A Physiologically Based Pharmacokinetic Model for Cobra Envenomation

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Abstract

Cobra bites are one of the leading contributors to the deaths worldwide attributed to venomous snake bites. This work discusses a pharmacokinetic model for the body where death from cobra envenomation occurs as a result of venom blocking nicotinic acetylcholine receptors caused the by binding of venom to the receptor sites. When approximately three fourths of the receptor sites in the skeletal muscle are bound by cobra venom, the synaptic chemical signal transmission for breathing ceases, causing death by suffocation. Death can occur as quickly as 30 minutes after envenomation if no treatment is administered. The proposed model is a physiologically based 2-compartment representation of the human body. The central compartment consists of the blood and organs and the muscle compartment contains the skeletal muscle in the body. An important aspect of this model is the inclusion of receptor sites in the muscle compartment to model the occurrence of death. By utilizing appropriate organ volumes and perfusion rates for each compartment in both rabbits and humans along with simple reactions following elementary rate laws, the proposed pharmacokinetic model can simultaneously track the distribution of cobra venom, antivenin, and free receptor sites in the body.