Chapter 4 Circulatory Shock

Stephen Lo

Introduction

Shock is a state where there is inadequate perfusion and therefore delivery of oxygen to the tissues. It is important to know that while blood pressure is a surrogate marker of tissue perfusion, it cannot be used in isolation as the sole determinant marker as it is crude and certainly not the most sensitive clinical sign. In this chapter, we will discuss the causes of shock as well as how to recognise it.

Causes of shock

There are 4 broad categories of shock:

1. Hypovolaemia
2. Obstructive shock
3. Cardiogenic shock
4. Distributive shock

Hypovolaemic shock

This where there is reduction in intravascular volume, leading to a reduction in the preload of the heart (the volume of the left ventricle at the end of diastole), leading to a reduction in stroke volume and therefore cardiac output. In isolated hypovolaemic shock, the compensatory mechanisms of the body are normally intact and are in the form of the sympathetic nervous system, where the body will defend the blood pressure. This is why blood pressure is a late sign of shock and cannot be used as a sole basis to determine shock.

Below is a table from the ATLS guidelines that show clinical signs associated with different volumes of blood loss.

Classes of haemorrhagic shock (as per ATLS)				
	I	II	III	IV
Blood loss (ml)	<750	750-1500	1500-2000	>2000
Blood loss (%blood volume)	<15%	15-30%	30-40%	>40%
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate (/min)	14-20	20-30	30-40	>35
Urine output (ml/hr)	>30	20-30	5-15	Negligible
Mental state	Slightly anxious	Mildly anxious	Anxious,	Confused,

			confused	lethargic

Note that you can lose 750ml of blood volume without clinical signs, and that blood pressure will only decrease after 1.5L of blood loss. There are two populations where blood pressure are even harder to interpret: the elder and the paediatric patient.

Elderly population

The elderly, because they are very likely to have pre-existing hypertension, which means a normal blood pressure of say systolic of 120 is actually relative hypotension for an 80 year old. They are also likely to be on medications such as beta blockers, which may mean they may not be able to mount a tachycardia, meaning that their heart rate may not be elevated even if they are in significant shock.

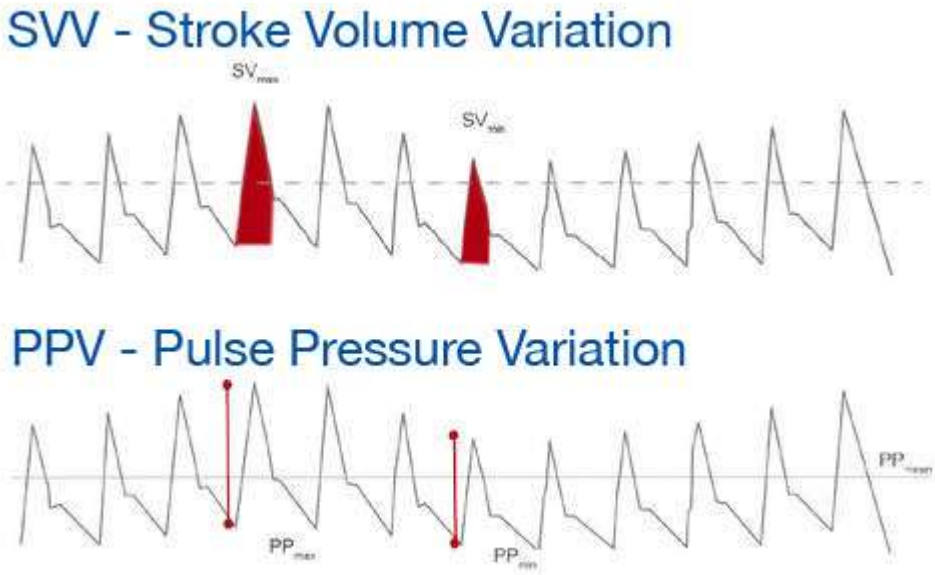
Paediatric population

The paediatric population have exceptionally good compensatory mechanisms and therefore hypotension is a very late sign of shock. Heart rate is an important sign, but also capillary refill, peripheral perfusion, presence of irritability are earlier signs of shock.

Further monitoring and testing

As well as clinical signs, there are bedside monitoring and testing that can help you to diagnose hypovolaemia

1. Central venous pressure (CVP): Studies have shown poor correlation of the CVP with the volume status of patients. Some people still use the trends to monitor fluid status.
2. Arterial blood pressure monitoring and pulse pressure variation: Inspiration and expiration of the patient leads to an altered preload. In a spontaneously breathing patient, during inspiration, the intrathoracic pressure drops and venous return and therefore blood pressure, increases. During expiration, venous return drops and therefore the blood pressure drops. Pulse pressure variation of 13% during inspiration and expiration is associated with a blood pressure response to fluid. Pulse pressure is the difference between systolic and diastolic blood pressure). The bigger the variation, the most likely the fluid responsiveness. In reality, we do not do calculations of pulse pressure on patients, but rather, look at the beat to beat variation of the arterial pressure tracing as the patient ventilates. A big difference, known as a “swing”, is a marker of fluid responsiveness.



