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Innervation of domestic fowl and guinea-pig ventricles

SIR,—Marked changes in the force of contraction of driven ventricular strips occurred when the suprathreshold electrical stimulation used to drive the strips was increased tenfold.

A strip was cut from the wall of the right ventricle of a chick (domestic fowl) or guinea-pig heart, one end being anchored to bipolar platinum hook electrodes, and the other (apical) end to a transducer for isometric tension recording.

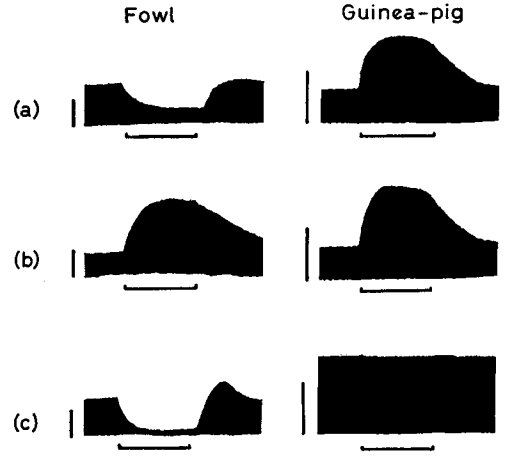

FIG. 1. Polygraph records of isometric tension of guinea-pig and chick ventricular strip preparations stimulated at constant rates. The stimulus strength was increased for periods of 2 min, indicated by the horizontal bars. Vertical calibrations 1 g. (*a*) Untreated preparations, (*b*) in the presence of 1 μg/ml atropine and (*c*) in the presence of 10 μg/ml guanethidine.

Strips were suspended in a solution of the following composition (g): NaCl 6·92, KCl 0·34, $CaCl_2$ 0·30, $MgCl_2$ 0·11, KH_2PO_4 0·16, $NaHCO_3$ 1·0, glucose 2·0, sucrose 4·5, water to 1 litre, and aerated vigorously with oxygen. The temperature was maintained at 41–42° for chick and 37–38° for guinea-pig ventricular strips, which were driven at constant rates of 4–6/sec with square wave pulses of 1–2 V and 5 msec duration.

Increasing the stimulation strength caused a decrease in the force of contraction of chick and an increase in the force of contraction of guinea-pig ventricular strips (Fig. 1a). Atropine, 1 μg/ml, had slight or no effect on the response of guinea-pig ventricular strips but in the chick the decrease was converted to a large increase in the force of contraction (Fig. 1b). Guanethidine, 2–10 μg/ml, abolished the increase in force of contraction in untreated guinea-pig strips but

the response was not converted to a depression. Guanethidine potentiated the depression observed in chick strips (Fig. 1c). In atropinised preparations from both species the increase in the force of contraction observed upon increasing the stimulus strength was abolished by the β-receptor blocking drug propranolol (0·1 μg/ml) and a slowly developing blockade was observed with the adrenergic neurone blocking drug guanethidine (2–10 μg/ml). The blockade produced by guanethidine was partially reversed by the addition of dexamphetamine, 1 μg/ml, to the bath.

Acetylcholine, 0·1 μg/ml, caused a marked depression of the force of contraction of chick ventricular strips—greater than the depression observed with 100 μg/ml of acetylcholine in guinea-pig ventricular strips. In the chick the depressions to both acetylcholine and increasing the stimulus strength could be abolished by atropine or hyoscine (1 μg/ml).

Triethylcholine, 250 μg/ml, which has been shown to compete with choline for the transport mechanisms involved in acetylcholine synthesis (Bowman & Hemsworth, 1965; Bull & Hemsworth, 1965), caused a slowly developing blockade of the depression observed upon increasing the stimulus strength driving chick ventricular strips. This blockade was reversed by the addition of choline (50 μg/ml) to the bath.

These results suggest that the decrease in the force of contraction observed upon increasing the stimulus strength driving chick ventricular strips is due to stimulation of cholinergic inhibitory fibres within the myocardium. Similarly the increase in force of contraction observed in untreated guinea-pig and atropinised chick ventricular strips is due to stimulation of adrenergic excitatory fibres within the myocardium. Thus the chick ventricle has both a parasympathetic and a sympathetic nerve supply while the guinea-pig has a sympathetic but little or no parasympathetic innervation. The latter finding is consistent with anatomical (Woollard, 1926; Nonidez, 1939) and physiological (Schreiner, Berglunde, Borst & Monroe, 1957; Sarnoff, Brockman, Gilmore, Linden & Mitchell, 1960; Vincenzi & West, 1963) evidence for the ventricles of other mammalian species.

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