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BRIEF COMMUNICATIONS

PROLONGED APNOEA WITH SUCCINYLCHOLINE DURING ELECTRO-CONVULSIVE THERAPY

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Introduction

Electro-convulsive therapy (ECT) is one of the important treatment modality for psychiatric patients, particularly in developing countries like India. The introduction of curare and subsequently succinylcholine chloride (Holmerg and Thesleff 1952) as an important adjunct in the administration of ECT, was a major advance in reducing the morbidity associated with ECT. The relative safety of succinylcholine, a depolarising neuromuscular blocking agent, in ECT, has been extensively investigated (Holmerg and Thesleff 1952, Pitts et al 1968). Side effects of succinylcholine (include bronchial secretion, bradycardia, bronchospasm, muscle pain and hyperkalaemia (Wylie and Churchill Davidson 1966). One rare serious complication of succinylcholine administration is prolonged apnoea due most often to an increased duration of action. This increased duration of action may result from decreased metabolism of succinylcholine as seen in genetic pseudocholinesterase deficiency syndrome (Lehmaun and Liddell 1969), exposure to or treatment with anticholinesterase agents (Pantuck 1966) or as a result of additive effects caused by other drugs having neuromuscu lar blocking side effects (Pittinger et al 1970). Shortly after clinical introduction of succinylcholine, reports appeared describing prolonged apnoea immediately following the drug's administration during surgical procedures (Argent et al 1955) or after ECT (Hurley and Munro 1952).

Three case histories, where prolonged apnoea was observed during ECT following administration of succinylcholine chloride are tabulated below:

Discussion

The postseizure apnoea was of normal duration during second ECT (given after seven days) in all the three cases. Hence the cause for prolonged apnoea with succinylcholine chloride like genetic pseudocholinesterase deficiency, liver disease or malnutrition in these cases are ruled out.

All the three cases had consumed organophosphorous insecticides. Organophosphorous compounds are cholinesterase

Table
Summary of Findings

Age/Sex/ Diagnosis	ECT given after days of Organophosphorous consumption	Dose of Succinylcholine	Duration of Apnoea	Next ECT given after	Dose of Succinylcholine	Duration of Apnoea
1) 22/M/End.Dep.	3 days	30 mg.	25 min.	7 days	30 mg.	3 min
2) 19/F/Schiz.	3 days	30 mg.	30 min.	7 days	30 mg.	3.5 min.
3) 39/M/MDP.Dep	3 days	30 mg.	20 min.	7 days	30 mg.	3 min.

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inhibitors. These compounds combine with SH radicle of the enzyme and act by causing irreversible inhibition of cholinesterases (Scott 1966). This causes clinically important reduction in cholinesterase activity to such a degree that prolonged apnoea occurs after succinylcholine administration.

Organophosphate anticholinesterase are rarely used clinically but are often used in insecticides and as weapon for chemical war (nerve gases). However in treatment of glaucoma topically administered organophosphate anticholinesterase may be prescribed from where sufficient absorption may take place leading to significant reduction in cholinesterase activity. Chessent et al (1974) and Packman et al (1978) have described a case each, where there was prolonged apnoea following succinylcholine administration during ECT, the cause being topically administered organophosphate compound for glaucoma.

These reports stress the importance of using a non-depolarizing muscle relaxant or waiting until serum cholinesterase activity returns to normal in such patients. In all the three cases the apnoea after succinylcholine was of normal duration after seven days. Hence if one has to administer succinylcholine for any reason it should be administered after seven days by which time serum cholinesterase activity would return to normal.

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