The influence of changes in hand temperature on the indirectly evoked electromyogram of the first dorsal interosseous muscle

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The evoked EMG response commonly decreases in amplitude during the first few minutes of anaesthesia. The purpose of this study was to determine if a relationship exists between changes in hand temperature, which are known to occur with induction of anaesthesia, and drift in the EMG signal. The indirectly evoked response of the 1st dorsal interosseous muscle was measured using a Datex™ Relaxograph® in 15 patients undergoing elective surgery. The test arm was wrapped in towels in order to minimize heat loss. Core body temperature, hand temperature, and T_1 were recorded at two minute intervals for the next 30 min. Patients then received a bolus of mivacurium $0.08 \, mg \cdot kg^{-1}$ and additional doses were given as needed. Complete recovery was defined as a TOF ratio >0.90. Regression analysis plotting Δtemperature against ΔT_1 was performed for each individual. The slope of the regression line for the relationship between $\Delta° C$ and ΔT_1 was then used to calculate a correction factor (CF) which might be used to "fine tune" the last measured T_1. The initial hand temperature averaged $30.8 \pm 1.4° C$ and this increased by $4.1 \pm 1.2° C$ over the next 30 min. During this period T_1 decreased by $24.8 \pm 5.9\%$ or $-6.05\%/° C$. The final mean T_1 value at the end of anaesthesia (uncorrected) was $70.6 \pm 7\%$ of control. The average corrected T_1 value was $94.7 \pm 8.5\%$ (range, 83–111%). It is concluded

that there was a correlation between $\Delta° C$ and ΔT_1 during the first 30 min of anaesthesia ($r^2 = 0.77$, $P < 0.0001$). However, in 5 of 15 individuals it was not possible to "temperature correct" the final T_1 value to within $\pm 10\%$ of control. Hence, while changes in muscle temperature probably play a major role in the T_1 drift seen with the Datex monitor, other factors remain to be identified.

La réponse évoquée à l'EMG diminue ordinairement pendant les premières minutes de l'anesthésie. Le but de cette étude était de déterminer la relation possible entre les changements de température de la main qui surviennent à l'induction, et la dérive du signal de l'EMG. La réponse indirecte évoquée au 1er muscle interosseux dorsal a été mesurée avec un Relaxograph™ Datex® chez 15 patients en chirurgie réglée. Le bras servant à l'expérience a été enveloppé dans des serviettes pour en atténuer la perte de chaleur. La température centrale, la température de la main et T_1 ont été enregistrés aux deux minutes pendant 30 min. Les patients ont alors reçu un bolus de mivacurium $0,08 \, mg \cdot kg^{-1}$ et des doses additionnelles administrées au besoin. La récupération complète a été définie comme un rapport de TOF $>90\%$. L'analyse de régression établissant la relation entre Δ température et ΔT_1 a été effectuée pour chacun des sujets. La pente de régression pour la relation entre $\Delta° C$ et ΔT_1 a été utilisée pour calculer un facteur de correction (FC) utilisable pour une mise au point finale du dernier T_1 mesuré. La température initiale de la main était en moyenne de $30,8 \pm 1,4° C$. Elle augmentait de $4,1 \pm 1,2° C$ pendant les 30 min suivantes. Pendant cette période, T_1 diminuait de $28,4 \pm 5,9\%$ ou $-6,05\%/° C$. La valeur finale moyenne de T_1 à la fin de l'anesthésie (sans correction) était de $70,6 \pm 7\%$ du contrôle. La valeur moyenne corrigée de T_1 était de $94,7 \pm 8,5\%$ (écart, 83–111%). En conclusion, il existe une corrélation entre $\Delta° C$ et ΔT_1 au cours des 30 premières min d'anesthésie ($r^2 = 0,77$, $P < 0,0001$). Toutefois, chez cinq des 15 sujets, il a été impossible d'appliquer la correc-

tion de température à la valeur finale de T_I à ±10% du contrôle. En conclusion, bien que les changements de température musculaire puissent jouer un rôle majeur dans la dérive de T_I observée avec le moniteur Datex, les autres facteurs en cause sont encore inconnus.

