Immediate effects of spinal cord stimulation in spinal spasticity

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Six patients with intractable spasms after spinal cord injury underwent implantation of an epidural spinal cord stimulation system. All the patients experienced good relief postoperatively. In three patients spinal cord stimulation consistently produced immediate inhibition of the spasms. This was evident within less than 1 minute of stimulation. Conversely, the spasms reappeared within less than 1 minute after cessation of the stimulation. The clinical observations were confirmed by polygraphic electromyographic recordings.

KEY WORDS spinal cord injury • spasticity • muscle spasm • spinal cord stimulation • myelopathy

Spinal cord stimulation (SCS) is a currently accepted treatment for some intractable painful conditions. Several reports have underscored the positive effects of SCS in some motor disorders. The primary indication for SCS for motor disorders has been multiple sclerosis; however, the use of this therapy for this condition has been controversial. Waltz reported good results with SCS in some extrapyramidal involuntary motor disorders, and Gildenberg had favorable effects in spasmodic torticollis. A few authors have reported on the effectiveness of SCS in spasticity.

At our institution we have implanted epidural SCS systems for the management of spasms in six patients with spinal cord injuries. The goal of this paper is to present objective evidence of some of the immediate effects that SCS has on spasms in patients with spinal spasticity. We define as "immediate" those effects occurring within seconds or minutes after the onset or cessation of SCS. Only three of our patients experienced immediate effects related to SCS.

Clinical Material and Methods

Six patients with handicapping spasms following spinal cord injury had an SCS system surgically implanted.* The patients ranged in age from 20 to 60 years. Trauma had occurred 9 months to 7 years prior to implantation. Five patients had an incomplete quadriparesis secondary to a cervical injury, but four of them were able to walk. One had a complete paraplegia at T-1. All six patients had severe spasms of the upper and/or lower limbs which significantly interfered with their rehabilitation. All the patients had previously undergone extensive physical therapy and medical trials with antispastic medications.

The implants were performed in the operating room under local anesthesia and under fluoroscopy. The electrode lead was made of a filament wire (MP-35 alloy of chromium, cobalt, and nickel) insulated with silicone rubber tubing; 5 mm of the filament was exposed at the tip. The monopolar cathode was inserted in the dorsal epidural space through a thin-walled No. 16 Tuohy needle. The needle was inserted through a midline approach at the T8–12 level. The electrode tip was placed at the lowest segment at which intraoperative SCS could elicit paresthesias in all four extremities. This usually resulted in a midline placement at the T1–2 area. In the patient with a T-1 paraplegic level the electrode tip was placed in the ventral epidural space. The radio receiver and the positive electrode were placed in a subcutaneous pocket in the flank. The internal system was activated through an external radio transmitter connected to an antenna applied to the skin around the receiver. The receiver produced a capacitatively coupled rectangular pulse.

* Spinal cord stimulation system manufactured by Clinical Technology Corp., Kansas City, Missouri.
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The transmitter produced a biphasic rectangular pulse (250 kHz). All the systems were inserted at a single procedure. Stimulation was started in the immediate postoperative period and carried out for several hours daily. Some patients, namely the ones who experienced immediate effects, preferred to have the stimulation on for most of the time, since this was the only way to keep the spasms under continuous control. The frequency of SCS was in the 75 to 100 Hz range with a pulse width of 100 to 250 μsec. The stimulus amplitude was set to a level where the subject felt paresthesias without direct motor effects. In the patient with complete paraplegia, the therapeutic SCS amplitude was set at a level below that which produced direct motor stimulation.

Pre- and postoperative evaluation included polygraphic electromyography (EMG) recording of the spasms, measurement of the torque and displacement generated during isokinetic voluntary movement and/or spasms, H reflex and H excitability curve, urodynamical studies, and videotape recordings.

Results

Summary of Cases

In three patients SCS produced an immediate significant reduction of the spasms. Good reduction of the spasms was also obtained in the other three patients; in these cases, however, a significant reduction of the spasms occurred only after a few hours of stimulation. Where immediate effects occurred, they could be demonstrated within seconds. These included cessation of the spasms a few seconds after the onset of stimulation and prompt recurrence when the stimulation was interrupted. A short case report of the three patients who achieved immediate relief from SCS is given.

