diseases and supersede other types of killed virus vaccines.

Ten years ago Freund and McDermott (1942) showed that allergy to horse-serum can be produced by inoculating it incorporated in a water-in-oil emulsion along with dead mycobacteria. The sensitisation was such that intradermal inoculation of horse-serum produced a tuberculin type of allergic reaction and this sensitivity lasted at least a year in guineapigs. As far as their investigation went, the presence of dead mycobacteria in the vaccine seemed to be necessary in order to produce the allergic state; therefore probably Friedewald's or Salk's vaccines do not produce allergy. Possibly a more acceptable adjuvant than mycobacteria could be found that would have this effect, and it seems likely that adjuvant, killed virus vaccines could be devised that would produce not only very high and prolonged antibody response but also a specific allergy to the virus. Such a vaccine might well produce just as good an immunity as a living vaccine. Indeed it would have one advantage over a living vaccine in that one could give a recall dose as soon as immunity waned to below a safe level, whereas with living vaccine it is not possible to revaccinate until the person has become susceptible again to the attenuated virus. Perhaps we will eventually be able even to improve on Nature and produce a better immunity than that following a natural attack of the disease.

In suggesting the development of allergenic vaccines against viruses I am assuming firstly that allergy will help in resisting invasion of the epithelial surfaces, and secondly that allergy will not be harmful in those individuals who develop the disease despite vaccination. Many pathologists would contest the second point even if they concede the first. Obviously this is a matter on which we require further information, but at present I see little reason for apprehension about the possible harmful effect of making a person allergic to a virus. If allergy were harmful one would expect to find second attacks of a disease often more severe than first attacks. Generally the opposite is the case, although there are probably exceptions to this rule. After all, the attenuated vaccines we use today produce allergy to the virus. However, a cautious approach is indicated and perhaps we should try allergenic vaccines first in veterinary work.

In a lecture of this sort some indulgence is usually granted in allowing speculation, and I have taken advantage of this. We all know, however, how unreliable even apparently reasonable speculation can be in the field of research.

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"... the public should realise that they have to play their part. If they want hygienic food handling, they should not be afraid of asking for it and of commenting when they see hygienic failures. How many restaurant customers have the moral courage to send back to the kitchen a cup or fork which is placed before them dirty? How many just wipe it on the tablecloth and say nothing? And the spending of a few minutes behind the counter of a snack bar shows forcibly how the public cough or sneeze germs over the food on display or tentatively touch it with fingers far dirtier than any employee would dare to have."—Dr. J. D. KERSHAW, medical officer of health for Colchester, in his annual report for 1951.

EXACT DETERMINATION OF THE CENTRAL VENOUS PRESSURE BY A SIMPLE CLINICAL METHOD

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THE central venous pressure is the pressure in the veins close to the right atrium of the heart: it is virtually the same as the pressure in the right atrium when this part of the heart is in diastole.

The central venous pressure depends on the quantity of blood returned to the heart in a given time (venous return) and on the ability of the heart to pump it out (competence). There is a reciprocal relation between the central venous pressure and the function of the heart.

The pressure in the atrium can be measured in relation to the atmospheric pressure (actual pressure) or to the intrathoracic pressure round the heart (effective pressure). The effective venous filling pressure is the algebraic difference between the actual pressure and the intrathoracic pressure (Henderson and Barringer 1913, Wiggers 1914).

In the heart-lung preparation the venous pressure regulates the cardiac output (Patterson and Starling 1914, Wiggers and Katz 1922). In the intact healthy body, on the other hand, hormonal and nervous factors exercise a great and sometimes overwhelming influence. When the heart becomes insufficient it behaves more like the heart-lung preparation of Starling, and the dependence of the cardiac output on the venous pressure is then seen quite clearly (Richards 1947). Although it would be more accurate always to measure the effective venous pressure, one must in practice be satisfied with the measurement of the actual pressure.

CLINICAL SIGNIFICANCE OF CENTRAL VENOUS PRESSURE

(1) *A single determination* of the central venous pressure tells us whether the right heart is efficient or not (either at rest or after exertion), and thus whether œdema and ascites are of cardiac, extracardiac, or mixed origin. It can also tell us whether an acute state of shock has a central origin (cardiac infarction or lung embolus) or is due to a decrease in the blood volume or to peripheral vasodilatation. Further, it is a valuable aid in the differential diagnosis of dyspnœa: in the presence of left cardiac insufficiency, with pulmonary congestion, the venous pressure is almost always raised; but, when the dyspnœa is pulmonary in origin, especially in asthma, emphysema, and chronic bronchitis, the venous pressure (during inspiration) tends to be strikingly low, provided there is no concomitant appreciable damage of the right heart.

