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ELECTRIC SHOCKS ARE INEFFECTIVE IN TREATMENT OF LETHAL EFFECTS OF RATTLESNAKE ENVENOMATION IN MICE

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E. K. JOHNSON, K. V. KARDONG and S. P. MACKESSY. Electric shocks are ineffective in treatment of lethal effects of rattlesnake envenomation in mice. Toxicon 25, 1347–1349, 1987. — Electrical shocks, even crudely delivered from 'stun guns' and gasoline engine spark plugs, have been reported to be effective in the treatment of snake bite. We thus applied similar electric shocks to mice artificially injected with reconstituted rattlesnake venom at various LD_{50} multiples. Those envenomated mice treated with electric shock survived no better than the controls. We thus found no evidence that electric shocks crudely administered had any life saving effect in mice.

THE USE of high voltage, low ampage electric shock in humans to treat snake bites has been proposed (GUDERIAN *et al.*, 1986). This involves application of electric shock to the bitten part, with reported instances of relief from pain and even life saving potential. As to the relief of pain, it has been suggested that the shock may really do no more than relieve the local discomfort and swelling, for which other less traumatic techniques already exist (SCHMUTZHARD, 1986). We present results on the implied life saving effects of electric shock, since even crude sources of the current, such as 'stun-guns', outboard motors, lawn mowers and auxillary lighting plants, have been reported to be efficacious (GUDERIAN *et al.*, 1986).

In an attempt to simulate the treatment of humans, male Swiss Webster mice weighing 20–25 g were injected intramuscularly into the right thigh with a range (0.75–6 mg/kg) of venom doses that included the approximate LD_{50}. The lyophilized venom, originally extracted and pooled from northern Pacific rattlesnakes (*Crotalus viridis oreganus*), was reconstituted by standard procedures (KARDONG, 1986*a*). A total of eight or, when near the actual LD_{50} level, sixteen mice were used at each dose level, half the mice being controls (venom only), the other half being experimentals (venom + electric shock). The injection volume was corrected so that each mouse received 50 μl containing the desired venom dose. The electric shock, 20–25 kV, <1 mA, was produced by a car electrical coil (transformer) run by a 12 V battery. The positive electrode was clipped to the tail and the negative to the skin of the shank distal to the site of venom injection. To each shocked mouse, a total of 10 such separate shocks of <1 sec duration were administered, all within

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60 sec of the venom injection. None of the shocked experimental mice survived at greater frequency than the control group. Twenty four hour survival of envenomated control and shocked mice were as follows: at 6.0 mg/kg, control (4/4), shock (4/4); at 3.0 mg/kg, control (4/4), shock (4/4); at 1.75 mg/kg, control (6/8), shock (7/8); at 1.5 mg/kg, control (0/4), shock (0/4). Parentheses indicate number dead/total number tested, respectively. Thus, we found no evidence that high voltage, low ampage electric shock is efficacious in treating the lethal effects of rattlesnake envenomation in mice. There are two possible reasons for this apparent absence of effect in mice.

First, mice may not be the most suitable model for evaluation of shock therapy or our electric shock in mice may not have accurately simulated the electric shock treatment used in humans. Perhaps a different schedule of shocks, changes in current or duration of shock would have changed our results. However, we used an automobile transformer to generate the high voltage, applied the shock to the envenomated region and applied it in a timely fashion. All of this seems to generally parallel the crude technique used in the Ecuadorian jungles and elsewhere (GUDERIAN *et al.*, 1986).

Second, our results may be negative because there is in fact no significant efficacy of electric shock for the lethal effects of snake bite. The common sequelae of crotaline snake bites include not only lethal effects, but also local tissue damage, such as hemorrhage, edema and myonecrosis (OWNBY, 1982). In the 34 cases of human snake bite in Ecuador treated with electric shock, GUDERIAN *et al.* (1986) report the absence or abatement of these local tissue responses, such as swelling, hemorrhage and serosanguinous bullae. In this investigation our results address only lethality and we made no attempt to evaluate local tissue responses in mice. It is still possible that shock treatment could prevent local tissue damage, while not affecting the venom components responsible for lethality. The supposed life saving effects of electric shock in humans, therefore, may be a misinterpretation of the cases of snake bite. In humans a large proportion of snake bites result in actual delivery of very little venom, despite visible evidence of fang puncture of the skin (REID, 1982; PARRISH, 1959; KARDONG, 1986*a*,*b*). For this reason, most strategies of medical treatment do not advise automatic administration of antivenom, but instead recommend treating the bite in proportion to the detectable signs of envenomation (RUSSELL, 1984). The reported life saving capabilities of electric shock in humans may therefore not be a consequence of the electric shock at all, but simply result from low levels of initial venom delivery.

We certainly agree that an alternative or supplement to antivenom for the treatment of snake bite would be helpful, especially in parts of the world where antivenom is not readily available. However, we could find no evidence in mice that electric shock crudely administered is a serious alternative for preventing the lethal effects of the venom.

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