Mount Sinai, only items specific to the current patients are to be placed in this area. As reported in the manuscript, this is *in addition* to terminal cleaning. We do not use a disposable sterile drape, an interesting caveat. Additional medications that have been prepared are also kept on the top of the anesthesia cart. Universal precautions, such as wearing a gown for patients in contact isolation, also apply at Dartmouth-Hitchcock Medical Center. We have various measures to control proper handling of controlled substances, an issue that is seemingly separate from infection control. All of our central lines are placed with central line dressings impregnated with chlorhexidine. Your investigation of stopcocks is interesting, timely, and in parallel with an ongoing study at our institution. In summary, based on the description which you provided of your infection control practices, the use of sterile drapes between patients serves as the only basis for variability in our infection control practices. Like use of gloves, this is unlikely to impact bacterial cross-contamination without intraoperative adherence to Centers for Disease Control and Prevention guidelines for hand hygiene. A more useful comparison for infection control practices at our respective institutions would be a report of hand hygiene compliance of anesthesia providers; the number one preventative measure for healthcare-associated infections.[2]

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Anesthesiology 2009; 110:1192–4

Increased Impedance on Nerve Stimulator Display May Actually Reflect a Decrease in Total System Impedance

To the Editor:—Tsui *et al.* in their paper "Electrical Impedance to Distinguish Intraneural from Extraneural Needle Placement in Porcine Nerves during Direct Exposure and Ultrasound Guidance" make a very interesting observation: An increase in electrical impedance on needle entry into a nerve.[1] However, although interesting, the finding is ascribed to an electrical parameter that it likely does not represent. As such, this is an example of being misled by some of our equipment that is routinely used without questioning what is actually being shown.

