Venous Return at Various Right Atrial Pressures and the Normal Venous Return Curve

ARTHUR C. GUYTON, ARTHUR W. LINDSEY, BERRY ABERNATHY AND TRAVIS RICHARDSON

From the Department of Physiology and Biophysics, University of Mississippi School of Medicine, Jackson, Mississippi

ABSTRACT

GUYTON, ARTHUR C., ARTHUR W. LINDSEY, BERRY ABERNATHY AND TRAVIS RICHARDSON. (U. Mississippi School Med., Jackson.) Venous return at various right atrial pressures and the normal venous return curve. Am. J. Physiol. 189(3): 609-615. 1957.—The normal venous return curve has been determined in 12 open-chest dogs with intact circulatory reflexes and in 14 open-chest areflex dogs. These curves show that venous return reaches a maximum value when the right atrial pressure falls to -2 to -4 mm Hg and remains at this maximum value down to infinitely low negative pressures. As the right atrial pressure rises to positive values venous return falls and reaches zero when the right atrial pressure has risen to equal the mean circulatory pressure. A venous return curve for the normal, intact dog has been tentatively formulated on the basis of these studies and previous studies in which individual points on the venous return curves of intact dogs have been measured.

When a change occurs in the hemodynamics of the circulatory system one cannot predict what will happen to the cardiac output unless he takes into consideration both the effect of this change on the ability of the heart to pump blood and also on the tendency for blood to return to the heart from the blood vessels. The ability of the heart to pump blood can be depicted by a curve showing cardiac output plotted against right atrial pressure, a type of Starling's curve used for many years to describe the functional ability of the heart. The tendency for blood to return to the heart from the circulatory system can be depicted by a 'venous return curve' which is a plot of blood flow into the right atrium against right atrial pressure.

The general characteristics of venous return curves are illustrated in figures 2-5 of this paper. All of these curves show that at negative right atrial pressures blood returns to the heart as rapidly as possible, but, as the right atrial pressure rises to positive values, a point is finally reached at which the back pressure from the right atrium is great enough to prevent all venous return.

If one can characterize both the cardiac output curve and the venous return curve in an animal, he can then predict by equating the two curves what the cardiac output and right atrial pressure will be. This procedure has already been explained in previous publications (1, 2), and it has proved to be especially valuable for analyzing quantitatively how much the cardiac output will be affected by exercise, sympathetic stimulation, transfusion, arteriovenous fistulae and many other factors that can change circulatory dynamics from the normal. Therefore, it has become important to record with as much care as possible the normal venous return curve to provide an accurate basis for these analyses.

METHODS

Thirty-one mongrel dogs of varying sizes, lightly anesthetized with sodium pentobarbital, were used in these studies. Arterial and venous pressures were recorded on a kymograph using mercury manometers with...
special zero adjustments and vibrators to eliminate friction losses. All pressures were measured with respect to the 'physiological zero reference point' which was defined in a previous paper (3) as 0.61 times the thickness of the chest anterior to the back surface of the animal.

The experiments of this study utilized open chest dogs employing the external perfusion system shown in figure 1. The right atrium was cannulated and all blood that flowed into the right atrium was perfused through a horizontal section of thin rubber tubing, then through a pump, through a rotameter type flowmeter, and finally back into the pulmonary artery. In other words, the system replaced the right ventricle. The pump was adjusted to a sufficiently high rate that it provided more than enough suction to keep the thin section of rubber tubing always in a semi-collapsed condition. This maintained the pressure at the beginning of the thin tubing at zero mm Hg with respect to the atmosphere, and by raising or lowering the tube, the zero pressure point of the perfusion circuit could be set at various hydrostatic levels thereby varying the right atrial pressure from very high positive values to very low negative values.

The animal was transfused with sufficient blood to fill the perfusion system and also to replace blood lost incident to the surgery.

