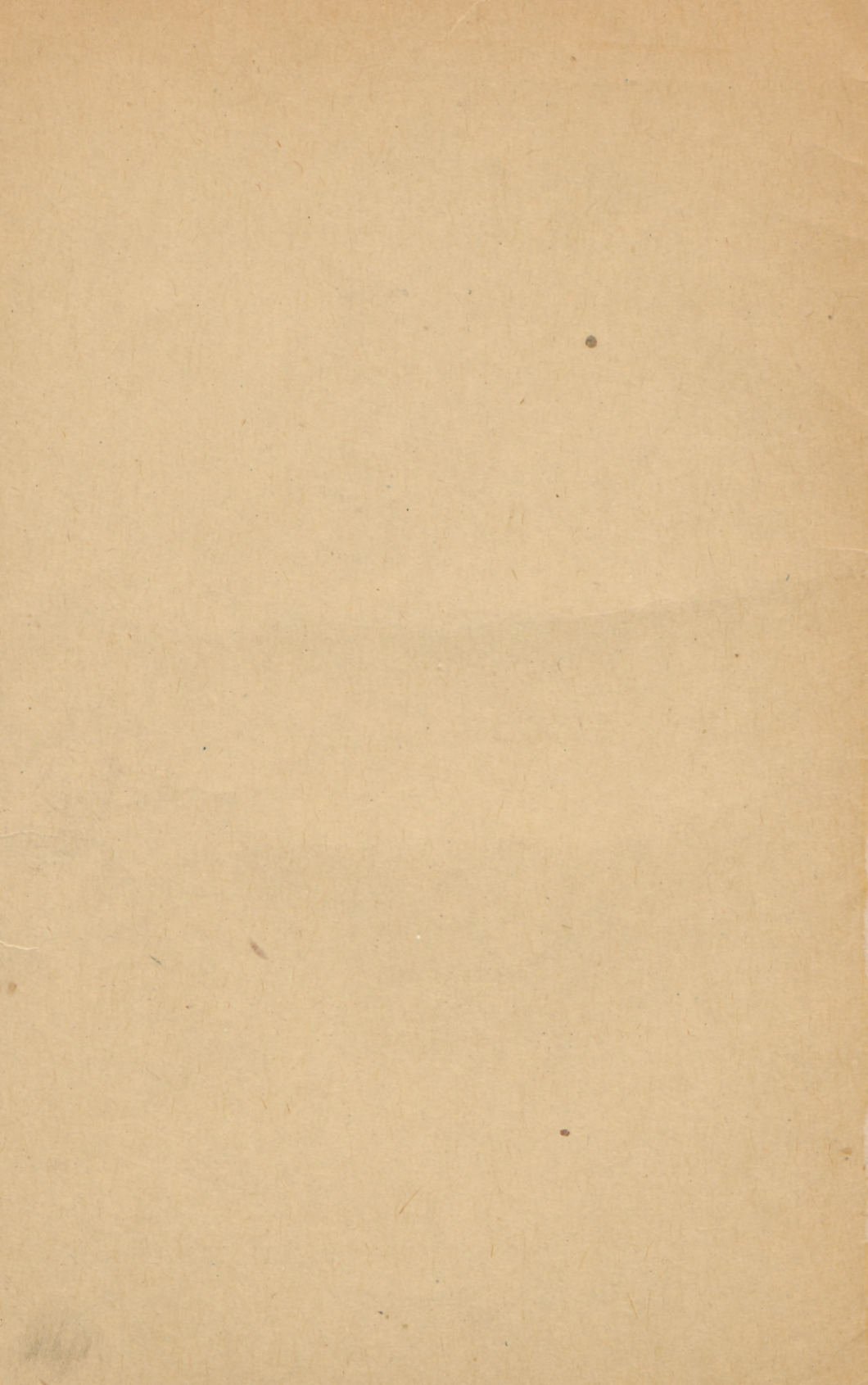


BRISSAUD (ED.)

Spasmodic laughter  
&  
weeping.  
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## Spasmodic Laughter and Weeping.

A LECTURE DELIVERED AT THE ~~SALTPETRIERE~~ BY PROFESSOR  
EDOUARD BRISSAUD.

TRANSLATED FROM THE REVUE SCIENTIFIQUE FOR THE CHICAGO MEDICAL RECORDER,  
WITH THE PERMISSION OF THE AUTHOR,

By HUGH T. PATRICK, M. D., CHICAGO.

