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Electroconvulsive therapy impairs systolic performance of the left ventricle

Purpose: In this observational study, we examined left ventricular systolic performance during electroconvulsive therapy (ECT), using an echocardiographic automated border detection system.

Methods: Nine ASA I or II patients scheduled for ECT were studied. Bilateral ECT was performed after the administration of propofol 1 $mg \cdot kg^{-1}$, succinylcholine 1 $mg \cdot kg^{-1}$, and assisted mask ventilation with 100% oxygen. Cardiac function was monitored by transthoracic echocardiography, prior to anesthesia induction and throughout the ECT procedure until ten minutes after the seizure.

Results: Increased end-systolic area and decreased fractional area change were observed at one minute after the seizure compared to the awake condition. No regional wall motion abnormalities were observed in all patients both at baseline condition and during the ECT.

Conclusion: Systolic performance of the left ventricle estimated by echocardiography decreased transiently in the immediate period after the electric shock.

Objectif : Examiner, dans une étude par observation, la performance systolique du ventricule gauche pendant une sismothérapie (ST) en utilisant un système échographique de détection automatisée des limites pariétales.

Méthode : Neuf patients ASA I ou II devant subir une TEC ont été étudiés. La TEC bilatérale a été réalisée après l'administration de 1 $mg \cdot kg^{-1}$de propofol, 1 $mg \cdot kg^{-1}$de succinylcholine et la ventilation assistée avec masque et 100 % d'oxygène. La fonction cardiaque a été évaluée par échocardiographie transthoracique, avant l'induction de l'anesthésie et tout au long de la TEC jusqu'à dix minutes après la période convulsive.

Résultats : L'augmentation de l'aire de fin de systole et la diminution de l'aire fractionnaire ont été observées une minute après la TEC en comparaison avec l'état vigile. Aucune anomalie régionale du mouvement de la paroi n'a été observée sous les conditions de base et pendant la TEC.

Conclusion : La performance systolique du ventricule gauche, estimée par l'échocardiographie, a diminué de façon transitoire dans la période suivant immédiatement l'électrochoc.

ELECTROCONVULSIVE therapy (ECT) is used for treating patients with psychiatric depression.[1] However, there have been many reports describing cardiac morbidity associated with ECT.[1] The cardiac morbidity of ECT is due to arrhythmias and instability of arterial blood pressure resulting in myocardial infarction and cardiac arrest.[1] Therefore, it would be important to assess the cardiac function during ECT.

There are two echocardiographic reports describing the regional wall motion abnormalities (RWMA) during ECT.[2–3] However, there has been no echocardiographic report describing the time course of cardiac function change during ECT.

Recently, an echocardiographic automated border detection system has been developed, which provides on line, beat-to-beat values of left ventricular cavity area.[4] These measurements can then be used to estimate load-dependent parameters of systolic performance such as fractional area change or ejection fraction.

The purpose of this study is to assess the time course of left ventricular systolic performance during ECT, using an echocardiographic automated border detection system.

Methods

After the approval of our institutional review committee and informed consent, nine ASA I or II patients scheduled for ECT were studied. Two men and seven women

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were studied (age 51 ± 8 yr; range 15–77, height 156 ± 2 cm; range 149–164, weight 47 ± 7 kg; range 31–60, values are mean ± SEM). All patients received their usual chronic medications (such as antidepressants) in the morning before the ECT. No patient received beta-adrenoceptor antagonists, or calcium-channel blocking drugs. All patients received 0.1 $mg \cdot kg^{-1}$ atropine sulfate *im* 30 min prior to ECT procedure.

Systolic and diastolic blood pressure, heart rate, arterial oxygen saturation measured by pulse oximeter and ECG were recorded continuously before ECT until the end of the procedure.

After the administration of propofol 1 $mg \cdot kg^{-1}$ over ten seconds and loss of consciousness, succinylcholine 1 $mg \cdot kg^{-1}$ was administrated, and assisted mask ventilation with 100% oxygen. Within two to three minutes after propofol injection, bilateral ECT was performed.

