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Correspondence

Central neuraxial blocks and anticoagulation

EDITOR:
Tyagi and Bhattacharya's review of anticoagulants and spinal-epidural blockade is very interesting, but a glance at the section about antiplatelet agents shows that inappropriate MEDLINE searches and cross-checking with former reviews were made [1]. Wulf sampled case reports (1966–1995) [2] and Vandermeulen and colleagues conducted a literature search on the National Library of Medicine's MEDLINE system (1906–1994) [3]. Both authors identified one case of spinal haematoma associated with acetylsalicylic acid therapy and central block [2,3]. Greensite and Katz described a case of a 68-yr-old male admitted for total knee replacement where a continuous epidural anaesthetic was planned [4]. Aspirin and other anti-inflammatory drugs had been stopped 5 months before surgery and preoperative prothrombin and partial thromboplastin times as well as platelet count were within normal limits. A 16-G Tuohy needle was advanced in the L2–3 interspace, but as gross blood appeared from the needle, it was immediately removed and reinserted into the L3–4 interspace. After operation medications included aspirin 650 mg twice daily: the first dose was administered 4 h after the end of surgery. Approximately 36 h after operation, sacral paraesthesia progressing to paraplegia occurred. An emergency myelogram demonstrated an extramedullary haematoma. At laminectomy, a large amount of blood was found in the subdural space extending from L2 to T12. The neurosurgeon noted no vascular abnormalities or tumours. This was the first spinal haematoma that resulted in cauda equina syndrome reported in association with a central nervous block in a patient without a vascular tumour who was taking aspirin. A MEDLINE search for 1966–2002 was performed for the following terms: 'Epidural', 'Spinal An(a)esthesia', 'H(a)ematoma'. Only two cases of confirmed spinal haematoma in conjunction with epidural or spinal anaesthesia in patients taking aspirin were identified [4,5].

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A reply

EDITOR:

We are grateful for the opportunity to respond to Dr Fattorutto's comments on our review [1]. His observations, however, are entirely misplaced and wrongly quoted. We will comment on his first observation about the data sources used for the two review articles on the subject of 'central neuraxial blockade in patients receiving anticoagulation' [2,3]. Wulf [2] not only sampled case reports from 1966 to 1995 using a MEDLINE search, but also cross-checked former case reports on the subject by continuous reading, over 10 yr, of the relevant anaesthesia journals. Similarly, Vandermeulen and colleagues [3] conducted a MEDLINE search and acquired further data by assessing previously published major reviews and large case series. Between 1906 and 1994, they identified 61 cases of spinal haematoma involving central neuraxial block. A similar overlapping approach for data collection was used by us to make the review [1] as detailed and complete as possible. A MEDLINE search for new cases (1995–2000), previous reviews with their cross-references [2–4] and other case reports gathered from relevant journals were included for the literature review.

Fattorutto refers to two earlier papers [2,3] that identify only one case of spinal haematoma with acetylsalicylic acid therapy and central neuraxial blockade. As correctly pointed out by us in our review [1], four of the 61 patients who developed spinal haematomas reported by Vandermeulen and colleagues were receiving antiplatelet therapy. While two of the patients had received aspirin (one along with concomitant systemic heparin therapy), one each had received ticlopidine and indomethacin. All three drugs incriminated are antiplatelet agents, and hence all four cases were included under the heading of 'spinal haematoma occurring in patients on antiplatelet therapy receiving central neuraxial blockade'. Wulf also reported three cases of spinal haematoma in patients with combinations of aspirin or other non-steroidal anti-inflammatory drugs and epidural anaesthesia. Probably Fattorutto has failed to appreciate that we reviewed all antiplatelet agents in relation to their effect on the incidence of spinal haematoma in patients receiving central neuraxial block, and not just 'aspirin and central neuraxial block'.

Our review reported that a single case of spinal haematoma in patients undergoing central neuraxial block with concomitant aspirin therapy [5] could be found by a MEDLINE search for 1995–2000. The last contention cited by Fattorutto that the MEDLINE search he conducted for 1966–2002 revealed only two cases [5,6] of this combination is a mere repetition of our observation. The second case referenced and detailed by him [6] was reported by Greensite and Katz in 1980 and thus obviously did not figure in our MEDLINE search conducted for 1995–2000. However, even this case report has been correctly referenced by us in the appropriate section of the text.

Thus, we do not agree to having used an inappropriate MEDLINE search or in having cross-checked incorrectly with former reviews on the subject. Rather, a detailed and more in-depth reading of the review is recommended before such observations are made.

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Intrathoracic blood volume in a patient with pulmonary embolism

EDITOR:

Intrathoracic blood volume (ITBV) is a reliable indicator of myocardial preload in critically ill patients [1,2]. The single transpulmonary thermodilution technique, which is increasingly used for haemodynamic monitoring, is sufficiently accurate for clinical estimation of ITBV and extravascular lung water (EVLW) [3,4]. We report a patient with low ITBV, despite high central venous pressure (CVP), which was first interpreted erroneously as hypovolaemia.

