Background: The prognostic importance of dyskinesia after acute myocardial infarction is unknown, and recommendations have been made that dyskinesia be included in calculations of wall motion index (WMI).

Objective: To determine whether it is necessary to distinguish between dyskinesia and akinesia when WMI is estimated for prognostic purposes following acute myocardial infarction.

Design: Multicentre prospective study.

Patients: 6676 consecutive patients, screened one to six days after acute myocardial infarction in 27 Danish hospitals.

Interventions: WMI was measured in 6232 patients, applying the nine segment model, scoring 3 for hyperkinesia, 2 for normokinesia, 1 for hypokinesia, 0 for akinesia, and −1 for dyskinesia. Calculation of WMI either included information on dyskinesia or excluded this information by giving dyskinesia the same score as akinesia.

Main outcome measures: Long term outcome (up to seven years) with respect to mortality.

Results: Dyskinesia occurred in 673 patients (10.8%). In multivariate analysis, WMI was an important prognostic factor, with a relative risk of 2.4 (95% confidence interval (CI), 2.2 to 2.7), while dyskinesia had no independent long term prognostic importance (relative risk 1.00; 95% CI, 0.89 to 1.12). For 30 day mortality dyskinesia had a relative risk of 1.23 (95% CI, 1.00 to 1.53) (p = 0.045).

Conclusions: Echocardiographic evaluation of left ventricular systolic function shortly after an acute myocardial infarct gives important prognostic information, but the presence of dyskinesia only has prognostic importance for the first 30 days.
Echocardiography

At the time of screening an echocardiographic examination was recorded on videotape by the investigator and sent to a core laboratory, where the WMI was calculated by two people using the nine segment model described by Heger et al. Segmental scores and WMI were calculated as described by Berning and Steensgaard-Hansen. Validation of this method has been reported previously. In the model, dyskinesia is represented by a score of −1, akinesia 0, hypokinesia 1, normokinesia 2, and hyperkinesia 3. Dyskinesia is distinguished from akinesia because it is characterised by outward movement during systole. Hyperkinesia was considered present when movement of the myocardium was more pronounced in the affected area than in other areas with normal function. With this reverse scoring system, a normal left ventricle has a WMI of 2.0. A WMI of 1.2 corresponds to an abnormal function. With this reverse scoring system, a normal left ventricle has a WMI of 2.0. A WMI of 1.2 corresponds to an abnormal function. With this reverse scoring system, a normal left ventricle has a WMI of 2.0. A WMI of 1.2 corresponds to an abnormal function.

RESULTS

Dyskinesia occurred in at least one segment in the 673 patients in whom an echocardiogram was analysed (10.8%), and in 765 segments overall, giving an incidence per segment of 1.36%. In patients without previous infarction, the incidence was 10.3%, and in patients without any symptoms of previous ischaemic heart disease (n = 3304)—that is, previous acute myocardial infarction, angina pectoris, or congestive heart failure—the incidence was 9.8%. Dyskinesia occurred mainly in the apex of the left ventricle and was recorded in 23.4% of patients with anterior acute myocardial infarction and in only 69 patients (3.6%) with inferior acute myocardial infarction (p < 0.01). In patients without previous acute myocardial infarction, these figures were 23.9% and 2.9%, and in patients without previous ischaemic heart disease, 23.4% and 2.5%, respectively (p < 0.001).

The main differences between the baseline characteristics of patients with and without dyskinesia are shown in table 1. Patients with dyskinesia were older and generally had a higher prevalence of other important risk factors, both previous disease and complications during the index infarction. Patients with dyskinesia were admitted later than patients without dyskinesia, but there was no difference in the time to echocardiography in the two groups.

There was a trend for less dyskinesia in patients given thrombolysis: in those treated with thrombolysis (n = 2567),
dyskinesia occurred in 10.2%; in those not treated with thrombolysis it occurred in 11.4% \( (p = 0.143) \). In patients without previous acute myocardial infarction the figures were 9.9% and 10.7%, respectively \( (p = 0.40) \), and in patients without known ischaemic heart disease, 9.3% and 10.4% \( (p = 0.27) \). Arrhythmias occurred more often in patients with dyskinesia \( (p<0.01) \). In patients without dyskinesia, median WMI was 1.5 (5th to 95th centile interval, 0.8 to 2.0). This was significantly different from the WMI (D-WMI) in patients who had dyskinesia \( (1.0, 0.6 to 1.4; p < 0.0001) \). If dyskinesia was excluded—that is, coded as akinesia—WMI (ND-WMI) was still significantly different from that in patients without dyskinesia \( (1.1, 0.7 to 1.6; p < 0.0001) \). Hyperkinesia was recorded in 24% of patients with dyskinesia and in 10% of those without \( (p < 0.01) \). If hyperkinesia was replaced by normokinesia when calculating WMI (NH-WMI), the NH-WMI for the dyskinetic group of patients was 1.0 \( (0.5 to 1.4) \), and for the non-dyskinetic group, 1.5 \( (0.8 to 2.0) \) \( (p < 0.001) \). If dyskinesia was excluded—that is, coded as akinesia—the wall motion index (ND-NH-WMI) was still significantly different from that in patients without dyskinesia \( (1.1, 0.6 to 1.4; p < 0.0001) \).

