

## RABBIT SYNDROME : PATCHY DISAPPEARANCE IN STAGE I NREM SLEEP

B. N. GANGADHAR<sup>1</sup>, M.B.B.S.

C. J. JOHN<sup>2</sup>, M.B.B.S., D.P.M.

SOMANATH CHATTERGI<sup>3</sup>, M.B.B.S.

M. S. KESHAVAN<sup>4</sup>, M.D. (Psych.)

H. S. NARAYANAN<sup>5</sup>, D.P.M., M.D.

### SUMMARY

A case which fulfilled the clinical description of Rabbit Syndrome investigated with sleep E.E.G is presented. Unlike the earlier reports, a patchy disappearance of the movement disorder during Stage I NREM sleep was noticed. The clinical differentiating features from tardive dyskinesia are discussed.

The "Rabbit Syndrome" described by Villeneuve (1972) as a late-onset neuroleptic-induced extrapyramidal syndrome, is characterised by rhythmic 5-5.5 per second involuntary movements of the oral and masticatory musculature that mimic the chewing movements of a rabbit. The rabbit syndrome is readily reversible with the discontinuation of neuroleptics (Villeneuve, 1972) and responds to antiparkinsonian drugs (Sovner and Dimascio, 1977; Jus *et al.*, 1979). There are reports that the Rabbit syndrome persists in stage I NREM sleep unlike tardive dyskinesia, another late-onset neuroleptic-induced movement disorder (Jus *et al.*, 1972; Villeunve *et al.*, 1973).

### Case Report :

Mr. S. R. a 38 year old unmarried male, consulted us on September 1980 with a recent onset of movement disorder confining to jaw and lips. He was attending out-patient department of National Institute of Mental Health and Neuro Sciences, Bangalore from 1967 onwards after an acute psychotic episode and had been on anti-psychotic medication as suggested to him

from time to time. He had no past history of any significant physical illness.

In 1967 he was treated with chlorpromazine (300-500 mgm/day) and improved over a period of three months. He resumed his occupation and was drug-free until 1973, when he developed a relapse of the psychotic illness with predominant schizophrenic features. He was treated with Chlorpromazine (300 mgm/day) and trifluoperazine (15 mgm/day) and a course of 5 ECTS. Trihexyphenidyl (6 mgm/day) was added as he developed parkinsonian symptoms. Patient made a good social recovery and was maintained on the same drugs for a period of one year.

He was alright till 1976 when he again developed florid schizophrenic features and had to be hospitalized. He was treated with trifluoperazine (30 mgm/day) and Chlorpromazine (100 mgm/day), and with a course of 6 ECTS. During his stay in hospital trihexyphenidyl (6 mgm/day) was added, as he developed parkinsonian symptoms. After discharge oral medications were gradually withdrawn and he was stabilized on fortnightly parenteral fluphenazine deconoate (25 mgm) and oral

<sup>1</sup>, <sup>2</sup>, & <sup>3</sup> Residents in Psychiatry

<sup>4</sup>Lecturer in Psychiatry

<sup>5</sup>Associate Professor in Psychiatry

National Institute of Mental Health Neuro & Sciences, Bangalore 29.

trihexyphenidyl (4 mgm/day). He was maintaining good improvement and was on regular medications for four years. During this period, he had an exacerbation of psychotic symptoms and was prescribed haloperidol (15 mgm/day) in addition to the parenteral fluphenazine deconate he had received a week earlier. It was six days after starting haloperidol, that he developed this movement disorder, for which consultation was sought.

At the time of evaluation, his mental status did not reveal any overt psychotic features. On physical examination he had an involuntary movement disorder which confined bilaterally to the oral and masticatory musculature. The tongue was not involved. The movement was rhythmic and more at rest with a tendency to diminish while engaged in conversation. There were no other neurological abnormalities. The movement disorder was thought to be a recent onset tardive dyskinesia and he was admitted for detailed evaluation and treat-

ment. Patient continued to receive haloperidol in the ward. On the second day of admission the patient had developed rigidity, tremors and bradykinesia in addition to these masticatory movements. He was given promethazine (50 mg) parenteral and within 45 minutes the parkinsonian symptoms and these masticatory movements disappeared completely. However the masticatory movement as such reappeared on the next day. At this stage a revised diagnosis of Rabbit syndrome was made as it fulfilled the clinical criteria. On the third day all drugs were withdrawn to facilitate a drug-free sleep EEG record.

The sleep EEG was recorded with a muscle electrode over the jaw. The jaw movements were rhythmic with a frequency of six per second (fig. 1). It was also seen that the movement disorder was not constantly present in Stage I NREM sleep. There were occasions when the movement was totally absent during this stage of sleep and this disappearance was patchy with

