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THE APPROACH TO THE STUDY OF HYSTERIA.*

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THE vicissitudes undergone by hysteria in the succession of the centuries have varied prodigiously, and he would be a bold clinician who to-day would announce their definitive end. Phases of medical doctrine run their course and have their day, but hysteria goes on. Doubtless in the present era of psychological grace we who have been conscious of the immense strides taken by psychopathological research are prone to believe, if not, perhaps, to feel entirely sure, that hysteria has at length yielded its secret—a discovery often heralded before, but never, we fain would pride ourselves, with so valid a pathogenesis on which to base the claim. If, for the nonce, however, we approach the question from the standpoint of pure empiricism, confidence may conceivably be somewhat shaken.

To this empirical approach objection cannot be raised. No reason exists why study of the objective clinical phenomena of the condition should not be prosecuted, deductions therefrom as to their nature drawn, and generalizations of a nosological character formulated, or, at least, attempted. Nothing, however, has been more typical of this period of psychological study, so far as hysteria is concerned, than the comparative absence of research of an objective and empirical kind. Can it be that acceptance of the former method dispenses with all necessity to conduct the latter? If this be the case, then psychological theory stands to lose in impressiveness. It is not so very long ago since a distinguished protagonist of new conceptions of hysteria, in a psychiatry club of which I was a member, listened with mild impatience to my exposition of certain neurophysiological peculiarities in hysterical symptoms, and closed discussion by declaring that since the neurosis had its demonstrable origin in a *vita sexualis* which did not run smooth its semeiology was of merely secondary interest and minor importance.

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Possibly the outstanding feature of hysteria as revealed to us by the records of former generations and the knowledge of our own is the changes which its clinical syndromes have suffered. This simple fact of observation must appear highly significant, as fruitful for theory as it is true in substance. The mediæval ecstatic, simulating in hands and feet the nail-prints of her Redeemer, is long since *démodée*, as is her eighteenth-century sister, melodramatically counterfeiting the emotional transports of a popular actress. I quote from Kay's *Portraits*, published in 1837 :—

At the first visit of the great Mrs. Siddons to Edinburgh, many were the fainting and hysterical fits among the fairer portion of the audience. Indeed, they were so common that to be supposed to have escaped might almost have argued a want of proper feeling. One night when the house had been thrown into confusion by repeated scenes of this kind, and when Mr. Wood (a popular surgeon) was most reluctantly getting from the pit (the favourite resort of all the theatrical critics of that day) to attend some fashionable female, a friend said to him in passing, "This is glorious acting, Sandy," alluding to Mrs. Siddons; to which Mr. Wood answered, "Yes, and a damned deal o't, too," looking round at the fainting and screaming ladies in the boxes.

What impresses in this echo from an almost unimaginable past is the insight displayed by the common-sense surgeon of that day. But let us come a little nearer our own time. The quotation is from a lecture on hysteria delivered at St. Bartholomew's Hospital in 1866, by Mr. F. C. Skey, F.R.S., a then celebrated member of its consulting staff :—

Thirty or forty years since . . . and for how many years anterior I know not, all the seaside towns were crowded with young ladies between 17 and 25 years of age and beyond it, who were confined to the horizontal posture, and wheeled about on the shore in bath chairs, on the supposition that they were the subjects of spinal disease. They were placed under much medical and dietetic discipline . . . and the large majority carried a pair of handsome issues in the back! Brighton, Worthing, Hastings, and other places on the South Coast were largely tenanted by these unfortunate females, to which a modest sprinkling of young gentlemen was attached. What has become of all these cases? They appear to have vanished just in proportion as the eyes of the surgeon have been opened to the absurdity of inferring that pain alone, which locates itself with remarkable precision in hysteria on a given vertebra, can indicate the presence of organic disease of the body of the bone without collateral evidence in its favour.

