Management of Acute Left Ventricular Failure

Acute left ventricular failure presents as pulmonary oedema due to increased pressure in the pulmonary capillaries. It is important to realise though that left ventricular failure and pulmonary oedema are not always synonymous as there are other causes for pulmonary oedema e.g. acute renal failure, acute respiratory distress syndrome (ARDS).

Acute heart failure may be de novo or it may be a decompensation of chronic heart failure.

Causes of Acute Heart Failure
- Acute myocardial infarction / ischaemia
- Acute mitral regurgitation
  - Papillary muscle rupture
  - Chordae rupture
- Arrhythmias
- Aortic dissection
- Cardiac tamponade
- Valve destruction (e.g. endocarditis)
- Myocarditis
- “Flash” pulmonary oedema e.g. renal artery stenosis, phaeochromocytoma

Initial Management
The patient should be sitting upright and assessed by the ABCDE approach.

A  Check the patient’s airway and administer high flow oxygen through a reservoir bag (also known as trauma mask).

B  Monitor the patient’s breathing and look for evidence of fatigue (if concerns then an urgent anaesthetic/ICU opinion should be sought). Pulse oximetry should be used.

C  Assess the patient’s circulation by measuring pulse and blood pressure and feeling their peripheries to check perfusion. The patient should be on a cardiac monitor to identify any arrhythmias. Insert an intravenous cannula.

Cardiogenic shock has a very high mortality (up to 90%) and the patient should be reassessed at regular intervals to identify evidence of developing shock so that appropriate management can be initiated quickly.

History
A detailed history is often not possible in the acute setting if the patient is very breathless. A preceding history of chest pain should be sought as well as a past history of ischaemic heart disease and valvular disease. The patient may describe a recent history of increasing dyspnoea with decreased exercise tolerance and orthopnoea.
Examination
The patient is acutely breathless and may have a cough productive of pink frothy sputum. They may be cold and clammy peripherally due to impaired peripheral perfusion. The patient is usually tachycardic, with a raised JVP and there may be a gallop rhythm (3rd heart sound). There will be widespread crackles in the chest and possibly a wheeze. Hypotension is a poor prognostic sign.

Initial Investigations

1. **ECG**
   Look for arrhythmias and ischaemia.

2. **Blood tests**
   FBC (exclude anaemia as a precipitating factor), electrolytes (will predispose to arrhythmias), and cardiac enzymes (if available in your hospital).
   Note that troponin levels can often be raised in patients with pulmonary oedema irrespective of the cause – a raised level does not tell you categorically that the underlying aetiology is that of an acute coronary syndrome. It remains a useful test, but should normally be delayed to 12 hours and the results interpreted alongside the ECG and any other symptoms the patient had.

3. **Arterial Blood Gases**
   The pO2 is usually low because the alveolar walls are thickened by oedema fluid, so diffusion of oxygen into the capillary is impaired.
   The pCO2 is also usually low, as this diffuses better than oxygen through the alveolar wall so its excretion is not so affected by oedema, and the tachypnoea associated with the hypoxia causes the pCO2 level to drop. A raised pCO2 is a bad sign, as it generally suggests the patient is tiring and no longer able to maintain a fast respiratory rate.
   The failing left ventricle is unable to deliver sufficient blood to the tissues, and the blood that does get there is low in oxygen. This means the tissues need to switch to anaerobic respiration to supply some of their energy needs, leading to a raised lactate and a metabolic acidosis.

4. **Chest X Ray**
Diffuse shadowing ("bat’s wings shadow") and kerly B lines due to interstitial oedema and alveolar fluid. The heart is often not enlarged in the acute setting. Pleural effusions may be present.

5. Transthoracic ECHO
If available this will confirm the diagnosis and may help identify the precipitating cause. This is the gold standard investigation when performed by experienced personnel.

Management

The initial approach to a patient with acute heart failure is similar to that of any acute illness, so should follow an ABC approach.
A – ensure airway patent
B – look: respiratory rate, chest wall movement, cyanosis
   Feel: tracheal position, percussion note
   Listen: air entry
   Monitor: oxygen saturations
   Intervene: apply high flow oxygen.
C – look: peripheral colour, JVP
   Feel: peripheral warmth, capillary refill time, pulse, apex beat, peripheral oedema
   Listen: heart sounds
   Monitor: blood pressure, heart rate
   Intervene: IV access, send bloods.
There are a few principles which govern the management of acute heart failure. Remember that the heart is merely a pump, the function of which is to generate pressure which then leads to flow. Flow is the movement of fluid (blood in this case) from an area of high pressure to an area of lower pressure. The greater the pressure difference, the greater the flow.

The heart itself is not able to dictate how much blood it has flowing into it – that is dictated by the pressure in the venous system. However, a well functioning heart is able to generate sufficient pressure to pump out the same amount of blood that had flowed into it. In heart failure, the heart has more blood coming back to it than it can cope with, and this leads to a build up of pressure before the heart, and a reduction in blood delivery distal to the heart.

