All that tics may not be Tourette's

SIR: Gilles de la Tourette syndrome (GTS) is characterised by multiple motor and one or more vocal tics lasting more than a year. Transient tic disorder (TTD) represents tics of duration less than a year, while in chronic multiple tics (CMT) they (vocal or motor) have been present for more than a year (American Psychiatric Association, 1987).

The hereditary nature of the syndrome was recognised by Gilles de la Tourette himself. There are still controversies about the possible phenotypes of the putative GTS gene(s), whether all cases of tics represent the GTS clinical spectrum, and what proportion of patients with tics have a disorder genetically related to GTS.

Family studies have reported that TTD and CMT are aetiologically related to GTS (Kurlan *et al*, 1988). While GTS is probably the most common cause of tics, they may also be due to other causes. Clinically the distinction between 'GTS tics' and other forms of tics is difficult. Some of these may represent habits, mannerisms, or physiological tics. GTS tics characteristically have a waxing and waning course, are suppressible, suggestible, and exacerbated by stress. While this is useful in clinical practice, such phenotypic definitions are far from optimum for genetic and linkage studies.

Eapen *et al* (1993), using a 'goodness of fit test', reported that the predicted and observed frequencies of GTS were not significantly different. However, when relatives with CMT and TTD were also included as affected, the observed rates were significantly different from the expected, indicating a poor fit for the data. This suggests that not all relatives with tics have a disorder that is genetically related to GTS. In these circumstances, motor tics (chronic and transient) may be phenocopies.

Although much of the genome has been excluded (Heutink *et al*, 1990; Pakstis *et al*, 1991), this may be a reflection of incorrect definition of the phenotypes. Definitive answers to some of these questions may have to await the development of a genetic marker for the GTS gene. In the meantime, if an 'endophenotype' can be identified, this will help link the clinical phenotype (the external clinical manifestation that can be observed) with the genotype (the underlying genetic mechanism).

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Brain, mind and behaviour

SIR: Fenwick (*BJP*, November 1993, **163**, 565–573) has written an intriguing article on the influence of new imaging techniques in broadening our understanding of major psychiatric illnesses, and suggests that this increased knowledge of brain function will have consequences for the expert medical witness who has to give evidence in court. He suggests that in the area of diminished responsibility, the concept of the guilty mind belongs to a non-scientific era and that the subtle brain malfunction demonstrated by new neurophysiological techniques may come to assume increasing importance in matters of criminal responsibility.

In support of his theory, Fenwick describes "The Case of the Miserable Teenager", and is critical of the jury, who were not convinced by his explanation of the offence and returned a verdict of guilty to murder rather than culpable homicide on the grounds of diminished responsibility.

We believe we were among the psychiatrists for the Crown in this case, and there are certain important omissions in Fenwick's account. We are constrained in our comments by issues of ethics and confidentiality, but confining our observations solely to what is already in the public domain through the press reporting of the incident, there were components of motivation, planning and subsequent concealment which may have influenced the jury in reaching the decision it did. These aspects potentially offer a different interpretation to that put forward by Fenwick.

We feel that it is prudent to be cautious in appraising Fenwick's theory, and there should be objective scientific evaluation and independent confirmation of his propositions before neuroimaging