No longer do the 'circus horses' of the Salpêtrière perform before visitors as in the palmy days of Charcot; no more does their contorted musculature respond to the application of divers metallic rods, as Gilles de la Tourette was wont to demonstrate; seldom indeed is the clinician witness to the elaborate and protracted hysterical fits whose theatrical features were drawn with artistic skill by Paul Richer. The intimate correspondence of the historical phases of hysteria with contemporary medical predilections is responsible for the alternate tragedies and comedies of its treatment, and Morestin's remark embodies a shrewd estimate of the situation: "Les hystériques suivent à leur manière le mouvement scientifique, et s'adaptent au progrès de la médecine." Since the day (and long before it) of the insensitive and bloodless marks that stamped the witch as Satan's own the poor hysteric has done her best, has

never failed to respond to the calls made on her. But to-day we seek the clue to the ailment in the unseen psyche, and she is somewhat at a loss accordingly ; her elaborated somatic manifestations are rather at a discount. A cold scientific environment besets her, instead of a world of emotional extravagance and limitless credulity. So a defence-hysteria is now à la mode, no less ego-centric if more utilitarian ; with an eye to the main chance, the hysteric turns to the possibility of compensation for trauma, or seeks to escape from unpleasant reality by a flight into neurosis—at least, so we are assured. The times have changed, and we, both physicians and hysterics, have changed with them.

Yet, when all this is said, it remains true in a deeper sense that if hysterical fashions vary, their kaleidoscopic patterns are composed of unaltering, instinctive and impulsive reactions ; thus can its clinical characters be recognized on the Greek vase depicting the ecstasies of the frenzied mænad, in the records of some New Testament miracles, in mediæval paintings of demoniac exorcism, in modern accounts of camp-meetings, spiritualistic séances and religious revivals, of disease-mimicry in school-girls and shell-shock amid twentieth-century warfare. Beneath surface diversities the essence of the phenomena involved has not been modified. And further, where old beliefs linger in the public mind, where superstition is rife and mysticism rampant, there still is to be found the favourable and appropriate milieu for hysterical exhibitions of antiquated form and guise even in this epoch of scientific enlightenment. The literature of demoniacal possession, recently collated and analysed by Oesterreich, contains not a few references of an authentic kind belonging to the last two decades only. Nor, in the nature of things, is it likely that such hysterical traits and expressions will really disappear ; human culture may progress, but a Messina earthquake or a world war will lay bare hidden reactions among some of the species, at all events. Leprosy, rabies and malaria are ancient diseases, but epilepsy and hysteria are older still ; are they as old as human nature itself ? Their antiquity and ubiquity might almost suggest this ; and since epilepsy is scarcely anything else than a physiological reaction, which anyone soever will exhibit provided he is injected with a convulsant, it becomes curiously interesting to discuss whether hysteria, too, is but a name for a reaction, capable of being expressed in dynamic, neurophysiological terms.

It is nearly twenty years since I wrote a paper in *Brain* on this subject, in which certain ideas respecting the objective study of hysterical phenomena were outlined.

A brief quotation will indicate what is meant :—

I believe we must depend on the objective signs of hysterical disease if we are to progress towards its unification. Janet himself holds that the intrinsic examination of cases of hysteria is the most accurate and scientific method. Now the chief point, as it seems to me, on which more light is wanted, is the mechanism of production of the symptoms. Granted that a particular mental state is accountable for the disturbances of function, how are these actually brought into being ? Granted

that amnesia or absentmindedness [in accordance with Janet's views then being examined] causes hysterical motor defects, why have we tremor in one instance and paralysis in another? And what are the objective features of these? Can we thereby learn anything as to the seat of the functional defect? I am convinced from the study of a number of cases that organic disease will teach us far more about hysteria than vice versa.

Of course, the physiological approach is only one of several. And in directing attention thereto for the moment I do not ignore that which seeks to utilize as much—as exclusively—as possible the assumption of 'unconscious mental processes' and of their influence on somatic pathways; nor that other which surveys the hysterical field from the standpoint of causation by more or less conscious processes of simulation. But it is permissible to examine the data without subscribing to any particular theory, and to ascertain whither such study leads. That physiological investigation is legitimate needs no apology whatever; both in former and latter times clinicians have busied themselves therewith, but few if any psychologists—with one noteworthy exception to be referred to later. Kretschmer's recent analysis of hysteria is concerned to a large extent with physiological considerations, though not at all on the lines now to be sketched.

