FUNCTION OF THE CORPUS CALLOSUM IN SCHIZOPHRENIA

DEAR SIR,

We read with interest the paper by Shagass *et al*, (*Journal*, May, 1983, **142**, 471–76) and their failure to replicate the work of Jones and Millar (*Journal*, 1981, **139**, 553–57) in relation to possible dysfunction of the corpus callosum in schizophrenia.

Jones and Miller reported finding a relative synchrony of contralateral and ipsilateral somatosensory evoked potentials (SEP's) in a schizophrenic group as compared with a definite latency in normal controls. Shagass, using a similar technique of vibrotactile stimulus to the index finger, found that few of his subjects had distinct ipsilateral responses. From his monopolar recordings he felt that possibly the vertex reference electrode used by Jones and Millar contributed to a spurious ipsilateral responses because it was not an inactive reference. This had been pointed out earlier by Desmedt and Brunko (1980) commenting on Salamy's (1978) technique of measuring interhemispheric conduction time. Shagass felt that this was consistent with evidence that distal parts of the limbs do not have callosal connections.

We have recently completed a study (to be published) on SEP's in eight control and six epileptic subjects with chronic psychosis. We used the more traditional technique of left and right median nerve stimulation with the vertex as common reference electrode. We found like Shagass that the ampitudes of the ipsilateral responses were smaller than the contralateral responses to such an extent that is was often difficult to make reliable ipsilateral latency measurements. Where such measurements could be made we found no significant differences between the latencies for ipsilateral or contralateral evoked responses in either the normal or patient groups. Results for the P1 and P2 peaks are shown in the Table. (Results quoted are for left and right median nerve stimulation averaged).

TABLE

	P1	P2	
Controls n=8	21 ± 4 m.sec.	46 ± 6 m.sec.	Contra
	23 ± 4 m.sec.	47 ± 5 m.sec.	Ipsi
	n.s.	n.s.	
Epileptics	27 ± 5 m.sec.	52 ± 8 m.sec.	Contra
with n=6	31 ± 6 m.sec.	56 ± 8 m.sec.	Ipsi
Psychosis	n.s.	n.s.	

Latencies were significantly greater for the patient group but this could be accounted for by the age difference between the groups (controls 29 ± 5 years, Epileptics 41±13 years).

Gulmann *et al* (1982) recently reported a study of ipsilateral and contralateral SEP's in ten controls and ten chronic schizophrenic patients using median nerve stimulation. They found, using bipolar recording, an ipsilateral response only corresponding to peak 5 of the contralateral SEP, i.e. at latency greater than 65 m.sec. This is somewhat longer than the peaks we looked at.

Our study left us like Shagass *et al* feeling that the use of SEP in comparing ipsilateral and contralateral responses as a technique for measuring callosal function needs further careful study before it is likely to be able to help us answer the question about possible corpus callosal dysfunction in schizophrenia.

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MUNCHAUSEN SYNDROME

DEAR SIR,

Munchausen Syndrome presenting a psycho-social symptomatology is now a well-described condition (Cheng and Hummel, 1978; Simpson, 1978; Snowdon *et al*, 1978). However it is infrequently referred to in text-books, and trainees often remain ignorant of its existence.

The most typical case would appear to be a single male, usually referred from the medical or surgical wards, often following an overdose or other suicide attempt. The mood is usually depressed and this is often associated with a feigned and dramatic bereavement such as multiple deaths of relatives in a car crash, often in a different country. The history, for one reason or another, cannot be corroborated.

I report such a patient.

V.M., a male aged 82, was referred to the psychiatric department following a paracetamol overdose, but his blood levels were found to be far below the expected amounts. He gave a history of periods of severe depression and anxiety following the death of his wife (an opera singer), daughter and stepson in a car-accident in Germany, eight years previously. He described various classical symptoms such as tearfulness, early morning wakening, diurnal variation of mood, ideas of guilt, loss of interest in his usual hobbies, and auditory hallucinations of voices, accusing him and ordering him to kill himself. He admitted to one previous psychiatric admission in Germay, where he had been treated with monoamine oxidase inhibition, but he could not remember the precise address of the hospital.

On examination he appeared very depressed and agitated, and was noted to exhibit restlessness, hand-wringing and a severe tremor. No informants could be interviewed, as the patient claimed to have no friends or relatives in this country and did not want us to contact his place of work.

A diagnosis of agitated depression was made and the patient was admitted to his catchment area hospital, where he was immediately recognised by the admitting doctor. It appeared that he had a history of numerous psychiatric admissions dating back at least six years, under various names. His last admission had taken place three months earlier, and was terminated when he was arrested in a bank for attempting to draw money under yet another false name.

On this admission he improved within a few days and was discharged to the care of his protion officer a fortnight later.

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RAPID TREMOR OF THE EYELIDS AFTER OVERDOSE OF FLUPHENAZINE

DEAR SIR,

A 20 year old man with 5 admissions, in the previous 17 months, and diagnosed variously as suffering from schizo-affective or manic-depressive (manic) psychosis, was admitted after an overdose of 80 × 5 mg fluphenazine tablets and 6 × 0.5 mg benztropine tablets, 2 days before. On examination he had a rapid twitching of his upper eyelids in both the closed and open positions, synchronous with tremor of his hands and tongue. There was a superimposed slow spontaneous blinking of 8 blinks per minute (normal ± 12 blinks per minute—Carney and Hill, 1982). Ocular movements were normal. The glabellar reflex showed non-habituation, and there were other Parkinsonian features. There were no signs of tardive dyskinesia; nor of psychosis.

He was given benztropine 2 mg intravenously. Within 20 minutes the eyelid tremor had markedly decreased. He reported feeling less stiff, and there was decreased jaw stiffness. The glabellar response remained non-habituated. He was subsequently given benztropine 2 mg bd orally. One week later, the tremor of the eyelids had ceased, but the fine tremor of the hands persisted, and blinking was still slow at 10 blinks per minute. The glabellar reflex habituated after 3 taps.

Blink rate is decreased in Parkinson's disease (Hall, 1945), the latter sometimes being indistinguishable from drug-induced Parkinsonism (Baldessarini, 1980), but eye blinking is increased in schizophrenia and tardive dyskinesia (Stevens, 1978), and in Gilles de la Tourette syndrome (Cohen *et al,* 1980).

Penders and Delwaide (1971) showed a return towards normal eye movements in Parkinsonian patients treated with L-dopa or amantidine. Conversely, dopamine blockade by neuroleptics reduces the blink rates and the thought disorder in schizophrenics (Karson *et al,* 1981a). Reduction of dopamine blockade as in Stevens' patients (Stevens, 1978) who were medication-free for 1–6 months, leads to an increase in blinkrate. Also, Karson *et al* (1981b) found an increased blink rate to apomorphine after haloperidol discontinuation. The central role of dopaminergic blockade in abnormal eye movements was further illustrated by the finding (Karson *et al,* 1983) of an inverse relationship between spontaneous blink rate and platelet monoamine oxidase activity.

Here, there was a decrease in spontaneous blinking, in keeping with the picture found in drug-induced Parkinsonism i.e. a dopamine blockade. The rapid tremor of the eyelids, synchronous with the tremor of the hands and ameloriated by benztropine, is thought to be an unusual extrapyramidal neuroleptic side-effect analogous to the perioral "rabbit" tremor described by Jus *et al* (1974), (which is also relieved by anti-Parkinsonism medication (Sovner and Di Mascio, 1978)). I think that the mechanism involved in the abnormal eyelid movements was a massive dopamine