The clinical nurse anesthetist as diagnostic sleuth

Two cases are presented where observations made during anesthetic management led to appropriate diagnoses that otherwise might not have occurred in a timely fashion. In the first case, an early diagnosis of myasthenia gravis was facilitated; while in the second case, an improbable response led to rethinking of the initial diagnosis. The role of the nurse anesthetist in being alert to the consequences of unusual clinical and theoretical responses on behalf of the patient is depicted.

Key words: Differential diagnosis, electromyography, monitoring, myasthenia gravis.

This is a report of the experiences with two recent patients where clinical observation under anesthesia led to reconsideration of initial medical diagnoses. The intent is to suggest that nurse anesthetists may occasionally find themselves in unique vantage points from which to strategically participate in the clarification and diagnosis of pathophysiologic processes.

Case I

A healthy 42-year-old female outpatient, weighing 67 kilograms, presented for a breast biopsy. The history and physical were completely unremarkable for anything other than two uneventful vaginal deliveries 20 and 24 years ago. The patient was employed as a high school science teacher and attended an aerobics class twice weekly. She was taking no medications, had no allergies and had a hemoglobin of 11.5 gm/dl.

The patient was brought to the operating room and, with the appropriate monitors in place, was induced with thiopental (3 mg/kg) and sufenta 10 μg. She was ventilated with nitrous oxide and oxygen (1:1) and isoflurane 0.5-3%; the plan was to mask ventilate for the duration of the procedure.

During the surgical preparation, vecuronium 2 mg was given intravenously to facilitate akinesis during a less hemodynamically intrusive level of isoflurane. It was noted that this dose of vecuronium was associated with a complete loss of electromyographic monitored neuromuscular function (Figure 1, Point B). The decision was made to intubate the patient, which was accomplished without vocal cord or diaphragmatic movement. A modified radical mastectomy ensued and 85 minutes following the first vecuronium dose (with approximately 50% recovery of initial twitch amplitude and train-of-four still significantly depressed), a “challenge” dose of vecuronium 0.5 mg was given to the patient (Figure 1, Point C), with resultant major decrement in neuromuscular function.

The surgical procedure was completed at the 145-minute mark (Figure 1, Point D), and 100% oxygen was administered, with the train-of-four ratio near 100% twitch height approximately 75% of control. Neostigmine 3.0 mg and glycopyrrolate 0.6 mg was given intravenously over 90 seconds. Within 5 minutes, the patient was reparaalyzed (Figure 1, Point E); she was sedated with midazolam 3 mg and transferred to the recovery room with controlled ventilation. Approximately 105 minutes later,
extubation criteria intact, the patient was extubated without incident.

An internist was consulted, and based on the findings intraoperatively, diagnostic work was initiated which revealed a high antiacetylcholine receptor antibody titer—24.8 pmol/ml, normally <0.2 pmol/ml—and the patient was diagnosed as "premyasthenic." She is to be followed very closely by her internist over the coming year.

Case II

A 19-year-old, 58 kg, mentally retarded man diagnosed four years earlier with ocular myasthenia gravis, because of persistent diplopia and ptosis, presented for a dental rehabilitation procedure. His medications consisted of thorazine 25-50 mg intramuscularly (as occasion requires for severe agitation) and a pyridostigmine “timespan” tablet (180 mg) each day for “ocular myasthenia,” which was withheld on the day of surgery. The patient appeared robust, his history and physical were unremarkable and his hemoglobin was 14.5 gm/dl.

Following premedication with midazolam 20 mg intramuscularly in the receiving room, an intravenous access was achieved and an additional 5 mg of midazolam was given intravenously prior to transfer to the operating room. With the appropriate monitors in place, induction commenced with thiopental 2 mg/kg, sufenta 20 µg and mask ventilation with isoflurane 1-4% in oxygen in preparation for intubation. Two attempts at intubation were unsuccessful because the patient appeared “tight.” Vital signs and end-tidal CO₂ were satisfactory, and a decision was made to titrate vecuronium to effect.

The history of myasthenia required the authors to proceed especially conservatively and yet repeated administration of vecuronium appeared to have marginal effect (Figure 2, Points A-G). Finally after 9 mg of vecuronium (freshly constituted for this case), the patient was successfully intubated (Figure 2, Point H) with a twitch height only approximately 50% depressed and four responses present to the train-of-four. A subsequent “challenge” of 2 mg of vecuronium was given at the 17-minute mark (Figure 2, Point I) with only a moderate effect noted.

The surgical procedure was completed after 150 minutes, and the patient was noted to have completely recovered from the vecuronium. Following discontinuation of the isoflurane, the patient rapidly aroused with obvious sustained strength in all muscle groups and was extubated without the administration of anticholinesterase.

An internal medicine follow-up discovered a normal antiacetylcholine receptor antibody titer and a normal response to a sedated edrophonium/ electromyographic challenge. A subsequent CAT scan revealed a tumor encapsulating each of the immediate retrobulbar areas, which was apparently causing this man’s ocular symptoms. He is currently being scheduled for resection of these tumors.

Discussion

Myasthenia gravis (MG) is a disorder of the neuromuscular junction, which probably is a result of an autoimmune-induced loss of acetylcholine receptors postsynaptically and/or an antibody effect at the receptor’s ion channel through which sodium and potassium traverse the cell mem-

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<td>Electromyographical representation of Case I</td>
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- A—Vecuronium 2 mg
- B—80% reduction in twitch height from control and complete loss of train-of-four response
- C—"Challenge" dose of vecuronium 0.5 mg
- D—Neostigmine 3 mg and glycopyrrolate 0.6 mg
- E—85% loss of twitch height from control following administration of reversal mixture

Note: Time across x-axis in hours and minutes
// shows time break on x-axis
brane. Because of the abnormal response to both depolarizers (resistance) and nondepolarizers (sensitivity), many authorities advocate avoiding the use of muscle relaxants in patients diagnosed with MG. Anticholinesterases, despite being a therapeutic mainstay in the management of MG, may paradoxically worsen the symptoms of MG when given to reverse the effects of a nondepolarizer.

Previous reports have suggested that anesthesia can play a role in the diagnosis of MG. Unrelated to MG, but in a diagnostic theme, a clinical episode has been reported where an anesthetist's astute observation and suspicion played a pivotal role in the discovery of a totally unsuspected condition—murder.

The purpose in reporting these cases was not to elaborate upon the management of MG but rather to illustrate two related examples of how nurse anesthetists might participate in the discovery, corroboration or reconsideration of a diagnosis based on enlighed observations made during the course of their clinical management. Similar opportunities may manifest during much different scenarios (e.g., asthma, hypertension, tumors of the oropharynx, etc.). We wish to encourage our colleagues to have a high index of suspicion and be alert to the consequences in patients who do not respond in the manner in which one has come to expect, whether it be from a clinical or theoretical perspective.

REFERENCES


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