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Bigeminus During Electroconvulsive Therapy Resolves Spontaneously

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Abstract

Electroconvulsive therapy (ECT) is a frequently used and effective therapy for patients suffering from affective disorders. ECT is known as a relative safe treatment in depressed patients even with pre-existing disorders of the cardiovascular system. However, ECT can induce significant hemodynamic changes, like increases in heart rate and blood pressure as well as cardiac arrhythmias. We describe the occurrence of a non-sustained, spontaneously resolving bigeminus immediately after administrations of ECT in a 62-year-old female patient with an episode of major depression and no specific cardiac history ((German J Psychiatry 2004; 7(3) 45-48).

Keywords: electroconvulsive therapy, ECT, arrhythmia, asystole, bigeminus

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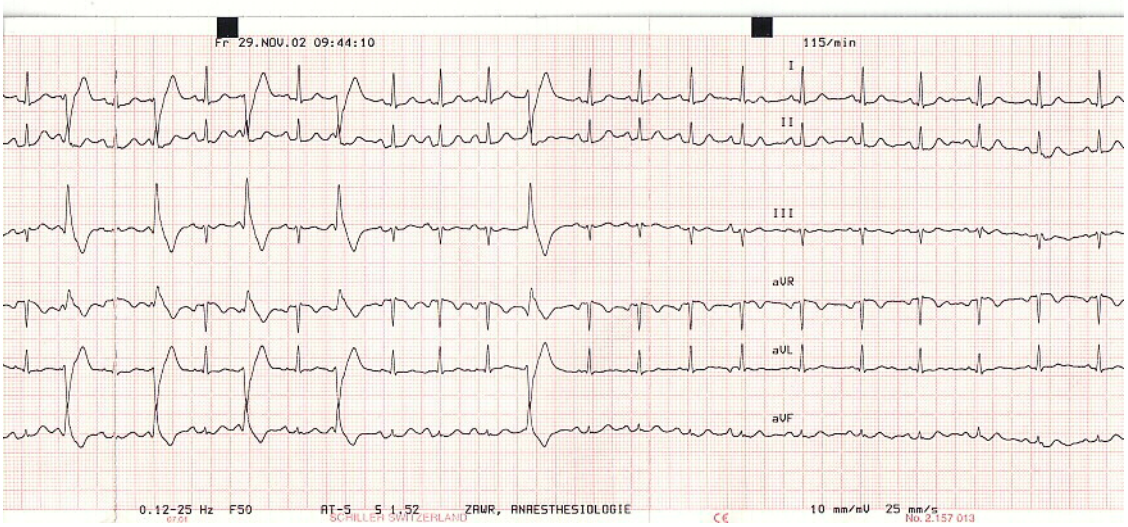
Introduction

Electroconvulsive therapy (ECT) is a safe and effective treatment modality for depression (UK ECT Review Group, 2003) even in patients with comorbid medical conditions such as coronary artery disease (Christopher, 2003; Cockey and Conti, 1995). Several complications can occur during ECT. Cardiac arrhythmias and changes of the electrocardiogram (ECG) have been described during and after ECT (Beale et al., 1994b; Steiner et al., 1993; Swartz and Manly, 1999). In California 35 cardiac arrests during ECT were reported between 1974 and 1983 (Kramer, 1985). ECT is associated with cardiovascular effects from parasympathetic and sympathetic stimulation, increased cerebral blood flow and oxygen consumption (Gaines and Rees, 1986; Knos et al., 1990), and increased intracranial (Gaines and Rees, 1986; Lovett Doust and Raschka, 1975), intraocular (Epstein et al., 1975; Gaines and Rees, 1986) and intragastric pressures (Gaines and Rees, 1986; Hurwitz, 1974). Hemodynamic changes occurring during electrically induced seizure activity are commonly attributed to adrenergic stress response (Fu et al. 1998).

We report a case of non-sustained bigeminus in the third ECT-treatment session of a patient without obvious pre-existing cardiovascular abnormalities.