(2) *Repeated determinations* of the central venous pressure over a period are an excellent measure of spontaneous changes in the state of the heart in cardiac patients and of the effect of rest in bed, salt-free diet, diuretics, digitalis, quinidine, and other therapeutic agents. An increase in venous pressure during intravenous infusion gives warning of overloading of the heart. The influence of the retention of water and sodium chloride on the circulation may easily be determined by following changes in the venous pressure in renal diseases (especially acute glomerulonephritis) and during treatment with adrenal cortical hormones or related compounds.

Any scientific investigation into the control of the circulation is incomplete if the changes in the venous pressure are not followed in detail.

PREVIOUS METHODS OF MEASUREMENT

The method to be used to measure the venous pressure in the above-mentioned types of cases should not be unpleasant for the patient, and the determination should not take too much time. The results obtained by experienced investigators must be accurate to at least 1 cm. of water; for an increase in the diastolic pressure in the right ventricle by 2·5 cm. of water may cause in normal people a rise of 40% in the cardiac output (Cournand et al. 1948). The methods in use hitherto have not fulfilled all these requirements, and this is why the measurement of the venous pressure has not become universal in routine clinical examination or scientific investigation.

Method of Moritz and von Tabora

For measuring the peripheral venous pressure a large needle is inserted into the cubital vein and connected to a manometer filled with sterile sodium citrate (Moritz and von Tabora 1910). The results obtained in normal people by numerous observers have varied very greatly and are 4–12 cm. of water higher than the pressure in the right atrium. The values obtained by the method of Moritz and von Tabora in cases of congestive heart-failure are sometimes improbably high. This is due to the distance between the atrium and the point at which the pressure is determined. As a result the speed of the blood flowing in the vein, and the diameter of the lumen of the vein, which may change by vasoconstriction or dilatation, exert a considerable influence on the value found. The supposition that there is always a linear relation between the peripheral venous pressure and the right atrial pressure cannot be upheld, as has been shown by Moia et al. (1950) and Pedersen (1952). If the vein collapses anywhere between the point where the pressure is determined and the right atrium, the linear relation fails immediately (Ryder et al. 1944). Negative pressures cannot be measured in the peripheral veins (Doupe et al. 1938).

Since the introduction of cardiac catheterisation into clinical methods (Forssmann 1929, Cournand and Ranges 1941, McMichael and Sharpey-Shafer 1944a) it has been possible to determine the mean pressure in the right atrium with a manometer attached to the cardiac catheter. It is clear, however, that this is impracticable as a routine method and especially as a method of following changes in the venous pressure over long periods.

Method of Lewis

Lewis (1930) laid the foundation for the determination of the central venous pressure by using the external jugular vein as a manometer to record pressure changes in the right atrium. He stated that in normal persons the zero (or atmospheric) pressure level of the fluid in the venous system is near the attachment of the second rib to the sternum at the angle of Louis. This would apply for any position of the body, lying, sitting, or standing. Although Lewis used the method of Moritz and von Tabora for *measuring* the venous pressure, he stated:

> "If therefore we can gauge the precise level at which the veins collapse we have a gauge of the filling of the venous reservoir, or to be more exact of right auricular pressure."

McMichael (1938) obtained as good results by Lewis's method as by that of Moritz and von Tabora. We are of the opinion that of the two methods that of Lewis is much to be preferred, provided that the technique is modified and standardised. The distance between the point where the pressure is recorded in the external jugular vein and the right atrium is short, and the lumen of the vein is large; hence pulsations caused by the contraction of the heart and by respiration are little altered. By always making the patient lie so that the vein is partly filled it is possible to measure negative pressures. The pressure in the external jugular vein during atrial diastole is thus almost equal to, and certainly closely related to, the pressure in the right atrium.

THE MODIFIED METHOD OF LEWIS

During the past ten years, investigations have been made in our clinic to develop a standard technique for the measurement of the central venous pressure. The provisional results have already been published by Allen (1948). Like Leishman (1946) we originally used a simple measuring device, which we later modified. Whereas Leishman found the same values as Lewis in normal people, Allen, with the standardised method, obtained results 5 cm. lower than those of either of these observers, and 6–10 cm. lower than those obtained by the method of Moritz and von Tabora for peripheral venous pressure.

