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Resumo	Envenomation by coralsnakes (Micrurus spp.) is characterized by blockade of peripheral neurotransmission mediated by the presence of α- and β-neurotoxins. However, little is known about their cardiovascular activity. Micrurus lemniscatus lemniscatus is a coralsnake found in the Amazon basin and occasionally causes envenomation in humans. In this study, we examined the hemodynamic, vascular and atrial responses to M. l. lemniscatus venom. Anesthetized rats were used for hemodynamic and electrocardiogram (ECG) recordings; in vitro experiments were carried out in rat isolated thoracic aorta and atria preparations. In vivo, venom (0.1 and 0.3 mg/kg) caused immediate and persistent hypotension that was maximal within the first minute with both doses being lethal after ~40 and ~20 min, respectively. ECG, heart and respiratory rates were not altered during the transient hypotension phase induced by venom but all altered prior to death. There was no evidence of myonecrosis in cardiac muscle tissue, pulmonary hemorrhage nor thrombosis in anesthetized rats exposed to venom. In vitro, venom (10 μg/ml) did not contract aortic strips nor affected the maximal responses to pre-contraction with phenylephrine (PE, 0.0001–30 μM) in strips with and without endothelium. However, venom (10 μg/ml) relaxed aortic strips with endothelium pre-contracted with PE. In aortic strips pre-contracted with PE, venom prevented acetylcholine (0.0001–30 μM)-induced relaxation in strips with endothelium without affecting relaxation induced by sodium nitroprusside (0.1–100 nM) in strips without endothelium. Venom (30 μg/ml) produced a transient increase of atrial contractile force without affecting atrial rate. Taken together these findings indicate a predominantly vascular action of the venom, most likely involving toxins interacting with muscarinic receptors.
Fomento	