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A COMPARISON OF ELECTROCARDIOGRAPHIC CHANGES IN PRE AND POST PROCEDURE PERIOD IN PATIENTS UNDERGOING ELECTROCONVULSIVE THERAPY

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ABSTRACT

The sensitive self excitable myocardial tissue is known to be affected by exogenous source of electrical stimulation resulting in moderate changes in rate, rhythm or altered conduction to very severe arrhythmias which are potentially fatal. The present non randomized prospective study was done on 100 subjects undergoing electroconvulsive therapy as part of their treatment for psychiatric ailments. ECG was recorded in pre and post ECT periods to assess evidence of electrical stimulation induced changes in ECG parameters. None of the patients had cardiac disease prior to ECT. Our study revealed definite and significant changes in the heart rate in the post ECT period. However contrary to the other published studies, no serious rhythm abnormalities were observed. The study concludes that at energy level (70-130 volts) used in modern ECT equipment and the duration (0.5-1.5 seconds) for which the electrical energy is passed through the human body, it does not cause significant changes in the electrical activity of the heart in North Indian subjects.

KEYWORDS*:* Electroconvulsive therapy, ECG, Cardiac disease

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1 INTRODUCTION

Cardiomyocytes are highly excitable tissues of the body, exhibiting the property of self excitation or autorhythmicity. Any change in the milieu of the myocardial tissues, internal or external is likely to trigger a response which is demonstrable as change in the state of the action potential. In clinical sciences there are many examples of modalities of treatment wherein deliberately an electrical current is passed through parts of the human body as part of a therapy protocol to bring about desired effects in the manner in which the specific tissue behaves. Frequently used electrical stimulation is used by the physiotherapists, psychiatrists and cardiologists, Electroconvulsive therapy is an important non-pharmacological intervention which is considered to be one of the most effective treatment for patients suffering from certain severe neuropsychiatric disorder like schizophrenia, major depression, catatonia or psychosis, prolonged or severe manic episodes, neurological disorder, severe psychomotor retardation and also in patients who need rapid and definitive response (e.g. in psychosis or a risk of suicide)[1]. Psychotropic medications produce significant improvement in most patients with major affective disorders and schizophrenia. However, a substantial minority of patients either do not respond to pharmacological treatment, suffer severe adverse affects that make medications intolerable, or have severe symptoms that benefit from urgent intervention and rapid response. For such patients, ECT offers a useful intervention[1]. It is well known that when an electrical current of any intensity is passed through the body, it is bound to affect, though temporarily, electrophysiological changes occurring in almost all tissues more importantly the vulnerable tissues like brain and heart. While the changes in the neuronal activity are the desired effects of Electroconvulsive therapy, the same may not be true of self excitable myocardial cells where disruption of its normal electrical events and may actually lead to fatal changes like triggering of fatal tachyarrhythmias. There are very few documented studies in India or abroad linking the cause of serious cardiovascular anomalies or mortality in the post ECT period[2]. The literature is mostly silent on linking of preexisting ECG changes to more morbid changes after the ECT. Though it is a routine procedure to get ECG done in every patient aged 40 years or above prior to conducting ECT, the practice of recording ECG in the post ECT period is not in vogue and therefore a lot of changes go unnoticed. The present study attempted to explore the possibility of occurrence of adverse electrophysiological changes observed in ECG of the patients who have undergone ECT with an intention to prevent mortality or morbidity due to ECT and make ECT a much safer procedure.

ECT involves the passage of a brief electrical current through brain to induce a generalized central nervous system seizure. Although favorable response to ECT can occur quickly, clinical benefits typically require multiple treatments administered over a period of several weeks[1] .The decision to administer ECT is based on an evaluation of the risks and benefits for the individual patient and involves a combination of factors, including psychiatric diagnosis, type and severity of symptoms, prior treatment history and response, identification of possible alternative treatment options and patient preference[3] .Two commonly used methods for administering ECT include a direct (unmodified) and a modified method. In Direct type of electroconvulsive therapy an electric current of 70 to 130 volts is passed for 0.5 to 1.5 seconds. It briefly throws the body into epilepsy-like seizures. No muscle relaxant and anaesthesia is used. Medication is given to reduce secretions. However, In Modified ECT, medication is given to reduce secretions from the mouth. Muscle relaxants and anaesthesia are given to reduce the overt epileptic /muscular convulsion and patient anxiety[4] .The goal of muscle relaxant is to achieve good muscle

relaxation and not total muscle paralysis[5]. Whether it is a direct ECT or a modified one, both are likely to affect the myocardial autorhymicity, excitability and conductivity for the short duration of time when the ECT is administered. Some of these changes triggered in the electrical activity of the heart may persist for a longer time and occasionally may lead to generation of fatal patterns. It becomes imperative to identify such changes induced as a result of ECT or to anticipate such changes in patients who may already be having a compromised or abnormal electrical activity of the heart. Such identification would help to take the necessary precautions before, during and after administering ECT in patients who are likely to be benefited by this mode of therapy.

