Anesthesiology
1996; 85:1253-9


# *Residual Postoperative Paralysis*

## *Pancuronium* Versus *Mivacurium, Does It Matter?*

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***Background:*** **Based on a train-of-four (TOF) ratio greater than 0.70 as the standard of acceptable clinical recovery, undetected postoperative residual paralysis occurs frequently in postanesthesia care units. In most published studies, detailed information regarding anesthetic management is not provided. The authors reexamined the incidence of postoperative weakness after the administration of long- and short-acting neuromuscular blockers because few, if any, such comparative studies are available.**

***Methods:*** **Ninety-one adult patients were studied. In group 1 (mivacurium, n = 35), anesthesia was induced with propofol/fentanyl and maintained with nitrous oxide, desflurane, and opioid supplementation. The response of the adductor pollicis to ulnar nerve stimulation was estimated by palpating the thumb. Mivacurium (0.20 mg/kg) was administered for tracheal intubation, and an infusion was adjusted to maintain the TOF count at 1. When surgery was completed, the infusion was discontinued. When a second twitch could be detected, 7.0 μg/kg atropine and then 0.5 mg/kg edrophonium were administered. At 5 and 10 min, the mechanical TOF response was measured. Additional measurements were recorded if possible. Patients were tracheally extubated and discharged from the operating room when they could respond to verbal commands and no TOF fade was palpable. In group 2 (pancuronium–desflurane anesthesia, n = 29), the protocol was identical to that of group 1, except that 0.07 mg/kg pancuronium was administered for tracheal intubation. Additional increments (0.5 to 1 mg) were given as needed. Antagonism was accomplished with 0.05 mg/kg neostigmine and 0.01 mg/kg glycopyrrolate. In group 3 (pancuronium propofol–opioid, n = 27), the protocol was identical to that of group 2, except that anesthesia was maintained with nitrous oxide and a propofol–alfentanil infusion. In all groups, patients were assessed until a TOF ratio of 0.90 or more was achieved**

***Results:*** **All of the patients in group 1 had TOF ratios greater than 0.80 on arrival in the postanesthesia care unit. Twenty of 35 patients had TOF ratios 0.90 or more while they were still in the operating room. Thirty-three of 35 patients had TOF ratios 0.90 or more within 30 min of reversal, and this value was reached in all patients by 45 min. Recovery parameters in groups 2 and 3 did not differ from each other. Hence data from these groups were pooled. Fifty-four of 56 patients who received pancuronium had TOF values of 0.70 or more, the remaining two patients had values of 0.6 to 0.7. In contrast to the mivacurium group, however, only four patients achieved a TOF ratio of 0.90 or greater while still in the operating room. Finally, eight of these patients did not achieve this degree of recovery within 90 min of reversal.**

***Conclusions:*** **These results suggest that if nondepolarizing neuromuscular blockers are administered using tactile evaluation of the TOF count as a guide, critical episodes of postoperative weakness in the postanesthesia care unit should occur infrequently even with long-acting relaxants. Nevertheless, if full recovery is defined as return to a TOF ratio of 0.90 or more, then short-acting agents would appear to offer a wider margin of safety. (Key words: Monitoring: neuromuscular; train-of-four. Neuromuscular relaxants: mivacurium; pancuronium. Postanesthesia care unit. Residual curarization.)**

AFTER nondepolarizing-induced neuromuscular block, an indirectly elicited train-of-four (TOF) fade ratio of 0.70 or more (at the adductor pollicis muscle) represents acceptable clinical recovery. More than 20 y ago, Ali and coworkers[1-3] indicated that obvious muscle weakness (ptosis, tracheal tug) was associated with TOF ratios less than 0.60. However, once this ratio had returned to a value greater than 0.70, they reported that sustained eye opening, head lift, and adequate mechanical respiratory reserve were consistently present. By this standard, Viby-Mogensen and associates[4] quickly noted that undetected residual paralysis occurred fre-

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Received from the Department of Anesthesiology, St. Vincent's Hospital and Medical Center of New York. Submitted for publication May 3, 1996. Accepted for publication August 6, 1996. Supported by a grant from the Glaxo Wellcome, Research Triangle Park, North Carolina.