The method chosen no doubt depends on the scientific taste and orientation of the individual physician, and I confess to having been influenced by a teacher who besought me in neurological work never to 'invert the pyramid.' For a number of years clinico-physiological observation of hysterical phenomena has occupied my time at intervals, and a few of the collected data may now be passed in brief review and their bearing on major hysterical problems examined.

MOTOR SYSTEM.

Hysterical paralysis is a clinical banality, but study of its intrinsic characters will be found illuminating. It assumes the form of paralysis in rigidity, or of paralysis in flaccidity. Otherwise expressed, the muscular weakness may or may not be accompanied by plain alterations in muscle tonus. When weakness or paralysis is associated with increased tone the condition clinically is usually, perhaps invariably, one of contracture. In flaccid cases the part affected seems emptied of tone, though not always perhaps to the degree met with in atrophic lower motor neurone affections, such as poliomyelitis. All intermediate stages between the 'toneless-flaccidity' and 'spasmodic-rigidity' extremes may be encountered.

So far as I can discover, my former chief, the late Dr. C. E. Beevor, was one of the first to direct attention to the occurrence in hysterical palsies of the phenomenon known as 'defective inhibition of the antagonists.' He had under observation the case of a girl aged 18 with incomplete right hysterical hemiplegia, and noted that whenever she was asked to perform a certain movement the first action detected was that of the antagonist muscles. For instance, on being told to extend the elbow, the first muscle observed to contract was the supinator longus, one of the antagonists to the movement of extension. This

was followed immediately by that of the triceps, and there succeeded a confusion of to-and-fro movements due to alternate or simultaneous contraction of extensors and flexors of the elbow. When she was requested to flex that joint the triceps sprang into contraction first. I was Dr. Beevor's house physician at the time, was duly impressed with the basic significance of this phenomenon for hysteria, and have made it a matter of routine investigation ever since. It is unquestionably the most common motor sign of the affection and can usually be demonstrated with ease. It underlies the vast majority of hysterical disturbances of active motility, being equally remarked in contractures; when the patient seeks to undo a contracted attitude the antagonists will be found to contract still further.

Beevor declared in his Croonian Lectures (1904) that he had never met with this condition of the antagonists acting before the prime movers except in hysterical paralysis, and thought it a diagnostic feature of that state. Oppenheim also admits its frequency under such circumstances.

The import of this physiological disorder resides in the fact of its obviously constituting a disturbance of the Sherringtonian law of reciprocal innervation, according to which as a given muscle contracts in a normal movement its antagonist is simultaneously de-innervated. In the experimental animal reciprocal innervation holds for all spinal and, more generally, decerebrate reflexes. But Beevor's view of its pathognomonic value for hysteria, though not discounted, must be considered in the light of another fact, viz., that, as was shown in my Croonian Lectures (1925), the phenomenon occurs also in athetosis. In that 'organic' affection the orderly innervation and de-innervation of prime movers and antagonists are interfered with. Further, and still more significantly, an endeavour was made in these lectures to show that athetosis results from disordered function of certain *cortical* reflex arcs, and that the underlying lesion *may* be of cortical site. Another condition attended by precisely the same physiological defect, as I have observed, depends on lesions of the superior frontal cortex sometimes associated with the symptom known as 'tonic innervation' and with apraxia. Thus it appears to be of deep interest for the study of hysterical physiology that a special symptom distinguishing it with such constancy is also met with in one or two 'organic' states whose site is cortical; and the inference seems fully warranted that hysterical palsies bear the physiological imprint of cortical defect of function.

A conclusion of this kind may appear *à priori* obvious from the standpoint of theory, but to have reached it on objective considerations alone is surely satisfactory, the more so as it furnishes an instance of explanation of hysterical physiology from the 'organic' side.

Another no less interesting motor sign consists in the shunting of the desired innervation to other groups of muscles, not confined to the actual antagonists. This is well seen, for example, in hysterical paresis of the arm; on the request to grasp the examiner's fingers tightly the patient gives the

impression of putting forth a great effort, but the grasp remains feeble, while it is easy to be assured of the fact that shoulder muscles and others equally remote from the long flexors are in strong contraction. This 'shunting' sign, as it may be termed, is highly characteristic of hysteria, but has its analogies in certain states of muscular weakness following structural lesions—yet under these latter circumstances it is seldom or never so pronounced as in the other.