Since 1948 we have measured the central venous pressure in a large number of normal persons, and our results are still lower than those reported by Allen, although only slightly so. Great care must be taken to have the patient lying in a good position, and the pressure is measured only when the patient is emotionally and bodily at rest. If attention is paid to these points, the results obtained by two experienced persons generally differ by not more than 0·5 cm.

Since the end of 1947 the measurement of the central venous pressure by the modified method has formed part of the routine examination of all patients, whether admitted to the clinic or attending as outpatients. It has been an indispensable link in our investigations into the relation between the circulation and the excretion of water and electrolytes (Borst 1948, Molhuysen et al. 1950, Blomhert et al. 1951).

Principle of Method

If the external jugular vein is to be used as an atrial manometer, two requirements must be fulfilled:

(1) There must be free communication between the neck veins and the right atrium. We therefore can only measure the pressure when the valves in the veins are open—i.e., during inspiration and falling atrial pressure ("negative" x and y wave or trough of the venous pulse tracing). During expiration and during atrial systole the central venous pressure rises and the valves of the veins close. Thus during expiration and atrial systole the filling of the vein does not reflect the atrial pressure; moreover the determination of the highest point to which the vein is filled is more difficult than the determination of the lowest point of complete collapse of the vein.

(2) There should theoretically be no flow of blood in the vein while the pressure therein is being measured. Therefore we obstruct the peripheral blood-stream by light pressure with the finger on the distal end of the vein just below the angle of the mandible. A new potential source of error is introduced by closing the upper end of the manometer, but this does not prevent the obtaining of a reliable reading, since the vein is not a rigid tube and does not require any significant pressure to cause it to collapse.

Reference Point

The pressures in the heart are measured in centimetres of water in relation to atmospheric pressure, which is regarded as nil (actual pressure). To eliminate hydrostatic factors the intracardiac pressure must be measured from a horizontal plane through the heart. This plane cannot be determined; therefore a fixed reference point is used on the thorax. The vertical distance from this point to the selected horizontal plane should be as constant as possible. Following Bloomfield et al. (1946) we chose a plane 5 cm. below the angle of Louis with the patient lying horizontal. In theory the plane should pass through the centre of the right atrium, but in practice this is often far from the case. In every person the position of the plane in relation to the reference point is different; moreover it varies with respiration and during the cardiac cycle. Bloomfield et al. measured in normal people the vertical distance from the angle of Louis to the midpoint of a straight line between the anterior tip of the ventricle and the lowest point of the atrium by lateral radiography. They found an average value of

5·82 ± 0·90 cm. In patients with cardiac enlargement the distance was 5·55 ± 1·50 cm., and in cases of emphysema 6·86 ± 0·80 cm. Richards et al. (1942) obtained slightly lower values in normals, using a similar method.

With our method it is not always possible to measure the venous pressure with the patient horizontal. The patient may be required to sit up more or less, according to the degree of elevation of the central venous pressure. If the pressure is low (as in many normal people), the foot of the bed must be raised 20–40 cm. to fill the veins of the neck to a point midway between the angle of the jaw and the clavicle. Since the external jugular veins have a slightly downward course to the heart when the patient is lying in the horizontal position they can still be used as an atrial manometer in the head-down position, so long as their cephalic ends lie above the horizontal plane through the entrance of the superior vena cava into the right atrium. The centre of the right atrium will not then be at the same vertical distance from the reference point as it is in the horizontal position, but the distance will not vary much from 5 cm. The error involved in sitting patients with a greatly raised venous pressure may be larger, but will be relatively small.

We can thus measure the central venous pressure by determining the distance between two horizontal planes. The one lies 5 cm. below the angle of Louis, and the other passes through the lowest point at which the external jugular veins collapse during normal inspiration, the blood returning from the head being excluded by light digital pressure.

Fig. 1—Incorrect position of patient. Note insufficient support from pillows, and kinking of external jugular vein.

Practical Details

It is essential to pay particular attention to the patient's position to eliminate the chance of pressure on the abdomen or "kinking" of the veins, both of which raise the measured pressure.

First, one determines approximately the position required so that, when the cephalic end of the external jugular vein is occluded, the vein collapses about midway along the line between the angle of the mandible and the clavicle. If necessary, the foot of the bed is lifted. Next, the position is improved by the use of pillows or cushions under the head, trunk, or lumbar region. The patient must be relaxed, and the spine slightly over-extended, while the head is so extended that the scalene muscles are taut but the sternomastoid muscles remain relaxed. The neck in particular must not be flexed, since the neck veins are then liable to be occluded at the thoracic inlet (fig. 1). Finally, the head is rotated about the axis of the spine into the position in which the pulsations in the vein are most clearly seen (fig. 2).

