REVIEW

Late potentials as predictors of risk after thrombolytic treatment?

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Late potentials represent low amplitude fractionated electrical activity and they appear at the end of or after the QRS complex. They can be recorded directly by catheter mapping or intraoperative endocardial and epicardial mapping¹⁻³ or indirectly from the body surface by "high-resolution electrocardiography"⁴⁻⁷ after myocardial infarction in patients with sustained ventricular tachycardia. These potentials originate from asynchronous electrical activity in bundles of surviving muscle, mostly at the border of a myocardial infarction. These surviving muscle bundles are separated by fibrous tissue that can form isolating boundaries between them.^{8,9} The low amplitude of these signals is probably the result of the large amount of connective tissue and few surviving muscle fibres beneath the electrode.⁸ They are considered as markers for an "arrhythmogenic substrate" that may become the site for re-entry.¹⁰ However, not all sites showing late potentials are arrhythmogenic; occasionally they are non-specific markers of local fibrosis.¹¹

Late potentials have rarely been seen in patients with normal left ventricular function.⁴⁻⁷ But they were found in 90% of patients with ventricular tachycardia or ventricular fibrillation after previous myocardial infarction.⁶ Late potentials were much less common in patients with previous ventricular fibrillation than in patients with sustained uniform ventricular tachycardia.⁶ The longer the duration of late potentials the faster was the rate of ventricular tachycardia. Late potentials were most common in patients with left ventricular aneurysms caused by coronary artery disease and in patients with other abnormalities of left ventricular contraction.¹² Late potentials were more common in patients with inducible ventricular tachycardia.¹³

Late potentials are strong predictors of arrhythmic events after myocardial infarction.⁷ This was convincingly shown in more than 2000 survivors of myocardial infarction. The presence of late potentials during recovery from myocardial infarction (detected by the use of high resolution electrocardiography) correlated with the subsequent occurrence of sustained uniform ventricular tachycardia or death from arrhythmia or both.

Clinical reports that showed a correlation between the absence of late potentials after myocardial infarction and the success of thrombolytic treatment are important because they indicate that effective thrombolytic treatment can prevent the development of an

abnormal electrophysiological milieu after myocardial infarction.¹⁴⁻¹⁶

Eldar *et al* reported on the effect of thrombolysis on the evolution of late potentials in the early postinfarction period.¹⁵ A hundred and fifty eight patients were prospectively studied during the first 10 days after acute myocardial infarction. The study population consisted of two non-randomised groups. Ninety three patients had been treated conservatively whereas 65 patients had been treated by intravenous thrombolysis. The incidence of late potentials in the first two days after infarction was not significantly different in patients treated conservatively (14%) and those treated by thrombolysis (11.8%). On days 7-10 the incidence of late potentials among patients treated by thrombolysis was unchanged; however, it had increased significantly in the conservatively treated group from 11.8% to 22.5% ($p < 0.01$). Volosin *et al* also evaluated the time course of late potentials after myocardial infarction.¹⁷ They found that in reperfused infarctions development of late potentials was related to the presence of Q waves and was independent of peak creatine kinase concentrations and infarct location. Late potentials sometimes were present only in the early infarction period; they may represent areas of transiently delayed myocardial activation that resolve with infarct healing.¹⁷

Gang *et al* reported a similar study in 106 consecutive patients aged < 75 years who were admitted to hospital with a first acute myocardial infarction.¹⁴ Forty four patients who had chest pain for less than four hours were treated with alteplase (recombinant tissue plasminogen activator) whereas another 62 patients who either did not qualify for or refused treatment with alteplase were treated conventionally. Thus, they were not a randomised sample of patients with acute myocardial infarction. Age, sex, site, and size of myocardial infarction, extent of coronary artery disease, and left ventricular ejection fraction were similar in both groups. Signal averaging was performed in all patients within 48 hours of admission to the hospital and again in the surviving patients before discharge. Only two (5%) of the 44 patients treated with alteplase had late potentials compared with 14 (23%) of 62 patients who were not ($p = 0.01$). The patency of the infarct-related coronary artery and administration of alteplase were the only independent predictors of the presence of late potentials.

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Preliminary data from Riccio *et al* on 172 patients in the Gruppo Italiano per lo Studio della Streptochinasi nell' Infarto Miocardico (GISSI-2) study point to similar findings.¹⁶ Twenty (16.5%) of 103 patients treated with streptokinase or alteplase had late potentials compared with 17 (28.9%) of 69 patients who did not receive thrombolytic treatment because of contraindications or late admission to the hospital.

