Britain is Talking About") illustrates its general interest.

Strober *et al* (1990) postulate genetic factors in the causation of anorexia nervosa and obviously this is also a possibility in the case of such identical twins.

Theander (1970), Crisp *et al* (1980) and Hudson *et al* (*BJP*, February 1983, **142**, 133–138) all agree on a 6–7% sibling risk in anorexia. The incidence of anorexic siblings may, however, be greater than some members of a family know or are prepared readily to admit.

Having been 'sensitised' by my contact with the family 'X', I found that no fewer than five of the 25 anorexic/bulimic patients referred to me over a one-year period turned out to have sisters who were also sufferers. In two of the 13 anorexic patients, their own condition had apparently been triggered by their anorexic sister's slimness (and perhaps concern and attention), and in another anorexic girl, her older sister's anorexia had been unknown to her. One bulimic girl had a bulimic sister (unknown to her parents) and another had a sister whose past bulimia was "a family secret" and only admitted after several interviews.

Case report. Family 'X' had four children: daughters 'B', 'C' and 'D', and 'D's' twin brother 'E'.

B was referred in 1974, at the age of 20 years, with a history of typical anorexia nervosa since the age of 16. Her pre-morbid weight was 60.8 kg (lowest weight 41.3 kg) at a height of 1.65 m. Her illness had apparently been triggered by jealousy of C's slimness. B recovered completely after treatment.

C was referred in 1980, at the age of 23 years, again with a typical history of anorexia since the age of 16. Her pre-morbid weight was 60.33 kg (lowest weight 27.7 kg) at a height of 1.7 m, and her anorexia had been triggered by the onset of B's anorexia. After treatment, C improved (weight 51.3 kg) but she married, moved to another city, and lost touch. Sadly, she apparently died the following year.

D, B's and C's younger sister, was referred in 1982, at the age of 17 years, with typical anorexia nervosa, apparently triggered by C's death. At referral her weight was 39.9 kg (normal weight 50.8 kg at a height of 1.69 m). She recovered with treatment, although she continued to be underweight and became a vegetarian.

B's and C's younger brother, and D's twin, E, was referred in 1986. Although his symptoms did not reach strict diagnostic criteria of anorexia nervosa, he had developed numerous curious rituals and beliefs about eating. He feared 'fatness' and had lost some weight to 57.6 kg (normal weight 68.0 kg at a height of 1.78 m). His symptoms had apparently been triggered off by the development of his twin's (D's) anorexia. He recovered completely with treatment and he, B and D remained well at enquiry in 1988.

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Neuroleptic malignant syndrome and carbamazepine?

SIR: Neuroleptic malignant syndrome (NMS) refers to a serious symptom complex which is sometimes seen in association with neuroleptic drugs. Symptoms include hyperthermia, muscle rigidity, altered mental status, autonomic dysfunction, elevated creatine phosphate kinase and leucocytosis. The pathomechanism has not yet been clarified but it has been thought that NMS develops after an acute dopaminergic transmission block in the basal ganglia and the hypothalamus due to treatment with dopamine receptor antagonists or withdrawal of dopamine receptor agonists (Weller & Kornhuber, 1992).

The association between carbamazepine (CBZ) and NMS has been suggested by O'Griofa & Voris (1991). However, the following should be kept in mind. CBZ is an anticonvulsant drug which is also used in psychiatric disorders and is therefore often given in combination with neuroleptic medication. The mode of action of CBZ is not yet completely understood, but stabilisation of the neuronal membranes by acting on the sodium and potassium channels most likely play a role (Macdonald, 1989; Schmutz, 1992). To our knowledge, there is no known action of CBZ on the dopamine receptor, so that CBZ is unlikely to be directly involved in the pathomechanism of NMS. Nevertheless, the records of CIBA contain 15 cases of NMS in which CBZ was part of the therapy. In most of these cases concomitant neuroleptic treatment could be held responsible for the development of NMS.

CBZ has been on the market for more than 20 years. To our knowledge the first case of NMS in

association with CBZ appeared in 1984, the time when the use of CBZ in psychiatric disorders became more common. Furthermore, we are not aware of any case of NMS where CBZ was given for only trigeminal neuralgia or epilepsy. It seems that CBZ alone is not likely to cause NMS, but that an underlying psychiatric disorder or concomitant neuroleptic treatment needs to be present. The combination of CBZ with dopamine receptor antagonists may, however, lead to increased neurotoxicity and warrants alertness for early signs and symptoms suggestive of NMS.

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CORRIGENDUM

BJP, December 1993, **163**, 833–844. The authors of the letter 'Dopamine D_2 receptor occupancy *in vivo* and response to the new antipsychotic risperidone' were transposed. Dr Busatto should have been placed as first author, Dr Kerwin as last author.

A HUNDRED YEARS AGO

Hypodermic injections of brain extract in mental diseases

The material was prepared by Messrs. Brady and Martin, and sent fresh twice weekly; it was called by them "cerebrine alpha". The injections began on August 20th, and were given twice daily for fourteen days to six patients. The arms were in all cases the seat of injection, and each was given under antiseptic precautions. The doses began at 5 minims, and were gradually increased to 15 minims.

The following complications resulted: Case B, the temperature rose 1° after each injection. An attack of syncope followed in the case of E on the tenth day, and a slight erythema followed the second injection in Case A. The pulse in each case was quickened, but no change was observed in respiration.

Case A (recurrent melancholia with fixed delusions) expressed himself after the sixth injection as "feeling much brighter." He conversed in a rational manner, went on improving, and was discharged recovered on November 2nd. In the remaining five cases the result was negative, and, beyond the above complications, nothing of interest transpired. There were two cases of chronic melancholia, two of primary dementia, one of acute mania.

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