

This testifies to the robustness of the main Anglo-Canadian findings.

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The importance of severity as a factor in post-traumatic stress disorder

SIR: There are several points that we would like to raise about the recent single case report by Spector & Huthwaite (*Journal*, July 1993, 163, 106–108), on the treatment of Y, a patient reported to have post-traumatic stress disorder (PTSD) following a road traffic accident. We do not feel there is sufficient evidence in the report that the subject's experience fulfils the part of the DSM-III-R criteria A, of being outside the range of normal human experience. There is no mention of the subject thinking that her life was in danger, or that she witnessed any horrific scenes. Without such information we are not convinced that this case satisfies this criteria for a diagnosis of PTSD although it clearly is an experience which would be markedly distressing to most people.

Secondly, PTSD is a condition which presents with a wide spectrum of severity and this can be overlooked when employing a categorical diagnosis which can depend on the presence or absence of a single symptom. Neither DSM-III-R or ICD-10 enables one to measure symptom intensity on a continuous scale. The severity of Y's PTSD is not considered in the article and we believe that this variable is vital in assessing treatment efficacy.

One research tool developed by the National Centre for PTSD, USA, which has been incorporated in validation studies and outcome research by the PTSD Unit at RAF Wroughton (Neal *et al*, 1993), is the Clinician-Administered PTSD Scale-1 (CAPS-1; Blake *et al*, 1990). This is based on DSM-III-R criteria but in addition has an intensity scale assessing severity. We would suggest that clinicians

involved in PTSD research consider the use of an instrument using both dichotomous and continuous scales. A revised computerised version of the CAPS-1 has been developed and validated at RAF Wroughton.

There is a need for well designed controlled studies into the treatment of this common and disabling condition. In our experience at the PTSD Units at RAF Hospital Wroughton and RN Hospital Haslar, even non-specific interventions, such as a simple psychiatric assessment, can result in a marked clinical improvement. Such an improvement could be misattributed to treatment methods in case reports or research studies where this is not considered.

BLAKE, D. D., WEATHERS, F. W., NAGY, L. M., *et al* (1990) A clinician rating scale for assessing current and lifetime PTSD: the CAPS-1. *The Behaviour Therapist*, 13, 187–188.

NEAL, L. A., BUSSUTTIL, W., HEREPATH, R., *et al* (1993) Convergent validity of measures of PTSD in a mixed military & civilian population. *International Journal of Traumatic Stress* (in press).

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Right hemisphere damage v. dysfunction in Tourette's syndrome

SIR: "Diagnosing a cerebral dysfunction on the sole basis of neuropsychological test results" is a questionable clinical method as Lanser *et al* point out in their report of absence of right hemispheric dysfunction in Tourette's syndrome (*Journal*, July 1993, 163, 116–118). They criticise previous reports interpreting Tourette's syndrome (TS) patient's poor performance on visual-perceptual tasks as suggestive of right hemispheric involvement in the pathogenesis of TS. To support their point, they report a neuropsychological comparison of individuals with TS and with "proven lesions of the right hemisphere (RH dysfunction)". Their "unexpected results" indicate that 7 to 16 children with "RH dysfunction" do not show any disturbance in right hemisphere functions (as assessed by their battery), and all of the children with TS perform "free from any neuropsychological signs or organic impairment". Since there are not many significant differences between the groups, they claim that this absence of difference should not lead us to identify TS as a RH dysfunction. However, there are many caveats which they do not discuss in the text.

Firstly, comparing two clinical groups in the absence of a normal control group is not "fair". Since there is not a one-to-one correspondence between

damage and *dysfunction*, even in elderly patients with brain lesions, it is not surprising to find a huge variability in terms of neuropsychological functioning in the "brain damaged" group. On the other side, psychiatric literature has an abundance of lateralised "soft" neurological findings, for example in obsessive-compulsive patients, in whom a "damage" has not been identified yet. However, increasing availability of finer imaging methods have made it clear that there are alterations in the brain structures of these individuals, and these alterations are probably associated with these "soft" findings which may be indicative of a "dysfunction without damage" (Insel, 1992).

