Cardiogenic shock

Rapid evaluation of hemodynamics is carried out in every emergency department in the world every single day. However, this usually consists of looking at general parameters such as blood pressure, pulse rate and perhaps oxygen saturation. Some clinical evaluation of perfusion may also be made, but how much better would it be if we knew exactly what the hemodynamics were? Because of the non-invasive nature of the USCOM 1A, and the speed with which such data can be acquired, the USCOM 1A is beautifully suited to the emergency environment. Let's take a look at a case that was presented in our own emergency department and see just how the USCOM 1A improves clinical management of the patient.

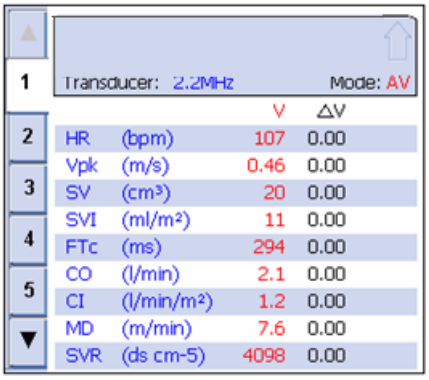
Presentation

Male, 68 years old, 76kg / 167lb. Forty minutes prior to admission he suffered acute onset of severe central chest pain and dyspnea. He had a past history of hypertension and angina. ECG shows antero-lateral ST elevation.

Observations:

- BP 96/53
- Pulse 108
- Respiratory Rate 32
- JVP clinically elevated
- SpO_2 86% on 10 l/min O_2
- He was confused and agitated
- Arterial gas analysis showed PaO_2 52, $PaCO_2$ 28, pH 7.18, Lactate 18
- CXR showed florid changes of pulmonary edema bilaterally

USCOM 1A Examination



The most obvious finding here is that his Cardiac Output (CO) and Cardiac Index (CI) are both low. The minimum Cardiac Index (CI) we should look for in a patient is 2.4 l/min/m^2. Clearly this patient's CI is well

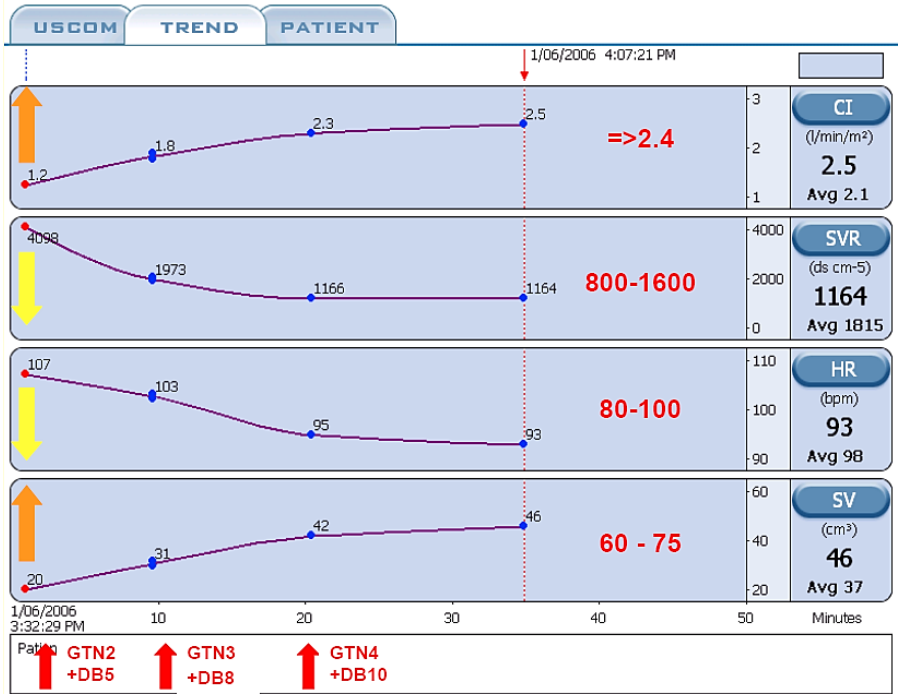
below this value. His Stroke Volume (SV) and Peak Velocity (Vpk) are both low, and his Systemic Vascular Resistance (SVR) is markedly raised.

What's going on here?

His myocardium is incapable of producing an adequate SV, and the low Vpk suggests a very low myocardial contractility (inotropy) status. His peripheral circulation is responding to the low CO by vasoconstriction, resulting in a high SVR of nearly four times that of normal. As a result of this, the blood flow in his aorta is much slower than normal as indicated by his Minute Distance (MD) (MD normal is 14-22 m/min). It's clear that this is a hypodynamic circulation.

But what is actually killing the patient? On a superficial level we might answer "cardiogenic shock" but what do we actually mean by this? We know that shock is any 'hemodynamic derangement leading to inadequate perfusion and oxygenation of the tissues'. So what then is this patient's oxygen delivery (DO_2)?

When we input the Hb and CVP we find that the DO_2 is only 372 ml/min (DO_2 normal is 667-923 ml/min). For a man of his size, a figure of even twice this value would be only just adequate.



Normal indexed values for a man of this age are what we should aim for and are in effect, our early goals in therapy. His CI is 1.2 and must be increased; his SVR is > 4,000 and must be reduced; his heart rate (HR) should be reduced and his SV needs to increase significantly.

So how did we treat him?

GTN2 and DB5 refer to glyceryl trinitrate infusion at 2 mcg/kg/min and dobutamine 5 mcg/kg/min.

Why did we choose these agents?

The USCOM 1A showed that his CO is inadequate and from clinical observation and his chest X-ray it is clear that his preload is already very high.

We urgently need to off-load him. Nitrates achieve this more rapidly than anything else. But why did we choose dobutamine? Well he needs one or other inotrope to increase his CI, but in the presence of a low blood pressure many people would opt for dopamine or norepinephrine or even perhaps an epinephrine infusion. However, the USCOM 1A shows that this is not appropriate as his SVR is very high. We need to vasodilate his arterial tree (reduce his afterload) if we hope to increase his SV, given that his myocardial contractility is low. The GTN will help a little but the most logical inotrope to use is dobutamine because of its vasodilator properties.

Repeat USCOM 1A shows that his SVR, SV, CI and HR are all going in the right direction. The infusions are then increased to 3 and 8 mcg/kg/min. Again, repeat measurement shows that we are making good progress.

Finally, the infusions are increased to 4 and 10 mcg/kg/min. Following this, we have achieved our early goal in terms of his overall hemodynamics.

His cardiogenic shock was achieved in just 35 minutes as the time scale on the trend screen shows. This treatment was carried out in the Emergency Department. By the time the patient was transferred to the Coronary Care Unit his immediate problem had already been solved.

His vital signs, laboratory results and radiology two hours post admission are interesting.

- BP 108/64
- Pulse 74
- SpO_2 96% (on 4 l/min O_2)
- CI = 2.8 l/min/m^2, SVR = 1082, SV = 66ml
- PaO_2 = 93, $PaCO_2$ = 35, pH = 7.38, Lactate = 1.6

His DO_2 is now 926 ml/min, an increase of 249%!

Over the next few hours his pulmonary edema resolved completely. Coronary angiography showed triple vessel disease which was not amenable to stenting. He subsequently underwent CABG x 3, on the 7th day after admission. He made an uneventful recovery and was discharged on the 21st day after presentation with no symptoms. At 6 month follow up he remained well with no angina.

Summary

This case shows that early diagnosis of a hemodynamic derangement, and rapid and appropriate treatment can be easily managed in the Emergency Department.