3. PICCO™ and Pulsed contour analysis: This is basically the use of a proprietary software and machine that analysis the contour of the arterial waveform and from this derives cardiac output, stroke volume variation (which is similar to pulse pressure variation) and systemic vascular resistance. PICCO calibrates the pulse contour to a thermal dilution method to obtain cardiac output, but some (Vigileo tm) just purely analyse the pressure waveform.
4. Echocardiogram: Transthoracic echo can estimate volume status by looking at the inferior vena cava diameter and compressibility as well as looking at the end diastolic diameter of left ventricle.

Treatment of hypovolaemia

Ensuring ABC is essential to keep the patient alive. In terms of hypovolaemic shock, it is important to have good IV access and fluid resuscitation. The type of resuscitation fluid (crystalloid or colloid) is not important. In the context of trauma, blood should be considered after 2-3L of intravenous fluid. For other cases, monitor the haemoglobin and aim for Hb>70g/L if they are not actively bleeding, or Hb>90g/L if they are. If they are haemorrhaging actively, you need to also consider the use of FFP, platelets, cryoprecipitate. If they are haemorrhaging rapidly, a massive transfusion protocol can be activated, and boxes of blood products will be delivered and can be given as they arrive. Vasopressors maybe needed to temporize the blood pressure, but it is not the end treatment, and should be only used to bridge between fluid resuscitation and bleeding source control. In a massive transfusion scenario, the other aspects that need to be considered include:

- Core temperature, keeping patients warm to prevent coagulopathy
- Watch for acidosis
- Consider anti-fibrinlytics, FVIIa, in addition to replacement of coagulation factors that are part of the massive transfusion protocol
- Watch for hypocalcaemia and hyperkalaemia that can occur in massive transfusion

While fluid resuscitation is initiated, the cause of hypovolaemia needs to be treated. In the context of trauma, this means identifying and treating the source of the bleeding. This means theatre/interventional radiology/endoscopy should be notified early.

Obstructive shock

This is also known as extracardiac shock. These are shock caused by pathologies around the heart and lungs. These include:

1. Tension pneumothorax
2. Pulmonary embolism/fat embolism
3. Pericardial tamponade

Tension pneumothorax

This occurs where a hole in the lung acts as a one way valve and air can enter the pleural space but cannot come out. As a result, pressure builds in the pleural cavity leading to a shift of the mediastinum causing obstruction flow from the vena cava and therefore reducing the return of blood to the right heart causing hypotension.

Important potential distinguishing signs of pneumothorax are distended neck veins, tracheal shift, unilateral air entry and hyper resonance to percussion (though hard to perform on a critically ill patient).

Confirmatory tests

A tension pneumothorax is a medical emergency and waiting for a chest X-ray may not possible if the patient is haemodynamically compromised, and therefore emergency decompression is needed. However, sometimes a bedside ultrasound of the lung may be feasible if there is doubt about the diagnosis and if the ultrasound is readily available. The aim of the ultrasound is to identify the sliding pleura, which is absent in a pneumothorax. If the patient is stable, then a CXR can be performed.

Therapy

An emergent decompression is required. This is common in the form of a needle decompression. This can be done by inserting a 14-16G IV cannula into the 2 intercostal space, mid-clavicular line, while aspirating for air with a syringe. Alternatively, a micro-thoracotomy can be performed at the safe triangle (between the anterior and mid axillary line, above the 5^{th} rib), after which a definitive intercostal drain can be inserted.

Pulmonary embolus

Normally, the thrombus is formed in the lower limbs, before it embolizes to the lung. Risk factors include hypercoagulability states, immobility and vascular irritability (Virchow's triad).

History is important if it is obtainable. Clinical signs include hypoxia, tachypnoea, tachycardia, raised JVP and shock.

Investigations include ECG which may show the classical S1QIIITIII, or though this not sensitive. Other changes include RBBB or Af, but the most common feature is tachycardia. Stable patients can be investigated with a CTPA, however unstable patients with clinical

suspicion maybe diagnosed on the basis of a bedside transthoracic with evidence of right ventricular dysfunction and dilatation.

Therapy

Patients who are cardiovascular stable, the first line therapy is heparin anticoagulation. This can be in the form of fractionated or LMWH. An alternative is an IVC filter and these are indicated for people who have:

1. Treatment failure on anticoagulation
2. Anticoagulation contraindicated

Thrombolysis can be beneficial in patients who are stable, but have evidence of right ventricular strain, but this benefit is in terms of morbidity, not mortality.

