Venous return: cardiomythology?

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'I just can't understand how they can be so muddled about the heart,' laments the cardiologist. 'They're bright students who enjoy cardiovascular physiology, but ask a few basic questions and they tie themselves in knots. What's gone wrong?'

Most of us who have to teach cardiovascular control will probably share this concern and probably also agree about the origin of the muddle; the conventional explanation of the circulation that assigns fictitious roles to vaguely defined variables. The main offender is 'venous return'.

No one would wish to deny the fundamental part played by the venous side in cardiovascular control. Inadequate cardiac output leads to venous excess,[1] an accumulation of blood at the input side of the heart that is then automatically dispelled by an increase in stroke volume. This is an almost perfect example of a classic direct negative feedback system, the error signal being accumulating venous volume arising from a discrepancy between what is being supplied to the heart and what it is actually pumping out. As Starling's experiments originally showed, this error signal stimulates its own reduction. An excess may be pathological in origin, as when cardiac output is reduced by an ischaemic event, or physiological, as when hypovolaemia or exercise triggers sympathetic venoconstriction propelling blood centrally. In either situation the increase in cardiac output reduces the venous excess that represents the error, and homeostasis – at least, of the heart – is achieved. So what harm does it do to talk about 'venous return' in this context?

The first problem is knowing what 'venous return' actually *is*. Is it a volume? Or a flow, like cardiac output? Most students opt for the latter, and have a vague feeling that a high level of venous return, in the sense of increased venous flow into the heart, will in itself drive cardiac output. Except in the very short term, however, venous return is necessarily the same as cardiac output: the heart can only pump out what it is given, and take in what it has expelled. The failing heart has both low output and input, as does the heart in a hypovolaemic circulation. Because it is not an error signal, venous return causes nothing: essentially synonymous with cardiac output, it is a redundant and misleading expression that encourages vagueness and confusion.[2] This is not a mere semantic quibble: loose phraseology reflects sloppy thinking.

The other cause of confusion is that venous return is so often introduced in the context of a misleading picture of the physical properties of veins. Students usually grasp the arterial side, where flow is essentially governed by laws of pressure difference and resistance that are closely similar to the laws of current flow in electrical circuits, which they are more or less comfortable with. $V = I \times R$ (voltage = current × resistance) translates directly into $MAP = CO \times TPR$ (mean arterial pressure = cardiac output × total peripheral resistance). So constricting arterioles is like increasing electrical resistance: for a given flow, the pressure must rise. Unfortunately, this is not how veins behave. Veins are predominantly capacitance vessels: as with electrical capacitors, pressure (voltage) is a function of the volume (charge) they hold. Whereas altering the tone of arterioles mostly affects resistance, in veins it mostly affects capacity, and it is volume rather than resistance that is controlled to regulate the circulation.[3] In this respect the venous side of the circulation is therefore physically completely different from the arterial: it is volume and pressure that dominate, not flow. This means that we need to think about the control of the circulation in terms of venous accumulation, not venous return.

Actually the more thoughtful student soon realises there is something fundamentally wrong with the conventional account. How, for instance, can nitrates possibly rescue the failing ventricle from its excessive preload? According to the conventional view, by causing venodilation and reducing venous resistance they should increase rather than decrease venous flow. And how can sympathetic venoconstriction – which ought to cause an increase in resistance with reduction in flow – possibly stimulate the heart to deal with a hypovolaemic circulation? But as soon as one realises veins are primarily capacitance vessels, the answers are clear. Nitrates increase venous capacity, allowing a greater volume of blood to reside in this part of the circulation, and reducing the volume of blood distending the cardiac chambers. Sympathetic activity reduces the volume of blood stored in the veins and shifts blood towards the heart. This creates venous excess, stretching the myocardium and augmenting cardiac

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Clin Med 2007;7:35–6

output until a new equilibrium is reached. Like power-assisted steering, the Starling mechanism simply provides the leverage that translates small alterations in venous sympathetic activity into powerful effects on stroke volume.

Our experience suggests that encouraging students to use the concept of venous excess has a dramatic effect in helping them to understand how the venous side contributes to cardiovascular homeostasis, thinking, as Starling did, in terms of volume and distension rather than venous flow.[4] Venous excess is a genuine error signal whose regulatory action is firmly grounded in experiment; it avoids the vague, essentially mythical concepts such as mean systemic filling pressure and venous return that cause students such confusion. Furthermore, venous excess is a tangible error signal the clinician can observe directly in the jugular vein.[5] After all, fluid management is performed with at least moderate success in the wards despite the absence of a venous returnometer.

In conclusion, we should resolve to hold firmly to experimental observations, and replace mythical abstractions with tangible clinical measurements. We can then give students an account of cardiovascular homeostasis that is not just correct, but easy to apply in the real world.

References

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