bustible material, also is present in the form of alcohol-based prep solutions and alcohol-based adhesive sprays,[5] alcohol-packaged suture,[1] drapery, gauze pads, sponges, adhesive tape (especially the plastic type), eye patches, disposable plastics, and paper products.[6]

Both experimental and clinical evidence support the potential hazard these combustible items pose. Plumlee[2] tested the combustibility of tincture of Benzoin spray and found that after a 5-s burst, the area sprayed could be ignited by cautery up to four minutes later; even after a 1-s burst, ignition was possible up to 90 s later. Hot wire cautery has been shown to generate enough heat to ignite any alcohol-based antiseptic which contains as little as 20 per cent alcohol. Therefore, any substance containing greater than 10 per cent alcohol must be considered potentially flammable.[7] Also, cotton drapes ignite within 3-s duration of contact with cautery if the oxygen concentration under the draping is 40 per cent or greater.[8]

We feel that the twin-fire can be explained by the combination of a highly enriched oxygen environment, combustible drapery, and electrocautery. Twin A was quite hypoxemic (Pa_{O_2} 28 torr); therefore, ventilation was with an $F_{I_{O_2}}$ of 100 per cent. The plastic occlusive drape completely enveloped the baby's head, encircling and being attached to the endotracheal tube. An audible leak around the endotracheal tube resulted in the creation of an enriched oxygen atmosphere under this drapery. When the dissection became immediately subjacent to the oxygen rich atmosphere, a flash fire in the operative field resulted. Analogous experiences were cited by Gupte,[3] who reported ignition of a pharyngeal gauze pack during intraoral diathermy which he attributed to leakage of oxygen and nitrous oxide around the endotracheal tube, and by Cameron and Ingram,[8] who reported ignition of surgical drapes when oxygen enriched gases were vented beneath them.

This case report serves to emphasize that the potential for fire still exists, despite the use of nonflammable anesthetics. The hazard may be decreased by: 1) using water-based prep solutions,[7] or if alcohol-based preps must be used, delaying draping until vapor dissipation has occurred[9]; and 2) preventing the accumulation of oxygen enriched anesthetic gases beneath the drapery.

References

1. Buyers RA: Fire in the operating room caused by fluid from suture packet. JAMA 237:531, 1977
2. Plumlee JE: Operating room flash fires from use of cautery after aerosol spray: A case report. Anesth Analg (Cleve) 52:202–203, 1973
3. Gupte SR: Gauze fire in the oral cavity: A case report. Anesth Analg (Cleve) 51:645–646, 1972
4. Bloch EC, Karis JR: Cardiopagus in neonatal thoracopagus twins: Anesthetic management. Anesth Analg (Cleve) 59:304–307, 1980
5. Jones M: Identifying electrical hazards and implementing safety measures–the theatre nurses role. Australas Nurses J 7:22–24, 1977
6. Perry LB, Gould AB Jr, Leonard PF: Discussion of case history number 82: nonflammable fires in the operating room. Anesth Analg (Cleve) 54:152–154, 1975
7. Briscoe CE, Hill DW, Paine JP: Flammable antiseptics and theatre fires. Br J Surg 63:981–983, 1976
8. Cameron BG, Ingram GS: Flammability of drape material in nitrous oxide and oxygen. Anaesthesia 26:281–288, 1971
9. Ogawa H: Accidental ignition of disinfectant in experimental laboratory. Cleve Clin Quart 37:219–222, 1970

Anesthesiology
55:458–461, 1981

Posttetanic Count (PTC): A New Method of Evaluating an Intense Nondepolarizing Neuromuscular Blockade

JØRGEN VIBY-MOGENSEN, M.D.,* PAUL HOWARDY-HANSEN, M.D.,† BENT CHRÆMMER-JØRGENSEN, M.D.,† HELLE ØRDING, M.D.,‡ JENS ENGBÆK, M.D.,† AAGE NIELSEN, M.Sc.§

The methods commonly used for monitoring neuromuscular transmission do not allow evaluation of an intense neuromuscular blockade. Thus the doses of nondepolarizing relaxant used for endotracheal intubation cause disappearence of the response to single, tetanic, and train-of-four (TOF) nerve stimulation for a variable

* Associate Chairman.
† Assistants in Anesthesia.
‡ Clinical and Research Fellow.
§ Statistical Research Unit, Danish Medical and Social Science Research Councils and Aalborg University Center.

