On the Necessity for a Modification of Certain Physiological Doctrines regarding the Inter-relations of Nerve and Muscle.

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ON THE NECESSITY FOR A MODIFICATION OF CERTAIN PHYSIOLOGICAL DOCTRINES REGARDING THE INTER-RELATIONS OF NERVE AND MUSCLE.

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For some years past I have endeavored to bring to the notice of the profession a view of the inter-relations of nerve and muscle—more especially of the vaso-motor nerves and the arterial muscles—which is entirely at variance with what is taught in our physiological text-books.

I should be unable to find any excuse or apology for attempting so bold a task, were it not that the proofs which I have to advance are drawn entirely from the authentic storehouse of physiological research. While the facts to be here advanced are the results of observation by the great Masters in this department of science, I hope to be able to show, conclusively, that the inferences or interpretations placed upon these facts are in some instances erroneous and ought to be modified or reversed.

In the examples now to be cited of an erroneous interpretation of authentic experiments, the idea evidently dominating the physiological mind was that a stimulus from nervous energy is necessary to induce muscular contraction. As a corrollary to this idea, of course, it followed that when the motor nerve supplying a muscle was cut, or paralyzed from any cause, the muscle thus deprived of nerve influence was rendered incapable of displaying its contractile power.

That such an idea was apparently justified by the behavior of the *voluntary* muscles is undoubtedly true; but not so in regard to the non-striated or involuntary muscles of organic life, which have been pronounced by physiologists to be paralyzed and powerless, at the very moment that the observers saw and recorded the palpable evidences of their more or less active contraction. In fact, so far from the current teaching of physiology being true, as regards the relations of motor nerves to involuntary muscles, the very reverse is true ; the actual fact being that *muscles of the involuntary class, as a rule, contract, not when stimulated by their proper motor nerves, but when these nerves are cut, or are paralyzed, or dead.*

THE ŒSOPHAGEAL AND GASTRIC MUSCLES.

To come now to the facts. The statement continues to be repeated in each succeeding text book on physiology, that section of the pneumogastric nerves (vagi) is followed by paralysis of the œsophagus and stomach. Now, on the theory uppermost in the minds of physiologists—referred to above—the œsophagus *ought* to be paralyzed here, and to be reduced to the condition of a mere flaccid tube. But that such is not the case is evident from the fact that after the operation, food and drink fed to the animal, "in a few moments are suddenly rejected by a peculiar kind of regurgitation." (Dr. Dalton's Phys., p. 473.) It needs no argument to prove that the sudden rejection of ingesta, in the manner stated, so far from being an evidence of paralysis, is really a proof of active contraction in the muscle.

But it is said that sometimes the ingesta are detained in the œsophagus for a time, and, "owing to paralysis of this canal," are not conveyed into the stomach. (Ib.) Dr. W. B. Carpenter, F. R. S., refers to this by stating that "if the pneumogastric be divided in the rabbit, on each side, above the œsophageal plexus but below the pharyngeal branches, and the animal be then fed, the food is delayed in the œsophagus which becomes greatly distended." (Hum. Phys., 5th Amer. Ed., p. 404.) Now the pharyngeal branches supply the upper part, and the œsophageal plexus, the lower extremity of this muscular tube. Mark what follows on section of the vagi between these two!

The upper part of the œsophagus, whose nerves are intact, admits the food and drink apparently in a normal manner, while the lower part of the tube, which has been deprived of nervous influence, contracts upon itself, and so lessens the calibre of the "canal" as to arrest the further passage of the superimposed ingesta, as a consequence of which the œsophagus "becomes greatly distended."

Whether the ingesta are thus forcibly detained or "forcibly ejected" would appear to depend on the point at which the vagi are cut. But in either case, the result, so far from being a proof of paralysis, really bears evidence of activity of the muscle. And this is confirmed by the observation of Dr. M. Hall, that "the simple contractility of the muscular fibre [of the œsophagus] occasions a distinct peristaltic movement along the tube *after its nerves have been divided*, causing it to discharge its contents when cut across." [Italics mine.] (Dr. Carpenter's Hum. Phys., 5th Amer. Ed., p. 404.)

Dr. Burdon Sanderson expresses the idea uppermost in the physiological mind, in stating that after section of the vagi "the muscular fibres of the œsophagus are paralyzed, so that regurgitation of food from the stomach is apt to take place." (Hand-book for Phys. Lab., Amer. Ed., p. 318.)

Dr. W. B. Carpenter seems to pass over this part of the subject lightly, and it is not till treating of the effects of section of the vagi on the gastric secretions that he plainly states that, "the first obvious effects of this operation are vomiting, (in animals that are capable of it) and loathing of food." (P. 423.) He also tells us, in another place, that the reopening of the cardiac orifice, on pressure from within, is one of the first of that series of reverse actions which constitute vomiting. (P. 404.) It is evident that the "pressure" referred to and the force necessarily required to eject the contents of the stomach and œsophagus could not come from "paralyzed" muscles, which the facts show to be really undergoing active contraction.

That nerve force is actually in abeyance in the act of vomiting was fully recognized by Dr. Anstie, who places it among the effects of paralysis of the medulla oblongata in narcosis. (Stimulants and Narcotics, p. 168.) While the vomiting of migraine, he says, "marks the lowest point of nervous depression." (Neuralgia, p. 39.)

Had those eminent physiologists, Drs. Todd and Bowman, doubts of the truth of the physiological theory of the day, and a prescience of what the future had in store, when they wrote: "The office of the gastric branches of the vagi nerves appears from Dr. Reid's experiments to be chiefly to *control* the movements of the muscular coat of the stomach." [Italics mine.] (Phys. Anat., p. 493.) That is precisely what the scope of this paper is designed to show—that in so far as the involuntary muscles, at least, are concerned, the function of nerve force is not to stimulate, but to restrain and control muscular activity; which all physiologists regard as an inherent endowment of muscular tissue.

THE BRONCHIAL MUSCLES.

Dr. Burdon Sanderson informs the readers of the "Hand-Book," that after section of the vagi "the muscular fibres of the bronchial tubes are in a similar condition" to those of the œsophagus and stomach. (P. 318.] Then it is evident that these muscular bands come under the rule or law laid down above, and contract, like other muscles of this class, when deprived of nervous influence.

THE NASAL MUSCLES.

It is a curious fact, that "owing to the great size of the velum pendulum palati, the horse is unable to breathe through the mouth." (Strangeway's Veterinary Anat., p. 209.) As a consequence, respiration is carried on in this animal exclusively through his nose; and when both the facial nerves are cut, or paralyzed, "the nostrils immediately collapse, and the animal dies by suffocation." (Bernard quoted by Dr. Dalton Phys., p. 458.)

A result very similar, so far as the closure of the nostrils is concerned, has occurred in the human subject, during paralysis of the facial nerve. Thus, Sir Thomas Watson, reporting the case of the girl, Jane Smith, says: "When she tried to snuff in air through her nose, not being able to keep the right nostril stiff and open, its sides came together, and no air passed up that side." (Lectures, Prac. Physic, p. 366.

A little reflection will show that this is necessarily due to muscular contraction. The effect produced is not to be accounted for by any filling up or stuffing of the nasal passage by relaxed or paralyzed muscles, because the muscles are on the exterior of the cartilages, and mucus membrane or fibrous tissue does not contract or respond to nerve action. The obstruction is caused by the cartilages of the nose coming together, for which the only adequate explanation is the action of the constricting muscles, which, as in other similar cases, assert their power when nervous restraint is removed.

SPASM OF THE GLOTTIS DUE TO NERVE PARALYSIS.

We now come to a still more striking illustration of the truth of the proposition laid down above. The aperture of the glottis is closed by one set of muscles and opened or dilated by another. The constricting muscles are the arytenoidei and crico-arytenoidei laterales, while the dilators of the glottis are the crico-arytenoidei postici.

Dr. Burdon Sanderson states that "the widening of the glottis is a condition of general muscular relaxation." He further states that the closing of the glottis is equally due to a general contraction of all the muscles ; so that the glottis is closed, "not because the postici crico-arytenoidei muscles and the other dilating muscles* do not act with the rest, but because they are overpowered by the constricting muscles. (Hand-book, p. 308.)

The situation thus depicted becomes quite remarkable and full of interest, when it is remembered that the sole motor nervous supply to both these sets of muscles passes through the inferior laryngeal (or recurrent) nerves, a branch of the pneumogastric, and that when this nerve is cut or paralyzed, the closure of the glottis takes place, as a result of spasm of both of the antagonizing muscles, as just stated.

On page 318 of the Hand-book the same eminent physiologist, describing the effects of section of the vagi, says : "The glottis is partially closed, just as it is in death." How the glottis is closed in death will appear from the fact, vouched for by Dr. Austin Flint, in the 5th edition of his "Practice of Medicine," when he says, the operation of passing a probang within the larynx, "is extremely difficult, if it be practicable, on the cadaver." (p. 294.)

There can be no doubt about the effect of the section referred to being of a paralyzing character, so far as the nerve is concerned, seeing that the simple section of the nerve during life, and the extinction of all nerve force in death, lead to precisely the same results as regards the closure of the glottal aperture. Dr. Burdon Sanderson adds that, "in animals with divided vagi life may be prolonged by tracheotomy," showing how complete and fatal is the spasm thus produced. Other evidence of similar import is not lacking. Thus, Dr Austin Flint, discussing the "danger of death from suffocation" in the "obstructed inspiration" occurring in nervous aphonia, says : "The condition is analagous to that after the physiological experiment of dividing both recurrent laryngeal nerves." (Prac. of Med., 5th Ed., p. 309:) The same author has "reported a case in which the left recurrent nerve being situated between a calcareous deposit and an aneurismal tumor, spasm of the glottis occurred so frequently and to such an extent as to prove fatal." (Ib., p. 371.)

Now since the recurrent nerve is the only motor nerve supplying these muscles, and since section or pressure on a nerve trunk cannot increase nerve activity—the nerve trunks being mere carriers and not producers of nerve force—it is evident that no other conclusion is possible than that the spasm here referred to is due to the absence of nerve force, and not to a stimulus from excited nerve action. And since nerve paralysis is thus shown to be directly the cause of spasm of the glottis, is it not necessary to infer that whatever is done by reflex action to cause spasm of the glottis must be of a paralyzing character to the nerve also? Thus, what is vaguely called "irritation," by which is usually meant an excitation or exaltation of nerve power, and which consists really in a perturbation of nerve force, must necessarily be an influence of a paralyzing character to the nerves it traverses. Such reflex "irritations" are usually attributed to brain lesions, to indigestible food, and other causes of a more or less debilitating character which may well arrest, rather than develop, the flow of nervous activity.

If it be true, that pain is "an expression of impeded and imperfect nerve energy, not of heightened nerve function," for which there is high authority (Anstie, "Neuralgia," pp. 12 and 163,) how much more is the perturbation of the nerve molecules, which constitutes "irritation," a disturbance of normal activities which is equivalent to paralysis.

