Harvey and Guyton got it wrong: why we need a 2 pump model of the circulation.

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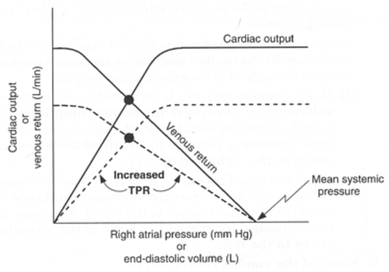
Introduction

In 1628, Harvey (De Motu Cordis) declared that **:** "It must of necessity be concluded that the blood is driven into a round by a circular motion in living creatures, and that it moves perpetually; and that this is the function of the heart, which by pulsation it performs; and lastly, that the motion and pulsation of the heart is the only cause"; although Guyton demonstrated that it was the need of the body tissues for oxygen was the real regulator of cardiac output, Guyton's model still subscribes to a 'one-pump' model of the circulation, in which the performance of the heart is determined by preload, heart rate, afterload and contractility.

Methods

Continuous haemodynamic data was collected at intervals of 12 seconds,20 seconds, 1minute, 10 minutes, 30 minutes and 1 hour in 250 patients presenting for elective major surgery. All patients had radial arterial lines, subclavian central lines and Flotrac arterial transducers (Edwards Lifesciences). Data was analysed graphically using the relationships MAP-CVP=CO x SVR, CO = SVxHR and MAP-CVP=SVx riVR (rate-independent vascular resistance). Monitoring was continued wherever possible in the postoperative period, and patterns of variation in blood pressure, stroke volume, cardiac output, SVR(MAP-CVP/CO) and rate-independent vascular resistance (MAP-CVP/SV)were studied.

Harvey's model: 'the motion and pulsation of the heart" is the only cause of motion of the blood. Harvey knew nothing of vascular resistance; if the heart drives the motion of blood and the circulation is passive, then $\Delta P = k\Delta Q$. The driving pressure is proportional to flow. This approximates the circulation in early life, when Resistance is low and relatively constant under controlled conditions. It fails in haemorrhage and sepsis.



Guyton's model: there is a 'hydraulic interdependence' between the heart and circulation. The vascular resistance (SVR/TPR) is the work against which the LV ejects. Guyton's concept is P=Q*R, where Q= SV*HR,and SVR =(MAP-CVP)/CO. Guyton's intersecting curves don't work if HR changes at constant SV.

Guyton's equation predicts that ΔP is proportional to HR. However, at constant SV, a change in HR produces no change in ΔP

Guyton predicts that at constant ΔP, if SVR increases,CO decreases; if SVR decreases, CO increases. Guyton's concept of SVR/afterload is only right if a fall in SVR increases SV, and a rise in SVR decreases SV. However, at constant SV, a change in HR increases CO and decreases SVR.

Both Harvey and Guyton use a 'one-pump model of the circulation. Guyton's concept of afterload breaks down on closer scrutiny. Guyton cannot have studied the elderly circulation, since after the menopause, the heart becomes stiff and acts as a conduit rather than a pump. The circulation becomes the primary driver of pressure in the circulation as the post-menopausal heart becomes stiff. The correct equation for the relationship between the heart, circulation and ΔP is: ΔP=SV*riVR, where SV is the cardiac contribution to pressure, and riVR is the elastance or rate-independent vascular resistance.

Reconciling 2 equations***: If MAP-CVP = (SV x HR) x SVR = SV x riVR, then SVR = riVR/HR.***

Any process which increases HR at constant elastance will decrease SVR,and any process which decreases HR at constant elastance will increase SVR. This produces a systematic error in the literature on inotropes, and explains why dobutamine appears to be a vasodilator and inotrope whenever it causes an isolated increase in HR.

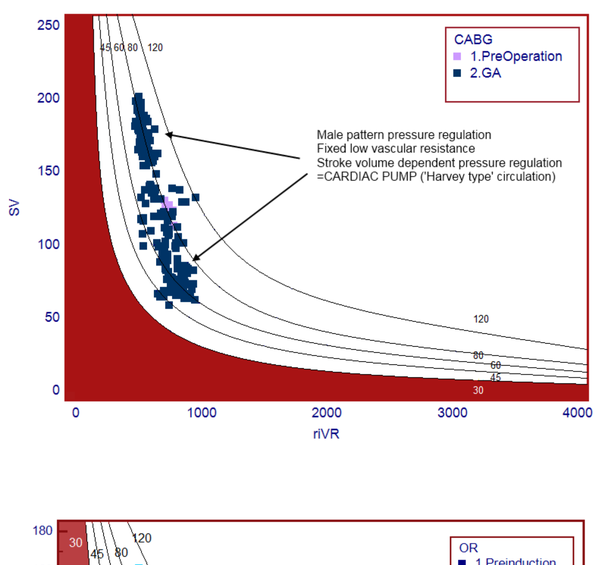
Harvey overlooked the vascular pump, and Guyton lacked the benefit of a dedicated SV monitor. Guyton's error arises from analysing total flow (CO) and not validating his model in the elderly, who become 'resistance dependent pressure regulators' as the stroke volume becomes fixed.

