Advances in Spinal Cord Stimulation

SPINAL CORD STIMULATION is a technique in which electrical stimulation is applied posteriorly to the spinal cord. It was first employed 30 years ago to manage chronic pain limited to several contiguous spinal segments. Although spinal cord stimulation is nondestructive and reversible, because of technical limitations, it did not gain wide acceptance until this decade. Recent improvements in equipment and in patient selection have decreased the risk of complications and improved efficacy.

Percutaneous placement now permits lead positioning without general anesthesia. Patients then provide important feedback during lead placement, enhancing the ability to locate the precise area of the cord responsive to stimulation. Percutaneous lead placement also permits a trial stimulation without committing the patient to incision for the introduction or removal of equipment.

Using pacemaker technology, internal pulse generators were developed for a totally implanted, self-contained system programmed externally. Alternatively, internal pulse receivers are implanted and powered by radiofrequency through a 7.6-cm (3-in) antenna above the skin, a system that does not require surgical intervention when the battery fails. Multilead systems allow the delivery of complex stimulation patterns for patients who are unresponsive to stimulation by a single lead. Flat, wide leads for more varied stimulation arrays and wider areas of coverage can be placed by laminectomy.

Patient selection is an essential component of managing pain syndromes with spinal cord stimulation. Patient suitability is assessed by psychometric testing, such as a specially adapted, validated version of the Minnesota Multiphasic Personality Inventory. A multidisciplinary patient evaluation and refined trial techniques allow a better prediction of which patients are likely to benefit from the procedure.

Spinal cord stimulation is also an effective adjuvant to standard therapies for refractory angina pectoris. Epidural leads are placed near T-1, and stimulation produces paresthesias in the aching area. Spinal cord stimulation substantially improves exercise capacity and quality of life while reducing the number of anginal attacks, ischemic electrocardiographic signs, and nitrate consumption. There is no evidence that it conceals the signs of acute myocardial infarction. In Europe, this technique is a routine supplement to conventional medical and surgical therapies for angina.

Similarly, spinal cord stimulation produces an antiischemic effect in peripheral arterial and severe vasospastic disease of the limb, including that of patients refractory to standard medical and surgical therapies. Patients with residual vascular compliance are most likely to respond to the procedure. In patients with peripheral vascular disease, spinal cord stimulation may increase exercise tolerance, aid in the healing of ischemic ulcers, and increase microvascular flow. Despite this, the primary target of spinal cord stimulation therapy in the United States in patients with peripheral vascular disease remains the management of pain associated with the disease.

In summary, pain from ischemic vascular diseases and chronic pain from syndromes such as the failed back, phantom limb, and complex regional pain syndrome I (reflex sympathetic dystrophy) can be effectively managed with spinal cord stimulation. To date, there have been about 100,000 implantations of spinal cord stimulation. It is likely that the use of the procedure will increase in the future because of improved efficacy and the increased number of applications.

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Use of β-Blockade to Prevent Death After Noncardiac Surgery

IN THE UNITED STATES each year about 30 million patients have noncardiac operations. Of these, about 1 million have diagnosed coronary artery disease, 2 to 3 million have two or more major risk factors for coronary artery disease, and another 4 million are older than 65. Despite advances in the diagnosis and treatment of coronary artery disease, the perioperative morbidity and mortality in this group remain high. The incidence of intraoperative ischemia is between 20% and 63%, and that of postoperative infarction can be as high as 37% with an associated mortality of 40% or higher. Of all possible predictors of an adverse outcome, postoperative ischemia has been identified as the most important, conferring a ninefold increase in the odds of having cardiac death, myocardial infarction, or unstable angina and a twofold risk of long-term sequelae. Thus, efforts at reducing adverse cardiac outcomes have concentrated on the preoperative evaluation and on reducing the incidence of postoperative ischemia.

In a number of studies, the effects of techniques for reducing perioperative myocardial ischemia, a possibly reversible cardiac risk factor, have been examined: anesthetics, postoperative sedation, prophylactic nitrates, calcium channel blockers, and β-blockers. Of these, intensive postoperative sedation, β-blockade, α_2-agonists, and adenosine analogues have shown reductions in the incidence or severity of perioperative myocardial ischemia. Until now, however, none of the clinically available therapies have shown a difference in mortality.

Recently a 200-patient, randomized, placebo-controlled, clinical trial showed that the prophylactic perioperative administration of atenolol reduced mortality after discharge from the hospital. The major reduction in the number of deaths from cardiac causes occurred during the first six to eight months (0% versus 8%, $P < .001$).