For haemodynamically unstable patients, thrombolysis should be given. If thrombolysis is contraindicated, then consideration needs to be given to surgical embolectomy, although this is rarely down by the cardiothoracic surgeons.

Pericardial tamponade

This is a complication that is seen in patients with cardiac surgery, and therefore must be considered in these patients who have post-op shock.

Clinical signs included distended neck veins and poor peripheral perfusion. Pulsus paradoxus may be present, but this will present in the ICU in the form of an arterial blood pressure "swing" similar to the one seen in hypovolaemia.

Monitoring and tests will show a raised CVP. A pulmonary artery catheter may show raised pulmonary diastolic pressures and a drop in cardiac index (ie. The cardiac output). The definitive diagnosis is made with a Transoesphageal/transthoracic echocardiogram.

Therapy

If the patient has arrested, than an emergent sternotomy is indicated. As a registrar, you may be the person that will perform this. We will have simulations during your orientation to train you for this procedure.

If the patient is shocked, but supportable, then surgeons, OR needs to be notified and patient needs to go back to theatre for an emergent chest reopening.

Cardiogenic shock

This is where the primary cause of the shock is due to cardiac dysfunction. This can be divided into:

1. Heart rate and rhythm
2. Myocardial wall dysfunction
3. Regurgitant lesions
4. Obstructive lesions
5. Shunts

Heart rate and rhythm

Tachycardia, bradycardia and arrhythmias can all cause shock. The purpose of this section is not to describe each rhythm in detail, but a general outline of how to manage these conditions.

Bradycardias

It is important to determine what rhythm the patient is. Possibilities include sinus bradycardia, junctional bradycardia or varying degrees of heart block.

In the non-cardiac patients, consider:

- Underlying cause: Is this a primary cardiac problem or a secondary problem? Primary cardiac problem include structural heart disease such as ischaemic heart disease, cardiomyopathy, primary conduction problem. Secondary problems include drugs, electrolyte disturbance, vagal response, raised intracranial pressure.
- Drug treatment: This includes atropine, glycopyrrolate, which are anticholinergics, or sympathomimetics including dopamine, dobutamine, isoprenaline, adrenaline.
- Pacing: If there is symptomatic bradycardia despite drug therapy, then pacing needs to be initiated. In an emergency situation, the patient may need cutaneous pacing, although if the patient is alert, sedation and analgesia need to be given. Otherwise temporary venous pacing wires will need to be inserted by the cardiologists.

In cardiac patients, often pacing wires will have been placed, as the risk of bradyarrhythmias is high. If there is haemodynamic compromise, either in terms of blood pressure or cardiac output, then the pacing rate can be increased. It is important to note that it is common to have only ventricular wires post-op and therefore there is loss of atrial kick. In some instances, patients may have better haemodynamics with a slow sinus rhythm, rather than a fast ventricularly paced rhythm.

Tachycardia

This can be divided to narrow complex tachycardia or broad complex.

Narrow complex tachycardia

Defined as a rhythm with a rate of >100/min, with a QRS complex <120ms. It originates from the conduction system upstream to and including the AV node, whereas broad complex tachycardia originates from below the AV node.

Causes of narrow complex tachycardia include:

- Supraventricular tachycardia (SVT), not Atrial fibrillation or flutter
    - Sinus tachycardia
    - Sinus nodal re-entrant tachycardia
    - Atrial tachycardia
    - AV node re-entrant tachycardia
- Atrial fibrillation
- Atrial flutter

