EFFECTS OF ELECTROCONVULSIVE THERAPY ON CARDIOVASCULAR SYSTEM

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ABSTRACT

Effects of electroconvulsive therapy on the pulse rate, blood pressure and electrocardiogram were studied in forty consecutive physically healthy inpatients undergoing ECT for their psychiatric disabilities. Tachycardia and elevated blood pressure occurred frequently. Sinus arrhythmias, atrial and ventricular premature beats and minor ST-T changes were also observed. All these changes reversed within thirty minutes.

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KEY WORDS : Electroconvulsive therapy; Cardiovascular system.

Introduction

Ever since its discovery, electroconvulsive therapy (ECT) has occupied a central place in the therapeutic armamentarium of psychiatrists. Despite abhorrence from some quarters, it is still being practised as one of the cheapest and safest, and yet effective therapeutic techniques in psychiatry [1]. It is the most consistently effective treatment for severe depression and does not have the cardiotoxic effects of tricyclic antidepressants, hence it has been advocated as the antidepressant therapy of choice for cardiac patients [2]. Yet occasional fatalities have been shown to be due to myocardial infarction [3]. In the present study, we tried to determine the effects of ECT on the pulse rate, blood pressure (BP) and electrocardiogram (ECG) in psychiatric inpatients.

Material and Methods

Forty consecutive male inpatients undergoing ECT for their psychiatric disorders were included in the study conducted at MH Bareilly from Jan 88 to Jan 90. None of the patients were suffering from ischemic heart disease, hypertension, diabetes mellitus or any other physical disease. They were physically examined and the following investiga-

tions were carried out : hemogram, urinalysis, Liver function tests (LFT), Aspartate aminotransferase (AST), Alanine aminotransferase (ALT), blood sugar (fasting and postprandial), blood urea, skull radiography and fundoscopy. Pre-anaesthetic check up was carried out by the anaesthesiologist.

All the patients were given ECT in overnight fasting condition between 8 AM and 10 AM. ECT was modified with injection atropine 1.2 mg IV stat, injection thiopentone sodium 5 mg/kg body weight IV and injection succinylcholine 1 mg/kg body weight IV. Electrical stimuli were administered through bitemporal electrodes. All patients were oxygenated during the procedure with a face mask. Pulse rate, BP and ECG were recorded before ECT, immediately after the cessation of convulsions, and 30 minutes after ECT.

Results

The mean age of the patients was 28.3 years while the range was 17-54 years. Maximum number of patients (n=19) were in the age group of 21-30 years. The psychiatric diagnoses of the patients are reflected in Table 1. None of the patients had heart disease, hypertension, diabetes mellitus or chronic medical illness. All were on appropriate medications

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(antipsychotics 33; antidepressant 11; anxiolytics 7) which were continued during the course of ECT but in reduced dosages.

TABLE 1

Psychiatric diagnosis of patients undergoing ECT (n=40)

Diagnosis	Number	Percentage
Schizophrenia	27	67.5
Affective psychosis (Depression)	8	20.0
Other non-organic psychoses	5	12.5

Before ECT, three patients had tachycardia (pulse rate more than 100/min). Blood pressure and ECG of all the patients were within normal limits before ECT.

Immediately after ECT, 36 patients had tachycardia (range 104-148/min). None of the patients had bradycardia. There was increase of systolic BP ranging from 20-60 mm of Hg and diastolic BP ranging from 0-50 mm of Hg. Other changes are shown in Table 2.

ECT was not stopped in any patient due to occurrence of transient arrhythmias. Correlation between arrhythmias and type of drug treatment is given in Table 3. There was no relationship between drug therapy and arrhythmias ($X^2 = 0.413$; $df = 2$; $p > 0.80$). Thirty minutes after ECT, the pulse rate, BP and ECG of all the patients reverted to within normal limits.

Discussion

Despite the relative safety of ECT, occasional fatalities are recorded. The mortality rate associated with ECT has been variously recorded around 1 : 20,000 treatment [3,4]. Some of these fatalities were due to myocardial infarction [5]. Five out of 5325 patients treated with ECT had a fatal outcome in one study as a result of cardiovascular complications due to ECT in the form of acute heart failure, vascular collapse, pulmonary oedema and cardiac arrhythmias [6]. Coronary atherosclerosis and thrombosis, cardiac arrest and coronary occlusion have been blamed in a few other cases [7]. Sudden vagal

TABLE 2

Electrocardiographic changes observed in patients (n=40) immediately after ECT

ECG change	Number	Percentage
Sinus tachycardia	36	90.0
Atrial premature beats	7	17.5
Ventricular premature beats	2	5.0
Sinus arrhythmias	5	12.5
Minor ST-T changes	4	10.0

TABLE 3

Correlation between arrhythmias and type of drug treatment in patients undergoing ECT (n=40).

Type of arrhythmia	Drug Treatment			Total
	Anti-psychotics (n=29)	Anti-depressants (n=7)	Anti-psychotics (n=4)	
Atrial premature beats	5	1	1	7
Ventricular premature beats	2	—	—	2
Sinus arrhythmias	4	1	—	5
Total	11	2	1	14

$X^2 = 0.413$ $df = 2$ $p > 0.80$ Not significant

stimulation during ECT is considered as a potential factor in cardiac arrest [8,9]. One of our earlier cases had suffered from cardiogenic shock but recovered [10]. However, in the present series, there was no case of cardiac arrest or serious cardiac complication.

