Plasma atrial natriuretic peptide in patients with acute myocardial infarction: effects of streptokinase

Sir,

Phillips et al studied plasma concentrations of atrial natriuretic peptide after acute myocardial infarction (1989;61:139–43). They did not report a significant correlation between plasma concentrations of atrial natriuretic peptide and peak concentrations of creatine kinase, which is an index of the severity of infarction. In their study, blood was sampled for the determination of atrial natriuretic peptide on the morning after admission, which was, as we understand, at least 0–32 hours after the onset of symptoms.

The exact timing of blood sampling for atrial natriuretic peptide is of great importance. We measured atrial natriuretic peptide concentrations at fixed times during 48 hours in 38 patients who were admitted to the coronary care unit within 4 hours 25 minutes after the onset of symptoms.1 Three hours after admission, the mean atrial natriuretic peptide concentration was significantly lower than it was on admission. Thereafter, atrial natriuretic peptide concentrations rose till 15 hours after admission. Both the atrial natriuretic peptide value on admission and the individual mean atrial natriuretic peptide value during the study period of 48 hours were significantly correlated with the maximum creatine kinase value.

We agree with the hypothesis of Phillips et al that acute myocardial dysfunction after myocardial infarction, by raising atrial pressures, causes a release of atrial natriuretic peptide from atrial storage granules. The ensuing decrease in atrial natriuretic peptide concentrations found in our study may possibly be attributed to depletion of these storage granules. At this stage, circulating atrial natriuretic peptide concentrations will decrease despite increased intracardiac pressures. Subsequently atrial natriuretic peptide is synthesised at a greater rate causing its concentrations in the blood to rise again. Our results imply that the correct interpretation of atrial natriuretic peptide values after acute myocardial infarction depends on the timing of blood sampling.

This letter was shown to the authors, who reply as follows:

Sir,

We found that plasma concentrations of atrial natriuretic peptide were higher in patients with acute myocardial infarction who were not treated with thrombolysis than in similar patients admitted with non-ischaemic chest pain and patients with myocardial infarction treated with streptokinase. Patients with ischaemic chest pain had intermediate concentrations of atrial natriuretic peptide. The finding of raised plasma atrial natriuretic peptide in the acute stages of myocardial infarction was confirmed by Tan et al and others.1