ACOUSTIC REFLEX AS A SIMPLE TEST FOR MUSCLE REPOLARIZATION

THESIS

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The Degree of M.Sc. in
(EAR, NOSE AND THROAT)

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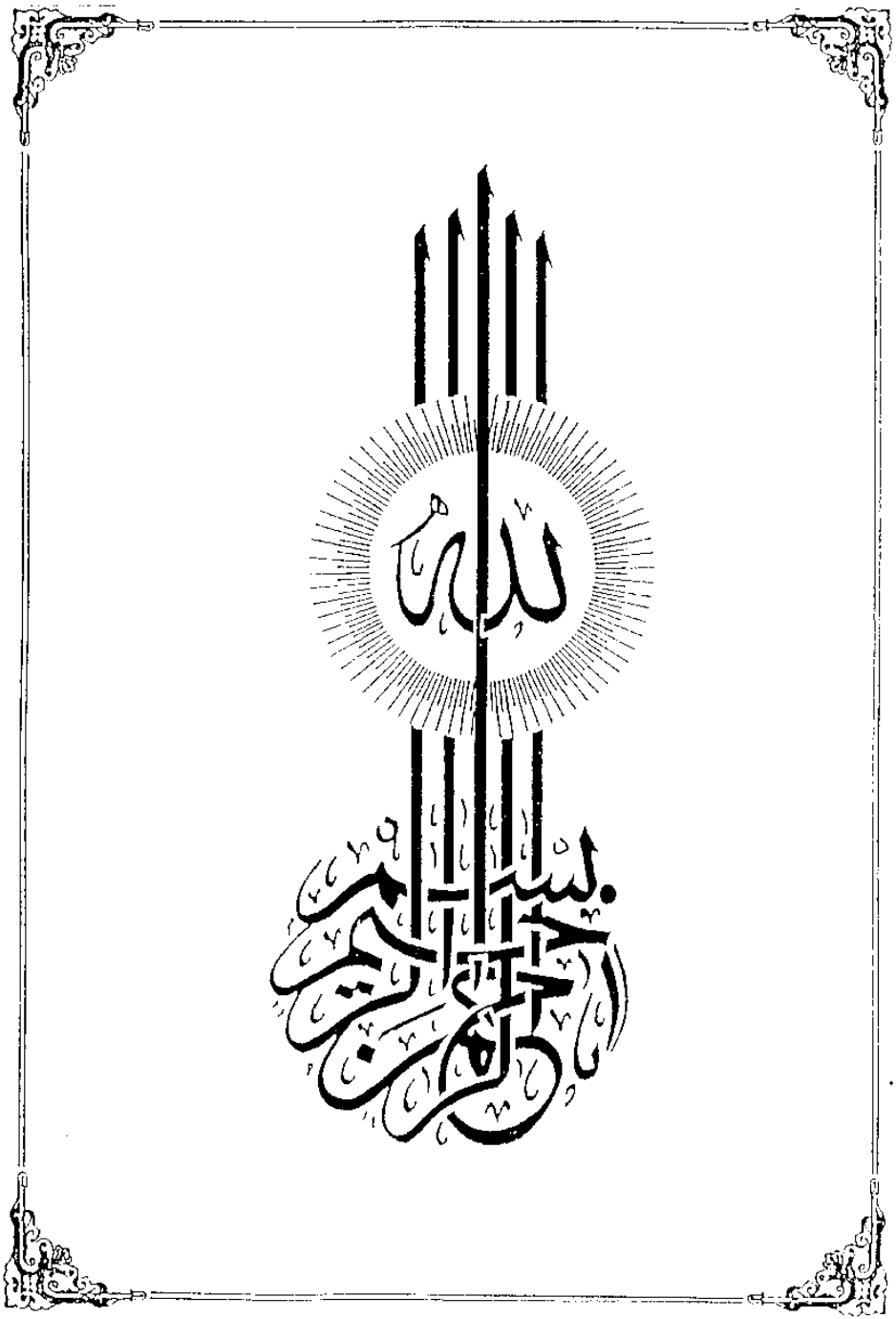

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ABSTRACT

Clinical use of the neuromuscular blocking drugs to produce muscle relaxation has changed the techniques of general anaesthesia during the past 35 years. Since deep anaesthesia can be avoided by their use.
An anaesthetist must therefore, have some basic knowledge for the rational use of the neuromuscular blockers to deal with complex problems that may arise in clinical practice as (1) Pharmacokinetics. (2) Action of the neuromuscular junctions. (3) Common and uncommon factors which modify the drugs. (4) Methods of reversing the blockade by proper use of neuromuscular facilitatry drugs. (5) and methods of monitoring the neuromuscular blockade.

