Electrocardiographic changes in acute coronary syndromes

Introduction

Acute coronary syndromes (ACS) remain one of the greatest global causes of mortality and morbidity. Rapid diagnosis and treatment are paramount. Despite scientific advances, the electrocardiogram (ECG) remains the cornerstone of ACS diagnosis, classification, risk stratification and management. In 2002, an expert committee of the American College of Cardiology and the European Society of Cardiology redefined ACS. The new definition incorporated the new diagnostic markers of myocyte necrosis, troponins, and also redefined the electrocardiographic criteria for ACS diagnosis (Alpert et al, 2000).

The old classification of myocardial infarction (MI) relied upon the presence or absence of Q waves on the final ECG, which was considered a broad surrogate of infarct severity. The new classification depends upon the presence or absence of ST segment elevation on the presenting ECG.

Figure 1 shows a schematic for the clinical diagnosis of ACS. Compared with the old classification this has important ramifications for immediate treatment of ACS in the emergency department as well as future management and prognosis. Patients with acute ST elevation MI (STEMI) should receive urgent reperfusion by thrombolysis or primary angioplasty unless contraindicated (Antman et al, 2004), whereas patients with non ST elevation MI (NSTEMI) or unstable angina are conventionally treated medically in the first instance with subsequent consideration of early invasive investigation and treatment (Bhatt et al, 2004). This article specifically focuses on morphological ECG changes during ACS; alterations in rhythm are not considered.

Not only does the ECG diagnose ACS and guide management, but it is able to risk stratify for the occurrence of future cardiac events, and assess the vulnerable arterial territory (Savonitto et al, 1999; Collinson et al, 2000; Hyde et al, 2003).

Dr Bejal Pandya *is Specialist Registrar in the Cardiology Directorate at The East Surrey Hospital and* **Dr Philip C Strike** *is Specialist Registrar in Cardiology, The Heart Hospital, University College London, London W1M 8PH*

Correspondence to: Dr PC Strike

Location of the infarction from the surface ECG

The surface leads of the ECG can identify areas of ischaemic myocardium during ACS. This in turn can be related back to the coronary anatomy and suggest the vessel in which the culprit lesion is located. (*Table 1*). Location of the ACS is related to prognosis and the extent of changes seen proportional to the amount of myocardium at risk (Topol and Van de Werf, 2002).

Types of ECG changes in ACS

ST elevation myocardial infarction

With the exception of life-threatening rhythm problems, the identification of ST segment elevation is the most important electrocardiographic information on the presenting ECG of an ACS patient. ACS arises in most cases from erosion or rupture of coronary atherosclerotic plaque and subsequent thrombus formation causing complete or incomplete coronary occlusion (Davies and Thomas, 1985). The degree and extent of ST elevation broadly correlates with the volume of myocardium at risk and requires prompt revascularization to open the occluded artery and minimize myocardial damage.

Figure 1. The spectrum of acute coronary syndromes according to electrocardiographic and biochemical markers of myocardial necrosis in patients presenting with acute cardiac chest pain. Reproduced with permission from Grech and Ramsdale (2003).

