

To the Editor:

We are writing with regard to the article by Adelson et al¹ entitled “Entrapment Neuropathy Contributing to Dysfunction After Brachial Plexus Birth Injuries.”

In the initial paragraph, the authors report that up to 90% of afflicted children have spontaneous functional recovery. This observation is unreferenced. Review of natural history studies would suggest that this is an overstatement.

In the following paragraph, the term “nonfunctional” is used to describe the involved extremity in an infant with a brachial plexus birth injury. The term nonfunctional is not clearly defined nor is it referenced in current literature. Specific parameters for surgical intervention are based on the type of injury and the specific degree of recovery during the first 8 or 9 months of life. The term nonfunctional is vague.

The authors do not describe the initial surgical procedure that was performed on children who subsequently underwent infraclavicular neurolysis. It would be interesting to know the intraoperative findings of the original procedure and what type of reconstruction was performed. Furthermore, it is important to know how they determined the quality of spinal nerve stumps that were used for grafting.

Looking at Table 3, one wonders how a late axillary/infraclavicular neurolysis would change passive range of motion of an extremity. In multiple cases, an obvious change in the extremity’s passive range of motion occurred postoperatively; unless the surgical exposure included tendon releases, it is unclear how neurolysis alone is responsible for changes in passive motion.

We wondered how measurements were obtained for active shoulder abduction.

Particularly perplexing is that active and passive external rotation of the shoulder is not noted in the article. External rotation of the shoulder is an essential requirement for normal shoulder elevation and the ability to bring the hand to the face. Recovery of external rotation of the shoulder is a basic goal of this surgery.²

In the final paragraph, the authors suggest that muscle weakness in the children with obstetric palsy is not related to the initial birth trauma that caused the nerve damage but may be caused by “dynamic or position-dependent compression of the axillary nerve.”¹ In over 100 cases of secondary procedures with transfers of latissimus dorsi–teres minor muscle tendon units to augment shoulder function, we have consistently noted infraclavicular scarring that extends well into the axilla and distally along muscle and fascial planes. The same findings have been seen in all children who have undergone a simultaneous shoulder and plexus procedure during infancy.³ It is obvious that the initial trauma was a causative factor, with hemorrhage, edema, and subsequent fibrosis.

With the exception of a review of the experience at Louisiana State University consisting primarily of adult brachial plexus cases,⁴ Adelson et al¹ cited no articles on brachial plexus birth injuries published in the last decade.

Finally, the electromyogram thresholds and the resultant decreases are expected during surgery, so the reported significant changes are spurious and do not indicate any real changes in nerve conduction.

In conclusion, it may be that the authors have recognized a potential source of additional muscle dysfunction in brachial plexus birth injuries; however, this article does not adequately prove this hypothesis.

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Author’s Response:

We appreciate the opportunity to respond to the recent letter regarding our article, “Entrapment Neuropathy Contributing to Dysfunction After Brachial Plexus Birth Injuries”¹ and thank the authors for their interest in our work and thoughtful and insightful comments. In our study, in evaluating children with birth palsy after a period of recovery, we have often observed limited active elevation of the shoulder despite satisfactory voluntary contraction of the deltoid as long as the arm remains in adduction. Alternating strength in other muscles as the result of similar positional changes, however, would be a very rare finding. The observation was interpreted as potential evidence of an isolated, dynamic compression neuropathy of the axillary nerve. In reporting the results from testing of that hypothesis, we also offer a treatment strategy for 1 specific late complication to birth palsy. At this time, the exact mechanisms behind the development of axillary nerve compression in the quadrangular space are incompletely understood. The authors of the letter raise a number of points, but for the most part, we are in agreement with their comments and thoughts, addressing them individually.

Whereas indeed it is thought that up to 90% of children afflicted with birth brachial plexus injuries have spontaneous functional recovery, the concern in the letter is that this is likely an overstatement and dependent on the definition of “recovery.” While it was an oversight that this statement was not referenced and may be caused by selection bias, there are a number of studies that note roughly this approximate 90% functional recovery.^{2,3}

Furthermore, it was noted that the term “nonfunctional” was used with regard to determining whether to proceed with surgical intervention. In the text, the term nonfunctional was placed in quotes because it has not been clearly defined in current literature and varies between manuscripts, and references were provided as to this variation. We differ with the letter authors in that references were provided from the current literature as to the concept of nonfunctional as an introduction to the concept. We also provided the definition of nonfunctional in the population of patients for surgical intervention in our study as “the inability to elevate the arm adequately to reach the hand to the mouth.” In addition, the initial surgical procedure from the center is described in the second paragraph in the introduction as neurolysis and nerve grafting to the supraclavicular brachial plexus. We did not describe in detail this surgical procedure because the focus of this particular article was to investigate additional factors that may potentially impede recovery in this patient population that had not recovered adequate function.