In about one-half of the experiments special precautions were taken to block the animal's vasomotor reflexes. To do this 20 ml of saline containing a total of 150 mg of Metycaine were injected intraspinally. This blocked all nervous outflow from the spinal cord including even the respiratory impulses over the phrenic nerve. Also, the blood pressure fell to an average of approximately 40 mm Hg. Artificial respiration was instituted, and an epinephrine drip at a rate of .00045 mg/kg/min. was started to reestablish the blood pressure at a value equal to that of the normal animal.

RESULTS

Venous Return in the Normal Open-Chest Dog. Figure 2 illustrates 12 venous return curves recorded in open-chest dogs. During each experiment, precautions were observed to maintain the right atrial pressure at a negative value of approximately -8 mm Hg thus promoting as much venous return as possible and thereby preventing progressive damage to the preparation. When a venous return measurement was to be made at any given positive right atrial pressure, the pressure was elevated rapidly to the desired value and held there only 8-10 seconds. By this time an equilibrium rate of flow had been established and the venous return measurement could be made. Prolongation of positive pressures for periods longer than this was extremely traumatic to the preparation and also elicited undesired vasomotor reflexes as evidenced by reactive changes in blood pressure and heart rate. After a sufficient number of points on each venous return curve had been recorded, the curve was plotted.

All the normal venous return curves obtained in this manner were averaged together by weighting each point on the curve in proportion to the weight of the animal; the resulting average curve is illustrated by the solid line of figure 4.

Venous Return in the Areflex, Open-Chest Dog. Fourteen dogs were prepared in the same manner as those above except that they were rendered areflex by administering spinal anesthesia as described under METHODS. The venous return curves in these dogs are illustrated in figure 3, and the average venous return curve obtained by weighting each point on the curves in proportion to the weight of the animal is illustrated by the dashed curve of figure 4.

Plateau of the Venous Return Curve. The curves of figures 2-4 illustrate that venous return reaches a maximum value and remains on a plateau at all right atrial pressures more negative than -2 to -4 mm Hg. Previous studies both from this laboratory and elsewhere have indicated that this is caused by collapse of the veins leading into the right atrium at these negative pressures (4, 5). Observation of the veins and right atrium of the open-chest dogs in the present experiments further corroborated these previous studies, for progressive collapse of the veins and right atrium was evident as the right atrial pressure was progressively decreased below the level at

Fig. 1. External perfusion system for controlling right atrial pressure and venous return.
which the venous return curve reached its plateau.

**Sharpness of the Upper Inflection in the Venous Return Curve.** The sharpness of the upper inflection in the venous return curve was shown to depend on two factors, *a)* the degree of venous pulsation and *b)* the difference in hydrostatic levels of the different veins entering the chest. In five experiments on areflex dogs additional to the ones reported above, the thin-walled rubber tube connected to the outflow of the right atrium was not used. As a result, the pulsation from the roller pump was transmitted backward into the right atrium. In these experiments, the inflection of the curve began at an average of +5 mm Hg and was not complete until the right atrial pressure fell to an average of +8 mm Hg, the exact values depending on the amplitude of the pulsations. Simply inserting the collapsible tube between the atrium and the pump removed most of these pulsations, though not all, and as a result the sharpness of the curve changed until the inflection now began at approximately +2 mm Hg and reached a plateau at +2 to −4 mm Hg. This is the effect which one would expect, for when the pressure is low during part of a pressure pulsation, venous return may reach a maximum and remain at this maximum regardless of how low the pressure falls. On the other hand, when the right atrial pressure rises high enough to overcome the venous collapse, return of blood to the ventricle decreases approximately in proportion to the elevation of pressure above the collapse point. These effects cause a phenomenon of ‘rectification’ that makes the upper inflection gradual rather than sharp.

The second cause of a gradual inflection rather than a sharp one seems to be the entry of some veins into the thorax at higher hydrostatic levels than others. Visual observation of the veins in the present experiments demonstrated that those veins entering the anterior part of the thorax began to collapse when the right atrial pressure was about +3 mm Hg, while the veins entering posteriorly collapsed only when the right atrial pressure reached −2 to −4 mm Hg. This is another reason why venous return curves do not reach a plateau suddenly and sharply at zero right atrial pressure, which one would expect if all veins were
at the same hydrostatic level and no pulsations were present.

