Studies in Experimental Pericardial Tamponade

Effects on Intravascular Pressures and Cardiac Output

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As intrapericardial pressure is increased by the introduction of saline into the pericardial cavity, cardiac output falls. Intravascular pressures, arterial and venous, pulmonary as well as systemic, approach the same value. This equalization of intravascular pressure is due to the decreasing ability of the heart to maintain normal distribution of blood within the vessels.

As early as 1889 Cohnheim demonstrated by injecting oil into the pericardial cavity that an increase in intrapericardial pressure causes a rise in peripheral venous pressure and a fall in peripheral arterial pressure. These same effects of cardiac tamponade have been confirmed by many others both in the experimental animal and in patients with pericardial effusion.

Cohnheim performed his experiments in curarized dogs with open chests, and under these conditions he observed a fall in pulmonary arterial as well as systemic arterial pressure. Moreover, he reasoned that pressure in the pulmonary vein must also fall, although he did not record the measurement of pulmonary venous pressure. He states "...and since in our experiment, as shown by the pulmonary curve, the right heart sends an abnormally small quantity of blood into the arteries of the lungs at each systole, the arterial pressure must fall, and with it*—despite the

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* Italicized in the original.

† The button used in these experiments was designed by Dr. Charles A. Hufnagel, Georgetown University Medical Center, Washington, D. C.
free end of this tube was led out of the chest by the same stab wound as that conducting the pericardial tube. The chest was then closed, the lungs having been fully inflated, and spontaneous respiration was allowed to resume. Cannulas were placed in a femoral artery and vein, and a cardiac catheter was introduced into a pulmonary artery. It was inserted by way of an external jugular vein, and passed through the right heart under fluoroscopic guidance. In many dogs a second catheter was passed to the right atrium. Saline manometers were used routinely to measure femoral venous and intrapericardial pressures, and in some instances to measure pulmonary venous pressure. Electromanometers were used to measure femoral arterial, pulmonary arterial, and right atrial pressures in all instances and in several experiments to measure "pulmonary capillary" and pulmonary venous pressures. The free end of the tube leading through the chest wall into the pericardial cavity was attached to a Y-connector, the other two arms of which were connected, respectively, to a saline manometer and a large reservoir of saline. Intrapericardial pressure was increased by raising the reservoir to the level desired. After several minutes of waiting to allow for inflow the reservoir tube was clamped and the intrapericardial pressure read from the saline manometer. While other measurements were being made the reservoir tube was left open so that, in spite of stretching of the pericardium or absorption of injected fluid, the intrapericardial pressure was maintained at the desired level. Postmortem examination was performed routinely and leakage from the pericardium was excluded in every case. All manometers, both saline and electrical, were set to the same zero point, the level of the dog board. Mean values were obtained by electrical integration for pressures recorded with electromanometers, and by averaging the extreme values for pressures obtained by saline manometers. Comparison of median pressures obtained arithmetically by the saline manometers with mean pressures obtained by electrical integration by the electromanometers does, of course, introduce an error. It is believed that our reported measurements are accurate to \( \pm 2 \) mm Hg. No attempt has been made to quantitate this error, since the changes in pressure to be reported are beyond the error involved in their measurement.

In eight dogs, the cardiac output was determined by the direct Fick method at selected points of intrapericardial pressure. Expired air was collected for three or more minutes in a Douglas bag through a balloon-sealed intratracheal tube, and oxygen consumption calculated. Blood samples were taken simultaneously from the femoral artery and the pulmonary artery over a one-minute period beginning one minute after the start of collection of the expired air. Analysis of these samples permitted calculation of the arteriovenous oxygen difference.

Results

Peripheral Arterial Pressure. The effect of pericardial tamponade on the femoral arterial pressure was observed in 18 dogs. In figure 1, mean femoral arterial pressures of four dogs are plotted against intrapericardial pressure. The curves shown are representative of all. It is to be noted that femoral arterial pressure is maintained during moderate cardiac tamponade, but eventually falls precipitously, in some experiments to a level almost as low as or equal to intrapericardial pressure. There is also a progressive reduction in pulse pressure seen in every case as pericardial pressure is increased.

