# E. C. T. AND DRUG INDUCED PARKINSONISM

B. N. GANGADHAR<sup>3</sup>, M.D.
J. ROY CHOUDHARY<sup>4</sup>, M.B.B.S.
S. M. CHANNABASAVANNA<sup>3</sup>, M.D.

### SUMMARY

A cross-sectional evaluation of the presence of drug induced parkinsonian symptoms in hospitalized patients was done. Patients who had received two or more ECTs had lower scores of parkinsonism when compared to those who were not receiving ECT. Since the patient groups were comparable on parameters which would influence the occurrence and development of drug induced parkinsonism, the lowered scores in one group could be attributed to the effect of ECT.

While ECT has been proved to be effective in depression (Gangadhar et al, 1982) as well as schizophrenia (Janakiramaiah et al., 1982) its benefits in other disorders are less proven. Yudofsky (1979) observed that ECT relieved both depressive and parkinsonian symptoms when given to a patient suffering from both. This observation that parkinsonian symptoms are relieved by ECT were confirmed on five patients by Balldin et al. (1980) who advocated its use in therapyresistent parkinsonism. In this context a recent observation (Small et al., 1982) that patients treated with ECT plus thiothixene had fewer drug induced abnormal movements than those treated with thiothixens alone is interesting. Is it possible that ECT treated patients while on neuroleptics develop lesser frequency or severity of drug induced EPS?

#### METHODS

At a cross section all first-admission in patients who were receiving ECT and a neuroleptic (N=17) were assessed on a EPS rating scale (Simpson and Angus, 1970). All these patients had already received at least two ECTs and were currently still on ECT treatments on alternate days. A control group of first admission patients (N=18), not receiving ECT

were chosen by a stratified random sampling, matching the dosage of neuroleptics they were receiving. The EPS scores were similary measured. Patient sample consisted of only functional psychotics other than depressives. Data on other factors affecting the onset and severity of drug induced parkinsonian symptoms, were also collected i.e. age, sex, duration of neuroleptic therapy, and current dosage of antiparkinsonian drug.

## RESULTS

The patients who were receiving ECT and the patients who were not receiving ECT were comparable onparameters which would normally affect the development and degree of extra-pyramidal reactions (Table 1).

With regard to neuroleptic drugs, the proportion of patients receiving different classes of neuroleptics were similar in both the groups. No patient was receiving haloperidal. Six patients in the ECT group and seven patients in the no ECT group were receiving thrifluperazine. The remaining patients were on chlorpromazine. Three patients in the ECT group and two patients in the no ECT group were receiving weekly fluperazine deconoate (25 mgm) injections in addition to chlorpromazine. The neuroleptic

<sup>1.</sup> Lecture

Junior Resident

<sup>3.</sup> Prof. and Head, Dept. of Psychiatry

777	T
LABLE	1

	ECT	NO ECT	
	(n=17)	(n == 18)	
Age (in years)			
	28.5±8.7	31.9±9.1	NS
Sex			
Male	7	8	
Female	10	10	NS
Dosage of Neurolepti Chlorpromazine mgn day.		528±181	NS
Duration of Neuro- leptic Therapy (days)	) 25. <b>4<u>十</u>20</b> .2	27.5 <u>+</u> 22.	0 NS
Dosage of Benzhexol mgm/day	1.9 <u>+</u> 2.1	3.1±2.2	NS
EPS score.	1.0±1.1	5.5 <u>±</u> 3.8	t=5.26 <0.001

dosage was converted into chlorpromazine equivalents and there was no statistically significant difference between the groups. The patients receiving ECT, on the average had received 5.2 ECTs each. The ESP scores in this group were significantly lower (p<0.001).

#### DISCUSSION

It has been established that parkinsonian symptoms develop due to a functional deficiency of dopamine activity (DA) and enhanced central cholinergic activity. Use of anticholinergic drugs is known to alleviate such symptoms. ECT has been shown to increase DA receptor sensitivity (Balldin et al., 1982) and hence might explain beneficial effect in parkinsonian

symptoms. A theoretical paradox does exist as to why it is also beneficial in schizophrenia (Janakiramaiah et al., 1982); Small et al., 1982), a disease believed to be due to increased central dopaminergic hyperactivity. Nevertheless, the observation that patients treated with ECT had lower mean EPS scores is encouraging in favour of use of ECT in conjunction with neuroleptics.

### REFERENCES

BALLDIN, J., EDEN, S., GRANERNS, A. K., MODIGH, K., SUANBORG, A., WALINDER, J., AND WALLIN, L., (1980). Electroconvulsive therapy in parkinson's with syndrome on-off phenomenon. J. Neural transmission, 47, 11.

Ballon, J., Granens, A. K., Gadsledt, G., Modich, K., and Wallnder, J., (1928). Neuroendocrine evidence for increased responsiveness of dopamine receptors in humans following electroconvulsive therapy. Psychopharmacology, 76, 371.

GANGADHAR, B. N., KAPUR R. L. AND KALYANA SUNDARAM, S., (1982). Comparison of ECT. and imipramine in endogenous depression—a double blind study. Brit. J. Psychiat., 142, 367.

JANAKIRAMAIAH, N., CHANNABASAVANNA, S. M., AND NARASIMHA MURTHY, N. S., (1982). ECT-Chlorpromazine combination versus Chlorpromazine alone in acute schizophrenic patients. Acta Psychiat. Scand., 66, 464.

Smeson, G. M., AND Angus, J. M. S. (1970).

Drug induced extrapyramidal disorder. Acta
Psychiat. Scand. (Suppl.), 212, 3.

SMALL J. G., MILSTEIN, V., KLAPPER, M., KELLANS, J. J., AND SMALL, I. F., (1982). EGT combined with neuroleptic in the treatment of Schizophrenia. Psychoparmacol. Bulletin, 18, 34.

YUDOFSKY, S. C., (1979). Parkinson's Disease, Depression and electroconvulsive therapy: A Clinical and neurobiologic synthesis. Comprehensive Psychiatry, 20, 579.