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In Reply:—The use of a feedback device for routine calibration of anesthetic gas monitor is common. Datex, Cricare, and Ohmeda all use a small amount of room air to maintain specified performance levels. Autocalibration occurs at 1.5, 2.5, 5, 10, 20, and 40 min intervals after Rascal II (Ohmeda). The BOC Healthcare Group, Edson, NJ) startup. Subsequent autocalibrations are initiated only at 80 min intervals. The Rascal II displays the message "CALIBRATING" on the screen, notifying the user of the interruption in the monitoring during the calibration process. The Rascal II does allow the clinician to postpone calibration at any point in the anesthesia. Nominally, 42 ml of room air is aspirated during the brief, 12-s calibration cycle. Only in the first three autocalibrations is argon used in the calibration process. Successful, subsequent autocalibrations do not use argon.

The Rascal II Operations and Maintenance Manual provides information on the use of room air in calibration. Ohmeda welcomes the comments of Stevens et al. in recognizing the potential sources of nitrogen in the breathing circuit and the need to monitor nitrogen in low flow situations.

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Spinal Meningitis Masquerading as Postdural Puncture Headache

To the Editor:—We report a case where vigilance by our Anesthesia Pain Management Clinic aided in prompt diagnosis and treatment of a patient with unrecognized spinal meningitis. A 39-yr-old healthy man underwent uneventful outpatient extracorporeal shockwave lithotripsy with combined spinal epidural anesthesia (27-gauge Whitacre spinal needle through 18-gauge Tuohy needle, Becton Dickinson, Franklin Lakes, NJ). Two days after surgery, the patient experienced a bilateral, occipital-temporal headache that worsened with an upright position. The patient was evaluated by his primary care physician and referred to the pain management clinic for an epidural blood patch with a presumptive diagnosis of postdural puncture headache. Further evaluation at the pain management clinic revealed acute development of photophobia and severe headache (6/10 on a verbal pain scale) while supine that worsened when upright (9/10). Vital signs were remarkable for a tympanic membrane temperature of 38.5°C. Physical examination was remarkable for an ill-appearing man with a positive Kernig sign of meningeal irritation. The patient’s spinal needle puncture site was nonerythematous and nonpeller.

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blood cell count was acquired, which revealed a leukocytosis (14,000/mm³) with 88% polymorphonuclearleucocytes. Based on these physical and laboratory findings, we decided to proceed with a diagnostic lumbar puncture rather than an epidural blood patch. Lumbar puncture revealed turbid cerebrospinal fluid with numerous gram-positive diplococci. Additional cerebrospinal fluid revealed 9,000/mm³ white blood cells (normal at our institution, 0–5/mm³). 184 mg/dl protein (normal, 18–58), and 11 mg/dl glucose (normal, 40–70). The patient was transferred to the internal medicine service, treated with vancomycin and ceftazidime, and discharged in good condition after 5 days in the hospital. Although no organisms were cultured from the cerebrospinal fluid, the Infectious Disease service believed that the patient’s findings were consistent with bacterial meningitis caused by Streptococcus pneumoniae.

Postural puncture headache is an uncommon (incidence 1–5%) but expected complication after spinal anesthesia with small, noncutting needles. Spinal meningitis is an extremely rare finding after spinal anesthesia. In this case, a causal relation between the patient’s meningitis and spinal anesthesia is unclear, because S. pneumoniae is the most common community-acquired pathogen for spinal meningitis in adults. Pneumococcal meningitis is a life-threatening medical emergency (approximately 25% mortality), and delay in instituting appropriate therapy worsens outcome. Prompt diagnosis and institution of antimicrobial therapy aided in this patient’s full recovery. As anesthesiologists expand into perioperative medicine, we encourage continued vigilance as consultants outside the operating suites.

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Drawing Conclusions from Pollock et al.: Limitations Imposed by Study Design

To the Editor — I read with interest the recent study of transient radicular irritation (TRI) by Pollock et al. 1 This is the first such study to be reported from the United States, and the results provide important confirmation of data from two European institutions. 2-4 These findings verify that transient neurologic symptoms frequently occur when lidocaine is used for spinal anesthesia and reinforce concern about the continued intrathecal use of this anesthetic. However, some aspects of the study’s design and analysis warrant comment.

The strength of a randomized trial rests on “designing interventions that have only one major difference between any two study groups.” 5 It is, therefore, surprising that the authors chose to administer a hyperbaric solution of 5% lidocaine with 0.2 mg epinephrine, a hyperbaric solution of 0.75% bupivacaine without epinephrine, and an isotonic glucose-free solution of 2% lidocaine without epinephrine. (Though not specifically stated, it is also likely that the glucose concentrations of the hyperbaric lidocaine and hyperbaric bupivacaine solutions differed.) Therefore, among the three experimental groups, there is no single comparison between any two that differs by only one relevant variable. This flaw in design hinders analysis of the potential effects of anesthetic agent, anesthetic concentration, glucose, baricity, and epinephrine, and sends the discussion into a tailspin of circular reasoning. For example, the possible contribution to TRI of one relevant factor such as epinephrine is ignored when interpreting the effect of a second, such as lidocaine concentration; conversely, the concentration of lidocaine is assumed to have no effect when interpreting the effect of epinephrine. The authors do offer a partial explanation for the choice of anesthetic solutions, stating that “Epinephrine was specifically included in only patients receiving 5% hyperbaric lidocaine in an attempt to determine whether the addition of epinephrine might increase the incidence of TRI.” However, this reasoning would be valid only if epinephrine were the sole variable in question (but, then, there would be no reason to systematically vary anesthetic concentration or glucose content).

Because of the study’s multiple variables, we must try to simplify interpretation by identifying factors likely to be irrelevant to the outcome variable. For example, the article references work in which it is demonstrated that glucose does not affect the potential of intrathecally administered lidocaine to induce sensory impairment in the rat. 6 However, caution must be used in extrapolating to transient clinical effects — as appealing as the concept may be, it has not been established that anesthetic-induced neurologic injury and transient pain/dysesthesia share a common mechanism. In addition, preliminary data generated in the same model sharply conflict with findings in the current study (i.e., in the rat, adding epinephrine increases neurologic impairment induced by intrathecal lidocaine.” Although we must be careful extrapolating from animal data, we must be even more cautious embracing unproven concepts, such as assuming that epinephrine might increase the incidence of TRI without entertaining the possibility that it might be protective.

The authors tried to “eliminate relative anesthetic potency as a possible cause of TRI” by basing their doses on potency data reported in an

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