Results

Data collected in elderly patients presenting for elective major surgery demonstrates that in both genders there is a progressive 'failure' of the heart as a pump with advancing age: SV shows a progressive fall and a corresponding rise in vascular resistance. The regulation of blood pressure changes with advancing age, and the relationship between HR and SV changes progressively in predictable ways with advancing age.

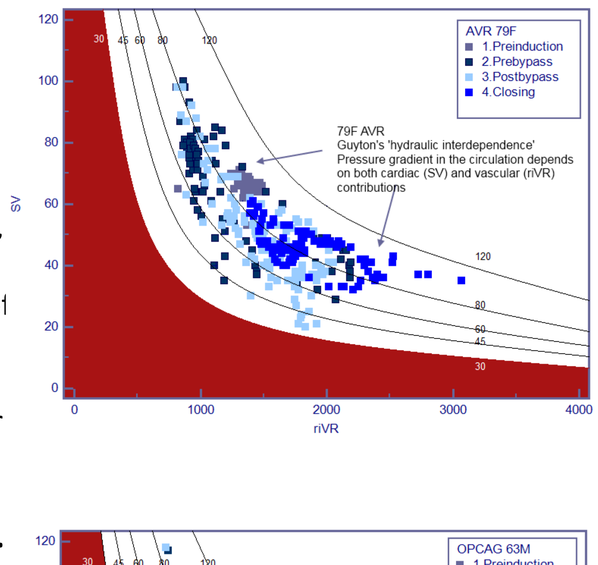
Conclusions

Regulation of blood pressure in youth is dependent on cardiac performance, and vascular resistance is relatively low and fixed. In old age it is the Stroke Volume that becomes low and fixed, and pressure varies with changing vascular resistance. This is important to intraoperative management of blood pressure, and to resuscitation. The change in blood pressure regulation cannot be reconciled with a single-pump model in the tradition of Harvey and Guyton. At any age, the regulation of blood pressure differs according to gender, and is consistent with the male capacity for higher work rates. Rather than seeing 'afterload' as a contributor to cardiac performance, we need a 2 pump model of the circulation, in which the circulation becomes the pump driving blood pressure as the heart fails.

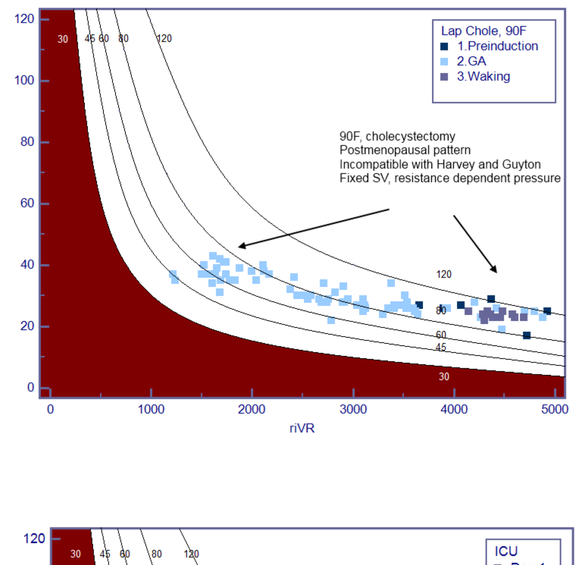


The Harvey model (ΔP = kΔQ)

In Harvey's physiology of the circulation, the performance of the heart is solely responsible for the motion of blood around the circulation**. If only the healthy, relatively young circulation is studied, this model is relatively defensible**



The Guyton model (hydraulic interdependence model ; P = Q*R): **Guyton offers a nuanced version of Harvey's 1628 model**, in which the vascular resistance imposes a workload on LV ejection. Guyton's concept underlies the therapy of heart failure and hypertension



The problem with Harvey and Guyton: **ΔP = kΔR**: the elderly circulation undergoes a fundamental change: there is a reduction in SV and SV range, the heart functions as a conduit rather than as a pump, the circulation is the primary driver of pressure and so displaces the heart as the primary 'pump'. This has no explanation in the Harvey and Guyton models. It can only be explained by baroreceptor feedback and neural control to achieve a baroreceptor 'set-point'