DETERMINATION OF THE VENOUS PRESSURE IN THE SITTING POSTURE

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The determination of the venous pressure (VP) is an accepted diagnostic aid in studying heart failure and conditions involving obstruction of the venous system. However, the classical methods of determining the VP directly (Moritz and Tabora, 1910; Lyons *et al.*, 1938, and others) require the patient to be in the supine position and cannot be applied to those with orthopnoea. Hence there is need for a method that can be used in the semi-sitting posture, which is convenient for all patients, including those unable to lie flat. This method, of course, has to give VP values equal or proportional to the standard values obtained in the supine position.

The methods that have thus far been proposed have failed to demonstrate a constant relationship between VP values in the sitting and supine postures for all types of patients.

Winsor and Burch (1946) described, for example, a method of measuring the VP in which the patient sits with his trunk at a 45-degree angle to the horizontal, but with his arm raised to the "phlebostatic level," i.e. almost horizontal. According to these authors the VP in this position is equal to that obtained in the supine in normal subjects. However, they found lower VP values in the sitting posture than in the supine in patients with severe heart failure. On the other hand Davis and Shock (1949), using the same method, found higher VP values in the sitting posture than in the supine in normal subjects, while in patients with right heart failure (high VP) they found equal values in both positions.

These contradictions are not surprising in view of the many variable factors that influence the VP in the sitting posture. These causes may raise or lower the VP depending on the condition of the patient. A few examples illustrating this variability follow.

(1) In the sitting position the hydrostatic pressure of the blood tends to distend the blood vessels in the lower parts of the body and force blood into them, and thus to lower the antecubital VP. The blood accumulates mainly in the small veins (Turner *et al.*, 1937). Constriction of the arterioles that control the blood flow to the veins counteracts this pooling of blood. It is therefore obvious that lowering of the VP due to this cause should be less pronounced in subjects with narrowed arterioles or rigid venules, as in patients with hypertension and sclerosis of small arteries or veins.

(2) An additional cause for the fall of the VP in the sitting posture is the increased filtration of plasma fluid through the capillaries in the lower parts of the body, resulting in a decrease in blood volume. The permeability of capillaries varies in different types of patients. It is increased in those with severe heart failure and high VP (Landis and Hortenstine, 1950). Therefore this type of patient should have a greater tendency to lowering of the VP on assuming the sitting posture than patients with normal capillary permeability.

(3) Compensatory mechanisms designed to stabilize pressure in the circulatory system do not function to the same degree in all patients. It is known, for example, that in patients with right-sided heart failure the mechanisms that keep the VP constant do not function properly. In these patients phlebotomy lowers the VP and intravenous infusion raises it, while in normal subjects these procedures do not alter it (Caughey, 1935; Richards *et al.*, 1937).

(4) Our observations in 16 patients (unpublished data) showed that in the sitting position, raising the arm to the horizontal causes almost always a rise in the antecubital VP, which rise, however, varies in extent from subject to subject, and may be as much as 5 cm. saline.

It follows, therefore, that the combination of these variable influences can give in different subjects varying results, so that in one the VP may rise on assuming the sitting posture, in another it may fall, while in a third there may be no change at all. This explains why no constant relationship between VP values in the two positions can be obtained for all kinds of patients.

It is evident, however, that a constant relationship between sitting and supine VP should exist in patients of the same type, in whom causes altering the sitting VP act in the same direction with the same intensity. It is also evident that by eliminating certain of these causes, such as of the elevation of the arm, it would be possible to reduce the tendency to conflicting VP results.

These considerations warrant the investigation of the VP in both positions by a comparable method with the aim of finding patients with similar differences between VP values in the two positions and of assembling them in clinically defined groups. This would make it possible to calculate the standard supine VP for patients of each group on the basis of the measurements in the sitting position.

METHODS

The antecubital VP was measured in the patients first in the 45-degree sitting position with the legs straight, and then in the supine position. The arm in both positions was held along the body in abduction of nearly 45 degrees. The apparatus consisted of the Moritz and Tabora saline mano-

FIG. 1.—Measuring the venous pressure (VP) in the supine posture according to the method of Moritz and Tabora (1910) (A), and in the sitting posture according to the present method (B).

The manometer tube (M) is filled with physiological saline from reservoir (R) and is connected to the patient's vein by a large-bore needle (N). C=clamp.

A spirit level (SL) is used to set the zero point (0) of the manometer at a level 5 cm. below that of the fourth sternocostal junction (J) for patients in the supine position, and 4 cm. below that level for patients in the sitting position (0').

