Case Reports

ECT-Induced Asystole from a Sub-Convulsive Shock

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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. Opinions differ as to the relative safety of electroconvulsive therapy in debilitated patients or in those with severe myocardial dysfunction. Gerring and Shields 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. 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.

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 radionuclide ventriculogram demonstrated the left ventricular ejection fraction to be only 22%. On examination the blood pressure was...
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 rhythm.

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, most of these occurred during the 1950s. There was no clear direct mortality in several recent large series which together totalled over 35,700 treatments. Nevertheless, isolated cases of death or severe cardiovascular complications continue to appear. In 1977, the American Psychiatric Association concluded that cardiac arrest is the leading reported cause of death associated with ECT.

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, 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. In addition to hypertension and tachycardia this sympathetic dominance may produce multifocal premature ventricular contractions and, rarely, ventricular tachycardia.

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

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. 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.

Her past medical history and physical examination were unremarkable: FEV₁ (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](image-url)

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

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