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In reply

We would like to thank Bouvet and Chassard for their interest in our review.[1,2] They raise an important point which is relevant to obstetric anaesthetists in their day-to-day practice. As Bouvet and Chassard point out, high neuraxial blockade has long been considered to be dangerous, especially at the high thoracic or low cervical vertebral levels due to the associated adverse cardiovascular and respiratory consequences. As the onset of Horner's syndrome (HS) results from interruption of oculo-sympathetic fibres which are said to exist between T1 and C8, one would expect concurrent hypotension and respiratory difficulty.

In our opinion one of the most interesting findings of the review is the lack of adverse features and the low level of cutaneous sensory blockade associated with the onset of HS during obstetric neuraxial blockade. The mean level of sensory blockade reported was only T4 and the level was as low as T12. In most cases, HS occurs despite the cutaneous level of blockade being significantly below the lower limit at which the oculo-sympathetic fibres emerge from the sympathetic chain. It is not completely clear to us why this large discrepancy between the level of cutaneous blockade and HS exists – we speculate that possibly the oculo-sympathetic fibres are more susceptible to the effects of local anaesthetic during pregnancy. Only 13% of cases of HS experienced systemic hypotension and only one a consequent fetal bradycardia, with all cases managed successfully using intravenous fluid and vasopressors. Finally, there were no reports of airway or ventilatory compromise associated with HS.

In answer to the practical question regarding the feasibility of an epidural top-up in the management of a parturient undergoing a category 1 caesarean section, our view remains that the presence of HS alone should not influence anaesthetic management and that these women should not be denied neuraxial anaesthesia. Many anaesthetists may not note the presence of HS after an epidural top-up in theatre. The presence of HS alone does not appear to be strongly associated with systemic hypotension or with adverse maternal or fetal outcomes. On the other hand, in our opinion, if HS co-exists with systemic hypotension, upper limb weakness or cranial nerve palsy, one should carefully consider the risks of proceeding to an epidural top-up compared to the risks of alternative anaesthetic management.

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In vitro intravenous fluid co-load rates with and without an intravenous fluid warming device

Delivery of an intravenous fluid co-load decreases hypotension after spinal anesthesia.[1] Fluid warming is recommended to reduce the incidence of hypothermia after cesarean delivery.[2,3] However, the increased tubing length and resistance of an in-line fluid warming device may reduce the speed of fluid administration and

Table 1 Time and flow rates for intravenous fluid administration

Groups	Time (s)	Flow rate (mL/min)	Mean difference (mL/min) (95% CI)	*P*-value
Control	271	178	Ref	Ref
Heated Ranger™	281	171	−7 (−4 to −10)	0.0003
Unheated Ranger™	292	165	−13 (−10 to −16)	<0.0001

decrease co-load efficacy. Therefore, we set out to evaluate the effect of a fluid warming device on intravenous flow rates during co-loading.

We conducted a randomized, in vitro study with three groups. To best approximate in vivo conditions, the control group consisted of our standard Y-type blood tubing set (Lifeshield™) attached to a 127 cm extension tubing set (SetSource™) and an 18-gauge peripheral intravenous catheter (Braun™). The experimental groups included a warming insert set (3 M Ranger™) between the blood tubing and extension set. To assess the effect of heat, the fluid warmer was turned on in the first experimental group (heated) and left off in the second experimental group (unheated).

Fluid in all groups was pressurized to 250 mmHg in a dual-chamber pressure infuser (3 M Ranger™). The primary study endpoint was the time required for 800 mL (10 mL/kg co-load and 80 kg standard patient weight) of lactated Ringers solution to accumulate in a graduated cylinder. The experiment was repeated in 16 runs per group, for a total of 48 measurements. To limit the effect of tubing fatigue or manufacturing anomalies, tubing sets and intravenous catheters were used for a series of four runs each. Histograms were utilized to screen for outliers. A two-sided t-test was performed to compare mean differences between the groups. Statistical analysis was performed in SAS® Version 9.4.

Histogram screening and subsequent statistical analysis identified a series of four runs with times that were significantly outside the normal distribution. This single series was associated with a single tubing set and was subsequently excluded from the statistical analysis because of a suspected manufacturing defect. The mean flow rates of the experimental and control groups are shown in Table 1. The 800 mL fluid co-load took 10 seconds (95% CI 10 to 16 s, $P = 0.0003$) longer to administer when a heated in-line warming device was added to a standard intravenous set (heated group: 281 s versus control group 271 s). The unheated in-line warming set increased co-load time by 21 seconds (95% CI 16 to 27 s, $P < 0.0001$) compared to no insert.

These results demonstrate a modest decrease in crystalloid flow rates with an in-line fluid warming insert. Although statistically significant, this difference appears unlikely to be clinically important enough to impact the ability to provide an effective co-load for spinal anesthesia. Given our findings, the benefits of perioperative normothermia likely outweigh the risks of a slower co-load. Further in vitro research would be necessary to compare other in-line fluid warming devices and different fluids (i.e. hetastarch, albumin, blood products, etc.), as well as to confirm these findings in vivo.

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Declarations of interest

None.

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