The choice of the latter value follows from consideration of the top diagram (C). A point (RA) (right auricle) 5 cm. below the fourth sternocostal junction in the supine position is only 3.5 cm. below that point in the subject sitting at a 45-degree angle to the horizontal. To these 3.5 cm. another 0.5 cm. is added to account for the sagging of the trunk when the trunk is elevated, giving a total of 4 cm. The height (P) of the fluid in the manometer above zero point, when equilibrium is reached, is the value of the VP.

meter, slightly modified. The reference level for the supine position was the level of the right auricle established 5 cm. below the fourth sternocostal junction (Moritz and Tabora, 1910). The level of the same point of the right auricle was taken as reference level also for the sitting posture. This level was established as a horizontal plane situated 4 cm. below the fourth sternocostal junction (Gitelson, 1951). The method and calculation of the zero level are illustrated by Fig. 1. Since in both positions the reference level is related to the same point of the right auricle, the VP values in both positions are comparable.

This report comprises the results of single and serial VP determinations in 60 patients. Fifty of these patients suffered from cardiovascular diseases, 39 were in different degrees of heart failure (Table I); five had cardiac cirrhosis. The remaining 10 were miscellaneous in-patients without cardiovascular disturbances.

TABLE I
RELATION OF THE VENOUS PRESSURE (VP) IN THE 45-DEGREE SITTING POSTURE TO THAT IN THE SUPINE IN VARIOUS TYPES OF PATIENTS

Diagnosis	Number of patients			
	Total	Group I Sitting VP higher than supine	Group II Sitting VP equal to supine	Group III Sitting VP lower than supine
<i>A. Type of disease</i>				
(1) No cardiovascular disease	10	2	6	2
(2) Rheumatic valvular heart disease (18 cases) and cor pulmonale (4 cases)	22	—	6	16
(3) Arteriosclerotic and/or hypertensive heart and vascular diseases	28	2	22	4
Total	60	4	34	22
<i>B. Degree of heart failure</i>				
(1) No failure or mild	27	4	21	2
(2) Moderate	14	—	8	6
(3) Severe	19	—	5	14
<i>C. Type of heart failure</i>				
(1) Left-sided purely (VP lower than 9 cm. saline)	9	1	7	1
(2) Right- and left-sided (VP higher than 9 cm. saline)	30	—	10	20

RESULTS

Relation of the VP in the Sitting Posture to that in the Supine (Table I).

(1) In all but four patients the VP was either equal in both positions (VP differences of 2 cm. saline or less) or lower in the sitting posture than in the supine (differences of more than 2 cm. saline).

(2) The VP was the same in both positions in the great majority of subjects with no heart failure (including normal subjects), with mild failure, or with severe but purely left-sided failure.

(3) The VP was significantly lower in the sitting posture than in the supine (differences up to 7.5 cm. saline) in the great majority of patients with severe, and in half the patients with moderate, predominantly right-sided, failure (Fig. 1).

(4) An especially high proportion with equal VP values in both positions was noted among patients with arteriosclerosis and/or hypertension (Table I). This was true also for those who had severe right-sided heart failure. Thus, only four of these patients had lower VP in the sitting posture. But even in these four the differences between the VP values in both positions were small and did not exceed 4.3 cm. saline, while in patients with severe heart failure without arteriosclerosis or hypertension these differences varied between 4.8 cm. and 7.5 cm. saline (Table II).

(5) Patients in heart failure with cardiac cirrhosis and ascites had relatively small differences between the VP in both positions (not more than 4.0 cm. saline).

(6) Old patients including those without obvious signs of arteriosclerosis and hypertension behaved like those with these signs, showing small or no differences in sitting and supine VP.

TABLE II
COMPARISON OF VENOUS PRESSURE DIFFERENCES IN THE 45-DEGREE SITTING AND SUPINE POSTURE IN SEVERE FAILURE WITH AND WITHOUT ARTERIOSCLEROSIS AND/OR HYPERTENSION

Type of patients	Number of patients	Venous pressure differences in cm. saline	
		Range	Mean
(1) Patients with arteriosclerosis and/or hypertension	8	0 -4.3	2.1
(2) Patients without arteriosclerosis and hypertension	7	4.8-7.5	5.5

Changes in the Venous Pressure when Sitting in Patients with Severe Heart Failure

Serial VP determinations in both positions performed on 15 patients with severe failure during treatment showed the following results.

(1) In all these patients, with clinical improvement and fall in body-weight the VP taken in both the sitting and supine posture fell (Fig. 2, 1-3, 5-10); when the failure increased and the body weight rose, then the VP in both positions rose (Fig. 2, 1, 3-5).

FIG. 2.—Relationship between venous pressure (VP) in sitting (circles, full lines) and supine (crosses, broken lines) postures and the body-weight (dark columns at bottom) in ten patients under treatment for severe heart failure.

