Correspondence

Anesthesiology
47:475, 1977

Electrical Stimulation and Sympathetic Hyperactivity

To the Editor:—We recently reported the case of a patient who, we believed, might have experienced an increase in regional sympathetic tone following transcutaneous electrical stimulation.\(^1\) Stilz and Carron, in a recent letter, questioned the existence of sympathetic hyperactivity in response to transcutaneous stimulation.\(^2\) Their criticism of our conclusion was threefold: First, they stated that they have seen decreased sympathetic tone in many patients following stimulation. This is a valid point. We, too, have seen significant increases in skin temperature and digital pulse wave amplitude in many patients, and are treating some of our patients with sympathetic dystrophy with transcutaneous stimulation. Second, they stated that they have not observed sympathetic dystrophy triggered by stimulation. Indeed, our reported case is the only one we have seen. We have, however, documented decreases in skin temperature, diminished pulse wave amplitude, and exaggerated sympathovagal reflexes in patients with chronic pain and healthy volunteers after stimulation. Third, they stated that spread of our patient’s pain to the arm and shoulder might represent the natural history of her disease. We feel this explanation is unlikely in light of this patient’s excellent health and her nine-year history of only localized pain without evidence of sympathetic hyperactivity.

It is frequently assumed that transcutaneous electrical stimulation acts at the dorsal horn level to inhibit activation of the action system.\(^3\) It is possible, however, that stimulation modulates pain in the periphery,\(^4\) or more centrally, e.g., via the central gray area,\(^5\) or as Stilz and Carron and I have suggested, through changes in sympathetic tone. Until the mechanisms of action of this treatment modality have been worked out, we should be aware of the possibility that a variety of effects, both beneficial and harmful, are possible.

We have documented both increases and decreases in sympathetic tone in our patients, and, while a single case report serves as scant evidence for the existence of post-stimulation sympathetic dystrophy, we should consider the possibility of such an entity unless the weight of future evidence disproves it.

Stephen E. Abram, M.D.
Assistant Professor of Anesthesiology
Medical College of Wisconsin
Milwaukee, Wisconsin 53226

REFERENCES


(Accepted for publication June 16, 1977.)

Inadequate Study of Coronary Vascular Resistance

To the Editor:—Whenever an investigation collects objective, numerical data but presents the results only as percentage changes from control, my suspicion is immediately aroused. The paper by Domenech et al.\(^1\) is certainly a case point. We are asked to accept an open-chest dog preparation "anesthetized" (?) with a total dose of thiopental, 17.3 mg/kg, over an unspecified time period as the "control" state for cardiac and coronary hemodynamic values. In my experience (more than 200 dogs), this dose given over a 2–4 min time span will not predictably produce enough anesthesia to keep all dogs from reacting to endotracheal intubation. In any event, these animals were doubtless in what most physiologists today would consider a "hyperdynamic" state, with heart rates of more than 100 beats/min and a mean aortic pressure above 120 mm Hg. Consequently, the changes produced by one concentration of halothane may well

475