ABC of clinical electrocardiography
Acute myocardial infarction–Part II

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This article describes the association of bundle branch block with acute myocardial infarction and the differential diagnosis of ST segment elevation.

Bundle branch block

Acute myocardial infarction in the presence of bundle branch block carries a much worse prognosis than acute myocardial infarction with normal ventricular conduction. This is true both for patients whose bundle branch block precedes the infarction and for those in whom bundle branch block develops as a result of the acute event. Thrombolytic treatment produces dramatic reductions in mortality in these patients, and the greatest benefits are seen in those treated early. It is therefore essential that the electrocardiographic identification of acute myocardial infarction in patients with bundle branch block is both timely and accurate.

Left bundle branch block

Left bundle branch block is most commonly seen in patients with coronary artery disease, hypertension, or dilated cardiomyopathy. The left bundle branch usually receives blood from the left anterior descending branch of the left coronary artery and from the right coronary artery. When new left bundle branch block occurs in the context of an acute myocardial infarction the infarct is usually anterior and mortality is extremely high.

The electrocardiographic changes of acute myocardial infarction can be difficult to recognise when left bundle branch block is present, and many of the conventional diagnostic criteria are not applicable.

Abnormal ventricular depolarisation in left bundle branch block leads to secondary alteration in the recovery process (see earlier article about bradycardias and atrioventricular conduction block). This appears on the electrocardiogram as repolarisation changes in a direction opposite to that of the main QRS deflection—that is, "appropriate discordance" between the QRS complex and the ST segment.

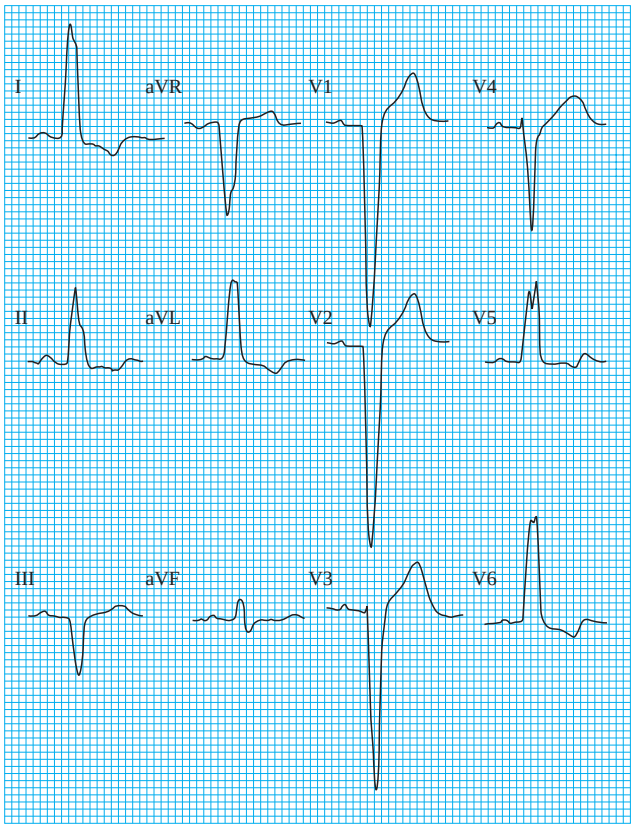
Thus leads with a predominantly negative QRS complex show ST segment elevation with positive T waves (an appearance similar to that of acute anterior myocardial infarction).

Recognition of acute ischaemia

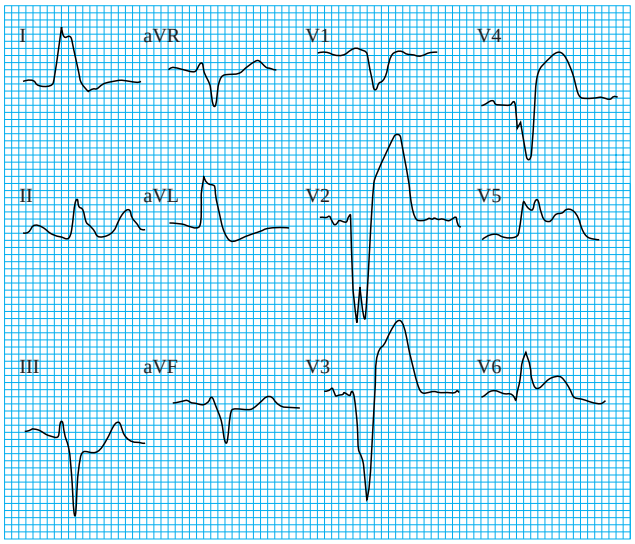
Many different electrocardiographic criteria have been proposed for identifying acute infarction in left bundle branch block, but none has yet proved sufficiently sensitive to be useful in the acute setting. However, some features are specific indicators of acute ischaemia.

