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Original Papers.

SOME PROBLEMS IN NEUROLOGY.

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No. II.—PATHOLOGICAL LAUGHING AND CRYING.

The problem presented by certain cases of abnormal emotional expression, in the guise either of exaggerated or uncontrollable laughing or crying, or, conversely, of paralysis (at least in part) of the same mechanism, has not attracted much attention in recent years; nor has much detailed criticism been offered, or advance made, on the position adopted by Nothnagel [1] forty years ago in reference to the latter, or by Brissaud [2] thirty years ago in respect of the former. From time to time doubts have been cast on the tenability of the hypotheses, yet little of a constructive nature has taken their place. The time seems opportune for a revision of the whole question, and for the offering of a somewhat different explanation of the syndromes. Again, as long ago as 1884, Professor William James [3] admitted with characteristic candour that if the hypothesis of the emotions suggested by him "is ever to be definitively confirmed or disproved it seems as if it must be by asylum physicians and nervous specialists, for they alone have the data in their hands." As far as I am aware, however, no attempt has been made thus to prove or disprove it in the light of neurological knowledge derived from clinico-anatomical facts, so that a tardy contribution from this viewpoint also may not be without interest.

DEFINITION AND DELIMITATION.

At the outset the ground must be cleared by specifying exactly what is meant, for the purposes of this communication, by pathological

laughing and crying. In the first place, reference is made solely to cases of organic nervous disease, cases in which, as a sequel to and consequence of a recognizable cerebral lesion or lesions, ' attacks ' of involuntary, irresistible laughing or crying, or both, have come into the foreground of the clinical picture. It must be clearly understood that a not unnatural result of severe or chronic nervous disease may be a general depression of spirits, favouring tearfulness and irritability and other manifestation of an emotionally altered psyche, but such conditions are not here considered. The emotional outbursts of the hysteric and the facile moods of the neurasthenic, likewise, are foreign to our subject. Patients, too, who suffer from cerebral arteriosclerosis, in particular those in whose cases other indications point to impairment of function of the basal portions of the brain from an *état lacunaire* or other vascular change, often exhibit signs of an abnormal emotional state, and while this is, without doubt, the direct result of the disease, the usually diffuse nature of the latter precludes its being utilised for topographical purposes. Occasionally, however, the symptoms make their appearance in an arteriosclerotic case after an ictus, or series of these, and in such examples the localizing value of the syndrome may be in no way inferior to that of other cases with a more restricted and clean-cut pathological process for a basis. Allusion, therefore, is made to the occurrence of exaggerated, forced, involuntary, uncontrollable laughing or weeping—the *Zwangslachen* and *Zwangsweinen* of German writers, the *rire et pleurer spasmodiques* of the French.

In the second place, the comparatively rare cases of organic disease in which there is conservation of voluntary facial movement with paresis or paralysis of the same musculature for the involuntary movements of laughing or smiling have long been known to the neurologist. This defect in the mechanism of laughing is clearly but a part, and it may be only a small part, of the total somatic expression of that particular emotional state, nevertheless as such it is deserving of close attention. Its significance will be duly considered in this paper, and a fresh explanation offered of its pathological physiology.

CLINICAL ILLUSTRATIONS.

I. Among the organic affections apt to be associated with the occurrence of pathological laughing or crying may be enumerated double hemiplegia, pseudobulbar paralysis and disseminated sclerosis ; their appearance after a single hemiplegia has also been observed, and, as remarked above, the symptom-complex is of moderate frequency in certain stages of basal degeneration from diffuse vascular processes. The exact nature of the morbid affection is of less importance than its site ; tumour growths, infective conditions, or vascular degenerations,

provided they are appropriately situated, may produce the symptoms indifferently.

By way of clinical illustration some personal cases may now be cited.

1. *Double Hemiplegia.*—One case of ' crying ' and one of ' laughing ' may be selected.

Case 1.—A woman of fifty-seven had suffered from left hemiplegia for one year, when a second stroke occurred involving the right side. Ever since the latter the daughter remarked that her mother had become, as she put it, ' hysterical,' laughing and crying at nothing.

On examination the patient was seen to have a distinctly vacant, apathetic facial expression at rest. She was able to move the facial muscles voluntarily on both sides, though there was slight weakness of the left corner of the mouth. On the slightest stimulus, even when the observer simply came to

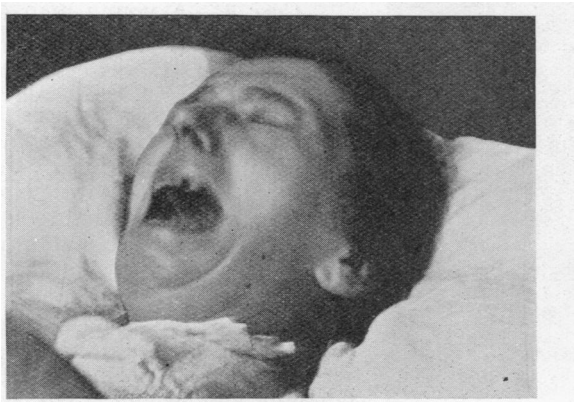

FIG. 1.—Case 1. Pathological crying in a case of double hemiplegia.

her bedside, she at once assumed a most lugubrious expression, her mouth opened widely, and a long, almost noiseless bout of weeping ensued, lasting for many seconds, even minutes, at a time (*Fig.* 1). During this spasmodic crying both sides of the face moved equally, and the eyes suffused with tears. Laughing attacks were extremely rare in comparison with this incessant weeping.

Case 2.—A man, aged sixty-seven, had two strokes in the same year (1916); the first was on the right, with very moderate aphasic disorder; the second was on the left, and comparatively mild. Ever since the first attack, and to an increasing degree since the second, he had exhibited characteristic involuntary laughing. Whatever the emotional stimulus, and however slight, he at once began to laugh, and laugh loudly. Thus on reading the war news he used at once to begin to smile, and the more serious and anxious the news, the more he laughed.

On examination there was some voluntary facial paresis on both sides, especially the left, some dysarthria, and some dysphagia, but during the laughing the facial movements were in no way restricted. A double extensor response was present.

2. *Pseudobulbar Palsy.*—The syndrome occurs in this affection with

greater frequency than in any other (*Fig.* 2). Hartmann [4] reports eight cases of pseudobulbar paralysis, in every one of which moderate or slight voluntary weakness of facial movement was associated with pronounced spasmodic laughing or crying, or both. Similar clinical examples have been recorded by Schaffer,[5] Weisenburg,[6] and many others. Instead of quoting personal cases I shall take this opportunity of referring in some detail to the remarkable instance of the affection reported by my former teacher, the late Dr. Charles Beevor.[7] As it was published in a somewhat out-of-the-way foreign journal it has never received the attention which its altogether unusual features amply justify. It so happens I am in a position to add some facts to Dr. Beevor's striking study of the case.

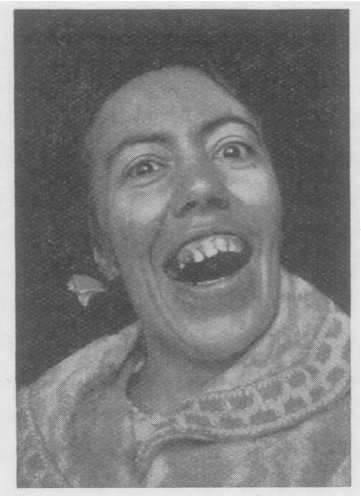

FIG. 2.—Pathological laughing in a case of syphilitic pseudobulbar palsy.

Case 3.—A young man of twenty-three, known to have been infected with syphilis, had three hemiplegic attacks, two involving the left side and one the right, as a result of which there was *complete* loss of all *voluntary* facial movements as follows: closing the eyes, elevation and retraction of the angles of the mouth, opening the mouth, closing the mouth; there was likewise absolute loss of voluntary biting, deglutition, phonation with elevation of the palate, and of voluntary inspiration and expiration (such as coughing). Both sides were affected equally. Contrasting with this profound degree of volitional palsy was the preservation of the emotional movements of laughing and crying. The patient was continually laughing; in fact, on the slightest provocation, or on none, he went off into a torrent of laughter, which made examination of his facial and respiratory condition very difficult. The reflex movements of coughing, sneezing, and yawning were readily obtained, but it was observed that in laughing the angle of the mouth was not retracted so well on the left side as on the right, indicating slight diminution of the emotional facial movement on the left, whereas in yawning the facial movement was symmetrical. As the patient was unable to open his mouth, it was the habit of the house physician to sit at the foot of the bed and yawn deliberately. Eventually the former caught the infection and yawned automatically, whereupon the Sister of the ward promptly took the chance of popping food into his mouth.

A further illustration of the phenomenal *rire spasmodique* may be added to the account. It so happened about this time that a peripatetic quack was touring the minor music halls of London and claiming to cure all and sundry complaints by means of electricity. The patient went to this individual, and was shown on to the stage. Diagnosed as hysterical by the electric 'expert,' he was submitted to a series of violent and extremely painful electrical applications, but the more they hurt the more he laughed, till at length he was quickly hustled off, and on his return the following evening was refused admission.

In view of its almost unique nature and significance from the standpoint of the mechanism of laughter and its localization, further reference is made to this case below.

3. *Single Hemiplegia.*—It is not a little curious to note how many cases have been recorded in which a single ictus seems to have precipitated the tendency to the symptoms under consideration. Examples have been given by Brissaud,[2] Broadbent,[8] Burzio,[9] Mills,[58] and others. In some instances, doubtless, there is more widespread vascular disease than the single stroke would indicate, and in some tumour cases, similarly, there is greater disturbance of cerebral function than the unilateral symptoms by themselves suggest. Yet the onset of the syndrome may undoubtedly coincide with the appearance of limb symptoms which are strictly one-sided.