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quently in the postanesthesia care unit (PACU). They observed that TOF fade ratios less than 0.70 were frequently found in patients recovering from traditional long-acting neuromuscular blockers when they arrived in the PACU. Other investigators confirmed this finding.[5,6] Additional work suggested that when neuromuscular blocking agents of intermediate duration were substituted for agents such as gallamine and pancuronium, the incidence of postoperative residual paralysis was reduced significantly.[7-10]

Nevertheless, reports continue to appear that show that residual weakness is still a common occurrence even with neuromuscular blocking drugs that have an intermediate duration.[11] In fact, at least one report suggests that TOF ratios less than 0.70 when patients arrive in the PACU may occur relatively frequently even after the administration of mivacurium.[12] These studies are disturbing because there is accumulating evidence that symptoms of residual weakness persist at TOF ratios as great as 0.90.[13] It is difficult, however, to evaluate most of these previously published reports. In most of them,[4-7] intraoperative monitoring of neuromuscular function with a peripheral nerve stimulator was not used or was used only sporadically. Even in studies in which peripheral nerve stimulator monitoring was used routinely, train-of-four counts (TOFC) at reversal were rarely reported. Barring this sort of information, we could argue that reports of residual weakness may merely represent an "artifact" of improper anesthetic management (intraoperative failure to monitor properly the extent of neuromuscular block) rather than a risk that is intrinsic to the drug.

Therefore we reexamined the issue of postoperative residual paralysis (including times to recovery of TOF ratios to more than 0.90) after the administration of nondepolarizing blockers of long and short duration because no comparable study has been published that also gives detailed information regarding anesthetic monitoring and management. We also compared the rates of recovery after antagonism of pancuronium-induced neuromuscular block in patients who did and did not receive a potent inhalation agent as part of their anesthetic management.

## Materials and Methods

We included 91 adult patients (ages 18 to 69 y) classified as American Society of Anesthesiologists physical status 1 or 2 who were having elective surgical procedures for which the administration of a muscle relaxant was appropriate. The expected total anesthesia time in all patients was expected to exceed 90 min. All patients were free of neuromuscular disease and within 25% of ideal body weight. Patients in whom difficulty with orotracheal anesthesia was anticipated were excluded from the protocol. Other exclusion criteria included a history of allergies to any study medication and current use of any drugs known to influence neuromuscular transmission. Institutional review was obtained before this project began, and all participants gave informed consent.

In group 1 (mivacurium–desflurane, n = 35), anesthesia was induced with 1.5 to 2.5 mg/kg propofol given intravenously plus 2 to 4 $\mu$g/kg fentanyl or 10 to 20 $\mu$g/kg alfentanil and maintained with inhalation of 60% to 65% inspired nitrous oxide plus desflurane (end-tidal concentration $< 5\%$) plus opioid supplementation as needed. Ventilation was controlled, and end-tidal $p_{CO_2}$ was maintained between 32 and 38 mmHg. Patients were sequentially assigned to one of three treatment groups. All anesthetics were administered by one of the investigators. The indirectly evoked response of the adductor pollicis muscle was estimated by palpating the slightly abducted thumb. Ulnar nerve stimulation was effected using a Fisher-Paykell constant-current nerve stimulator at a milliamperage deemed appropriate by the clinician (30 to 50 mA). Mivacurium (0.20 mg/kg) was administered by infusion pump over approximately 90 s, and the patient's trachea was intubated approximately 1.5 min later. A mivacurium infusion (5 $\mu g \cdot kg^{-1} \cdot min^{-1}$) was begun as soon as some evidence of neuromuscular recovery was present and was then adjusted to provide a level of twitch depression at which one palpable response to TOF stimulation was obtained. Relaxant administration was guided solely by the train-of-four count as palpated at the thumb.

When the surgical procedure was complete, the infusion was discontinued and patients were allowed to recover spontaneously. As soon as a second twitch was palpable, 0.5 mg/kg edrophonium preceded by 7 $\mu$g/kg atropine were administered intravenously. Five minutes later, the actual TOF ratio was quantitated for the first time. The indirectly evoked mechanical response of the adductor pollicis muscle was measured using a Life-Tech Myotrace APM linear force transducer (Houston, TX), and a Gould WindoGraf electrophysiology monitor (Valley View, OH) was used to record responses. Because accurate mechanomyographic measurements require a stable preload and minimal voluntary muscle

activity, anesthetic depth was adjusted so that at least one additional TOF recording 10 min after reversal could be obtained in the operating room. This consisted of the administration of 10 to 30 mg propofol if spontaneous patient activity was detected. In some patients, this may have slightly delayed their arrival in the PACU. Additional intraoperative TOF measurements were recorded if conditions permitted. Patients were tracheally extubated and discharged from the operating room when they could respond to verbal commands and no fade was palpable on TOF stimulation.

