and the relationship of this reaction with the other light reactions that this species exhibits.

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[1] Fischer, P.-H., "Vie et Moeurs des Mollusques" (Paris, 1950). Thorpe, W. H., "Learning and Instinct in Animals" (London, 1956). Fraenkel, G. S., and Gunn, D. L., "The Orientation of Animals" (Oxford, 1940).
[2] Kerz, M., *Experientia*, **6**, 427 (1950).
[3] Waterman, T. H., *Science*, **3**, 252 (1950).
[4] Wellington, W. G., *Ann. Ent. Soc. Amer.*, **48**, 67 (1955). Wellington, W. G., *et al.*, *Canad. J. Res. Zoology*, **29**, 339 (1951).
[5] Vowles, D. M., *Nature*, **165**, 282 (1949). Waterman, T. H., *Trans. N.Y. Acad. Sci.*, **14**, 11 (1951). Stephens, G. C., *et al.*, *Ann. Ent. Soc. Amer.*, **46**, 75 (1953). Carthy, J. D., *Discovery*, **18** (3), 105 (1957).

Neuro-Anatomical Site in Tick Paralysis

Previous studies[1] have shown that tick paralysis, induced in mongrel dogs by applying the wood tick *Dermacentor andersoni* Stiles, is due to failure in transmission at the neuro-muscular junction because: (*a*) the anterior tibial muscle in a paralysed dog responded to direct but not to indirect stimulation through the peroneal nerve; (*b*) the motor nerve fibres conducted an impulse.

More recent investigations have demonstrated the following: The muscle contracts on rapid close intra-arterial injections of acetylcholine; neostigmine does not facilitate transmission in a fully paralysed muscle and causes only a slight improvement in a partially paralysed one; neither cathodal nor anodal current facilitate transmission in a fully paralysed muscle; the paralysis is intensified by curare, but is uninfluenced by pentamethonium; tetanization via the nerve slightly facilitates transmission of subsequent individual pulses in both the partially and completely paralysed muscle and in the former the tetanus is well maintained. These results indicate that the neuro-muscular block is dissimilar to that produced by the competitive and depolarizing blocking agents and anticholinesterases.

Functional activity of the end-plates is indicated by (*a*) contraction of the muscle on close intra-arterial injection of acetylcholine, (*b*) their depolarization by succinylcholine.

Table 1 shows the response of the normal, partially and completely paralysed anterior tibial muscle to close intra-arterial injections of acetylcholine. Increasing paralysis apparently produces an increasing sensitivity of the muscle to acetylcholine, the paralysed muscle behaving like a denervated one.

End-plates could be located in the paralysed anterior tibial muscle by sweeping its undersurface with a wick electrode after an injection of succinylcholine, the technique being similar to that described by Burns and Paton[2]. Usually four or more end-plate regions could be located in this way. The depolarizing effect of the succinylcholine was then permitted to wear off. In an apparently paralysed muscle, usually one or more muscle fibres contracted on nerve stimulation and the site of minimum latency of the action potential corresponded to only one of the located end-plate regions. Neither action nor end-plate potentials could be detected at the other located end-plate regions. The findings suggest that the probable mechanism for the paralysis is due to failure in the liberation or synthesis of acetylcholine at the nerve terminals.

Table 1. Sensitivity of Anterior Tibial Muscle to Rapid Close Intra-arterial Injection of Acetylcholine

Anterior tibial muscle	No. of experiments	Approximate average dose (μgm.) of acetylcholine to produce	
		Threshold contraction	Contraction = maximal muscle twitch
Normal	4	5	50
Partially paralysed	3	1	15
Fully paralysed	1	?	1

In an attempt to localize exactly the site of the neuro-muscular block, the anterior tibial muscle was stimulated directly in a completely paralysed animal before and after the administration of curare. No difference could be detected in the magnitude of the mechanical responses indicating that the block is probably located at the nerve terminals in contradistinction to that in botulism poisoning, where the block is evidently proximal to the terminals[3].

The only effective method, known to date, of producing tick paralysis is by applying the wild ticks. When a saline extract (1 gm./ml.) of 128 engorged female ticks removed from paralysed dogs was injected intra-arterially (3 ml. × 1 ml.) and intravenously (1, 10 and 10 ml.) into a normal anæsthetized dog, it produced no detectable effect on the mechanical response of the anterior tibial muscle during indirect stimulation. Evidently continuous secretion of a 'toxin' by the ticks is necessary to produce paralysis because rapid recovery occurs on removal of the ticks.

As the tick 'toxin' appears to interfere with the liberation of acetylcholine at the neuro-muscular junction, one might expect that it would also interfere with acetylcholine liberation at other sites, for example, ganglionic and neuro-effector cholinergic junctions. Stimulation of the central end of the cervical sympathetic, proximal to the superior cervical ganglion, resulted in a normal dilation of the pupil and retraction of the nictitating membrane in a paralysed dog and stimulation of the peripheral end of the vagus in the neck caused normal cardiac slowing. In addition, if acetylcholine liberation in sympathetic ganglia was diminished in a paralysed animal one might expect to find a low blood pressure. The blood pressure in five paralysed dogs were within normal limits (130, 150, 100, 170 and 190 mm. mercury). Evidently the tick 'toxin' exhibits a specific predilection for the neuro-muscular junction.

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[1] Murnaghan, M. F., Proc. Tenth Internat. Congr. Entomol. Montreal, Canada, August 1956 (in the press).
[2] Burns, B. D., and Paton, W. D. M., *J. Physiol.*, **115**, 41 (1951).
[3] Burgen, A. S. V., Dickens, F., and Zatman, L. J., *J. Physiol.*, **109**, 10 (1949).

Anthocyanins in Blackcurrant Fruits

Robinson and Robinson[1] investigated the anthocyanin pigments of the fruit of the blackcurrant (*Ribes nigrum*) and stated that the skins contained a cyanidin-3-bioside. Cyanidin was the only aglycone found; but it was thought possible that small amounts of delphinidin were present. Fouassin[2] has recently examined blackcurrant pigments by paper