“x” descent of CVP: An indirect measure of RV dysfunction?

Sir,

A 38-year-old female with severe aortic stenosis and mild pulmonary hypertension was scheduled for an aortic valve replacement. It was noticed on the continuous central venous pressure (CVP) monitor, that the “x” descent of the CVP waveform did not decline as steeply in the postbypass phase as it did in prebypass phase [Figures 1 and 2]. Additionally, intraoperative TEE examination of the tricuspid valve revealed a Tricuspid annular plane systolic excursion (TAPSE) of 18 mm and 12 mm in prebypass and postbypass period, respectively, with no tricuspid regurgitation.

TAPSE was obtained by visualizing the lateral edge of the tricuspid annulus in the deep trans-gastric view and using M-mode to measure movement of tricuspid annulus during the cardiac cycle. TAPSE is used routinely as a simple method of estimating RV function, with a lower reference value for impaired right ventricular (RV) systolic function of 16 mm [1] [Figure 3]. The TEE finding observed in this case suggested post bypass RV dysfunction.

The CVP waveform, when interpreted correctly, can have meaningful clinical implications. A typical CVP waveform consists of five phasic events, three positive waves (a, c and v) and two negative deflections (x and y descent). Each wave corresponds to certain mechanical cardiac event [2-4] [Figure 4].

The a wave is the first positive deflection as a result of the atrial contraction which occurs at end diastole and follows the p wave on the electrocardiogram. The C wave is second positive deflection as a result of tricuspid bulge in atrium due to isovolemic contraction of ventricle and coincides with QRS complex of electrocardiogram. The X descent is due to downward movement of the lateral annulus of the tricuspid valve during RV contraction and atrial relaxation. It coincides with ST segment on electrocardiogram. The V wave is third positive deflection due to rapid atrial filling when tricuspid valve is closed in ventricular systole and follows the T wave of electrocardiogram. The Y descent is due to rapid ventricular filling as tricuspid valve opens in early diastole and coincides with TP segment of electrocardiogram [Figure 5]. Central venous pressures is reliably measured at end-expiration on a calibrated monitor screen or paper recording. Interpretation of CVP waveforms can indicate

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**Figure 1:** Prebypass CVP waveform (blue) x descent marked with arrows

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**Figure 2:** Postbypass CVP waveform (blue) X descent marked with arrows. Steepness of x descent is decreased compared to prebypass

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**Figure 3:** Schematic diagram showing TAPSE measurement

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**Figure 4:** CVP waveforms with corresponding cardiac events and ECG
underlying disease process. In junctional rhythm, there is delayed retrograde atrial depolarisation. This delay may cause RA to contract against closed tricuspid valve, creating large cannon a wave. Tricuspid regurgitation may give rise to large CV complex with disappearance of x descent, as pressure generated during RV contraction is detected at the catheter tip.

X descent is determined by right atrial and right ventricular function, volume loading conditioning and pericardial forces. In the present case, volume loading conditions were in the same range in both phases as assessed by transesophageal echocardiography. The transducer system was properly damped to rule out any effect of overdamping on pressure waveform by doing fast flush test. Pre-CPB and post-CPB heart rates were comparable and rhythm was normal sinus. So there was minimal effect of heart rate variation on CVP waveform. Generally, tachycardia reduces the length of diastole and parts of the CVP pressure waveform merge, while bradycardia causes each wave to become more distinct. Also, because the pericardium was open, so there was no effect of pericardial force on chamber pressure. Hence, anything affecting RV systolic function will also affect “x” descent wave. In this case, decreased slope of “x” descent may be due to RV dysfunction which might be the result of poor RV protection during CPB or RV strain because of increased afterload [Figure 6]. While maintaining acid-base balance and adequate arterial pressure, dobutamine was started at 3-5 μg/kg/min which improved RV function over a period of time. The slope of the “x” descent in CVP gradually recovered to baseline and TAPSE reverted to its preoperative range. As the RV function improved postoperatively, the patient’s hemodynamics gradually got stabilized.

In this case, we observed that slope of “x” descent in CVP correlated well with RV function after ruling out other affecting factors. However a precise monitoring grid is needed to measure the timing and slope of “x” descent. Further research is needed to better understand the relationship between qualitative and quantitative measurements of RV function as related to CVP waveforms.

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