Figure 1. ECG, 80 Seconds After ECT Stimulus: Spontaneous Conversion From Bigeminus Into Sinus Rhythm



Case Report

A 62-year-old Caucasian female (162cm height, 61 kg body weight) with a 46-year history of bipolar affective disorder, currently suffering from a major depressive episode, was admitted because of catatonia after long-time neuroleptic medication. She was previously successfully treated with ECT at the age of 32. Other medical diagnoses included hypothyreosis and non-insulin dependent diabetes mellitus. There were no cardiovascular abnormalities before admission to hospital. Medication at hospitalization was: bromocriptin 2.5 mg 3x/d, levothyroxin 0.025 mg 1x/d, lorazepam 1 mg 2x/d, and gliclazid 30 mg 1x/d. There were vesicular breath sounds on auscultation; respiratory rate was 20 breaths per minute. Cardiac examination was unremarkable. Blood pressure (BP) at admission was 110/60 mmHg, with a heart rate (HR) of 100 beats/minute. Initial ECG and chest radiograph were normal. Laboratory values on admission were unremarkable with the exception of thyroid stimulating hormone (TSH) level <0.03 mU/l (norm: 0.35-4.50 mU/l). Free triiodothyronine (FT3) and free thyroxin (fT4) were normal: 5.2 pmol/l (norm: 2.9-6.5 pmol/l) and 22.6 pmol/l (norm: 9.5-25.0 pmol/l), respectively.

The clinical state deteriorated during her hospitalization. Extrapyramidal symptoms developed (hypertonia, bradykinesia, resting tremor) as well as tachycardia and arterial hypertension. She was transferred to the intensive care unit for 6 days and after stabilization, to the regular medical unit with metoprolol 10 mg t.i.d. as additional medication.

This patient was scheduled for another series of ECT treatments. Standard monitoring procedures for ECT included the measure of BP, pulse oxymetry and ECG. After preoxygenation for 5 minutes anesthesia was induced with alfentanil (0.5 mg) and etomidate (0.3 mg/kg body weight). Succinylcholine 1mg/kg body weight was used for muscle relaxation. Oxygen was administered by a facemask. A psychiatrist induced the seizures using a MECTA Spectrum 5000 ECT stimulator (unilateral fronto-vertical stimulation of the non dominant hemisphere). By performing a dose finding algorithm (Beale et al., 1994a), a stimulation energy of 44 J resulted in a tonic-clonic seizure of 38 seconds duration.

Two days later, the patient arrived for the second treatment as planned. BP and HR were 110/55 mmHg and 80 beats/minute (sinus rhythm), respectively. Oxygen saturation before ECT was 96%. Immediately after the stimulation the patient developed a bradycardia of 40 beats per minute for 40 seconds. The bradycardia resolved spontaneously within 2 minutes. Heart rate recovered to 75 bpm. Recovery from anesthesia was uneventful. Before the next scheduled treatment, two days later, HR was 52 beats/minute and BP 105/55 mmHg. In addition to the routine ECG-monitoring we recorded the ECG before anesthesia, after induction and during the entire time, during and 3 minutes after ECT-treatment. Anesthesia and ECT were performed as before. Immediately after ECT we documented a bigeminus with a HR of 213 bpm lasting 20 seconds. BP was 155/80 mmHg. One minute later the HR was 129 bpm and decreased without intervention. The bigeminus converted spontaneously to a stable sinus rhythm after 80 seconds (see Figure 1). The patient recovered from anesthesia without any further events and was transferred to the ward after 45 minutes of close hemodynamic monitoring with stable cardiovascular parameters.

Postinterventional evaluation demonstrated normal creatine kinase, troponin and serum electrolytes. A 24 hour Holter monitoring of ECG documented a normofrequent sinus rhythm with frequent single ventricular beats (maximum 43 per hour), one couplet, AV-block I°, one episode of bradycardia with missing P-wave, and two episodes of asymptomatic AV-block II°. A transthoracic echocardiography showed normal left-ventricular function with an ejection

fraction of 70%, diastolic dysfunction and clinically insignificant aortic and mitral valve insufficiencies.

The patient was scheduled again for an ECT treatment three days later. No electrocardiographic changes were observed during following treatments. The depressive illness had improved markedly after this cycle of ECT. However, 6 weeks later there was a relapse requiring low frequency maintenance ECT once every four weeks for one year and maintenance treatment with venlafaxine 75 mg per day.

