INTRODUCTION

Rat ileum

In many organs with sympathetic innervation, nicotine and ACh produce sympathomimetic effects in the presence of atropine (Hoffman et al. 1945; Kottegoda, 1953 a, b.; Burn and Rand, 1960; Brandon and Rand 1961; Gillespie and MacKenna, 1960). These sympathomimetic effects of the two drugs appeared to be indistinguishable, being abolished following chronic sympathetic denervation or treatment with reserpine and it is generally thought that both nicotine and ACh act at the same site and in a similar manner to cause release of NA and thus to produce sympathomimetic effects. In fact, the action of ACh in releasing NA from the postganglionic sympathetic fibre has been described as 'nicotine like' (Burn and Rand, 1962).

In an earlier paper from this laboratory it was shown that ACh in the presence of hyoscine produced sympathomimetic effects (Gokhale et al. 1967). In the experiments to be described certain differences between the sympathomimetic

effects of ACh on the one hand and those of nicotine and DMPP on the other were observed. These differences suggest that while ACh acts directly to cause release of NA from peripheral store in the adrenergic terminals, nicotine and DMPP act by releasing ACh which in turn liberates NA.

Rabbit ileum

The majority of motor nerves have been shown to release ACh as the transmitter substance. This is true (a) of nerves to skeletal muscle (b) of parasympathetic nerves, both pre- and postganglionic, and (c) of preganglionic sympathetic nerves. The sympathetic postganglionic fibres, apart from those to the sweat glands, alone have NA as their main transmitter. That there may have been some evolutionary change is evident from the observations of Young (1936) who found that in two teleost fishes, Lophius piscatorius and Uranoscopus scaber, the splanchnic innervation of the intestine was motor. Burnstock (1958) showed that the same was true in the trout, another teleost fish. Since the motor effect was abolished by atropine, Burnstock (1958)

concluded that the innervation was cholinergic. A similar observation was made by Burn and Rand (1960) on the intestine of chicken.

Boatman et al. (1965) reported that stimulation of postganglionic sympathetic nerves to perfused hindleg vessels of newborn dog caused vasodilatation from 1 to 2 weeks. Burn (1968 a) made similar observation on newborn rabbit intestine by stimulating periarterial nerves. Although in the adult rabbit, the relaxation of the intestine caused by sympathetic nerve stimulation is due to release of NA, there is evidence that ACh is also released (Burn, 1968 b).

Burn argued (1968 b), "if it is true that there has been change in sympathetic function in the course of evolution and that the motor responses of the intestine seen in the trout and chicken have become changed to an inhibitory action in the rabbit, then, since ontogeny recapitulates phylogeny some sign should be seen in the newborn rabbit".

The present study confirms and extends Burn's (1968 a) observations. Further, an attempt was made to determine the time course required for the development of sympathetic nerves and of endogenous NA content in the intestine of newborn rabbit. The ability of the intestine of newborn rabbit to accumulate NA added exogenously was also investigated.