The vascular distensibility of the hand was measured in normal subjects before, during and after reduction of the effective blood volume. Evidence is presented which indicates that the veins become less distensible when the effective blood volume is reduced and it is suggested that this mechanism mobilizes blood normally contained in the "vascular deadspace" of the veins.

**Method**

The vascular distensibility of the hand was measured by comparing the increase in hand vascular volume resulting from intermittent obstruction of the venous return with the associated increase in hand venous distending pressure. This technique has previously been described in detail (Watson, 1961).

**Expression of results.**

The increase in hand volume during venous obstruction was expressed graphically against the simultaneous increase in vascular distending pressure: these parameters were measured at intervals of 0.2 to 0.5 sec throughout the period of venous obstruction. A curve was obtained representing the vascular volume-pressure relationship of the hand during each period of venous obstruction. The gradient of this curve represents the change in hand volume resulting from change in vascular distending pressure and was therefore equal to the hand vascular distensibility. As the vascular volume-pressure relationship of the hand was commonly curvilinear (fig. 1) it was necessary to measure the gradient of the resulting line at a constant hand volume so that comparable values could be obtained under different circumstances. In all the results presented below, vascular distensibility was measured at a constant hand volume 1.5 ml above the resting volume found at the beginning of the investigation. The values for vascular distensibility shown at intervals of 1 minute in figures 2 and 4 represent mean values obtained by analysis of the two or three episodes of venous obstruction occurring in the previous minute.

*End-tidal Pco₂* was derived from the record of an infra-red carbon dioxide analyzer (Ird-o-meter) sampling continuously at 60 to 120 ml/min from the external nares or mouth. The infra-red analyzer had previously been calibrated with various mixtures of carbon dioxide in air, the precise proportions of which had been determined with a Haldane apparatus. The calibration curve obtained conformed with that provided by the manufacturer.

**Arterial blood pressure** was measured with a sphygmomanometer: the pneumatic cuff was placed around the opposite upper arm and the systolic and diastolic blood pressures determined by auscultation.

The effective blood volume was reduced by pooling blood in the legs: this was achieved by inflating pneumatic cuffs placed around the thigh to a pressure which did not exceed the diastolic arterial pressure.

**Procedure.**

The subject sat in a comfortable chair in a quiet laboratory. His legs were dependent and a venous occluding cuff was placed around each thigh. After a period of 30 minutes an estimation was made of the vascular distensibility of the hand of the relaxed normal subject by analysis of twenty consecutive episodes of venous occlusion imposed within a period of 10 minutes. The cuffs on the
legs were then inflated to 60–80 mm Hg; this pressure was increased to the diastolic blood pressure over a period of 5 to 10 minutes. The cuffs remained inflated for 10 to 15 minutes and were then suddenly deflated. After deflation of the leg cuffs the legs were elevated. Throughout the procedure the venous return from the hand was occluded two or three times each minute so that frequent estimation of hand venous distensibility was made.

In one subject, after a baseline value for hand vascular distensibility had been obtained, 1 pint of blood was removed from the antecubital vein of the opposite arm before the leg cuffs were inflated. This subject fainted after the leg cuffs were inflated. Measurement of the venous distensibility of the hand was made in this subject during the removal of the blood and during the period in which the leg cuffs were inflated both before and during the faint.

Vascular volume-pressure relationship of the hand obtained in three normal subjects (A, B, C).
- ● before the leg cuffs were inflated;
- X after the leg cuffs had been inflated for 7 minutes;
- ▲ 10 minutes after the leg cuffs had been released.
Subjects.
The investigation was performed on four normal male subjects aged 27 to 44 years. None had clinical evidence of disease of the respiratory or circulatory systems.

RESULTS
Figure 1A, B, C shows the hand vascular volume-pressure relationship of the three subjects who did not faint when the effective blood volume was reduced. In all these subjects a reduction in baseline hand volume occurred after inflation of the leg cuffs. This reduction was significant (P<0.05) in two subjects (figs. 1A and 1C) and insignificant in one subject (fig. 1B). The curve representing the vascular volume-pressure relationship of the hand fell towards the pressure axis after inflation of the leg cuffs indicating that the vascular distensibility had decreased. This decrease in vascular distensibility, measured as the decrease in the gradient of the vascular volume-pressure relationship, was highly significant (P<0.01) in all subjects. After the pressure in the occluding cuffs on the thighs was released the venous distensibility increased but in only one subject did it attain its previous level. No marked change occurred in the arterial blood pressure of any of these three patients. The end-tidal PCO₂ remained constant (± 2 mm Hg).

Figure 2A, B, C expresses the vascular distensibility of the hand of these three patients against time. The signal marks the period for which the leg cuffs were inflated. In all subjects the vascular distensibility of the hand decreased during the period of simulated oligaemia. In one subject (fig. 2A) a transient decrease was observed in vascular distensibility immediately after the leg cuffs were inflated. This was possibly a consequence of apprehension. When the pressure in the leg cuffs was released a rapid increase in vascular distensibility occurred but the first resting value was not attained.

