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was unsatisfactorily low. This means that more trials are still needed to define the actual prognostic value of blood pressure in the very old. It is likely that only the results of randomized controlled trials of adequate size, aimed at assessing the risks and benefits of lowering blood pressure in hypertensive subjects over the age of 80, will provide us with convincing evidence on whether very old subjects with elevated blood pressure should or should not be treated.

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Primary ventricular fibrillation: a reason to be cautious

See page 919 for the article to which this Editorial refers

It has been stated that the prognosis of ventricular fibrillation in the initial stages of acute infarction is always favourable [1,2]. This lethal arrhythmia was estimated to occur (in the pre-thrombolytic era) in about 5% of all infarctions after admission [3]. Because immediate treatment with defibrillation resulted in a short-term favourable prognosis in the resuscitated patient, strategies were developed to create a safer environment for patients in the early stages of the infarction. The establishment of coronary care units offered continuous ECG monitoring and availability of defibrillators. As most deaths due to ventricular fibrillation occur before admission to hospital, efforts were undertaken to reduce the delay to admission after the onset of chest pain. Mobile units, capable

of defibrillating when necessary, were to be used to transport the patient to hospital.

It is now evident that early reperfusion by thrombolytic agents reduces early death and the probability of in-hospital fibrillation, as demonstrated by several multicentre trials on thrombolytic therapy [4]. Therefore, early thrombolytic therapy or angioplasty are important next steps, even when this does not necessarily reduce the incidence of primary ventricular fibrillation [4].

This takes us to the question, why do patients with acute infarction have ventricular fibrillation in the early stage? Ventricular fibrillation, generally considered the result of random reentry, has no obvious other reason in this condition than the acute infarction, and occurs not as a result of shock or heart failure [3]. The electrical environment that favours this arrhythmia is created by the dynamics of

the infarction, and its interplay with the remote myocardium. This leads to inhomogeneity of recovery of excitability. In the early stages of infarction, the occlusion of the artery, with the subsequent ischaemia and beginning of necrosis, or the reocclusion of an open artery might be responsible for ventricular fibrillation. However, reperfusion can also herald the onset of ventricular fibrillation, but this idea was not corroborated by recent data with angiographic studies^[4]. Early ventricular fibrillation is not preceded by ventricular tachycardia based upon a fixed reentry circuit, as often seen when a patient has a cardiac arrest subsequent to an old infarction. Personally, we have never seen a fast, monomorphic ventricular tachycardia in an acute infarction, unless the patient had a scar from a previous infarction. Acute ischaemia, remote from the infarcted region, is another possible explanation for ventricular fibrillation, and has been used as an excellent animal model to induce this arrhythmia^[5].

However, clinically important ischaemia, not related to the infarct artery, is infrequently detected, even when its presence is aggressively investigated. Remote ischaemia, and possibly hibernating myocardium, play a role in the pathogenesis of late and exercise-induced ventricular fibrillation, and relevant stenoses must be corrected whenever possible^[2,6]. Late fibrillation is more common when congestive heart failure is involved. Electrolyte disturbances, polypharmacy and haemodynamic factors can play a role in triggering the arrhythmia. Such arrhythmias have a worse prognosis than the early form, with an in-hospital mortality of up to 75%. Primary ventricular fibrillation has been associated with low potassium, but this may be an epiphenomenon^[7]. The idea that hypertrophied, non-infarcted tissue plays a role in the pathogenesis is of interest, and may have repercussions in the prognosis of these patients^[8].

It has been said that the incidence of early ventricular fibrillation has been decreasing over the last few years, 'because we are doing the right things' (personal communication). This included (in the pre-thrombolytic era, which still persists for more than 50% of all patients, as they do not receive thrombolysis) effective pain relief, sedation, beta-blockers and possibly nitrates. Therefore, the Danish study from Jensen *et al.* in this issue is still relevant, even though no thrombolytics were administered (as the study was conducted in the years 1977 to 1988)^[9]. Primary ventricular fibrillation occurs in the same proportion as mentioned above in this thrombolytic era (5 to 8% in the GISSI-2 trial^[10]).

