Peripheral Venoconstriction in Human Congestive Heart Failure

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A method for measuring venous "tone" in the limb segment of man in vivo was used to study the problem of cardiac decompensation. Venoconstriction was present in patients with heart failure and it tended to remit as the patients improved.

VENOCONSTRIC TION has been suggested as an explanation for a variety of observations made on animals and humans suffering from cardiac decompensation.1-4 We have used the method described by Litter and coworkers for measuring peripheral venous distensibility6 to study this aspect of congestive heart failure in man. The method measures the increment of limb volume occurring in response to a known increment in local effective venous pressure. The experimental conditions were such that the starting point for each determination was a low, constant local effective venous pressure which had been rendered independent of central venous pressure. This characteristic of the method was important in that it allowed comparison of data obtained from different patients with widely divergent central venous pressures.

The observations here reported indicate that the peripheral veins of patients with congestive heart failure are constricted as compared with those of patients who have no cardiovascular disease and that this venoconstriction tends to remit with recompensation.

METHODS

All patients were studied in the sitting position since orthopnea prevented some from lying supine. The forearm rather than the leg was studied as the edema of the lower extremities of some of the patients might have affected the results.

The subject was seated in a comfortable chair with his forearm inserted into a water-filled, variable-depth, limb-segment plethysmograph which was 16.5 cm. long. A 5 cm. wide arterial occluding cuff around the wrist was inflated to suprasystolic pressure just prior to each determination. Volume changes of the limb segment were recorded on a kymograph by a heated stylus attached to a Brodie bellows. The bellows was calibrated with known volumes of air introduced into the system. The patient's hand was supported by sand bags and the partially flexed elbow was supported by a rigid, padded block with two of its sides enclosed to prevent motion of the elbow. The forearm was placed at the level of the shoulder. A venous occluding cuff 12.5 cm. wide was placed around the upper arm. It was held firmly in place by adhesive tape and gauze. Great care was taken to prevent the cuff from exerting pressure on the arm in the uninflated state. Pressures in this cuff were controlled with a water manometer. Venous pressure was measured in the opposite arm, which was at the same level as the plethysmographic arm, either just prior to, or throughout, each experiment. Venous pressure related to the level of the plethysmographic arm is referred to as the natural local venous pressure. The plethysmographic water level was at least 5 cm. higher than the level of natural local venous pressure. Whenever possible, venous pressure was recorded in the supine position and related to the right atrium by the method of Lyons and associates.7 When the patient was unable to assume a supine position, venous pressure was related to the right atrium by the method of Winsor and Burch.8 Pressures related to the right atrium are referred to as the venous pressure. When this pressure exceeded 16 cm. of saline it was considered to be elevated.

The experimental procedure for obtaining peripheral venous distensibility was as follows: The plethysmographic water level was adjusted as noted above. Since external pressure around the forearm then exceeded the natural local venous pressure, the arterial inflow forced the local venous pressure to a level high enough to exceed slightly the applied external water pressure. The effective pressure (internal pressure minus external pressure) within the veins and their contained volume had then reached a low, constant and reproducible value. This pressure will be referred to as the effective local venous pressure. When measured directly, this value was found to be less than 1 mm. Hg. An equivalent starting point for each determination was then assured. The venous congesting cuff on the upper arm

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was inflated at 1 mm. Hg increments until the first small increase in forearm volume occurred. This cuff pressure (whatever it happened to be as related to atmospheric pressure) was called cuff zero pressure. Cuff pressure was then raised from cuff zero pressure by 30 mm. Hg pressure in 5 mm. Hg increments. Each of these pressure increments resulted in a volume increment of the forearm and as soon as a new forearm volume was reached the next 5 mm. Hg increment of pressure was introduced into the cuff. The total of the volume increments of the forearm segment venous system in response to the increase of 30 mm. Hg effective local venous pressure was thus measured, starting at an effective venous pressure of less than 1 mm. Hg. The volume of the forearm segment contained within the plethysmograph was obtained by subtracting the capacity of the plethysmograph with the forearm in place from its previously determined total capacity. The total forearm venous volume increase was converted to cubic centimeters per 100 cc. of tissue and is referred to as venous distensibility. A small distensibility was interpreted as representing relative venoconstriction and a large distensibility represented relative venodilatation.

**Subjects**

Twenty hospitalized patients with signs and/or symptoms of congestive heart failure during or prior to testing were studied on 33 occasions. The types of heart disease which caused the cardiac decompensation were distributed as follows: five hypertensive, three arteriosclerotic, four rheumatic, one syphilitic, five chronic pulmonary artery hypertensive, and two degenerative. The results of this study were essentially the same in all the types of heart disease studied. In 20 experiments the patients had elevated venous pressures while in 13 experiments the patients had normal venous pressures. Six of these 13 had some clinical evidence of cardiac failure while the rest appeared free of failure on clinical examination at the time of the experiment. In general, the patients with heart failure in the normal venous pressure group were not as severely incapacitated as were those in the elevated venous pressure group.

