Physical examination of venous pressure: A critical review

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Objective To explain why investigations of the measurement of central venous pressure (CVP) usually reveal a discrepancy between the clinician's estimate of CVP from physical diagnosis and supine measurement with a catheter. Data from MEDLINE search, personal files, and bibliographies of textbooks on physical diagnosis and cardiology were used.

Results The most important reasons for this disagreement are the failure to standardize the external reference point used by the clinician to indicate "zero" venous pressure and the failure to recognize that venous pressure often depends on the position of the patient during examination. During physical examination clinicians tend to underestimate the CVP, as measured by a catheter in the same patient positioned supine, especially when the measured value is high. This occurs because the venous pressure of patients with heart failure, in contrast to that of healthy individuals, demonstrates an exaggerated postural fall when the patient is in the more upright positions that are necessary to visualize the elevated neck veins. The cause of this postural instability, increased venoconstriction from sympathetic tone, also helps explain two other physical findings of the jugular veins, the abdominojugular test, and Kussmaul's sign.

Conclusions Clinicians should avoid making decisions about degrees of CVP elevation that are imprecise and difficult to reproduce. Instead, they should determine during physical diagnosis merely whether the CVP is elevated. Until further research is done, the best definition of elevated CVP is that of Sir Thomas Lewis—when the top of the external or internal jugular veins is >3 cm of vertical distance above the sternal angle, the CVP is abnormally high. (Am Heart J 1998; 136:10-8.)

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Although clinicians began to associate conspicuous neck veins with heart disease almost 3 centuries ago,[1,2] the practice of actually measuring a patient's venous pressure during physical examination is only several decades old. Even Sir James Mackenzie, who in the late 1800s described most of what we now know about bedside diagnosis of the jugular veins—the a, c, and v waves, venous sounds, cannon A waves, venous waveforms in heart disease, and bedside diagnosis of atrial fibrillation (by examination only of the pulse and neck veins, before the era of electrocardiography)[3,4]—totally ignored the concept of measuring venous pressure. According to Mackenzie, neck veins were either prominent, a sign of heart failure, or they were normal; more refined descriptions of venous pressure simply were not useful.

Venous pressure became more important to clinicians in the twentieth century after direct cannulation of the antecubital vein allowed clinicians to measure pressure directly by manometry[5] and after Ernest Starling's investigations between 1912 and 1914 that linked venous pressure to cardiac output.[6-9] In his book "The Failure of Circulation," Tinsley Harrison further endorsed Starling's ideas[10] and, along with others, encouraged clinicians to regard the elevated venous pressure as an early and essential finding of heart failure.[11,12] The treatment of heart failure became less empirical and more rational, and venous pressure became the objective end point that clinicians monitored frequently, often on graph paper, after administration of digitalis, phlebotomy, or diuretics.[13-15]

Physical examination and direct cannulation of the venous system are both used to measure venous pressure. Several studies have compared these techniques,[16-20] most concluding that physical examination is inaccurate and unreliable. This article will review the shortcomings of these investigations, showing that both methods are clinically useful but provide data that are not directly comparable. Furthermore, it will discuss the pathogenesis of elevated venous pressure and the mechanisms of two physical findings involving the jugular veins, the abdominojugular test, and Kussmaul's sign. Understanding these issues first requires an appre-

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Table I. Proposed external reference points for measurement of venous pressure

