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Case Report

The successful use of spinal cord stimulation to alleviate intractable angina pectoris

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The first reported use of electricity for its numbing effect was in AD 46 by a Roman.[1,2] Since then electro analgesia has been used in the USA in the 1850s and peripheral nerve stimulation has been used sporadically since. In 1965 Shealy and colleagues demonstrated that spinal cord stimulation (SCS) in cats blocked behavioural responses to acute pain,[3,4] and the same group subsequently implanted a spinal cord stimulator in a patient in 1967, reducing his chest wall cancer pain.[5] With these results SCS has been used with mixed results in a variety of pain conditions. In 1984, Sandric and colleagues reported that patients undergoing SCS for various conditions who also had ischaemic heart disease showed improvements in their ECG, and two had no further angina.[6] Murphy and Giles (1987) were the first to report the technique being used specifically for angina.

This is the first reported use of a Medtronic spinal cord stimulator for angina in Northern Ireland.

CASE REPORT A 52-year-old man with intractable angina was referred. He had two acute myocardial infarctions and coronary artery bypass grafting (CABG) in March 1989. There was a gradual return of angina since 1993. There was further angiographically demonstrated coronary artery disease progression and so CABG was performed again in 1995 but it was not possible to bypass all the occlusions. Subsequently he had frequent episodes of angina, requiring hospital admission for analgesia and nitrate infusions for a total of 78 days over the previous calendar year.

METHOD

The patient was given pre-medication of clonazepam 1mg. orally 2 hours prior to the procedure. Continuous ECG, blood pressure and S_pO_2 were monitored throughout and he received oxygen by face mask during the insertion. The epidural space was identified with fluoroscopic assistance using loss of resistance to air with a wingless 16 guage epidural needle with a 7.5mm terminal orifice (to allow advancement and withdrawal of the electrode without danger of electrode damage). The needle tip was placed in the mid-line of the epidural space. The electrode was advanced under x-ray control so that its tip lay just to the left of the midline at C7 and a stimulation trial carried out (the wire has 4 electrodes (0,1,2,3) each of which can be positive or negative, on or off, allowing variable stimulation over a fairly wide area). The aim was to achieve stimulation (paraesthesia) over the area where angina was felt; this was achieved. When satisfactory stimulation was achieved a 2cm incision through skin was made caudal and cephalad to the epidural needle, a subcutaneous pocket created, needle withdrawn ensuring no movement of the electrode wire and the wire end attached to a subcutaneously tunnelled cable which emerged 10cm from the midline and attached again to the external hand held stimulator. After 4 days of trial stimulation the patient reported no episodes of angina and an increased exercise tolerance, so a permanent receiver was implanted subcutaneously. The patient was further instructed in how to use the stimulator.

RESULTS

Over a 36-week follow-up period the patient has had angina on a much reduced frequency and on one occasion it was found that the electrode wire had migrated cephalad, and after suitable withdrawal satisfactory stimulation had been re-achieved his angina was reduced again to its new

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much reduced state. His exercise tolerance has also increased. His frequency of hospital admission has also been reduced to 3 admissions totalling 9 days over an 8 month period.

COMMENT

The mechanism of pain relief by SCS is unknown.[8] One concern is that angina, (a potential warning signal) of impending myocardial ischaemia) would be masked. In a series of 50 patients with intractable angina in whom spinal cord stimulation was performed for 1-57 months, ten subsequently died due to acute myocardial infarction.[9] In nine spinal cord stimulation did not conceal precordial pain and in one no information about precordial pain could be obtained.[9] In other series continuous ECG monitoring has demonstrated less ischaemic changes during exercise in those with SCS and intractable angina when compared to a non stimulated group,[10, 11, 12] and furthermore, less ischaemia provoked by increased pacing frequency in those with concomitant pace makers in situ when compared to a control group.[13] When monitored electrocardiographically after adenosine infusion, decreases in left ventricular ejection fraction were less marked in the SCS group when compared to those unstimulated.[14] It does therefore seem that the effect of SCS is more than a simple masking effect and may actually improve myocardial oxygen supply thereby reducing angina.

SCS is expensive. The price of an electrode and receiver is around £5,300. However, it reduced suffering and hospital admissions in this case. Hopefully this case has highlighted potentially useful analgesic effects of SCS in a patient with intractable angina.

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