VASCULAR DISTENSIBILITY OF THE HAND DURING PRESSURE BREATHING

BY

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The distensibility of a system is the change in volume which results from unit change in distending pressure when sufficient time is allowed for the system to become stable. The vascular distensibility of the hand is therefore the change in hand blood volume which results from unit change in the pressure distending the vessels of the hand.

METHODS

In this investigation the vessels of the hand were intermittently distended by inflating a pneumatic cuff around the wrist so that venous return from the hand was obstructed. The rate of increase of hand volume was initially equal to the hand blood flow but decreased as the venous pressure approached the pressure within the pneumatic cuff as some blood then leaked proximally.

**Hand blood volume** changes were measured as hand volume changes with a water-filled, stirred plethysmograph of 5.16 litres capacity maintained at a temperature of 34 ± 0.5°C. The water within the plethysmograph exerted a hydrostatic pressure of 8 to 11 cm H₂O upon the back of the hand, which was at the level of the subject's manubrium sterni. Hand volume changes were derived from pressure changes in the air contained in the turret of the plethysmograph. Ambient temperature was within the range 22 to 25°C and constant throughout each investigation.

**Hand venous pressure** was measured through a nylon catheter of 0.8 mm internal diameter introduced percutaneously on the dorsal or radial aspect of the radiocarpal joint and directed peripherally, negotiating at least one venous valve until the end of the catheter lay about 5 cm distal to the site at which the vein was punctured. Care was taken that the end of the catheter was not wedged within a venous tributary or within the sinus of a venous valve. The catheter was periodically flushed with 0.2 ml of saline containing 0.01 mg/ml of heparin.

The vascular distending pressure, or *transmural venous pressure* of the hand is the difference between the pressure within the vein and the pressure within the loose extravenous connective tissue of the back of the hand.

Between episodes of venous obstruction:

\[ P_D = P_V - P_T \]

where \( P_D \) = vascular distending pressure;
\( P_V \) = measured venous pressure;
\( P_T \) = pressure in the extravenous connective tissue.

The pressure within the loose connective tissue and within the hand vein was always equal to the hydrostatic pressure of 8 to 11 cm H₂O imposed by the column of water within the plethysmograph above the back of the hand (Watson, unpublished data). This indicates that the imposed hydrostatic pressure was transmitted directly both to the connective tissue and to the veins of the hand. Between episodes of hand venous tamponade the vascular distending pressure was therefore zero, or near zero, indicating that the veins were collapsed (Ryder, Molle and Ferris, 1944).

During episodes of venous obstruction the hand volume increased and the pressure within the plethysmograph rose. This increase in pressure within the plethysmograph was transmitted directly to the loose extravenous connective tissues of the back of the hand (Watson, unpublished data). The increase in venous distending pressure at a given instant during an episode of venous obstruction was therefore derived from the measured increase in venous pressure by sub-

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tracting the measured increase in pressure within the plethysmograph.

$$P_D = P_v - P_T$$

where $P_D$ = increase in vascular distending pressure at a given instant during venous obstruction;

$P_v$ = measured increase in venous pressure at a given instant during venous obstruction;

$P_T$ = simultaneous measured increase in pressure within the plethysmograph.

An occluding pneumatic cuff was placed round the wrist immediately proximal to the plethysmograph and inflated to 40 mm Hg for 10 to 20 seconds: the cuff remained deflated for at least 10 seconds between consecutive inflations.

All pressures were measured with capacitance transducers (Southern Instruments) and recorded with a four-channel direct writing pen unit. The pens were 16 cm long, working in an arc of 4 cm and had a peak-to-peak response time of 0.07 sec. The paper speed usually employed was 5 mm/sec, which allowed hand volume and hand venous pressure to be compared at intervals of 0.2 sec during an episode of venous obstruction.

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 through the period of venous obstruction. A curve was obtained representing the vascular volume-pressure relationship of the hand during each period of venous occlusion. The gradient of this curve represented change of hand volume resulting from change in vascular distending pressure and was therefore equal to the hand vascular distensibility.

Each volume-pressure relationship presented in this paper represents mean values derived from analysis of twenty consecutive episodes of venous tamponade made under constant conditions, unless otherwise stated.

