Editorial

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# 'Advances in understanding and management in obstetric anaesthesia': The great myth of our times

Even after more than one century of Kreis' demonstration of spinal anaesthesia in obstetrics[1], understanding the complexity of the anatomy, physiology, pharmacology and application of neuraxial anaesthesia (NA) is still debatable. The reported incidence of hypotension and bradycardia after NA is 33% in the obstetric and 13% in the non-obstetric population.[2] According to traditional teaching, this hypotension is a consequence of a decrease in venous return (VR) and cardiac output (CO) (pre-load reduction theory), caused by the NA-induced preganglionic sympathetic nerve blockade[3,4] and supine hypotension syndrome of pregnancy (SHSP).[5]

Studies[6-8] between 1940 and 1970, mainly in animals and few volunteers and patients showed that systemic vascular resistance (SVR) is reduced by 5%–20%, stroke volume (SV) is reduced by 5%–25%, heart rate (HR) is reduced by 5–25%, CO is reduced by 10%–30% and arterial blood pressure (BP) is reduced by 15%–30%. It is well known that mean arterial pressure (MAP) = CO × SVR. CO depends on four variables, namely, preload, afterload, contractility and HR. The sympathetic blockade involving entire thoracolumbar outflow in NA reduces the preload by venodilation and afterload by a reduction in SVR, but the cardiac contractility is not affected.

The CO is largely determined by factors that depend on the venous side of the circulation, which was established originally by Starling's classic experiments.[9,10] Starling's studies were conducted in an isolated heart preparation, supplied with blood from a venous chamber, which could be raised or lowered to adjust the right atrial pressure (RAP). Here, the reservoir is raised or lowered to keep RAP constant. In his experiment, RAP increased the CO, but it did not mean that VR increased CO. Guyton's model demonstrated a relationship between RAP and VR, i.e., reduction in RAP increases VR[11] [Figure 1]. In the venous system, there is a driving pressure, the mean systemic filling pressure (MSFP) which is determined by stressed volume in veins and its compliance and resistance. Certain volume of fluid has to be added to fill the vascular bed to the point where its presence exerts a force on the vessel walls. This volume is known as the 'unstressed volume'. Any volume that is above this level is the 'stressed volume', which will exert an increasing degree of pressure on the venous vascular bed[12] [Figure 2]. VR is determined by the formula: $VR = MSFP - RAP/V\Omega$ where MSFP is mean systemic filling pressure, RAP is right atrial pressure, VR is venous return and VΩ is venous resistance.[13] We now realise the blood flow in the venous side is determined by MSFP and RAP since venous resistance is very low. In spinal hypotension, the VR can be increased by raising MSFP either by adding fluid which increases the stressed volume or by vasoconstriction of the veins. Since the venous capacitance and stressed volume are regulated by sympathetic tone, after NA venodilation will be maximal. If the veins lie below the level of the right atrium (RA), gravity will cause pooling of the blood peripheral veins and if the veins are above the level of the RA, there is backflow of the blood into the heart by gravity. In humans, in the supine position, blood flow into the inferior vena cava (IVC) is gravity dependent, and blood will drain by gravity into the IVC from liver, spleen and mesenteric veins, even if the sympathetic 'squeeze' on this splanchnic venous reservoir system is lost due to NA. Only the kidneys and lower limbs lie below the IVC. Hence, the lower limbs can trap blood after sympathectomy. Trendelenburg positioning of 10°–20° will facilitate the VR from lower limbs. In pregnant woman lying supine, the splanchnic capacitance veins will drain directly into the IVC through the hepatic veins; these are not compressed by the uterus and will therefore not affect VR.

In the management of NA-induced hypotension, the preload reduction theory suggested three ways to prevent NA-associated hypotension: (1) infusion of fluids, either colloids or crystalloids, to compensate for the venous blood that trapped in the lower limbs. However, findings that have emerged from fluid loading studies do not support this strategy. Preloading the patient with intravenous fluids can cause haemodilution and reduction in blood viscosity which increase CO transiently; however, hypotension eventually ensues by a further decrease in SVR by haemodilution. Colloid preloading may be more effective than crystalloid preloading.[14,15] The timing of fluid administration (pre-loading vs. co-loading) does not seem to impact on the incidence of spinal hypotension.[16] Although these findings have some clinical relevance, no fluid loading regimen has been 100% effective in preventing spinal hypotension.[15,17,18] (2) Leg compression was attempted but was relatively ineffective, despite the success of the anti-G suit in preventing lower limb pooling and hypotension in aerospace medicine.[19-21] (3) In obstetric anaesthesia, the post-spinal hypotension (SHSP) is attributed to aortocaval compression, preload reduction and reduced CO. Lateral uterine displacement is traditionally advocated to reduce IVC compression. However, more recent findings[22] using femoral vein ultrasound demonstrate that significant vena caval compression occurs in only 30% of supine women. Although uterine displacement is widely used, it does not reliably prevent hypotension after spinal anaesthesia.[17,23] The Cochrane collaboration suggests that 'there is limited evidence to support or clearly disprove the value of the use of tilting or flexing the table, the use of wedges and cushions or the use of mechanical displacers larger