Overall survival curves according to the presence or absence of dyskinesia are shown in fig 1. Patients with dyskinesia had a 30 day mortality of 19.2%, and a one, three, and five year mortality of 31.3%, 44.9%, and 54.9%, respectively. In patients without dyskinesia the corresponding figures were 9.4%, 19.9%, 32.2%, and 41.2%. In univariate analyses the relative risk associated with dyskinesia was 1.6 \( (1.4 to 1.7) \) \( (p < 0.0001) \).

This apparently adverse prognosis associated with dyskinesia was completely explained by patients with dyskinesia having more depressed left ventricular systolic function: in a multivariate model including dyskinesia and ND-WMI, only ND-WMI had prognostic importance. In this analysis, the relative risk of dyskinesia was 1.00 \( (0.90 to 1.11) \), and that of ND-WMI, 4.1 \( (3.8 to 4.6) \).

The results of multivariate backward selection analyses where dyskinesia was forced into the model, after including all variables that were either of prognostic importance in univariate analyses or showed significant differences between the groups, are shown in table 2. Dyskinesia did not have any long term prognostic significance. The relative risk of ND-WMI was 2.5—equal to that obtained in a multivariate analyses including D-WMI and the same set of variables. If hyperkinesia was replaced by normokinesia when calculating WMI (NH-WMI), dyskinesia did not have prognostic importance in multivariate analyses \( (relative \ risk 0.99, 0.88 to 1.11; p = 0.85) \).

The prognostic importance of dyskinesia in the various subgroups is shown in table 3. Dyskinesia did not add prognostic information at any level of WMI. As dyskinesia mainly occurred in anterior Q wave infarction, multivariate analyses were repeated in the patients with an anterior infarct \( (n = 2445) \). Again dyskinesia did not add prognostic information. Dyskinesia did not add any long term prognostic information in patients treated with or without thrombolysis. This was also the case in the “first infarct” and “no previous ischaemic heart disease” subgroups.

As the multivariate analysis reflected a time period of up to seven years, we could have overlooked the possibility that dyskinesia was a marker of early mortality. To test for the short term prognostic importance of dyskinesia, multivariate models were constructed looking only at death occurring up to 30 days after the infarct. In univariate analysis, dyskinesia was associated with a relative risk of 2.2 \( (1.8 to 2.7) \). The results of multivariate analyses including ND-WMI are shown in table 4. The factors that had prognostic importance for the first 30 days differed slightly from those found important for long term prognosis, though the only major difference was the
The principal finding of this study is that left ventricular systolic function measured by the wall motion index shortly after acute myocardial infarction is a powerful predictor of long term mortality; however, no useful prognostic information was obtained by distinguishing between akinesia and dyskinesia as components of the WMI. Intuitively, dyskinesia would be expected to result in a worse prognosis as it leads to a reduction in the left ventricular ejection fraction. Removing dyskinesia from the WMI by changing it to akinesia should have shown that dyskinesia had independent prognostic importance. However, this was found only in multivariate analysis of 30 day mortality. Dyskinesia had no prognostic value for long term mortality. Thus dyskinesia is a weak prognostic factor that extinguishes rapidly over time after acute myocardial infarction.

Variables describing regional left ventricular dysfunction and used for prognostic purposes include akinesia, dyskinesia, and aneuryism formation. These variables are related, but describe different facets of dysfunction. In contrast to akinesia, dyskinesia is characterised by outward movement of a wall segment during systole. Defined in this way dyskinesia includes aneurism formation; aneurysms, however, show bulging during diastole as well, and a thinner myocardium. Infarct expansion is a definite outward bulging and alteration in the curvature of the infarcted left ventricular segment, with hinge points persisting between both systole and diastole. These phenomena and their relation to long term prognosis need to be considered in the light of any improvements resulting from revascularisation procedures.

