Baricity is the density of the local anesthetic solution divided by the density of cerebrospinal fluid (CSF). Research from the Ottawa Hospital cited by Dr. Tinits defined the densities of CSF, local anesthetic solutions, and common additives.[1] The density of CSF in the parturient at term is 1.00033 ± 0.00010 g·mL^{-1}, while the densities of water and saline are 0.9933 and 0.9995 g·mL^{-1} respectively. The densities of isobaric bupivacaine, fentanyl, and saline solutions are 0.9993 g·mL^{-1}, 0.9932 and 0.9995 g·mL^{-1}, respectively. While we did not directly measure the density of the solutions used in this study, the components of the 4.5 mg bupivacaine solution are relatively hypobaric when compared to CSF, but range within 0.0003 g·mL^{-1} of solutions considered isobaric by clinicians. As such, it would seem unlikely that a strongly hypobaric solution was responsible for the observed cephalad extension of anesthesia.

With regards to the use of tetracaine, a 1% solution in saline has a specific gravity of 0.9995 g·mL^{-1}. Given the relatively small difference in density between saline and water noted above, a 1% tetracaine solution in water could be expected to be of a similar density to the solutions employed in our study. We cannot comment directly on the use of tetracaine for Cesarean delivery, but our anecdotal experience suggests that traditional doses of 12 to 15 mg are associated with a higher frequency of hypotensive events and deeper levels of motor block than with bupivacaine. Continuous spinal-epidural anesthesia is, indeed, a useful technique but was not the subject of the present paper.

With respect to the comments by Drs. Bruyère and Benhamou, we recognize that lower doses of fentanyl have been advocated for both Cesarean and labour analgesia. During the design phase of our trial, we were influenced by the 6% rate of analgesic supplementation reported in Ben-David's study that used 25 μg doses of fentanyl.[2] As there was evidence supporting the use of larger doses of fentanyl,[3,4] we opted to proceed with the 50 μg dose. The results of our study suggest this larger dose was not associated with poor neonatal outcomes and, while not reported in the manuscript, we found no evidence of maternal respiratory depression or naloxone use in our cohort. Subjects receiving the 50 μg dose of fentanyl in either arm of our study preferred the present technique to their previous Cesarean delivery. In the spirit of continuous quality improvement, we have re-evaluated our dose of opioid in light of the high rate of pruritus and nausea reported in the present study. Our current clinical practice is to administer 5-mg isobaric bupivacaine combined with fentanyl 10–15 μg and morphine 100–150 μg intrathecally. We have yet to formally assess the impact of this change on side effects, but we have not observed an increased requirement for postoperative analgesic supplementation.

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Diagnosis of brain death with the electroencephalogram

To the Editor:

In a recently published, single-case study, Rimmelé *et al.*[1] claim that the electroencephalogram (EEG) is not an adequate test to confirm the diagnosis of brain death. Since additional angiography disclosed residual cerebral blood flow (CBF), it was concluded that electrocerebral silence was misleading and, although clinical criteria were fulfilled, the patient was not assessed as being brain dead. I submit that the authors' conclusions are not warranted.

The patient was examined neurologically and two EEGs were recorded shortly after injection of etomidate, 20 mg. The half-life of etomidate is 0.5 to 1.25 hr and that of its metabolites is 4.5 hr, so it is not appropriate to state that any sedation was absent. Usually we measure plasma levels to ensure they are below therapeutic range or we wait four times the longest half-life. Here, clinical examinations, as well as EEGs, may still have been influenced by sedatives.

Apparently, the movement of the right arm was spinal - as the authors themselves admit - and did

not necessitate another exam. It was an example of residual reflex activity or automatism.[2]

If, indeed, there was no significant residual sedative action and if the clinical exam, as well as the EEGs were valid, there was absolutely no need for an angiography. Performing superfluous investigations always raises the predicament that discrepancies may create a dilemma that can only be solved by clinical acumen. It is more likely that the angiographic results were misleading. There are cases where primary parenchymal damage ensues without brain swelling, such that brain death occurs, not via cerebral edema, transtentorial herniation and lack of perfusion, but via cellular death. The limitations of angiography in diagnosing brain death have been repeatedly addressed.[3] There could be persistence of CBF despite brain death and angiography may be misleading because of reperfusion.

Thus, most countries world-wide consider the EEG as a reliable and valid adjunct in the clinical diagnosis of brain death.[4] In Germany, an EEG is even considered mandatory in primary infratentorial damage. Precise quality standards have been developed to exclude any misinterpretations.[5] On the other hand, angiography, being potentially harmful, is being abandoned by many. I consider the conclusions drawn by Rimmelé *et al.*[1] to be unwarranted and believe there is no need to change the use and the established standards of EEG in the diagnosis of brain death.

