Pressure-Volume Curves of the Arterial and Venous Systems in Live Dogs

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ABSTRACT

Pressure-volume curves have been recorded for the arterial and the venous systems of live dogs. The quantity of blood in the arterial system of a 12-kg dog was found to average approximately 60 cc greater when the heart was pumping blood normally than when the circulation of blood was at a standstill. Likewise, under normal dynamic conditions the quantity of blood in the venous system of the 12-kg dog averaged 60 cc less than that under static conditions. The capacitance \( \frac{dV}{dP} \) of the venous system, as calculated from the pressure-volume curves, averaged between 18 and 30 times as great as the capacitance of the arterial system. This means that for each unit change in blood pressure the venous system can hold \( 18-30 \) times as much blood as can the arterial system within the range of pressures measured in this study.

Among the most important problems in circulatory dynamics has been the attempt to quantitate how much blood shifts from one part of the circulatory system to another part under different conditions. For instance, when the arterial pressure is increased, how much extra blood is stored in all of the arteries? On the other hand, when the venous pressure is increased, how much does the blood volume increase in the venous system?

Recent evidence from this laboratory has indicated that the volume change for a given change in pressure in the over-all venous system is probably 18 times or more greater than is the volume change in the arterial system for a similar change in pressure (1). Furthermore, some of the calculations necessary for predicting venous return to the heart depend mathematically on knowing the 'capacitances' \( \frac{dV}{dP} \) of the different major portions of the circulatory system, and especially is it desirable that the ratio of the capacitances of the venous and arterial systems be known (2).

Therefore, the purpose of the present study has been to record as accurately as possible the pressure-volume curves of the arterial and venous systems in live dogs.

METHODS

In general, the method used for recording the pressure-volume curves of the venous and arterial systems was to stop the heart suddenly by maximal stimulation of the vagus nerves at 60 stimuli/sec, and then to record the rapid fall in arterial pressure and the rapid rise in venous pressure for the following few seconds before vasomotor effects could take place (about 8 sec.). The resulting curves were pressure-time curves. To convert these curves into pressure-volume curves, a special procedure was devised as follows: A minute or so after the dog had recovered from the first recording of the pressure-time curves, a second set of pressure-time curves was recorded but in a slightly different manner: during the recording of these curves, blood was made to flow through an external shunt from the aorta into the right atrium at a known and constant rate of flow. This flow through the external shunt caused the slopes of the curves to be much greater than those of the previous set of curves. By measuring the differences in these slopes at each pressure level, and knowing the rate of flow through the external shunt, it was possible to calculate the changing rate of blood flow through the peripheral vessels as the arterial and venous pressures fell. Then, the determined rates of blood flow through the external shunt and peripheral vessels were integrated into pressure-volume curves. The precise mathematics of this procedure is given in the appendix to this paper.

The system for transferring blood at a constant
rate through the external shunt consisted basically of a mercury column 3000 mm in height. At the top of the mercury column was a mercury bulb and at the bottom was a similar mercury bulb. The two bulbs were half filled with blood from a donor animal. The upper bulb was connected to a catheter inserted into the aorta, and the lower bulb was connected to a catheter inserted into the right atrium. A fixed resistance was placed in the mercury column between the two bulbs, and, because of the tremendous head of pressure, the rate of mercury flow through this resistance remained within 3% of its calibrated value despite changes in the arterial pressure during the generation of the pressure-time curves. When the mercury was allowed to flow, blood passed out of the arterial system into the upper bulb and it entered the venous system from the lower bulb at the same time.

Nineteen dogs anesthetized with sodium pentobarbital and heparinized with approximately 3 mg/kg of heparin were used in these studies. Arterial and venous pressures were measured with either a Statham strain gauge or with a Sanborn electromanometer, and the records were recorded on a Sanborn polyviso recorder. Arterial pressure was recorded from a catheter inserted through a carotid artery into the arch of the aorta, and venous pressure was recorded from a catheter inserted through an external jugular vein into the right atrium.

