Is It Possible to Predict Cerebral Dysrhythmias by Measuring Pulse and Blood Pressure Before and After Electroconvulsive-therapy ?

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A number of patients who receive electroconvulsive therapy (ECT) have a prior abnormal Electroencephalograms (EEG). This study tries to find the relation between patients with abnormal EEG who received ECT and their pulse rate, systolic, and diastolic blood pressure. Three groups of patients (39 patients) with major depression, who received ECT were studied. All patients received bilateral ECT under general anesthesia. The first group of patients had abnormal EEG and clinically had seizures. In this group, the relationship between means of pulse rate, systolic, and diastolic blood pressure before and after ECT were statistically significant where z-test results were -3.18, -3.06, and -2.93 respectively. The second group of patients had normal EEG but had doubtful seizures. The relationship between means of pulse rate and diastolic blood pressure were statistically significant where z-test results were -3.18 and -2.9. The third group of patients had normal EEG and clinically had no evidence of seizures. The relationship between means of pulse rate, systolic, and diastolic blood pressure were statistically not significant where z-test results were -1.18, -o.8, and -o.82 respectively. Conclusion: It is possible to predict the presence of abnormal EEG from marked increase in pulse rate and blood pressure one hour after ECT

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INTRODUCTION

It is well known that electroconvulsive-therapy (ECT) is associated with a hyperdynamic state characterised by arterial hypertension, tachycardia, and increased cerebral blood flow rate and velocity (Viguera et al., 1998). Moreover, ECT increases neuronal energy consumption and alters systemic hemodynamics (Satio, 1996). ECT produces a hyperdynamic cardiac state in which there is a significant increase in blood pressure, a shortening of the pre-ejection period (PEP), and an increase in rate pressure product (RPP). Changes in the PEP and the RPP are compatible with increased cardiac contractility and oxygen consumption, respectively. These effects are apparently mediated by an ECT-induced increase in circulating catecholamines, particularly epinephrine (Mulgaokar, 1985).

Past research focused on characterizing the cognitive deficits caused by ECT, understanding their causes, and defining ways of ameliorating the deficits. Recommendations on evaluating the effects of mediating variables such as blood pressure rise, and assessing the influence of background variables such as age, sex, and brain abnormality were considered to be essentially useful (Calev, 1994).

Electrically induced seizures have been used widely to treat psychiatric

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disease since its introduction in 1938. Seizure activity is postulated as the therapeutic aspect of this form of treatment, but it is accompanied by untoward physiologic consequences. The cardiovascular responses of such sequel consist of generalized autonomic nervous system stimulation with initial parasympathetic outflow, followed immediately by a sympathetic response. In certain patients the sequence described may result in an initial bradycardia or even asystole, followed by tachycardia, dysrhythmia, and hypertension. The initial rise in systolic pressure is proportionately greater than that in diastolic pressure, and is more pronounced in hypertensive than in normotensive patients (Prudic et al.,1987). The cerebrovascular system as well, responds with a marked increase in cerebral blood flow to the increased cerebral oxygen consumption and the dramatic elevation of intracranial pressure. Accordingly, the general anesthesia for electroconvulsive therapy (ECT) should be administered only in locations equipped for support of the unconscious patient and treatment of complicaton (Gaines & Rees, 1992).

Larsen and colleagues (1984) investigated the relationship between the duration of the ictal tachycardia, the motor response, and the electroencephalography (EEG), defining a cardiovascular seizure endpoint as the time point associated with the most rapid decrease in heart rate.

