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Clinical Study

Prevention of brisk hyperactive response during selective dorsal rhizotomy in children with spasticity: Isoflurane versus sevoflurane maintenance anesthesia

Deniz Konya [a], Arzu Gercek [b,*], Adnan Dagcinar [a], Nigar Baykan [b], Memet M. Ozek [a]

[a] *Department of Neurosurgery, Marmara University, Institute of Neurological Sciences, Istanbul, Turkey*
[b] *Clinic of Anesthesiology, Marmara University, Institute of Neurological Sciences, M.Ü Nörolojik Bilimler Enstitüsü P.K. 53 Maltepe, Istanbul 34854, Turkey*

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Abstract

In children with spasticity, deep tendon reflexes are hyperactive and even stimulation of normal dorsal rootlets can produce exaggerated full-strength, single-twitch responses in the muscles they innervate. This phenomenon is called the brisk hyperactive response (BHR). The aim of this study was to compare the efficacy of 2 volatile anesthetics, isoflurane and sevoflurane, for suppressing the confounding effect of BHR during selective dorsal rhizotomy (SDR) in children with spasticity. The subjects were 54 consecutive children of American Society of Anesthesiology physical status III who were scheduled for SDR. After tracheal intubation, each child was randomly assigned to Group I (isoflurane; $n = 27$) or Group S (sevoflurane; $n = 27$). There was no significant difference between the mean operation times in Groups I and S (200 ± 40 vs. 220 ± 35 minutes, respectively; $p = 0.0559$). Thirteen patients in Group I (48.1%) and 5 in Group S (18.5%) exhibited BHR during stimulation of the dorsal rootlets (odds ratio 4.086; $p = 0.0418$). Three of these 18 patients (2 in Group I and 1 in Group S) experienced hypertension and tachycardia simultaneously with BHR (odds ratio 4.086; $p = 1.0$). The results suggest that sevoflurane is more effective at preventing BHR and might be a better choice for anesthetic management of children with spasticity undergoing SDR.

Keywords: Brisk hyperactive response; Children; Management; Selective dorsal rhizotomy; Spasticity; Volatile anesthetics

1. Introduction

Interruption of the central nervous system descending inhibitory pathways results in rearrangement of spinal reactivity, leading to spasticity and overactivity.[1] Spasticity should be treated because it limits patients' functional capacity and can, therefore, lead to many other problems: inactivity, pressure sore formation, cardiovascular problems, respiratory infection, thrombophlebitis, bladder/bowel problems, osteoporosis, fixed contracture, pain, social isolation, further loss of muscle strength, and difficulties obtaining care. The patient's quality of life progressively deteriorates if she or he is left untreated.

The purpose of treatment for spasticity is deafferentation of the posterior horns, which reduces the pathological reflex responses that cause spasticity.[2] Posterior rhizotomy was first performed in 1913 by Foester, who carried out the operation primarily to relieve spasticity.[3] The technique has been improved and modified to become known as selective dorsal rhizotomy (SDR). In this technique, the surgeon sections the rootlets that supply the muscle groups clinically identified as being involved with the disabling spasticity. SDR is now widely used for children with lower-extremity spasticity, particularly those who are resistant to conservative treatment or have significantly impaired functional ability.[2,4–7]

* Corresponding author. Tel.: +90 532 6417334; fax: +90 216 4214382.
E-mail address: arzu.gercek@marmara.edu.tr (A. Gercek).

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To identify a muscle's response to electrophysiological stimulation, there must be no intraoperative muscle relaxant. This means that, during surgical treatment for spasticity, no muscle relaxant agent can be given after induction of anesthesia. However, children with spasticity have hyperactive deep tendon reflexes and even stimulation of a normal dorsal rootlet can result in an exaggerated full-strength, single-twitch response in the muscle it innervates, called the brisk hyperactive response (BHR).[8] The BHR causes no additional disability in a child with spasticity but makes intraoperative testing difficult and, therefore, must be suppressed throughout electrophysiological stimulation. One challenge of anesthetic management for SDR in these children is the need to suppress BHR while not interfering with abnormal muscle responses.