RESULTS

Pressure-Volume Curves in the Arteries. Figure 1 illustrates a number of representative arterial pressure-volume curves recorded from different dogs. Also, figure 3 illustrates a composite curve constructed from nine separate arterial pressure-volume curves recorded from different animals, these curves all corrected to that of a 12-kg dog. In making these corrections, the capacitance \((dV/dP)\) of the arterial system was considered to vary approximately in proportion to the weight of the animal. This is probably a reasonable assumption in the light of present knowledge as will be discussed below.

Pressure-Volume Curves in the Veins. Figure 2 illustrates a number of representative pressure-volume curves for the venous systems of different dogs. Also, in figure 3 is represented a composite curve constructed from nine different venous pressure-volume curves corrected to that of a 12-kg dog. The character of the initial portions of these curves is not that of usual pressure-volume curves recorded in isolated veins. The probable reason for this is the following: the venous pressure recorded in the right atrium immediately after the heart is stopped is not equal to the mean distributed pressure throughout the veins because many of the veins entering the thorax are still collapsed immediately after the heart is stopped. However, as has been shown in previous studies (3), after the right atrial pressure rises high enough to overcome this collapse, venous pressure as measured in the right atrium is then probably as satisfactory a measure of the mean distributed pressure throughout the major veins as can be attained. For this reason, only the latter part of the curves recorded from the veins are considered to depict pressure-volume relationships.

Relationship of the Capacitance \((dV/dP)\) of the Arterial System to the Capacitance of the
Venous System. Figure 3 is constructed to show the simultaneous pressure rise in the venous system and pressure fall in the arterial system as blood shifts from the arterial system into the venous system. It will be observed from these relative curves that the over-all ratio of capacitance of the venous system to the capacitance of the arterial system was approximately 18 to 1. However, discounting the first portion of the venous pressure-volume curve which is probably invalid for reasons discussed above, one finds that the ratio of capacitances of the two systems is approximately 30 to 1 for the remainder of the curves. In other words, the volume in the venous system changes approximately 30 times as much as it changes in the arterial system for an equivalent pressure change in the two systems.

Relationship of Arterial Capacitance to Weight. Figure 4 illustrates the relationship of arterial capacitance to weight of the dogs used in the present study, showing that the capacitance tends to increase with weight of the dog. This is the effect one would expect because the blood volume increases with the weight of the dog, and capacitance is the product of blood volume times distensibility of each segment of the vascular system. This curve illustrates especially the mathematical difference between the ‘capacitance’ \( dV/dP \) of the arterial system and the ‘distensibility’ \( dV/(dP \times V) \) of the arteries, for the general effect of growing older and larger is decreased arterial distensibility, which is opposite to the effect on the capacitance.

Even though the capacitance generally increases with weight of the dog, the data available from the present study is far too little to determine whether this relationship is a linear one or a curvilinear one.

Venous capacitance also varied generally in proportion to weight, though the data was less evident than that for arterial capacitance. This difference presumably occurred because venous pressure-volume curves depend on many other factors in addition to the distensibility of the veins—such as compression of the veins by the tissues—these factors varying greatly from one animal to another.

Volume of Mobilizable Blood in the Arteries. In seven of the dogs removal of blood from the arterial system by the mercury shunt system caused the pressure to fall first to a plateau at a level of approximately 3-4 mm Hg and then suddenly to fall to negative pressure values, as illustrated at the end of curve A in figure 5A. It was assumed that these negative pressures were probably due to exhaustion of most of the blood from the aorta and some of the blood from the other arteries. The total volume of blood transferred before negative pressures appeared in the aorta, including that transferred through the peripheral blood vessels and through the shunt, averaged 75 (± 19) cc for a 12-kg dog before the negative pressures suddenly appeared at the end of the curve. This value of 75 cc, therefore, is a very rough estimation of the easily mobilizable blood in the large arteries. Obviously, it is much less than the total volume of blood in all of the arteries.
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Fig. 5. Mathematical and graphical method for changing pressure-time curves into pressure-volume curves. Curves represented are: A, pressure-time curves with affixed tangents at the 60 mm Hg levels; B, rate of emptying of the arterial system into the venous system at each second after stopping the heart; C, volume of blood which has left the arterial system at each second after stopping the heart; D, derived pressure-volume curve. (See text for explanation.)