In group 2 (pancuronium-desflurane, n = 29), the protocol was identical to that of group 1 except that 0.07 mg/kg pancuronium was administered for tracheal intubation. Additional increments (0.50 to 1 mg) were administered in a manner designed to keep the TOFC as close to 2 as possible. Reversal was accomplished with 0.05 mg/kg neostigmine plus 0.01 mg/kg glycopyrrolate.

In group 3 (pancuronium-propofol, n = 27), the protocol was identical to that of group 2, except that desflurane was omitted from the anesthetic sequence. Anesthesia was maintained with nitrous oxide plus a propofol-alfentanil infusion.

In all groups, each patient was assessed until a TOF ratio of 0.90 or greater was achieved. If this value was not attained in the operating room, subsequent measurements were made in the PACU until this level of recovery was reached. Only patients who had a TOF count in the range of 1 to 3 at the time of reversal were included in the study.

Data were analyzed using appropriate tests, with $P < 0.05$ considered statistically significant. Continuous objective variables (such as mean TOF ratios 10 min after reversal) were analyzed using single factor-factorial analysis of variance and the Scheffé F test for multiple comparisons or by a two-tailed two-sample Student's *t* test if applicable. Differences in frequency distribution between various groups (*e.g.*, the incidence of TOF ratios less than 0.90 at 30 min after reversal for mivacurium *versus* pancuronium) were subject to chi squared analysis.

## Results

There were no significant differences in the demographics of the three groups of patients, nor were there differences in the times from the initial bolus of relaxant to anticholinesterase administration (table 1).

**Table 1. Patient Demographics and the Duration of Surgery**

| | Group 1 (n = 35) | Group 2 (n = 29) | Group 3 (n = 27) |
|---|---|---|---|
| Age (yr)* | 39.9 ± 10.5 | 43.7 ± 11.1 | 41.0 ± 11.2 |
| Weight (kg)* | 69.3 ± 14.3 | 69.1 ± 11.1 | 71.4 ± 14.5 |
| Sex (M/F) | 10/25 | 10/19 | 6/21 |
| Duration (min) initial bolus to reversal* | 143 ± 59 | 147 ± 61 | 158 ± 67 |

* Mean ± SD. No statistical differences between the three groups were present.

*Group 1 (Mivacurium)*

The average time (± SD) from the initial bolus to cessation of infusion was 143 ± 59 min (range, 57 to 332 min), and reversal was attempted 4.6 ± 3.2 min later. Total cumulative mivacurium dosage averaged 0.69 ± 0.27 mg/kg or 5.28 ± 1.55 $\mu g \cdot kg^{-1} \cdot min^{-1}$.

The mean TOF count at reversal was 2 ± 0.4. Train-of-four ratios 5 and 10 min after reversal were 0.57 ± 0.12 (range, 0.34 to 0.84) and 0.76 ± 0.11 (range, 0.41 to 0.94), respectively. In 20 of 35 patients, a TOF ratio of 0.90 or more was attained before transfer to the PACU. In these persons, the average time to a TOF ratio of 0.90 or greater was 13.6 ± 3.8 (range, 8 to 22) min. In the remaining 15 patients, the average TOF ratio on arrival in the PACU was 0.94 ± 0.05 (range, 0.84 to 1.0), and this occurred 27.7 (range, 23 to 33) min after antagonism with edrophonium. Thirty-three of 35 patients had TOF ratios of 0.90 or greater within 30 min of reversal, and this value was reached in all of them by 45 min.

*Groups 2 and 3 (Pancuronium)*

Patients who received a nitrous oxide-desflurane-based anesthetic required a smaller cumulative dose of pancuronium than did patients anesthetized with nitrous oxide-propofol-opioid (0.088 *vs.* 0.118 mg/kg). This difference, however, was not statistically significant when calculated on a micrograms-per-kilogram-per-minute basis. The recovery times observed in groups 2 and 3 were indistinguishable (table 2), and thus recovery data from these groups were pooled to compare them with data from the mivacurium protocol.