GENTLEMEN :—I am going to study with you to-day spasmodic laughter and weeping, particularly the varieties which are observed in hemiplegia of cerebral origin. Those of you who follow this service had the opportunity of seeing a few days ago an old lady, a hemiplegic, transferred to the hospital wards for some slight indisposition, gastric trouble, I believe. This woman had been admitted to the infirmary for a paralysis of the right side, preventing her from earning her living. Here she had been very well until this last attack, moving about with tolerable facility despite her infirmity. When she entered the hospital ward we noticed an exaggeration of her contractures, which ordinarily were not very pronounced, and as soon as we began to question her she began to weep most copiously and was shaken with sobs. She was seemingly plunged in the deepest despair and this without apparent provocation. The moment she was addressed with tenderness, or was even looked at she burst into tears. At present, she seems consoled ; at least she does not weep any more, and has returned to the infirmary in her former condition, except that she retains from this transitory ailment a propensity to laugh without adequate cause. Her hemiplegia persists but is not more pronounced than before. Episodes of this nature are of common

occurrence in the history of hemiplegics and they are observed at the Saltpêtrière especially in winter, in the course of any indifferent illness or indisposition. We see all at once an exaggeration of the reflexes, the paralysis increases with a tendency to contracture and sometimes a singular laughing and crying is added which is nothing but an exaggeration of the spasmodic condition.

This laughing and crying although not constant in hemiplegia is very frequent and this spasmodic condition, which is evidenced by an increase of sensitiveness and of reflex phenomena nearly related to it, is encountered also in other diseases. We have here the necessary and sufficient elements for an attempt at central localization whatever may be the cerebro-spinal affection with which we have to deal.

It is of this question that I have decided to talk to you and my wish to do so is increased by the fact that I have recently seen the report by Bechterew of a case of uncontrollable laughter.<sup>1</sup> I had already studied this subject with some care and had presented my results to the Congress of Limoges. I do not enter into the question of priority and would not have mentioned these details had I not seen that work earlier than mine had been published by Bechterew in Virchow's Archives.<sup>2</sup> Far from claiming priority for my communication I am happy to concede it to Prof. Bechterew and regret that I did not know of his interesting paper at the time of the Congress of Limoges. I would add that my conclusions while differing slightly from those of the professor of Kasan, agree with them in the main and I am glad to be in accord with him on this point. The case of Bechterew is as follows: A young man affected with cerebral hemiplegia of the left side, of syphilitic origin, was heavy and somnolent. He had a marked tendency to weep and looking at him sufficed to cause a burst of tears. Besides he was taken with spells of unconquerable laughter, veritable spasms occurring several times during the day. They did not seem to be an ordinary reflex action; tickling, for instance, did not provoke them, and they always occurred from psychic influence; a spontaneous influence, autochthonous as Bechterew calls it, often so fugitive that the patient could not remember the cause of his laughter and found it out of place. The attacks lasted half an hour, an hour, two hours, with but short intermissions. Specific treatment was employed, the hemiplegia improved and at the same time the attacks of spasmodic laughter disappeared. The conclusions drawn from this observation, the autopsy is wanting, are that the location of the lesion corresponded to the centers for expres-

sion in the anterior part of the thalamus. Here in fact we find the system of nuclei where converge on the one hand the fibers which transmit cortical stimulation and on the other those which conduct reflex excitation. Is the cerebral lesion the direct cause of the tendency to irresistible laughter or is it the indirect cause by preventing the action of the inhibitory centers? Prof. Bechterew adopts this latter hypothesis because to provoke the laughter a psychic influence was necessary, however trivial.

But whatever our conclusions may be, for the moment we will simply bear in mind the fact of this uncontrollable laughter, and the fact is not rare. We see in hemiplegics sometimes spasmodic laughter, sometimes weeping, occurring in spells, occasioned by the awakening of an image stored away probably in the frontal lobes. The phenomenon is familiar; it is known that these patients are the subjects of a peculiar sensibility, but it has not been specially studied as an entity and with regard to the interesting question of localization.

We know nothing of the mental state of our fellow creatures except by means of the expressions which they furnish us. Left by ourselves would we even have emotions? The question is still sub judice and the judges will ever be divided. But it seems that these manifestations of the two extremes, sadness and joy, are to be the point of departure for this physiological research. If from the analysis of certain simple facts we can draw some conclusions as to the variations of the emotional function, perhaps we shall succeed in finding one of the unknown quantities in the great problem of the psychic life.

