Does pain relief with spinal cord stimulation for angina conceal myocardial infarction?

C Andersen, P Hole, H Oxhoj

Abstract

Objective—To investigate the possibility that spinal cord stimulation (SCS) used for pain relief can conceal acute myocardial infarction (AMI).

Design—Prospective evaluation of patients treated with SCS.

Setting—University hospital.

Patients—50 patients with coronary artery disease and severe, otherwise intractable angina treated with SCS for 1–57 months.

Main outcome measures—Necropsy findings, symptoms, serum enzyme concentrations, electrocardiographic changes.

Results—Ten patients were considered to have had AMI. In nine of these SCS did not conceal precordial pain and in one patient no information about precordial pain could be obtained.

Conclusion—There was no evidence that SCS concealed acute myocardial infarction.

(Br Heart J 1994;71:419–421)

The perception of cardiac pain during myocardial ischaemia is mediated via sympathetic afferent nerve fibres.1 High thoracic epidural anaesthesia can block cardiac afferent sympathetic fibres; this technique therefore reduces cardiac pain during myocardial ischaemia.2 The same effect is achieved by spinal cord stimulation (SCS),3 which has become a well established treatment for chronic pain. SCS was first reported as a successful treatment of otherwise intractable angina pectoris in 1987.4

Precordial pain is the cardinal symptom of acute myocardial infarction (AMI) and it is possible that effective pain relief by SCS may conceal acute myocardial infarction.2 Currently several centres use SCS to treat angina: other centres are reluctant to use SCS because they fear that the stimulation could conceal an acute myocardial infarction.

We sought evidence of acute myocardial infarction in patients treated with SCS for angina.

Patients and methods

The study was approved by the local ethics committee and the patients gave their informed consent before enrolment. We studied 50 patients (40 men and 10 women) treated with SCS for otherwise intractable anginal pain (table 1). Eighteen of the patients had had one myocardial infarction and 15 had had two or more. Forty two of the patients had previously been treated with percutaneous transluminal coronary angioplasty (PTCA) or coronary artery bypass graft surgery (CABG) or both. In all the patients SCS treatment was started because further medical treatment or revascularisation was regarded as impossible.

The patients were reviewed after an average of 29 (range 1–57) months of SCS treatment. The minimum observation time for patients who did not die or stop SCS treatment (6 patients) was 12 months. During the observation period electrocardiographic changes and cardiac enzymes, if available, were reviewed for every hospital admission and visit to the casualty ward. All admissions to hospital in the 3 years before SCS treatment were also reviewed.

The patients were seen by one of us every 1 to 4 months. At these visits they were questioned about symptoms and visits to other medical institutions. If the patient had consulted a general practitioner, information about the visit was obtained. Because of severe chest pain 44 of the patients were treated with opioids before SCS treatment. A reduction in dose during SCS was used to assess symptomatic relief.

When possible the diagnosis of acute myocardial infarction was established according to WHO criteria that is, pain, ECG changes, increase in myocardial enzymes (creatine kinase B > 20 U/l and lactate dehydrogenase i > 170 U/l).6 If this information was unavailable, acute myocardial infarction was diagnosed if the patient died suddenly or acute heart failure developed.

A 12 lead ECG was recorded at follow up and compared with the ECG recorded immediately before SCS treatment was started. Any alterations in Q wave, QRS configuration, ST/T segment, and T wave were evaluated.

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Table 1 Data on Patients

<table>
<thead>
<tr>
<th>Variable</th>
<th>AMI (n = 10)</th>
<th>Non-AMI (n = 40)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>57 (47–71)</td>
<td>61.5 (43–78)</td>
</tr>
<tr>
<td>F/M</td>
<td>9/1</td>
<td>9/31</td>
</tr>
<tr>
<td>CABG (%)</td>
<td>8 (80)</td>
<td>31 (78)</td>
</tr>
<tr>
<td>PTCA (%)</td>
<td>1 (10)</td>
<td>9 (23)</td>
</tr>
<tr>
<td>AMI (%)</td>
<td>9 (90)</td>
<td>24 (60)</td>
</tr>
<tr>
<td>Hospital admissions before/with SCS*</td>
<td>3/0/1-6</td>
<td>3/0/1-9</td>
</tr>
<tr>
<td>Opiate consumption before/with SCS†</td>
<td>47/50-2</td>
<td>30/4-0-4</td>
</tr>
<tr>
<td>Digoxin (No of patients (%))</td>
<td>4 (40)</td>
<td>10 (25)</td>
</tr>
</tbody>
</table>

*Mean number of hospital admissions per patient per year.
†Median opiate consumption equivalent to mg morphine per patient per 24 h.
Table 2 Data on the 10 patients with acute myocardial infarction

