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Case Reports

ECT-Induced Asystole from a Sub-Convulsive Shock

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Key Words: TREATMENT: therapy, electroconvulsive COMPLICATIONS: arrhythmia, asystole

Electroconvulsive therapy (ECT) is a safe treatment process in which the risk entailed is said to be generally no greater than that associated with the use of short-acting barbiturates[1-4]. Opinions differ as to the relative safety of electroconvulsive therapy in debilitated patients or in those with severe myocardial dysfunction. Gerring and Shields[5] identified a group of patients at high risk for the development of cardiovascular complications, namely myocardial ischaemia and/or arrhythmias following electroconvulsive therapy. In this group, which included patients with a history of angina, myocardial infarction, congestive heart failure, arrhythmias, rheumatic heart disease or an abnormal baseline electrocardiogram, the complication rate was 70%. However, Dec *et al.*[2] found electroconvulsive therapy to be safe, effective and well tolerated in a group of elderly debilitated patients, one-quarter of whom had severe cardiovascular disease including poor ejection fractions and recent myocardial infarctions.

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We report here a case of a patient with severe underlying cardiac disease in whom the administration of a sub-convulsive shock resulted in asystole of approximately fifteen seconds' duration on one occasion and marked slowing of the heart rate on a subsequent occasion.

CASE REPORT

A 60 kg, 75-year-old male was admitted to hospital for treatment of severe depression, being the third such episode within the previous six months. A series of eight electroconvulsive therapy (ECT) treatments had been administered elsewhere at the time of his initial diagnosis. Since then, his condition had proved refractory to treatment with both tricyclic antidepressants and monoamine oxidase inhibitors.

The patient had a 30-year history of hypertension, for which treatment with nifedipine had recently been discontinued. Twelve months previously he had suffered an inferior myocardial infarction. On admission to the ward an ECG showed the presence of an old inferior infarct, first degree atrioventricular block, frequent premature ventricular contractions, right bundle branch block and very poor R wave progression across the precordial chest leads. A radionucleide ventriculogram demonstrated the left ventricular ejection fraction to be only 22%. On examination the blood pressure was

145/90 mmHg, pulse irregular at 80 beats/minute, and jugular venous pressure elevated 4 cm. The chest was clear to auscultation. In view of the severity of his psychiatric condition he was felt to be at extreme risk of suicide. After consultation between the anaesthetist, cardiologist and psychiatrist, it was agreed to proceed with a second course of ECT.

On the first occasion the patient was premedicated with nifedipine 10 mg sublingually 20 minutes prior to treatment in order to attenuate any hypertensive response. A 16 gauge intravenous line was inserted and monitoring devices including a blood pressure cuff, precordial stethoscope, ECG (lead CM5) and EEG attached. Atropine 0.4 mg was administered intravenously. Following pre-oxygenation, anaesthesia was induced with etomidate 12 mg and succinylcholine 40 mg. Both pulse and blood pressure remained stable at pre-induction levels (BP 130/85 mmHg, pulse 80 beats/minute). Therapy was to be delivered from the MECTA instrument, which delivers a constant current, bi-directional square wave brief pulse stimulus. The convulsive parameters selected were pulse frequency 40 Hz, pulse width 1.5 msec and pulse duration 2 sec.

With the application of electro-shock the patient failed to convulse and suddenly became asystolic. A precordial thump was administered with no effect. Atropine 0.8 mg and ephedrine 15 mg, already at hand, were then administered intravenously. After approximately fifteen seconds, which included a brief period of external cardiac massage, normal sinus rhythm returned.

Two days later a successful treatment was performed. On this occasion the patient was premedicated with atropine 0.6 mg subcutaneously one hour prior to treatment and once again received nifedipine 10 mg sublingually pre-therapy. Immediately before treatment he received atropine 1.6 mg intravenously in incremental doses which increased his pulse rate from 65 to 85 beats/minute. After induction of anaesthesia with etomidate 15 mg and succinylcholine 50 mg, two stronger electro-shocks (pulse frequency 60 Hz) were delivered back to back. The patient sustained a modified generalised grand mal convulsion for a period of 50 seconds during which his heart rate increased to 105 beats/minute.

Prior to his third treatment the patient received atropine 0.6 mg subcutaneously, nifedipine 10 mg sublingually, and atropine 1.2 mg IV. On this occasion he failed to convulse with the application of the electro-shock, but there were no changes in either pulse rate or rythm.