To return to our patients; I must at once guard you against one source of error. The hemiplegics in question do not belong to those cases of senile dementia in whom small foci of softening occur day by day, and in whom the intelligence decreases *pari passu*. They do not represent that type of "whimpering senility" of which Diderot stood in such horror. Our patients possess all their faculties although at first view deprived of them. If we question them closely we see that the memory, the association of ideas, in a word the understanding is intact or very nearly so. Senile dementia, a misnomer since it may occur at all ages from organic cerebral disease, has also its silly laugh and inexhaustible tears, so well described by M. Magnan, but that is not the object of our consideration at present.

We shall see that many hemiplegics suffer cruelly from this disposition to excessive laughing and weeping and that they recog-

nize how ridiculous are these tempestuous manifestations. A mild pleasantry quite incongruous with such bursts of gaiety, induces an explosion of mirth which it seems will never end. Some of these subjects in spite of their interest in intellectual pursuits cannot, for example, go to the theater for fear of rendering their vicinity insupportable by their boisterous laughter and tears.

A hemiplegic in the full possession of his faculties recently confided to me how painful for him was this hyper-sensitiveness, and related the following: A lady mentioned to him that her little dog had just died. "Indeed," said he, "Oh, that is too bad." Was it this expression of condolence which by a sort of suggestion awakened a train of sad ideas? At any rate all the machinery of expression was set in motion; the simple sadness of the face induced tears, the tears, sobs, and worse still, this unfortunate man was no longer master of his sphincters. I could cite many analogous cases in which laughter took the place of tears.

Thus in a general way the laughter or the weeping in these cases is indeed the manifestation of an emotion, sad or gay, but more demonstrative than it should be. It is a voluntary expression but excessive, as manifested by the spasmodic phenomena.

Before studying these morbid manifestations I wish to say a word about the corresponding physiological processes: But we must acknowledge that the ultimate mechanism of laughing and crying is but poorly understood. Laughter, especially, cannot be experimentally studied "for laughter is peculiar to man." Weeping is also said to be one of our exclusive peculiarities. The assertion may be contested for if there are no animals that laugh, there are some that weep, and under such conditions that they would seem to thus express an emotion. A whole class of mammals, particularly ruminants secrete tears in abundance.

In man laughter and weeping are accompanied respectively by a special facial expression. But have not animals an analogous faculty? Some anthropoids have most significant facial expressions, not only the grimace which amuses us, but true expressive movements which merit closer study, much more so indeed than the problematical language of these animals.

But to return to the laughter and weeping of man. They enter into the category of muscular, that is motor, manifestations which are the necessary consequence of every emotion. This does not pass beyond the limits of simple physiology and pathology. There is nothing rash in saying, as I have just done, that a "movement of the soul" cannot be represented except by a muscular

action or its equivalent, secretion for instance, and is necessarily thus represented. Gratiolet<sup>3</sup> in a work, already old, on physiology and the movements of expression affirmed that every emotion manifested itself by an act.

I will give you a fresh example of this truth. A medical student whom I know intimately and who for the past six years has been affected with a syphilitic hemiplegia tells me that he has given up reading novels; the misfortunes of the heroine produce a torrent of tears and he is in a transport of joy over the happy passages. However, he does not read aloud but only with the eyes and it is simply the emotion which he feels that brings into play his muscular apparatus. In his case it is a spasm which occurs instead of the imperceptible vibration which we all feel. Thus, as Féré has demonstrated, in pleasure and pain, joy and sadness, there is a "necessity of reaction."<sup>4</sup> In subjects in a condition of spasm the least contraction tends to become general. You know besides, as the experiments of Charcot and P. Richer have shown, that a contraction provoked in certain muscles of the face may induce a whole series of appropriate attitudes; and this brings us to our subject, for the acts of laughing and weeping have a primary facial localization and then tend to become general. It would seem that in describing laughter one might repeat the lecture of that master of philosophy, M. Jourdain; you will see that in constructing these vulgar facts we may find information of the greatest interest.

