GILLES DE LA TOURETTE'S SYNDROME TREATED BY OPERANT CONDITIONING

DEAR SIR,

Dr Fernando (*Journal*, May 1976, 128, 436–41) implied that the role of behaviour therapy in the treatment of Gilles de la Tourette syndrome is not clearly established. We should like to describe the case of a boy whose vocal tics were controlled by an operant conditioning programme used by his parents in the home.

Sean, aged 11, was referred because of socially disruptive vocal tics which at school distracted the other children and led to comments by neighbours.

Sean was born by elective caesarian section at 36 weeks because of placenta praevia. His parents were then in their late 30s. At the time of referral his sisters, who were 12 and 14 years older, had left the family, and his 21-year-old brother, who was a successful pop guitarist spent little time at home, but was the apple of his mother's eye. Sean was unplanned and initially unwanted, as his mother had to give up her job and readjust to coping with the demands of a baby.

Although he did not mix with other children, and had difficulty in settling in his infant school, there were no overt problems until Sean was 9, when he was referred to a dermatologist because he was breaking off his hair. He had no close friends and tended to run to teachers for support, as he was being picked on by other children at his school. He was nervous in class, and stammered, and his concentration was poor. Both the school and his parents noticed that he developed motor tics at this time. He was transferred to a different primary school, and his tics stopped, but started again 3–4 months later. They gradually became more frequent and especially his loud vocal tics. At his secondary school, Sean was attention-seeking and disruptive in class. He was under-achieving but of average intelligence. In the diagnostic interview Sean was sullen and inarticulate, but no vocal or motor tics occurred. He said he resented not having the same freedom as his older brother. Both parents were very distressed by Sean's behaviour and felt guilty because of his anomalous position in the family as the youngest child of elderly parents.

Because of the parents' anxiety about the use of haloperidol, we decided to try an operant conditioning programme under the supervision of a psychologist. The aim was to make Sean more conscious of his tics when they occurred and to help him control them by rewarding him if he was able to suppress them. Before designing the programme, the psychologist observed Sean at home to determine the exact nature and frequency of his vocal tics. They proved to be a loud 'barking' sound, emitted at approximately 30-second intervals. Sean said he was conscious of their occurrence but could not voluntarily inhibit them.

In order to obtain a baseline, Sean's parents were asked to record the number of tics during frequent observation periods. This record of Sean's tics showed that their frequency diminished as Sean became tired. At the weekend his tics were 'constant' but they did not occur when Sean was asleep.

A reward system of stars and money for a tic-free period was agreed between Sean and his parents. The required tic-free period was gradually lengthened as Sean became more successful at controlling his tics. The parents were given detailed written instructions of the programme, and daily telephone contact was maintained to give advice and support and to enable alterations in the programme to be made where necessary.

After six weeks, Sean's parents discontinued the programme. Almost six months after this, Sean's vocal tics have not returned and there has been no symptom substitution, while the motor tics have not changed in frequency or severity.

Although the programme was not used in school, control of Sean's tics improved in the classroom setting. This is in contrast to the Rosen and Wessner (1973) study, in which teachers carried out a programme to control the tics of a boy within the classroom setting, but there was no resulting change in behaviour in the home setting. Sean's parents and teachers say he is much happier, and has become very popular with his peers since overcoming his problem.

His parents felt that the programme had been easy to apply and had quickly resolved a very distressing situation.

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REFERENCES

FERNANDO, S. J. M. (1976) Six cases of Gilles de la Tourette's syndrome. *British Journal of Psychiatry*, 128, 436–41.

ROSEN, M. & WESSNER, C. (1973) A behavioural approach to Tourette's syndrome (1973). *Journal of Consulting and Clinical Psychology*, 41, 2, 308–12.

SPEECH IN DEPRESSIVE STATES

DEAR SIR,

Szabadi, Bradshaw and Besson state 'Although motor retardation involves the retardation of speech, there have been no attempts to measure the speed of speech in a quantitative fashion in depressed patients'.