Received from the Department of Anesthesia, Herlev Hospital, University of Copenhagen, DK-2730 Herlev. Accepted for publication March 13, 1981.

Address reprint requests to Dr. Viby-Mogensen: Department of Anesthesia, University of Copenhagen, Herlev Hospital, DK 2730 Herlev, Denmark.

Key words: Neuromuscular relaxants: pancuronium. Neuromuscular transmission: stimulator, nerve. Monitoring: Stimulator, nerve; train-of-four; posttetanic facilitation; posttetanic count.

period of time. During this time, the magnitude of neuromuscular blockade cannot be evaluated by the traditional stimulation forms.

Heisterkamp *et al.*[1] found that the enhancement of posttetanic twitch tension (posttetanic facilitation) in partially curarized patients remained constant regardless of the dose of non-depolarizing muscle relaxant or magnitude of neuromuscular blockade. If this also holds true for an intense neuromuscular blockade, the response to posttetanic twitch stimulation after the injection of a non-depolarizing muscle relaxant must appear earlier than the response to pretetanic twitch or TOF nerve stimulation.

The present study was designed to evaluate the neuromuscular blockade during the period of no response to single or TOF stimulation by quantifying the degree of posttetanic facilitation.

Materials and Methods

Thirty healthy adult female patients (ASA class I) undergoing gynecologic operations were studied. Informed consent was not obtained as the anesthetic procedure was that normally used and monitoring of neuromuscular function during anesthesia is routine at our hospital. Our study plan was approved by the Ethical Committee at the hospital. No patient had neuromuscular disease or received any drug that might alter neuromuscular function. They ranged in age from 27–56 years (mean 43 years).

One hour after giving 0.2 mg/kg diazepam orally, anesthesia was induced with 3–5 mg/kg thiopental and maintained with nitrous oxide 50 per cent and halothane 0.75–1.50 per cent inspired concentration. Ventilation was controlled.

Following induction of anesthesia, the ulnar nerve was stimulated at the wrist through percutaneous electrodes connected to a nerve stimulator.[2] The adduction force of the resultant thumb twitch was measured by a displacement transducer, and recorded on a polygraph. For TOF stimulation, a series of four supramaximal single stimuli (rectangular pulses of 0.2-ms duration) was applied to the nerve at 2 Hz for 2 s every twelfth second.[3] When the response to TOF nerve stimulation was stable (usually after 8–12 min), the height of the first twitch of the train was taken as the standard control (control twitch height). Thirteen patients (Group I) were then given 0.08 mg/kg pancuronium and 17 patients (Group II) 0.1 mg/kg pancuronium, intravenously. The trachea was intubated when the response to TOF nerve stimulation had disappeared. Four min later the mode of stimulation was changed to 1 Hz single twitch stimulation (fig. 1). After 1 min, a tetanic stimulus (50 Hz) was applied for 5 s. Three seconds later, the single twitch was again applied for 1 min followed by 4 min of TOF nerve stimulation. This pattern of stimulation (TOF, single twitch, tetanus, single twitch, TOF) was continued until the first twitch of the TOF reached 10–20 per cent of control twitch height.

Regression analysis was used in the statistical analysis of the data from Group II. Prediction regions were obtained by considerations similar to those used in random coefficient regression models.