To see best the lowest point at which the external jugular vein collapses it is nearly always necessary to make the examination in subdued lighting with oblique or tangential illumination from a torch held as shown in fig. 3.

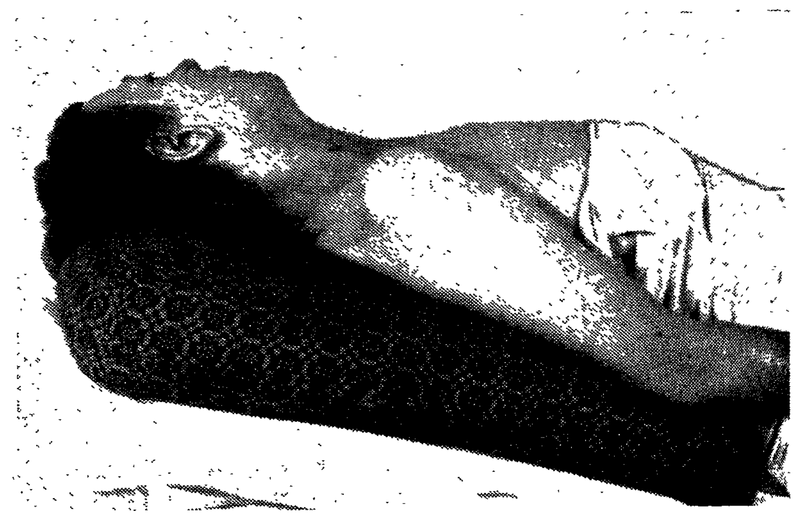

Fig. 2—Correct position of patient on firm cushions (central venous pressure is apparently above normal).

The waves of the venous pulse (fig. 4) are caused by the intermittent building-up of the centripetal blood-stream against the closed valves in the veins, and the troughs or negative waves appear abruptly when the valves open, since the pressure in the atrium is lower than that in the veins. One can distinguish *a*, *c*, and *v* waves, with *x* and *y* troughs. For our determinations we use the *x* and *y* troughs, for at these points of the cycle the valves are open. When the cephalic end of the vein is occluded by finger pressure (fig. 3), the pressure in the vein falls and becomes almost equal to the lowest pressure which occurs in the atrium—i.e., when the end of inspiration coincides with the *x* or *y* trough of the venous pulse. The pulsations in the vein disappear almost completely as the return of blood through the valve is prevented. There are, however, many exceptions to this; blood often enters the vein through patent tributaries, and blood collects once more above the closed valves, whereby the vein may again collapse when the valves open. The superficial tributaries may normally be occluded successfully at the same time. By raising the finger which was preventing the return of venous blood the vein is again filled, and pulsations are seen at a higher level once more. Repeated observations of the lowest point at which the vein collapses may thus be made, allowing its position to be determined with accuracy. The pressure gradually falls slightly at rest in patients with a raised central venous pressure, having been raised somewhat by the preceding activity. As a check, one may determine the pressure in the contralateral external jugular vein, or in the internal jugular vein. In the latter case slightly more pressure must be exerted by the finger occluding the peripheral end of

Fig. 3—Interruption of centripetal blood-stream. Note position of torch to give oblique lighting.

the vein. A mark is made on the skin to indicate the lowest point of collapse, followed by the actual measurement of the pressure.

For the determination of the difference in level between the point of venous collapse and the reference point we use the instrument shown in fig. 5. A small spirit-level is incorporated in the horizontal arm. The method of use is obvious from the figure.

Although it is simple, the determination of central venous pressure, like any clinical method, has to be learnt by experience.

Difficulties in Finding Correct Point of Collapse

(1) The external jugular vein may be kinked at the thoracic inlet if the neck is flexed too far forwards, or if it is too far extended, whereby the muscles of the neck are not relaxed. It is also possible for the head to be insufficiently or excessively rotated. Occasionally no pulsations are seen while the vein is distended. By improving the position of the patient or the rotation of the head, pulsations can usually be brought into view and the vein made to collapse after interruption of the centripetal blood-stream. The vein may be kinked even if pulsations are seen, because the posterior facial vein divides into two parts, of which the posterior helps to form the external jugular vein, and the anterior helps to form the common facial vein, which joins the internal jugular vein. Blood may thus reach the atrium by the alternative path; the intermittent flow causes pulsations,

