VENOUS EXCESS

"The road of excess leads to the palace of wisdom"
(William Blake, Proverbs of Hell).

Reddi & Carpenter's venous excess model of cardiac output

DR EUGENE'S CONGRESS

Dr E. is just back from the ICM Congress. Dr E. especially loved making new friends and drinking the local beer with them ('networking'). A guy on the podium had said that central venous pressure is a hopeless guide to the adequacy of the circulation.

- What is the role of CVP in haemodynamic physiology?

The short answer is that the pressure in the central veins is the effective weight of the venous excess volume from which the right atrium is filled during diastole. The trouble is that Dr E had not been to Cambridge University and so had not been taught the concept of venous excess.

- So please explain the venous excess paradigm!

THE ARTERIAL SIDE

Let's start with self evident and unarguable fact that the cardiac output is the product of stroke volume and heart rate;

- $CO = SV \times HR$

The often-measured arterial blood pressure is a product of CO and vascular peripheral resistance;

- AP = CO x PR

Reflect on these simple formulae;

- CO = SV x HR
- AP = CO x PR

They suggest causal relationships, but they don't tell you the direction of causality. For instance, we would not presume a rise in ABP implies a rise in Qt. We apply some physiology.

On the arterial side of the circulation there is a negative feedback loop regulating AP via arterial baroreceptors that modulate brain centres that control HR (the vagal brake) and the general sympathetic (predominantly vasoconstrictor & positive chronotropic) tone.

- let's see that in diagrammatic form (the red box);