The letter writers commented that late axillary nerve decompression would unlikely change the passive range of motion found in Table 3. We agree that a neurolysis alone would not be expected to improve passive motion. Indeed, it is clearly stated in the text that tenotomies were performed on the surrounding tissues for decompression and that also the surgical exposure included tendon releases.

The measurements of passive and active range of motion were performed in the clinic at follow-up by the lead author. We are in agreement with the letter that external rotation is essential for normal shoulder elevation and the ability to bring the hand to the face. Similarly agreed is that the recovery of external rotation is 1 basic goal of reconstructive procedures for birth palsy. We do not, however, suggest that muscle weakness in children with obstetric palsy would not be related to the initial birth trauma and nerve damage. In our final paragraph, we only suggest that “dynamic or position-dependent compression of the axillary nerve may be one or more of the potential etiologic factors.” In our series of secondary procedures

that included transfer of the latissimus dorsi–teres muscle conjoined tendon to augment shoulder function, we identified infraclavicular scarring, vascular compression, and tendinous shortening that were identified as potential etiologies of compression of the nerves. These procedures can improve passive, but certainly not active, elevation of the arm. In addition and as a part of the procedure, we performed a decompression of the axillary nerve at its passage through the quadrangular space and subsequently documented improved function of the deltoid muscle.

In this article, our neurophysiological data confirm intraoperative improvement in axillary nerve conduction. Whereas the letter authors comment that electromyography threshold changes would be expected during surgery, this type of intraoperative neurophysiological study has, to the best of our knowledge, not previously been reported in the literature.

Lastly, this was a retrospective report, with only 10 participating patients, and we do not claim that this 1 small study proves the hypothesis. Our goal was to raise the question as to other potential causes of lasting functional impairment in birth palsy, of which axillary nerve entrapment may be one. A dynamic and position-dependent, as opposed to static, compression of the axillary nerve represents an explanatory model as to why the deltoid would be functional with the arm in adduction but loses the ability to actively contract when the arm is actively or passively abducted. Conversely, we believe that the augmentation of shoulder function is not solely caused by the tendon transfers but rather raises the question of other potential factors (ie, secondary regional scarring). Our findings of neurophysiological improvement as well as functional improvement even in patients who did not have tendon transfers provide what we believe is preliminary data to indicate that even late neurolysis and nerve decompression may improve the outcome in these children and that further study is warranted.

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To the Editor:

I read with interest the article by Davids entitled, “Quantitative Gait Analysis in the Treatment of Children With Cerebral Palsy” in the recent issue of the *Journal*.¹ Although I was impressed with the arguments put forward in support of gait analysis, I am deeply concerned about what Davids says in the last paragraph of the article. In response to the question he poses, “Should every child with cerebral palsy have a quantitative gait study before surgery designed to improve ambulation?” he responds with an emphatic “Yes.”

Is this realistic in the context of the world at large? I am privileged to work in one of the more affluent institutions of this country. The hospital has sophisticated facilities for magnetic resonance imaging scan, computed tomographic scan, in-vitro fertilization, renal transplantation, coronary angioplasty, and the like. This is all a far cry from what is available in most hospitals in this country and in many parts of the world. Yet, in our center, we do not have the funds to refurbish a very basic gait laboratory set up 15 years ago, which is now defunct. Do I stop offering surgery to more than 250 children with cerebral palsy I see each year because I cannot get a preoperative gait study?

I am aware that Davids is helpless in finding solutions for remedying the gross disparities in the health care delivery systems in different parts of the world. However, I do believe that he

and others who are actively involved in gait analysis in cerebral palsy can help those of us who work in areas where routine gait analysis is just not going to be available in the foreseeable future. They need to strive to identify clinical criteria for performing specific operations based on the knowledge gained from gait analysis.

Gage² emphasized that the understanding of lever-arm dysfunction in cerebral palsy came from gait analysis. But is gait analysis essential in deciding when to do a femoral derotation osteotomy to improve lever-arm dysfunction? Cook et al³ demonstrated that in 77% of instances, a decision to perform a derotation osteotomy can be made on clinical grounds. This is an example of where information and knowledge derived from gait analysis can be used in situations where gait analysis is not available. Similarly, the awareness of co-spasticity of the hamstrings and the rectus femoris clearly came from gait analysis. However, is it not possible to diagnose a stiff-knee gait due to this phenomenon without gait analysis? Are there no clinical features that will enable one to diagnose a stiff-knee gait? In our center, we have diagnosed such cases clinically, and the outcome of distal rectus transfers has been gratifying.

