Letter to the Editor

TO THE EDITOR

Vecuronium induced prolonged paralysis in two pediatric intensive care patients

Neuromuscular blocking drugs (NMBD) are widely used to facilitate endotracheal intubation in intensive care units. These drugs are known to be useful especially in children with seizures, traumatic head injury, and respiratory failure.1,4 However, recently published reports give information about patients who had prolonged muscle weakness after discontinuation of NMBDs.3,4 These reports point out that the prolonged paralysis extends beyond the weaning period, necessitates rehabilitation, and increases the hospitalization period in most adult, and a few pediatric, critically ill patients. Although NMBD have been thought to be responsible for prolonged paralysis in those patients, critical illness neuropathy, infections, steroid myopathy, and other drugs should be considered in the differential diagnosis.4,9 We report two patients with spontaneously resolved prolonged paralysis attributed to vecuronium therapy in our pediatric intensive care unit.

Patient 1: A two-month-old girl who was mechanically ventilated for respiratory failure due to Klebsiella pneumoniae pneumonia in our pediatric intensive care unit. She had been operated on for anomalous origin of left coronary artery from the pulmonary artery that was reimplanted to the aorta. She was admitted to our unit with pneumonia the second day after heart surgery. We applied vecuronium infusion to achieve neuromuscular blockade. Midazolam infusion was also given for sedation. Neurological examination was unremarkable. She received high-dose corticosteroid therapy (2 mg/kg/day, prednisolone, 14 days) for bronchial hyperreactivity. Vecuronium was given at a dose of 0.1 mg/kg/hour, for 26 days (total dose was 374.4 mg). At the time of vecuronium cessation her neurological examination revealed hypotonicity, absence of active movements of head and extremities, and absent deep tendon reflexes (DTR). Her DTR, extremity movements returned to normal three days after cessation of vecuronium, respectively. Creatinine kinase was 52 IU/L (0-170). Electromyogram and muscle biopsy could not be performed. She had sufficient respiratory effort and was easily extubated. He returned to normal daily activities without neurological deficit only seven days after vecuronium cessation.

Prolonged paralysis has been reported in patients managed in intensive care units,1,2,5-7 Currently available literature consists of only case reports. No randomized trials are available, making it difficult to draw definite conclusions about underlying mechanisms. Prolonged paralysis attributed to NMBD may be related to associated factors, such as multi organ failure especially renal/hepatic, concomitant drugs (mainly aminoglycosides and corticosteroids), acid-base, electrolyte disturbances and atrophy as a result of non-use of muscles. These factors act by altering NMBD clearance and/or potentiating the neuromuscular junction.3,7,10 The presence of hypokalemia, respiratory acidosis, hypermagnesemia and hypothermia and the administration of too many antibiotics can result in failure of reversal or the recurrence of blockade due to NMBD. The most common antibiotics blamed for prolonged NMBD effect are aminoglycosides, tetracyclines, lincomycin, and polymyxin. Numerous other pharmacologic agents potentiate the effects of NMBD: local anesthetics, calcium channel blockers, nitroglycerine, high-dose corticosteroids, furosemide, and alkylating cytotoxic agents.2,5 We consider that prednisolone could have contributed to the prolonged effect of vecuronium in Patient 1. The safe dose of NMBD may vary from patient to patient, drug medication and also dependent concomitant drugs and conditions.

In conclusion, we have to use NMBD for intubation and mechanical ventilated patients in pediatric intensive care units. However, these drugs have some morbidity in long term use. Neuromuscular blocking drugs, such as vecuronium, should be stopped when patients recover, to prevent prolonged paralysis.


TO THE EDITOR

Re: Exacerbation of Pre-existing Epilepsy by Mild Head Injury

Drs. Tai and Gross recently reported an exacerbation of pre-existing epilepsy in a series of patients following mild injury to the brain. The authors lay claim to a causal connection by way of cerebral insult rather than the effects of stress.

Unfortunately there was no assessment of seizure frequency in a group of control individuals receiving injuries other than to the brain. The authors suggest that because the increase in seizure frequency was prolonged following the brain injury, it is unlikely that the increase was solely due to stress. However, an adjustment reaction following injury may be prolonged for a period of years, notably in those designated as having post-traumatic stress disorder. Neuronal plasticity changes may take place in the limbic circuitry of chronically stressed individuals regardless of injury or type of injury.

It is possible that, unwittingly, Drs. Tai and Gross may have included two, or even three, injured individuals without brain trauma in their series of five, namely those without a documented blow to the head. The authors assumed there was brain injury solely as a result of deceleration. However, brain injury without head contact in adults is so rare that it is almost never seen in a clinical setting in civilian life.

The authors may be right in supporting a direct relationship between exacerbation of seizure disorder and a minor injury – regardless of whether or not there was trauma to the brain.

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RESPONSE

Exacerbation of Pre-existing Epilepsy by Mild Head Injury

While we had considered the possible role of stress, none of our patients met DSM-IV-TR criteria for post-traumatic stress disorder and, therefore, it remains our opinion that the most likely explanation for seizure exacerbation was head trauma. As our series was retrospective, some accidents occurred years before presentation. Based on the nature of the accidents, we suspect some degree of head trauma likely was present in all patients. We presented this series because we were struck by the temporal relationship between minor accidents and exacerbation of seizures in epileptic patients. Further study is required to ascertain whether what has previously been considered trivial head injury can provoke seizures in epilepsy patients.

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