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In Reply:—We appreciate the merit of the observation made by Blanck and Gardner regarding the ischemia-related injury to porcine cardiac sarcolemma.

The study of Blanck and Gardner supports our findings of increased binding capacity of the voltage-sensitive calcium channels (VSCC) to isradipine calcium channel blocker after ischemia.[1] However, they suggest a different explanation for this phenomenon, based on the parallel increase in 5′-nucleotidase, which might have originated from previously sequestered channels exposed by ischemia.

The preparation used by us is constituted of highly purified vesicles of sarcolemma membranes,[2] almost totally deprived of other cell constituents. As previously described in the literature, the histologic changes observed after 10 min of regional ischemia in canine heart are usually minimal, with less than 2% infarcted tissue. In such experimental protocol, the suggestion made by Blanck and Gardner of sequestered channels from a hidden pool is therefore, although possible, less likely than the mechanism offered in our article. We proposed the explanation of differential unmasking of latent channels in the sarcolemma.

The valuable article of Hoehner *et al.*[3] mentioned the theory of artifactual observation but gave it a low priority, because they found, very similar to our findings, "the same crude homogenate protein content in ischemic and nonischemic tissues." The similar amount of sarcolemmal membranes in the ischemic and control tissues suggests, in our opinion, that the increase in binding capacity of the VSCC originates by a conformational change due to biochemical process in the sarcolemmal vesicles rather than by exposure of membranes from a hidden pool.

In summary, our findings of increased calcium channel blocker binding to sarcolemmal VSCC and the fact that halothane anesthesia might prevent the ischemic changes in the myocyte correlate with other studies emphasizing the protective role of halothane during myocardial ischemia. As we stated in the original article, the mechanism underlying the increase in binding capacity of VSCC is not completely clear and deserves further investigation.

Benjamin Drenger, M.D.
Director, Cardiothoracic Anesthesia Unit
Department of Anesthesiology and Critical Care Medicine
Hadassah University Hospital
P. O. Box 12000
91120 Jerusalem, Israel

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Elastance *Versus* Compliance

To the Editor:—A recent case report[1] suggests that the plea by Lanier and Warner that the anesthesia community discontinue its use of the term "compliance" in connection with the intracranial space has been heard.[2] Others who predicted that the misuse of the term compliance was too "well entrenched" to be changed have been proved wrong.[3] Elastance (elastance = $\Delta P/\Delta V$) is the correct term when referring to the pressure changes that occur inside the cranium in response to changes in the volume of the intracranial contents, and compliance (compliance = $\Delta V/\Delta P$), the inverse of elastance, is incorrect. However, the report by Sperry *et al.* suggests that we are