![Fig. 1. Relation of femoral arterial pressure to intrapericardial pressure in pericardial tamponade.](image)

Peripheral Venous Pressure. Mean femoral venous pressure was measured in 18 dogs, and the values obtained are plotted in figure 2. Pressure in the peripheral veins begins to rise as intrapericardial pressure approaches the resting venous pressure, and the venous stays above the intrapericardial, although by an ever decreasing increment, until both reach the same level at the height of tamponade. The pressure attained in the femoral vein as flow is brought to a standstill is in the region of 20 mm Hg.

Right Atrial Pressure. The mean right atrial pressure as recorded in eight dogs is similarly plotted in figure 3. Right atrial pressure rises with increasing intrapericardial pressure in the same manner as peripheral venous pressure.
However, the right atrial mean pressures are slightly lower than simultaneous venous levels. The pressure gradient between femoral vein and right atrium gradually decreases throughout the experiment, until the two pressures become equal at maximum tamponade.

Fig. 2. Relation of peripheral venous pressure to intrapericardial pressure in pericardial tamponade. In figures 2, 3 and 4 the diagonal line is the line of equality of intravascular and intrapericardial pressures.

Fig. 3. Relation of right atrial pressure to intrapericardial pressure in pericardial tamponade. See legend to figure 2.

Pulmonary Venous Pressure. In figure 4, mean pulmonary venous pressure as measured in 12 dogs is plotted against intrapericardial pressure. These mean pressures, whether obtained by averaging the extreme values measured with a saline manometer or by electrical integration with an electromanometer, are observed to rise as intrapericardial pressure is increased. It may be noted that the pressures in the pulmonary veins and right atrium are generally of the same order of magnitude and that when there is a difference between them, the pulmonary venous pressure is slightly higher than the right atrial pressure. The pressure attained in the pulmonary veins at the height of tamponade is in the range of 20 mm. Hg. In several dogs “pulmonary capillary” pressure was measured according to the technic of Hellems and co-workers. In each of these cases, “pulmonary capillary” pressure rose during tamponade in exactly the same manner as pulmonary venous pressure.

Fig. 4. Relation of pulmonary venous pressure to intrapericardial pressure in pericardial tamponade. See legend to figure 2.

Pulmonary Arterial Pressure. By analogy with the systemic circuit, one would expect the pulmonary arterial pressure and the pulmonary venous pressure to approach the same point as tamponade is produced. This does occur, as observed in eight dogs, but whether pulmonary arterial pressure rises or falls to approach this point depends upon the mean pressure level in the pulmonary artery before the induction of tamponade. In some dogs there is a decline, that is, the mean pulmonary arterial pressure is lowered by the procedure, but in most experiments the mean pulmonary arterial pressure eventually rises as the circulation is brought to a standstill. Since the “normal” mean pulmonary arterial pressure is in
the same range as the pressure observed throughout the vascular bed when circulation has ceased, the changes produced in pulmonary arterial pressure by tamponade are not dramatic.

A clearer understanding of the circulatory dynamics of pericardial tamponade is obtained by examining the simultaneous changes in all pressures in a single dog during a single episode of tamponade. Figure 5 illustrates the effects of gradually increasing intrapericardial pressure on femoral arterial, pulmonary arterial, right atrial, and pulmonary venous pressures in a single dog. Right atrial and pulmonary venous pressures rise steadily as pericardial pressure increases. In this experiment pulmonary arterial pressure does not change significantly. In this animal, as in all, femoral arterial pressure declines, slowly at first, then more and more rapidly. All curves approach a single point at an intrapericardial pressure of 22 mm. Hg when all intravascular pressures are equal at about 20 mm. Hg. At this point the absence of any pressure gradients must mean that circulation has ceased. Even so, at this point the process is still reversible. If intrapericardial pressure is rapidly reduced, all intravascular pressures gradually return to their original levels, and for a time peripheral arterial pressure may actually exceed its pretamponade level.