Case 4.—A woman of fifty had an attack of right hemiplegia and aphasia, and thereafter became peculiarly lachrymose, bout after bout of weeping succeeding each other all through the day, with copious tears. Thus, when I used to come to her bedside, this was the unvarying signal for a fresh outburst of uncontrollable crying; in fact, whenever she was spoken to the same occurrence was noted. On examination there was slight volitional weakness of the right side of the face, but the asymmetry was not observed during the bouts of weeping. Before the hemiplegia no such emotional overaction had ever been noticed.

Case 5.—A male patient, aged fifty-nine, presented in a fairly typical form the thalamic syndrome in association with a left hemiplegia. Thus, in addition to the corticospinal symptoms (extensor response, etc.), there was found on examination objective diminution to painful and thermal stimuli over the same side, with highly characteristic over-reaction; an athetoid attitude of the hand was also noted, and occasional involuntary movements of the same. He complained of constant and severe 'burning pins and needles' in the left arm and left side of the face.

In addition, the patient presented the symptom of involuntary laughter in a marked degree. This had made its appearance after the stroke, but it should be stated it was greatly augmented when, some two years later, weakness of the right side, associated with some sensory change, began to develop.

Though involuntary laughing or crying is not regarded as in any sense a usual or even occasional accompaniment of the so-called 'thalamic syndrome,' there is no reason why it should not sometimes happen that the two are combined, and the above is an instance in point. A similar example came under my notice a number of years ago, in which spasmodic weeping took the place of the laughter of case 5.

By way of contrast, allusion may here be made to a series of cases published by Féré,[10] under the title of '*Le fou rire prodromique.*' In these the onset of irresistible emotional overaction *preceded* the development of hemiplegia. In the first case an elderly gentleman of sixty-four began to suffer from *rire spasmodique*, being convulsed with laughter over trifles and failing altogether to inhibit the performance. A few

months later this was followed by right hemiplegia and, subsequently, by left, and death ensued from pseudobulbar paralysis. The second case was that of a man, also aged sixty-four, in whom uncontrollable explosions of laughter, from minimal stimulation, were followed always by an irresistible desire to sleep. These phenomena were of daily occurrence for some four months, when a severe left hemiplegia supervened. In view of the particularly interesting fact that, thereafter, all involuntary explosive laughter ceased up to the date of the patient's death, eighteen months later, it is regrettable that further exact details of the neurological condition were not given.

No case of this description has come under personal notice, but I have seen a case of disseminated sclerosis in which risibility, amounting on occasion to involuntary laughing, was the first symptom to attract attention.

4. It is unnecessary to furnish clinical examples from cases of other organic affections of the nervous system, but an exception may be made in respect of disseminated sclerosis, because of the frequency of the syndrome in that disease.

Case 6.—In the case of a man of thirty-two, with the typical symptoms of the affection, attention was first directed to the emotional change by the fact that when reading of a perfect stranger's death he would begin to weep; with the narration of amusing incidents exaggerated laughing would set in. Under observation bursts of long, uncontrollable, but almost noiseless laughter took place at the veriest trifles. In the course of my examination I asked the routine question whether he had any difficulty with the bladder, and replying in the affirmative, he added he had already 'ruined four pairs of trousers,' and went off into an apparently interminable series of peculiar hollow laughs, which convulsed the whole ward as well as himself. So facile became the mechanism, so completely without control, that he would laugh whenever he began to speak, as though the stimuli of contracting muscles were sufficient to set it off.

II. It is time, however, to turn to cases characterized by volitional normality but emotional abnormality of facial movement.

Hitherto, in the majority of the quoted cases, it has been remarked that any asymmetry of voluntary facial action disappeared when the features have been innervated under the influence of stimuli of the emotional order. The occurrence of facial paresis or paralysis in emotional expression is a very old observation. Almost a century ago Sir Charles Bell [11] wrote: "As you find the portio dura in possession of distinct properties, all of them related to respiration, breathing, speech, and expression, you will not be surprised that these functions should occasionally be differently affected; as, for example, a man will continue to possess the power over the nerve, as the nerve of speech, and yet he will be incapable of expressing the usual signs in laughter or in crying. In short you find that your patient sometimes exhibits

paralysis of the side of the face only when he smiles or laughs, at other times it is not observable." An old but impressive clinical illustration is furnished by the case recorded by Stromeyer [12] in 1837, concerning a girl of twelve years, "in whom the right side of the face continued expressionless in emotions, and showed no increased action in accelerated respiration after running, going up stairs, etc. Nevertheless, the child was as able to control the muscles on this side as those on the left; she could move the angle of the mouth, dilate her nostrils, wrinkle her forehead, and contract her eyebrows at will. . . . On compressing the epigastric region, it appeared that the right

FIG. 3.—Case 7. Normal volitional facial movements.

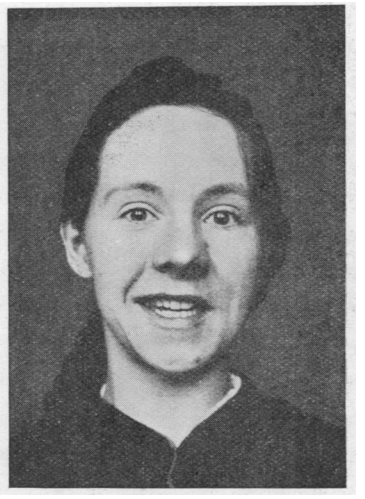

FIG. 4.—Case 7. 'Mimic palsy' of left side of face in laughing.

half of the thorax scarcely took any part in the forcible (involuntary) thoracic respiration which was induced by the pushing back of the diaphragm. When this kind of examination was made, the apathy of the one half of the face was particularly manifested at the nares; whilst the right one remained immovable, the left one expanded fully at every act of inspiration." Ancient though it is, this case is particularly informative and will be referred to again.

Numerous examples of the combination of voluntary control with involuntary paralysis have since been recorded (Nothnagel,[13] Bayerthal,[14] Mills,[15] Borst,[16] Nonne,[17] Monrad-Krohn,[18] and many more). Of various examples that have come under personal observation three only will be cited.

Case 7.—A young woman of twenty-seven had suffered for six months from increasing headache, giddiness and attacks of vomiting. Her cerebra-

tion became slower and her memory poor. For about the same time weakness and paræsthesiæ of the left limbs had been observed.

On examination the optic discs were clear but hyperæmic. Objective evidence of a slight left hemiparesis (arm and leg) was obtained. When the patient showed her teeth, closed her eyes, etc., no paresis of the left face was discoverable, but on emotional stimulation a striking asymmetry was at once shown, the left side exhibiting a considerable degree of 'mimic paralysis' (*Figs.* 3 and 4). The later course of the case indicated more definitely the presence of a cerebral tumour in the region of the right internal capsule and right regio subthalamica.

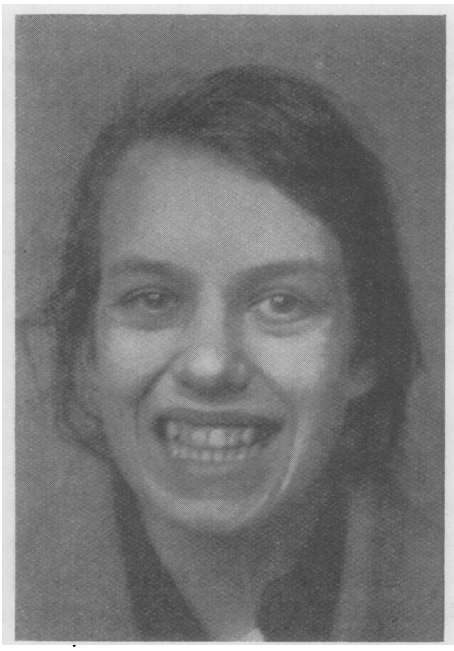

FIG. 5.—Case 9. Normal volitional facial movements.

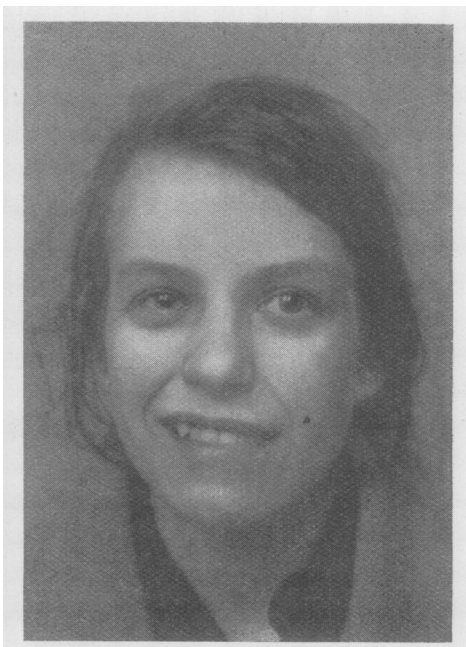

FIG. 6.—Case 9. Emotional or 'mimic' palsy of left side of face in laughing.

Case 8.—A male patient of forty-one, known to have had syphilis, suffered from a stroke on the right side involving arm and leg, and not long thereafter developed a highly typical right posthemiplegic hemitremor. On examination it was found that the left pupil was inactive to light and reacted poorly with convergence, whereas the reactions of the right pupil were normal. Lateral conjugate deviation was good in both directions, but upward and downward movement was very poor indeed. Voluntary movement of the facial musculature was normal on the two sides, whereas on smiling the right facial movement was minimal, and on laughing the difference was notably accentuated. That is, the right side of the face exhibited expressional paralysis.

Case 9.—A girl, aged seventeen, developed the characteristic symptoms of intracranial tumour in the shape of headache, giddiness, vomiting and papillœdema. The presence of a double Argyll Robertson pupil, nystagmus,

tremor, inco-ordination in finger-nose test (left), etc., pointed clearly to a mesencephalic localisation.

On volitional movement of the face no defect was observable, but on expressional movement (laughing) the left side of the face moved only slightly, whereas the right side moved normally (*Figs.* 5 and 6). At the autopsy a tumour was found situated in the tegmentum and upper pons, involving the left side more than the right (*Fig.* 7).