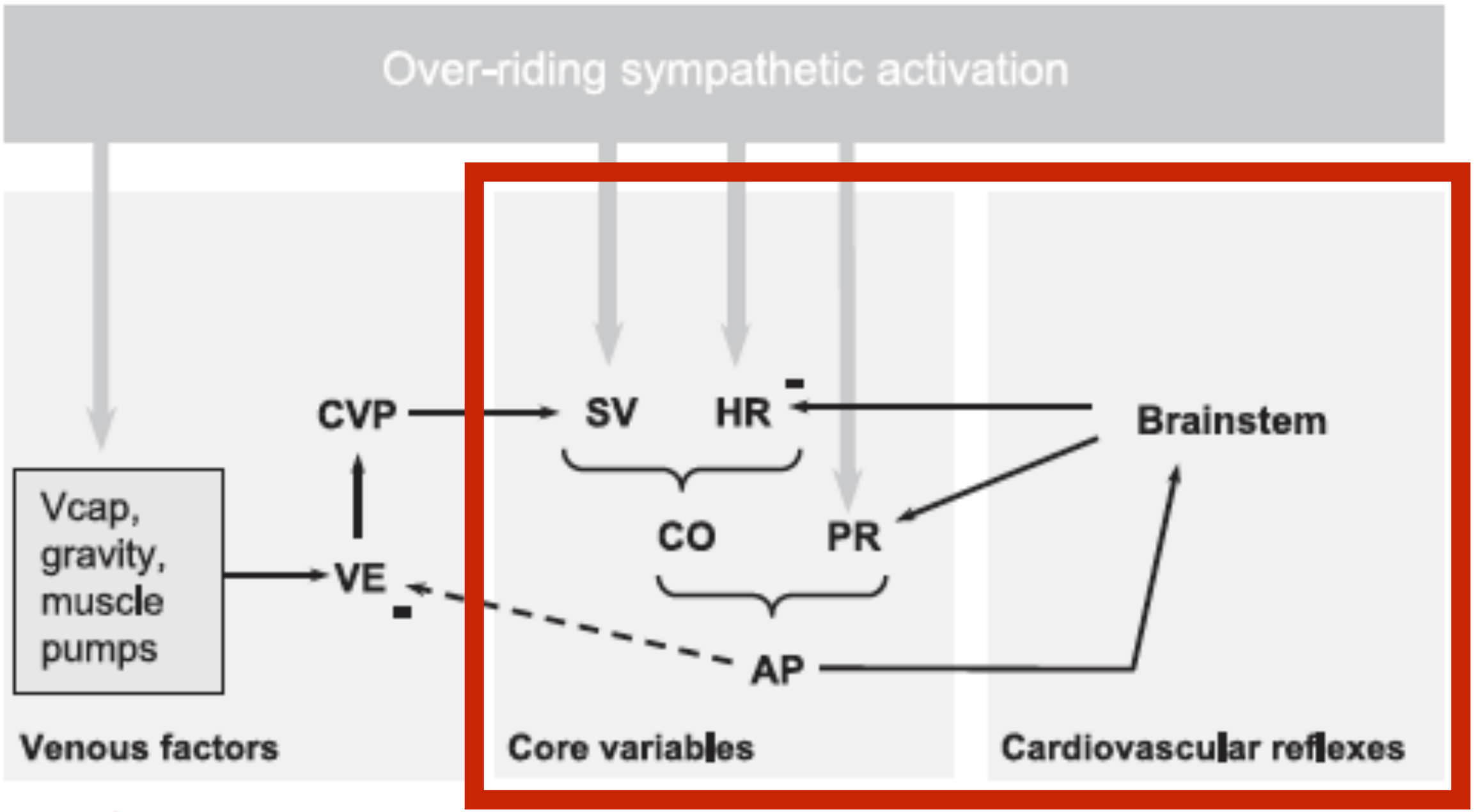

Fig. 5. The functional relationships between the main circulatory variables (VE, Vcap, SV, PR, AP). The relationships among SV, HR, CO, PR, and AP are true by definition; the arrows show physiological regulatory mechanisms, and the dashed line indicates the fact that arterial excess (distension) must cause venous deficit. CVP, central venous pressure.

Example: the mechanism by which a fall in resistance results in increased cardiac output can be explained as follows

- PR⇣ ▷ AP⇣ ▷ Baroreceptor activity⇣ ▷ HR⇡ and hence CO⇡
- Try another yourself; what happens when stroke volume is reduced?

If you worked out that

- SV↓ ▷ CO↓ ▷ AP↓ ▷ Baroreceptor activity↓
  ▷ HR↑ and hence CO↑

you've got the idea. But now we go with Dr Eugene into less-well appreciated territory, the influence of the venous side of the circulation on stroke volume.

THE VENOUS SIDE

At the Congress a Very Important Guy on a podium had talked about venous return, but this baffled Dr Eugene. If it is true that increasing venous return leads to an increase in cardiac output and if it is also true that increasing cardiac output leads to an increase in venous return, we have a positive feedback loop that offers no control. For regulation we need an 'error signal' that can be part of a negative feedback loop.

The difference between venous return and cardiac output is a measure of mismatch between the rate of blood entering the great veins and the rate of leaving. If the two are equal, then we are in a happy steady state. It is only when venous return exceeds cardiac output, or cardiac output exceeds venous return, that issues arise.

A difference between venous return (VR) and cardiac output will lead to an accumulating error signal, $\int(VR - CO)\,dt$, representing the volume of blood on the venous side waiting to be pumped. Reddi & Carpenter call this the venous excess (VE), and the rate at which it is growing they call the venous accumulation rate (VAR).

The Starling mechanism of the heart guarantees that the heart pumps out whatever is put into it, in other words that stroke volume is equal to the input of blood to the right atrium.

Input of blood to the right atrium depends on CVP (modified by heart rate if there is not enough time during diastole for pressures to equilibrate)

… and CVP depends on venous excess, which we've just defined.

- Venous excess is a component of the blood volume (BV), rises and falls with venous capacitance (Vcap), is gravitationally dependent on patient posture, and can be boosted by muscle pump activity during exercise.

The effect of an intravenous bolus infusion can therefore be explained as follows;

- BV⇡ ▷ VCap⇣ ▷ VE⇡ ▷ CVP⇡ ▷ SV⇡

- … and perhaps HR⇡ via the Bainbridge effect, then CO⇡ AP⇡ and HR⇣ to baseline.

To complete the picture, we need to add some more general mechanisms:

- first, the emergency sympathetic actions such as cardio-acceleration and increased force of contraction, vasoconstriction, and venoconstriction, which act globally on the circulation when the blood supply to the brain is threatened;

- second, the long-term mechanisms regulating blood volume, in particular the hormonal responses to long-term hypotension and to decreased venous volume.