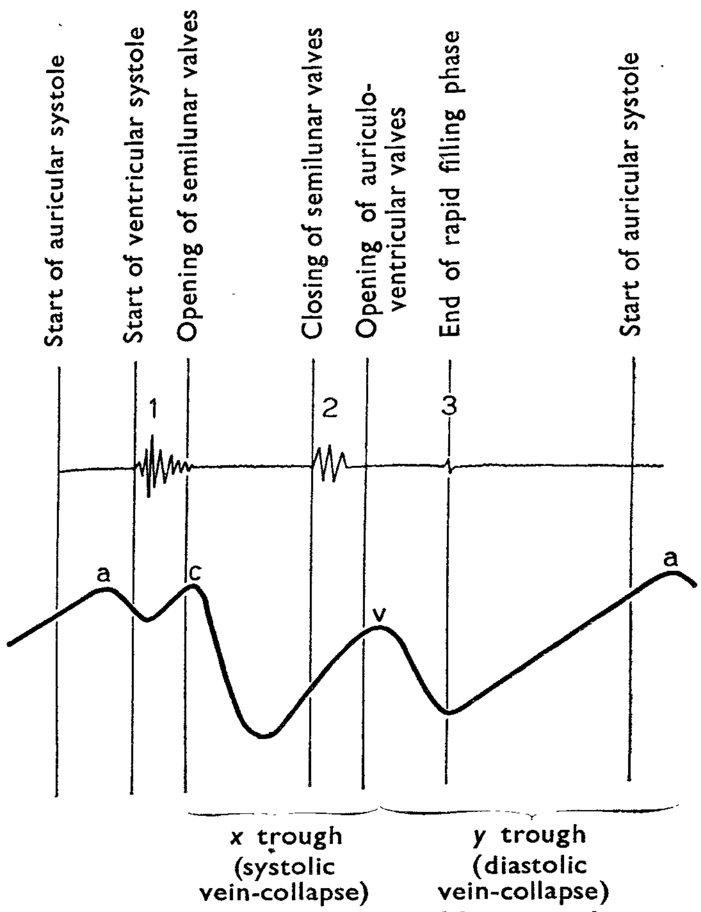

Fig. 4—Jugular venous pulse and heart sounds.

but the external jugular vein will not collapse at the "lowest point." The point of collapse is then usually not sharply defined until the venous occlusion at the thoracic inlet is overcome, and a direct route established for the flow of blood from the external jugular vein to the right atrium.

(2) The measurement of the pressure may be difficult if the external jugular vein is poorly developed and pulsates poorly (in 14% of the patients in our series). In this event sometimes the internal jugular vein should be used.

(3) In severe shock the pulsations in the external jugular vein are small and the vein is contracted. It

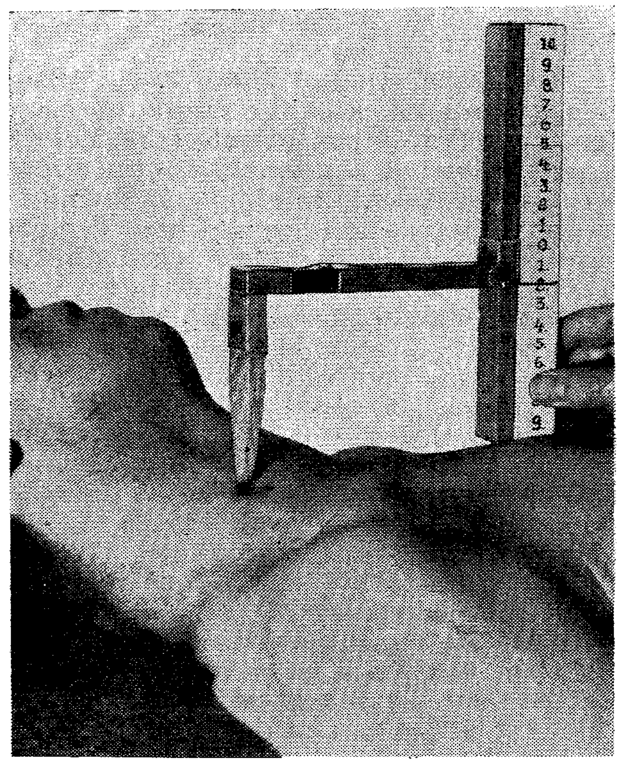

Fig. 5—Method of measuring central venous pressure. A spirit-level is incorporated in the horizontal arm. Central venous pressure is +3 cm. of water (2 cm. below sternal angle).

may be impossible to measure the central venous pressure in such cases.

(4) Sometimes there are no pulsating or collapsing veins in the neck, as a result of which it may be impossible to measure the venous pressure at all (6% of our cases).

