Eur Arch Psychiatr Neurol Sci (1984) 234:147–148

European Archives of Psychiatry and Neurological Sciences

Electrocardiographic Changes Following Electroconvulsive Therapy

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Summary. This paper reports a study of the effects of ECT (electroconvulsive therapy) upon cardiac function (as judged by electrocardiographic changes) in 24 patients who were given 139 treatments in all. Of the 24 patients 13 were studied over a 24-h period using monitoring equipment. No potentially dangerous arrhythmias were encountered. Modified ECT was found to give rise to sinus tachycardia, which was considerably more prolonged than previously reported. The tachycardia was shown in some cases to be accompanied by a depression of the ST segment and may hence be potentially harmful to those with pre-existing ischaemic heart disease. In such patients consideration should be given to the prior administration of β-adrenergic blocking drugs and/or oxygen.

Key words: Electroconvulsive therapy – Electrocardiography – Tachycardia

Introduction

Electrocardiographic (ECG) changes due to electroconvulsive therapy (ECT) have been studied by many investigators (Bridenbaugh et al. 1972; Hansoti et al. 1973; McInnes and James 1972; McKenna et al. 1970; O'Melia 1970; Pitts 1972; Strian et al. 1977; Witzum et al. 1970). Although a variety of abnormalities have been reported, ECT has usually been regarded as a safe treatment except in those patients who have suffered from a recent episode of coronary thrombosis (Freeman 1979).

While Part I of the current study seeks to replicate the findings reported in the literature in which most of the patients studied were observed for only a short time, both before and after ECT, Part II reports the finding in 13 patients who were monitored continuously over a 24-h period, i.e. for 12h before and after ECT was administered.

Part I

Method

A total of 11 mental hospital in-patients (4 males, 7 females, mean age 57.5 ± 12.5 years; range 28–77 years; 9 Caucasians, 2 Asians) were studied while receiving 5 to 9 ECT treatments (76 in all). Their psychiatric diagnoses were: endogenous depression (7 cases); neurotic depression (2); schizophrenia (1); and schizo-affective disorder (1). A past history of coronary thrombosis was noted in 2 patients, and 1 still suffered from frequent attacks of anginal pain.

In all cases modified ECT was given in accordance with current practice. Thus prior to application of the electrical stimulus, 0.6mg atropine, together with 65mg methohexitone were given intravenously, followed by 35mg succinylcholine.

A 12-lead ECG tracing was recorded both before and 60min after ECT. Standard lead II was recorded continuously after the initial 12-lead ECG until 1min after ECT and again at 5, 15 and 30min for 60s on each occasion.

Results

All patients showed a significant increase in heart rate immediately after ECT ($P<0.05$ on the sign test); the mean rate being 96 beats/min (bpm) just before anaesthesia and 133 bpm, immediately after ECT (Fig. 1). In 10 of the 11 patients the heart rate had decreased by 15min and continued to fall until 60min had passed. Nonetheless, in 6 patients the 60min rate still exceeded the pre-anaesthetic rate and in only one instance was it lower than in the pre-anaesthetic rate.

Except for sinus tachycardia, 3 of the 11 patients showed rate-dependent ST segment depression. There was, however, no evidence of any residual effect after 60min.

Part II

Method

A total of 13 other mental hospital in-patients (6 males, 7 females, mean age 52.4 ± 17.7 years, range 24–87 years, 11

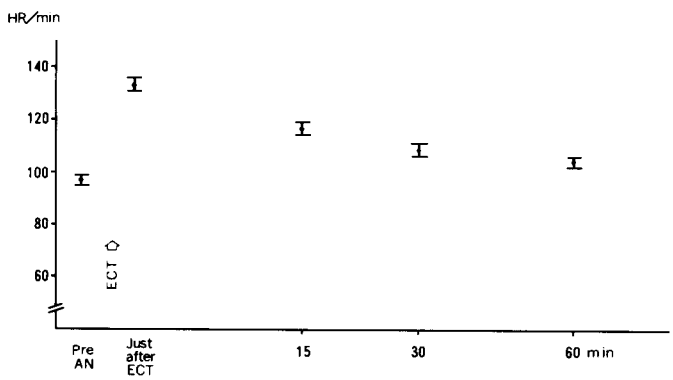

Fig. 1. Heart rates before and after ECT in Part I

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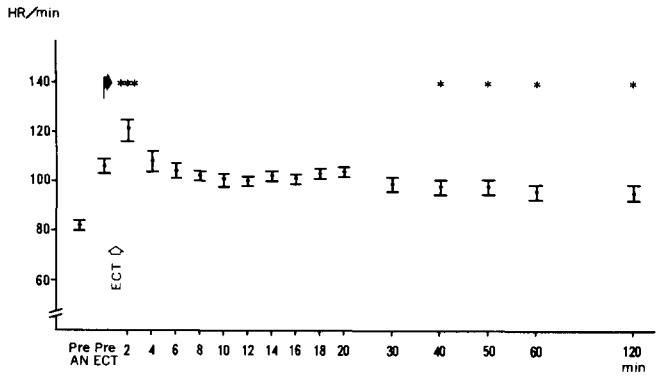

