SVR versus rate-independent vascular resistance

Why SVR is a False Measurement of Total Peripheral Resistance.

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Introduction

Calculation of peripheral resistance is based on Ohm's Law popularised by Guyton, viz. MAP-CVP = CO x SVR. Guyton argued that pressure varied with heart rate. His formula implies that peripheral resistance varies inversely with heart rate. It is problematic that this formula uses the average systemic pressure gradient, and divides it by the volume pumped by the heart per minute to derive systemic vascular resistance, introducing time as an intrinsic component of resistance. The way to remove time from the calculation of systemic vascular resistance is to use SV rather than CO for calculation of vascular resistance, represented here as rate-independent Vascular Resistance.

Methods

Haemodynamic information was studied in 300 patients at intervals of 12 seconds to 1 hour. In excess of 100,000 values were plotted graphically to determine the relationships described by Ohm's Law. The traditional formula MAP-CVP = CO x SVR was plotted, but the same data was plotted using the alternative formula MAP-CVP = SV x riVR. The alternative formula is based on the assumption that vascular resistance is not time dependent, and the pressure gradient in the circulation is the product of Stroke Volume and the distensibility of the vascular tree (riVR). The results of 2D plots were examined in a variety of clinical settings .

Did Guyton err?

Guyton's concept of afterload (the downstream 'work' against which the LV pumps) is fundamental to his intersecting venous return and ventricular function curves,and fundamental to the treatment of heart failure and hypertension. It all derives from the equation "MAP-CVP=CO x SVR"
Or: "MAP-CVP= SV x HR x SVR"
This predicts:(1) that Systemic perfusion pressure (MAP-CVP) varies with HR
(2) that SVR varies reciprocally with SV (Guyton)
(3) that SVR varies reciprocally with HR (contra Guyton)

However, if the systemic perfusion pressure remains constant while HR changes, then (1) is false. If SVR varies inversely with HR at a constant SV (Figs 1,2) then Guyton's concept of SVR becomes invalid. Experimentally, the relationship between SV and SVR has been examined by pacing subjects at a constant rate, but this intrinsically alters the HR-SV relationship, and the change in SV and SVR cannot be attributed to a second intervention (e.g.fluid loading)

Pressure in the circulation is instantaneous, constantly changing and is not time dependent; intuitively, resistance should behave similarly, but Guyton;s equation introduces time into the calculation of vascular resistance, thus:
MAP-CVP = (SV x HR) x SVR, so
$\Delta P = V/t \times R$
Guyton;s formula results in a resistance with the units Pt/V.

If resistance is not heart rate dependent, then Ohm's Law should be written:
MAP-CVP = SV x riVR
where riVR is systemic elastance or 'rate independent vascular resistance', and has the units P/V, or 1/Compliance. This can be reconciled with Guyton thus:
MAP-CVP= (SV x HR) x SVR = SV x riVR.
Solving this equation yields the relationship: ***SVR = riVR/HR***

If this relationship is correct, then any increase in HR will lower SVR at a constant elastance,and any decrease in HR will increase SVR at a constant elastance.

What difference does riVR as a measure of vascular resistance make?
(1) Contradicts Guyton's hydraulic model and supports baroreceptor/neural control of blood pressure
(2) When riVR is used as a measure of arterial resistance, it is apparent that inotropy, vasoconstriction and vasodilation are primarily a HR artifact. Vasoactive drugs should be classified as cardiotropic,vasotropic and chronotropic.

Results

If heart rate changes at a constant Stroke Volume, the systemic pressure gradient does not change. SVR and rate-independent VR (riVR) move in opposite directions when heart rate changes. SVR falls if heart rate rises, and rises where heart rate falls. This appears to explain the observation that vasoactive drugs that lower heart rate (noradrenaline,metaraminol) are vasoconstrictors, and vasoactive drugs that raise heart rate (dobutamine, adrenaline, dopamine) are vasodilators. If riVR is instead used to measure vascular resistance, many of the differences described for vaso-active agents are seen to be imaginary. In tachyarrhythmias, SVR decreases, even though patients are often clinically vasoconstricted, but riVR increases as Stroke Volume falls.

Conclusions

SVR is an erroneous construct. The use of SVR to measure vascular resistance has led to widespread misconceptions about the effects of vaso-active drugs. The use of rate-independent vascular resistance (riVR) correlates much better with clinical observation, and suggests that the differences between vaso-active drugs have been misunderstood. If the alternative formula is accepted, then it must be concluded that heart rate and circulatory pressure are unrelated, except in so far as HR impacts on Stroke Volume.

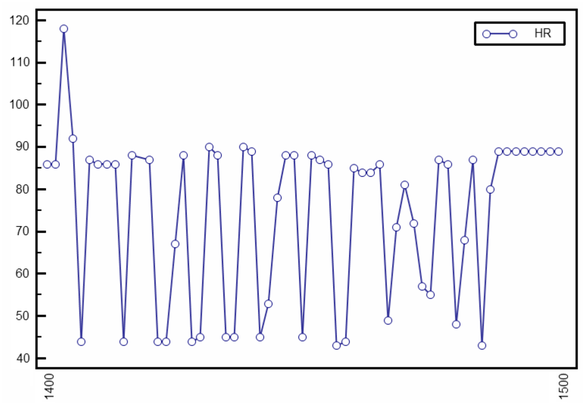

Figure 1.79M, OPCAG, recurrent dysrhythmia during surgery, plotted every minute.