Broad complex tachycardia

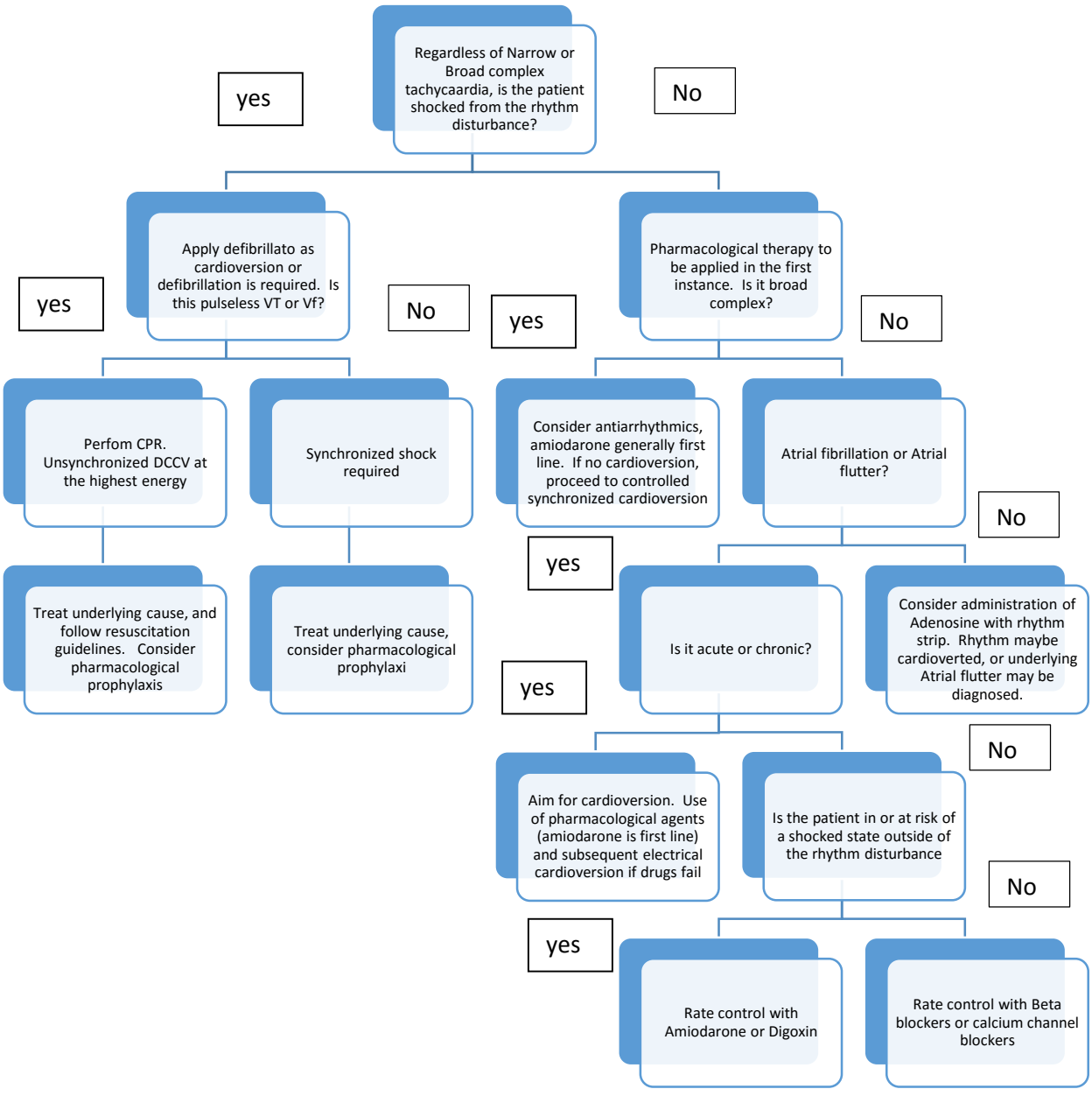
Defined as rate of >100/min with QRS>120ms. It originates from below the AV node.

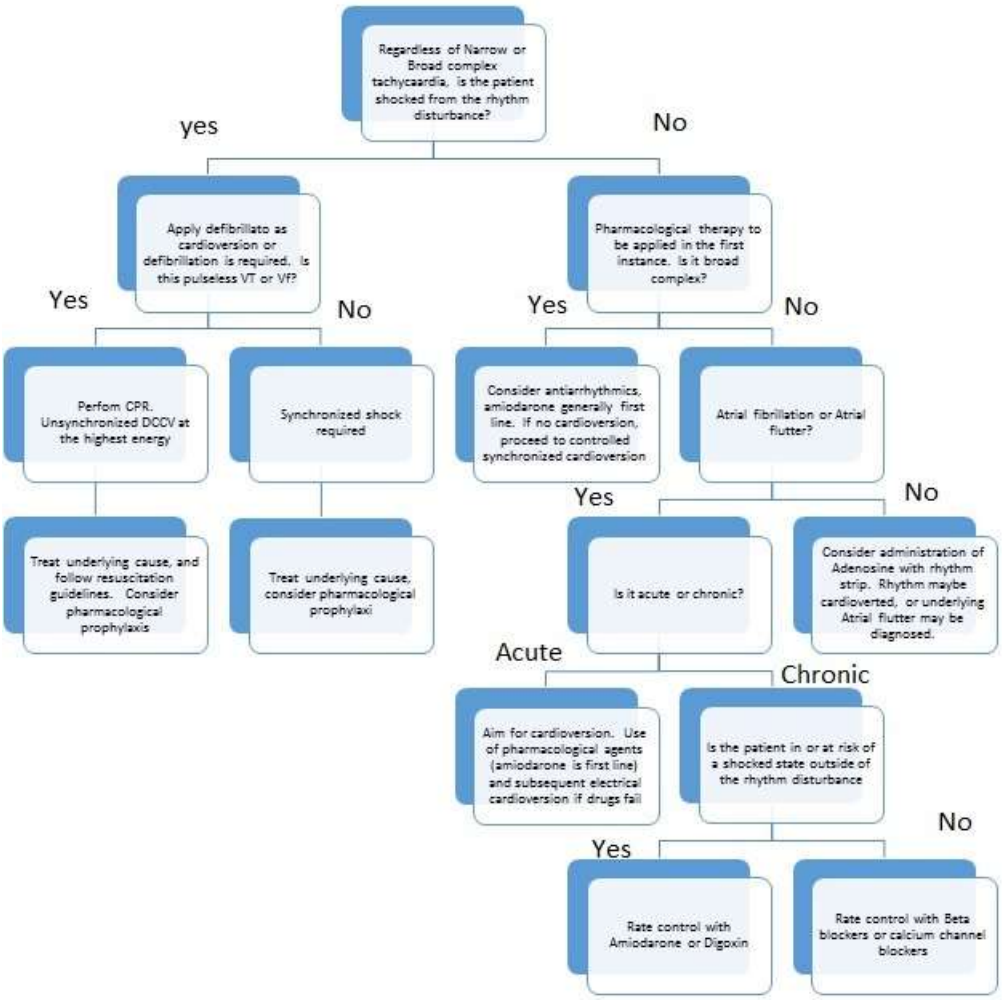
Causes include:

