
Vectorcardiographic monitoring of ST segment changes during transient hypotension following thrombolysis with streptokinase

Reperfusion is an important prognostic factor for patients with acute myocardial infarction (AMI), treated with thrombolysis[3]. Continuous dynamic vectorcardiographic monitoring is a non-invasive method for observing changes in cardiac ischaemia. We applied this method to examine alterations in ischaemia during thrombolysis, with particular emphasis on the relationship between reperfusion and the transient drop in blood pressure occurring occasionally during thrombolysis with streptokinase.

Patients treated with streptokinase were monitored with continuous vectorcardiography (VCG) during treatment. For VCG analysis we used the MIDA system, which gives an online, dynamic analysis of QRS complex and ST-segment changes[3]. The two vectorcardiographic parameters studied were QRS vector difference (QRS-VD) and ST vector magnitude (ST-VM). The QRS-VD depicts the total change within the QRS complex. The ST-VM is the deviation of the ST segment 60 ms after termination of the QRS complex.

We defined VCG reperfusion as: a ST magnitude recovery of at least 25% within 90 min from initiation of thrombolysis, and/or a QRS-VD slope greater than 6 μV·h⁻¹, or reaching a QRS-VD plateau within 2 h[3]. We only studied early abnormalities, registering the occurrence of transient peaks (Fig. 1). We defined a transient peak as: a rapid increase in the ST-VM occurring during the first 15 min of thrombolysis, and to a level exceeding the initial ST-VM value.

Blood pressure was monitored every 10 min during thrombolysis. In case of any discomfort, additional blood pressure measurements were carried out. We defined a streptokinase-induced blood pressure drop as a decrease in systolic blood pressure exceeding 50 mmHg, or to value less than 90 mmHg. During a 12-month period, 58 patients were monitored with VCG during thrombolysis with streptokinase. Twelve patients were excluded from the analysis, because the VCG monitoring during thrombolysis was incomplete. Of the remaining, 27 patients exhibited VCG signs of reperfusion, and 19 showed no signs of reperfusion; of these three had no AMI.

Nineteen patients experienced a drop in blood pressure, according to the definition mentioned above. Of these, 14 patients had reperfusion patterns, but five showed no signs of reperfusion; two patients had no AMI. The patients with and without transient hypotension were comparable in respect to clinical data.

In all patients with a transient blood pressure drop, the VCG exhibited a simultaneous transient peak. This means that 52% of patients with reperfusion exhibit a transient peak, compared to 26% of patients without reperfusion. The normal ECG showed a simultaneous marked increase in ischaemia, expressed as an aggravation of ST-segment elevation. In 11 patients with a transient peak we found a similar, but less marked, rise in the QRS-VD trend curve.

Streptokinase-induced hypotension has previously been regarded as a sign of reperfusion[3]. We found, however, only a weak correlation between transient hypotension and

![Figure 1](image-url)

*Figure 1* The arrow marks a ‘transient peak’ at the ST-VM trend curve. A simultaneous peak is seen at the QRS-VD trend curve.
reperfusion. The phenomenon is even seen in patients without AMI. We found that a transient drop in blood pressure induced an aggravation of the ischaemia, and as such may be regarded as hazardous for the patient.

In studies analysing ECG-patterns during thrombolysis, similar observations, of an additional ST segment elevation during early reperfusion, have been made. In these studies, it has been demonstrated that the benefit of reperfusion is limited in patients with additional ST-segment elevations\[9\]. This early ST elevation is surely a counterpart to the transient peak we describe.

In our study we observed that patients with hypotension had a transient peak in the initial ST-VM trend curve. It occurred irrespective of signs of reperfusion or AMI, and seems to be a consequence of the hypotensive reaction. We therefore assume that the transient peak can be explained as streptokinase-induced hypotension.

As we are well aware, only part of the variability in the ST-VM trend curve is explained by drop of blood pressure. The variability could also be a consequence of unstable angina pectoris, or alteration in patency of the coronary arteries as previously described by Krucoff et al.\[10\]. It is important to note that we only studied the initial changes, and not the occlusion/reocclusion pattern described by Krucoff et al.

We conclude that VCG monitoring allows the effect of blood pressure drop during thrombolysis to be examined. We found that transient hypotension accentuates the ischaemic burden. A transient peak is interpreted as a sign of alteration in myocardial ischaemia, not as evidence of reperfusion.

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References

A case of infective endocarditis complicated with anterior mitral valve leaflet abscess

Infective endocarditis is a microbial infection of intact or degenerated cardiac values, the endothelium surrounding congenital or acquired cardiac defects and the endothelium of vascular malformations\[2\]. Although there are many complications of the disease, to our knowledge there are few reported cases complicated by an abscess of the cardiac valve leaflet\[3\].

We report a case of infective endocarditis very probably complicated by an abscess of the anterior mitral valve leaflet in addition to embolic complications.

A 19-year-old white male presented to the emergency room semi-conscious with fever, abdominal pain, weight loss, motor aphasia and a possible diagnosis of infective endocarditis.

His physical examination revealed a body temperature of 38.8°C, an apical third heart sound, a grade 2/6 pansystolic murmur on the apex, a right hemiparesis, cutaneous embolic phenomena and a sacral grade 2 decubital ulcer.

Circulating blood cells showed a haemoglobin of 9 gm·dl\(^{-1}\), and a haematocrit of 28%. His white blood cells were 12 200 mm\(^{-3}\) with a left shift on the differential count. Erythrocyte sedimentation rate was 75 mm·h\(^{-1}\). His ECG and chest X-ray were normal. A head computed tomography scan showed multiple infarctions. Blood cultures were drawn. His echocardiogram revealed a 1:3 x 0·7 cm mobile vegetation and a prolapse of the anterior mitral valve leaflet into the left atrium. Abdominal ultrasound showed a 12 x 7 cm splenic abscess which was drained and cultured. Three of four blood cultures grew methicillin resistant Staphylococcus aureus (MRSA) as did the splenic drainage specimen. On the 12th day of admission, there was no cessation of fever. A Doppler echocardiogram revealed a lesion compatible with an abscess on the anterior mitral valve leaflet (Fig. 1): grade 2--3/4 mitral insufficiency, and grade 1--2/4 tricuspid insufficiency. Surgery was ruled out because of the patient’s poor clinical condition. Three weeks after admission his fever ceased and on the 42nd day of admission the patient left hospital, against medical advice. He was put on oral ciprofloxacin 500 mg four times a day and rifampicin 600 mg daily and was requested to return to the clinic one month later. The patient failed to return and died at home 3 months later; no autopsy could be performed.

There are many complications of infective endocarditis but an abscess

![Figure 1](https://example.com/figure1.png)

**Figure 1** Circular lesion, compatible with an abscess, on the anterior mitral valve leaflet.