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-Jarich 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
Heart transplant recipients without evidence of cardiac reinnervation is another reason to challenge the ventricular baroreceptor hypothesis in vasovagal syncope. Finally, the group of Morita et al. made the interesting observation that during hypotensive haemorrhage in unanaesthetized animals, there was a decrease in afferent renal nerve activity. This decrease was not prevented by vagal denervation which seems to indicate that it does not depend on stimulation of cardiac receptors. On the other hand, the decrease in afferent renal nerve activity was prevented by blockade of opiate receptors by naloxone. The latter observation, among others, raised the question of a possible role of endogenous opioid mechanisms in vasovagal attacks.

In 1994, Wallbridge and colleagues demonstrated an increase in plasma /?-endorphin levels prior to the onset of syncope in subjects with vasovagal attacks induced by tilt-testing. In a follow-up to these preliminary results, the same group reports in this issue on the failure of naloxone to modify the vasovagal response in well characterized subjects with frequent spontaneous syncope and a reproducible vasovagal response to tilt-testing. Their observation leads to the conclusion that endogenous opioid mechanisms are not an important trigger for vasovagal events in humans, but probably relate to co-release with adrenocorticotrophic hormone from the pituitary in response to stimulation of low-pressure atrial baroreceptors by relative central hypovolamia.

I found this paper interesting for three different reasons: (1) In spite of its frequency and of its easier diagnosis since the introduction of head-up tilt-testing, vasovagal syncope remains a condition which is poorly understood. The hypothesis implying that the mechanism of syncope may be similar to the Bezold-Jarich reflex is undoubtedly attractive but is contradicted by several experimental or clinical observations. Further research is justified in that area. (2) Opioids may play an important role in modifying baroreceptor physiology and substantial experimental literature supports the concept of a sympathoinhibitory action of these agents. Their effects, however, are not easy to investigate since opioids do not act in isolation but rather as neuromodulators. They surely deserve further attention (3) The discussion on the mechanisms of the vasovagal syncope raises renewed interest in the fascinating and broad discipline of ‘neurocardiology’. The complexity of the numerous electrophysiological mechanisms and neuroendocrine systems involved in cardiovascular regulation also constitutes an attractive area for future research.

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Heart failure patients: why do they fatigue, how do they get better?

See page 1678 for the article to which this Editorial refers

The study of heart failure has changed. First we assumed that poor myocardial contractility causes all the symptoms, and that treatments that increased contractility would both make patients feel better and live longer. This was a mistake. Later the importance of the body's reflex responses to the ventricular dysfunction were appreciated and their importance...