TUTORIAL

Weaning from Cardiopulmonary Bypass: Problems and Remedies

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Weaning a patient from cardiopulmonary bypass (CPB) is an important step of surgical procedures. It requires the anaesthesiologist to apply the basic principles of cardiovascular physiology and pharmacology. The goal is a smooth transition from the mechanical pump back to the heart as the source of blood flow. Weaning off CPB should always be conducted in a coordinated fashion. The surgeon directs the weaning process with input from anaesthesiologist and perfusionist. A skillful anaesthesiologist usually shares the control of weaning with the surgeon. Weaning from pump involves optimising cardiovascular variables. The time for optimisation is compressed to minutes or seconds, and decision must be made quickly to avoid myocardial injury or damage to the other major organ systems. The subject will be dealt under following headings.

1. Preparation for weaning
2. Final checklist before terminating CPB
3. What to look for during weaning?
4. Measuring cardiac function
5. Factors contributing to failure to wean

Preparation

Preparation for separation from CPB must be based on a clear understanding of the patients preoperative condition and the events of the operative course. Weaning off CPB is initiated after review and adjustment of certain patient linked variables, such as temperature, laboratory data, and data obtained from monitors.

Temperature

Core Temperature should be greater than 36°C before terminating the CPB. Shell temperature should be at least 33°C. Ending CPB when patient is cold causes prolonged hypothermia due to equilibration of the cooler, vessel-poor areas with the warmer better perfused vessel-rich areas, after active rewarming is discontinued. Nasaopharyngeal temperature correlates with brain temperature but may be artificially elevated during rapid rewarming and should not be used for determining the temperature at which CPB should be discontinued unless it is stable for 20–30 min. Venous return temperature can be used in a similar manner to help confirm the core temperature. The nasopharyngeal temperature should not exceed 38°C as this may increase the risk of postoperative central nervous system (CNS) dysfunction.

Laboratory data

CPB results in many metabolic abnormalities. Metabolic acidosis with considerable base deficit is likely to occur after prolonged CPB. Acidosis can depress the myocardial function, interfere with inotropic action and increase pulmonary vascular resistance (PVR). Serum sodium, potassium and ionised calcium should be measured. Routine administration of calcium after CPB is not recommended. Ionised calcium levels should be evaluated after rewarming to help direct therapy. Calcium levels are affected by pH. Low pH will increase calcium levels. Most studies suggest that administration of calcium produces an elevation in systemic vascular resistance (SVR) primarily, when the ionised calcium level is in low normal range or higher.[1] Calcium chloride administered in the doses of 5-15 mg/kg can counteract the negative inotropic action of hyperkalaemia. Patients on digoxin therapy can have life threatening dysrryhthmias after calcium administration. Augmentation of reperfusion injury is possible. Calcium should preferably be administered 15 min after aortic cross-clamp release.

In patients with poor renal function, repeated doses of hyperkalaemic cardioplegias may result

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in significant hyperkalaemia. Potassium can induce atrioventricular conduction block. Hyperkalaemia with potassium levels < 6 mEq/L does not require treatment in the presence of normal renal function. Hypokalaemia should be avoided before the termination of CPB.

Hyperglycaemia which is common during CPB usually returns to normal after CPB. However, hyperglycemia should be aggressively treated both during and after CPB with insulin in diabetics. Severe hyperglycaemia increases osmolality and induces osmotic diuresis and CNS dysfunction.

The optimal haemoglobin concentration during CPB is usually accepted as being 6-8 g/dL, although there is no minimum safe level. The haemoglobin concentration usually should be greater than 7.0 g/dL before terminating CPB. If it is less than this value, transfusion is advised to maintain oxygen carrying capacity after CPB. Patients with incomplete revascularisation, anticipated low cardiac output (CO) and end-organ damage may benefit from higher haemoglobin concentrations.

