

LETTER TO THE EDITOR

INFLUENCE OF VAGAL TONE ON ADRENALINE-INDUCED VENTRICULAR TACHYCARDIA IN RATS AND GUINEA PIGS*

Sir,

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Vagal tone is important in the sensitivity of the myocardium to arrhythmogenic agents (1). We found that ventricular tachycardia (VT) due to benzene and adrenaline developed significantly earlier in guinea pigs than in rats, though its duration was same. We have examined the vagal tone in rats and guineapigs and its relationship with VT, induced by benzene and adrenaline.

VT was induced in Wistar rats and albino guinea pigs using the method described by Wegria and Nickerson (4). Rats (200–250 g) and guinea pigs (400–450 g) of either sex were anaesthetised with urethane (1.5–1.6 g/kg, ip). Positive pressure artificial respiration (45/min, at 1 ml/100 g) was maintained by a rodent respirator (Harvard apparatus-Model 681 A). The jugular vein was cannulated for drug administration. Standard lead II ECG was recorded on a Grass Polygraph (Model 7D) and heart rate was monitored from the 'R' wave of ECG. The animals were made to inhale benzene vapours for 2 min from a Woulfe's bottle connected to the inlet of the respirator. This was followed immediately by administration of L-adrenaline bitartrate (10 µg/kg, iv). Time for onset and duration of VT (sec.) were noted. In selected groups, vagosympathetic trunks were exteriorised and bilateral or left or right unilateral vagotomy was performed, 10 min before the induction of arrhythmia. Another set of animals was pre-treated with atropine sulphate (2 mg/kg, iv) 10 min before the induction of the arrhythmia. The results were analysed using Students 't' test.

The basal heart rate in rats was 335 ± 15 /min, whereas that in guinea pigs was 293 ± 8 /min. In rats onset of VT was significantly delayed in comparison to guinea pigs (Table I). However, there was no major difference in the duration of VT.

In rats, the bilateral vagotomy resulted in a significant decrease in the time for onset of VT, whereas the duration of VT was increased. In right vagotomised rats there was a decrease in the time for onset and an increase in the duration of VT but these changes were not statistically significant. However, left vagotomy had little effect on the

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TABLE 1: Effect of bilateral vagotomy (B.V.), left vagotomy (L.V.), right vagotomy (R.V.) and atropinisation (ATR) on the heart rate, time (sec) for onset and duration of ventricular tachycardia (VT) (Mean \pm SEM) induced by benzene and adrenaline in rats and guinea pigs. Parentheses indicate the number of animals in a group.

	Rat	Guinea pig
<i>Time for onset of VT</i>		
Control	10.26 \pm 1.19 (20)	4.20 \pm 1.12 (20) ^a
B.V.	5.94 \pm 1.31 (20)*	2.92 \pm 1.68 (20)
L.V.	11.88 \pm 3.13 (8)	5.00 \pm 3.61 (5)
R.V.	7.43 \pm 2.87 (6)	6.25 \pm 2.39 (4)
ATR	4.30 \pm 0.57 (6)***	4.17 \pm 1.54 (6)
<i>Duration of VT</i>		
Control	57.67 \pm 2.24 (20)	50.96 \pm 3.28 (20)
B.V.	79.38 \pm 8.17 (20)*	52.50 \pm 4.83 (20)
L.V.	33.75 \pm 5.88 (8)***	40.00 \pm 5.06 (5)
R.V.	62.86 \pm 6.43 (6)	43.75 \pm 8.00 (4)
ATR	92.30 \pm 3.50 (6)***	40.00 \pm 5.70 (6)
<i>Heart rate (% change)</i>		
Control	100.00 \pm 0.00 (20)	100.00 \pm 0.00 (20)
B.V.	114.57 \pm 2.49 (20)***	98.81 \pm 2.31 (20)
L.V.	95.51 \pm 1.23 (8)**	97.69 \pm 2.08 (5)
R.V.	107.61 \pm 2.06 (6)**	96.16 \pm 2.45 (5)
ATR	109.11 \pm 2.69 (6)***	95.09 \pm 1.24 (6)**

*P < 0.025;

**P < 0.005;

***P < 0.001 in comparison to control.

^aP < 0.001 in comparison to rat.

onset whereas it decreased the duration of VT. In contrast, in guinea pigs the time taken for onset as well as duration of VT were unaltered after bilateral or left or right vagotomy.

In rats bilateral and right vagotomy caused significant increase, whereas left vagotomy caused a significant decrease in the heart rate. However, in guinea pigs bilateral, left or right vagotomy failed to cause any significant change in the heart rate. The results in rats clearly show that right vagus has greater deceleratory influence on the heart than that of left vagus and this observation is in conformity with the previous reports (2, 3). No attempt was made in their work to explain why left vagotomy was followed by bradycardia in rats. There was no significant change in heart rate after unilateral or bilateral

vagotomy in guinea pigs suggesting a near absence of cardioinhibitory action of vagus. Singh *et al.* (3) also reported species difference in vagal tone, which was greater in dog and cat than in rabbit and guinea pig.

Vagal tone seems to have considerable influence on the development of VT. Atropinisation decreased the time for onset and increased the duration of VT in rats whereas both the parameters were unaffected in guinea pigs. In rats there was a significant increase in the heart rate after atropinisation but in guinea pigs there was a decrease in heart rate. It was concluded from the results that guinea pigs, which have poor vagal tone are more susceptible to VT than rats which exhibit considerable vagal tone. After abolition of vagal influence either by bilateral vagotomy or atropinisation, arrhythmia developed earlier and its duration was increased. The relative susceptibility of guinea pigs to benzene and adrenaline induced VT, thus may be due to the comparatively lower vagal tone.

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