The fourth treatment was delivered utilizing an older electro-shock apparatus which delivers a sinewave current. The patient was premedicated with atropine 0.6 mg subcutaneously. On arrival in the treatment room, he was given nifedipine 10 mg sublingually followed 20 minutes later by atropine 2.0 mg intravenously in incremental doses. Once again, he failed to convulse. With the application of the electro-shock there was a marked slowing in pulse rate from 96 beats/minute to 42 beats/minute.

Subsequent treatments performed under ketamine anaesthesia with atropine administered one hour before treatment in a dose of 0.6 mg subcutaneously, nifedipine 10 mg sublingually 20 minutes before treatment and intravenous atropine in a dose range of 1.4-2.0 mg intravenously immediately prior to treatment produced convulsions without bradyarrhythmias.

DISCUSSION

While several hundred deaths have been reported over the years in association with ECT,[6-10] most of these occured during the 1950s. There was no clear direct mortality in several recent large series which together totalled over 35,700 treatments.[11-14]

Nevertheless, isolated cases of death or severe cardiovascular complications continue to appear.[15,16] In 1977, the American Psychiatric Association concluded that cardiac arrest is the leading reported cause of death associated with ECT.[17]

The case report here demonstrates that profound autonomic responses may occur in response to ECT. The usual course of events is that the electro-shock stimulus induces a period of vagal dominance, with consequent bradycardia and perhaps a transient fall in blood pressure. This period of vagal activity may also be marked by asystole of a few

seconds' duration, atrial premature contractions, atrial fibrillation and atrial flutter,[18,19] and lasts for the duration of the tonic phase of the fit. Most patients then proceed to a subsequent clonic phase during which there is central sympathetic discharge, accompanied by a rise in circulating catecholamines.[19-21] In addition to hypertension and tachycardia[22] this sympathetic dominance may produce multifocal premature ventricular contractions and, rarely, ventricular tachycardia.[18,19]

The outstanding feature of the present case is that asystole was induced in a patient who failed to achieve a grand mal convulsion. On a subsequent occasion there was a marked slowing in pulse rate with the delivery of a subconvulsive shock in the presence of a previously administered large dose of atropine. Experimental work in animals shows that ECT-induced asystole can be prolonged in the presence of a high spinal anaesthetic, which prevents the peripheral release of catecholamines.[21] A case of asystole in association with the administration of propranolol 1.0 mg intravenously followed by a subconvulsive shock has also been reported.[23] The failure to proceed from the tonic (parasympathetic) phase to the clonic (sympathetic) phase of a convulsion leaves parasympthetic discharge unopposed. Presumably this places a patient with myocardial conduction abnormalities at greater risk of developing severe bradyarrhythmias.

While we cannot exclude the possibility of the prior administration of nifedipine provoking asystole, we feel this to be unlikely. The previous course of therapy was conducted in the presence of nifedipine therapy of several months' duration. Nifedipine is largely a vasodilator and exhibits no depressant effect on conduction. Its administration frequently results in a net reflex tachycardia.[24] Nevertheless it is possible that the heart may, in the presence of acute doses of nifedipine, be susceptible to asystole initiated by parasympathetic preponderance.

In conclusion, we feel that when administering ECT to patients with severe myocardial dysfunction it is most important that a convulsion follows the electro-shock. The failure to achieve a convulsion places these patients at greater risk of the development of vagal arrhythmias, including asystole. The administration of larger than normal doses of atropine prior to treatment seems to afford some degree of protection against the development of these arrhythmias, but cannot by itself be totally relied upon.

