxia, hypovolaemia, ischaemia or infarction, acute great closure reperfusion or inotropic agents.

Ventricular arrhythmias can range from simple premature ventricular complexes (PVCs) to ventricular tachycardia (VT) or ventricular fibrillation (VF). Single PVCs do not pose a significant risk of life-threatening ventricular arrhythmia. Complex ventricular arrhythmias may make patients prone to sudden death, especially in the long term. Patients with sustained ventricular arrhythmias have a poor prognosis in both the short and long term.

Asymptomatic or haemodynamically stable patients do not usually require acute treatment of PVCs or even short runs of nonsustained VT, although any reversible causes should be corrected. Patients with VT accompanied by haemodynamic instability need immediate synchronised cardioversion. Amiodarone is reserved for haemodynamically stable patients with VT or an uncertain rhythm. Ventricular fibrillation should be treated with electrical defibrillation.

Long-term management of sustained ventricular arrhythmias: antiarrhythmic agents, electrophysiological studies or placement of implantable cardioverter-defibrillator

Bradyarrhythmias are uncommon in the immediate postoperative period. In most cases, a temporary epicardial pacemaker is sufficient. In a small percentage of patients, a permanent pacemaker may be necessary, especially in those with sinus node dysfunction, atrioventricular conduction disturbances after either CABG or valve surgery.

### Hypertension
Causes of postoperative hypertensions are multifactorial and may include:
- withdrawal from preoperative antihypertensive medications
- pain
- hypoxaemia, hypercapnia
hypothermia.

Arterial vasoconstriction usually plays a central role in acute postoperative hypertension.

Hazards of untreated postoperative hypertension include: increased myocardial work and oxygen consumption, myocardial infarction, rhythm disturbances, increased bleeding, and even suture line disruption.

Treatment of postoperative hypertension:

1. deepening sedation
2. pain control
3. preventing of hypothermia, postoperative warming
4. antihypertensives

Vasodilators available for the treatment of perioperative hypertension

A. Nitrovasodilators
   Nitroglycerine is the first agent to treat hypertension for patients undergoing after coronary revascularization (anti-ischaemic effects; not always effective – NTG primarily causes venodilation rather than arterial dilation; patients tend to develop tolerance to NTG).

Sodium nitroprusside is a nonspecific venous and arterial vasodilator. But theoretically nitroprusside can cause coronary steal. In patients with renal failure elimination of sodium nitroprusside is reduced, thus making the patient vulnerable to the toxic effects of the drug’s metabolites (cyanide and thicyanate)

B. Dihydropyridine-type calcium channel blockers (e.g. nicardipine, clevidipine)
   They selectively relax arterial resistance vessels without negative inotropic or dromotrophic effects. They result in generalized vasodilation of the renal, cerebral, intestinal and coronary vascular beds. Nicardipine and clevidipine are recommended by some experts as first-line treatment for cardiac surgical patients with acute hypertension requiring immediate control.

C. Dopamine agonists
   Fenoldopam is a short-acting dopamine agonist that causes arterial-specific vasodilation by stimulating D₁ receptors. Fenoldopam increases renal blood flow to produce diuresis and natriuresis. In one trial in cardiac surgical patients, the investigators concluded that fenoldopam did prevent acute kidney injury in high-risk population of patients undergoing cardiac surgery. Higher doses of fenoldopam may be needed for severe hypertension but may be associated with undesirable increases in heart rate.

D. α₁-adrenergic antagonists: urapidil (available in Europe, not approved in the US)
E. α-adrenergic antagonists (e.g. prazosin, doxazosin)
F. angiotensin-converting enzyme inhibitors (e.g. enalaprilat)
G. angiotensin II antagonists
H. atrial natriuretic peptide (nesiritide)
I. adenozone
J. hydralazine
K. minoxidil
L. diazoxide

NOTE:
It is important to ensure that the patient’s intraarterial pressure is monitored adequately when any vasoactive agent is administered. Vasoconstriction or poor perfusion of the extremities may create a discrepancy between central aortic and peripheral arterial pressure. A radial artery catheter may be “positional” if hand positioning is suboptimal, or “damping” of the tracing may occur as a result of poor perfusion of the distal extremities.
Occasionally during the perioperative period, the cardiac anaesthetist or surgeon must replace a peripheral catheter (e.g. insert a femoral artery catheter) to ensure that the effects of vasoactive therapy are monitored accurately.

