have been exited. We think it is more likely that the catheter was inserted correctly and later eroded through the vein and entered the right hemithorax. The lack of symptoms until 4 days after catheter placement would make the later erosion secondary to motion of the head and neck highly suspect. Her iv infusion had been kept at a rate of approximately 1.0 l/day of normal saline prior to her respiratory arrest, which may not have greatly exceeded the absorptive capacity of the pleural surface; however, accumulation of fluid in the chest must have contributed to her respiratory compromise. She had no other iv lines in place, and fortunately no medication had been given iv. The amount of fluid visible in the right chest in figure 1 was detectable on physical examination immediately before the first radiograph was obtained, but the chart failed to document examinations during the postoperative period. The composition of the pleural fluid is compatible with a mixture of normal saline infused before transfer to the medical service, approximately 300 ml of 5% dextrose in water infused after transfer but before the second radiograph was obtained, and blood (the ratio of red blood cells in pleural fluid to blood is equal to the ratio of pleural fluid protein to serum protein).

In summary, insertion of a central venous line by any technique, including external jugular cannulation, can result in significant complications. The clinician should be alert for any malfunctions in the catheter (especially lack of venous return) and review roentgenographs taken for any purpose for proper catheter placement. In addition, clinical evaluation, which should have detected the complication prior to respiratory arrest, is part of the daily care of the patient.

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Anesthesiology
62:674–677, 1985

Aseptic Meningitis Following Spinal Anesthesia—A Complication of the Past?

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Meningitis following well-conducted spinal anesthesia is a rare but serious complication. Major surveys of regional anesthesia have reported thousands of spinal anesthetics free of this complication.[1,2] Aseptic meningitis is a clinical syndrome whose acute onset and clinical symptoms mimic septic meningitis. Its differential diagnosis from bacterial meningitis can be critical in light of the rapid progression and often fatal course of an untreated septic meningitis.

In the earlier years of spinal anesthesia, aseptic meningitis was a not uncommon and well-reported complication following spinal anesthesia. By 1947 Thorsen[3] referenced more than 100 reported cases in the medical literature and Orkin (as quoted by Goldman and Sanford[4]) noted an incidence of 0.26% of aseptic meningitis in a summary report on approximately 46,000 spinal anesthetics. As a readily appreciated syndrome following spinal anesthesia, purulent sterile meningitis has all but been lost to a generation of anesthesiologists, with the last reported case in 1970.[5] The following is a case report and a discussion of the decline in the incidence of aseptic meningitis.

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Received from the Charles A. Dana Research Institute, Department of Anaesthesia, Beth Israel Hospital and Harvard Medical School, Boston, Massachusetts. Accepted for publication December 13, 1984.

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Key words: Anesthetic techniques: spinal. Complications: aseptic meningitis.

Report of a Case

A 32-year-old man came to the operating room for a repair of a ruptured left Achille's tendon. His medical history was unremarkable. He was afebrile, 188 cm tall, and had no evidence of localized infection in his lower back area. Laboratory results included a white blood cell count of 8,000/mm^3 with normal white blood cell differential count and a negative urinalysis.

The patient requested a regional anesthetic, and after premedication with diazepam 7.5 mg iv he was placed in the left lateral decubitus position. The skin of the lumbar area was prepped with three washes

with a 1% iodine solution and draped in a sterile fashion. Dural puncture was performed with a disposable 22-gauge spinal needle from a hospital-prepared block tray via the L3–L4 interspace with minimal difficulty and confirmed by clear, free-flowing cerebrospinal fluid (CSF). No paresthesia occurred, and a total of 5 ml of a hyperbaric 0.25% dibucaine solution was injected without complaints or break in sterile technique. Satisfactory analgesia ensued to a T12 level on the left, and the patient was turned to the prone position. The surgical repair was carried out without incident.