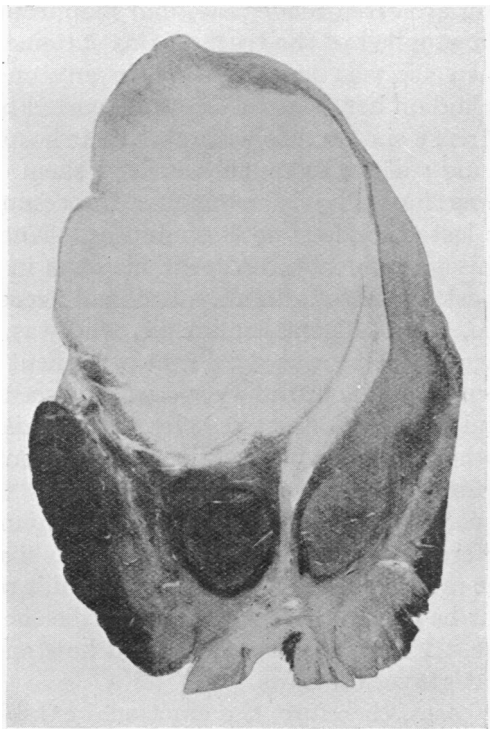

FIG. 7.—Case 9. Tumour of mesencephalon, involving mainly the left side, and interfering with the non-volitional faciorespiratory path through the tegmentum on that side.

It is worth noticing that in all the reported instances, as far as I have been able to ascertain, this paresis or paralysis of involuntary facial movement has been on one side only. No bilateral case seems to have been observed; none has come under my notice, and a similar remark has been made by Spiller.[19]

THE EMOTIONAL FACTOR IN PATHOLOGICAL LAUGHING AND WEEPING.

The natural question that must arise for discussion is whether the emotional outbursts of which descriptions have been given correspond

to, or reflect, the mental state of the individual concerned at the moment of their expression. Is there a strict relationship between the patient's feelings and their exteriorization as noted by the observer? Are such overwhelming laughter and tears activated by appropriate stimuli, or do they in their turn induce the appropriate frame of mind, or have they any emotional content at all? To enable us to come to some decision the following considerations appear pertinent.

1. The stimuli are often inadequate and inappropriate. Instances have already been supplied of the truth of this statement. One patient (case 4) cried when she was spoken to, when any one sat beside her, when a hand was laid on her arm. In a case reported by Giannuli,[20] the patient, a man of sixty-six, used to walk about the hospital with his eyes glued to the ground; if he so much as raised them to meet anyone else's gaze he was immediately overcome by compulsory laughter, which sometimes lasted for four or five minutes. Knowing the irresistible nature of this phenomenon, he spent his days in endeavouring to avoid even the most trifling of stimuli. Brissaud [2] recounts the history of a patient of his, an intelligent hemiplegic, who was told incidentally by a lady that her little dog was dead; in a moment the fountains of emotion were opened; a mournful visage was succeeded by tears, and tears by sobs, and sobs, unfortunately, by a Rabelaisian effect on his sphincters. Another of Brissaud's patients, a student with syphilitic hemiplegia, was forced to abandon novel reading; "les malheurs de l'héroïne le font éclater en sanglots, ses joies lui donnent de véritable transports." Reference has been made (case 6) to spasmodic laughter accompanying the mere attempt to speak; Giannuli's patient, similarly, laughed as soon as he opened his mouth to describe the pain he suffered from renal disease. I have already mentioned how one of my patients (case 2) laughed at grave war news.

In not a few cases, therefore, the emotional exhibition develops at the bidding of stimuli so minimal as to escape detection; in others, it is motivated by impulses appropriate enough if trifling, but it is excessive out of all proportion to the impressions originating it; in still others, a stimulus of a particular quality is followed by an emotional outburst of a contradictory sort, to which it is not appropriate. Intelligent patients suffering in this fashion are often conscious of the insufficiency of the psychical impulse, and the more intelligent they are the more painfully aware do they become of the incongruity in their affliction.

2. From what has been said it will be understood that the apparent, visible emotion does not necessarily correspond to the patient's real feelings at the time—an observation which has often been made. *Apropos* of some cases of disseminated sclerosis, Oppenheim [47] says that "the patient has to laugh against his will, although his mood is not gay; this distresses him greatly." A patient of sixty-three, whose case is

given by Dupré and Devaux,[21] a pseudobulbar with excessive attacks of laughing and crying, indicated by gestures of impatience and denial how much he was annoyed and ashamed at his performances, and how he suffered in mind at the constant caricaturing of his real feelings by their outward expression. A patient of my own (case 5) told me, with every sign of disgust, how one day his daughter had hinted plainly enough that she thought " Dad was putting it on a bit " ; incensed at her unbelief, he rose from his chair to give her a box on the ears, but, his legs giving way, he had to throw his arms round her neck to keep himself from a fall, and in this (for his angry state of mind) irritating and ignominious position he burst into explosive laughter. I have endeavoured to ascertain from intelligent patients whether when thus overcome by laughter against their will and in opposition to their real feeling they do not, in spite of the latter, end by experiencing the emotional state commonly associated with laughter, and I am satisfied it is not so, in some instances at least. On the other hand, Moutier [22] was informed by a young pseudobulbar patient that a single or brief attack of *pleurer spasmodique* left him quite indifferent, whereas prolonged and repeated bouts had the effect of saddening him and of bringing on tears legitimately motivated by the thought of the infliction under which he laboured.

Thus if the exaggerated laughter or weeping of the hemiplegic or pseudobulbar may, and often enough does, correspond to his emotional mood of the moment, it is not so always ; we can scarcely suppose that inextinguishable laughter represented the feelings of the patient (case 3) undergoing painful treatment at the hands of a showman.

3. As a further point, it is important to note in some instances the invariability of the emotional response, whatever the stimulus. Some of the sufferers can only laugh, others can only weep. Why this should be so is not easy to determine. Every one knows that laughter and tears are said to be ' near each other.' Crile's [23] theory in respect of normal emotional activity is that both laughter and crying have the purposeful effect of utilizing released kinetic energy, and of ' working it off ' until it is neutralized. Hence one may pass into the other almost indifferently. In some pathological cases, none the less, the mechanism seems to be ' set ' for one only. On the whole, a rough generalization suggests that in cases of disseminated sclerosis the manifestation is one of cheerfulness ; in pseudobulbars laughter and tears occur indifferently ; in arteriosclerotic cases tearfulness seems to predominate. Be this as it may, the evidence here adduced points to not infrequent conflict and incongruity between the patient's state of mind and its outward exhibition.

The difference in type, however, must not be taken too absolutely. Brissaud [24] gives an amusing description of a patient with extremely marked *pleurer spasmodique*, whose facies during the ' attack ' became

'*affreusement grimaçant.*' In the bed next to him was a case of *rire spasmodique.* The latter patient used to roar with laughter at the weeping of the former, and this on occasion led the first to change his *pleurer* to an equally phenomenal *rire,* though the tears and the lachrymose physiognomy to some extent remained through it all.

It is important, at the same time, to be assured of the fact that the emotional display is a genuine manifestation of feeling. No one who has seen these attacks of involuntary laughing or weeping can doubt the reality of their emotional content. Let it not be supposed they are mere 'play-acting.' On the contrary, prolonged exhibition of every manifestation of grief, in facial expression, respiratory accompaniment, and secretion of tears, or, alternatively, equally patent demonstration of hilarity, in features, respiratory movement, rosy and suffused countenance, and tears too, it may be, is too definite to be mistaken for the mere 'shell' of a mental state empty or devoid of emotional tone. I cannot, therefore, agree with Bianchi [60] when he declares that the "weeping and laughter of such sufferers are only *simulacra* of the real emotions." The display gathers impetus as it proceeds, and if it is initiated by trifling stimulation, and in a comparatively cold emotional atmosphere, its avalanche nature is conclusive proof of the involvement of the entire mechanism, somatic and visceral, of emotional expression. In a word, it differs from legitimate emotional performances solely in its inevitability, its frequency, its uncontrollable character, the occasionally contradictory relation of 'cause' and 'effect,' and the extreme facility with which it is induced; in expression and accompaniments it is identical.

BEARING OF THE PHENOMENA ON THE JAMES-LANGE HYPOTHESIS.

The theory of the emotions associated with the names of Dr. Carl Lange, of Denmark, and Dr. William James, of America, is too well known to require any elaboration or detailed mention in this place. Since, however, it seems to some extent to be misunderstood, or, at least, incorrectly applied, I have taken the opportunity of re-reading the originals in a recent convenient reprint.[25]

According to Lange, emotion is the product of (1) a cause—a sensory impression which usually is modified by memory or a previous associated image, and (2) an effect—viz., vasomotor changes, which in their turn produce changes in bodily and mental functions. He asks, "What lies between these two factors, or does anything lie between them?" As is known his answer is that nothing lies between; the bodily phenomena are aroused immediately by the cause, so that the emotion consists exclusively of the functional disturbances of the body. "Take away the bodily symptoms from a frightened individual; let

his pulse beat calmly, his look be firm, his colour normal, his movements quick and sure, his speech strong, his thoughts clear, and what remains of his fear ? " Whether a mental or a physical impression induces the reaction, the chief requisite for the formation of an emotional state remains the same for both, viz., the stimulation of the vasomotor centres. " We owe all the emotional side of our mental life, our joys and sorrows, our happy and unhappy hours, to our vasomotor system. If the impressions which fall upon our senses did not possess the power of stimulating it, we would wander through life unsympathetic and passionless, all impressions of the outer world would only enrich our experience, increase our knowledge, but would arouse neither joy nor anger, would give us neither care nor fear."