Fig. 2. Heart rates before and after ECT in Part II. The rate at 2 min is significantly ($P<0.001$) higher than the pre-ECT rate. Those at 40, 50, 60 and 120 min are significantly ($P<0.05$) lower than the pre-ECT rate

Caucasian, 2 Asians) were studied while receiving 1 to 8 ECT treatments (63 in all). Their psychiatric diagnoses were: endogenous depression (11), schizophrenia (1) and obsessional neurosis (1). None had a previous history of cardiac disease, although 2 were fibrillating at the time treatment was given. The same treatment procedure as described in Part I was used.

However, in this second group of patients the ECG was recorded continuously from 12 h before ECT was given until 12 h after treatment, using Oxford Instruments Holter monitoring equipment. The tapes were reviewed by one of us (A.J.F.P.) with respect to the presence and types of arrhythmias throughout the 24-h period of ECT monitoring. Heart rates were noted before anaesthesia, immediately before ECT was given, every 2 min for 20 min after ECT and again at 30, 40, 50, 60 and 120 min afterwards.

Interference and other technical mishaps resulted in only 37 of the 63 taps being suitable for complete analysis.

Results

Apart from the 2 patients who were in atrial and flutter fibrillation throughout, all other patients were in sinus rhythm. No instances of malignant ventricular arrhythmias (Lown and Wolf 1971) were detected at any stage either before or after ECT. However, the mean heart rate of the group increased significantly ($P<0.001$) from pre-anaesthesia (82 bpm) to pre-ECT (106 bpm) with a further significant increase ($P<0.001$) from pre-ECT to a peak rate at 2 min post-ECT (121 bpm) (Fig. 2). Thereafter the mean rates did not exceed the pre-ECT mean rate, although remaining highly significant ($P<0.0001$) in excess of the pre-anaesthetic rate at 120 min after ECT was given.

Although a considerable variation in peak rates (88 to 170 bpm) and in percentage rise between pre-anaesthetic and peak rates (21%–115%) was observed, there was relatively little variation found in individual patients ($P=0.1$ and 0.2 respectively, Friendman two-way analysis of variance).

Discussion

In this study the only important effect of modified ECT on cardiac rate and rhythm was a variable but highly significant increase in heart rate. We have shown that this tachycardia is produced in part by the drugs administered, but also that the ECT itself is coincidental with, and presumably produces, a significant and immediate further increase in heart rate. Although this further increase disappeared within 4 min after ECT, the rate remained highly significantly in excess of the pre-anaesthetic rate for more than 2 h. It may therefore be possible that the anaesthetic agents induced the sinus tachycardia. However, Witztum et al. (1976) found that the increase in heart rate following ECT could not be augmented by the increased dose of methohexitone. It seems plausible that both anaesthetics and ECT contributed to the rise in heart rate.

It seems inappropriate to draw any further conclusion from heart rate changes any later than 2 h post-ECT as the patients were by then fully mobile. Even so the effects of ECT on the heart rate in this study were considerably more prolonged than in those previously reported (Rollason et al. 1971).

Although the increases in rate immediately after ECT would not be sufficient to embarrass myocardial oxygen supply in a normal heart, the ST segment depression observed in some cases suggests that an ECT induced tachycardia might be potentially harmful in the presence of coronary artery disease. In such circumstances consideration should, therefore, be given to the simultaneous administration of a β-adrenergic blocker and/or oxygen.

Despite this, in these two studies ECT was found to be a safe procedure in psychiatric practice.

Acknowledgement. This project was supported by a research grant of the West Midlands Regional Health Authority. The authors wish to thank Professor Sir William Trethowan for his supervision, and Dr. N.W. Imlah, Medical Director, and all consultant psychiatrists of All Saints Hospital, Birmingham, for giving permission for access to their patients. Thanks are also due to Dr. U. Smith, Consultant Anaesthetist, and the staff of ECT section of All Saints Hospital.

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Received February 24, 1984