Best Practice & Research Clinical Anaesthesiology 28 (2014) 297–303

Contents lists available at ScienceDirect

Best Practice & Research Clinical Anaesthesiology

journal homepage: www.elsevier.com/locate/bean

9

Update on volume therapy in obstetrics

Wendy H.L. Teoh, MBBS, FANZCA, FAMS, Senior Consultant, Adj. Assistant Professor [a, b, *],
Martin Westphal, MD, PhD, Professor, Chief Medical Officer [c, d],
Tim G. Kampmeier, MD, Research Fellow [c]

[a] *Department of Women's Anaesthesia, KK Women's and Children's Hospital, Singapore*
[b] *Duke University – NUS Graduate Medical School, Singapore*
[c] *Department of Anaesthesiology, Pain Medicine and Intensive Care, University Hospital of Muenster, Muenster, Germany*
[d] *Fresenius Kabi AG, Bad Homburg, Germany*

Keywords:
hypotension
spinal anaesthesia
caesarean delivery
preload
coload
crystalloids
colloids
HES
volume
obstetrics

Symptomatic hypotension (maternal nausea, vomiting, dizziness and dyspnoea) during spinal anaesthesia for caesarean delivery remains a prevalent clinical problem. Severe and sustained hypotension can lead to impairment of uteroplacental perfusion, foetal hypoxia, acidosis, neonatal depression and further adverse maternal outcomes of unconsciousness, pulmonary aspiration, apnoea and cardiac arrest. Mechanical methods aimed at countering the effects of aortocaval compression do not reliably prevent maternal hypotension. Intravenous crystalloid preloading (given prior to administration of spinal anaesthesia) has poor efficacy, and focus has changed towards decreased use of crystalloid preload and ephedrine, to increased use of coload (given at the time of spinal administration) with colloids or crystalloids, and early use of phenylephrine. The recent multicentre, randomised, double-blinded CAESAR trial demonstrated the efficacy of a mixed 500 ml 6% hydroxyethyl starch (HES) 130/0.4 + 500 ml Ringer's lactate (RL) preload in significantly reducing hypotension, compared to a 1-l RL preload, without adverse effects on coagulation and neonatal outcomes in healthy parturients undergoing caesarean delivery under spinal anaesthesia.

* Corresponding author. Department of Women's Anaesthesia, KK Women's and Children's Hospital, 100 Bukit Timah Road, 229899, Singapore. Tel.: +65 81217986; Fax: +65 62912661.
E-mail addresses: teohwendy@yahoo.com (W.H.L. Teoh), martin.westphal@gmx.net (M. Westphal), tim.kampmeier@gmail.com (T.G. Kampmeier).

http://dx.doi.org/10.1016/j.bpa.2014.07.004
1521-6896/

Introduction

In contemporary obstetric anaesthesia practice, neuraxial techniques are the preferred method for providing anaesthesia for caesarean delivery over a general anaesthetic. Compared to an epidural or combined spinal–epidural (CSE), single-shot spinal anaesthesia is most commonly employed as it is easy to perform and provides rapid-onset dense neuroblockade [1,2]. The goal for adequate surgical anaesthesia is a sensory blockade to the T5 dermatome. This is typically achieved by administration of intrathecal hyperbaric bupivacaine ranging from 10 to 12 mg with an opioid adjuvant [3]. All neuraxial techniques are associated with hypotension from the resulting sympathetic blockade. In an effort to alleviate this, the administration of fluids and vasopressors remains the cornerstone of treatment of spinal-induced hypotension, in tandem with avoidance of aortocaval compression by effecting left lateral uterine displacement and use of calf compressors to aid venous return.

As the mass of local anaesthetic influences the spread of the block and causes hypotension, lower doses of 4.5–6.6 mg bupivacaine [4–6] as part of a single-shot spinal technique have previously been used for caesarean delivery, but this has raised safety concerns of whether lower doses should be used just because they can be used, due to the potential for unnecessary conversion to general anaesthesia. Lower spinal doses of local anaesthetics, however, can safely be utilised as part of a CSE technique especially if it is imperative to maintain stable intraoperative haemodynamics (e.g., severe pre-eclamptics and high-risk parturients with cardiac problems) as the epidural catheter allows analgesia supplementation in the event of prolonged surgical duration and breakthrough pain [7,8].

Incidence and physiology of maternal hypotension revisited

Hypotension is arbitrarily defined in the literature as a systolic blood pressure <100 mmHg or <80% of the baseline value [9], and is a common sequela after spinal anaesthesia in 55–90% of parturients [10]. Reductions in blood pressure of >30% baseline have also been described in nearly 50% of women undergoing spinal anaesthesia [11]. The risk factors for hypotension include increased sympathetic tone, increasing age, obesity, higher blocks and higher birth weight, but not multiple gestation [12]. The clinical signs of spinal hypotension are usually limited to nausea/vomiting and/or dyspnoea. However, hypotension when severe and sustained can lead to impairment of uteroplacental perfusion (as the vessels are maximally dilated, of low resistance and exhibit no autoregulation), resulting in foetal hypoxia, acidosis, neonatal depression or injury [13] and adverse maternal outcomes of unconsciousness, pulmonary aspiration, apnoea and cardiac arrest.

It was traditionally taught that hypotension occurred as a result of decrease in venous return and cardiac output after a spinal anaesthetic. Strategies to increase venous return (leg lifting and mechanical compression of the lower extremities) and aggressive intravascular volume loading have proven largely ineffective in the treatment of arterial hypotension [14]. Tamilselvan et al. [15] studied maternal cardiovascular indices and reported that the corrected ejection time (a measure of ventricular filling) did not change after the onset of spinal blockade, refuting a significant decrease in venous return as the cause of maternal hypotension.

We now know that neuraxial anaesthetic techniques produce hypotension through blockade of sympathetic fibres which control vascular smooth muscle tone. Preganglionic sympathetic fibre blockade primarily causes an increase in venous capacitance, which shifts a major part of blood volume into the splanchnic bed and lower extremities reducing venous return to the heart; there is also decreased resistance in arterial pre- and postcapillary resistance vessels. The resulting decreased systemic vascular resistance contributes significantly to arterial hypotension, and Langesaeter et al. [16] showed that this was associated with an increased cardiac output [16]. Dyer et al. further showed that this increased cardiac output correlated with increased heart rate changes after vasopressor administration, emphasising the importance of heart rate as a surrogate indicator of maternal cardiac output [17,18].

Intravascular volume loading strategies

Fluid administration during caesarean delivery is indicated not only to offset the dehydration of preoperative fasting but also to maintain cardiac output during the onset of spinal blockade. Most