Cardiopulmonary bypass

Coronary bypass grafting may be performed 'on-pump' with the use of cardiopulmonary bypass (CPB) or 'off pump' without it. There is no evidence that one technique is superior to the other. Off pump coronary artery bypass grafting (OPCAB) is usually indicated when there are contra- indications to CPB and aortic cross-clamping such as: severely atheromatous ascending aorta, porcelain aorta (calcified), patient's refusing blood transfusion or those patients deemed at very high risk of stroke. Open chamber heart valve surgery always requires CPB.

Cardiopulmonary bypass is now the norm for CABG because it provides a bloodless and motionless surgical field that makes it much easier for the surgeon to construct high quality vascular anastomoses. After surgical exposure the patient is fully anticoagulated with a bolus of systemic heparin. An arterial cannula is placed, usually into the ascending aorta, and one or two venous cannulae are placed into to the superior vena cava, inferior vena cava or right atrium. Blood is drained under the force of gravity to a venous reservoir that is open to atmospheric pressure. From the venous reservoir blood passes through a pump then an oxygenator. The oxygenator acts as an artificial lung removing of carbon dioxide and adding oxygen.

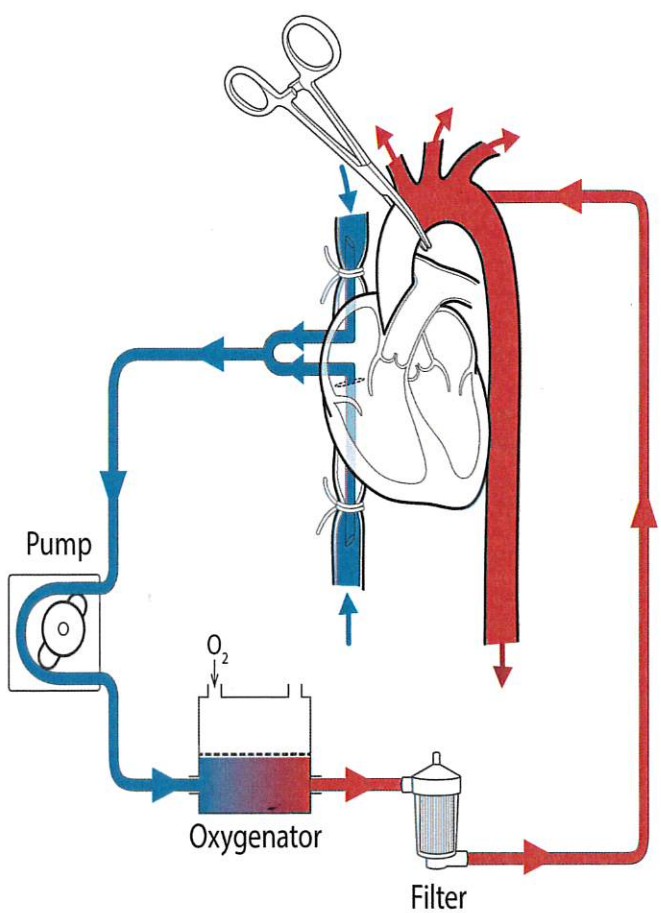

Figure 36: Basic schematic of the cardiopulmonary bypass circuit

Following initiation of CPB a cross clamp may be applied to the ascending aorta and the heart rendered asystolic with the administration of cardioplegia solution. The period during which the aortic cross clamp is applied is the 'ischaemic time'. The coronary

circulation has been isolated by application of the cross clamp. It receives no blood flow during the period of aortic cross clamping though may receive some oxygen delivery if blood cardioplegia is used (in preference to crystalloid cardioplegia). Inadequate myocardial protection during this period can lead to myocardial stunning and severe cardiac dysfunction on separating from CPB. Even with the aortic cross-clamp applied, gradual distention of the LV will occur due to return from the bronchial circulation. A left ventricle vent is inserted into the LV to drain blood from the left ventricle into the venous reservoir. This prevents LV distension that can adversely affect cardioplegia delivery and myocardial protection.