Author	Suggested reference point*	Evidence
Von Recklinghausen[29] (1906)	Midway between xiphoid and back†	No data offered
Moritz[5] (1910)	5 cm below thoracic surface at fourth costal cartilage† thoracic surface usually pierces the junction of vena cava and right atrium	In cadavers with varying chest dimensions, a needle inserted at this point and parallel to the anterior
Young[30] (1923)	8 cm posterior to sternum†	No data offered
Harris[31] (1928)	Anterior axillary line†	No data offered
Eyster[32] (1929)	Junction of anterior and middle thirds of anterior-posterior dimension at fourth intercostal space†	No data offered
Taylor[14] (1930)	Midaxillary line†	No data offered
Griffith[33] (1934)	2½ inches below sternal angle†	No data offered
Lyons[28] (1938)	10 cm anterior to skin of the back†	Usual location of right atrium in dissections of frozen cadavers, and author's experience that reference to posterior body wall minimized variation in pressure measurements in patients with widely divergent chest dimensions
Holt[22] (1940)	10 cm anterior to skin of the back‡	Point minimizing variation of venous pressure in 10 individuals with varying chest dimensions who were positioned supine and then prone
Winsor[23] (1945)	"Phlebostatic axis"—intersection of plane parallel to anterior surface midway between back and xiphoid and cross-sectional plane through fourth ntercostal space at sternum‡	Point minimizing variation of venous pressure in 90 individuals with varying chest dimensions who were placed in various positions from supine to 90 degrees upright
Borst[34] (1952)	5 cm below sternal angle‡	No data offered

*Identification of reference points assumes the patient is supine.
†Reference point "accurate" only when patient supine.
‡ Reference point "accurate" in any position.

ciation of three important concepts: central venous pressure, zero points, and reference points.

Central venous pressure

The central venous pressure (CVP) refers to the mean vena caval or right atrial pressure, which is equivalent to right ventricular end-diastolic pressure in the absence of tricuspid stenosis. The higher the CVP, the greater the passive diastolic filling of the right ventricle and, according to Starling's cardiac function curves in normal hearts, the greater the right ventricular stroke volume of the subsequent beat. CVP is expressed in millimeters of mercury (mm Hg) or centimeters of water (cm H_2O) above atmospheric pressure (this article uses cm H_2O; 1.36 cm H_2O = 1.0 mm Hg). Because this article discusses investigations of both the systemic venous pressure and right atrial pressure, the term "venous pressure" will refer to the former and "CVP" to the latter.

Physiologic zero point

The physiologic zero point is the location in the cardiovascular system where the CVP is tightly regulated, changing little if at all during the volume shifts that occur when the patient stands or sits. To obtain reproducible measurements that are independent of position, the "zero" mark of the manometer or electronic system used to measure venous pressure should lie at the same vertical height of this point.

In 1956, Guyton et al.[21] studied 38 anesthetized dogs and found that the zero point for each dog was at an identical anatomic location—in the right ventricle just beyond the tricuspid valve. When this point was used for both the axis of rotation and the zero level of the manometer, there was minimal variation of venous pressure as the dog was rotated horizontally about a longitudinal axis and then vertically about the transverse and anterior-posterior axes (the variation in venous pressure, for example, during rotation of the animal head-up and then head-down was < 1 cm H_2O).

There are few studies of the zero point in human beings, if it even exists, although most clinicians assume that it lies in the right atrium. In investigations where the zero mark of the manometer was kept on the phlebostatic axis, a line representing the intersection of the cross-sectional plane through the fourth intercostal space at the sternum and the coronal plane midway between back and xiphoid—a line that traverses the posterior right atrium of most individuals—the

venous pressure of healthy adults changes less than 1 to 2 cm H_2O whether the individual is supine, prone, or in various positions between supine and upright.[22-24] Whether the right atrium is the actual zero point, however, is uncertain because the zero point should logically lie in the patient's midline to minimize the hydrostatic pressure changes that would occur when an individual rolls from side to side[25]; most of the right atrium, of course, lies in the right chest. The fact that Guyton included in his experiments such rotation about the longitudinal axis is probably what led him to select a more midline structure, the right ventricle, as his zero point.

Just as the precise location of the zero point in human beings is unknown, the mechanism for the normal postural regulation of pressure at this point remains inadequately investigated. Of several possible mechanisms, the most important is the vast distensibility of the normal systemic veins, which contain two thirds of the total blood volume and as capacitance vessels can easily accommodate or donate blood with little or no change in pressure. Starling's cardiac function curves may also come into play: if the CVP should fall with standing, the stroke volume of the right ventricle would also abruptly fall because of decreased right ventricular distension. Because systemic venous return would continue, however, the reduced right ventricular output would tend to raise the CVP to its original value. Finally, of the 500 to 750 ml of blood that accumulates in the legs with standing, 75% comes from the pulmonary circulation and only 25% from the systemic circulation, thereby minimizing the affect on CVP.[26] Although peripheral venoconstriction with standing would also help regulate CVP, one study showed this didn't occur.[27]