Coagualation abnormalities occur during and after CPB. Patients who are on antiplatelet drugs or in whom there is a 'long pump run' can have thrombocytopaenia. Fresh frozen plasma and cryoprecipitate must be available in the operating room, if clotting factor deficiencies are anticipated. Desmopressin can be used to increase platelet aggregation in patients with platelet abnormalities. These blood products and desmopressin should be given only after complete heparin reversal. Post bypass bleeding is usually due to three factors, inadequate surgical haemostasis, inadequate heparin reversal or platelet dysfunction.

The Final Check List

The weaning process should be gradual as the cardiac function is usually not normal at the end of CPB. The final check list may be as follows:

1. Oxygen flow meter must be on, supplying 100% oxygen.
2. Lungs are ventilated with 100% oxygen – confirm visually.
3. Note the quality of breath sounds.
4. All laboratory data (as mentioned above) within acceptable limits – optimal metabolic state.
5. Patient's temperature – sufficiently rewarmed.
6. Proper de-airing of heart and great vessels.
7. All equipments and drugs are ready. (Always keep an extra syringe pump for an unanticipated emergency use)

What to Look for During Weaning

It is important to restart all the monitors like pulse oximeters, capnometer, apnoea alarms, oxygen and ventilator alarms. Plethysmography takes time to appear when sensors are placed on extremities. A pulse oximeter waveform appearing immediately after termination of CPB is always a sign of good peripheral perfusion and adequate rewarming. There might be a larger gradient between end-tidal carbon dioxide ($EtCO_2$) and arterial carbon dioxide tension ($PaCO_2$) at the end of bypass. A rapidly increasing height of capnogram is a sure sign of good CO during the termination of CPB.

Pressure transducers should be zeroed and calibrated and their levels should be checked in relation to the operating table. Pressure waveforms are best displayed using overlapping traces with identical scales. Coronary perfusion pressure may be estimated by the vertical height difference between the arterial diastolic pressure and pulmonary capillary wedge pressure (PCWP) or left atrial pressure (LAP). The vertical height between the pulmonary artery mean pressure and central venous pressure (CVP) waveforms might estimate the right ventricular work. The slope of the rise in central aortic pressure during systole may give some indication of left ventricular (LV) contractility. Valvular regurgitations can be diagnosed by the detection of 'V' waves during the diastolic phases on waveforms of filling pressures. A decreased pulse pressure suggests LV failure. Radial artery pressures may not be accurate following CPB. During the first 30 minutes, the radial artery tends to underestimate both the systolic and mean central aortic pressure.[2] Clinically significant radial artery hypotension should be confirmed with central aortic pressure

measurement. A kinked arterial catheter or extension tubing should also be ruled out. Visual inspection of heart should be done at every point. A vigorously contracting heart with abnormal arterial trace and an arterial line which is difficult to aspirate, should lead to suspicion of a kinked catheter or line.

In patients with good preoperative LV function, PCWP of 8-12 mm Hg or CVP of 6-12 mm Hg is often normal. In non-compliant ventricles higher filling pressure (>20 mm Hg) may be needed to achieve higher filling volumes. In such cases both left and right sided filling pressures should be monitored. The CVP/LAP ratio should be observed. The normal ratio is less than 1. If the ratio is more than 1, interventricular septum may be pushed to the left, limiting the LV filling and decreasing CO.

ECG should be carefully assessed for rate, rhythm, conduction and evidence of ischaemia. Cardiac rate and rhythm must be optimised. Heart rate of 80-100 beats/minute often is needed for adequate CO. Patients who have undergone coronary artery bypass graft (CABG) surgery might tolerate heart rates in the higher range after revascularisation than prior to the bypass. Patients with severely limited stroke volume (after ventricular remodeling) may require even higher rates. Sinus tachycardia should be treated (>120/min) before terminating the CPB. Adequate preload should be tried first to see whether heart rate can be reflexly decreased. Other causes include light planes of anesthesia (Fast-tracking), hypoxia, hypercapnia, inotropes, anaemia and ischaemia.