The views advanced so plausibly by James are not entirely identical. He does not postulate the intervention of stimulation of vasomotor centres, presumably those in the medulla, but rather holds that " particular perceptions produce widespread bodily effects by a sort of immediate physical influence, antecedent to the arousal of an emotion or emotional idea." One of his great arguments, as we know, is the purely speculative one of inability to picture an emotion without the consciousness of all the feelings of its characteristic bodily symptoms. " What kind of an emotion of fear would be left if the feelings neither of quickened heart-beats nor of shallow breathing, neither of trembling lips nor of weakened limbs, neither of goose-flesh nor of visceral stirrings, were present, it is quite impossible to think. Can one fancy the state of rage and picture no ebullition of it in the chest, no flushing of the face, no dilatation of the nostrils, no clenching of the teeth, no impulse to vigorous action, but in their stead limp muscles, calm breathing, and a placid face ? "

A third variant of the theory is that elaborated by Sergi,[26] who considers Lange's views too restricted, and includes in the mechanism other medullary centres for organic life than the vasomotor, viz., the respiratory and the vegetative or sympathetic centres.

It will be seen that the hypothesis is, in a way, described with slight inaccuracy as the ' peripheral theory ' of the emotions. Both Lange and Sergi assume the intervention of the bulbar centres *before* the peripheral elements are set in motion ; James, too, admits cortical activity *before* the periphery is reached by reflex currents. Yet all seem to be convinced that no emotion is felt in consciousness until the cortex is in its turn again reached, this time by visceral impressions. James, for example, gives the following *résumé* of his position : " An object falls on a sense-organ and is apperceived by the appropriate cortical centre ; or else the latter, excited in some other way, gives rise to an idea of the same object. Quick as a flash, the reflex currents pass *down* [italics mine] through their preordained channels, alter the condition

of muscle, skin, and viscus ; and these alterations, apperceived like the original object, in as many specific portions of the cortex, combine with it in consciousness and transform it from an object-simply-apprehended to an object-emotionally-felt."

Thus the first stimulus is ecto-peripheral, followed by an endo-peripheral stimulus, and emotions are not felt till the impression aroused by the latter reaches the cortex. But between these is an efferent impulse to the viscera and certain skeletal muscles, so that an in reality somewhat complicated degree of neural activity, both central and peripheral, must precede the arousing of the emotional feeling. A diagram from Kirchhoff [27] will render the idea more clear (*Fig.* 8).

Objections to the theory outlined above have been raised from different sides.

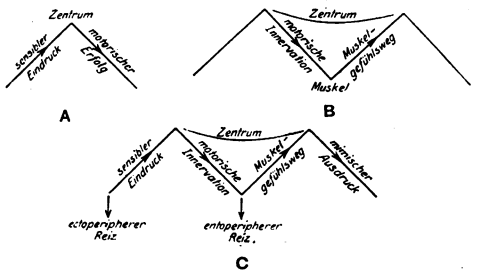

FIG. 8.—(Kirchhoff.)
A. Ordinary reflex arc.
B. The so-called reflex 'circle' of Bell.
C. The reflex 'chain' for the expression of emotion.

(1) Psychological difficulties have been emphasized by not a few who are competent to criticize. Sully,[28] for example, is convinced that the presence of an element of feeling at the very beginning of an emotional experience can sometimes be clearly observed. Pleasurable emotion can be started by 'agreeable sensations,' *viâ* eye and ear, and by the 'agreeable perceptions' which grow immediately out of these. When we laugh at some absurd incongruity in speech or manners "the perception which starts the laugh is an emotional perception," and "is flooded from the very first with the gladness of mirth." Further, once we are exhausted with laughing at a comedian we may be physically incapable of any further manifestation of emotional feeling, and yet we may still feel the full appeal of his funny stories, of his amusing antics. The objection has been raised by Störring [29] that Lange and those who agree with him reduce emotions and feelings to sensations, even though they be complex sensations of a particular kind. He maintains that emotions represent fusions of organic sensations and affective elements, and that it is impossible to peel away the mass of organic sensations from the total in such a way as to justify the statement that nothing is left. To enter at large into these and other psychological criticisms that have been offered is, however, outside the purpose of the present communication.

(2) Physiological objections have been formulated by Sherrington,[30] Cannon,[31] and Bianchi,[60] among others.

Sherrington, it will be remembered, by appropriate experimentation, removed completely the sensibility of the viscera and of all the skin and muscles behind the shoulder in a number of dogs, yet this procedure resulted in no obvious diminution of an emotional character. " A mere remnant of all the non-projecting or affective senses was left, and yet emotion persisted." His conclusion is that organic and vascular reaction, though not the actual excitant of emotion, strengthens it. Cannon's researches have been devoted more particularly to an analysis of the visceral components of emotional states, and he has shown, in terror, rage, and intense elation, for instance, that the responses in the viscera " seem too uniform to offer a satisfactory means of distinguishing states which, in man at least, are very different in subjective quality." Since *various* strong emotions are expressed in the diffused activities of a *single* division of the autonomic system the bodily conditions which have been assumed, by some psychologists, to distinguish emotions from one another " must be sought for elsewhere than in the viscera."

(3) The argument from clinico-pathological cases is the one to which attention is here specially directed.

Consideration of the clinical examples cited somewhat briefly above, will show, as I believe conclusively, that in some instances at least the outward expression by no means corresponds to the patient's real feelings. It may be repeated that even though the emotional states of the cases are pathological, morbid, produced by disease, or what you will, they are in quality identical with normal emotions. Yet more than one patient has protested against the laughter or tears being taken as the index to his actual affective state. The conclusion, I suggest, is unmistakable, that the bodily reverberation, as James calls it, is not *per se* the emotion ; the latter is not, so to speak, the mental symptom of the former. With all the outward appearances of mirth and hilariousness, and, further, with concomitant activity of the visceral mechanisms in facial expression and respiratory movement, and, in addition, with simultaneous activity of visceral components, as witness rosy countenance and sparkling eye, the individual may not only not feel happy, but his state of mind may be in patent conflict with the apparent emotion. It is clear, therefore, that the James-Lange hypothesis must be materially modified if it is to be brought into line with observations such as have here been recorded. There can be in these pathological cases no complete fusion between the peripheral and the cerebral components. It is obvious that the emotional framework may be activated and come into full play without its afferent impulses being synthetized with the pre-existing mental state into a harmonious whole. The trigger is touched ever so lightly, and the neural pattern is released into exaggerated action ; in spite of this, the patient may remain mentally detached, largely, if not always entirely, uninfluenced by the somatic and visceral

currents streaming centre-wards. From the standpoint of the clinician, therefore, I find myself in accord with the physiologist when he declares that "the reverberation from the trunk, limbs, and viscera counts for relatively little . . . as compared with the cerebral reverberation to which is adjunct the psychical component of the emotional reaction." Indeed, it might be said that some of my own and of the reported cases of others indicate the possibility of dissociation between the psychical and the physiological elements in the emotion.

Under normal conditions, practically all writers agree on the reinforcing and intensifying of the emotional cerebral state by the advent of somatic and visceral impulses (cf. Mott [32]), but our study of certain diseased conditions of organic origin must lead us to accept with caution the deductions as to the genesis of emotions made by the introspective method of the pure psychologist.

THE MECHANISM OF EMOTIONAL EXPRESSION.

In the expression of the emotions of joy and of sorrow, the only two with which we are here concerned, somatic and visceral factors are to be distinguished, though the latter, perhaps, are less in evidence or less intense in their activity than in other emotions that might be named. In the case of laughter there is, on the somatic side, involvement of facial and respiratory musculatures. It is unnecessary to describe the exact features of the former, familiar as it is to every one. As for the latter, the automatic rhythm of the respiratory centre in the medulla is rudely interrupted by prolonged inspirations, followed by short and broken expirations. Coupled with the respiratory movements are laughter sounds of laryngeal origin and of varying character and pitch. If the laughter is overwhelming, other muscles beyond those of face and respiratory apparatus will be implicated; in fact, there may be a good deal of diffused movement, even to the extent of rolling on the floor. For our purpose, however, attention need only be directed to face and chest. On the visceral side, capillaries and arterioles are dilated; the eyes sparkle and increased glandular secretion is observed; the skin reddens and glows.

In striking contrast, as far as the skeletal musculature is concerned, is the expression of sorrow; the facial movements of the latter are the reverse of those of laughter, while in respect of respiration there are short and interrupted inspiratory movements, succeeded by prolonged expirations—again the reverse of the other emotion. Further, there is a general inhibition, face and chest apart, of the rest of the voluntary musculature. On the visceral or vegetative side a degree of hypofunction results from a widespread vasoconstrictor effect, according to Lange, and is explanatory of the pale colour, sunken features, sensations of cold, lassitude, etc., that accompany sorrow.

Now the objective study of the facial and respiratory movements in the various types of case here dealt with is a *sine qua non* for the understanding of the difficult problems connected with the question of mimetic centres as opposed to centres for voluntary movement, and of the localization of the lesions producing the clinical phenomena under discussion. The no less important matter of the central representation of the visceral system is not at present, unfortunately, capable of the same objective examination.

A theoretical question not without practical bearing may be briefly touched on in passing. Which contributes more to the total emotional feeling in a normal or pathological case, the facial and respiratory movement, or the visceral activity ? Or are their respective quotas approximately equal ?

The evidence I wish to adduce is based on the investigation of a whole series of pathological cases in which free movement of the facial musculature is impeded by organic disease. It has been a routine matter in examination of such cases to inquire into the patient's feeling under the influence of appropriate stimuli. Among the material examined have been cases of facial diplegia, facial myopathy, myasthenia gravis, and of paralysis agitans and postencephalitic Parkinson's disease. The conclusion in each instance of bilateral facial impairment has been that the patient can readily feel and be acutely conscious of experiencing a particular emotional state such as that associated with hilarity and joy in spite of the minimal expression in the face. Moreover, the facial element may, as in the case of the ' snarling smile ' of myasthenia, be a positive distortion of the normal movement, yet the feeling is in no degree lessened or altered. A facial diplegic, as one has often seen, may preserve a mask-like countenance and yet be moved by ' inward ' laughter. Romberg,[33] for example, mentions the complete absence of expressional movement in one of his cases of facial diplegia, and says the patient " was very sensitive on this point, and termed it his greatest misfortune that he was forced to be joyful or sad without making any demonstration of his feelings to his fellow creatures." Similarly, Sir Charles Bell quotes a case from Dupuytren's clinique, that of a girl of sixteen, with facial diplegia, whose countenance bore a serious character, contrasting forcibly with her frame of mind ; " she retained her good humour and sometimes laughed heartily . . . as if behind a mask, her face being quite immoveable and grave, whilst the emotion and sound of laughter prevailed."

