Recovery from homonymous hemianopia in MS – A 2 year follow-up study with fMRI and DTI

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ABSTRACT
A 30 year old patient with early relapsing-remitting MS presenting with hemianopia and a large new lesion in the optic radiation was followed over 2 years clinically, with fMRI and DTI. While the patient showed a fast clinical recovery there was a time lag and incomplete recovery from an initial reduction of activated cortical voxels on fMRI, while permanent fiber damage in the optic radiation was demonstrated by DTI. DTI and fMRI can provide new information on the relationship of structural and functional recovery in MS.

INTRODUCTION
In patients with early relapsing-remitting multiple sclerosis (MS) most clinical relapses are followed by complete clinical remissions. Incomplete recovery and the accumulation of residual deficits may lead to disability relatively early in the course of the disease. To date there is only limited knowledge about the contribution of destructive and adaptive processes involved in the pathophysiology and functional recovery after the relapse. Evaluation of white matter anisotropy by diffusion weighted (DW) MRI and of cortical activation by functional MRI (fMRI) provide new means to assess both structural and functional aspects of MS lesions.

MATERIALS AND METHODS
A 30 year old patient with early relapsing-remitting MS presented with a new hemianopia to the right. Inkeeping with the clinical deficit a large new contrast enhancing lesion in the postgeniculate visual pathway along the posterior horn of the lateral ventricle was demonstrated on MRI. She was treated with i.v. methylprednisolon and made a realtively fast clinical recovery. FMRI, DTI and visual field testing were performed 4 times over 2 years: In the early recovery phase when the hemianopia had improved to an upper quadrant field defect, 2. after 9 weeks when there was no clinical field defect detectable on confrontation or on Goldmann perimetry, 3. after 21 weeks when the patient reported slight further improvement in that reading for longer periods had become less tiring.4. after 24 months.
MRI was performed with a 1.5 T Magnetom VISION, SIEMENS.
1) Proton density-, T_2-, T_1-weighted
2) DTI FLAIR-SE-EPI acquisition (TR/TI/TE = 6000/2000/110 ms, slice thickness TH = 5 mm, FOV = 240x240 mm^2, MAT = 128x128, interp. 256x256) containing gradient lobes for DW (b = 0, 1030 s/mm^2). Amplitude images were averaged from 8 measurements.
3) T_1-weighted after 0.2 mmol/kg Gadodiamid-DTPA.
fMRI scans were acquired using a multislice 2D EPI sequence with 19 axial slices (3 mm thickness, 1 mm gap, 64x64 matrix, FOV 220 mm, TE = 66 ms, Talairach normalization). As input we used the well established simple visual 6 Hz checkerboard stimulation. It was investigated in one block of 100 measurements alternating rest and activation in 5 groups of 10 circles each. Activated pixels were identified by correlating raw data with a convolved reference function on a pixel by pixel basis (BrainVoyager 2.6) (2,3).

RESULTS
Color coded DTI demonstrated loss of fibers of the optic radiation on the left. On follow-up MRI there was resolution of the initial inflammatory edema and residual enlargement of the posterior horn of the lateral ventricle indicating atrophy and fiber loss in the lesion. (Fig. 1) In the acute stage, there was a significant decrease of the BOLD response (mean signal intensity as well as # of activated pixels) in the left visual cortex including MT/MST. This improved incompletely on examinations 2 -4. (Fig. 2)

DISCUSSION
The optic radiation is an anatomical structure that can be closely related to visual function. It can be particularly well demonstrated with DTI due to its highly organised fibers. In MS it offers the opportunity to relate structural and functional characteristics of inflammatory/ demyelinating lesions to the clinical deficit.
Despite complete clinical recovery from hemianopia (examination 2) there was evidence of fiber destruction and subclinical compromise with less activity in the left visual cortex. This suggests compensatory capacities of the affected parts of the visual system.

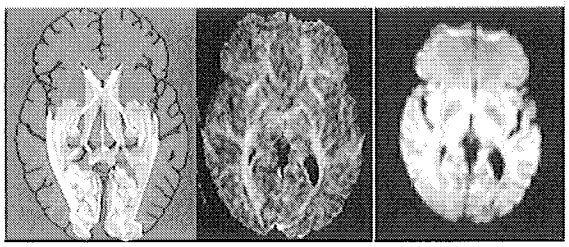

Fig. 1 Anatomic sketch – Color coded DTI – Fibre tracking of optic radiations showing reduction of fibers on the left

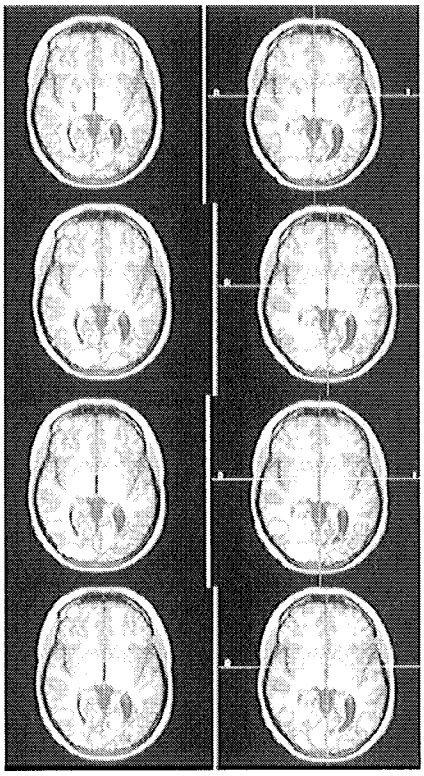

Fig. 2 fMRI follow-up over 2 years, gradual recovery of cortical activation in the left visual cortex

REFERENCES
1. van Gelderen P, Vleeschouwer MHM. MRM 31,154,1994
2. Bandettini, P.A., et. al., MRM 30, 161,1993
3. Goebel R, NeuroImage 3, S604, 1996