Normal Sinus rhythm is always preferable. In patients with left ventricular hypertrophy, sinus rhythm is essential as 'atrial kick' can contribute up to 40% of CO. Atrial pacing may be considered if there is no atrioventricular conduction block. Supraventricular tachycardias and atrial fibrillation (acute) should be cardioverted. Esmolol, calcium channel blockers and adenosine may also be used. Some of these agents may decrease contractility. Digoxin is effective but has long duration of onset of action. Presence of new ventricular dysrrhythmias should arouse suspicion of ongoing ischaemia in the absence of any electrolyte abnormalities. Amiodarone can be used in the pump if there are intractable ventricular arrhythmias. It can produce severe depression of ventricular function.

Measuring Cardiac Function

Inspection of the heart visually or by echocardiography provides valuable information about contractility, wall motion abnormalities, conduction, preload and valvular function.

Cardiac function may be assessed by measuring CO. A cardiac index (CI) of more than 2.0 L/min/m^2 is ideal for termination of CPB. If heart rate is high, a normal CO can exist despite a low stroke volume, therefore calculation of stroke volume index might be useful. Signs of adequate tissue perfusion should be present as soon as CPB is terminated. A good plethysmograph from a sensor placed on the finger is a sure sign of excellent peripheral perfusion. Arterial blood gases and pH should be measured just before CPB is terminated. Lactic acidosis and gas exchange abnormalities should be ruled out. Mixed venous oxygen saturation (SVO_2) or tension measurement provides a good guide for CO assessment. Many heart-lung machines have the provision to monitor continuous SVO_2. Decreased urine output after CPB should be evaluated immediately. A mechanical defect in the urinary drainage system should be ruled out. A distended bladder on visual inspection and low serum potassium should arouse suspicion of a kinked catheter. The ideal perfusion pressure for adequate tissue perfusion should be individualised. Patients with renal insufficiency, cerebrovascular disease or hypertension may require higher perfusion pressures, but at the risk of increased bleeding.

Factors Contributing To Failure To Wean

Total CPB is physiologically a circulation in series. Blood flows from vena cavae to oxygenator and to aorta. Partial CPB converts circulation to parallel. Blood flows from vena cavae both into right ventricle and oxygenator and into lungs and aorta. Termination of CPB finally converts the circulation back into series. The duration of parallel circulation is determined by the ability of the ventricle to withstand the partial preload diverted

to heart and sustain the circulation. It is important to realise that the CPB performs basically two vital functions, respiration and circulation. Inability to restore any of these two functions can potentially lead to failure to wean from CPB.

Respiration

Restoring respiration and establishing ventilation and oxygenation are the most important steps during weaning from CPB. Analgesic and anaesthetic requirements are increased during rewarming phase. Benzodiazepines should be added to avoid awareness during rewarming. Vaporisers should be turned off 10 min. prior to termination of CPB. Anaesthesia machine should be switched on. All alarms must be reactivated. Adequate oxygen should be flowing through the machine and the airway circuit should be properly connected. Failure to wean from CPB can occur due to inadvertent continuous administration of volatile anaesthetic during weaning from CPB.

The lungs should be inflated manually to evaluate compliance and eliminate macroatelectasis. Lungs should be examined for both inflation and deflation and re-expanded with two or three sustained breaths (10-15 sec each) to a peak pressure of 30-35 cm H_2O with visual confirmation of bilateral lung expansion and resolution of atelectasis. In patients with internal mammary artery grafts, care must be taken to prevent lung over distension, which may cause graft avulsion. Patients with a history of bronchospastic disease can have increased airway resistance during weaning from CPB. An inadvertently distended left ventricle during CPB or persistent L to R shunt on CPB can decrease lung compliance and increase airway resistance. Pre-existing cardiac conditions like large L to R shunts and obstructed anomalous pulmonary venous connections can cause bleeding into lungs, leading to decreased compliance. An increased LAP either due to depressed cardiac function or mechanical mitral valve obstruction can lead to acute pulmonary oedema and decreased pulmonary compliance. Pneumothorax or pleural effusion can also impair lung function. Inspection of lungs for bilateral inflation is very essential as unilateral inflation or lung collapse can lead to severe hypoxaemia immediately after termination from CPB. This is particularly important in children and short statured adults. Endotracheal suction may be required and this should be done carefully in the anticoagulated patient to avoid mucosal trauma and bleeding. Prophylactic use of beta agonists in both intravenous and inhalational forms should be considered to treat increased airway resistance and slow deflating lung. CPB should be continued until the airway pressures return to normal. In patients undergoing reoperations pleural adhesions might hinder evacuation of pleural effusion accumulated during CPB.