The external reference point

Over the last century, investigators have proposed numerous landmarks to help clinicians locate the level of the right atrium, most as reference points for directly measuring the supine antecubital venous pressure with a catheter (Table I). The evidence for these reference points varies, resting in some cases on anatomic dissection,[5,28] in others on a trial-and-error search for the zero point,[22,23] but in most, unfortunately, on no data, the landmark simply representing a convenient point.[14,29-34]

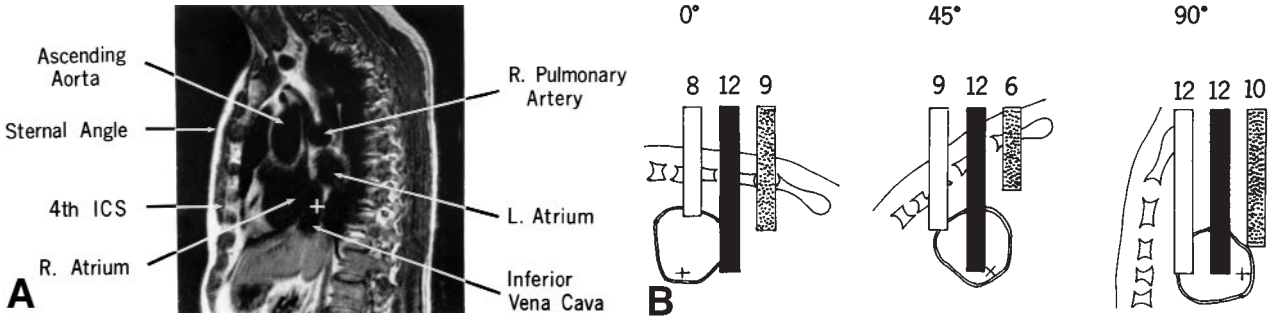
Sir Thomas Lewis, a pupil of Mackenzie, proposed in 1930 a simple bedside method for measuring venous pressure designed to replace the manometer, which he found too burdensome for general use. He observed that the top of the jugular veins of normal individuals (and the top of the fluid in the manometer) always came to lie within 1 to 2 cm of vertical distance from the sternal angle, whether the individual's position was supine, semiupright, or upright. If neck veins were higher than this, Lewis concluded the patient had elevated venous pressure.[12] A modification of this technique, commonly cited in textbooks[35] and review articles[36] and sometimes called the "method of Lewis" states that the CVP equals the vertical distance between a point 5 cm below the sternal angle and the top of the neck veins,[13,34,37,38] although Lewis did not make such a claim.

Obviously, the measurement of venous pressure is only as good as the reference point used, and studies reveal that these external reference points identify sites that differ in the supine patient by several centimeters vertically (if one clinician, for example, uses a reference point 4 cm above that used by a second clinician on the same patient, the first clinician's CVP measurement will be 4 cm lower even if he or she agrees about the position of the neck veins or manometer fluid). When the various landmarks in Table I were compared with the position of a right atrial catheter on lateral chest radiographs of patients in supine position, the method of Lewis identified a point consistently 1 to 2 cm anterior to the catheter whereas the phlebostatic axis identified a point consistently 2 to 3 cm posterior.[39,40] The best approximation of the position of the catheter in one study was 43% of the total anterior-posterior dimension from the anterior surface at the fourth intercostal space.[40]

The changing relation between zero point and reference point

Even if clinicians use identical reference points, the vertical distance between the patient's zero point and reference point depends on which angle the patient is positioned in during examination, thus introducing another potential variable for interobserver disagreement. Some of this variation reflects a fluid-filled and mobile heart that actually drops in patients in upright position because of gravity. For example, Guyton found the right atrial pressure fell 0.7 to 1.0 cm H_2O with the hind end of the dog down compared with the supine position, but again fell 0.4 to 0.6 cm H_2O with the head end down.[21] If these changes were caused by Guyton's failure to locate the dog's zero point precisely, the pressure in the two positions would vary in opposite directions. Instead, the heart (and physiologic zero point) probably dropped with respect to the zero mark on the