I am willing to admit that we may not always get it right while making such decisions on clinical grounds alone. Should we consider this as an admission of defeat? Cook et al³ noted that in 40% of instances, the surgical decision making was altered by gait analysis. Looking at it in another way, "They got it right in 60% of instances," and we may have to adopt this positive outlook in areas where there is no hope of getting gait analysis on our children with cerebral palsy.

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Author's Response:

Mr Joseph's interest in and observations concerning my Current Issues position paper that appeared recently in the *Journal of Pediatric Orthopaedics* are appreciated.¹ His thoughtful comments raise issues that have been debated in and out of the medical literature for over 15 years.^{2,3}

Why do I believe that all ambulatory children with cerebral palsy (CP) should have quantitative gait analysis (QGA) included as part of the clinical decision-making process? I can identify 2 reasons. First, without QGA, the clinician must rely upon data generated from the physical examination and observational gait analysis for clinical decision making. It has been shown that data, such as range of motion, generated from the physical examination of children with CP have poor intraobserver and interobserver reliability.⁴ In addition, it has been shown that there is poor correlation between physical examination data and objective kinematic data (eg, popliteal angle measurement and knee extension in stance phase).⁵ We should not perform a hamstring lengthening to improve a popliteal angle measurement, but rather to improve knee extension in the loading response and terminal swing subphases of the gait cycle. We know from QGA that the 2 are not always closely correlated. In addition, experienced clinicians performing video-based observational gait analysis have been shown to be limited in their ability to identify the majority of kinematic parameters used in clinical decision making.⁶ Second, it is difficult to determine the outcome after an intervention if there is no objective, quantitative measure before the intervention. Mr Joseph notes that "in our center, we have diagnosed...(stiff-knee gait)...clinically...(ie, without QGA)...and the outcome of distal rectus transfers has been gratifying." How does he know this? What does he compare before and after? Would this approach be acceptable in other areas of orthopaedics, surgery, or internal medicine in this modern era that favors an evidence-based approach to clinical decision making and assessment

of outcome? Communities with clinically "gratifying" outcomes for ambulatory children with CP that have subsequently introduced QGA to the decision-making process have noted a significant number of cases with iatrogenic gait deviations (eg, crouch gait), usually after inappropriate or incomplete orthopaedic surgical treatment.^{7,8}

Quantitative gait analysis provides more and better data for clinical decision making and individualized assessment of outcome in children with CP. Without QGA, clinical decision making and outcome assessment are less objective, less consistent, and cannot be efficiently shared between centers. Previous paradigms do not generate adequate objective information to meet current standards for evidence-based medical practice. For these reasons, all ambulatory children with CP should have QGA included as part of the clinical decision making and outcome assessment processes. Unfortunately, as Mr Joseph points out, not all ambulatory children with CP can receive QGA. I can identify 2 situations in which this is so. First, the health care system truly cannot afford to provide the service. In such a situation, the orthopaedist should not "... stop offering surgery... to children with CP." Instead, the surgeon does what Mr Joseph suggests, which is to stay up on the current related medical literature and use what can be safely applied in the particular clinical setting. Second, the health care system can afford to provide the service but chooses not to. As I noted in the Current Issues article, this is usually justified based upon a cost-benefit analysis of the provision of the particular service.¹ Why is it an acceptable standard in many communities to routinely obtain a magnetic resonance imaging (MRI) study of the knee before a knee arthroscopy, yet not insist on a comprehensive QGA before surgery to improve ambulation in children with CP? The cost of each study is similar, yet the decision making before a single-event multilevel surgery for children with CP is significantly more complex than that required before a knee arthroscopy. It can be argued that the value of the QGA is greater than the MRI, yet only the latter is funded. I believe that this is because the information from the MRI is easier to understand and apply

than that from the QGA. In addition, the QGA primarily benefits a societal group (ie, children with disabilities) that is relatively small and not politically influential. Availability of, and access to, QGA are actually social issues that are determined according to societal priorities.

As noted in the “Medical Professionalism in the New Millennium: A Physician Charter,” physicians should be committed to continually improving the quality of and access to care, and the just distribution of societal resources, for all of our patients.⁹ Mr Joseph’s hospital has adequate resources for “sophisticated facilities for MRI... CT...in-vitro fertilization... renal transplantation... (and) coronary angioplasty” but does not have the funds to “refurbish a very basic gait laboratory... which is now defunct.” Unless orthopaedists demand QGA for children with CP, it will not be

provided. The resources are limited, the distribution process is political, and I sympathize with Mr Joseph’s challenges and frustrations. Although I recognize that all children with CP cannot receive QGA to assist in clinical decision making and assessment of outcome, I continue to believe that they should.

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