Kersschot *et al* used programmed ventricular stimulation with three extrastimuli to test for the presence of an arrhythmogenic substrate.¹⁸ Twenty six days after infarction the induction rate for sustained ventricular arrhythmias in general (48% *v* 100%) and uniform ventricular tachycardia (29% *v* 67%) in particular was much lower in 21 patients treated with intravenous and intracoronary streptokinase than in 15 control patients. But contradictory results were reported by Treese *et al* who studied 84 of 127 patients who had been randomised to intravenous and intracoronary thrombolysis with or without transluminal coronary angioplasty.¹⁹ Group A consisted of 64 patients who showed primary recanalisation of the infarct related vessel with persistent patency at a subsequent follow up angiogram: group B consisted of 20 patients who showed late reopening ($n = 5$) or a closed infarct related vessel ($n = 15$) (caused by late reocclusion in nine). There was no difference between the frequency of ventricular extrasystoles in both groups but repetitive ventricular extrasystoles were more common in group A (21 of 64 patients, 32.8%) than in group B (2 of 20 patients, 10%; $p < 0.05$). However, in contrast with the data of Kersschot *et al*,¹⁸ there was no difference in the incidence of inducible ventricular tachycardia.

The results of these preliminary studies indicate that successful early reperfusion may prevent the formation of an arrhythmogenic substrate as shown by the presence of late potentials on high resolution electrocardiography or the results of programmed ventricular stimulation. They^{14-16 18 19} raise some important questions:

- (a) Why does thrombolytic treatment seem to affect the incidence of ventricular late potentials and the results of programmed ventricular stimulation?
- (b) What is the mechanism for the improvement in prognosis after successful thrombolytic treatment?²⁰ Is this improvement in prognosis the result of a reduction (albeit small) in infarct size?^{21 22} Or are other mechanisms responsible—such as changes in the electrophysiological milieu?

Thrombolytic treatment aims at opening an occluded coronary artery and thus at reducing infarct size, with the ultimate goal of improving prognosis. Reduction in cardiac mortality was shown in various studies of thrombolytic treatment though this was not a consistent finding.^{20 23} Outcome was governed by the time from onset of myocardial infarction to thrombolysis, by the patency rate achieved, and possibly by the site of infarction. However, we do not know whether im-

provement in prognosis after thrombolytic treatment is due to a reduced mortality from heart failure or from arrhythmic causes. In addition, the mechanism underlying an improvement in prognosis has not yet been adequately examined.

The reduction in infarct size achieved by early thrombolysis is relatively small. Ritchie *et al* reported a reduction in infarct size (as a percentage of the left ventricle) from 19% in controls to 15% in patients treated with streptokinase.²¹ Bassand *et al* found that the left ventricular ejection fraction was higher (53%) in a group treated with anistreplase than in a group that was not given fibrinolytic treatment (47%).²² The infarct size was reduced by 31%. In the GISSI trial there was no difference in ejection fraction at the predischARGE examination in those assigned to streptokinase and those assigned to standard care.²⁴ However, patients treated with streptokinase had smaller ventricular volumes and a smaller regional wall motion index than patients given standard treatment. These effects persisted for up to 6 months. Another effect of thrombolytic treatment may be the prevention of aneurysm formation.²⁵ This beneficial effect was also seen after late thrombolysis (4–10 hours).

It has been suggested that some of the beneficial effects of alteplase such as improved left ventricular function²⁶ or low mortality²⁷ may be due to some direct myocardial cellular mechanism that is independent of its ability to lyse intracoronary thrombi.²⁸⁻³³ The assumption was that alteplase has a direct protective effect on the myocyte or that it dissolves microthrombi.²⁸ These findings are still controversial. However, they may have major implications for the interpretation of the mechanisms of alteplase.³³

Current data suggest that a patent infarct related coronary vessel reduces the likelihood of ventricular tachycardia even when infarction of the myocardium is developing. Because studies on thrombolytic treatment were not designed to establish the mechanisms of the observed reduction in total mortality, no definite conclusions can be drawn. This is why studies by Gang *et al*, Eldar *et al*, and Riccio *et al* are of great importance because they suggest a potential mechanism for the improvement in prognosis after successful thrombolytic treatment.¹⁴⁻¹⁶ The correlations between the presence of late potentials and arrhythmic events after myocardial infarction and the low incidence of late potentials in patients with successful early reperfusion suggest that the improvement in prognosis may be due to the prevention of the development of an arrhythmogenic substrate.^{6 10}

