daily during at least 6 months in cases of recurrent pericarditis and during 3 months in cases of first episodes of pericarditis.

Our results were recently reported,¹ and they confirm the findings of Guindo et al.¹ and Rodriguez de la Serna et al.² Eleven patients with recurrent pericarditis were included in the study. Previous treatments (nonsteroidal anti-inflammatory drugs in 10 patients and corticosteroids in one patient) had been unable to prevent a total number of 32 episodes of pericarditis and had induced two cases of severe erosive gastritis and one of corticosteroid-dependence. After colchicine was started, no new recurrences and no side effects occurred during a mean follow-up of 10 months (range, 3–24 months).

In cases of a first episode of pericarditis (19 patients), the ability to treat the acute phase was good when certain etiologies such as postpericardiotomy syndrome, viral pericarditis, and idiopathic pericarditis were concerned. Diarrhea occurred in one patient on the first day, and colchicine was stopped. When a specific treatment was required (e.g., for tuberculosis or pancreatitis), efficacy of colchicine became evident only after initiation of the specific treatment. In these 19 cases, ability to prevent recurrences was less evident because two recurrences occurred during a mean follow-up of 5 months (range, 1–12 months). In the first case, the patient had stopped colchicine after 8 days without medical advice and had experienced a recurrence at 6 months. In the second case, the recurrence happened at 3 weeks, after a transitory improvement of clinical and biological signs during one week.

We agree with Dr. Guindo that a further large, double-blind clinical trial is warranted that compares colchicine to a nonsteroidal anti-inflammatory drug like aspirin. Yet we think that this trial should be performed not only in cases of recurrent pericarditis but also in cases of a first episode of acute pericarditis.

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References

Reply
It was with great interest that we read the letter of Millaire and Ducлюx, and it is greatly encouraging for us to know that our results¹,² have been confirmed by other authors. Furthermore, the results in this study turned out as “dramatic” as our own, as there were no new recurrences in any of the 11 patients with recurrent pericarditis when treated with colchicine.³ However, we cannot expect colchicine to be effective in all patients. Since our paper was prepared³ we have included five new patients and recurrence has just appeared in one despite colchicine treatment.

On the other hand, with regard to Millaire and Ducлюx’s patients,³ it is not surprising that colchicine was not effective in those patients with specific pericarditis (e.g., tuberculosis), because the etiopathogenic mechanism is totally different from idiopathic pericarditis.

Despite the excellent results obtained in the prevention of idiopathic recurrent pericarditis, it should be kept in mind that both Millaire and Ducлюx’s series and our own are open-label. Therefore, as already explained in our article, and in agreement with Adolph’s editorial,⁴ before accepting that colchicine is the panacea for the prevention of recurrent pericarditis, a large double-blind clinical trial is mandatory. We are presently starting two multicenter double-blind placebo controlled studies, involving more than 20 hospitals. Our objective is twofold. First, we hope to confirm our results in those patients who have presented with recurrent pericarditis (secondary prevention). Second, and most important, we hope to determine whether administration of colchicine during 1 month, together with conventional anti-inflammatory treatment, prevents recurrence (primary prevention) in patients with acute pericarditis. If the results are similar to those obtained to date, we should be able to alter the natural history of the disease with a relatively simple, safe, and cheap treatment (1 mg/day colchicine for 1 month), thus avoiding disturbing recurrences in the majority of patients.

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References

Does Reperfusion Induce Myocardial Necrosis?
In his editorial comment,¹ Dr. Miura points out two aspects of our study² that, in his opinion, make the proximal (reperfused) and the distal (nonreperfused) regions of the ischemic territory less than completely comparable with respect to determinants of myocardial infarct size.

Firstly, he points out that the ischemic period was 5-minutes longer in the nonreperfused than in the reperfused region. Also, our methods did not detect the effect of the 5-minute difference in the duration of ischemia on the extent of necrosis.

In dogs 2, 3, and 4 listed in our Table 4, the transmural extents of necrosis progressed respectively by 27.4% in 60 minutes or 2.3% in 5 minutes, by 30.6% in 90 minutes or 1.7% in 5 minutes, and by 16.6% in 60 minutes or 1.4% in 5 minutes. These values explain why, given the considerable natural variability in the dynamics of necrosis, our methods could not detect the 2% progression of necrosis during 5 minutes of additional ischemia. It is, however, important to realize that the magnitude of putative extension of necrosis, attributed to reperfusion injury on the basis of the effect of oxygen free radical scavengers, is in the range of 70% to 120% of the necrosis caused by ischemia alone. Reperfusion injury, resulting in a 2% extension of necrosis, would not be worth the attention it has been receiving in recent years.

Secondly, Dr. Miura states that the subepicardial collateral blood flow “tended to be” higher in the proximal (reperfused) region than in the distal (nonreperfused) region. Collateral blood flow distribution was studied in six dogs. In three dogs the subepicardial collateral blood flow was higher in the
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