Figure 3 shows the vascular volume-pressure relationship of the subject who fainted. The vascular distensibility was significantly decreased (P<0.05) by the removal of 1 pint of blood. A further highly significant decrease in vascular distensibility (P<0.01) was found when the leg cuffs were inflated. When the subject fainted no significant change of the reduced distensibility was found. Figure 4 expresses the values obtained for vascular distensibility and the arterial blood pressure against the time in minutes. The signals show the periods in which blood was withdrawn, in which the leg cuffs were inflated, and in which the subject fainted. During reduction of effective blood volume a progressive decrease in the vascular distensibility of the hand was found: in this time there was no marked change in the arterial blood pressure. Loss of consciousness was associated with dramatic fall of the arterial blood pressure but with no change in the vascular distensibility of the hand: during the period of low arterial blood pressure, the pressure in the pneumatic cuff occluding the venous return from the hand was reduced to 25–30 mm Hg to avoid impeding the arterial blood flow to the hand. The end-tidal PCO₂ fell from 42 mm Hg to 34 mm Hg during the 3 minutes preceding loss of consciousness.

The patient recovered consciousness when his legs were raised and the pint of blood was rapidly retransfused. The arterial blood pressure rose, the vascular distensibility increased and the end-tidal PCO₂ rose to 39 mm Hg.

DISCUSSION
The technique used in this investigation is similar to that employed by Sharpey-Schafer (1961) for determining the distensibility of the capacity vessels of the forearm. It is related to the plethysmographic methods of determining “venous” distensibility in vivo (Clark, 1933; Capps, 1936; Kidd and Lyons, 1958; Glover, Greenfield, Kidd and Whelan, 1958; Eckstein and Horsley, 1960). The vessels investigated are the capacity vessels which consist largely of veins (Litter and Wood, 1954). As the hydrostatic pressure due to the column of water within the plethysmograph above the back of the hand was transmitted directly both to hand tissues and to veins, the venous distending pressure was near zero between episodes of venous tamponade, indicating that the veins, or low pressure capacity vessels were collapsed (Ryder, Molle, and Ferris, 1944), so that a reasonably constant baseline hand venous volume was assured (Clark, 1933).

The effective blood volume was reduced by trapping blood in the legs. The volume of blood trapped was not measured but has been esti-
Change in the vascular distensibility of the hand of three normal subjects (A, B, C) expressed against time. The subjects are the same as in fig. 1A, B, C respectively. Fig. 2A shows a transient decrease of vascular distensibility after the leg cuffs were inflated and is probably due to apprehension. The signal indicates the period for which the leg cuffs were inflated.
Volume-pressure relationship of the hand obtained in the subject who fainted.

- before the leg cuffs were inflated;
- after 1 pint of blood had been removed in 5 minutes;
- 5 minutes after inflation of the leg cuffs;
- while unconscious 6 minutes after inflation of the leg cuffs.

Change in the vascular distensibility of the hand and arterial blood pressure of the subject who fainted, expressed against time. No change occurred in arterial blood pressure until just before the subject fainted, although progressive reduction of vascular distensibility occurred in this time. When the subject fainted the arterial blood pressure fell suddenly, but no change was found in vascular distensibility.
VASCULAR DISTENSIBILITY OF THE HAND

mated as 500 to 600 ml (Ebert and Stead, 1940; Sharpey-Schafer, 1951). Arteriolar constriction in skin and muscle normally occurs when the effective blood volume is reduced, and maintains the arterial blood pressure despite a reduced cardiac output (Barcroft and Edholm, 1945). Our results suggest that these changes are associated with alteration in the distensibility of the capacity vessels of the hand. If the changes observed in the hand are typical of the systemic capacity vessels as a whole, reduction of distensibility will result in mobilization of blood from the "vascular deadspace" of the systemic capacity vessels, which may contain 60 per cent of the total blood volume (Landis and Hortenstine, 1950). This would allow an adequate central venous pressure to be maintained with a smaller total blood volume.

When fainting occurs during reduction of the effective blood volume no further fall is found in the cardiac output, the blood flow through muscles increases markedly and the arterial blood pressure falls (Sharpey-Schafer, 1951). The observations presented here suggest that no change in the distensibility of the capacity vessels of the hand occurs under these circumstances and that syncope is not a consequence of sudden increase in the volume of blood contained in the systemic veins. As such an increase would result in a fall of venous return and hence of cardiac output, the findings are compatible with the observation that little change in cardiac output occurs during oligaemic syncope.

SUMMARY

The distensibility of the capacity vessels of the hand was measured in three normal subjects when the effective blood volume was reduced by 500 to 600 ml.

The capacity vessels of the hand became less distensible during reduction of blood volume.

The distensibility of the capacity vessels of the hand was measured in one normal subject who fainted when the effective blood volume was reduced by about 1000 ml. The capacity vessels of the hand remained relatively indistensible throughout the period of unconsciousness.

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REFERENCES


