Injury to the central nervous system can result in spasticity in the extremities distal to the site of neurologic damage. Spasticity usually requires treatment because it interferes with patients’ ability to position satisfactorily, causes discomfort, and hampers patient transfers. Spasticity is usually adequately controlled with oral medications such as baclofen or benzodiazepines. When the spasticity is refractory to oral agents, it may be treated with an implanted drug delivery system that continuously infuses baclofen into the cerebrospinal fluid. Continuous infusion of morphine into the cerebrospinal fluid has also been used to treat spasticity. Tolerance may develop to both baclofen and morphine administered intrathecally, and an increasing amount of drug may be required to provide satisfactory control of spasticity. This report describes the successful relief of spasticity with intrathecal fentanyl in a patient whose spasticity was resistant to chronically administered intrathecal baclofen.

**Case Report**

A 42-year-old man with a 23-year history of an incomplete spinal cord injury at the fifth cervical level was referred to the Pain Service for evaluation of spasticity in May 1989. His spasticity had been effectively managed with oral baclofen until 5 yr previously, when increasing spasticity required treatment with a combination of oral baclofen, diazepam, and clonidine. At the time of referral he was refractory to these medications. Neurologic evaluation and magnetic resonance imaging failed to disclose pathology that would account for the increase in his symptoms. The patient’s spasticity was interfering with wheelchair transfer, preventing him from sitting upright, and contributing to the development of decubitus ulceration. Consequently the patient was referred for evaluation and treatment of his spasticity. The patient received intrathecal baclofen 50 μg and fentanyl 40 μg administered 1 week apart. The response to each agent was evaluated by clinical examination and with the flexion dynamometer, an instrument designed to measure and document spasticity accurately. Baclofen and fentanyl both produced dramatic temporary reduction in spasticity (Fig. 1). In August 1989 a spinal pump (Medtronic Inc.) was subcutaneously implanted. The pump infuses baclofen intrathecally at a continuous rate. The infusion rate can be adjusted electrically using a programing computer and radiofrequency interface similar to that used to encode a cardiac pacemaker. The patient’s response to the intrathecal baclofen infusion at the rate of 200 μg/24 h was cessation of spasticity and profound lower extremity flaccidity. Subsequently the rate of delivery of baclofen was reduced to 140 μg/24 h to provide enough muscle tone to facilitate transfers. The daily requirement of baclofen increased steadily over the past 12 months until the patient was currently receiving 720 μg of baclofen every 24 h. Despite the increased baclofen requirements the patient’s spasticity persisted. The pump was examined according to the manufacturer’s recommendations and was found to be in proper working order. In April 1991 the patient inadvertently received an overdose of intrathecal baclofen (1,440 μg/24 h) at twice the intended infusion rate. In the ensuing 12 h the patient subsequently noted the progressive absence of spasticity, weakness in his arms, and difficulty breathing. He observed progressive weakness affecting his legs initially, and then his arms, and finally his muscles of respiration. The pump was turned off, and the patient was admitted to the hospital for observation. He was alert and oriented. Pulmonary function tests were performed and the patient underwent clinical examination and evaluation with the flexion dynamometer, which confirmed physical examination findings of profound lower limb flaccidity. His symptoms improved over the next 4 h; the pump was reprogrammed to deliver the intended 24-h dose of baclofen, 700 μg; and the patient was discharged from the hospital. Within 24 h he noted a return of spasticity, and he returned 1 week later for an increase in the baclofen infusion rate. Prior to adjusting the pump, fentanyl 40 μg was injected into the cerebrospinal fluid, causing a marked diminution in spasticity within 30 min. Figure 1 is a graphic representation of the flexion dynamometer spasticity measurements performed over the preceding 18-month period including the initial evaluation.

**References**


Force

Degrees Extension

Fig. 1. Evaluation of spasticity by the dynamic flexometer. As the knee is passively extended over a 3-s interval, the degrees of extension and amount of force (kg) required to extend the leg are simultaneously plotted. Baseline spasticity evaluation prior to starting baclofen infusion (crosses). Results after intrathecal injection of 50 μg baclofen (plus signs), May 1989. Intrathecal fentanyl 40 μg, May 1989 (circles) and the same dose April 1991 (triangles). Profound relaxation obtained with a baclofen overdose April 1991 (asterisks).

