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Sensory Deficit in Conservatively Treated Neonatal Brachial Plexus Palsy Patients

Letter to the Editor

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We read the article by Brown et al. on hand sensorimotor function in older children with neonatal brachial plexus palsy (NBPP) with interest. The authors concluded that sensory function in NBPP may be impaired and challenge the common notion that sensory recovery is good in NBPP. These conclusions confirm our earlier ones.

Brown et al. did not find significant differences using Semmes-Weinstein filaments, whereas we did, and they found differences for stereognosis, whereas we did not. This may be because their population included more severely affected NBPP patients than ours; our population was older than theirs, and test applications differed in details. Brown et al. addressed the limitations of timing differences and concluded that a large effect size (Cohen d) indicated clinically important differences. However, Cohen d’s designation of effect size need not reflect practical importance, so it remains doubtful whether these timing differences impair function in daily life.

We had addressed two additional themes regarding sensory function in NBPP. The first was the origin of the common notion that sensory function is good in NBPP: this was likely due to authors overemphasizing the few unimpaired functions at the cost of many impaired ones. The second theme was why sensory deficits in NBPP do not follow the adult pattern with distinct sensory deficits following root and nerve innervation. We attributed the absence of sensory “gaps” in NBPP to a characteristic unique to NBPP: a neuroma in continuity allowing axons to reinnervate target regions, albeit with cross innervation. We had stressed that in this respect the sensory and motor abnormalities of NBPP are quite similar.

Finally, Brown et al. call attention to a possible contribution of altered central nervous system to explain the tactile impairment in NBPP. We found evidence for a central impairment affecting motor function in NBPP and agree that it may also affect sensory function. However, peripheral factors are likely to explain part of the sensory impairment in NBPP through reduced numbers of peripheral axons and extensive cross innervation. The latter may well contribute to altered central processing.

References