Decrease of Venous Return Caused by Right Atrial Pulsation

By Arthur C. Guyton, M.D., Jimmy B. Langston, Ph.D., and Oliver Carrier, Jr., B.S.

With the technical assistance of J. B. Abernathy

For almost as long as the problem of venous return has been studied, two different factors have been claimed to be important in determining venous return to the heart.1-3 These are: (1) the force of the systemic arterial pressure pushing blood through the systemic circulation toward the heart, and (2) factors on the venous side of the heart that affect venous return, including rhythmic suction of blood from the veins. These two effects have also been called vis a tergo ("force from behind") and vis a fronte ("force from the front"). The importance of the vis a tergo has never been doubted by any investigator; this has been reviewed in detail in a monograph by Franklin1 and in a review by Landis and Hortenstine.2 However, the importance of rhythmic suction of blood into the heart as the result of either cardiac or respiratory suction has been very controversial.

Recently Brecher, in his monograph on venous return,3 has emphasized the importance of rhythmic suction, both cardiac and respiratory, as one of the important factors in promoting venous return to the heart. He has especially shown that suction resulting from respiratory movements enhances venous return and has also demonstrated that the heart can actually suck blood from the veins during certain phases of the cardiac cycle.4 Also, O'Brien has confirmed the ability of the left ventricle to suck blood into its cavity.5

Furthermore, Brecher's studies indicate that rhythmic cardiac and respiratory suction of blood from the veins not only increases instantaneous flow of blood into the heart during the period of suction but also increases the mean venous return integrated over a period of time.

The present studies were designed partly to determine the mechanism by which rhythmic suction increases the mean venous return. This could occur in one or both of two different ways: (1) The rhythmic suction could aid in filling the heart and in this way increase the efficacy of the heart as a pump, thereby reducing the mean right atrial pressure. The reduced right atrial pressure, in turn, would then be the cause of the increased venous return. (2) The rhythmic pulsation induced by rhythmic suction in the right atrium and veins could possibly have some direct effect on blood flow along the veins to increase the rate of venous return. To paraphrase these two mechanisms: is it the rhythmic pulsation itself that increases venous return, or is it reduced mean right atrial pressure that increases the return?

To separate these two possible mechanisms as well as to determine the basic effect of atrial pulsation on venous return, artificial pulsations were created in the right atrium by causing rapid inflow and outflow of blood to the right atrium, using a mechanical pulsator. The pulsations were not synchronous with cardiac action and therefore did not increase the effectiveness of the heart as a pump. Furthermore, the mean right atrial pressure was controlled independently of the degree of pulsations. Therefore, it was possible to determine whether pulsation per se, rather than changes in mean right atrial pressure, had a beneficial or harmful effect on venous return. The results of the present...
experiments indicated that pulsations per se have detrimental effects on blood flow from the peripheral veins to the right atrium. However, they also showed that very slight reduction in mean right atrial pressure, which can easily result from rhythmic suction, can cause marked increase in venous return.

RECTIFICATION OF VENOUS PRESSURE CAUSED BY PULSATILE RIGHT ATRIAL PRESSURE, AND THE EFFECT OF THIS ON VENOUS RETURN

To make the results to be presented in this study more meaningful, it is necessary to discuss at this point the phenomenon of rectification that occurs in any hydraulic, electrical, or pneumatic circuit in which pulsation causes repetitive resistance changes during different phases of the pulse cycle. This phenomenon, as it applies to the present studies, can be explained as follows:

First, figure 1(A) shows a segment of a vein as it enters the thoracic cavity and then empties into the right atrium. To the left is the right atrial end of the vein, and to the right is the peripheral end. When the right atrial pressure is elevated, the vein becomes distended, as indicated by the dark outlines. However, when the right atrial pressure falls to a sufficiently negative value, the atrial end of the vein collapses. The dark curve of figure 1(B) shows the relationship of the nonpulsatile right atrial pressure to the venous pressure immediately peripheral to the collapse point. This curve was derived from data obtained in over 200 separate experiments in which pulsations per se have detrimental effects on blood flow from the peripheral veins to the right atrium.
this laboratory, which showed in essence that, as long as the right atrial pressure is above 0 mm. Hg, there is no measurable difference between the right atrial pressure and the venous pressure immediately before entry of the veins into the thorax. However, as the right atrial pressure falls below 0 mm. Hg, a disparity does develop. By the time the right atrial pressure has fallen to approximately —4 mm. Hg, the central veins reach complete collapse, and the pressure immediately peripheral to the collapsed veins never falls significantly below 0 mm. Hg. Further decrease in right atrial pressure, even down to values as low as —20 to —30 mm. Hg, will not cause further decrease in the venous pressure beyond the collapsed segment.

Superimposed on this graph is a horizontal sine wave right atrial pressure pulsation, and superimposed to the right is the theoretical resultant venous pressure pulsation immediately peripheral to the collapsed segment of the vessel. The entire diagram in figure 1(B) is a "rectification diagram"; it illustrates rectification of alternating positive and negative right atrial pressure to give a mean venous pressure considerably above the mean right atrial pressure. In this instance, the mean atrial pressure is 0 mm. Hg, and the mean venous pressure beyond the collapse point is 6\(\frac{1}{2}\) mm. Hg.

Figure 1(C) illustrates the effect on venous pulsation of progressively increasing the amplitude of the right atrial pulsation. The dashed line in this figure shows a progressively rising mean venous pressure as the pulsatile right atrial pressure increases to very great pulsations. At the very large pulsations, the mean venous pressure in the figure rises to as high as +10 mm. Hg.

Finally, in figure 1(D) is shown another type of rectification diagram, illustrating rectification of the alternating flow component of venous return as the right atrial pressure pulsates. In this figure, the dark curve is an average venous return curve as determined in this laboratory, and it represents the effect of nonpulsatile right atrial pressure on venous return. The plateau in the curve at negative right atrial pressure is caused by collapse of the veins, which can be readily understood from the discussion of figure 1(B) and (C). If we superimpose rhythmical pressure pulsations on the right atrial pressure axis, as shown by the horizontal sine wave, then the venous return can be altered even though the mean right atrial pressure remains constant. The superimposed pressure pulsations in this figure vary from —6 mm. Hg to +2 mm. Hg, which is a reasonable physiological range for normal right atrial pulsation in the closed-chest animal. The vertical pulsatile curve to the right in the figure shows the theoretical effect of the pressure pulsations on venous return. Note that when the mean right atrial pressure is —2 mm. Hg but is not pulsating, the venous return is approximately 1,200 ce./min. However, when the right atrial pressure is pulsating, the instantaneous venous return falls from 1,200 to 800 ce./min. during the positive phase of the pressure pulsation and rises only from 1,200 to 1,250 ce./min. during the negative phase of the pulsation. As a result, the average venous return falls from 1,200 ce./min. to approximately 1,030 ce./min., despite the fact that the mean right atrial pressure remains exactly constant. This curve illustrates that even normal pressure pulsations, if they cause alternating collapse of the veins, could be a detrimental influence on venous return, though probably not seriously so because of their small amplitude.

It should be especially noted that the concepts presented in figure 1 are almost identical with those that have been discussed in detail by Holt and Dumaresq. Furthermore, these concepts apply to negative pressures caused by respiratory suction as well as those caused by cardiac suction. It should be noted again from the figure that collapse of all the central veins ordinarily is not complete until the mean right atrial pressure falls to an average of —4 mm. Hg. This fact must be emphasized because the collapse phenomenon is often misconstrued to mean collapse of all veins instantly and completely at all values of right atrial pressure below 0 mm. Hg.
VENOUS RETURN AND ATRIAL PULSATION

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There are at least two reasons for the gradual, rather than instantaneous, transition from the noncollapsed to the collapsed state, as follows: First, a small amount of negative force is required actually to pull all the surfaces of the veins together; this is particularly true of the veins in the liver. Secondly, different veins enter the heart and thoracic cavity at different hydrostatic levels, which would also cause a gradual, rather than sudden, transition from the noncollapsed to the totally collapsed state.

