Recovery characteristics following antagonism of vecuronium with edrophonium, neostigmine or pyridostigmine

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Vecuronium-induced neuromuscular blocks in 42 ASA physical status I and II females were antagonized with atropine and equipotent doses of edrophonium, neostigmine and pyridostigmine. Recovery characteristics including speed of reversal, dysrhythmias and mean arterial pressure changes were studied. Edrophonium was associated with the fastest neuromuscular recovery. No clinically significant cardiovascular changes were found in any group.

It is imperative that any newly developed drug, along with its effects, dosage and interactions with other agents, be evaluated and documented. Vecuronium (Norcuron®) is one of the new neuromuscular blocking agents in use. It is a nondepolarizing agent of rapid onset and short duration, with minimal adverse systemic reactions. Secondary to its pharmacodynamics, vecuronium continues to gain increasing use and popularity as a surgical muscle relaxant.

Reversal of vecuronium can be accomplished with one of the anticholinesterase agents. The choice of which to use has been a personal one because there is limited documentation as to which of the reversal agents works best with vecuronium. A recent investigation by Kopman illustrates the point that ease of reversal is a function of several factors including the type of blocking agent used, the level of paralysis, the anticholinesterase chosen and the experimental conditions of the body. Miller has emphasized that a myriad of studies have evolved concerning neuromuscular blocking agents, their antagonists and multiple combinations of the factors just mentioned. Being able to apply these data to clinical anesthesia practice is of prime importance. In this study, edrophonium, neostigmine and pyridostigmine were evaluated as antagonists of vecuronium to determine if there was a significant difference between the three agents with regard to speed of reversal and cardiovascular stability.

Methods

This study utilized a quasi-experimental research design. The reversal agents served as the independent variables. Evaluation of the return of the train-of-four, the presence/absence of dysrhythmias and changes in mean arterial pressure (MAP) served as the dependent variables. Forty-two female patients undergoing laparoscopy were investigated after giving informed consent to a study approved by the Clinical Investigation and Human Use Committees of the affiliated hospital. All patients studied were ASA physical status I or II, non-obese and free of neuromuscular disease, upper extremity dysfunction, and hepatic or renal dysfunction by history. The patients were normothermic and taking no medications known to interfere with neuromuscular transmission. Serum potassium, calcium and magnesium were within normal levels.
The patients were assigned to one of three groups using a computer-generated, random number table. Group E received edrophonium 0.5 mg/kg; Group N received neostigmine 0.043 mg/kg; and Group P received pyridostigmine 0.21 mg/kg. All groups concomitantly received atropine 0.015 mg/kg as the anticholinergic agent. A CRNA assistant prepared the reversal agent in a specially coded syringe with identical volumes in comparative syringes. The researchers were not aware of the group assignment of each individual patient.

The vecuronium, atropine and anticholinesterase agents were obtained from identical respective lots. All patients were monitored with the same automatic auscultatory blood pressure monitor (Dinamap® by Critikon), Hewlett Packard Capnometer® 47210A, neurotechnology peripheral nerve stimulator and a Medar APM® force displacement transducer. All monitors were calibrated and maintained to manufacturer's specifications.

The patients received no premedications and had nothing by mouth after midnight. In the operating room "prehold" area, an intravenous line was started in a convenient site of each patient's arm. A blood pressure cuff was placed on the other arm and electrocardiogram (ECG) electrodes were applied to monitor lead II. Continuous monitoring of ECG was accomplished using a Hewlett Packard monitor 78534B. Blood pressure was ascertained and recorded every five minutes using a Dinamap® monitor during the surgical procedure. Blood pressure was measured more frequently if needed using the "demand" mode.

Patients were preoxygenated with 6 L/min. by mask. Fentanyl 2-3 μg/kg and droperidol 1.25 mg were given intravenously. Anesthesia was induced with sodium thiopental 5 mg/kg. Once the patient was asleep, 27-gauge needle electrodes were placed over the ulnar nerve at the wrist and a supramaximal stimulus was delivered. The muscle twitch of the adductor pollicis muscle was recorded using a Medar APM® force displacement transducer connected to a Hewlett Packard preamplifier and recorder unit. Control values for twitch height and train-of-four were recorded. Vecuronium 0.05-0.10 mg/kg was given for intubation. When twitch height was less than 25% of control, intubation of the trachea was performed using direct laryngoscopy.

