Pressure-volume curves of systemic and pulmonary circuit

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In order to measure MSP and MPP, the heart is stopped, and blood is translocated from the systemic arterial circulation to the venous segment of the systemic circuit. When blood is translocated in this manner, the systemic arterial pressure will fall and the systemic venous pressure will rise. The point at which the two pressures are equal is termed the MCP. If the equilibrium pressure is determined in the isolated systemic circuit, it is termed the MSP. Likewise, the equilibrium pressure in the pulmonary circuit is defined as the MPP.

The equilibrium pressure minus the respective atrial pressure has been shown to represent the gradient for venous return (GVR) for the respective circulation. Thus, for the systemic circulation $GVR = MSP - RAP$ and for the pulmonary circulation $GVR = MPP - LAP$. (RAP and LAP are atrial pressures prior to fibrillation.)

Flow is proportional to these pressure gradients divided by the resistance to venous return. Therefore, under conditions of a constant resistance to venous return in each circulation, they indicate the venous return to each side of the heart and, in a competent heart, the cardiac output (7, 9, 10).

Guyton et al. have demonstrated the effects of sympathetic stimulation, changes in blood volume, hypertension, and other circulatory alterations on MCP (5, 9, 10). In the present study, the effects of acute blood volume changes on MSP, MPP, and the corresponding pressure gradients for venous return were investigated.

METHODS

Eight mongrel dogs weighing between 16–20 kg were used in this study. The dogs were anesthetized with 30 mg/kg sodium pentobarbital and the chest was opened at the fourth intercostal space. A 4-cm incision was made in the pericardium through which a flexible tube, impregnated with Silastic was placed around the aorta and pulmonary artery. One end of the tube was tied to the anterior portion of the fourth rib, and the other end was subcutaneously brought through the posterior...
Thorax near the spinal column. This tube was later to be used to occlude flow in these two vessels. Subsequently, a polyethylene catheter filled with heparin was secured in the left atrium and the free end sutured subcutaneously. The pericardial and thoracic incisions were sutured, and the animals were allowed to recover 5–7 days.

After recovery from surgery, the dogs were again anesthetized with 28–30 mg/kg sodium pentobarbital and heparinized with 5 mg/kg of sodium heparin. The experimental design is shown in Fig. 1. The distal end of the left atrial catheter and the tubing around the great vessels were exposed. A large catheter was placed in the right femoral artery and advanced to about the level of the diaphragm. This catheter was connected to the input side of a Sigmamotor pump. The output of the pump was connected, via two large catheters, to both the right and left femoral veins. A 1-liter blood reservoir was also connected to the input side of the pump so that the blood volume of the animal could be rapidly changed. Catheters for recording pressure were placed in the right atrium, pulmonary artery, and aorta. The left atrial pressure was recorded through the catheter placed in the left atrium during surgery. Pressures were recorded by Statham pressure transducers and recorded on a Grass model 5 polygraph. The zero reference point for the pressure measurements was 0.61 times the anterior-posterior thickness of the chest (11).

The blood volumes of three animals were determined with T-1824. Since these determinations were within 7% of the estimated blood volume obtained by dividing the dog’s weight (in kg) by 12, blood volumes of the remaining five dogs were estimated by this procedure.

Equilibrium pressures for the two circulations were measured as has been previously described. The heart was fibrillated with a shock of 110 v ac applied through two needle electrodes inserted into the skin on the left anterior chest wall over the area of the heart. Immediately after inducing fibrillation, the ascending aorta and pulmonary artery were occluded by tightening the loop around them. In the systemic circuit, blood was translocated from the femoral artery to the femoral veins by activating the previously described Sigmamotor pump. This pumping procedure was necessary to effect a rapid equilibration of the pressures within the systemic circulatory system. If the equilibrium pressure is not attained within 8 sec, intense vasomotor activity is initiated and the pressures rise, thus voiding the measurement. Since the resistance to blood flow in the pulmonary circuit was relatively low, the pulmonary pressure equilibrated within 2 or 3 sec of fibrillation and pumping was not necessary. Once the pressures within each system reached equilibrium, the tension on the loop was released, and the heart defibrillated with a shock of 440 v ac for 0.1 sec applied through two large electrodes located over the anterior and posterior thorax, as shown in Fig. 1.

The animal was then allowed sufficient recovery time for the measured parameters to return to their control values (approximately 15 min).

In order to determine the effect of changes of blood volume, the animals were either hemorrhaged or transfused as described below. If an increase in blood volume was desired, the blood reservoir was opened and blood was pumped into the venous circuit very rapidly prior to fibrillation. If a decrease in blood volume was desired, blood was pumped from the femoral artery into a beaker.
PRESSURE-VOLUME CURVES

An average of three determinations was made on each animal.

RESULTS

Mean systemic pressure. Figure 2 shows a typical recording obtained during this study. The arrows indicate when the transfusion was begun and the application and removal of the fibrillating current. In this particular animal the blood volume was increased by 10%. After transfusion and fibrillation, the right atrial and left atrial pressures are elevated while the mean arterial and pulmonary arterial pressures are falling. The X's indicated the points at which RAP = AP and LAP = PP. In this particular record, the blood transfusion, fibrillation, and equilibration of pressures occurred within 6 sec. The control MSP obtained in these experiments was 6.4 ± 0.9 mm Hg and the control MPP was 9.9 ± 1.1 mm Hg.

