in hemodynamic variables. Further study is warranted, comparing AAA with AOD patients using intraoperative echocardiography and cross-clamping below the celiac axis.

On the basis of the changes in SVR and SV, less intraoperative hemodynamic stress from aortic occlusion was seen in the AOD patients in this study than in the AAA patients. We attribute this protective effect to the greater degree of periaortic collateral vascularization seen by preoperative aortography in the AOD group, and we believe that the amount of collateral vascularization seen on preoperative aortograms can selectively predict the magnitude of change in SV and SVR that will occur intraoperatively with cross-clamp application. However, since both groups of patients in our study subsequently required afterload reduction, invasive hemodynamic monitoring remains useful in CAD patients undergoing infrarenal aortic surgery.

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Accidental Hyperthermia as a Complication of Extracorporeal Shock-wave Lithotripsy under General Anesthesia

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Extracorporeal shock-wave lithotripsy (ESWL) is a new treatment for renal calculi.[1,2] We report an unusual and potentially dangerous hyperthermic complication.

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Case Report

A 25-yr-old woman presented for ESWL treatment of a left lower pole renal calculus. The patient was allergic to codeine, meperidine, and oxycodone, but was otherwise healthy. She took no medication except for intermittent courses of trimethoprim-sulfamethoxazole. General anesthesia was induced with thiopental 5 mg/kg iv and the trachea was intubated after succinylcholine 1 mg/kg iv. No trismus was noted during intubation. Anesthesia was maintained with oxygen 1.0 l/min, nitrous oxide 2.0 l/min, and enflurane 1.5%. Anesthesia depth was judged as adequate by a stable arterial blood pressure and heart rate, and by lack of movement. After 30 min, treatment was interrupted for a routine electrode change. Despite the lack of stimulation, the heart rate gradually increased from a baseline of 80 to 95 bpm, with systolic arterial blood pressure decreasing slightly from 105 to 95 mmHg. The enflurane was decreased to 0.5% and the patient coughed. Fentanyl 50 μg was administered, and the enflurane increased to 2% when shock-wave treatment resumed. Despite increasing the concentration of enflurane, the heart rate continued to increase. The

patient was diaphoretic and lacked muscular rigidity. The pharyngeal temperature was measured at 39.0° C, the patient having been afebrile prior to immersion. The water in the lithotripter bath was noted to be exceptionally warm, and subsequently measured at 40.3° C. Bath temperature is normally maintained at 35–37° C. The patient was lifted from the tank and cooled by evaporation using cold tap water and fans. Her temperature peaked at 39.7° C, and then decreased to 37.2° C over 25 min. Water inflow to the tank was determined to be 62.0° C. The patient remained under general anesthesia with a stable arterial blood pressure of 100/60 mmHg, and heart rate declined as she cooled. She was again placed into the lithotripter bath after it was adjusted to 37° C, and the procedure was completed without incident. Recovery from anesthesia was rapid and uneventful, except for mild nausea that responded to prochlorperazine. The patient was informed of the events and observed overnight. No untoward sequelae were noted. She remained in the hospital, and repeat ESWL was carried out using a 1% lidocaine spinal anesthesia without incident 3 days later.

Discussion

No cases of accidental hyperthermia due to immersion while under general anesthesia have been reported. Whole body hyperthermia for cancer therapy has been studied.[3,4] Patients undergoing this mode of therapy are enclosed in a water-perfusion suit and their esophageal temperature is raised to 41.8° C for several hours.[4] Reported physiologic changes at elevated temperatures include sweating, increased minute ventilation, and decreased urine flow.[5] Hemodynamic changes include tachycardia to 140–160 bpm with constant stroke volume, resulting in a 50–100% increase in cardiac output.[4] Commonly encountered complications include superficial cutaneous burns, post-treatment lethargy, fatigue, and vomiting.[4] Only minimal elevations of SGOT, SGPT, bilirubin, and alkaline phosphatase have been reported.[4] In contrast to controlled hyperthermia, heatstroke is far less benign.[5] Cardiovascular effects of hyperthermia include hyperdynamic response with low peripheral resistance, hypotension, and cyanosis. The hemodynamic changes may precipitate angina or congestive heart failure in those with underlying disease. Rhabdomyolysis and acute renal failure may precipitate disseminated intravascular coagulation. Hepatocellular injury is common. The damaging effects of hyperthermia involve the brain diffusely, but with disproportionate effects on cerebellar function.[6] Irreversible brain damage, presumably due to protein denaturation, is common when temperatures of 42.2° C are reached.[6] Heat injury appears to be manifest at core temperatures greater than 40° C; our patient did not appear to exceed this temperature based on measured esophageal temperature.

Malignant hyperthermia is a feared complication of general anesthesia, and was considered a possibility in this patient. In the presence of an obvious external source of heat and the absence of clinical findings other than increased temperature and heart rate, we chose to treat this patient by removal from the bath and external cooling. Her rapid response to this course of action reassured us that no further therapy was required.