- Atrial tachycardia with aberrant conduction
- Ventricular tachycardia
- Ventricular fibrillation

Management of tachycardias in the ICU setting

It is important to diagnose and treat the underlying cause of the tachycardia in addition to the tachycardia itself. Treatment of the rhythm itself is dependent on whether the patient is haemodynamically stable or not.


Regardless of Narrow or Broad complex tachycaardia, is the patient shocked from the rhythm disturbance?
yes
No
Apply defibrillato as cardioversion or defibrillation is required. Is this pulseless VT or Vf?
yes
No
Pharmacological therapy to be applied in the first instance. Is it broad complex?
yes
No
Perfom CPR. Unsynchronized DCCV at the highest energy
Synchronized shock required
Consider antiarrhythmics, amiodarone generally first line. If no cardioversion, proceed to controlled synchronized cardioversion
Atrial fibrillation or Atrial flutter?
Treat underlying cause, and follow resuscitation guidelines. Consider pharmacological prophylaxis
Treat underlying cause, consider pharmacological prophylaxi
yes
No
Is it acute or chronic?
Consider administration of Adenosine with rhythm strip. Rhythm maybe cardioverted, or underlying Atrial flutter may be diagnosed.
yes
No
Aim for cardioversion. Use of pharmacological agents (amiodarone is first line) and subsequent electrical cardioversion if drugs fail
Is the patient in or at risk of a shocked state outside of the rhythm disturbance
yes
No
Rate control with Amiodarone or Digoxin
Rate control with Beta blockers or calcium channel blockers



Haemodyanmically compromised from rhythm

In treatment of any abnormal rhythm with tachycardia, consideration is whether there is significant haemodynamic compromise from the rhythm disturbance. This is because in the ICU, many patients are already haemodynamic compromised and requiring inotropes and so in this instance, consideration needs to be given to the change in the dose of inotropes required. Electrical defibrillation/cardioversion is the first line treatment in significant haemodynamic disturbance. If the patient is conscious, then a careful anaesthetic needs to be given. In **any instance**, if the patient is pulseless, CPR needs to be commenced and electrical cardioversion is required. Treatment is then directed towards the underlying

cause, with the correction of electrolyte (in particular potassium), and consideration of prophylaxis such as magnesium and antiarrhythmics.

Haemodynanmically uncompromised

This means that there is time for pharmacological therapies to be given, an elective cardioversion can be considered if pharmacological methods fail.

If the rhythm is acute, then the goal is to cardiovert the patient. The first line therapy in general is electrolyte correction and Amiodarone, with the exception of SVTs which is not Af/A flutter, where Adenosine can be considered. Adenosine can revert SVTs and sometimes unmasks atrial flutter that may appear as a simple SVT. Adenosine should not be given to patients with significant asthma as it can cause bronchospasm. If this fails then an elective cardioversion should be considered. This should be in consultation with the consultant in charge.

If the patient is in chronic atrial fibrillation or flutter, then the main aim is to rate control patients. In patients who are unstable from other conditions, e.g. sepsis, than rate control drugs with a less tendency to cause hypotension should be used. These include amiodarone and digoxin. Should there be no concern of hypotension, and sometimes even hypertension, than beta blockers and calcium channel blockers should be considered.