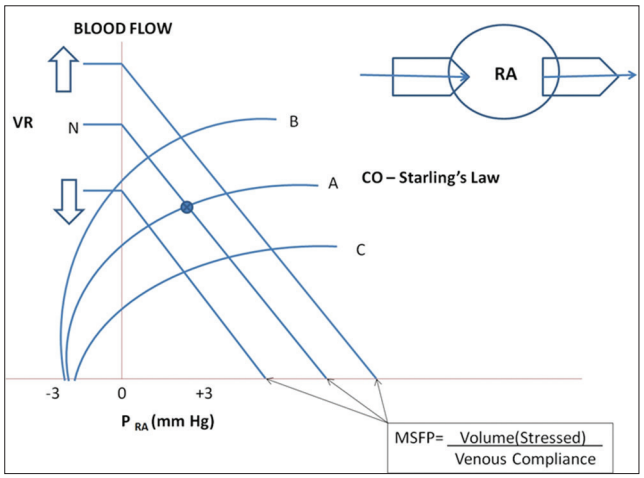


**Figure 1:** Guyton's model versus Starling's law. A: Normal VR and CO, B: Increased CO due to increased VR or sympathetic stimulation, C: Decreased CO due to decreased VR or cardiac dysfunction, MFSP: Mean systemic filling pressure, VR: Venous return, $P_{RA}$: Right atrial pressure, CO: Cardiac output, RA: Right atrium

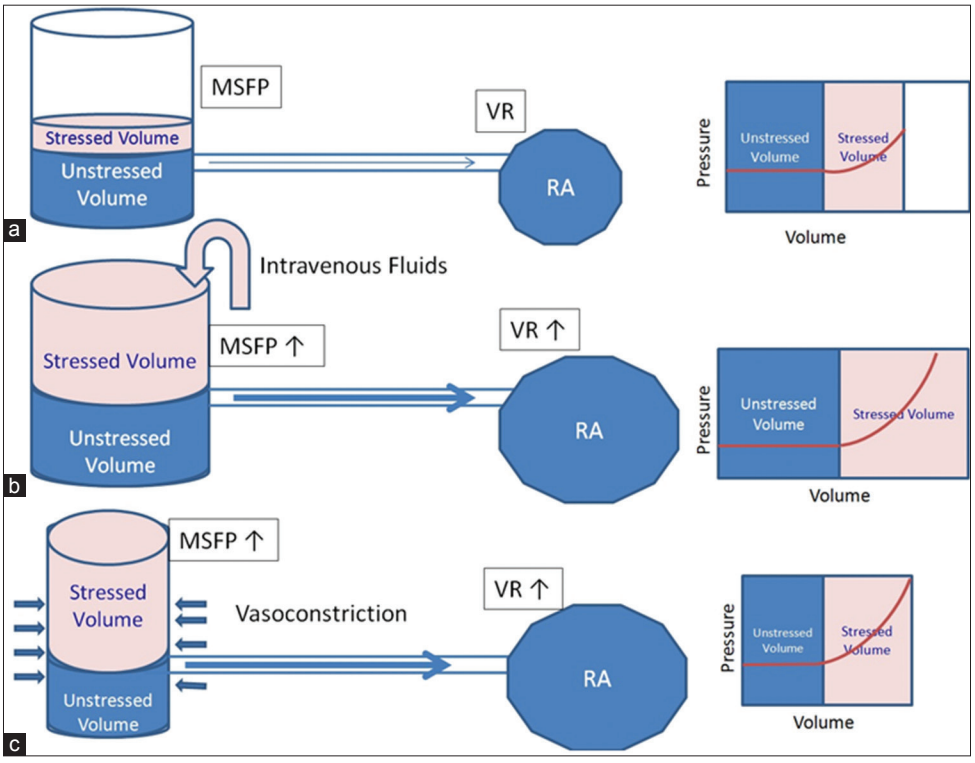


**Figure 2:** (a) Stressed volume maintaining the VR. (b) Intravenous fluid increases MSFP and VR. (c) Vasoconstriction increases MSFP and VR. MFSP: Mean systemic filling pressure, VR: Venous return, RA: Right atrium

studies are needed to confirm these findings'.[24] It is, therefore, clear that therapies based primarily on the concept of preload and CO reduction do not reliably prevent hypotension after NA.