The factors that govern the genesis of late potentials during the evolution of myocardial infarction are still poorly understood. Gang *et al* suggested several mechanisms that might lead to the absence of late potentials in patients with successful reperfusion (that is those in whom the infarct related artery was open).¹⁴ They postulated that the absence of late potentials may be related to reperfusion because it eliminates ischaemia, allows the

return of various plasma constituents to the ischaemic area, permits a rapid washout of the toxic products of anaerobic metabolism, causes contraction-band necrosis, may increase the amount of haemorrhage within the infarct zone, and may cause the generation of oxygen free radicals.¹⁴

Late potentials originate from small areas within the myocardial infarction in which surviving muscle cells are interspersed with fibrosis. The duration and the degree of local impairment of blood flow govern the extent of necrosis and subsequent fibrosis which may establish an abnormal electrophysiological milieu. Because thrombolysis aims to restore blood flow to the myocardium as fast as possible, early intervention may influence the genesis of an abnormal electrophysiological substrate in different ways. First, blood flow may be reduced for too long to allow recovery of the myocardium; this leads to total necrosis without the survival of any muscle fibres. In this situation no chronic arrhythmogenic substrate should develop. Secondly, incomplete recovery may lead to regional survival of the myocardium after thrombolysis. This may increase the likelihood of a patchy infarct and thus of an "arrhythmogenic substrate". Thirdly, restoration of blood flow may be achieved early enough to allow complete recovery of the myocardium which should prevent the occurrence of an "arrhythmogenic substrate".

Current data suggest that detection of late potentials by high resolution electrocardiography is a way of assessing the likelihood of arrhythmic events after myocardial infarction. Several studies even suggest that it is a more powerful predictor for ventricular instability than long term electrocardiographic recording.⁶ High resolution electrocardiography seems to be a powerful tool for identifying the presence of an arrhythmogenic substrate after a patient with acute myocardial infarction has been treated by thrombolysis.¹⁰ Prospective studies using this new technique should help to clarify whether in patients with similar degrees of left ventricular impairment outcome is worse in those with late potentials.

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LETTERS TO THE EDITOR

- The British Heart Journal welcomes letters commenting on papers that it has published within the past six months.
- All letters must be typed with double spacing and signed by all authors.
- No letter should be more than 600 words.
- In general, no letter should contain more than six references (also typed with double spacing).

Balloon dilatation of tricuspid stenosis caused by carcinoid heart disease

SIR,—Mullins *et al*'s conclusion that balloon dilatation of the tricuspid valve is safe and effective for tricuspid stenosis caused by carcinoid may not be consistent with clinical and haemodynamic data in their patient (*Br Heart J* 1990;63:249–50). They state that the jugular venous pulse showed a prominent systolic wave. This clearly means that tricuspid regurgitation was a clinically important abnormality. This accords with the haemodynamic data because in the basal state the 'v' wave in the right atrium was 44 mm Hg whereas the 'a' wave was 24 mm Hg. They say that the chest x ray showed pulmonary oedema. This is an unusual and unlikely feature of primary tricuspid valve disease, especially when there is no additional left sided disease. Because there was no mention of myocardial or valve disease affecting the left heart I presume that these structures were normal.

Right ventricular hypertension in carcinoid heart disease affecting the right side of the heart is possible only if there is stenosis of the pulmonary valve. The pulmonary valve was normal in this case, so what was the cause of the right ventricular pressure of 52 mm Hg? In addition, a right ventricular diastolic pressure of 18 mm Hg is rather surprising in the presence of important tricuspid stenosis without any additional cause for diastolic restriction of the right ventricle.

After dilatation of the tricuspid valve the 'a' wave in the right atrium remained at 24 mm Hg whereas the height of the 'v' wave fell from 44 to 36 mm Hg. This needs to be explained. Mullins *et al* claim that the severity of tricuspid regurgitation did not change after dilatation so what was the cause of reduction in the height of the 'v' wave?

Though after dilatation the mean pressure drop across the tricuspid valve fell from 10.5 to 7.6 mm Hg and the valve area increased from 0.9 to 1.4 cm², are Mullins *et al* justified in calling this dilatation "effective"? The reduction in the mean pressure drop could have been solely related to the reduction in the height of the 'v' wave, especially as the height of the 'a' wave was unchanged. Without knowledge of the cardiac output the effectiveness of dilatation cannot be judged solely on the values of the pressure drops.