Secondly, the battery of tests administered by Lanser *et al* is not the best predictor of the level of functions predominantly subserved by the right hemisphere. For example, scoring of the Bender Visual Motor Gestalt Test (and drawing) as normal v. below age level v. organic may be helpful in clinical practice; but for the purpose of estimating the level of contribution to the clinical picture by a single hemisphere, this scoring may not be sufficient. Just think of the possible overlaps between "below age level" and "organic" categories.

Thirdly, finding a neuropsychological impairment similarity between the right brain damaged and dysfunctional children and the TS children does not necessarily suggest a "RH dysfunction". The cause and nature of the damage (as acute onset events such as head trauma) and patient characteristics (as age or gender) may determine the consequences in a different way than they would in TS. The evidence until now suggests pre/perinatal insult and genetic factors play a significant role in the *development* of TS, in addition to several other factors which may affect the course of an illness (Leckman *et al*, 1992). This early and silent onset might be followed by substantial changes in cerebral organisation as suggested by recent magnetic resonance imaging studies (Peterson *et al*, 1993). This 'new order' in the brain *might* result in a "RH dysfunction-type" output. However, this does not necessarily indicate a pathogenesis similar to the "right brain damage" caused by a tumor or infarction.

The data reported are certainly not suggestive of a "RH dysfunction". But it would not also be possible to say the opposite, considering the limitations of group compositions and instruments as mentioned above. A recent example for a more comprehensive assessment battery can be found in Randolph *et al*'s (1993) report of correlations between tic severity and some neuropsychological measures like executive functions, visuospatial/constructional and attentional vigilance measures; the latter two of which

have been reported to be predominantly subserved by right hemisphere (Pardo *et al*, 1991).

INSEL, T. R. (1992) Toward a neuroanatomy of obsessive-compulsive disorder. *Archives of General Psychiatry*, **49**, 739-744.

LECKMAN, J. F., PAULS, D. L., PETERSON, B. S., *et al* (1992) Pathogenesis of Tourette syndrome: clues from the clinical phenomenology and natural history. *Advances in Neurology*, **58**, 15-24.

PARDO, J. V., FOX, P. T. & RAICHLER, E. M. (1991) Localization of a human system for sustained attention by positron emission tomography. *Nature*, **349**, 61-64.

PETERSON, B. S., RIDDLE, M. A., COHEN, D. J., *et al* (1993) Reduced basal ganglia volumes in Tourette's syndrome using 3-D reconstruction techniques from magnetic resonance images. *Neurology*, **43**, 941-949.

RANDOLPH, C., HYDE, T. M., GOLD, J. M., *et al* (1993) Tourette's syndrome in monozygotic twins: relationship of tic severity to neuropsychological function. *Archives of Neurology*, **50**, 725-728.

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Compulsive spitting as a neuropsychiatric symptom in Indian psychiatric practice

SIR: In Indian settings, many unusual psychiatric symptoms have been reported, for example asneezha, Bhanmati sorcery, Sati, Ascetic syndrome, Koro, Dhat syndrome, and so on (Bhatia, 1992; Neki, *Journal*, September 1973, **123**, 257-269).

We have recently come across the symptom of compulsive spitting in a number of psychiatric patients. There were: three cases of seizure disorder (spitting occurred as a part of aura); five cases of schizophrenia (in whom spitting occurred as a type of motor stereotypy); one case of mania (spitting occurred as a part of grandiosity—that this world has nothing worthy of him); two cases of anxiety state (spitting occurred as a part of stereotyped behaviour); three cases of obsessive-compulsive disorder (spitting as a manifestation of compulsion to prevent the entry of any dust in the mouth); three cases of tic disorder which includes one case of Tourette's syndrome (spitting occurred as a manifestation of motor tic).

BHATIA, M. S. (1992) *Dictionary of Psychiatry, Psychology and Neurology*. Delhi: CBS Publishers and Distributors.

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