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The Clinical Use of Muscle Relaxants

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Relaxants have been in clinical use for almost a quarter of a century. During this time significant advances were made in the understanding of the anatomy of the neuromuscular junction (n.m.j.)[1], the physiology of the transmission process, the mode of action of neuromuscular blocking agents (n.m.b.a.) and the various factors which influence the action of these agents at the n.m.j.

These developments necessitated the revision of some of the concepts of the clinical use of relaxants. As in other fields of medicine, however, there has been a considerable time lag between the availability of scientific information and its application to clinical practice.

The purpose of this presentation is to attempt to bridge this gap and to consider, on the basis of presently available experimental data and clinical experience, the rational use of n.m.b.a. and their antagonists.

Depending on their chemical structure, the clinically used quaternary ammonium-type n.m.b.a. have been divided into two groups [4] (Fig. 1). The first group consists of the relatively bulky pachycurares which were assumed to interfere with the depolarization phase of n.m. transmission and to produce a non-depolarization block. These agents were also called non-depolarizing or antidepolarizing relaxants [17]. The second group consists of the less-bulky leptocurares which are structurally more similar to acetylcholine than the pachycurares. These compounds produce a prolonged depolarization of the post junctional membrane, interfere with the repolarization phase of n.m. transmission, and are usually referred to as depolarizing relaxants [49]. It was assumed that acetylcholine and the non-depolarizing and depolarizing n.m.b.a. all act at the same receptor sites [17, 49].

Further studies, however, revealed that the mode of action of the leptocurares is more complex. It was shown by THESLEFF [55, 58] that despite the continued presence of the depolarizing agents at the n.m.j. and the persistence of the n.m. block, the postjunctional membrane becomes repolarized and, at the same time, loses its sensitivity to acetylcholine and other

[1] The following abbreviations will be used in the text: neuromuscular (n.m.); neuromuscular blocking agents (n.m.b.a.); neuromuscular junction (n.m.j.).