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Sympathomimetic action of tetanus toxin

Clinical reports by Kerr, Corbett & others (1968) revealed that in tetanus, there may be labile hypertension, tachycardia and irregularity of cardiac rhythm. We have looked for evidence of action of the toxin on the sympathetic nervous system. Tetanus toxin (Haffkein's Institute), 20 000 MLD/ml, given in amounts of 0·01 to 1·0 ml, produced 15-25% reduction of flow in the hind limb of the white rat perfused with oxygenated Ringer-Locke solution at 37°. The reduction was approximately doubled after pretreatment with cocaine (1 mg), but there was no reduction of flow in animals pretreated with reserpine (1 mg), 24 h before, or with phenoxybenzamine (1 mg) 45 min before the toxin.

A triphasic response was seen in the systemic blood pressure of the dog treated with (1.0 ml, of toxin per kg). There was an immediate sharp rise of 10 mm of mercury, then a similar fall below the pre-injection level and later a more sustained pressor effect of 30-40 mm of mercury for 15-20 min. There was no tachyphylaxis. Pre-treatment with phenoxybenzamine (10 mg/kg) abolished the delayed pressor effect without affecting the earlier two phases. Similar observations were made on the rat. In about one quarter of the dogs, administration of toxin produced only a depressor response; when these animals were pretreated with mepyramine, the injection of toxin produced a sharp transient pressor effect; this was followed by a more sustained pressor effect which was abolished by pretreatment with phenoxybenzamine.

Injection of low doses of toxin (0.01-0.5 ml) in the perfused frog or rabbit heart produced positive inotropic and chronotropic effects with higher doses of toxin (0.5 ml) there was a subsequent depression. These effects were blocked by suitable doses of β -adrenergic blocking agents like dichloroisopropyl noradrenaline or pronethalol.

The toxin also produced contractions of the smooth muscles of the dog spleen and the guinea-pig vas deferens which were antagonized by phenoxybenzamine.

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