The transthoracic echocardiograph (Hewlett Packard SONOS 5500®, 3.5 MHz transducer, Andover, MA, USA) used in this study, had automated border detection system (Acoustic Quantification® system), which could trace the endocardial border continuously, compute the cross-section area of the left ventricular cavity, measure the R-R intervals from the corresponding displayed electrocardiographic tracing.[4]

Left ventricular parasternal short-axis images were recorded at the mid-papillary muscle level for measurements. While viewing the real-time images, the investigator activated automated border detection and adjusted the time gain compensation controls of the ultrasonograph to facilitate the automatic tracking of the highlighted indicator on to the echocardiographic image of the left ventricular endocardial border. Using the track ball of the ultrasonograph, the investigator outlined a region of interest that contained the left ventricular cavity throughout the cardiac cycle, excluding other cardiac chambers. The areas of left ventricle (LV) were then calculated from each cardiac cycle and displayed in a waveform. All studies were performed by the same operator (Y.K.) to eliminate interobserver variability and improve reproducibility. To minimize intraobserver variability, we recorded the acoustic quantification on video tape and re-assessed the validity of the gain setting. As all patients in this study were lean, we could observe LV cavity clearly in all cases.

The following left ventricular indexes were measured for each patient: (1) end-systolic area (ESA), (2) end- diastolic area (EDA), (3) fractional area change (FAC); FAC=(EDA-ESA) / EDA X 100.

According to Mulier *et al.*,[6] we used the quotient of systolic blood pressure and (EDA-ESA) as an index of left ventricular afterload. Data are expressed as means ± SEM.

After confirmation of equal variance among the group by the Bartlett test, changes in the variables were compared with analysis of variance for repeated measures with a P value <0.05 considered statistically significant. Post-hoc testing was performed using Scheffe's test.

Results

There were no significant differences in EDA, heart rate and the quotient of systolic blood pressure and (EDA- ESA) (Table). Increased ESA and decreased FAC were observed at one minute after the electrical shock compared to the awake condition. Systolic blood pressure was increased from one to four minutes after the electrical shock, compared to the awake condition. Diastolic blood pressure was increased from one to three minutes after the electrical shock, compared to the awake condition.

The Figure shows the relationship between the changes in FAC and systolic blood pressure. There was a relative correlation between the changes in FAC and

TABLE Time course for changes of hemodynamic variables

Measurement time	*(1)*	*(2)*	*(3)*	*(4)*	*(5)*	*(6)*	*(7)*	*(8)*
End-diastolic area (cm^2)	7.5 ± 0.8	7.8 ± 0.8	9.3 ± 0.9	8.2 ± 0.7	8.4 ± 0.9	8.8 ± 0.6	8.9 ± 0.8	7.8 ± 0.7
Endsystolic area (cm^2)	3.1 ± 0.3	3.5 ± 0.4	5.5 ± 0.6*	4.7 ± 0.4	4.1 ± 0.6	4.0 ± 0.4	3.7 ± 0.4	3.4 ± 0.4
Fractional area change (%)	58 ± 5	55 ± 4	44 ± 4*	49 ± 5	52 ± 5	56 ± 4	60 ± 5	56 ± 4
Heart rate (beat·min)	92 ± 5	91 ± 6	106 ± 7	104 ± 7	99 ± 5	97 ± 4	96 ± 5	97 ± 4
Systolic BP (mmHg)	112 ± 4	108 ± 4	147 ± 10*	145 ± 6*	144 ± 7*	137 ± 6*	131 ± 6	121 ± 4
Diastolic BP (mmHg)	61 ± 4	63 ± 3	81 ± 8*	87 ± 8*	80 ± 8*	80 ± 5	73 ± 4	65 ± 3
Systolic BP / EDA-ESA	47 ± 17	48 ± 10	62 ± 11	57 ± 9	57 ± 11	55 ± 17	49 ± 13	48 ± 12