The first facial manifestation, at the labial commissure, consists in a slight contraction of a tegumentary muscle, the zygomaticus. The debut is always here, that is, in the region supplied by the filaments of the inferior facial; at first only a smile confined to the "corners of the mouth," then spreading, gaining the throat region and the phenomenon, at first limited to the face, attains the upper glottis and then the lower which dilates for the passage of the air expelled by the diaphragm. When this latter muscle comes into play it is evident that the excitation, starting from the nucleus of the facial has reached that of the phrenic nerve passing by way of the nuclei of the pneumogastric and spinal accessory. This is the moment of the full hearty laugh. Finally are produced manifestations still more boisterous and generalized. The motor column of the cord itself is attacked and the entire body participates in the spasm. We "hold the sides" and are "doubled up." This is the Homeric laugh of the Olympian gods at the sight of halting Vulcan trying to fill the place of Ganymede.

If now we pass in review the phenomena characteristic of weeping we shall see them succeed each other in an order quite analogous. This time the debut is a depression of the mouth by the chin muscles, the depressor labii inferioris and triangularis menti which by their contraction produce in the sub-buccal region the significant undulations which, especially in children, precede the torrent of tears. The excitation is propagated to the muscles of the glottis, the diaphragm and finally the sobs burst shaking convulsively the entire body the same as the laugh.

I have reserved the tears themselves for the last. But the flux of tears also depends upon the motor nerve. It occurs with a suddenness and abundance that can be occasioned only by a vaso-secretory nerve, such as is the chorda tympani for the salivary function. The lachrymal secretion occurs under conditions exactly analogous to those of the salivary secretion in the memorable experiments of Claude Bernard and Vulpian. We know that the chorda tympani corresponds closely to a motor nerve in origin, relations, distribution and function. It is a filament of the facial as is the branch of the ophthalmic which supplies the great lachrymal gland. Likewise blushing is essentially a phenomenon of vasodilatations and not the result of passive congestion such as is seen in the beginning of asphyxia. Thus all these effects depend upon the same gray motor column. Let us attempt then to locate the centers which preside over their production. I present to you a section representing the optic thalamus with the posterior segment of the internal capsule; below, the posterior commissure and the tubercula quadrigemina in the middle, the aqueduct of Sylvius surrounded by its gray matter. I spoke to you in my last lecture of the nuclei of the motor oculi communis distributed one above the other in this region; they form part of the same gray column that we find lower down, constituting first the origin of the facial, then that of the pneumogastric, the glosso-pharyngeal and further down the spinal accessory; farther still the origin of the phrenic which we shall see comes into play to shake the thorax in violent laughter or sobbing. With these few anatomical facts we may at once examine the process of laughing from the simple smile, a slight facial contraction, to the burst of laughter which convulses like a clonic spasm, a process always following the same track, always descending for the short diagonal of the rhomboidal floor of the fourth ventricle down to the gray matter of the cervical cord. And the stimulus of these bulbo-spinal nuclei is not arrested here since we may have a generalized spasm, a veritable



convulsion, the "crazy laugh" the "epileptic laugh." Finally I would add that the action of the gray motor column brought into play in laughter and weeping always begins at the level of the nucleus of the fifth pair, never higher. Above this the motor column corresponds to the intrinsic and extrinsic eye muscles and saving in exceptional cases the gray nuclei of the aqueduct do not participate in the action of the ganglionic masses in the expression of emotion. Nor does the nucleus of the patheticus take any part in this excitation. The fourth pair is misnamed for it has never added one whit to the pathos of any face.

Before inquiring as to the communications which serve to connect the cortical centers with the bulbar nuclei, let us endeavor to see the relation of these nuclei to expression itself. Investigators have sought for a long time to determine the location of the decussation of the motor tracts coming to these nuclei of expression. As the movements of facial expression are bilateral, at least in frank laughing and weeping, we must admit that the decussation, complete or incomplete, is perfectly symmetrical. The anatomical details are but little known and are still being studied, but recent experiments by Muratow have shown that the decussation of the motor tract for the face, superior and inferior, excluding the eye muscles, takes place just below the corpora quadrigemina, that is, just below the region which is never interested in these reflex phenomena of laughter and weeping.