RELATION OF VASO-MOTOR NERVES TO THE ARTERIAL MUSCLES.

I propose to show here, on the very best physiological authority, that what is known as "paralytic hyperæmia" is—contrary to the accepted opinion—venous and not arterial.

I need not delay to offer proof that the middle muscular coat of the arteries is under the control of the vasomotor nerves of the sympathetic, which regulate the calibre of these tubes ; or that the chief vasomotor centre is in the medulla oblongata, with probably lesser centres in the spinal cord. These are among the well-authenticated facts of recent physiology. It is in determining the action or play of this mechanism, that I have the temerity to claim that our physiologists have made an "unscientific use of the imagination."

The theory of the text-books is that when the influence of the vasomotor centre is cut off from the arterial muscle in any way, hyperæmia of the arteries results. Thus in destruction of the nervous centres by the operation of

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* There are no "other dilating muscles" than the crico-arytenoidei postici.

"pithing"—as a result of section of the spinal cord just below the medulla, and on section of the chief vasomotor nerve trunks, in the body or viscera, it is claimed that the corresponding arteries are more or less dilated. Dr. Burdon Sanderson contents himself with stating that under these circumstances, "the arteries are relaxed," and again, that they "become permanently larger." (Hand-book, pp. 245-256.) Other physiological teachers, such as Prof. Kuss, say that here the arteries are "dilated," while Dr. Sidney Ringer, in his excellent "Therapeutics," has it that "the arteries remain widely dilated." (5th Amer. Ed., p. 312.) We shall presently see how far these statements are justified by the facts.

SECTION OF THE CERVICAL SYMPATHETIC.

To M. Claude Bernard and Dr. Brown-Sequard we are largely indebted for what is known on this subject, as observed by them in the famous experiment on the cervical sympathetic. Dr. Brown-Sequard enters into the details at great length in his "Physiology and Pathology of the Central Nervous System." Yet nowhere in this work, in regard to this or any other section of cord or nerve, does he once assert that the arteries are dilated. In the pages devoted to it he refers to the contemporary experiments on this subject by Waller, Donders and his pupils, by Kussmaul and Tenner, Moritz and Schiff, yet he makes no mention of an allusion to dilated arteries by any of these eminent observers. This is surely significant. With him it was always "the blood-vessels" which are "paralyzed" and "the blood-vessels" which are "dilated." He says that "the hanging down of an animal, by holding it up by its hind legs, in producing a congestion of the brain, produces very nearly all the effects of this section." (P. 143.)

From these considerations it will be evident, first, that it was by no means apparent—was indeed a matter of great difficulty to determine accurately what particular "vessels" were enlarged, hidden as they mostly were beneath the skin and its subjacent tissues. Nay, it is not too much to say, that the statement that it is the arteries that are enlarged is purely hypothetical, and not based upon an actual demonstration of the facts. Secondly, it will be also evident from the statement just quoted from Dr. Brown-Sequard, that venous hyperæmia, the result of the blood being forced out of the arteries by their partial contraction, "very nearly accounts for all the effects of this section." The truth of this will not only appear from what is to follow now, but from the effect of other sections to be noted.

Notwithstanding an increased afflux of blood, and consequently a relative elevation of temperature, with heightened sensibility, "the intimate acts of nutrition appear to be modified in nothing. * * * Nor does it appear that this hyperæmia, however intense or prolonged it may be, has ever the effect, save under exceptional circumstances, of determining by itself the development of inflammatory action." (M. Charcot, Lect. Nerv. Sys., pp. 90-91.) This could hardly be the case if the hyperæmia were arterial.

Among the effects of this section on muscles, as recorded by Dr. Brown Sequard, are contraction of the pupil, retraction of the eye-ball, partial closing of the eye-lids, contraction of "almost all the muscles of the eye," and also of the muscles of the angle of the mouth and nose; contraction of the erectile muscles of the ear, and others. Now, seeing that it is *contraction*, and not relaxation of all these muscles, which follows section of this nerve, the law of analogy would require that the muscles of the arteries supplied by this nerve be contracted also; otherwise the anomaly would exist of the same nerve producing contraction in a large number of muscles and relaxation in a single instance. Why should the arterial muscle be regarded as an exception among so many others, especially when all the facts of the case are compatible with arterial contraction and venous fullness?

As for the second part of the experiment, in which the hyperæmia is dissipated by faradization of the distal end of the cut nerve, that is easily accounted for. The terminal branches of the cut sympathetic evidently influence the muscles of the head and face over a wide area. As is well known, the effect of faradization is to set up a succession of rapid contractions and relaxations in muscular tissue. The pressure thus brought to bear on the swollen veins would amply suffice to force their contents onwards, and thus to dissipate the venous congestion. Examples of this very result are not lacking. Thus when Kolliker applied one pole to the umbilical artery and vein of a fresh human placenta, there followed contractions by which the veins forced out their contents and changed into bloodless strings." (Meyer's Elec. Hammond, p. 88)

The following quotations from Rosenthal's "Diseases of the Nervous System," Vol. II., Wood's Library, have a peculiar fitness here: "Kussmaul and Tenner have shown in a series of experiments, by placing a watch-glass in the opening of a trephined skull, without allowing the air to enter (Donder's plan,) that compression of the carotids causes capillary anæmia and venous hyperæmia of the brain and meninges." (P. 64.)

"In Verneuill's patient, upon whom ligature of the carotid was performed for a tumor of the parotid gland, persistent contraction of the pupil developed shortly afterwards, with rise of temperature and vascular dilatation upon

the temple and gums, and abundant perspiration upon the side of the face, corresponding to the operation. All these symptoms can be produced experimentally upon animals by dividing the cervical sympathetic." (P. 26.)

Here is a remarkable proof that the section referred to causes arterial contraction (and not dilatation,) seeing that the other effects of the section are equivalent to those produced by ligature of the carotid.

SECTION OF THE SPLANCHNICS.

In a "demonstration of the vasomotor functions of the splanchnics nerves," the chief editor of the "Hand-book for the Physiological Laboratory" (Amer. Ed., p. 258), informs his readers that these nerves contain vasomotor fibres which "are distributed to the arteries of the abdominal viscera."

We approach this "demonstration" expecting to find that when these nerves are cut the predicted results will follow in the arteries they supply being more or less "relaxed" or "dilated." What is our disappointment to find in all that follows in this chapter of the "Hand-book," the arteries are never once alluded to! Thus the very pith and point of the so-called "demonstration" is entirely ignored! What occurs is thus stated by Dr. Burdon Sanderson: "After section of both nerves the vessels of all the abdominal viscera are seen to be dilated." What "vessels" are these? Not the arteries, because Dr. B. S. continues: "*The portal system is filled with blood;* the small vessels of the mesentery and those which ramify on the surface of the intestine are beautifully injected; the vessels of the kidney are dilated, and the parenchyma is hyperæmic; all of which facts indicate, not merely that by the relaxation of the abdominal blood-vessels, a large proportion of the resistance to the heart is annulled, but that *a quantity of blood is, so to speak, transferred into the portal system, and thereby as completely discharged from the systemic circulation as if a great internal hemorrhage had taken place.*" (P. 260.) [Italics mine.]

It needs no italics to give point and force to this remarkable admission. It is merely stating, with a little circumlocution, that the arteries are empty and the veins are full! The "beautiful injected vessels," which the learned editor so much admired, are not arteries but veins, the blood in which has become "bright red, like arterial blood," as Prof. Kuss explains of venous blood in the mesentery, "because oxygenation has been effected simply by exposure to the air." (Lec. Phys., p. 326.)

The contraction and emptiness of the arteries, after section of their vasomotor nerves, is thus proved on the very highest authority. Where now is the justification of the assertion that after a section of this kind the arteries are dilated and hyperæmic?

Whatever obscurity there might be as to the actual results of section of the cervical sympathetic, for obvious reasons, there can be no mistake as the results here.

Now the law of uniformity of cause and effect, demands that what is true of the relative state of the arteries and veins after section of the splanchnics, must be true also after section of the cervical sympathetic;—and since the arteries are thus shown to be empty and the veins full in the former case, the same condition must be held to prevail also in the latter.

It is worthy of note, in this connection, that both after section of the spinal cord, and after section of the splanchnics, blood pressure falls, and in both cases may be restored by faradization of the divided cord or nerve. It is evident from this, that the fall of blood pressure (as shown by the kymograph in the carotid) on section of the cord, is not to be regarded as an indication of arterial relaxation, as appears to have been done; because blood pressure fell also after section of the splanchnics, where we know positively that arterial dilatation could not have taken place.

It may be asked, how could faradization of the spinal cord or of the nerve, restores the pressure or tension in the arteries, if the heart and arterial system were already empty? Dr. Burdon Sanderson supplies the answer, indirectly, in stating: "It is seen that after section of the cord the heart is flaccid and empty, and that its cavities fill and its action becomes vigorous, when the vascular contraction caused by excitation of the peripheral end [of the cut cord] forces the blood forward so as to fill the right auricle." (P. 251.) Now the only blood which could be "forced forward so as to fill the right auricle," is *venous* blood from the distended portal system. Thus it will be seen that all the facts fit, and as it were, dovetail into each other, in establishing that nervous paralysis and contraction of the arterial muscle go together the result being hyperæmia, not of the arteries but of the veins.

The explanation just quoted from the Hand-book, as to the forcing forward of the venous blood, as an effect of the faradic current, confirms the explanation made above, as to the dissipation of the venous hyperæmia by the same current after section of the cervical sympathetic.

STATE OF THE ARTERIES IN DEATH.

Not only are the arteries invariably as empty as their physical structure will permit them to be, when their nerves are cut or paralyzed in the living body, but such is also their condition *in death* of the body, when nerve force is extinct. This is a fact too well known to need any special proof. It is a fact, however, which ought to be explained by those who hold that in a condition of nerve paralysis the arteries are "dilated" and hyperæmic.

THE OPERATION OF PITHING.

What has just been said of the contracted and empty state of the arteries is true also after the operation of "pithing" (in which the medulla and spinal cord are destroyed); as any one can easily satisfy himself, as I have done, by actual experiment. This is inadvertently proved to be the case by Dr. Burdon Sanderson in his account of an experiment designed to prove the contrary. Two frogs are taken. One is "pithed," in the other the nervous centres are uninjured. In both the heart is carefully exposed and the single ventricle slit open, so as to show the state of the great vessels. The experiment is intended to prove that in the pithed frog the arteries are "relaxed" and full of blood. On Dr. Burdon Sanderson's showing, the results are these: In the pithed frog, "although the heart is beating with perfect regularity and unaltered frequency, it is empty, and in consequence, instead of projecting from the opening in the anterior wall of the chest, it is withdrawn upwards and backwards towards the œsophagus." The heart and its appendages "are alike deprived of blood;" but on opening "the rest of the visceral cavity," "*the intestinal veins are distended.*" In these, "the whole mass of blood has come to rest, *out of reach of the influence of the heart.*" (P. 246.) How significant is this! If the arteries were dilated, and consequently full of blood, this blood could not be said to be "out of reach of the influence of the heart." But this is not all. The Hand-book continues: "In the frog deprived of its central nervous system *only a few drops of blood escape*—the quantity, that is to say, previously contained in the heart and in the beginning of the arterial system. In the other, *bleeding is not only more abundant but continues for several minutes after the section.*" (Pp. 246-296.) [Italics mine.]