Subsequent to the test injection of intrathecal fentanyl, baclofen was gradually discontinued and the pump filled with fentanyl with an infusion rate of 50 μg/24 h. The patient initially demonstrated a favorable response to the intrathecal fentanyl, but during the next 2 weeks required a 3-fold increase in the amount of fentanyl needed to control his spasticity. At this point the fentanyl was discontinued and the pump refilled with preservative-free morphine (1,000 μg/ml). The patient demonstrated a marked reduction of spasticity to the initial continuous infusion of morphine (300 μg/24 h).

During the last 2 months the patient's spasticity has been well controlled using intrathecal morphine, although the required amount of morphine has slowly increased to 600 μg/24 h. At the time of this writing the patient has not received baclofen for 3 months.

**DISCUSSION**

The patient described in this case report exhibited tolerance to baclofen, requiring a steadily increasing amount of intrathecal baclofen to achieve a satisfactory reduction in spasticity. His treatment was complicated by an inadvertent baclofen overdose after nearly 2 yr of continuous treatment. The baclofen overdose caused profound flaccidity and was associated with respiratory muscle weakness and sedation. Because of the escalating baclofen requirement, fentanyl 40 μg was injected intrathecally to determine if intrathecal opioid was a viable treatment option. The fentanyl, administered after 18 months of continuous baclofen infusion, produced relaxation similar to the initial intrathecal fentanyl (given 18 months previously) and the baclofen overdose. Intrathecal fentanyl was associated with neither weakness nor sedation. The amount of baclofen required to provide reduction in spasticity equal to fentanyl was associated with adverse effects such as sedation and respiratory weakness (table 1). Intrathecal fentanyl produced no weakness or sedation, unlike baclofen, which required a toxic dose to reduce spasticity.

The pattern of symptoms associated with the baclofen overdose was one of ascending weakness with very mild sedation occurring as a late symptom. Baclofen overdoses have been reported elsewhere, and intravenous physostigmine has been used to reverse the associated respiratory depression and somnolence. It appears from the previous report that the mechanism of reversal of respiratory depression was associated with an increase in the level of consciousness and not from a reversal of neuromuscular weakness. In our patient, weakness and not somnolence was the predominant problem, and physostigmine was not administered. However, the effect of physostigmine on baclofen-induced muscle weakness is not known.

Although intrathecal baclofen and opioids have been used in the evaluation and/or treatment of spasticity, their clinical interaction has not previously been evaluated in humans. The development of tolerance to chronically administered intrathecal baclofen and morphine has been described. The patient demonstrated tolerance to baclofen as indicated by increasing baclofen requirements, deteriorating spasticity in the presence of an appropriately functioning infusion system, and an evaluation by a spinal cord injury physician that failed to reveal any change in the patient's baseline medical condition that could explain the increasing dosage requirement.

A number of possible explanations can be formulated in an attempt to explain the antispastic efficacy of intrathecal fentanyl despite tolerance to baclofen. The most likely of these is the lack of cross tolerance between intrathecal fentanyl and baclofen, which might be expected because these drugs act at distinct receptors and activate different intracellular pathways. On the other hand, there may be a synergistic interaction between baclofen and fentanyl that resulted in profound relaxation following intrathecal fentanyl in the presence of a baclofen intrathecal infusion. Finally, these results may simply be the result of 40 μg of fentanyl being supramaximal in its an-

<table>
<thead>
<tr>
<th>Mechanics</th>
<th>Baseline</th>
<th>Baclofen Overdose</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC (l)</td>
<td>4.0</td>
<td>3.1</td>
</tr>
<tr>
<td>FEV₁ (l)</td>
<td>3.1</td>
<td>2.6</td>
</tr>
<tr>
<td>FEV₁/FVC (%)</td>
<td>79%</td>
<td>80%</td>
</tr>
<tr>
<td>Peak flow (l/s)</td>
<td>6.6</td>
<td>4.5</td>
</tr>
</tbody>
</table>

FVC = forced vital capacity; FEV₁ = forced expiratory volume in the 1st s after the start of expiration.
tispastic effects and thereby obscuring any cross-tolerance effects between baclofen and fentanyl. A formal dose–
response study with and without baclofen coadministration would be required to differentiate between these
possible explanations.