Figure 3 illustrates the effect of changes in blood volume on MSP. The MSP decreased from the normal value of 6.4 mm Hg to 0 mm Hg with a decrease in blood volume of only 18%. The MSP doubles (6.4 → 12.8) with an increase in blood volume of 18%. An increase in blood volume of 35% will approximately triple the MSP.

If the effect of these changes in blood volume on right atrial pressure is taken into consideration and the right atrial pressure is then subtracted from the MSP, the difference is the gradient for venous return for the right heart (SGVR). The effect of changes in blood volume on SGVR is shown in Fig. 4. The control SGVR was 6.4. The SGVR fell to zero when the blood volume was reduced by 20%. An increase in blood volume of 19% doubles the SGVR, and an increase of 38% triples it.

Mean pulmonary pressure. The results of studies of MPP are shown in Figs. 5 and 6. Figure 5 shows the effect of changes in blood volume on MPP. The control MPP was found to be 9.9 mm Hg in the normal dog. The MPP fell from 9.9 mm Hg to zero when the blood volume was decreased by only 12%. However, since the pulmonary system is enclosed and under an average pressure of -5 mm Hg when referred to the atmosphere, the true zero pressure for MPP would be -5 mm Hg rather than

![Diagram of pressure-volume curves with annotations]

FIG. 2. Recording of MSP and MPP determination. The animal was rapidly transfused with blood to 10% above his control volume, and MSP and MPP determined as explained. Arrows indicate the event occurring as noted in the top tracing.

FIG. 3. Changes in MSP as a result of percentage changes in blood volume. Open circles indicate values obtained when the animal was rapidly hemorrhaged and closed circles represent measurements obtained when the animal was transfused. Eight dogs were used and an average of three determinations made on each animal.
FIG. 4. Pressure gradient for venous return to the right side of the heart when blood volume was rapidly changed. The control right atrial pressure and the MSP were used to calculate the gradient for venous return.

0 mm Hg. Thus, the MPP in Fig. 5 has been extrapolated to the true zero reference level. The MPP then is at true zero when the blood volume is reduced by 18%. Thus, zero pressure levels are obtained in both the systemic and pulmonary systems at the same decrease in blood volume. This is more clearly illustrated by a comparison of the pressure gradients in each circulation (Figs. 4 and 6). The MPP rose to twice normal, 18.8 mm Hg, when the blood volume was elevated by 12%. When the blood volume was increased by 24% the MPP tripled from its control value to 29.7 mm Hg.

When concomitant changes in left atrial pressures are subtracted from MPP, the pulmonary gradient for venous return is obtained (PGVR). The control PGVR was 6 mm Hg. The effect of changes on PGVR is shown in Fig. 6. The PGVR falls to zero when the blood volume is decreased by 20%, but doubles in value to 12 mm Hg when the volume is increased by 14%. Increasing the blood volume by 21% triples the PGVR.

DISCUSSION

Several findings may be gleaned from these experiments. The results of the experiments on the effect of changes in blood volume on MSP and MPP, shown in Figs. 3 and 5, provide pressure-volume relationships for the systemic and pulmonary circulations, respectively. Of particular importance is the steepness with which the pressures in the circulation respond to changes in volume. For instance, if the blood volume is only decreased to 82% of normal, the MPP falls completely to -5 mm Hg and the MSP drops to 0 mm Hg. These values represent the zero level of pressure in the respective circuits. When the blood volume is increased, the MSP increases 6.4 mm Hg (the control value) for each 18% increase in blood volume, and the MPP increases by 9.9 mm Hg (the control value) for each 12% increase in blood volume. It can be seen from Figs. 3 and 5 that the slope of the MPP and blood volume curve is 35% steeper than the slope of the MSP and blood volume curve. This, at first glance, would appear to be unusual since the pulmonary circulation is generally considered to be highly distensible. However, this finding may be explained by the inequality of the ventricular function curves for the two sides of the heart. If the ventricular function curves of the right and left hearts relating minute ventricular output to atrial pressure are compared, it can be seen that the function curve for the right heart is the steeper curve. Indeed, the curve for the right heart is almost 50% steeper than that of the left. An increase of only 2 mm Hg in the right atrial pressure will double the output of the right heart, whereas an increase of almost 4 mm Hg in the left atrial pressure is necessary to cause a similar increase in output of the left heart (11).

Thus, when blood volume is increased, the output of the right ventricle momentarily exceeds the output of the left ventricle. The difference between the output of the right and left hearts remains dammed in the pulmonary circulation until the left atrial pressure rises to a level that will result in the output of the left ventricle being
pressure-volume curves for the two circulations are very steep. Even very small changes in blood volume exert large changes in the two pressure gradients for venous return and, thus in cardiac output. Indeed, changes in blood volume, less than present methods of measurement can detect, exert significant changes in cardiovascular dynamics.

These studies were conducted with rapid changes in blood volume and represent purely hemodynamic effects of instantaneous changes in volume. Within 8 sec from the change in volume, other factors begin to intervene. Pressoreceptor reflexes, stress relaxation, and reverse stress relaxation all tend to minimize the effect of changes in blood volume. These studies can be compared with those of Chien (1) which were conducted in intact animals with reflexes fully active. His studies showed that cardiac output fell to zero only after a loss of 30% blood volume. In the present studies, a loss of blood volume of only 18 per cent was sufficient to reduce cardiac output to zero. In summary, it can be seen that very small changes in blood volume can result in very large instantaneous changes in cardiovascular dynamics, but the reflex control systems of the circulation minimize these changes to a large extent within a few seconds thereafter.

**REFERENCES**