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Re: 'A study of factors influencing surgical cesarean delivery times in an academic tertiary center'

We read with interest the article by Gonzales Fiol et al.[1] published online in the journal in January 2018. The authors conducted a retrospective review of caesarean deliveries over a 12-month period to determine caesarean delivery times and identify factors influencing operative time and preference for a particular anaesthetic method. Although the authors examined the influence of several pertinent variables on operative time, we consider that premature conclusions were drawn that were not supported by the data.

Firstly, when undertaking a linear regression analysis, a key assumption is the normal distribution of the residuals (the distance of the predicted value from the measured value). Given the skewed distribution of operative time and the low coefficient of determination (R^2), we suspect the residuals of the data were not normally distributed, thus violating this condition. The authors did not report the assessment of this assumption.

Secondly, as identified by the authors, the variables studied account for a small part (18%) of the variation in caesarean delivery operative time. In presenting a predictive model in the form of the decision tool in Fig. 2, the authors infer clinical utility. However, a coefficient of determination (R^2) of only 0.18 suggests the predictive model is a poor fit and does not predict operative time accurately. Furthermore, the statistical model has not undergone an assessment of its predictive power. The convention when preparing and assessing a predictive statistical model is to undertake an analysis on a split data set.[2,3] Firstly, a "training" subset of data is used to formulate the model and then its performance is validated on a "testing" subset of data. If this had been undertaken, the authors could have demonstrated the model's ability to accurately identify cases of greater than 90 minutes duration, accompanied by the sensitivity and specificity. This would have evidenced the true utility and transferability of this model into clinical practice.

We believe this paper does not validate the predictive model proposed and suggest it is an inadequate tool to influence clinical decisions such as anaesthetic technique.

S. Singh, A. Clark
Department of Anaesthesia and Intensive Care, University Hospital Crosshouse, Kilmarnock, Scotland, United Kingdom
E-mail address: sharandeepsingh@nhs.net

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Ed. The authors of the article to which this letter refers did not respond to the invitation to reply.

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Dosing an unintentional intrathecal catheter with programmed intermittent epidural bolus settings may not produce hypotension

Maintenance of epidural analgesia by programmed intermittent epidural boluses (PIEB) delivers identical volumes and doses of epidural medication at scheduled intervals, and demonstrates more extensive epidural spread than continuous infusion.[1] There is limited information on how an unrecognized intrathecal catheter might present during PIEB settings,[2] especially if the catheter was placed as part of a combined spinal-epidu-

ral (CSE) technique and the epidural test dose was omitted.[3,4]

A healthy term primigravid woman in labor requested neuraxial analgesia and had an uncomplicated CSE technique. A 19-gauge open-tip flexible wire-reinforced catheter (Arrow FlexTip Plus®, Reading, PA, USA) threaded easily and aspiration directly from the catheter, without a filter, showed no return of cerebrospinal fluid (CSF) or blood. An epidural test dose was not given. Our institutional PIEB regimen was established using a CADD-Solis Infusion Pump (Smiths Medical, St Paul, MN, USA). The regimen uses 0.0625% bupivacaine with 2 μg/mL fentanyl, a 10 mL PIEB bolus every 45 min, starting 30 min after epidural placement, and a 5 mL patient-controlled epidural analgesia (PCEA) bolus with a 10-min lockout interval. The patient received three PCEA doses over the first two hours. She noted her legs were completely numb about two hours after placement but was unconcerned and did not notify a nurse. The woman's blood pressure was measured every 5 min for 15 min and then every 30 min. She received one-on-one nursing care.

The nurse attending her noted a denser motor blockade about five hours after initiation and called the anesthesiologist, who demonstrated a block to ice to T2 and was able to easily aspirate 5 mL of clear fluid from the epidural catheter. A review of her blood pressure readings over five hours showed only mild asymptomatic hypotension, in the range of 90–133 mmHg for systolic and 51–62 mmHg for diastolic blood pressure. She had reported intermittent mild nausea which she did not relate to the PIEB or PCEA doses. She delivered vaginally 2.5 h later, remaining comfortable without additional analgesia. The intrathecal catheter was removed the following day, she did not develop a post-dural puncture headache, and there were no adverse maternal or neonatal outcomes.

In this case, an inadvertent intrathecal catheter with PIEB drug administration presented with dense motor blockade. This is in contrast to another case report in which the woman presented with profound symptomatic hypotension.[2] In that case, uneventful catheter placement as part of a CSE technique was followed by hypotension, with new-onset nausea and dyspnea, after the fifth PIEB dose. The passage of a soft-tip, flexible, wire-reinforced epidural catheter through an intact dura is considered unlikely,[5] so the authors theorized that the catheter had been placed subdurally initially and had then punctured the arachnoid mater and migrated intrathecally. An alternative hypothesis is that the dura was unknowingly "nicked" by the Touhy needle and the catheter later migrated through this weakened spot. A further hypothesis is that the small dural hole intentionally created during the CSE technique provides an entry point for the catheter, although the catheter diameter in this and the other report[2] was 19-gauge whereas the spinal needles used were 26- and 27-gauge. In both cases the catheter functioned as if positioned epidurally for several hours, prior to the patients developing symptoms suggesting intrathecal drug administration, with an intrathecal location proven by aspiration of CSF.

Intravascular migration of catheters is a well-known complication of epidural analgesia and occurs more frequently with stiff, compared with flexible, catheters. The intrathecal migration rate of stiff and flexible catheters is unknown and assumed to be much lower.[6–8] The other similar report involved a different catheter (B Braun Perifex® FX Springwound, B-Braun Medical Inc., USA) to the one that we used, which suggests that all flexible wire-reinforced catheters are at risk of intrathecal migration after insertion.

