ultrasound examination at the T3 level identified the area between the T3 transverse process (TP) and the erector spinae muscle (ESM). Using an aseptic technique after local anesthesia, an insulated needle (Pajunk, SonoPlex Cannula, Geisingin, Germany) was introduced using an in-plane approach with a high frequency transducer (Sonosite, M Turbo, Bothell, MA) until bone contact was made. Hydrodissection with saline (Fig. 1) revealed linear spread above the TP and below the fascia of the ESM. After a negative aspiration test, 20 mL of a mixture containing plain lidocaine 0.5% and bupivacaine 0.25% with 40 mg triamcinolone was injected. Assessing by means of response to cold, dermatomal spread from C5 to T3 was revealed.

The patient had a positive response to treatment at two and eight weeks after the procedure, describing a global reduction in pain of 85% proximally (cervical and scapular area) and 50% distally (forearm) at two weeks and 90% reduction of both symptoms at eight weeks. The patient tolerated the physical therapy program and no emergency room visits were recorded. She remained symptom- and recurrence-free 12 weeks after the ESP injection.

To our knowledge this is the first report of an ESP block used for spinal-related pain in the obstetric setting. This block may be a suitable alternative for the management of acute radicular symptoms in pregnant patients and avoid the potential risks of surgical intervention during pregnancy. Further prospective studies are necessary to define the role of ESP block for spinal-related pain in non-pregnant and pregnant patients.

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Acute hypoglycemia during cesarean delivery in a patient with type-1 diabetes mellitus

Insulin resistance increases during pregnancy due to high levels of cytokines produced by the placenta.[1] In patients with type-1 diabetes mellitus (DM) requiring administration of insulin, the amount of insulin increases as pregnancy progresses, while the risk of a hypoglycemic attack increases after placental delivery as a result of a rapid reduction in insulin resistance.[2] We managed a 39-year-old patient (gravida 9, para 6), at 37 weeks' gestation, who had type-1 DM (high hemoglobin$A1_C$ of 70.5 mmol/mol). The DM had been diagnosed during her sixth pregnancy, during which her blood glucose was controlled using 20 units of a rapid-acting insulin analogue before every meal and 48 units of a long-acting insulin analogue before sleep. She had an uneventful current pregnancy, free of hypoglycemia events, and her blood glucose levels ranged from 4.5 to 7.4 mmol/L after 32 weeks' gestation. Her blood glucose was managed using 24 units of rapid-acting insulin analogue before every meal and 55 units of long-acting insulin analogue administered before sleep. On the day of delivery, five days after hospitalization for oligohydramnios, her pre-prandial level was 5.6 mmol/L and 24 units of rapid-acting insulin analogue were administered. After 90 minutes without oral intake and no blood glucose measurement, she underwent an urgent cesarean delivery (followed by tubal ligation) for late deceleration of the fetal heart rate. Spinal anesthesia was performed using 11 mg 0.5% hyperbaric bupivacaine, 10 μg fentanyl and 0.1 mg morphine, achieving a T4 sensory level. Intra-operatively, a total of 1050 mL of intravenous Ringer solution (450 mL before delivery and 600 mL until the end of surgery) was administered. Systolic blood pressure was maintained at 110 mmHg or more with 50 μg boluses of phenylephrine. Intra-operatively, her arterial oxygen saturation was 98–99% on oxygen at 5 L/min administered via face mask. She delivered a healthy baby with a blood glucose level of 3.8 mmol/L. Seventy minutes

after placental delivery, in the operating theatre, she began snoring and was not responding to verbal and physical stimulation. Her blood pressure was 128/86 mmHg and arterial oxygen saturation 99%. Her blood glucose was checked and found to be unmeasurable (Medisafe FIT®, Terumo, Tokyo, Japan), meaning that the concentration was below 1.1 mmol/L. Following rapid intravenous administration of 20 mL 50% dextrose, her blood glucose increased to 5.6 mmol/L. She regained consciousness and was discharged to the obstetric ward, after receiving an additional 20 mL. Ninety-five minutes after the placental delivery, in the obstetric ward, her blood glucose decreased to 3.8 mmol/L and a further 20 mL of 50% dextrose was administered. Subsequently, she resumed oral intake of food and did not experience further hypoglycemic events. On the first postoperative day her insulin requirement was 8 units of rapid-acting insulin analogue before every meal and 10 units of long-acting insulin analogue administered before sleep.

Several alternative diagnoses were considered for her sudden loss of consciousness, including cerebrovascular disease and high spinal block. This acute hypoglycemic event may be explained by a combination of factors. The patient's insulin requirements were higher during this pregnancy than during previous ones, possibly related to greater release of cytokines by the placenta during the current pregnancy. Insulin sensitivity increases suddenly following placental delivery.[2] In addition, high levels of placental tumour necrosis factor-alpha play an important role in diabetic patients in the disappearance of insulin resistance immediately after placental delivery.[3–5] Furthermore, decreased levels of catecholamines after spinal anesthesia and intrathecal fentanyl may contribute to acute hypoglycemia.[6] We did not have a maternal blood glucose level available before placental delivery, but it was likely equal to or more than 3.8 mmol/L, similar to the umbilical vein blood glucose level.[7] This suggests an abrupt fall in maternal blood glucose after delivery. She had not experienced hypoglycemia at the time of her sixth delivery, when the DM was diagnosed, possibly related to the absence of sympathetic inhibition induced by anesthetics and the decreased levels of tumour necrosis factor-alpha. To avoid acute hypoglycemia following placental delivery in patients with type-1 DM, this phenomenon must be considered. Furthermore, we recommend measuring pre-operative maternal blood glucose levels again after placental delivery. Finally, administration of fluid containing glucose or a dextrose and insulin titrated infusion should be considered, while monitoring the blood glucose and for symptoms of hypoglycemia.

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Propofol, Zola, and the modern obstetric rapid sequence induction

We read with great interest the results of the latest national United Kingdom survey of practice on the conduct of rapid sequence induction (RSI) for caesarean section[1] and its findings of continuing heterogeneity in anaesthetic practice with respect to this clinical context. Last year, at King's College Hospital, we carried out a similar study which was recently presented at the 2019 Winter Scientific Meeting of the Association of Anaesthetists.

The case against thiopentone is bolstered by examples of inappropriate dosing, with underdosage carrying a risk of awareness[2] and inappropriately high dosing being implicated in maternal deaths.[3] We consider that these dosing misjudgements may well reflect a lack of familiarity with thiopentone. As the adage goes, the safest drug in a clinical scenario is often the one most familiar to the individual administering it.

Desai et al. noted that the proportion of anaesthetists who theoretically support an alteration of first-line induction agent from thiopentone to propofol is double that of those who have already effected this change in practice (82% vs. 41%) – and that this speaks to unknown barriers to the use of propofol in this context. No doubt fear of criticism in an already litigious subspecialty plays a role. We suggest that a parallel exists between this and 'Zola's triggers', namely the reasons that lead patients to consult a doctor.[4] One of these trig-