

# Spinal Cord Stimulation for Intractable Angina Pectoris

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The successful use of spinal cord stimulation (SCS) in patients with stable angina pectoris refractory to medical management who are poor candidates for percutaneous or surgical angioplasty has been well described (1). Janfaza et al. (2) recently described a critically ill patient with unstable angina pectoris who was treated with SCS. The patient's condition improved and stabilized, and the patient was discharged from the hospital and survived for at least 6 wk. To the best of our knowledge, there are no other reports of the use of SCS in such critically ill patients. We describe two similar patients treated with SCS but with different outcomes.

## Case Report 1

A 79-yr-old man was transferred to the cardiology service for treatment of intractable angina pectoris. He had experienced increasingly frequent and severe substernal chest pain, dyspnea, and diaphoresis worsened by mild exertion and mental stress for 8 days before this transfer. His electrocardiogram (EKG) showed new ST-segment depression in leads V3–V6. He was receiving IV heparin, nitroglycerin, and morphine in addition to oral metoprolol and nifedipine at the time of admission. An urgent cardiac catheterization showed severe three-vessel disease in addition to an 80% left main occlusion.

The patient had suffered two previous myocardial infarctions and a cerebrovascular accident (CVA), and he had bilateral 100% carotid artery occlusions. He had a medical history of noninsulin-dependent diabetes mellitus, hypertension, severe peripheral vascular disease, and a 4.0-cm abdominal aortic aneurysm.

Despite aggressive medical management, the patient continued to have severe and protracted episodes of chest pain at rest. His lesions were not amenable to percutaneous angioplasty, and he declined coronary artery bypass grafting because of the high risk of recurrent CVA. He decided not to be resuscitated or intubated in the event of a sudden decline in his status.

On the 10th day after admission, the acute pain service was consulted. After a thorough discussion with the patient

and his family regarding the risks, potential benefits, and uncertainty about the efficacy of the procedure under the current circumstances, the patient and his family agreed to a trial of SCS. The patient's heparin was stopped for 4 h, and coagulation variables were determined to be normal.

The patient was taken to the operating room, monitored, lightly sedated by an anesthesiologist, and placed in the prone position. Anesthesia was accomplished by local anesthetic infiltration. A 15-gauge Tuohy needle was inserted at the T4-5 interspace. Position was identified and confirmed fluoroscopically throughout the procedure. The epidural space was identified using a loss-of-resistance to air technique. A Pisces Quad-Plus Electrode (Medtronic, Inc., Minneapolis, MN) was inserted into the epidural space and advanced under fluoroscopic guidance until the tip lay at the C7-T1 interspace and approximately 3 mm to the left of midline. A trial paresthesia was found to encompass the entire area of anginal pain. Electrode configuration was 0 = off; 1 = positive, 2 = negative, 3 = off. Pulse width was 300 ms and rate was 50 Hz. The threshold for paresthesia was 1.5 V, and the maximally tolerable paresthesia was 1.9 V. The procedure was well tolerated by the patient.

Stimulation was kept constant at 1.6 V; if the patient had an episode of angina, the voltage was increased to 1.9 V until it resolved. The patient's IV medications were discontinued. Oral nitrates and morphine sulfate were used as needed. The patient had fewer and less severe episodes of angina. He was transferred out of the coronary care unit (CCU).

Three days after placement of the trial electrode, the patient experienced worsening angina unrelieved by SCS, and he was transferred back to the CCU. IV heparin was resumed. His EKG indicated new ischemic changes, and he had signs and symptoms of congestive heart failure. His chest pain required SCS and IV morphine and nitroglycerin for control. On the fifth day after SCS implantation, the patient died quietly.

## Case Report 2

An 86-yr-old woman was admitted with a 1-wk history of uncontrolled left shoulder pressure radiating into her left arm and hand. At the time of admission, she was receiving metoprolol, amlodipine, and ticlopidine. She was treated with IV heparin, nitroglycerin, and morphine.