ST segment elevation in association with a positive QRS complex, or ST segment depression in leads V1, V2, or V3 (which have predominantly negative QRS complexes), is not expected in uncomplicated left bundle branch block and is termed "inappropriate concordance."

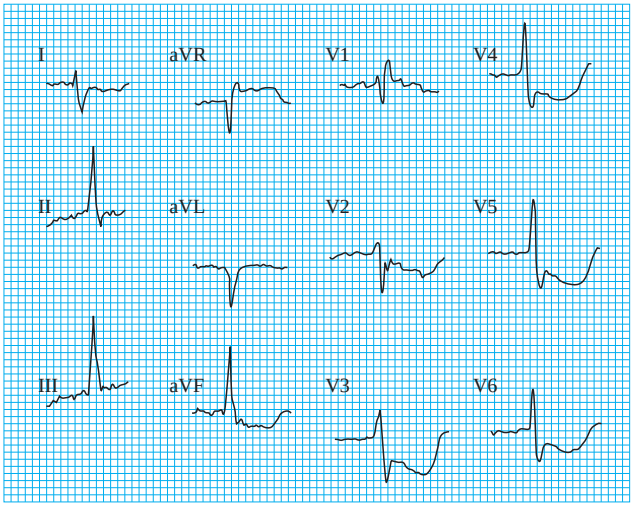
Inappropriate concordance strongly indicates acute ischaemia. Extreme ST segment elevation (≥5 mm) in leads V1 and V2 also suggests acute ischaemia. If doubt persists, serial electrocardiograms may show evolving changes.



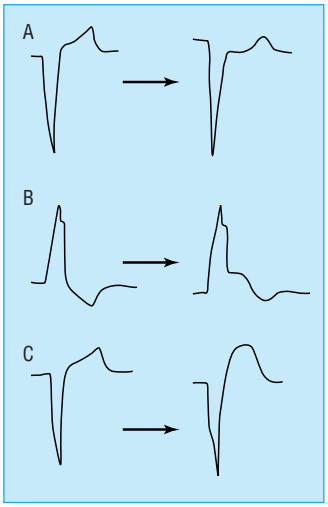
Appropriate discordance in uncomplicated left bundle branch block (note ST elevation in leads V1 to V3)



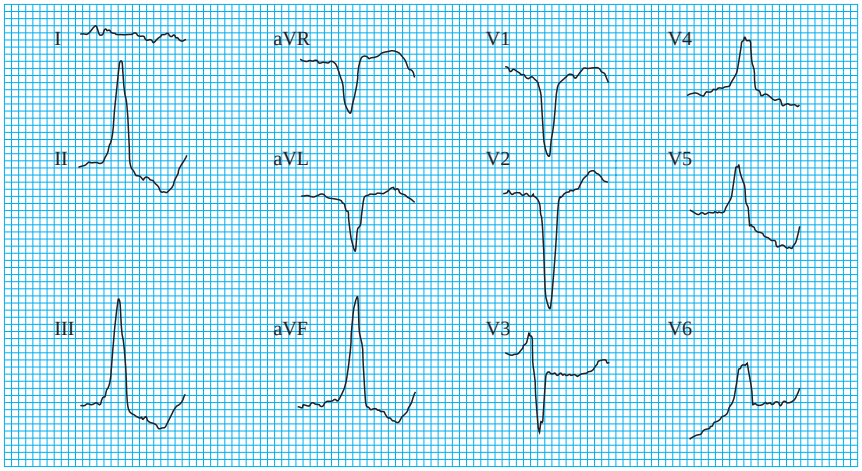
Acute myocardial infarction and left bundle branch block. Note that the ST segments are elevated in leads V5 and V6 (inappropriate concordance) and grossly elevated (> 5 mm) in leads V2, V3, and V4; note also the ST segment depression in leads III and aVF



ST segment depression in precordial leads in 68 year old man with chest pain



Inappropriate concordance in lead V1 in patient with left bundle branch block (A); inappropriate concordance in lead V6 in patient with left bundle branch block (B); and exaggeration of appropriate discordance in lead V1 in patient with left bundle branch block (C)



Development of left bundle branch block in same man shortly after admission (note ST segment depression in lead V3; this is an example of inappropriate concordance)

Right bundle branch block

Right bundle branch block is most commonly seen in association with coronary artery disease, but in many cases no organic heart disease is present. Uncomplicated right bundle branch block usually causes little ST segment displacement and neither causes nor masks Q waves. Thus it does not generally interfere with the diagnosis of acute myocardial infarction, though it may mask a posterior myocardial infarction.