The fact that both of these physiological traits are such as can be imitated by the normal subject has led some observers (v. Hösslin, Bassett-Jones and Llewellyn) to regard the presence of either as proof of simulation, or at least of an attempt to deceive. But in my opinion this view betrays a merely superficial insight into their meaning. The criterion of imitational capability is worth exactly nothing. With practice the normal subject can so empty his musculature of tonus that a limb can become as flaccid as in poliomyelitic paralysis—indeed, the detonization may proceed so far as to result in actual loss of the knee-jerk, as Jacobson has conclusively shown. Here is an 'organic' condition—loss of deep reflexes—which can be effected temporarily by the aid of 'volition' (I regret introducing that highly ambiguous word at this stage, and shall have something more to say of it later). Where, then, is the value of imitation as proof that hysteria and simulation are akin or identical? One might as legitimately claim that atony with loss of a tendon reflex is hysterical and evidence of malingering, since it is not outside the faculty of normal man to reproduce it. The variety artiste on the stage can so deplete his muscles of tone as to give an excellent rendering of a dummy figure, a jointless doll—as atonic and as seemingly 'paralytic' as any state of 'organic' disease could ever be. With practice, again, a very fair reproduction of some of the salient features of Jacksonian epilepsy can be made.

The real point, I submit, is that in respect of motor phenomena imitability characterizes more especially those whose physiological seat is cortical, and that symptoms of escape or release at lower levels can be reproduced with perhaps less facility. However this may be—and it is undesirable at present to lay any stress on the generalization—both the 'shunting' and the 'antagonist contraction' signs indicate, as is here contended, impairment of cortical function, coupled with defective inhibition. The hysteric cannot inhibit antagonistic innervation, nor can he prevent displacement of innervating currents in an erroneous direction.

A word, next, on those forms of motor disability which consist of 'positive' movements. Hysterical tremor, for instance, considered physiologically, presents no separate or contrasting features when set alongside those of so-called 'organic' type. I have frequently found intention-tremor clinically indistinguishable from that of disseminated sclerosis. The reputed differentia of variability, of influence by psychical and emotional stimuli, of dependence on the attention paid to it, and others, are demonstrably invalid. Numerous 'organic' tremors can be affected by a whole series of factors, exhibit marked

fluctuation and fluidity, are highly irregular, shift their incidence, and are aggravated when the subject is under observation. A hundred years ago James Parkinson himself noted the disappearance of the tremor of paralysis agitans in consequence of mere change of posture. As a matter of observation, not a few hysterical movements of this kind sometimes seem as fixed and as invariable as those deemed the result of structural lesions. Now it is within common knowledge that hysterical subjects exhibiting such movements do not complain of fatigue, or at least appear to be less conscious of it than does a normal subject who executes them intentionally; moreover, the Parkinsonian and the torticollic, by way of instance, frequently (though not constantly) admit muscular fatigue and aching. Kretschmer, I consider, is mistaken in asserting that "a rapid, rhythmical and somewhat constant shaking tremor, in general, will absolutely not allow itself to be imitated voluntarily under normal experimental requirements, either by healthy or by nervous persons." On the contrary, we can easily convince ourselves of the possibility of imitation by actual practice. But he rightly points out that under the latter circumstances it leads to such a degree of fatigue that its continuance becomes impossible. The difference in this respect seems to depend on the fact that in hysteria, as in 'organic' states, tremor is an escape-phenomenon of an infra-cortical level, and that the experimenter is presumably utilizing neomotor mechanisms to reproduce the activities inherent in palæomotor mechanisms of a lower physiological grade; fatigue sets in more readily in the former case because such artefactive tremor forms no part of their habitual function. At any rate, it seems feasible from a physiological point of view to attribute the 'positive' movements of hysteria to failure of transcortical inhibition when their type is high, and of corticifugal inhibition when their type is low.