The methods used for monitoring up tell now based on electric nerve stimulation is are: ulnear nerve, posterior tabial nerve and facial nerve stimulation. The most commonly used is stimulation of the ulner nerve via certain gauge needles placed subcutaneously and a supramaximal stimuli of duration 0.3 ms are applied at a frequency of 0.15 HZ. and electromyographic (E.M.G.) activity of the adductor pollicis muscle is recorded continuously so detection of repolarisation of the body muscles is obtained after the end of the relaxation state.

The idea of this work is to find an easier way for the muscle repolarisation through recording the acoustic muscle reflex at the begining of repolarization state.

We do our work on 25 middle aged patients 10 females and 15 males and they are Ear. Nose and Throat free also they are free from neuromuscular diseases, that is evidenced by clinical examination, tympanometery examination, electromyographic testing and stapedius reflex measurement.

All patients was subjected to anaesthesia with a same dose of thiopenton sodium (Intraval) in a dose of 1 gram for every patient and suxamethonium (succinylcholin) as a depolarizing muscle relaxant in a dose of 1 mg/kgm of body weight.

We recorded the time of succinylcholin injection, the time of return of acoustic muscle reflex after the action of the succinylcholin was ended and the time of return of the spontaneous respiration.

REVIEW OF LITERATURE

PHYSIOLOGY OF NEUROMUSCULAR TRANSMISSION
(Figure 1,2,3,4,5 and 6)

The basis for the neurohumoral theory of neuromuscular transmission has been put by Dale and his collegues in (1936), who demonstrated that release of acetyl-choline at the neuromuscular junction occurs on stimulation of motor nerves and the action of this chemical is intercellular or in the subneural space. The neuromuscular theory suggests that the acetyl-choline depolarized the end plate, induces a current and this initiates muscular contraction. This was confirmed by the work of Dowan (1936).
Another electro-chemical theory had been postulated by Nashmansoh, (1950), who suggested that the electric current generated at the axonal terminal bridges the subneural gap and liberates acetylcholine at the post-junctional membrane leading to depolarization of the end plate.

Del Castillo and Katz (1956) searshing for an evidence of both the chemical and electrical theories concluded that there is good evidence that electric current fails to pass across the synapse and there is no experimental support for the claim that electric transmission can produce accross the neuromuscular junction. Also Goodman