The Brugada syndrome, which is familial, occurs particularly in young men and is characterised by right bundle branch block and ST segment elevation in the right precordial leads. There is a high incidence of death as a result of ventricular tachyarrhythmias

Differential diagnosis of ST segment elevation

ST segment elevation has numerous possible causes. It may be a variant of normal or be due to cardiac or non-cardiac disease. A correct diagnosis has obvious advantages for the patient but is also particularly important before the use of thrombolytic treatment so that unnecessary exposure to the risks of thrombolytic drugs can be avoided.

The interpretation of ST segment elevation should always be made in the light of the clinical history and examination findings. There are often clues in the electrocardiogram to differentiate the ST segment elevation of acute ischaemia from other causes; for example, reciprocal changes (see last week's article) may be present, which strongly indicate acute ischaemia.

Causes of ST segment elevation

- Acute myocardial infarction
- "High take-off"
- Benign early repolarisation
- Left bundle branch block
- Left ventricular hypertrophy
- Ventricular aneurysm
- Coronary vasospasm/Printzmetal's angina
- Pericarditis
- Brugada syndrome
- Subarachnoid haemorrhage

Serial electrocardiography or continuous ST segment monitoring is also useful as ischaemic ST segment elevation evolves over time. Old electrocardiograms are also useful for comparison.

"High take-off"

Care is required when interpreting ST segment elevation in right sided chest leads as the ST segments, particularly in leads V2 and V3, tend to be upsloping rather than flat. Isolated ST segment elevation in these leads should be interpreted with caution. (For more information on "high take-off" see the second article in this series.)

Benign early repolarisation

A degree of ST segment elevation is often present in healthy individuals, especially in young adults and in people of African descent. This ST segment elevation is most commonly seen in the precordial leads and is often most marked in lead V4. It is usually subtle but can sometimes be pronounced and can easily be mistaken for pathological ST segment elevation.

Benign early repolarisation can be recognised by its characteristic electrocardiographic features: elevation of the J point above the isoelectric line, with high take-off of the ST segment; a distinct notch at the junction of the R wave and S wave, the J point; an upward concavity of the ST segment; and symmetrical, upright T waves, often of large amplitude.

Antecedent myocardial infarction

The ST segment elevation associated with acute infarction usually resolves within two weeks of the acute event, but it may persist indefinitely, especially when associated with anterior myocardial infarction. In these patients a diagnosis of left ventricular aneurysm should be considered. Care should be taken when interpreting the electrocardiogram within two weeks of an acute event, and comparison with old electrocardiograms may be useful.

Acute pericarditis

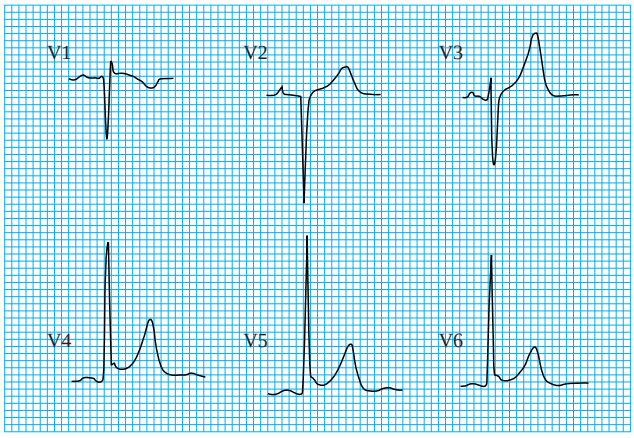
Acute pericarditis is commonly mistaken for acute myocardial infarction as both cause chest pain and ST segment elevation. In pericarditis, however, the ST segment elevation is diffuse rather than localised, often being present in all leads except aVR and V1. The elevated ST segments are concave upwards, rather than convex upwards as seen in acute infarction. Depression of the PR segment may also be seen.