Although ictal EEG monitoring was described as early as 1939 (Fleming et al.,1939), it was not seriously considered for routine clinical use until the pioneering studies of multiple monitored ECT (MMECT) by Blachly and Growing in the mid 1960s(Blachy & Gowing, 1966). The scalp-recorded EEG of low voltage signal that originates from synchronus activity within the dendritic fields of groups of adjacent large cortical pyramidal cells, has a spatial resolution of approximately one square inch of underlying cortex (Gaines & Rees, 1992). A recording made during the waking state would consis usually of a mixture of lower amplitude alpha and beta activity, plus the effects of muscle and movement artifacts. Therefore during baseline pre-ictal EEG recording a mixed fast and slow activity is indicative of anesthesia effect. Following the electrical stimulus, during which the EEG amplifiers are blocked by the effects of the stimulus, a brief pre-ictal period of low amplitude fast activity may sometimes be seen. The next phase, is the epileptic recruiting rhythm, a period of low to moderate amplitude activity in the alpha or beta range, believed to be associated with the synchronizing effects of thalamo-cotical projections during the early stages of seizures generalization (Kiloh et al., 1981).

The post-ictal phase begins immediately upon seizure termination. Although the immediate post-ictal EEG, appears flat, the amplification used is much lower than that encountered with routine non-ictal EEG recording. The degree of this post-ictal suppression varies considerably across patients, and is influenced by ECT type. When minimal suppression is present, the immediate postictal EEG is characterized by a mixture of low amplitude fast and slow activity which may obscure the seizure endpoint, particularly if a termination phase is present(Weiner et al.,1986).

The aim of this study is to illustrate of the relationship between cerebral dysrhythmias and changes in pulse rate and blood pressure before and after ECT.

SUBJECTS AND METHODS

Subjects in this study were selected from patients who received at least 3 ECT sessions in the psychiatric department at Erfan and Bagedo General Hospital (a private hospital in Jeddah, Saudi Arabia). Thirty nine in-patient who were admitted with a diagnosis of major depression according to DSM-IV criteria.

Patients who received less than 3 ECT sessions were excluded. Electroconvulsive therapy sessions for all patients included in this study were every other day. Twenty of these 39 patients had an element of suicidality. Twelve of them had had suicidal thoughts. Eight of them had had suicidal attempts. Five of them had both suicidal thoughts and attempts. These 39 patients were chosen randomly from 342 inpatients who received ECT in the period from July 1997 to March 1998. The age range was from 18 to 45 year old. Confounding variables like age, and sex were considered to be relatively similar in the three groups of patients. Twenty five of them were males and 14 were females. All of them were physically healthy subjects e.g., no diabetes, no hypertension, no collagenic diseases, no other neurological deficits (except in the group with cerebral dysrhytmias), no cardiac diseases,etc. All the patients were not on psychotropic medication before the first ECT session. This was done to avoid the effects of these psychotropics on pulse, blood pressure and seizure duration. Bilateral ECT was the modality used for all cases.

Methods

Patients were examined 1-2 days before starting the ECT course. Physical examination was done to exclude any organic disease. Routine laboratory investigations and x-rays (complete blood picture, erythrocyte sedimentation rate, blood urea & creatinine, random blood sugar, serum sodium & potassium and plain x-ray chest) were done (American Psychiatric Association, 1990). Electroencephalograms (EEG) were done for all patients before the start of ECT sessions. Diagnostic criteria of major depression from Diagnostic and statistical Manual-IV (DSM-IV) were applied on every patient in this study before the ECT(American Psychiatric Association, 1994).

Patients in this study were divided into 3 groups:

The first group: It consisted of 13 patients who had definite changes in EEG in the form of focal activity, spikes, or generalized cerebral dysrhythmia. Clinically seven of them had shown generalized seizures activity and six of them had shown partial seizures activity. These diagnoses were according to the International Classification of Epileptic seizures-revised (1989) (Commission on classification and terminology of the International league against epilepsies, 1989).

The second group: It consisted of another 13 patients who had normal or (no pathology) in their EEG. Clinically it was undetermined whether they had had either partial or generalized seizures.

The third group: It consisted of 13 patients who had normal EEG and clinically had neither partial nor generalized seizures.

In a good number of patients who received ECT, hemodynamic changes persists for less than 60 minutes following ECT session (Viguera et al., 1998). So, measuring of pulse and blood pressure was performed 60 minutes before and after ECT. The duration of ECT stimulus in every session was assessed automatically in seconds through the Thymatron device.

