we made the mistakes of not pointing out that with our method our upper normal value in serum is 225 IU, and of not expressing the proposed cutoff point as 88.8% of that value, instead of the absolute number of 200 IU, which is valid only for laboratories using exactly the same method we use.

As the normal upper limit for serum for the group of Romero et al is 460 IU, the cutoff point of 200 IU, which represents only 43% of their normal upper limit, obviously results in a very low specificity, only partially improved when they use 307 IU, equivalent to 66.6% of their normal upper limit. We presume that with a cutoff point of 408 IU or 58.8% of their upper normal limit in serum, the results in their patients may be similar to ours. Specificity should increase while a good sensitivity is assured by the low cutoff point of 45 mg/dL for cholesterol.

We think the same explanation is valid for the letter of Drs. Garcia-Pachon and Padilla-Navas, but we cannot be sure because they do not report their upper normal value.

Without pretending to justify our mistakes, we think it is necessary to point out that similar errors are, surprisingly, present in most of the articles reviewed by us: only four report their upper normal limits for serum, which range from 237 to 460 IU3,4,6,7 and only two explicitly express the cutoff point for LDH as a proportion of the upper normal limit in serum.3,5 In another three, this proportion may be calculated4,6,7 and in only three the criteria for pleural LDH of two-thirds the normal upper limit for serum, stated by Light8 in 1983, are applied exactly.3,5

We will not go into further detail because we think that these aspects will be fully considered in the meta-analysis that is being conducted by John E. Heffner, who has requested from us and other groups all the data concerning laboratory methods and the individual results of patients.

To conclude, we would like to emphasize that all future studies involving pleural LDH should be required to report the upper normal limit for serum of that particular laboratory and to express the cutoff point as a proportion of that value.

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Pseudoaneurysm of the Left Ventricle After Mitral Valve Replacement

Surgical Repair or Medical Follow-up

To the Editor:

I read with great attention and interest the article by Fazia and coworkers (February 1996)1 and the case reports of Bline and coworkers (February 1996)2 concerning long-term survival without emergency surgical repair of the four patients with left ventricular pseudoaneurysm following myocardial infarction and mitral valve replacement.

Treatment of these patients, especially with a large pseudoaneurysm, by heart transplantation prior to decompensation, seems to be a very safe solution of the problem for centers with a cardiac transplant program.

I would like to describe herein a patient who developed subannular pseudoaneurysm of the left ventricle after mitral valve replacement. In this case also, “unexpected” 6-year medical follow-up was possible without any surgical attempt.

A 47-year-old woman was first operated on 16 years ago for closed mitral valvotomy. She underwent reoperation in 1989 and the heavily calcified mitral valve was replaced with 29-mm mechanical prosthesis (Medtronic). A De Vega annuloplasty was also done for the insufficient tricuspid valve. The patient had an uneventful recovery. In July 1990, however, she presented as New York Heart Association class IV, and a chest radiograph revealed right lateral enlargement of the cardiac silhouette. Echocardiographic studies and cardiac catheterization showed a subannular posterior left ventricular pseudoaneurysm formation and paravalvular leakage. Her functional status improved with medical therapy (New York Heart Association class II to III) and the patient did not want surgery. Cardiac catheterization was repeated in July 1995. On the follow-up, the sizes of the pseudoaneurysm were not changed, and there is (+/+++) mitral regurgitation due to the paravalvular leakage (Fig 1).

We collected 26 similar cases reported between 1969 and 1994.3,4 The time intervals elapsed from the last surgical procedure on mitral valve to the diagnosis of the pseudoaneurysm vary from 2 weeks to 7 years. One of them was found at autopsy.4 Fifteen cases were treated surgically. Rereplacement of the prosthetic mitral valve and closure of the pseudoaneurysm with a pericardial or polytetrafluoroethylene or Dacron or Teflon patch were generally done. Five of the patients did not survive. Another one survived with a residual channel from left ventricle to pseudoaneurysm. Ten patients were followed-up medically from 5 months to 10 years, without any catastrophe, and there are two reported spontaneous closures.5,6

Prompt surgical repair is said to be the first choice of therapy. Recommended surgical technique of repair is removal of the previous prostheses, internal closure of the orifice of the pseudoaneurysm using a patch, and reimplantation of a new valve. However, especially for patients who had several previous surgical interventions, medical follow-up and echocardiographic survey of the dimensions of the pseudoaneurysm may be safer.

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Communications to the Editor
Is Aging a Risk Factor for Silent Ischemic Cardiopathy?

To the Editor:

One of the most credited theories about the origin of silent myocardial ischemia is the raising of the pain threshold. The relationship between silent ischemia and old age has not yet been defined clearly, but age is often considered a risk factor for this disease.

In our study, we assessed the course of pain threshold and pain tolerance in subjects affected by silent ischemia and attempted to define the role played by age.

We studied 23 male subjects (mean age: 64.7±5.5 years) affected by silent ischemic cardiopathy and 20 male patients (mean age 68.5±8.1 years) with symptomatic cardiac ischemic disease. We evaluated the pain threshold in three different sites (forearm, shoulder, precordium), using electromyographic equipment with a protocol that provides short (0.05 ms) and low-frequency (0.5 Hz) endermic electric stimuli. This was obtained by taking five measurements and calculating the mean of the three central ones.

Pain tolerance was then determined by increasing the stimuli. Exclusion criteria were the presence of significant mental disorders such as excessive anxiety or depression and this was determined using the Hamilton test and the Assessment Scale of Emotivity and Wellbeing in the Elderly (cutoff point was, respectively, at 18 and 6).

Values of pain threshold were assessed using the same method in a group of 40 healthy subjects, five for each age decade, between 10 and 90 years.

Our data show a significant difference of pain threshold and tolerance between subjects with silent and symptomatic cardiopathies (34.7±12.6 mA vs 25.2±12.5 mA; p<0.001 for the threshold; and 68.5±21.2 mA vs 46.0±22.3; p<0.001 for tolerance). The fact that the significance of our results is superior to other studies can be due to the particular method of stimulation used and to the uniformity of the sample studied (sex, age, exclusion of subjects with anxiety and depressive symptoms). No difference

3 Spellberg RD, O'Reilly RJ. Pseudoaneurysm of the left ventricle following mitral valve replacement. Chest 1973; 62: 115-17
6 Tai YK, Mok CK, Chow WH. Left ventricular pseudoaneurysm after replacement of the mitral valve: long-term survival and spontaneous closure. Int J Cardiol 1989; 25:319-52

FIGURE 1. Echocardiographic evaluation of the patient showing a pseudoaneurysmal formation and mitral regurgitation. LV=left ventricle; RV=right ventricle; LA=left atrium; AO=aorta; PMV=prosthetic mitral valve; MR=mitral regurgitation; arrow=pseudoaneurysmal formation.

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