

“Belt and braces”: prophylactic vasoconstrictors after spinal blocks in the elderly

A Milner 

University of Pretoria and Steve Biko Academic Hospital, South Africa

Corresponding author, email: anamil@iafrica.com

Churchill: “Those who fail to learn from history, are doomed to repeat it.”

South Africa is following the global trend of an aged population explosion (Figure 1). Consequently, anaesthetists have to provide safe anaesthesia to this growing group, whose failing physiology may impact pharmacokinetic and pharmacodynamic parameters.

The cornerstone of safe anaesthesia is tissue oxygenation, an endpoint, which ensures aerobic metabolism. Aerobic saturation is essential for the production of adenosine triphosphate (ATP), the energy required by all metabolic processes, which is derived via a mitochondrial chain where oxygen is the initiator of the entire process.² The formula for oxygen delivery below identifies the parameters required to ensure that the endpoint, i.e. oxygen supply to all tissues is maintained.²

$$DO_2 = \text{Cardiac output} \times \{ (1.31 \times \text{Hb} \times S_aO_2 \times 0.01) + 0,0025 \times P_aO_2 \}$$

During the aging process, the oxygen delivery (DO_2) to tissues slowly declines as:

1. Cardiac Output gradually decreases due to:

A. Compromised myocyte contractility:

- Contractility is hampered as myocyte collagen and elastin decline, resulting in decreased left ventricular end-diastolic volumes and ejection fractions.
- Downregulation of myocyte beta-receptor function also occurs with age. This becomes relevant during

hypotensive episodes when there may be a slower and exaggerated response to a drop in blood pressure.

- Heart rate variability is decreased by lessened baroreceptor function and vagal tone.

B. Afterload:

- Deteriorating function of endothelial cells in arterial walls results in increased reactive oxygen species and a decrease in nitric oxide production. The resultant peripheral vasoconstriction and elevation of blood pressure causes increased afterload and increased work of the heart.³
- In addition, baseline noradrenaline levels are also elevated, which exaggerates the afterload resistance.⁴

C. Preload:

- Due to ‘venous stiffening’, less blood is returned to the heart and hypotension may be exaggerated.⁴

2. Oxygen saturation (S_aO_2) decreases with age as the pulmonary gas exchange deteriorates:

- Ventilation/Perfusion (V/Q) mismatches increase.
- Alterations occur in lung compliance, resistance, and lung mechanics.⁴

3. Partial pressure of oxygen (PaO_2) is thought to decrease by 0,4 mmHg/year.⁵



Figure 1: The steady increase of life expectancy in South Africa¹

Unfortunately, the aging process results in other pathological conditions that may also impact or be impacted by induced hypotension e.g. coronary artery disease, valvular dysfunction, heart and conduction system failure, and decreased perfusion to the kidney.³

Hypotension after spinal anaesthesia is the result of lumbar sympathetic chain blockade. The incidence of significant post-spinal hypotension in geriatric patients appears to vary widely between 5 and 80% and is often difficult to predict.⁶ Therefore, the article in this edition of the SAJAA by Filani et al. has merit for suggesting that patients over 65 years of age, should receive a prophylactic infusion of 50 µg/ml phenylephrine at 1 ml/min, lasting 10 min after spinal anaesthesia. Phenylephrine is a potent vasoconstrictor with alpha-1 and alpha-2 agonistic effects. Bradycardia is a common side-effect and may be exaggerated in the elderly who have depressed heart rate variability. Predictably, the study also found that the lowest dose trialed, i.e. 50 µg/ml, had the least side-effect profile.⁷

Drugs such as phenylephrine with short half-lives will rapidly reach plasma steady-state concentration, therefore, giving the drug as an infusion over 10 minutes may delay side-effects. Also, if severe bradycardia does occur during the infusion, the infusion may simply be terminated.⁷

So, in conclusion, due to the unpredictable response of sympathetic fibre blockade in the elderly who received spinal anaesthesia, the “belt and braces” approach of giving prophylactic vasoconstrictor immediately after administration of the spinal local anaesthetic, can not be faulted.

ORCID

A Milner  <https://orcid.org/0000-0003-1621-6511>

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