That PACU Patient Looks Floppy

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Objectives

1. Understand the prevalence, risk factors, and complications of residual neuromuscular blockade.
2. Describe treatment options to prevent and treat residual neuromuscular blockade
3. Identify the role of suggamadex in preventing residual neuromuscular blockade

A 62yo female is brought to the post-anesthesia care unit following a four-hour multilevel lumbar fusion. You have assumed care of the patient during her recovery so that your partner can be released of duty. Fifteen minutes after arrival, the bedside nurse requests that you evaluate the patient because of uncoordinated movement.

1. What are the possible causes for uncoordinated movement in the PACU?
2. How would you differentiate between and what are the common risk factors for each etiology?

The patient appears to be hemodynamically stable and has an oxygen saturation of 98% on 4L O2 by nasal cannula. You review the intraoperative record and note that the 84 Kg patient received 50mg of rocuronium at the time of induction with intermittent doses throughout the case. Their last dose of rocuronium was 20mg approximately 90 minutes prior to extubation. The patient was documented to have 4/4 twitches on TOF testing prior to receiving a 2.5mg dose of neostigmine. There is no further documentation of neuromuscular function in the chart and the patient was extubated 10 minutes after reversal. You suspect the patient may have residual neuromuscular blockade.

3. How can you evaluate the patient for residual weakness in the PACU?
4. How does the validity of this clinical evaluation compare to twitch monitoring?
5. What is the optimal TOF ratio prior to extubation?

6. How accurate is the perception of TOF ratios and what is the "zone of blind paralysis?"

You call the CRNA who staffed the case to enquire about neuromuscular blockade monitoring prior to extubation. The CRNA states that the patient was monitored at the eye and that there were four strong twitches after neostigmine was administered. They do admit, however, that there may have been mild fade.

7. What are the implications of differing anatomic locations for twitch monitoring?
8. Is there any connection with the location monitored and residual neuromuscular blockade?
9. How do you determine the dose of neostigmine for reversal of neuromuscular blockade?
10. Are there any negative consequences to administering neostigmine to a patient who has had spontaneous recovery of neuromuscular function?

The patient continues to maintain adequate oxygenation despite her uncoordinated movements.

11. How does residual neuromuscular blockade effect PACU and hospital length of stay?
12. Does her adequate oxygenation reflect a relative protection from pulmonary complications?

Your eager call resident questions why the patient would receive an antiquated medication like neostigmine.

13. How does the introduction of suggamadex alter the incidence of residual neuromuscular blockade?
14. How does the mechanism of suggamadex differ from neostigmine?
15. How would you decide whether to reverse a patient with suggamadex or acetylcholinesterase inhibiting agents?
16. What are the relative and absolute contraindications to suggamadex?

You decide to defer re-intubation given the patient's relative stability and adequate oxygenation. You elect to treat the patient's residual neuromuscular blockade pharmacologically.

17. Is it safe to administer suggamadex after acetylcholinesterase inhibitors?
18. Are there any potential negative consequences?

The patient receives additional neostigmine and regains full motor function. She becomes concerned that her care was inappropriate.

19. How would you discuss the cause of her weakness?

Discussion Guide:

The patient with uncoordinated movement in the PACU could be suffering from an array of different complications. These etiologies range from the common problems, such as residual neuromuscular blockade, to more rare complications such as cerebrovascular accidents, extrapyramidal symptoms, or even serotonin syndrome. Residual neuromuscular blockade remains the most prevalent cause with an incidence of approximately 40% (1). A review of the patient's history and anesthetic record, as well as a thorough physical exam should help in identifying the specific etiology and preferred treatment.

The accurate evaluation of neuromuscular blockade is vital to ensuring proper reversal at the time of extubation. TOF monitoring throughout the operative period allows for timely re-dosing of neuromuscular blockers as well as planning for extubation. Unfortunately, subjective TOF testing has been shown to be inadequate for predicting residual neuromuscular blockade. The presence of fade in the TOF has been shown to be imperceptible to anesthesia providers even when it exceeds 0.50. TOF fade of between 0.40 and 0.90 has been described as the "zone of blind paralysis" because of the inability of anesthesia providers to reliably detect differences over this range (2). A TOF ratio of less than 0.90 is currently used to define residual neuromuscular blockade. Below this threshold, the incidence of signs and symptoms of residual blockade rises dramatically (3).

The choice of location for TOF monitoring has been shown to affect the incidence of residual neuromuscular blockade. Multiple studies have demonstrated a faster recovery of fade when the TOF monitoring has been performed at the eye muscles as opposed to the adductor pollicis. (4-6). Thilen et al. demonstrated an odds ratio of 5.48 for residual neuromuscular blockade when TOF monitoring was performed at the orbicularis oculi as compared to the adductor pollicis (7). The adductor pollicis remains the preferred site for TOF monitoring.

The ability to detect residual neuromuscular paralysis by clinical evaluation is also limited. The tests that are often recommended include handgrip, leg lift, and sustained head lift (8). Kopman et al, however, demonstrated that otherwise healthy individuals could adequately perform these clinical tests across a broad range of train-of-four fade ratios. Some patients were able to sustain head lift and leg lift with train-of-four fade ratios of 0.50 (3).

The successful reversal of neuromuscular blockade with anticholinesterase medications appears to be dependent on both timing and dose. Reliance on spontaneous recovery of neuromuscular function without pharmacological reversal has been shown to be inadequate (9-11). McClean et al demonstrated that risk of

postoperative complications significantly reduced when reversal was performed with neostigmine after recovery of the second twitch on TOF and at a dose of less than 60ug/kg (12). Reversal higher doses of neostigmine have been associated with worsening pulmonary outcomes and prolonged PACU stays despite evidence adequate oxygenation (13).

The introduction of suggamadex in the US market has brought a unique mechanism of action to the reversal of rocuronium and vecuronium. The use of suggamadex returns the neuromuscular junction to its native state, without the risk of residual weakness from increased acetylcholine activity or adverse events associated with muscarinic activity that are associated with acetylcholinesterase inhibitors. In comparative trials, suggamadex has been shown to produce a TOF ratio of >0.9 in significantly less time than neostigmine (5 min vs. 50 min). This has clear implications on the prevalence of residual neuromuscular blockade in the recovery room (14). The use of suggamadex is contraindicated in patients with prior anaphylaxis to the medication and in those with severe renal impairment with a creatine clearance <30mL/min (Merck Product insert). The use of suggamadex for rescue of residual neuromuscular blockade following reversal with acetylcholinesterase inhibitors has not been studied. There are two case reports of successful rescue in this situation, but further studies should be performed (15).

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