# **Brief Review**

# Pain management for children following selective dorsal rhizotomy

Jeremy M. Geiduschek MD,\*
Charles M. Haberkern MD,\*†
John F. McLaughlin MD,† Lawrence E. Jacobson MD,\*
Ross M. Hays MD,†‡ Theodore S. Roberts MD§

Selective dorsal rhizotomy (SDR) is a neurosurgical procedure used for treating lower extremity spasticity in patients with cerebral palsy. The purpose of this paper is to present a review of our institution's first three years' experience with postoperative pain and spasticity management in patients who have undergone SDR. The medical records of the 55 patients who had an SDR during the study period were reviewed. The basis of postoperative analgesia was morphine, with the majority of patients receiving continuous morphine infusions (20-40  $\mu g \cdot k g^{-1} \cdot h r^{-1}$  (n = 49), 60  $\mu g \cdot k g^{-1} \cdot h r^{-1}$  (n = 1)). Four patients used a patient-controlled delivery system. One patient had successful analgesia with epidural morphine. Ketorolac (1  $mg \cdot kg^{-1}$  iv loading dose followed by 0.5  $mg \cdot kg^{-1}$  iv every six hr for 48 hr) was used as an adjunct to morphine in six patients. For management of postoperative muscle spasm, an intravenous benzodiazepine was used (diazepam 0.1 mg · kg-1 (n = 2), or midazolam infusion 10-30  $\mu g \cdot kg^{-1} \cdot hr^{-1}$  (n = 1)51)). All patients were cared for on a ward where nurses were familiar with the use of continuous opioid and benzodiazepine infusions. All patients received continuous cardiorespiratory monitoring as well as frequent nursing assessment. There were no episodes of postoperative apnoea or excessive sedation. We have found the use of continuous infusions of morphine

# **Key words**

ANALGESIA: pain service, PCA; ANALGESICS: morphine, ketorolac; PAIN: dorsal rhizotomy.

From the Departments of Anesthesiology\*, Pediatrics†, Rehabilitation Medicine‡ and Neurological Surgery§, University of Washington School of Medicine, Children's Hospital and Medical Center, Seattle, WA.

Address correspondence to: Dr. Jeremy M. Geiduschek, Department of Anesthesiology, Children's Hospital and Medical Center, P.O. Box C5371, Seattle, WA 98105.

Portions of this work were supported by Grant R01NS27867 from the National Institute of Neurologic Disorder and Stroke. Accepted for publication 9th March, 1994. and midazolam, along with adjunct ketorolac, to be effective in treating postoperative pain and muscle spasms following SDR.

En neurochirurgie, on traite la spasticité de l'infirmité motrice cérébrale par la rhizotomie dorsale sélective (RDS). Cet article revise nos trois premières années d'expérience avec le traitement de la douleur et de la spasticité postopératoires chez des patients qui ont subi une RDS. Nous avons relevé les dossiers de 55 patients. Dans la majorité des cas, on a utilisé la morphine en perfusion continue (20-40  $\mu g \cdot kg^{-1} \cdot h^{-1}$  (n = 49), et 60  $\mu g \cdot k g^{-1} \cdot h^{-1}$  (n = 1) comme analgésique postopératoire principal. Quatre patients se sont auto-administré la morphine. Un patient a reçu une analgésie épidurale continue à la morphine. Chez six patients, du kétorolac (dose initiale de 1 mg·kg-1 iv suivie de 0,5 mg·kg<sup>-1</sup> à toutes les six h pour 48 h) a été ajouté à la morphine. Pour traiter la spasticité postopératoire, nous avons utilisé une benzodiazépine (diazépam 0.1 mg · kg<sup>-1</sup>, (n = 2) ou une perfusion de midazolam 10-30  $\mu$ g · kg<sup>-1</sup> · h<sup>-1</sup> (n = 51)). Tous les patients ont été traité dans leur service par des infirmières familières avec les perfusions continues de morphiniques ou de midazolam. Tous les patients ont été monitorisés en continu et évalués fréquemment par le personnel infirmier qui n'a pas décelé d'apnée ou de dépression respiratoire. Nous avons trouvé que les perfusions continues de morphine et de midazolam avec l'ajout de kétorolac étaient efficaces pour traiter la douleur postopératoire et les spasmes musculaires après RDS.

