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Atypical tetanus--towards an understanding.

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Tetanus toxin is a neurotoxin of extraordinary potency. In nanogram amounts it releases the central nervous system from inhibitory control enabling muscles which normally propel the skeleton to be driven into a state of persistent contraction, and the patient into a state of protracted immobility. The clinical picture of trismus, muscle rigidity, and reflex spasms is classical. On occasions however, this picture is modified as in cephalic tetanus, where one sees areas of local muscle paralysis in the presence of typical muscle overactivity elsewhere in the body. Such a paradoxical situation argues against a single or simple mode of action for tetanus toxin. How is atypical tetanus explained?

:: Experimental data

Animals differ in their susceptibility to tetanus, a fact attributed to the ease with which various peripheral nerves can transport the toxin centripetally to the brain and spinal cord. In any one species, graded doses of toxin induce different effects. Thus sublethal doses produce classical tetanus with recovery within six weeks. Ten minimal lethal doses (10 MLD) induce an identical picture, but one which is rapidly

fatal. Pathological study of such animals is revealing. A number of anterior horn cells belonging to the limb into which tetanus toxin was injected show evidence of chromatolysis, whilst those of the contralateral limb do not.[7] This suggests that high concentrations of toxin transported centripetally are in some way responsible for nerve cell damage. Histochemistry of such cases shows select atrophy of Type I muscle fibres.[3] These are slow twitch fibres dependent on aerobic metabolism whose characteristics are determined both by their innervation as well as by the pattern of neuronal discharge they receive. Electrophysiological studies during life indicate a lesion at the neuromuscular junction whereby the release of the neurotransmitter, acetylcholine, to Type I fibres is prevented.[5]
Finally when animals are subjected to very large doses of toxin (1000 MLD) paralysis of all muscle types develops rapidly and the animals die without developing a single tetanic spasm.

:: Human data

In human tetanus, patients may harbour many MLDs of toxin and yet survive because of the intensive treatment they receive. If this fact is kept in mind then it will be seen that animal studies have their human counterpart. Peripheral nerves in severe tetanus when studied electrophysiologically show evidence of an axonal neuropathy.[6] The lesion is mild and in all probability reversible. In contrast to animals it is not located at the myoneural junction and its exact site at present remains speculative. Since the majority of the nerve fibres continue to conduct impulses, these patients demonstrate typical muscle overactivity and not paralysis.
In cephalic tetanus, one does see local areas of muscle paralysis. That this is a reflection of a high local concentration of toxin is based on two observations. First, the site of maximum paralysis is found adjacent to the precipitating lesion. Second, affected muscles in the stage of recovery are first transformed into a state of overactivity before returning to normal.[2] The facial nerve is unusually vulnerable to this lesion perhaps because of its special function related to facial expression. However this may be, cephalic tetanus is usually mild and does not signify a bad prognosis.
The discussion to date has focussed on the effects of toxin on the somatic nervous system. These can be abolished by using neuromuscular blocking agents, but only serve to uncover the actions of toxin on the autonomic nervous system. Here the picture is one of excessive sympathetic discharge with increased levels of catecholamines.[1] In severe tetanus the system shows extreme lability. Continuous EEG monitoring suggests sudden failure of autonomic centres in the hypothalamus and brain stem, since an abnormal trace invariably precedes episodes of hypotension or cardiac standstill.[4] Hypoxia, infection, or acid base disturbance may trigger this failure at certain points in time.
Toxin through its action on the hypothalamopituitary axis also creates on altered endocrine environment. As the disease peaks, hormone levels of FSH, LH, testosterone and estradiol fall severely. Prolactin and cortisol levels remain within the normal range but appear to be inadequate considering the hyperactive and stressed state of the tetanus patient. Temporary hypothalamic failure would be a plausible explanation for these findings.
The above examples illustrate the widespread physiological disturbance that accompanies tetanus. In severe disease, effector neurones exposed to high concentrations of toxin become temporarily unresponsive to other neuronal influences. This feature is seen to underly atypical tetanus in its different guises.

:: Summary

Tetanus toxin abolishes the synaptic transmission of inhibitory impulses within the central nervous system. Evidence is provided which suggests that wherever high local concentrations of toxin exist, effector neurones are unable to relay messages to target organs, thereby treating focal areas of paralysis. Different neurones vary in their susceptibility to this effect of toxin. The concept plausibly explains the syndrome of cephalic tetanus. It also may account for paroxysmal autonomic failure and temporary hypothalamic endocrine failure seen at times in severe tetanus.

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