

any symptomatic improvement, the patient was referred back to ECT. She was premedicated with atropine, 0.5 mg im and 0.4 mg iv, and then was given 0.9 mg/kg methohexital and 0.5 mg/kg succinylcholine. Electric stimulation of sufficient intensity to elicit a generalized seizure was used during the treatment. The patient tolerated a course of 13 treatments without cardiac complications and with good remission of symptoms.

Cardiac arrest is a well-documented, although rare, complication of ECT. In the present case, the tendency to bradycardia and asystole was probably heightened by the use of beta blockade. The subconvulsive electrical stimulation also could have contributed. Experimental data support the notion that an adrenergic mechanism is involved in the phenomenon of vagal escape² and that, in the presence of sympathetic blockers, a shock-induced activation of the autonomic nervous system can lead to a parasympathetic mediated cardiac arrest. This does not normally occur with ECT because the seizure elicits a marked peripheral sympathetic response that results in a rise in heart rate. With a subconvulsive shock, the central parasympathetic mechanism was unopposed and resulted in a slowing of the heart rate; a phenomenon exacerbated by beta blockade. It seems probable that propranolol was involved, at least in part, in the pathogenesis of the asys-

tole. Therefore, we feel that one should exercise caution when using the combination of propranolol and ECT.

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Respiratory Depression Following Only 0.4 mg of Intrathecal Morphine

To the Editor:—Intrathecal and epidural opiates have been used successfully to produce postoperative analgesia. However, respiratory depression is a problem with this technique with doses of 1 mg or more. However, we observed a case of respiratory depression with a much smaller dose, 0.4 mg, of intrathecal morphine. A 74-year-old patient scheduled for a peripheral orthopedic procedure was premedicated with 5 mg of diazepam orally, 2 h preoperatively. Analgesia was obtained using 0.8 ml heavy lidocaine (*i.e.*, 40 mg lidocaine) inserted via a spinal needle at L₂₋₃ with the patient in the left lateral position and maintained 15 degrees head up. This was followed by 0.4 mg of preservative-free morphine in 5% dextrose water. Flunitrazepam, a total of 1 mg, was given iv intermittently during the procedure.

Postoperatively, inspired oxygen was increased with the help of a 28% ventimask. It was instructed that she be kept slightly head up. Accidentally the patient was put

head down for half an hour. Two-and-a-half hours later, the patient was cyanotic, with a respiratory rate of 6 breaths·min⁻¹, an arterial blood pressure of 70/50 mmHg, and a heart rate of 46 beats/min. Naloxone 0.4 mg was given iv and 0.4 mg im. This resulted in immediate improvement in respiratory rate to 10 breaths·min⁻¹, arterial blood pressure to 100/50 mmHg, heart rate to 50 beats/min, and her color returned to normal. No further naloxone was required.

The intrathecal morphine was the only narcotic given during the perioperative period. Pain relief lasted for 36 h.

The cause of the respiratory depression is most likely due to rostral spread of the morphine following the normal flow of cerebrospinal fluid from the lumbar region to the basal cisterns and which may then enter the ventricular system.¹

The large difference in incidence of respiratory depression following epidural as compared with intrathecal morphine is probably due to the fact that morphine does not as readily enter the cerebrospinal fluid when

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injected epidurally due to the physical barrier presented by the dura, and by the simultaneous uptake of morphine into the general circulation by Batsons epidural venous plexus. Morphine, 8 mg, injected extradurally reaches a peak cerebrospinal fluid concentration at 120 min with approximately 3,500 ng/ml, whereas at 10 min, 1 mg of intrathecal morphine has a concentration of 40,000 ng/ml². Thus, if one can extrapolate, then even 0.4 mg of intrathecal morphine still will have a very high cerebrospinal fluid concentration and readily will produce respiratory depression if encouraged to reach the fourth ventricle by coughing, straining, or, as occurred in this patient, by being put in the Trendelenberg position when using a hyperbaric solution.

The original reports of intrathecal morphine were using 0.5–1 mg³, but doses as high as 20 mg⁴ have been given intrathecally. The effectiveness of doses of 0.5–1.0 mg has been demonstrated clearly.³ No dose response curve for intrathecal morphine, however, has been made yet, thus establishing the minimal effective dose for intrathecal use. The presently recommended dose is 0.5–1 mg. However, with very high concentrations of morphine entering the cerebrospinal fluid from intrathecal

administration, and extrapolating this against cerebrospinal fluid concentrations that are effective after epidural injection, then doses of possibly 0.05–0.1 mg may well be as effective and much safer.

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Bupivacaine Cardiotoxicity May Be More Related to Technique than to the Drug

To the Editor:—Drs. Conklin and Ziadlou-Rad described a case that they felt illustrated several points that are pertinent to the prevention of bupivacaine cardiotoxicity.¹ I would maintain that, while certain of the steps they advocate might lessen the incidence of large intravascular drug boluses associated with epidurals, there are still assumptions in both their original and amended manner of performing the injection sequence that should be examined.

At the outset of their procedure, the authors placed a catheter in what they felt was the epidural space and then aspirated. Finding no evidence of blood or other fluid, they injected small amounts of chloroprocaine as test doses based on the claim that “. . . we have found that in parturients, 5 ml of 3% chloroprocaine uniformly produces signs of CNS toxicity without producing seizures.”

This claim was not substantiated in any verifiable manner. That would seem to require deliberate intravascular injections of at least mildly toxic doses of local anesthetics; and, because of this, I am not sure that their statement can be supported in any but an anecdotal manner. Also, their statement seems to imply that all parturients respond in like fashion to a fixed dose of drug. This would be

contrary to clinical experience. Thus, when authors state that some amount of anesthetic uniformly produces symptoms and that those symptoms are uniformly limited, they assume a burden of proof I do not believe they can support.

If one cannot prove the test dose to be completely reliable in detecting an intravascular catheter, then to inject the entire blocking dose at one time, as Conklin and Ziadlou-Rad did, seems inappropriate. The authors partially address this by acknowledging that “fractionating” the full dose may lessen the risk inherent in a single large injection. However, they do not ask the next logical question: How much is a safe increment? I assume that their 5 ml per minute of 0.75% bupivacaine represents a guess rather than an empiric observation, since they did not make a claim for it similar to the one made for 5 ml 3% chloroprocaine. Had they applied the same standard of observed safety to their bupivacaine dose as to their test dose, I think they might have injected the bupivacaine in a different manner.

A test dose of a local anesthetic represents the largest safe increment that one knows to give. Even though it cannot be proven infallible as an indicator of an intravenous injection, the next increment is given with a much