The Effects of Short-Term Venous Congestion on Forearm Venous Volume and Reactive Hyperemia Blood Flow in Human Subjects

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SUMMARY  Congestive heart failure is associated with a reduction in limb venous volume at an effective venous pressure of 30 mm Hg (VV[30]). Further, an attenuated arteriolar dilation in response to a metabolic stimulus has been demonstrated. It was the purpose of this study to determine to what extent the chronic elevation in venous pressure seen in heart failure might explain these abnormalities of the limb circulation.

Ten normal human volunteers were subjected to venous congestion of one arm for three hours at 70 mm Hg. A mercury-in-rubber strain gauge plethysmograph was used to measure forearm VV [30] and forearm blood flow at rest after release of five minutes of arterial occlusion (the reactive hyperemia response). Congestion reduced VV [30] 22%, resting forearm blood flow 49% and peak reactive hyperemia blood flow 28%. Thus, chronic venous congestion per se may significantly reduce limb venous volume as well as resting and reactive hyperemia blood flow.

PERIPHERAL EDEMA is a common finding in congestive heart failure. This results from the sodium and water retention and chronic venous congestion seen in this syndrome. Recently, it has been suggested that minimal amounts of edema which might not be clinically apparent could possibly account for most of the reduced limb venous volume and a portion of the limited metabolic arteriolar dilation seen in symptomatic heart failure patients. To test this hypothesis, limb venous congestion for three hours was induced in normal human volunteers and its effect on the forearm venous volume and reactive hyperemia blood flow response was evaluated by a plethysmographic technique. Our studies demonstrated that venous congestion for this brief duration resulted in significant reductions in forearm venous volume as well as resting and reactive hyperemia blood flow.

Methods

Studies were performed on ten normal healthy male subjects between the ages of 21 and 40 years. All studies were performed in a 27° room with the subjects supine in a basal post absorptive state and with both forearms elevated to the level of the anterior chest. Forearm blood flow was measured bilaterally with a mercury-in-rubber strain gauge plethysmograph by the venous occlusion technique as previously described. Circulation to the hand was arrested by a wrist cuff for at least one minute before measurements were made. Following the determination of 8 to 12 blood flow recordings simultaneously in both forearms, the upper arm cuffs were rapidly inflated to 250 mm Hg to institute arterial occlusion for a period of five minutes. At the termination of the ischemic period, blood flow was simultaneously restored rapidly to both forearms and forearm blood flow measured at 5 seconds, 15 seconds, and 15 second intervals for the next two minutes. This procedure was repeated in ten minutes and the values for resting and peak blood flow averaged. Ten minutes later, the venous volume of the forearm at a pressure of 30 mm Hg (VV [30]) was determined by suddenly inflating both upper arm cuffs to 30 mm Hg above cuff zero. Equilibration of venous pressure with cuff pressure was permitted for three minutes at which time the venous volume remained constant. In all instances, it was determined that before venous volume determinations the veins were collapsed and venous pressure in the elevated forearm was reduced to less than 1 mm Hg. This procedure was repeated in ten minutes and the duplicate results averaged.

Following determination of basal blood flow, peak reactive hyperemia blood flow and forearm venous volume, the upper arm cuff of one forearm was inflated to 70 mm Hg for three hours. The arm selected for the three-hour venous congestion was alternated between dominant and nondominant forearm sequentially in the subjects studied. Following the period of congestion, a ten-minute stabilization period was allowed prior to the repeated determination of blood flow and venous volume in a manner identical to that prior to venous congestion.

Venous pressure was measured just prior to release of arterial occlusion and the determination of the reactive hyperemia blood flow response to ensure that it had not changed significantly. In an additional subject, venous congestion was performed similar to the experimental group to determine the amount of edema formation by means of continuous plethysmograph recording. Edema volume was determined by subtracting VV [30] at three minutes from the total change in limb volume.

Statistical comparisons were made using Student’s t-test for paired (values compared before and after congestion in the same arm) and group data (values compared between arms at the same time). Data reported are mean values ± the standard error of the mean.

Results

Venous volume at a venous pressure of 30 mm Hg (VV [30]) prior to the three hour venous congestion period was 4.26 ± 0.35 ml/100 ml in the control arm and 4.62 ± 0.48
1. **FIGURE 1.** Forearm venous volume at a pressure of 30 mm Hg (± SEM) measured simultaneously in two forearms before (pre) and following (post) three hours of venous congestion of one forearm. Values obtained from the forearm subjected to congestion are seen in panel A and those from the control uncongested forearm are in panel B. P values refer to comparisons between adjacent bars.

ml/100 ml following congestion of the contralateral forearm. In the experimental arm VV [30] was 4.60 ± 0.40 ml/100 ml prior to congestion, a value similar to that in the control forearm (fig. 1). Following three hours of venous congestion, VV [30] was significantly reduced to 3.60 ± 0.28 ml/100 ml (P < 0.05), a value which was also significantly less than that measured simultaneously in the control limb (P < 0.05).