Myocardial wall dysfunction

This is where the heart fails to pump effectively to deliver blood to the vital organs. The common causes of acute and potentially reversible myocardial wall dysfunction seen in the ICU include:

1. Myocardial ischaemia and infarction
2. Drugs
3. Metabolic derangement e.g. severe acidosis
4. Septic cardiomyopathy
5. Myocardial stunning post cardiac arrest
6. Post-cardiac surgery (e.g. prolonged bypass, inadequate myocardial protection)
7. Infection related cardiomyopathy (specific organisms particularly viral infections)
8. Valve related

This can involve both the right and left ventricle. For the left ventricle and can be divided into systolic dysfunction where the heart can't squeeze and pump, and diastolic dysfunction, where the heart can't relax and fill.

Assessment of cardiac function

Assessment of metabolic derangement can be done by doing electrolytes and blood gas, and an ECG may give you a clue to the possible causes of poor cardiac function. However, the best way to assess cardiac function is the echocardiogram. This can potentially diagnose the cause of myocardial dysfunction, quantify the dysfunction as well as making measurements of cardiac output. Other ways of monitoring can include cardiac output monitors such as pulse contour analysis, pulmonary arterial catheter with intermittent or continuous thermal

dilution. Blood tests such as lactate and mix venous saturation can also be used to determine whether there are signs of poor organ perfusion.

Management of myocardial dysfunction

The key is to treat the underlying cause of the myocardial dysfunction. In the meantime, supportive management should be instituted. This means that:

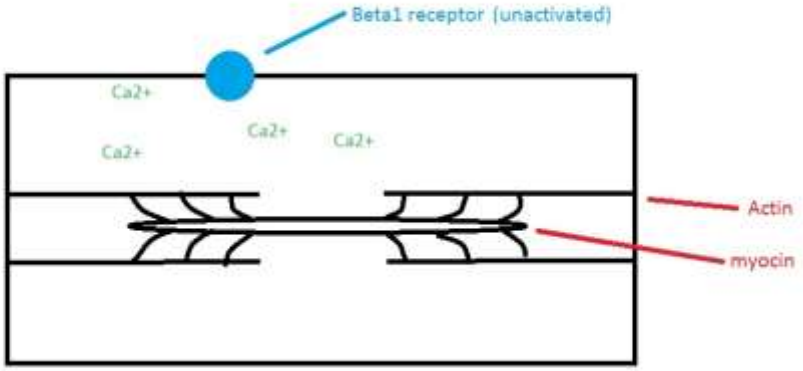
1. Preload should be optimized. Make sure the patient is not hypovolaemic. Inadequate preload is a common cause of decreased cardiac output, and this will be more so if the myocardium is dysfunctional.
2. Afterload management: Systemic pressure should be targeted such that coronary perfusion pressure can be maintain to avoid myocardial ischaemia, while excessive afterload can increase myocardial workload (which the heart may not be able to produce) and increase in oxygen demand. For the right heart, the afterload constitutes the pulmonary artery pressure and hence factors that affect pulmonary pressures should be taken into account. This include any underlying causes of pulmonary hypertension, but also metabolic causes such as PaCO2, PaO2 and pH. Low PaO2, pH, and high PaCO2 can all cause pulmonary hypertension.
3. Rhythm: Try and preserve sinus rhythm if possible. Sinus rhythm will produce the best cardiac output as the atrial kick is preserved. The HR target that should be achieve varies from pathology to pathology.
4. Contractility: Contractility can be increased temporarily by inotropes.

Inotropes

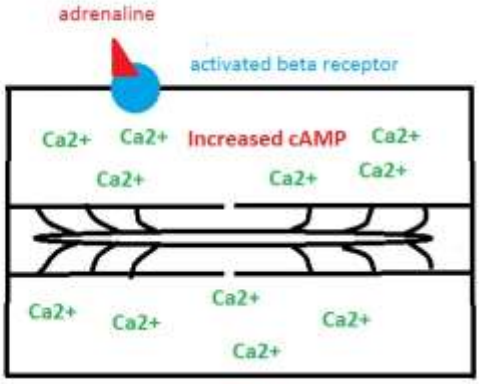
These are drugs which increase the contractility of the heart. We will discuss briefly the pharmacology of these drugs and then discuss the different drugs.

