AE Sung Park has been a pioneer in the treatment of spasticity in patients with cerebral palsy (CP). In this issue is another article that helps define the role of selective dorsal rhizotomy (SDR) in the treatment of hypertonicity in children. Dr. Park and other authors have to date published ample evidence that SDR decreases Ashworth scores, increases range of motion, and does not cause significant weakness in selected patients with spastic diplegia.

This study addresses a frequent question that parents ask before any procedure: “will my child require further surgeries?” O’Brien, et al., demonstrate that patients with more significant motor involvement will have nearly a twofold increase in the necessity for orthopedic surgery over the ensuing 5 to 9 years.

In 2005, children with hypertonicity have multiple treatment modalities available: SDR, botulinum toxin, phenol injections, intrathecal baclofen, and oral medications (including baclofen, Dantrium, Valium, and Zanaflex), as well as a number of orthopedic surgical procedures. O’Brien, et al., restrict their inquiries to the use of only one of these other modalities before and after a patient undergoes SDR. It would be very helpful to identify how many patients in each of these groups required additional treatment for their hypertonicity after their surgery.

Several technical questions arise regarding the study. How were the patients selected? The authors state they performed 177 SDR procedures but only 158 patients participated in the follow up. The inclusion/exclusion criteria, ambulatory status, and parental satisfaction of the 11% of patients not included are not mentioned. A questionnaire was sent out. Details on the validation of the questionnaire used to assign the gait score and assess the number and type of orthopedic procedures performed are not supplied. For example, if a patient takes steps in physical therapy or at home without assistive devices but mainly uses crutches, some confusion could exist if he or she is given a score of 10 or 9. How was consistency in the assignment of the gait score ensured in a self-administered questionnaire?

The study identifies patients who underwent orthopedic procedures; it omits patients who had procedures recommended but whose families declined them, and patients who did not receive follow-up care and would have benefited from additional orthopedic surgery. The authors do not discuss how the type of procedure listed in the response to the questionnaire was validated. Last, it would be remarkable if the authors were able to get complete responses from 100% of the patients, but compliance and completeness issues are not addressed.

Why did more severely affected children require more orthopedic surgery? If we look at the data in one group, those younger than 4 years old who were not independent walkers after SDR, we can identify several interesting points. The majority (27 [73%] of 37) of the orthopedic procedures in the nonambulatory patients who were younger than 4 years old involved soft-tissue lengthening, which are procedures for residual or recurrent hypertonicity or for residual contractions. The occurrence of contractures that cannot be treated with serial casting or other therapy in children younger than 4 years of age after rhizotomy is quite small. A conclusion could be inferred that the patients in the nonambulatory group younger than 4 years old therefore had a significant incidence of residual or recurrent hypertonicity. In contrast, four of 19 patients in the ambulatory group underwent seven procedures, of which only three were soft-tissue lengthening. These findings are repeated in every group, although the need for procedures to correct fixed contractures increases with age.

In the late 1980s and early 1990s, SDR was the only effective procedure for hypertonicity (“when all you have is a hammer, everything looks like a nail”). Pediatric neurosurgeons, including those in our clinic, offered SDR to a wide range of patients who exhibited hypertonicity. The procedure was shown to have long-term efficacy in patients with spastic diplegia. In our experience, SDR has not been shown to be effective in treating hypertonicity of spinal origin, such as spinal cord injury and hereditary spastic paraparesis. We have found that a good initial decrease in tone occurs, but the hypertonia returns within several years. Selective dorsal rhizotomy has also not proved effective in treating the hypertonicity arising from traumatic brain injury, anoxia, or movement disorders such as dystonia or choreoathetosis.
Pure spastic diplegia results from periventricular leukomalacia. This syndrome causes injury to the white matter tracts next to the ventricles, the closest fibers being those of the cortical spinal tract for the lower extremities. Spasticity, by definition, is a part of the upper motor syndrome that occurs when the cortical spinal tract is damaged. Periventricular leukomalacia has a spectrum of severity and is often associated with the effects of intraventricular hemorrhage in the newborn, which can cause injury to the thalamus and basal ganglia. A child with spastic diplegia who has significant cognitive difficulties, involvement of the upper extremities, or truncal hypotonia has an injury to the brain that involves more than the periventricular cortical spinal tracts to the lower extremities. Patients who have involvement of the supplementary motor systems in addition to or instead of the cortical spinal tracts may experience a variety of movement disorders, including rigidity, dystonia, and athetosis. Many of these patients present with complex, combined movement disorders and demonstrate hypertonicity due to combinations of spasticity with dystonia or athetosis. After the spasticity is reduced, the dystonia or athetosis becomes apparent. This complexity leads to failure to meet the functional goals the family and spasticity team had identified.