Figure 1



A, T1-gated sagittal magnetic resonance image of a 43-year-old man, just to right of midline. *White cross* marks phlebostatic axis. **B**, Schematic drawings that illustrate changing vertical relations between heart and three external reference points as patient moves from supine (0 degrees) to semiupright (45 degrees) to upright (90 degrees). Only anterior thoracic wall, manubrium, intercostal spaces, and right atrium, adapted from magnetic resonance image, in **A** appear in drawings (*cross* in posterior right atrium marks phlebostatic axis). Three reference points are (1) point 5 cm posterior to anterior body wall at fourth intercostal space (*left, open bar*), (2) phlebostatic axis (*center, solid bar*), and (3) 5 cm below sternal angle ("method of Lewis," *right, stippled bar*). CVP at phlebostatic axis (12 cm H_2O) is assumed to remain constant in different positions, and right atrium is presumed to not drop from gravity in upright position. Height of vertical bar, labeled in centimeters of water above each bar, depicts measured CVP according to each external reference point.

manometer in both the head-end down and hind-end down positions but not in the supine or prone positions.

In addition, unless the external reference point exactly pinpoints the individual's zero point, simple geometry dictates that the vertical distance between the two will change in different positions. Fig. 1 illustrates these geometric relations in one patient, demonstrating that if the clinician uses the sternal angle as the reference point, the venous pressure will seem to fall 2 to 3 cm in the 45-degree position, compared with the supine and upright position, simply because the vertical distance between sternal angle and the physiologic zero point (right atrium) is greatest at 45 degrees (using method of Lewis in the example in Fig. 1, the measured CVP is 9 cm H_2O at 0 degrees, 6 cm H_2O at 45 degrees, and 10 cm H_2O at 90 degrees, even though the actual CVP at the physiologic zero point—the phlebostatic axis in this example—remains constant). Similarly, if the clinician chooses a reference point in the supine patient that is even with the fourth intercostal space but anterior to the zero point (e.g., 5 cm below fourth intercostal space), the venous pressure will seem to rise as the patient sits up (in Fig. 1, 8 cm H_2O at 0 degrees, 9 cm H_2O at 45 degrees, and 12 cm H_2O at 90 degrees).

In fact, the measured CVP does fall about 3 cm H_2O at 45 degrees compared with the supine position when the sternal angle is the external reference point.[41] Other studies show that the fall in CVP of the same individual, when measured supine and then semiupright (45 degrees), is 2 to 3 cm greater if the sternal notch is the external reference point instead of the midaxillary line at the fourth intercostal space (a point near the phlebostatic axis).[42,43] When the external reference point is 5 cm posterior to the fourth intercostal space, a site anterior to the zero point, the measured CVP does indeed rise as the patient moves from supine to upright.[44]

These data emphasize that the clinician's estimate of venous pressure, whether by physical diagnosis or catheter measurement, depends greatly on the external reference point selected and the angle used during the examination. This principle holds true even if the CVP at the zero point is perfectly stable in different positions, an assumption that, though proven in healthy individuals, may be erroneous in sick patients.

Bedside examination versus direct measurement of venous pressure

Clinicians can identify the jugular veins in 72% to 94% of patients[20,34] and measure the CVP with fair to moderate interobserver agreement (kappa statistic 0.3 to 0.65).[20] Table II presents data from the five clinical studies that compared direct measurements of CVP to clinicians' estimates from physical diagnosis. Importantly, the

Table II. Venous pressure: Bedside examination versus direct measurement

Reference	*n*	External refernce point: For the clinician	External refernce point: For catheter measurement	Findings*
Clinician's estimate vs catheter measurement				
Davison[16] (1974)	39	Method of Lewis	5 cm below sternal angle in supine patient	Difference in measurements†: <2 cm = 57% <3 cm = 93% <4 cm = 100%
Ducas[18] (1983)	25	Patient at 45 degrees, method of Lewis	5 cm below sternal angle in supine patient	Difference in measurements‡: <5.4 cm = 86%
Cook[20] (1990)	50	Not stated	Not stated	Correlation coefficient, bedside vs catheter value: 0.65 to 0.74
Clinician's accuracy in predicting venous pressures§				
Connors[17] (1983)	62	Not stated	Not stated	42.7% Predictions within measured range
Eisenberg[19] (1984)	103	Not stated	Midaxillary line	55% Predictions within 2.7 cm H_2O of measured range