From cases of this kind it may legitimately be argued that the time-worn controversy as to the actor's feeling the emotional quality of his part by assuming a suitable facial expression can be dismissed in a few words. An artificial assumption of an emotional facies is practically a negligible element ; only when the psychical component is

fused with the appropriate visceral component can emotion be felt acutely, and the latter is less significant than the former; mere portrayal of an emotion may deceive the audience, but never the actor himself.* A little acquaintance with the observed facts of clinical neurology serves to emphasize the inaccuracy of the idea which Edgar Allan Poe [34] puts in the mouth of one of his characters: "When I wish to find out how wise, or how stupid, or how good, or how wicked is any one, or what are his thoughts at the moment, I fashion the expression of my face as accurately as possible in accordance with the expression of his, and then wait to see what thoughts or sentiments arise in my mind or heart, as if to match or correspond with the expression."

SOME THEORIES OF THE MOTOR PHENOMENA.

Clinical study reveals the existence of three types of interrelated motor disorder in connection with emotional facial movement.

1. In the ordinary case of organic hemiplegia, the face on one side is paresed or paralysed for voluntary movement, but not for emotional expression; in other words, there is volitional asymmetry, but involuntary symmetry. Careful examination sometimes shows that three, rather than two, stages can be distinguished; thus we may observe (*a*) volitional asymmetry; (*b*) at the commencement of emotional movement, as in smiling, there may be involuntary asymmetry, the sound side moving before the other; (*c*) when, however, laughing is well established the stage of involuntary symmetry is reached.

2. In the ordinary case of double hemiplegia, or of pseudobulbar palsy, we meet with a condition of affairs as in (1) above, except that the voluntary paresis or paralysis is bilateral.

3. The third group is constituted by the class of case referred to at the outset, where voluntary control is perfect, while emotional facial expression is unilaterally paresed or paralysed; in other words, the condition is one of volitional symmetry and involuntary asymmetry.

An explanation that is sometimes given for the phenomena as observable in cases belonging to the first group is that physiological couples are not as a rule separated by unilateral disease; that is to say, the facial musculature of the two sides acts normally, in emotional expression, as a physiological couple, and is presumably represented bilaterally in the cerebral cortex, or wherever it be; hence unilateral lesions underlying hemiplegia will not throw out one-half of the pair. This view, known as Broadbent's hypothesis, is supposed to give an apt explanation of the relative conservation of voluntary movement in the upper face, as opposed to the lower, in hemiplegia, and has been widely

* This was admirably exemplified in a charming *lever de rideau* entitled *A Touch of Truth*, which preceded the popular *Bunty Pulls the Strings* at the Haymarket Theatre during the greater part of the latter's long run in 1911.

applied in other directions, mainly, of course, in reference to *volitional* action. Its original formula [35] is as follows: "where the muscles of the corresponding parts on opposite sides of the body constantly act in concert, and act independently, either not at all, or with difficulty, the nerve-nuclei of these muscles are so connected by commissural fibres as to be *pro tanto* a single nucleus. This combined nucleus will have a set of fibres from each corpus striatum [read, cerebral cortex] and will usually be called into action by both, but it will be capable of being excited by either singly, more or less completely according as the commissural connection between the two halves is more or less perfect."

The application of this theory to the matter of conservation of mimic expression (a *non-volitional* movement) in unilateral facial hemiplegia may be criticized on the ground that it is going beyond Broadbent's original contention, but the criticism is not valid.

Consideration of the cases of the other groups, however, has naturally led to the development of the idea that there are separate and distinct paths for emotional and for volitional facial movement.

1. As long ago as 1865 it was pointed out by Saunders [36] that the facial muscles have three distinct modes of action: as respiratory muscles, reflex; as muscles of expression, emotional; and as voluntary muscles in the strict sense. He postulated in the peripheral trunk of the seventh a distinct set of fibres for each of these different kinds of action, each connected independently with different excitor centres, so that one might cease to function through disease, but not the others. Long before him, Sir Charles Bell, as already remarked, had noted the different types of activity, and had offered an explanation that is not, perhaps, very clear. Bell [37] says in one place: "We must determine whether even the portio dura of the seventh nerve may not lose one faculty and retain another. I suspect that the influence of passion, as those of smiling or laughing, is lost in consequence of affections that do not destroy the entire power of the nerve." Elsewhere he declares that: "We really have no reason to conclude that the one property of a nerve requires a finer organization than another. I should rather suppose that this power of expression is constituted with a finer relation to the condition of the mind and of the body; and, therefore, we may suppose is more easily affected by slighter derangements."

Bell's view is probably not incorrectly described as a theory ascribing differences in function to *differences in degree of affection* of the seventh, and in this respect is classifiable, with that of Saunders, as a peripheral theory.

The hypothesis is certainly untenable. If the trunk of the facial nerve is peripherally involved, it is involved for all modes of activity without any doubt. The only exception, or apparent exception, that I know of is that furnished by a case recorded by Spiller.[38] He has

observed in some instances of pressure on the facial nerve from extra-cerebellar tumours that emotional expression may be more impaired on the affected side than volitional expression. This he considers indicative of " a certain stage of peripheral facial palsy," but he admits that " the impairment of facial emotional movement may result, in part at least, from the pressure of the tumour upon the medulla oblongata and pons." A converse condition seems to have been observed by Monrad-Krohn,[39] who says that " in some few cases of peripheral facial paresis one may once in a while find a faint suggestion of this dissociation, inasmuch as the emotional innervation seems to result in a slightly *stronger* [italics mine] movement than the voluntary innervation."

Since my acquaintance with Spiller's article, frequent examination of analogous cases has failed to reveal any instance corroborating the observation. It must be remarked, moreover, that as regards both Spiller's and Monrad-Krohn's cases the difference is one of slight degree only between voluntary and involuntary innervation, not one of preservation and loss respectively, or *vice versâ*. The problem, it may therefore be taken, bears rather on the possibility of separate, central, paths for the two main varieties of facial action; in Sherrington's terminology, separate 'private paths' converging on a 'final common path.'

2. In 1879 Nothnagel,[1] influenced by the much older conceptions of Sir Charles Bell and of Romberg, assumed that the simplest way to explain the ordinary motor phenomena in single hemiplegia, where the face is unilaterally paralysed for voluntary but not for mimic movement, was to postulate the existence of a 'psychoreflex' facial path, distinct from the facial division of the pyramidal tract; he thought the optic thalamus and its connections with the cortex were situated on this 'psychoreflex' path, which in hemiplegia was unaffected. To explain the converse syndrome, viz., unilateral emotional paralysis with retention of voluntary control, he tentatively suggested that " perhaps in such cases there is a local lesion in the optic thalamus." This view, commonly referred to as Nothnagel's theory, has since been applied far and wide, but its pathological basis, by which alone it can finally be proved or disproved, has never been quite satisfactory. It must in fairness be recorded that Nothnagel himself did not go so far as to place 'mimetic centres' in the optic thalamus—the form in which the theory is usually expressed; his exact words, quoted above, indicate the uncertainty in his own mind as to the facts.

Cases of his own on which he subsequently relied [40] are anything but unequivocal. One was that of a man of twenty-four, with typical emotional palsy of the left face, voluntary movement being normal; there was also weakness of the left limbs, with considerable loss of sensibility, and with astereognosis. *Post-mortem*, the right optic thalamus was enlarged

by a tumour to twice the size of the left; in addition, however, the internal capsule was compressed, and a great part of it, posteriorly, as well as of the corona radiata leading to it, was softened. In a second case, with a similar facial syndrome, a glioma of the thalamus was discovered, but there is no report of the condition of the internal capsule.

Nothnagel's views have found support from Strümpell, Bruns, Bechterew,[41] and many more, and have been repeated in successions of text-books. Cases supposedly buttressing them have been published by Nonne,[17] Kirchhoff,[42] Raimann,[43] and others, yet not many of these will stand rigorous investigation. For example, Kirchhoff localized in the median nucleus of the thalamus the hypothetical 'mimetic centre'; his patient was a man of fifty-six, with slight left hemiplegia; there was asymmetry on smiling, but in laughter the asymmetry disappeared. In spite of this, Kirchhoff believed the case supported Nothnagel's theory, since an area of softening was found in the anterior and median part of the right optic thalamus; in addition, however, part of the genu of the capsule, the head of the caudate, and the upper third of the lenticular nucleus were softened. Nonne's case was that of a man of fifty-one, with a history of several strokes, in whom the combination of attacks of weeping and laughing with facial asymmetry on emotional movement occurred; a number of softenings were discovered at the autopsy, including one which implicated two-thirds of the right thalamus. A case of Raimann's is equally untrustworthy, because of the multiplicity of the lesions. The case published by Borst[16] is also unsatisfactory, since right-sided athetosis and ataxia were combined with right 'psycho-facial paralysis'; to account for the symptoms there was found a tumour of the third ventricle, compressing and invading the left thalamus, the left crus, and the upper half of the pons.

Hopeless as it is to disentangle specific mechanisms from widespread disorders of function entailed by equally widespread lesions, the general localization of the above-mentioned cases ought not to be ignored; a good example of 'involuntary crying' caused by a thalamic tumour has been recorded by Weisenburg and Guilfoyle.[44] On the other hand, one or two records may now be cited in which the thalamus has been (presumably) intact. In the Dupré-Devaux case of pronounced *rire et pleurer spasmodiques* a marked *état lacunaire* of each putamen was associated with softenings in the anterior limb of the left internal capsule, while the thalami were unaffected. Burzio's[9] patient, a woman of twenty-four, suffered from severe left hemiplegia and from irresistible attacks of laughing; the lesion was a vast softening of the right lenticular nucleus, with involvement of the posterior limb of the capsule, while all its anterior limb fibres were degenerated; cortical softenings were also seen in right frontal and postcentral gyri. The thalamus was

apparently normal. Similar *rire spasmodique* in a case of disseminated sclerosis was attributed by Touche [45] to the presence of plaques in the anterior and posterior segments of both internal capsules, in lenticular nuclei, etc., but no change was detected in thalamus or regio subthalamica.