The use of integrated evoked electromyography (EEMG) as a clinical monitor of neuromuscular block was first described by Lam *et al.* in 1981,[1] and the first commercial adaptation of this device was described three years later by Pugh *et al.*[2] It is now generally accepted that train-of-four (TOF) ratios measured with this technique and those recorded with more traditional mechanical recordings give similar information.[3,4] There is less agreement on the validity of recordings of single twitch height because of drift of the EEMG amplitude with time. Edmonds *et al.*[5] noted many years ago that the EEMG wave-form decreases both in latency and in amplitude during the first few minutes of anaesthesia. At the end of an anaesthetic, it is not uncommon to find that single twitch (T_1) may only recover to 70–80% of control despite TOF ratios exceeding 0.90. This phenomenon has been particularly worrisome since its aetiology has not been elucidated, and the magnitude of the observed drift in T_1 does not appear to be predictable. Various suggestions regarding the responsible mechanism ranging from an alteration is muscle preload,[6] to changes in electrode impedance with time, or to some unspecified central effect of general anaesthesia on neuromuscular transmission have been hypothesized, but the cause of this phenomenon has remained uncertain.

Smith *et al.*[7] reported that induction of anaesthesia is associated with an increase in hand skin temperature as well as the temperature of first dorsal interosseous (DI) muscle. They found this increase in hand temperature was inversely related to the magnitude of the evoked EEMG of the first DI. However, they were not able to demonstrate a correlation between *changes* in hand temperature and the extent of T_1 drift with time.

We decided to reexamine the observations and conclusions of Smith *et al.* for several reasons. First, the temperature changes they describe averaged about 2°C and were accompanied by decreases in T_1 of only about 15% from control. It has been our experience that, if care is taken in wrapping the arm in towels after induction of anaesthesia (passive warming), hand temperature may increase by more than 5°C, and that under these circumstances much greater decreases in T_1 may be expected. Second, preliminary observations in our department revealed a correlation between alterations in hand skin temperature (Δ°C) and changes in twitch height (ΔT_1). If a predictable relationship exists between Δ°C

and ΔT_1, it may be possible to apply a "correlation factor" to EEMG T_1 values if the final hand temperature as well as the temperature at the time of initial instrument calibration are both known. This study was primarily an attempt to investigate the latter premise.

Methods

Fifteen ASA PS 1–2, adult patients undergoing elective surgical procedures were included in the study. All patients were free from neuromuscular disease, were within 18 to 65 yr of age, and 15% of ideal body weight. The protocol was approved by our hospital's Human Subject Review Committee. Anaesthesia was induced with alfentanil 40 μg · kg^{-1} plus propofol 2.0–2.5 mg · kg^{-1} *iv*, and tracheal intubation was accomplished without the use of muscle relaxants. Anaesthesia was maintained with nitrous oxide (65–70% inspired), propofol 75–100 μg · kg^{-1} · min^{-1}, plus fentanyl supplementation *iv* as needed. Ventilation was controlled, and PETCO$_2$ maintained between 32–38 mmHg. The blood pressure cuff was placed on the contralateral arm, *iv* lines were in the ipsilateral antecubital fossa.

The indirectly evoked integrated compound action potential of the first dorsal interosseous muscle to supramaximal stimulation of the ulnar nerve at the wrist was measured and recorded using a Datex Relaxograph® monitor. Supramaximal nerve stimulation was achieved using the nerve stimulator incorporated into the Datex unit (pulse width 100 msec, constant current, 0–70 mA range). The test hand was immobilized, and approximately 200 to 300 g of resting tension were applied to the thumb with a Velcro strap. Train-of-four stimulation was given every 20 sec during the period of observation, and single twitch depression, indicated by the height of the first TOF twitch/control twitch height (T_1/T_c), and TOF fade were continuously recorded. Hand temperature was measured using an Electromedics, Inc.® surface probe (cat. no. 2404, YSI 400 compatible) placed on the palm, just distal to the thenar eminence. This thermistor has an accuracy of ±0.2°C. All electrodes were applied at least ten minutes before any data collection. Immediately after induction of anaesthesia, control twitch height (T_c) and TOF fade ratio (T_4/T_1) were established, and hand temperature was recorded. The test arm was then wrapped in several layers of towels and sheets (from finger tips to the axilla) in order to minimize heat loss. Active warming of the hand was not employed. An oesophageal temperature probe was also placed in order to measure core body temperature. Hand temperature was recorded at two-minute intervals until temperature and T_1 were stable for at least three minutes (±0.1°C, and ±1% T_1 respectively) or until 30 min had elapsed from the initial calibration of the Datex® unit. No neuromus-

cular blocking agents were administered during this 30-min test period. Once the stabilization period was complete, patients received an initial bolus of mivacurium 0.08 mg · kg^{-1} and additional doses were given as needed clinically. At the end of the surgical procedure, if the TOF ratio had not returned spontaneously to a value of ≥0.90, antagonism of residual block was achieved with increments of edrophonium 0.25 mg · kg^{-1} until that endpoint was attained.