So far as the multiplex syndromes categorized as the hysterical fit are concerned, their range from fugues at the one extreme to immobility and lifelessness at the other covers so wide a symptomatic field as perhaps to render attempts at synthesis precarious. Reduced, however, to their lowest terms, one and all are indicative of release of function from conscious control. Differences between hysterical and epileptic fits are matters of degree and not of kind. In each, the depth to which unconsciousness proceeds is variable, and if more profound in the average major epileptic seizure may be serviceable in clinical diagnosis but not in nosological differentiation. Both show convulsive and quasi-purposive movements on occasion, the latter predominating in hysteria: and while cyanosis, tongue-biting, and micturition frequently distinguish the one, and automatisms the other, the distinctions resolve themselves into the simple question of which mechanisms are prone to be involved, and which to escape, in either. Otherwise expressed, clinical differences stand for modifications in site and extent, but not for intrinsically or essentially different processes. A general resemblance between the attitude in extensor rigidity of many hysterical attacks and that of the decerebrate animal cannot be denied, for it is a matter of direct observation; moreover, it is to be expected, since

if for any reason, through any agency, neuronic control of infracortical mechanisms is in abeyance, these are bound to exhibit on release their specific functional qualities. The physiological characters of some hysterical fits may be taken as evidence of transient (perhaps incomplete) decortication or decerebration, nor can I think any other interpretative view permissible. For the student of hysterical physiology the problem is to examine and determine to what extent, down to what levels, neuronic dissociation can be effected by the processes of hysteria.

SENSORY SYSTEM.

The empiricist taking the sensory derangements of hysteria at their face value notes the neuralgias and topalgias on the one hand, and the anæsthesias on the other. He is struck by their variety and variability, their capacity for rapid change, and—so opinion has it—their failure to correspond to anatomical schemes; they are alleged to conform with psychical but not physiological dispositions. As illustration of this latter statement we see cases in which the breast but not the nipple is anæsthetic, the front of the body but not the back, and we are familiar with the 'glove' and 'stocking' anæsthesia of the textbooks.

But ere assuming therefrom that hysteria is a 'disease of the mind,' a disorder on the plane of mental images, we do well to recall that the physiological arrangement of the mechanisms underlying conduction of sensory impulses varies at successive neural stages. Peripheral loss of sensibility can be distinguished from spinal loss, spinal from thalamic, and thalamic from cortical. In respect of the last of these the clinician can prove from actual experience that a re-arrangement or re-representation of lower sensory schemata takes place, and that with some forms of cortical lesion the distal parts of the opposite limbs are much more affected than the proximal. This fact of itself at once reduces the differential value of 'glove' and 'stocking' anæsthesia, apart from the other fact that it may be found in syringomyelia, as was demonstrated years ago by Brissaud. Further, as regards the arm more especially, evidence can be adduced which clearly suggests the existence in the cortex of a pre-axial and post-axial segmentation, and errors of reference in either direction can be shown to occur with certain cortical lesions. A front-and-back schematization is not only inherently possible, but is rendered probable by studies of Redlich, Vierheller, and others, in both spinal and prespinal cases 'organic' in origin. We recognize the existence of 'sidedness' in sensory disorder of the highest level, and that on an 'organic' basis: *allocheiria* or, preferably, *dyscheiria*, considered distinctive of hysterical states, I have seen characteristically in disseminated sclerosis. Some evidence has accumulated favouring the conception of what may be called a longitudinal segmentation of the trunk in the cortex.

We need not, however, enter at large into details of this kind. The point that emerges is simply this, that in the light of such data it is rash to presume a psychogenic basis for sensory disorders because of peculiarities in their seg-

mental type when there is presumptive evidence of physiological arrangements in the cortex to explain their occurrence.

Some reference is here desirable to the not infrequent concomitance of hysterical disturbances of sensation with absence of bleeding when the cutaneous zone affected is pricked with a pin. So ancient and notorious an observation as this becomes unusually instructive when set beside a further clinical fact, viz., that precisely the same combination has been seen in connection with 'organic' sensory change caused by a lesion in the formatio reticularis of the medulla. I have reported elsewhere the analysis of a case of occlusion of the left posterior inferior cerebellar artery in which the patient discovered for himself that he did not bleed in the analgesic area when the skin was punctured by a needle. I quote from the original record :

Mr. M. had often noticed that pricking of the left face, in addition to its being felt as pressure merely, never resulted in any bleeding. He had frequently experimented with himself in this connection, and says the same thing is true of the right side of the body. About three weeks ago, however, coincident with the diminution of the subjective sensations on the left face, he found that pricking was followed by bleeding for the first time since the onset. I re-examined him from this point of view, and can vouch for the fact that blood is much more readily obtained by pricking on the left hand than on the right, whereas there is no difference now between the two sides of the face.