The average time (± SD) from the initial bolus to reversal of residual block was 152 ± 63 (range, 54 to 383) min. The total cumulative pancuronium dosage averaged 0.10 ± 0.032 mg/kg. The mean time between reversal and the last incremental dose of pancuronium

**Table 2. Recovery Parameters: Pancuronium *Versus* Mivacurium**

| | Group 1 (n = 35) Mivacurium–Desflurane | Group 2 (n = 29) Pancuronium–Desflurane | Group 3 (n = 27) Pancuronium–Propofol | Groups 2 and 3 Combined Data (n = 56) | Probability Group 1 *versus* Groups 2 and 3 |
|---|---|---|---|---|---|
| TOF ratio @ 5 min* | 0.57 ± 0.12 | 0.51 ± 0.16 | 0.49 ± 0.12 | 0.50 ± 0.14 | <0.05 |
| TOF ratio @ 10 min* | 0.76 ± 0.11 | 0.66 ± 0.16 | 0.65 ± 0.10 | 0.65 ± 0.13 | <0.001 |
| Last TOF in OR | 0.85 ± 0.10 | 0.71 ± 0.14 | 0.72 ± 0.09 | 0.71 ± 0.12 | <0.0001 |
| Time (min) to first TOF measurement in the PACU*,† | 19.7 ± 7.9 | 31.9 ± 9.35 | 28.0 ± 5.8 | 30.0 ± 8.0 | <0.0001 |
| First TOF in PACU† | 0.93 ± 0.03 | 0.86 ± 0.08 | 0.84 ± 0.07 | 0.85 ± 0.08 | <0.0001 |

Values are mean ± SD.

TOF = train-of-four; OR = operating room; PACU = postanesthesia care unit.

* Post reversal.

† Or last value in OR if the TOF ratio was ≥0.90 before transfer to the PACU.

was 32 ± 31 min (range, 5 to 180 min). The mean TOF count at reversal was 2.1 ± 0.8. Train-of-four ratios 5 and 10 min after reversal were 0.51 ± 0.15 (range, 0.20 to 0.92) and 0.66 ± 0.14 (range, 0.30 to 0.92), respectively. Only 4 of 56 patients had TOF ratios of 0.90 or more before transfer to the PACU. In the remaining 52 patients, the average TOF ratio on arrival in the PACU was 0.85 ± 0.08 (range, 0.63 to 0.97), and this averaged 33.6 ± 8.2 min (range, 20 to 52 min) after neostigmine antagonism. Only 2 of 56 patients had an initial measured TOF ratio in the PACU of less than 0.70. In eight participants, a TOF of 0.90 or more was not attained by 90 min after recovery, and in five of these participants this value was less than 0.85.

Most patients recovering from pancuronium demonstrated recovery to a TOF ratio of 0.70 or more within 30 min of neostigmine administration. However, at this point only one third of the patients had a TOF ratio of 0.90 or more (table 3).

## Discussion

### *Mivacurium*

Our findings are consistent with other published reports that examined antagonism of mivacurium-induced neuromuscular block with edrophonium.[14-16] Kopman and associates[17] reported that when 0.5 mg/kg edrophonium is administered when the fourth response to TOF stimulation is first detected during recovery from mivacurium-induced block, the recovery time from the first palpable response at the thumb to a TOF ratio of 0.90 averaged about 15 min. In the present study, we have data for 26 persons at exactly 15 min after antagonism (the remaining nine patients were in the process of emerging from anesthesia and data could not be collected at that time for technical reasons). Although the present protocol differed in some respects from that of Kopman and associates,[17] the mean observed TOF value of 0.87 ± 0.09 was similar to their findings. In addition, the protocol that we used in the current study represents what is perhaps a worst-case scenario. Antagonism was attempted on average only 4.6 min after the termination of a mivacurium infusion sufficient to maintain the TOF count at one palpable response. In contrast, the last incremental dose of pancuronium in groups 2 and 3 had been given on average 32 min before neostigmine administration.

Of the 35 patients studied in group 1, only one had a TOF ratio less than 0.65 on discharge from the operating room (0.58, at 10 min after antagonism). However, when this parameter was remeasured in the PACU 18 min later, we recorded a value of 0.95. The lowest TOF value that we measured in any patient in group 1 on arrival in the PACU was 0.84.

**Table 3. Ratios 30 Min Postreversal: Observed Frequencies**

| | Pancuronium (n = 56) | Mivacurium (n = 35) |
|---|---|---|
| TOF ratio < 0.90 | 37 | 2 |
| TOF ratio ≥ 0.90 | 19 | 33 |

*P* value (pancuronium *vs.* mivacurium) < 0.0001.