A 34-yr-old male with abdominal and chest trauma was transferred to our institution. He underwent splenectomy and mini-thoracotomy for removal of a left-sided pleural haematoma that caused compression atelectasis. Unfortunately, the patient developed the adult respiratory distress syndrome (F_iO_2 60%, with a positive end-expiratory pressure of 10 cmH_2O) and septic shock on day 19. At that stage, extended haemodynamic monitoring, including the transpulmonary thermal-dye dilution technique (femoral artery catheter 4-F, Pulsiocath PV 2024L®; Pulsion Medical Systems, Munich, Germany), was instituted. A chest spiral-CT scan was performed in the search for an infectious focus. No pulmonary abscess was found and there were no signs of central pulmonary embolism. Staphylococci were isolated from blood cultures and the antibiotic therapy was changed appropriately. The patient developed an abdominal compartment syndrome that required laparotomy. The abdomen was not closed. Disseminated intravascular coagulation (DIC) with transfusion requirements developed (D-dimers 3 $mg\,L^{-1}$) and the cardiopulmonary function deteriorated further. ITBV was very low (Table 1) and fluid challenges were given to optimize myocardial preload. Although CVP increased progressively, ITBV remained low. This was interpreted as persistent hypovolaemia and fluid loading was continued. However, cardiac output (CO) did not increase and even decreased. Echocardiography was performed and revealed right heart failure as indicated by poorly contracting and fluid overloaded right heart chambers. The left heart was markedly underfilled. The arterial to alveolar PCO_2 difference was first interpreted – especially in view of the computed tomographic scan findings the day before – as a result of pulmonary gas exchange disturbance and it increased progressively (Table 1). Pulmonary embolism was suspected as an underlying cause, but unfortunately the patient died several hours later in hypodynamic shock despite high vasopressor and inotropic support. Autopsy revealed lung oedema and purulent pneumonia. The epicardial coronary arteries were normal but hypoxia-induced patchy necroses within the myocardium were found. A necrotic area, about 5 cm under the diaphragm, with Gram-positive cocci was found. Pulmonary fat embolism was excluded, but repeated multistage massive peripheral pulmonary thromboembolism was considered as the major cause of death.

In our patient, ITBV was low despite high CVP and this was first erroneously interpreted as hypovolaemia. In fact, pulmonary embolism, which was clinically suspected late in the course, could be confirmed *post mortem*. A previous study showed that fluid loading might be an appropriate measure in acute massive pulmonary embolism as indicated by an increase in CO in 12 of 13 patients [5]. In that study, the mean right

Table 1. Haemodynamic and pulmonary variables during the clinical course.

Fluid challenge		BP (mmHg)	CVP (mmHg)	CO ($L\,min^{-1}$)	ITBV ($mL\,m^{-2}$)	EVLW ($mL\,kg^{-1}$)	PaO_2/F_iO_2 (mmHg)	$\Delta a–etPCO_2$ (kPa)
1	Before	115/58 (72)	9	–	–	–	176	2.9
	After	112/53 (69)	14	–	–	–	128	4.1
2	Before	110/60 (78)	19	6.8	792	7.5	118	6.2
	After	95/55 (67)	21	6.4	842	13.1	92	7.3
3	Before	100/50 (67)	25	5.6	735	14.3	117	6.7
	After	105/75 (88)	27	3.8	678	11.8	110	7.0

BP: blood pressure, systolic, diastolic, mean; CVP: central venous pressure; CO: cardiac output; ITBV (normally 800–1000 $mL\,m^{-2}$): intrathoracic blood volume; EVLW (normally <9 $mL\,kg^{-1}$): extravascular lung water; PaO_2: arterial oxygen tension; F_iO_2: inspired oxygen fraction; $\Delta a–etPCO_2$: arterial to end-tidal PCO_2 gradient.

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atrial pressure increased from 9 to 17 mmHg after a volume challenge of 500 mL dextran. In contrast to our case, these patients received simultaneously heparin and thrombolytic therapy. In our patient, DIC-related multistage pulmonary artery obstruction was diagnosed by the pathologist from macroanatomical changes at autopsy. The patient did not receive thrombolysis and fluid loading so may therefore have been without positive effects on CO. We used the transpulmonary indicator dilution technique by which ITBV, as an indicator of myocardial preload, can be obtained. In principle, a low ITBV is measured when the perfused intravascular space is actually reduced. Recently obtained animal experimental data show that ITBV, measured by the transpulmonary thermo-dye dilution technique, was reduced following occlusion of large pulmonary artery branches, while EVLW was underestimated [6]. In our patient, repeated pulmonary embolism during sepsis-related DIC may be considered as responsible for the reduced pulmonary perfusion bed. This would explain the reduced ITBV and underestimated EVLW. The observed increase in EVLW during fluid loading may be interpreted as increasing oedema in the remaining perfused pulmonary bed which was still underestimated. This correspondence demonstrates that if the intravascular capacity is reduced, ITBV must consequently decrease. Theoretically, in this case the decrease in pulmonary blood and left heart volumes must have exceeded the increase in right heart volume. In general, ITBV does not differentiate between the different compartments within the chest. It might be normal if the right heart and the pulmonary tree have an excess of volume whilst the pulmonary venous circulation and the left heart are underfilled. Therefore, in the presence of right heart failure, ITBV may not be regarded as representative of left heart preload.

