

Asystole in Electroconvulsive Therapy: Report of Four Cases

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Background: Asystole is an uncommon but potentially fatal complication of electroconvulsive therapy (ECT). Although the risk of asystole can be reduced with anticholinergic medications, the recent emphasis on new modifications of technique (first, the use of subconvulsive stimuli to titrate the seizure threshold, and second, pretreatment with intravenous β -blockers) may increase the risk of asystole in ECT patients.

Method: I present four new cases of asystole in ECT and outline a scheme for anticipating and preventing asystole.

Results: An episode of asystole did not prove to be an obstacle to further uncomplicated ECT.

Conclusion: If risk factors contributing to asystole are reduced and adequate doses of intravenous atropine are on hand, a patient's ECT treatments need not be interrupted.

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Electroconvulsive therapy (ECT) is an effective treatment for depression and is associated with a low mortality of approximately 3 deaths per 10,000 patients.¹ Cardiovascular complications are cited as the leading cause of death in ECT.¹ In the early days of treatment, the death rate was considerably higher, up to 0.8% in some series,¹ perhaps related to the lack of modern modifications including muscle relaxation, positive pressure ventilation, and use of anticholinergic medications. Continuous electrocardiogram (ECG) monitoring was not routinely available during the early development of the treatment; hence, many early deaths could only be ascribed to *cardiac arrest*,^{2,3} a nonspecific term encompassing pulseless ventricular tachycardia, ventricular fibrillation, and electromechanical dissociation.⁴

The advent of routine continuous monitoring of the ECG during ECT clarified that asystole was indeed a cause of cardiac arrest in ECT. Correspondingly, the 1978

American Psychiatric Association (APA) Task Force on ECT included anticholinergic pretreatment as part of the suggested routine to guard against bradycardia.³ Now, however, there is a swing away from routine use of anticholinergics,⁵ as reflected in the report of the 1990 APA Task Force on ECT⁶ that recommended an individualized approach regarding pretreatment: anticholinergic pretreatment is specifically indicated only for patients receiving sympathetic blocking agents.

Contemporary reviews of the cardiovascular effects of ECT have emphasized hypertension and tachycardia, instead of asystole, as the principal stress during ECT.⁷ The focus on these hyperdynamic responses during ECT may be related to the availability of new parenteral sympathetic blocking agents such as labetalol⁸ and esmolol.⁹ In comparison, relatively little has been written recently regarding the risk for asystole and bradycardia during ECT.

Asystole is defined in textbooks on electrocardiography as a period of electrical silence of unspecified duration between QRS complexes.^{4,10,11} Strictly speaking, the 1-second interval between ventricular contractions in a patient with a sinus rhythm of 60 beats/minute would represent a 1-second asystole. This definition is too broad to be of any clinical significance; yet, this appears to be the definition used in some articles describing ECG responses in ECT.^{12,13} At the other extreme, some authors argue that asystole up to 15 seconds should not be viewed as pathologic during ECT.¹⁴ Clinical usage of the term *asystole* usually conveys pathologic significance, and in this paper, I will define asystole as a period of ECG electrical silence lasting at least 10 seconds. This definition is admittedly arbitrary, but it emphasizes the infrequent occurrence of asystole since an interval of this duration is rarely recorded during most ECT sessions.

Herein I present four new cases of asystole during ECT. Continuous monitoring of the EEG was performed during each procedure with the MECTA SR1 brief pulse device (MECTA Corporation, Portland, Ore.). The ECG was continuously monitored throughout the induction, stimulus, and poststimulus period on a Spacelabs ECG oscilloscope (Spacelabs, Seattle, Wash.), and in addition, the ECG was recorded after the stimulus on the strip chart of the MECTA SR1 device. A MEDLINE search of the English-language literature since 1965 that used the key words *ECT* and *asystole* revealed four additional cases of asystole that lasted 10 seconds or more. From these

sources, I compiled a list of probable risk factors for asystole in ECT and suggest ways to manage this risk.

CASE REPORTS

Case 1

Ms. A, a 55-year-old, 70-kg black woman chronically hospitalized with medication-resistant DSM-IV-defined¹⁵ schizoaffective disorder, depressive type, was treated with ECT for her paranoia, dysphoria, and anhedonia. Her medical history was unremarkable. She enjoyed a good response to her first course of ECT, but unfortunately she relapsed within 1 month. Prior to her first course of ECT, Ms. A was noted to have a 12-lead ECG and an at rest ECG rhythm within normal limits, but during the tachycardia phase of the ECT seizure, she developed a wide-complex sinus rhythm interpreted as rate-dependent bundle-branch block. Her relapse after the initial successful course of ECT led to a recommendation for another course of treatment.