In previous reports of patients with transmural anterior acute myocardial infarction, dyskinesia has been found in 21.5%, 25%, and up to 35% of cases, and in 1% of segments. The incidence of aneurysm formation is 12.4% in necropsy studies. The low incidence of dyskinesia in the present study may partly reflect the fact that the study was performed after generalised use of thrombolytic treatment had become the norm. It may also be relevant that on average echocardiography was performed three days after the infarct, at a time when dilatation of the left ventricle is not yet complete and when infarction expansion is still progressing. Interpretation of the type of infarct related wall motion in the days following acute myocardial infarction is further complicated by the fact that low sympathetic drive may improve function and high sympathetic drive decrease it (by increasing myocardial ischaemia), as with dobutamine stimulation,13-15 Assuming a similarity between the echocardiographic observations during the days after acute myocardial infarction and during dobutamine stimulation, some regions may show improved wall motion during the acute phase.16 Also, the echocardiographic pattern of an akinetic segment at rest may become dysskinetic during peak stress. As both akinesia and dyskinesia most probably represent infarcted myocardium, motion in this area may be determined by the function of myocardium remote from the infarct site. Thus a hyperkinetic area may change an akinetic area to a dysskinetic area mechanically.

In this study we observed dyskinesia with the same frequency in patients treated with thrombolysis or not. In previous studies, patients treated with thrombolysis did not have a significantly lower rate of dyskinesia, but there was slightly better wall motion of the infarct areas if complete or near complete reperfusion (TIMI grade 3 flow) was obtained by thrombolysis,17 especially if collateral supply was present.18 However, in one study a relation between thrombolytic treatment (or high TIMI flow) and the size of the infarct was already present from day 2 to 3.19 The reason for this difference may be that measurement of the perimeter of the infarct is a more sensitive marker of dyskinesia than echocardiographic estimation.

The decreasing systolic function of the left ventricle during the first days or weeks after acute myocardial infarction, reflected in the decrease in left ventricular ejection fraction or increase in left ventricular end systolic volume, is mainly caused by the disappearance of hyperkinesia.20 Echocardiographic demonstration of changes in the infarct area within the first hours and days following symptoms shows that 66% of initially hypokinetic segments remain unchanged up to 10 days after the infarct and most of the rest improve.21 In contrast to this is the finding that anterior segments with dyskinesia have an 83% risk of aneurysm formation within one year.22 In the present study we drew no distinction between dyskinesia and aneurysm (as echocardiography was done on average three days after the infarct). Previously, formation of an aneurysm23 and infarction expansion24 have been shown to be of prognostic importance. As dyskinesia does not apparently have any long term prognostic significance it is possible that the majority of our patients did not develop an aneurysm. Our finding of only short term prognostic importance for dyskinesia could be explained by the formation of aneurysms in some patients, with their distal short term prognosis,25 and a larger group without progression to aneurysm formation.

Conclusions
Dyskinesia and akinesia represent infarcted myocardium, and they are thus important factors in prognosis. However, when used in the wall motion index score for determining prognosis, a distinction between these two phenomena does not seem to be justified. This is in contrast to the opposite end of the scoring system, where a distinction between hyperkinesia and normokinesia is advantageous.

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Table 4 Relative risk of 30 day mortality after acute myocardial infarction: results of multivariate analyses with non-dyskinetic wall motion index (ND-WMI)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Relative risk (95% CI)</th>
<th>p Value</th>
</tr>
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<tbody>
<tr>
<td>Dyskinesia</td>
<td>1.23 (1.00 to 1.53)</td>
<td>0.045</td>
</tr>
<tr>
<td>Age†</td>
<td>1.04 (1.03 to 1.05)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Systemic hypertension</td>
<td>1.2 (1.0 to 1.5)</td>
<td>0.03</td>
</tr>
<tr>
<td>Previous AMI</td>
<td>0.8 (0.6 to 0.9)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Patient delay†</td>
<td>1.009 (1.005 to 1.014)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Thrombolysis</td>
<td>0.8 (0.6 to 1.0)</td>
<td>0.02</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>2.9 (2.2 to 3.8)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Ventricular dilatation</td>
<td>3.6 (2.9 to 4.4)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>1.2 (1.0 to 1.5)</td>
<td>0.02</td>
</tr>
<tr>
<td>Non-dyskinetic WMI*</td>
<td>3.7 (2.9 to 4.8)</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

*Relative risk is for unit for continuous variables.
†Patient delay is time from start of symptoms until arrival at hospital.
AMI, acute myocardial infarction; CI, confidence interval; WMI, wall motion index.
Pyoderma gangrenosum presented as a refractory wound infection following permanent pacemaker implantation

Pyoderma gangrenosum (PG) is a rare painful ulcerative cutaneous disease of unknown aetiology. An underlying systemic disorder is associated in up to 50% of cases. We present a case of PG which developed after permanent pacemaker implantation in an 85 year old woman with a background history of monoclonal gammopathy and polylymphagia rheumatica. This was initially treated as a severely infected pacemaker site, which led to extraction of her pacemaker system. Her wound only started to heal after she was treated with systemic steroids. PG has been reported following various surgeries but to the best of our knowledge none has been reported following permanent pacemaker implant. Such diagnosis should be born in mind as it may avoid unnecessary pacemaker system extraction, a lesson we have now learned.