ST segment elevation in pericarditis is thought to be due to the associated subepicardial myocarditis. The zone of injured tissue causes abnormal ST vectors; the end result is that leads facing the epicardial surface record ST segment elevation, whereas those facing the ventricular cavity (leads aVR and V1) record ST segment depression. The absence of widespread reciprocal change, the presence of PR segment depression, and absence of Q waves may be helpful in distinguishing pericarditis from acute myocardial infarction.

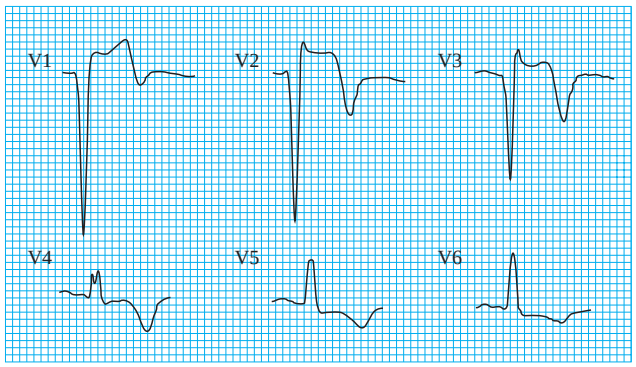
Other causes of ST segment elevation

The characteristic features of left ventricular hypertrophy are also often misinterpreted as being caused by acute ischaemia. ST segment elevation in the precordial leads is a feature of left ventricular hypertrophy and is due to secondary repolarisation abnormalities.

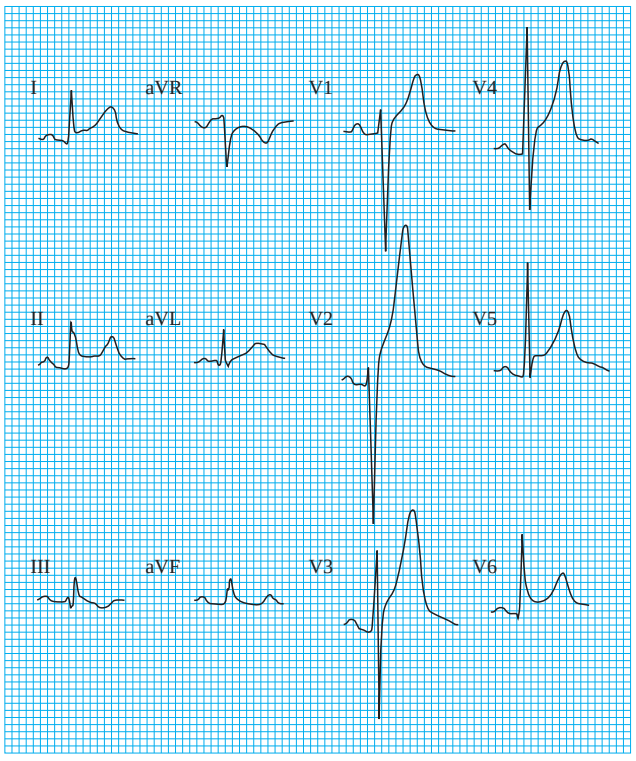
ST segment abnormalities are seen in association with intracranial (particularly subarachnoid) haemorrhage. ST segment elevation or depression may be seen; a putative explanation is that altered autonomic tone affects the duration of ventricular repolarisation, producing these changes.



Benign early repolarisation

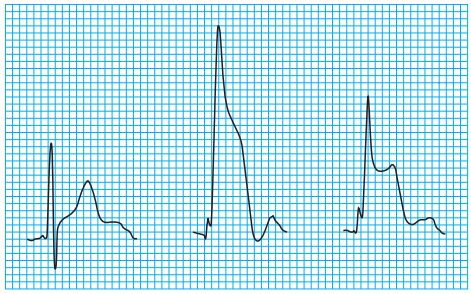


Persistent ST segment elevation in anterior chest leads in association with left ventricular aneurysm



Acute pericarditis with widespread ST segment elevation and PR segment depression (see lead II)

Printzmetal's angina (vasospastic angina) is associated with ST segment elevation. As the changes are due to coronary artery spasm rather than acute infarction, they may be completely reversible if treated promptly. ST segment abnormalities may be seen in association with cocaine use and are probably due to a combination of vasospasm and thrombosis.