Fluphenazine was the only medication prescribed to Ms. A at the time of the first ECT of the second course. She was premedicated with 0.2 mg of intramuscular glycopyrrolate 1 hour prior to the treatment. Anesthesia included 70 mg of intravenous methohexital and 90 mg of intravenous succinylcholine. The electrical stimulus was delivered with bifrontotemporal electrode placement. Stimulus dose titration that used subconvulsive stimuli was planned for this first treatment as a means of defining her convulsive threshold. Ms. A's heart rate was 107 beats/minute and blood pressure was 140/80 mm Hg just prior to induction of anesthesia. The initial stimulus parameters were 1.0 msec pulse width, 40 Hz frequency, 0.5 sec stimulus train duration, and 0.8 A current. This first stimulus was subconvulsive and was immediately followed by a progressive slowing of Ms. A's heart rate, which led to a 30-second asystole. The asystole resolved after atropine 0.75 mg i.v. was administered, and sinus rhythm returned to greater than 90 beats/minute. Although further stimuli were not attempted at this treatment session, subsequent ECT treatments using fully convulsive stimuli and 0.4 mg of intravenous atropine pretreatment were completed without asystole or bradycardia.

Case 2

Mr. B, a 45-year-old, 96-kg white man, was admitted with DSM-IV-defined bipolar disorder, manic phase, consisting of florid excitement with intrusiveness, irritability, clang and loose associations, and pan-insomnia unresponsive to medications. His medical history was otherwise unremarkable, and a 12-lead ECG was within normal limits. Bifrontotemporal ECT was initiated after withdrawal of valproic acid. Each of the first 12 treatments, all bifrontotemporal, was fully convulsive, and the cardiovascular responses were unremarkable despite the omis-

sion of anticholinergic medication. At the 13th bifrontotemporal ECT treatment, Mr. B's heart rate was 110 beats/minute after infusion of his routine 60 mg of methohexital, but transiently slowed to 30 beats/minute immediately after infusion of the routine dose of succinylcholine 100 mg. Mr. B received atropine 0.4 mg intravenously before his 14th bifrontotemporal ECT session, in response to the slowing noted at the prior treatment. His heart rate was 139 beats/minute immediately after methohexital 60 mg was administered. Infusion of succinylcholine 100 mg was followed by the usual spread of muscle fasciculations from the neck into the chest, but this time the patient experienced a 10-second asystole *before* the stimulus. The asystole resolved before any intervention could take effect. The patient was subsequently treated at that session without further difficulty, and four more uncomplicated ECT treatments were given after intravenous pretreatment with 0.8 mg of atropine.