**Renal insufficiency**

In a healthy individual, about 20% of the total cardiac output (~1 L/min) is received by the kidneys. The percentage of oxygen consumption versus total-body oxygen utilization does not usually exceed 10%. Despite such a low oxygen extraction ratio, the kidneys are extremely sensitive to hypoperfusion occurring as a result of hypotension.

Common causes of renal injury in cardiac surgical patients include:
- fluid loss
- myocardial depression
- peripheral vasodilation caused by anaesthetics and long-term treatment with angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers
- systemic inflammatory response
- loss of pulsatile flow during cardiopulmonary bypass.

Perioperative renal dysfunction, when it occurs, can have a serious consequences. Postoperative acute renal failure in cardiac surgical patients is associated with longer ICU stay, longer overall hospital stay and increased mortality.

In a multicentered study of patients who underwent coronary revascularization with or without vascular surgery, Mangano and coworkers found higher mortality in patients with acute renal failure requiring dialysis (63%) and those with renal dysfunction (19%) than in patients who had neither (0.9%). Patients with postoperative renal failure had significantly longer hospital and ICU stays than did patients who had no renal failure or dysfunction in the postoperative period.

Risk factors that are commonly associated with postoperative renal dysfunction after cardiac surgery include:

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<th>Intraoperative factors</th>
<th>Postoperative factors</th>
</tr>
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<tbody>
<tr>
<td>- preexisting renal insufficiency</td>
<td>- the need for emergency surgery</td>
<td>- hypovolaemia</td>
</tr>
<tr>
<td>- type 1 diabetes mellitus</td>
<td>- cardiopulmonary bypass time exceeding 3 hours</td>
<td>- hypotension secondary to either hypovolaemia or LCOS, and embolic phenomena</td>
</tr>
<tr>
<td>- age &gt; 65 years</td>
<td>- poor cardiac function</td>
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<tr>
<td>- major vascular surgery</td>
<td></td>
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<tr>
<td>- arteriopathy</td>
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<tr>
<td>- genetic predisposition</td>
<td></td>
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<tr>
<td>- recent exposure to nephrotoxic agents (e.g. radiographic dyes, bile pigments, aminoglycoside antibiotics, nonsteroidal anti-inflammatory drugs)</td>
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</table>

**Damage to nephrons in the medullary region of the kidney is the commonest cause of acute tubular necrosis.**

Hypoxia is a common cause of damage to nephrons in this region.

It is thought that adequate hydration and maintenance of normovolaemia can help in the prevention of postoperative renal dysfunction in cardiac surgical patients. In patients at high risk for acute renal failure, perioperative care should optimize cardiac output and, if possible, reduce the duration of cardiopulmonary pump.

The definition of acute renal failure include three useful criteria:
1) a serum creatinine level greater than 0.5 mg/dL above its preoperative value
2) a serum creatinine level greater than 50% above its preoperative value
3) a serum creatinine level higher than 2.0 mg/dL.

Another definition of acute renal failure involves a classification scheme that uses the acronym “RIFLE”.

<table>
<thead>
<tr>
<th>GFR criteria</th>
<th>Urine output criteria</th>
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<tbody>
<tr>
<td><strong>Risk</strong></td>
<td>Plasma creatinine increased 1.5x or GFR decrease &gt; 50%</td>
</tr>
<tr>
<td></td>
<td>&lt; 0.5 mL/kg/h x 6 hrs</td>
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<tr>
<td><strong>Injury</strong></td>
<td>Plasma creatinine increased 2x or GFR decrease &gt; 25%</td>
</tr>
<tr>
<td></td>
<td>&lt; 0.5 mL/kg/h x 12 hrs</td>
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<tr>
<td><strong>Failure</strong></td>
<td>Plasma creatinine increased 3x, acute plasma creatinine ≥ 4 mg/dL, or acute rise ≥ 0.5 mg/dL</td>
</tr>
<tr>
<td></td>
<td>&lt; 0.3 mL/kg/h x 24 hrs or anuria x 12 hrs</td>
</tr>
<tr>
<td><strong>Loss</strong></td>
<td>Persistent acute renal failure = complete loss of kidney function &gt; 4 weeks</td>
</tr>
<tr>
<td><strong>ESKD</strong></td>
<td>End-stage kidney disease (&gt; 3 months)</td>
</tr>
</tbody>
</table>

