Toxicon, Vol. 30, No. 10, pp. 1257–1279, 1992.
Printed in Great Britain.

0041-0101/92 $5.00 + .00

EXPERIMENTAL TREATMENT PROTOCOLS FOR SCORPION ENVENOMATION: A REVIEW OF COMMON THERAPIES AND AN EFFECT OF KALLIKREIN-KININ INHIBITORS

M. ISMAIL, AMAL J. Y. FATANI and T. T. DABEES

Department of Pharmacology, College of Pharmacy, King Saud University, Riyadh 11451, Saudi Arabia

(*Received* 13 *January* 1992; *accepted* 3 *April* 1992)

M. ISMAIL, A. J. Y. FATANI and T. T. DABEES. Experimental treatment protocols for scorpion envenomation: a review of common therapies and an effect of kallikrein-kinin inhibitors. *Toxicon* **30,** 1257–1279, 1992.—Nine fatal cases from the sting of the scorpion *Leiurus quinquestriatus* are presented. All victims showed association of CNS and cardiovascular manifestations. Either the CNS or the cardiovascular effects could occur first in the early phases of the scorpion envenoming syndrome; the CNS manifestations, however, always preceded the terminal hypotension and cardiac arrest. Pharmacokinetic studies in rabbits following s.c. injection of the labelled venom showed that rapid absorption took place with about 70% of the maximum blood concentration reached within 15 min. Intramuscular injection of antivenom did not significantly affect the absorption of the venom or the other pharmacokinetic parameters. The total area under concentration time curve was not significantly different from that following i.v. injection, showing that nearly complete absorption of the venom from the s.c. site would occur in 7–8 hr. The i.v. infusion of venom into anaesthetized rats, at a rate comparable to the absorption rate from s.c. sites, allowed the determination of the minumum lethal dose (MLD) with reasonable accuracy. In rescue experiments, anaesthetized rats were injected s.c. with multiple MLD of venom and infused i.v. with drugs commonly used in the treatment of scorpion envenomation. The prepared potent specific antivenoms, but not the commercial polyvalent antivenom, rescued all animals from the lethal effect of the venom, even when injected late. Atropine, atropine + phentolamine, chlorpromazine, hydrocortisone and indomethacin were able, in varying degrees, to rescue some rats injected with 2 MLD of venom. Phentolamine, propranolol, hydralazine and calcium gluconate significantly prolonged the survival time, but did not rescue any animals. Chlorpheniramine, saline and 1/4 saline + 5% dextrose were without any effect. Aprotinin, the kallikrein-kinin inhibitor, was able to rescue half of the animals from the lethal action of the venom. Electrocardiographic studies showed that *L. quinquestriatus* venom, irrespective of the route of administration, causes myocardial ischaemia and either inferior or anterior wall infarction. This was associated with an initial moderate and a terminal severe bradycardia together with a variety of rhythm and conduction defects. Except for minor and transient electrocardiographic changes, either the prepared antivenoms or aprotinin protected rabbits and rats from the cardiac effects of the venom.