Digital Hemodynamics in the Normotensive and Hypertensive States

II. Venomotor Tone

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The single factor implicit in the sustained hypertensive state, regardless of etiology, is an increase in peripheral vascular resistance. It is generally agreed that this results from a narrowing of the small arteries and arterioles. Although the small veins and venules possess anatomically and physiologically similar smooth-muscle elements, they have not been implicated in this increase in vascular resistance.1-3

Burch4-7 has recently demonstrated the usefulness of rheoplethysmography in evaluating the status of the postcapillary digital vessels. A study of the tone of the small veins and venules in the normotensive and hypertensive states was therefore undertaken.

Theory

When the occluding cuff adjacent to the plethysmographic cup is suddenly inflated, the volume of inflowing blood is trapped within the digital vessels enclosed by the cup, and a volume-time course curve of the inflow (Iv) is recorded. As the inflowing blood accumulates in the small venous reservoir, the pressure in the venules and small veins begins to rise, and the arteriovenous (AV) gradient is reduced. Over the course of a number of pulse cycles this reservoir, if in a relatively relaxed state, is able to accommodate the quantity of inflowing blood. Thus the AV gradient does not critically approach zero, the pressure within the venous system remains below the occluding cuff pressure, and the rate of inflow (Ir) does not reach zero (fig. 1).

If, however, the postcapillary system is “tight” reflecting an increase in venomotor tone, the same quantity of inflowing blood quickly fills the venous reservoir, thereby reducing the AV gradient, and the pressure in this system increases to a value equal to that of the occluding cuff pressure. Outflow is therefore initiated, and the volume of the digit no longer increases. The rheoplethysmogram in this latter situation illustrates a “plateau” of the inflow volume (Iv), and a rate of inflow (Ir) which reaches zero (fig. 2). It might therefore be expected that there exists a spectrum of venomotor activity between these two extremes.

It should also be mentioned that similar phenomena occur with extremely low degrees of arterial inflow. In this situation the diastolic pressure and, therefore, the AV gradient is low enough to result in essentially no volume change during diastole. Such a curve is observed in a subject with a markedly constricted digital vasculature.6

Volume-Pressure Curves

Twelve normotensive and 13 hypertensive subjects were studied in the fasting state under controlled environmental conditions. The normotensive subjects were free from intercurrent cardiovascular disease. None of the hypertensive patients were in congestive heart failure at the time studied. All digital flows were recorded on the plateau of an alpha wave and the ascent of a beta wave. Digital blood flow was recorded from the right index finger as previously described.8

Progressive cuff pressures (20 to 80 mm. Hg for the normotensive and 20 to 100 mm.
Hg for the hypertensive subjects) were employed. At each pressure rheoplethysmograms were recorded until the inflow volume ($I_V$) reached a plateau. Artifact curves were recorded for each respective pressure. These records were then analyzed for the exact volume inflow ($I_V$) where the "plateau" occurred, and where the rate of inflow ($I_R$) first reached zero. This volume represents the quantity of blood, over and above the resting volume, necessary to cause the pressure within the venous reservoir to rise above the existing occluding cuff pressure, thus resulting in leakage.

Digital small vein pressures were determined by the manner previously described. A volume-pressure curve for each subject was then drawn, the inflow volume ($I_V$) being plotted at which the rate of inflow ($I_R$) first equaled zero against the increase in venous pressure over the resting pressure, i.e., the pressure in the occluding cuff minus the digital small-vein pressure previously determined.

**Results**

The volume-pressure curves and a curve of the mean values for the normotensive subjects are shown in figure 3. Figure 4 illustrates similar curves for the hypertensive patient. The normotensive subjects demonstrated that from 0 to 40 mm. Hg there occurred relatively large volume changes, yielding a typical sigmoid-shaped curve. The hypertensive subjects differed in that there was in general a uniformly rising curve, indicating progressive pressure rises for each volume increment. In addition, the hypertensive curves were all lower on the graph, demonstrating that the veins actually held less blood than in the normal patients.

*Figure 1*

Typical rheoplethysmogram with "relaxed" postcapillary vessels.
Review of Previous Studies

The rheoplethysmograms of the 14 normotensive and 17 hypertensive subjects studied and previously reported were reinvestigated. Each record was analyzed to determine the inflow volume ($I_V$) where the rate of inflow ($I_R$) initially reached zero. In this study only two consecutive pulse cycles were evaluated.

Theoretically, if this volume ($I_V$ where $I_R = 0$) was found to be consistently lower in the hypertensive as compared to the normal subject, this would indicate a tighter venous reservoir.

However, the hypertensive group had been evaluated with a higher occluding cuff pressure (100 mm. Hg) than the normotensive group (60 mm. Hg). If anything, this would tend to minimize the existence of an increase in veno-motor tone, simply because the pressure in the post-capillary vessels would have to exceed 100 mm. Hg instead of only 60 mm. Hg before leakage could occur.