Case Reports

Case 1. This 65-year-old man had an immediate SCS effect on abdominal muscle spasms triggered by cutaneous stimulation. He had sustained traumatic T-1 paraplegia in 1980. Shortly after the injury he developed spasms affecting the abdominal, trunk erector, hip flexor, adductor, and hamstring muscle groups. A bilateral section of the iliopsoas muscle, femoral, and obturator nerves provided good relief from the spasms in the lower limbs. The truncal and abdominal spasms were not affected, however, and remained severe and frequent.

The SCS electrode was placed at the midline in the ventral epidural space at T-5. When SCS was applied the spasms completely disappeared. The effects of SCS occurred within seconds but also disappeared within seconds when the stimulation was interrupted. Figure 1 upper shows an EMG recording with surface electrodes from the abdominal muscles. Cutaneous stimulation of the thigh was applied so as to trigger a severe spasm and was then carried out continuously throughout the test. When the spasm was severe and sustained, SCS was applied. This produced immediate cessation of the spasm. When the stimulation was stopped, the spasms reappeared immediately. There was no spasm while the SCS was on (Fig. 1 lower). When the pulse amplitude was decreased, a spasm appeared in the abdominal muscles. The spasm was maximal when spinal stimulation was interrupted, and stopped within seconds when SCS was resumed. The effects were unchanged at a 6-month follow-up examination.

Case 2. This 25-year-old man had an immediate SCS effect on extensor spasms of the lower extremities triggered by proprioceptive stimulation (knee tap). He had suffered quadriplegia after a C-5 fracture in 1978. The patient had a clinical picture of a central cord syndrome associated with Brown-Séquard components. He had good strength in the right limbs and could walk with the help of crutches. Severe extensor spasms affected the lower limbs and, to a lesser extent, the upper limbs. The spasms were severe and frequent enough to make walking very difficult. The SCS electrode was placed in the dorsal epidural space at the T-1 level at the midline.

FIG. 1. Electromyographic recordings from the abdominal muscles in Case 1. ON/OFF (small arrows) = cutaneous stimulation given to trigger spasms. Cutaneous stimulation is being given continuously throughout the recording. ON/OFF (large arrows) = spinal cord stimulation (SCS). The thick line on the spinal stimulation tracing is an artifact from the electrical stimulation. Upper: Cutaneous stimulation triggers a spasm (A) which is abolished about 3 seconds after SCS is applied (B). The spasm reappears promptly when SCS is stopped. The baseline artifact is due to the SCS. Lower: The spasm of the abdominal muscles (A) starts when the stimulation (B) is tapered (at C) and is maximum when the stimulation is off.
The deep-tendon reflexes were hyperactive. Figure 2 shows the EMG recording of a knee jerk with the patient in the sitting position. An after-discharge contraction of the quadriceps femoris and of the hamstrings followed the knee jerk. Stimulation applied for a few seconds caused the after-discharge to disappear in the quadriceps and to decrease in the hamstrings.

The effects of SCS with the patient in the supine position, thus increasing the stretch of the quadriceps, are shown in Fig. 3. While the SCS was on, the knee tap elicited a brisk monosynaptic reflex with no after-discharge and minimal activity on the hamstrings (Fig. 3-1). Stopping the stimulation caused progressively increasing excitability of the spinal cord; a long-lasting spasm of the quadriceps and hamstrings followed the monosynaptic component of the knee jerk when the SCS had been off for 60 seconds.

Figure 4 shows the same pattern in a continuous isokinetic dynamometer recording of the torque and angle generated at the knee. The peaks in the torque tracing are due to successive knee taps given to elicit a patellar reflex. Without SCS, a knee tap caused a prolonged extensor spasm, as shown by the prolonged downward deflection of the knee angle. With SCS, the knee taps elicited a few beats of clonus but there was no extensor spasm. When SCS was stopped, extensor spasms reappeared in response to successive taps after about 30 seconds.

At his 6-month follow-up examination, the patient still experienced immediate effects with SCS. The overall level of excitability of the spasms, however, had significantly decreased.