This randomized prospective study was conducted to compare the efficacy of 2 volatile anesthetics (isoflurane and sevoflurane) in the setting of SDR in children with spasticity. We assessed each agent's ability to suppress the confounding effect of BHR during electrical stimulation of dorsal rootlets while not interfering with abnormal muscle responses. We hypothesized that sevoflurane would prevent BHR more effectively because previous work has shown that this agent has a more significant muscle relaxant effect than isoflurane.[9]

2. Materials and methods

2.1. Study design

The study was approved by our Institutional Research Board and written informed consent was obtained from the parents of each participant. Fifty-four consecutive American Society of Anesthesiology physical status III children with spasticity scheduled to undergo SDR were enrolled prospectively.

2.2. Anesthetic management

Each child was premedicated intravenously (IV) with midazolam (70 μg/kg) and atropine (15 μg/kg) and closely monitored by nurses. Parents were allowed to stay with the patient until just before the operation started in order to ease the child's anxiety. A eutectic mixture of local anesthetics was applied to the dorsum of the patient's hand 45 minutes before the operation and a 22 G IV catheter was inserted. Anesthesia was induced with IV propofol 2 mg/kg, vecuronium 0.1 mg/kg, remifentanil 1 μg/kg, dexamethasone 0.2 mg/kg and ondansetron 0.1 mg/kg. After tracheal intubation, the child was randomly assigned to 1 of 2 groups by asking the parents to select a sealed envelope. In Group I ($n = 27$), anesthesia was maintained with 0.5% isoflurane and 50% dinitrogen oxide (N_2O) (in O_2), as well as a remifentanil infusion (0.25 μg/kg per min). In Group S ($n = 27$), anesthesia was maintained with 0.6% sevoflurane and the same combination of 50% N_2O in O_2 and remifentanil. No vecuronium (muscle relaxant) was given after induction to avoid inhibiting muscle response to stimulation of dorsal rootlets.

Standard monitoring was carried out, including 3-lead electrocardiography (lead II recorded), invasive blood pressure monitoring, pulse oxymetry, capnography, and body temperature monitoring via an esophageal probe. Urine output during the operation was also recorded. All patients were mechanically ventilated using 10 mL/kg tidal volume and a rate appropriate for the child's age to keep end-tidal CO_2 between 30 mmHg and 35 mmHg. Maintenance fluids (0.9% NaCl) were administered.

We defined BHR as hyperactive deep tendon reflexes and exaggerated full-strength, single-twitch responses in the muscle innervated by the dorsal rootlet stimulated (see Section 2.3). When we observed BHR during rootlet stimulation, we took specific steps to assess the effect of the volatile anesthetic on BHR: (i) kept the concentration of the volatile anesthetic agent constant; (ii) injected a 1 mg/kg bolus of propofol to suppress the BHR; (iii) started an infusion of propofol at 4 mg/kg per minute. If hypertension and tachycardia accompanied the BHR, we administered a single bolus of remifentanil (1 μg/kg).

At the end of the surgery, we infiltrated the wound edges with 5 mL of 0.25% bupivacaine hydrochloride. Remifentanil infusion was stopped during dressing of the wound. The lungs were ventilated with 100% O_2 and neuromuscular blockade was reversed with IV neostigmine (30 μg/kg) and atropine (15 μg/kg). Patients were extubated when they exhibited adequate spontaneous ventilation and opened their eyes. Any complication throughout the peri-operative period was recorded.

After recovery from anesthesia, each child was observed for 24 hours in the Neurological Intensive Care Unit. Postoperative analgesia was achieved with IV pethidine hydrochloride and paracetamol suppositories. The Children's Hospital of Eastern Ontario Pain Scale was used and a score greater than 4 indicated pain.[10]

2.3. Surgical management

We performed surgery with the patient in the prone position. Once the surgical field was disinfected with 10% povidone-iodine and sterile draping was positioned, the operation was started. A skin incision was made from L1 to S1 and paravertebral dissection was carried out. An electric saw was then used to perform en bloc laminotomy with a flap extending from L1 to L5. The dura mater was opened and the anterior (motor) and posterior (sensory) nerve roots were identified at each level from L2 to S1. For intraoperative recording of motor responses, individual dorsal roots were stimulated using a 1-s constant 50 Hz current at the threshold level of 6 mA to 10 mA (Aesculap, Bethlehem, PA, USA). Compound muscle action potentials were recorded using surface electrodes placed bilaterally on various muscle groups of the lower extremities (Supplementary Material Fig. 1). A physiotherapist assessed each patient's muscle responses during the