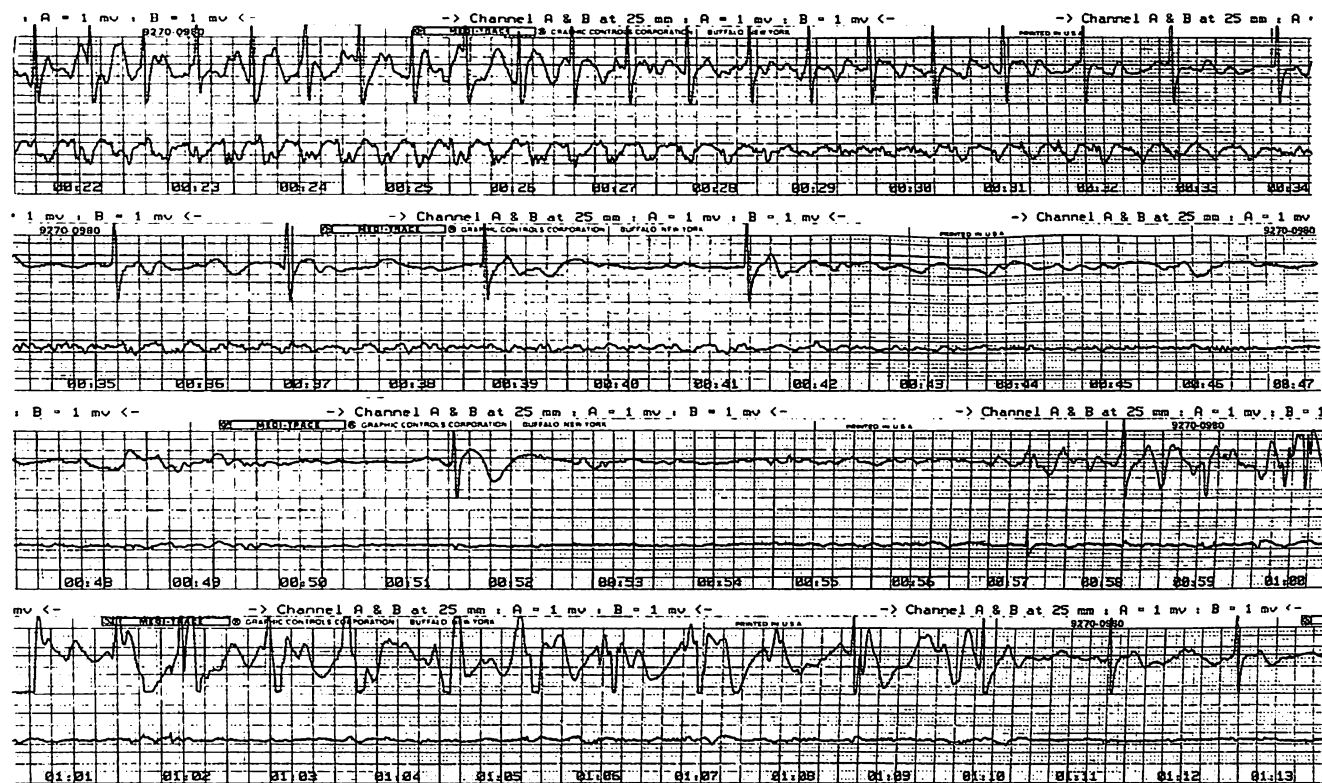
Case 3

Mr. C, a 71-year-old, 83-kg white man, was admitted with total anhedonia, social withdrawal, insomnia, paranoid ideas, and nihilism consistent with DSM-IV-defined major depression, recurrent, severe with psychotic features. Prior episodes had responded well to ECT, and ECT was again recommended for his depression. His prior medical history was remarkable only for non-insulin-dependent diabetes mellitus, controlled with glyburide. The first three treatments were uncomplicated and included glycopyrrolate 0.1 mg intramuscularly and methohexital 80 mg and succinylcholine 100 mg intravenously. Bifrontotemporal electrode placement was employed, and fully convulsive stimuli were achieved on each of the first three treatments. Thirty milligrams of the ultra-short-acting β -blocker esmolol was given intravenously before the stimulus to control blood pressure during each of the first three ECT treatments. Medications given during the fourth bifrontotemporal ECT treatment were identical to those given in prior sessions except that only 20 mg of esmolol was used. Mr. C's heart rate was 101 beats/minute prior to induction of anesthesia and throughout the 50-second EEG seizure. A 10-second asystole immediately followed the seizure (Figure 1). The asystole spontaneously resolved before any intervention could take effect, and 1 minute after the end of the seizure, Mr. C's heart rate rose to 60 beats/minute. Mr. C subsequently received six more uncomplicated bifrontotemporal ECT treatments after intravenous pretreatment with 0.4 mg of atropine and omission of esmolol.

Case 4

Ms. D, a 77-year-old, 80-kg white woman, was admitted with DSM-IV-defined major depression, single episode, severe, comprising a 2-month history of depressed mood, insomnia, and poor appetite unresponsive to medi-

Figure 1. Asystole in Mr. C at the Conclusion of His ECT Seizure*



*Upper channel = ECG; lower channel = EEG. Time elapsed is in seconds. Recording segments are continuous.

cation. Her medical history included diabetes mellitus that was treated with insulin and hypertension treated with lisinopril. Her ECG revealed first degree A-V block and a possible remote septal infarct. All of her ECT treatments were rendered with right unilateral electrode placement. During the first three uncomplicated treatments, Ms. D received glycopyrrolate 0.1 mg intramuscularly one hour before ECT and 60 mg of both methohexital and succinylcholine intravenously. At the fourth ECT session, Ms. D received 0.2 mg of intravenous atropine with the routine dose of anesthetic and relaxant. The dose of relaxant was judged to be insufficient; therefore, an additional 20 mg of succinylcholine was given before the stimulus. Finally, Ms. D received 30 mg of esmolol just prior to the stimulus. Her heart rate was 70 beats/minute before induction and rose to 100 beats/minute during the 61-second seizure. Ms. D experienced a 10-second asystole immediately after termination of the seizure. The asystole resolved spontaneously before any intervention could be made. Ms. D subsequently received five more uncomplicated right unilateral ECT sessions accompanied by 0.4 mg of i.v. atropine, and β -blockers were avoided.

DISCUSSION

The details of these cases invite the following general comments. First, asystole can occur during a course of ECT even if the patient has had several prior uncomplicated treatments in which essentially the same technique had been used (Cases 2 and 3). Second, asystole can occur at multiple junctures of the procedure including during induction of muscle relaxation (Case 2), immediately after a subconvulsive stimulus (Case 1), and in the immediate postictal period (Cases 3 and 4). Correspondingly, the ECT treatment team must remain vigilant throughout the entire procedure. Third, an episode of asystole need not interrupt the treatment course; all of our cases went on to receive further uncomplicated treatment. Indeed, Mr. B received ECT within minutes after experiencing his succinylcholine-related asystole.

