

Clinical Case 3: USCOM in the Intensive Unit – Case Study



Post-op hypotension and ischaemia? Hypovolaemia? Septicaemia? Cardiac?

In intensive care, we have to look at the patient's cardiopulmonary system as a whole rather than looking at individual parameters; it is all too easy to focus on one parameter but miss the big picture.

In this case, a 46 year old male, 118 kg, type 1 diabetic was admitted to ICU with hypotension (85/40) following incision and drainage of a large axillary abscess under general anesthesia. His ECG showed ST depression in the anterior and lateral chest leads, with a normal heart rate and normal conduction. Here are his admission haemodynamics.

The image shows a screenshot of a medical monitor displaying haemodynamic data. The screen has a blue header bar with the date and time '28/03/2007 - 11:16:37 AM' and a home button icon. Below the header, there are two main sections. The first section, labeled '1', shows 'Transducer: 2.2MHz' and 'Mode: AV'. The second section, labeled '2', is a table of haemodynamic parameters. The table has three columns: the parameter name and units, the current value, and a change value (ΔV). The values are: HR (bpm) 67, Vpk (m/s) 2.8, SV (cm³) 260, SVI (ml/m²) 136, FTc (ms) 559, CO (l/min) 17, CI (l/min/m²) 9.1, MD (m/min) 49, and SVR (ds cm-5) 181. All values are in red text.

		V	ΔV
1	28/03/2007 - 11:16:37 AM		
	Transducer: 2.2MHz		
	Mode: AV		
2	HR (bpm)	67	0.00
	Vpk (m/s)	2.8	0.00
	SV (cm³)	260	0.00
	SVI (ml/m²)	136	0.00
	FTc (ms)	559	0.00
	CO (l/min)	17	0.00
	CI (l/min/m²)	9.1	0.00
	MD (m/min)	49	0.00
	SVR (ds cm-5)	181	0.00

So what are the numbers telling us? The most obvious finding is the cardiac output of 17 L/min, with a cardiac index of 9.1 (normal is 2.8-3.5 at this age). This strongly suggests that the hypotension is not due to primary cardiac failure! However, with his low diastolic blood pressure (which determines myocardial perfusion) and such a high CO it is not surprising that his ECG shows ischaemic changes.



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Perhaps the next question is why is he so hypotensive when he has such a high CO? The answer lies in the first law of haemodynamics; $BP = CO \times SVR$. His CO may be very high but his SVR at just 181 is very low indeed. His CO is over 3 times normal but his SVR is only one sixth of normal. The product of CO x SVR is therefore only just over half of the normal value, so he is hypotensive.

The FTc of 559 shows that he is either well pre-loaded, indeed it suggests he is fluid overloaded. The Ftc is also high in left ventricular failure, but that hardly seems likely here. The MD, which is the mean aortic flow velocity, shows that this is a very hyperdynamic circulation.

He is clearly septic with peripheral vascular collapse. The heart has increased its output to maximum, but this is just not enough to overcome the degree of peripheral vasodilation.

As regards treatment, whilst the first thought might be to just use a vasoconstrictor (pressor) agent, you must always beware of underlying myocardial depression which is often found in septicaemia. Whilst the patient is this vasodilated, there may not seem to be much evidence of this, but as the SVR rises and the afterload that the heart must work against increases, so the true state of myocardial function may become apparent. Consider dopamine or noradrenaline as the situation unfolds, and repeat the USCOM regularly!

As a final observation, why is his heart rate only 67? He is a type 1 diabetic so it is unlikely to be due to a β -blocker. In fact, this is often a sign of myocardial depression and is not uncommon in septicaemia. A similar pattern may also be seen in pancreatitis, ketoacidosis, envenomation and poisoning, amongst others, where latent myocardial depression may be hidden below a vasodilated vascular state.

