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Depolarisation of the motor endplate region and neuromuscular block

In experiments on the cat's gracilis muscle decamethonium iodide has been found to cause a depolarisation of the muscle fibre localized to the endplate region. At first this depolarisation is sharply localised and is associated with increased exitability and spontaneous activity of the muscle fibre. Later the depolarization spreads to involve progressively more of the fibre membrane adjoinging the endplates, although it never invades the whole muscle fibre. The depolarized area now becomes less excitable to direct electrical stimulation, blocks the propagation of a directly excited muscle action potential across the endplate region, and raises the propagation threshold to the endplate potential elicited by nerve stimulation. With these changes, neuromuscular block occurs. The depolarization can be removed, and neuromuscular transmission restored, by the application of an anode to the endplate region. Application of a cathode deepens the block.

These characteristic features of the action of decamethonium are also produced by the injection of acetylcholine, by tetanization of the motor nerve in the presence of anticholinesterases, or by the application of a cathode to the endplate region for longer than a few seconds; i.e.by any long-lasting depolarization of the endplate region. The phenomena described (transmission block, associated with local depolarization; initially increased local excitability followed by decreased local excitability; nspread of depolarization with time; reversal of block by an anode and intensification by a cathode) may therefore be regarded as the necessary consequences of the presence of a maintained depolarization at a junctional region.