Recognition of this potentially dangerous problem was prompted by the patient's unexplained tachycardia, and apparent increase in anesthetic requirement. We were not previously using continuous temperature monitoring of patients undergoing general anesthesia for lithotripsy, due to technical problems in positioning the patient with the temperature probe in place. Such monitoring might have provided earlier indication of the temperature rise. The mechanism of this patient's increased anesthetic need is presumably increased cardiac output with resultant decrease in blood concentration of inhaled anesthestic. Cardiac output rises by 12% with every degree C rise in body temperature.[7] A high cardiac output results in increased uptake of anesthetic, thus lowering the alveolar concentration.[8] The increase in cardiac output is shunted primarily to muscle and skin vessels, and, consequently, a higher inspiratory concentration is required to maintain a constant blood level.

We have modified our procedures by adding a temperature probe with a readout of water temperature within view of the anesthetist, and requiring continuous esophageal temperature monitoring in all patients undergoing general anesthesia for lithotripsy. Discussions are underway with the manufacturer to include either limit alarms or a failsafe mechanism to prevent excessively hot water from entering the bath.

In summary, a 25-yr-old woman undergoing extracorporeal shock-wave lithotripsy was accidentally subjected to gradually increasing water bath temperature. Increased heart rate and decreasing arterial blood pressure were noted, and anesthetic requirements increased. These findings are consistent with the vasodilatation and increased cardiac output expected with elevation of core temperature. Prompt recognition and treatment are essential if patient injury is to be avoided. We recommend that patient and water bath temperature be monitored by the anesthesiologist during extracorporeal shock-wave lithotripsy.

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Neurologic Dysfunction in Postpartum Patients Caused by Hypomagnesemia

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The use of epidural analgesia to provide pain relief during labor and delivery is increasing in popularity. When a patient who received epidural analgesia develops neurologic complications in the postoperative period, the anesthesiologist is confronted with the complex problem of differential diagnosis. When faced with such a situation, one should consider other causes of neurologic dysfunction, other than those complications commonly attributed to the epidural technique. In this report, we present two patients who manifested neurologic dysfunction following epidural anesthesia that was most likely due to hypomagnesemia.

CASE REPORT 1

A 22-yr-old, 52 kg, gravida 2, para 1, A.S.A class I patient was admitted to the hospital for labor and delivery at 34 weeks of gestation. Her prior surgery included a cesarean section 3 yr prior to the present admission. She was not allergic to any medications. Physical examination was normal. Pelvic examination revealed 4.0 cm dilatation of the cervix and ruptured amniotic membranes. Her vital signs included an arterial blood pressure of 100/70 mmHg and a heart rate of 86 bpm. For pain relief, she requested lumbar epidural analgesia. Utilizing loss of resistance technique, a 17-G epidural needle was inserted into the L3-L4 interspace. During the insertion of the epidural needle, an accidental dural puncture occurred. The needle was then inserted into another epidural space and an intermittent lumbar epidural analgesia (ILEA) with 0.25% bupivacaine was administered. Eight hours later, she delivered spontaneously a healthy male infant. Twenty-four hours following the delivery, she complained of nausea, vomiting, and headache. The diagnosis at this time was spinal headache. She was advised to stay in bed and drink fluids. Because the headache persisted the next day, an autologous epidural blood patch (EBP) was performed with 10 ml of blood. During the night, 12 h following the EBP, she complained of vertigo and requested pain medications. The next morning (20 h following the EBP), she had two episodes of generalized seizures. She was given incremental doses of diazepam (20 mg total) and 500 mg of phenytoin IV to control the seizures. She remained in post-ictal coma for 2 h and then slowly became fully oriented. She denied any history of drug addiction, alcohol abuse, or seizure disorders. She had no evidence of preeclampsia. Neurological examination, CT scan of the head, drug screening, lumbar puncture and analysis of the CSF and EEG were all normal. The only abnormal laboratory finding was a low magnesium level of 1.3 meq/l (normal at this lab 1.8–2.4). Serum electrolytes were within normal limits. She was given 4 gm of magnesium sulphate iv bolus injection. Later on, she received oral magnesium supplementation. In the next 2 days, she completely recovered, and her anti-convulsive medications were slowly discontinued over a period of time. Upon discharge, her serum magnesium level was 2.0 meq/l.

CASE REPORT 2

A 22-year-old, 65 kg, gravida 1, para 0, A.S.A class I patient was admitted at term in early labor. Upon examination, her cervix was 4 cm dilated, and the membranes had ruptured. Her vital signs included an aretrial blood pressure of 110/80 mmHg and a heart rate of 92 bpm. For relief of pain, she requested lumbar epidural analgesia.

Utilizing loss of resistance technique, a 17-G epidural needle was inserted into the L3-4 interspace. A 19-G epidural catheter was then introduced through the needle without difficulty. No parasthesias were elicited on placement of the needle, catheter, or injection of the local anesthetic. Neither blood nor cerebrospinal fluid could be aspirated through the catheter. A test dose of 3 ml of 0.5% bupivacaine with epinephrine was injected. Five minutes later, an additional dose of 6 ml of 0.25% bupivacaine with epinephrine was injected. Following this, the patient experienced relief of pain. After receiving two more injections of the local anesthetic, the patient spontaneously delivered a healthy male infant. She recovered from the effect of the anesthetic in about an hour, and was released to the ward. She offered no complaints on the day following the epidural anesthesia.

Two days following the epidural anesthesia, while holding the baby,

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