

REVIEW

Central venous pressure – evaluation, interpretation, monitoring, clinical implications

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Abstract: Physicians need to understand, evaluate and address hemodynamics in every patient and even more importantly in patients that are critically ill. Being able to determine and interpret central venous pressure is one of the most useful bedside evaluation skills, even in the 21st century (Fig. 3). Full Text (Free, PDF) www.bmj.sk.

Key words: central venous pressure, hemodynamic monitoring.

Central venous pressure (CVP) is a number that describes the pressure of the blood in the thoracic vena cava near the right atrium of the heart. It can be simplified that CVP equals right atrial pressure. CVP reflects the amount of the blood returning to the heart and the ability of the heart to pump the blood in the arterial system. It is a good approximation of right atrial pressure, which is a major determinant of right ventricular end diastolic volume and right ventricular preload. Change in CVP = change in VOLUME / change in venous COMPLIANCE. In other words CVP is increased by venous blood volume or by increase in venous tone (Fig. 1).

Some of the case scenarios for increased CVP are hypervolemia, forced exhalation (transient), being on ventilator (PEEP), tension pneumothorax, pleural effusion and heart failure. Decreased CVP is usually seen in hypovolemia, septic shock, deep inhalation (transient) and increased venous compliance. The inhalation/exhalation variance is even more important in patients with chronic obstructive pulmonary disease (COPD) or any patients that have to strain to exhale. Physicians should be aware that during exhalation in a patient with asthma or COPD the CVP will be elevated due to straining and should be evaluated during the inhalation phase of respiratory cycle during non-forced inhalation to avoid falsely elevated readings.

There are several waves on the CVP tracing that are related to the cardiac cycle (Fig. 2):

+ a wave: This wave is due to the increased atrial pressure during right atrial contraction. It correlates with the P wave on an EKG.

+ c wave: This wave is caused by a slight elevation of the tricuspid valve into the right atrium during early ventricular contraction. It correlates with the end of the QRS segment on an EKG.

- x descent: This wave is probably caused by the downward movement of the ventricle during systolic contraction. It occurs before the T wave on an EKG.

+ v wave: This wave arises from the pressure produced when the blood filling the right atrium comes up against a closed tricuspid valve. It occurs as the T wave is ending on an EKG.

- y descent: This wave is produced by the tricuspid valve opening in diastole with blood flowing into the right ventricle. It occurs before the P wave on an EKG.

Noninvasive measurement of CVP is quick, practical and useful, but not exact. The examiner should use the internal jugular vein when possible and be able to identify this structure based on the waveform of CVP as opposed to the waveform of the arterial pressure, which is different. It is important to distinguish that the venous waveform disappears upon gentle pressure on

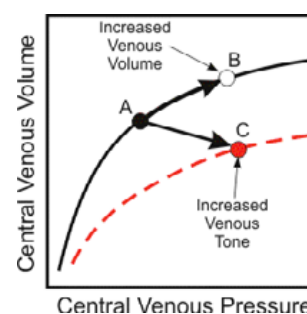


Fig. 1. Klabundle R: Cardiovascular Physiology Concepts. <http://www.cvphysiology.com>.

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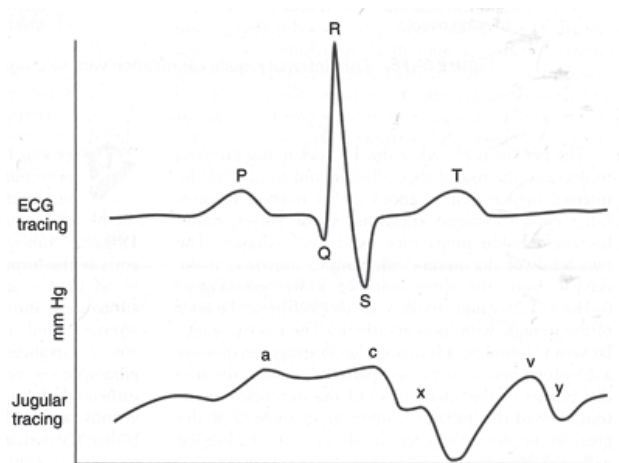


Fig. 2. <http://www.healthsystem.virginia.edu/internet/anesthesiology-elective/cardiac/cvcphys.cfm>.

the base of the neck (pressure generated with the back of the extended index finger just above the clavicle). The pulsation of the carotid artery persists, as the pressure in the carotid arteries is much higher as in the jugular veins. One can speculate that the distance between the right atrium and the sternal angle (angle between manubrium and corpus sterni) varies with position. For the purpose of the estimation of CVP it can be regarded as constant and to be 5 cm (Fig. 3). The examiner needs to know the anatomical location where to look. We should aim for the internal jugular vein but may use external jugular vein instead, as it is sometimes easier to spot. The difference is usually minimal but we should always look on both sides to make valid measurement. Positioning of the patient and proper light is critical. Start at 45° angle of the whole torso elevation (not only head and neck) and position as needed to achieve visualization for the CVP (if CVP is elevated may need to erect patient more, if it is decreased might need to flatten patient further). In a healthy individual at a 45° angle of the upper body the CVP will be approximately in the middle of the neck (distance between the clavicle and the mandible). This will be approximately 6–9 cm of water column, which ultimately equals the vertical distance in centimeters from the right atrium. Measurement consists of drawing a virtual horizontal line at the sternal angle and another one at the level of the CVP (upper level of the blood filling the jugular vein). The vertical difference between those 2 lines in centimeters should be added to the arbitrary distance between the right atrium and sternal angle (5 cm).