Basal forearm blood flow was similar in both forearms prior to congestion and was significantly reduced only in the congested limb following application of the experimental intervention (control limb: 4.6 ± 0.6 to 4.6 ± 0.8 ml/min/100 ml; congested limb: 4.9 ± 0.5 to 2.5 ± 0.3 ml/min/100 ml, P < 0.02 (fig. 2B). The basal flow in the congested limb was also significantly less than that measured simultaneously in the control forearm (P < 0.05).

Peak reactive hyperemia blood flow (PRHBF) following release of five minutes of arterial occlusion was similar in both limbs prior to congestion (fig. 2A). However, following three hours of elevated venous pressure, PRHBF was significantly reduced when compared to the precongestion value (P < 0.01) as well as when it was compared to PRHBF measured simultaneously in the control limb (P < 0.02) (control limb: 28.0 ± 2.7 to 29.7 ± 2.9 ml/min/100 ml; congested limb: 29.8 ± 2.2 to 22.4 ± 1.8 ml/min/100 ml, P < 0.01).

In the subject in whom a continuous plethysmographic recording was performed during venous congestion, edema fluid collected rapidly over the first hour and gradually thereafter. Edema volume at 15, 30, 60, 120 and 180 minutes was 1.34, 2.43, 4.27, 5.72 and 6.53 ml/100 ml, respectively.

**Discussion**

An elevated venous tone at rest and during exercise has been noted in heart failure patients for many years. Although it has been thought that this resulted from an enhanced activation of the sympathetic nervous system, it was recently demonstrated that local factors were the major determinant of the reduced venous volume seen in heart failure. In fact, one preliminary report has suggested that neurogenic venous tone in heart failure may even be normal.
at rest. In our studies, it was demonstrated that venous congestion at 70 mm Hg for only three hours could result in a 22% reduction in venous volume (fig. 1). Following this brief period of congestion, venous volume was 3.6 ml/100 ml, a value still nearly twice that observed in heart failure subjects. Therefore, short-term venous congestion cannot completely explain most of the reduced venous volume seen in heart failure. Further studies are indicated to determine what other factors may be involved.

The cause of the reduced venous volume produced by short term venous congestion was most likely edema fluid accumulation since limb volume increased approximately 6.5%. However, it is possible that the three hour period of stretch of venous smooth muscle (the Bayliss phenomenon) may be partially responsible. This is a less likely explanation since the venous pressure was allowed to return to control levels after the three hour congestion period and prior to repeat determination of venous volume.

A second observation was that three hours of venous congestion could reduce forearm blood flow at rest. Congestion also reduced blood flow when it was augmented by the metabolic arteriolar dilator stimulus of the reactive hyperemia response (fig. 2). These findings confirm in humans similar observations in a canine preparation. The reduction in PRHBF in the human studies was 25%, a value similar to the animal studies, where it was reduced by 33%. The greater reduction seen on the animal studies may be explained by the slightly longer congestion period to which the animals were subjected (four hours). Importantly, the animal studies did answer questions that could not be easily answered in humans. For example, it was determined that venous congestion resulted in an increase in interstitial pressure to positive values and that it did not alter arterial sodium content. In the animal studies it was suggested that the slight loss in limb temperature during congestion could not account for the reduction in metabolically-augmented limb blood flow. Rather, it was suggested that some other consequence of the congestion, such as the interstitial fluid accumulation, was the cause of this reduction.

It is well known that when venous congestion is maintained up to the point when arterial occlusion is instituted, the subsequent reactive hyperemia response is reduced. This occurs because the pressure drop on the arterial side of the circulation due to run-off of blood into the capacitance vessels is diminished when venous pressure is initially quite high. This observation has led to the hypothesis that the reactive hyperemia response is secondary to two factors, accumulation of metabolites and diminished stretch of vascular smooth muscle. Therefore, care was taken to insure that venous pressure in the limb had returned to basal values before the reactive hyperemia response was determined.

It appears that in normal human subjects short-term venous congestion and subsequent edema fluid accumulation can result in qualitatively similar changes in limb vascular dynamics as are shown in heart failure patients. These reductions in forearm venous volume, resting, and metabolically augmented blood flow, though qualitatively similar, are quantitatively less.

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