(5) Unilateral or bilateral congested veins without pulsations, even in the sitting position, indicate obstruction to the return of venous blood to the heart, from pressure on the large veins and heart—e.g., mediastinal tumours, enlarged lymph-glands, and aortic aneurysm—or thrombosis of the veins.

RESULTS

Of a series of 400 outpatients:

1. The central venous pressure was determined without difficulty in 321 (80%). The external jugular veins were well developed, pulsated well, and collapsed clearly.
2. The pressure was determined less easily or with difficulty in 55 patients (14%). The external jugular veins were less well developed, pulsated less actively, or collapsed less clearly, and in some cases pressure had to be measured in the internal jugular vein.
3. The pressure could not be measured in 24 patients (6%). There were no visible neck veins or it was impossible, in spite of every attention to the position of the patient, to find a vein which pulsated or collapsed well.

In 332 cases two experienced observers determined the central venous pressure independently. The results showed no difference in 195 (59%); a difference of not more than 0·5 cm. in 104 (31%); and a difference greater than 0·5 cm. in 33 (10%). Thus in 90% of the

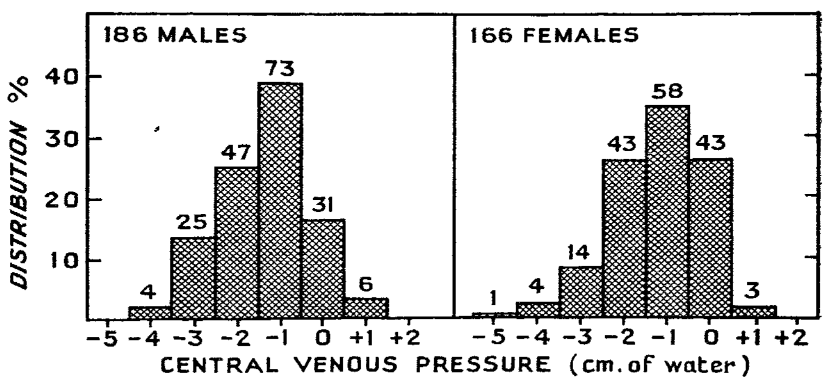

Fig. 6—Distribution of normal values of central venous pressure.

cases investigated the duplicated determinations differed by not more than 0·5 cm.

NORMAL CENTRAL VENOUS PRESSURE

We have taken as normal the results obtained in 352 completely investigated outpatients (186 males and 166 females) who showed no abnormality of the circulatory or respiratory systems, and in whom no definite anæmia or decrease in the plasma-proteins was demonstrable.

Since the error in measurements is generally not more than 0·5 cm., the results are grouped at intervals of 1 cm.: from +1·5 to +0·5 cm., from +0·5 to −0·5 cm., from −0·5 to −1·5 cm., . . . from −4·5 to −5·5 cm. The distribution of the normal figures for these males and females is given in fig. 6, which shows that the greatest frequency is found in the interval from −0·5 to −1·5 cm. in both sexes.

The result of the calculation of onesided confidence intervals for the percentages of males and females with a central venous pressure between +0·5 and −3·5 cm. of water is as follows: with a confidence coefficient of 0·005 (0·5%) at least 88% of the males have a central venous pressure between +0·5 and −3·5 cm. of water, whereas with a confidence coefficient of 0·1 (10%) at least 91% lie between +0·5 and −3·5 cm. of water. For the females these figures are 89% and 92% respectively.

In a series of 165 men and 145 women not only was the central venous pressure and the age known, but also the diameter of the thorax at the level of the angle of Louis. The relations between the central venous pressure on the one hand, and the age and the thoracic diameter on the other were statistically investigated. There was found to be a definite relation. To eliminate this factor of dependency of the central venous pressure on age and diameter of the thorax a correction table was compiled from our observations (Molhuysen 1952). New histograms were constructed, but these did not differ materially from the original histograms, and we have given up using them.

Daily Variation

The changes in central venous pressure during the day are fairly large, but seldom more than 1 cm. in normal persons. Patients with a raised central venous pressure often show changes of several centimetres.

RELATION BETWEEN CENTRAL VENOUS PRESSURE AND MEAN PRESSURE IN RIGHT ATRIUM IN NORMAL PERSONS

Several investigators have measured the mean pressure in the right atrium with a manometer attached to a cardiac catheter, with the subjects lying down. In table I we compared their results with the normal values for the central venous pressure determined by our method.