A scatter diagram was constructed plotting change in hand temperature (independent variable) against any alteration in EEMG (dependent variable). Regression analysis using the method of least squares was performed, forcing the line of regression through the origin. The slopes of the lines of regression (±95% confidence limits) and the coefficient of determination (r^2) were then determined for each individual. Average ±SD values for these variables were calculated. Similar calculations were also performed using pooled data from all individuals.

The slope of the regression line (pooled data) for the relationship between Δ°C and ΔT$_1$ was then used to calculate a correction factor (CF) which might be used to "fine tune" the last measured T$_1$ value. For example: If the relationship between temperature and twitch height is such that each degree centigrade increase in temperature results in a 6% decrease in twitch height (slope = −6.0), then a CF of 1.0638 must be applied (94% × 1.0638 = 100%). In like fashion, if the hand temperature increased by 3.5°C from the time of initial instrument calibration to end of the anesthetic, a CF of 1.2233 would be employed (1 + [3.5 × 0.0638] = 1.2233). Hence, if the final T$_1$ value at "full" recovery was 81% of control, the "corrected T$_1$" would be 99.1% (81 × 1.2233 = 99.09). Corrected T$_1$ values were calculated for all patients. The average values and 95% confidence limits of these values as well as the suggested CF were then calculated. Full T$_1$ recovery was defined as return of the TOF ratio to a value >0.90.

Results

Hand temperature and twitch height (T$_1$) vs elapsed time
Initial palmar skin temperature at the time of instrument calibration (within the first two minutes after induction of anaesthesia) was 30.8 ± 1.4 (SD)°C. Over the next 25 to 30 min hand temperature increased by an average of 4.1 ± 1.2°C, and stabilized at a value 1.2 ± 0.4°C below core temperature. Approximately 70% of this temperature change occurred within the first ten minutes after instrument calibration (Figure 1 and the Table).

At the time when temperature was deemed to have stabilized, the decrement in T$_1$ amplitude averaged 24.8
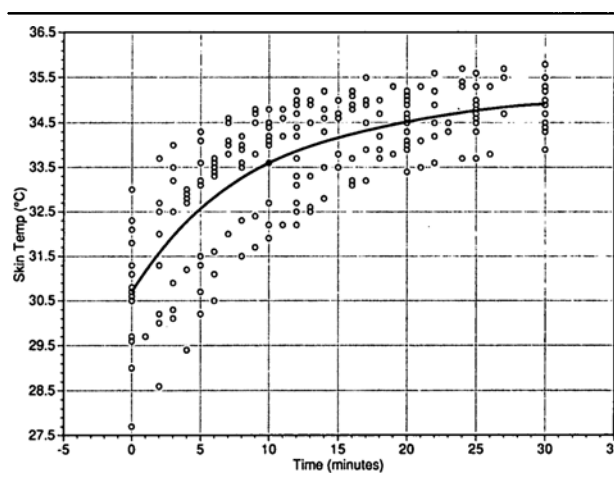

FIGURE 1   Change in palmar skin temperature as a function of time from induction of anaesthesia. Curve fit is the result of a fourth-degree polynomial "best fit" linear least squares analysis. By 25–30 min, hand temperature had stabilized. Data points may overlap.

± 5.9 percent from control. At ten minutes post-calibration, T$_1$ had decreased by 15.7 ± 3.9% or approximately 63% of the total decrease noted in the first 25–30 min (Figure 2).

Hand temperature vs twitch height
Regression analysis of 208 pooled individual observations from 15 subjects yields a slope for the relationship of twitch height to hand temperature of −5.61% T$_1$/°C (r^2 = 0.77, $P < 0.0001$) (Figure 3). The slope as determined by averaging the 15 individually determined temperature-twitch relationships was −6.10% T$_1$/°C (95% confidence limits, −5.68 to −6.51).

At the end of anaesthesia (mean time from initial calibration = 94 ± 6 min), a CF of 6.5%/°C was used to correct the final T$_1$ values. The final average T$_1$ value at the end of anaesthesia (uncorrected) was 70.6 ± 7% of control. The average corrected T$_1$ value was 94.7 ± 8.5% (range, 83–111%). While there was a correlation between Δ°C and ΔT$_1$ during the first 30 min of anaesthesia, in 5 of 15 individuals it was not possible to "temperature correct" the final T$_1$ value at full recovery to within ±10% of control.