ECT procedure

It was administered bi-fronto-temporally every other day which was usually started at 8 AM. All patients were fasting for at least 6 hours prior to the ECT treatment. Patients received a variable number of ECT sessions ranging from 3-12 sessions.

Pre-medication and anaesthesia: Thiopental sodium (Intraval) was used as an anaesthetic agent in a dose of 4-6 mg/ kg body weight diluted in 20 ml saline and given very slowly intravenously to induce a degree of anaesthesia as light as possible. Flexidyl was used as a skeletal muscle relaxant in a dose of 0.5 mg/kg body weight. Moreover, 100% oxygen was given from the onset of anesthesia until the resumption of adequate spontaneous breathing (American Psychiatric Association, 1990).

The device used was Thymatron. The ECT amount of energy, which was given to the 39 patients, in every session, ranged from 50 to 350 millicoulombs (10% - 70% of total energy). The duration of the ECT stimulus was automatically controlled by the device of ECT(Swartz & Abrams, 1994). Duration of seizures was assessed by using a stop watch.

Statistical analysis

Sistical analysis was performed using the SPSS-PC + package. The data was expressed as mean values SD and value of < 0.005 were considered to be statistically significant. Z-test was used to compare between different means.

RESULTS

In the first group of patients who had definite pathology in EEG and clinically had features of either partial or generalized seizures; the mean values of pulse, systolic, and diastolic blood pressure before ECT (table 1) were 81.01 $\pm$ 7.43, 112.27 $\pm$ 9.61, 73.7 $\pm$ 5.5 respectively. While the mean values of pulse, systolic, and diastolic blood pressure after ECT were 92.3 $\pm$ 4.76, 120.54 $\pm$ 8.39, 80.02$\pm$ 5.36. The difference between these means before and after ECT were statistically significant where p-values were 0.001, 0.002, 0.003 respectively and z-test values were -3.18, -3.06, -2.93 respectively.

In the second group of patients who had no pathology in EEG and clinically had doubtful features of seizures; the mean values of pulse, systolic, and diastolic blood pressure before ECT (table 2) were 84.06 $\pm$ 6.66, 112.96 $\pm$ 14.42, and 76.26 $\pm$ 6.81 respectively. While the mean values of pulse, systolic, and diastolic blood pressure after ECT were 92.34 $\pm$ 2.83, 118.55 $\pm$ 10.54, 82.23 $\pm$ 5.51. The differences between the means of pulse and diastolic blood and diastolic blood pressure before and after ECT were statistically significant where p-values were 0.001 and 0.004 respectively and z-test values were -3.18 and -2.9. It is interesting to note that, the difference between the means of systolic blood pressure before and after ECT,however was statistically not significant where p-value was 0.04 and z-test -2.05.

In the third group of patients who had no pathology in EEG and clinically had no evidence of seizures; the mean values of pulse, systolic, and diastolic blood pressure before ECT (Table III) were 89.85 $\pm$ 7.01, 117.3 $\pm$ 8.73, 79.6 $\pm$ 6 respectively. While the mean values of pulse, systolic, and diastolic blood pressure after ECT were 87.28 $\pm$ 4.93, 113.77 $\pm$ 8.39, 79.86 $\pm$ 8.97 respectively. The difference between the means of pulse, systolic, and diastolic blood pressure were statistically not significant at

Table 1
Comparison between Averages of Pulse Rate, Systolic and Diastolic Blood Pressure Before and After ECT in group I (n = 13 patients)

	Pulse	Systolic blood pressure	Diastolic blood pressure
Before ECT	81.01±7.43	112.27 ± 9.61	73.7 ± 5.5
After ECT	92.3 ± 4.76	12054 ±8.93	80.02 ± 5.36
p-values	0.001	0.002	0.003
Z-test	-3.18*	- 3.06	- 2.93*

* Level of significance is P <0.005

Table 2
Comparison between Means ± Standard Deviations of Pulse Rate, Systolic, and Diastolic Blood Pressure Before and Diastolic Blood Pressure Before and After ECT in Group II (n = 13 patients)

