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The action of some derivatives of ergot in peripheral vaso-motor exhaustion.

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In a previous report on the effects of adrenalectomy on the number and duration of anemic responses to temporary occlusion of the head arteries in the cat,[1] it was shown that the failure of blood-pressure to rise above spinal level was due to failure at the myo-neural junction, and not to exhaustion of the medulla; in animals subjected to adrenalectomy, injection of small doses of adrenalin so restored general conditions that the cardio-vascular and nervous mechanisms again became functionally competent, and several more occlusions could be done before there was again failure at the periphery. If, after subsequent failure of vaso-motor response, adrenalin was again injected, several more rises of blood-pressure could be obtained on occlusion of the head arteries.

The question arose as to whether the specific action of adrenalin was necessary to restore vaso-motor tone under such conditions, or whether any vaso-constrictor, acting at the myo-neural junction, would be effective. In several animals, therefore, after adrenalectomy and the typical peripheral failure of blood-pressure, tyramine (Burroughs-Wellcome), a preparation of ergot, was injected intravenously, under conditions similar to those in which adrenalin had been injected. In every case, there was an immediate and very adequate vaso-constrictor effect. Blood-pressure rose, and although it did not attain the height produced by the injection of adrenalin, it was maintained at a high level for a much longer period of time and was equally effective. The corneal reflex returned, and spontaneous respiration gave evidence of restored bulbar function. The anemic rise in blood-pressure could then be elicited several times before pressure again fell permanently. With another injection of tyramine, the entire process could be repeated, and so on until bulbar exhaustion was produced.

[1] Coombs, H. C., *Am. J. Physiol.*, March, 1925. (In press.)

Fluid extract of ergot (John Wyeth and Bro.) was likewise injected in doses of 15-30 minims in 5 cc. of Ringer's solution, under similar conditions of peripheral failure; but although there was a slight rise in blood-pressure, it was in no way comparable to that produced by tyramine, either in height or duration, and completely failed to effect any restoration of cardiovascular function.

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The rôle of the dorsal spinal nerve roots in bulbar anemia.

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Pike[1] has shown that in the spinal cat there is a certain reflex element concerned in the maintenance of blood-pressure, by the fact that section of the dorsal roots of the spinal nerves in the thoracic region causes a fall of about 10 millimeters of blood-pressure. Wickwire[2] has demonstrated that section of the dorsal roots of the spinal nerves from C5 to L2 does not abolish the compensation of heart rate to high and low blood-pressures.

A series of experiments was made to see whether division of the dorsal roots of the spinal nerves would affect the cardiovascular response to bulbar anemia, produced by temporary occlusion of the arteries to the head.[3] Laminectomy was done through the thoracic region, and the head arteries were isolated as usual. The blood-pressure rise in a control occlusion of the head arteries was then recorded, and as soon as recovery was initiated, the dorsal roots were divided in the thoracic region, and occlusion of the head arteries was repeated. Such difference in response as was shown between the two occlusions was due to the low pressure which resulted from the extensive operation,

[1] Pike, *Quart. J. Exper. Physiol.*, 1913, vii, 1.

[2] Wickwire, *Am. J. Physiol.*, 1920, liii, 355.

[3] Stewart, Guthrie, Burns and Pike, *J. Exper. M.*, 1906, viii, 289.