Is it not evident that in the case of the pithed frog, the arterial system promptly emptied itself into the now "distended veins," and had "only a few drops of blood" left to drain away through the open ventricle (the frogs being both suspended); while in the case of the other frog, whose nervous system was intact, this arterial contraction did not take place, and the arteries continued to bleed for several minutes till drained of blood.

The "Hand-book for the Physiological Laboratory," from which I have quoted so often, occupies to-day a leading place as an exponent of physiological science. The reader who studies the details of the experiment just quoted will be surprised to find, that here again, in an experiment specially designed to prove that "all the arteries are relaxed," the condition of the arteries is completely ignored, and never once alluded to! The arteries *ought* to be "relaxed," "dilated," and even "widely dilated" here, on the theory of the text-books, but they are empty and contracted, their final act being, as in death from other causes, "to drive their contents into the veins." (Kuss. Phys., p. 181.)

AN EXPERIMENT OF DR. BROWN-SEQUARD.

In this connection I must notice in the briefest manner, an experiment of Dr. Brown-Sequard in which the doctrine here supported is confirmed in a remarkable manner.

In a dog, a section was made of a lateral half of the spinal cord just below the medulla. The result was, extreme hyperæmia of the "blood-vessels," to use Brown-Sequard's term, of one posterior limb, while the "blood-vessels" of the other posterior limb displayed a state of spasm and ischæmia quite as extreme. "Very often the spasm persists for days," wrote the observer, "and it may be so great that the circulation is almost entirely suspended," so that "the cutting of the skin hardly gives a drop of blood."

The question at once arose, was the paucity of blood in one limb due to the excess of blood circulating in the other, or *vice versa*? Was the spasm on one side, or the dilatation on the other, the primary or direct effect, through the spinal vasomotor nerves of the half section of the cord?

In order to solve this question, Dr. Brown-Sequard made "direct experiments." Among others he ligatured the iliac artery feeding the dilated blood-vessels of the hyperæmic limb, thus directing "almost the whole of the blood coming from the aorta" into the iliac artery of the limb in which the circulation was so much diminished. Notwithstanding this, the spasm was but partially overcome: "the temperature rose but little;" and "it was quite evident the small arteries near the toes did not allow the blood to pass freely."

Here was complete evidence, not only that there was spasm, but also that this spasm was arterial. Although the vasomotor mechanism of the spinal cord is as yet only very imperfectly understood, there seems no reason to doubt

that this active contraction of the arterial muscle was here, as elsewhere, due to nervous paralysis, the result of the half section of the spinal cord.

MORE ABOUT THE ARTERIAL MUSCLES.

It will be obvious that the relative state of the arteries and veins in the foregoing experiments is incompatible with what M. Charcot calls "the paralytic dilatation" of the arteries, as a result of vasomotor nerve section, and could not occur, if after this section the arteries remained "widely dilated," and "permanently larger," as asserted by other authorities already quoted. If this were the condition of the arteries, it is evident that they would be wholly incapable of contracting upon their contained blood, so as to force it forwards through the capillaries and into the veins; an act depending entirely upon arterial contraction, because the force of the heart has already expended itself, and the capillaries have no muscular walls; while, that the veins are merely passive, is shown by the fact they have no vasomotor nerves, and their calibre is not, as in the case of the arteries, regulated by nerve influence. (Dr. M. Foster's Phys., pp. 265-263.) Thus all the facts show that the arteries, so far from being "dilated" and "paralyzed" are undergoing active contraction.

Some recent authorities appear to suggest the modified idea that the dilation of the arteries, instead of being "permanent," as alleged by some authorities, is a temporary effect—"an opening of the flood-gates," so to speak, in order to facilitate the transmission of blood to the veins. Thus Dr. M. Foster writes: "The section of the splanchnic nerves causes the mesenteric and other abdominal arteries to dilate, and these being very numerous, a large amount of the peripheral resistance is taken away and the blood pressure falls accordingly; a large increase of flow into the portal veins takes place and the supply of blood to the face, arms and legs is proportionately diminished." (Phys., 3rd Amer. Ed., pp. 240 and 220.)

It would appear that here, as elsewhere, "the fall of blood pressure" is regarded as evidence of "lessened peripheral resistance," and a proof that the arteries are "dilated," the fallacy of which will presently appear.

We read again: "When the nervous system is destroyed, dilation of the splanchnic vascular area causes all the blood to remain stagnant in the portal vessels; and probably these as well as other veins are rendered unusually lax, so that the blood is largely retained in the venous system, and very little reaches the heart." (Ib., p. 367.) And further: "When in the frog, the brain and spinal system are destroyed, very little blood comes back to the heart, as compared with the normal supply, and the heart in consequence appears almost bloodless and beats feebly . . . the veins become abnormally distended and a large quantity of blood becomes lodged and hidden as it were in them." (Ib., p. 263.)

Here is the secret, both of the emptying of the arteries and of the fall of blood pressure. The blood comes to rest in the more capacious venous system, (Ib., p. 154,) "out of reach of the influence of the heart." Now seeing that the rapidity of the arterial circulation is such that only one-seventh of a second is required for blood to pass from the heart to the radial pulse, how long, think you, would be required to empty the arterial system of the pithed frog, seeing that at first little blood, and very soon no blood, finds its way back through the heart, into the arterial trunks? Why, the time required would be counted by seconds rather than by minutes. There would be no time and no necessity for the terminal arteries to dilate; the emptying of the arteries and the fall of blood pressure being amply accounted for by the fact that *blood is passing out of the arterial system faster than it is being returned to it.*

A precisely similar condition to that just described as resulting from nerve destruction, occurs also in the fatal stage of asphyxia. Here, too, the arteries are "contracted" and empty, and the large veins are so distended that "if cut into they spirt like arteries." (Dr. Burdon Sanderson, Hand-book, etc., p. 332.) And here also, Dr. M. Foster tells us there is a fall of blood pressure in the midst of general arterial contraction. He says: "On account of the increasing slowness and feebleness of the heart, the blood pressure, in spite of the continued arterial contraction, begins to fall; since less and less blood is pumped into the arterial system." (Phys., p. 445.) It will be seen that the parallel between the two cases is complete, and that the plain facts, as given by the highest authorities, do away completely with the assumption that, here, the fall of blood pressure is to be regarded as a proof of arterial relaxation.

Even in the slower forms of death, when the process of emptying the arteries is more gradual, there is still no evidence of, and no necessity for, a dilation of the terminal arteries to give exit to the blood; for, granting that contraction of the terminal arteries would tend to hinder the outflow of blood, this effect would be counteracted by the stronger contraction of the larger arterial trunks above, forcing the blood through and out of the numberless terminal branches ending in the capillaries.

The facts thus far presented refer only to the great vasomotor areas of the cervical sympathetic and splanchnics. It seems unnecessary to attempt to discuss the lesser and local vascular mechanisms, about which little is known, and that little comes to us under the ægis of an erroneous theory. The greater always includes the less. What happens when the life of the chief nervous centres is killed, either by sudden and intended destruction, or in death from ordinary causes, happens also in a more limited area when local or subordinate centres are killed or paralyzed. Since in the former case the arteries are found contracted and empty, the same rule must be held to hold good in the case of the individual nerve and artery.

THE STIMULATION (?) OF ASPHYXIA.

Is it not a strange proposition to put forward in the name of medical science, that an animal dying of asphyxia is actually undergoing a high degree of nervous excitation. Yet such is actually the teaching of the text-books in physiology to-day! Dr. Burdon Sanderson treating of asphyxia says: "One of the effects of diminishing the proportion of oxygen in the blood is to excite the vasomotor centre, and thus to determine general contraction of the small arteries. The immediate consequences of this contraction is to fill the venous system." As the process advances "the heart's contractions become more and more ineffectual till they finally cease, leaving the arteries empty and the veins distended." (Hand-book, etc., p. 333.)

There is no mention here of arterial relaxation or dilation, to facilitate the outflow of blood. On the contrary "the immediate consequences" of "a general contraction of the small arteries" is "to fill the venous sysem," and in a few minutes "the arteries are empty and the veins dilated," the animal being dead. This is precisely the condition which we have seen in a former page to be the direct result of destruction of the nervous centres. It is a process which invariably prevails in the dying and is complete in death. Thus according to Paul Bert, quoted by Prof. Kuss, "death is always owing to asphyxia." (Phys., p. 330.)

Why has it been assumed by physiologists that in this rapid sinking into death the nervous centres are undergoing an unusual excitation? Because as we have just seen, there is "a general contraction of the small arteries," and other spasms and contractions of the respiratory muscles fixing the chest and arresting respiration; and in accordance with the theory of the day, these spasms and contractions of the muscles, depend on active discharges of nerve force, stimulating the muscles to contract. How is this assumed extraordinary activity of the nerve centres to be accounted for in an animal actually dying? There is a "physiological law" which declares that the activity of an organ is directly dependent upon its receiving a due supply of arterialized blood (Dr. C. B. Radcliffe); and Dr. W. B. Carpenter has said of venous blood, that "it exerts a depressing influence upon the nervous centres," from which they are at length "completely paralyzed." (Hum. Phys., p. 537.)

One would have imagined that bad blood, deficient in oxygen and loaded with carbonic acid, would have been the very last thing which a physiologist would have chosen, as a pabulum from which to generate an excess of nerve force! and doubtless the choice was embarrassing enough. But necessity compels. The exigency of the theory is inexorable. Muscular contraction without nervous stimulation is deemed impossible, and there being nothing else to fall back upon, it has been assumed that impure, non-arterialized blood plays the part of a stimulant to the nervous centres.

Accordingly we find a recent and popular writer—Dr. J. Milner Fothergill—in his "Antagonism of Therapeutic Agents," declaring that "the more venous the blood the greater the activity of the respiratory centre. The effect of venous blood is to augment the natural explosive decomposition of the nerve cells. . . . The effect of defective arterialization causes more rapid as well as deeper breathing; more perfect and extensive respiration is set up until properly oxygenated blood is procured." This author would almost lead one to believe that a kindness was done to the rabbit in having its vagi cut. He says, "When the vagi are cut, the respiration is modified; it becomes deeper and more prolonged, fuller and more complete." (P. 88.)

But unfortunately this view of an apparently improved respiration is wholly delusive; for, as Dr. Burdon Sanderson tells us, "notwithstanding the vigor of the respiratory movements, the blood becomes more or less venous,"—the animal is dying, and does die, "commonly before the end of the first day." (Hand-book, p. 317.)