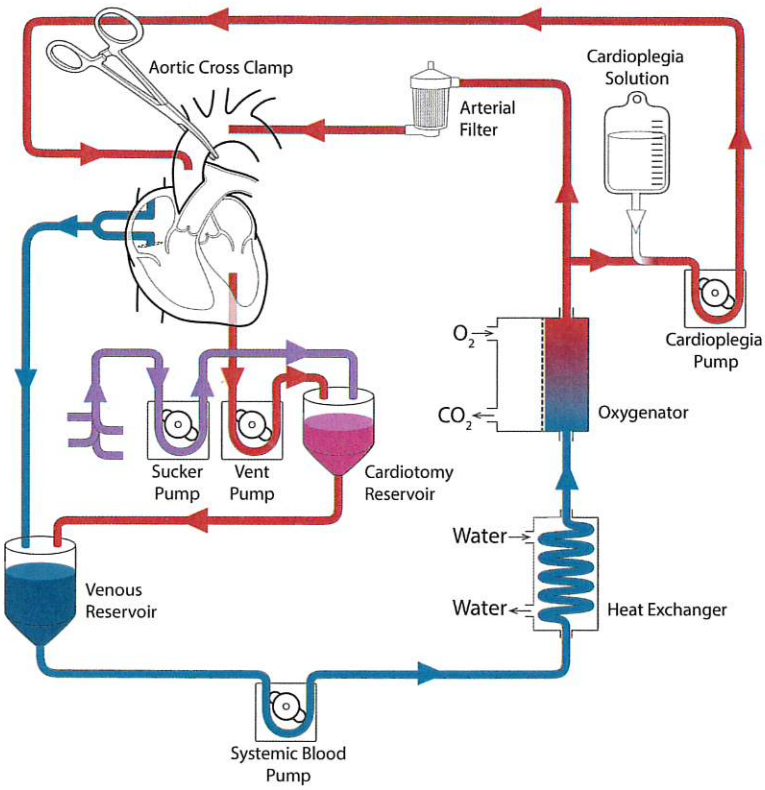

Figure 37: Detailed schematic of the cardiopulmonary bypass circuit

During CPB, blood in the extracorporeal circuit can be heated or cooled. For certain procedures such as aortic arch and valve surgery the patient will be cooled by the bypass machine to provide myocardial and neurological protection. On separating from CPB the native lungs must be adequately ventilating, the heart rhythm and ejection re- established with a normal temperature, adequate haemoglobin concentration, acid-base status and electrolyte profile achieved. The blood flow into the CPB is progressively decreased and more blood is allowed to pass across the pulmonary circulation to the left heart until the venous line is clamped and the patient is 'off bypass'.

Once the operating teams regard the patient to be stable, the CPB cannulae are removed and the heparin is reversed with protamine. Administration of protamine may cause hypotension, pulmonary vasoconstriction and anaphylaxis so the drug should be administered cautiously. After protamine reversal a second ACT is taken to check it has returned to the baseline value. Overdose of protamine may paradoxically worsen bleeding tendency due to the unbound molecule's inhibitory action on platelets. It is therefore important to dose the protamine correctly. Pump blood can be administered after CPB. This is the remaining CPB volume and still containing heparin

so if administered additional protamine may be required.

Complications of CPB

Include:

- Haemodilution
- Bleeding and platelet dysfunction
- Myocardial stunning and cardiac dysfunction
- Systemic inflammatory response syndrome
- Pulmonary complications including atelectasis, lung collapse, ARDS
- Macro- and micro-embolism
- Post-operative hypothermia
- Potassium load from the cardioplegia which may predispose patients with renal dysfunction to post- operative hyperkalaemia

Haemodilution

The prime volume for CPB, i.e. the volume of the circuit, varies between models but it is usually 1.2-1.5 litres. Some centres use lower prime circuits or use autologous priming in which the patients own blood is used to prime the circuit. If the circuit is primed with crystalloid a degree of haemodilution is inevitable and this is more marked for smaller sized patients. Some consequences of haemodilution are beneficial – such as more favourable flow characteristics at lower haematocrits, but some are detrimental. These include reduced oxygen carrying capacity, but this may be offset by the improved flow characteristics and a degree of dilutional coagulopathy. Some centres will use mannitol for circuit priming which may cause high diuresis in the initial post-operative period.

Bleeding and platelet dysfunction

Bleeding during and after CPB may be caused or exacerbated by systemic heparinisation, hypothermia and haemodilution that are required for the surgical procedure and / or conduct of heart-lung bypass. Bleeding and coagulopathy are more common after prolonged bypass (greater than 120 minutes). Furthermore, CPB causes marked platelet dysfunction, in addition to the consumption of clotting factors, platelets and fibrinogen.

Despite liberal systemic heparinisation, clotting cascade activation still occurs in the extracorporeal circuit and gradually produces a consumptive coagulopathy. The combined insult of surgery and CPB can cause hyperfibrinolysis – though it is now common to prophylactically infuse antifibrinolytic therapy such as tranexamic acid, during the perioperative period in an attempt to reduce this complication.