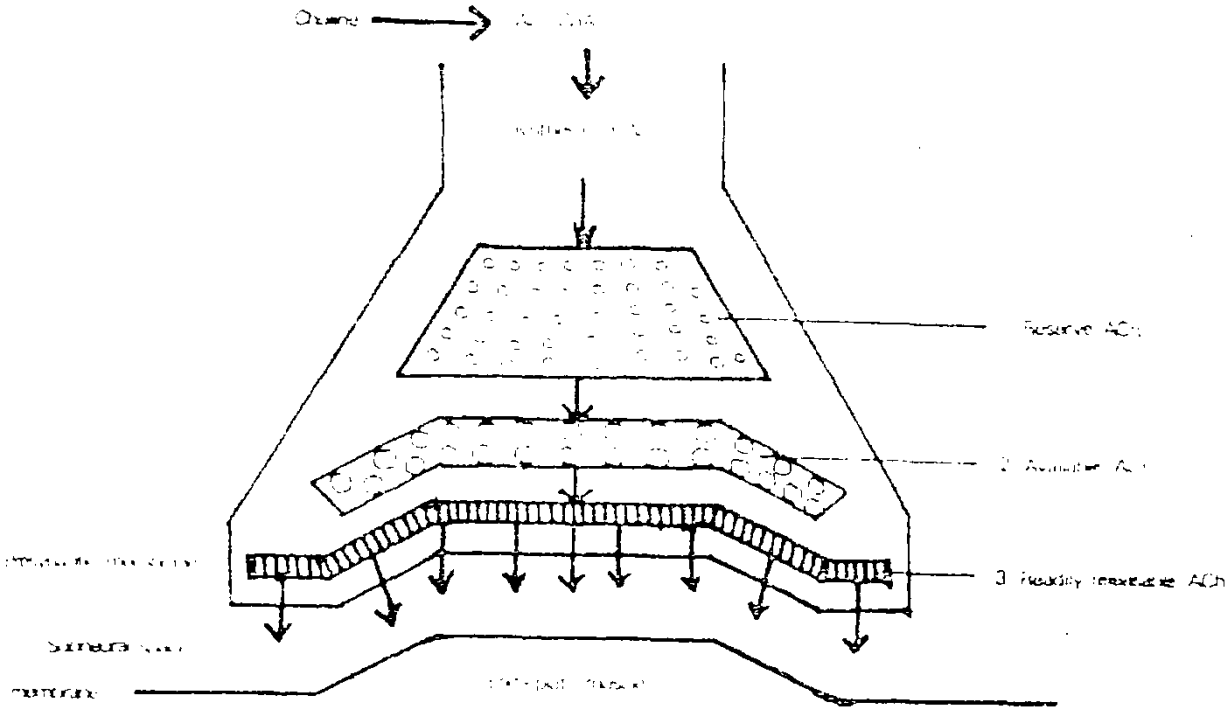

Figure (1)

Diagram of acetylcholine production, stores and flow in cholinergic nerve terminals.

and Gilman (1970) added that there is indirect evidence that most post-synaptic and post-junctional membrane are electrically inexcitable.

Neurohumoral mechanism of neuromuscular transmission :-

Acetylcholine plays the essential role in neuromuscular transmission. Most of the acetylcholine concerned with neuromuscular transmission is synthestized within the motor nerve terminals, only small amount being formed in the axon and moved to the terminal by axoplasmic transport. Synthesis is effected by acetylation of choline (actively transported across the nerve terminal membrane), (Birks and Macintosh, 1957 and 1961 by acetyl co-enzyme A (from the mitochondria), with choline-o-acetyl transferase as a catalyst, while the ultimate source of choline is in plasma, up to 50% of that which is actively transported into the nerve terminal is derived from the hydrolysis of previously released acetylcholine, moreover, the rate of uptake is increased by nerve stimulation and the energy transformation that associated with integration of acetylcholine are through ATP (Nach Manson, 1962).

Synthesized acetylcholine appears to exist in a precursor form in the resting state, and it is stored in some molecular form in the axoplasm, and part in

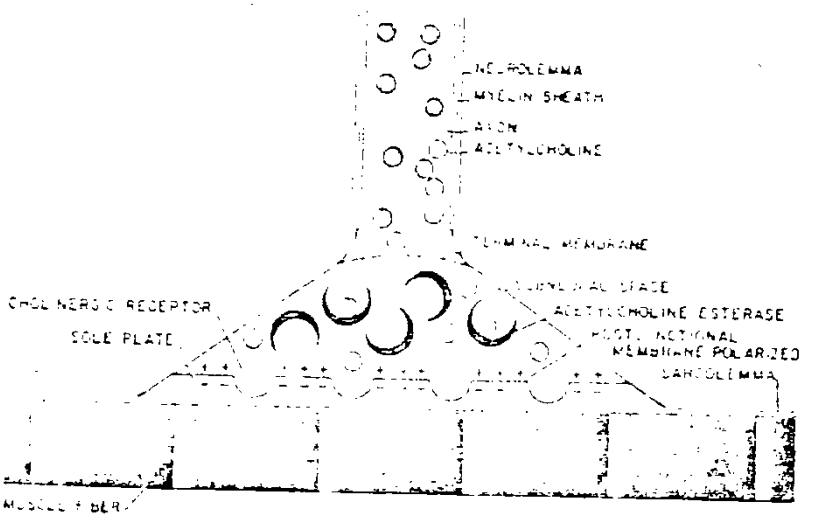

Figure (2)

Schematic representation of the resting end plate.

Note that the post-junctional membrane is polarized

vesicles in the form of multimolecular packets or quanta which are bound and inactive and these vesicles are characteristic of pre-junctional nerve terminals (Taylor, 1960). Three pools of vesicles appear to exist readily releasable acetylcholine (15-20%), available acetylcholine and reserve or depot from which the available acetylcholine is replenished (Hubbard and Wilson, 1973). Both calcium and magnesium are essential for fusion of vesicles and nerve membrane and for extrusion of acetylcholine packets through the micro - tubular membrane system of Birks into the subneural space, so reduce calcium or increase magnesium concentration may reduce the release of acetylcholine or may abolish it completely (Delcatillo and Engback, 1964), Katz and Miledi, 1964 and 1965).