The effects of changes in intrapericardial pressure may also be described by charting the intravascular pressure existing simultaneously at each of several points within the circulation. Figure 6 is a diagram of the mean intravascular pressures at five such points before and during pericardial tamponade. This diagram shows that as intrapericardial pressure is increased, arterial pressures fall and venous pressures rise. These changes in pressure are due to a decreasing effect of cardiac output upon the distribution of blood within the vessels. It is, therefore, necessary to consider the effects of changes in intrapericardial pressure upon the cardiac output.

**Cardiac Output.** As intrapericardial pressure is increased, cardiac output decreases (table 1). This fall in cardiac output is due to a pro-
The heart rate increases as tamponade is produced, but this increase is insufficient to maintain normal minute output. Figure 7 shows that stroke output approaches zero at an intrapericardial pressure of about 20 mm. Hg. This level agrees with the value at which all intravascular pressures become identical, as described earlier.

Peripheral Resistance. Comparison of the effect of increasing pericardial pressure on arterial blood pressure and cardiac output shows (table 1) that the arterial pressure is maintained early in the experiment when cardiac output has already declined. Maintenance of arterial pressure in the face of a decreased cardiac output can only be accomplished by an increase in peripheral resistance. That this occurs in pericardial tamponade has been shown previously11, 13 and is demonstrated by the calculated values for peripheral resistance given in table 1.

The measurements of simultaneous pulmonary arterial and pulmonary venous pressures in these experiments permit calculation of the pulmonary resistance. Calculated pulmonary resistance during pericardial tamponade showed no change which was not explainable by the systematic error involved in measurement of the pressures and flow by the methods used.

**DISCUSSION**

The pressure within any segment of the vascular tree is determined by the capacity of that segment and the volume of the blood within it, whether that volume is put there momentarily by the action of the heart or lies stagnant when there is no effective ventricular propulsion of blood. As Starling pointed out, in pericardial tamponade intravascular pressures approach an identical (and therefore “flowless”) point. This trend is explained by the steadily lessening ability of the heart to perform its function as a pump. As cardiac output declines, and finally ceases, the volume of blood within the vascular tree is redistributed until all pressures, systemic and pulmonary, arterial and venous, are identical. If at this point pericardial pressure is reduced, restoration of normal pressures is accomplished by restora-

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**TABLE 1**

<table>
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<tr>
<th>Dog Number</th>
<th>Pericardial Pressure mm. Hg</th>
<th>Cardiac Output L/min.</th>
<th>Pulse Rate beats/min.</th>
<th>Mean Femoral Arterial Pressure mm. Hg</th>
<th>Mean Right Atrial Pressure mm. Hg</th>
<th>Total Peripheral Resistance*</th>
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* The values given are those obtained by using the following formula:

\[
Resistance = \frac{\text{Cardiac Output}}{\text{(Mean Femoral Arterial Pressure} - \text{Mean Right Atrial Pressure})}
\]

with pressures and cardiac output expressed in the units used in the table.
tion of normal volume distribution by a recovery of cardiac function.

The mechanism by which pericardial effusion decreases cardiac output is not yet clear. Nerlich\textsuperscript{16} has stressed the lessened gradient for venous inflow into the atria. Changes in resistance to flow within the heart have been inferred.\textsuperscript{18} Such problems are at present under study in this laboratory.

**Summary**

1. Peripheral arterial, peripheral venous, right atrial, pulmonary arterial, and pulmonary venous (and occasionally "pulmonary capillary") pressures have been measured in dogs during experimental pericardial tamponade. Cardiac output was measured at selected points in many experiments.

2. With increasing intrapericardial pressure pulmonary venous pressure (and "pulmonary capillary" pressure) has been found to rise in every case. This rise in pulmonary venous pressure is of the same order of magnitude as the rise in right atrial pressure.

   Depending on its resting value, mean pulmonary arterial pressure may remain unchanged, rise, or fall to approximate that level of pressure which exists throughout the vascular tree at the height of tamponade.

   As intrapericardial pressure increases, all intravascular pressures, pulmonary and systemic, venous and arterial, approach the same value of about 20 mm. Hg.

3. Cardiac output falls as intrapericardial pressure is elevated. This is due to a decrease in stroke volume.

4. The equalization of intravascular pressure is due to a progressive decline in the performance of the heart in maintaining normal distribution of blood within the vascular tree.

**Acknowledgment**

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