Atropine premedication and the cardiovascular response to electroconvulsive therapy

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Summary
A report by the Royal College of Psychiatrists recommended avoiding atropine premedication during electroconvulsive therapy (ECT). We have examined the cardiovascular effects of ECT with or without atropine premedication. Consenting patients \( n = 30 \) were allocated randomly before their third ECT session to receive atropine or no premedication. The rate pressure product (RPP) was recorded before anaesthesia, before ECT stimulus and at 1-min intervals thereafter for 5 min. Patients who did not receive atropine had significantly lower RPP values after all stimulus recordings. Administration of atropine or not explained 32\% of the variance of summated RPP after the stimulus. There was no clinically significant bradyarrhythmia in those who did not receive atropine. Our findings support the recommendation of the Royal College of Psychiatrists. The study suggests that when threshold determination is not needed, avoiding atropine effectively contains potentially harmful cardiovascular responses, \((\text{Br. J. Anaesth.} 1998; 81: 466–467)\).

Keywords: premedication, atropine; brain, electroconvulsive therapy; cardiovascular system, responses

Cardiovascular changes occur consistently during electroconvulsive therapy (ECT). Acute increases, albeit transient, occur in heart rate and arterial pressure, and hence rate pressure product (RPP), an index of myocardial oxygen consumption.\(^1\) During modified ECT, a reduction in arterial oxygen saturation can occur even after adequate ventilation. The increase in RPP during ECT can create an imbalance between myocardial oxygen supply and demand. Treatment variables, such as ECT laterality (bilateral vs unilateral) and anaesthetic agent (thiopental vs methohexital) do not differentially reduce heart rate or arterial pressure after ECT.\(^2\) Stimulus laterality may not offer a means of controlling cardiovascular responses during ECT. Other strategies aimed at reduction of heart rate and arterial pressure include pretreatment with nifedipine, labetolol or esmolol. Although these methods are effective in the attenuation of the cardiovascular response, they are usually reserved for patients with compromised cardiac function.

Atropine premedication produces anticholinergic-mediated tachycardia. This effect, in addition to the intense sympathetic response after the ECT stimulus, can potentially contribute to greater myocardial work load. In contemporary ECT practice, subconvulsive stimuli used in stimulus titration, increases the risk of bradycardias.\(^3\) However, there may be advantages in withholding atropine in subsequent ECT sessions where subconvulsive stimulation is absent. There are reports that atropine could compromise cardiac stability\(^4\) and for this reason current guidelines from the Royal College of Psychiatry recommend avoiding atropine during ECT.\(^5\) We have examined the effect of atropine on RPP in a randomized, controlled design during the third ECT session.

Methods and results
We studied 30 consecutive right-handed patients (mean age 27.3 (range 15–50) yr; 15 males) referred for ECT after obtaining written informed consent. Patients continued to receive concurrent psychotropic drugs before the ECT session; these were neuroleptics \((n = 23)\), tricyclic antidepressants \((n = 5)\) and trihexyphenidyl \((n = 6)\). All patients were ASA grade I. During the third ECT session, patients were allocated randomly to one of two equal groups to receive atropine premedication or no atropine.

Modification of the ECT procedure was achieved using atropine 0.15 \(\mu\)g kg\(^{-1}\) (if given), thiopental 3 mg kg\(^{-1}\) and succinylcholine 1 mg kg\(^{-1}\) i.v., in that order. The psychiatrist and anaesthetist were blinded to the treatment groups. Intermittent positive pressure ventilation with 100\% oxygen was provided until resumption of spontaneous and regular breathing. ECT was administered using a constant current bidirectional brief pulse ECT device (mean stimulus dose 154 (SD 50.2) mC). The choice of ECT stimulus laterality was determined by the referring psychiatrist. In the third ECT session, patients received either threshold bilateral \((n = 18)\) or suprathreshold (twice the threshold) right unilateral \((n = 12)\) ECT; all developed a seizure in response to the first stimulus in the session. Seizure duration was monitored using an EEG from F\(_3\) and F\(_4\) sites referenced to ipsilateral mastoids (mean duration 65.5 (SD 38.1) s). Cardiovascular monitoring was performed using an

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Automated cardiac monitor (pulse oximetry, non-invasive arterial pressure (AP), ECG lead II tracing and heart rate). The product of heart rate and corresponding systolic AP was RPP. Cardiovascular recordings were made before anaesthesia (just before administration of the premedication), before stimulus application (45 s after injecting the premedication and just before stimulus application) and five times at 1-min intervals after the stimulus.

For statistical analysis we used the software package SPSS (v. 6.0). RPP values in the two groups were compared using two-way repeated measures analysis of variance (RMANOVA). Summated RPP (arithmetic sum of RPP at the five recordings after the stimulus) was subjected to linear regression using age, sex, atropine status, stimulus laterality, stimulus dose, EEG seizure duration and pre-anaesthesia RPP as the independent variables. Significance (a) was fixed at 5% or lower.

The two groups were comparable in stimulus dose and laterality, in addition to seizure duration. Significant increases in RPP occurred during the procedure (occasion effect: $F = 9.7; df = 168, 6; P < 0.001$). The increases in RPP were smaller in the group that did not receive atropine (group effect: $F = 10.5; df = 28, 1; P = 0.003$) (fig. 1).

In the multivariate, stepwise, linear regression model, only atropine premedication emerged as a significant predictor ($B = 14343; t = 3.6; P = 0.001$) accounting for 32% of the variance ($r^2$) in the summated RPP after the stimulus.

There were no clinically significant cardiac arrhythmias in either group.

Comment

Routine atropine premedication during ECT has been recommended by Abrams who argued that the risk-benefit analysis favours the use of anticholinergic agents. This is contrary to the the Royal College of Psychiatry's recommendation of avoiding atropine premedication during ECT.

We found that mean RPP was attenuated when atropine premedication was withheld. Linear regression analysis showed that atropine alone substantially contributed to RPP after the stimulus. Withholding atropine did not affect seizure duration or produce bradycardia. An advantage of our study is that automated cardiac monitoring eliminated rater bias.

Sustained increases in RPP in patients given atropine increase myocardial workload and oxygen demand. Withholding atropine demonstrably controlled RPP without compromising cardiac stability. Therefore, routine administration of atropine in ECT sessions not involving threshold determinations in ASA I patients is not recommended. However, caution is advised in extrapolating our results to older patients and those who are not ASA I.

References