*Values in centimeters of water.
†Only data from right internal jugular shown. Results from right and left internal and external jugular veins were similar. Regression equation: y = 0.64x + 1.54, where x is measured CVP and y is clinician's estimate from examination of the external jugular vein on expiration ($r = 0.77$).
‡Neck veins were not seen in 40% of patients positioned at 45 degrees; all these patients had measured values <7 cm or >26 cm, too low or too high to be visualized in this position. Mean error of clinician's estimate minus catheter measurement (± SD) = −0.5 ± 4.2 cm H_2O.
§Defined as low, normal, and high: <0 cm, 0 to 9.5 cm, and >9.5 cm (Connors), respectively, and <2.7 cm, 2.7 to 8.2 cm, and >8.2 cm (Eisenberg), respectively.

patients in these studies were probably among the most difficult to examine. Over 90% were in the intensive care unit, and many were on mechanical ventilators. Entry criteria included either the clinical need for right atrial catheterization (which tends to select unstable patients or those with confusing examinations because they have not responded to initial therapy)[17,19] or patients who already had an internal jugular catheter in place (which may obscure the neck veins during examination).[16,18,20] Nonetheless, these studies are relevant because the patients recruited are among those with the most pressing need for accurate measurements of CVP.

Four of the five studies concluded that bedside diagnosis was inaccurate and unreliable,[16,17,19,20] the clinician's estimate (in centimeters of water) being within only several centimeters of the measured value.[16,18] Results were similar whether the clinician had examined the external or internal jugular vein.[16] When clinicians were asked to assess the CVP as either low, normal, or high, they were accurate only about half of the time, although more so if the venous pressure was predicted to be high (77% to 80% accuracy) than low (3% to 38% accuracy).[17,36] Accuracy improved when mechanically ventilated patients were excluded.[17,20]

Why disagreement occurs

Table II reveals that only two of the five studies specified which reference point they used for both the catheter and bedside examination, and only one described the angle for the bedside examination—methods that, again, almost guarantee significant disagreement. It is very unlikely that the clinicians in these studies used the same technique, as illustrated in a different study in which experienced intensive care nurses identified their own favorite midaxillary or midthoracic reference point on the same individual[45]: the nurses' reference points differed by as much as 7 cm vertically, a discrepancy that surprisingly persisted even after attempts to standardize their technique.

For two of the studies[17,19] it is difficult to conceive how the clinician could ever distinguish low and normal CVP during examination, because low was defined as <0 cm H_2O in one study and <2.7 cm H_2O in the other, levels that actually make the jugular veins invisible to the examiner. Accordingly, the prediction might well have rested more on the patient's clinical presentation (e.g., hematemesis and shock) than actual inspection of the neck veins.

Other reasons for disagreement, although less significant, include the failure of two studies[16,20] to specify whether the CVP was measured electronically or by manometry. Manometric measurements are on average 2 cm higher, partly because of a meniscus effect and partly because of the difficulty of identifying the mean pres-

sure in the manometer's bobbing saline column.[46-48] In addition, because blood flows toward the heart, the jugular venous pressure (which the clinician examines) must exceed the right atrial pressure (which the catheter measures). On average, the right internal jugular venous pressure is 1 cm higher and the left 2 cm higher than the right atrial pressure.[49-52]

Most important is the observation that when disagreement occurred, the clinician tended to underestimate the measured value,[16,17,19,20,35] the difference being the greatest when the measured value was high.[16] Because the catheter measurements were always taken from supine patients, but physical diagnosis involved positioning the patient at whatever angle allowed best visualization of the neck veins, it is important to determine whether sick patients have the same postural regulation of venous pressure (within 1 to 2 cm H_2O) as normals.