Reversible ST segment elevation associated with coronary artery spasm

The ABC of clinical electrocardiography is edited by Francis Morris, consultant in emergency medicine at the Northern General Hospital, Sheffield; June Edhouse, consultant in emergency medicine, Stepping Hill Hospital, Stockport; William J Brady, associate professor, programme director, and vice chair, department of emergency medicine, University of Virginia, Charlottesville, VA, USA; and John Camm, professor of clinical cardiology, St George's Hospital Medical School, London. The series will be published as a book in the summer.

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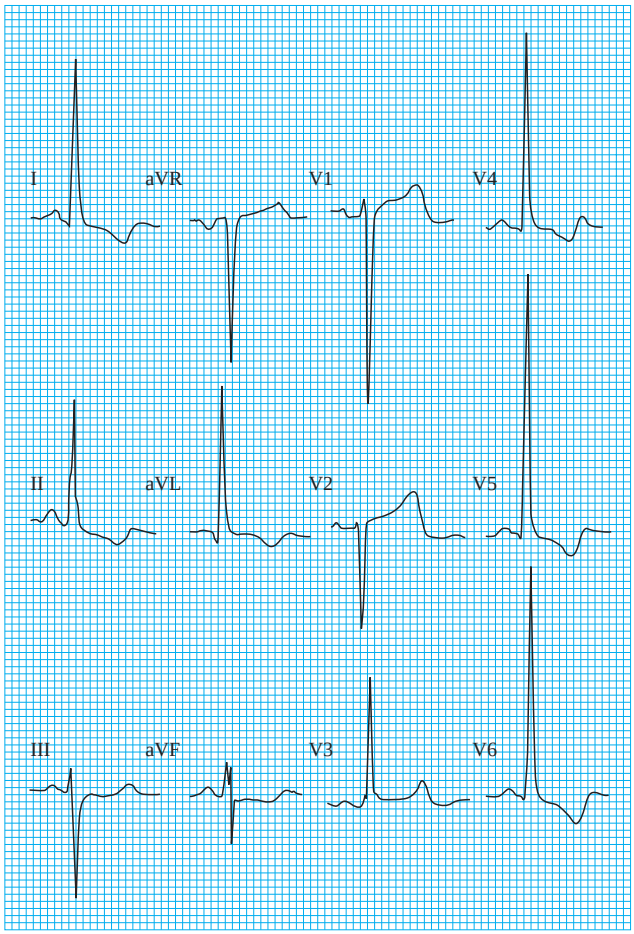

ST segment elevation in leads V1 to V3 in patient with left ventricular hypertrophy

A memorable patient

As in a mirror

Recently, I saw a young woman. She had come in to inform me of her progress, as she knows how long hospital letters take to get through. She knows this because she works in the NHS. A month earlier, she had been diagnosed with lupus erythematosus. She has been given some rudimentary information at the clinic and some prednisolone. She has been told that her blood tests and antibody measurements were not in her notes but that they'd been seen and were "OK."

As you might expect of an intelligent young graduate working in the health service, she had done some research. She had read about fetal problems in lupus and had asked her consultant which antibodies had been checked, in readiness for the future. She was told it wasn't relevant.

I then began to tell her things about how this was her body and her chronic disease and that if she thought something was relevant to ask, it was. She was the one living with her lupus, not any doctor. She looked fairly blankly at me—understanding, but not thinking she could do it, not really listening.

I then, I'm not sure why, shared my own secret. I told her that I am recovering from optic neuritis and that, according to my consultant neurologist and ophthalmologist, I have a 50:50 chance of going on to develop multiple sclerosis. I told her that, at one appointment at the hospital, I was asked by an associate specialist in ophthalmology if I had had any weakness or loss of balance, as this might be widespread neurological disease. That was it—nothing else, no more information.

She then really opened up. She told me how her perspective had changed. If her younger sister worried about assignments she just wanted to shout at her because it was so unimportant. I told her that when patients came in after having a cold for three days and wondering why they weren't better, I felt like yelling at them. She told me about her fears for a family life later, having babies with congenital heart block. I told her about my fears of a puerperal relapse. I know I was near to tears at one point to find that someone felt just like me and that my feelings were normal.

I realised that I should take my own advice. It is my disease, and if I want anything I have the right to ask for it. I then deserve a reasoned explanation about why it would or wouldn't be beneficial so that I can make my own decisions. (Fortunately, the neurologist I saw did just that.) I am the one whose life is affected. I am the one who has to live with it.

I may tell more patients with chronic disease about my own experience to see if it helps them to tell me about their worries and it lets me help them come to terms with it. I just hope now that my patient feels empowered to talk with her consultant, not just be talked at by her consultant.

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