SHORT REPORT

Heart rate differences between right and left unilateral electroconvulsive therapy

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Abstract
Left and right unilateral electrode placements were alternately applied in electroconvulsive therapy given to 21 men with melancholia. Accompanying heart rate elevations were greater following right unilateral treatment than left unilateral, apparently because of longer persistence of peak rates. This is consistent with right cerebral hemisphere superiority in the control of heart rate activity in neurologically intact humans.

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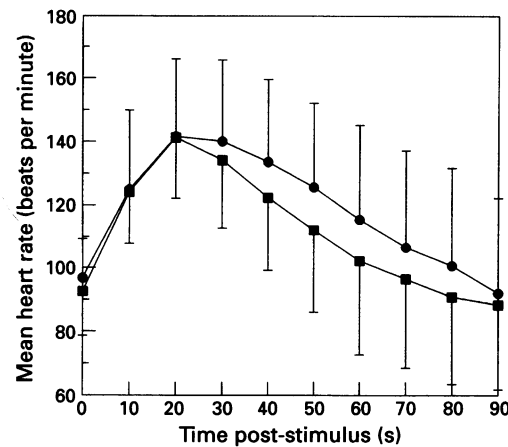R D Lane
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In a previous comparison of heart rate changes with bilateral and right unilateral electroconvulsive therapy (ECT)[1] we found greater postictal heart rates with bilateral ECT, but no differences in the magnitude or persistence of the peak heart rate. Evidence for lateralisation of control of sympathetic nervous outflow to the heart[2-4] implies that differences between the hemispheres may have contributed. This led us to compare differences in heart rates associated with left and right unilateral ECT.

Method
We studied 21 male veterans consecutively referred for ECT in the routine course of treatment for melancholia, who had no history or evidence of neurological or cardiovascular disease, and who had a resting heart rate below 120 beats per minute measured under anaesthesia immediately before ECT. This limit was set in recognition of the cardioacceleratory effects of the anaesthetic agents, and the need to allow a potential for heart rate increase. Informed consent was obtained. Because of systematic variations that tend to be largest between the first and second ECTs, the first ECT was excluded from study and given with bilateral electrode placement. The second and third treatments were administered alternately with left and right unilateral temporal-paravertex electrode placements,[1] according to a randomised, balanced design. Anaesthesia was induced with 0·0044 mg/kg (limit 0·4 mg) glycopyrronium, 1·0 mg/kg methohexitone, and 0·5 mg/kg succinylcholine. Identical anaesthetic doses were used

for both study conditions. Seizures were induced with a brief pulse instrument set for 378 mC charge (0·9 A, 1 ms pulse width, 140 pulses per second for 3 s), excepting one patient with a high threshold who received 504 mC (same stimulus for 4 s).

Electrocardiograph monitor paper recordings were examined blind to treatment electrode placement for the following measures, relative to termination of ECT stimulus: prestimulus heart rate under anaesthesia, peak heart rate, time to maximum heart rate, three-beat average heart rate centred at each of nine 10-second intervals, and minimum heart rate during these intervals. The duration of seizure was measured as the time between stopping the ECT stimulus and the ending of tonic and clonic motor activity including a limb occluded by cuff from succinylcholine.[1] The duration was at least 22 s in every trial.

ECT anaesthesia typically lasted for five to 10 minutes, so that all patients were fully unconscious during the period of measurement. Anaesthesia was conducted in the same manner in both study sessions.

Results
Seizure duration was not significantly different (p = 0·33), between left (54·5 (SD 21·7) s) and right (61·6 (SD 34·9) s) unilateral ECT; one patient (192 s) generated this apparent, but non-significant, difference. The figure displays the heart rates measured. Paired t tests revealed no significant differences in baseline heart rate (p = 0·073), peak heart rate elevation over baseline (p = 0·50), or time to reach peak heart rate (p = 0·11). Repeated measures of analysis of variance on heart rate elevation (above baseline) revealed a significant variation over time of left-right differences (p = 0·035)—that is, a significant interaction between electrode placement and time. It also showed the expected significant main effect of time (p < 10^{-6}), that corresponds to the increase in heart rate induced by seizure.