REFERENCES

1. Concensus Conference. Electroconvulsive therapy. JAMA 1985; 254:2103-2108.
2. Dec GW, Stern TA, Welch T. The effects of electroconvulsive therapy on serial electrocardiogram and serum cardiac enzyme values. JAMA 1985; 253:2525-2529.
3. Holden C. A guarded endorsement for shock therapy. Science 1985; 228:1510-1511.
4. Pippard J, Ellam L. Electroconvulsive treatment in Great Britain. Br J Psychiat 1981; 139:563-568.
5. Gerring JP, Shields HM. The identification and management of patients with a high risk of cardiac arrhythmias during modified ECT. J Clin Psychiat 1982; 43:140-3.
6. Impastato DJ. Prevention of fatalities in electroshock therapy. Dis Nerv Sys 1957; 18 (suppl):34-75.
7. Raskin N. Electric shock casualties. J Nerv Ment Dis 1958; 126:360-366.
8. Wyant GM, Macdonald WB. The role of atropine in electroconvulsive therapy. Anaesth Intens Care 1980; 8:445-450.
9. Maclay WS. Death in treatment. Proc Roy Soc Med 1953; 46:13-20.
10. Hussar E, Pachter M. Myocardial infarction and fatal coronary insufficiency during electroconvulsive therapy. JAMA 1961; 204:1004-1007.
11. McLeave DJ, Blakemore WB. Anaesthesia for electroconvulsive therapy. Anaesth Intens Care 1975; 3:250-256.
12. Pitts FN. Medical aspects of ECT. Sernin Pshychiatry 1972; 4:27-42.
13. Heshe J, Roder E. Electroconvulsive therapy in Denmark. Br J Psychiatry 1976; 128:241-245.
14. Turek IS, Hanlon TE. The effectiveness and safety of electroconvulsive therapy. J Nerv Ment Dis 1977; 164:419-425.
15. Marks RJ. Electroconvulsive therapy: physiological and anesthetic considerations. Can Anesth Soc J 1984; 31:541-548.
16. Gaines GY, Rees DI. Electroconvulsive therapy and anesthetic considerations. Anesth Analg 1986; 65:1345-1356.
17. American Psychiatric Association Task Force Report: Electroconvulsive therapy. Washington, D.C. American Psychiatric Association 1978.
18. Perrin GM. Cardiovascular and other physiologic changes accompanying EST. Acta Physiol Scand 1961; 36:10-45.

19. Pitts FN, Desmarias GM, Stewart W. Induction of anesthetic with methohexital and thiopental in electroconvulsive therapy. N Engl J Med 1965; 273:353-360.
20. Valentine M, Keddie KM, Durine D. Comparison, techniques in electroconvulsive therapy. Br J Psychiatry 1968; 114:201-206.
21. Anton AH, Wy DS, Redderson CL. Autonomic blockage and the cardiovascular and catecholamine responses to electroconvulsive therapy. Anesth Analg 1977; 56:46-54.
22. Egbert LD, Dumas PA, Ginter GC, Eckenhoff JE. Modification of the circulatory response to electrode therapy by thiopental. Anesthesiology 1959: 20:309-312.
23. Wulfson HD, Askanazi J, Finick AD. Propranolol prior to ECT associated with asystole. Anesthesiology 1984; 60:255-256.
24. Kapur PA. Pharmacology of anti-anginal drugs. 1987 Review Course Lectures, Int Anesth Res Soc. 1987; 59-64.

Achalasia and Anaesthesia, A Case Report

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Key Words: OESOPHAGUS: achalasia; ANAESTHESIA: complications

Achalasia of the oesophagus is a rare condition with a reported incidence of eight cases per 100,000 population.[1] It is characterised by symptoms related to oesophageal obstruction and respiratory complications secondary to aspiration of oesophageal contents.

We report a case of previously undiagnosed achalasia which had resulted in tracheal compression mimicking asthma which was treated by oesophagoscopy and hydrostatic dilatation under general anaesthesia.

CASE REPORT

An 18-year-old girl presented with a two-year history of wheeze and dyspnoea on exertion. A diagnosis of asthma was made and she was treated with oral bronchodilator therapy with no improvement in symptoms or peak expiratory flow rate.

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Her past medical history and physical examination were unremarkable: FEV_1 (2.6 litres), FVC (3.6 litres) and peak expiratory flow rate (225 litres per minute) were all decreased and unaffected by bronchodilators. A PA chest radiograph was reported as

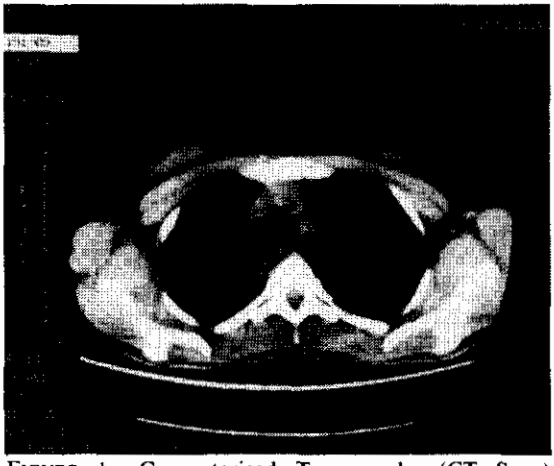

FIGURE 1.—Computerised Tomography (CT Scan) showing a grossly dilated oesophagus, with food residue within it, indenting the membranous trachea posteriorly.