

Title: LOCALIZED HYPOTHERMIA POTENTIATES NONDEPOLARIZING NEUROMUSCULAR BLOCKADE
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Introduction. Previous studies have shown that non-depolarizing neuromuscular blockade (NNMB) is potentiated by generalized hypothermia. (1,2) Apparent differences in the level of NNMB may be noted in a patient when the monitored limbs differ in temperature, even when core temperature is normal. Therefore, we measured integrated evoked electromyographic (IEMG) responses in upper extremities (UE) of differing temperatures to determine whether localized hypothermia altered the assessment of NNMB.

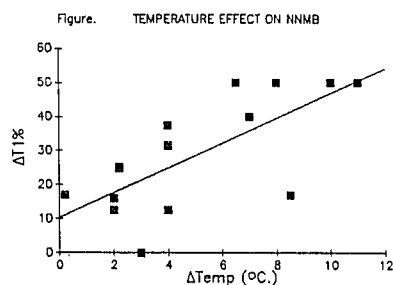
Methods. After receiving approval from the Committee on Studies Involving Human Beings, 15 ASA I-III neurosurgical patients, ages 20-65, with non-lateralizing neurological exams, had bilateral upper extremity (UE) monitoring of NNMB with the Datex NMT 221 (Puritan-Bennett) IEMG monitors (1 for each UE). Supramaximal train-of-four (TOF, 0.5 Hz) stimuli of 0.1 msec duration were delivered through surface electrodes applied over the ulnar nerve at the wrist of both UEs following induction and at 20 second intervals thereafter for the remainder of surgery. The IEMG quantitated electrical activity over the hypothenar aspect of each hand; twitch height was expressed as a percent of unparalyzed control twitch height (T1%). Temperature in each UE was measured with surface thermistors applied over the adductor pollicis and shielded with YSI 4009 heat reflecting shields (Yellow Springs Instrument).

The anesthetic regimen consisted of 70% N₂O in 30% O₂ and meperidine 1-3 mg/kg or fentanyl 5-7 µg/kg; vecuronium was administered for muscle relaxation in a bolus of 0.05-0.08 mg/kg, then followed by an infusion adjusted to keep the T1 of one extremity at 10% of unparalyzed control. Isoflurane was administered in low inspired concentrations for BP control in 11 patients. The intravenous catheter was placed in the warm UE. The BP cuff (when used) was also placed on the warm UE. The contralateral UE was cooled to a surface temperature no lower than 22°C by exposure to ambient temperature; or, by combinations of ice and air convection. Bilateral UE, core (esophageal), and ambient temperatures, as well as T1%, were recorded throughout surgery. For data analysis, the difference in T1% between the warm and cold extremity ($\Delta T1\%$) was calculated. Patients were then assigned to 1 of 2 groups based on the extent of UE temperature differences ($\Delta Temp$): low $\Delta Temp$ ($<6^\circ C$) or high $\Delta Temp$ ($\geq 6^\circ C$). Measurements of $\Delta T1\%$ at 25%, 50% and 75% spontaneous recovery from NNMB in the warm UE were compared between the 2 groups using the Mann-Whitney U test.

Results Mean core temperature was $35.8 \pm 0.62^\circ C$ (SD) in the patients studied. Mean warm UE temperature was $32.9 \pm 1.3^\circ C$ (SD). $\Delta Temp$ ranged

from $0.2-11^\circ C$ between the 2 UEs. The relationship between $\Delta Temp$ and $\Delta T1\%$ at 50% recovery of the warm UE is shown in the Figure. There was a significant difference in measured NNMB when low $\Delta Temp$ patients were compared to high $\Delta Temp$ patients ($p < .002$). The difference in NNMB between low $\Delta Temp$ and high $\Delta Temp$ patients was also significant at 25% recovery of the warm UE ($p < .001$) and 75% recovery of the warm UE ($p < .008$). Regression analysis at 25%, 50% and 75% recovery showed a positive correlation between $\Delta Temp$ and the $\Delta T1\%$ (Table and Figure). In addition, 4 patients with $\Delta Temp \geq 6^\circ C$ showed no clinical evidence of spontaneous recovery in the cold UE despite steady recovery in the warm UE.

Discussion. Localized hypothermia in the monitored extremity potentiates NNMB as measured by IEMG. While the question may be raised as to whether IEMG measurement underestimates neuromuscular recovery, prior studies (2,3,4) as well as our observations of gross mechanical evoked responses indicate that the mechanical response to TOF actually lags behind IEMG during spontaneous recovery from NNMB. These findings have important clinical implications, insofar as the patient's level of NNMB is usually monitored in an accessible extremity. This extremity may be significantly colder because of exposure to ambient temperature. If administration or reversal of non-depolarizing relaxants is based on monitoring in a cold extremity, 1) an inadequate general level of NNMB may exist, and 2) furthermore, patients may be presumed (incorrectly) not to be "ready" for reversal of NNMB.



Table

$\Delta Temp$ vs. $\Delta T1\%$			
		r	p
25% recovery	(N=15)	.722	<.01
50% recovery	(N=14)	.710	<.01
75% recovery	(N=12)	.608	<.05

References:

- 1) Miller RD, Van Nyhus LS, Eger EI: Anesthesiology 46:333-335, 1977.
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- 3) Weber S, Muravchick S: Anesth Analg. 65:771-776, 1986.
- 4) Kopman AF: Anesthesiology 63:208-211, 1985.