The patients' ages are shown at the top and their diagnoses indicated by the letters: (A) for arteriosclerotic, (H) for hypertensive and (R) for rheumatic heart diseases. In all these patients the sitting VP rose or fell concurrently with the supine VP and the bodyweight. In the first four patients the differences between VP's in the two positions are large and constant. In the other patients the differences are smaller and disappear in some with the fall in VP to normal. Patient A.M. had cardiac cirrhosis.

(2) During heart failure the VP differences in the two positions remained more or less constant for every subject (Fig. 2, 1–6). But in arteriosclerotic and hypertensive subjects these differences tended to diminish when recovery began (Fig. 2, 7–10).

DISCUSSION

The main points illustrated by these observations are that the presence of right-sided heart failure predisposes to lower sitting VP; that vascular sclerosis and hypertension tend to prevent such a drop of the VP; and that in heart failure, the sitting VP changes in the same direction as the supine VP, and as the body weight.

These findings are intelligible if we consider the causes altering the VP in the sitting posture, mentioned above. Hence, the drop of the VP in the sitting position in young patients with right-sided heart failure may be attributed to disturbances in compensatory mechanisms, to increased distension and permeability of small vessels in the dependent parts of the body, causing a great decrease in circulating blood volume. This is in line with the findings of Wollheim (1950) who observed in patients with right heart failure a decrease in the supine VP after the patients had sat up for 30 minutes. This was in contrast to normal subjects in whom the VP in these circumstances remained unchanged.

The stability of the VP during changes in position in arteriosclerotic and hypertensive patients may possibly be explained by relative rigidity or increased tonus of small vessels, which hinder the pooling of blood in the lower parts of the body. This agrees well with the results of Turner *et al.* (1937), who found that the increase in volume of a finger on lowering the hand is less in arteriosclerotic and hypertensive subjects than in others.

The discrepancy between the results of Davis and Shock (1949) and of Winsor and Burch (1946) may perhaps be explained on the same basis. The former investigators included inmates of a home for old people among their subjects and thus may have investigated a larger number of arteriosclerotic patients. In these subjects a similar VP in both sitting and supine positions when the arm is lowered may be anticipated on the basis of our experiments. But a higher sitting VP is expected when the arm is elevated, in accordance with the method they used.

The small differences between the sitting and supine VP in patients with cardiac cirrhosis, hepatomegaly, and ascites may be attributed to increased intra-abdominal pressure in the sitting posture, which results in increase in central VP, an effect similar to the hepatojugular reflux. This may hinder or prevent the fall of VP in the sitting posture.

Some practical implications of these observations are as follows.

(1) The changes in the sitting VP obtained by this method provide a fair indication of the changes in the degree of heart failure during treatment.

(2) Sitting VP values may be substituted for “standard” supine values in old, arteriosclerotic and hypertensive patients, at least in those without signs of severe right heart failure.

(3) In patients with signs of right heart failure the supine VP values may be calculated from those of the sitting VP as follows: supine VP equals the sitting VP plus the difference in the VP’s in these positions. This difference has to be established by occasional measurement.

(4) On comparing supine and sitting VP, and assuming with Moritz and Tabora that supine values higher than 9 cm. are abnormal, it follows that values below 5 cm. in the sitting posture are normal, while those above 10 cm. are abnormal for all patients; values between 5 and 10 cm. may be normal, especially in old arteriosclerotic and hypertensive patients, or abnormal, particularly in young patients with normal pressure and right ventricular failure.

SUMMARY

The antecubital venous pressure was measured with a saline manometer in the 45-degree sitting posture with the arm lowered, and compared with that pressure measured in the supine posture.

The reference level was a horizontal plane situated 4 cm. below the fourth sternocostal junction for the sitting posture, and 5 cm. below this junction for the supine.

There was a clear tendency to a lower venous pressure in the sitting posture as compared with the supine in patients with predominant failure of the right ventricle. In patients with purely left-sided failure the venous pressure was the same in both positions. Most patients with arteriosclerosis and hypertension, even those with right-heart failure, and most old subjects had the same venous pressure in both postures.

The sitting venous pressure repeatedly estimated during the treatment of patients with severe heart failure changed in the same direction as the pressure measured in the supine posture, and as the bodyweight.

Pressures less than 5 cm. saline in the sitting posture appeared to be normal, while those above 10 cm. were abnormal for all patients. Values between 5 cm. and 10 cm. were normal, especially in patients with hypertension or arteriosclerosis, or abnormal, particularly in young subjects with right-sided failure.

It appears then that the determination of the venous pressure in the sitting posture provides a fair indication of the degree of heart failure, and makes possible the calculation of the standard supine venous pressure for certain groups of cardiac patients. Changes in the sitting venous pressure reflect alterations in the degree of heart failure during treatment.

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