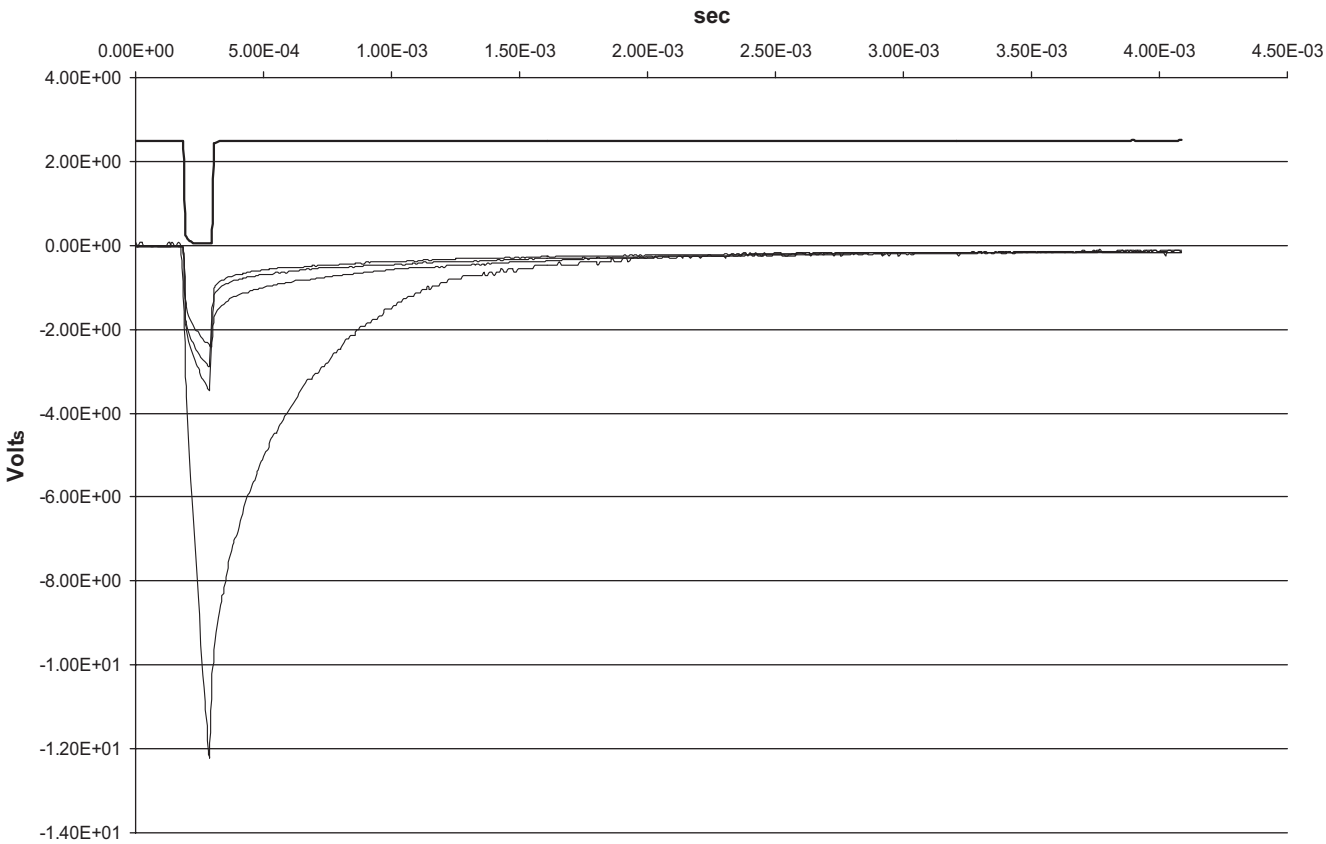

Fig. 1. Voltage responses for a 0.489 mA square waveform current pulse measured across a fixed 4.92 kΩ (displaced + 2.5 V for display purposes) and across a tissue/electrode system using a 22-gauge Stimuplex needle (D. Braun Medical, Bethlehem, PA) inserted to a depth of 5 mm with several return electrode configurations.