The vessels investigated by this technique are those which could be distended by the imposed vascular distending pressure: they are therefore those vessels which normally contain blood at a pressure less than the highest vascular distending pressure imposed. As the highest vascular distending pressure was about 30 mm Hg this technique investigates those vessels in which the blood pressure is usually low. As the capacity of these vessels is large they are called the low pressure capacity vessels, and consist of veins, venules and capillaries. The volume of the veins and venules investigated considerably exceeds that of the capillaries (Litter and Wood, 1954).

Oesophageal pressure was used as an index of intrapleural pressure and was measured by the method of Dornhorst and Leathart (1952). The oesophageal catheter was passed pernasally and its oesophageal end adjusted until records of intrathoracic respiratory pressure changes were obtained with minimum cardiac pulsation. The mean oesophageal pressure was obtained from the oesophageal pressure record by determining the area beneath the trace and calculating the height of the rectangle having similar length (duration) and height (pressure).

End-tidal $P_{CO_2}$ 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 end of the patient's tracheostomy tube, or from the mouthpiece of the subject.

The totally paralyzed patients received intermittent positive pressure respiration from an experimental respiration pump (Watson, Spalding and Smith, 1962). The mean intrathoracic pressure was raised by preventing the tracheal pressure from returning to atmospheric pressure during expiration. This was achieved by immersing the outlet of the respiration pump to a known depth in a column of water. The imposed inspiratory tracheal pressure was raised to maintain a constant alveolar ventilation, so that the end-tidal $P_{CO_2}$ remained constant. Throughout the investigation the respiratory rate was 13/min.

Continuous positive pressure breathing was imposed upon normal subjects by causing them to breathe through a Siebe-Gorman valve placed in an air flow line which was continuously flushed at 50 l./min with air. The pressure within the air flow line was varied by immersing the outlet to a variable depth in a column of water.

Subjects.

Six conscious patients with respiratory paralysis or severe weakness of the muscles of respiration were investigated. Each received IPPR
through a cuffed tracheostomy tube (Spalding and Smith, 1956) which provided an airtight seal in the trachea. Two patients suffering from chronic poliomyelitis had no clinical evidence of disturbance of the control of the circulation and had a normal response of the arterial blood pressure to Valsalva’s manoeuvre. Two patients had severe polyneuritis and two patients had complete traumatic lesions of the spinal cord in the cervical region: these four patients had a grossly abnormal response of the arterial blood pressure to the Valsalva manoeuvre. Six normal subjects aged 27 to 39 years were also investigated. All subjects and patients lay down throughout the investigation.

Experimental procedure.

After the apparatus had been set up, no measurement of capacity vessel distensibility was made for about 30 minutes. Twenty consecutive records were then analyzed. After this the mean intrathoracic pressure was raised to the desired level and twenty consecutive records again analyzed. The intrathoracic pressure was then restored to its initial level and further measurement made. This procedure was repeated with different values for mean intrathoracic pressure. Care was taken to keep the end-tidal Pco₂ constant in each patient (±2 mm Hg) throughout these investigations.

All values given here represent the mean values obtained from twenty consecutive episodes of venous tamponade when a steady state had become established. The time taken for the observed changes in distensibility to occur is also described.

RESULTS

Elevation of the mean intrathoracic pressure to 5 cm H₂O caused no change in the vascular volume-pressure relationship of the hand in four of the six normal subjects. Further elevation of the mean intrathoracic pressure caused the capacity vessels of the hand to become less distensible. The results shown in figure 1A were derived from one of these four subjects, and are typical. Elevation of the mean intrathoracic pressure to 5 cm H₂O was associated with a decrease in the distensibility of the capacity vessels of the hand in the two remaining normal subjects. This decrease became more marked when the intrathoracic pressure was further increased. The results shown in figure 1B were derived from one of these two patients. The reduction in distensibility was highly significant (P<0.01) on all occasions. Similar significant results were obtained in the two totally paralyzed patients with normal nervous control of the circulation receiving IPPR (figs. 2A, 2B). The distensibility of the capacity vessels began to decrease about 10 seconds after the intrathoracic pressure was increased and had usually attained

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![Figure 1A](https://academic.oup.com/bja/article-abstract/33/12/600/245414/001.png)

![Figure 1B](https://academic.oup.com/bja/article-abstract/33/12/600/245414/002.png)

Examples of mean vascular volume-pressure relationship of the hand in two normal subjects. Each curve was derived from analysis of twenty consecutive periods of venous obstruction, each lasting about 10 seconds.
Examples of mean vascular volume-pressure relationship of the hand in two subjects with respiratory paralysis but with normal nervous control of the circulation. Each curve was derived from analysis of twenty consecutive periods of venous obstruction, each lasting about 10 seconds.