The timing of initiation of mechanical ventilation is controversial. Some physicians believe that ventilation should begin when pulsatile flow resumes in order to avoid hypoxaemia. However, the changes in arterial blood gases are well within the limits of clinical acceptability and do not necessitate ventilation while pulsatile flow is resumed during full CPB.[3]

The pulse oximeter or the CPB circuit venous oxygenation also can be used to assess the need for ventilation during partial CPB. Sometimes overzealous ventilation on partial CPB can lead to severe respiratory alkalosis, an unwanted physiological effect during rewarming.

Circulation

Termination of CPB is a stressful period, needing the restoration of complete and efficient mechanical activity of heart, which might be still recovering from a physiological insult of chemical quiescence and surgical trauma. In the presence of normal respiratory mechanics, the mechanical and electrical activity of the heart is responsible for maintaining the oxygenation of tissues. It is the duty of the anesthesiologist along with the other members of the team to be vigilant and quick to respond to any indications, suggestive of difficulty in the weanning process.

Difficulty in weaning form CPB can be either anticipated or unanticipated. In a retrospective study,[4] patients were grouped into three groups depending on the level of difficulty to wean from

CPB. In group A, patients offered no difficulty to discontinuation from CPB. In these patients pump flow was gradually reduced and stopped and venous line clamped. Final adjustment of cardiac output is made off pump, by slowly administering residual volume from the oxygenator until ideal preload is attained.

In group B, patients had mild to moderate degree of cardiac dysfunction. They required some support to disconnect from the pump. These patients required elaborate preparations prior to termination of CPB. CO was estimated before turning off CPB. Preload was adjusted according to the filling pressures. Inotropes were started. Pump flow was reduced only after making sure that interventions made were working. Pump flow was decreased in small increments after waiting for sufficient time at each step. CO was assessed with the help of LAP or PCWP and/or transoesophageal echocardiography (TOE). Pump was stopped only after making sure that cardiac function was stable for adequate time with minimal support from CPB. A good rule is, "The first attempt to switch off CPB is the best one".

In group C, patients had severe ventricular dysfunction that proved difficult to terminate CPB, despite physiological and pharmacological support. In these patients CPB was prolonged. Intense pharmacological support in combination with rest to the myocardium may convert some of these patients into group 'B'. These non-responders may eventually need mechanical circulatory support. Patients in this group may be recategorised by the use of TOE by the end of rewarming. Patients showing some cardiac activity on TOE, might be able to separate from CPB after prudent pharmacological and mechanical support using intra-aortic balloon pump (IABP). Patients showing no cardiac mechanical activitiy should go on to mechanical circulatory support.

Myocardial function continues to be the single most important determinant of successful weaning from CPB. It is crucial for the anaesthesiologist to gather information from preoperative charts about the extent of myocardial dysfunction and take into account the intraoperative events like effectiveness of myocardial protection and adequacy of surgical repair, to plan the treatment options for weaning from CPB. In a review of 12,471 patients Christakis et al[5] highlighted the predictors of mortality following CABG surgery in relation to preoperative LV function. Female gender, left main stenosis, emergency surgery, and reoperation were predictors of mortality in patients with normal (>40%) ejection fraction (EF). The type of myocardial protection used was found to be an important predictor of mortality in patients with EF between 40% and 20%. In patients with EF <20%, the only predictor of death was emergency surgery. In a retrospective study[6] in patients who underwent CABG surgery, the predictors for the need for inotropic support are preexisting low EF, dilated ventricles, high left ventricular end diastolic pressure (LVEDP), long aortic cross clamp time, old age, and female gender. It has been clearly shown in a number of studies that there is a decline in the ventricular function immediately after CPB, making the time during termination of CPB a vulnerable point. Ventricular function is impaired by aortic cross clamping. The ischaemia due to cross clamping can result in myocardial stunning. In cases of acute myocardial infarction ventricular function might improve due to revascularisation. But this benefit might be offset with the possibility of myocardial stunning due to cross clamp or cardioplegia. Ventricular function usually improves after valve replacement. Ventricles with chronic aortic regurgitation suffer from prolonged volume overload resulting in eccentric ventricular hypertrophy. In patients with mitral regurgitation the acute afterload mismatch produced by replacing the mitral valve and removing the low pressure relief effect frequently results in ventricular dysfunction.