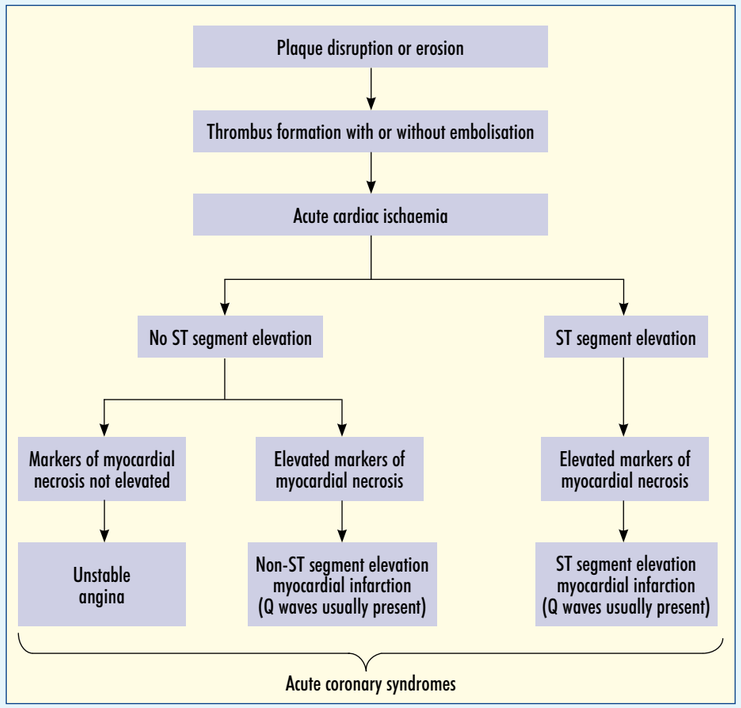


Table 1. Anatomical and electrocardiographic correlations in acute coronary syndromes

Anatomical territory	Coronary artery	Leads affected
Anterior	Left anterior descending artery or circumflex artery	V2–V4
Anterolateral	Left anterior descending artery	V4–V6, I, AVL
Lateral	Left anterior descending artery or circumflex artery	I, II, AVL, V6
Inferior	Right coronary artery or circumflex artery	II, III, AVF
Inferolateral	Right coronary artery or circumflex artery	II, III, AVF, AVL, V4–V6
Inferoseptal	Circumflex artery	II, III, AVF, V1–V3
Posterior	Circumflex artery or right coronary artery	V1, V2

STEMI is defined as new or presumed new ST elevation in two or more contiguous leads of >0.2 mV in V1, V2 and V3 and 0.1 mV in other leads or new onset left bundle–branch block (LBBB) (Alpert et al, 2000). This is classically convex in shape in STEMI. With prompt treatment it is possible for the ECG to normalize once more after recanalization of the occluded artery but a wide spectrum of ECG findings may be seen including T wave inversion, Q wave formation, loss of R wave amplitude, and persistent ST elevation (which may suggest ventricular aneurysm formation). *Figure 2* shows a normal ECG, and *Figure 3* shows STEMI in the anterior and lateral leads. At angiography this correlated with proximal occlusion of the left anterior descending coronary artery.

Left bundle–branch block

In this condition the lateral left wall and the septum of the left ventricle are delayed in depolarization. This results in a widened QRS complex throughout all leads, and can render the ECG difficult to interpret. In the presence of a clinical history suggestive of ACS it should be treated as a new occurrence unless there is information or examination findings to suggest otherwise. The changes associated with ischaemia such as T wave inversion and ST depression can be obscured by the LBBB. It is possible, however, to identify acute changes on a background of old LBBB, with differences in ST elevation or depression changing the pre-existing pattern.

Figure 2. An example of a normal electrocardiogram.

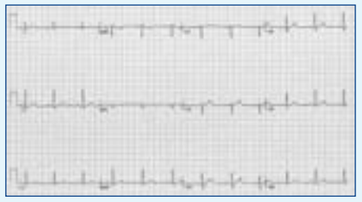


Figure 3. Electrocardiogram showing anterolateral ST elevation.

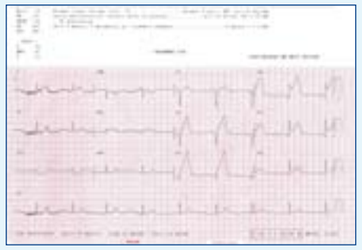
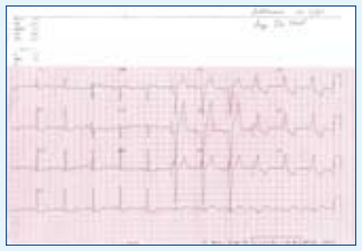


Figure 4. Electrocardiogram showing left bundle–branch block.



