

TITLE: EFFECTS OF PHYSIOLOGIC ALTERATIONS ON NEUROGENIC MOTOR EVOKED POTENTIALS IN SWINE
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Neurogenic motor evoked potentials (NMEP) are a recently described method of monitoring the integrity of the anterior spinal cord during surgery. The efficacy of NMEPs in detecting motor tract dysfunction during spinal surgery has been shown in animals and humans. While this technique is gaining popularity, the effects of alterations in PaCO₂, MAP, and temperature on NMEP are not well known. This experiment examines the effects of PaCO₂, MAP, and temperature changes on the amplitude and latency of NMEPs.

With approval from the Clinical Investigation Animal use Committee, eight hogs (18-24 kg) were anesthetized and instrumented. Anesthesia was maintained with a ketamine infusion and pancuronium bromide. Monitors included femoral arterial line, PA catheter, pulse oximeter, mass spectrometry and temperature (core). NMEPs were obtained utilizing a Nicolet Compact 4 Clinical Averager. Stimulating electrodes were placed within the T₂ and T₃ interspinous ligaments after excision of the spinous processes. Paired bipolar recording electrodes were placed over each sciatic nerve under direct vision via incisions over both hips.

PaCO₂ -After obtaining baseline NMEPs, at a PaCO₂ of 40mmHg, ventilation was manipulated to raise or lower PaCO₂ in 10mmHg increments (20-70mmHg). NMEPs were obtained at each level of PaCO₂. MAP and temperature were maintained.

MAP -MAP was lowered using nitroprusside and esmolol infusions. Infusions were titrated to lower the MAP in 10mmHg increments. NMEPs were obtained at each level of MAP to a MAP of 30mmHg. PCO₂ and temperature were maintained.

Temperature -Temperature was recorded from the PA catheter thermistor. A cooling blanket was used to gradually lower core temperature 1°C at a time. At each 1°C decrease (36-31°C) NMEPs were obtained. PCO₂ and MAP were maintained. Amplitude was measured as greatest positive to negative peak deflection. Latency was measured as the initial positive or negative deflection from baseline.

Hypercapnia nor hypocapnia had any statistically significant effect on either latency or amplitude as measured by T-testing and ANOVA.

Incremental decrease in MAP reveal preservation of latency but there was predictable (p<0.05) decrement in amplitude beginning at a MAP of 60mmHg and reaching 50% reduction in amplitude at 30mmHg.

Temperature effects on NMEPs differed from that of MAP effect in that latency was markedly prolonged (r=.8 ANOVA, p<.001) without predictable effect on amplitude (ANOVA p=.23).

Knowledge of the effects of physiologic variables on NMEPs is important to aid in interpretation of each response. There was no significant effect over a wide range of PaCO₂ (20-70mmHg). This is not surprising since O₂ responsiveness at the spinal cord level appears to be 50% that of the central vasculature. With the use of deliberate hypotension in spinal surgery to decrease blood loss, it may be clinically significant as far as monitoring NMEPs to drop MAP near the lower limits of autoregulation. It may be difficult to assess whether changes in NMEP are related to surgical manipulation or hypoperfusion.

With changes or temperature amplitude varied widely but had no predictable change. Conversely, latency showed a linear increases with decreasing temperature (p<.005).

In summary, with the exception PaCO₂ changes, variation in blood pressure and temperature within the clinical range may have tremendous effects on NMEP.

Reference. Owen JH et al. Spine 13:1111, 1988.

TITLE: PRESSURE WAVEFORM LOCALIZATION OF MULTI-ORIFICED CVP CATHETERS
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Introduction. Venous air embolism (VAE) is a potentially catastrophic surgical complication with an incidence of 8-15%. (1) Early diagnosis and treatment is imperative to prevent significant morbidity or death. Definitive therapy relies on the aspiration of air through a central venous (CVP) catheter. A multi-orificed CVP catheter positioned .5-2.0 cm below the sino-atrial (SA) node is the optimal location for air aspiration (2). The only methods currently available for accurate localization are intravenous ECG (IVECG) or x-ray confirmation. Use of a right ventricular (RV) pressure wave form as a guide for placement of a CVP catheter may be a potentially reliable and simple technique. We attempted to define the distance from the loss of a RV waveform to the appearance of a diagnostic IVECG indicating catheter localization at the SA node prospectively in 15 patients.

Methods. After approval of our Institutional Review Committee, 15 patients scheduled for craniotomy were evaluated. An introducer sheath was inserted into the brachial vein and a multi-orificed CVP catheter advanced while simultaneously monitoring IVECG and pressure waveform until a RV pressure tracing was obtained. The catheter was withdrawn slowly until the RV trace was lost, and then by .5cm

increments until a biphasic p wave was obtained on IVECG. The distance from loss of the RV pressure trace to the appearance of a biphasic p-wave was measured. Patient data, time for insertion, success rate and complications were also recorded.

Results. Results are summarized in Table 1. Average distance from loss of a RV pressure waveform and appearance of a biphasic p wave was 3.0 ± .1 cm. Success rate for placement of the catheter into the central circulation was 88%.

Discussion. The results of our study indicate that utilizing pressure monitoring alone can provide another accurate method of localizing CVP catheters for VAE treatment. The distance from loss of RV pressure tracing to the SA node was very consistent with little variability in this patient population. This technique is simple, reliable, and does not require additional materials or specialized CVP catheters with ECG monitoring capability. In addition, because catheter migration can occur with arm movement during surgery, and may be as much as 5cm (3); pressure waveform localization provides an easy method for intraoperative repositioning of CVP catheters.

TABLE 1 (Mean ± SEM)

Height	72.2 ± 3.2 inches
Distance	3.0 ± .10 cm
Insertion Time	6.8 ± 4.0 min.

References:

1. McNeice WL, Adv. Anesth. 4:151-184, 1987.
2. Bunegin L, Anesth. 55:343-348, 1981.
3. Kalso E, Acta. Anaesth. Scand. 26:354, 1982.