2 AIM

To record the electrocardiography (ECG) parameters in patients undergoing electroconvulsive therapy (ECT) and to compare the changes if any with ECG done in the pre ECT procedure period.

3 MATERIALS AND METHODS

The study was conducted at Christian Medical College, Ludhiana at the Department of Physiology and Dept of Psychiatry. This Non-Randomized comparison study was conducted over a period of two years commencing from October 2009.100 cases of electroconvulsive therapy at the department of Psychiatry were included in the study (50 of modified ECT and 50 of unmodified ECT)An informed consent for inclusion in the study was taken. Each patient was reassured of the non invasive nature of the test and any questions regarding the procedure by the patient, if any, were duly addressed to allay any apprehension. Relevant details of his/her history, prevailing illness, diagnosis, ongoing medication, associated or concurrent medical illness was recorded. A detailed general physical examination as well as systemic examination was done and observations were documented as per the protocol of the study.Each patient was screened for indication for the ECT by the Psychiatrist who also complied with the pre requisites and consent of ECT in each case.Prior to ECT, vital signs of each patient were recorded including Heart rate, Blood Pressure and Respiratory rate. The patient was allowed to relax and was explained the process of recording ECG in order to minimize the anxiety that may be there in his/her mind.Electroconvulsive therapy was given by the Psychiatrist using constant current, brief pulse ECT machine which is able to deliver a wide range of electrical dose in the range of 70-130 volts. The ECT machine used is MEDICA BPE-791.ECT was scheduled between 8:00am and 11:00am. Patients were kept fasting for at least 8 hours prior to ECT .Dentures , contact lenses or any other ornaments were removed prior to the procedure . The procedure room was equipped with a defibrillator and drugs necessary for cardiopulmonary resuscitation. Electrodes were placed bifrontotemporal with the centre of each electrode about 1 inch above the mid point of an imaginary line drawn from the tragus to the external canthus. The electrical stimulus given was 70-130 volts and the current was allowed to flow up to 0.5-1.5 seconds.

The brief pulse square wave stimulus was used for this purpose, which rises and falls abruptly and delivers its charge typically in 1 msec. Because the current is off during most of the stimulus train, brief pulse square wave stimuli deliver a fraction of sine wave stimuli of equal current, yet have the same therapeutic quality. A standard 12 lead EKG was recorded on BPL CARDIART 108T / MK-VII machine prior to ECT in each case . A repeat ECG was done 10 minutes after completion of ECT allowing for muscle tremor to settle.The following parameters in Pre and Post ECT, Electrocardiogram were compared: Changes in rate, Changes in rhythm, Changes in electrical axis (QRS axis), changes in morphology of the waveforms, conduction defects if any and ST segment changes were noted. The observed results were analyzed using students paired t-test.

4 OBSERVATIONS AND RESULTS

Majority of the patients included in the study belonged to age group of 21-30 years (83 males, 17 females) who had no cardiac morbidity. There was no ECG evidence of cardiac disease in the few subjects between the age 51-60 years included in the study. The heart rate in the pre ECT period (min 60/min - max 122/min; mean 87.36/min) was compared to HR of post ECT period (min 75/min - max 150/min; mean 107.38/min) which was statistically significant ($p < 0.001$).There was no significant occurrence of arrhythmia in the post ECT period. This was inferred by looking at the final statistical analysis of p-value of QRS duration as 0.103 implying indirectly that ECT of the given strength had no significant effect on ventricular depolarization in the present study.The pre ECT electrocardiogram showed minimum P wave duration of 0.04 sec, amplitude 0.1 mv and maximum duration 0.8 sec and amplitude 0.2mv with the mean value of 0.0848 sec and 0.105 mv . The corresponding values in the post ECT period showed minimum P wave duration 0.04 sec, amplitude 0.1 mv and maximum duration 0.8 sec and amplitude 0.3 mv with the mean value of 0.844 sec and 0.108 mv. On statistical analysis this change was observed to be insignificant (p value for duration & amplitude came out to be 0.878 and 0.162 respectively) showing the safety of ECT on cardiac depolarization.QRS Duration and amplitude in the pre and post procedure period were compared. The QRS duration in the pre procedure period has a minimum value of 0.08 sec, maximum value of 0.12 sec and mean of 0.1128 sec. Amplitude had minimum value of 0.4 mv, maximum of 3.2mv and mean value of 1.696 mv. In the post procedure period the minimum value of duration was 0.08 sec, maximum of 0.12 sec and mean of 0.1096 sec. Amplitude had minimum value of 0.4mv, maximum 3.0 mv and mean of 1.766 mv. The final statistical analysis showed the p-value for duration was 0.103 and for amplitude 0.070 showing that the change was insignificant.