Let it be kept in view that the theory of the day explicitly teaches that "the muscles receive from the nervous system a preternatural stimulus to action" (Dr. Pereira, Vol. 2, p. 541,) and that spasm and convulsion "are dependent upon excessive activity of the spinal centres:" (Dr. W. B. Carpenter, Ib., p. 846,) and we shall see presently to what apparent absurdity this doctrine has led. In one of Kussmaul and Tenner's experiments, the carotid arteries are ligatured with the effect of inducing "immediate loss of consciousness and general and violent convulsions," which are promptly recovered from, and nervous control over the muscles restored, as soon as the ligatures are

untied and blood is admitted to the brain. Dr. M. Foster's view of this experiment is, that here "the nervous centres being no longer furnished with fresh blood, become rapidly asphyxiated through lack of oxygen." And yet strangely enough he holds that in this almost fatal condition of "rapid asphyxiation" the nervous centres are undergoing stimulation! for he adds: "similar anæmic" convulsions are seen after sudden and large loss of blood from the body at large; the medulla being stimulated by the lack of arterial blood." (Phys., p. 441.) Surely such a view as this may be gravely challenged, even when put forward on high physiological authority! Dr. M. Foster remarks in another page, in his chapter on "Death," that "blood is not only useless but injurious unless it be duly oxygenated." (P. 833.) And again he says of venous blood that if it "continues to be driven through a muscle the irritability of the muscle is lost even more rapidly than in the entire absence of blood. It would seem that venous blood is more injurious than none at all." (P. 126.) Why should nerve function be augmented by what is useless and injurious, not only to muscle, but to every other tissue in the body?"

THE CHEYNE-STOKES RESPIRATION.

What seems a lower depth of absurdity, if possible, has yet to be reached in the explanations of the Cheyne-Stokes respiration. I quote here from Dr. L. Sansom's "Physical Diagnosis of the Heart," by whom Traube's theory on this subject is said to be "the most plausible." According to Traube, "the first thing which occurs is the establishment of a condition of impaired irritability of the respiratory centre through mal-oxygenation; the long respiratory arrest gives time for the accumulation of carbonic acid in excess in the blood. Arrived at a certain maximum this begins to stimulate, slowly and imperfectly at first and afterwards in increasing degrees, the centre, so that it developes the respiratory efforts till they culminate in dyspnœa. Then as the centre ceases to be stimulated or becomes exhausted, dyspnœa again supervenes." (P. 37.)

It will be observed that here the *deficiency* of oxygen and subsequently the *presence* of carbonic acid are made to play opposite and antagonistic parts! The lack of oxygen (instead of stimulating the medulla, as supposed by Dr. M. Foster), first enfeebles the respiratory centre, in the medulla, and then, the same blood, still deficient in oxygen, but now loaded with carbonic acid, counteracts the previous depression, and tones up the weak nerve centre, so that, ere long, it displays extraordinary activity. But, unfortunately, this exhilarating pabulum—carbonic acid—is soon exhausted, and the nerve centre resumes its former feebleness till a new supply can be procured. The physiologist is certainly quite impartial, and allows the rivals to have their "innings" turn about. How such nonsense as this "most plausible theory" could find a place in physiological literature seems explicable only on the exigency of the hypothesis so long in vogue.

Filehne's theory in explanation of this state is more complicated, and at least equally absurd. Instead of the respiratory centre being stimulated (as Traube says), it is the vasomotor centre which is excited by the presence of carbonic acid. Arterial contraction follows, till "a gradually increasing anæmia of the respiratory centre" is brought about. This anæmic condition excites the respiratory centre "and inspiration becomes more and more deep," till oxygen is supplied to the blood; "the arterial spasm is thus relieved," owing to the freshly oxygenated blood failing to stimulate the vasomotor centre (so as to contract the arteries), as the carbonic acid had previously done. With the relief of arterial spasm, and a consequent normal dilation of the arteries, "the anæmia of the respiratory centre passes off, and with it the exaggerated impulse to respiration, and breathing once more becomes superficial." (P. 137.) In other words, the respiratory centre functionates best when it is supplied not only with non-arterialized blood, but when it has too little even of that; as soon as the anæmia passes off, and this nervous centre gets a fair supply of blood, it ceases to act—suspends business—till the better times of bad blood and deficient blood come round again, when it is moved to activity once more!

There is still another explanatory theory to be noticed, which I find referred to editorially in the *Canada Lancet* for February, 1886: "Bramwell, who follows the teaching of M. Foster and others, supposes that the respiratory centre consists of two portions, one accelerating (or motor), and one inhibitory. He further believes that these two portions are acted on in opposite directions by the blood, whether arterial or venous. Thus while venous blood stimulates the discharging cells of the centre and depresses the inhibitory portion, arterial blood acts in exactly the opposite direction."

"At the close of the period of apnœa, the discharging portion of the centre is stimulated by the venous blood," with its excess of carbonic acid, and this same blood, at the same time, is depressing the rival, or inhibitory part of the centre. The motor or discharging portion of the centre triumphs; respiration becomes established and even exaggerated. Unhappily the victor fails to "hold the fort." As soon as the blood becomes "fully oxygenated," the "in-

hibitory portion becomes stimulated, and gradually overpowers the discharging portion," so that "the respirations grow weaker and weaker until the state of apnœa results." Then the suspension of breathing restores the venous character of the blood and accumulates a store of carbonic acid, the stimulation of which reanimates the centre previously depressed by the presence of oxygen in the blood Such appears to be the scope of this theory.

In this, as in the previous explanations, arterial blood is made to play the part of a depressor and paralyzer of the respiratory process, which it is constantly tending to arrest; but while paralyzing one portion of the respiratory centre it is stimulating another; and a similar double character is attributed to the action of venous blood. Thus during the brief time from the beginning of apnœa to the culmination of dyspnœa—a period rarely exceeding one minute—the blood passing to the brain is called upon to exert four different and even diverse effects; first as venous blood stimulating one part of the respiratory centre and paralyzing another portion of the same centre: reverse effects being produced a few seconds later by the same blood on its becoming oxygenated.

One is really at a loss to understand how such an explanation could have been admitted to a place in physiological literature. Again it is the exigencies of an erroneous theory which have led to such a complicated and unsatisfactory hypothesis.

If it be asked how the state of apnæa is induced by forced vigorous respirations, if it be not due to an excess of oxygen introduced into the blood, and how the opposite condition, or demand for air by breathing, seems to attend the absence of oxygen and the presence of venous blood, I can only answer as to the last that if no better explanation than that venous blood is a stimulant has yet been found, some better explanation is surely to be looked for. And as to the state of apnæa referred to, I find Dr. Austin Flint stating that "according to Hoppe-Seyler, apnœa, in the limited sense above mentioned, is to be attributed, not to an excess of oxygen in the blood, but to fatigue of the respiratory muscles." (Prac. of Med., 5th Ed., p. 70.)

A NEW THEORY SUGGESTED.

Dr. Sansom regards the condition of the respiratory centre in this case as one of paresis and direct exhaustion. He shows that during the apnœal period "the arteries are strongly contracted." The proof of this is found in the rise of arterial tension; in the depression of the "great fontanelle" of the head, and also in the arrest of the process by the inhalation of nitrite of amyl, which dilates the arteries. On the theory of these pages, arterial contraction is due to vasomotor nerve depression or paralysis; and accordingly we find here that the vasomotor centre, as well as the respiratory centre, is depressed in function. It has been amply shown above, that contraction of the arteries occurs in the dying and is complete in death. It is also one of the prominent phenomena during the last stages of asphyxia and is invariably attended by venous fullness. The condition present during the stage of apnœa in the Cheyne-Stokes respiration, with its contracted arteries and dilated veins, appears to correspond very closely to that present as death approaches and in the latter stages of asphyxia. The original paretic and exhausted condition of the respiratory and vasomotor centres is aggravated by the further depression caused by mal-oxygenation of the blood; which, when venous and loaded with carbonic acid, is invariably a depressing, and never a stimulating agent to nerve function. Vasomotor nerve failure induces contraction of the arterioles, systemic emptiness and venous engorgement, as the foregoing examples abundantly prove; and as a consequence, the great mass of the blood "becomes lodged and hidden as it were" in the great venous trunks. At that moment death is very near, but as the heart continues to beat, it is fair to assume that a small quantity of blood still finds its way through the lungs, and, from its very scantiness, is capable of being aerated by means of the exchanges of gases still going on in the lungs, owing to the presence of residual air, during the temporary, partial or complete arrest of respiration. As a consequence, the quantity of blood reaching the nerve centres, though small, is at least partially oxygenated, and serves to revive the function of these centres "imperfectly at first," but with momentary improvement.

The effect of this revival on the vasomotor centre, is to facilitate the dilatation of the arterioles; in which the pulmonary vessels share, permitting, ere long, the inrush of venous blood from the distended vena cava and portal system, and its transmission onwards through the heart and lungs.

This corresponds to the period of increase in respiratory function, in which the laborious efforts of a feeble mechanism have been mistaken for an "exaggerated impulse" from excited and overacting or "exploding" nerve centres.

Meanwhile, impure blood from the venous reservoirs (finding an entrance through the now fairly dilated pulmonary vessels,) begins to fill the lungs in such a quantity (as it is drawn onwards by an inequality of pressure, towards

the as yet unfilled arteries), that the whole mass of blood, failing to be arterialized with sufficient rapidity, again becomes unfit for the maintenance of nerve-function and the perpetuation of processes depending upon it.

In such a case, a previously weak organ or centre is the first to suffer. The medulla oblongata is such an organ in this case, and its contiguous centres for respiration and circulation fail together; bad blood and deficient blood, acting on centres previously paretic, or enfeebled, have done their work, and again the respiration is suspended. The vasomotor centre is again so functionally weakened that it loses control of the arterial muscle—the "inherent contractile force," which all physiologists assign to muscular tissue, thus freed (as in the examples enumerated above), induces "the strong arterial contraction" referred to by Dr. Sansom, which contraction of the artery is all the stronger the nearer nerve force is to cease in the extinction of life.

This arterial, or systemic contraction, again empties the lungs and refills the venous reservoirs, from which the blood is again drawn, at first slowly and then again more rapidly, as the process repeats itself.

Here, then, is an explanation of the Cheyne-Stokes respiration based upon sound, though as yet unacknowledged, physiological principles, according to which paretic and enfeebled nerve centres are helped by their appropriate pabulum—oxygenated blood—and are overwhelmed and have their function suspended by what is naturally calculated to poison and paralyze them, impure, venous blood, deficient in oxygen and loaded with carbonic acid.

THE INTESTINAL AND UTERINE MUSCLES.

In sustaining the contention that, as a rule, muscles of the involuntary class contract, not when stimulated by their appropriate nerves, but when deprived of nerve energy, I have not yet alluded to the involuntary muscular fibres of the intestines and uterus. The antagonism of nerve and muscle is not here so evident as in the cases already cited, but here the relations of nerve and muscle have not as yet been completely investigated. (Dr. J. Brunton.)

