leading to platelet aggregation, and platelet–fibrinogen interaction leading to thrombus formation that need to be addressed to fully understand the pathophysiology of arterial ischaemia. Findings such as obtained in this study help provide yet another piece of the puzzle.

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References
variables, indicating that a single coronary lesion resulting in an extensive infarction is sufficient to create the substrate for ventricular tachycardia.

**The methodology**

The study analyses SPECT data to assess residual myocardial ischaemia, and that is appropriate. However, the question arises whether programmed ventricular stimulation is the best method to assess electrical instability. The sensitivity of programmed ventricular stimulation is sufficient to reproduce clinical arrhythmias in patients with an old myocardial infarction who suffer from spontaneous sustained ventricular arrhythmias. But the positive predictive value of programmed stimulation to identify patients who may later develop ventricular arrhythmias is poor (their references[4,6]). In terms of methodology, the major limitation of the study by Paganelli et al. was the inclusion of patients on the basis of results of programmed ventricular stimulation, and not on the basis of spontaneous arrhythmias. Although a statistical relationship was found between residual myocardial ischaemia and inducibility, we do not know how frequently acute ischaemia triggers spontaneous ventricular arrhythmias.

There is no doubt that acute ischaemia may cause ventricular arrhythmias serving simultaneously as the trigger and the substrate. There is also no doubt that acute ischaemia may serve as a trigger of arrhythmias based on other substrates, like a ventricular tachycardia based on a reentry mechanism in a patient with an old myocardial infarction. However, there are differences between the two types of arrhythmias. Acute ischaemic arrhythmias are polymorphic, while reentry arrhythmias based on an infarction substrate are mostly monomorphic. Acute ischaemic arrhythmias do not recur if ischaemia does not recur, while a monomorphic reentrant tachycardia will recur any time a trigger occurs, be it ischaemia, changes in heart rate, electrolyte disturbances, and spontaneous or physician-induced extrasystoles. It is the difference between spontaneous and physician-induced events that forms the core of this discussion.

A study performed at our institution and not referred to in the paper by Paganelli et al. showed that antianginal therapy (medication and revascularization) was not sufficient to prevent recurrences of sustained ventricular arrhythmias in patients with coronary artery disease and an old myocardial infarction[3]. That was shown by prospectively giving antianginal medication and implanting an automatic defibrillator in all patients with sustained ventricular arrhythmias who underwent bypass surgery. After a mean follow-up period of about 2 years, almost half of the patients received an appropriate shock from their device. In other words, should a cardioverter-defibrillator not have been implanted in these patients, 50% would have died because of a recurrence of their ventricular arrhythmia. Thus, in patients known to have a healed myocardial infarction, correction of all causes of apparent and silent ischaemic events leaves one or two patients exposed to the risk of dying suddenly from arrhythmias unrelated to acute ischaemia.

We have therefore to conclude that the study by Paganelli et al. shows a statistical association between residual myocardial ischaemia and inducibility of sustained ventricular arrhythmias during programmed ventricular stimulation after a recent myocardial infarction. Unfortunately, that is not the same as proving a cause-to-effect relationship. Coronary disease and myocardial infarction have three pathophysiological manifestations: ischaemia, mechanical dysfunction and electrical instability. The three are closely related and each patient requires a complete therapeutic approach to all three manifestations of the disease. One should not conclude that antianginal therapy alone is sufficient to prevent late ventricular arrhythmias after myocardial infarction.

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