Correspondence

Postoperative rocuronium reparable paralysis

To the Editor:

We read with interest the report by Arndt *et al.*¹ Recurarization appears to occur frequently and we would like to provide two further reports.

Patient #1 sustained a gunshot to her neck causing an intimal flap injury to the carotid artery. During stent placement under conscious sedation an intracranial AV fistula was discovered during arteriography. Subsequently, invasive radiological ablation under general anaesthesia after awake fiberoptic intubation, proceeded uneventfully. After standard neuromuscular reversal and tracheal extubation, the patient was transported awake, sitting up, and talking in the SICU. Fifteen minutes after extubation, she was unable to open her eyes and noted difficulty breathing. Bag mask ventilation was begun. Fiberoptic intubation under controlled ventilation via an LMA proceeded uneventfully. The ulnar nerve response revealed one weak twitch to TOF stimulation. Twenty minutes later, the patient was again able to open her eyes, lift her head and cough. The trachea was extubated uneventfully.

Patient #2 presented for breast biopsy under general anaesthesia. Anaesthesia was induced with propofol and fentanyl, followed by an intubation dose of rocuronium (0.6 mg·kg⁻¹) and anaesthesia was maintained with nitrous oxide-oxygen-sevoflurane. Vancomycin (1 g) was given preoperatively. Post surgery, normal TOF, sustained tetanus and head lift (>5sec) was evident after standard reversal. After awake extubation, the patient was transported to PACU. Within 20 min, she became progressively dyspneic and weak as evidenced by depressed ulnar twitch responses. Calcium chloride (1.5 mg·kg⁻¹) followed by 10 mg pyridostigmine and 0.2 mg glycopyrrolate was given with no immediate improvement. Subsequently, the trachea was reintubated after 5 mg midazolam and the lungs were ventilated until spontaneous neuromuscular recovery. Extubation proceeded uneventfully.

In summary, we describe two cases of rocuronium reparable paralysis in which neuromuscular recovery was demonstrated prior to PACU arrival. Vancomycin-induced neuromuscular potentiation should be given serious consideration when used with rocuronium.²

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REPLY

We were interested in Dr. Coveler's response to our article on "Postoperative rocuronium reparable paralysis...". It is our opinion that postoperative rocuronium reparable paralysis is not infrequent. However, the aetiology may be multifactorial. We may be on the learning curve in using this drug correctly, or rocuronium may possess a pharmacological profile making reparable paralysis more likely. From Dr. Coveler's description, it would appear that good clinical practice was followed and postoperative paralysis should have been unlikely.

The reason we wrote our article was to warn clinicians of possible rocuronium reparable paralysis. It would appear that our experience at Wisconsin is not unique. Most clinicians are comfortable using atracurium and vecuronium for neuromuscular blockade and the recovery profile makes post-operative paralysis unlikely. However, one of my colleagues reminded me of the "ventilator farms" in the recovery room when vecuronium was initially introduced, due to clinicians needing to learn how to use this drug. The basic question to which I do not have an answer is, does rocuronium possess an unique pharmacology making post-operative reparable paralysis more likely, or are we still on the learning curve?

I hope our article and Dr. Coveler's experience will prompt others to investigate rocuronium to address the issue of postoperative paralysis. I still use rocuronium, but with caution.

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Masquerading neuropathies

To the Editor:

The recent clinical report by Kahn illustrating the potential for postoperative epidural analgesia with local anaesthetic/opioid infusions to confound the recognition of perioperative neurological complica-

tions, leads one to ask if relatively high concentrations of local anaesthetic should ever be used for postoperative analgesia. Opioids and local anaesthetics have been shown to be synergistic in animal models and at least additive in several human postoperative analgesia trials.²⁻⁴ However, recent reports have shown that an additive analgesic interaction can be attained with relatively low concentrations of bupivacaine (0.1–0.3 mg·ml⁻¹) infused at a rate of 1–5 mg·hr⁻¹.⁽³⁻⁵⁾ It may also be necessary to titrate the local anaesthetic and opioid separately to obtain the greatest degree of analgesic synergy. Fixed mixtures of opioids and local anaesthetics make it difficult to optimize the benefits of each analgesic and reduce side effects.³⁻⁵ This may be best accomplished by utilizing a patient controlled analgesia delivery system for the opioid component.

Kahn's report points out the potential for high concentrations of bupivacaine to produce confusion in the evaluation of postoperative neurological deficits. Concentrations of bupivacaine > 0.06% do not appear to add much in analgesic benefit and contribute to a greater incidence of motor block and hypotension.³⁻⁸ I would suggest that any patient developing motor deficit in the postoperative period should have the local anaesthetic discontinued from the epidural analgesic solution. The patient should be reevaluated within two hours to examine for regression of the nerve block. Furthermore, concentrations of bupivacaine > 0.06% are not warranted and should be abandoned in the delivery postoperative epidural analgesia.

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REPLY

Synergistic effects of combined opioid-local anaesthetic epidural mixtures are more evident in thoracic than in lumbar epidural¹ infusions. However, the optimum concentration of each drug has yet to be determined. In an ideal setting, titration of each drug could best achieve the desired result. This may be impractical on a busy Acute Pain Service, and expensive, as two infusers and two drug mixtures are required. Our experience with very low concentrations (0.01–0.03%) of bupivacaine resulted in inconsistent analgesia, depending on the surgery, the site of the epidural catheter, drug mixtures and infusions rates. In addition it is labour intensive. Despite these low concentrations, sensorimotor blocks with lumbar epidural catheters still occur.²

The references quoted to reduce concentrations of bupivacaine do not support the recommendations. Reference #3, by addressed the fentanyl sparing effects of various concentrations of bupivacaine with no mention of the deleterious effects of bupivacaine 0.125%. Although lower concentrations of bupivacaine were used, high infusion rates of up to 22 ml·hr⁻¹ were needed, raising the question, was less drug used? Reference #4³ is an abstract, non-peer reviewed article, with few patients enrolled in each group. Reference #7⁴ et al. suggested using bupivacaine 0.2% with fentanyl in thoracic epidurals for thoracotomies. References #6⁵ and #8⁶ demonstrated that low dose bupivacaine did not improve analgesia. Perhaps higher doses are needed. In a follow up study,⁷ bupivacaine 0.125% was found to improve analgesia after thoracic and abdominal surgery, with minimal sensorimotor disturbance.

Using 8–12.5 mg·hr⁻¹ epidural bupivacaine as a continuous infusion would not be considered high dose. In a recent review Stevens and de Leon-Casasola,⁸ recommended 6–10 mg·hr⁻¹ bupivacaine 0.2% for epidural infusions for postoperative analgesia. When used according to the guidelines in the case report,⁹ the incidence of motor blocks was low. We have found this regimen to pro-