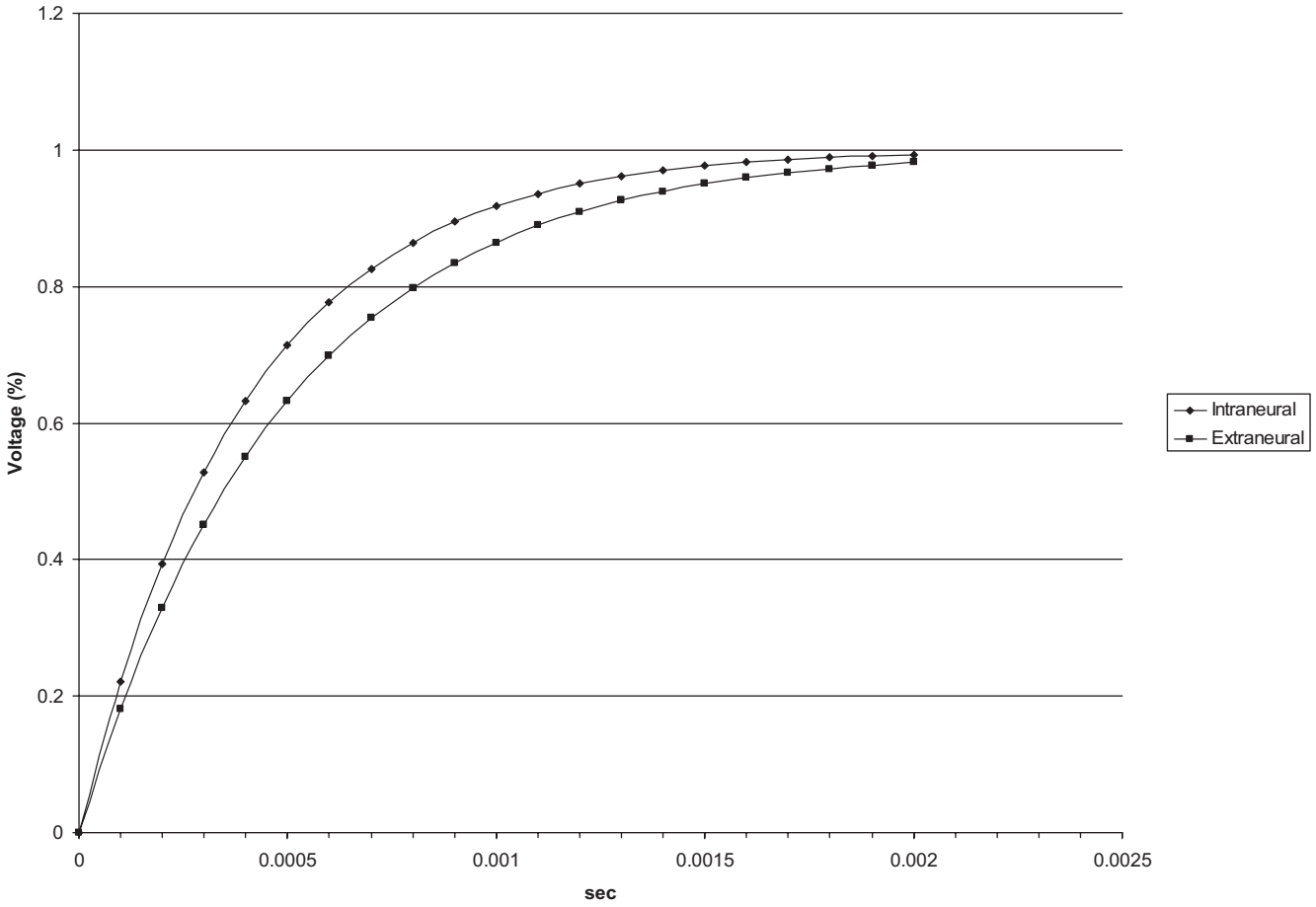

Fig. 2. Charging curves with C = 5 × 10^{-8} F and R = 1 × 10^{4} Ω for the extraneural curve and C = 1 × 10^{-6} F and R = 4 × 10^{2} Ω for the intraneural curve (values according to Prokhorov *et al.*[4]). Note that at 0.0001 s, there is a 20% difference between the developed voltages. Impedances: extraneural = 3 MΩ, intraneural = 160 kΩ.

For reasons not clear to me, a few years ago some nerve stimulators began displaying something called "impedance." At the time, this struck me as odd, since these devices produce square waveform outputs. Now, with the publication of Tsui *et al.*, this issue needs to be addressed, because it is leading to confusion.

It is well established both theoretically and experimentally that nerves display a *reduced* impedance when compared to surrounding tissues.[2–4] Yet, Tsui *et al.* carefully document an abrupt increase in "impedance" on needle penetration of a nerve, as determined by a Stimuplex HNS 12 (D. Braun Medical, Bethlehem, PA). So, how do we resolve this apparent contradiction? Although this format does not allow for a comprehensive discussion of the essential issues, there are some details that provide clarification of the disparate data.

All commercially available nerve stimulators for regional anesthesia are constant current devices: They put out a square waveform pulse of user-defined current amplitude. Although such an output results in a square waveform voltage pulse when the current is directed across a resistance, tissue represents a parallel resistance capacitance circuit. When a square waveform current pulse is directed across tissue, the resulting voltage pulse demonstrates a charging component, as shown in figure 1. Note that the voltage pulse requires 2–2.5 ms to achieve its plateau value in response to a square waveform current pulse. Consequently, the 100–200 μs pulse durations of commercial nerve stimulators are not adequate to allow the developed voltage to reach the plateau value. To calculate the impedance of such a circuit, the plateau value must be achieved, reflecting the resistive component of the resistance capacitance circuit. Knowing the resistance, the capacitive component may be approximated from the slope of the charging curve. It may only be approximated, because the actual charging curve is defined by the multiple electrical path components, shown in equation 1, and their individual charging time constants (τ), where τ is defined as the product of R and C.

$$V = \sum C_0 e^{-t/\tau_0} + C_1 e^{-t/\tau_1} + C_2 e^{-t/\tau_2} + \cdots + C_n e^{-t/\tau_N} \quad (1)$$

What the nerve stimulators appear to be doing when displaying "impedance" is taking the maximum developed voltage at the end of the current pulse, dividing it by the known applied current to yield the "impedance" according to Ohm's Law.

$$\frac{E}{I} = Ze^{j\phi} \quad (2)$$

where E is voltage, I is current, Z is impedance, j is the square root of –1, and φ is the phase angle. However, the voltage maximum for the applied current has not been reached during the period of the nerve stimulator pulse, so the displayed value clearly does not represent the electrical impedance of the system. What Tsui *et al.* observed was when the needle tip was intraneural, the developed voltage was higher at pulse termination than when the needle was extraneural. This was not a measure of system impedance, but was actually a function of the reduced time constant associated with the changes in capacitance and resistance of the nerve, modeled in figure 2.[4]

The reduced effective system time constant with intraneural needle tip placement resulted in a more rapid rate of rise of the voltage curve, even though its final value may be unchanged or even reduced. With intraneural placement, the capacitance increased, the resistance decreased, and

the resulting impedance was actually reduced, according to equation 3.

$$Z = \sqrt{R^2 + X_C^2} \quad (3)$$

where X_C is the capacitive reactance and is defined by:

$$X = \frac{1}{2\pi fC} \quad (4)$$

where f is the frequency of the applied waveform. Dependent on the magnitude of the capacitive and resistive changes, the overall impedance decreased dramatically.

