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Effect of Subarachnoid Catheter Position on the Efficacy of Intrathecal Baclofen for Spinal Spasticity

Paul G. Loubser, M.B., Ch.B., Raj K. Narayan, M.D.†*

* Associate Professor, Department of Anesthesiology, The University of Texas Health Science Center Medical School; Director of Anesthesiology Services, The Institute for Rehabilitation and Research.

† Associate Professor, Department of Neurosurgery, Baylor College of Medicine.

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Address reprint requests to Dr. Loubser: Department of Anesthesiology, Baylor College of Medicine, 6550 Fannin, Suite 1003, Houston, Texas 77030.

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SEVERAL studies have demonstrated that intrathecal baclofen reduces spasticity secondary to spinal cord injury.[1–4] Long-term control of spasticity is achieved by continuously infusing baclofen using a surgically implanted infusion pump and subarachnoid catheter. Pharmacologic agents that are administered intrathecally migrate in a rostral direction, following cerebrospinal fluid (CSF) flow patterns. However, it has been unclear whether the position of the spinal catheter orifice (site of drug delivery) has any influence on the degree of spasticity reduction. Previously, most experience with intrathecal baclofen has been limited to delivery of drug caudad to the spinal cord injury. Theoretically, delivery of intrathecal baclofen cephalad to the spinal cord injury should also be associated with significant spasticity reduction, because suprasegmental (above the spinal cord injury) excitatory reflexes

Table 1. Ashworth Scale

Grade	Degree of Muscle Tone
1	No increase in tone
2	Slight increase in tone, giving a "catch" when affected part is moved
3	More marked increase in tone, but affected part easily flexed
4	Considerable increase in tone, but passive movement difficult
5	Affected part rigid in flexion or extension

Total grade is calculated by summing grades for hip flexion, hip abduction, knee flexion, and ankle dorsiflexion on each side and then dividing by 8.

would be inhibited. However, we wish to report our experience with a case that argues against this supposition and indicates that the catheter tip should, preferably, be placed below the level of spinal cord injury.

Case Report

A 35-yr-old man with T9 incomplete paraplegia of 3 yr duration was referred to our center for spasticity control with intrathecal baclofen (Lioresal intrathecal, Medtronic, Minneapolis, MN). Spinal cord injury developed secondary to compression by a spontaneous epidural abscess. The patient presented with severe spasticity of the lower extremities that was refractory to all traditional oral pharmacologic agents, including diazepam (Valium, Roche, Nutley, NJ), clonidine (Catapres, Boehringer Ingelheim, Ridgefield, CT), dantrolene (Dantrium, Norwich Eaton, Norwich, NY), and baclofen (100 mg/day; Lioresal, Ciba-Geigy, Summit, NJ). Spasticity interfered with the patient's functional abilities and personal independence, and produced considerable pain and discomfort. The use of intrathecal baclofen was approved by the Institutional Review Board for Human Research, and informed consent was obtained from the patient.

Table 2. Reflex Scale

Score	Description of Reflex Response
0	No response
1	Hyporeflexia
2	Normal response
3	Mild hyperreflexia
4	Four beats clonus
5	Unsustained clonus, >4 beats
6	Sustained clonus

Total score is calculated by summing scores from knee and ankle reflexes on each side and then dividing by 4.

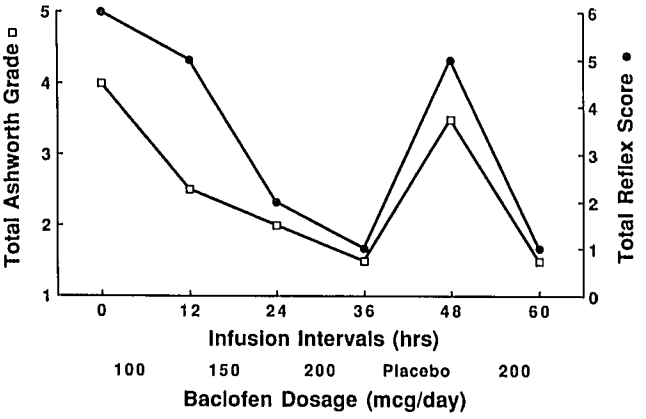

Fig. 1. Temporary percutaneous intrathecal infusion of baclofen demonstrates optimal spasticity control at 200 μg/day and negative placebo response.

Indium (Amersham, Arlington Heights, IL) cisternography was performed to ensure that the spinal canal was patent. Indium (0.5 ml) injected into the lumbar intrathecal space flowed rostrally toward the cervical region over the next 12 h, demonstrating the absence of spinal canal obstruction. A temporary percutaneous trial of intrathecal baclofen infusion was then performed over a period of 3 days. A 23-G polyethylene catheter was placed intrathecally *via* the L4–L5 lumbar vertebral interspace and threaded cephalad for 2–3 cm to lie opposite the L3 vertebral body. Following confirmation of adequate placement of the catheter, an intrathecal infusion of baclofen was commenced. The dosage of baclofen was titrated upward every 12 h in 50-μg/day increments until optimal control of spasticity was observed. Normal saline was also infused for a 12-h period to examine the presence of any placebo effects. Spasticity was evaluated clinically using Ashworth grading and a reflex scale every 12 h (tables1 and 2). The infusion of baclofen and placebo was double-