Inflection in the Lower Part of the Venous Return Curve. It is evident from the figures that a slight concave inflection is found at the lower end of most venous return curves, and in some cases the inflection is marked. It was not possible to determine the entire genesis of this inflection, but, as was true in relation to the upper inflection of the curve, the more intense the right atrial pulsations, the more intense and more elongated was the lower inflection. This appeared to result from "rectification," for as the right atrial pressure approached the value at which all venous return ceased, the blood flowed in spurts rather than steadily. Under these conditions, it would not be mean right atrial pressure which would determine venous return but, instead, the pressures during the portions of the cycle when the blood is flowing. The mean value of these pressures can be several millimeters of Hg less than the true mean pressure, which could explain much of the lower inflection in the curve.

DISCUSSION

Validity of the Venous Return Curves. Because the dogs used in these experiments were subjected to traumatic surgery prior to measuring the venous return curves, one would question whether or not the measured curves are reasonably accurate for a normal dog. Yet, extreme precautions were observed to keep the circulatory systems as nearly normal as possible during the course of the experiments. Any animal which evidenced a cardiac output not in the normal range for open-chest dogs or an arterial pressure less than normal was discarded. Also, except while measurements were actually being made, every attempt possible was observed to prevent the development of positive pressure in the right atrium, for it has long been the experience in this laboratory that the circulation of open-chest animals can be maintained essentially normal only so long as venous flow into the heart is unimpeded. Indeed, the suction applied to the right atrium by the external perfusion system of the present experiments was found to delay deterioration of the preparation rather than to enhance it. This indicates that even the normal rise in right atrial pressure incident to opening the chest can be a deteriorating factor on the circulation, and that the present experimental procedure, in which the right atrial pressure was...
NORMAL VENOUS RETURN CURVE

Fig. 4. Average venous return curves. Solid line represents the average curve recorded in normal open-chest dogs, and the dashed line represents the average curve recorded in areflex, open-chest dogs.

maintained at negative values, more nearly reduplicates normal circulatory conditions than does the usual open-chest preparation.

To prevent distortion of the venous return curve during the course of its measurement by changing vasomotor reflexes, two precautions were observed: in the animals which were not rendered areflex all measurements were made within 8–10 seconds after the right atrial pressure was elevated to a positive value, and then the pressure was immediately returned to a negative value. Previous studies have shown that vasomotor reflexes do not become significant in this short time in the dog (6, 7). The second precaution was to determine normal venous return on 14 areflex dogs in which the vasomotor reflexes were totally abrogated but in which normal vasomotor tone was maintained by continuous epinephrine drip. It was gratifying to note the close correspondence, as illustrated in figure 4, between the curves obtained in the areflex dog and those obtained in dogs which still had intact reflexes. This gives at least some assurance that the venous return curves determined in these studies are a good indication of the ability of the circulation to return blood to the heart in the open-chest dog.

Venous Return Curve of the Intact Dog. To determine an entire venous return curve in an intact dog would probably be an almost impossible task because, first, it is very difficult to raise and lower the right atrial pressure of an intact animal without greatly altering some other characteristic of the circulation, and, second, it is almost impossible to make rapid and very accurate measurements of cardiac output in a few seconds time in the intact animal; failure to make rapid measurements leads to distortion of the curve because of changing vasomotor tone during the course of the measurements.

Yet, it has been possible to determine quite accurately a number of individual points on the venous return curve of intact dogs. The most important of these points is the terminal point of the curve at zero venous return. At this point the right atrial pressure equals the mean circulatory pressure which is defined as the equilibrium pressure throughout the circulation at which all blood flow would cease. In a previous study, the mean circulatory pressure was measured in 18 dogs within a few seconds after pumping by the heart had been stopped, and the average of these determinations when referred to the physiological zero reference point was 7.0 ± 0.9 mm Hg (7). Observing the curves of figure 4, it can be seen that the mean pressure at which venous return reached zero in the open-chest dogs was less than 1 mm greater than this value, which shows that this very important terminal point of the curve is essentially the same for open-chest and normal intact dogs.