It has always been accepted that early ventricular fibrillation ('primary ventricular fibrillation') has no influence on prognosis after hospital discharge

following acute myocardial infarction. Supportive data in this field has been overwhelming. Indeed, when correctable mechanisms are the reason for ventricular fibrillation, one could support the hypothesis that the prognosis of a patient is unaltered compared to his companion in the hospital who did not have ventricular fibrillation. It has to be stressed, however, that other authors described an in-hospital mortality of 23% after primary ventricular fibrillation^[3]. Death came early in this study. It was associated with age, a higher incidence of previous infarctions, and with heart block. Recurrent ventricular fibrillation was also observed in 17%, in spite of antiarrhythmic therapy. Nevertheless, the long-term survival after hospital discharge (and this is the key point) was comparable with those without early ventricular fibrillation (85% over 3 years).

Why is the Danish study so different? First, it has to be said that the high mortality incidence is early (within the first 60 days). This is in accordance with previous data showing high in-hospital mortality. It can be hypothesized that the early post-infarction phase is characterized by persistent instability, not only from the ischaemic point of view, but also from the rhythmological point of view.

When primary ventricular fibrillation accompanies the infarction, an aggressive approach is necessary: ischaemia correction, beta-blockade, unloading of the ventricles with ACE-inhibitors, and potassium repletion have to be provided. Perhaps a full, non-invasive rhythmological investigation is necessary, followed by invasive testing whenever doubt persists about which arrhythmia mechanism was responsible for the ventricular fibrillation.

A real bias in the study of Jensen *et al.*, however, is the fact that antiarrhythmic drugs were prescribed for 'Lown III-IV' in the patients with primary ventricular fibrillation. This may be why mortality was increased. The fact that the increased mortality was only increased within the first 2 months fits in the recent analysis of CAST data, showing that new ischaemic events probably account for the higher mortality in patients treated with antiarrhythmic agents^[11,12]. Therefore, it must be concluded that after primary ventricular fibrillation reperfusion should be achieved, and the presence of an open artery verified. When other cardiac abnormalities exist (infarction, hypertrophy) further measures are warranted, but additional antiarrhythmic treatment (with drugs) is as obsolete in this indication as for all other asymptomatic patients after infarction.

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Angina pectoris and the personality factor: the relevance of psychosocial factors in myocardial ischaemia

See page 911 for the article to which this Editorial refers

Psychosocial factors have long been known to play a role in determining the clinical profile of patients with ischaemic heart disease. 'What is in my brain, that is in my heart' (Shakespeare). Studies have confirmed that personality, stressful life events, work-related and social factors can all modulate the clinical manifestations of ischaemic heart disease. The paper by Billing *et al.*^[1] in the present issue provides interesting information along these lines.

Using structured psychological interviews, the authors evaluated 767 patients from the Angina Prognosis Study in Stockholm (APSIS) and 50 matched healthy controls. They found that patients with stable angina had experienced significantly more stressful events, and had more psychosomatic symptoms than healthy controls. These findings extend to anginal patients, observations obtained to date mainly on patients with acute myocardial infarction. Furthermore, since the study group comprised approximately the same proportion of male and female patients, the

authors were in a position to assess the role of gender on the psychosocial traits of patients with angina.

Females rated less type A behaviour and hostility than males. Type A people are characterized as highly competitive, ambitious, and in constant struggle with their environment. By contrast, the type B personality is defined by greater passivity and is less disturbed by environmental stress. Interestingly, similar psychological features have been described in female patients with syndrome X (i.e. exercise-induced angina and ischaemic-like ST segment changes with normal coronary arteries) by Corlando *et al.*^[2]. In their patients, the authors found a higher stressful event score preceding the onset of anginal episodes, an excess of family and social difficulties and inhibition in the expression of emotions. They also found low scores for irritability, hostility and neuroticism — again suggesting that the personality and behavioral substrate of ischaemic heart disease can be very different in women and men.

In the study of Billings *et al.*, patients with typical stable angina were selected. Chest pain is not only an important clinical symptom, but is also a