When the right atrial pressure is rhythmically raised and lowered at very slow frequencies, 1 to 20 cycles per minute (c.p.m.), it is very easy to show that this phenomenon of rectification always occurs and that the average venous return always becomes reduced. However, in the normal animal the pulsatile right atrial frequencies are often as high as 200 c.p.m., which has made it necessary to determine whether or not the rectification phenomenon still holds at these higher frequency values. This has been the purpose of these studies.

Methods

Experiments were performed in 15 open-chest dogs, weighing approximately 15 Kg., anesthetized with 30 mg./Kg. of sodium pentobarbital and heparinized with 5 mg./Kg. of heparin.

A large glass cannula was sutured in the lateral wall of the right atrium, and blood was pumped (by a Sigmamotor pump) from this through an external perfusion system, and thence back into the pulmonary artery. In this external perfusion system was a rotameter for measuring flow, an electric heater for maintaining normal blood temperature, and damping systems both at the inlet and outlet of the pump to reduce the transmission of pump pulsations into the right atrium or pulmonary artery. The proximal end of the pulmonary artery was tied so that all the blood entering the right atrium had to flow through the external perfusion system and the flowmeter in the perfusion system constantly measured total venous return (or cardiac output). The volume of blood pumped per minute could be varied from 6 L. per minute down to zero, thereby altering the mean right pressure from as low as -30 mm. Hg up to a positive value high enough to stop completely all venous return (usually about +10 mm. Hg).

To introduce pulsations into the right atrium, blood was rapidly injected and removed through a sidearm of the large right atrial cannula. The frequency of this pulsatile inflow and outflow was 60, 100, 160, or 240 c.p.m., and the volume per pulse was continuously variable from 0 to 68 cc. The pulsating device was a pulsatile air pump that gave sine wave pulsations; these pulsations were transferred to the blood by an exchange chamber divided by a rubber dam, with blood on one side of the rubber and air on the other side.

Both mean and pulsatile right atrial pressures were recorded on a Sanborn recorder, using a Statham strain gauge. This strain gauge was attached to a hardened plastic catheter (almost as hard as glass) that was inserted into the right atrium through the auricular appendage. Arterial pressure was monitored by an additional strain gauge connected to a hardened catheter inserted in the femoral artery. All pressures were referred to a zero hydrostatic level 0.61 times the chest thickness in front of the back, a point chosen for reasons discussed previously.

Results

Figure 2 illustrates artificial pressure pulsations induced in the right atrium by the apparatus described above. The degree of pulsation, its frequency, and the mean right atrial pressure could be varied at will.

DEMONSTRATION OF RECTIFICATION IN VENOUS RETURN

Figure 3 illustrates simultaneous recordings of venous return (cardiac output) and right atrial pulsations. Note that, as long as the positive phase of the right atrial pulsations remained less than -4 mm. Hg, there was no decrease at all in venous return from the control value despite the presence of the large pulsations. However, just as soon as the positive peak of the right atrial pulsations
began to rise about \(-4\) mm. Hg, and especially when it rose above \(0\) mm. Hg (which is precisely the same range in which the venous return curve illustrated in figure 1(D) begins to curve downward), the venous return began to fall. As the right atrial pulsations rose still higher, the venous return progressively fell. The principal point of figure 3 is that even an instantaneous elevation of right atrial pressure above the point at which the venous return curve begins to turn downward exerts a detrimental effect on venous return. A second point of the figure is that whenever the right atrial pressure is very negative, even extreme pulsation has essentially no effect on venous return. The effect occurs only when the pulsation is in the pressure range at which the venous return curve bends from the plateau phase to the downsloping phase. This, it must be noted, is in the normal right atrial pressure range.