Dr. M. Foster states that section of the vagi "renders difficult the passage of food along the œsophagus," and causes "a spasmodic contraction of the cardiac orifice of the stomach; in other words, the tonic action of the sphincter is increased"; (Phys., pp. 346, 347),—facts which sustain what has been already stated above as to the non-paralization of the muscles concerned, after section of their nerves. The peristaltic movements of the intestine, he states, may occur "wholly independent of the central nervous system," and are "at bottom automatic," (p. 348). We have it on the authority of the late Dr. W. B. Carpenter, F.R.S., that "the intestinal tube from the stomach to the rectum is not dependent upon the nervous centres either for its contractility or for its power of exercising it, but is enabled to propel its contents by its own inherent powers." (Hum. Phys., p. 410). So also of the uterus, the contractions of which are not due to a reflex activity of the spinal cord, but to its own inherent power of contraction; parturition having taken place after destructive injury and paralysis of the cord, and even after somatic death. (Ib., pp. 979, 980). In these cases, also, the nerve would seem to be useless as the ally of the muscle, but would play an important part in controlling and regulating, by antagonizing, its contractile energy.

I must notice, in this connection, an observation of Dr. M. Foster regarding the bladder. He says:—"The escape of the fluid [from the bladder] is, however, prevented by the resistance offered by the elastic fibres of the urethra, which keep the urethric channel closed. Some maintain that a tonic contraction of the sphincter vesicæ aids in, or, indeed, is the chief cause, of this retention. The continuity of the sphincter vesicæ with the rest of the circular fibres of the bladder suggests that it probably is not a sphincter, but that its use lies in its contracting after the rest of the vesical fibres, and thus finishing the evacuation of the bladder. On the other hand, the fact that the neck of the bladder can withstand a pressure of twenty inches of water so long as the bladder is governed by an intact spinal cord, but a pressure of six inches only when the lumbar cord is destroyed or the vesical nerves are severed, affords very strong evidence in favor of the view that the obstruction at the neck of the bladder to the exit of urine depends upon some tonic contraction maintained by a reflex or automatic action of the lumbar spinal cord." (Phys., p. 448).

But this experiment admits of a very different inference. We have just seen, on the authority of Dr. M. Foster, that section of the chief motor nerves of the stomach "increases the tonic action of the sphincter" of the stomach, as we had before seen it does of the entire contractile tissues of that viscus. We have a right to look for a similar increase of tonic contraction in the bladder, when deprived of its nervous connection with the spinal cord, or when the latter is paralyzed. Admit that here, as in the examples cited above, the spinal nerves exercise a restraint over the contractile fibres of the bladder, tending to prevent its contraction. With this restraint intact, the bladder

is able to bear a pressure of twenty inches of water before the sphincter is overcome; whereas, with nerve influence withdrawn by section or paralysis, and the muscular fibres of the bladder set free to contract, (as in the case of the œsophagus and stomach), the resistance at the outlet, though also relatively increased, is overcome by the superior expelling force from above, with the aid of only six inches of water pressure.

The same principle applies to involuntary discharges from the rectum, which Drs. Todd and Bowman say is due not to paralysis of the sphincter, against which the fæces are driven, but to the "active pressure of the parts above which are not paralyzed." (Path. Anat., p. 180) The "parts above" are the intestinal muscles, which, in the last stages of exhausting disease, (when such discharges usually occur), have attained their freedom, just as the arterial muscles do under like circumstances, owing to the general decadence of nervous energy.

VOMITING OF PREGNANCY.

With the evidence before us as to the contraction of the gastric muscle on severance of its nerves, vomiting in general may surely be regarded as due to nerve depression rather than to nervous excitation. An additional observation in proof of the same is to be found in the fact that injury of the vagus may produce constant vomiting, (Bryant's Surgery, Amer. Ed., p. 208); and further, that vomiting is mentioned by Dr. C. Bastian among the symptoms of hemiplegia. (Brain Disease, p. 56.) An explanation of the vomiting of pregnancy would be found if we might assume that a monopoly of nerve energy was being expended in the uterus, owing to the extraordinary development taking place in that organ, thus starving the gastric nerves, so to speak, which, no longer able to restrain the gastric muscle, permit the untimely and abnormal contractions of that viscus. That this occurs chiefly in the early months of pregnancy might be accounted for by the unusual demand rather suddenly made upon the nervous resources, which tend to equalize their expenditure, as the months go on, and the organism becomes accustomed to its new condition.

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." (Rational Therapeutics, p. 52.) 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 play 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 imper-

fectly." 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." (Therapeutics, 5th American Ed., p. 499) 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." (Dis. Nerv. Syst., p. 539.)

Dr. Ringer further furnishes strong evidence that paralysis, and not over-action, is the condition of the nerve centres in tetanus. He instances "certain poisons, like gelseminum and buxus sempervirens, which produce *at the same time* both weakness of natural co-ordinated reflex action, cord paralysis and *tetanus*." He says "it is impossible that the tetanus should depend on stimulation of the cord, for we have seen that the tetanus was preceded by considerable depression of the cord and continues until the depression ends in extinction of all cord function"; or, as he says again, the tetanus "occurred in a dying cord." (London Lancet, Feb. 17, 1887, p. 228; Braith Retros., July, 1887, p. 98).

In strychnia poisoning, death occurs from asphyxia, (Fothergill, Antag. Ther. Agents, p. 55), with its contracted and empty arteries and engorged veins;—the precise condition of the vascular system produced by destruction of the spinal cord, as in pithing, as already shown in a previous page. Do not the foregoing facts shew that strychnia does not kill as a stimulant, or excitant, of the spinal cord? Moreover, medical literature clearly shows the value of alcoholic stimulants in strychnia poisoning, but I cannot delay to quote it. On the other hand, chloral hydrate, which has some reputation in these cases, is "not by any means antagonistic" to the action of strychnia. It acts by simply lessening the contractile energy of the muscles, like other anæsthetics, by de-oxidizing the blood, and thus retarding the chemical processes in the muscle, whereby its contractile force is generated. In this way the convulsions are arrested, and time gained for the elimination of the poison. But dangerously large doses—seven or eight grammes—(about two drachms)—are required for this purpose. (Lyman's Anæsthetics, Wood's Library, pp. 264, 267, 275.) "Strychnia affects paralyzed, sooner than unparalyzed muscles," writes Dr. Ringer: but this is not exact. Strychnia does not affect the muscles at all, as Dr. R. himself shows; and the muscles are not paralyzed in the cases to which he refers. What he means is that strychnia induces twitches and spasms in muscles whose nerves are enfeebled, sooner than in muscles whose nerves are acting normally. Why is this? If strychnia were a stimulant, would it not sooner excite vigorously acting nerves than enfeebled ones? But since its effect is to cause "depression of the motor nerves," nerves already suffering in this way have their vital activity more easily extinguished, and their muscles set free, than is the case with healthy nerves. The same thing is equally true of the other paralyzer, electricity. Twitches, tremors, spasms and tetanus are all but varying stages of nerve paralysis and of muscular freedom.

ELECTRICITY A PARALYZING AGENT.

Prof. Tyndall tells us that a mere trace of iron in the coils of a galvanometer, of even such splendid instruments as those used by Prof. Du Bois Reymond in his researches on animal electricity, caused a fallacious deflection of the needle, to the extent of thirty degrees and more. (Heat as a Mode of Motion, p. 34.) It is therefore not to be wondered that erroneous conclusions were sometimes arrived at in experiments so beset with fallacies, even when conducted apparently with the greatest care.

So mysterious a force, which exhibits itself alike in the lightning's flash, in a tiny spark and in the quiver of the eminently sensitive protoplasm of a muscle, might well excite wonder and enthusiasm. As investigation proceeds, however, the exaggerated ideas as to the important part played by electrical currents in the phenomena of nerve and muscle, and even of life itself, which prevailed some years ago have been rapidly on the decline among students of electro-physiology; but will doubtless linger long in the popular and even in the professional mind.

But electricity is not nerve force, nor can it cause the generation of nerve force, which is impossible in a mere nerve trunk separated from its nervous centre. This must be obvious. If it produce effects equivalent to a loss of vital action such as occurs in the death or destruction of portions of the nervous system, it must be classed as a sedative and not as a stimulant. In the experiments about to be mentioned the currents employed are those used for ordinary physiological and therapeutic purposes.

The effect of such a current applied to the inferior laryngeal nerves is to induce spasm of the muscles of the glottis. "The rima is completely closed." (Dr. B. Sanderson, Handbook, p. 308.) That is to say, it does precisely

what we have seen above is done by section and paralysis of these nerves. Applied to the lower ends of the vagi it causes contraction of the œsophagus and stomach and "in most cases vomiting." (Meyer's Prac. Elec. Hammond, p. 87.) Just as we have before seen, results from section of those nerves. We have had proof that section of the spinal cord and of vaso-motor nerve trunks induces contraction of corresponding arterioles. Similar effect is produced by electrization of the same parts, the calibre of the arteries being sometimes reduced to one-sixth of their normal size. (Weber-Meyers, Ib., page 88.)

Dr. M. Foster tells us that section of the spinal cord at the medulla, or in the dorsal region, arrests the secretion of urine; and such a section of the cord is of course a paralyzing act. He also tells us that electrization of the spinal cord below the medulla also arrests the secretions of urine. Then is not this a paralyzing act also? It is unnecessary to multiply examples. Shall we continue to call an agent a stimulant and refer to it as an excitant of nerve activity, the ordinary effects of which on nerves are equivalent to nerve section, nerve paralysis and death!

MILD CURRENTS PARALYZE.

It is sometimes said that powerful currents may paralyze and even kill, but that mild or weak currents merely stimulate or excite. Is there any proof of this? Where in the records of electro-physiology do we find a claim for opposite effects from weak and strong currents? It is true that we are cautioned against the depressing effects of long continued applications of even mild currents. But this is not to the present point. The short *seance*, with its mild currents, may and probably does afford a stimulation of increased vigor, but this is mainly due to the moderate exercise which it gives the muscles and their consequently improved nutrition (Drs. Beard and Rockwell); perhaps also in some degree to the mental impressions of the patient. The longer *seances*, with stronger currents, are fatiguing and exhausting in proportion as they are depressing or paralyzing.

Is it not true that the weakest current which can affect a muscle at all, causes a momentary contraction of the muscle; and that the strongest current that can be borne during life, or that can be brought to play upon a still irritable nerve and muscle after death, simply produces a more vigorous effect of the same kind; the contraction becoming continuous in spasm or tetanus? It is never contraction on one hand and relaxation on the other, unless, indeed, other conditions intervene and muscular contractile energy is at an end. As a matter of fact, weak and strong currents act precisely in the same manner, and differ only in the lesser or greater contraction of the muscle which they produce. The process is a uniform one, as indeed it must be, since a purely physical force cannot change its character, and play fast and loose in the mode of its operation.