Anesthesia was maintained with $N_2O$ 67% and $O_2$ 33% via a semiclosed circle absorber system. Fentanyl 50-100 μg or sodium thiopental 50-75 mg were supplemented as needed. If twitch height returned to 25% of control before the end of the surgical procedure and additional muscle relaxation was needed, vecuronium 0.01 mg/kg was given. Controlled ventilation was maintained with respiratory rate and tidal volume measured by the Ohmeda 5400 Volume Monitor® on the Ohio Modulus II® anesthesia machine. Ventilations were delivered to maintain end-tidal $CO_2$ at 35-40 torr as measured by Hewlett Packard end-tidal $CO_2$ monitor. A nasopharyngeal temperature probe was placed and temperature was measured on the Hewlett Packard monitor. Temperature was maintained between 35.5 and 37.5°C by use of a K-thermia® blanket.

On removal of the laparoscopic trocar, the train-of-four was evaluated and a continuous recording of ECG and train-of-four at 10 second in-

### Table I
Demographic comparison

<table>
<thead>
<tr>
<th></th>
<th>Edrophonium</th>
<th>Neostigmine</th>
<th>Pyridostigmine</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (years)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>21-36</td>
<td>21-37</td>
<td>21-37</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>27.7 ± 4.1</td>
<td>27.2 ± 5.2</td>
<td>28.7 ± 4.2</td>
</tr>
<tr>
<td><strong>Height (inches)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>60-67</td>
<td>62-69</td>
<td>62-70</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>64.4 ± 2.3</td>
<td>64.8 ± 2.1</td>
<td>65.8 ± 2.5</td>
</tr>
<tr>
<td><strong>Weight (kg)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>41-73</td>
<td>45-79</td>
<td>46-77</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>61.5 ± 8.4</td>
<td>61.7 ± 10.2</td>
<td>62.1 ± 10.0</td>
</tr>
</tbody>
</table>

SD = Standard deviation
p value <0.05

Journal of the American Association of Nurse Anesthetists 128
tervals was initiated. MAP and heart rate were then recorded every minute. With the closure of the incision, the prepared reversal agent was administered intravenously over 20 seconds. When neuromuscular recovery was reached (T4 ratio = 0.75), the N₂O was discontinued and 100% O₂ was delivered with controlled or assisted ventilations until spontaneous ventilation returned.

The time from injection of reversal agent to return of T4 ratio to 0.75 was ascertained. The mean reversal time of each group was computed and analyzed using a Student’s t-test and analysis of variance (ANOVA) to determine whether statistically significant differences existed. Statistical significance regarding the incidence of dysrhythmias and MAP changes was examined using chi-square tests with 2x3 contingency tables. Level of significance for all analyses was p < 0.05. Statistical analysis was computed on an IBM PC computer using the Epistat® software program.

**Results**

Thirty-eight ASA physical status I and four ASA physical status II females undergoing laparoscopic procedures were randomly assigned to one of three groups: Group E (n=13), Group N (n=12) and Group P (n=13). ANOVA showed no statistical differences in demographic data between the three groups (Table I). Four patients were not included secondary to a lack of spontaneous return of neuromuscular activity at time of reversal. Data on these patients were not included in the statistical analysis.

Anesthesia was maintained with mean doses of the agents indicated in Table II. Mean surgical time was 32.8 minutes with a range of 11-72 min-

<table>
<thead>
<tr>
<th>Table II Anesthetic doses, surgical time and recovery data</th>
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</thead>
<tbody>
<tr>
<td>Sodium thiopental mg/kg</td>
</tr>
<tr>
<td>Range</td>
</tr>
<tr>
<td>Mean ± SD</td>
</tr>
<tr>
<td>Fentanyl µg/kg</td>
</tr>
<tr>
<td>Range</td>
</tr>
<tr>
<td>Mean ± SD</td>
</tr>
<tr>
<td>Vecuronium mg/kg</td>
</tr>
<tr>
<td>Range</td>
</tr>
<tr>
<td>Mean ± SD</td>
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<tr>
<td>Surgical time (minutes)</td>
</tr>
<tr>
<td>Range</td>
</tr>
<tr>
<td>Mean ± SD</td>
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<tr>
<td>T4 ratio at reversal</td>
</tr>
<tr>
<td>Range</td>
</tr>
<tr>
<td>Mean ± SD</td>
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<tr>
<td>T4 recovery to 0.75 (sec)</td>
</tr>
<tr>
<td>Range</td>
</tr>
<tr>
<td>Mean ± SD</td>
</tr>
<tr>
<td>Dysrhythmias present (number)</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>mean arterial pressure changes ± 20%</td>
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SD = Standard deviation
* = Differs from Groups N and P; p <0.05
** = Differences found between the groups per chi-square; p <0.05
utes. Mean T4 ratio prior to reversal for each group was: Group E = 0.246, Group N = 0.241, Group P = 0.114. ANOVA showed no statistical differences between the three groups (Table II).