The diagnosis of acute MI in the context of LBBB has been described by Sgarbossa et al (1996). *Figure 4* shows an example of LBBB, in this case there is ST elevation anteriorly as well. The patient with a paced ventricular complex also presents a significant challenge, as the paced QRS will have evidence of LBBB. This problem may be overcome by reducing the pacing rate to enable non-paced QRS complexes to be visible, although this should be performed very cautiously as the patient may be compromised at slower paced rates or have no underlying escape rhythm.

Non-ST elevation myocardial infarction

There are several ECG changes that may be seen; these changes may be dynamic and repeated ECGs may be useful in the diagnosis in conjunction with troponin measurement. The changes seen include:

1. ST depression, here there is sagging of the ST segment below the isoelectric line. *Figure 5* shows an example of anterior ST depression in a patient with critical left anterior descending coronary artery disease
2. T wave inversion. *Figure 6* shows anterior T wave inversion in a patient following an ACS caused by severe left anterior descending artery disease

Figure 5. Electrocardiogram showing ST segment depression.

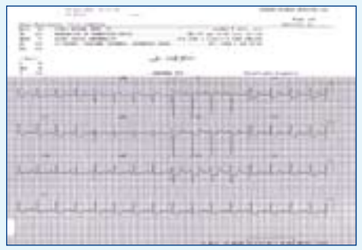


Figure 6. Electrocardiogram showing T wave inversion.

3. Normal ECG. It is possible to sustain a NSTEMI without ECG changes. ACS involving the circumflex artery is most likely to be electrocardiographically 'silent'. Other changes such as hyperacute T wave changes are particularly important as these may be the earliest abnormality seen and may progress to ST elevation so repeat ECGs are necessary.

The magnitude of ECG changes seen is linked to prognosis and also severity of coronary disease (Kaul et al, 2001, 2003). These patients represent a group at very high risk of developing further cardiac events and patients should be considered for early invasive investigation and revascularization (Yusuf et al, 1998; Collinson et al, 2000; Diderholm et al, 2002; Wiviott et al, 2004). Changes such as ST depression may be seen in reciprocal leads to ST segment elevation and can reflect a large threatened territory or widespread severe coronary artery disease. The ECG should not just be used for diagnosis, but also for assessing the efficacy of treatments such as thrombolysis.

Unstable angina is diagnosed from dynamic ECG changes as described above occurring in the absence of elevation of markers of myocyte necrosis.

Caveats

There are several conditions that can make ECG interpretation difficult and these should always be considered if appropriate. These include paced rhythms, the presence of left ventricular hypertrophy and strain, changes caused by previous ACS, previous coronary artery bypass surgery, use of digoxin and a wide variety of normal variants such as high ST segment take off. There is also poor representation of the right ventricle as well as the posterior and apical segments of the left ventricle (Zimetbaum and Josephson, 2003). It is also important to bear in mind that the initial ECG is diagnostic of MI in only 50% of cases so serial ECG examinations should be performed in addition to measurement of serum markers.

Although the ECG changes outlined above are indicative of ACS, other cardiovascular disorders may present with similar findings and it must be stressed that the diagnosis requires careful history taking, examination and appropriate use of clinical investigations. These other conditions include pericarditis, pulmonary embolism and aortic dissection. Any doubts should be discussed with specialist cardiac centres immediately.

Conclusions

The ECG plays a vital role in the diagnosis and the treatment of both acute and chronic coronary disease. It can guide the clinician in risk stratifying the patient and allow, in conjunction with serum cardiac enzymes, diagnosis of an ACS with increased sensitivity and specificity and facilitation of early and potentially life-saving treatment. BJHM

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Conflict of interest: none.

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KEY POINTS

- The electrocardiogram (ECG) is a quick, simple and non-invasive test.
- The ECG guides immediate medical therapy based on the presence or absence of ST elevation.
- The ECG provides anatomical and prognostic information.
- The ECG must be repeated during acute coronary syndromes to assess response to treatment.