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Drugs

MUSCLE RELAXANT ACTION The classical experiments of Claude Bernard established that curare acted at the myoneural junction. Langley, in 1905, and Dale and associates, in 1936, postulated that the drug acted by occupying cholinergic receptor sites on the postjunctional membrane. In recent years, Hubbard and other workers have suggested that the presynaptic effects of muscle relaxants may be of major importance. Paton and Zaimis, in 1952, proposed that neuromuscular blockade resulted from competition between acetylcholine and curare-like drugs for the receptor sites. This theory was elaborated in 1961 by Paton, who proposed that the point of equilibrium in such a dynamic state would depend upon the rate of reaction between the drug and the receptor. If the competition theory is correct, it must fulfill certain criteria: 1) the equilibrium–dose ratio between the agonist, in this case acetylcholine, and the antagonist, a curare-like drug, must be a constant; 2) the rate of association and dissociation of a drug with a receptor site must be of the same order if the amount of entropy in the system is constant. It has always been assumed that the degree of paralysis produced by a drug is dependent upon the blood, and hence the extracellular fluid (ECF), level of the agent, and that lowering the blood and ECF concentrations would, therefore, reverse the paralysis. The present work suggests that this assumption is not valid for anesthetized human patients. *(Feldman, S. A., and Tyrrell, M. E.: A New Theory of the Termination of Action of the Muscle Relaxants, Proc. Roy. Soc. Med. 63: 692 (July) 1970.)*