Case 3. This 20-year-old man had relief of extensor spasms of the lower extremities triggered by cutaneous
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stimulation. He had suffered C-5 quadriparesis second-
ary to a C-5 compression fracture 9 months prior to
implantation of the SCS system. He had only a trace
of movement in the toes and no other voluntary move-
ment below the level of the injury. Deep touch and
position sense were intact, while pain and temperature
sensation were abolished.

In the first 2 months after the injury the patient
developed extremely severe spasms affecting all four
limbs and the abdominal and trunk muscles. The
spasms were consistently extensor in the upper limbs
but alternating flexor or extensor in the lower limbs.
The spasms had an extremely low threshold and lasted
several minutes. The effects of SCS on a spasm triggered
and maintained by cutaneous stimulation of a lower
limb are shown in Fig. 5. The spasm was completely
inhibited within a few seconds after the onset of stim-
ulation and recurred in the hamstrings immediately
after cessation of SCS. The effects were unchanged at a
6-month follow-up examination.

Discussion

Our experience shows that, in selected cases, epidural
SCS does produce significant immediate inhibitory ef-
fects on spinal spasms. In such cases there is an imme-
diate direct correlation between the onset or cessation
of SCS and the inhibition or reappearance of the
spasms. These effects have been observed in patients
with clinically complete and incomplete spinal cord
injury.

The exact mechanisms of action of SCS are presently
unclear. In spinal spasticity the reticulospinal pathways
that control the segmental and supraspinal input to
the alpha and gamma motor neurons are interrupted
to various degrees, according to the extent and topo-
ographical location of the lesion. The resulting imbalance
is responsible for the hyperactivity of the segmental
spinal circuits as seen in spinal spasticity. Spinal
cord stimulation brings on a powerful inhibition of the
hyperexcitable spinal circuits which, in some cases, has
immediately evident clinical effects. This could be due
to direct activation of the descending inhibitory retic-
ulospinal pathways or it could take place through long-
loop mechanisms. The activity of SCS is not limited to
the level of stimulation, but spreads both cranially and
caudally within the spinal cord. In Case 3 we observed
marked reduction of extensor spasms in the upper
extremities with the electrode tip at T-3.

The amount of current necessary to produce clini-
cally evident effects must be such that the stimulation
spreads to the dorsal columns and/or to the dorsal part
of the gray matter of the spinal cord. The best results
are obtained when the patients feel paresthesias in all
those areas where the spasms are to be affected. If the
current is increased to where direct stimulation of the
motor tracts occurs, motor contractions at segmental
and supraspinal levels are produced. This is usually
of no advantage to the patient. In some cases the
threshold between the therapeutic level and the motor
stimulation level can be very narrow. In Case 2 a pulse
width of 250 μsec elicited contraction of the abdominal
muscles and paresthesias at the same threshold of stim-
ulation. Only by reducing the pulse width to 100 μsec
were we able to consistently elicit paresthesias without
direct motor effects.

Fig. 4. Patellar reflexes with the hips extended and the
knees flexed 90° in Case 2. Continuous recording with the
Cybex II isokinetic dynamometer during successive knee taps
(peaks on the torque recording). Downward deflection in the
knee angle tracing = extension of the knee. When spinal cord
stimulation (SCS) is off the first knee tap triggers an extensor
spasm. Knee taps when the SCS is on elicit brisk knee jerks
but no spasms. Spasms recur about 30 seconds after cessation
of SCS and progressively increase in intensity.
FIG. 5. Electromyographic recording in Case 3 showing extensor spasm of the lower extremity triggered by cutaneous stimulation. A = angular displacement of the knee (upward deflection indicates extension); Q = quadriceps; and H = hamstrings. ON/OFF (small arrows) = cutaneous stimulation given to trigger spasms; ON/OFF (broad arrows) = spinal cord stimulation (SCS). Immediate cessation of the spasm is seen on starting SCS. The spasm reappears in the hamstrings when SCS is stopped.

Prior to implantation of the SCS system, all the patients in this study had undergone extensive trials with antispastic drugs and physical therapy. The effects of SCS on the spasms in these cases compared favorably with the acute effects of a dorsal rhizotomy or myelotomy. The significant advantage is that SCS is not a destructive procedure and that, at least in short-term follow-up studies, its effects can be tailored to individual needs. If the long-term follow-up results show the persistence of these results, SCS might become the procedure of choice for spinal spasms that do not respond to noninvasive measures.

References

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