This finding of Tsui *et al.*, although it does not demonstrate increased impedance on intraneural needle tip placement, is interesting, because it shows that the complex system time constant changes on close approach to neural tissue when using externally applied electrical fields, an observation I have also made.[5] Also, individual time constants, equation 1, may be derived from the observed charging/decay voltage curve through additional mathematical methods (logarithmic peeling), and the time constants contributed by the nerve, the insulated needle, or the remaining tissue electrical path determined separately.[6]

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In Reply:—We are very pleased that our report has stimulated the important comments made by Dr. Cory. We would like to take this opportunity to address the correspondent's concerns and to clarify the simplified electrical circuit depicted in the original manuscript.[1]

Using a clinically relevant low frequency (2 Hz) stimulation, we had hoped to detect any possible warning signs of intraneural needle placement by understanding how to interpret the displayed impedance from one of the common commercial stimulators. We were most encouraged to note the distinct impedance change displayed on the stimulator upon the needle entering the intraneural compartment. The specific concern expressed in the above letter relates to the proposed inaccuracy in the interpretation of the displayed impedance. From observations based on human data, Dr. Cory posits that the maximum voltage may not have been reached within such short pulse duration (0.1 ms).

First of all, it is important to clarify that the simplified circuit of the original manuscript represents only the resistive portions of the circuit. Strictly speaking, an accurately depicted circuit would be much more complex and include the capacitance and inductance of the many tissue types (fig. 1). However, we believed such complex electrical circuitry may have distracted readers from the primary goal of the research. Despite this, there was no intention on our part to undermine the research methodology. Specifically, the effect of capacitance in the porcine model on the dynamic time-dependent component of impedance is negligible when compared with that in humans. This is based on the observed rapid rise in the voltage–time curve, which has a maximum voltage plateau phase near 0.1 ms in a porcine model (fig. 2).[2] Therefore, the displayed impedance from the stimulator is less affected by an increase in pulse duration and is a reasonable approximation. This is in contrast with our unpublished human volunteer data (fig. 3). In humans, the voltage–time response curve takes longer to reach the maximum voltage plateau phase (2–2.5 ms). Therefore, the displayed impedance will change substantially, along with the prolonged pulse duration for extended periods. This is why we clearly pointed out the limitations of our investigation in the manuscript as *"we anticipate that there may be substantial interspecies differences in EI... alternatively a percentage change in EI from the extraneural compartment in humans indicative of intraneural placement would be of high clinical value."* This may rectify the confusion to which Dr. Cory refers, as he may have missed or was unaware of such interspecies differences.

We thank the correspondent for his helpful comments, and we are grateful for the opportunity to clarify our results. We must also emphasize that it was never our intention to relate the absolute mechanism of the complex circuit. Instead, our intent was to examine the

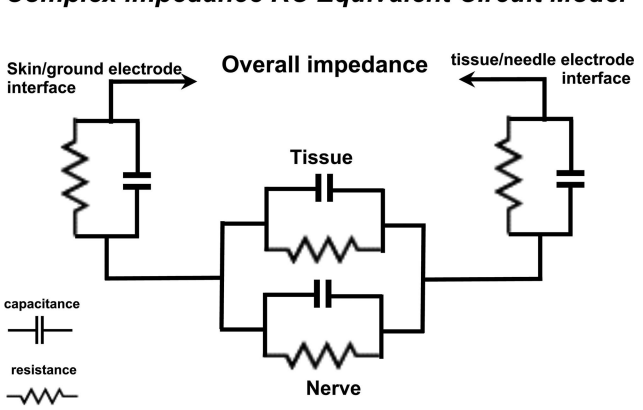

Fig. 1. Schematic complex impedance resistance–capacitance equivalent circuit model.

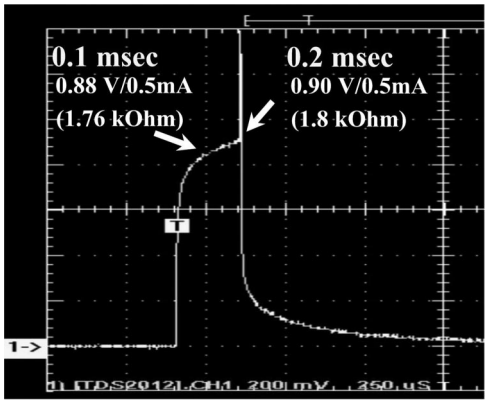

Fig. 2. Voltage–time curve in a porcine model. Example of the voltage response after applying 0.5 mA with a 0.2 ms pulse width *via* an 18-gauge insulated needle placed extraneurally. Adapted from Tsui *et al.*,[2] with permission from Elsevier.