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## Understanding the role of antivenom in treating neuromuscular dysfunction in snake envenoming.

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Neuromuscular paralysis in snake envenoming is typically a descending paralysis that rapidly becomes life-threatening. Based on experimental evidence, most snake neurotoxins either structurally damage the motor nerve terminal (pre-synaptic) or antagonize the nicotinic acetylcholine receptors in the motor-endplate (post-synaptic) of the neuromuscular junction, resulting paralysis.

The role of antivenom in prevention and reversal of paralysis in snake envenoming has never been supported with high quality clinical evidence. Observational studies have described the failure of antivenom in prevention and reversal of paralysis. However, lack of proper case-authentication, unquantifiable clinical parameters with no serial measurements of venom concentrations and the absence of neurophysiological parameters have made it difficult to distinguish whether the failure of antivenom is due to the inadequate neutralization of venom neurotoxins in the circulation (poor efficacy) or it is related to an insult that is impossible to be reversed by antivenom (poor effectiveness). However, once the observational studies were equipped with serial measures of objective clinical features, venom concentrations and sensitive neurophysiological tools such as single-fiber electromyography, the quality of the clinical evidence could be improved. In confirmed Indian krait (Bungarus caeruleus) bite and Russell's viper (Daboia russelli) envenomings, despite early antivenom clearing venom antigens from circulation, antivenom neither prevented nor reversed the paralysis as evident clinically and neurophysiologically. The above observations showed reliable clinical evidence of poor effectiveness of antivenom in treating paralysis due to pre-synaptic neurotoxins, despite the antivenom being efficacious.

In snakebites with pre-synaptic neurotoxicity, by the time antivenoms are given, already started neurotoxin mediated damage is unlikely to be reversed. However, efficacious antivenoms still have a place in trapping the neurotoxins within the circulation preventing further neuromuscular damage. Similar studies are needed on envenomings by Asiatic cobra (Naja sp.), to determine the effectiveness of antivenom for post-synaptic neurotoxicity.