That some vasomotor change had occurred over the left face and right body and limbs seems certain—presumably a degree of vasoconstriction. The association of the same vasomotor state with hysterical anæsthesia is deeply interesting. I remember a case of complete hysterical paraplegia in a young woman of 22 where the degree of analgesia was so profound that a needle was passed through the calf without its being perceived, and without causing any bleeding. After a single application of the wire-brush the analgesia disappeared and bleeding occurred at once whenever the skin was pricked.

It seems only one conclusion is permitted by comparison of these two cases ; an alleged stigma of hysteria turns out to occur also from 'organic' lesions of the medulla and loses any specificity it is thought to possess ; moreover, the inference can justifiably be drawn, I submit, that bulbar centres and mechanisms are involved in certain cases of hysteria, and that these in part belong to the neurosympathetic system.

REFLEX SYSTEM.

Anyone with clinical experience will allow that reflectivity frequently exhibits departures from what is normal. In hysterical hemiplegia or paraplegia the observation is now old and well-established that the plantar response is often lost : with repeated stimulation no reaction is obtainable, and this, too, when the foot is neither specially cold nor anæsthetic. I have been familiar with this for many years, as also with the fact that the abdominal reflex may be reduced (or even lost) in hemiparetic cases, usually in association with sensory diminution on the same side—an observation substantiated by

Monrad-Krohn. Of the occurrence of hysterical ankle clonus no doubt can be entertained. Interference with sphincteric action, too, needs no comment ; incontinence of urine was present for six weeks in the case of a hysterical paraplegic under my care, and retention is no rarity.

Dr. C. S. Myers is the psychologist referred to above who, almost alone of his class, has paid any attention to these phenomena, or evinced appreciation of their importance. At the end of the War he said that "in this country at least we have been paying so much attention to the mental aspect of the war neuroses that a detailed examination of the accompanying bodily symptoms has been generally neglected." His own observations showed him the frequency with which the plantar reflex fails, and he also remarked in many cases of asymmetry as regards the two that both knee-jerk and abdominal reflex appeared weaker on the same side as the feeble flexor or abolished plantar response, and that diminished appreciation of cutaneous stimuli was usually in evidence also.

I long ago satisfied myself that distinctions between 'organic' and hysterical ankle clonus formerly relied on could not be trusted ; Babinski's criterion that the former is present each time the patient is examined, whereas the latter varies and may not be elicited on occasion, has, from my personal experience, turned out to be valueless. Nor does irregularity of rhythm carry any differential weight. Now if diminution of the abdominal reflex is depended on as an early sign in frontal tumours, disseminated sclerosis, and pyramidal conditions generally, I cannot see why the same phenomenon in cases of hysteria should not bear the same meaning or import. It is unnecessary now to advert to the rather disputed question of the development of an extensor plantar reflex in hysteria, but loss of the plantar reaction must signify some physiological change. A similar reduction characterizes many cases of 'organic' hemiplegia at the acute stage, and according to Ganault the reflex is completely abolished in 10 per cent. No reason whatever can be advanced for accepting these physiological specialties in 'organic' affections and denying them equal significance in cases of hysteria.

For simplicity's sake a few comparatively common, every-day symptoms of hysteria have been selected for examination from a neurophysiological angle. This sketchy and compulsorily limited review has perhaps sufficed to demonstrate how cortical, infracortical, mesencephalic, bulbar, spinal and neurosympathetic mechanisms may severally be disordered in function as a result of processes which we call hysterical. No doubt in one sense this is a mere platitude ; for after all what others are there for hysteria to usurp than those of the person concerned ? But it is something, surely, to be able to show that differentiating criteria as between organic and hysterical conditions have lost much of their specificity, that symptoms thought exclusively hysterical

reappear under circumstances never assigned to the interference of the psyche, and that many of the somatic manifestations of that 'neurosis' have their counterpart in known physiological derangements of one grade or another. In one at least of its aspects the affection becomes less mysterious when its activities can be traced through different physiological levels.