With the development of bi-polar electrical stimulation techniques for spinal rootlet stimulation, selective dorsal rhizotomy (SDR) has re-emerged as a therapeutic option for surgically treating lower extremity spasticity in patients with cerebral palsy and related conditions. <sup>1,2</sup> The procedure involves a lumbosacral laminectomy and dural incision to expose lumbar and sacral spinal nerve roots. Intraoperative electromyography is used to isolate dorsal rootlet bundles associated with sustained muscular con-

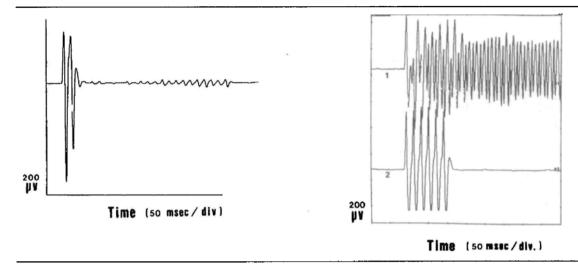


FIGURE (Left) A normal H-reflex response from the electromyogram of the adductor longus following L<sub>3</sub> root stimulation at 50 Hz. The first few stimuli result in muscle contraction. Subsequent stimuli are inhibited from producing further contractions. This rootlet was not sectioned. (Right) An abnormal incremental response to stimulation of an S<sub>1</sub> nerve rootlet at 50 Hz. Channel 1 (top) illustrates a failure to inhibit subsequent motor responses. Channel 2 (bottom) shows an additional response recorded simultaneously from the contralateral gastrocnemius muscle. This abnormal rootlet was subsequently sectioned.

traction or with diffusion of contractions to muscle groups not belonging to that nerve's segmental distribution. Rootlet bundles (containing 3-5 rootlets) that do not have normal inhibitory responses to electrical stimulation are sectioned (Figure).

Patients undergoing SDR pose a challenge in managing pain in the acute postoperative period. There are several components to the postoperative pain in this patient population: somatic pain at the operative site, dysaesthesia and hyperaesthesia of the lower extremities, and distress and discomfort resulting from intermittent muscle spasms of the lower extremities. The muscle spasms when severe cause tension at the surgical site and worsen any incisional pain that might already be present. Here we present a summary of our first three years' experience in caring for children who have undergone this procedure.

## Methods

The hospital records of the 55 children who underwent SDR at Children's Hospital and Medical Center, Seattle, Washington, between October 1988 and August 1992 were reviewed. Physician and nursing progress notes as well as pharmacy records were examined to determine first, the frequency, duration, and dosages of medications administered, and second, the incidence of apnoea or bradypnoea, sedation, nausea, emesis, and pruritus.

#### Results

Patient age at the time of operation ranged from 3 to 22 yr (median age 5 yr): 35 were male and 20 were female. Forty-three patients carried a diagnosis of spastic diplegia

TABLE I Past medical history

| Aetiology or associated factor          | Number of patients |
|---|--------------------|
| Prematurity (gestational age <36 wk)    | 34*                |
| - Intracranial haemorrhage              | 12                 |
| - CNS infection                         | 2                  |
| - Anoxia                                | 5                  |
| - Postnatal factors                     | 2                  |
| - Known prenatal factors                | 1                  |
| - Unknown                               | 15                 |
| Term delivery (gestational age ≥ 36 wk) | 21*                |
| - Small for gestational age             | 4                  |
| - Other prenatal factors                | 6                  |
| - Perinatal infection                   | 1                  |
| - Transverse myelitis                   | 2                  |
| - Other postnatal factors               | i                  |
| - Unknown                               | 8                  |

<sup>\*</sup>Some patients had more than one associated factor.

and 12 had spastic quadriplegia. In 32 cases, an aetiology of cerebral palsy could be inferred from the medical history (Table I). Thirty patients had normal intelligence, 13 had developmental delay and/or learning disability, and 12 had mental retardation. Seventeen were ambulatory, 25 were able to walk aided with support, and 13 were non-ambulatory.

All patients received general anaesthesia for SDR. The anaesthesia consisted of a combination of isoflurane, nitrous oxide, oxygen, and an opioid (fentanyl or sufentanil). No neuromuscular blocking agents were administered after induction of anaesthesia. Rootlet stimulation

was done in a method similar to that described by Fasano. At the end of the surgical procedure following dural closure, 15 patients had bupivacaine (0.25% or 0.5%) with epinephrine 5  $\mu$ g·ml<sup>-1</sup> (0.3–1 ml·kg<sup>-1</sup> body weight) instilled into the surgical wound. All these patients had evidence of motor blockade in the immediate postoperative period. All patients undergoing SDR except one received postoperative care on a surgical ward, where the nursing staff was familiar with problems associated with the procedure. One patient required admission to the Intensive Care Unit for management of hypertension.

