

USCOM in the Emergency Department: Clinical Case 1 Chest Pain? Cardiac Shock











The measure of life.

Rapid evaluation of haemodynamics is carried out in every emergency department in the world every single day. In the main however, this usually consists of looking at some 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 haemodynamics were doing? Because of the non-invasive nature of the USCOM, and the speed with which such data can be acquired, the USCOM 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 improves clinical management of the patient.

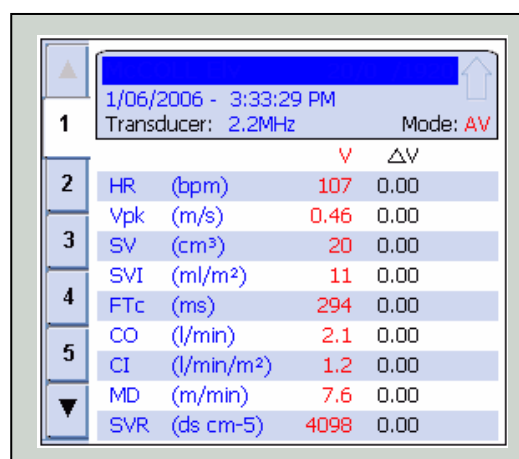
Male, 68 years old, 76kg. Acute onset of severe central chest pain and dyspnoea 40 minutes prior to admission. He had a past history of hypertension and angina. ECG shows ST elevation antero-laterally.

Observations:

-  BP 96/53
-  Pulse 108
-  Resp Rate 32
-  JVP clinically elevated
-  SpO₂ 86% on 10 l/min O₂
-  He was confused and agitated
-  Arterial gas analysis showed PaO₂ 52, PaCO₂ 28, pH 7.18, Lactate 18
-  CXR showed florid changes of pulmonary oedema bilaterally

This is the USCOM data screen.

What can you see?



		V	ΔV
1	1/06/2006 - 3:33:29 PM Transducer: 2.2MHz Mode: AV		
2	HR (bpm)	107	0.00
	Vpk (m/s)	0.46	0.00
3	SV (cm ³)	20	0.00
	SVI (ml/m ²)	11	0.00
4	FTc (ms)	294	0.00
	CO (l/min)	2.1	0.00
5	CI (l/min/m ²)	1.2	0.00
	MD (m/min)	7.6	0.00
	SVR (ds cm-5)	4098	0.00



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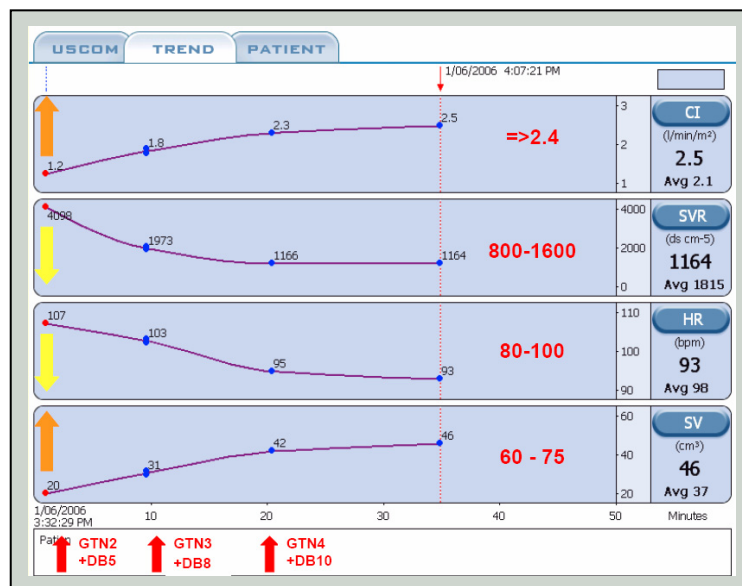
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The most obvious finding here is that his Cardiac Output and Cardiac Index are both low. The minimum Cardiac Index we should look for in a patient is 2.4 l/min/m². Clearly this patient comes nowhere near this. His Stroke Volume is low, his Peak Velocity is low, and his SVR is markedly raised. What's going on here?

His myocardium is incapable of producing an adequate Stroke Volume, and the low Peak Velocity suggests a very low myocardial contractility (inotropy) status. His peripheral circulation is responding to the low Cardiac Output by vasoconstriction, giving him a high SVR of nearly four times normal. As a result of this, the blood flow in his aorta is much slower than normal as indicated by his MD (the 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 "haemodynamic derangement leading to inadequate perfusion and oxygenation of the tissues". So what is this patient's oxygen delivery?

To calculate this we use the Oxygen Delivery formula. When we plug in the numbers in this case we find that the Oxygen Delivery is only 372 ml/min. For a man of his size, a figure of even twice this value would be only just about enough.



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The normal values for a man of this age and size are what we should aim for and in effect, our early goals in therapy. His Cardiac Index at 1.2 must be increased; his SVR at over 4,000 must be reduced; his heart rate should be reduced and his Stroke Volume 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 at 5 mcg/kg/min. Why did we choose these agents?

The USCOM shows that his Cardiac Output is inadequate and from clinical observation and from his chest X-ray it is clear that his preload is already very high.

We need to off-load him urgently. Nitrates achieve this more rapidly than anything else. But why did we choose dobutamine? Well he needs one or other inotrope to increase his Cardiac Index, but in the presence of a low blood pressure many people would opt for dopamine or noradrenaline or even perhaps an adrenaline infusion, but the USCOM shows that this is not appropriate. His SVR is so high that we need to vasodilate his arterial tree (reduce his afterload) if we hope to increase his Stroke Volume, 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 shows that his SVR, Stroke Volume, Cardiac Index and heart rate 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 haemodynamics.

As the time scale on the trend screen shows, the treatment of his cardiogenic shock was achieved in just 35 minutes and this 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. The importance of diagnosing the haemodynamic problem and treating it appropriately and rapidly is obvious.










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

-  BP 108/64
-  Pulse 74
-  SpO₂ 96% (on 4 l/min O₂)
-  CI = 2.8 l/min/m²
-  SVR = 1082
-  SV = 66ml
-  PaO₂ = 93, PaCO₂ = 35, pH = 7.38, Lactate = 1.6

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

Over the next few hours his pulmonary oedema resolved completely.

Coronary angiography showed triple vessel disease, 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.



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