Objective evidence of adequacy of surgical repair should always be obtained before termination of CPB. The surgeon remains an important source of information in this regard. For CABG surgeries, the quality of distal anastamosis, degree of under revascularisation, and presence of badly diseased coronaries are important factors that determine the difficulty in CPB termination.

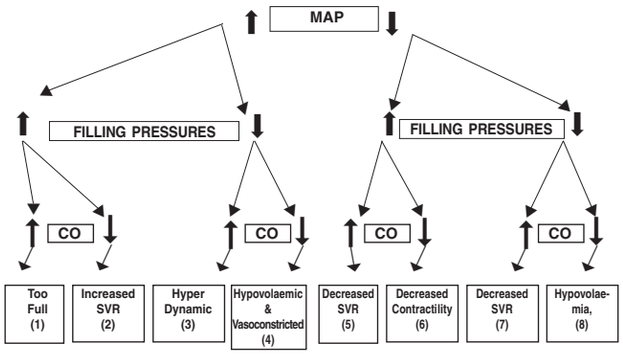
The commonest reasons for failure to establish adequate circulation at the termination of CPB may be due to:

- LV failure
- RV failure
- Inappropriate vasodilation

Left Ventricular Failure

Treatment: The causes of LV failure are shown in table 1. Ephedrine 10-20 mg or 10 µg of bolus epinephrine is often given to increase contractility before commencing the infusion.

Epinephrine or dopamine may be appropriate if the heart rate is normal and SVR is low or normal. Dobutamine and milrinone may be more appropriate if SVR is increased. Norepinephrine or phenylephrine may need to be added to milrinone if SVR is low. Nitroglycerin should be started if ischaemia is present. Figure 1 shows the algorithm for diagnosing various haemodynamic causes and their management during weaning from CPB.



TREATMENT -

Situation (1): Wait, try diuretics, venodilators
Situation (2): Use inodilators or vasodilators
Situation (3): Wait, Deepen plane of anaesthesia
Situation (4): Use volume and vasodilators
Situation (5): Wait, vasoconstrict if blood pressure is too low
Situation (6): Check preload, use inodilator, "go back" on pump, IABP, LVAD
Situation (7): Use vasoconstrictors
Situation (8): Use volume, for RV failure consider iNO, PGE1, RVAD

Fig. 1. Schematic representation to assist the diagnosis and treatment of haemodynamic disturbances during weaning from CPB
IABP: intra-aortic balloon pump, LVAD: left ventricular assist device, INO - inhaled nitric oxide, PGE: prostaglandin E, RVAD: right ventricular assist device

Table 1. Causes of left ventricular failure

Ischaemia	A. Graft Failure	B. Inadequate Coronary Blood Flow Myocardial Damage	C. Myocardial Ichaemia Leading To
	1 Clotted graft,	1. Incomplete revascularisation	1. Incomplete myocardial preservation during CPB
2. Distal graft occlusion	2. Inadequate coronary perfusion pressure		2. Evolving myocardial infarction.
	3. Air in the graft	3. Emboli in native coronary arteries	
	4. Kinked graft	4. Coronary spasm	
Valve failure	A. Prosthetic Valve 1. Sewn in reverse 2. Paravalvular leak 3. Immobile disk	B. Native Valve 1. Acute MR (Papillary muscle dysfunction)	
Gas exchange problems.	A. Hypoxaemia 1. Inadequate FiO_2 2. Mechanical Ventilator failure 3. Airway disconnected 4. Bronchospasm 5. Pump lung	B. Hypoventilation 1.Leak around ET tube 2.Tube moved up in trachea 3.$ETCO_2$ port disconnection	
Inadequate preload			
Reperfusion injury			
Miscellaneous causes of reduced contractility	A. Medications (-blockers) B. Acidosis C. Electrolyte disturbance D. Pre-existing LV failure.		