T wave parameters in the pre ECT period showed a duration 0.12 sec, amplitude 0.3 mv and maximum duration 0.27 sec and amplitude 0.6 mv with the mean value of 0.2152 sec and 0.36 mv in the pre ECT period. In the post ECT period showed minimum T wave duration 0.1 sec, amplitude 0.2 mv and maximum duration 0.3 sec and amplitude 0.5 mv with the mean value of 0.211 sec and 0.354 mv. The P value for duration & amplitude came out to be 0.051 and 0.322 respectively which was not significant. There was no significant change in T- wave amplitude and duration. There was no peaking or inversion showing the safety of ECT on ventricular repolarization.We did not observe manifestations of increased cholinergic activity by reduction in heart rate or any episode of asystole or occurrence of premature ventricular contractions. PR- interval had a minimum value of 0.08 sec, maximum of 0.16 sec and mean of 0.112 sec in pre period. In post period, minimum was 0.2 sec, maxmimum was 0.4 sec and mean value of 0.352 sec. P- value came out to be 0.013 which was not significant showing the safety of ECT on AV conduction time.QT- interval had a minimum value of 0.2 sec, maximum of 0.4 sec and mean of 0.363 sec in pre ECT period. In post ECT period, minimum was o.2 sec, maximum was o.4 sec and mean value of 0.352 sec. P- Value came out to be 0.007 which was insignificant.No ST-segment elevation or depression were observed in the post ECT period implying that ECT given at the energy level used in our study did not have significant effect on ventricular pre repolarization phase.

5 DISCUSSION

Many changes are known to occur as a result of transient flow of electrical current as happens in electroconvulsive therapy procedure. Disorders of cardiac rate and rhythm includes sinus tachycardia, sinus bradycardia, atrial tachycardia, ectopics[2]. The other main cardiac complications known to occur are asystole, cardiac rupture, myocardial infarction [4] ,arrhythmias, instability of arterial blood

pressure, cardiac arrest, life threatening multifocal premature ventricular beats, bigemini, trigemini[5] ,transient left ventricular systolic dysfunction[6] ,ventricular ectopy, transient ST segment depression, transient wall motion abnormalities on echocardiography[7] and global left ventricular hypokinesia .Cardiovascular and pulmonary complication are the potential cause of death and morbidity after ECT. Reported cases of severe cardiovascular complications or mortality have shown variable results [4,8-10] pointing to cardiac arrest or cardiac insufficiency.Both Parasympathetic and sympathetic cardiac arrhythmias are frequently observed during and immediately after ECT[6]. The most pronounced is sinus bradycardia, occasionally leading to stand still for several seconds. Other vagal arrhythmias that tend to appear in the immediate post ictal phase, include atrial premature contraction, paroxysmal atrial tachycardia, atrial flutter and atrial fibrillation. Sympathetic arrhythmias reported include sinus tachycardia, bigeminy, Trigeminy, ventricular tachycardia, ventricular fibrillation, and PVCs during sinus tachycardia. T-wave amplitude changes and ST segment depression are also described [11]. Myocardial oxygen consumption as assessed by the rate pressure product raises 200-400% while receiving the ECT induced seizure. The presence of beta blockers, and anticholinergic agent (e.g. Atropine, glycopyrrolate) help to prevent cardiac stand still due to unopposed parasympathetic tone. None of the cardio specific enzymes (CPK - MB, LDH-l, and LDH-2) are reported to be elevated after modified ECT[11].

