Pericardiocentesis

Pericardiocentesis is a procedure whereby fluid is aspirated from the pericardial cavity via a needle. It may be performed: (1) when there is a large pericardial effusion, (2) to relieve cardiac tamponade, or (3) to obtain fluid for analysis.

The role of echocardiography in this procedure is to: 1. confirm the presence, size and distribution of the effusion, 2. identify the ideal puncture site and trajectory angle for needle entry, 3. confirm the needle location within the pericardial cavity, 4. reassess the amount of residual fluid following the procedure.

In particular, the ideal site and trajectory angle for needle entry is based on: (1) the shortest distance from the body surface to the pericardial effusion where fluid accumulation is maximal, and (2) a straight trajectory path that avoids vital structures such as the liver, myocardium and lungs.

Most commonly, pericardiocentesis is performed from the subcostal and para-apical locations (Fig. 12.21). The injection of a small volume of saline can be performed to confirm the location of the needle within the pericardial space (Fig. 12.22). This is especially important if a bloody fluid return is noted following needle insertion.

Important Note: Aortic dissection is a major contraindication for pericardiocentesis due to the risk of intensified bleeding and extension of the dissection[12].

Cardiac Tamponade

Cardiac tamponade occurs when there is an increase in the intrapericardial pressure (IPP) due to accumulation of an effusion, blood, clots, pus, gas or combinations of these within the pericardium. This ultimately leads to compression of the heart, impeded diastolic filling of both ventricles, systemic and pulmonary congestion, and a decreased stroke volume and cardiac output.

Pathophysiology of Cardiac Tamponade

In order to understand the pathophysiological changes that occur with cardiac tamponade, consideration of the transmural pressures, changes in right and left heart filling with respiration, and ventricular interdependence is required.

Transmural Filling Pressures

The transmural filling pressure describes the difference in pressure between the inside and the outside of a walled structure. Therefore, for the heart the transmural filling pressure (TMFP) can be used to describe the pressure difference between the intracavitary pressure (ICP) and the IPP.

In the normal situation, the intrathoracic pressure (ITP) is transmitted to the pericardial sac so the IPP equals the same as the negative (subatmospheric) ITP. Therefore, as the ICP is usually positive, there is a positive TMFP which is higher than the ICP. This positive TMFP maintains the shape of the cardiac chambers and prevents them from collapsing at end-diastole when the ICP falls to zero (Fig. 12.23).

With cardiac tamponade, the IPP is increased. As the IPP rises, the ICP also rises in an attempt to maintain a positive TMFP and an adequate cardiac output. However, further increases in the IPP result in a fall in the TMFP which will result in impeded diastolic filling of the heart and a subsequent reduction in cardiac output. When the TMFP becomes negative there is collapse (compression) of the cardiac chambers (Fig. 12.24).

Right and Left Heart Filling with Respiration

In the normal situation, there is augmentation of right heart filling with inspiration, a decrease in right heart filling with expiration, and minimal variation in left heart filling with respiration.

During inspiration, as the diaphragm descends there is an increase in the intrathoracic pressure (IAP) and a reduction in the IPP which leads to an augmentation of systemic venous return and increased filling of the right heart. With expiration, the opposite occurs so as the diaphragm ascends, there is an increase in the ITP and a decrease in the IAP which leads to a reduction in the systemic venous return and a decrease in right heart filling.

There is minimal variation in left heart filling with respiration. This is because changes in ITP are transmitted to both the pericardial sac (and the cardiac chambers) and the pulmonary veins to the same degree (Fig. 12.25, top). For instance, as the ITP decreases with inspiration the IPP and ICP also decrease to the same degree; and as the pulmonary veins are also contained within the thoracic cavity, the decrease in ITP is also reflected in the pulmonary veins to the same degree. Likewise with expiration, as the ITP increases so does the IPP, ICP and pulmonary venous pressure to the same degree. Therefore, the effective filling gradient (EFG) of the left heart changes only slightly during respiration. This slight respiratory variation in the left heart EFG is based on changes in right heart filling with inspiration as described above.

In particular, increased RV filling during inspiration causes the ITP to rise very slightly to the left; as a result there is a slight inspiratory reduction in LV filling and LV stroke volume as well as a slight inspiratory fall in the systemic arterial pressure (< 10 mm Hg).

In the case of tamponade, the normal respiratory changes described above are accentuated. That is, with inspiration left heart filling is decreased more than normal and right heart filling is increased more than normal. These changes occur because the IPP is increased. Therefore, the normal decline in the ITP during inspiration is not fully transmitted to the pericardial sac and to the cardiac chambers; however, the ITP decline is transmitted to the pulmonary veins as normal. As a result, the increased IPP is counteracted by the ICP which does not fall as it should but because the pulmonary venous pressure falls...