During the postoperative period all patients had continuous ECG monitoring of heart rate and thoracic impedance monitoring of respiratory rate (Hewlett Packard 7800 Series Cardiorespiratory Monitor, Medical Products Group, Waltham Massachusetts and after 1989, Spacelabs 90903A Cardiorespiratory Monitor, Chatsworth, California). In addition, all patients after January 1991 were monitored with continuous pulse oximetry (Spacelabs 90903A Cardiorespiratory Monitor, Pulse Oximetry Module, Chatsworth, California; or Nellcor N-180 Pulse Oximeter, Hayward, California).

Assessment of patient analgesia was made subjectively by ward nurses without the use of a standardized pain scale. The majority of patients were able to communicate verbally whether or not analgesia was adequate. Sedation was considered excessive if the patient required vigorous stimulation to be aroused, or was unarousable. The first two patients had their postoperative pain and spasticity management coordinated by the neurosurgeon. All subsequent patients were followed by an anaesthetist on the Acute Pain Service.

All 55 patients received morphine as the basis of analgesic therapy (Table II). Fifty patients were treated with a morphine infusion, alone or in conjunction with other therapy as noted below. All infusions were instituted at 20 μg·kg<sup>-1</sup>·hr<sup>-1</sup>. Inadequate analgesia was treated with a combination of supplemental morphine by bolus (0.05-0.1 mg·kg<sup>-1</sup>) and increases of the infusion rate by  $5-10 \mu g \cdot kg^{-1} \cdot hr^{-1}$ . Adjustments above 40 μg·kg<sup>-1</sup>·hr<sup>-1</sup> required consultation with the Acute Pain Service anaesthetist. One patient required a maximum infusion rate of 60 µg·kg<sup>-1</sup>·hr<sup>-1</sup> for analgesia, while the remaining 49 patients were comfortable at rates of 20-40 µg·kg<sup>-1</sup>·hr<sup>-1</sup>. Once the morphine infusion rate was increased above 20 μg·kg<sup>-1</sup>·hr<sup>-1</sup>, it was not decreased unless side effects occurred or until oral analgesics were tolerated. Of the 50 patients who had morphine infusions, 14 received supplemental morphine by bolus on two or more occasions, and 10 on one occasion. Twentysix patients required no supplementation.

Four patients of normal intelligence, aged 7-22 yr, used patient-controlled analgesia (PCA). All had a basal in-

TABLE II Methods of postoperative analgesia

| Early postoperative analgesia and spasticity management                          | Number of patients $(n = 55)$ |
|--|-------------------------------|
| Morphine infusion only   | 2                             |
| Morphine infusion + diazepam   | 2                             |
| Morphine infusion + midazolam infusion<br>Morphine infusion + midazolam infusion | 40*                           |
| + ketorolac  | 6                             |
| Epidural morphine + midazolam infusion   | 2*                            |
| PCA morphine + midazolam infusion  | 4                             |

<sup>\*</sup>Includes one patient started on epidural morphine and changed to an intravenous morphine infusion on the first postoperative day.

fusion of morphine ( $16-27 \mu g \cdot kg^{-1} \cdot hr^{-1}$ ) in addition to PCA doses of morphine (unit dose  $0.015-0.025 \text{ mg} \cdot kg^{-1}$ , lockout interval 8 min). Cumulative morphine administered in the patients using PCA was  $20-50 \mu g \cdot kg^{-1} \cdot hr^{-1}$ .

Two patients were treated with epidural morphine injected through an epidural catheter placed by the surgeon under direct vision following dural closure and prior to skin closure (patients #16 and 17 in the series). One patient had satisfactory analgesia for three days (six doses of epidural morphine; 70 µg · kg<sup>-1</sup> · dose<sup>-1</sup>) before starting oral analgesics. The other patient developed a leak of clear, non-glucose containing fluid around the catheter insertion site immediately after injection of preservative-free morphine. This epidural catheter was removed on the first postoperative day, and the patient subsequently received an intravenous morphine infusion.

