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).

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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 asneeza, 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).

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