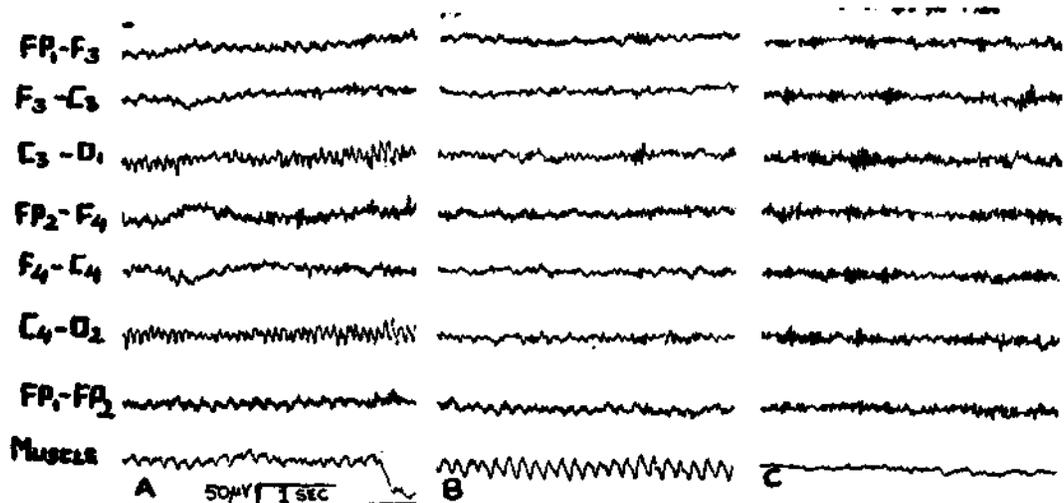


Fig. 1.—E. E. G. tracings illustrating the relation between the involuntary movement and sleep. The last channel records artefacts from an electrode placed on the masseter. Recording done with "10-20 electrode placement" TC 0.03 High Frequency filter 75.

(a) awake, with 9-10 Hz alpha and 6 Hz myogenic artefacts.

(b) Stage 1 NREM sleep, with absence of alpha and low index beta. Note the rhythmic 6 Hz involuntary movement artefact.

(c) Stage 1 NREM sleep with abundant beta, with disappearance of the involuntary movement.

no predilection for early, middle or late parts of this stage of sleep. From the fifth day of admission trihexyphenedyl (8 mgm per day) was started and the movement disorder disappeared totally. He was discharged by the tenth day. He continued to remain symptom free on further weekly follow-ups without antipsychotics. Trihexyphenedyl was withdrawn over a period of four weeks, and the movements did not reappear.

#### COMMENTS

The clinical description of this case is in keeping with the earlier description of Rabbit syndrome, including its response to antiparkinsonian drugs (Villeneuve, 1972; Sovner and Dimascio, 1977; Jus *et al.*, 1979). As the movement did not persist throughout the stage I NREM sleep a differentiation from tardive dyskinesia needs to be made at a clinical level. Unlike Rabbit syndrome the initial movements of orofacial tardive dyskinesia occur in tongue and later spread to other parts (Ayd, 1967). Also tardive dyskinesia tends to worsen on withdrawal of neuroleptics and might even be suppressed by antipsychotics (Baldessarini and Tarsy, 1975). Even though its early symptoms are completely reversible (Quitkin *et al.*, 1977), tardive dyskinesia worsening with anticholinergic antiparkinsonian drugs is well emphasised (Baldessarini and Tarsy, 1975). The latter feature certainly differentiates Rabbit syndrome from tardive dyskinesia, as the former specifically improves with anti-parkinsonian drugs.

We maintain the view that the pathophysiology of Rabbit syndrome is similar to that of the drug induced parkinsonism

(Jus *et al.*, 1972). The coexistence of the drug-induced parkinsonism and improvement in both with antiparkinsonian drugs, as in this case substantiates this view. It is interesting to note that unlike drug-induced parkinsonism Rabbit syndrome occurs as a late-onset neuroleptic induced movement disorder. Hence a separate terminology may be still valid.

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#### REFERENCES

- AYD., F. J., JR. (1967). Persistent dyskinesia. *Med. Sci.*, 18, 32.
- BALDESSARINI, J. R. TARSY, D. (1975) Mechanisms underlying tardive dyskinesia. *The Basal Ganglia, Research Publications: Association for research in nervous and Mental disease.* (Ed.) Yahr. D. Melvin. New York: Raven Press, Vol. 55, 433.
- JUS. A., JUS, K. AND FONTAINE, P. (1979). Long term treatment of tardive dyskinesia. *J. Clin. Psychiat.* 40, 72.
- JUS. K., VILLENEUVE, A. AND JUS, A. (1972). Tardive dyskinesia and Rabbit Syndrome during Wakefulness and sleep. *Am. J. Psychiat.* 129, 765.
- QUITKIN, F., RIFKIN, A., GOCHFELD, L., KLIENT, D. F. (1977). Tardive Dyskinesia: are first signs reversible. *Am. J. Psychiat.* 134, 84.
- SOVNER, R., DIMASCIO A. (1977). The effect of Bzotroprine Mesylate in the Rabbit syndrome and tardive dyskinesia. *Am. J. Psychiat.*, 134; 1301.
- VILLENEUVE, A. (1972). The Rabbit Syndrome: a peculiar extrapyramidal reaction. *Canad. Psychiat. Assoc. J.*, 17 (Suppl), 69.
- VILLENEUVE, A., JUS, K. AND JUS, A. (1973). Polygraphic studies of tardive dyskinesia and of the Rabbit syndrome during different stages of sleep. *Biol. Psychiat.*, 6, 259.