The patient had a history of sinus node disease and had a dual-pacing, dual-sensing, dual-mode pacemaker. She had previously suffered a CVA and a myocardial infarction and had undergone percutaneous coronary angioplasty. She had a history of hypertension and peripheral vascular disease.

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An urgent cardiac catheterization and an attempt at revascularization via percutaneous angioplasty resulted in closure of the right coronary artery. The patient's creatinine phosphokinase level was increased, consistent with an acute myocardial infarction. She decided not to be resuscitated or intubated in the event her condition worsened. She was deemed to have a high risk of death and stroke with surgical bypass, and the acute pain service was consulted about the implantation of a SCS device.

Informed consent was obtained, and the device was implanted as with the previous patient. Electrode configuration was 0 = off; 1 = positive, 2 = negative, 3 = off. Pulse width was 200 ms and rate was 80 Hz. The threshold was 2 V, and the patient's maximally tolerated voltage was 2.1 V. Paresthesia covered her entire painful area.

IV medications were gradually discontinued. The patient had fewer episodes of angina, which were all easily aborted with SCS. Four days after her trial electrode was placed, the patient was returned to the operating room, and an Itriel 2 generator (Medtronic, Inc., Minneapolis, MN) was implanted into the right buttock area. She was transferred out of the CCU. The following evening, she developed palpitations while her SCS device was on at the threshold voltage, and an EKG showed ventricular tachycardia. The patient declined any treatment and subsequently developed left shoulder pain, which was incompletely but adequately relieved with her SCS device increased to 2.1 V. She became unconscious and died approximately 10 min later.

## Discussion

Both of these patients had symptoms of ischemia when their cardiac status deteriorated. This supports the work of other investigators who have shown that SCS does not mask the symptoms of ischemia but improves the function of the heart (3,4). There is a single report of interference between an unipolar pacemaker and unipolar SCS (5). The safe use of a dual-pacing, dual-sensing, dual-mode pacemaker in conjunction with quadripolar SCS electrodes has been described (6). It is unlikely that the arrhythmia observed in our second patient was the result of pacemaker interference, although it is possible.

Janfaza et al. (2) reported the six-week survival of a single critically ill patient treated with SCS. Our cases suggest relief of pain but no, or minimal, survival advantage in this critically ill population. Although both patients experienced improvement of their anginal symptoms with SCS, the effect was not sufficient to overcome the severity of their ischemic disease.

There is evidence demonstrating pain relief, low morbidity, and improved function of the heart in patients with stable angina treated with SCS (1). Our patients had unstable angina and were considered terminally ill, with a life expectancy estimated in days to weeks. It may be that SCS is not indicated in end-stage unstable angina because of its inability to prolong life, as demonstrated in the patients presented, but this remains undetermined. The precise niche for SCS in the treatment of angina is still being investigated.

## References

1. Eliasson T, Augustinsson L-E, Mannheimer C. Spinal cord stimulation in severe angina pectoris: presentation of current studies, indications and clinical experience. *Pain* 1996;67:169-79.
2. Janfaza DR, Michna E, Pisini JV, Ross EL. Bedside implantation of a trial spinal cord stimulator for intractable anginal pain. *Anesth Analg* 1998;87:1242-4.
3. Sanderson JE, Brooksby P, Waterhous D, et al. Epidural spinal electrical stimulation for severe angina: a study of its effects on symptoms, exercise tolerance and degree of ischemia. *Eur Heart J* 1992;13:628-33.
4. Anderson C, Hole P, Oxhoj H. Does pain relief with spinal cord stimulation for angina conceal myocardial infarction? *Br Heart J* 1994;71:419-21.
5. Romano M, Zucco F, Rota Baldina M, et al. Technical and clinical problems in patients with simultaneous implantation of cardiac pacemaker and spinal cord stimulation. *PACE* 1993;16:1639-44.
6. Romano M, Brusa S, Grieco, et al. Efficacy and safety of permanent cardiac DDD pacing with contemporaneous double spinal cord stimulation. *PACE* 1998;21:465-7.