Effect of Vasopressin on the Pressure-Volume Curves. It is quite difficult to record pressure-volume curves by the present method when pressor drugs are administered to the animal because, first, control conditions must be maintained extremely constant between the two intervals of heart stoppage and, second, because pressor drugs, in general, prevent satisfactory stoppage of the heart by vagal stimulation. Nevertheless, in two experiments in which the arterial pressure was elevated with vasopressin such pressure-volume curves were determined. Curves from one of these experiments are illustrated in figures 1 and 2. In both instances the curves had a slope \( \frac{dP}{dV} \) considerably greater than the average slope in normal dogs. In other words, as would be expected, the capacitances \( \frac{dV}{dP} \) of the arterial and venous systems were reduced.

DISCUSSION

Validity of the Pressure-Volume Curves. During measurement of the pressure-volume curves, some blood flow still occurred in the arteries and veins. Consequently, the pressures in the small vessels of the arterial and venous systems were not in complete equilibrium with the measured pressures in the aorta and right atrium respectively. Yet, the pressure differences were probably of little importance in recording the arterial pressure-volume curves, for even when the rate of blood flow through the arteries is quite rapid, the pressure differences among the larger vessels of the arterial tree are not great, and the large arteries are probably responsible for most of the characteristics of the pressure-volume curves for two reasons: first, the volume of blood in the major arteries of the systemic arterial system is much greater than the volume of blood in the small arteries (4). Second, it has been found that the distensibilities of the small arteries are far less than the distensibilities of the large arteries (5). Thus, because capacitance is equal to distensibility times volume, the total capacitance of all the small arteries calculates to be insignificant in comparison with the total capacitance of the large arteries. Therefore, it is believed that the pressure-volume curves recorded for the systemic arterial system are quite valid.

On the other hand, the picture is different for the veins, because as long as blood flows through the veins the pressures in different parts of the venous system are significantly different from each other. The cause of these differences is mainly the total collapse of many of the large veins, these collapsed veins offering considerable resistance to the flow of venous blood toward the heart (3). Therefore, for a second or more after the heart is stopped—that is, while the right atrial pressure is still low—right atrial pressure undoubtedly does not represent the mean pressure throughout even the large veins. Yet, it has been found previously that when the collapsed veins fill with blood the pressures
throughout the major veins equilibrate (3). Furthermore, after approximately the first second during the generation of a venous pressure-volume curve, the flow of blood from the peripheral veins toward the central veins decreases greatly, this also minimizing the pressure differences throughout the venous system. Therefore, the recorded venous pressure-volume curves are probably valid only during the latter half of their extent if this much, which means that the venous curves should be used only to illustrate general principles.

**Characteristics of the Arterial Pressure-Volume Curves.** The arterial pressure-volume curves recorded in this study exhibited almost precisely the same characteristics as published curves recorded from arterial segments (4, 6), aortas of dead animals (5, 7, 8), and aortas of live animals (9, 10). Also, the characteristics of the arterial curves recorded in this study were not greatly different from those of curves recorded for the pulmonary system (11). In general, the slope of the curves \(dP/dV\) was almost a linear function, which was also true of the different previously published arterial pressure-volume curves within the pressure range of the present studies (4 to 10).

**Characteristics of the Venous Pressure-Volume Curves.** For reasons discussed above the first portions of the venous pressure-volume curves are probably inaccurate because of collapsed veins in the venous system. However, during the latter part of the recorded pressure-volume curves, the slope of the curve \(dP/dV\) is reasonably linear. A similar linear function has been noted by other research workers studying isolated veins, using the same pressure range as that used in these experiments (4, 10, 12, 13).

**Importance of the Capacitances \(dV/dP\) of the Venous and Arterial Systems.** It is evident from figure 4 that approximately 60 cc more blood is present in the arterial system of a 12-kg dog when the heart is pumping blood than when it is not pumping blood. It is this extra 60 cc of blood in the arterial system which stretches the arterial walls and thereby elevates the pressure up to the normal arterial pressure. On the other hand, under dynamic conditions the quantity of blood in the venous system of a 12-kg dog is approximately 60 cc less than that present in the venous system under static conditions. Thus, the average volume of extra blood which the active heart depletes from the venous system and maintains continually in the arterial system has been measured for the dog.