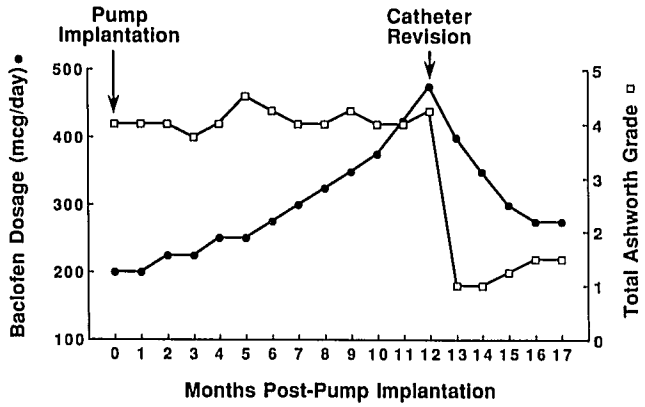

Fig. 2. After pump implantation, optimal spasticity control could not be achieved, despite an increase in intrathecal baclofen dosage to 475 μg/day. After catheter revision, optimal spasticity control was achieved with 275 μg/day.

blinded, so that neither the patient nor the investigator evaluating spasticity were aware of baclofen dosages or infusion of placebo.

The results shown in figure 1 demonstrate that optimal control was achieved at a dosage of 200 μg/day. The response to placebo was negative. After a successful temporary trial, the patient was taken to the operating room and, under general anesthesia, an infusion pump (Synchromed, Medtronic, Minneapolis, MN) was subcutaneously implanted in the right lower abdomen. The pump was connected to an intrathecal catheter that was subcutaneously tunneled around the right flank entering the intrathecal space at L3–L4. Technical difficulty was encountered in placing the intrathecal catheter tip at T12. However, once the catheter was threaded cephalad to lie in the region of T4, approximately five segments cephalad to the actual level of spinal cord injury, repeated aspiration/observation of CSF flow from the catheter hub was obtained. The positioning of the catheter tip was confirmed radiographically. After recovery from surgery, intrathecal baclofen infusion was commenced again using the infusion pump. However, using similar clinical assessment scales, the quality of spasticity reduction achieved with 200 μg/day was considerably less than that obtained during the temporary trial. Gradually, the dosage of baclofen was increased over the next few months, until a maximum of 475 μg/day was reached. In spite of this dosage, optimal spasticity control could not be achieved. Radiographic studies confirmed subarachnoid location of the catheter, as well as the absence of catheter breakage or obstruction, and close examination of the infusion pump software verified accurate delivery of baclofen to the intrathecal space. Twelve months after pump implantation, the patient was taken back to the operating room and the catheter length was shortened to position the tip at the T12-L1 vertebral interspace level. After recovery from surgery, intrathecal baclofen, recommenced at 400 μg/day, immediately produced complete flaccidity of both lower extremities (fig. 2). Over the next 5 months, the baclofen dosage was gradually decreased to 275 μg/day to slightly increase lower extremity muscle tone while maintaining optimal spasticity control (fig. 2).

Discussion

The findings in this patient indicate that the actual delivery site of intrathecal baclofen significantly affects the amount of spasticity reduction in spinal cord injury. In this instance, optimal control of spasticity was achieved when intrathecal baclofen was delivered below, rather than above, the level of thoracic spinal cord injury. Our observations probably relate to the pharmacodynamics of baclofen in the intrathecal space, because anatomic or mechanical problems associated with the infusion pump/intrathecal catheter ensemble were excluded.

The exact mechanism of intrathecal baclofen's clinical effects are not clearly understood. Baclofen may ligand with spinal cord GABA receptors, producing GABA-mimetic effects,[5] or inhibit glutamate,[6] an excitatory neurotransmitter. After the gradual release of baclofen from the spinal catheter orifice, baclofen flows rostrally, obeying the hydrodynamics of the CSF flow.[7,8] This hypothesis is supported by clinical findings in quadriplegic patients that, because baclofen migrates cephalad from the lumbar region, spasticity reduction initially occurs in the lower extremities, followed some 4–6 h later by similar effects in the upper extremities.

Spasticity is thought to be produced by altered descending inhibitory and excitatory suprasegmental influences causing increased responsiveness of deep tendon and cutaneous reflexes below the level of injury.[9] In this patient, intrathecal baclofen administered in the region of T4 had very little effect on lower extremity spasticity, despite a greater than 100% increase in daily dosage. In contrast, administration of intrathecal baclofen below the lesion in the region of spinal segments where the hypertonicity occurred was associated with much more efficacious spasticity control. The daily volume of baclofen solution infused intrathecally averaged 0.6 ml in this patient, while the total spinal subarachnoid CSF volume is approximately 75 ml.[10] Therefore, with administration of intrathecal baclofen rostral to the spinal cord injury, it is unlikely that much drug would reach more caudad segments of the spinal cord, even by diffusion along concentration gradients.

This report indicates that intrathecal catheters should be placed in the lumbar region for lower extremity spasticity control. A potential drawback of this practice is that, to produce simultaneous reduction of spasticity in upper extremities, as in quadriplegic patients, sufficiently large amounts of baclofen need to be administered to flow rostrally and reach cervical spinal segments at effective concentrations.

This may produce complete flaccidity of lower extremities, which is undesirable, because a certain amount of spasticity maintains muscle bulk and bone mineralization. A possible solution to this problem in quadriplegic patients would be to use two catheters: one delivering intrathecal baclofen in the lower lumbar segments, and the other in the upper thoracic and lower cervical segments.

In summary, this report describes an important clinical finding pertaining to intrathecal pharmacotherapy with baclofen, in which control of spasticity was significantly improved when the site of drug delivery was moved from above to below the level of spinal cord injury.

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