analyzer. The vacuum pump maintains fine regulation of the desired 50 or 150 mL/min aspiration rate by drawing largely from the sample port and partly from air inside the monitor. The air is added after the sample is analyzed. When the “Auto Cal in Progress” is activated, circuit gas is sampled more rapidly for 15-20 s, and a higher exhaust gas flow also occurs.

Exhaust gas volume also exceeds sample gas volume with the Raman scattering analyzer (RASCAL II, Ohmeda, The BOC Healthcare Group, Edison, NJ). The exhaust gas contains air during regular use and argon during the calibration procedure. Sampling from the patient is interrupted during calibration. The RASCAL II has a single nominal aspiration rate of 210 mL/min.

In a small survey of 3 POET II, 3 POET IQ, and 2 RASCAL II monitors used daily in our operating rooms, we obtained the following data. With the POET monitors, exhaust exceeded sample flow rate by 4-19 mL/min at the 50 mL/min setting and 9-19 mL/min at the 150 mL/min setting. With the two RASCAL II monitors, the exhaust exceeded sample flow rate by 16 and 23 mL/min. When sampling oxygen only with the POET analyzers, nitrogen concentration of the exhaust gas ranged between 9.3% and 19% at the 50 mL/min setting and 11% and 18% at the 150 mL/min setting. With the RASCAL IIs, the exhaust gas nitrogen values were 5.9% and 11%.

The monitors functioned at or very near manufacturer’s specifications. However, returning the monitor exhaust to the circuit constantly adds air to the circuit. With the RASCAL II, a small amount of argon also is added during its calibration cycle.

Wendell C. Stevens, M.D.
J. A. Nash, M.B., Ch.B.
Richard Bunney, A.B.E.T.
Department of Anesthesiology
Oregon Health Sciences University
Portland, Oregon 97201

(Accepted for publication August 29, 1996)

In Reply: — The use of air for routine calibration of an anesthetic gas monitor is common. Datex, Criticare, 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, Edison, NJ) startup. Subsequent autocalibrations are initiated only after 80-min intervals. The Rascal II displays the message “CALIBRATING” on the screen, notifying the user of the interruption in patient monitoring during the calibration process. The Rascal II does allow the clinician to postpone calibration at any point in the anesthetic. 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.

Dan Hatlesad
Respiratory Gas Product Manager
Ohmeda Medical Systems
1315 West Century Drive
Louisville, Colorado 80027

(Accepted for publication August 29, 1996)

Anesthesiology
1996; 85:1493
© 1996 American Society of Anesthesiologists, Inc.
Lippincott-Raven Publishers

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-year-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 nontender.

Anesthesiology. V 85, No 6, Dec 1996

Anesthesiology
1996; 85:1493
© 1996 American Society of Anesthesiologists, Inc.
Lippincott-Raven Publishers
Correspondence

Blood cell count was acquired, which revealed a leukocytosis (14,000/mm³) with 83% polymorphonuclear leukocytes. 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 tests revealed 9,000/ mm³ white blood cells (normal at our institution, 0–5/mm³), 184 mg/dl protein (normal, 15–88), 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 Staphylococcus 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 relationship 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.

Spencer S. Liu, M.D.
Anne Pope, M.D.
Department of Anesthesiology, Mailstop B2-AN
Virginia Mason Medical Center
1100 Ninth Avenue
Seattle, Washington 98111

References


(Accepted for publication August 29, 1996)

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. 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. 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.” 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 isobaric 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 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 all 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. 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 anesthesia potency as a possible cause of TRI” by basing their doses on potency data reported in an

Anesthesiology, V 85, No 6, Dec 1996