McMichael and Sharpey-Shafer (1944b) took the horizontal plane through the angle of Louis as their zero pressure level. Their figure is therefore increased by 5 cm. to make it comparable with those of other investigators, who, like ourselves, used a horizontal plane 5 cm. below the angle of Louis as zero pressure level.

Because in our method the pressure in the jugular vein is determined when the pressure in the atrium is lowest—i.e., during inspiration and ventricular systole—it is to be expected that our results will be lower than the mean pressure in the atrium. The pressure in the right atrium changes during the cardiac cycle by about 3–4 mm. of mercury (Cournand et al. 1951). Our results, which are slightly lower than those of Allen (1948), agree satisfactorily with those of Bloomfield et al. (1946), account being taken of the above factors.

TABLE II—CENTRAL VENOUS PRESSURE AND MEAN RIGHT ATRIAL PRESSURE MEASURED SIMULTANEOUSLY

Case no.	Sex	Age (yr.)	Diagnosis	Size of catheter	Pressure (cm. of water) Right atrium	Pressure (cm. of water) Central venous
1	F	23	Coarctation of aorta	9	−2·5	−2
2	M	23	Single ventricle	9	0	−1
3	F	25	Patent ductus arteriosus	9	0	0
4	F	14	Pulmonary stenosis	8	−2	−2
5	F	12	Pulmonary stenosis	8	0	−1
6	M	21	Pulmonary stenosis	8	+3	−1·5
7	M	33	Pulmonary stenosis	9	−1	−1
8	M	12	Pulmonary stenosis	8	+3	−1
9	F	33	Mitral stenosis	8	−0·5	−1
10	M	29	Atrial septal defect	10	+2	+0·5
11	F	15	Patent ductus arteriosus	8	+1·5	0
12	F	20	Patent ductus arteriosus	8	0	−1

SIMULTANEOUS MEASUREMENT OF CENTRAL VENOUS PRESSURE AND MEAN PRESSURE IN RIGHT ATRIUM

In 11 cases of congenital heart-disease and 1 of mitral stenosis we measured the central venous pressure with the patient lying down and at the same time the mean pressure in the right atrium by connecting an intracardiac catheter to a water manometer. The results are given in table II.

Except for 1 patient the pressure in the jugular vein was equal to, or less than, the pressure in the atrium. In 8 cases the difference was 1 cm. or less, in 2 cases 1·5 cm., and in 2 cases 4 and 4·5 cm. One of these latter showed a raised mean pressure in the right ventricle. We could not ascertain whether the systolic pressure in the right atrium was higher than normal in the last 2 cases. In general there was very good agreement between the central venous pressure and the mean right atrial pressure.

FACTORS INFLUENCING CENTRAL VENOUS PRESSURE

In discussing several factors which may exert an influence on the central venous pressure one must distinguish patients with normal cardiac function from those with cardiac insufficiency.

Respiration

During normal respiration the changes resulting from changes in the depth of inspiration are less than 0·5 cm. Although we observed differences of 0·5–2·0 cm. between the pressure measured during inspiration and that measured during expiration, it is questionable, as already stated, whether the pressure during expiration can be measured by our method, since the valves will close immediately, when there is a reversal of direction of blood-flow. This is not a serious objection to the method. If the respiration is normal, the mean difference between the minimum pressure in the right atrium during inspiration and the minimum pressure during expiration is not

TABLE I—NORMAL VALUES OF CENTRAL VENOUS PRESSURE AND MEAN ATRIAL PRESSURE

Reference	Site of measurement	Method	Pressure (cm. of water)	No. of cases
Richards et al. (1942)	R. atrium	Water manometer	From +0·8 to +6	9
McMichael and Sharpey-Shafer (1944b)	R. atrium	Water manometer	Av. +1	?
Stead et al. (1945)	R. atrium	Water manometer	Av. +3·1 (from 0 to +8·5)	18
Bloomfield et al. (1946)	R. atrium	Hamilton manometer	From −2·7 to +2·7	9
Allen (1948)	Jugular vein	Lewis-Borst	From −1 to +2 (80%)	55
Present investigation	Jugular vein	Lewis-Borst	From −3·5 to +0·5 (90%)	352
Lagerlöf and Werkö (1948)	R. atrium	Hansen manometer	Av. +3·7	13

more than 1·6 cm. of water; though an extreme value of 2·4 cm. of water has been found (Battro et al. 1949).

During forced inspiration the central venous pressure falls several centimetres. The values obtained with our method will then be much lower than those of the mean right atrial pressure. This is seen sometimes in patients with emphysema, bronchial asthma, or chronic bronchitis. If there is severe dyspnœa it is impossible to measure the pressure, because the veins are sucked completely empty in whatever position the patient is placed.