Postural regulation of venous pressure in sick patients

The CVP significantly drops when patients with intravascular volume depletion or heart failure sit up. For example, in a study of 16 patients with volume depletion, the CVP fell almost 10 cm H_2O in the semiupright position (45 degrees) compared with the supine position.[41] This large drop in measured pressure is not entirely caused by varying vertical separation of the sternal notch (the reference point used) and the zero point, because in the same patients after fluid resuscitation but using identical methods the pressure fell only about 3 cm H_2O (i.e., that predicted in Fig. 1).

Similarly, in a study that used the phlebostatic axis as reference point in 110 subjects, the venous pressure did not change when normal adults and those with class I heart failure sat up but fell almost 8 cm H_20 when patients with class III to IV heart failure sat up.[24] Faced with severely orthopneic patients unable to lie down for supine measurements of venous pressure, other investigators have attempted to determine the upright venous pressure in patients with heart failure but have abandoned the effort because the postural drop observed sometimes brought the upright venous pressure into the normal range.[24,33]

These observations help explain why the clinician's estimate from physical diagnosis tends to underestimate the measured value. In patients with heart failure, for example, direct measurements revealing an elevated CVP in the supine position are being compared with clinicians' estimates obtained from a patient in a more upright position. This upright position allows visualization of the elevated jugular veins but also changes the vertical relation between the zero and reference points and may cause a postural fall in pressure.

The mechanism for this postural instability also helps explain two other physical findings involving jugular venous pressure, the abdominojugular test and Kussmaul's sign.

The abdominojugular test, Kussmaul's sign, and postural instability of venous pressure

During the abdominojugular test, the clinician presses firmly over the patient's mid-abdomen for 10 seconds, a maneuver that probably increases venous return by displacing splanchnic venous blood toward the heart. The CVP of normal individuals usually remains unchanged during this maneuver, rises for a beat or two, or even falls slightly.[18,53-55] If the CVP rises and stays elevated throughout the maneuver, the test result is positive, a result correlating with elevated right atrial pressure in some studies,[56] elevated left atrial pressure in others,[54] and the clinical diagnosis of heart failure in still others (sensitivity 0.24 to 0.73; specificity 0.96 to 1.0).[18,57] In many patients with a positive test result, greater abdominal pressure causes a greater increment in venous pressure.[58]

Kussmaul's sign is the paradoxic elevation of CVP during inspiration. Although classically associated with constrictive pericarditis, it occurs only in the minority of such cases[59,60] and is found in other disorders such as severe heart failure,[60,61] pulmonary embolus,[62] and right ventricular infarction.[63-65]

These two signs and the postural instability of venous pressure reflect a systemic venous system whose pressure is unusually sensitive to small changes in venous volume. In contrast to normal individuals, any increase of venous return in these patients—whether from abdominal pressure in those with a positive abdominojugular test, from inspiration in those with positive Kussmaul's sign, or from lying down from the upright position in those with heart failure or volume depletion—causes abnormal increments in venous pressure.

In hyperadrenergic states, such as volume depletion or heart failure, the systemic veins (especially of the extremities) are much more constricted and less distensible, reducing extremity blood volume and increasing central blood volume.[66,67] This basal venoconstriction, caused by sympathetic stimulation and the tissue edema

of heart failure,[66] is probably the main factor that elevates CVP in the first place. For example, in healthy individuals the CVP remains constant or changes minimally after rapid intravenous infusion of 1000 ml of saline,[68] yet venous pressure increases promptly, by almost 100%, after intravenous administration of norepinephrine.[69,70] In a study of patients convalescing from a myocardial infarction, repeated measurements of peripheral vein distensibility revealed an inverse relation between the supine CVP and compliance of the veins; the more distensible the vein, the lower the CVP.[71] In patients with hemorrhagic shock, there is a complete lack of relation between CVP and intravascular volume[72-74]; the normal or high CVP of some patients in shock presumably reflects intense peripheral venoconstriction.

