BOLD fMRI and fcMRI in the Pediatric Brachial Plexus Injury Population: Evaluating Cortical Sensory Network Plasticity

Jacques A. Machol IV, MD, Rupeng Li, MD, Ph.D., Nicholas Flugstad, MD,
Ji-Geng Yan, MD, PhD, James S. Hyde, PhD, Hani Matloub, MD

Abstract

Background: Brachial plexus birth palsy is a debilitating injury. The most common of these palsies are upper lesions involving the C5-C6 roots of the brachial plexus (1,2). Nerve root avulsions may occur; these cannot be repaired primarily. Principal goals of reconstructive procedures include restoration of shoulder abduction and biceps flexion (3). There are many options for nerve transfer for neurotization of injured nerves (4). Previous work using blood oxygen level dependent (BOLD) functional magnetic resonance imaging (fMRI) in the rat model has demonstrated a high degree of brain remodeling following peripheral nerve injury and repair (5-6). We present our initial findings using air-puffer somatosensory stimulation for 3T BOLD fMRI assessment in a pre-operative pediatric brachial plexus injury patient.

Methods: An 11-month-old female with left brachial plexus birth palsy was evaluated. The patient was noted to have injury to the C5-C6 nerve roots resulting in 0/5 shoulder external rotation and posterior deltoid function. This pathology was confirmed via pre- and intra-operative EMG and during post-scan surgical exploration.

3T BOLD fMRI imaging was performed prior to surgery. EPI imaging was obtained while a timed air-puff mechanism delivered somatosensory stimulus to the C5-C6 dermatome (lateral deltoid region). This was completed in duplicate for the injured side (left) and then again, in duplicate, for the healthy side (right). The primary somatic sensory cortex in the post central gyrus (S1) was the region of principal evaluation. These functional images were utilized to contrast the injury side cortex to the non-injury side cortex. The injury patient's S1 cortical function was then compared to a healthy adult using the same somatosensory stimulus BOLD fMRI protocol. Functional connectivity MRI (fcMRI) was also performed.

Results: The BOLD fMRI investigation demonstrated a significant difference between the injury side primary somatic sensory cortex function when compared to the non-injury side in a brachial plexus birth palsy patient. This variance is also illustrated when compared to a healthy adult subject undergoing the same functional imaging parameters. fcMRI demonstrated significant intra-cortical connectivity differences.

Conclusions: This novel application of BOLD fMRI and fcMRI has demonstrated inter-cortical somatosensory differences in a high brachial plexus birth palsy patient. The 3T BOLD fMRI model proposed is applicable to demonstrate cortical sensory pathology in the pre-operative patient with nerve root injuries. Ultimately, this model will be utilized to prospectively investigate cortical sensory plasticity after nerve transfer for brachial plexopathy treatment.

References:

1. Foad SL, Mehlman CT, Ying J. The epidemiology of neonatal brachial plexus palsy in the united states. J Bone Joint Surg Am. 2008 Jun;90(6):1258-64.
2. Hoeksma AF, ter Steeg AM, Nelissen RG, van Ouwerkerk WJ, Lankhorst GJ, de Jong BA. Neurological recovery in obstetric brachial plexus injuries: An historical cohort study. Dev Med Child Neurol. 2004 Feb;46(2):76-83.
3. Narakas AO. Injuries of the brachial plexus and neighboring peripheral nerves in vertebral fractures and other trauma of the cervical spine. Orthopade. 1987 Feb;16(1):81-6.
4. Wood MB, Murray PM. Heterotopic nerve transfers: Recent trends with expanding indication. J Hand Surg Am. 2007 Mar;32(3):397-408.
5. Bandettini PA, Wong EC, Hinks RS, Tikofsky RS, Hyde JS. Time course EPI of human brain function during task activation. Magn Reson Med. 1992 Jun;25(2):390-7.
6. Kwong KK, Belliveau JW, Chesler DA, Goldberg IE, Weisskoff RM, Poncelet BP, et al. Dynamic magnetic resonance imaging of human brain activity during primary sensory stimulation. Proc Natl Acad Sci U S A. 1992 Jun 15;89(12):5675-9.

Disclosure/Financial Support
Funded by grant 2RO1EB000215-23A1 NIH United States (Dr. James Hyde).