	Pulse	Systolic blood pressure	Diastolic blood pressure
Before ECT	84.06 ± 6.66	112.96 ± 14.42	76.26 ± 6.81
After ECT	92.34 ± 2.83	118.55 ± 10.54	82.33 ± 5.51
p-values	0.001	0.04	0.004
Z-test	- 3.18*	- 2.05	- 2.9*

* Level of significance is P <0.005

Table 3
Comparison Between Means ± Standard Deviations of Pulse Rate, Systolic, and Diastolic Blood Pressure Before & After ECT in Group III (n = 13 patients)

	Pulse	Systolic blood pressure	Diastolic blood pressure
Before ECT	89.85 ± 7.01	117.3 ± 8.73	79..6 ±6
After ECT	87.28 ± 4.93	113.77 ± 8.39	79.86 ± 8.97
p-values	0.24	0.42	0.42
Z-test	- 1.18	- 0.8	- 0.82

Level of significance is P <0.005

p-values of 0.24, 0.42, 0.42 and z-test values were -1.18, -0.8, 0.82 respectively.

The mean durations of ECT seizures were assessed in the three groups of patients(per seconds). These durations were 51.8 ± 4.5, 44.2 ± 6.4 and 29.4 ± 4.4 repectively. There was a significant increase in the mean duration of seizures in the first group where, z-test value was 29.7 and p-value was < .00001. In another word, the mean duration of the ECT seizures in the first group with epileptic seizures and abnormal EEGs was significantly higher than that in the other two groups.

DISCUSSION

In this study it was found that there is a relation between the increase in pulse and blood pressure following ECT and the presence of cerebral dysrhythmias. It is known that the brain, heart and blood vessels bear the brunt of the physiological impact of ECT, hence, patients with compromised cerebral and cardiovascular function constitute the population at greatest risk during the procedure. Blood pressure and heart rate fall during the stimulus, then rapidly rise to reach rate-pressure product level (RPP= heart rate X systolic blood pressure) that are several multiples of the baseline. Sharp increases in cerebral blood flow and metabolism, and in cerebrovascular permeability, contribute to an abrupt but transient concurrent increase in intracranial pressure. Electro-convulsive therapy initially may increase cerebral metabolic rate of oxygen more than cerebral blood flow and that rapidly increasing blood pressure transiently may overwhelm cerebral pressure autoregulation (Satio, 1996). An initial vagally driven sinus bradycardia is rapidly replaced by a pronounced sympathoadrenal tachycardia. Normally, EEG patterns immediately following the electrical stimulus are characterized by an invariant sequence of amplitude build- up, hypersynchronous large spikes, bursts of spike and slow-wave complexes, and suppression (or flattening) as the synchronized neural activity generalized and then abates. Several hours of cerebral hypometabolism ensue, accompanied by generalized EEG slowing and reduced cerebral blood flow(Abrams,1991).

Fu, 1998 and colleagues noticed an acute hypertensive response are often observed immediately after electroconvulsive therapy in 110 treatments evaluated on 22 patients. However Prudic and colleagues, studied 34 patients with primary, major depressive disorder where the pulse rate and blood pressure were assessed prior and following seizure induction at every treatment. They found that, pretreatment pulse rate was strongly predictive of peak postictal change in both pulse rate and blood pressure, while pretreatment blood pressure was not. Patients with high pre-ECT pulse rate had smaller peak postictal pulse rate and blood pressure increases (Prudic et al.,1987). Moreover, Fuenmayor and colleagues, found that in some patients without heart disease, ECT procedures are associated with a significant and transient decrease in left-ventricular systolic and diastolic function (Fuenmayor et al., 1998). The results in the prior two studies seemed contradictory with that in this study. But it is important to notice that Prudic and colleagues, studied patients with major depression without mentioning the EEG patterns or any clinical features of partial or complex seizures in these patients, while Fuenmayor and colleagues studied the effects of ECT on left ventricular functions in 11 patients only.