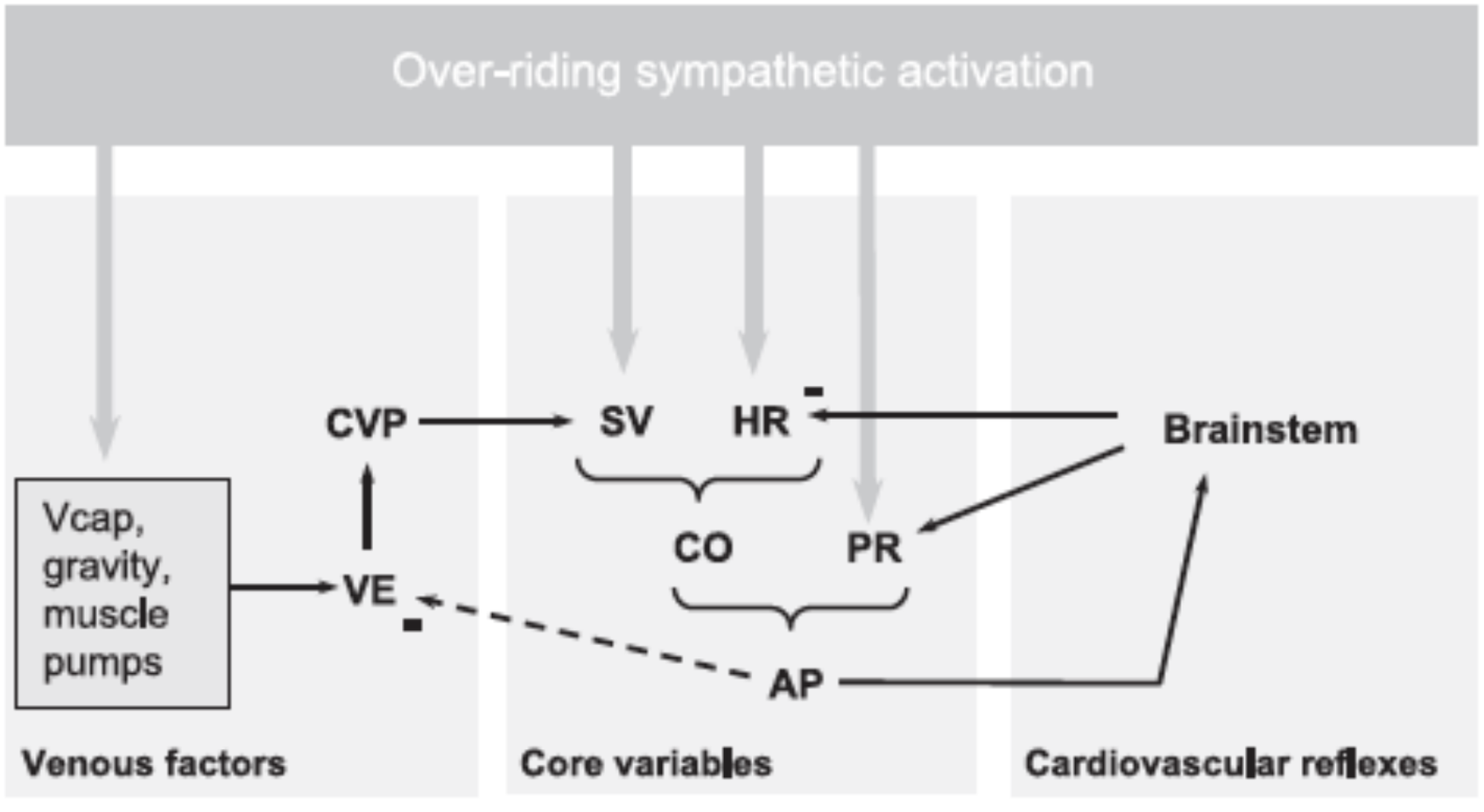

Fig. 5. The functional relationships between the main circulatory variables (VE, Vcap, SV, PR, AP). The relationships among SV, HR, CO, PR, and AP are true by definition; the arrows show physiological regulatory mechanisms, and the dashed line indicates the fact that arterial excess (distension) must cause venous deficit. CVP, central venous pressure.

"The finished system contains nothing mysterious, no abstract, unmeasurable variables. By systematically working through it, students can very easily work out for themselves the effects of exercise, hemorrhage, and other perturbations."

ROUNDS

On the next day's round Professor B'Staad asked Dr Eugene to explain the haemodynamic consequence of placing a patient in a head-down tilt. Being a conscientious student of the venous excess model, Dr Eugene was able to oblige. Prof B'Staad (MB ChB Cantab) was impressed.

If you too could answer Professor B'Staad, you really are an ace student and completely ready to engage in the clever guys' debate about whose interpretation of Arthur Guyton's classic experiments you favour.

Or you could just rest on your laurels and stick with this one.