Myocardial stunning and cardiac dysfunction

During cardiac surgery reduced myocardial contractility may be multifactorial. Patients with coronary artery disease - prior to revascularization - a reduction in the myocardial oxygen supply-demand ratio during anaesthesia may render areas of the myocardium ischaemic leading initially to reduced diastolic function and then reduced contractile function. During aortic cross-clamping, inadequate myocardial protection e.g. suboptimal cardioplegia delivery, may lead to areas of myocardial stunning defined as reversible reduction in contractile function not attributed to infarcted or ischaemic tissue. A greater degree of inadequate protection may produce areas of infarcted myocardium. Upon revascularisation, the myocardium may suffer further insult from reperfusion injury from the

production of oxygen free radicals. A key feature of myocardial stunning is that the impaired myocardium can be 'recruited' i.e. contractility improved, with the administration of inotropes. In open heart surgery, small air emboli may migrate along the native right coronary artery or the right coronary graft due to their upper most position, which can lead to inferior ST segment changes on the ECG and a degree of haemodynamic instability that usually spontaneously resolves.

Systemic Inflammation

The extracorporeal circuit of CPB induces a variety of inflammatory mediators that can result in a systemic inflammatory response picture following separation from bypass. The foreign material of the circuit activates tissue factor and factor XII, leading to initiation and amplification of the clotting cascade. Platelets are activated and degranulate causing platelet adherence and further propagation of the clotting cascade. Neutrophils are activated and release a variety of vasoactive inflammatory mediators and cytokines, which stimulates the complement pathway.

This clinically manifests as a 'warm shock' in the post- operative period with warm dilated extremities and a brisk capillary refill. There will be low filling pressures, low SVR and evidence of a reasonable cardiac output. Appropriate microbiological investigations and broad spectrum antibiotics may be appropriate as initially the clinical picture mimics septic shock which is conceivable in the setting of a concurrent infection that was not picked up pre-operatively. Furthermore, following CPB there is evidence of reduced humoral and cellular immunity that leaves individuals susceptible to new onset or worsening of existing infection. Optimal filling and vasopressor therapy are appropriate in the setting of a good cardiac output and a low SVR.

Pulmonary complications

During full cardiopulmonary bypass the lungs need not be ventilated. A variable amount of atelectasis is inevitable though sometimes lobar collapse (usually the left lower lobe which is ascribed to the weight of the heart compressing it) can occur. Technological advances have dramatically decreased the incidence of post-CPB ARDS though it still remains a potential complication. Postulated mechanisms include activation of inflammatory cascades including complement activation that produces a secondary ARDS. The alveolar capillary membrane permeability increases dramatically leading to non-cardiogenic pulmonary oedema. Treatment is supportive, with lung protective ventilation strategies and a restrictive fluid balance. It is important to rule out *cardiogenic* pulmonary oedema that may be due to a surgically correctable lesion or amenable to anti-heart failure medical therapy.

Macro- and micro-embolism

It is difficult to quantify the incidence of embolism during CPB given that there is likely to be a great proportion of subclinical events. Systemic emboli may affect the brain, heart, viscera or limbs. Patients with heavily calcified aortas are at increased risk of stroke – usually from dislodging a plaque during instrumentation of the aorta. Platelet and white cell aggregates from the extracorporeal circuit can be returned to the patient causing systemic embolism. Air emboli may be generated in the CPB circuit and also from within the surgical field. Indeed, it is fairly common to see transient inferior territory ST segment elevation and corresponding myocardial territory

hypokinesia coming off CPB that is due to air travelling up the right coronary artery (which is uppermost).

Hypothermia

Hypothermia is used in cardiac surgery for myocardial, neurological and end-organ protection by reducing cellular metabolic requirements. Though prior to separating from CPB patients are rewarmed to 37°C, they can often suffer rebound hypothermia due to regional blood flow differences during rewarming. Hypothermia has many detrimental consequences including:

- Increased incidence of arrhythmias
- Vasoconstriction leading to increased afterload and poor peripheral perfusion
- Coagulopathy due to platelet dysfunction and reduced clotting cascade enzymatic function
- Shivering which decreases myocardial oxygen supply and increases demand

Therefore, active rewarming methods should be employed (e.g. forced air warmers) to achieve normothermia.
Hyperthermia should be avoided as data from other patient groups suggests increased risk of neurological complications.