Right Ventricular Failure

Treatment: The causes of RV failure are shown in table 2. Any signs of RV ischaemia should be treated. Coronary air embolism must be treated with epinephrine boluses (10-50 µg) into the pump before termination of CPB. Wait for ECG to return to normal before weaning. Patients with pulmonary hypertension need strategies to manipulate pulmonary vascular resistance (PVR). Hyperventilation, accomplished by high respiratory rate, (because high tidal volume increases PVR) can decrease PVR. Avoid hypoxaemia and acidosis. Higher preload is needed. Consider inhaled nitric oxide or prostaglandin infusion for reducing PVR. Resistant RV failure might necessitate the use of RV assist device.

Table 2. Causes of right ventricular failure

1.	Pulmonary Hypertension
	Chronic mitral valve disease
	L-R shunts
	Pulmonary embolism
	Acute mitral regurgitation
2.	Air Embolism of right Coronary artery
3.	RV Ischaemia or Infarct
4.	RV Outflow Obstruction
5.	Tricuspid Regurgitation

RV: right ventricle, L-R: left to right

Inappropriate Vasodilation

It prevents achievement of an adequate blood pressure despite an acceptable CI. The commonest causes are, pre-existing medications (angiotensin converting enyme (ACE) inhibitors), electrolyte abnormalities, acid–base disturbances, sepsis, idiopathic conditions (factors related to CPB) and hyperthermia. Treatment includes use of vasopressors like norepinephrine, phenylphrine and vasopressin. It is important to avoid use of inodilators in these patients.

Some of the 'predictors' for failure to wean from CPB are :

1. Preoperative EF < 0.45 or diastolic dysfunction
2. Female patient undergoing CABG (tendency for incomplete revascularisation due to smaller and more diseased coronary arteries)
3. Elderly patient
4. Use of ACE inhibitors
5. Ongoing ischaemia or evolving infarct in pre-CPB period.
6. Prolonged CPB (>2-3 hours).
7. Incomplete revascularisation (small vessels or distal disease)
8. Valve replacement with small valve or suboptimal valve repair
9. Incomplete myocardial preservation during cross-clamping
   a) ECG not asystolic
   b) Prolonged ventricular fibrillation prior to cross clamp
   c) Warm myocardium
      i. LV hypertrophy
      ii. High grade coronary artery stenosis
      iii. Choice of grafting order
      iv. Noncoronary collateral flow – washout of cardioplegia
      v. Poor LV venting – LV distension.

Preparations For High-risk Patients

1. It is a good practice to have ready syringes of ephedrine (3 mg/ml)and epinephrine (10 µg/ml), so that boluses can be used until decision to start a continuous infusion is made.
2. Need for additional monitoring (LAP, PA pressure) considered.
3. All inotropes available on the shelf (like amrinone, milrinone) with some extra supply of syringe infusion pumps.
4. IABP must be inside the OR, switched on and ECG source connected (always connect a second ECG cable in patients with preoperative LV dysfunction for rapid insertion of IABP). Presence of femoral arterial line will make the IABP insertion easy before terminating CPB.
5. Inotropes must be started and IABP inserted early when difficulty is anticipated and confirmed with TOE.
6. Early use of IABP should be preferred over incremental doses of one or more inotropes, so that the heart rates remaing in physiological limits.
7. Preemptive use of milrinone has been shown

to improve cardiac function during and after cardiac surgery.[7]

Weaning from CPB, should be a co-ordinated effort and adequate preparations should be made in patients with anticipated difficulty to wean. Problem situations during weaning include excessive vasodilation, vasoconstriction, acute haemodynamic deterioration and hypoxaemia. An efficient and vigilant team will be successful in weaning difficult cases from CPB if prudent physiological concepts are applied.

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