After an uneventful recovery room stay, during which time he had no complaints, remained afebrile, and the motor and sensory effects of the spinal anesthetic had worn off, he returned to the ward. Twelve hours after the introduction of spinal analgesia, he was febrile and complained of chills, headache, and a stiff neck. Examination revealed a clear mental status, photophobia, nuchal rigidity but negative Kernig and Brudzinski signs, and an otherwise normal neurologic and general examination. The patient had received one preoperative and one postoperative dose of cephalothin.

Blood cultures, urinalysis and urine culture, a chest roentgenogram, and an immediate lumbar puncture were performed. The CSF was grossly cloudy and revealed a white blood cell count of 600/mm^3 (68% polymorphs, 4% lymphocytes, and 27% monocytes); red blood cells 380/mm^3; protein 112 mg/dl; glucose 92 mg/dl (with systemic glucose 122 mg/dl). No organisms were seen on Gram's stain of spun sediment of CSF.

While the findings of a normal glucose and the absence of organisms in the CSF is atypical for septic meningitis, our failure to appreciate the diagnosis of aseptic meningitis and our concern of leaving untreated a possible bacterial meningitis led us to initiate antibiotic therapy with cefotaxime 2 g iv every 4 h.

By the next afternoon, approximately 12 h later, his symptoms had dissipated completely, and his fever had defervesced. Antibiotic therapy was continued until the CSF cultures showed no growth at 48 h. Antibiotics were discontinued, and the patient was observed for an additional 48 h. He remained afebrile and clinically well off antibiotics and was discharged. At 1 month postdischarge, he was feeling well.

Discussion

Aseptic meningitis is a clinical syndrome rather than a specific disease, characterized by symptoms of fever, headache, nuchal rigidity, and photophobia. When associated with spinal anesthesia, it generally has an acute onset within 24 h of the dural puncture and a self-limited and benign course. The diagnosis is confirmed by examination of the CSF: no organisms are seen on microscopy and none are grown from cultures. A marked pleocytosis with polymorphonuclear cells predominates in the initial phase of the illness, with a shift toward mononuclear predominance if a second lumbar puncture is performed 12–72 h after the onset of the symptoms.[6,7] This patient's clinical course was very similar to previously reported cases[4,5,8,9] and entirely compatible with the diagnosis of aseptic meningitis. Although a second lumbar puncture was not performed due to the patient's refusal, the sterile CSF cultures and rapid recovery in the absence of continued antibiotic therapy confirm the diagnosis.

Of the numerous etiologies attributed to this type of aseptic meningitis, the weight of the evidence suggests that a chemical irritation of the subarachnoid space, most likely from detergent-contaminated needles and syringes, is responsible for the majority of cases. However, from the birth of spinal anesthesia to the late 1940s, this syndrome was a regularly occurring complication with an accepted incidence rate of one case to every 400th anesthetic and undoubtedly had multiple etiologies including toxic local anesthetic agents and additives, large, long-beveled spinal needles enhancing traumatic taps along with inadequate sterilization of equipment. During the mid-1950s the frequency of reports of aseptic meningitis decreased drastically after a series of studies indicated that chemical contaminants were likely at fault. Hurst[10] reported an experimental study on monkeys in which different agents, including detergents and disinfectants, were injected intrathecally and produced symptoms and histologic changes consistent with aseptic meningitis. Denson *et al.*[11,12] demonstrated that syringes washed and then sterilized without vigorous rinsing to remove residual detergent were capable of producing varying degrees of arachnoiditis in the majority of monkeys studied. They suggested that, unless due precautions were taken to remove all detergents or antiseptics from the equipment, chemical meningitis and arachnoiditis were likely complications.