Once more, a considerable number of thalamic lesions are reported in the literature which have not been associated with symptoms such as are under discussion, and which need not here be particularized.

Vague and lacking in precision, therefore, as must apparently be the conclusions to be drawn from the data thus briefly sketched, the possible *rôle* of the thalamus, i.e., of some part of it, in the genesis of involuntary laughing and crying is not to be lightly dismissed, as we shall shortly see.

3. Another hypothesis was advanced by Brissaud [2] to account for the phenomena. According to him, integrity of the thalamus is

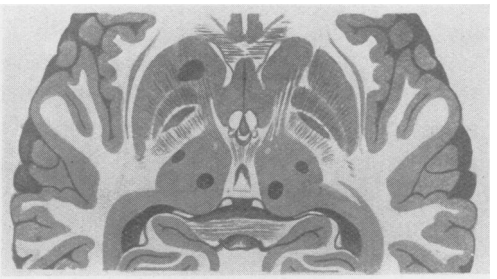

FIG. 9.—Lesions in Giannuli's case of *rire spasmodique*.

essential to the appearance of spasmodic laughter or weeping; the causative lesion is one involving the anterior limb of the internal capsule, in that part where he places his *faisceau psychique,* or frontothalamic tract of control over thalamic centres. One of his cases was that of a man of forty-eight, with right hemiplegia and complete aphasia; extremely marked uncontrollable weeping was an additional feature. *Post-mortem,* softenings were found in the left putamen, extending completely across the anterior segment of the internal capsule; symmetrical lesions were discovered in the right putamen, but these only touched the anterior limb of the capsule. Many small cortical lesions were also noted, though their exact position is not given. In the case recorded by Giannuli [20] and used by him to support Brissaud's hypothesis, there was a considerable softening in one anterior capsular limb, yet both thalami also were patently the seat of degenerative disease (*Fig.* 9) —in contradiction of Brissaud's contention, just mentioned, for integrity of the thalamus in spasmodic laughing and crying.

In his lectures on pseudobulbar paralysis Brissaud [46] appears to

place centres for involuntary expression of emotion in the basal ganglia (' noyaux opto-striés '), and to argue that the syndrome we are concerned with is a product of ' irritation ' : " il signifie toujours une irritation capsulaire " (anterior segment). He regards the phenomena as on a par with the tendency to spasm shown in any case of hemiplegia : " si les centres en question ne sont pas détruits mais simplement excités par une lésion de voisinage, ils traduisent le spasme hémiplégique en déchaînant le rire et le pleurer."

Notwithstanding its plausibility the hypothesis will not bear serious investigation. Apart from the primary objection to the view that the symptom is ' irritative ' and not of the nature of a ' release-phenomenon,' Brissaud depends for support of his thesis on anatomical connections of the cortex with the corpus striatum and on anatomical views of the ansa lenticularis which have not been confirmed by subsequent investigation. The diagrams illustrating his article assume links in neuronic chains that remain to-day quite speculative from an anatomical standpoint.

Oppenheim,[47] who with Siemerling described exaggerated laughing and crying in pseudobulbar palsy in 1886, supposes it due to " lesions of the centres or interruption of the tracts which have an inhibitory effect upon the bulbar centres," without specifying further the exact position of one or the other. He states, moreover, that these facial movements may be affected in every possible way " according as the morbid foci have an irritating or a paralysing effect." It will be seen, we think, that here again the student is offered a theory which is more than a little nebulous.

4. Reference has been made above to the work of Hartmann [4] on pseudobulbar paralysis. Arguing from the experiments of Bickel, who observed the ' explosive ' character of voluntary movements in animals after the production of sensory ataxia by appropriate cortical lesions, Hartmann is inclined to regard the absence of centripetal impulses as a factor in the pathogenesis of the phenomena. He believes that a defect on the *afferent* side through the optic thalamus is responsible for the release of involuntary emotional activity. In this respect he is in agreement with von Monakow,[48] and, in a way, with Lewandowsky [49] ; the latter argues that peripheral sensibility has a much greater influence on mimic expression than on voluntary innervation of the facial musculature, and is led to suppose that since the optic thalamus is only a ' Schaltstation ' of sensibility on the way to the cortex, and not an autonomous organ, lesions productive of amimia or of expressional overaction may be situated there, though they may not ; they may also occur in the cortex or in subcortical fibre-systems. He is doubtful whether mimic reactions may be obtained at still lower levels.

The opinion expressed by Bechterew [50] is that the syndrome is dependent on two factors : (1) removal of voluntary control, and (2) the

influence of exaggerated involuntary stimuli. Unfortunately he has not vouchsafed the reader precise information as to how these come into action; quoting a personal case in which other symptoms than the involuntary *rire* and *pleurer* pointed to a pontine localization (no pathological confirmation), he expresses the view that the involvement of fibres passing from the optic thalamus to ' deeper lying centres ' (not specified) allows the exaggeration of emotional display. In another communication he states, negatively, that *rire spasmodique* is certainly not due to implication of the voluntary paths to the facial muscles.

From these, and from other writings that might be cited but that do not call, perhaps, for any detailed reference, the student of the subject will appreciate the truth of Lewandowsky's final comment, that more exact observations are required, and that the hypotheses usually advanced are characterized by indefiniteness and absence of precision.

OUTLINE OF A SUGGESTED THEORY.

In order to aid understanding of a possible theory explaining both amimia and mimic overaction, it is desirable to indicate the way in which, as I conceive it, the subject should be approached.

1. Omitting in this place consideration of the visceral components of laughing and weeping, I think it imperative to note the participation of both facial and respiratory mechanisms in the act of laughter (or weeping), both physiological and pathological.

The physiological association of facial and respiratory musculatures in the expression of emotion scarcely calls for any comment, so obvious is it. Bell called the seventh the " facial nerve of respiration "; when the lower face (mouth and nose) is paralysed it was described by him as " paralysis of the respiratory functions of the facial." The implication of the face in sneezing, the facial spasms occurring with respiratory gasps *in extremis*, the collaboration of the facial apparatus with the other in ordinary breathing and speaking, are simple instances of the action of this important synkinesis. The seventh nerve is united functionally with the tenth, and also on occasion with the eleventh and certain upper cervical spinal groups. For simplicity's sake, we may allude to it as the faciorespiratory mechanism. We note that its normal activities are involuntary, i.e., it is under voluntary control only to a limited extent. Laughter may be ' stifled,' tears may be ' restrained,' no doubt; at any rate, practice may enable the individual to inhibit its function to a varying degree; ordinarily speaking, however, the faciorespiratory mechanism goes off ' on its own,' whether the circumstances be physiological or pathological.

The localization of the ' nœud ' of this mechanism is still uncertain; we have to postulate a centre linking the seventh nucleus in the pons with the motor nucleus of the tenth (nucleus ambiguus) in the medulla

and the phrenic nuclei in the upper cervical cord, etc. By all analogies this ' centre ' must be supranuclear ; for the sake of argument we may suppose it has an upper pontine site.

2. Our second preliminary consideration is to bear in mind the existence and function of the respiratory centres proper, for ordinary automatic breathing, situated in the medulla. With their normal action must also be associated co-operation on the part of the larynx and the face, otherwise normal breathing might partake of the noisy character observed in various diseased conditions.

The most recent work on the localization of the respiratory centres is that of Lumsden,[51] who has shown, by numerous experiments on cats, rabbits, dogs, and monkeys, the somewhat elaborate nature of the arrangements. Thus, he has demonstrated that ordinary rhythmical respiration—quiet, unconscious breathing—depends on several factors. There is (*a*) an inspiratory mechanism at the level of the striæ acousticæ ; this he calls the ' apneustic centre,' because when this group of nerve cells is cut off from above, prolonged tonic contraction of the inspiratory muscles ensues (' apneusis '). The level of the striæ acousticæ is upper medullary. (*b*) Just below this there is a separate expiratory centre (medullary), the existence of which has long been suspected and is now apparently established. (*c*) Both (*a*) and (*b*) are controlled by a higher centre in the upper half of the pons, styled by Lumsden the ' pneumotaxic ' centre, because it regulates normal quiet breathing. When it is cut off from (*a*) by appropriate section, respiration takes the form of a series of prolonged inspirations, each followed by two or three relatively quick respirations of abnormal type. Lumsden has shown that this cycle repeats itself with great regularity. Evidently, then, the pneumotaxic centre produces normal respiration by inhibiting the activity of the apneustic centre below (behind) it. (*d*) A fourth, ' gasping,' centre, situated below (*b*) at the level of the apex of the calamus scriptorius, is regarded by Lumsden as a ' relic,' and need not further concern us.

No mention is made by this writer of the position on transverse section of the various groups and tracts the functions of which he has so excellently demonstrated, nor is there any allusion to concomitant implication of the face in respect of the activity of the pneumotaxic centre—not that this, perhaps, was to be expected. It is therefore impossible, without further investigation, to say what relation, if any, there may be between the pneumotaxic centre of Lumsden and the postulated co-ordinating centre for the faciorespiratory mechanism referred to above.

3. Our next consideration bears on the influence of voluntary action on the respiratory centre in the pontomedullary apparatus. Its automatic activity is set aside *voluntarily* when we deliberately hold our breath, or when we voluntarily pant, cough, yawn, sigh, take deep

breaths, etc. Further, its activity is set aside *involuntarily* when we are convulsed with laughter, or when we give way to crying, sobbing, howling. Both in the former and the latter case facial movement is involved; we innervate the facial musculature *voluntarily* for the purposes specified, and the face takes its share in the *involuntary* expression of joy or sorrow.