Apart from hypotension, reduction in HR is observed frequently in obstetric NA. HR is invariably affected if the sympathetic block is above T4, involving cardiac sympathetic fibres. However, the finding of elevated resting HR in denervated, transplanted hearts indicates that sympathetic tone is not generally required to maintain normal resting HR, which is less than the intrinsic automatic firing rate of the sinoatrial node.[25] Reduction in HR can occur even in low spinal block due to intrinsic cardiac reflexes that are volume sensitive. These includes local Bezold–Jarisch reflex that causes an acute reduction in SA node output in response to decreased LV stretch, the reverse Bainbridge reflex that causes an increase in vagal tone in response to reduced RA stretch; and other un-named intrinsic cardiac reflexes.

Thus, based on improved understanding of the physiological changes that occur following spinal anaesthesia, important advances have been made for preventing spinal hypotension during caesarean delivery. There is a paradigm shift from preload reduction theory to afterload reduction as the primary mechanism of NA-induced hypotension. In this situation, a rapid acting alpha-agonist such as phenylephrine is the best option in obstetric NA to restore baseline haemodynamics. Fluids are only given for volume replacement in hypovolemic patients. Since bradycardia has been identified as the main precursor of cardiac arrest in NA, epinephrine (0.1–0.2 mg) should be considered early in the treatment of severe bradycardia, especially if conventional doses of atropine are not effective.

The non-pregnant uterine circulation exhibits autoregulation, alternately vasoconstricting or vasodilating in response to a number of different stimuli.[26] However, the mature placenta is a high capacitance, low-pressure organ with no autoregulatory and little vasoconstrictor ability.[27] It is generally accepted that uteroplacental perfusion depends on maternal CO rather than on BP. Robson *et al*. demonstrated the presence of a correlation between decreased maternal CO and foetal acidosis during caesarean deliveries under spinal anaesthesia, but no correlation between maternal hypotension and foetal acidosis.[4] Subsequently, studies[28,29] showed that the maternal CO, HR and SV increased during the first 15 min after induction of spinal anaesthesia. The initial compensatory vasoconstriction in the upper limbs and a redistribution of blood from slow to fast beds could be the reason for the increase in CO. Clinicians who rely solely on monitoring of BP may under-appreciate this. This issue of the journal contains a similar study of cardiac index (CI) variability under epidural and combined spinal-epidural (CSE) in parturients in labour, which throws light into the reliability of BP monitoring in relation to CI.[30] CSEs were associated with a significant decrease in CI as compared to epidurals for labour analgesia, even though both groups showed a similar decrease in BP. The authors question whether BP monitoring alone provides an accurate reassurance for adequate placental perfusion. However, the limitation in this study is lack of assessment of uteroplacental blood flow with ultrasound Doppler which would have revealed more facts than meets the eye.

Another new frontier in obstetric anaesthesia, the hybrid operation room is analysed interestingly by an article in this issue.[31] The increasing rate of caesarean delivery in India from 8.5% to 31.0%–50.2%.[32,33] and attendant placental implantation abnormalities, coupled with increasing cardiac comorbidities in the obstetric population, has driven the innovation of more integrated hybrid operating rooms to take care of high-risk parturients during delivery. Hybrid operating rooms are very extremely complex working environments where a large team of surgeons, intervention radiologists, anaesthesiologists, nurses and technicians work seamlessly together. Caesarean deliveries with an increased risk of haemorrhage, cardiovascular disease or intracranial pathology need to be operated in an OR with facilities for cardiac catheterisation, neuroangiography and magnetic resonance imaging. A hybrid OR also gives surgeons and medical staff the flexibility in planning and carrying out different procedures ranging from ureteral stents to internal iliac artery catheterisation without transporting patients between floors or shunting the patients.

Although the procedural benefits of having a hybrid OR can be extremely great, it requires a significant investment of both space and money. The expected cost of building a hybrid OR is 120% more than that of a traditional surgical OR, and the operating costs for each hybrid OR adds 90% to standard OR costs.[34] The installation costs range from US $1.2 million to $5.0 million depending on the devices that are installed.[35] The utility and cost benefit also depends

on the critical cases turnover. The application of hybrid procedures and use of the hybrid OR is expected to grow with the evolution of more imaging technology. But it's feasibility in Indian scenario in terms of cost-effectiveness has to be seen.

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