A 71-year-old male patient underwent aortic valve replacement (Mitroflow bio prosthesis) and mitral valve replacement (St Jude Epic bio prosthesis) because of heart failure due to severe aortic valve stenosis. The medical history included hypertrophic cardiomyopathy and IGA nephropathy with end stage renal disease for which he was treated by continuous ambulatory peritoneal dialysis (CAPD). During the operation the patient was haemodynamically unstable because of stunning due to pre-existent heart failure with diffuse myocardial damage (postoperative serum creatine kinase MB fraction maximum 150 U/l, without ischaemic ECG changes). He was treated with dobutamine en norepinephrine. In the ICU, dobutamine was replaced by milrinone to reduce the risk of diastolic dysfunction in hypertrophic cardiomyopathy. In the ICU the cardiac output was 2.5 litres per minute. The central venous pressure (CVP) was 6 mmHg, the pulmonary artery pressure (PAP) was 50/20 mmHg and the pulmonary artery occlusion pressure (PAOP) was 18 mmHg. The patient gradually stabilized and on day four postoperatively we tried to create a negative fluid balance by the use of continuous haemodiafiltration (CVVHDF). This, however, led to renewed haemodynamic instability on which a fluid challenge of 500 ml was given in 30 minutes. During this fluid challenge the following curves were noticed on the monitor. It appeared that the CVP curve had significantly changed from a normal venous pressure pattern to the pattern showed in Figure 1.

What is your diagnosis?

**Diagnosis**

For a differential diagnosis we thought of right ventricular volume overload with tricuspid regurgitation due to volume overload, suggested by, what we first thought, prominent v-waves in the CVP-curve. However, this diagnosis had to be rejected since the patient had atrial fibrillation; a-waves should not be present and, with tricuspid regurgitation the x descent should not be apparent. Moreover, closer examination of the CVP curve showed that the pattern did not fit in at all with any known physiologic or pathologic state.

We discovered that the volume resuscitation had been performed with a volumetric infusion pump (Braun Infusomat) through the CVP lumen, with a rate of 999 ml/hour.

The discontinuous flow pattern of the infusion pump created a pulsatile pattern in the CVP curve. After discontinuation of the volume resuscitation, we observed a prompt normalization of the CVP curve (Figure 2).

**Conclusion**

Volume resuscitation administered with a volumetric pump may interfere with CVP measurements and may be mistaken for tricuspid insufficiency. Close examination of the CVP curve in search of a- and v - waves may reveal the true diagnosis.