Results

The response to posttetanic twitch stimulation in 13 patients given 0.08 mg/kg pancuronium intravenously (Group I) appeared in an average of 37 min before the first reaction to TOF stimulation. The posttetanic twitch response was 40 per cent of control twitch height at the time, when the first response to TOF reappeared (fig. 2). Because it appeared possible during intense neuromuscular blockade to predict time to initial response to

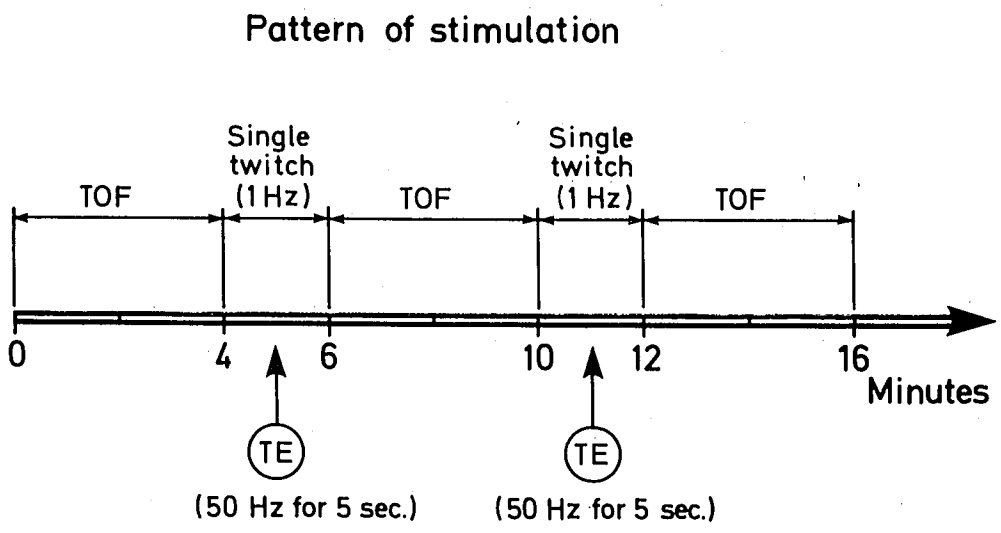

FIG. 1. Pattern of stimulation. TOF: train-of-four nerve stimulation. TE: Tetanic stimulation (50 Hz for 5 s). See text for further explanation.