### *Pancuronium*

When this investigation was first conceived, we expected that our results would be little different from those of Bevan and colleagues[10] or Viby-Mogensen and

coworkers.[4] We expected that a significant percentage of the patients who had received pancuronium would arrive in the PACU with measured TOF ratios of less than 0.70. We also predicted that further recovery to a TOF ratio of 0.90 or more after pancuronium would be slow. Certainly other investigators have presented compelling evidence that clinical signs of residual weakness are common after reversal of even modest doses of traditional long-acting relaxants.[18] Our results, therefore, were a distinct surprise. Although the last recorded TOF ratio in the operating room was less than 0.60 in 8 of 56 persons, on arrival in the PACU only two patients had measured values of less than 0.70, and in both cases the TOF ratio exceeded 0.60. In fact, 35% of patients had TOF ratios of 0.90 or more when this parameter was first measured in the PACU. These findings regarding the incidence of residual weakness after pancuronium are perhaps the most favorable ever reported. Because we do not question the accuracy of earlier workers' results, an attempt to reconcile our findings with those of other investigators is required.

Although we cannot completely explain these discrepancies, we believe that the low incidence of residual paresis encountered in this study was at least in part a function of careful and standardized intraoperative management. All the anesthetics in the study were administered or supervised by one of only four clinicians. We decided at the outset not to try to monitor the results of our department as a whole. It was our feeling that as the number of participants in a study expands beyond a small number, the potential for protocol violations increases greatly. Thus we standardized our anesthetic technique. Because incremental doses of pancuronium were never administered unless two (and usually three) evoked responses to indirect muscle stimulation were detected, the average cumulative dosage of pancuronium in groups 2 and 3 was low, averaging only 0.10 mg/kg in 2.5 h. In addition, we tried to time incremental doses so that two evoked responses would be present when reversal was initiated. Although this goal was not always achieved, a TOFC of at least 1 was always present before neostigmine was administered.

Because our results in group 2 were so unexpected, we considered another explanation. After antagonism of residual nondepolarizing neuromuscular block, recovery is primarily determined by two processes: direct antagonism by the anticholinesterase and a spontaneous decrease in the plasma concentration of the blocking agent, with the latter becoming the major determinant at profound levels of block.[19] Several years ago, Gencarelli and coworkers[20] proposed a third possibility. They posited that potentiation of neuromuscular block by volatile agents might provide an additional safety margin for patients because the anesthetic produces a component of relaxation that is readily reversible with time independent of the use of acetylcholinesterase inhibitors.

This last mechanism would seem to be most relevant to inhalation drugs of low solubility such as desflurane and sevoflurane. Because drug elimination is so rapid at the end of anesthesia with these new drugs, any potentiation should quickly dissipate. A recent study by Wright and associates[21] suggests that this may be so. These authors found that when the end-tidal anesthetic concentration of desflurane was abruptly reduced from 1.25 to 0.75 minimum alveolar concentration or from 0.75 to 0.25 minimum alveolar concentration, $T_1$ increased significantly during a steady-state vecuronium infusion. Because there is no reason to suggest that Wright and associates' results would be different if pancuronium had been the nondepolarizing blocker investigated, the use of desflurane as part of our anesthetic regimen in group 2 might be partially responsible for the low incidence of residual paresis that we recorded after administering pancuronium. This is why we included group 3 in this investigation.

Although this line of reasoning is initially attractive, no published studies have documented a lower incidence of residual neuromuscular block after the administration of potent volatile anesthetics. In fact, when antagonism is attempted during continued administration of inhaled anesthetics, recovery may actually be delayed.[22-24] As is evident from table 2, we could not demonstrate any differences between the two groups receiving pancuronium in regard to TOF fade ratios 5 and 10 min after reversal, or in mean TOF ratios when patients arrive in the PACU.