In our patient, right heart overload and poor left heart filling – findings consistent with relatively acutely increased right heart afterload during pulmonary embolism – were found by echocardiography. Accordingly, echocardiography should be considered when other cardiovascular monitoring techniques yield inconclusive or contradictory information. In conclusion, whenever ITBV is low and CVP high, particularly when CO does not increase following fluid loading, other possible reasons for reduced central blood volume (i.e. pulmonary embolism, tension pneumothorax, etc.) should be suspected and further diagnostic measures considered.

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Prophylactic continuous intravenous ephedrine infusion for elective Caesarean section under spinal anaesthesia

EDITOR:

Maternal hypotension is the most frequent complication of a spinal anaesthetic for Caesarean section. A range of stratagems is currently used to prevent or minimize hypotension, but there is no established ideal technique. Different approaches include prehydration with crystalloids or colloids, lower limb compression, and pharmacological prophylactic protocols using ephedrine, phenylephrine, angiotensin II or metaraminol [1–4].

Loughrey and colleagues described the prophylactic administration of intravenous (i.v.) boluses of ephedrine for elective Caesarean section under spinal anaesthesia [1]. They concluded that a prophylactic

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bolus of ephedrine 12 mg i.v. given at the time of the intrathecal block – associated, if necessary, with rescue boluses – leads to a lower incidence of hypotension during spinal anaesthesia compared with i.v. rescue boluses alone.

We use compound sodium lactate solution (500 mL) given as a preload over 10 min before the intrathecal injection of hyperbaric bupivacaine. The bolus method is chosen over an i.v. infusion to provide a fast and reliable early prophylactic dose of vasopressor as the rapid sympathetic blockade develops.

In the recent study of Kee and colleagues, the smallest prophylactic i.v. ephedrine dose was 30 mg. In that study, the parturient was preloaded with lactated Ringer's solution 20 mL kg^{-1} and the spinal block was performed using hyperbaric bupivacaine 10 mg and fentanyl 15 µg [5]. In another trial by Vercauteren and colleagues, a single i.v. dose of 5 mg limited the severity of hypotension in prehydrated subjects receiving hyperbaric bupivacaine 6.6 mg and sufentanil 3.3 µg [6]. Jackson and colleagues concluded that volume preloading was not essential to prevent spinal-induced hypotension in Caesarean section. They used a prophylactic infusion of ephedrine 60 mg in Hartmann's solution (500 mL) given according to maternal arterial pressure [7].

During 2001, our Medical Centre performed 181 Caesarean section deliveries of which 94% were under spinal anaesthesia. The usual policy we adopted consisted of prehydration with lactated Ringer's solution (300 mL) over 10 min. At the same time, hyperbaric bupivacaine 0.8–1 mL 1%, combined with fentanyl 25 µg, was given intrathecally plus a slow baseline infusion of ephedrine 25 mg in 500 mL lactated Ringer's solution i.v. The aim of the ephedrine infusion was to keep arterial pressure at a baseline level and never below 90 mmHg systolic. This infusion was continued until the extraction of the baby and then it was gradually stopped. We did not observe rebound hyper- or hypotension. The mean total dose of ephedrine used was 12.5 ± 2.3 mg (mean ± SD). Only five patients required rescue boluses of ephedrine 5 mg because of symptoms suggestive of hypotension, e.g. nausea and vomiting, without any recorded fall of arterial pressure.

We agree that the prophylactic ephedrine dose is safe and efficacious in reducing the incidence of maternal hypotension, but we also believe that an infusion of the drug, according to the patient's requirements, is much safer than the bolus technique. In our patients, the preload was reduced to 300 mL – similar to Jackson and colleagues' study [7] in which the minimal preload was 200 mL – while the concentration of ephedrine in our protocol was 0.05 versus 0.12 mg mL^{-1}.

In conclusion, further randomized control trials would be useful to determine the optimal dose of ephedrine, the method of administration, the volume and the kind of preload.

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ERRATUM:

Krenn H, Deusch E, Balogh B, Jellinek H, Oczenski W, Plainer-Zöchling E, Fitzgerald RD. Increasing the injection volume by dilution improves the onset of motor blockade, but not sensory blockade of ropivacaine for brachial plexus block. *Eur J Anaesthesiol* 2003; **20**: 21–25.

Unfortunately the statement in the Methods section on page 22 that 'All blocks were performed by one investigator (H. K.) using the interscalene approach described by Winnie and colleagues [9]' is incorrect because axillary plexus block, not interscalene block, was used in the investigation. This sentence should be replaced by 'All blocks were performed by one investigator (H. K.).' This error, for which the authors accept responsibility, is very much regretted.