Consistent with the straightforward waxing and waning of a right-sided cardioacceleratory influence, the percentage of patients showing greater heart rate elevation with right than left unilateral ECT in each 10 s interval rose steadily from 47·6% in the first interval

to a maximum of 71·4% in the fourth interval, and then fell continuously to 33·3% in the ninth interval. The probability is 0·0273 that random variations would generate this pattern of a single peak within nine consecutive epochs, which is the ratio of seven possible patterns with one peak to 2^8 total possible patterns of change from one epoch to the next. There was no substantial difference in minimum heart rates observed between right and left unilateral ECT (average 0·6 per minute).

Discussion

Significantly more persistent or greater heart rate elevation accompanied, and was more common with, right unilateral ECT than left unilateral ECT. The figure indicates that the heart rate decelerations were essentially the same with both treatments. This parallelism suggests that the difference can be attributed to longer persistence of peak heart rate with right unilateral ECT, although other explanations are possible. As in our previous study,[1] electrode placement did not affect peak heart rate, which therefore again appears to be at a physiological maximum for response to induced seizures. These results presumably mirror sympathetic neural stimulation of the heart, as muscarinic blockade should have essentially removed vagal influences.

Although the heart rate difference between left and right unilateral ECT occurred during the seizure, the bilateral to right difference occurred postictally, apparently after conclusion of all seizure-induced neural effects. This suggests different causes. The observed greater persistence of heart rate elevation with right unilateral ECT is consistent with the predominantly right sided mechanisms that mediate sympathetic cardioacceleration with electrical stimulation of the brainstem.[2 3] Further, the cardioacceleratory sinoatrial node of the heart is innervated by a greater number of sympathetic fibres from the right side of the brain than from the left.[5]

Bilateral ECT presumably generates greater postictal catecholamine levels than unilateral ECT, in view of corresponding differences in postictal prolactin levels,[6] and circulating catecholamines might have con-

tributed to the observed postictal heart rate differences.[1] Nevertheless, heart rate elevations can persist for several minutes post-seizure after bilateral adrenalectomy.[7] Thus the greater heart rate elevation with bilateral than right unilateral ECT may have followed the greater right hemisphere stimulation associated with the former.[8]

Neurally stimulated cardioacceleration persists about a minute after the stimulation ends,[2] consistent with the observed timing of heart rate differences between right and left unilateral ECT. There is a close correspondence between conclusion of ECT-induced cardioacceleration and termination of brain electrical seizure activity,[9] when metabolic and EEG measured electrical activity abruptly drop by about 82%, to approximately a third of baseline.[10] The results are also consistent with the greater cardioacceleratory responses seen with right than left insular (subtemporal) stimulation seen in humans during neurosurgery.[11]

The elevated metabolic, EEG, and neurochemical components of ECT seizure activity suggest intense widespread physiologically propagated stimulation.

The measured consequences are presumably a summation of the effects of stimulations at the many brain sites[2 3] that can affect cardiac activity. Differences between right and left unilateral ECT correspond to incomplete seizure generalisation through the brain[9] to the opposite hemisphere. With both neural and endocrine mechanisms, our present results suggest that electrode placement lateralises brainstem stimulation as well as cortical stimulation—that is, right unilateral ECT stimulates the right brainstem more than left unilateral ECT. Cases where there is no difference between hemispheres may result from particularly effective generalisation to the opposite hemisphere.

The possibility that observed right–left heart rate differences were generated by longer seizure activity with right unilateral ECT is not supported by the observation that the length of cuffed limb seizures was essentially the same for the two treatments. Cessation of cuffed limb movement coincides with the end of seizure activity in the motor strip and correlates highly with EEG measurements of total seizure endpoint.[9]

Seizures presumably also elevate heart rate during and after the seizure by raising levels of circulating catecholamines. Combined concentrations of adrenaline and noradrenaline in the blood after the ECT seizure[12] tend to exceed 1800 pg/ml, the minimum required to induce cardioacceleration.[13] Left-right brain differences in the control of catecholamine release are unknown.

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