The rectification phenomenon was demonstrated at each pulsatile frequency in each of the 15 experiments, and at each frequency it was demonstrated at as many as four to seven different pulse volumes. In no experiment did we fail to demonstrate the effect.

**Effect of Pulse Volume on Venous Return**

Figure 4 illustrates the effect on venous return of changing the pulse volume. In this experiment, the mean right atrial pressure was \(-7\) mm. Hg, and the pulse frequency was 100 c.p.m. The mean right atrial pressure of \(-7\) mm. Hg was slightly below the pressure level at which the venous return curve began to slope downward. Therefore, superimposed pressure pulsations of sufficient pulse pressure would be expected to reduce the venous return because of the rectification phenomenon. Note that even a 10-cc. pulse volume reduced the venous return to 90 per cent of normal. Higher pulse volumes, 22 to 68 cc. per pulse, reduced the venous return to as little as 20 per cent of normal.

At first, it was not evident how pulsatile right atrial pressure and the rectification phenomenon could cause venous return to fall below 50 per cent of normal because, theoretically, normal flow of blood to the heart should occur at least 50 per cent of the time during positive and negative pulsation. However, two observations demonstrated the cause of the excessive decrease in venous return when the pulsatile volumes were extremely large. First, at the large pulsatile volumes
the volume of blood entering the right atrium at each positive phase of the cycle was in the order of two times as great as the amount of blood that the right atrium itself would hold, and during the course of the experiments the veins beyond the right atrium could be seen to dilate markedly during the positive pulses. Thus, during each positive pulse, a large quantity of blood actually flowed backwards from the right atrium into the venous tree, and all of this blood had to be removed from the venous tree during the negative pulse phase before any net flow of blood into the right atrium could result. Secondly, if we refer again to figure 1(C) it becomes evident that the pressure rectification phenomenon can cause mean right atrial pressures to rise actually higher than the mean value, 7 mm. Hg, usually required to stop all venous return. Indeed, in several of the individual smaller dogs pulsatile volumes of 68 cc. per pulse did cause zero net return to the heart.

The effect of pulse volume was demonstrated in every experiment and at each pulse frequency with almost no quantitative difference from dog to dog.

**EFFECT ON VENOUS RETURN OF VARYING THE MEAN RIGHT ATRIAL PRESSURE DURING ATRIAL PULSATATION**

Figure 5 illustrates the effect on venous return of progressively raising the mean right atrial pressure from —28 mm. Hg up to +12 mm. Hg under the following conditions: (a) while there was no artificially induced pulsation, (b) while pulse volumes of 4 ml. at 60 e.p.m. were applied, (c) while pulse volumes of 8 ml. at 60 e.p.m. were applied, and (d) while pulse volumes of 22 ml. at 60 e.p.m. were applied.

Note in this figure that, when no artificial pulsation was applied, raising the mean right atrial pressure caused no detrimental effect on venous return until the right atrial pressure reached —1 mm. Hg. Then further rise in right atrial pressure caused rapid decrease in venous return; this effect was also illustrated for the typical venous return curve in figure 1. When progressively greater pulse volumes were applied, the venous return began to decrease at lower and lower mean right atrial pressures. This result can be explained in the following way: when pulsation in the right atrial pressure is extreme, even though the mean pressure is very low, the positive pressure peaks can still rise high enough to cause rectification in the venous return, thereby decreasing venous return.

Note especially in this figure that all of the curves come very nearly together when the mean right atrial pressure has risen into the range of +5 to +9 mm. Hg. Actually, in this range the deviation of the curves from each other is within the limits of experimental error, and in some of the other experiments the curves did overlap each other. The reason for this was discerned by studying the pulse records. It was found that at these high right atrial pressures the negative peaks of the pressure pulsations did not fall to a low enough pressure level ever to cause collapse. This readily explains why the right hand ends of the curves run almost together.

