sinoatrial node still gives rise to discussion. Anderson and Ho indicate a discrepancy between the depicted location of the sinoatrial node in the schematic dorsal view of the human atria (their Fig. 1) and its description relative to the sulcus terminalis.

The crista terminalis is sometimes indicated on the outer surface of the right atrium by a shallow groove, the sulcus terminalis or terminal groove[11]. In their schematic drawing, Anderson and Ho depict the atria in a postero-superior view. The sinoatrial node is depicted in the sulcus terminalis which is curved convex towards the right atrial appendage. However, in agreement with the drawings of the posterior view of the heart in Anderson’s and Becker’s unrivalled colour atlas of Cardiac Anatomy[2], the sulcus terminalis makes a convex curve away from the right atrial appendage (compare section line 1, Fig. 1 of our original article).

In all our preparations, the human sinoatrial node was located on the epicardial side superior to the crista terminalis (and thus superior to the sulcus terminalis). We therefore showed that the sinoatrial node preparation should be dissected parallel to and inferior to the crista terminalis (section line 1, fig. 1 of our original article).

Our statement that the sinoatrial node is located 'beneath the epicardium of the sulcus terminalis' may be misinterpreted. We meant to describe a location on the epicardial side superior to the sulcus terminalis rather than a location in the sulcus terminalis. Using the described and depicted section lines we dissected over 100 preparations. The sinoatrial node or the major part of the sinoatrial node was included in all these preparations. Therefore, based on our experience, we consider that the depicted section lines will not cause any disappointment.

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Perfusion heterogeneity in syndrome X
We refer to the paper by J. G. Meeder[11] describing perfusion heterogeneity in syndrome X. Their non-invasive approach using positron emission tomography is interesting.

For the detection of perfusion heterogeneity in syndrome X, as well as for the follow-up of the effect of therapy, another non-invasive approach can be used, namely BSPM (Body Surface Potential Mapping). This method is substantially cheaper.

We present an example of BSPM in a 42-year-old woman who had suffered for several years from
atypical chest pain on exercise as well as at rest. Her stress test was positive, but she rejected coronary angiography. She was eventually seen by a psychiatrist.

Her BSPM examination revealed extensive ischaemia on repolarization isopotential maps probably of syndrome X origin.

Nitrates and beta-blockers were prescribed and after 2 weeks of this therapy there was substantial regression of her condition. However, regression of ischaemia in the coronary microcirculation was gradual, but within a year these signs disappeared almost completely.

For the BSPM, we used 80 unipolar electrodes placed at regular intervals on the anterior and posterior chest wall. Figure 1 represents a normal repolarization map, with the anterior chest wall situated on the left half of the map and the posterior wall on the right. For better orientation, six standard precordial electrodes, V1 to V6, are depicted in the form of small black squares.

Figure 2 shows the BSPM findings before treatment and Fig. 3 one year later. The substantial improvement is quite apparent.

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Myocardial reperfusion injury: experimental evidence and clinical relevance

We agree with the general conclusions that reperfusion injury is a clinically relevant phenomenon and that continuing research directed at the manifestations of myocardial reperfusion injury may have considerable therapeutic implications[1]. However, we would question the view that reperfusion arrhythmias are of small significance in patients undergoing reperfusion therapy.

The incidence of reperfusion arrhythmias in man is low compared to animal studies for the reasons pointed out by Hansen, in particular the duration of antecedent ischaemia[2]. The major focus of treatment of acute myocardial infarction these days is rapidity of opening occluded coronary arteries, which is likely to increase the number of reperfusion arrhythmias on the basis of the bell-shaped curve. However, as Hansen indicates, most large-scale studies of intravenous thrombolysis for acute myocardial infarction have shown a lower incidence of ventricular fibrillation in the active treatment group compared with placebo or control, even though thrombolysis is associated with a far greater likelihood of reperfusion.

Before we dismiss reperfusion arrhythmias following thrombolysis as clinically unimportant, it should be pointed out that most thrombolytic trials were not designed primarily to assess the impact of thrombolysis on serious rhythm disturbance, particularly during the early stages of hospitalization. Data are generally not available or, more commonly, have been pooled over a long time period. One trial that assessed the effects of thrombolysis on early and late rhythm disturbance was the German APSAC trial comparing intravenous streptokinase with heparin, given within 4 h of the onset of symptoms[3]. In the first 24 h of treatment the incidence of ventricular fibrillation in the thrombolytic group was 6-8% compared with 4-6% in the heparin group, but the difference was lost after the first 24 h (2.5% vs 2.7%), giving an overall incidence of 8-0% and 7-3% respectively. Similarly, in the ISAM thrombolytic trial, the incidence of ventricular fibrillation in patients receiving thrombolysis was 3-6% in the first 3 h after start of treatment compared with 3-4% in the placebo arm, falling to only 0-7% in the thrombolytic arm compared...