The treatises on this subject bear ample evidence of the paralyzing effects of electrization when even weak currents are used, as could only be the case for therapeutic purposes. Althaus found that the electric current produced an anæsthetic and paralyzing effect on the ulnar and sciatic nerves. Drs. Beard and Rockwell tell us that "in rhinitis, pharyngitis and laryngitis,"—where only very mild currents are admissable,—"they have for years been accustomed continually to make use of the benumbing effects of electrization." (Med. and Surg. Elec., p. 123.) Even "weak electrization of the upper part of the neck may arrest respiration," as well as produce spasm of the glottis and of the muscles of inspiration. (Ib., p. 133.) Currents necessarily weak, because applied to the neck of "a sensitive young lady," induced anæmia of the brain, with drowsiness and other effects indicative of arterial contraction. (Ib., p. 134.) Other authors equally allude to the "paralyzing effects of the constant current,"—(Valentine, Matteucci, Eckhard, Meyers.)

From these considerations I hold that there is no evidence whatever that weak and strong currents produce opposite effects, or that one may paralyze and the other stimulate.

DIRECT AND INVERSE CURRENTS.

A great deal has been written about the different effects of direct and inverse currents. Dr. J. Russell Reynolds, in reply to the question, "What current should I use to relieve pain and spasm, the direct or inverse?" answers:—"All I have to say is that so far as I have seen it does not make the smallest difference. Theoretically it makes a very great difference, but practically it makes none." (Clinical Uses, etc., p. 18.) Now, I think that the evidence showing that both these currents are paralyzing is indisputable. Take the direct current first. A nerve-muscle preparation is prepared. To the middle of the nerve trunk a salt solution or the poles of an induction battery are applied, and in either case the effect is so regulated as just to fail to cause a contraction of the muscle. If, now, the poles of a galvanic battery are applied to the distant end of the nerve-trunk, the P. pole furthest from the muscle, so as to produce a direct current, throwing the lower end into catelectrotonus, the muscle will contract at once.

Hence the direct current is said to increase the irritability of the nerve. But electricity is not nerve force, and nerve force cannot be generated in a mere nerve trunk. The true change in the nerve is not one of increased strength or vigor ; it is simply that the feebly paralyzing action of the salt solution or of the induction battery has been supplemented or re-inforced by the additional paralyzing wave of the direct current, and nerve force is for the moment annulled. What is just asserted is nothing new. Thus, "According to Volta, both directions of the current are depressing in their effects." (M. Meyer, p. 57). Prof. Matteucci found that "the direct current" not only "diminished the excitability of nerves," but produced in them "a temporary paralysis." (Braith. Epit., Vol. II, p. 661). Dr. W. B. Carpenter wrote "The direct current weakens and at last destroys the excitability of a nerve." (Hum. Phys., p. 351). So much for the direct current.

The inverse current produces in the nerve trunk, between the electrodes and the muscle, a condition of analectrotonus, which is admittedly one of "diminished irritability," which term is in itself an acknowledgement of lowered vital activity, which can only be accounted for as a degree of paralysis, and is induced by weak as well as relatively strong currents. Dr. C. B. Radcliffe states of M. Eckhard : —"This very able physiologist has ascertained that so long as the inverse galvanic current is closed it is impossible to produce contraction of the muscle by pinching, pricking or otherwise acting on this part of the nerve . . . . which is consequently in a state of suspended irritability. (Epilepsy, etc., p. 75). This is a state of paralysis, because "a nerve that is deprived of its irritability can neither receive impressions nor transmit them." (Ib., p. 78).

Drs. Beard and Rockwell say that "in regard to the differential action of the ascending and descending currents there has been an almost infinite amount of shallow observation and impulsive writing." These writers offer ample evidence that the effects in question are due, *not to current direction*, but to *the physical effects of the poles*, at one of which acids accumulate and alkalies at the other.

TWO EXPERIMENTS.

Here are two experiments which show that the combined effects of strychnia and electrization are equivalent to the destruction of the spinal cord. In a rabbit undergoing the convulsions of strychnia poisoning the spasms will be at once arrested on breaking up the spinal cord by a wire thrust into the spinal canal. If instead of destroying the spinal cord in this manner it be subjected to electrization the spasms will be averted, or arrested if already present The rabbit dies, but without the characteristic spasms. (Matteucci, Periera, Radcliffe.) Is a powerful electric current needed here? Not at all. Quite a moderate current will suffice ; because the strychnia poison is causing general contraction of the arterioles (Fothergill), filling the veins and deoxygenizing the blood. Asphyxia is also setting in from the same cause, joined with fixation of the chest by spasm of its muscles, whose motor nerves are being paralyzed (Ringer.) Electrization produces parallel effects and intensifies the fatal processes already in operation. A weak current suffices to complete the arterial emptiness, the venous engorgement and the non-oxydization of the blood. The spasms cease probably because such blood as is now present is inimical to the life of the muscle, and destroys its contractile energy more rapidly than no blood at all. (Foster, Phys., pp. 126, 833.)

If the theory of the day were true the rabbit ought not to have died ! With the stimulating and vitalizing action of an electric current, added to the previous exhileration of strychnia stimulation the rabbit should have lived and flourished, in the interests of the theory, which alas! as usual, is found to be out of harmony with the facts.

Why does Dr. J Russell Reynolds say that "it would be very unwise to use any form of electricity during the period of shock?" (Lect. on Clin. Uses, p. 84.) Why do eminent authorities discourage its employment in cases of suspended animation, as in apparent death from drowning? (Dr. Ringer, Ther., p. 792.) Why does Dr. B. W. Richardson, F.R S., of London, write: "I feel it too unreasonable to recommend galvanic action as a means of resuscitation in threatened death from chloroform" . . . . fearing least under the semblance of restoring life he should clench death ! (Med. Times and Gazette, 1861; Braithwaite, Jan., 1873, p. 256.) These are precisely the conditions under which a "stimulant, tonic and vitalizer" should be eagerly sought for and diligently employed ! It is evident that electrization is none of these, and therefore it is forbidden "in any form."

I think I am justified in claiming for the foregoing facts that they prove, as fully as any doctrine in physiology can be proved, that electrization as ordinarily employed is a paralyzing process.

BENEFICIAL EFFECTS OF ELECTRICITY.

Electricity is no doubt a valuable therapeutic agent, and, like other paralyzing agents, does good in appropriate cases. But its beneficial effects may all be accounted for in strict accordance with its *role* as a paralyzer of nerve activity. Thus, it eases pain in a perturbed nerve by temporarily paralyzing it. It lowers the activity of the

vaso-motor nerves, and by thus setting free the contractile energy of the muscle it reduces the calibre of the arterioles, lessening or curing congestion, and consequently starving the hypertrophic growths. In other cases, by a momentary arrest of nerve action in the motor trunks, it induces prompt spasmodic contractions in the muscles, thus exercising them, and by attracting blood and pabulum to wasted muscles or tissues in the same way, it improves their nutrition. In chronic indurations and hyperplastic growths the purely chemical effects of the opposite poles, or electrodes, so modifies the nutritive activities of the tissues as to prove beneficial in restoring a more normal condition. Thus the curative effects of electrical treatment are all accounted for in strict accordance with its *role* as a paralyzing agent. To proclaim it, therefore, as "nature's own tonic," or to laud it as a "vitalizer" or extol it as the ally of nerve force, may be pardonable in the instrument makers, but is to be condemned on the part of scientific medicine.

HOW THERAPEUTICS HAS SUFFERED.

It has sometimes been remarked that the department of therapeutics lags behind other branches of the medical art. Perhaps it will be pardoned if I venture to suggest that therapeutics has suffered greatly from the adoption of the *dictum* that electricity is a *stimulus* to nerve function How much of a huge and hypothetical inhibitory system has found, perhaps, its chief support in this very error. When electricity stopped the heart, some mechanism had to be found for the arrest of its action by a stimulus. On what must the excitation expend itself? Not on the proper motor ganglia of the heart, which a stimulus would drive faster. To 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 stand-still. 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! (Dr. M. Foster, Phys., p. 232). 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. (Foster, Phys., p. 121). 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." (Phys., p. 94). Is this con-

traction 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 is the product of the death of albuminoid bioplasm." (Disease Germs, pp. 136, 137). If this be true of fibrin, it may fairly be assumed to be true also of myosin, which closely resembles the former. Coagulated plasma, or myosin, is dead, and if the muscle also be dead, and its inherent contractile power at an end, in what manner does dead myosin acting on a dead muscle produce so perfect a counterfeit of muscular contraction, that one of the keenest observers of the day pronounced it, "The most steady and persistent contraction which muscle can possibly exhibit," (Anstie, Stim. and Narc., p. 70); so perfect a counterfeit, indeed, that our eminent English physiologist, the late Dr. Carpenter, employed the microscopical appearances of muscle during rigor mortis as the chief basis for his description of the changes taking place in ordinary muscular contraction, as he himself has told us. (Hum. Phys., 5th Amer. Ed., pp. 307, 308).

Again, the reaction of a living muscle in repose is neutral, or alkaline, but after exercise, or tetanus, the reaction becomes acid, an effect in some way depending upon the chemical processes in the muscle associated with its contraction. In rigor mortis the reaction becomes "most distinctly acid" also. But if the muscle be already dead and these chemical changes at an end, what is the source of the acidity? To the presence of this acid, the coagulation of the myosin and the rigidity of the muscle are of late attributed. But since the acidity is the *result*, or *effect*, of muscular contraction in the living muscle, how can it be the *cause* or starting point of the contraction and stiffening in the dead muscle?

Dr. Lauder Brunton finds that muscle plasma "coagulates too quickly in the muscles of warm-blooded animals to allow of its preparation from them." Now, rigor mortis does not usually set in for several hours after death,—Dr. Brown-Sequard found it to be ten hours in four rabbits,—and its onset may even be artificially delayed. The statement, therefore, is only explicable on the supposition that coagulation of the muscle plasma and rigor mortis do not occur together—that is, as cause and effect. It would seem to be implied that the muscle plasma coagulates too early to be the cause of rigor mortis. Dr. Brunton further shews that the muscle plasma may coagulate without producing rigor mortis. In an experiment, detailed on page 363 of the Hand-book, it is shewn that, if half a fresh muscle be immersed for a few minutes in water at a temperature of 104° Fah., the reaction will be acid, as Dr. Brunton says,—"from development of rigor mortis." The other half of the muscle is to be placed for a similar time in boiling water; and here the reaction "will be alkaline." Dr. B. adds,—"Before rigor mortis had time to set in the albumen of the muscle was coagulated. This coagulation set free a quantity of alkali, hence its reaction." Dr. Brunton's exposition of this experiment, if correct, would be fatal to the myosin hypothesis, since if the coagulation of the muscle plasma be attended by an alkaline reaction while in rigor mortis, the reaction is strongly acid, the former could not be the cause of the latter, and they must be regarded as separate and distinct processes.