The progress of medical knowledge has been so gravely hampered by persistent use of the two terms 'organic' and 'functional' that it is high time they should be discarded for ever. Scarcely less objectionable is the alternative antithesis of 'structural' and 'nutritional.' (I have studiously avoided in this discussion the word 'functional' even as reputedly synonymous with 'hysterical.') In their place, if terminology is required, we may with advantage employ the pair of opposites 'static' and 'dynamic.' It must be clear on due consideration that innumerable nervous symptoms stand for dynamic alterations in function which cannot at present be correlated with any recognizable lesions. In the case of a major epileptic fit manifesting itself all over the body, so to speak, no one can imagine for a moment that there are corresponding 'lesions' all over the nervous system. The display is a physiological reaction to some nociceptive excitant whose site of initial action is probably extremely limited, but a dynamic, and transient, extension follows over a wide region of normal nervous tissue. In a number of disease-conditions associated with static lesions the symptoms themselves exhibit fluidity of the most obvious kind, as has already been alluded to in connexion with the tremors of paralysis agitans. How then can we link them to the cellular changes we detect under the microscope?

Applying this general semeiological conception to hysteria, we note at once the prominence of the dynamic and the comparative absence of any static elements therein. We are impressed by the strictly physiological inhibitions and excitations of that state and by their ranging over higher and lower mechanisms alike: but at the same time their essentially unfixed and fluid character stands out saliently. How can we explain the rapid cure of hysterical disabilities—or shall I say their clinical disappearance—how understand that nervous mechanisms are as it were ready to resume duty at a moment's notice, after perhaps some years of disuse, unless on the hypothesis that function is abrogated dynamically but not statically? There is a block somewhere, a pure inhibition, in the case of negative symptoms: or, on the other hand, inhibition is removed and release-phenomena come into being as positive symptoms. Neither can be correlated with lesions of a static kind, but that physiological derangement of a dynamic character is invariably present seems almost self-evident, and from the illustrations already furnished may be considered objectively proved. The difference, then, from so-called 'organic' conditions does not reside in the co-existence of static change, for this need not accompany all the manifestations of that class, but rather in the manner of onset of the symptoms and the manner of their removal.

ETIOLOGY.

We can trace through the symptomatological diversities of hysteria the action of ordinary physiological processes, precisely the same as are encountered in the dynamic symptoms of affections we categorize as having a basis in recognizable disease of tissues ; but as soon as we approach the question of their origin and pathogenesis difficulties accumulate. In these days a majority of psychiatrists and neurologists presumably adopt the view of their ideogenic origin, and regard this as almost axiomatic. Perhaps the most commonly accepted theory is that they are conditioned by one or other kind of sexual conflict, of which, to take concrete examples, some may be enumerated as follows : jilting in love, jealousy, lovers' quarrels, a sister's marriage, fear of pregnancy, marital unhappiness and so forth. To enlarge the catalogue would be both easy and superfluous. Frankly, this strictly sexual etiology appears incapable of universal application to questions of hysterical pathogenesis. Why and how such psychical incidents should produce loss of the plantar reflex, or local vasoconstriction and cutaneous anæsthesia, eludes comprehension. On the view that hysterical symptoms are nothing else than reactions to stimuli, it still is difficult to understand why psychical excitations of this completely restricted sexual kind should be capable by themselves of evoking responses ranging from fugues to retention of urine. With some knowledge of the extensive psychological literature on the subject I have not met with any serious effort amid its profuseness to correlate etiology and symptomatology. Between the one and the other stretches a pathogenic hiatus of a glaring kind, which cannot be said to facilitate acceptance of the sexual theory.

If the etiological net is cast a little wider, we might adopt the hypothesis that hysterical reactions are determined by the emotional life in an unrestricted sense. Emotional experiences and impulses, originating in whatever fashion, associated with sexuality or not, do unquestionably help to engender the development of hysterical syndromes ; they may be taken as exciting, if not predisposing, causes. It is, perhaps, easier for the trained neurologist to comprehend the physiological effects of emotional disturbance, since he has at least a little knowledge of the mechanisms of the neuraxis underlying the expression of the emotions. Radiation of emotional effect can be translated into anatomo-physiological terms ; the somatic and neurosympathetic accompaniments of emotional action range widely and are capable of objectivation. Varieties of shock have their reverberation in known high and low level neuronc systems. Nevertheless individual hysterical reactions, monosymptomatic types, are not easily correlated with the causative action of so *general* an excitant or inhibitant as emotion. Of course I recognize and for that matter have myself observed the occurrence of hysterical symptoms (such as aphonia or amaurosis, for example) in which it has been feasible to link the *form* of the reaction to some pre-existing incident connected with the particular part of the body affected, but in numerous others it is impossible to trace any such

association. A comprehensive study of hysterical semeiological types in relation to etiological variants is urgently needed.