This is not so. Hutt and Coxon (1965) did just that. They reported on spontaneous talking speed and on reading speed in a manic-depressive patient. Spontaneous speech proved to be a most sensitive predictor of mood change. Reading speed by contrast did not alter with mood. They used a simple tape recording. The more complex conversion of the results employed by Szabadi, Bradshaw and Besson is not required.

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REFERENCES

1. HUTT, C. & COXON, M. W. (1965) Systematic observation in clinical psychology. *Archives of General Psychiatry*, **12**, 374–8.
2. SZABADI, E., BRADSHAW, C. M. & BESSON, J. A. O. (1976) Elongation of pause-time in speech: a simple, objective measure of motor retardation in depression. *British Journal of Psychiatry*, **129**, 592–7.

LITHIUM TREATMENT OF CYCLICAL VOMITING IN A MENTALLY DEFECTIVE PATIENT

DEAR SIR,

Miss J is aged 29 and mentally defective with an intelligence quotient of about 60. Persistently recurrent episodes of vomiting began when she was nearly 19 and were initially attributed to anxiety about a dental extraction. These became progressively more serious, did not respond to treatment and repeatedly brought about prostration and severe electrolyte disturbances. The nursing staff noted that vomiting occurred almost exclusively when J was depressed. At such times her skin appeared pasty and her squint became more marked; she looked miserable, was sometimes doubly incontinent, and vomited persistently. Vomiting was accompanied by severe polydipsia, far exceeding that required for fluid replacement. Staff also described brief episodes of elation lasting between a few days and two weeks in which she was talkative, friendly and familiar.

Investigation, which included chest and skull X-ray, air encephalogram, barium meal, intravenous pyelogram, cholecystogram and tests of renal, adrenal and hepatic function, failed to reveal any cause for vomiting and we decided to treat her as a case of manic depressive psychosis with lithium and to monitor its effect on her mood state and vomiting.

For two years nursing staff recorded her mood state twice daily at the end of each daytime nursing shift as either elated, normal or depressed; and episodes of vomiting were also noted. Symptomatic treatment with prochlorperazine and chlorpromazine was continued throughout but during the second year lithium was also prescribed and a mean blood level of 0·65 mEq/l maintained.

In the first year there were 91 recordings of elated mood, 191 of depressed mood, 421 of normal mood and 63 recordings of vomiting. During the second year there were 57 recordings of elated mood, 59 of depressed mood, 592 of normal mood and 31 recordings of vomiting. The therapeutic effect of lithium is not usually apparent immediately on starting treatment and if the results for each year are compared, when the ratings for the first three months are omitted, the results are even more striking. In the first nine-month period there were 79 recordings of elated mood, 175 of depressed mood, 282 of normal mood and 60 recordings of vomiting, whereas in the second nine-month period there were 25 recordings of elated mood, 23 of depressed mood, 492 of normal mood and 24 recordings of vomiting ($\chi^2 = 385{\cdot}7$, $P < {\cdot}001$). In the first year there was a clear relationship between vomiting and depression. In the second year this relationship disappeared.

There is a similarity between this patient's symptoms and cyclical vomiting in children. Mitchell (1) described cyclical vomiting in children as recurrent rather than regularly cyclical, accompanied by symptoms of anxiety and leading rapidly to a state of severe ketosis and dehydration. The patient in this study showed these somatic features but also affective swings. Taken along with the accompanying polydipsia the syndrome is suggestive of a hypothalamic timing and synchronizing mechanism (Jenner (2)). Although the presentation was atypical for manic depressive psychosis, treatment with lithium resulted in partial stabilization of mood, reduction in frequency of vomiting and an impressive though unmeasured reduction in severity and quantity of vomitus. It may be worth treating resistant cases of cyclical vomiting in children with lithium.

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REFERENCES

1. MITCHELL, R. G. (1973) *Disease in Infancy and Childhood* (by Ellis and Mitchell), 7th Edition. Edinburgh and London: Churchill Livingstone.
2. JENNER, F. A. (1968) *International Review of Neurobiology, ii*, 129.