The common AC electrocardiograph, in the case of subepicardial or transmural ischaemia, where there is no healthy myocardial tissue between the recording electrodes and the afflicted myocardium, records ST-segment elevation. If the ischaemia is subendocardial, in which case healthy, non-ischaemic myocardium exists between the recording electrode and the ischaemic myocardium, the electrocardiograph records ST-segment depression.

It is well known that ST-segment elevation enables more accurate location of the ischaemic wall than does ST-segment depression. Even following experimental occlusion of the anterior descending branch and recording from the epicardial surface, ST-segment depression is not a reliable indication of the severity of the reduction in blood supply.1

Here we describe a case with ST-elevation on the anterior wall leads of the ECG, which, however, later proved to be due to complete occlusion of the proximal right coronary artery.

Case report

A man aged 45 years, a smoker with moderate hypertriglyceridaemia and exceptionally low HDL, moderately obese, came to the outpatients’ department eight hours after the onset of epigastric pain with reflection to the wrists, accompanied by sweating and weakness. This had already been linked with an ECG picture showing around 3 mm ST-segment elevation in leads V1-V4, a sharp-peaked T, Qr in III, aVF, with 0.5 mm ST-segment elevation in III and incomplete right bundle branch block (Figure 1). Haemodynamically he was Killip I. Because of the disappearance of the symptoms and the time elapsed, conservative treatment was decided upon. One hour later, however, the problem recurred, with a clear increase in the height of the T wave on the anterior wall leads.
wall leads and a negative T on I, aVL, with no other changes on the inferior leads. In view of this the patient was taken to the haemodynamic laboratory where, following initial catheterisation of the left coronary net, complete occlusion of the proximal right coronary artery was discovered, with co-dominance of the left and right coronary net (Figure 3). The obstruction was opened successfully and a stent was implanted (Figure 4, 5). The symptoms receded, with disappearance of the elevation on the precordial leads and restoration of the T wave on I and aVL (Figure 2). Maximum CPK was 1660 (MB fraction: 147). The patient’s subsequent hospitalisation was uneventful.

**Discussion**

The right ventricle is involved in around 25% of inferior myocardial infarctions. The most sensitive indication of occlusion of the proximal right coronary artery is lead V4R, which has a sensitivity of 82-100% and a specificity of 68-77%. The appearance of a greater elevation in lead III than in lead II strongly implicates the right coronary artery, with occlusion of the proximal or middle part (sensitivity 89% and 80%, respectively), while the coexistence of ST-segment elevation in V1 (without elevation in V2) – five out of 69 total inferior infarctions in one series of patients – is indicative of occlusion of the proximal right coronary artery with 100% specificity.7

Our patient had no ST-segment elevation on the inferior wall leads (apart from 0.5 mm in III), an observation that initially suggested an anterior wall infarction and contributed to the decision for invasive treatment. In another series of patients similar to ours, with ST-segment elevation on the anterior wall leads and no significant elevation on the inferior leads, coronary angiography revealed occlusion of the proximal right coronary artery in 7% of cases.8 When the right coronary artery is occluded these anterior wall elevations decrease in magnitude from V1 to V5, in contrast to occlusion of the anterior descending branch, which is associated with ST-segment elevation ≥1 mm most commonly in V2 (sensitivity 91-99%), and less often, in decreasing order of frequency, in V3, V4, V5, aVL, I, V1 and V6.9

Two damage currents interact after acute central occlusion of the right coronary artery. One originates from the necrosis in the inferior wall, while the other is created by the concomitant damage in the right ventricle. In most cases the former predominates, because of the greater tissue loss in the corresponding region. This results in neutralisation of the ST-segment elevation in lead III.
elevation on the left precordial leads, which one would expect to see on account of the right ventricular damage. When, however, the extent of the damage in the inferior wall is small or absent, the elevations in the anterior wall are reflected back more easily. Rare cases of isolated right ventricular infarctions on a substrate of a non-dominant right coronary artery, which supplies no blood at all to the left ventricular wall, show ST-segment elevation exclusively on the anterior wall leads, with no ECG changes in the inferior leads.

The anatomical region “seen” by lead V1 (right paraseptal) receives “double perfusion” from septal branches of the anterior descending and from the conus artery. This is the reason that in anterior infarctions we do not see elevation in V1. In a third of cases, however, as an anatomical variation, the conus artery is so small that it makes no appreciable contribution. In such cases, acute occlusion of the left anterior descending branch is associated with elevation in V1, making it difficult to determine the culprit vessel based on the particular way the anterior wall elevations are distributed, as described above.

Thus, lead V1 reveals lesions in the right ventricle when the damage to the inferior wall is limited, whereas it is one of the leads affected, in about one third of cases of anterior wall infarction, when there is a hypoplastic conus artery.

References


Figure 3. Right coronary artery before opening of the proximal total occlusion.

Figure 4. Right coronary artery after opening of the proximal total occlusion.

Figure 5. Anatomically intact left coronary artery.

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