At very low mean right atrial pressures, the venous return was also unaffected by pulsations of different amplitude. Here, it was found that the positive peaks of the pulsations, whatever their amplitude, failed to rise high enough ever to open up the collapsed veins. Therefore, no rectification could occur in this pressure range either.

On the other hand, when the mean right atrial pressure was near the zero level, the disparity between the different curves was at its greatest. It is in this range that the pres-
sure pulsations caused collapse of the veins during the negative half of the cycle and filling of the veins during the positive half of the cycle thus resulting in typical rectification.

Qualitatively (and almost quantitatively), precisely the same results were demonstrated in all of the experimental animals.

**EFFECT OF PULSE FREQUENCY ON VENOUS RETURN**

In all animals, artificial atrial pulsations were applied at 60, 100, and 160 c.p.m. In three of the experiments a frequency of 240 c.p.m. was also used. Furthermore, at each of these frequencies, the effects of at least five different pulse volumes were studied at all ranges of mean right atrial pressure from −28 up to +12 mm. Hg. In these studies, we could discern no consistent difference between the effect of one frequency versus another. If anything, the depressant effect on venous return was greater at higher frequencies than at lower frequencies, the cause of which was not apparent. However, these results do show that even at higher frequencies the rectification phenomenon still occurs equally as well as at lower frequencies. This finding is important because many of the pulsatile components of the normal right atrial pressure wave do have relatively high frequencies.

**SUMMARY OF RESULTS**

Over 250 separate records were made of the effects on venous return of artificially imposed right atrial pressure pulses. Pulse volumes were varied from 0 to 68 cc. per pulse, mean right atrial pressures from −28 to +12 mm. Hg, and frequencies from 60 to 240 c.p.m. In not one single instance did the artificial superimposition of pulsations increase the venous return. On the contrary, the venous return was always depressed whenever the mean right atrial pressure was in the normal physiological range (−3 to +3 mm. Hg). The greater the degree of pulsation, the lower the mean right atrial pressure had to fall to prevent the depressant effect on venous return.

The records and curves shown in figures 2 through 5 were all repeated almost precisely in each one of the 15 animals. There were no qualitative differences in the results; the only differences were quantitative and even then only minor ones.

**Discussion**

Under the conditions of these experiments, artificial pulsations in the right atrium always reduced venous return when the mean right atrial pressure was in the normal physiological range. These experiments showed that the rectification phenomenon discussed in the introduction to this paper does apply to venous return—that is, a rise in right atrial pressure above −4 mm. Hg, even for a small fraction of a second, depresses venous return, while a fall in right atrial pressure below this level fails to increase venous return; as a result, the average venous return falls when the right atrial pressure is pulsatile.

However, it must be recognized that the artificial pulsations induced in the right atrium in these experiments were not synchronized with the heart beat, and the results could have been entirely different with synchronized pulsations. Indeed, at least two separate types of synchronous right atrial pulsations have definitely been shown to aid cardiac function and thereby to increase venous return. For instance, atrial contraction occurring immediately before ventricular systole adds to the priming volume of the right ventricle and thereby increases the effectiveness of the heart as a pump. This, in turn, decreases the mean atrial pressure, consequently increasing venous return. Likewise, Brecher has shown almost conclusively that the downward thrust of the atrioventricular junction of the heart during ventricular systole causes blood to be sucked out of the central veins into the right atrium. This constitutes a type of right atrial pulsation, and it increases the effectiveness of the heart as a pump, thus also increasing the rate of venous return.

Now, let us return to the two mechanisms discussed in the introduction by which right atrial pulsation could benefit venous return. First, do the synchronous pulsations discussed...
in the above paragraph increase venous return by lowering the mean right atrial pressure, or, secondly, do they benefit venous return by some direct effect of the pulsations, independent of mean right atrial pressure changes, on blood flow in the veins? In the present experiments the pulsations were not synchronized with cardiac action. However, there is no reason to believe that the effect of these pulsations on the veins should have been different from the effect of the natural pulsations from the heart. Yet, the pulsations caused only harm to venous return and never benefit. Therefore, we conclude that right atrial pulsation per se causes no direct effect on the veins to benefit venous return.