It remains for us to inquire if the facial has different branches for laughter and for tears. No; in all probability the same nerve fibers and the same nuclei serve for these two opposite expressions, but, varying with the case, they create different motor combinations. We may say in a general way that the expression of laughter is due rather to the action of the levators and weeping to that of the depressors. And if we consider the muscular actions of laughing and of weeping as a whole we might make a somewhat similar statement of the other muscles engaged, the nuclei of the pneumogastric and spinal accessory supply muscles of inspiration and expiration. It is the first which are employed in the act of sobbing, and the second in laughing; for whether it be bursts of laughter, or a passion of weeping, it is the respiratory muscles which are in action. In short, although the nuclei of the motor column always stimulate their corresponding muscles our emotions are not expressed by means of such and such a nucleus taken as a whole. It would seem that they choose certain cells of each group. But if we are moved by more complex emotions, several

of these groups of cells may be discharged together, and if an emotion is extremely intense the neighboring groups cannot remain intact and the muscles of the face which serve for a frank laugh or cry combine their action so as to produce a complex result; we "laugh till we cry," we "weep for joy." Let me add that as great emotional agitation, so also do certain pathological conditions give rise to a double expression of sadness and joy. Thus the bulbar phenomenon is not always simple; the activity of the nuclei is not always uniformly localized. The reason lies in the fact that the medulla oblongata does not act as a purely reflex center but obeys commands from above, from the cerebral cortex. A direct bulbar stimulus has no effect but a reflex contraction without any manifestation of expression, as experiments have decisively shown. These orders which produce psycho reflex acts are transmitted, without doubt, by the optic thalamus. The connections of the thalamus with the bulbar nuclei are so arranged that for every expression, simple or complex, there exists a center of command. From this center are sent out the impulses to the bulbar nuclei, these impulses being discharged by a psychic stimulus.

Suppose for a moment a lesion exclusively irritative and confined to the medulla. You would see nothing but a spasm, a grimace resembling at once the act of laughing and of crying. All these groups of cells would act at the same time because the thalamus does not intervene to make a choice. Do we find such phenomena in neuropathology? Without a doubt, and I have already shown you the fact on another occasion apropos of a case of amyotrophic lateral sclerosis. You remember the characteristic meaningless laugh and weeping of Charcot's disease. It is due to the irritation of all the bulbar nuclei for facial expression. It is neither frank laughter nor tears, but a mixture of both.

While the complex phenomena of laughter and weeping are the consequence of an action of the cerebral cortex, we must not conclude that they are always of exclusively cortical origin. Laughter and weeping are often of a purely reflex nature; that is to say that they occur under circumstances in which the cerebral hemispheres are entirely neutral. Tickling, for instance, provokes the most frank laugh, a laugh nevertheless over which the cerebrum exercises no control whatever. There are still other kinds of activity of this system of bulbar nuclei which are, like laughter and weeping, sometimes psychic in nature, sometimes reflex. Yawning is one of these acts, with an established mechanism in which participate the muscles supplied by the seventh, tenth,

eleventh and twelfth cranial nerves, and a whole series of cervical nerves. Sometimes it is reflex, sometimes psychic; one yawns from ennui, and one yawns from want of sleep. Spasmodic yawning is also observed sufficiently often in hemiplegia. Nor is it simply a symptom added to the ordinary spasmodic phenomena of epilepsy, (Féré),<sup>5</sup> or hysteria, (Charcot,<sup>6</sup> Gille de la Tourette, Guinon et Huet).<sup>7</sup> It seems indeed to depend upon an organic lesion but I will not insist upon this point for the present. I confine myself to the mention of it as a symptom; as a complex systematic act, the frequency and intensity of which are exaggerated as are the laughter and weeping of hemiplegia mentioned in the beginning of the lecture, and which may be produced by a mechanism purely reflex or psycho-reflex.

To return to the laughter. It is about demonstrated that its center of coördination is in the thalamus. It is here, too, that the stimulus of tickling is reflected; but the laugh thus provoked has no relation to the pathological laugh we are studying.