A stiff, constricted peripheral venous system not only makes the supine CVP higher but also renders the CVP more sensitive to changes in venous return. Because venous return diminishes in the upright position, the CVP falls. This postural instability is only one example of how CVP depends on venous return. Exercise also increases venous return and causes exaggerated increments in venous pressure of patients with heart failure compared with healthy individuals.[56,66] Similarly, leg elevation fails to alter the venous pressure of normal individuals but increases it in those with heart failure.[56] Normal individuals have a positive abdominojugular test result after they receive a sympathomimetic agent,[75] and, conversely, patients with heart failure and a positive test result have a fall in venous pressure and a blunted increment during abdominal pressure after administration of a vasodilator.[76]

In patients with constrictive pericarditis the abdominojugular test result is also markedly positive in those with a positive Kussmaul's sign,[60] and the venous pressure rises promptly with leg elevation.[56] In patients with heart failure, Kussmaul's sign is positive in only those with the most markedly positive abdominojugular test.[60] Kussmaul's sign may actually represent an inspiratory abdominojugular test.[77] Whereas increases in intraabdominal pressure do not increase the CVP of normal individuals,[53,54] patients with constrictive pericarditis and Kussmaul's sign demonstrate a marked dependence of the two variables: removal of ascites causes the CVP to fall, and any measure that increases intraabdominal pressure—gentle abdominal compression, an abdominal corset, or a deep inspiration—causes the CVP to rise.[77]

Venoconstriction alone, however, does not explain why Kussmaul's sign occurs in most cases of right ventricular infarction[64,65] but relatively fewer cases of heart failure,[60] conditions that presumably share similar degrees of adrenergic tone. This finding suggests that the constraining effects of a normal pericardium on a dilated right ventricle (e.g., right ventricular infarct or severe congestive heart failure) or of a diseased pericardium on a normal-sized right ventricle (constrictive pericarditis), along with venoconstriction, are essential to Kussmaul's sign. Perhaps the fixed right ventricular distention of these patients eliminates the normal regulatory role the Starling cardiac function curves have on CVP: instead of augmenting ventricular distention and output, therefore, any increase in venous return only exaggerates the increment in CVP already caused by venoconstriction.

Conclusion

There are so many reasons for disagreement between direct measurements and the clinicians' estimates of CVP that trying to precisely compare the two—without first standardizing reference points and the angle of examination—is fundamentally unsound. Mackenzie and Lewis were probably correct when they advised clinicians to determine during physical examination only whether the patient's CVP was increased, thus avoiding decisions about degrees of elevation that are imprecise and difficult to reproduce.

How does the clinician know the CVP is abnormally elevated? Of all the suggested reference points (Table I), the sternal angle has the important advantage of being very easy to locate reproducibly during examination. More recent investigations have confirmed Lewis' original observation that the supine pressure of central veins (the subclavian or innominate vein or superior vena cava) lies at a height within 1 cm vertically of the sternal angle in most healthy individuals.[78] If the top of the neck veins is more than 3 cm above the sternal angle, venous pressure is abnormally elevated.[78] Although the vertical distance between the sternal angle and heart varies in different positions, this is not a disadvantage for the clinician trying to determine if the CVP is elevated because the only potential error is for the clinician to underestimate the measured supine value.

The phlebostatic axis is the landmark demonstrating the least postural variation of venous pressure in normal individuals, yet it is unlikely clinicians will consistently locate this landmark. Even using flexible right-angle triangles and a standard patient position, intensive care nurses trying to locate a similar point, the anterior axillary line at the fourth intercostal space, disagreed by several centimeters in both horizontal and vertical directions.[45]

For the unstable patient in the intensive care unit who has failed to respond to initial treatment, who requires repeated measurements of CVP, and in whom identification of a low CVP is important for management, direct measurements with a catheter are superior to physical examination (Table II). For the clinic or ward patient, however, with edema, dyspnea, or ascites—medical problems in which detection of elevated CVP has diagnostic importance—examination of the neck veins at the bedside is much more convenient and probably as useful. Further research must identify a value of venous pressure that is clinically useful in these patients, but in the meantime, Lewis' assertion that a measurement >3 cm H_2O above the sternal angle is abnormal, whatever the patient's position between supine and upright, seems to be a good starting point that would tend to only underestimate the measured supine value.

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