Hardlicka and colleagues studied the recording during a series of ECT, and persistent changes after finishing the series of ECT. They found that EEG screening has shown that the patients with abnormal EEG do not respond well to ECT. In the interparoxysmal recordings during a series of ECT the associations of the generalised slowing in the range of the theta and delta activity with the therapeutic effect were looked for, particularly in the past. Persistet changes after finishing the ECT are usually examined especially to look if ECT does not cause persistent abnormalities of the EEG recording which would be an evidence for brain damage or for epileptic kindling (Hardlicka et al, 1998). The importance of this study is that, it can explain lowering of seizure threshold in patients with cerebral dysrhythmias. Accordingly, the results of the present study,can be explained by the phenomenon of kindling where the patients with abnormal EEG are vulnerable to develop a low threshold of seizure activities following ECT. So, post ECT serial or status cerebral dysrhythmias may develop and lead to persistent increase in both pulse rate and blood pressure following ECT sessions.

Another explanation of the increase in pulse and blood pressure in patients with abnormal EEG (following ECT) is, the over stimulation of sympathetic tone in the brain stem of these patients. The synergistic interaction between ECT and disturbed brain electricity (which is accompanying with cerebral dysrhythmias) leads to elevation of pulse and blood pressure after ECT in this particular group of patients.

A third explanation of increase in pulse and blood pressure in patients with abnormal EEG (following ECT) is the increase of duration of ictal phase in these patients. This may lead to more stimulation of cardiovascular center in the brain stem. Consequently, these patients with abnormal EEGs are more vulnerable to increase in pulse rate and blood pressure following ECT.

Although measurement of blood pressure and pulse associated with ECT are easy to take, there have been few studies about these measurements, and they may have untapped potential for evaluating ECT and ECT patients. On reviewing medline from 1982 to 1999 and other literature about ECT, few references discussed the relation between hemodynamic changes and abnormal EEG during and after ECT (Hrdlicka et al., 1998). This area is in need of further research work applied on more patients treated by ECT and who have abnormal EEG.

Conclusion:

It is better to use EEG in the electroconvulsive therapy for screening patients before starting treatment. Moreover, the EEG correlates of the ECT recording of the paroxysm, immediate post-paroxysmal recording, interparoxysmal recording and during a series of ECT are needed to be done.

For psychiatrists who do not use EEG monitoring during ECT, they can predict abnormal EEG from marked increase in pulse rate, and blood pressure after ECT.

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REFERENCES

Abrams, R. (1991) Electroconvulsive therapy in the medically compromised patient. Psychiatr Clin Nor Am ,51: 871-85.

American Psychiatric Association(1994). Diagnostic criteria from DSM-IV. American Psychiatric Association. Washington, DC.

American Psychiatric Association(1990) Task Force on Electroconvulsive Therapy: the practice of electroconvulsive therapy: recommendation for treatment, training, and privileging. Washington, DC, American psychiatric Press;

Blachy, P. H, and Gowing D(1966) Multiple monitored electroconvulsive treatment. Compr Psychiatry, 7: 100-9.

Calev, A. (1994) Neuropsychology and ECT. past and future research trends. Psychopharmacol Bull ,30: 461-9.

Commission on classification and terminology of the International league against epilepsies(1989) Proposal for revised classification of epilepsies and epileptic syndromes. Epilepsia,30:389.

Fleming, GWTH. Golla, F. L. Walter, W. G. (1939) Electric-convulsion therapy of schizophrenia. Lancet ,2: 1353-5.

Fuenmayor, A. J. Fakin, Y. Moreno, J. Fuenmayor AM(1998) Effects of electroconvulsive therapy on cardiac function in patients without heart disease. Cardiology, 88: 254-7.

Fu W. Stool, L. A. White, P. F. and Husain M. M. (1998) Is oral clonidine effective in modifying the acute hemodynamic response during electroconvulsive therapy? . Anesth Analg ,86: 1127-30.

Gaines, G. Y. and Rees, D. I. (1992) Anesthetic considerations for electroconvulsive therapy. South Med J, 85: 469-82

Hrdlicka, M, Moran, M, and Blatny, M (1998) Use of EEG in electroconvulsive therapy. Detska Psychiatricka Klinika,137: 628-31

Kiloh, LG, McComas AJ, Osselton JW, et al (1981) Clinical Electroencephalography, 4ed . London, Butterworth.