Results

Six of the 28 plethysmograms on the 14 normotensive subjects and 18 of 34 plethysmograms on the 17 hypertensive subjects revealed a rate of inflow ($I_R$) equal to zero during two consecutive pulse cycles. The inflow volumes ($I_V$) where $I_R = 0$ are shown in figure 5. It is apparent that in spite of the higher occluding cuff pressure employed, the hypertensive consistently exhibited a smaller volume inflow at the point where the zero rate of flow occurred; thus indicating a tighter venous reservoir.

The 22 normotensive and 16 hypertensive plethysmograms that did not show a zero rate of flow during the two pulse-cycle sequence were considered to represent a more relaxed venous system.

![Diagram](image)

**Figure 2**

Typical rheoplethysmogram with “tight” postcapillary vessels.
Volume-pressure curves in normotensive subjects.

Volume-pressure curves in hypertensive subjects.

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Gradation of Venomotor Tone

With these differences in mind it then seemed reasonable to grade the degree of venomotor tone existent in the hypertensive group.

In comparing varying degrees of venomotor activity, it is essential to establish a reference volume inflow ($I_v$). For this we selected the mean volume inflow, $25 \text{ mm}^3/\text{5 ml part/sec.}$, shown in figure 4, the plot of the volume-pressure curve for the hypertensive group.

The following criteria were designed to semiquantitate the level of existing venomotor tone during the course of a two pulse-cycle sequence.

Grade III: Those patients whose inflow volume ($I_v$) was $25 \text{ mm}^3/\text{5 ml part/sec.}$ or lower, and whose rate of inflow ($I_R$) equaled zero within two pulse cycles.

Grade II: Those patients whose inflow volume ($I_v$) was $25 \text{ mm}^3/\text{5 ml part/sec.}$ or less, and whose rate of inflow ($I_R$) decreased below $10 \text{ mm}^3/\text{5 ml part/sec.}$ but not to zero within two pulse cycles. We arbitrarily chose $10 \text{ mm}^3/\text{5 ml part/sec.}$ as the point below which one could assume a sharp decrease in the AV gradient, presumably due to a rapidly rising pressure in the venous reservoir.

Grade I: Those patients whose inflow volume ($I_v$) was greater than $25 \text{ mm}^3/\text{5 ml part/sec.}$, irrespective of whether their rate of flow reached zero at larger volumes.

Results

Table 1 illustrates these results. The numbers represent individual experiments (an average of a minimum of 5 flow studies, on a series of normotensive and hypertensive subjects). Columns one and two illustrate that 33.3 per cent of the hypertensive group possessed grades II or III venomotor tone while less than 5 per cent of the normotensive group demonstrated venomotor activity greater than grade I. It must be remembered that the hypertensive subjects were studied at a $40 \text{ mm. Hg}$ greater cuff pressure ($100 \text{ mm. Hg}$) than the normotensive ($60 \text{ mm. Hg}$), thus tending to minimize the degree of existing venomotor tone. Column three illustrates 18 observations on a group of hypertensive subjects using an occluding cuff pressure of $60 \text{ mm. Hg}$. Fifteen of the 18 studies showed grade-III venomotor tone.

Discussion

These observations suggest that the small veins and venules are active participants in the over-all increase in peripheral resistance in the sustained hypertensive state.

The hypertensive volume-pressure curves showing minute changes in volume associated with relatively large pressure increases reflect the presence of both vеноconstriction and venomotor tone in digits of these subjects. On the other hand, the large rises in volume with associated small pressure increments seen in the normotensive curves demonstrate a relaxed digital venous reservoir.

The volume-pressure relationships determined in this study are accurate only if the
resting status of the digital vasculature is similar in the two groups. In other words, if venous engorgement existed, or if more AV shunts were open, or if resting venous pressure were considerably higher in one group than the other, the curves could not be compared. Previous work from our laboratory has shown that such differences do not exist. Essentially the digital vasculature of normal and hypertensive subjects is similar except for the increased peripheral resistances and digital arterial pressures noted in the latter.

The validity of these determinations is further supported by a review of the rheoplethysmograms of the normotensive and hypertensive groups studied with different occluding cuff pressures. In this study the pressure in the venous reservoirs of the hypertensive subjects had to rise 40 mm. Hg higher than the normotensive before leakage past the occluding cuff could occur. In spite of this, significantly smaller volumes of trapped blood resulted in leakage and a rate of inflow equal to zero in the hypertensive group.

It is appreciated that the method of grading venomotor tone as performed in this study is a purely arbitrary one. Nevertheless, it serves to illustrate that hypertensive subjects differ in the amount of tone of their veins, and may serve to distinguish those with high neurogenic tone, if such can be released following nerve block. Such studies are presently in progress.

This work demonstrates that the smooth muscle of the small veins in the sustained hypertensive state is probably, although to a lesser degree, under the same influences as the arterioles.

Summary

By use of the technic of rheoplethysmography, it has been demonstrated that the small veins and venules participate in the over-all increase in peripheral resistance in hypertension.

An arbitrary grading of the degree of venomotor tone in hypertension has been established.

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