

these unfortunate occurrences, may be due to heart clot, owing partly to the hyperinotic state of the blood in pneumonia and partly to debility of the muscular walls of the heart from parenchymatous degeneration of its muscular tissue, or to endocarditis. The heart in this damaged condition may still be capable of doing its work with the body at rest in a recumbent posture; but any sudden elevation of the body to the erect posture, imposing an extra strain upon the organ, might cause a fatal paralysis. The practical lesson is obvious.

ON THE NECESSITY FOR A MODIFICATION OF CERTAIN PHYSIOLOGICAL DOCTRINES REGARDING THE INTERRELATIONS OF NERVE AND MUSCLE.

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HOW ARTERIAL SEDATIVES ACT.

Ergot of rye is an agent which produces in a marked degree contractions of involuntary muscular fibre everywhere, but whose effects are especially seen in the arterioles and uterus. Must not a uniform law or rule govern the occurrence of such contractions? We have seen that they occur best under a deprivation of nerve action, and are never so complete as in the general death of the body. How then can ergot be regarded as a stimulant? Who would ever think of administering it in cases of faintness and exhaustion as a restorative of nerve energy? Must it not act, like nerve section and nerve paralysis, in lessening the tone of the vascular and motor nerves, so setting free the contractile energy of the arterial and uterine muscles, which contract accordingly?

Dr. Sidney Ringer grows enthusiastic over the action of aconite in acute congestion of the tonsils, and that, too, in doses too small to reduce the action of the heart. Aconite undoubtedly causes contraction of the arterioles, and accordingly on the theory of the day it must be classed as a stimulant, as it actually has been by some authors, Dr. Edward Meryon, M.D., F.R.C.P., for instance, who holds that "it stimulates the dormant fibres of Remak and by so doing diminishes the calibre

of the arterioles" (a). Errors of this kind must be charged to the misleading guidance of an erroneous theory. Aconite is a profound paralyzer, and in small doses, by lowering the activity of the vaso-motor nerves, it frees the contractile power of the muscular bands of the arterioles, which contract accordingly, lessening or curing congestive states.

Is not this precisely the *role* of the galvanic current, when brought to bear upon the cervical sympathetic, say in exophthalmic goitre? The thyroid gland and its appendages are being overfed by dilated arteries. Bring about contraction of these arterial tubes, by lowering the activity of the vaso-motor nerves in the way just indicated, and the congestion and hyperplasia are relieved if not cured. But the electric current, for therapeutic purposes, has been classed as a stimulant! So has strychnia; so ought to be prussic acid, for it, too, causes spasms and convulsions of muscle! So is fatal hemorrhage. All stimulants, as well as aconite, on the theory of the day! It would require a volume to elucidate these points, and I must condense what I have to say into a few paragraphs.

STRYCHNIA A PARALYZING AGENT.

Dr. Harley has shown that strychnia probably acts by preventing the oxygenation of the blood, which Dr. C. B. Radcliffe very properly holds cannot be the *role* of a stimulant. Dr. Ringer tells that "after traumatic and strychnia tetanus the functions of the motor nerves and muscles are depressed; the motor nerves conveying impressions imperfectly." But may not this motor nerve depression be due to a reaction from previous over excitement? Dr. Ringer says no! and adds, "Strychnia directly depresses motor nerves, for large doses kill without exciting convulsions, when the motor nerves are found to have lost their conductivity" (b). Which in physiological language means that the nerves are paralyzed. Dr. W. A. Hammond has recounted an experiment performed by himself and Dr. S. Weir Mitchell, which, he says, "shows that the action of strychnia is to destroy the nervous excitability from the centre to the periphery" (c). Dr. Ringer further furnishes

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(a) Rational Therapeutics, p. 52.

(b) Therapeutics, 5th American Ed., p. 499.

(c) Dis. Nerv. Syst., p. 539.