Moreover, mortality during the first postoperative year (3% versus 14%, $P = .005$) and two years (10% versus 21%, $P = .019$) was also reduced. Cardiovascular event-free survival during the two-year follow-up was improved by the perioperative administration of atenolol (68% versus 83%, $P = .008$). This study is important because it provides a simple, safe, inexpensive therapy for preventing perioperative myocardial ischemia and also reducing mortality. Before this study, risk factor identification followed by coronary revascularization was the only option. Patients who were found to require coronary revascularization before a noncardiac operation incur the risk of two operations rather than a single one. Now a medical regimen can reduce the morbidity and incidence of cardiac death that can be associated with noncardiac surgical procedures.

Who should receive prophylactic perioperative β-blockade? Any patient who is already on β-blockade therapy should continue to receive it. In addition, any patient qualifies who has known coronary artery disease as shown by a previous myocardial infarction, typical angina, or atypical angina with electrocardiographic changes indicating ischemia in response to exercise or scintigraphic evidence of a myocardial perfusion defect. Moreover, any patient qualifies who is at risk for coronary artery disease because of previous or current vascular operations or the presence of at least two of the following risk factors (in addition to male sex): age 65 years or older, hypertension, current smoking, serum cholesterol level of 6.20 mmol per liter (240 mg per dl), or diabetes mellitus. Who should not be given prophylactic perioperative β-blockade? Anyone with a known sensitivity to β-blockers, acute congestive heart failure, acute bronchospasm, third-degree heart block without a pacemaker, a heart rate below 55 beats per minute, or a systolic blood pressure below 100 mm of mercury should not receive this therapy. Care should be taken when administering it to patients with a history of asthma.

What types of operations qualify for prophylactic perioperative β-blockade? All patients scheduled to undergo a major noncardiac operation requiring general anesthesia and a hospital stay qualify. It is unclear if patients scheduled for minor operations should receive this therapy.

How long does a patient need to be on prophylactic perioperative β-blocker therapy? Patients were administered atenolol for seven days postoperatively. Episodes of myocardial ischemia are most frequent during the first 48 hours postoperatively, but can continue for at least seven days. The optimum duration of therapy is unknown, but seven days should be considered a minimum. Furthermore, patients with a known history of coronary artery disease may benefit from indefinite β-blockade.

What drug should be used? Atenolol was chosen because it was a long-acting drug that had proven efficacy in preventing death after myocardial infarction. Other drugs such as metoprolol tartrate have similar efficacy. It is unclear if a short-acting drug such as esmolol hydrochloride would have similar effects. Notably, the use of esmolol would increase the cost and complexity of administration. How should the drug be administered? An intravenous dose of atenolol of 5 to 10 mg should be administered 30 minutes before the surgical procedure while monitoring the blood pressure and heart rate. A second dose of 5 to 10 mg of intravenous atenolol should be administered postoperatively. If the patient is not to take food or liquids orally, intravenous administration should be continued twice a day. Once the patient is able to take oral medications, oral atenolol, 50 or 100 mg, with the dosage guided by the heart rate and blood pressure, can be used. It is important to continue administering the β-blocker for a full seven days postoperatively. It is unclear if an oral dose of atenolol given in the immediate preoperative period will be adequately absorbed in time to provide optimal protection.

The difficulty with adopting prophylactic perioperative β-blockade for patients at risk for perioperative cardiac morbidity and mortality will be the postoperative administration of the drug. This therapy requires prolonged β-blockade to prevent postoperative myocardial ischemia. It will be important to convince our surgical colleagues to continue this therapy into the postoperative period. The clinical practice of avoiding postoperative β-blockade so that tachycardia can guide volume replacement is clearly detrimental to the long-term survival of patients at risk for cardiac morbidity. Controlling hemodynamics during the operative and postoperative period is critical to avoiding perioperative cardiac morbidity and death.

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'Practice Guidelines for Blood Component Therapy' Summarized

DESPITE THE DECREASING potential for blood components to transmit viral disease, remaining concern (and concern regarding other possible complications: transfusion reactions, the transmittal of bacterial or parasitic diseases, immunosuppression, and cost) has prompted several professional societies and governmental organizations and regulatory bodies to produce "guidelines," "strategies," and "practice parameters." These documents have not addressed the specific needs of anesthetized patients, however. The American Society of Anesthesiologists responded to this need by convening a task force in 1994 that produced an evidence-based document, "Practice Guidelines for Blood Component Therapy." The panel included community- and university-based anesthesiologists, representatives from other specialty organizations, and a methodologist. After review of the rele-