Cardiovascular changes after electroconvulsive therapy may be due to the following reasons:

1. The effect of electroconvulsive therapy on autonomic system causes a number of cardiovascular system changes and makes it particularly vulnerable to exacerbation and even increased risk of death[12].
2. Electroconvulsive therapy produces a brief period of increased parasympathetic activity followed by increased sympathetic activity[13].
3. The parasympathetic activity that precedes the sympathetic outflow poses a risk of causing bradycardia and at times can lead to asystole[14]. Increased sympathetic outflow contributes to hypertension and tachycardia and cases of bigemini, trigemini and supraventricular tachycardia have been reported in conjunction with electroconvulsive therapy[6].
4. Electroconvulsive therapy causes reduction in the venous return to the heart by the raised intrathoracic pressure caused by the convulsion and by intermittent positive pressure ventilation(IPPV)[15].
5. The hemodynamic changes occurring during electrically induced seizure activity are also attributed to adrenergic stress response.
6. Electroconvulsive therapy also provokes a rise in plasma catecholamines and induces a considerable increase in myocardial oxygen demand[16].

Cardiovascular complications (e.g., acute myocardial infarction, ventricular fibrillation, Asystole, ruptured aneurysm) are responsible for most of the mortality occurring in association with ECT. Caution is required in patients with arrhythmias, left-heart atrioventricular conduction defects, ischemia, heart failure, or other atherosclerotic pathology.The most frequent complications during ECT prior to modem anesthetic techniques and effective medical management were cardiovascular. Other significant cardiovascular risk factors that must be assessed include uncompensated congestive heart failure, severe valvular disease, unstable angina, uncontrolled hypertension, fragile vascular aneurysms, and clinically significant cardiac arrhythmias[17]. A thorough medical evaluation is critical prior to considering ECT for high-risk cardiac patients.131 In addition to a careful history, physical examination, and appropriate blood work, an ECG and a chest radiograph is useful. Functional cardiac testing may be needed in selected cases where ischemic disease is suspected.132 Optimal medical therapy in preparation for ECT is mandatory to minimize risk during and after treatment. Usual cardiac medication should be continued, unless there

are specific contraindications[18] Cardiac pacemakers generally have a protective effect during ECT, improving the heart's rate and rhythm but it is advisable that a cardiac electrophysiologist should be consulted prior to treatment[19]. In patients with permanent pacemakers, a temporary conversion to fixed rate pacing is done to minimize the interference with pacemaker functioning[20]. In patients with automatic internal cardioverter-defibrillator, the device should be deactivated before the electrical current is applied and reactivated in the early recovery period[21]. In a study of 26 patients with pacemakers only one serious cardiac event in the form of supraventricular tachycardia occurred. With proper pre-ECT cardiac and pacemaker/ defibrillator assessment, ECT can be safely and effectively administered to patients with an implanted cardiac device. Appropriate medical consultation should be sought to minimize risks for such patients (e.g., digitalization for congestive heart failure, antiarrhythmic therapy or cardioversion for atrial fibrillation). The decision to administer ECT will then be, as always, a matter of informed consent by the patient and relatives with due consideration of the risks of giving or not giving ECT[22].

There exists no specific data to guide the safe use of ECT in patients with recent myocardial infarction. The physiology and histopathology of the developing lesion suggest that the risk of ventricular fibrillation or rupture is greatest during the first 10 days post infarction. On the other hand, Ungerlieder[23] inadvertently gave ECT without ill effect to a patient with an acute myocardial infarction; he also described a second patient given ECT three days post-infarction without sequelae. Patients with preexisting ischemic disease and conduction disorders were at risk for ischemia and arrhythmias, respectively.

6 CONCLUSION

We conclude that there is definite and significant change in the heart rate in the post ECT period but this change remains within the homeostatic range and therefore does not adversely affect the cardiac activity or the haemodynamic parameters significantly. In contrast to several studies which showed the presence of arrhythmias in the post ECT period, some of which are reported to be fatal also, no such arrhythmias were observed in our study using the defined safe energy level and defined protocols for ECT given by modern ECT equipment like MEDICA BPE-791 used in the present study. There were no significant changes in the duration and amplitude of p wave, QRS complex and T wave in the post ECT period. No significant changes were seen in PR or QT interval. ST segment remained isoelectric in the post ECT period showing that the present protocol of ECT did not induce any obvious myocardial ischemia.It is thus inferred that at energy level (70-130 volts) used in modern ECT equipment and the duration (0.5-1.5 seconds) for which the electrical energy is passed through the human body, it does not cause significant changes in the electrical activity of the heart in North Indian subjects.

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