The assertion that we are all potential hysterics, often put forward both before the War and since, is explained by Kretschmer to mean that everyone carries within him old, instinctive, primitive forms of reaction more or less covered up and controlled by the acquisitions of culture. Most of those who have had experience of hysteria will agree that it is apt to appear at certain physiological periods under circumstances of an emotional kind. Yet at times it undoubtedly arises unexpectedly: I have seen hysterical hemiplegia in a King's prizeman at Bisley. To the generalization, however, it is impossible to give formal consent; on the contrary, I submit that the evidence favours the view that the hysteric is born and not made, and that an unstable constitutional background, a 'temperament,' if the word is preferred, is the soil from which hysterical growths spring. And it seems feasible to suggest that this determinant may be in reality of a physiological and not a psychological kind—that it is constituted by an inherent lability of reaction, a lack of neuronie cohesion and firm integration, an abnormal facility in dissociation of neural systems, such as undoubtedly characterize hysterical semeiology from the viewpoint of nervous function. When we are told that the "acquisitions of culture" cover up the "primitive" within us, it appears to me preferable to say that transcortical inhibition dominates the neuraxis; when the hypoboulia of the hysteric, his weak will, is paraded as explanatory of hysterical pathogenesis, a physiological exposition of the data might, in my view, equally well be sought; we might speak rather of failure of one cortical element to inhibit another—a state of affairs the extreme likelihood of the occurrence of which is postulated by Sherrington for various fields of cortical action. That 'volition' seems constantly, or at least repeatedly, to take a share in the production of hysterical symptoms is notorious, and is responsible for the not unnatural view that simulation enters largely therein; but it is conceivable that the physiological basis of the 'will' is inhibition; that a 'voluntary' movement signifies merely one which is transcortically permitted; that 'letting go' rather than 'doing' represents the true meaning of a 'volitional' act. With a conception of this kind, developed along physiological lines, the peculiar features of hysterical motor disorder receive a simple explanation; we do not mix up a psychological impairment—a weak will—with a physiological derangement—defective inhibition of antagonists—but take the former to signify failure of transcortical control and elucidate the latter thereby. An ingenious explanation, on purely physiological grounds, of the phenomena of hysterical palsy was published by Graham Brown and R. M. Stewart in a paper which appeared at the end of the War and seems not to have attracted the attention it certainly merits.

The general tenor of the argument here outlined with some restraint is that physiological processes lie behind nervous manifestations at all levels; that the cortex, considered as a ganglion, cannot differ inherently in function

from other, infracortical, ganglia except as regards complexity ; there is no reason to suppose nature makes a jump from one order to another totally different at the cortical stage. Reflex actions are there to be expected, in analogy with those located inferiorly. The present trend of physiological research is to reduce steadily the number of functions once ascribed to 'volition' ; for example, mechanisms controlling posture and balance are now taken away from it and assigned to reflex arcs independent of the higher levels. So far as the cortex is concerned, I am by no means alone in arguing that much cortical activity partakes absolutely of a reflex character ; and I fancy that what for want of a better expression we term the 'will' is being driven from one transcortical ditch to another as it struggles to survive. It seems logically sound to seek to interpret cortical activities in physiological terms alone, and to avoid use of the psychological term 'volition' altogether. Admitting that many of the phenomena of hysteria belong obviously to the highest grade of activity, are so like 'volitional' actions as to be thought indicative of deception or simulation, it is suggested they represent transcortical disorder of function, on the strict analogy of functional release at lower levels. An approach from below upwards, as it were, has much to commend it, for the gap between physiological and psychological systems is scarcely likely to be bridged from the psychical side.