thus may constitute the first line of defence of the body to infection.

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Effect of Bromide Ions on Junctional Transmission

Muchnik and Gage[1] have recently suggested that bromides act as sedatives by reducing the amount of transmitter substances released at synapses in the central nervous system. The evidence in favour of this suggestion is that the replacement of external chloride by bromide ions reduces the quantal content of the end-plate potential (e.p.p.) of frog skeletal muscle fibres. This result, however, was obtained in experiments in which neuromuscular transmission was already blocked, and the quantal content of the e.p.p. greatly reduced, by the presence of magnesium ions and a subnormal concentration of calcium ions. It therefore seemed of interest to investigate the action of bromide ions on synaptic transmission in an otherwise normal ionic environment.

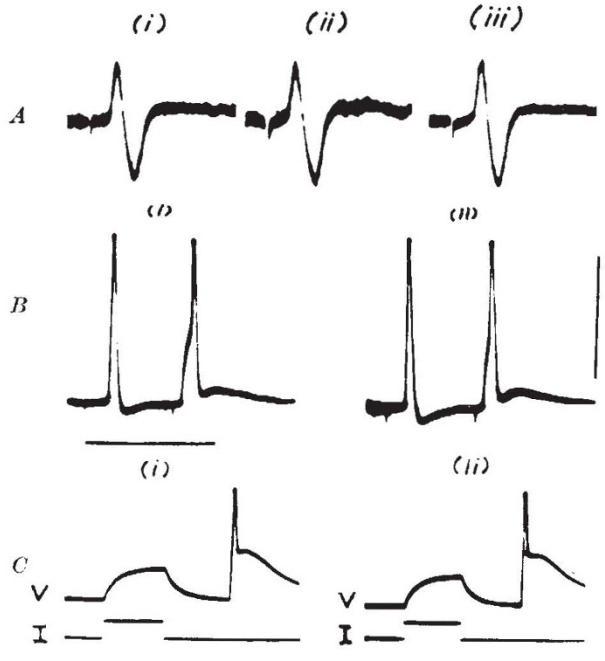

Fig. 1. *A*, Extracellular responses recorded from a postganglionic ramus of the tenth sympathetic ganglion in response to preganglionic stimulation. In (i) bath contained 114 mM Na^+, 2 mM K^+, 1·8 mM Ca^{2+}, 117·2 mM Cl^-, 2·4 mM HCO_3^- and 2 g/l. glucose; (ii) 35 min after replacement of 113·6 mM Cl^- by Br^-; (iii) 12 min after return to original solution. *B*, Intracellular response of a sympathetic ganglion cell to an antidromic stimulus (applied to the sciatic nerve) followed by that to an orthodromic stimulus; (i) in "Cl- solution" same composition as in *A*(i); (ii) 14 min after exposure to "Br- solution", same composition as in *A*(ii). *C*, V, Intracellular response to a depolarizing current pulse, I (applied through the recording electrode), followed by that to an orthodromic stimulus; (i) in "Cl- solution" (ii) 11 min after exposure to "Br- solution"; note absence of change in resistance of cell. Time calibration, 25 ms in *A*, 100 ms in *B* and *C*. Voltage calibration, 2·5 mV in *A*, 50 mV in *B* and 100 mV in *C*. The current pulse in *C* was about 5×10^{-10} A.

The preparation used was the lumbar portion of the sympathetic chain of the frog, including the eighth to tenth ganglia, and the sciatic plexus[2,3]; orthodromic stimuli were applied to the chain between the eighth and ninth ganglia and responses were recorded either from small groups of post-ganglionic fibres by means of an electrode inserted into a post-ganglionic ramus[4,5] or from individual cells by means of an intracellular electrode[2,3]. Examples of recordings obtained in the first way are shown in Fig. 1*A* and they suggest that replacement of more than 95 per cent of the external chloride by bromide does not impair synaptic transmission through sympathetic ganglia. The intracellular records in Fig. 1*B* and *C* point to the same conclusion and the fact that the initial rate of rise of the orthodromic action potential is unaffected by the replacement of chloride with bromide ions suggests that this procedure does not change the amount of transmitter released. Furthermore, in five experiments in which the preparation was bathed in "Br- solution" the average number of quanta released per stimulus was found to be between ten and fifty-four. This range is similar to that found in earlier experiments[6] in "Cl- solution" (six to thirty-four) by the same method.

It would be of interest to know whether the difference between the present result and that of Muchnik and Gage resulted from the difference between the junctions studied or the presence of different concentrations of divalent ions. It is significant, however, that the presynaptic effect of bromide ions on transmission is not general and, in the absence of any evidence related to synaptic transmission within the central nervous system, it would be unwise to suppose that such an effect accounted for sedation.

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Miniature End-plate Currents in Voltage-clamped Muscle Fibre

THE acetylcholine which is released from presynaptic terminals after an action potential causes an increase in the conductance of the subsynaptic membrane of frog skeletal muscle, and the current which flows across this shunt depolarizes the membrane to give the characteristic end-plate potential (e.p.p.). The time course of this end-plate current (e.p.c.) in the frog has been determined in voltage clamp conditions by Takeuchi and Takeuchi[1]. It is now recognized that acetylcholine (ACh) is released from presynaptic terminals in the form of unit packages or quanta[2]. These quanta are released at a low rate from the "resting" terminal and each quantum causes a miniature end-plate potential (m.e.p.p.). After an action potential in the presynaptic terminals, the rate of release of quanta is increased for several milliseconds[3]. The time course of the end-plate current therefore reflects not only the time course of the conductance change caused by each quantum of ACh but also the time course of the release