I present you here a patient whom you have already seen. She is a hemiplegic, but her hemiplegia is not of cortical origin; it is due to a lesion of the basal ganglia which has produced the facies called "pseudo-bulbar." You observe that her hemiplegia is left-sided, and her gait decidedly spastic. Question her, and she replies with the greatest difficulty, moving the tongue with effort and drooling saliva. This pseudo-bulbar paralysis is the result of a lesion which has cut, above the motor decussation, the fibers which pass from one hemisphere to the other to produce the bilateral movements of the face. The result has been a bilateral facial palsy. This paralysis, however, is not complete; the patient can articulate, but it is exceedingly difficult to understand her. You know enough of the history for us to study together this peculiar group of bulbar symptoms that she presents in such a marked degree. The neighbors of this woman in the ward are hysterical cases who, by the way, are far from being as sensible as she is. The other day, during our visit, one of them presented a typical picture of hysterical delirium. She assumed the most fantastic attitudes, with gestures pompous and comical; she made solemn salutations and bowed as if at mass with imperceptible solemnity. Our patient, although habituated to such scenes, could not help laughing, and having begun, you remember what followed. The explosion of her apparent gaiety filled the ward with ringing laughter, and she became, in spite of her efforts, much noisier than the hysterical subject.

This variety of laughter is absolutely identical with that of the patient whom I just presented; the hemiplegia which is the cause of this hilarity is so by reason of a lesion of the basal ganglia; the connection of the thalamus with the cortex is respected. The result of the cortical stimulus is a laugh to-day; yesterday it was tears; she was discouraged and indulged in a fit of uncontrollable weeping.

The other patient before you is also a hemiplegic whose face, as you see, is absolutely impassive. He can still walk but with very short steps. However it is not his hemiplegia to which I would call your attention. Notice the physiognomy, in which we do not see evidence of a single emotion; an immovable mask. The eye muscles are normal but deglutition is difficult and speech almost impossible; the intellect is intact and he comprehends perfectly his attacks. He is a laugher and a most pronounced one. Simply the word "laugh" spoken to him causes a burst of merriment that he is unable to control; he laughs till he chokes. He is menaced at all times with these attacks, is never sure of himself, and avoids everything which might induce them, for once begun he is bound to submit to his irrational laugh until it is exhausted. It is like an epileptic fit fated to pass through the cycle of convulsive manifestations and which nothing can arrest once the aura has appeared.

Finally I show you a young woman presenting the complete symptom complex of the hemiplegic form of multiple sclerosis. The spasmodic condition dates back three or four years. In contrast to what you have just seen in the preceding patients, she has no immobility of the face; on the contrary she talks with a grimace, puckering and drawing her lips in an affected manner. Her bulbar nuclei are not paralyzed but she is subject to the same attacks. The first time that we saw her she filled the ward with laughter that nothing could stop.

Thus we see a burst of spasmodic laughter in subjects whose faces are impassive, and we find the same phenomenon in this woman whose face, on the contrary, is in perpetual motion. Can a lesion explain these apparent contradictions? I will endeavor to demonstrate that it may. But first I wish to say a word about the anatomical arrangement of the lower levels of the cerebral hemispheres.

The figure represents a horizontal section of the left hemisphere. You recognize the island of Reil, the subcortical white matter, the claustrum, external capsule, lenticular nucleus and cau-

date nucleus which is nothing but the continuation of the putamen; posteriorly we find the cauda of this nucleus and internally the optic thalamus and ventricle. We know perfectly the course of the pyramidal tract in the internal capsule, the motor tracts for the inferior and superior extremities and for the face excepting the fibers for the orbicularis.

All these known divisions belong to the posterior limb of the capsule; in front is a terra incognita, the anterior limb which toward the peduncle becomes the "innominate tract" still called the "psychic tract." This tract does not always degenerate after cortical lesions. It is distinctly connected with the frontal lobes and is at first constituted in part by the fibers of the anterior thalamic region which rejoin it after having been distributed in the thalamus and which form the anterior root of this ganglion. Laterally from this tract, separated by a bundle of capsular fibers is seen Arnold's tract which extends to the vicinity of the corpora quadrigemina. We do not know which of these two tracts it is that conducts the stimulus from the frontal cortex to the centers of coördination in the optic thalamus, but we may be sure that the inferior fibers of one of them must fill this role. For in cases of complete destruction of the anterior limb of the capsule, see what we observe; whatever effort the patient may make he cannot succeed in stimulating the center of expression for the opposite side; one hemisphere only is active and he laughs on one side only. On the contrary let us suppose a lesion involving the knee of the internal capsule, the voluntary motor tract for the face, the geniculate tract. The psycho-reflex movements will then be possible and the patient will retain the power of facial expression. The symptoms will be those of pseudo-bulbar paralysis from a unilateral cerebral lesion.