Over the past decade, comprehensive pediatric spasticity centers have integrated the multiple modalities available for the treatment of spasticity. Patients are treated using the approaches that best meet their individual goals or the goals of their caretakers. The goal is never reduction of hypertonicity, because reduction of hypertonicity is the tool used to improve function or quality of life. These centers have identified the ideal candidate for SDR. The ideal patient is 3 to 7 years old, has spastic diplegia, was born premature, and has adequate strength, irrespective of the need for assistive devices. Many of the patients who respond best to SDR have a very narrow stance because of high adductor tone and scissoring, thus preventing independent walking or standing, so they require assistive devices for balance. Most of these children do not require such devices after SDR. O’Brien and colleagues failure to find a difference in the rate of orthopedic surgery in 2- to 3-year-old patients based on pre-SDR ambulatory status supports this observation. At our center, adequate strength means that patients can reciprocal crawl, tall kneel, side sit independently, rise to stand with stand by assistance, and do a graded squat stand. If the child cannot perform these functions, then intrathecal baclofen or botulinum and stretching with reevaluation in 1 year is recommended. Patients with spastic quadriaparesis and problems with hypertonia are not referred for SDR surgery, irrespective of their ambulatory status.

O’Brien, et al., offer strong support for this approach. They demonstrate a twofold increase in residual or recurrent hypertonicity in their group with more involvement (children requiring assistive devices after rhizotomy). They show that 30% to 40% of those patients require additional procedures for their hypertonicity. Their data support restricting SDR surgery to patients with pure or nearly pure spastic diplegia, and recommending other approaches to patients more significantly affected.

Many pediatric spasticity centers no longer recommend SDR. In my conversations with these centers, the reasons stated always reference marginal or poor outcome due to recurrent or residual spasticity or to weakness inhibiting function postoperatively. In addition, the patients failed to meet the goals set up by the family with the spasticity clinic team. O’Brien and colleagues’ study gives strong support for careful patient selection for SDR and for limiting the surgery to patients with nearly pure spastic diplegia who are projected to have enough strength to walk independently afterward. Other patients will likely have mixed hypertonia and may have better functional outcomes with other procedures or therapies for decreasing their hypertonicity.

RESPONSE: We thank Dr. Turner for his interest in our work on spastic CP in general and his comments regarding this paper in particular. We find his comments interesting, but also find some of his statements regarding SDR to be inaccurate, anecdotal, and not based on medical evidence.

It is important to note that in this era of evidence-based medical practice, SDR has emerged as the only surgical treatment for spastic CP, satisfying rigid treatment criteria regarding reduction of spasticity and improvement of motor function. Throughout the past 18 years, SDR has undergone unprecedented scientific scrutiny as a surgical treatment option, with at least 59 related reports in the English literature. No other surgical treatment, including intrathecal baclofen, has endured such intense scrutiny. Evidence accumulated about SDR to date clearly indicates that the beneficial effects of this procedure include reduction of lower-limb spasticity, improved lower-limb range of motion, improved motor function as indicated by the Gross Motor Function Assessment, and improved functional performance according to validated outcome measures such as the Pediatric Evaluation of Disability Inventory. If Dr. Turner performs an unbiased review of the literature, he will see medical evidence that overwhelmingly favors SDR.

We ask that readers of this letter who are considering SDR for the treatment of CP spasticity rely on published, objective data from medical centers of high renown and not on the personal opinions of others. In particular, we view with skepticism opinions that relate to treatments that are heavily advertised by strong corporate commercial interests yet have unproven efficacy, such as intrathecal baclofen.

No reliable evidence exists to support the empirical use of “botulinum toxin, phenol injections, intrathecal baclofen, and oral medications (including baclofen, Dantrium, Valium, and Zanaflex)” in the long-term treatment of spastic diplegia. Botulinum toxin is used for the treatment of spastic CP. It can reduce lower-limb muscle tone in young children, but its benefits are short lasting and the long-term beneficial effects in children with spastic CP are unknown. Beneficial effects of antispasticity medications on spastic CP have never been shown in clinical stu-