<table>
<thead>
<tr>
<th>Case No</th>
<th>Age</th>
<th>Sex</th>
<th>Previous AMI</th>
<th>CABG</th>
<th>PTCA</th>
<th>SCS (month)</th>
<th>Survival/cause of death</th>
<th>CK-B</th>
<th>LD-1</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>49</td>
<td>M</td>
<td>2</td>
<td>1</td>
<td></td>
<td>21</td>
<td>Alive</td>
<td>34</td>
<td>219</td>
</tr>
<tr>
<td>2</td>
<td>54</td>
<td>M</td>
<td>1</td>
<td>2</td>
<td></td>
<td>13</td>
<td>Alive</td>
<td>56</td>
<td>294</td>
</tr>
<tr>
<td>3</td>
<td>55</td>
<td>M</td>
<td>1</td>
<td>1</td>
<td>1/1</td>
<td>25</td>
<td>Alive</td>
<td>51</td>
<td>495</td>
</tr>
<tr>
<td>4</td>
<td>59</td>
<td>M</td>
<td>1</td>
<td>2</td>
<td></td>
<td>27</td>
<td>Alive</td>
<td>131</td>
<td>901</td>
</tr>
<tr>
<td>5</td>
<td>49</td>
<td>F</td>
<td>-</td>
<td>2</td>
<td></td>
<td>37</td>
<td>Heart failure</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>6</td>
<td>53</td>
<td>M</td>
<td>3</td>
<td>-</td>
<td></td>
<td>-</td>
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</tr>
<tr>
<td>7</td>
<td>57</td>
<td>M</td>
<td>1</td>
<td>-</td>
<td></td>
<td>6</td>
<td>AM*</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>8</td>
<td>61</td>
<td>M</td>
<td>1</td>
<td>1</td>
<td></td>
<td>9</td>
<td>Heart failure</td>
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<td>9</td>
<td>66</td>
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<td>AM*</td>
<td>81</td>
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<tr>
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<td>71</td>
<td>M</td>
<td>1</td>
<td>2</td>
<td></td>
<td>12</td>
<td>VF</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

*Confirmed at necropsy; †Sudden death.

CK-B, creatine kinase isoenzyme B; LD-1, lactate dehydrogenase; VF, Ventricular fibrillation.

STATISTICAL ANALYSIS

The groups with and without acute myocardial infarction were compared by rank sum test (Mann-Whitney) and observations before and with SCS were evaluated by the Wilcoxon/Pratt test. Differences were regarded as significant if \( P < 0.05 \).

Results

We evaluated 108.6 patient-years of SCS treatment. During the observation period 10 patients were diagnosed as having an acute myocardial infarction while they were being treated with SCS: six of them died (table 2). Two patients died of other causes: one of pneumonia and one of progressive heart failure.

In the four survivors and one of the patients who died acute myocardial infarction was verified by serum enzyme concentrations; the other patients died before a blood sample was taken. In an additional patient acute myocardial infarction was verified at necropsy: the diagnosis was based on clinical observations in the remaining four patients. Two of these died in intractable ventricular fibrillation, and two patients in heart failure died suddenly. Nine of the ten patients with acute myocardial infarction recognised that the precordial pain during their acute myocardial infarction was being clearly different and definitely more severe than their usual angina and that it was not alleviated by SCS. Furthermore, the pain at acute myocardial infarction was accompanied by unusual symptoms such as dyspnoea, general weakness, etc. Patients with previous myocardial infarction reported that some symptoms were the same as at the last myocardial infarction.

One patient (case 7) died suddenly; it is not known whether he experienced any chest pain. He was admitted to hospital for a transurethral resection of the prostate and was well when he was given premedication. About one hour later he was found dead in the ward. Acute myocardial infarction was verified at necropsy (table 2). One patient refused hospital admission; she was treated at home by her general practitioner and died suddenly in heart failure. This death was attributed to AMI (case 5, table 2).

During the observation period 37 of the 50 patients were admitted to hospital. During the observation period there were 153 admissions. The mean number of admissions for chest pain or angina or observation for acute myocardial infarction during the 3 year period before SCS treatment and during the period with SCS treatment were not significantly different (\( P = 0.6 \)) in the 10 patients with acute myocardial infarction and the patients without (table 1).

During SCS treatment about 90% of the visits to the casualty ward or the general practitioner were for prescriptions or socio-economic problems or both. The median number of contacts with a physician was one per month. There were, however, large variations between individual patients (0–30 per month).

During the follow up period five patients had their antianginal medication adjusted. One patient had CABG because coronary angiography showed occlusion of a previously inserted bypass graft.

The symptomatic relief achieved by SCS treatment (assessed from the reduction in opiate consumption) was significant; there was, however, no difference between the 10 patients with acute myocardial infarction and the others (table 1). Forty three of the 50 patients reported that SCS treatment reduced angina. All 10 patients who had an acute myocardial infarction during SCS treatment claimed to have a considerable reduction in angina.

There were no differences in the number of previous acute myocardial infarctions or in the number of bypass operations between the ten patients in the acute myocardial infarction-group and the other 40 patients (table 1).

The electrocardiograms recorded before SCS were abnormal in most of the patients, with a high incidence of Q wave and ST-T abnormalities (table 3). In the patients without acute myocardial infarction a comparison of the follow up electrocardiograms with those recorded before SCS showed no significant changes in Q wave, T wave inversion, or new bundle branch block (table 3). In the group with acute myocardial infarction follow up electrocardiograms could be recorded only in the four patients who survived: one patient developed bundle branch block, two had T wave changes, and in one no changes were seen in the electrocardiogram. The four patients who survived their acute myocardial infarction have not been admitted to hospital since.

Discussion

Recognition of sympathetic afferent fibres as the pathway for cardiac pain led to efforts to
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Br Heart J 1994 71: 419-421
doi: 10.1136/hrt.71.5.419

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