Pharmacology of inotropes

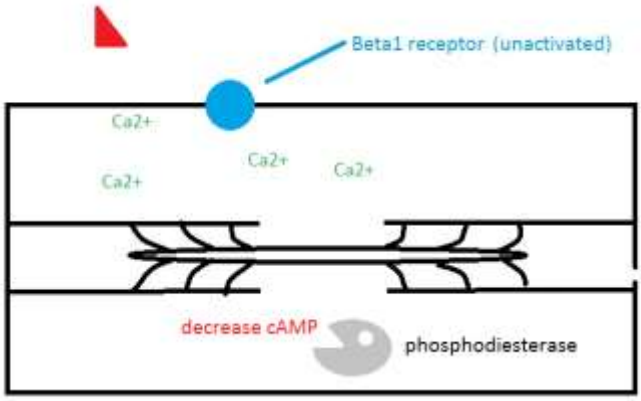
The cardiac myocyte are the basic cellular units of the myocardium. Contraction occurs because of interactions between two important structures; the actin and myosin. The myosin “walks” along the actin and therefore shortens the myocyte causing contraction. This contraction is activated by increased by intracellular ca2+. The intracellular calcium normally enters the cell on depolarisation which can occur through the myocardial conduction pathways (ie. SA node/AV node/Purkinje fibres) or on its own (automaticity). The beta adrenergic activation modulates this pathway, but does NOT initiate depolarisation



The strength of the contraction depends on the concentration of intracellular calcium. The higher the concentration, the greater the contraction and therefore the strength of the contraction of the myocardium can be modulated by controlling intracellular calcium. This naturally occurs through the sympathetic system by the activation of the Beta1 receptors of the myocardial cell membrane by releasing adrenaline at the sympathetic nerve endings as well as systematically through the adrenal glands.



Activation of the Beta1 adrenal receptors activates increases cAMP, a secondary messenger within the cell that activates a pathway that increases intracellular calcium.



cAMP is broken down by phosphodiesterase within the cell and hence once the beta1 receptor activation terminates, the cAMP will be broken down and intracellular calcium will return to baseline levels.

We will now look at the common inotropes and how they increase contractility of the myocardium:

1. **Catecholamine and sympathomimetics**: These bind onto the beta1 adrenal receptor and cause an increase in intracellular calcium by increasing cAMP. Note that aside from the cardiac effects, the beta receptor also has metabolic effects. The common side effects of beta selective sympathomimetics include hyperglycaemia, lactic acidosis, hypokalaemia, tachycardias and arrhythmias.
    a. Adrenaline: This is the naturally occurring inotrope. It is given as a continuous infusion. At lower doses, beta1 adrenergic effects predominates, but at higher doses, more and more alpha1 adrenergic effects occurs. This means that at higher doses, it will cause constriction of arterioles and cause increase in systemic vascular resistance and hence acts as a vasoconstrictor as well.
    b. Dobutamine: it is a synthetic sympathomimetic that has a pure beta effect. This means it does not have any vasocontriction effects, and as beta adrenergics actually vasodilates peripherally, sometime blood pressure can drop, rather than rise.
    c. Dopamine: Like adrenaline, at low doses it predominately is an inotrope, but at higher doses, it vasoconstricts. Studies have shown this drug to be more arrhythmogenic and could potentially cause nausea and vomiting through the dopamine receptor. It can be used peripherally, and is acceptable to be used in some wards outside the ICU.
2. **Phosphodiesterase inhibitors:** These work by inhibiting the breakdown of the secondary messenger of cAMP and therefore increasing its concentration, resulting in higher calcium concentration and therefore contractility. In addition to inotropy, it also causes vasodilatation, both systemically and pulmonary vasculature, reducing

afterload and further augmenting cardiac output. However, this again, may actually reduce blood pressure due to the drop in systemic vascular resistance. The only drug used in this class for inotropy is Milnirone. The other important side effect is arrhythmia and it is renally excreted, and therefore caution is advised for patients with renal impairment.

3. **Calcium sensitizer:** The only drug in this class is levosimendan. The exact mechanism is not known, but that for a given intracellular calcium concentration, the contractility is increased. In addition, it also vasodilates. It has an active metabolite with a long half-life and can last for a week after a 24hr infusion. The use of this drug should be always after approval of the ICU specialist.