Cardiac function is determined by preload (CVP), afterload, heart rate and cardiac contractility. Being able to evaluate CVP at the bedside (at least estimate) is very useful skill and saves time as well as simplifies differential diagnosis process in various clinical situations. CVP evaluation puts light and sense to managing patients with indeterminate chest X-ray/radiographs (CXR) findings, elevated beta natriuretic peptide (BNP) levels, shortness of breath complaints and evaluating volume status. If

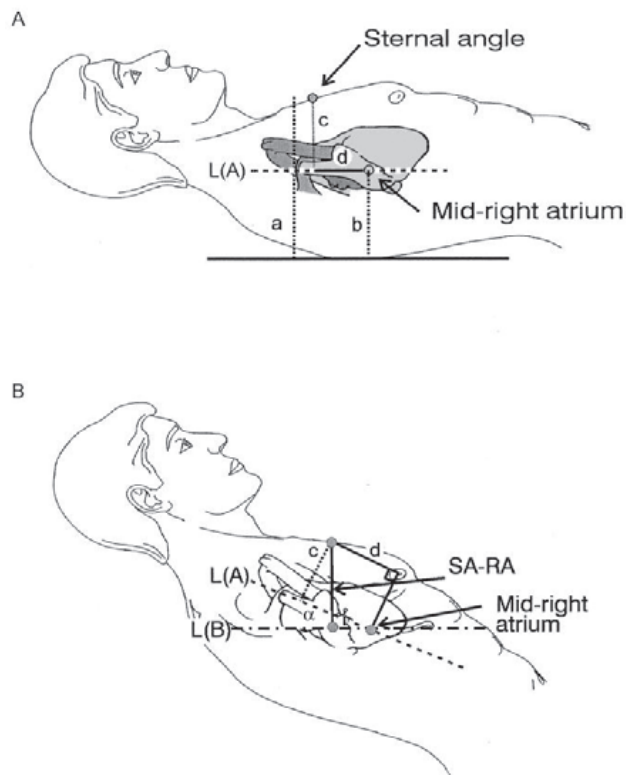


Fig. 3. Ratika S, Magner P, Matzinger F, Van Walraven C. How Far Is the Sternal Angle from the Midright Atrium? *J Gen Intern Med* 2002; 17 (11): 861–865.

patient has normal or high CVP it is very unlikely that he or she is having significant hypovolaemia. On the other hand if the CVP is low it is useful to perform orthostatic blood pressure measurements and after confirming severe hypovolaemia start aggressive volume resuscitation. Many times CXR is having diffuse interstitial infiltrates and differential diagnosis includes pulmonary congestion/edema. If patient has normal or low CVP this diagnosis can be excluded with very high level of confidence. Similar case applies to elevated BNP, marker widely used to confirm or rule-out congestive heart failure (CHF). In patients with normal or low CVP it is very unlikely that elevated BNP is due to CHF exacerbation, more likely it is due to different conditions such as pulmonary embolism (also creating heart strain) or renal insufficiency (BNP is cleared via kidneys). It is essential to be able to determine the volume status of patients presenting with common clinical problems such as hyponatremia and syncope. Patient with hyponatremia, hypovolaemia and low serum osmolality (hypovolaemic hyponatremia) who is on diuretics has most probably low sodium as a side effect of diuretics. The same goes for syncope in such patients, as it is very common that overdiuresed patients experience orthostatic syncopal spells. In cases with low-normal or decreased CVP it is very useful to use orthostatic blood pressure measurements.

When should CVP be measured exactly using invasive methods? In patients with hypotension who are not responding to

initial intravenous fluid resuscitation, in continuing hypovolaemia secondary to major fluid shifts or loss and in patients requiring infusions of inotropes or vasopressors. Those are the patients usually admitted to critical care units. Exact measurement of CVP involves inserting central venous catheters and using hemodynamic monitors. Catheters are usually introduced either in subclavian or internal jugular vein with the tip positioned in vena cava just before right atrium. CVP, measured invasively, is sufficient to guide fluid therapy in the majority of patients (may elect to use Swan-Ganz catheter but there is conflicting evidence in recent literature). It is hard to set normal or average CVP number. The measurement is determined by venous return, influenced by intrathoracic pressure and right ventricular compliance. Complete heart block, atrial fibrillation, tricuspid stenosis and regurgitation will lead to an inaccurate reading, although the diagnosis of these disorders can be made from the CVP waveform (Fig. 2).

Central venous pressure should be regarded as a trend. Multiple fluid boluses might be necessary before the CVP reaches the target range and patient can be switched to a maintenance fluid infusion. In patient with sepsis it is imperative to replenish vascular volume sufficiently before starting vasopressors. It is not uncommon to volume under-load and under-resuscitate pa-

tient to a target CVP. "Normal CVP" = 5–10 cmH₂O. While treating septic patients clinicians should use ~10 cmH₂O in non-ventilated patient, and ~15 cmH₂O, if the patient is on positive pressure ventilation (CPAP, BiPAP). If there is a question of cardiac disease, cardiac hypertrophy or dilatation or if the patient is older, aim higher ~15plus cmH₂O. In many young patients, it is often not possible to raise the CVP above 10 cmH₂O because of high efficiency of the cardiovascular system.

In conclusion, it is imperative in today's practice of medicine to be time efficient and to use modern diagnostic methods. For example, diagnosing a cause of a cardiac murmur based on auscultation alone is almost unheard of in current practice and every patient with such problem is evaluated with echocardiography, which is very appropriate. On the other hand it is essential not to forget the "art of medicine" and the bedside skills. Measuring CVP (noninvasive) is a good, simple, reliable bedside test that saves time and helps interpret different and sometimes conflicting findings. Evaluating and addressing hemodynamics (invasive monitoring) is essential in majority of patients hospitalized in critical care units. Aim of this article was to emphasize importance of hemodynamic monitoring in clinical practice as well as to motivate the audience to "brush-up" on this essential and very valuable skill.

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