Paton and Wand (1967) have shown that only 20-25% of the receptor pool need to be affected by the transmittor to produce transmission to all fibres in a muscle, thus there is a large receptor reserve or a margin of safety for transmission.

At the motor end plate, it appears to be the complex formed by acetylcholine and the membrane receptor that induces the change in membrane permeability results in

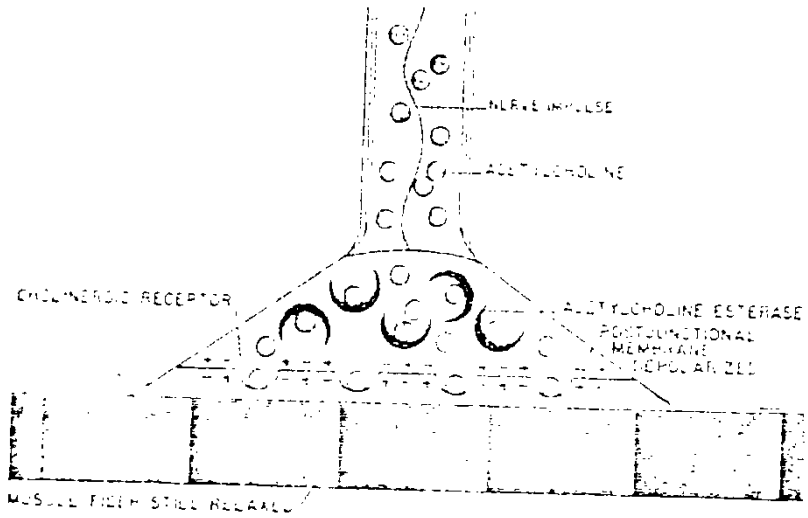

Figure (3)

The effect of nerve impulse on the end-plate.

Note :

a) The increased acetylcholine concentration at the end-plate.

b) Cholinergic receptors are occupied by acetylcholine.

c) Post-junctional membrane is depolarized.

d) Muscle fibre is still relaxed.

an increase in Na^+ and K^+ permeability produce a depolarization potential the current sink created by this local potential depolarized the adjacent muscle membrane to its firing level.

Action potential are generated on either side of the end plate are conducted away from the end-plate in both directions along the muscle fibre. The muscle action potential in turn initiates muscle contraction (Ganong, 1981). The reaction of acetylcholine and receptor substance is completed in a fraction of millisecond and is followed immediately by hydrolysis by cholinesterase (Collins, 1976). The enzyme itself is persumed to have an esteric and anionic receptor site like that of the receptor protein. The anionic site strongly attracts the positively charged onium head of acetylcholine, the esteric site fixes the drug and produce hydrolytic cleavage of the molecula, not all the acetylcholine is destroyed by the cholinesterase of the end plate, a variable proportion diffusing away from the synaptic area to be hydrolysed by plasma choline esterase Figure (6).

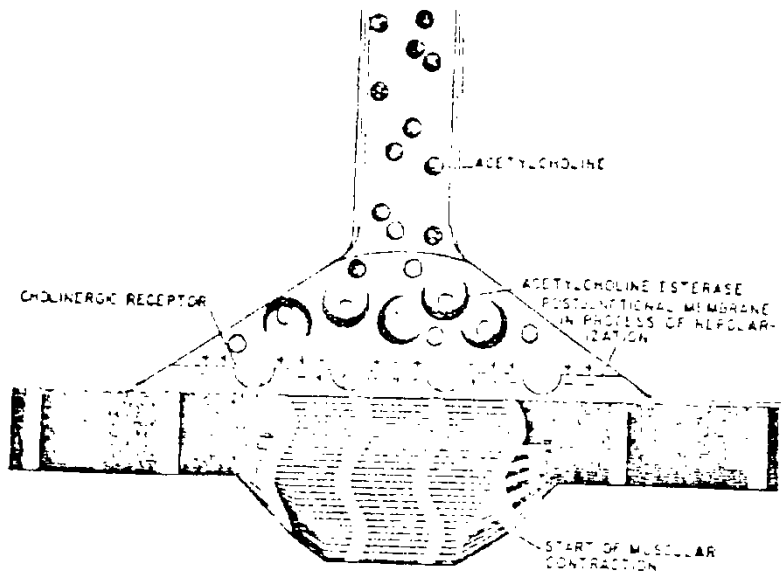

Figure (4)

This diagram is 2.5 to 3.0 millisonds later than in Fig.(5). Post-junctional membrane almost completely repolarized, muscle fibre just starting to contract.