With the widespread improvements in hospital-based equipment preparation, including multiple-rinse cycle cleansing procedures and water-soluble detergents, reports of aseptic meningitis appeared sporadically during the 1960s, but cases tended to cluster that suggested a breach in technique. Goldman and Sanford[8] reported five cases of chemical meningitis on an obstetric service during an 11-month period and attributed it to residual phenolic disinfectants on the syringes used for intrathecal injection. The fact that the anesthesia department in the same hospital omitted the disinfectant in their equipment preparation and reported no cases of aseptic meningitis following spinal anesthesia for surgical procedures during this same period supports their claim. More demonstrative was a series of three cases occurring within a 24-h period following spinal anesthesia for vaginal delivery.[13] The anesthesiologist involved concluded that a breach in technique in the preparation of the spinal trays was the likely etiology, since all three had been prepared by the same person on the same day and no such complication ever had occurred before this day nor in the subsequent 13-month interval. Austin and Sokolowski[8] attributed their two cases of aseptic meningitis following spinal anesthesia to contaminated needles and syringes with alkyl-arylsulfonate detergent and noted the absence of this complication in the anesthesia department, which omitted the detergent in their equipment preparation protocol. Gibbons[9] similarly reported an "epidemic" of chemical meningitis with

three cases in a 3-week period. A careful review of their procedures uncovered a recent change in the washing and rinsing procedures of needles and syringes and led to the observation of residue on the equipment thus processed. No further cases of meningitis occurred when the use of the detergent was discontinued. The evidence, while circumstantial, strongly suggests detergent-contaminated equipment as the likely etiology in these cases.

Following our case we investigated likely etiologies. The dibucaine used was from a recently manufactured lot and, while the remainder of the actual ampule used was no longer available, ampules from that lot had not resulted in other cases of aseptic meningitis, nor had the manufacturer received complaints regarding the anesthetic. Spinal trays at our hospital are prepared by our central processing department. Spinal needles are disposable, but a 5-ml glass syringe is reused for intrathecal injection of the local anesthetic. These syringes are prepared sterilely by undergoing machine washing with detergent at more than 80° C, and then multiple-rinse cycles, the last with distilled water. Although one detergent (Liqua-Safe®) primarily is used, a second detergent (Amsco-sonic®) may be substituted if there is a lack of supply of the former. Several glass syringes processed in the usual fashion with Liqua-Safe® revealed no detectable residue detergent by spectrophotometric analysis. However, examination of several syringes processed in an identical fashion using Amsco-sonic detergent revealed gross contamination with residue detergent. Further investigation of the detergents showed that these cleaning compounds had different physicochemical characteristics. In addition to different absorption spectrae between 200 and 600 nm, the absorbance of diluted Amsco-sonic® solutions are sensitive to temperature changes, while Liqua-Safe® solutions are not. This indicates that Amsco-sonic® detergent undergoes changes in its physical characteristics with temperature fluctuations while Liqua-Safe® does not. The instability in absorbance is due to the formation of molecular aggregates in Amsco-sonic® solutions when temperature of the solution is increased. Thus, we found undiluted and diluted Amsco-sonic® solutions turn completely turbid when temperature is increased to 35° C, and these solutions remain turbid even when temperature of the solutions is increased to 100° C. Liqua-Safe® solutions remained clear at all temperatures up to 100° C. It is likely that during the raised temperatures of the cleaning process of our glass syringes, aggregates of Amsco-sonic® detergent form, and these insoluble aggregates can result in aseptic meningitis when injected as contaminants of a local anesthetic solution. We believe that the finding of spectrophotometrically identifiable detergent residue on our spinal tray glass syringes is direct evidence for chemical contamination as the etiology of this patient's aseptic meningitis.

While the prognosis of aseptic meningitis associated with spinal anesthesia is good, with full neurologic recovery the rule, much of its importance to the clinician lies in its differential diagnosis from septic meningitis. Bacterial meningitis remains a rare but potentially lethal complication of spinal anesthesia. The differential diagnosis hinges on examination and culture of CSF. It is vital then that the patient with fever, meningismus, and a history of recent spinal anesthesia undergo a prompt diagnostic lumbar puncture. Furthermore, despite the absence of organisms on direct microscopy and the evidence that polymorphonuclear cells predominate in the CSF early in the course of aseptic meningitis, we do not agree with the practice of withholding antibiotics anticipating rapid clinical improvement.[7] Many perioperative patients are on antibiotics prophylactically allowing a partially treated bacterial meningitis to masquerade as aseptic meningitis. Additionally, untreated septic meningitis can progress rapidly to a fatal outcome. We suggest initiation of appropriate antibiotic therapy promptly following diagnostic work-up and continuation until clinical improvement ensues and cultures yield no growth at 48 h. Thereafter, the diagnosis of aseptic meningitis reasonably can be assumed and observation off antibiotics is appropriate. Appropriate antibiotic therapy must anticipate that bacterial meningitis in patients with recent spinal anesthesia commonly is due to Pseudomonas species or unusual nosocomial organisms.[14]

