

Modification by atropine and practolol of changes in heart rate evoked by cutaneous nerve stimulation

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Norman & Whitwam (1973) demonstrated that the increase in heart rate evoked by stimulation of the radial nerve contains a vagal component which is dependent on the initial heart rate and blood pressure. Fussey, Kidd & Whitwam (1969) showed that stimulation of cutaneous nerves evokes activity in cardiac sympathetic nerves and Peiss & Manning (1964) observed increases in heart rate when peripheral nerves were stimulated in vagotomized animals.

The present study was designed to demonstrate both vagal and sympathetic mechanisms in evoked heart rate responses. Eight dogs, anaesthetized with thiopentone and chloralose, were artificially ventilated and paralysed with suxamethonium. A cutaneous branch of the left radial nerve was exposed, desheathed, cut distally and immersed in mineral oil. The vagus nerves were exposed in the neck and placed on thermodes. Femoral arterial pressure, an electrocardiogram and heart rate measured beat-by-beat from the R-R interval were recorded. The changes in heart rate and blood pressure evoked by trains of stimuli applied to the radial nerve (intensity 40 V, pulse duration 0.5 ms, frequency 20 Hz, duration 5 or 30 s) were observed; in intact preparations these changes were often transient and tended to return towards control values during continuing stimulation of the afferent nerve.

On cooling the vagus nerves to 1°C there was an increase in the mean resting heart rate from 125

to 148 beats/min. Stimulation of the radial nerve then produced a further average increase in heart rate of 28.6 beats/min (129 observations, range 4-80 beats/min) beginning on average in 1.76 s of the onset of stimulation. The maximum heart rate occurred on average in 6.1 s. In four preparations given atropine (0.5 mg/kg i.v.) stimulation of the radial nerve evoked a mean increase in heart rate of 19.1 beats/min (37 observations, range 3-46 beats/min) beginning on average in 1.9 s of the onset of stimulation and reaching a peak within 8.5 s. In three preparations given practolol (2 mg/kg i.v.) stimulation of the radial nerve evoked a mean rise in heart rate of 18.4 beats/min (34 observations, range 0-42 beats/min) beginning on average in 1.4 s and reaching a maximum in 3.5 s. A fourth dog given practolol showed no evoked response in heart rate. The evoked increase in heart rate seen after either atropine or practolol was abolished when both drugs were administered.

This study confirms that there are vagal and sympathetic components in the heart rate response to cutaneous nerve stimulation which can be blocked selectively by atropine and practolol respectively.

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References

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