

## Vasodilatation and Hypotension: Blood Pressure is not all it's Pumped up to be

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It is commonly and widely held knowledge that blood pressure determines flow. However, at the risk of being burnt at the stake as a heretic it is time to examine and question this 'knowledge'.

Consider the following comparison.

	<b>Astronomy</b>	<b>Circulation</b>
Knowledge	The earth is flat	Pressure determines flow
Conclusions	If sail too far then will fall off the edge of the earth	Hypotension can lead to organ hypoperfusion
Evidence	Some sailors failed to return from voyages	Hypotensive patients have suffered morbidity
Alternative interpretations	Sailors got lost or drowned	Morbidity caused by other factors or treatment - vasoconstrictors
Conflicting data that do not fit the model	If the earth is flat why are other landmasses not visible?	Why does flow increase when there is vasodilatation?

**Table 1**

The unshakable 'fact' that pressure is required to drive a fluid (gas or liquid) from a region of higher pressure to a region of lower pressure warrants examination. While it is true that fluid motion and pressure gradients are associated, that association does not address cause and effect.

The driver for flow comes from the application of forces. Newton concluded that a mass would not change velocity (accelerate) in the absence of an applied force. Even the Jedi knights in Star Wars acknowledged this fact in their departing the farewell "May the force be with you".

In considering the role of perfusion in meeting tissue demands it is the volume of blood per unit time ( $m^3 \text{ sec}^{-1}$  or  $ml.min^{-1}$ ) that is important rather than the velocity of blood flow ( $m.sec^{-1}$ ). Apart from affecting transition time through vascular beds the speed is relatively insignificant when compared with the volume of flow.

Flow as it leaves the ventricle represents cardiac output, which is determined by myocardial contractility, preload (venous return) and after load (vascular resistance) as depicted by the cardiac function curves of the Frank-Starling Law.

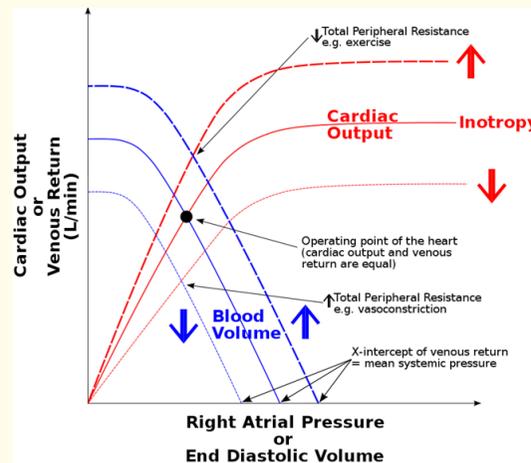


Figure 1

In the circulatory system the propulsive forces that propel the mass of blood ejected from the ventricles are produced by contraction of the ventricles and elastic recoil of the large arteries. The resistive forces that oppose the propulsive forces include vascular resistance, viscosity, and tissue frictional resistance. It is the balance of these forces that determines blood flow, the velocity of which is not constant (pulsatile). The opposing forces (mainly vascular resistance) in the presence of ventricular contraction are what cause there to be a pressure and pressure gradient [1]. In other words, pressure is the result of propulsive forces acting across resistive forces. This is described in the relationship

$$P = F/A$$

where pressure or strain (P) is the result of force (F) acting over an area (A). The pressure is the dependent variable and varies directly with force and inversely with cross-sectional area, both of which are the independent variables. From the diagram above it can be seen that increasing total peripheral resistance (after load) reduce both venous return and cardiac output.

While there are a number of equations relating pressure and flow they are simply arithmetic relationships and do not address cause and effect.

Interestingly, clinicians measure the severity of a vascular constrictive lesion in terms of the pressure gradient – the greater the gradient the less the flow and the greater the concern. How do we reconcile that here we have a high gradient but poor flow? Treatment of the poor flow is to remove/release/bypass the constriction, which will lower the gradient and improve flow [2]! This is nicely demonstrated with a balloon. The rate of gas flow (deflation) can be increased by reducing the outlet constriction leading to a lower pressure gradient but increased outflow.

What of the pressure and pressure gradients in the pulmonary circulation? The cardiac output of both left and right ventricles (in the absence of cardiac failure) is the same yet the pressure and pressure gradient on the pulmonary side is only one fifth of that on the systemic side. Clearly this is a reflection of the differences in resistance. Consequently, it is the resistance, in the presence of adequate ventricular function, that determines flow and not the pressure or pressure gradient. The venous side of the circulation is also low pressure and flow in dependent areas is assisted by the contribution of muscle contraction (pumps).

The main function of the circulation is to supply blood flow to tissues and organs according to their needs. In order to achieve this efficiently the body needs to be able to divert blood flow in accordance with those needs. The way it achieves this regulation is by alterations in vascular tone [3]. It increases resistance to areas of lower demand (increasing the upstream pressure and consequently the pressure gradient) and reduces the resistance to areas of greater demand (lowering the upstream pressure and consequently the pressure gradient).

The differences between pressures and gradients on the arterial side compared with the venous side are a reflection of function. The role of the arterial tree is to distribute blood to the organs/tissues while that of the veins is to collect the returning blood to the heart. Arterial smooth muscle and sympathetic innervation allows for significant changes in vessel calibre and consequently resistance, whereas diversion is not a function of the venous drainage system and venous constriction serves essentially to mobilize reserves of blood volume.

This has clinical significance because vasodilatation and hypotension are not an uncommon accompaniment to induction and maintenance of anaesthesia. The reason for this is two-fold. The first is a direct effect on the vascular smooth muscle to relax it; and the second is an indirect effect via the sympathetic nervous system, which is depressed along with global depression of cerebral activity. The resulting vasodilatation reduces peripheral resistance (after load) and results in hypotension.

The resultant hypotension has led to the widespread use of vasoconstrictors, which have a direct effect on vascular smooth muscle to increase tone leading to increasing resistance and consequently to increased after load. The rationale is to 'normalize' the blood pressure in the belief that firstly, vasodilatory hypotension has resulted in diminished flow and secondly, that administration of the vasoconstrictor will improve the flow [4]. Not only is this an unnecessary expense it may also be detrimental in converting a high flow situation to a low flow one as well as increasing myocardial work.

It is argued that we cannot measure flow (easily), whereas blood pressure is easily measured. As a result blood pressure is rationalized as a surrogate for flow. It would appear that blood pressure is an extremely poor surrogate and in fact may be misleading. We know that flow to organs is regulated according to tissue needs so how is the brachial artery pressure related to regional flows in the different vascular beds when flow, resistance, and pressure vary from bed to bed according to each of their varying needs?

Deliberate hypotensive anaesthesia has been practiced for decades. Well designed outcome studies are necessary to elucidate the benefit of normalizing blood pressure in the context of anaesthesia induced vasodilatation. Currently the studies assume that hypotension is harmful and consequently their protocols call for use of vasoconstrictors [6]. In these studies it is unfortunately not possible to differentiate between harm due to the hypotension and harm due to the vasoconstrictors and so the conclusions drawn are not valid.

In the presence of automated 'routine' thinking, without question, our patients may be being subjected to less than optimal outcomes. It is always beneficial to ponder, question, and explore in an attempt to improve health care.

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