Examples of mean vascular volume-pressure relationship of the hand in two subjects with severe polyneuritis.

Examples of mean vascular volume-pressure relationship of the hand in two subjects with complete traumatic lesion of the cervical part of the spinal cord.
a steady state within 2 minutes. The increase in distensibility of the capacity vessels found when the intrathoracic pressure returned to normal took a similar time.

No change occurred in the distensibility of the capacity vessels of the hand either in the two patients with acute polyneuritis (figs. 3A, 3B) or in the two with complete traumatic lesions of the spinal cord in the cervical region (figs. 4A, 4B), when the mean intrathoracic pressure was raised. These patients were less tolerant of elevation of mean intrathoracic pressure, complaining of faintness with values of intrathoracic pressure less than those tolerated by patients with normal nervous control of the circulation.

**DISCUSSION**

Blair, Glover and Kidd (1959) found no alteration of the distensibility of the forearm capacity vessels during continuous positive pressure breathing. Ernsting (1957) presented evidence of reduction of venous distensibility of the hand during positive pressure breathing with airway pressures considerably in excess of those investigated here. Neither investigator measured intrathoracic pressure.

In this investigation mean oesophageal pressure is used as representing mean intrapleural pressure. The conclusion of most recent investigators is that the mean oesophageal pressure is 1 to 2 cm H$_2$O higher than the mean intrapleural pressure in subjects lying supine (Mead and Gaensler, 1959; Knowles, Hong and Rahn, 1959).

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; Kidd and Lyons, 1958; Glover et al., 1958; Eckstein and Horsley, 1960). The vessels investigated are the low pressure capacity vessels which consist almost entirely of veins (Litter and Wood, 1954). The veins were collapsed by the hydrostatic pressure imposed by the water within the plethysmograph, so that a reasonably constant baseline hand venous volume was assured (Clark, 1933).

During intermittent positive pressure respiration the mean intrathoracic pressure is raised (Opie, Spalding and Smith, 1961). Elevation of the mean intrathoracic pressure imposes a stress upon the circulation by impeding venous return to the right side of the heart (Lauson, Bloomfield and Cournand, 1946; Brecher and Mixter, 1953). The volume of blood displaced into the “vascular deadspace” of the veins by a given rise in venous pressure depends upon the distensibility of the systemic veins. Under normal conditions it is possible that 50–60 per cent of the total blood volume is within the low pressure capacity vessels (Landis and Hortenstine, 1950). If the observations made upon the hand are representative of the systemic muscular veins, the volume of blood pooled in the veins as a result of elevation of intrathoracic pressure during pressure breathing is reduced by the decreased venous distensibility. Patients receiving IPPR who have abnormal nervous control of the circulation do not modify the distensibility of the capacity vessels of the hand in this way. It is therefore probable that the reduction in distensibility is under nervous control and is possibly due to an increase in venomotor tone.

When the mean intrathoracic pressure of conscious patients receiving IPPR is raised, the transmural central venous pressure remains almost unchanged until the mean oesophageal pressure reaches 4 to 6 cm H$_2$O (Watson, Spalding and Smith, 1962), provided that the nervous control of the circulation is normal. Patients without normal nervous control of the circulation suffer progressive fall in transmural central venous pressure as the mean intrathoracic pressure is raised. It is probable that in patients with normal nervous control of the circulation one of the factors maintaining a constant transmural central venous pressure when intrathoracic pressure is raised is the reduction of systemic venous distensibility.

**SUMMARY**

The distensibility of the capacity blood vessels of the hand was measured in six normal subjects and in six patients with respiratory paralysis during pressure breathing.

In subjects and patients with normal nervous control of the circulation, pressure breathing was associated with a reduction in distensibility of the capacity blood vessels of the hand.

In patients with demonstrably abnormal
nervous control of the circulation no change was found in the distensibility of the capacity blood vessels of the hand during pressure breathing.

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EDITORIAL ANNOUNCEMENT
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