meet the exigency of the theory it was necessary to imagine a purely hypothetical system of inhibitory nerves, the excitation of which, by antagonizing the proper motor ganglia of the heart, would bring it to a standstill. It is worthy of notice that in this experiment "the most marked effects are produced when the electrodes are placed on the boundary line between the sinus venosus and the auricles." (*a*) Now this is the precise location of the chief motor ganglion of the heart in the frog, —the animal in which this observation has been made, so that the assumed stimulus has to pass over the proper motor ganglion in order to reach the supposed inhibitory ganglia, farther away in the septum dividing the auricles! It needs explanation why, under these circumstances, the "stimulus" should ignore the motor ganglion in order to excite its rivals, which are further out of reach of the current.

The theory of the day on this subject, or rather the "temporary hypothesis," as Dr. M. Foster calls it, necessitates that the action of drugs be wrought out amid the struggle for supremacy between two rival nerve factions or camps, as it were, with results which are far from encouraging. For instance, a recent physiological work on the "Action of Medicines," informs us in the opening paragraph regarding belladonna, that "It paralyzes the motor nerves in frogs at the same time that it excites the spinal cord; after they recover from the motor nerve paralysis the tetanic symptoms of spinal stimulation appear"! Would it not be well to try how far the results might be simplified on the view that, under the circumstances, the heart's action ceased from paralysis of its motor ganglia;—thus dispensing for a time with this part of an inhibitory incubus, which threatens to become unmanageable through its very complexity?

THE VOLUNTARY MUSCLES.

The foregoing considerations have reference especially to the relations of nerves to involuntary muscles. Why it is that muscles of the voluntary or striated class do not also pass promptly into a state of spasm or contraction when their motor nerve trunks are cut, or when the body is dead, I am unable to explain; unless it be admitted that here the motor nerve trunks are more than mere carriers of nerve force—are in fact, with the nuclei and nerve plates at their endings, miniature magazines of nerve energy, which continue for a time to restrain the muscle after section of the nerve trunk or after somatic death.

POST-MORTEM MUSCULAR CONTRACTION.

If such an hypothesis were admitted it would serve to explain certain phenomena for which an explanation is necessary, such as the remarkable contractions of muscles which are known to occur in certain cases after death. There can be no doubt that the activity of both nerve and muscle survives for a time the death of the organism. The life of the nerve, which is more intimately dependent upon vital conditions, succumbs before that of the less vital and more enduring contractile power of the muscle (*b*). And as one fasciculus, or one muscle, or one group of muscles attains its freedom, the contraction which follows gives rise to the movements referred to.

RIGOR MORTIS.

Is a muscle contracted or shortened when it passes into rigor mortis? All observers agree that such is the case, and Dr. M. Foster tells us that the shortening and contraction "may be considerable." (*c*) Is this contraction and shortening the last act of the muscle in dying, or does it occur after the actual death of the muscle—that is, in a dead muscle? Let us consider the latter view first, since it appears to be the one in favor by our physiological teachers at the present time.

If the muscle be dead, not only is its nerve force extinct, because nerves die first, and consequently there can be no stimulus from nerve energy to cause the muscle to contract, and further, the chemical changes in the muscle which generate its contractile force must also have ceased to operate, so that its contractile power is at an end. In the assumed absence of contractile energy, it has become customary to attribute the death-stiffening to coagulation of the muscle plasma in the muscle. This would account for the rigidity of the muscle, but would fail to account for the contraction and shortening admittedly present. Muscle plasma, in the living muscle, bears the same relation to the myosin of dead muscle that certain albuminous substances in the circulating blood do to fibrin, after blood is drawn off in a vessel. According to Dr. Lionel Beale, fibrin is "non-living matter, and

(*a*) Dr. M. Foster, Phys., p. 232.

(*b*) *Ib.*, p. 121. (*c*) *Ib.*, p. 94.