We write not to disparage the PIEB technique but to encourage vigilance. There are few descriptions of the effect of unintentional PIEB dosing of an intrathecal catheter. Given the greater epidural spread of local anesthetic with PIEB, boluses administered by the pump theoretically pose a greater risk of high block than a continuous infusion. A safety feature of PIEB dosing may be that symptoms develop slowly and allow recognition of intrathecal spread before progression to respiratory distress. Intrathecal migration of an epidural catheter poses a bigger risk after manual boluses given for inadequate pain control or cesarean delivery, and identification of catheter migration is vital. Finally, while not fail-safe,[9] all hand-delivered doses should be given incrementally after direct aspiration of the catheter, even if previous aspiration tests have been negative.

E. Dinges, M. Foulks, C. Kent
Department of Anesthesiology and Pain Medicine, University of Washington, Seattle, WA, USA
E-mail address: edinges@uw.edu

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Benign paroxysmal positional vertigo presenting as persistent vomiting in a parturient using epidural analgesia

A healthy 26-year-old woman, G1P0, was admitted in labor and an epidural catheter was inserted uneventfully. Maintenance of epidural analgesia was with levobupivacaine 1.25 mg/ml and fentanyl 1.25 μg/ml at a rate of 6 mL/h, using patient-controlled epidural analgesia (6 mL on demand at a 15 min lockout interval). This proved effective and without significant complication.

Nausea and vomiting were noted two hours after the insertion and these symptoms persisted for 10 hours despite intravenous fluid administration (normal saline 1 L) and anti-emetic treatment (intravenous metoclopramide 10 mg). No other gastro-intestinal symptoms, such as abdominal pain, tenderness or diarrhea, were noted. Her blood pressure was within normal limits and she had a mild tachycardia. We considered it unusual for a parturient to have persistent nausea and vomiting so did a brief neurological examination, in order to rule out serious causes such as increased intracranial pressure induced by pre-eclampsia. Horizontal rotatory nystagmus was a significant finding and the woman volunteered that vertigo, nausea and vomiting were induced by changing position. Benign paroxysmal positional vertigo (BPPV) was our provisional diagnosis and the Dix-Hallpike test was performed. With the patient sitting upright, we held her head in 45° rotated to the left. She was then instructed to keep her eyes open while she was taken backward to lie down with a pillow under her shoulders to extend her head 20°. Up-beating, left horizontal rotatory nystagmus with a five-second latency and lasting for nearly 30 seconds was induced, and the patient experienced vertigo and vomiting again after the maneuver. Left posterior canal BPPV was diagnosed due to the positive result of the left-sided Dix-Hallpike test. Following the diagnosis, we carried out the Epley maneuver to treat the patient (Fig. 1). The initial maneuver was the same as the Dix-Hallpike test, holding the woman's head with 45° rotation to the left when she sat upright, before taking her backward to a supine position with her head rotated 45° to the left and extended 20° by the pillow. This position was maintained for 30 seconds before turning her head 90° to the right for 30 seconds. Next, we had the patient roll on her right shoulder and turn her head a further 90° to the right, making her head facing down at a 45° angle for 30 seconds. Finally, we brought her to the upright sitting position to complete the maneuver. Her symptoms were relieved after the treatment. She delivered her infant 18 hours after commencing epidural analgesia and was discharged three days later without a recurrence of BPPV.

Benign paroxysmal positional vertigo may be caused by cupulolithiasis. It is the most common cause of dizziness or vertigo, and some studies suggest an association with estrogen change and pregnancy.[1–3] Prolonged bed rest may be another cause of BPPV in pregnant women, especially if they are usually advised to sleep on their left side to decrease vena caval compression. The gold standard to diagnose BPPV is the Dix-Hallpike test and treatment is by the Epley maneuver or with antihistamines and benzodiazepines. In our opinion, the Epley maneuver is the first-line treatment, not only because of cost-effectiveness but because it is a physical rather

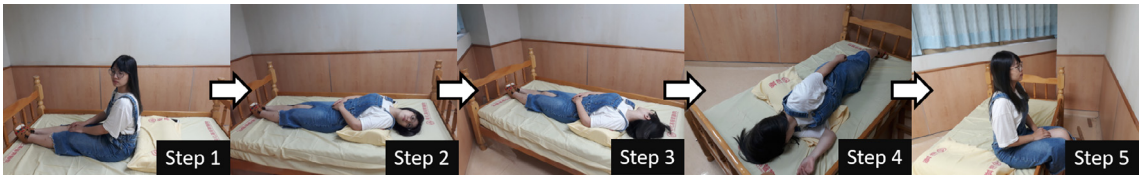

Fig. 1 Dix-Hallpik test and Epley maneuver. Step 1: Let the patient sit upright, with her head rotated 45° to the left. Step 2: Take the patient backward to a supine position with her head rotated 45° to the left. Use a pillow under her shoulders to keep her head in 20° of extension. Step 3: Ask the patient to turn her head to the right until it is rotated 45° to the right. Step 4: Move the patient to the right lateral decubitus position, with her head still rotated 45° to the right. Step 5: Help the patient to sit upright, with her head in the neutral position. Dix-Hallpik test for left posterior canal BPPV: Step 1 to 2. Epley maneuver for left posterior canal BPPV: Step 1 to 5, maintaining each step for 30 seconds. For diagnosing and treating right posterior canal BPPV, use the same steps but reverse the left and right sequence. BPPV: benign paroxysmal positional vertigo.