Succinylcholine-related asystole has been described in other procedures,^{16,17} but Mr. B may represent a unique instance reported in ECT. Multiple, closely spaced boluses of succinylcholine may increase the risk of asystole and may have played a role in the asystole experienced by Ms. D.¹⁶

Table 1. Characteristics of Eight Patients With ECT-Related Asystole of 10 or More Seconds

Case	Inadequate Anticholinergic ^a	β -Blocker	Subconvulsive	Bilateral Electrode Placement	Preexisting Conduction Defect	Continued ECT
Wulfson et al, 1984 ¹⁸	Yes	No	Yes	Yes	No	Yes
Wells et al, 1988 ¹⁹	No	No	Yes	Not specified	Yes	Yes
Liebowitz and El-Mallakh, 1993 ²⁰	Yes	Yes	No	Yes	No	Not specified
Kaufman, 1994 ²¹	Yes	Yes	No	Yes	No	Yes
Ms. A	No	No	Yes	Yes	Yes	Yes
Mr. B	No	No	No	Prestimulus asystole	No	Yes
Mr. C	Yes	Yes	No	Yes	No	Yes
Ms. D	No	Yes	No	No	Yes	Yes

^aAs per APA Task Force Guidelines, 1990.⁶

Other possible contributing factors for asystole in these cases include subconvulsive stimuli (Case 1), doses of anticholinergic agents smaller than those recommended by the APA Task Force (Case 3),⁶ concurrent use of β -blockers (Cases 3 and 4), preexisting ECG conduction deficits (Cases 1 and 4), and bifrontotemporal electrode placement (Cases 1, 2, and 3). I found four other instances of ECT-related asystole of 10 seconds or longer, and the possible contributing factors in these cases are summarized along with my cases in Table 1.¹⁸⁻²¹ Inspection of Table 1 reveals that, with the exception of Mr. B, two or more possible contributing factors were present in each instance. I speculate that contributing factors, in a variety of combinations, may be additive in their risk for asystole. The relative contribution of each factor at present is unknown.

The role of anticholinergics^{12,22,23} and β -blockers²⁴ in ECT hemodynamics has been described elsewhere and will not be reviewed here. The roles of subconvulsive stimuli and bilateral electrode placement, however, merit some discussion. Subconvulsive stimuli are an inherent part of defining the convulsive threshold with the stimulus titration technique.²⁵ Although usually well tolerated with right unilateral electrode placement, subconvulsive stimuli reliably produce a transient slowing of the patient's heart rate, presumably by direct entrainment of the vagus nerve.²⁶ Large numbers of ECT practitioners have adopted stimulus titration techniques.²⁷ Whether the slowing of heart rate could be exaggerated with bilateral as opposed to right unilateral subconvulsive stimuli is unknown. Bilateral electrode placement is in many respects a more profound treatment than right unilateral ECT as manifested in its more reliable antidepressant effect^{28,29} and its greater tendency for producing confusion,³⁰ as well as greater synchronization and amplitude of the ictal EEG³¹ and greater release of prolactin associated with bilateral placement.³² The greater physiologic effects of bilateral ECT may be related to a more diffuse intracranial radiation of electrical current compared with a narrower band of current with right unilateral,³³ and the more diffuse radiation of current with bilateral ECT may be more

likely to influence the hypothalamic control of the vagus nerve. Usually, slowing of the heart rate in the immediate postictal period is *not* as great with bilateral as right unilateral ECT,³⁴ but I speculate that in some instances the greater generalized cerebral effects of bilateral ECT could lead to a greater degree of postictal bradycardia when other contributing factors listed in Table 1 are present.

Some authors have taken a benign view of asystole,¹⁴ and I agree that ICU admissions are not necessarily indicated after an asystole. In fact, all of my cases tolerated asystole without residual complications. Still, I believe that these relatively short periods of asystole are influenced by the same factors that could eventually result in a fatal asystole. Hence, asystole should continue to be taken seriously.

In closing, ECT practitioners should be vigilant for asystole at multiple stages of the ECT session, regardless of how many successful prior treatments a patient may have received when identical technique was used. The tone of the pulse oximeter provides an ideal means for continuous auditory monitoring of heart rate while the ECT practitioner is engaged in other tasks during the procedure. The cumulative number of contributing factors listed in Table 1 should be limited whenever possible. Atropine should always be immediately available for intravenous injection. Intramuscular glycopyrrolate may be an excellent sialagogue, but intravenous atropine provides superior protection against bradycardia.²³ Finally, asystole does not imply the need to interrupt the course of treatment if subsequent treatments are managed with larger doses of intravenous atropine and/or reduction of other contributing risk factors.

Drug names: esmolol (Brevibloc), fluphenazine (Prolixin), glyburide (Diabeta, Micronase), glycopyrrolate (Robinul), labetalol (Normodyne, Trandate), lisinopril (Prinivil), methohexital (Brevital sodium), succinylcholine (Anectine and others), valproic acid (Depakene and others).

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Supplement 2

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