Six patients received ketorolac iv (1 mg·kg<sup>-1</sup> loading dose followed by 0.5 mg·kg<sup>-1</sup> every six hours for 48 hr) in addition to morphine iv.

In addition to opioid therapy, postoperative management of these patients included the administration of a benzodiazepine. This was prompted by the postoperative course of the first two patients who experienced severe muscle spasms characterized by uncontrolled intermittent contractions of lower extremity muscles followed by either crying or complaining of pain. After this experience, all subsequent patients received benzodiazepine therapy in anticipation of the development of postoperative muscle spasms. The third and fourth patients in the series received intermittent doses of intravenous diazepam (0.1 mg·kg-1). The remaining 51 patients received an infusion of midazolam instituted at  $10-30 \mu g \cdot kg^{-1} \cdot hr^{-1}$ . Persistent spasms that were subjectively assessed to cause patient discomfort were treated with a combination of midazolam boluses (0.5-2 mg iv) and increases of the midazolam infusion rate. Six patients required infusion rates greater than 30 µg · kg<sup>-1</sup> · hr<sup>-1</sup>; one patient required 60 μg·kg<sup>-1</sup>·hr<sup>-1</sup>. Nine patients received additional boluses of midazolam for spasms not relieved by the infusion alone. None of the patients treated with ketorolac required a midazolam infusion of  $>30~\mu g \cdot kg^{-1} \cdot hr^{-1}$  or additional midazolam boluses.

None of the patients had an episode of apnoea or excessive sedation. Nausea and/or vomiting occurred in 31 patients, who were treated with metoclopramide *iv* (0.1 µg·kg<sup>-1</sup>·dose<sup>-1</sup>); ten of these required treatment beyond the first operative day. Twelve patients developed pruritus, which was treated with diphenhydramine (0.5-1 mg·kg<sup>-1</sup> *iv*, maximum 50 mg). Indwelling bladder catheters were placed in all patients for monitoring urine output during the surgical procedure, and these remained in place until morphine and midazolam infusions were discontinued. One patient had urinary retention following discontinuation of the urinary catheter. One patient developed an erythematous macular rash consistent with a drug eruption. One patient developed "hyperactive" behaviour which resolved with discontinuation of midazolam.

Intravenous therapy (morphine with or without midazolam) was continued for two to five days (median three days), although, attempts were not made to discontinue it before the second postoperative day. At this time oral analgesics and benzodiazepines were ordered to be given as needed, particularly in conjunction with physical therapy which was instituted on the fourth postoperative day for all patients (Table III). At the time of discontinuation of intravenous therapy, all patients were tolerating a clear liquid diet. Duration of hospital stay ranged from five to 12 days (median seven days).

### Discussion

In our institution, SDR is performed on patients who have a velocity-dependent ("clasp-knife") spasticity with few other motor impairments and a perceived potential for improvement in mobility or in wheelchaire positioning.

Postoperative care is directed towards providing safe and effective control of pain and spasticity, as well as facilitating early mobilization, physical therapy, and discharge from the hospital. The evolution of our pain and spasticity management programme has improved our ability to attain these goals. We have learned that the discomfort children have following SDR results not only from the laminectomy but also from dysaesthesia, hyperaesthesia and intermittent muscle spasms of the lower extremities. The latter three components of postoperative pain are presumed to be secondary to sectioning of nerve rootlets. These problems are most intense during the first three postoperative days and then subside quickly. Initially, the contribution of muscle spasms to postoperative discomfort was not anticipated, but it was markedly alleviated by treatment with benzodiazepines. Complaints of pain during injection of diazepam in two patients prompted a change to intravenous infusion of midazolam.

TABLE III Oral analgesics and benzodiazepines

|  | Number of patients |
|--|--------------------|
| Oral analgesics                          | 55                 |
| - Nothing required (acetaminophen with   |                    |
| codeine prescribed)                      | 2                  |
| - Acetaminophen with codeine             | 47                 |
| - Acetaminophen with oxycodone           | 5                  |
| - Meperidine                             | 1                  |
| Oral benzodiazepines                     | 53*                |
| - Diazepam                               | 30                 |
| - Lorazepam                              | 2                  |
| - Nothing required (diazepam prescribed) | 21                 |

<sup>\*</sup>Not prescribed for first two patients.