Right heart afterload support

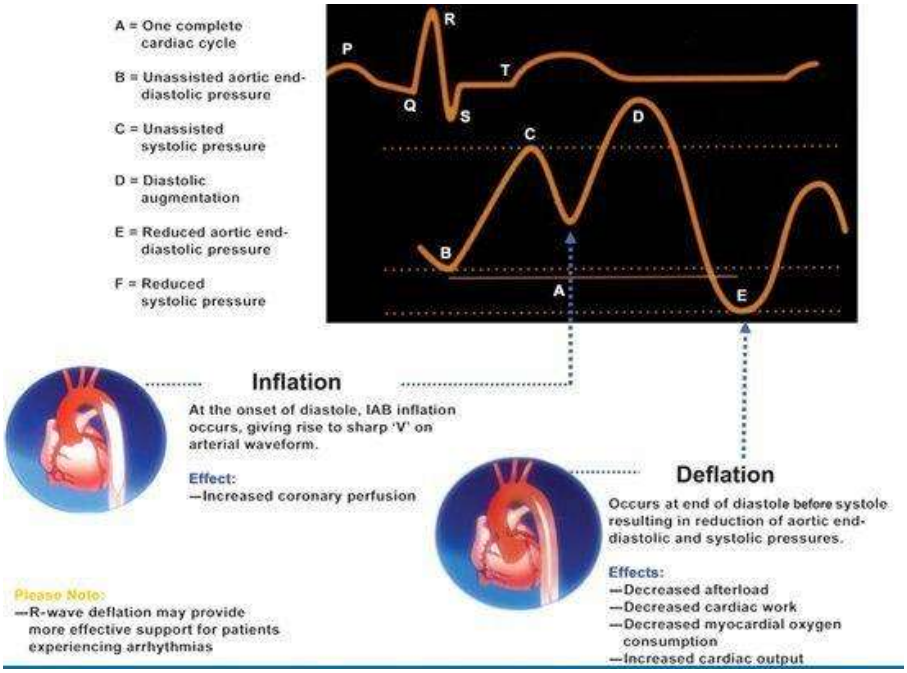
The right heart can also be supported by decreasing afterload i.e. reduction of pulmonary pressures. As with any other pathology, the key is to treat the underlying cause, however in the meantime, there are supportive measures which can be instituted.

1. Ensuring normal pH, low normal paCO2 and normal pO2.
2. Inotropes: Levosimendan, milnirone both reduces pulmonary pressures as well as it's inotropic effects
3. Inhaled Nitric oxide: This is delivered through a specialized delivery system attached to the ventilator. Nitric oxide (NO) is a direct vasodilator of the pulmonary vasculature and hence reduces pulmonary vascular pressure and therefore right heart afterload.
4. Nebulised iloprost: This is a synthetic analogue of prostacyclin (PGI2), which is a pulmonary vasodilator and can be delivered as a nebuliser every 3-4hrs.

Mechanical cardiac support

Intra-aortic balloon pump

This is described in detail under the basic science and equipment section. The balloon pump is inserted percutaneously through the femoral artery and sits in the aorta, just below the left subclavian artery. It acts by reducing systemic afterload, while improving coronary perfusion by increasing diastolic pressure at the aortic root. Unlike other organs, the heart muscle is supplied by blood flow during diastole, rather than throughout the cardiac cycle. This is because during, systole, both the right and left ventricle contracts and is therefore under high pressure. Hence blood flow is less during this time and is greater when the heart relaxes during diastole.



Inflation/Deflation Timing and the Cardiac Cycle
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The IABP inflates during diastole and this causes a back pressure to the aortic root and therefore improves blood flow to the coronaries, the time when most of the perfusion to the heart occurs. During systole the heart deflates, creating a negative pressure, or in another words "suck" the blood flow from the left ventricle causing a reduction in afterload and therefore the work the heart has to do.

Ventricular assist devices (VADs) and ECMO

These are extra corporeal circuits which can bypass the heart and generate blood flow to perfuse organs. They give the heart/ventricle rest so that they can recover, as long as they have reversible pathology.

Distributive shock

This is where there is inadequate organ perfusion is due to low peripheral resistance. In the pure form, where there are no other causes of shock, the peripheries are warm but blood pressure is low. The common causes of distributive shock in the ICU include:

- Septic shock
- SIRS (non-infectious)
- High spinal injury
- Anaphylaxis
- Drugs
- Vasoplegia post cardiac bypass

If it is possible, treat the underlying cause, particularly in sepsis, where source control is the key. However, to support the pressure it is important to maintain adequate preload and avoid hypovolaemia and the use of vasopressors.

Vasopressors

This is the class of drugs which causes vasoconstriction and therefore increase in total peripheral resistance. There are two receptors which are targeted: the alpha1 adrenergic and vasopressin receptor.

Alpha 1 agonists

These receptors are on the smooth muscles of the arterioles and causes vasoconstriction. There are a variety of drugs which we use that acts on these receptors.

1. Noradrenaline. This has predominately alpha 1 adrenergic effect, but also has some beta effect. In most instances, this is a good balance as vasoconstriction will increase afterload and a decrease in cardiac output, which can be counteracted somewhat by the beta 1 effect. It is often the first line therapy for distributive shock, but it is potent and requires a central line.
2. Metaraminol: This is a synthetic vasopressor which has similar effects to noradrenaline, but not as potent and can be used through a peripheral line. It is used as temporizing measure in patients with no central access in the ICU.
3. Phenylephrine: This is a synthetic vasopressor that has no beta effects. It is not potent, and so can be used through a peripheral line. It is sometimes used in patients who have LVOT obstruction or Systolic Anterior Motion (SAM) to avoid increase in contractility which can increase the obstruction.

Vasopressin

This acts on the vasopressin receptor, another vasoconstricting receptor. This is used in patients where there is refractory distributive shock, despite a high dose infusion of noadrenaline.