Mean times from administration of the reversal agent to a T4 ratio of 0.75 can be compared for each group in Table II. ANOVA showed a significant difference between the groups. Further analysis with Student's t-test demonstrated that the reversal time in Group E was significantly less than Group N or P. However, Group N was not statistically different from Group P (Table II).

Dysrhythmias were noted with all reversal agents. Chi-square analysis showed no statistical difference between the groups with p > 0.05. Mean arterial pressure changes of 20% were noted with all agents. Chi-square analysis showed a statistical difference between the groups with p < 0.05 (Table II).

Discussion. Results showed that edrophonium had a significantly faster neuromuscular recovery time than either neostigmine or pyridostigmine in the presence of a vecuronium-induced neuromuscular block. This is consistent with the findings of Cronnelly et al. and Harper et al. in the reversal of other non-depolarizing neuromuscular blocking agents. Baird et al. showed that edrophonium was twice as fast as neostigmine in reversing vecuronium in cats. This is consistent with the authors' results of edrophonium being 2.3 times faster than neostigmine.

Ferguson et al. studied the reversal of pancuronium-induced neuromuscular blocks and found that edrophonium was faster than neostigmine, which was faster than pyridostigmine in the return of the T4 ratio. The authors' study differed from these findings in that there was no difference in the speed of reversal between neostigmine and pyridostigmine in vecuronium-induced blocks.

At the neuromuscular junction, acetylcholine is normally hydrolyzed into acetic acid and choline by the enzyme acetylcholinesterase. During the hydrolysis, the quaternary group of the choline combines by electrostatic attraction to the anionic site of the acetylcholinesterase, and the carbamate group interacts at the esteratic subsite. The choline splits off and the acetylated enzyme which remains rapidly hydrolyzes to form acetic acid and regenerated active enzyme. The choline is taken up by the presynaptic nerve terminal for the formation of more acetylcholine, and the acetic acid is dispersed into the extracellular fluid.

Anticholinesterase agents inhibit acetylcholinesterase by reversibly combining at either the active center site or a peripheral ionic site. Acetylcholine cannot be hydrolyzed as long as the anticholinesterase agents are combined with the acetylcholinesterase sites. Therefore, the concentration of acetylcholine increases at the neuromuscular junction.

Edrophonium is a potent, reversible, competitive inhibitor of acetylcholinesterase which competes with acetylcholine for the anionic site. Its duration of inhibition is brief due to the reversibility of binding. Both neostigmine and pyridostigmine transfer a carbamate group to the enzyme, which chemically combines at the esteratic site. The acetylcholine cannot bind to the enzyme until the carbamate enzyme is hydrolyzed. This hydrolysis takes place at a slower rate than the hydrolysis of acetylcholine, thus producing a longer lasting inhibition of the acetylcholinesterase. This correlates with past thoughts that edrophonium did not have a sufficient duration of action to effectively reverse neuromuscular blocking agents.

Edrophonium, neostigmine and pyridostigmine have been shown to increase miniature end-plate potential frequency, quantal release, size and rate of the available acetylcholine stores of the nerve terminal. The effects of these drugs on the nerve terminal do not appear to result from the inhibition of acetylcholinesterase. Neostigmine has also been found to cause direct stimulation of the end-plate region, which has not been found with either edrophonium or pyridostigmine.

A 1982 study showed that the magnitude of antagonism produced by edrophonium increased with dose; however, the duration of antagonism was in a linear relationship only to a dose of 0.125 mg/kg. If the duration of antagonism was directly proportional to the duration of enzyme inhibition, then greater blood levels produced with increased edrophonium dosage should continue to lengthen the time of inhibition and, therefore, antagonism. This study also found that edrophonium appears to have predominantly presynaptic effects.