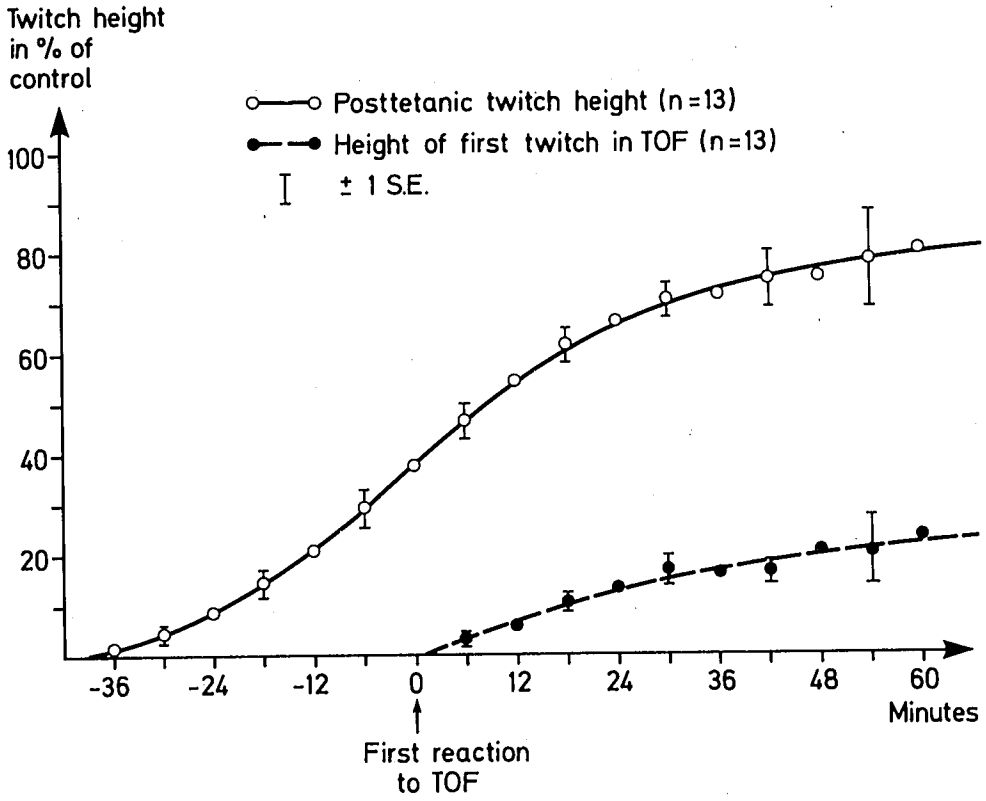

FIG. 2. Relationship between the first posttetanic twitch response (upper curve) and the response to train - of - four nerve stimulation (TOF, lower curve) in 13 patients given 0.08 mg/kg pancuronium intravenously (Group I). For clarity the curves have been drawn so that time zero is the time for first response to TOF nerve stimulation. Note that the response to posttetanic twitch stimulation appeared an average of 37 min before the first reaction to TOF nerve stimulation.

TOF nerve stimulation from the reaction to posttetanic twitch stimulation, an additional 17 patients (Group II) were studied. In Group I the injection of 0.08 mg/kg pancuronium intravenously did not cause the response to posttetanic twitch stimulation to disappear in all patients. The dose of pancuronium was therefore increased to 0.1 mg/kg in patients in Group II.

The statistical analysis showed for each of the 17 patients in Group II a close linear relation between time to first detectable reaction to TOF nerve stimulation and the square root of the posttetanic twitch height in per cent of control. Evaluation of posttetanic twitch response in relation to a control twitch response presupposes access to monitoring equipment. However, the number of posttetanic responses can be counted (the posttetanic count: PTC). We therefore related time to first response to TOF nerve stimulation to PTC. For each patient there was a close linear relation between time (t) to first reaction to TOF nerve stimulation and the square root of PTC:

$$\sqrt{PTC} = \alpha t + \beta \qquad \text{(equation 1)}$$

However, the lines varied between patients, and it was decided to neglect the relatively small variation about the individual lines.

Solving equation 1 with respect to t yields

$$t = \frac{\sqrt{PTC} - \beta}{\alpha} \qquad \text{(equation 2)}$$

By approximating the right hand side of equation 2 by a constant plus a linear expression in α and β, and using the estimates of the parameters of the distribution of (α, β) estimates of the mean and standard deviation of t were calculated for different values of $\sqrt{PTC}$.

Figure 3 summarizes the results, showing mean and 95 per cent prediction region of the time to first reaction to TOF nerve stimulation as a function of the number of posttetanic responses.

DISCUSSION

The degree of posttetanic facilitation is dependent on how often the tetanic stimulus is applied, frequency of the tetanic stimuli, duration of tetanic stimuli, and the time lapse between the conclusion of the tetanic stimulus and the first posttetanic single stimulus. These variables should therefore be kept constant for reliable monitoring of neuromuscular function.[2] A tetanic stimulus of 50 Hz was chosen for our study because Stanec *et al.*[4] have shown that at this frequency tetanus is well-maintained and there is little or no posttetanic facilitation in the absence of blocking drugs.

Our results show that the posttetanic twitch is present when the response to a tetanic stimulus (50 Hz) has disappeared. The reason for this is presumably that the mobilization of acetylcholine from the reserve to the readily available stores caused by the tetanic stimulation outlasts the period of tetanic stimulation.[5]