GFR – glomerular filtration rate, ESKD – end-stage kidney disease

Postoperative acute renal failure should be prevented whenever possible. Several treatment modalities has been suggested to prevent or ameliorate postoperative renal dysfunction:
- maintain adequate oxygen delivery (by ensuring cardiac output, adequate oxygen-carrying capacity, and proper haemoglobin saturation)
- suppression of renovascular constriction (by ensuring adequate volume of preload and use of infusions of mannitol, calcium entry blockers, and angiotensin-converting enzyme inhibitors)
- renal vasodilation (by dopaminergic agents, prostaglandins, and atrial natriuretic peptide)
- maintain renal tubular flow, prevent tubular obstruction (by loop diuretics and mannitol)
- decrease renal tubular oxygen demand (by the use of loop diuretics and mild coolig)
- attenuate ischaemic reperfusion injury as a result on the release of oxygen free radicals and calcium ions.

Unproven pharmacologic therapies include dopamine, dopexamine, the loop diuretics, mannitol, calcium channel entry blockers, ACEIs and atrial natriuretic peptide.

Central nervous system dysfunction
Many patients undergoing cardiac surgery are at risk for central nervous system injury as a result of advanced age and comorbid diseases, in addition to the surgical risk of embolization, hypoperfusion and the inflammatory response associated with cardiopulmonary bypass.

**Type I central nervous complications** (fatal or nonfatal stroke or transient ischaemic attack) occur
- in 3.1% of CABG patients
- in 8.4% of patients undergoing CABG combined with another open cardiac surgical procedure
- in 10% of patients undergoing double or triple valve replacement without CABG.

Risk factors for stroke after cardiac surgery: age, history of neurologic disease, vascular disease, diabetes mellitus, previous CABG, unstable angina or pulmonary disease.

**Type II central nervous complications** (neurocognitive dysfunction) occurs more frequently. In one study 53% of CABG patients showed evidence of new neurocognitive dysfunction at the time of discharge from the hospital. After initial improvement in many patients, 42% had persistent neurocognitive deficits 5 years after surgery.
Data are inconsistent regarding whether the risk of neurocognitive disorders is higher after oper versus closed cardiac procedure.

Common causes of central nervous system injury or dysfunction in cardiac surgical patients are thought to be:
- microemboli (e.g. mobilization of atheromatous material, entrainment of air from the operating field, injection of gas into the venous reservoir)
- cerebral hypoperfusion (e.g. global hypotension, occlusion by atheromatous embolus leading to a stroke)
- cerebral hypothermia or hyperthermia (e.g. during aggressive rewarming)
- inflammatory effects (caused by interaction of blood with the foreign surfaces of the pump oxygenator)
- cerebral oedema (results from hypoperfusion, hyponatraemia)
- pharmacologic effects (anaesthetic-related cognitive damage, proteomic changes)
- dysfunction of the blood-brain barrier (diffuse cerebral inflammation).

Increasing understanding of the etiology of injury and dysfunction in the central nervous system has prompted the development of methods to monitor intraoperative brain function and guide interventions to ameliorate postoperative focal deficits and cognitive dysfunction:

- **TEE or epiaortic echocardiography** with a hand-held probe placed on the ascending aorta or aortic arch can be used to visualize protruding atheromas, atheroma burden of the aorta. The severity atheromatous disease of aorta is a strong predictor of death and stroke after CABG. These techniques are superior to surgical palpation in detecting such diseases.

- **Transcranial Doppler (TCI)** involves ultrasonic scanning of blood flow through the middle cerebral or common carotid arteries. TCI can detect emboli moving through arteries. TCI enables to measure cerebral blood flow velocity and document changes of cerebral blood flow from baseline during cardiac surgery.

**Respiratory insufficiency**
A midline sternotomy (or thoracotomy) causes significant reductions in total lung capacity, vital capacity (VC), forced expiratory volume in 1 second (FEV₁) and functional residual capacity. These changes may result in postoperative atelectasis and at least mild hypoxaemia.

Pain management should be included in postoperative period to minimize pulmonary complications. Sternotomy pain may limit the patient’s ability to cough or perform deep-breathing exercises, and incisional leg pain from harvesting of saphenous veins may prevent early ambulation, thereby increasing the risk for pulmonary complications.

Traditional management after cardiac surgery has included overnight mechanical ventilation. It is clinically important to have a period of mechanical ventilation to allow rewarming and emergence from anaesthesia, optimize cardiac function and ensure cardiac stability and the absence of unacceptable of bleeding.