Studies by Cronnelly and Morris have demonstrated that equi-antagonistic doses of neostigmine and edrophonium have the same duration of action. Pyridostigmine, however, had a 40% longer duration of antagonism than either edrophonium or neostigmine. Previous practices and studies that resulted in inadequate duration of action of edrophonium were based on doses less than equi-antagonistic. Edrophonium 0.5 mg/kg has been found to be equivalent to neostigmine 0.043 mg/kg for the reversal of nondepolarizing neuromuscular blocking agents.

Edrophonium has been gaining popularity as

Journal of the American Association of Nurse Anesthetists
a reversal agent for several reasons. It has a more rapid onset of action and has less muscarinic effects, thereby requiring less anticholinergic drug to counteract them. The assumption is that a faster recovery with less cardiovascular instability can be obtained with edrophonium.

Edrophonium is a smaller compound with a molecular weight of 166 compared to 223 for neostigmine. It has been suggested that the factor that limits the speed of onset of action may be the rate at which the cation diffuses from the plasma to the receptor sites. The smaller molecular size and higher molar dose of edrophonium may provide a more rapid diffusion to the site of action. Edrophonium has an onset of action of 0.8 - 2 minutes, neostigmine, 8 - 11 minutes, and pyridostigmine, 12 - 16 minutes. Differences in onset of action may be due to times required for enzymatic inhibition.

Studies of the reversal of alcuronium, using train-of-four, showed a more rapid recovery of first contraction response and achievement of plateau with edrophonium than either neostigmine or pyridostigmine. Studies of the reversal of d-Tubocurarine demonstrated a significantly faster onset of action of edrophonium 0.5-1.0 mg/kg over neostigmine 0.043 mg/kg and pyridostigmine 0.21 mg/kg. Comparative studies of the antagonism of pancuronium with neostigmine, pyridostigmine and edrophonium showed a significantly more rapid return of first response of train-of-four and T4/T1 ratio with edrophonium. In studies with cats, edrophonium was found to produce full antagonism of vecuronium twice as rapidly as neostigmine.

Numerous dysrhythmias were noted in each group and warrant delineation. Group E showed two episodes of junctional rhythm, two of first degree atrioventricular (AV) block, four of second degree AV block and three of sinus tachycardia. Group N showed two occurrences of junctional rhythm, one of first degree AV block, one of second degree AV block, one of third degree AV block, one of sinus bradycardia and three of sinus tachycardia. Group P demonstrated two episodes of junctional rhythm, one of second degree AV block, one of premature ventricular contractions, three of sinus tachycardia and one of sinus bradycardia. Of all rhythms seen, none caused hemodynamic compromise as evidenced by the fact that all patients maintained an adequate MAP, and all rhythms reverted to normal sinus rhythm without intervention. Although the occurrences were not statistically different between groups, all criteria for chi-square analysis were not met. Specifically, observed values were not greater than or equal to five. This must be considered in the interpretation of these findings.

With the inhibition by anticholinesterase, the subsequent increase in acetylcholine occurs not only at the neuromuscular junctions, but also at the cholinergic receptors of the parasympathetic nervous system. If this is allowed to occur unchecked, one of the most clinically significant results is bradycardia secondary to vagal stimulation. An anticholinergic agent, usually atropine or glycopyrrrolate, is given with the anticholinesterase agent. Edrophonium has been found to require less atropine than either neostigmine or pyridostigmine to prevent muscarinic effects. It has been suggested that an antagonist that requires less atropine would be associated with fewer cardiac dysrhythmias.

Several studies have shown conflicting conclusions, including (1) that there is no significant difference in dysrhythmias between groups reversed with edrophonium or neostigmine; (2) that there is a higher incidence of dysrhythmias with the neostigmine group; and (3) that there are no dysrhythmias but wider fluctuations in heart rate with neostigmine. Fogdall et al. demonstrated that the cardiac muscarinic side effects of neostigmine and pyridostigmine were similar during antagonism of a d-Tubocurarine-induced blockade.

The authors chose atropine as the anticholinergic agent to reduce the number of variables in this study. The dose of 0.015 mg/kg was selected to assure adequate antimuscarinic protection for the neostigmine and pyridostigmine groups. In spite of this, no difference was found in the incidence or type of dysrhythmias between the groups.

Analysis of MAP changes was statistically significant between groups. Group E showed the largest number of patients with MAP changes, while Group N had the fewest. Changes in all groups were well within accepted clinical limits. The majority of changes were increases, which correlates with the general findings of Mirakhur et al. and Cozanitis et al. These researchers suggested a causal relationship between these changes and the lightening of anesthesia. As with dysrhythmia analysis, all criteria for chi-square analysis were not met.

