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ERGOTAMINE TARTRATE IN THE TREATMENT OF NARCOLEPSY

Dear Sir,

Amphetamine and its related compounds have long been used in the drug therapy of narcolepsy. However, although they have proved effective in treating sleep attacks they have not been so effective in treating REM sleep-related manifestations such as cataplexy, hypnagogic hallucination and sleep paralysis. On the other hand, it was shown by Akimoto *et al* in 1960 that imipramine is markedly effective in treating these conditions, and at the same time it was disclosed that these manifestations are derived from REM sleep abnormalities (Hishikawa *et al*, 1966; Suzuki, 1966; Guilleminault *et al*, 1976).

We have recently encountered two cases of narcolepsy in which manifestations such as sleep attacks, cataplexy, hypnagogic hallucination and sleep paralysis were markedly improved with ergotamine tartrate only (Cafergot tablets, each containing 1 mg of ergotamine tartrate and 100 mg of anhydrous caffeine; Sandoz, Basel and Sankyo Co., Tokyo) and Bellergal tablets (each containing 0·1 mg of bellafoline, 0·3 mg of ergotamine tartrate, and 20·0 mg of phenobarbitone, Sandoz, Basel and Sankyo Co., Tokyo).

Case 1. A woman aged 48 had typical narcolepsy which had evolved at the age of about 23. This patient had been treated orally with 6 tablets daily of methylphenidate hydrochloride (each tablet containing 10 mg of the agent) for the preceding several years; however, because of gradual acquirement of tolerance, two or three sleep attacks had been occurring every week. In our out-patient clinic she was treated with 3 mg daily of ergotamine tartrate and 5 tablets daily of Cafergot, and each treatment resulted in a marked improvement in the manifestations within several days.

Case 2. A 23-year-old student had typical narcolepsy which had evolved at the age of about 15. This patient had been medicated with 6 tablets daily of methylphenidate hydrochloride for the preceding several years. However, because the effect of the medication had been gradually reduced, the patient was additionally medicated with 3 tablets daily of Cafergot in the out-patient clinic. This additional medication caused the manifestations to be markedly improved. In this case the medication with Bellergal and also that with ergotamine only were tried, and proved effective.

In both these cases there occurred no particular changes in the background patterns on the EEG after the medication, compared with the patterns before the medication: thus the medication probably acted to inhibit REM sleep. In two cases of periodic somnolence medication with Cafergot improved the manifestations. Ergotamine tartrate, which has been used as a drug with an angiotonic action, may be considered the treatment of choice in narcolepsy with acquired tolerance to the routinely used drugs. A study is in progress in our Department of the CNS actions of ergotamine tartrate in narcolepsy.

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RESPIRATORY VENTILATION

Dear Sir,

We note with interest the letter of Mitchell-Heggs *et al* (*Journal*, July 1977, pp 108–9). We, Damas Mora *et al* (1976), were anxious to draw attention to the fact that mood affects respiratory ventilation and hence arterial PCO_2. Our intention was to make research workers cautious, as this could complicate chemical comparisons of psychiatric patients and controls (Damas Mora *et al*, 1977).

We concede that technical inadequacies limit precise quantification, but feel that dog bites man requires less evidence than the converse. We certainly do not understand why our critics feel we are opposed

to the careful examination of the relation between the limbic system's function and the control of respiration. Indeed, like others, we are currently looking at the changes in the sensitivity setting of the respiratory centre in various mental states.

Our first paper, however, is somewhat defective in not clearly stating that the catheters were 0·5 cm in the nostril in nose-breathing subjects. The paper also fails to indicate how one figure for the end-tidal PCO_2 over 5 minutes was obtained. It was the mean for the expiratory CO_2 peaks over a period in which variation was minimal.

'Of course, there is no substitute for measurement of arterial blood itself', but practical reasons have led those interested in the study of the chronic psychogenic hyperventilation syndrome (see Lewis, 1957) to use methods in fact based upon the principles outlined by Tyndall (1865). This appears to be true of much research in general medicine.

We confess that capillary blood and end-tidal PCO_2 showed limited correlations, but we assume that this arose because respiration altered during blood sampling, and because the arterialization was not always complete. It must be emphasized that the correlation was with the mean end-tidal PCO_2 during the blood sampling when that too was more difficult to evaluate.

The catheter is likely to have had effects 'on the respiratory variables and mental state of a subject who was already in a "nervous state" ' but would this have been minimized by taking arterial blood?

Intra-subject variability also presents a problem, but one covered by the statistics.

Certainly benzodiazepines act for long periods, but the respiratory effect is reported by some to be limited and brief. Furthermore, the literature is contradictory (see Steen *et al*, 1966; Dalén *et al*, 1969). Intravenous chlordiazepoxide in the cat (Florez, 1969) and intramuscular lorazepam in man (Gasser *et al*, 1975) have no significant effect on respiration. The demonstration in a short communication to the Medical Research Society by Guz *et al* (1977) was not available to us. They showed effects of diazepam (not nitrazepam) on PCO_2 and ventilation. It does make us hesitate about possible drug effects on our patient sample, as some had nitrazepam the night before, but this may be similar to effects in other psychiatric studies and is not relevant to our second study of the effects of overbreathing. The matter nevertheless deserves closer scrutiny and more experiments.

Respiratory disease, too, could have been more common in our patients than in our controls. On clinical examination it did not seem to be so.

It may be unkind to state it, but nevertheless it is so, that the referees twisted our arms to write about drug effects on the blood brain barrier due to PCO_2. That speculation was not in our original text. It is impossible to please everyone, though, but we do thank Mitchell-Heggs *et al*, for their helpful remarks on our study.

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CAPGRAS' SYNDROME

Dear Sir,

I was struck by Dr Christodoulou's comment (*Journal*, June 1977, **130**, pp 556–64) that one of his patients demonstrated 'reduplicative paramnesia', a phenomenon I also recently noted in a patient whom I diagnosed as having Capgras' syndrome in 1975, and who presented a year later with reduplicative paramnesia (he was in hospital in Chicago but insisted he was actually in New York and that there were two New York Cities). Hayman and Abrams (1) suggested prosopagnosia as a possible cerebral mechanism for Capgras' syndrome, but these recent observations require consideration of reduplicative paramnesia as an alternative explanation.

In their review and case reports of reduplication, Weinstein and Kahn (2) include the delusion of doubles as a form of reduplication for person, and note that most cases of reduplication of body parts reported in the literature have occurred in association with right cerebral brain damage. Viewing Capgras' syndrome as a reduplication for person would