

(*Journal*, May 1969, p. 541) are consistent with the general body of evidence which indicates that the thalamus of the dominant hemisphere, and its ventrolateral nucleus in particular, take part in the speech functions of the dominant hemisphere (Bell, 1968). In Table I they indicated that eight patients had post-operative dysphasia, and they also reported that the patients submitted to thalamotomy 'showed significantly more deterioration than the controls on speech', but unfortunately they did not indicate whether the lesions in these cases were in the dominant or the non-dominant hemisphere. However, an immediate post-operative change in cognitive function indicating impairment in the auditory-verbal modality resulted from lesions in the dominant hemisphere.

Although the findings are not exceptional, their conclusions are. The authors argue that because the auditory-verbal impairment was transitory it could not have been due to the lesion in the nucleus ventralis lateralis. Surely a transitory disorder is the typical result of any single lesion that affects speech? The transitory nature of the dysphasia after a cortical excision in the dominant parietal lobe has certainly not deterred others from claiming that this area is involved in speech functions.

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SLEEP PATTERNS IN PREGNANCY

DEAR SIR,

Karacan *et al.* (*Journal*, August, 1969, p. 929-35) report that in 'late' pregnancy subjects experience a prolonged sleep latency, a greater number of awakenings, a shorter total sleep time and a 'suppression' of stage 4 sleep. These authors offer several elaborate explanations of their findings, including 'a sub-clinical depression' and 'hormonal changes' late in pregnancy; but they rightly state that 'the mechanism of these sleep disturbances is unknown'.

I too would like to speculate as to cause, but in a more mundane fashion.

Women in their last trimester support an abdominal protuberance of some size, and once in bed are faced with the task of moving their extra mass from side to side. This manoeuvre undoubtedly required some skill and dexterity. I would suspect that any woman embarking on such manoeuvres throughout

the night would have a prolonged sleep latency, a greater number of nightly awakenings, less total sleep time and a 'suppression of stage 4 sleep'.

Although without hard data to support my conclusions I feel that anyone who has spent one or more nights sharing the bed of a 'woman with child' will surely find heuristic value in my speculations.

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DERMATOGLYPHICS AND SCHIZOPHRENIA

DEAR SIR,

One of the major inquiries of a neurogenetic unit of a psychiatric hospital would concern cytogenetic studies in schizophrenia. Recent publications by Kaplan, Judd and others, however, have reported data suggesting that there are inconsistent differences in karyotype pattern found in cases of schizophrenia. Thus, our original intention to screen cytogenetically the newborn offspring of schizophrenic women in this hospital had to be abandoned.

The observations by Raphael and recently by Sanks on dermatoglyphic aberrations (digital ridge dysplasia) in schizophrenic children suggested other possibilities of screening the newborn children.

This preliminary investigation on 17 pregnant women (diagnosed as different types of schizophrenia) and 14 newborn children who were examined, produced quite interesting findings. Nine mothers showed the typical digital ridge dysplasia, while 6 out of 14 investigated children showed this abnormality. Questions regarding pedigree, racial differences and sex differences of offspring, etc., should be discussed in more extensive investigations.

The findings suggest, however, that one should undertake dermatoglyphic tests on a larger scale, either in the described form with pregnant women or in special schools for emotionally disturbed children and their parents. The eventual value of these investigations lies in the possibility of an earlier diagnosis of a schizophrenic tendency in children, particularly of parents with this disease.

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