

## S29. Diffusion tensor imaging: perspectives in neuropsychiatry

*Chairs:* W.-K. Strik (CH), I. Agartz (S)

### S29.1

Possibilities and limitations of DTI: a methodological introduction

E. Formisano. *The Netherlands*

No abstract was available at the time of printing.

### S29.2

DTI in neuropsychiatric disorders

K.O. Lövlblad. *Switzerland*

No abstract was available at the time of printing.

### S29.3

Abnormal brain white matter in schizophrenia: a diffusion tensor study

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Disturbances of interconnections between brain structures have been proposed to explain the diverse brain abnormalities found in schizophrenia on functional and structural brain imaging studies. MR diffusion anisotropy imaging (DTI) is sensitive to disruptions of commissural white matter pathways and can be used to study axonal connectivity, myelin packing and fiber orientation.

The fractional anisotropy and the mean diffusion coefficient were measured in the cerebral volume of 20 schizophrenic and 24 healthy subjects, men and women, using DTI. In addition, 3D SPGR was used for segmentation of brain tissue into grey and white matter and cerebrospinal fluid.

In the schizophrenic patients, the fractional anisotropy was reduced in the splenium of the corpus callosum and in the adjacent occipital white matter. The segmentation revealed no tissue deficits in the volume of the reduced fractional anisotropy. The mean diffusion was globally increased in the total white and grey matter volume of the brains of the schizophrenic patients compared with the healthy subjects.

The findings support the view that global and regional white matter abnormalities occur in schizophrenia. Results from other DTI studies in schizophrenia will be briefly reviewed.

### S29.4

DTI in hallucinating schizophrenic patients

D. Hubl. *Switzerland*

No abstract was available at the time of printing.

### S29.5

Microstructure of temporo-parietal white matter as a basis for reading ability: evidence from DTI

T. Klingberg. *Sweden*

No abstract was available at the time of printing.

## S30. Treatment and management of patients with somatoform disorders – Part II

*Chairs:* A. Lundin (S), A. Barsky (USA)

### S30.1

Treatment and management of the somatizing patients – introduction

A. Lundin\*. *Danderud Hospital AB, Department of Rehabilitation Medicine, Stockholm, Sweden*

Awareness of the impact of somatisation and medically unexplained symptoms in public health is growing among health care providers. There is today a growing need for further development of methods for treatment and management of these conditions. As medically unexplained symptoms appear at all levels of care – in primary care as well as in different specialist settings and psychiatry – and with different degrees of severity, interventions will have to be differentiated and relevant to the context in which they appear. The allocation of relevant diagnostic and treatment resources will be discussed.

The symposium will present results of different treatment modalities, tailored for specific somatic syndromes of medically unexplained nature. An overview of the diagnostic panorama, and how somatisation presents in different medical settings will be given as an introduction.

### S30.2

Treatment of patients with functional gastro-intestinal disorders

F. Creed. *UK*

No abstract was available at the time of printing.

### S30.3

Treatment of chronic fatigue syndrome: why not?

M. Sharpe\*. *University of Edinburgh, Department of Psychiatry, Scotland, UK*

Chronic fatigue syndrome (CFS) is a descriptive diagnosis for chronic disabling medically unexplained fatigue. CFS is now known to overlap with other so-called functional syndromes including irritable bowel syndrome and fibromyalgia. There is also a substantial overlap with the so-called psychiatric syndromes of depressions and anxiety. There has been some progress in finding biological correlates of the condition, but its aetiology remains controversial. A wide range of treatments both pharmacological and non-pharmacological has been attempted. There is limited evidence for antidepressant drugs and steroids, but the strongest evidence is for a rehabilitative cognitive behavioural type treatment, which includes graded increases in activity. There are now a number of trials demonstrating the superiority of this over usual care and other non-specific treatments. Cognitive behavioural rehabilitation is not a panacea but it is of substantial benefit to many patients.

However, the availability of CBT for CFS remains extremely limited. There are number possible reasons for this including the lack of support for this treatment from patient organisations. The interesting question therefore is not how do we treat CFS but why not?