Larson, G, Swartz, C, Abrams R(1984) Duration of ECT induced tachycardia as a measure of seizure length. Am J Psychiatry, 141: 1269-71.

Mulgaokar GD, Dauchot PJ, Duffy JP, and Anton AH(1985) Noninvasive assessment of electroconvulsive-induced changes in cardiac function. J Clin Psychiatry ,46: 479-82.

Prudic, J, Sackheim, H. A. Decina P, et al., (1987) Acute effects of ECT on cardiovascular functioning: relations to patient and treatment variables. Acta Psychiatr Scand ,75: 344-51.

Satio S (1996) Regional cerebral oxygen saturation during electroconvulsive therapy: monitoring by near-infrared spectrophotometry. Anesth Analg, 83 : 726-30.

Swartz, C. M, and Abrams R(1994) ECT instruction manual. Fifth edition. USA; Somatics, Inc.

Viguera, A. Rordorf, G. Schouten, R. Welch, C. and Drop, L. J. (1998) Intracranial haemodynamics during attenuated responses to electroconvulsive therapy in the presence of an intracerebral aneurysm. J Neurol Neurosurg Psychiatry, 64:802-5.

Weiner, R. D. Rogers, H. J. Davidson, J. R. et al., (1986) Effects of electroconvulsive therapy upon brain electrical activity. Ann NY Acad Sci, 462: 270-81.

هل من الممكن التنبؤ بوجود اضطراب فى إيقاع المخ عن طريق قياس النبض وضغط الدم قبل وبعد العلاج بالصدمات الكهربائية؟

فى هذه الدراسة كان هناك عددا من المرضى الذين لديهم اضطراب فى تخطيط الدماغ خضعوا للعلاج بمنظم إيقاع المخ (العلاج بالصدمات الكهربائية). حاولت هذه الدراسة أن تجد علاقة بين المرضى المصابين باضطراب فى تخطيط الدماغ وبين النبض وضغط الدم الاندفاعى والانبساطى لهؤلاء المرضى. تم تقسيم المرضى المصابين بالاكتئاب الجسيم وعددهم ٣٩ مريضا إلى ثلاث مجموعات. كل هؤلاء المرضى خضعوا للعلاج بمنظم إيقاع المخ ثنائى الجانب تحت مخدر عام، المجموعة الأولى من هؤلاء المرضى كان لديهم نوبات صرعية إكلينيكية واضطرابات فى تخطيط الدماغ، وقد وجد فى هذه المجموعة من المرضى فروقا إحصائية ذات دلالة بالنسبة لمتوسطات النبض وضغط الدم الاندفاعى والانبساطى قبل وبعد العلاج بمنظم إيقاع المخ حيث كانت قيم اختبار "زد" ٣,٠٥ - ٢,٩٣ على الترتيب. المجموعة الثانية كان لديهم تخطيطات سليمة للدماغ؛ ولكن كان هناك شك فى إصابتهم بنوبات صرعية، وقد وجد فى هذه المجموعة من المرضى فروقا إحصائية ذات دلالة.

بالنسبة لمتوسطات النبض وضغط الدم الانبساطى قبل وبعد العلاج بمنظم إيقاع المخ؛ حيث كانت قيم اختبار "زد" ٣,١٨ - ٢,٩ على الترتيب. المجموعة الثالثة كان لديهم تخطيطات سليمة للدماغ، ولم يشكوا من أى نوبات صرعية طوال حياتهم؛ ولم يوجد فى هذه المجموعة من المرضى أى فروق إحصائية ذات دلالة لنفس المتغيرات السابقة قبل وبعد العلاج بمنظم إيقاع المخ. ونجد من هذه الدراسة أنه من الممكن التنبؤ بوجود تغيرات فى تخطيط الدماغ عند وجود زيادة واضحة فى النبض والضغطين الانبساطى والاندفاعى بعد العلاج بمنظم إيقاع المخ.