Discussion

Changes in hand temperature with induction of anaesthesia
It is now well established that induction and maintenance of general anaesthesia are usually associated with marked peripheral vasodilatation, and a transfer of heat from the body core to peripheral tissues. Matsukawa *et al.* demonstrated, under propofol-fentanyl anaesthesia, that fore-

TABLE

Patient #	Initial temp °C	Final temp °C	Δ°C at 30 min	ΔT_1(%) at 30 min	Final T_1 as % of initial T_1*	T_1 after CF† Applied (%)
1	29.6	34.3	4.7	−27.5	69	103
2	29.7	33.8	4.1	−26.0	69	102
3	31.3	35.3	4.0	−31.6	58	84
4	32.1	35.0	2.9	−15.0	81	99
5	31.8	35.5	3.7	−28.0	65	83
6	30.6	34.9	4.3	−30.6	65	92
7	31.1	35.8	4.7	−26.3	72	91
8	32.3	35.0	2.7	−13.3	84	96
9	30.5	34.5	4.0	−22.5	72	97
10	30.8	35.2	4.4	−25.0	65	93
11	29.0	34.3	5.3	−23.5	71	105
12	30.7	34.7	4.0	−21.7	76	97
13	32.1	35.2	3.1	−26.5	65	85
14	27.7	34.7	7.0	−35.4	66	111
15	33.0	35.0	2.0	−19.4	80	84
Average	30.8	34.9	4.1	−24.8	70.6	94.7
SD	1.40	0.51	1.19	5.92	7.1	8.5
Range	27.7–33.0	33.8–35.8	2.0–7.0	−13–(−)35	58–84	83–111

*In all cases, TOF ratio $\geq$0.90.
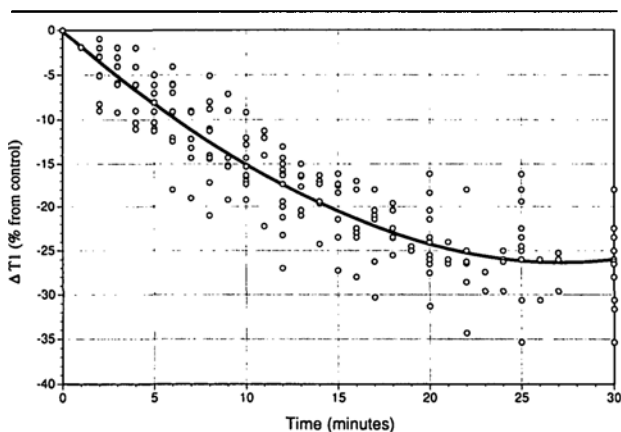†Correction factor (CF) = +6.5% per Δ°C decrease.

FIGURE 2   Change in indirectly evoked single twitch amplitude (T_1) as a function of time from induction of anesthesia. Curve fit is the result of a fourth-degree polynomial "best fit" multiple linear least squares analysis. By 25–30 min, T_1 had stabilized. Data points may overlap.
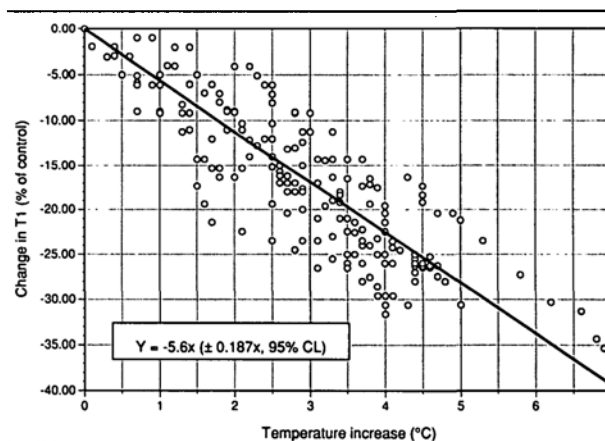

FIGURE 3   Change in indirectly evoked single twitch amplitude (T_1) as a function of time from induction of palmar skin temperature. Linear least squares analysis. The coefficient of determination (r^2) was 0.77 ($P <$ 0.0001). Data points may overlap.

arm blood flow increases on average by more than four-fold with induction of anaesthesia.[8] Smith *et al.*[7] demonstrated that following induction with propofol and maintenance with nitrous oxide and enflurane that the temperature of the first dorsal interosseous (first DI) muscle will increase by 2°C during the first 30 min of anaesthesia. Despite stable nasopharyngeal temperatures (mean 36.0, range 35.9–36.2°C), the temperature of the first DI rose from 33.5°C (33.1–33.9) to 35.5°C (35.3–35.8) during that interval. We have now docu-

mented that, if some attempt is made to insulate the arm from the ambient environment, increases in hand temperature of $\geq$4.5°C are not uncommon.

