Because we consider the last paragraph of our editorial germ-an to our concern, reiteration will serve to emphasize its vital importance:

"In the absence of such a reporting system and lacking new scientifically based data to supersede the old, we strongly urge manufacturers of electro-medical equipment to continue to design their systems so as to meet the 1975 AHA committee recommendations and the 1978 ANSI standard. Although we understand the desirability for standards of individual countries to be in harmony with a single international standard, that alone cannot justify a relaxation of limits that may be hazardous to some patients. In the absence of credible data, the increases in risk current permitted by the new standard constitute unconsented-to human experiments to determine the safe range of such currents. There is a clear need for clinical studies (ethically approved) to provide robust information to resolve this issue. We strongly urge clinical investigators and members of industry to collaborate in gathering the appropriate data and designing a reporting system to establish a new realistic basis for safe current limits."

In summary, both dog and human studies dictate the need to limit current through patient leads to 10 μA under single-fault conditions. As recommended previously by the AHA, enclosure leakage current under single-fault conditions should also be limited to 10 μA. With no accessible conducting surface, the leakage current should be limited to 100 μA, providing a reasonable safety factor for strong sensations as documented by Tan and Johnson. These current limitations are within the capabilities of modern engineering design and have been met by electrocardiographs of various manufacturers for many years.

**References**


**Myocardial Ischemia Caused by Exercise Versus Total Coronary Occlusion**

The study by Borges-Neto et al reports interesting findings regarding the extent of myocardial ischemia caused by exercise versus total coronary occlusion, but the conclusions expressed in the "Discussion" section and in the "Summary" may be misleading to clinicians. Indeed, they suggest that studies investigating the "myocardial demand" side of myocardial ischemia may prove to be inadequate prognostic indicators because the extent of myocardium in jeopardy from a stenotic lesion would be larger in the case of sudden coronary occlusion than that expected from exercise studies. Although this latter proposal is certainly true (as it would be true that ischemia caused by the occlusion of a normal vessel would obviously be larger than that induced by exercise in the same territory), it does not reflect the object of radionuclide exercise studies or the reality of progression of coronary artery disease. The main object of radionuclide studies is to detect coronary stenoses that are likely to cause signs or symptoms of myocardial ischemia (ie, stenoses >50%). If such stenoses are more likely to become totally occluded than less severe ones, it must be remembered that, contrary to the occlusion of mere plaques (<50%), the phenomenon of occlusion of severe stenoses is seldom acute and is therefore not comparable to the sudden occlusion caused by balloon inflation during coronary angioplasty: In many instances, because of the development of collateral circulation, total occlusion of a tight stenosis is clinically well tolerated and does not result in myocardial infarction. There remains true that radionuclide exercise studies are unable to detect those atheromatous plaques <50%, which may indeed become unstable and result in abrupt coronary occlusion and myocardial infarction. However, from a "public health" standpoint, exercise radionuclide studies remain outstandingly good prognostic indicators, as recently documented in large series of patients.

Nicolas Danchin, MD
Pierre-Yves Marie, MD
Departments of Cardiologie A and Médecine Nucléaire
Cha Nancy-Brabois
Vandoeuvre-lès-Nancy, France

**References**


**Reply**

We agree with the conclusion of the letter from Drs Danchin and Marie that exercise radionuclide studies remain outstanding prognostic indicators in patients with coronary artery disease. Our group was among the first to report this finding, which has been repeatedly confirmed by many subsequent studies. We disagree that the main object of radionuclide studies is to detect coronary stenosis because our data consistently show physiological markers of ischemia to be stronger prognostic indicators than any descriptor of coronary artery anatomy.

Using physiological tests, it has been possible to dichotomize large populations of patients with known coronary artery disease into those with a low and high risk of subsequent cardiac death. The absolute risk of low- and high-risk subsets is determined by the total risk within the population and the threshold of test result recognized as a positive or negative predictor. Irrespective of the specific noninvasive tests used or the approach used to define low-