Identification of the epileptogenic tuber with diffusion-weighted MRI in patients with tuberous sclerosis and epilepsy

F. Jansen¹, B. Kees¹, O. van Nieuwenhuizen¹, G. Huiskamp¹, A. van Huffelen¹, J. van der Grond¹
¹University Medical Center, Utrecht, Utrecht, Netherlands

Purpose
Patients with tuberous sclerosis complex (TSC) and drug-resistant epilepsy may be considered candidates for epilepsy surgery. However, as most patients with TSC have multiple, potentially epileptogenic, tubers this option is often rejected. In this study we tested whether diffusion-weighted MRI (DWI) enables differentiation of epileptogenic tubers from inert ones.

Methods
In four patients with a clear unifocal interictal spike activity, identified with simultaneous high-resolution electro-encephalography (EEG) and magneto-encephalography (MEG) recording, fluid-attenuated inversion recovery (FLAIR) MRI and DWI scans were performed. In these four patients, apparent diffusion coefficient (ADC) maps were calculated in the identified epileptogenic tuber and compared with those in non-epileptogenic tubers and regions of normal appearing cortex.

Results
In the four patients both FLAIR, DWI, and ADC maps showed multiple tubers. Examples of images of one patient are shown. On the trace DW images and ADC maps, tubers were characterized by decreased and increased signal intensity respectively, as compared with the surrounding normal appearing white and gray matter (figure 1). The trace ADC of the four epileptogenic tubers (mean 1099 mm/s, SD 35.0) was significantly higher (p<0.001) than that of 18 non-epileptogenic tubers (mean 926 mm/s, SD 69.4). Furthermore, the trace ADC of the non-epileptogenic tubers was significantly higher (p<0.001) than the trace ADC of 16 regions of normal appearing cortex (mean 784 mm/s, SD 61.7) (figure 2).

Conclusions
DWI is a promising tool in differentiating epileptic from inert tubers in TSC. The histopathology of tubers may explain the increase in diffusivity in TSC as this reflects an increase of extracellular space, and a loss of structural organization. The increase of ADC in epileptogenic tubers may be explained by an even higher loss of neurons or by edema, caused by the seizures themselves. DWI may be of clinical importance for the identification of epileptogenic tubers in patients with TSC and intractable epilepsy and thus may facilitate the selection of candidates for epilepsy surgery.

References

Figure 1. FLAIR image and corresponding ADC map of one patient. Tubers are shown as hyperintense regions. The epileptogenic tuber is located in the right temporo-occipital region.

Figure 2. Box plot of the mean ADC of the 1. epileptogenic tubers, 2. non-epileptogenic tubers, 3. normal appearing cortex, in the four patients (p values< 0.001).