A potential pitfall in our study was that muscle-skin temperature gradients probably were not constant during the initial 30 min of observation. The gradient between skin and muscle temperature is difficult to predict. Heir *et al.*[9] found that skin temperature averaged 2.2°C below muscle temperature, which in turn was 0.8°C below core temperature. However, it appears that in that investigation

the hand under study was exposed to ambient operating room temperatures. Our initial measurement of skin temperature was made with the hand exposed to room air. Subsequent temperature determinations were made after wrapping the arm and hand in several layers of cotton blankets. Hence, while average skin temperature increased by 4.1°C during the initial 30 min of observation, it is possible that changes in muscle temperature were less dramatic. While we recognized this difficulty in advance, our goal was to validate a noninvasive approach to EMG monitoring, and we elected not to place im temperature probes.

Changes in T_1 with time

When determining "baseline" or control values for evoked muscle twitch amplitude it is necessary to establish that these values are stable over time. Failure to establish baseline stability presents the researcher with potential difficulties. For example, if an investigator wishes to administer an anticholinesterase antagonist at 25% recovery of T_1, the extent to which the control value has "drifted" over time is unknown. Hence, it is not possible to determine the correct time to induce reversal. If, following complete reversal (no fade on TOF stimulation), T_1 returns to only 80% of control, it is apparent that drugs given at "25% recovery" were in fact administered at 31% of control.

The need for a period of baseline stabilization is not unique to electromyography. When recording mechanical responses to indirect stimulation, a different problem is encountered. It has been known for many years that the force of twitch tension will increase with repeated stimulation. This is known as the staircase phenomenon or treppe.[9] Thus, when performing mechanomyographic (MMG) studies, twitch height tends to increase with time. The extent of twitch augmentation is a function of stimulation frequency and the total number of stimuli applied. Generally, however, a stable mechanomyographic "preparation" can be assured if the nerve-muscle unit is first conditioned by 1 Hz stimulation for two to three minutes (*Savarese JJ*, personal communication). The need to spend more than five minutes establishing a stable baseline is usually unnecessary when doing MMG studies.

The magnitude of the indirectly evoked mechanical twitch response is not immune to changes in muscle temperature. Heier *et al.* observed that T_1 did not decrease until muscle temperature decreased below 35.2°C. Below this temperature twitch tension decreased in a linear manner.[10-12] Similarly, Erikkson *et al.* reported that prolonged TOF nerve stimulation did not change the mechanical twitch response in patients with a normal central and peripheral temperature. A peripheral skin temperature <32.0°C with sustained and normal body temperature is, however, associated with changes in both twitch tension and TOF ratio that may be a source of error when evaluating neuromuscular function.[13]

There is mounting evidence that the EMG is also greatly influenced by muscle temperature. While decreases in muscle temperature are associated with a reduction of isometric force, this is accompanied by an increase in the peak-to-peak amplitude of the compound action potential.[14] Engbaek *et al.*[15] also found that the EEMG signal was highly temperature dependent. They reported (in an animal model) that a 1°C decrease in muscle temperature was associated with a 6% increase in T_1 from control levels. They suggested that the failure of the EEMG to return to 100% of control during clinical studies might be in part due to changes in muscle temperature.

It was our hypothesis at the start of this investigation that these temperature changes were the dominant cause of the drift seen in the EEMG when employing the Datex monitor. While in general we did find a correlation between T_1 drift and temperature, we were unable in any specific patient to apply a temperature correction factor with any confidence. In five of 15 individuals it was not possible to "temperature correct" the final T_1 value at full recovery to within ±10% of control. The extent of this uncorrected "drift" in T_1 was not a function of time since calibration, and was unrelated to any other modifying factor that we could identify.

In view of the above findings, investigators using the Datex Relaxograph to record changes in T_1 should report the conditions under which their data were obtained in detail. How long was the period of signal stabilization? What was the hand temperature at the time control responses were recorded? To what extent did hand temperature change during the period of observation? Did T_1 return to control at the end of the observation period? What was the TOF ratio at the time when T_1 was determined to be fully recovered? If T_1 did not return to 100% of control at a time when the TOF ratio had recovered to a value ≥0.90, how did the investigator "normalize" the data? Unless answers to these questions are provided, meaningful interpretation of the investigator's data may not be possible.

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