Though the area of the tricuspid valve increased, its absolute value of 1.4 cm² still indicates a critical tricuspid stenosis.¹

What is more impressive is the fact that despite an 'a' wave of 24 and a 'v' wave of 36 mm Hg in the right atrium after balloon dilatation the "patient is doing well with reduction of dyspnoea and pulmonary and peripheral oedema". Why did the pulmonary oedema resolve after the release of the tricuspid stenosis?—when it seems logical to expect an increase in pulmonary oedema because of increased flow in the pulmonary circuit.

Unless these discrepancies are sorted out I cannot accept that this was predominantly a case of tricuspid stenosis requiring balloon dilatation. Moreover, on the basis of these data alone I believe that it is not correct to call this dilatation effective and recommend it for carcinoid valve disease.

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- 1 Kitchin A, Turner R. Diagnosis and treatment of tricuspid stenosis. *Br Heart J* 1964;26: 354–79.

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

SIR,—We are grateful for Dr Dalvi's interest in the case we presented. He raises three points for debate.

Firstly, was there significant tricuspid stenosis? Tricuspid stenosis was confirmed by a transvalvar gradient of 10.5 mm Hg and an orifice area of 0.9 cm² estimated by Doppler echocardiography. The criticism that the presence of a cardiac catheter across the valve during the haemodynamic study leads to an overestimation of the gradient can be levelled at all gradient estimations. This criticism, however, is not valid when the orifice area is estimated by Doppler echocardiography at a different time and the method is not subject to the same measurement constraints.

Secondly, why did the patient have a raised right ventricular diastolic pressure and pulmonary oedema? These abnormalities are not a feature of pure tricuspid stenosis. This elderly woman, presenting with carcinoid, also had poor left ventricular function, the cause of which was not investigated further. We postulated that fluid retention caused by tricuspid stenosis and poor ventricular function was responsible for the pulmonary congestion.

Thirdly, was the tricuspid dilatation successful? We showed a reduced mean gradient and an increase from 0.9 cm² to 1.4 cm² in orifice area estimated by Doppler echocardiography. The patient's symptoms of fluid retention and dyspnoea rapidly improved. The increase in tricuspid valve orifice area was maintained on a later echocardiogram.

This patient was terminally ill and in a very poor clinical state. Our aim was to palliate the symptoms associated with her severe tricuspid valve disease. The small improvement in tricuspid valve orifice was sufficient for this purpose.

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Diagnosis of a pseudoaneurysm of the right ventricular outflow tract

SIR,—Sreeram *et al* reported the diagnosis of a pseudoaneurysm of the right ventricular outflow tract (1990;63:129–31). Their colour Doppler picture certainly shows clearly the narrow communication between the right ventricle and the pseudoaneurysm.

Those who do not have a colour Doppler machine may be interested to know that a communication such as this can also be detected by contrast echocardiography, as we reported a few years ago.¹

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- 1 Garty BZ, Berant M, Weinhouse E, Levinski L. False aneurysm of the right ventricle due to endocarditis in a child. *Pediatr Cardiol* 1987;8:275–7.

This letter was shown to the author, who replies as follows:

SIR,—As Berant and Garty point out in their letter, the use of contrast echocardiography may be a valuable adjunct to the investigation of patients in whom a pseudoaneurysm of the right ventricular outflow tract is suspected.

While contrast echocardiography is a useful alternative non-invasive approach to diagnosis where colour flow mapping is not available, we suggest that colour flow mapping is a more appropriate technique because it can be quickly and easily carried out as part of the ultrasound examination itself. It does not require an intravenous line and administration of a contrast agent. Furthermore, the contrast effect produced can be variable in such patients and thus the results of such studies could be misleading. We expect colour flow mapping will prove to be a more sensitive and specific technique.

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CORRECTION

Review. Late potentials as predictors of risk after thrombolytic treatment? Günter Breithardt, Martin Borggrefe, Ulrich Karbenn (September 1990 issue, volume 64: pages 174–6)—We regret that an editorial error was introduced into this article. Lines 9, 10, and 11 of the second paragraph on page 174 should have read: There was an inverse relation between the duration of late potentials and the rate of ventricular tachycardia.