Another group of 10 hospitalized patients with no cardiovascular disease but of roughly comparable age and sex distribution was studied. These patients were considered to be normal controls, though they did suffer from a variety of diseases. The results obtained in this group were similar to those obtained in other studies carried out on the lower limbs of supine normal young male subjects.6

**Results**

The peripheral venous distensibility in patients with heart failure who had elevated venous pressures averaged 2.9 cc. per 100 cc. of forearm with a standard deviation (S.D.) of ±0.6 cc. Venous pressures in this group ranged from 17.0 to 38.0 cm. of saline. There was no apparent correlation between the level of the venous pressure and the degree of venoconstriction. The values obtained in those patients who had definite clinical evidence of decompensation, but normal venous pressures averaged 3.2 cc. per 100 cc. of forearm with a S.D. of ±0.6 cc. Patients with heart disease, but no evidence of failure at the time of the study, averaged 4.4 cc. with a S.D. of ±0.9 cc. Finally, the patients who had no cardiovascular disease had an average value of 4.4 cc. per 100 cc. forearm with a S.D. of ±0.4 cc., (fig. 1).

Four patients from the group with heart failure and elevated venous pressure were
studied again after recompensation and their venous distensibility rose from 2.3 cc. to 3.4 cc., 2.1 cc. to 4.4 cc., 3.6 cc. to 4.8 cc., 3.2 cc. to 4.3 cc. per 100 cc. of forearm, respectively. One patient with elevated venous pressure was improved by therapy and venous distensibility rose from 2.4 to 3.0 cc. per 100 cc. of forearm (fig. 2).

**DISCUSSION**

These studies indicate that the peripheral venous systems of patients with congestive heart failure are less distensible than those of patients without cardiovascular disease. However, there was no apparent relationship between the degree of venoconstriction and the magnitude of venous pressure elevation in the congestive heart failure patients. Indeed, patients with other signs of cardiac decompensation, but normal venous pressure frequently though not invariably showed decreases in distensibility of the same order of magnitude as those observed in patients with elevated venous pressures. This decreased distensibility or venoconstriction tended to remit as the patient improved and in fact it often returned to the range of control values after successful therapy. Thus the status of the peripheral veins could be roughly correlated with the overall clinical status of an individual patient’s decompensation.

The evidence presented establishes a firm basis for the belief that peripheral venoconstriction occurs in patients with congestive heart failure. However, whether or not this peripheral venoconstriction is also generalized cannot be determined by this method. One can state that at a given venous pressure within the range of the pressures utilized for this study (0 to 30 mm. Hg) the contained volume of the peripheral vein system in congestive heart failure patients was less than that in normal subjects. These studies do not suggest a causative mechanism for the observed venoconstriction, nor do they clarify its relationship to the numerous other physiologic responses to the failing heart.

**SUMMARY AND CONCLUSIONS**

A method for the measurement of venous distensibility which effectively isolates the peripheral venous system from the influences of central venous pressure changes has been applied to the problem of human congestive heart failure.

Two groups of patients with heart failure were studied. One group was composed of patients who had elevated venous pressures and the second consisted of patients with normal venous pressures. Some of the patients were restudied after improvement occurred in the congestive state. A group of patients who had heart disease but were not in failure at the time of testing were studied.

A control group composed of hospitalized patients who had no cardiovascular disease was also studied.

The patients with heart failure had peripheral venoconstriction as compared with the control patients. This decreased venous distensibility returned toward or to normal as compensation of the heart failure occurred. Venoconstriction was observed in heart failure patients who had normal peripheral resting venous pressures as well as in those with elevated peripheral venous pressures.

**Summario in Interlingua**

Un metodo pro le mesuration del distensibilitate venose, que isola efficacemente le peripheric systema venose ab le influentia del variationes in le pression venose central, esseva applicate al problema del congestive disfallimento cardiac in humanos.

Esseva studiate duo gruppus de patientes con disfallimento cardiac. Un del gruppus consisteva de patientes con elevate pressiones venose. Le patientes del secunde gruppo hava normal pressiones venose. Alicunes del patientes esseva restudiate post que illes hava attingite un melioration de lor stato congestive. Esseva studiate un gruppo de patientes con morbo cardiac, non in stato de disfallimento al tempore del tests. Etiam un gruppo de controlo esseva studiate, consistente de patientes hospitalisate qui non hava morbo cardiovascular.

Le patientes con disfallimento cardiac hava venoconstriction peripheric in comparation con le patientes de controlo. Iste reducite distensibilitate venose retornava verso o usque a
valores normal in tanto que compensation del disfallimento cardiac esseva effectuate. Veno-constriction esseva observate in patientes de disfallimento cardiac qui habeva normal peripheric pressiones venose in reposo etiam in patientes qui habeva elevate peripheric pressiones venose.

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