Values are mean ± SEM

EDA=end-diastolic area, ESA=end-systolic area, HR=heart rate, BP=blood pressure

(1), awake; (2), one minute after the propofol administration; (3), one minute after electrical shock; (4), two minutes after electrical shock; (5), three minutes after electrical shock; (6), four minutes after electrical shock; (7), five minutes after elecrical shock; (8), ten minutes after electrical shock

*P <0.05 compared to period (1)

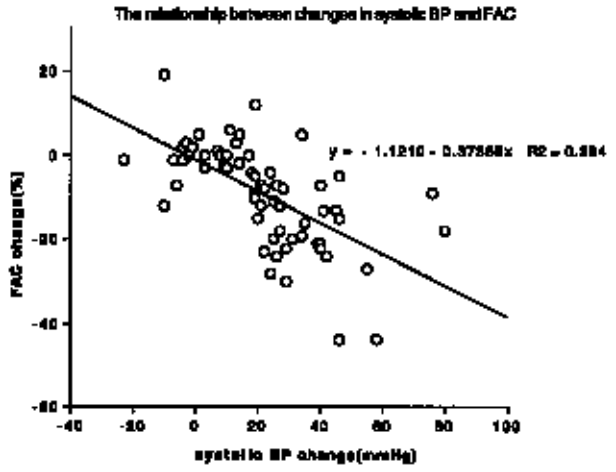

FIGURE The Figure shows the relationship between the changes in fractional area change (FAC) and systolic blood pressure (BP). There was a relative correlation between the changes in FAC and systolic BP (r^2=0.384).

systolic blood pressure (r^2=0.384). No RWMA or ST segment changes were observed in all patients both at baseline condition and during the ECT procedure.

Discussion

This decreased systolic performance observed in this study might be attributable to sympathetic hyperactivity which induces tachycardia, an increase in systemic vascular resistance and hypertension at this period. ECT provokes a rise in plasma catecholamines, and induces a considerable increase in myocardial oxygen demand.[1] This increased myocardial oxygen demand is likely to increase the risk of myocardial ischemia in patients with coronary artery disease. The decreased systolic performance observed in the present study might worsen with myocardial ischemia after ECT.

In this study we used propofol at a dose of 1 $mg \cdot kg^{-1}$ as a hypnotic drug. Fredman *et al.*[5] reported that propofol at a dose of 0.75 $mg \cdot kg^{-1}$ improved hemodynamic stability after ECT. In contrast, Mulier *et al.*[6] reported that propofol at a dose of 1.4 $mg \cdot kg^{-1}$ reduced systolic arterial blood pressure mainly through its negative inotropic properties. This discrepancy might be attributable to the difference in a propofol dose and in premedical drugs such as beta-adrenoceptor antagonists or atropine.[7]

The decrease in FAC observed at one minute after the ECT would be derived from two mechanisms. Both administration of propofol at a dose of 1 $mg \cdot kg^{-1}$ and increased systolic blood pressure shown in the Figure could induce a decrease in myocardial contractility.

There have been two reports describing RWMA during ECT. Messina *et al.*[2] reported a 45% incidence of new RWMA immediately after ECT. In contrast, O'Connor *et al.*[3] reported that new RWMA were seen in only one of 26 (4%) ECT sessions. They also reported that patients without RWMA at baseline had no new RWMA after ECT. In the present study, we did not examine all segments of the LV because we focussed on systolic performance during ECT. However, we could not detect any RWMA in the section we observed. This partial result is consistent with O'Connor's report. A more extensive study will be necessary to determine whether new RWMA could be observed after ECT.

Although we realize that automated border detection system might underestimate or overestimate EDA and ESA, an echocardiographic automated border detection system is a promising non-invasive assessment of cardiac performance.[8] And, many investigators have also observed that accurate, real time measures of left ventricular area dimension can be obtained with echocardiographic automated border detection system.[9]

In summary, we found that systolic performance of the LV, estimated by echocardiography is decreased transiently in the immediate period after ECT.

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