Letters

Transient global amnesia and migraine: familial incidence

Sir: The pathophysiology of transient global amnesia is unclear: often an ischaemic aetiology has been suggested, but epileptic and migrainous mechanisms have also been invoked. A familial incidence in this syndrome has rarely been reported.^{1,2} We describe a family with an unusual incidence of transient amnesic attacks and migraine.

ER, a 72-year-old woman with 10 years of schooling, was admitted to hospital in March 1985, after a temporary attack of altered behaviour dominated by disturbance of memory. Two hours before she had been at home, conversing with her daughter-in-law, when suddenly she became strange, astonished and did not recognise some household objects as familiar; she asked her daughter-in-law about her presence in the house. She was not able to remember having had a walk in the morning and having prepared a trip to Milan for the next day in order to attend an opera at La Scala Theatre (she had been very excited about this on the previous day). On admission she was still confused, disoriented in time but not in space. General examination, ECG and neurological examination were normal. She asked the same questions over and over again, showing great difficulties in grasping simple information. Other high cerebral functions, clinically evaluated, were preserved. Retrograde amnesia was present, covering a period of several weeks. During the examination she began to recover and after 1 h she was apparently normal. Blood parameters, EEG, CT brain scan and cerebro-vascular Doppler sonography, subsequently performed, were normal. Neuropsychological tests for memory (verbal and spatial span, Rey 15 Words, Rey Complex Figure), administered a month later, gave normal findings; at this time, only amnesia for the period of the attack still remained. The patient's history revealed that she had suffered from common migraine until the menopause, and her mother and two siblings had suffered from vascular headaches (common and classic migraine). Furthermore, a brother of hers, MR, a 76-year-old healthy man, with 7 years of schooling, had suffered from an acute, transient attack of amnesia at the age of 52: after a windy day spent hunting with some friends, he had a 6-8 hour long episode of amnesia, without loss of consciousness, during which he was able to speak, walk and

drive. We examined this man, who had no physical abnormalities. Neuropsychological tests for memory proved normal. He was still able to describe the amnesic episode in sufficient detail, even though it happened more than 20 years before. His medical history was uneventful except for that episode and for migraine which in adolescence had been so marked that he had to interrupt high school. A sister had also had an episode of memory disturbances when she was young, but she had died many years before and we had no details about her. We think that, while ER suffered from a classical transient global amnesia attack, MR had probably had transient global amnesia with partial recollection of the episode, analogous to other rare observations.^{3,4}

Previous reports about familial transient global amnesia^{1,2} suggest that familial incidence may be explained by a high frequency of risk-factors for cerebro-vascular disease in the same family. Alternatively, it is possible that episodes of transient global amnesia in members of a same family are a coincidence, because of the frequent occurrence of this condition, which is certainly more frequent in medical practice than has been described. However, we would argue that the migrainous hypothesis of transient global amnesia is also compatible with a familial incidence of this syndrome and the presence of transient global amnesia and migraine in the same family supports this explanation. Even though the high frequency of both conditions can also be invoked to explain their association, this argument is not very convincing. We think that the hypothesis of a single mechanism underlying both transient global amnesia and migraine (the transient global amnesia-migraine connection⁵), as suggested by their presence in the same family, is an interesting one and should be the subject of further investigations.

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Taurocyamine in cerebrospinal fluid of neurological and psychiatric patients.

Sir: In 1981 Mori *et al*¹ reported that ω -amino acids could be the precursors of the corresponding guanidino compounds. Taurocyamine could be derived from taurine. Taurine, considered as an inhibitor of impulse transmission in the central nervous system,² occurs also in cerebrospinal fluid (CSF). Taurocyamine has been demonstrated in the CSF of uraemic patients³ and in some epileptics⁴ but not in normal neurological patients.⁵ The purpose of our study was to evaluate the presence of taurocyamine in CSF of patients with neurological and psychiatric diseases.