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Reply:

We sincerely thank Dr. Lang for his comments regarding our case report in which a preliminary, clinical diagnosis of brain death was confirmed by two electroencephalograms (EEGs) and subsequently ruled out by cerebral angiography.[1]

Contrary to Dr. Lang's correspondence, the statement, "EEG is not an adequate test to confirm the diagnosis of brain death", is not drawn from our "single-case study", but obtained from recent Canadian recommendations for brain death diagnosis[2] based on recent and exhaustive reviews of literature.[3,4] In all of these documents, EEG is no longer recommended as an ancillary test, notably because of the numerous false positives and false negatives observed with this assessment.

Concerning the etomidate injection, it was performed 22 hr before the first EEG and 27 hr before the second one. Therefore, we can state decisively that the results of the EEGs and the clinical examinations were not influenced by sedatives. Dr. Lang stated that the movement of the right arm was "apparently" spinal. Our purpose for performing the angiography was to replace the word "apparently" by the word "certainly".

Due to the persistence of cerebral blood flow found at angiography, it was concluded that the patient was not brain dead. Indeed, the pathophysiological definition of brain death in many countries remains the irreversible interruption of cerebral blood flow.[2,5]

In conclusion, our case report must be considered an illustration of the limitations of the EEG for brain death diagnosis. Once again, the complexity of brain death diagnosis is revealed and differing opinions exist amongst experts from different countries. Therefore, an international harmonization of criteria for brain death diagnosis is essential.

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Survey of attitudes of anesthesiologists to peripheral nerve blocks

To the Editor:

The issue of potential risk to patients receiving peripheral nerve blocks is of great importance. Brull *et al.*[1] recently published an estimate of neurological risk related to regional anesthesia and observed rates as low as 0.03 per 100, following supraclavicular block, to an alarming 2.84 per 100, after interscalene block. Yet, despite this significant risk, Brull *et al.*[2] reported that only 58% of academic anesthesiologists performing regional anesthesia routinely disclose the risks of permanent neuropathy to their patients undergoing continuous nerve block; and only 43% of the anesthesiologists surveyed disclosed the risk of paralysis.

As a group, anesthesiologists are clearly the most familiar with the potential risks and benefits of regional anesthesia. However, often patients are incompletely informed about potential complications.[1] Proper, informed consent is imperative; as we cannot assume patients have the same risk tolerance as do anesthesiologists. The analysis of the risk/benefit ratio takes into consideration many factors, such as the patient's pain tolerance and the expected intensity of postoperative pain.

Concern regarding the incidence of neuropathy, and other complications related to peripheral nerve blocks, led us to conduct a survey of our department members. We sought to assess our specialty's attitudes about regional anesthesia. Specifically, we evaluated our acceptance of peripheral nerve blocks for patients and compared that to our acceptance of similar techniques for ourselves, if undergoing a surgical procedure.

Following Research Ethics approval, a survey was circulated by mail to all anesthesiologists at four academic hospitals in Ottawa. Of the 83 surveys sent, 59 were completed (response rate 71%). The Table summarizes the principal results. Responders had a wide range of clinical anesthesia experience (from one to over 20 yr in practice). Of the 59 who responded, 12 (20%) rarely, if ever, performed blocks; 36 (61%) performed one to five blocks/month; six (10%) performed six to ten blocks/month; and only five (8%) performed > ten blocks/month. The Table shows the types of peripheral nerve blocks administered by anesthesiologists in our institutions. When questioned about being a block recipient, 36% of respondents were happy to receive all blocks; 61% indicated they would decline one or more blocks; and 3% would refuse all blocks. The main blocks causing concern were lumbar plexus psoas compartment, proximal sciatic, and supraclavicular blocks, which are also the least frequently performed blocks on patients at our institutions. The most common reasons given by anesthesiologists for refusing a peripheral nerve block included: risk of injury to lung, blood vessels, or other structures (63%); risk of nerve injury (44%); and the expected discomfort from nerve block placement (22%). Of lesser concern were: local anesthetic toxicity (5%); injury to, or from, an insensate limb (5%); and infection (0%).

TABLE Anesthesiologists' acceptance of peripheral nerve blocks for patients and for themselves

Peripheral nerve block	*Number (%) of anesthesiologists who perform each block (n = 59)*	*Number (%) of anesthesiologists who would decline each block for themselves (n = 59)*
Interscalene	45 (76)	5 (8)
Supraclavicular	14 (23)	15 (25)
Infraclavicular	14 (23)	10 (16)
Axillary	32 (54)	5 (8)
Elbow	7 (11)	10 (16)
Wrist	15 (25)	7 (11)
Intravenous regional anesthesia	49 (83)	7 (11)
Psoas compartment	0 (0)	22 (37)
Femoral	51 (86)	6 (10)
Proximal sciatic	10 (16)	13 (22)
Popliteal	26 (44)	4 (6)
Ankle	39 (66)	9 (15)

Few studies have addressed anesthesiologists' attitudes and preferences relating to anesthesia techniques for themselves.[3] This survey demonstrates that a large proportion of anesthesiologists are willing to accept the potential risks of peripheral nerve blocks.