CORRESPONDENCE

Triple Gloves

To the Editor—Dr. Jacoby’s concern regarding contamination of anesthesia equipment by personnel wearing soiled gloves is appropriate, as is his comment that soiled gloves should be removed promptly. During the course of an anesthetic, there are two events during which heavy glove-soiling occurs—induction of general anesthesia with subsequent airway management, and emergence from general anesthesia and tracheal extubation. I have found that prior to these two events, by donning two or three pairs of thin latex gloves at one time, I can prevent contamination of my anesthesia equipment. When my gloves become soiled, they are quickly removed, exposing fresh clean gloves underneath. I do not have to stop what I am doing to put on clean gloves, and I am not distracted from the care of my patient. I recommend this method as a way to prevent contamination of anesthesia equipment with soiled gloves while maintaining a protective barrier between anesthesiologist and patient and without the distraction of stopping to don clean gloves.

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REFERENCE

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More on Pharmacokinetics with a Pocket Calculator

To the Editor—Pierre Maitre and I recently described an algorithm for using a pocket calculator to predict anesthetic drug concentrations. Our original program for the Hewlett-Packard (HP) 41CX was not of sufficient quality to make generally available. I have since implemented these algorithms in a user-friendly program for the HP 48SX calculator. Interested readers may contact me for the program listing. Alternatively, readers may send their HP 48SX calculator to me, and I will, at no cost, download the program into their calculator and return it with instructions.

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Prolongation of Cocaine Effect

To the Editor—It appears that some of the information contained in the recent article by Fleming et al. has filtered to the street, as indicated by the following case referred to the New York City Poison Control Center.

A 25-yr-old woman (gravida 8, para 4) at 36 weeks gestation ingested 20 ml 70% chlorpyrifos solution (an organophosphate insecticide) while smoking crack (crystalline cocaine) in an effort to prolong the effect. Approximately 1 hr later she complained of nausea and weakness. Vital signs were stable with the exception of a pulse rate of 115 beats per min. Shortly, she vomited, developed fasiculations, and had a generalized tonic-clonic seizure. Continuous fetal monitoring showed no fetal distress. After tracheal intubation she received atropine 4 mg, diazepam 10 mg, phenobarbital 900 mg, and 2-pralidoxime 2 g followed by 1 g every 8 h for 24 h as per our recommendation. There was a great deal of concern regarding her anesthetic management should an operative delivery be required. Fortunately, during our deliberations and within a few hours of admission, vaginal delivery of a healthy baby occurred. There was no evidence of neuromuscular deficiency in the baby. The mother recovered uneventfully. Plasma cholinesterase concentrations are listed in the table.

The use of cocaine and organophosphates simultaneously may result in a confusing toxicologic picture: the signs of sympathetic nervous system excess following cocaine and the symptomatology of acetylcholine excess can counterbalance one another, and both can give significant central nervous system symptomatology. The intent of ingesting the chlorpyrifos was to prolong the effect of the crack she smoked. It

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appears that this was based on the assumption that the organophosphate would prevent the rapid breakdown of the cocaine by inhibiting plasma cholinesterase, thereby leading to a prolonged or intensified high. As seen in Table 1, the duration of cholinesterase blockade from organophosphates can be prolonged. For the anesthesiologist, this may pose difficulties if succinylcholine is used.

Hence, in the elective setting, we suggest that a history of cocaine use should prompt a question regarding the concomitant use of organophosphates or similar toxins. A positive response may dissuade the anesthesiologist from using succinylcholine, depending on the time since exposure to the toxin. In the setting of emergency operations in the cocaine-intoxicated individual, it may be prudent to question the patient (or emergency personnel) if he or she took anything to “make the cocaine last longer.” If so, use of succinylcholine should be tempered by the knowledge that a prolonged effect may occur and that there may be an unusually poor response to standard intubating doses of nondepolarizing muscle relaxants. One should consider this also in the differential diagnosis of a sensitivity to succinylcholine, and delay evaluation of plasma cholinesterase levels until the full history is elucidated or sufficient time has passed to allow regeneration of plasma cholinesterase.

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Complete Sympathetic Blockade?

To the Editor—A recent article by Peters et al.1 demonstrates that vasopressin antagonism results in a profound decrease in blood pressure in dogs with high epidural anesthesia. I find this information of interest in light of attempts to understand hemodynamic changes in patients undergoing epidural anesthesia. However, I must take issue with one of the principal statements in this article1 and an earlier article from the same laboratory.2

Specifically, Peters et al. claim that “sympathetic efferents were largely, if not completely eliminated by high epidural anesthesia.” They state that this was demonstrated by a 20% decrease in plasma norepinephrine concentration. This does not prove a complete or almost complete sympathectomy, but rather suggests the contrary. That Peters et al. found the plasma epinephrine concentrations unchanged and found only a 20% decrease in norepinephrine argues that the sympathectomy produced by this high level of 0.5% bupivacaine epidural anesthesia was incomplete. These findings are similar to those of similar series of experiments we performed using the same dog model.3 In these experiments, we found only a moderate decrease in plasma catecholamine concentrations, despite a high level of epidural block. When our blocked dogs then were subjected to a severe stress of hypercarbic acidemia and hypoxemia, the catecholamine response seen in unblocked dogs was attenuated, but not completely eliminated. Again, this suggests an incomplete sympathectomy, despite evidence of a high epidural block.

Peters et al. further cite as evidence of complete sympathectomy paralysis of the membrana nictitans, since they are innervated by sympathetic nerves originating from the T1 to T3 spinal segments.4 Having some experience with experimental epidural anesthesia in dogs and with these particular dogs used by Peters et al., I have observed that extensive epidural blockade produces only a partial paralysis of the membrana nictitans. If one induces general anesthesia in a dog with a high epidural block, one observes that this partial paralysis of the membrana nictitans then becomes complete, indicating that the block of the sympathetic efferents by epidural anesthesia was only partial. This concept is not new and was proposed by Bromage over ten years ago.5

Whether epidural anesthesia produces a complete or only partial sympathetic block is not of just theoretical interest. In the treatment of patients with reflex sympathetic dystrophy (RSD) and other disorders of sympathetic function, we assume that a complete sympathetic block with epidural anesthesia has been achieved once a sensory or motor blockade is present. This is the basic assumption underlying the “differential epidural block.”6 If epidural sensory or motor anesthesia does not guarantee complete blockade of sympathetic fibers, this may explain some of the treatment failures in cases of “severe RSD.” Sympathetic efferent fibers may be more resistant to epidural anesthesia than previously assumed. This recent data questions a long-held assumption and provides additional impetus for further investigation of the effect of epidural anesthesia on the sympathetic nervous system.

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