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risk' group and a mere 5% in the control group acute events are likely to have been very rare.

It is easy to concur with the authors that accidentally detected lesions that have recently progressed can and should be dilated without a significantly increased risk of recurrence. The community of angioplasty operators is grateful for these data, endorsing a long established practice. However, the paper does not minimize the acute problems or increased likelihood of restenosis when it comes to plaques dilated at the very moment of progression. The authors are far too experienced not to have conveyed this caveat clearly in the discussion.

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Arrhythmogenic right ventricular disease, dysplasia and cardiomyopathy

See page 1717 for the article to which this Editorial refers

This issue contains an interesting article on long-term changes in the ECGs of patients with ventricular arrhythmias originating in the right ventricle[1]. The authors describe their patients as having arrhythmogenic right ventricular disease but quote extensively references concerning arrhythmogenic right ventricular dysplasia. In fact the term arrhythmogenic right ventricular disease refers to a larger group of patients than the classical arrhythmogenic right ventricular dysplasia, which is clearly identified by its specific histological structure rarely available in a common clinical series[2].

In addition to the presence of ventricular tachycardia (of unknown origin) the authors' criteria for inclusion are based on contrast angiography. This is also an interesting approach in clinical diagnosis since angiography is strongly dependent on the underlying anatomical structure and has generally been considered as the 'gold standard' for the diagnosis by showing evidence of segmental abnormalities[3,4].

In addition to this approach the authors have included in their selection the so-called 'more diffuse forms'. We are concerned that these more diffuse forms may confuse the issue. Diffuse dilatation of the right ventricle alone is not a sign of arrhythmogenic right ventricular dysplasia. It could be the result of different forms of idiopathic cardiomyopathy mostly involving the right ventricle without replacement of myocardial fibres by fatty tissue[5]. Even segmental abnormalities could be the result of a localized form of healed myocarditis, ischaemia or cardiomyopathy[6]. Only specific segmental abnormalities, such as outpouching bulges, microaneurysms, or deep fissures in the infundibulum or at the apex are markers of arrhythmogenic right ventricular dysplasia. However, such comments are not of academic importance since the prognosis of these various subgroups may be different. For instance, the development of myocarditis recently demonstrated in a subgroup of cases superimposed on arrhythmogenic right ventricular dysplasia could lead to progressive modification of the ECG that is not the result of the dysplastic phenomenon (replacement of right ventricular musculature by adipocytes and fibrous tissue) but a different phenomenon that could affect both right and left ventricles[7]. Therefore, the changes observed in some of the cases may not be the result of evolution of the basic disease, but the consequence of a different phenomenon acting.
as a complication of arrhythmogenic right ventricular dysplasia. Further studies are obviously necessary to better identify these subgroups.

The ECG may be an interesting diagnostic tool with which to identify the disease in a screening process, to delineate its clinical forms, to follow its evolution in order to identify its complications and finally to have a better evaluation of its prognosis.

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Endogenous opioids, catecholamines and vasovagal syncope

See page 1729 for the article to which this Editorial refers

The term vasovagal syncope was coined by Lewis in 1932[1] when he described fainting attacks attributable to hypotension and bradycardia. A few years later, Barcroft and colleagues[2] studied the haemodynamics of syncopal attacks induced in healthy subjects by bleeding or placement of tourniquets to the legs. Before the onset of the faint, heart rate increased and so did vascular resistances. Blood pressure showed very few changes in spite of a significant decrease in cardiac output. The onset of syncope was abrupt. It was caused by a sudden, profound fall in arterial blood pressure related to sudden vasodilatation and accompanied by bradycardia.

More than half a century later, the mechanisms responsible for initiating a vasovagal syncope are still poorly understood. Before the faint, there generally is evidence of stimulation of the sympathetic system which may result from acute changes in circulating blood volume or emotional stress. Why does this sympathetic stimulation abruptly give way to vasodilatation and bradycardia?

Several different hypotheses have been put forward[3,4]. An abnormally sensitive response to stimulation of arterial baroreceptors has been suggested. An abnormally sensitive carotid sinus reflex has, for example, been observed in a large proportion of patients with unexplained syncope and the carotid sinus syndrome may represent one form of vasovagal attacks. Stimulation of areas within the hypothalamus may result in tachycardia, hypertension and muscle vasodilatation, but, in the experimental animal, this response never develops into a vasovagal attack. The most favoured hypothesis nowadays is that vasovagal attacks are caused by the Bezold-Jarisch reflex: stimulation of (hypersensitive?) cardiac ventricular receptors by powerful contractions on a nearly empty ventricle may trigger the profound depressor response. The explanation, although plausible, remains controversial[5]. It seems for example, that relatively few ventricular receptors are excited during haemorrhage or occlusion of the caval veins in the cat and the reflex responses to such mechanical stimulation are not enhanced by sympathetic stimulation. Moreover, if the hypotension was simply a reflex phenomenon there is no reason why it should persist after the stimulus has disappeared i.e. after bradycardia and hypotension have developed. The demonstration of the presence of vasovagal reactions during passive upright tilt in