Finally if the two geniculate tracts are involved, either by a double symmetrical lesion or by a single one at their point of decussation, the subject will be unable to execute a single voluntary movement of the face. Nevertheless he will have a tract conducting the stimulus of psychic impressions to the internal capsule and as far as the center for the coördination of facial expression; thus he will still be able to excite his bulbar nuclei, but their action will be deranged because they have, so to speak, lost their head; it is impossible for the patient to master his hilarity by voluntary inhibition and the "bulbar laugh" without limit and without control is the result.

Such is the lesion which I infer in the hemiplegics of whom I

just spoke, a lesion purely irritative when located in contact with fibers of the anterior limb of the internal capsule and paralyzing when it cuts the posterior limb. You see then why our patients have immobility of the face and why, also, when stimulated, excessive laughter or tears result. Ordinarily, indeed, they can be quiet for it is rare that the voluntary motor tract is totally destroyed but the veriest trifle sets them off and the laugh terminates in an explosion such as you have just witnessed with hiccough, laryngeal constriction, salivation, even vomiting, becoming a truly convulsive attack when the process extends to all the muscles.

Prof. Bechterew assumes a lesion of the thalamus in his case. I admit that I can scarcely imagine such a location; that is, a destructive lesion of the center of distribution of movements. If, in a hemiplegic, there were a lesion at the level of these centers, a lesion not destructive, but simply irritating, I admit that it might produce these irritative troubles of the movements of expression; but how at the same time explain the hemiplegia of Bechterew's case? It is absolutely necessary that there be a lesion of the internal capsule. It appears to me then much more reasonable to suppose a lesion of the capsule destroying the voluntary motor tract and in contact with the fibers for expression without destroying them. If, in cerebral affections, spasmodic laughter or weeping may depend upon this lesion, let me add that it may be seated high up or low down. The lower down it is toward the medulla the less we will find differentiated the expressions of joy, pain, hate, terror, etc., corresponding to the infinitely delicate play of the centers for the tegumentary muscles of the features. And this is explained by the well-known disposition of the various "systems of projection," the fibers converging more and more as we descend. If then we have a lesion located at the point of convergence we must admit that all the emotions will be expressed by a single play of feature. This is the case in the striking facies of pseudo-bulbar paralysis.

The localization which we have just studied produces in addition to the paralysis spasmodic troubles such as are caused by any irritative affection, as amyotrophic lateral sclerosis. For example you remember in the last patient presented we found the signs of multiple sclerosis of the hemiplegic form. There is besides a whole category of patients in whom we find this bulbar type, viz., idiots, who also have attacks of laughter. From chronic lesions of the capsule, of the ganglia of gray matter or of the cortex they realize the pathogenic conditions of an unstable physiognomy. Simple

atrophy, porencephalus, chronic diffuse encephalitis, etc., may be the cause of the phenomena. In these patients as in the preceding the lesion affects that part of the capsule which is frequently called the "psychic tract," the projection of Arnold's tract, or the anterior root of the thalamus. Here I must say mea culpa. At the time when I was studying with Prof. Charcot, the degenerations of the pes pedunculi our attention was directed to this bundle to which, as yet, no name had been given. I scarcely knew how to designate it and called it the "psychic tract." The name has been adopted in several classical works, but I am the first to recognize the fact that it is not entirely satisfactory. It certainly is the most direct path of communication between the frontal cortex and the gray matter of the base and medulla. If it takes no essential part in the elaboration of mental acts it at least plays a role in the external manifestation of these acts; a connecting link between certain centers where thought is born and others less noble that automatically present its reflections, it is the indispensable balance wheel of the movements which, rightly or wrongly, have been called the psycho-reflexes. For this and this only it seems in a measure to merit the title.

<sup>1</sup> Soc. of Neurol. and Psych. of Kasan, April 24, 1893.

<sup>2</sup> Die Bedeutung der Sehhügel auf Grund von experimentalen und pathologischen Daten. Virchow's Arch., 1887, vol. 110, p. 102.

<sup>3</sup> De la physionomie et des mouvements d'expression, Paris, 1869.

<sup>4</sup> Sensations et mouvements, Paris, 1887.

<sup>5</sup> Nouvelle Iconographie de la Salpêtrière, 1888, t I, p 163.

<sup>6</sup> Leçons du Mardi, 1888-89 et Clinique des maladies du système nerveux t I. p. 429.

<sup>7</sup> Nouvelle Iconographie de la Salpêtrière, 1891, No. 3.







