(Combinations do occur, but were not the subject of this paper.) I hope the authors will clarify their message.

DAVID H. SPODICK, M.D.
The St. Vincent Hospital
Worcester, Massachusetts

References

GIK and Pacing Response

To the Editor:

The paper on the protective effect of glucose-insulin-potassium on the pacing response by Chiông and colleagues (Circulation 54: 37, 1976) is an example of the high standards and meticulous work of Dr. Parker's cardiology division. It is unfortunate, therefore, that it is flawed by the exclusion of one element vital to any reliable result: administration of a placebo infusion with double blinding. This could have been given to the same patients or to matched patients with the order of administration determined by randomization. It is particularly unfortunate that, among all nonsurgical specialists, cardiologists remain conspicuous for their reluctance to accept appropriate design and control of clinical trials.

DAVID H. SPODICK, M.D.
The St. Vincent Hospital
Worcester, Massachusetts

References

The authors reply:

We appreciate Dr. Spodick's comments on our paper and share his concern about the reproducibility of the pacing-induced ischemic syndrome. The administration of a placebo infusion to the patients who received the glucose-insulin infusion was not attempted since this procedure would have prolonged the study by at least another hour. We are in the process of obtaining evidence that, in this clinical experimental model, the ischemic syndrome is reproducible in two consecutive pacing periods.

M.A. CHONG, M.D.
R.O. WEST, M.D.
J.O. PARKER, M.D.
Queens University
Kingston, Canada

SVT in Children

To the Editor:

In a recent paper, Gillette claimed: "to date, no studies have been reported with regard to the mechanism of supraventricular tachycardia in children utilizing the new electrophysiologic techniques."

I write to draw your attention to the fact that the pioneering work of the cardiologists at the Hopital Lariboisiere in Paris has been neglected. This article, in English in an American journal, should, I feel, have been noted by the reviewers even if missed by the author. Work that has been reported during the past decade would thus have received due recognition. Furthermore, fundamental advances in the understanding of the mechanisms of tachycardia especially relevant to children could have been discussed.

DENNIS KRIKIER, M.D.
Hammersmith Hospital,
London, England

References

ST-Segment Mapping — A Cardiologist’s Tease

To the Editor:

The informative editorials on ST-segment mapping by E. Braunwald, PR Maroko and HA Fozzard and DS Das Gupta, are important contributions to those engaged in future research and conceptual thinking. ST mapping is a very attractive tool because within few moments of global ischemia of the left ventricle, the “skirt” of ST-segments is elevated for clinicians to peer within and size up. However attractive this tool may be, it is an electrophysiological system which has not been clarified. I certainly agree with Braunwald that we earnestly need to obtain an electrical model of the sequential changes that take place from the onset of ischemia to the stage of chronic infarction.

ST-segment mapping, being an electrophysiological system, does not chart the highly complex biologic system. A patient with acute myocardial infarction is subject to a myriad of intrinsic and extrinsic influences which may alter ischemic injury. The changes in ST-segment elevation produced by these factors may mislead the unwary clinician. To my knowledge, there has been no sophisticated study employing simultaneous intramyocardial, epicardial, and precordial ST-segment mapping correlating with CPK activity, sympathetic stimulation, alterations in pH, electrolytes, hypoxia, temperature changes, with and without ischemia. This type of carefully conducted study could clear some of the hanging clouds and provide us with a more lucid picture of the factors affecting ST segments and the usefulness of ST-segment mapping as an index of myocardial ischemia. We have been prematurely cautioned (rather, misinformed,) by Fozzard and Das Gupta that “ST-segment mapping is not a reliable measure of myocardial ischemia.” Their conclusion should not be taken seriously; at the best, it is theoretical. I certainly agree that at the present time we cannot convert ST-segment changes into a quantitation of necrotic myocardium. There is good evidence, however, that epicardial ST-segment mapping reflects the ischemic injury and precordial ST-mapping projects the underlying electrophysiology.

We should certainly take heed of the limitations of ST mapping in programming future research using the tool to quantitate myocardial necrosis. ST-segment changes, at the present state of our knowledge, cannot be used to indicate grams of infarcted myocardium. We have to take into consideration both spatial and non-spatial factors.

Computers are a big help. Automated precordial mapping may provide a rapid and accurate analysis and minimize interobserver variability. One could use as many electrodes as the computer could handle, or the torso could accommodate, to map every millimeter of the ischemic myocardium.

AMAR KAPOOR, M.D.
Victoria General Hospital
Dalhousie University
Halifax, Canada

References

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AMAR KAPOOR, M.D.
Victoria General Hospital
Dalhousie University
Halifax, Canada

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A Kapoor

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