In the case of the synchronous pulsations which do benefit venous return, the increase in return could easily result from decreased mean right atrial pressure resulting secondarily from increased effectiveness of the heart as a pump. In support of this concept, the present experiments, as well as previous ones from this laboratory, showed that only a 1 mm Hg fall in right atrial pressure enhances venous return about 14 per cent when this pressure is in the positive range and to a lesser extent until the veins collapse completely at about −4 mm Hg. Therefore, we can see that even slight enhancement of cardiac effectiveness as a pump could easily exert a tremendously important effect on venous return.

It is reasonable to suspect that the systolic suction effect demonstrated by Brecher also reduces the mean right atrial pressure sufficiently to cause the increased venous return which he observed. Unfortunately, he did not publish records or information giving the effect of systolic suction on the mean right atrial pressure. However, in other studies in which he discussed the importance of negative intrathoracic pressure and of inspiration on venous return, he did publish records from which it could be discerned that the mean right atrial pressure had fallen enough to account for the increased venous return. Here again, we must keep in mind that a 1-mm Hg fall in right atrial pressure can cause as much as 14 per cent increase in venous return.

To conclude this discussion, it is our belief that much of the controversy over the relative importance of venous pulsations in affecting venous return has actually been a problem of semantics. There is no doubt that right atrial contraction immediately before ventricular systole increases the effectiveness of the heart as a pump and, therefore, also increases venous return. Yet, it would be completely wrong to suppose that right atrial contraction increases venous return by virtue of some effect that the resultant pulsations have on the peripheral veins. By the same token, suction of blood from the central veins into the right atrium during ventricular systole makes increased quantities of blood available to the right ventricle when the A-V valves open, and this, too, undoubtedly increases the effectiveness of the heart as a pump. When the heart becomes a more effective pump, it not only increases the cardiac output but also reduces the mean right atrial pressure as well, and the overall effect is enhancement of venous return. Here again, it must not be assumed that this systolic suction of blood from the veins increases venous return because of some pulsatile effect on the veins.

From the above discussion, the reader will immediately recognize that much of the misunderstanding that has developed in relation to venous return could easily have resulted from semantics rather than from conflicting experimentation. We suggest, for instance, that the terms *vis a tergo* and *vis a fronte* are very misleading and do not fit well into modern concepts of circulatory dynamics. Furthermore, venous return should never be considered separately from cardiac output except under transient conditions, and the idea that cardiac output is controlled by the heart while venous return is controlled by peripheral factors is completely untenable. Three separate, independent, and different circuit analyses of the circulation have now been published, all of which analyze the factors affecting venous return and cardiac output simultaneously rather than separately. The results
of these analyses are almost identical despite their different origins, and all of them have demonstrated that the concepts of *vis a tergo* and *vis a fronte* are almost hopelessly inadequate. However, if ever we do discuss venous return separately from cardiac output, then it is certainly much better to talk in terms of pressure gradients within the systemic circulation than to talk about a positive force at one end of the circulation and a sucking force, endowed with especially important pulsatile properties, at the other end.

**Summary**

Over 250 records were made in 15 dogs to show the effect of artificially induced right atrial pressure pulsations on venous return. In not a single instance did the induced pulsations increase venous return, but often it decreased venous return. On the other hand, a very slight decrease in mean right atrial pressure down to the pressure level that the veins collapse completely was shown to increase venous return markedly. These experiments indicate that the benefit to venous return which results from rhythmic cardiac or respiratory suction is caused by a decrease in mean right atrial pressure.

**References**

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Circ Res. 1962;10:188-196
doi: 10.1161/01.RES.10.2.188

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1962 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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