From the pressure-volume curves which have been recorded in this study it is also possible to calculate how much the mean pressures of the venous and arterial systems will be affected by shifting a known quantity of blood between the two respective systems. Because the capacitance of the venous system, as measured in the present study, is 18–30 times as great as the capacitance of the arterial system, shifting blood from one system to the other should cause 18–30 times as much change in arterial pressure as in venous pressure. This ratio of pressure changes in the two systems has been corroborated by many previous studies, studies in which pumping by the heart was suddenly stopped by electrical fibrillation of the heart, by vagal stimulation of the heart, or by constriction of the central blood vessels mechanically (1).

Finally, the capacitance measurements of this study are valuable for mathematical analysis of factors affecting venous return to the heart and cardiac output. In a previous study from this laboratory the mathematical relationship for calculating the rate of venous return to the heart was developed as follows (2):

\[
VR = \frac{MCFP - RAP}{R_1C_1 + (R_1 + R_2)C_2 + \cdots + \frac{R_1 + R_2 + \cdots + R_nC_n}{C_1 + C_2 + \cdots + C_n}}
\]

in which \(VR\) is venous return, \(MCFP\) is mean circulatory filling pressure, \(RAP\) is right atrial pressure, \(C\) is the capacitance of each respective part of the circulatory system, and \(R\) is the resistance of each respective part of the circulatory system. It will be noted that the capacitances of the different portions of the circulatory system are among the most important factors which determine venous return and cardiac output. These effects have been discussed in the previous paper, pointing out that an approximate analysis of venous return can be computed when the capacitances of the arterial and venous systems are known.
Mathematics of the Method for Deriving Pressure-Volume Curves From Pressure-Time Curves. The mathematics of the method for deriving the pressure-volume curves of this present study is illustrated by figure 5 and may be described as follows: the heart of each dog was suddenly stopped by vagal stimulation two separate times, and two separate sets of pressure-time recordings for arterial and venous pressures were made. During one set of recordings blood was shunted from the arterial system into the venous system as described under methods. The rate at which blood was shunted from the arterial system into the venous system remained constant at approximately 15 cc/sec, until the arterial pressure fell to equal the rising venous pressure. The other set of recordings was made without shunting blood from the aorta into the venous system. Therefore, during one set of recordings the quantity of blood which flowed from the arterial system to the venous system, including both the blood flow through the peripheral vessels, and during the other set of recordings it was equal to the natural rate of flow through the peripheral vessels, and during the other set of recordings it was equal to the natural rate of flow plus an extra, known rate of flow.

Figure 5A illustrates the two respective arterial pressure-time curves recorded from the same dog; curve A was recorded with the external shunt open, and curve B was recorded with the external shunt closed. At any given pressure level on curve A the rate of blood flow (dV₁/dt) from the arterial system to the venous system is the same as that which leaves the arterial system, the values for volume change as determined from simultaneous arterial curves may be used in plotting the desired pressure-volume curve for the arterial system.

Solving for dV₁/dt, the equation may be expressed as follows:

$$\frac{dV_1}{dt} = K \frac{dP_1}{dP_1 - dP_2}$$

By making appropriate measurements from tangents at many points on the recorded pressure-time curves and using formula 4, the rate of blood flow from the arterial system to the venous system at many points along curve A can be derived, and it is possible to plot the rate of volume change in the arterial system against time, as illustrated in figure 5B. Then, using graphical methods for integrating curves, the curve of figure 5B is integrated to give the total volume change in the arterial system plotted against time as shown in figure 5C. Using curve A of figure 5A and the curve of figure 5C, both the pressure and the total volume change can be determined at each interval of time after the heart is stopped, and from these values the volume of blood in the arterial tree at each pressure may be plotted as shown in figure 5D, thus giving the desired pressure-volume curve for the arterial system.

A similar method may also be used for calculating pressure-volume curves of the venous system. Also, because the quantity of blood which enters the venous system is the same as that which leaves the arterial system, the values for volume change as determined from simultaneous arterial curves may be used in plotting the venous curves.

REFERENCES