However, currently many patients are extubated within 3 to 6 hours of arriving in the postoperative care unit (so called “fast-tracking”). In planning for fast tracking, one should avoid high-dose narcotic anaesthetic techniques, and postoperative doses and timing of analgesic and sedative administration must be appropriate.

Only a small percentage of patients after cardiac surgery require prolonged mechanical ventilation. Independent risk factors for prolonged ventilation include:
- advanced age
- preoperative ventilation
- elevated serum creatinine
- EF less than 30%
Valve surgery, previous cardiac surgery, urgent or emergency surgery, recent myocardial infarction, peripheral vascular disease, use of cardiopulmonary bypass, current smoking, FEV1 less than 70% of predicted value.

Prolonged ventilation is most commonly due to cardiac dysfunction and cardiogenic pulmonary oedema. Patients with marginal cardiac function may require diuresis, afterload reduction, or inotropy. More severe postoperative respiratory insufficiency causing difficulty weaning from mechanical ventilation may be due to pulmonary-related reasons:

- Intrinsic pulmonary problems (e.g., severe COPD, atelectasis, pneumonia, or surgical trauma to the lungs),
- ARDS related to cardiopulmonary bypass (postperfusion lung syndrome) or transfusion (TRALI – transfusion related lung injury),
- Pulmonary embolism.

Nonpulmonary complications such as persistent postoperative bleeding, neurologic complications (including stroke and delirium), renal insufficiency/failure, gastrointestinal complications, and sepsis may also result in a need for prolonged mechanical ventilation.

Pain
Sources of pain after cardiac surgery: sternotomy incision, chest tubes, leg incisions
The stress response and enhanced sympathetic tone are common effects of postoperative pain after cardiac surgery. Pain increases the heart rate, PVR, myocardial work, and myocardial oxygen consumption. Thus pain can contribute significantly to postoperative cardiac morbidity after cardiac procedure.

Post-cardiac surgical pain can also negatively affect the respiratory system. A restrictive defect occurs that is characterized by loss of lung volumes, thereby contributing to atelectasis. The patient’s pain may cause voluntary reduction of muscular movement in the thorax and abdomen, a phenomenon called "splinting". Such splinting interferes with patient’s ability to cough effectively and clear secretions. This problem can lead to lobar collapse, pneumonia and increased duration of hospitalization.

Unrelieved pain also has psychological effects. Pain-related anxiety, depression, and sleep deprivation may contribute to delirium in patients in the ICU.

Opioids remain the “gold standard” for pain control but they have side effects that include nausea, vomiting, urinary retention, decreed gastric motility, pruritus, sedation and respiratory depression. Interest has emerged in the use of regional anaesthesia.

Bleeding/coagulopathy
Inadequate surgical hemostasis is the most common reason for blood loss after cardiopulmonary bypass. Exposure of blood to the surfaces of bypass circuitry is a profound stimulus for inflammatory upregulation. Activation of the haemostatic system is a component of the normal inflammatory response.

Platelet dysfunction is frequently implicated in postbypass bleeding. Exposure of blood to the oxygenator and cardiopulmonary circuitry and hypothermia cause platelet activation, degranulation and aggregation. Relative
thrombocytopenia commonly occurs after cardiopulmonary bypass as a result of haemodilution, platelet sequestration, adhesion and destruction.

Another cause of coagulopathy is **thrombin production and fibrinolysis**. Thrombin is produced by the extrinsic and intrinsic coagulation pathways and by activated platelets and is only partially suppressed by heparin during cardiopulmonary bypass. Ongoing thrombin generation during cardiopulmonary bypass results in low-grade consumption of multiple factors in the coagulation cascade. If cardiopulmonary bypass is prolonged, these factors may be depleted.

Endothelial cells themselves are also vulnerable to inflammatory activation by the effects of cardiopulmonary bypass and hypoxia. Fibrinolytic activity is also increased by cardiopulmonary bypass. Contact activation of factor XII, prekallikrein and high-molecular-weight kininogen leads to fibrinolytic activation of endothelial cells to produce tissue plasminogen activator (t-PA) and lysis of fibrin strands and the fibrin precursor fibrinogen. With excessive fibrinolysis, depletion of fibrinogen can result in consumptive coagulopathy. In addition fibrinolysis exerts important harmful effects, because fibrin degradation products and plasmin inhibit platelet function.

**REFERENCES**