Thus we get the idea of a *double control* over the faciorespiratory synkinesis: (*a*) a voluntary control when we choose to inhibit automatic movement, and (*b*) an involuntary control when that automatic movement is forced to give way to the expression of emotion.

(1) *Voluntary Control.*—The path followed by volitional impulses to facial and respiratory muscles is undoubtedly the familiar corticopontine, corticobulbar, and corticospinal tract. In particular, the geniculate bundle of the pyramidal tract, from the operculum and lower end of the precentral gyrus, *viâ* the genu of the internal capsule, conveys these impulses to the appropriate nuclei. As we have seen, voluntary breathing sets aside ordinary breathing, hence we must postulate, on the principle of reciprocal innervation, a synchronous inhibition of the automatic pontobulbar centre. The anatomical course taken by the latter, inhibitory, impulses is less certain, but of their reality there can be no question. It will be remembered that Hughlings Jackson [52] explained the interesting observation he made on respiratory movement in hemiplegia by the existence of double sets of respiratory fibres passing from the brain in this way.

Lesions, therefore, of the geniculate bundle anywhere in its course—especially if they are bilateral—will impair volitional control over the musculatures concerned in the expression of emotion, with the result that the involuntary action of the same mechanisms will tend to become abnormal. Pseudobulbar paralysis is the disease of the geniculate bundles which, we have already seen, is particularly prone to be accompanied by the phenomena of *rire et pleurer spasmodiques.* If the reader will refer again to case 3 above he will note there was *absolute* voluntary paralysis of face and of respiratory apparatus—hence emotional seizure of the same parts was entirely unchecked, and the patient's existence was one long roar of laughter. An old observation recorded by Magnus,[53] in 1837, presents certain analogies to my case 3, and may be briefly outlined. The patient was a widow of fifty who had had two 'strokes,' with the result that there was *complete* bilateral paralysis of the face and tongue; yet she smiled and laughed, often violently, the paroxysmal laughter ending in a peculiar, grunting sound of which she was ashamed, and which she would willingly have suppressed; it continued, however, even after the movements of laughter had ceased.

It is clear, then, that the more absolute the faciorespiratory paralysis, the more exaggerated is the involuntary innervation of the same

mechanism. In this connection Monrad-Krohn has shown that the emotional innervation is often distinctly exaggerated on the paretic side in hemiplegia, and has proved (by the ' slow-motion ' cinematographic camera) that emotional movement is actually quicker on the side showing voluntary paresis. On the other hand, for the exhibition of ' uncontrollable ' laughter or tears a degree of volitional paresis or paralysis is not quite essential, though it is certainly usual ; the involuntary action of a normal laugh may break down normal control ; the quivering lip of the child is indicative of a balance between the action of the voluntary and the involuntary processes which may be tipped over in either direction by a trifle.

(2) *Involuntary Control.*—The careful experiments of W. G.

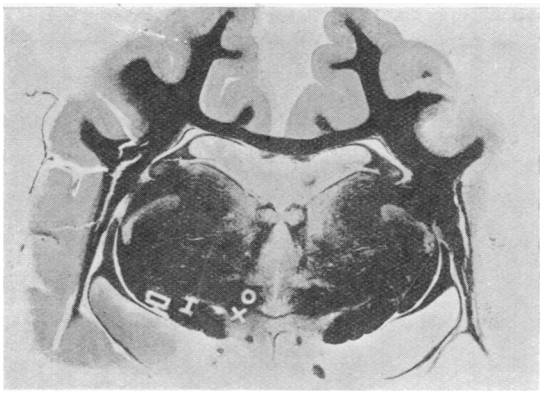

FIG. 10.—(Spencer.)
O = position of respiratory path, stimulation of which produces slowing and arrest.
X = ditto, stimulation causing acceleration. These are extrapyramidal tracts.

Spencer,[54] in 1894, determined the existence of four paths from the cerebral cortex to the respiratory mechanism. Of these, one is undoubtedly the voluntary path just mentioned, from the motor cortex *viâ* the genu of the capsule ; its stimulation produces, in the ape, a sort of ' holding the breath,' or, as Spencer calls it, " overinspiratory tonus." Two of the other tracts follow an entirely different course ; one is an ' arresting ' and the other an ' accelerating ' path. The former arises from the under surface of the frontal lobe, the latter from the sensory cortex. Spencer has traced the two throughout their course ; they come together towards the middle line at the mesial aspect of the lower optic thalamus, bordering on the third ventricle, and run down, near the midline of the tegmentum, to the medulla. Both are far removed from the voluntary tract for respiratory innervation in the capsule and crus. More exactly, the route followed by the arresting path is from a spot on the under surface of the frontal lobe where the olfactory tract runs into

the temporosphenoidal lobe, along the ' olfactory limb ' of the anterior commissure (where it decussates), by the side of the infundibulum, past the nucleus ruber below and external to the aqueduct in the plane of exit of the third nerve, and so to the medulla. As for acceleration, " commencing especially from a point on the convex surface of the cortex within the sensorimotor area, the effect may be followed back through the lenticular nucleus where it borders on the outer and ventral portion of the internal capsule ; the strand runs at first externally and then ventrally to the motor portion of the internal capsule, and so reaches the tegmentum. The lines from the two sides meet in the interpeduncular grey matter at the level of and just behind the plane of the third nerves."

The figures reproducing Spencer's photographs (*Figs.* 10 and 11)

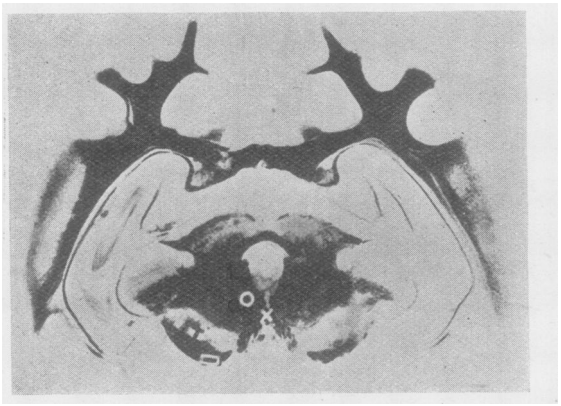

FIG. 11.—(Spencer.)
O and X as in Fig. 10. Note the distinction of these paths from the respiratory tracts in the crus (indicated by ⊏ and ⊥).

indicate the position of the arresting and accelerating respiratory paths and show their distinction from the voluntary tract for respiratory innervation in the capsule and crus.

I believe it is a feasible speculation that these are the paths for emotional activation of the faciorespiratory mechanism. They are separate from the paths for voluntary control ; they come towards the midline in the regio subthalamica and tegmentum ; stimulation of them produces unvaryingly the phenomena of arrest and acceleration noted above. As far as the respiratory element in involuntary laughing and crying is concerned their appropriate excitation and inhibition will explain the mainly expiratory character of the former and the mainly inspiratory character of the latter.

Clinical proof of the reality of the faciorespiratory involuntary synkinesis, and of the possibility of its unilateral paralysis, is furnished

by Stromeyer's remarkable case, already mentioned. It remains to ascertain if we have any experimental evidence bearing on the association of the face with involuntary respiratory tracts. In a valuable paper [55] entitled 'Note on the Physiology of the Basal Ganglia and Midbrain of the Anthropoid Ape, especially in reference to the act of Laughter,' Graham Brown has given us certain data that bear on our subject. He has demonstrated in the normal animal that tickling in the hollow of the shoulders, armpit, etc., causes the chimpanzee to respond by retraction of the lips, as in smiling, while, at the same time, the respiration becomes more rapid and slightly vocal. "The sound given is that of 'Ha, Ha, Ha,' but not said as we say it—rather whispered. There can be little doubt that this reaction to tickling is equivalent to the act of laughter." *

The same investigator, working on the exposed surface of the mesencephalon after transection, has found that between the internal boundary of the red nucleus and the mid-longitudinal dorsiventral plane of the neuraxis there is a small and very strictly circumscribed area, not much more than 1 mm. across, unipolar stimulation of which suddenly changes the normal slow, deep, and steady respiration to fast and shallow breathing (Fig. 12). The abdominal muscles of both sides of the body appear actively to contract and relax during the reaction, which stops always with cessation of stimulation. Graham Brown says specifically that "the sound of this breathing was very similar to the 'Ha, Ha, Ha' of the laughing chimpanzee." In a personal communication he states that to the best of his recollection the facial muscles were retracted at the mouth at the same time.

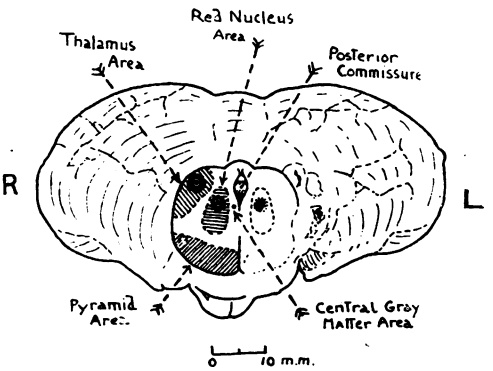

FIG. 12.—(Graham Brown.) Stimulation of the spot indicated as 'central grey matter area' produces a modification of the chimpanzee's breathing closely akin to 'laughing.'

Here, then, though there is some slight uncertainty, it would appear that stimulation of a specific descending tract in the mesencephalon causes the animal to make both the facial and the respiratory movements of laughing, apart altogether from the corticobulbar pathways. Graham Brown has also found within the extreme caudal end of the optic thalamus two spots, of which stimulation applied to the dorsal one causes

* There is a fine photograph of a 'laughing' chimpanzee on page 92 in Crile's book on the Emotions (see reference 23).

very vigorous and ' hollow ' breathing, while excitation of the ventral spot gives a slowing of respiration.