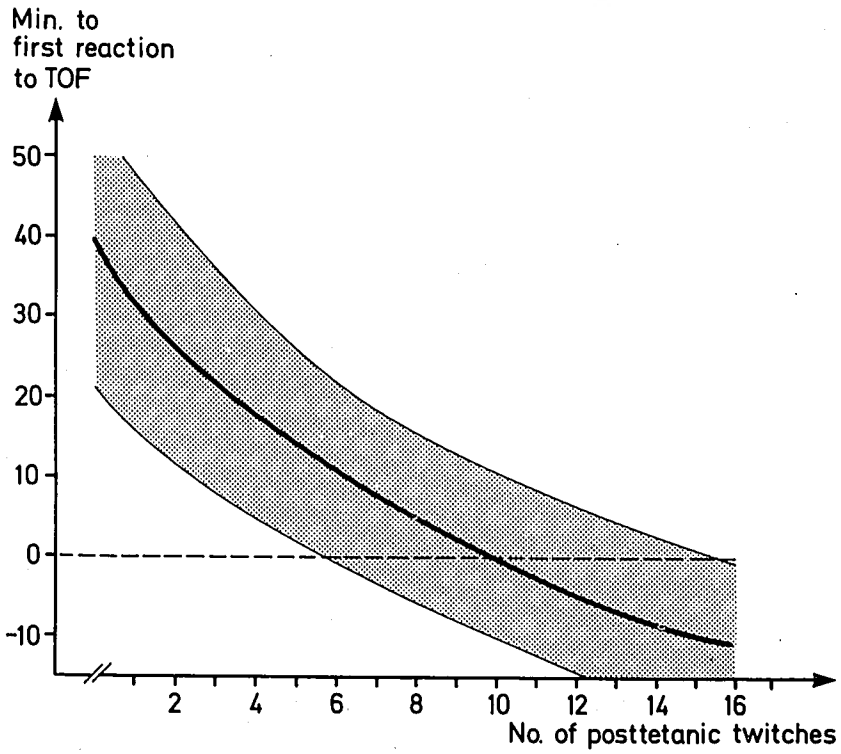

FIG. 3. Relationship between minute to first reaction to train-of-four (TOF) nerve stimulation and number of postetanic responses (PTC) in 17 patients (Group II). The mean curve and 95 per cent confidence limits are shown.

Tetanic stimulation may in itself influence recovery from a nondepolarizing neuromuscular blockade.[6] Therefore, this stimulus should not be applied too often. In this study we applied the tetanic stimulus every 6 min. The possibility exists that this relatively frequent application may influence recovery of the neuromuscular transmission in the investigated arm. This problem needs further investigation. However, studies in progress comparing the recovery time in both arms, with the one stimulated by TOF nerve stimulation every twelfth second and the other by intermittent tetanic stimulations (50 Hz for 5 s) every 6 to 10 min, indicate that there is no clinically significant difference in recovery time between the two arms.

In summary, the results of this study indicate: 1) An intense nondepolarizing neuromuscular blockade can be evaluated by the response to posttetanic single twitch stimulation; 2) Magnitude of posttetanic facilitation was negatively correlated with time to first response to TOF nerve stimulation; and 3) Time to return of response to TOF nerve stimulation may be derived from the number of posttetanic twitch responses present (PTC).

REFERENCES

1. Heisterkamp DV, Skovsted P, Cohen PJ: The effect of small incremental doses of *d*-tubocurarine on neuromuscular transmission in anesthetized man. ANESTHESIOLOGY 30:500–505, 1969
2. Viby-Mogensen J, Hansen PH, Jørgensen BC et al: A new nerve stimulator (Myotest). Br J Anaesth 52:547–550, 1980
3. Ali HH, Utting JE, Gray C: Stimulus frequency in the determination of neuromuscular block in humans. Br J Anaesth 42:967–977, 1970
4. Stanec A, Heyduk J, Stanec G, et al: Tetanic fade and post-tetanic tension in the absence of neuromuscular blocking agents in anesthetized man. Anesth Analg (Cleve) 57:102–107, 1978
5. Waud BE, Waud DR: Physiology and pharmacology of neuromuscular blocking agents, Muscle Relaxants. Edited by Katz RL. Amsterdam, London, New York, Exerpta Medica, 1975, pp 1–58
6. Feldman SA, Tyrell MF: A new theory of the termination of action of the muscle relaxants. Proc R Soc Med 63:692–695, 1970