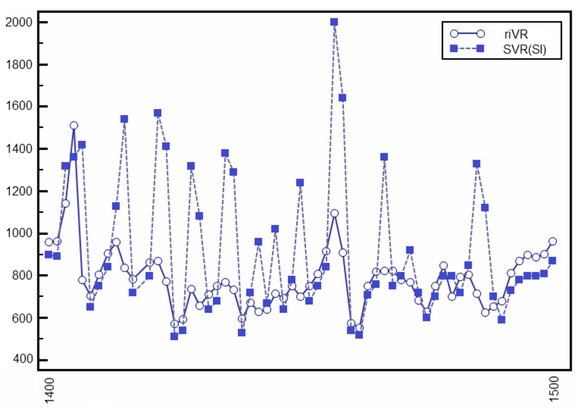

Figure 2.79M,OPCAG, SVR versus riVR plotted every minute using Guyton(SVR) and using riVR.

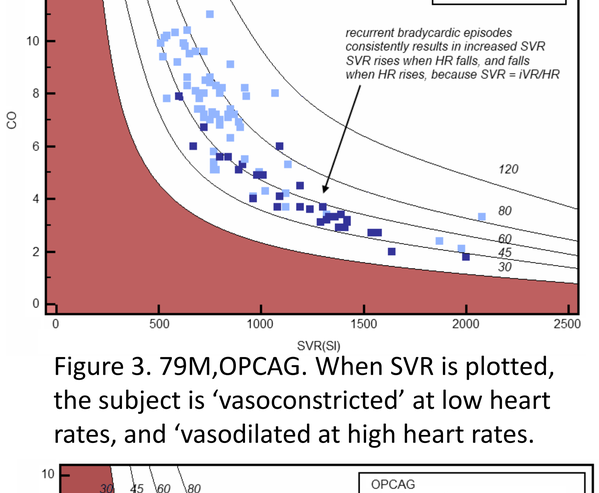

Figure 3. 79M,OPCAG. When SVR is plotted, the subject is 'vasoconstricted' at low heart rates, and 'vasodilated at high heart rates.

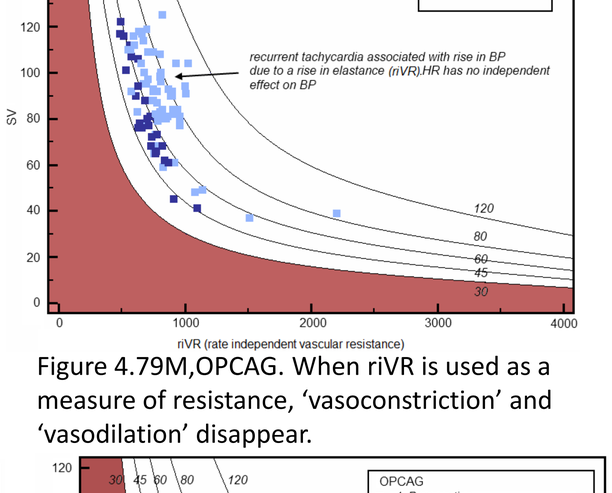

Figure 4.79M,OPCAG. When riVR is used as a measure of resistance, 'vasoconstriction' and 'vasodilation' disappear.

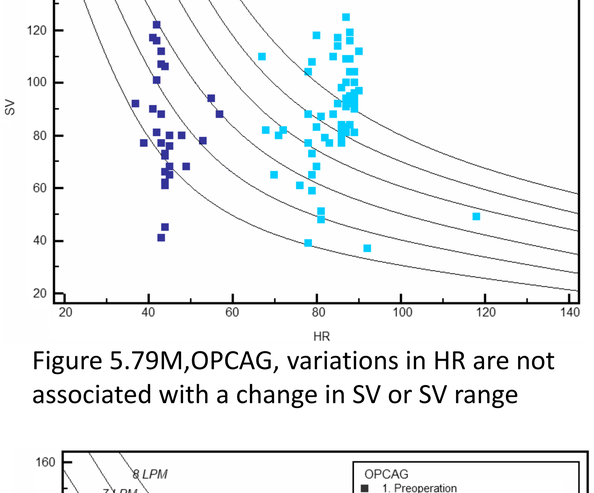

Figure 5.79M,OPCAG, variations in HR are not associated with a change in SV or SV range

Figure 6. 86M,EF 30%,OPCAG; intraoperative AF causes 'vasodilation'

Figure 7.86M,EF30%,OPCAG; when resistance is corrected for HR, 'vasodilation' of AF disappears.

Figure8. 86M,OPCAG. Rapid AF is associated with increased CO, even though SV falls.