The finding of tachycardia following ECT is in agreement with the majority of earlier studies [9,11]. However, in the present study, none of the patients had bradycardia after ECT which was reported in an occasional patient in few studies [9,11].

In the present study, there was a rise of both systolic and diastolic BP after ECT which is in agreement with the study of Fink [12]. In contrast to this, two studies observed an increase in systolic BP only while one study did not find any significant rise or fall of BP [11].

Transient arrhythmias are common after

ECT. In our series only 35% of the patients developed arrhythmias compared to 56% reported by Shah et al [11]. This could be due to the younger age of our patients as also the absence of pre-existing heart disease.

The arrhythmias may be due to reflexes activated when large amount of blood trapped in the periphery during the seizure are sucked into the great veins and right atrium. The possibility also exists of direct stimulation of intracranial areas which influence cardiac rate through the vagus. An alternative explanation is the stimulation of hypothalamic centres through the release in the cerebro-spinal fluid of posterior pituitary hormones. Respiratory acidosis and hypercapnia, sympathetic discharge and hypoxia are other explanations [9]. A confounding variable could be the psychotropic drugs which our patients were receiving. At therapeutic levels, tricyclic antidepressants have an antiarrhythmic effect similar to that of Type 1 antiarrhythmic drugs (e.g. quinidine and procainamide).

Corresponding ECG changes include reversible prolongation of P-R, QRS, QTc, ST-T wave changes, and development of AV and bundle branch block [2,13]. Toxic levels of tricyclic antidepressants can result in any and all types of rhythm disturbances [2,14]. The antipsychotic drugs (especially phenothiazines) have been associated with a variety of ECG changes including prolongation of P-R and QT intervals, widened QRS complex, ST segment depression, T wave changes, variable degrees of heart block and serious ventricular arrhythmias. However, the most common ECG changes are benign and reversible T wave alterations. As in the case of the tricyclic antidepressants, the ECG changes with antipsychotic drugs are also dose dependent [2,15]. The dosage of tricyclic antidepressants and antipsychotics given to our patients was at the lower side of therapeutic range. Moreover, the ECG changes were noted exclusively after ECT. The ECGs of all patients at baseline and thirty minutes after ECT were normal. There was also no correla-

tion between the type of drug and cardiac arrhythmias (Table 3).

An increase in size of 'P' wave in the ECG after ECT was seen in 5% patients. It is probably a sign of dilatation of the atria due to increase in venous return and venous pressure [11].

REFERENCES

1. Shukla GD. Adverse effects of ECT. In : Gangadhar B, ed. Proceedings of the national workshop on ECT Bangalore : National Institute of Mental Health and Neuro Sciences 1992 : 83-102.
2. Hackett TP, Rosennaum JF, Tesar GE. Emotions, Psychiatric disorders and the heart. In : Braunwald E, ed. Heart disease 3rd ed. Philadelphia : WB Saunders Co. 1988 : 1883-900.
3. Theryan P, Saju PJ, Dutta S, John JK, Karuvilla K. Physical morbidity with unmodified ECT- A decade of experience. Indian J Psychiat 1993; 35 : 211-4.
4. Hashe J, Roeber E. Electroconvulsive therapy in Denmark. Br J Psychiatry 1976; 128 : 241-5.
5. Hussar AE, Pachter M. Myocardial infarction and fatal coronary insufficiency during electroconvulsive treatment. JAMA 1968; 204 : 1004-5.
6. Alexander SP, Lawrence NG, Lewis WH. Deaths following electrotherapy. JAMA 1956; 161 : 577-9.
7. Arneson GA, Butler T. Cardiac arrest and electroshock therapy. Am J Psychiatry 1961; 177 : 1020-2.
8. Cropper CF, Hughes M. Cardiac arrest (with apnoea) after ECT. Br J Psychiatry 1964; 110 : 222-4.
9. Altschule MD. Further observations on vagal influences on the heart during electroshock therapy for mental disease. Am Heart J 1950; 39 : 88-91.
10. Chaudhary S, Augustine M. Cardiogenic shock with imipramine. Indian J Psychiat 1990; 32 : 364-5.
11. Shah VD, Mansurre AM, Hakim IM et al. Cardiovascular and electrocardiographic changes after electroconvulsive therapy (ECT)- a series of 50 cases. Indian J Psychiat 1977; 19 : 51-3.
12. Fink M. Vascular effects. In : Convulsive therapies : Theory and Practice. New York : Raven Press, 1979; 155-7.
13. Marshall JB, Forker AD. Cardiovascular effects of tricyclic antidepressant drugs : Therapeutic usage, overdose and management of complications. Am Heart J 1982; 103 : 401-5.
14. Siddiqui JH, Vakassi MM, Ghani MF. Cardiac effects of amitriptyline over dosage. Current Therapeutic Res 1977; 22 : 321-4.
15. Fowler ND, Mc Call D, Chou T, Holmes JC, Hanenson IB. Electrocardiographic changes and cardiac arrhythmias in patients receiving psychotropic drugs. Am J Cardiol 1976; 37 : 223-9.