Instillation of bupivacaine into the surgical wound after dural closure in 15 patients resulted in excellent analgesia for several hours. However, as the epidural block subsided we found it difficult to attain adequate analgesia and spasm control expeditiously by instituting morphine and midazolam infusions; these patients would often need several boluses of both medications to become comfortable. As a result, we have discontinued the instillation of bupivacaine, and we initiate morphine and midazolam infusions immediately after the patient awakens in the postanaesthesia care unit.

Recently, we have begun to administer ketorolac in addition to morphine and midazolam in the early post-operative period. Ketorolac (0.9 mg·kg<sup>-1</sup> iv) has been shown to have equianalgesic properties to morphine (0.1 mg·kg<sup>-1</sup> iv) in children following a variety of surgical procedures.<sup>3</sup> Ketorolac has a morphine-sparing effect when used to treat postoperative pain in adults.<sup>4,5</sup> It is interesting that none of the patients who received ketorolac required adjustments of their midazolam infusion rate to help control muscle spasms.

Other therapeutic modalities directed towards postoperative analgesia in patients undergoing SDR have been described. Harris et al. have reported the use of intrathecal morphine (7-23 µg·kg<sup>-1</sup>) for postoperative analgesia in 15 children. The duration of analgesia was variable and two children required naloxone administration, one for apnoea and the other for excessive somnolence.6 Others have reported good results with epidural morphine and continuous epidural infusions of fentanyl administered through catheters placed rostral to the upper border of the dural incision at the end of the procedure. <sup>7,8</sup> Following the episode of fluid leakage around an epidural catheter in our seventeenth patient in the series, we have not used epidural analgesia. Also, we have been reluctant to use epidural opioids following an intravenous opioid-based anaesthetic, since this combination may increase the incidence of respiratory depression.9

The combination of continuous *iv* infusions of morphine and midazolam, with adjunctive ketorolac therapy, may be used to provide effective management of acute postoperative pain and muscle spasm following SDR. As SDR becomes more common, the summary of our experience should be useful to others.

# Acknowledgements

The authors wish to thank Elliot J. Krane, M.D. for his valuable comments and suggestions about the manuscript. They also wish to thank Donald C. Tyler, M.D., Helen Karl, M.D., Maureen Pomietto, R.N., M.S.N., and the anaesthesiology fellows and nursing staff at Children's Hospital and Medical Center, Seattle, for providing excellent postoperative analgesia to the patients described in this paper.

#### References

- 1 Fasano VA, Broggi G, Zeme S. Intraoperative electrical stimulation for functional posterior rhizotomy. Scand J Rehabil Med Suppl 1988; 17: 149-54.
- 2 Peacock WH, Arens LJ, Berman B. Cerebral palsy spasticity. Selective posterior rhizotomy. Pediatric Neurosciences 1987; 13: 61-6.
- 3 Watcha MF, Jones MB, Lagueruela RG, Schweiger C, White PF. Comparison of ketorolac and morphine as adjuvants during pediatric surgery. Anesthesiology 1992; 76: 368-72.
- 4 Gillies GWA, Kenny GNC, Bullingham RES, McArdle CS. The morphine sparing effect of keterolac tromethamine: a study of a new, parenteral non-steroidal anti-inflammatory agent after abdominal surgery. Anaesthesia 1987; 42: 727-31.
- 5 Burns JW, Aitken HA, Bullingham RES, McArdle CS, Kenny GNC. Double-blind comparison of the morphine sparing effect of continuous and intermittent i.m. administration of ketorolac. Br J Anaesth 1991; 67: 235-8.
- 6 Harris MM, Kahana MD, Park TS. Intrathecal morphine for postoperative analgesia in children after selective dorsal root rhizotomy. Neurosurgery 1991; 28: 519-22.
- 7 Sparkles ML, Klein AS, Duhaime A-C, Mickle JP. Use of epidural morphine for control of postoperative pain in selective dorsal rhizotomy for spasticity. Pediatric Neurosciences 1989; 15: 229-32.
- 8 Klein AS, Vollers MJ. Epidural fentanyl infusion after dorsal rhizotomy for postoperative analgesia in children. J Neurosurg Anesthiol 1989; 1: 195-6.
- 9 Cousins MJ, Cherry DA, Gourlay GK. Acute and chronic pain: use of spinal opioids. In: Cousins MJ, Bridenbaugh PO (Eds.). Neural Blockade in Clinical Anesthesia and Management of Pain. 2nd ed. Philadelphia: JB Lippincott, 1988: 955-1029.