Other variables in the reversal of neuromuscular blocking agents have been documented. Antibiotic interactions with muscle relaxants have been well documented with no one mechanism of action found for all antibiotics. Some are thought to produce neuromuscular blockade by inhibition of acetylcholine release from the presynaptic nerve terminal and by stabilization of the postjunctional membrane, whereas others have just a postsynaptic effect. Neostigmine can successfully antagonize some but not all of these blocks.

Local anesthetics and antidysrhythmics en-
hance the neuromuscular block of both nondepolarizing and depolarizing agents. Quinidine in particular has been noted to potentiate neuromuscular blocks, and edrophonium has been ineffective in antagonizing a nondepolarizing block after the administration of quinidine.17

Electrolyte imbalances also will affect neuromuscular blockade and its antagonism. Hyperkalemia will enhance a depolarizing muscle relaxant and oppose the action of the nondepolarizing agents. Magnesium enhances the relaxant properties of both depolarizing and nondepolarizing agents. Hypocalcemia has also been found to augment muscle relaxants.8

Respiratory acidosis enhances nondepolarizing neuromuscular blocking action and opposes reversal by neostigmine.16 Antagonism is also blocked by metabolic alkalosis, but this may actually be related to electrolyte changes associated with the pH abnormality.8

Volatile anesthetic agents also augment the neuromuscular block produced by nondepolarizing muscle relaxants. A study by Rupp et al. demonstrated that enflurane was more potent than isoflurane or halothane in augmenting a vecuronium-induced neuromuscular blockade. This study also showed that increasing the concentration of volatile anesthetic had less effect on a vecuronium-induced block than on blocks induced by pancuronium or d-Tubocurarine. This study suggests that the end-tidal concentration of isoflurane (Forane®) from 0.5% to 1.5% had minimal effect on the neuromuscular block induced by vecuronium.18

Limitations to this study exist. The sample group was chosen from an available volunteer female population. The sample size was small. Four researchers collecting data along with several CRNA assistants preparing the reversal agents may have altered consistency in this study. The exclusion of data from four subjects must also be considered a limitation.

T4 ratio at the time of reversal was not controlled in this study and return of single twitch height (T1/Tc) was not calculated. Data from four subjects were not used due to a lack of spontaneous return of neuromuscular activity at the end of the surgical procedure. ANOVA showed that there was no difference in the T4 ratio between the groups; however, the range within the groups was 0.0-0.63. Baird et al. found that the magnitude of antagonism is dependent on the amount of spontaneous recovery of muscle twitch at the time of administration of the anticholinesterase.19 Kopman was unable to produce satisfactory reversal of neuromuscular blockade with edrophonium 0.5 mg/kg, when spontaneous return of the fade ratio was less than 0.10.20

In Kopman’s more current work, recovery with neostigmine was faster than with edrophonium when continuous infusion of pancuronium, vecuronium or atracurium had maintained a T1/Tc of 0.10.1

Studies by Graham et al. have suggested that the site of action of competitive neuromuscular blockers to produce twitch and train-of-four depression during onset and recovery of neuromuscular blockade may be different for each neuromuscular blocking agent.21 For these reasons, the comparison of findings between studies must cite the mode of neuromuscular stimulation being measured, which neuromuscular blocking agent was used, and the degree of control of muscle relaxation at the time of reversal.

Recommendations for future study include using a larger sample size comprised of male and female patients of ASA physical status I through IV. An alternative study could compare various anticholinesterase-anticholinergic combinations. Controlling the T4 ratio at time of reversal would be another consideration as well as comparing with T1/Tc.

Conclusions
Before incorporating the results of this study into individual practice, the reader should acknowledge the limitations present in this study. Variations in patients and/or anesthetic practice may alter recovery characteristics following antagonism of vecuronium. Being aware of the differences in recovery times may assist the practitioner in deciding when to administer the antagonist.

It was demonstrated within the population studied that edrophonium antagonized vecuronium-induced neuromuscular block faster than did neostigmine or pyridostigmine. No difference in reversal time was found between neostigmine and pyridostigmine. Only edrophonium was associated with statistically significant MAP changes; however, there were no clinically significant cardiovascular changes in any group.

REFERENCES

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