units, as well as other types of monitors, employ RF interference suppression circuits on the inputs to their amplifiers. These circuits generally take the form of capacitive filters. The installation of an inductor in series with these filters can produce a resonant circuit which, without adequate resistive damping or other protection, could affect the input amplifiers of the monitor. While the use of inductors, as outlined by Hall *et al.* and ourselves, may limit problems encountered with ECG electrodes in the presence of ESU's, a critical analysis of the effects of defibrillation potentials (under the conditions of worst placement of the paddles relative to the electrodes) on such modified leads needs to be made. The use of inductors in ECG leads does constitute a design modification which should, perhaps, be verified with the ECG manufacturer before use.

We are grateful to Drs. Hall, Malhotra, and Hedley-Whyte for drawing these matters to our attention and for permitting a further discussion of this most important problem of RF burns.

BRYAN FINLAY, PHD., P.ENG.
DARRELL COUCHIE, C.E.T.
LARRY BOYCE, C.E.T.
EDWARD SPENCER, C.E.T.
TOM AVEY
Department of Biomedical Engineering
University Hospital
London, Ontario, Canada N6G 2K3

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Serum *d*Tc and Neuromuscular Blockade in Man

To the Editor:—The conclusion of Matteo, Spector, and Horowitz[1] that the intensity of neuromuscular blockade from *d*-tubocurarine (dTc) can be causally related to serum *d*Tc concentration must be questioned.

The classic isolated-arm experiments of Feldman and Tyrrell[2] showed conclusively that rapid lowering of blood concentration of a nondepolarizing relaxant does not speed up return of neuromuscular transmission. In their view, the rate of recovery is governed instead by the strength of the drug–receptor association. While Feldman and Tyrrell did not actually measure plasma levels of *d*Tc, it is difficult to fault their line of reasoning.

The results of Matteo *et al.* do not necessarily challenge Feldman's work. The time course of the decay of plasma *d*Tc concentration following a *single bolus* injection closely parallels the dissociation rate of the drug–receptor complex. I suspect that if Matteo *et al.* had compared a single bolus of 0.8 mg/kg with a steady infusion of, perhaps, 0.015 mg/kg/min they might have reached very different conclusions.

AARON F. KOPMAN, M.D.
Department of Anesthesiology
Long Island Jewish-Hillside Medical Center
New Hyde Park, New York 11040

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To the Editor:—Matteo, Spector and Horowitz are to be congratulated on acheiving a radioimmunoassay technique for the estimation of serum *d*Tc. However, I cannot agree that because they demonstrate a coincident correlation between the decrease in serum *d*Tc and a diminishing neuromuscular block that one can claim that the intensity of neuromuscular blockade necessarily can be related to the serum concentration of the drug. What they demonstrated was that following injection of *d*Tc the serum levels of drug in similar individuals fall at approximately the same rate and that the coincident recovery of neuromuscular blockade, following an injection of *d*Tc, also occurs at similar rates in different individuals, a somewhat surprising finding[1,2] especially considering the variable extents of respiratory alkalosis.[3,4]

In order to demonstrate that neuromuscular blockade is related to serum concentration of a drug it is necessary to show that alterations in serum concentration, such as a sudden reduction, cause commensurate changes in neuromuscular blockade. This in fact has been demonstrated not to be the case with *d*Tc and other nondepolarizing muscle

relaxants at the neuromuscular junction and at muscarinic receptors.[5,6] The demonstration that it is possible to have very different intensities of neuromuscular blockade in the two arms of the same person while the serum concentration of drug is unchanged[5] is proof that recovery from neuromuscular block is not directly related to reduction in concentration.

S. A. FELDMAN, F.F.A.R.C.S.
Magill Department of Anaesthetics
Westminster Hospital
Westminster Medical School
(University of London)
Page Street
London, SW1P 2AP
England

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To the Editor:—Our data do not challenge Feldman and Tyrrell's work. It is not possible to compare their results with ours since kinetics in the whole body are so different from kinetics in their isolated-arm experiment.

There is no doubt that a *d*-tubocurarine—receptor complex is formed, but we believe that in the whole body there is a point when dynamic equilibrium between the concentrations of *d*Tc at the endplate, in the extracellular fluid, and in the serum is reached.

We see nothing surprising about the apparent correlation between serum *d*Tc concentration and intensity of neuromuscular blockade. To be sure, when a dose of *d*Tc is given as a bolus the initial rapid distribution of *d*Tc to specific and nonspecific binding sites would make the serum *d*Tc concentration measured *at that time* unrelated to events occurring at the neuromuscular junction. During recovery phase, however, *d*Tc must leave the neuromuscular junction and enter the extracellular fluid, and eventually the plasma compartment. This is the phase during which we measured serum *d*Tc concentration and neuromuscular transmission and showed a good correlation.

We do not see the point of suddenly changing serum *d*Tc concentration, and this is not a usual clinical situation. If this occurred, the kinetics would become unstable and measurements would not be valid.

RICHARD S. MATTEO, M.D.
Department of Anesthesiology
College of Physicians and Surgeons of Columbia University
New York, New York 10032

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An Off Day

To the Editor:—Knowledge of the fastidious review process of the Editorial Board of ANESTHESIOLOGY leads me to rely heavily on an accurate abstract for surveying information content of published articles. Imagine, then, my dismay on reading the entire article "Cardiovascular Effects of Isoflurane in Surgical Patients," by Graves *et al.* (ANESTHESIOLOGY 41:486–489, 1974). Table 3, which summarizes all their data, contains 28 cells, in each of which some measure before and after isoflurane is compared, the mean difference presented, and the statistical significance of the difference noted. In at least ten of the 28 cells errors in simple subtraction occur, and in at least half of these, increases are reported as decreases or *vice versa*. Further, in the first paragraph of Discussion, decreased stroke volume is considered "depression of the heart" and is expected to be reflected in decreased arterial pressure. To top this, "increased diastolic pressure . . . suggested incomplete cardiac emptying." Based on observed increases in