Investigation of vibration induced artifacts in clinical diffusion weighted imaging of the brain

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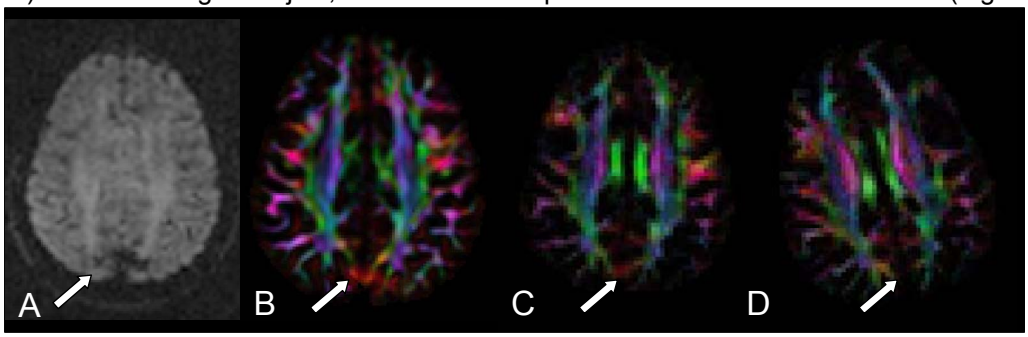
Introduction: A vibration induced artifact on diffusion tensor MRI (DTI) metrics has been reported in the literature. The artifact is consequent to spurious localized signal-loss in the diffusion-weighted images acquired with a horizontal component of diffusion sensitization. It manifests as increased diffusivity in the left-right direction, increased FA, and red appearance in directionally encoded color (DEC) maps, mainly in parietal-occipital regions. This artifact was described on a 3T Siemens Trio and attributed to the vibrations of the patient table from the gradient switching associated with diffusion preparation period[1]. To our knowledge this artifact has not been reported on GE or Philips scanners. We noticed that a highly publicized case report of DTI detected brain plasticity[2] may have been based on data corrupted by this artifact, although the data was obtained on a GE scanner. Briefly, a 39-year old man who spontaneously recovered from being in a minimally conscious state for 19 years was scanned twice. There was increased FA in the parietal-occipital region with red appearance in the DEC map at Time 1 that was not present at Time 2. This was interpreted by the authors, not as an artifact, but as a "transitional stage of an ongoing process…of possible axonal regrowth." Notably, this article has been cited 13 times with calls to change medical dogma[3], and entering the discussion of an Institute of Medicine exploratory meeting to set policy[4], and the authors have been interviewed on a national television program. This prompted us to review systematically our clinical DTI data that we have acquired on both GE Signa HDX and Siemens 3T Trio scanners. We examined historical data and tested the effect of correction strategies—as suggested by Gallichan[1]—on a single subject to optimize our DTI protocol prior to starting a new prospective study.

Methods: Archival GE data was acquired at low (3mm isotropic) and higher (2.5mm isotropic) resolution with various FOV, TR and TE acquired on pediatric subjects (99 autistic, 78 typical) on a 1.5T GE Signa and adult subjects (various GE scanners). Archival Siemens data was collected before and after a manufacturer upgrade to the scanner bed intended to eliminate the vibration artifact[5]. 42 pediatric datasets (23 typical, 19 epilepsy) were obtained on a 3T Siemens Trio ranging in age from 7-16 years. DTI data was acquired with 30 directions at b=1000s/mm^2 and 5 b=0s/mm^2, repeated twice for 70 brain volumes, no averaging, 2.5 mm isotropic. On the same Siemens scanner, we conducted tests on a single subject (age 32 male). Parameters that were kept constant include 41 diffusion directions, 31 b=1100s/mm^2, 5 b=300s/mm^2, and 5 b=0s/mm^2, parallel imaging. We examined using full k-space acquisition, then keeping full k-space also increased the TR, used bipolar gradient acquisition, and altered head position by pitch and roll $\pm$30 degrees. We conducted a systematic visual inspection of raw images and report the percentage of datasets where the artifact was evident.

Results: The artifact was noted in 7% of the pediatric GE datasets, none of the adult GE datasets, and 50% of the Siemens data. Unexpectedly, there was a higher percentage of datasets affected after the upgrade (71%, 20/28) compared to pre-upgrade (7%, 1/14). For the single subject, the artifact was present in the raw diffusion data (Figure Panel A) and DEC map (Panel B). The artifact was reduced when using full k-space acquisition (Panel C), but was eliminated by altering the roll (Panel D). Increasing the TR, setting the gradient to bipolar, and altering the pitch did not affect the artifact.



Discussion: We expand on a systematic vibration artifact previously described in the literature in three important ways. First, the artifact is more widespread than first described because it is not limited to Siemens scanners. Although less frequent in the GE data, the artifact is present and because of its low incidence, it may be missed more often. We suggest that regardless of scanner manufacturer, data should be systematically inspected for this artifact. Secondly, we offer another potential solution to reduce the artifact as we found that previously proposed solutions to avoid the artifact—manufacturer bed upgrade and full k-space acquisition—did not entirely eliminate the problem. We contend that altering the roll position of the head reduces the amplitude of the vibrations within the brain and disrupts the directional bias of the artifact. Other factors, e.g. patient weight, may affect the prevalence of the artifact. Lastly, the artifact may be the basis for a clinical misinterpretation that has been cited as evidence to change policy and practice. The speed that this article was propagated was assisted by the inherent interest of the case details; however, it also illustrates the danger of premature clinical interpretation of DTI data. We suggest that repositioning the patient by adjusting the roll would be a simple and practical method to determine if such findings were indeed axonal regrowth or artifact.

References: 1. Gallichan D et al, 2010, Hum Brain Mapp, 31(2):193-202. 2. Voss HU et al, 2006, J Clin Invest, 116(7):2005-11. 3. Laureys S, 2006, Curr Opin Neurol, 19(6):520-7. 4. Finns JJ, 2011Trans Am Clin Climatol Assoc.;122:336-46. 5. Liu KC et al, 2011, US Patent No. 7,924,008 B2