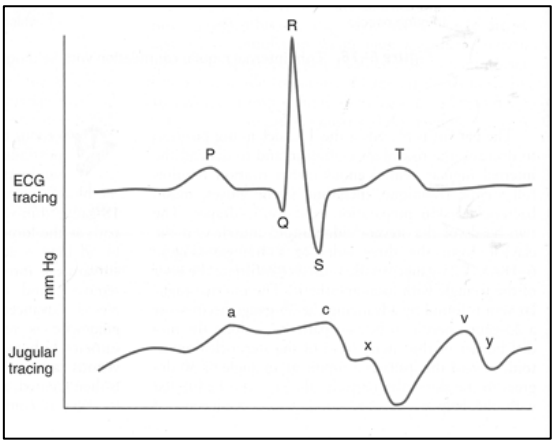
CVP Interpretation

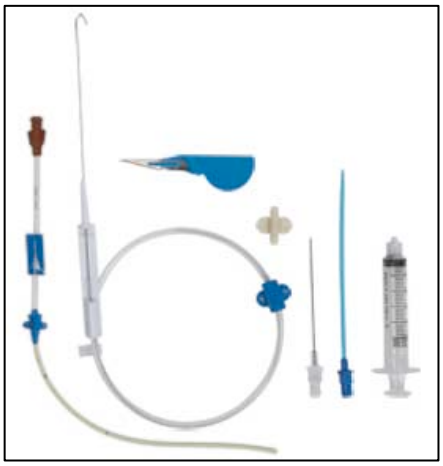
Anesthetic Pearls: Anesthetic Interpretation and Management of the CVP Trace



Central lines are used for many surgeries that require large vascular access, sizeable fluid shifts, access for blood tests / analysis, and approach to the heart for PA catheter placement. The interpretation of CVP tracing is essential to the management of various cardiopulmonary pathologies. The most important technical consideration for accurate recording of the CVP is determining the proper external reference level for the pressure transducer and the midchest / mid-atrial position is commonly chosen.

CVP Interpretation:

1. CVP waves:
   - "**a**" wave results from right atrial contraction
   - "**c**" waves result from tricuspid valve closure and isovolumic right ventricular contraction
   - "**v**" waves result from ventricular ejection, which drives venous filling of the right atrium



2. Pressure, Flow, and Volume – The CVP trace reflects cyclic changes in right atrial pressure (<u>not</u> volume). Right atrial pressure rises because of increasing volume at a constant chamber stiffness ("**v**" wave), or increasing chamber stiffness at a constant or decreasing chamber volume ("**a**" wave).

3. Wave Summation and Extra Peaks – The three distinct CVP peaks ("**a**", "**c**", "**v**") and two troughs ("**x**", "**y**") are discernible in the normal venous pressure trace, these characteristic classic features are altered by changes in heart rate and conduction abnormalities. A short PR interval on the EKG causes fusion of "**a**" and "**c**" waves, and tachycardia reduces the duration of diastole leading to an abbreviated "**y**" descent and thereby causing "**v**" and "**a**" waves to merge.

4. CVP-Cardiac Cycle Relationship: CVP waves refer to the time in the cardiac cycle when the wave begins, rather than when the wave reaches its peak.

CVP Diagnostic Details:

A. Arrhythmias: In atrial fibrillation the "**a**" wave disappears and the "**c**" wave becomes more prominent, since atrial volume is greater at end-diastole and onset of systole, owing to the absence of atrial contraction.

B. Tricuspid Valve Disease: A broad, tall systolic "**c-v**" wave is described, which begins in early systole and obliterates the systolic "**x**" descent in atrial pressure.

C. Right Ventricular Ischemia and Infarction: These conditions cause systemic arterial hypotension and disproportionate elevation of CVP compared to PA wedge pressure. Often the CVP will exceed the wedge pressure and display prominent "**a**" and "**v**" waves, the former resulting from atrial contraction into a stiff right ventricle and the latter resulting from tricuspid regurgitation.

D. Pericardial Constriction: The restraining pericardium limits venous return to the heart, which reduces stroke volume and cardiac output. Central venous pressure is elevated, there is <u>end-diastolic pressure equalization in all cardiac chambers</u>, and the trace resembles that seen with right ventricular infarction.

E. Cardiac Tamponade: Increased pericardial fluid pressure couples changes in atrial and ventricular volume. As a result, total cardiac volume does <u>not</u> change during diastole when blood is transferred from atrium to ventricle, and the CVP becomes monophasic and displays a single prominent "**x**" descent and an attenuated or absent "**y**" descent.

F. Respiratory Influences: During spontaneous breathing, inspiration causes a decrease in pleural and pericardial pressures that is transmitted to the right atrium and produces a decline in transduced CVP.