Regardless of the mechanism, the ability of intrathecal
fentanyl to reduce spasticity in this patient with tolerance
to intrathecal baclofen may have important clinical
implications. The majority of patients with implanted
intrathecal infusion devices have demonstrated resistance
to conventional oral medications for controlling their
spasticity. Furthermore, they usually develop tolerance
to intrathecal baclofen. In this patient, the favorable re-
response to intrathecally infused opioid despite the develop-
ment of tolerance to baclofen has proven very useful.
The opioid treatment has provided satisfactory relaxation
without the need for baclofen for at least 3 months. It
appears that intrathecal opioid therapy may be a viable
alternative therapy to provide a respite from baclofen.
Prior to the commencement of intrathecal opioid therapy,
baclofen should be discontinued gradually to prevent
withdrawal symptoms. After a respite from baclofen the
opioid could be discontinued and the baclofen resumed
at a lower dose. A prolonged treatment plan could rou-
tinely involve rotation of intrathecal baclofen and opioid
when clinical tolerance becomes apparent. The rate of
reoccurrence of tolerance to baclofen following a respite
and the effect of chronic prior exposure of baclofen on
the development of tolerance to intrathecal opioids are
not known.

In conclusion, this case report describes a patient with
severe spasticity who developed tolerance to intrathecal
baclofen but who retained the ability to respond with re-
laxation to intrathecal fentanyl. This may have important
clinical implications for the prolonged management of
spasticity in patients with implanted intrathecal infusion
devices.

REFERENCES
   BMJ 4:15–18, 1971
2. Pierson GA, Fowlks EW, King PS: Long-term follow-up in the
   Rehabil 47:143–149, 1968
3. Penn RD, Savoy SM, Corcos D, Latham M, Gottlieb G, Parke B,
   Kroin JS: Intrathecal baclofen for severe spinal spasticity. N
4. Erickson DL, Blacklock B, Michaelson M, Sperling KR, Lo JN:
   Control of spasticity by implantable continuous morphine pump.
5. Penn RD, Kroin JS: Long-term intrathecal baclofen infusion for
   of spasticity with intrathecal morphine sulfate. Local Spinal
   Therapy of Spasticity. Edited by Muller H, Zierski J, Penn RD.
   New York, Springer-Verlag, 1988 pp 137–142
7. Chabai C, Schwid HA, Jacobson L: The dynamic flexometer: An
   instrument for the objective evaluation of spasticity. ANESTHESIOLOGY
8. Muller-Schwefeke G, Penn RD: Physostigmine in the treatment
9. Chabai C, Jacobson L, Schwid HA: An objective comparison of
   intrathecal lidocaine versus fentanyl for the treatment of lower

Anesthesiology
76:314–316, 1992

Incidental Discography during Celiac Plexus Block

PETER R. WILSON, M.B., B.S., PH.D., F.F.A. R.A.C.S.*

Celiac plexus blockade with local anesthetic or neurolytic
agents is widely used as a diagnostic and therapeutic maneu-
ver for patients with upper abdominal pain and upper
abdominal carcinoma. Numerous techniques of celiac
blockade have been described, including the classical two-
needle posterior approach,1,2 a modified posterior ap-

* Associate Professor of Anesthesiology, Mayo Clinic and Founda-
tion.
Received from the Mayo Clinic and Foundation, Rochester, Min-
nesota. Accepted for publication October 14, 1991.
Address reprint requests to Dr. Wilson: Department of Anesthesi-
ology, Mayo Clinic and Foundation, Rochester, Minnesota, 55905.
Key words: Anesthetic technique: celiac plexus block. Complications.

proach,3 a posterior transaortic method,4 and an anterior
approach.5 Some of these methods require needle contact
with the vertebral body as an indicator of position,
whereas others require radiologic control with fluoroscopy
or computed tomography scan. All techniques should re-
quire a functional test dose of local anesthetic before a
neurolytic agent is injected.6

Because these needles must traverse many structures in
reaching the celiac plexus, numerous complications,
including postural hypotension, chest pain, failure of
ejaculation, urinary difficulty, pneumothorax, neurologic
damage, pleural effusion, paraplegia, and aortic pseudo-
aneurysm have been reported.7–12 These present case re-