How, then, do we explain the almost total absence significant weakness on arrival in the PACU in our patients recovering from pancuronium anesthesia? First, we believe that a low incidence of residual paralysis can only be achieved if intraoperative monitoring of neuromuscular function is done with some care. A recent article by Shorten and Merk supports this.[25] These investigators, using a protocol similar to ours, tried to maintain intraoperative TOF counts of 1 to 2 (tactile) using incremental pancuronium during nitrous oxide–enflurane anesthesia. They reported that only 3 of 20 patients arrived in the PACU with TOF values less than 0.70, whereas in a control group (no peripheral nerve

stimulator was used), 9 of 19 patients arrived in the PACU with TOF values less than 0.70. Unfortunately, Shorten and Merk do not report the TOF counts at which reversal was attempted or the dose of anticholinesterase administered. Not all investigators have been able to demonstrate that neuromuscular monitoring makes a difference. Pedersen and associates[26] found that the use of a peripheral nerve stimulator had no effect on the dose of relaxant given during anesthesia, on the need for supplementary doses of anticholinesterase in the PACU, on the time from end of surgery to end of anesthesia, or on the incidence of residual postoperative paralysis evaluated clinically.

We cannot explain Pedersen and associates' results except to note that "small" differences in methodology may in fact prove to be important. Visual estimation of the TOFC may not correlate with the value obtained by tactile evaluation of the adductor pollicis muscle.[27,28] Similarly, the site used for neuromuscular monitoring is important. The degree of block at the orbicularis oculi may have little correlation with that found at the adductor policis,[29] and even the hypothenar muscles may have different sensitivities to blocking drugs compared with the first dorsal interosseous muscle.[30] Thus our results may only apply when neuromuscular monitoring consists of tactile evaluation of the TOFC at the thumb. We also believe that our results should not be extrapolated unduly. All our patients were classified as ASA physical status 1 or 2 between the ages of 18 and 69. In addition, several other details of drug administration are worth noting. Reversal was never attempted unless at least one evoked response to ulnar nerve stimulation was present, the average interval between the last dose of pancuronium and neostigmine administration averaged approximately 30 min, the initial "intubating dose" approximated a single $ED_{95}$ bolus, and the total cumulative dose of pancuronium given rarely exceeded two times the $ED_{95}$.[31] A final caveat: 10 min after reversal of pancuronium, considerable paresis may still be present. At this point, 25% of patients had TOF ratios less than 0.60, and 5% had TOF ratios less than 0.40. Thus our findings may in part reflect a longer reversal to PACU arrival time than that experienced by other investigators. The clinical message is that when possible, antagonism should be initiated as soon as the need for muscle relaxation is over, not during application of the surgical dressing.

Nevertheless, administering pancuronium under what might be described as a best-case scenario, we could not confirm the high incidence of pancuronium-induced residual weakness in the PACU described by most other investigators.[4-6]

## Conclusions

A recent discussion of pharmacoeconomics in the practice of anesthesiology asserted that ". . . we [often] opt for more expensive ways of accomplishing given ends without achieving commensurate benefits for the patient. An example is the current practice of using the newer, more expensive intermediate-acting neuromuscular blocking drugs (*e.g.*, atracurium and vecuronium) for procedures having a duration greater than two hours, when arguably most patients would have an equally satisfactory experience with an older, off-patent, less expensive long-acting relaxant (*e.g.*, pancuronium)."[32] The results of our investigation may be viewed by some as providing substance to this position. Although return to a TOF ratio of 0.90 or more is more rapid after mivacurium-induced neuromuscular block, some might argue that recovery times after antagonism of pancuronium-induced block are fast enough for all practical purposes. Our position is more cautious, and we believe that certain caveats must be remembered.

First, as we noted, our results with pancuronium probably represent a best-case situation. Our findings should not be extrapolated to the extremes of age, or to patients with diminished renal or hepatic function. Second, the findings of previous investigators[4-10] cannot be dismissed out of hand. Our results represent the outcome that can be achieved using a rigid protocol of drug administration and neuromuscular monitoring. They may not be representative of outcome studies involving larger numbers of clinicians, some whom may feel free to be more "creative" in their anesthetic management. In addition, there is a common misapprehension that a TOF ratio equal to 0.70 is synonymous with full clinical recovery. This is clearly not so. Although a TOF ratio of this magnitude probably assures return of respiratory mechanics (vital capacity, peak expiratory flow rate, and so on) to near control values,[3] bothersome symptoms of residual weakness are likely to be present at this level of recovery. Work in progress from our department with unpremedicated awake volunteers indicates, for example, that significant visual disturbances persist until the TOF ratio exceeds 0.90.

Our results suggest that if nondepolarizing blockers are administered using tactile evaluation of the TOFC as a guide, critical episodes of residual weakness in the

PACU should occur infrequently even with long-acting relaxants. Nevertheless, if full recovery is defined as return to a TOF ratio of 0.90 or more, then short-acting agents offer a wider margin of safety.

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