Finally, our investigation to pinpoint the etiology of this complication revealed our dependence on both the manufacturers of anesthetic agents and equipment used routinely for spinal blocks and the techniques in our central processing department. While past experience has demonstrated a high degree of reliability, this case has lead us to reevaluate our practice. Prepackaged sterile disposable spinal trays have been marketed and in clinical use for years and thus far have not been associated with a case of aseptic meningitis in the published literature. The use of these trays does not eliminate the possibility of contaminated equipment, nor does it lessen our dependence on medical supply companies. To eliminate such complications completely remains our goal, but regardless of whether disposable equipment is used or not, close and constant attention to the details of equipment preparation, sterilization, and technique is mandatory.

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Anesthesiology
62:677–678, 1985

Errors in Installation of a New Gas Delivery System Found after Certification

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Bulk supplies of oxygen and nitrous oxide frequently are delivered via pipelines to operating rooms. Stringent standards have been formulated for the installation of central gas supply systems by the National Fire Protection Association (NFPA).‡ Certification of newly installed systems by an engineering firm is a common practice, most often on a voluntary basis by the hospital. In many states, evidence of satisfactory specifications is required for licensure of the hospital facility. The Joint Commission on Accreditation of Hospitals and most insurance companies require an inspection to verify compliance with published standards.

Errors during installation can lead to crossed medical gas and vacuum pipelines, leaks in the pipelines or concealed connectors, contamination of delivered gas by other gases, water, hydrocarbons, or particulate matter.[1] Pressures may exceed or fall below designed limits and must be monitored by sensitive alarm systems. The adaptors that connect piped gases to the machine may be installed with improper fittings or reversed. Several of these problems have resulted in patient deaths and have been addressed in publications.[1–4]

The present report describes errors detected 6 weeks after certification of the gas delivery system in a new operating room suite by an engineering firm.

REPORT

In mid-February, 1984 a new patient care facility was nearly complete. The operating room suite contained preassembled quick-connect Chemetron® gas delivery system with ceiling-mounted hose reels. Over the next 3 months, construction continued with interim inspections by an engineering firm. A number of gas leaks and alarm system deficiencies were noted and corrected. On June 14, 1984, all medical gases and vacuum systems were certified as satisfactory. After that date, the contractor remained on site, completing ceiling roentgenographic installations and mounting stainless steel cover plates in the ceiling, through which the gas hoses and connectors were passed.

On July 29, 1984, one day prior to move-in, the Anesthesiology Team conducted a final inspection to verify the integrity of gas lines, identity of the delivered gases, and proper function of connectors. Three defects were found in 14 operating rooms. A vacuum outlet was inoperative due to particulate matter in a check valve. An oxygen port to a cardiopulmonary bypass location delivered no gas at all. Subsequent dismantling of the pipe-interface in the ceiling revealed an absent spring device in a check valve. In a third room,

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Received from the Department of Anesthesiology, Albert Einstein Medical Center, Northern Division, Philadelphia, Pennsylvania, and ECRI, Plymouth Meeting, Pennsylvania. Accepted for publication December 13, 1984.

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Key words: Anesthetic, gases: nitrous oxide. Oxygen: delivery systems.

‡ NFPA 56F: Nonflammable Medical Gas Systems, National Fire Protection Association. Batterymarch Park, Quincy, Massachusetts 02769, 1979.