Although the pressure in the atrium falls during inspiration, this change is partly compensated for by the more rapid return of blood to the heart. The effective pressure in the right atrium increases in inspiration and falls in expiration (Lauson et al. 1946).

Posture

So long as the jugular vein collapses between the angle of the mandible and the clavicle, it is possible to determine the central venous pressure by our method. If the central venous pressure is measured first with the patient horizontal, and the body is then elevated to 30° from the horizontal, small changes will be found, usually not more than 0·25–0·50 cm. and never more than 1 cm. (Allen 1948). When the central venous pressure is raised, the patient will have to be more or less sitting, whereby the vertical distance from the centre of the atrium to the reference point alters and the result will differ from that which would be obtained with the patient lying down. When the central venous pressure is much raised, the change in position of the heart will exert relatively little influence on the result. The venous return falls slightly with the patient sitting, and the pressure is found to be slightly lower than with the patient lying down. The reverse obtains for the Trendelenburg position.

Exertion

In 376 outpatients with a normal circulatory system the central venous pressure did not rise after twenty "knees-bends" but remained constant or fell by at the most 0·5 cm. This fall may be explained by the deeper inspiration.

In cardiac patients with a raised central venous pressure a rise of several centimetres was seen after exertion (drawing up of the legs) followed by a slow return during 1-5 minutes to the original level. If the central venous pressure is normal in a patient at rest, one can discover by the effect of exertion whether there is latent cardiac insufficiency.

Pressure on Liver: Hepatojugular Reflux

In the same group of 376 patients with normal cardiac function no rise of the central venous pressure was seen during pressure on the region of the liver, if the patients continued to breathe quietly.

In the presence of latent or manifest cardiac insufficiency the central venous pressure rose several centimetres. Cessation of the pressure was followed by a rapid return of the venous pressure to the initial value. We apply this test in conjunction with the exertion test to diagnose latent cardiac insufficiency.

Emotion

In patients with normal cardiac function no measurable change in central venous pressure due to emotion was observed. In the presence of impaired cardiac function and a raised central venous pressure we often observed a further rise of several centimetres under the influence of emotion.

SUMMARY AND CONCLUSIONS

By a modification of the method of Lewis it is possible to measure the central venous pressure rapidly, accurately, and in a manner not unpleasant for the patient. We successfully measured the pressure in 376 (94%) of 400 outpatients and failed to obtain a reading in only 24 (6%).

In 332 cases with a normal circulatory system the central venous pressure was determined by two experienced observers. In 299 (90%) of these cases the difference between the two readings was 0·5 cm. or less. In 33 (10%) the difference was greater than 0·5 cm.

The central venous pressure was determined in 352 outpatients with normal circulatory and respiratory systems and without anæmia or decreased plasma-proteins, and the results were analysed statistically. In 90% of persons in whom a normal central venous pressure is to be expected the pressure lies between +0·5 and −3·5 cm. of water in relation to a horizontal plane 5 cm. below the angle of Louis.

There was a demonstrable relationship between the central venous pressure, age, and diameter of the thorax. Since the distribution of normal results is little affected by the elimination of these relationships it is unnecessary to take them into consideration in practice.

The normal values for the central venous pressure are lower than those of the mean pressure in the right atrium, since by the modified method of Lewis the pressure is measured when the atrial pressure is at its lowest—i.e., when the end of inspiration coincides with the *x* or *y* trough of the venous pulse. If this fact is taken into consideration, there is seen to be good agreement between the normal values and the values for the mean pressure in the right atrium.

The mean pressure in the right atrium was measured simultaneously with the central venous pressure in patients with congenital heart-disease and mitral stenosis but without evidence of cardiac insufficiency. In 8 cases the difference between the two readings was 1 cm. or less; in 2 there was a difference of 1·5 cm.; and in 2 other patients a difference was found of 4 and 4·5 cm.

Factors which may influence the central venous pressure are discussed.

We are indebted to Prof. D. van Dantzig, Prof. J. Hemelrijk, and Mr. T. J. Terpstra, of the statistical department of the Mathematisch Centrum, Amsterdam, for the statistical analysis of our results, and to Dr. A. P. M. Verheugt, cardiologist of the Onze Lieve Vrouwe Gasthuis, Amsterdam, for permission to compare our method with that of cardiac catheterisation. This work was aided by a grant from the Netherlands Organisation for Pure Research.

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