Discussion

Our report illustrates one potential risk related to electroconvulsive treatment: the induction of disturbances of the cardiac rhythm.

The anesthesiological management of patients for ECT is generally uncomplicated. However, not only anesthetic drugs, but also ECT itself can be associated with severe side effects. As a result of etomidate's reduced cardiovascular depressant properties and ability to enhance seizure activity, the acute hemodynamic response to ECT is accentuated compared with the barbiturates and propofol (Ding and White, 2002). The use of the rapid-acting, short duration muscle relaxant succinylcholine can cause severe bradycardia from increased parasympathetic autonomic nervous system outflow (Gabrielli et al., 2002).

Although very rarely, ECT itself is associated with cardiovascular mortality (Partridge et al., 1991). A survey estimated an overall mortality rate from the ECT treatment of 0,002% (Abrams, 1997). A mortality rate of 3-4 deaths per 10.000 patients treated was demonstrated (Crowe, 1984; Fink, 1985). A large scale study of 22.210 ECT treatments in Denmark found one death (Heshe and Roeder, 1976). In comparison, the mortality rate under general anesthesia approaches less than 1 per 10.000 anesthetics (Kubota et al., 1994). However, most importantly the mortality of inadequately treated depressed patients is more than double that of depression treated adequately with antidepressant medication and ECT (Avery and Winokur, 1976), and ECT is probably more effective than drug therapy (UK ECT Review Group, 2003).

When cardiac complications occur during ECT, they are mostly due to stimulation of the autonomic nervous system (Knos et al., 1990). During the first 10 to 15 seconds after electrical discharge (during the tonic phase of the seizure), there is a marked dominance of the parasympathetic system with subsequent, often marked, bradycardia, occasional asystole and a resultant fall in BP which is followed by sympathetic stimulation (Gaines and Rees; 1986; Gerring and Shields; 1982; Selvin, 1987; Steiner et al., 1993; Tang and Ungvari, 2001). Within 10 to 12 seconds of the sympathetic surge, caused by epinephrine and norepinephrine release, sinus tachycardia and arterial hypertension may develop (Anton et al., 1977; Gaines and Rees; 1986; Jones and Knight, 1981; Selvin, 1987; Steiner et al., 1993). Plasma epinephrine increases to 15 times normal levels, and plasma norepinephrine peaks can become 3 time higher than under normal resting conditions, with peak levels occurring within 60 seconds of electrical stimulation. Concentration of epinephrine decrease towards normal values 10 minutes after ECT, and norepinephrine levels remain increased for twice as long (Gaines and Rees; 1986; Jones and Knight, 1981; Selvin, 1987). These hemodynamic changes produce an abrupt increase in myocardial oxygen consumption. It is common practice to administer a short acting beta-blocker or a mixed alpha-beta-blocker to blunt the catecholamine stress response (McCall et al., 1991).

In our patient, the short bradycardia corresponded with the immediately parasympathetic response after ECT. Short phases of asystole occur rarely and bradycardia frequently resolves spontaneously before therapeutic intervention (Tang and Ungvari, 2001). Despite the treatment with metoprolol of our patient a tachycardic bigeminus occurred during sympathetic surge. Since the BP remained stable and the HR decreased continuously, we decided that no intervention was necessary. Esmolol (a beta-blocker with a rapid onset and brief duration of action) could be used in such situations. A similar case has recently been reported. After the development of intermittent bigemini over a period of 30 seconds after conclusion of seizure, a normal sinus rhythm established. Two minutes later this patient suddenly became asystolic but and was successfully resuscitated (Gabrielli et al., 2002). We suggest that in our case, despite of normal laboratory markers a transient myocardial ischemia caused by increased myocardial oxygen consumption after ECT seizure was the cause of bigeminus.

In conclusion, life-threatening multifocal premature ventricular beats, bigemini, and trigemini can occur during the sympathetic surge after ECT (Huuhka et al., 2003). These are serious arrhythmias that warrant a treatment with beta-blocking drugs when persisting. Any risk, which may be associated with ECT, must however be weighed against the – often underestimated - morbidity and mortality of untreated treatment resistant depression.

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