So the first principle is to reduce the amount of blood returning to the heart. Blood flows passively from the central veins, through the right atrium into the right ventricle during diastole. The factors that govern how much blood returns to the heart are displayed here:

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Peripheral venous tone          Volume of venous blood
                                 ↓                             ↓
Central Venous Pressure         Right Ventricle
(Pressure range 0-10mmHg)       (diastolic pressure 0-5mmHg)
                                 ↓                             ↓
Blood flow
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Therefore, if the pressure difference between the central venous pressure and the right ventricular pressure were reduced, less blood would flow into the right side of the heart and the ventricle would have less work to do – this is also known as reducing preload. This can be achieved by reducing peripheral venous tone, or reducing venous blood volume.

Nitrates are dilators of the venous system and so will reduce venous tone. Frusemide is also a dilator of the venous system and it is this effect which causes initial relief in symptoms. Through its diuretic effects, frusemide will also reduce the volume of blood and cause further reductions in preload – it takes longer for this effect to be appreciable though.
Venodilation will lead to a greater volume of blood remaining in the venous system – the veins are sometimes referred to as capacitance vessels because they have this capacity to accommodate volume, and this does not cause problems.

The second principle is to promote outflow of blood from the ventricle. We will consider the left ventricle in the following description, but the principles are similar for the right ventricle.

The balance of pressure for ventricular outflow is as follows:

![Flow diagram]

- Ventricular systolic pressure
- Flow
- Aortic pressure

The ventricular systolic pressure is generated by the ventricular walls contracting and squeezing on the blood contained within.
The aortic pressure is governed by how much blood is pushed into it (from the heart) and how much blood escapes out of the other end (through the arterioles and into the tissues).

Remember that flow is directly proportional to the pressure difference, so if there is a big difference between ventricular pressure and aortic pressure, then more blood can flow out of the heart – put another way, the stroke volume will increase.

In heart failure, the ventricle can not generate a high enough pressure to expel all of the blood that is being delivered to it. If we were able to drop the aortic pressure, that will increase the pressure difference between ventricle and aorta and improve the function of the heart.

Opiates such as diamorphine and morphine work this way – this is because they reduce sympathetic activity. The sympathetic system has a range of effects, an important one of which is to cause vasoconstriction of arterioles, which acts to keep blood in the arterial system and raise the aortic pressure that the heart is pumping against. Reducing this sympathetic activity therefore reduces aortic pressure.

Nitrates, as well as being venodilators (dilators of the venous system), are also arterial vasodilators. They can therefore also help to improve ventricular outflow.

This principle of reducing the pressure the left ventricle has to overcome is known as reducing afterload.

Another way of increasing ventricular outflow is to increase the pressure generated within the ventricle. This is how inotropic drugs such as dobutamine work, however there are many important issues and dangers associated with these drugs, so they should only be administered by experts.

The third principle is to treat any underlying cause. This may be an acute coronary syndrome, an arrhythmia or an acute valvular lesion.

These patients are usually best looked after in a critical care setting – the coronary care unit, high dependency or sometimes the intensive care unit. They should have cardiac and oxygen saturation monitoring. Urinary catheterisation and urine output monitoring is also very useful.
There are a number of advanced therapies that are occasionally used in these critical care settings, including intra-aortic balloon pumps, inotropic drugs and CPAP.

**Acute Right Ventricular Failure**

Isolated acute right ventricular failure is a lot less common than either left ventricular failure or congestive (right and left ventricular) failure. It should be suspected when there is a high venous pressure (raised JVP), but no evidence of pulmonary oedema. It most commonly occurs with an inferior MI.

Diagnosis can be confirmed with an echo, or occasionally invasive cardiac pressure monitoring with a pulmonary artery catheter (sometimes called a swan-ganz catheter).

The approach to acute right ventricular failure is a little different to left ventricular failure. Since the left ventricle is working well, it will be able to deal with any blood it receives from the right side and pulmonary oedema is therefore not a concern. Problems can arise when there is very little blood coming through the right side of the heart, meaning the left side has hardly any blood to deal with and it therefore can not supply the needs of the systemic circulation. The priority in this case, therefore, is to get as much blood through the right side of the heart as possible—which means ensuring that the filling pressures are high. This is the opposite to the therapy advocated in left ventricular failure, but that is because back-pressure in the peripheral venous system does not have such serious consequences as in the lungs. High filling pressures are achieved by avoiding drugs such as nitrates and diuretics, and giving boluses of IV fluids. A slow and steady approach is taken, with boluses of around 200ml of fluid being delivered, with re-assessment of haemodynamic parameters after each bolus. This is performed most safely in a critical care setting (ie. CCU), sometimes with invasive monitoring.