A comparison of the results obtained by Spencer and by Graham Brown shows, as far as the mesencephalon and tegmentum are concerned, the existence of resemblances in anatomical position and in objective phenomena sufficiently impressive to outweigh such discrepancies as still remain. Their correlation with the work of Lumsden is difficult, as already hinted, because of the absence in the latter's experiments of evidence pointing to a particular localization on transverse section at the level of the upper pons.

Our general conclusion may be couched in the following terms: there are corticifugal paths to the faciorespiratory centres in the pons and medulla that are independent of the voluntary cortico-ponto-bulbar tracts to the same nuclei; on excitation they will either arrest or accelerate, i.e., interfere with, the normal rhythmic activity of the respiratory centre; the available evidence warrants the speculation that they are the routes taken by emotional impulses to modify the faciorespiratory synkinesis in the direction either of laughter or the reverse. Their exact course remains for further substantiation; it is perhaps noteworthy that they make their way separately towards the midline skirting the lower optic thalamus (in the case of one) and passing by the lower regio subthalamica to the tegmentum, and so to more caudal levels of the neuraxis.

APPLICATION TO DISEASED CONDITIONS.

1. Pathological laughing and crying are allowed by lesions of the voluntary paths from the motor areas of the cortex or by any state in which these exercise imperfect control. Laughing and crying then become ' uncontrollable.' Their common appearance in pseudobulbar paralysis is readily understood, because of the usual volitional facial, etc., weakness.

It must, however, be pointed out at once that this is not a complete explanation of the facts. Some cases of bilateral facial weakness of central origin are not particularly prone to develop the exaggerated emotional display of which we are speaking, while it may appear in other diseases in which bilateral voluntary control is not in any way impaired, or not obviously impaired, by the morbid state. In the case of the former it is likely either that the individual is not by constitution particularly ' emotional,' or that voluntary control, though impaired, is still adequate, or that, possibly, disease is affecting the activity of the non-voluntary paths as well as of the voluntary. In this connection Féré's case of ' fou rire prodromique,' referred to above, is of interest in view of the cessation of the uncontrollable laughter subsequent to the development of a severe left hemiplegia. It would have been of value to ascertain

the position of the lesion or lesions which thus caused the emotional exhibition to stop.

In the case of the latter—as, indeed, in normal persons—the emotional stimuli evidently overwhelm the control mechanism, and we must suppose either an irresistible quality in them, heightened by disease, or a constitutional peculiarity on the part of the individual—disease apart, or defect of cortical control not discoverable in tests for volitional facial innervation, or, perhaps, defect on the afferent side to the cortical emotional 'centres' from which the faciorespiratory paths arise.

2. The reverse condition, unilateral (or bilateral) emotional palsy, is brought about by a lesion of the appropriate involuntary system whose possible course has already been indicated, the voluntary cortico-ponto-bulbar tract being normal. *Ex hypothesi*, this may occur anywhere from the cortex to at least as low as the pons.

For example, the clinical evidence in my case 8 is strongly suggestive of a lesion in the mesencephalon (paralysis of upward and downward conjugate movement of the eyes, unilateral Argyll Robertson pupil, hemitremor) under the anterior corpora quadrigemina. The patient showed unilateral, right, emotional facial palsy. Similarly, in case 9, a tumour of the mesencephalon was in a position to effect unilateral 'mimic' paralysis (see *Figs.* 5 and 6). The remarkable instance of the same condition recorded by Mills [15] was associated with various other symptoms and signs, and was due to a destructive lesion involving, *inter alia*, the mesencephalon. Allusion has already been made to the occurrence of the paralysis in cases in which the thalamus, speaking loosely, has been implicated. If the hypothesis advanced in this communication is trustworthy, the condition should not occur in all thalamic cases by any means, but only in those where the lesion is so placed as to engage the tracts specified.

We have seen they do not pass right through the thalamus—at least in the case of the arresting path—but rather skirt it mesially and ventrally. The argument, therefore, opposing Nothnagel's original speculation on the ground of the occurrence of negative thalamic cases loses much of its value. The lesion causing mimic palsy does not occupy the same site as that causing the thalamic syndrome—an explanation which has suggested itself to Gordon Holmes [56] and others, and which is borne out by the considerations here advanced. The two, however, may doubtless be combined, though no definite instance has as yet come under my notice.

Ex hypothesi, further, mimic paralysis may originate in a cortical lesion, but, so far as I have seen, no such case has yet been recorded. Pathological investigation of all cases exhibiting mimic palsy is a *desideratum*.

The possibility of a combination of (1) and (2), viz., the occurrence

of facial asymmetry in uncontrollable laughter, is realized in case 3 above, in which, with highly characteristic *rire spasmodique*, the angle of the mouth was not retracted so well on the left side as on the right; a similar combination was observed in the case reported by Nonne.[17]

It is of particular interest to note the general grouping of the cases of involuntary, mimic, paralysis discussed above (cases 3, 7, 8, 9, Mills' case) in relation to the distribution of the posterior communicating artery. Beevor has shown that this vessel supplies the anterior third of the crusta, and all the part between it and the third ventricle at the midline; frequently, too, it irrigates the anterior half of the internal nucleus of the optic thalamus. Now we have seen that the respiratory tracts discovered by Spencer come together within this area; hence a lesion in the distribution of the posterior communicating artery may be regarded as likely to exteriorize itself, *inter alia*, by unilateral mimic paralysis. The explanation given by Beevor for the phenomena of his striking case rests on a different interpretation from my own; he thinks Spencer's respiratory arresting and accelerating paths are those of *volitional* control, and that their involvement caused the paralysis of voluntary respiration in his patient. With this view I am not in accord, as will have been seen; I believe, however, their partial implication explains the incomplete mimic paralysis which his case certainly exhibited.

From the argument here advanced it will be gathered that any hypothesis for the placing of actual 'mimetic centres' in the thalamus is unnecessary. The thalamus cannot be more than a link in the chain—if, indeed, it is actually as much. The afferent paths for appropriate impulses from eye, ear, skin (tickling), etc., lead through the thalamus to the cortex. The laughter-producing stimulus is cortically appreciated, and its expression through the involuntary faciorespiratory mechanism is mediated, *ex hypothesi*, by the efferent arresting or acclerating tracts already described. The arresting tract in its descent skirts, or perhaps runs through, the lower mesial margin of the thalamus—the palæo-thalamus, be it noted, which borders on the third ventricle, and which, according to Tilney and Riley,[57] "seems to be invested with a functional responsibility related to the development of the emotions and the emotive expressions." On the afferent side neuronal systems are relayed in the thalamus, but it is not certain if the same obtains on the efferent side, hence the possibility of a 'short-circuit' from sensory to motor path in that ganglion itself remains undecided. Whether such a short-circuit would explain the 'explosive' character of spasmodic laughing and crying, the exaggerated response to trifling emotional stimuli, is equally uncertain. The argument from the pseudobulbar cases is that some defect of volitional faciorespiratory control heightens the facility of the explosive phenomena, yet, as we have seen, it is not always *per se* sufficient. It is conceivable, therefore, as some have maintained, that

failure of corticothalamic inhibition is responsible for undue ' liveliness ' of the thalamus, and for the exhibition of involuntary emotional exaggeration.

For myself, however, I am of the opinion there is more to be said for the participation of the cortex in the production of abnormal emotional activity. We cannot take it that the cortical origins of the arresting and accelerating respiratory tracts of Spencer are physiologically, though anatomically, separate, and we may ask—using Mills' expression—where is the rendezvous ? In an ingeniously developed argument, that veteran neurologist [58,59] contends that in the right hemisphere mainly, in the midfrontal region, are centres for the representation of movements especially concerned with the expression of emotion. He gives the term ' movement ' a broad significance, as applying both to skeletal and to visceral, vascular, and secretory activity. On the other hand, Bianchi,[60] whose claim to speak with authority also is acknowledged, declares that " to maintain that the frontal lobe plays a part in the essence and mechanism of the emotions . . . is a bold hypothesis in which there is a good deal of mere conjecture and certainly no basis of proof."

Be all this as it may, and however much in the matter is still obscure, our facts have led us to suggest that there are corticifugal paths for the expression of the emotions *viâ* the faciorespiratory apparatus, distinct from those for voluntary innervation of the same nuclei, and as a necessary corollary we presume the existence of a cortical nodal point co-ordinating them. Its situation is at present indeterminate, yet it is likely to have some definite position. In this connection I echo with approval the words of Mills, who declares he is not one of those who believe that the problem of emotion, or of any other great mental process, is to be explained by regarding it in some vague way as a complex expression of the action of the cerebral cortex as a whole.

There is clinico-pathological, and experimental, evidence suggesting that non-volitional control over the normal activity of the faciorespiratory mechanism is exercised from the cortex by routes that pass separately downwards to come together towards the midline in the regio subthalamica and tegmentum.

It is not certain that these actually pass through the thalamus in man, though it is understandable that some thalamic lesions may be so placed in that ganglion as to interfere with them as a vicinity effect.

We have no information as yet to show these paths are interrupted by a thalamic relay, nor is it known that emotional impulses can pass from sensory to motor side at this level ; it is possible, perhaps, but not probable.

It is outside my immediate purpose to deal at any length with certain modifications of the act of laughter or of crying due to sensory,

ataxic, and spastic conditions as affecting the musculatures involved in the emotional performance. Neurologists have occasion, every now and then, to observe cases in which laughter is mainly inspiratory, or 'hollow,' or 'noiseless,' others in which it is prolonged by rigidity of the facial muscles (as in some cases of progressive lenticular degeneration, paralysis agitans, etc.)—the so-called 'spastic smile'—and others in which an obvious element of ataxia or dysmetria is introduced. The laughter of cerebellar patients, of those with Friedreich's disease, etc., not infrequently assumes a peculiar character. As any motor mechanism may be impaired by defect on the sensory or the cerebellar side, so may that concerned in involuntary emotional expression. This part of the subject, however, is quite subsidiary to the main problem here discussed, and must for the present be left aside.

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