Points on the upper part of the curve at the beginning of the plateau can be determined by measuring simultaneously the cardiac output and right atrial pressure in normal dogs. This has been done many times in this laboratory and others. The usual right atrial pressure, referred to the physiological reference point, is approximately —2 mm Hg (3). The cardiac output as determined by the Fick and Stewart methods ranges about 30% higher than the measurements of this study (8), which is the result to be expected because of the surgical trauma of the experiments. Therefore, if one should plot the normal values for cardiac output and right atrial pressures against each other, the plotted points would fall on curves about 30% higher than those recorded for the open-chest dog.

The only portion of the venous return curve which seems to remain in doubt for the intact dog is the downward slope. All experiments to date have shown this portion of the curve to be almost a straight line except for a minor
inflection pointed out under RESULTS. A relatively straight line was also predicted by mathematical analysis of venous return (9), the formula for which indicates that as long as the thoracic veins do not collapse venous return should be proportional to the mean circulatory pressure minus the right atrial pressure. Therefore, the doubtful portion of the venous return curve in the intact dog can probably be assumed to be almost a straight line.

In summary, on the basis of discrete points determined in intact animals, it appears that venous return curves for intact dogs should be almost exactly the same as those measured in the present experiments except that they should range about 30% higher. The solid curve of figure 5 illustrates the curve for the normal dog as it would be predicted from this analysis and from the mean value determined by Wiggers (8) for the circulatory index of dogs, 2.87 l/min/m².

**Predicted Venous Return Curve of the Human Being.** Obviously, the venous return curve determined in dogs can indicate only a general pattern of what one would expect in the human being. In dogs the terminal point on the curve at zero venous return—that is, the mean circulatory pressure point—remains almost precisely 7.0 mm Hg regardless of the size of the dog (7), which indicates that the size of the animal does not affect this point. Unfortunately, the mean circulatory pressure has never been determined in the normal human being, but static pressure measurements made in dead persons have been approximately the same as those measured in dead dogs when the dogs had essentially the same circulatory conditions as the human beings prior to death (10). All of these facts indicate that the terminal point of the normal human venous return curve should be essentially the same as that for the dog.

The level of the plateau of the normal human venous return curve can be predicted from the normal cardiac output which is about 5.5 l/min. or 3.0 l/min/m² (2). The dashed curve of figure 5 represents the estimated normal venous return curve for the human being based on this analysis. Because of the drastic procedures required to determine the normal venous return curve, it is doubtful that it will ever be measured in its entirety in the human being. For this reason an estimate of the curve is probably the best that can be accomplished.

**Equating the ‘Normal’ Venous Return Curve With Cardiac Output Curves.** A venous return curve is almost meaningless unless it is used simultaneously with a cardiac output curve. Figure 6 illustrates the normal venous return curve equated with three different cardiac output curves. The ‘normal’ cardiac output curve of the figure depicts the approximate ability of the normal, unstimulated heart to pump blood. The ‘equilibrium point’ at which the venous return curve crosses this output curve describes the actual cardiac output and...
the actual right atrial pressure under these conditions. If the heart is subjected to extreme sympathetic stimulation, which increases its efficiency as a pump, the cardiac output curve becomes the upper one. If the venous return curve remains constant, then the effect will be described by the point at which the venous return curve crosses the new cardiac output curve. In other words, the cardiac output will rise to slightly above normal, and the right atrial pressure will fall to slightly below normal. Finally, if the heart’s ability to pump blood should be greatly decreased, thereby decreasing the output curve to the lower one of figure 6 then the venous return and cardiac output curves will equate at a relatively low cardiac output and an elevated right atrial pressure. 

Unfortunately, analyses of the effects of different circulatory factors on cardiac output and right atrial pressure by these two types of curves are usually not this simple, for ordinarily any change which affects the heart will cause reactive changes in the venous return curves and vice versa. The general trends of these different changes in venous return curves with changing hemodynamic conditions have been presented previously (1, 2), but precise analyses must await further more accurate studies.

REFERENCES