The study includes the analysis of cerebrospinal fluid samples of patients with neurological and psychiatric diseases (see table). The neurological patients were classified according to the Ninth Revision of The International Classification of Diseases.⁶ The psychiatric patients were classified according to the Third Edition of the Diagnostic and Statistical Manual of Mental Disorders.⁷ CSF was obtained by lumbar puncture. 3 ml of CSF were dried in a rotatory vacuum evaporator at 30°C and dissolved in 0.8 ml 0.2 N sodium citrate buffer, pH 2.2. The sample was directly applied to the chromatographic system. The separation of taurocyamine was carried out with 0.2 N sodium citrate buffer, pH 3.28 on a 69 × 0.9 cm chromatographic column with 55 cm bed of spherical resin (flow rate 50 ml/hr, 30°C) in an automatic amino acid analyser (Beckman, Unichrom) modified for guanidino compounds analysis. The colour reaction developed with Sakaguchi Reagent has been described by Durzan.⁸

Taurocyamine was eluted after 48 min. Under our conditions taurocyamine can be estimated at a concentration as low as 4 nmol/ml when using 3 ml CSF and read on the chromatographic pattern of a CSF specimen with and without addition of standard.

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Table Diagnoses and number of cases

Neurological patients		Psychiatric patients	
Diagnosis	Number	Diagnosis	Number
Sleep disorders	4	Affective disorders	19
Multiple sclerosis	3	Schizophrenic disorders	39
Central nervous system neoplasms	5	Paranoia	4
Meningitis	7	Brief reactive psychosis	9
Viral encephalitis	3	Neuroses	18
Extrapyramidal diseases	9	Adjustment disorders	5
Infantile cerebral palsy	5	Personality disorders	6
Infantile spinal muscle atrophy	3	Organic mental disorders	10
Peripheral nervous system diseases	14	Substance use disorders	5
Epilepsy	20	Other psychiatric disorders	8
Other neurological diseases	6	Total	123
Total	79		

taurocyamine at a concentration of 200 nmol. A small peak, perhaps urea, is seen after 40 minutes. Quantitative determination of taurocyamine is not without problems and overestimation can occur.⁹ Taurocyamine could not be estimated in any CSF of the patients examined. This means that high levels of taurocyamine as described in CSF of uraemic patients³ can be ruled out for patients with neurological and psychiatric diseases.

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Familial carpal and tarsal tunnel syndrome

Sir: The familial occurrence of nerve entrapment at the carpal and tarsal tunnel seems to have been rarely if ever described. Pedigrees of families liable to pressure palsies¹ have included evidence of sub-clinical abnormalities under the carpal tunnel but these patients had symptomatic palsies predominantly of the sciatic and ulnar nerves. We have recently studied a patient whose family members show a predilection to either the carpal tunnel syndrome or the tarsal tunnel syndrome.

A 61-year-old housewife was referred to one of us (MLS) with a history of pain in the foot on both sides characteristic of the tarsal tunnel syndrome. She was otherwise healthy, of average build without notably abnormal wrists or ankles. Electrophysiological study confirmed local abnor-

mality,² and significant asymmetry of the distal latencies to flexor hallucis and abductor quinti minimi.³ A sural sensory action potential was normal in amplitude and latency. The patient had had successful surgery for bilateral carpal tunnel syndrome. She had four sisters: one was asymptomatic. An elder sister had been treated for unilateral carpal tunnel syndrome, as had one younger sister. The youngest in the family, a woman aged 43 years, had had treatment for a unilateral carpal tunnel syndrome and a unilateral tarsal tunnel syndrome. The patient's mother had just been diagnosed as having the carpal tunnel syndrome. Unfortunately these other members of her family do not live in the UK and could not be examined.

This family show a striking predisposition to entrapment at the carpal tunnel and at the tarsal tunnel. There was no obvious abnormality of the wrists or ankles of the patient examined, and no stigmata of any connective tissue or skeletal disease. The other family members are not thought to be abnormal in any other way. Nevertheless it seems likely that the cause lies in the geometry of the carpal and tarsal tunnels, rather than undue nerve sensitivity to pressure stretch since none of the family members gave a history of other nerve pressure palsies and the patient studied had no evidence of diffuse neuropathy. Computed tomography of the wrists has shown that patients with the carpal tunnel syndrome have a smaller than normal space.⁴ The occasional coincidence of the carpal tunnel syndrome and tarsal tunnel syndrome has been reported before,⁵ but not its familial incidence.

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Fig Pedigree of family. Square = male, circle = female, black symbol = proband, CTS = carpal tunnel syndrome, TTS = tarsal tunnel syndrome, numbers = age in years.