The foregoing difficulties certainly seem to create distrust in the myosin hypothesis; and we now turn from it, with its dead muscle and inert myosin, to the other aspect of the case, under which the complete cessation of nerve activity and the final contraction of the muscle marks the onset of rigidity. "The rigidity, the loss of suppleness and the diminished translucency," observable in the muscle in this state, are reasonably accounted for by the condensation of tissue which is here permanent, as the contraction is continuous. That a certain relaxation subsequently occurs, during which meat or game, which is at first tough, becomes more tender and toothy, is attributed by M. Rosenthal to the action of the acid referred to, which relaxes the connective tissue which holds the fibres together, so that the latter separate more readily. (Muscles, etc., p. 87-8). This is but the beginning of the chemical change which ends muscular contractility in the ruin of putrefaction. The following remarkable series of conditions are common both to muscular contraction and to rigor mortis. In both the reaction becomes acid. In both carbonic acid is set free in the muscle. In both the temperature rises,—often markedly so in rigor mortis). In both the muscle is contracted and shortened; in some cases, as in death from cholera, "rigor mortis may be said to be simply

a continuation of the cramps and contractions occurring during life." (Wood's Prac., Vol. I, p. 717). In both, glycogen is converted into sugar. Do not all these coincidences in appearances and effects point strongly to a similarity of processes in muscular contraction and cadaveric rigidity? Of course the parallel is not complete in every particular. It is said that the muscular sound emitted during ordinary muscular contraction is absent. This sound is attributed to vibration of the muscle substance. Might it not be due in part to the altered circulation in the ordinary muscle during contraction, for it is well known that the blood channels, under certain circumstances, give out a musical note? In rigor mortis, of course, the circulation of the blood ceases, as does also the removal of waste products. That the muscle substance continues to vibrate in rigor mortis is evident, because chemical changes are still taking place there, as is shewn by what is said above, and especially by "a marked accession of heat"; (Foster, p. 542); and "heat is only another form of motion," (Rosenthal, p. 42). So that, after all, it would seem as if the atoms of the muscle continue to vibrate, even though no sound is audible.

That indefatigable observer, Dr. Brown-Sequard, some time ago, related to the Biological Society of Paris, "some experiments he had made, by a special instrument, to determine the movements of single muscles in the body after death. He found that there was a very considerable degree of contraction and relaxation, as much, for example, as two and a half millimetres in a muscle measuring only six millimetres in length. He thought that the results of his experiments disproved the theory of coagulation in the muscular tissue as the cause of cadaveric rigidity. (N Y. Med. Rec., Jan. 9, 1886).

I am not necessitated to prove that rigor mortis is due to post-mortem contraction of the muscles; but in the absence of any other satisfactory explanation of this state, I am entitled to refer to it in support of my thesis; and I would ask those who dissent from this view, and who, in consistence with their theory, must hold that nerve stimulus is necessary to muscular contraction, to account for the presence of nerve force under the conditions referred to.

SPASMS IN VOLUNTARY MUSCLES.

It would, perhaps, be no difficult task to shew that even voluntary or striated muscles pass into a state of partial spasm or contraction during life, much oftener than might at first sight appear, under a form of "irritation," which may very properly be regarded as consisting in a lowering of nerve activity.

"Irritation" is not increased nerve action. A splinter under the nail is attended by a loss of tactile sensibility. A mote in the eye irritates, but it obscures vision. Why should indigestible food oppressing the digestive functions of a child be regarded as a source of increased nervous "discharges"? Such sources of irritation ought to be considered as depressing rather than exciting nerve action; a view of the case for which authorities have been already quoted, and others are to follow.

Dr. Anstie wrote, "convulsive action of the muscles, as everyone knows, are very common complications of neuralgia," and the same acute observer held that "pain is not a true hyperæsthesia; on the contrary, pain involves a lowering of nerve function." (Neural., p. 12).

Dr. Hilton, in his work on "Rest and Pain," points out that the irritation of peritonitis induces contraction of the abdominal muscles. In the same way, pleuritis renders the chest walls fixed by spasmodic contraction of its muscles; while the muscles of an inflamed joint, he says, "are invariably contracted, and continually tend to increased flexion of the limb, not because such a position is easiest for the patient, which is not always the case, but owing to a reflex perturbation transferred to the muscles of the adjoining surface." (P. 96). That peripheral irritations *do* produce nerve paralysis must be admitted on the authority of Dr. Brown-Sequard. (Lect. Cent. Nerv. Syst., pp 160, 170), and others.

What is the "irritation" in these cases but a mild form of nerve paresis, just as "the irregular muscular action" which shows itself in tremor, fibrillary contractions, or in spasm, denotes the failure of the ordinary nervous restraint over the corresponding muscles.

Why should "morbid conditions of the medulla oblongata" avowedly depending on "defective nutrition" be supposed to give rise to "explosive and atactic manifestations of nerve force," (Anstie, Neural., p. 156), when they are much more naturally explained as depending upon nerve failure? The weak point in the theory of the text-books, is that nerve force is required to be displaying the full activity of robust health, and even more, in exaggerated "discharges" and "explosions" at the very time there is the most undoubted evidence of nerve failure and exhaustion. Why, in cases of "early and late rigidity" of muscles, should a clot in the brain be held to be an exciting irritant, seeing that the brain tissue is wholly insensitive, and may be cut, pricked or seared with a red-hot iron with-

out eliciting any signs of pain? It is difficult to express here the multitude of facts which show the very frequent association of paralysis and spasm in disease of the brain and spinal cord. The paralysis is of the nerve and the spasm of the muscle—conditions very embarrassing to the theory of the day, but consistent and harmonious states in the theory of these pages. Is there not much significance in the statement of Seguin, that "a lesion of the lateral columns of the spinal cord produces paralysis with contracture" of muscles. Why? Because, as Dr. Brown-Sequard has shewn, "the motor fibres run on the exterior of the cord in its antero-lateral columns." (Erichsen, Concus. Spine, pp. 29, 30). Motor nerve disease and destruction induces contraction of the muscle, which later on becomes atrophied, partly, no doubt, from inaction.

It is on record, too, that while injury of the vagus nerve induces contractions of the gastric muscle, injuries of the spinal accessary nerve are attended by spasms of the trapezius or sterno-mastoid muscles. (Bryant's Surgery, p. 208). Other examples of a similar kind are not lacking.

One might imagine that Dr. B. W. Richardson, F.R.S., intended to endorse the theory of these pages when he wrote as follows regarding the convulsions of the drowning. He says:—"The convulsive movements that are seen are unconscious movements; they are the same as those which mark the period of stupor, in death by hanging, by noxious vapors, by concussion; and they are simply the results of action of muscles from which *the controlling power of the nervous centres has been removed.*" (Braithwaite, July, 1871, p. 255). [Italics mine]. Dr. Henry M. Lyman, A.M., M.D., would appear also to have had a commendable distrust, if not an entire disbelief, in the theory of the text-books, when, in referring to "a temporary increase of muscular movement directly caused by the abolition of some special source of nervous impulse," he says:—"Witness the tremendous *liberation of muscular movement* which follows a *paralysis* of the influence of the brain, by the sudden decapitation of a fowl, for example." (Anæsthetics, Wood's Lib., p. 26). [Italics mine].

One of Dr. Ferrier's experiments is so much in point here, that, at the risk of being tedious, I cannot forbear a brief reference to it. The right brain of a monkey had been exposed and subjected to faradization. Next day the animal "was found perfectly well." "Towards the close of the day following, on which there were signs of inflammatory irritation and suppuration, it began to suffer from choreic spasms," which rapidly assumed an epileptiform character. Next day hemiplegia became established with the usual symptoms of "paralysis of the left arm and partial paralysis of the left leg." "On the day following paralysis of motion was complete over the whole of the left side, and continued so till death, nine days after." Dr. Ferrier says, "In this we have a clear case of vital irritation producing precisely the same effects as the electric current, and then destruction by inflammatory softening resulting in complete paralysis, etc." (Functions of Brain, pp. 200, 202).

On Dr. Ferrier's view, the stage of apparent inflammatory action was accompanied by increased production and discharge of nerve energy, as seen in the choreic and epileptiform spasms. But "Recent studies show that the inflammatory process is a destructive and depressive one, so far as the tissues are concerned; that it does not irritate and kindle into increased activity the protoplasm of the cells, but rather the reverse." (Editorial, N. Y. Medical Record, Jany. 30, 1886, p. 128). So that it is now definitely understood that the inflammatory process in brain tissue does the reverse of Dr. Ferrier's view, and paralyzes rather than excites nerve energy.

Observe here, that the spasms of the muscles, on Dr. F.'s own shewing, began to occur contemporaneously with the "signs of inflammatory irritation and suppuration," and as this term "irritation," (on so good an authority as the able editor of the N. Y. Medical Record), must now be interpreted to mean depression and lowering of cell activity, it follows that the spasms referred to occurred from the absence or failure of nerve energy, and not from its undue excitation. Observe, too, that Dr. Ferrier held that this "vital irritation," as he saw it, but which we now know is depression or paralysis, produced "precisely the same effects as the electric current." Another evidence of the paralyzing character of electricity!

THE EPILEPTIC PAROXYSM.

With the experiments on the cervical sympathetic and splanchnic nerves before us, how can we say that the anæmia, or rather ischæmia, of the brain, which ushers in the epileptic seizure, is due to "excessive action of the spinal centres," compelling the spasm or contraction of the arterial muscles on which this ischæmia depends? Have we not had proof that the arterioles contract best when their vaso-motor nerves are cut, or are paralyzed, or dead; and if so, are we not bound to hold that not excess but failure of nerve power is the proximate cause of the epileptic paroxysm? And is not the question of such excess or failure of nerve force a most practical one in determining the treatment?

How far is our comparative failure to cure this terrible disease due to our approaching it under the ægis of an erroneous theory—that nerve force here needed to be depressed rather than exalted? It is well for mankind that in this, as in some other instances, our practice has sometimes been directly at variance with the theory of the day. Thus we find Dr. Anstie assuring us that "our best anti-spasmodics are stimulants"; and that "alcohol is one of the best remedies possible in the convulsions of teething in children." (Stim. and Narcot., pp. 123, 129).

NO "MORBID" NERVE FORCE.

Spasms and convulsions frequently take place in the very act of dying, and under circumstances in which nerve force ought to be regarded as at a low ebb; as, for example, in uræmic blood poisoning. It is customary in some quarters to attribute these or other spasms to "a morbid irritability" or "a morbid nerve force"; as if the central nervous ganglia were capable of producing two kinds of nerve force, one normal and the other "morbid," and the spurious variety of attaining extraordinary power just in proportion to the complete failure of nerve force proper. A little reflection, I think, will show that this is untenable. Nerve force may be increased or diminished: its condition may be one of excess or of failure, but that it may present a duplicate of itself, and its *alter ego* produce effects, for which nerve force proper is inadequate, and yet is responsible, is surely yielding too much to the exigency of an erroneous theory.

Medical literature presents numerous examples of this appeal to a "morbid nerve action," and it is rather surprising to find such a writer as the late Dr. Anstie referring to "the explosive disturbances of nerve force which give rise to the convulsions of tetanus" as "something quite different in kind" from healthy nerve action. (Neural., p. 8). Now, if a nerve centre be thrown into action otherwise than by the exercise of its normal activity, then it is no longer the nerve centre which is acting, but a power extraneous to itself; a modern Archæus for which scientific medicine ought to have no place. And if tetanus be really due to an explosive activity of the nervous centres which are discharging nerve force with unwonted activity, surely to administer stimulants in such a case ought to be injurious, if not fatal! And yet we find that Dr. W. A. Hammond of New York has produced statistics in which "stimulants" stand at the very head of the list of curative agents in tetanus. (Dis. Nerv. Syst., 4th Ed., p. 541). Here again the theory of the day is surely out of joint with the clinical facts.

CHLOROFORM AND RELAXATION OF ANÆSTHESIA.

I have been asked how the rigidity, at first, and subsequently the relaxation, of the muscles during anæsthesia are to be accounted for on this theory. The answer is easy. The rigidity is due to the partial paralysis of motor nerve influence, setting the contractile power of the muscle free to act This occurs at a comparatively early stage of the process. The relaxation which attends complete anæsthesia is due to the loss of contractile power on the part of the muscle, owing to the absence of oxygen in sufficient quantity in the blood; for chloroform tends to prevent the oxygenation of the blood, (Ringer's Ther., p. 286), and renders it venous in character. In this way the chemical processes on which the generation of contractile force in the muscle depends are retarded. (Lyman's Anæsthetics, p. 28; Bryant's Surgery, Amer. Ed., p. 318). Dr. M. Foster states that "blood is not only useless, but injurious, unless it be duly oxygenated." And again, "if venous blood be driven through a muscle the irritability of the muscle is lost even more rapidly than in the entire absence of blood." (Phys., pp. 883, 126). This, I think, will be accepted as a satisfactory explanation. in strict accord with physiological facts. The relaxation, however, is not so great but that faradization of the muscle will induce a further degree of contraction; showing that the contractile energy of the muscle, though weakened, is not lost. That the contractile power of the muscle is thus lowered offers a bar to the prolonged or complete administration of chloroform during parturition, for obvious reasons.

The mode in which anæsthetics induce arterial contraction, as explained by Dr. Henry M. Lyman, may be quoted as follows:—"Chloroform acting through the blood upon the nervous apparatus in the walls of the vessels, tends to paralyze the sensory endings of the nervous fibrils. This means a diminution of the normal impulses, which should continually reach the central intraparietal ganglia," in consequence of which "the motor cells no longer experience the inhibitory influence which they should receive from the periphery of their territory, and a liberation of a motor impulse excites muscular contraction, and we have vascular spasm," etc., as the result, (Anæsthesia, etc., page 27). This, of course, is purely hypothetical. The motor nerve fibrils in the muscular bands are ignored altogether, while a purely imaginary "inhibitory" system is invoked to meet the exigency of the occasion. How much better to hold that the motor nerve fibrils also are more or less paralyzed, and the arterial muscle directly set free to contract; thus dispensing with the inhibitory apparatus altogether

THE NERVE-MUSCLE PREPARATION.

It is impossible here to enter on a critical analysis of the experiments on nerve and muscle, which a careful examination will show to be wholly consistent with the views here advocated. When in a nerve-muscle preparation, the muscle is made to contract by applying to the nerve trunk the shock of electricity, the corrosion of a chemical agent as a quick stroke, what is there to show that the effect on the nerve is not to cause a temporary cessation of nerve influence, rather than the production of a stimulus? There is really nothing, and the character of the impulse is merely a matter of inference. Even in what is called the rheoscopic frog, where contraction in one muscle imparts an influence whereby another muscle is made to contract, the molecular or electrical wave may as well be paralyzing as stimulating.

THIS THEORY NOT NEW.

In hastening to conclude, let me state that, whether this theory of the antagonism of nerve and muscle be true or false, I am not entitled to the praise—or blame—of originating it It was broached so long ago as 1832 by Dr. West, an English physician, and is said to have met with some countenance from Sir Charles Bell. Dr. C. B. Radcliffe, F.R.S., in his work on "Epilepsy, Paralysis and Pain," has warmly adopted the views of Dr. West, and offers some strong evidence in support of the proposition, that "there is reason to believe that ordinary muscular contraction is associated with a deprivation of nervous influence, and not with a contrary state of things." (P. 95). I have here endeavored to support the same thesis, but with evidence drawn from other sources.

OBJECTIONS TO THIS THEORY.

1. It has been objected to this theory that "a muscle can contract when irritation is directly applied without the intervention of nerves." Now, I am not in the least disposed, or obliged, to dispute this assertion, for reasons which will appear later on. My thesis has much to gain, and nothing to lose, by the fullest admission of the independent irritability of muscular tissue. But it is exceedingly difficult, if not at present impossible, to say when a still irritable muscle has been deprived of "the intervention of its nerves." Certainly such is not the case in the experiments edited by Dr. M. Foster, in the Hand-book heretofore referred to, where the experimenter, in order to produce the ideo-muscular contraction, is to choose "a muscle which has been much exhausted by treatment or by long removal from the body," and to "wait till neither muscle nor nerve give any ordinary contraction with an electric stimulus." It cannot be held to be proven that in such a nerve-muscle there is not still remaining a force in the weakened nerve sufficient to control the equally weakened muscle.

CURARE AND THE MOTOR NERVE ENDINGS.

2. It has also been objected that, while the motor nerve endings are paralyzed by curare, the muscle does not contract, as it ought to do if this theory were correct. To this I have to reply, that if the muscles are not found contracted it is partly due to the insufficiency of the poisoning of the motor nerves, and partly to the fact that curare diminishes the contractile energy of the muscle. (Rosenthal, Muscles, etc., p. 254). Nicotine and conine act precisely like curare, (Ib., p. 253), and in the final action of these three poisons, motor nerve paralysis and spasm, or convulsion of the muscles, occupy a prominent place. (Ringer). The special results vary, of course, in different animals. Nicotia sometimes acts like an anæsthetic; (Stille and Maisch, p. 372); and the same is doubtless true of the others. Now, anæsthetics induce muscular relaxation by deoxydizing the blood; and nicotine is known to disorganize the red corpuscles which are the oxygen carriers It is doubtless in this way that, under the slow action of these poisons, muscular relaxation is brought about. If death be rapidly produced by curare, convulsions occur. (Stille and Maisch). Here the motor nerves are paralyzed before time has been afforded for the poison to lower the irritability of the muscle, which passes into tonic or clonic spasms according to its freedom, thus behaving as it "ought" to do. Is not this a sufficient answer to the objection?

But more remains to be said. The experiments with curare are not so conclusive as to be beyond the reach of criticism. They were intended to prove the independent irritability of muscle, which is now generally an accepted fact among physiologists. M. Rosenthal asserts that these experiments (and those of Kuhne upon the sartorious muscle), do not prove this; which is equivalent to stating that it is not proved that curare paralyzes the motor nerve endings.

More direct evidence upon this point is that of Dr. Onimus, who, not long ago, "read a paper before the Academy of Medicine, Paris, upon electro-muscular contractility and the action of curare. Contrary to the opinion of M. Claude Bernard, Dr. Onimus believed that curare does not act on all parts of the motor nerves, but only on their trunks;—the nerve centres and terminal filaments being unaffected." (N.Y. Med. Record, 1880, p. 73).

In view of these authoritative opinions, (and doubtless of others to which I have not access), it is evident that this objection falls to the ground and loses the weight which otherwise might attach to it.

But suppose it were established beyond doubt that the influence of the nerve were completely eliminated from the muscle in any case, and that the contractile protoplasmic masses of the muscle were left wholly to themselves, and their life being not yet extinct, that they gave token of that still flickering life when comparatively rudely assailed by a shock of electricity or a corrosive or injurious agent,—what then? Such signs of irritability, elicited under such circumstances, would not militate against my thesis; for such would be the behaviour to be expected from still living protoplasm, wherever found, and would in no way disprove the contention that in the association of nerve and muscle in the organism the *role* of the nerve is to restrain or control the protoplasmic energy of the muscle so long as their mutual relations continue. For, after all, "the contraction of muscular tissue is, in fact, a limited and definite amœboid movement, in which intensity and rapidity are gained at the expense of variety." (Dr. M. Foster, Phys., p. 63).

Indeed, I think the rational view of the situation just depicted, turns the argument the other way; and tends to show that in the joint *role* of nerve and muscle the function of the nerve is *not* to goad or stimulate the muscle to contract. To suppose this is to assign to nerve energy the relative value of the fifth wheel in the coach. Such enduring power of contractility as the muscle here exhibits evidently needs no supplementary aid from the nerve. What it really *does* need, however, is restraint, control and co-ordination for the purposes of the organization of which it is a part.

OTHER OBJECTIONS.

A further objection has been suggested, on the ground that on a nervous impulse reaching a muscle, an electric current is generated during the period immediately preceding the contraction of the muscle; but this is an objection which is only of any force on the assumption that electricity is a stimulant. There is nothing in the action taking place here to show that the electric current is a stimulant rather than a paralyzer. There is simply a "freeing of the forces in the muscle," just as the spark of electricity frees the forces bound up in gunpowder, and so fires the train. (Rosenthal, p. 250).

As for the additional plea that nerve force and muscle force are too much alike for us to consider one a paralyzing and the other a contracting agent: that is merely begging the question. Nothing whatever is known regarding the nature of these forces; and the intimate structures of nerve and muscle are so widely different as to justify the idea that the product, so to speak, of each, is equally diverse.

This theory has been objected to as a proposed addition to the inhibitory system of the text-books. This is a mistake. If the views here enunciated were adopted, the huge incubus of the present inhibitory hypothesis could be in great part swept away, to the great advantage both of physiology and therapeutics.

If it be claimed that on the cutting of the spinal cord or of a nerve trunk, the "irritation" set up at the point of cutting, or the generation of electrical currents as the result of chemical change in the transverse section, act as a stimulus, and that contraction of the corresponding muscle is thus produced, such a claim must be regarded as untenable for the following reason:—The acts just referred to cannot be stimulating acts, because they are attended by precisely similar effects as are produced in the muscle by death from any cause, in which condition, it is needless to say, nervous activity is not increased. The proof of this has already been sufficiently vouched for, and need not be repeated here.

Of course, I do not pretend that all difficulties vanish in the light of the theory here advocated. There are very serious, if not insurmountable, difficulties in the theory of the text-books; as the facts of the foregoing pages fully shew. What I claim is, that the view here presented rests on a rational basis, and, though presented very inadequately, and under many disadvantages, has the merit of furnishing a key to many obscure phenomena in the organism, and is entitled to the fair and candid consideration of the members of our profession.

LINDSAY, ONT., CANADA,
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