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DIFFERENTIAL DIAGNOSIS OF FUNCTIONAL AND ORGANIC NERVOUS DISORDERS*

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It would be well at the outset of the discussion to define the terms "functional" and "organic." Strictly speaking, all symptoms of illness are, of course, functional, in that they are manifestations of disordered function; but here I propose to use the word in a narrower sense as indicating those symptoms which are psychogenic. By "organic" will be meant symptoms which result from abnormal physical alterations in the nervous system. The word "abnormal" is advisedly used, for all forms of activity of the nervous system must be associated with physical changes. These morbid alterations may be so gross that they can easily be detected by the pathologist, and have long been recognized. Others are less obvious, and, like those in the basal ganglia in paralysis agitans, have only come to light in recent years through the development of histological technique. There is still another group of cases in which the symptom is believed to be organic, although structural changes may or may not be found post mortem; this group is largely composed of disorders of function which are transient and recurrent. During the attacks there may be present signs which are accepted as of organic significance—for example, in epilepsy, loss of consciousness, abolition of tendon reflexes, or extensor plantar responses—while in others, such as narcolepsy, there may be no physical signs of this order.

It is therefore clear that, although the pathologist has done much in the differentiation of organic from functional nervous disorders, the main burden has rested on the clinician, and still does. By the sifting of accumulated experience gained at the bedside, through careful investigation of the natural history of disorders, the characteristics of individual symptoms, and the searching for physical signs, he has led the way in differential diagnosis. The physical signs of organic disease are often crude and are comparatively few, but they are gradually becoming more refined and are increasing in number. But it is not only on the elicitation of physical signs that the physician ought to depend for differential diagnosis. Symptoms, including the patient's own sensations, have for long been taken seriously by the clinician, and will, in the future, yield information of the greatest value, for frequently they indicate the earliest stages of disordered function before structural changes develop of sufficient severity to give rise to physical signs. This leads up to the importance of careful history-taking, the methodical investigation of symptoms, and their temporal relationships. An adequate history should, of course, include an account of the patient's past illnesses and his habits,

and of the health of his family. One should, in addition, attempt to get some idea of the main psychological influences that have affected him, particularly if diagnosis is uncertain. In other words, a proper medical history is one which recapitulates the life of the patient as a whole, of which his present illness forms but a part. "Disease" is a vital process with a natural history, and symptoms are not a series of isolated events.

Clinical Criteria of Differential Diagnosis

What, then, are the indications which have been proven to be of use in the separation of functional and organic nervous illnesses? The psychological aspect of the problem will be dealt with by Dr. Petrie, but let me say that the history of a psychoneurotic almost always shows a tendency, from childhood onwards, to face trouble badly. In the past, he may have been able to carry on with his work, and may not have broken down before; nevertheless, emotional disturbances have produced symptoms of a psychogenic kind. In fact, to borrow a definition, a neurotic is a person who is unable to strike a sensible bargain with existence. Dr. T. A. Ross, as the result of his great experience, stresses this point by saying that in an obscure case the diagnosis of a psychoneurosis should not be made in the absence of a psychogenic history. It may be argued, with justice, that every individual has his breaking-point, and that an anxiety, resulting from a threat to himself or to those to whom he is deeply attached, may precipitate a mental illness. But it is questionable whether the illness would persist for long if the individual possessed an average capacity for adaptation as shown by his behaviour in the past. If the present illness was not preceded by emotional strain, and if the past history shows that the ill person had passed through experiences distressing to him, and yet was able to adjust himself to them in an adequate manner, the symptoms which now trouble him, however much they may resemble those of a psychoneurosis, should not readily be accepted as such.

For example, sleeplessness at night with or without lethargy during the day is now a well-recognized symptom of lesions of the hypothalamus from various causes, and, when due to encephalitis lethargica, is often, especially in children, associated with irritability, tearfulness, and fear. Fatigability, a common complaint in neurotic persons, may also be an organic manifestation, as in Parkinsonism and myasthenia gravis. Certain headaches—for instance, those of intracranial hypertension—pains in the trunk and limbs as in tabes or polyneuritis, and the paraesthesia in subacute combined degeneration, are of the same order, but a careful analysis of such symptoms in the process of history-taking and a knowledge of features

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of organic significance should prevent many mistakes in diagnosis.

A single symptom such as sudden diplopia or misty vision in one eye, which persists for a few weeks, ought, especially in a young adult, to make one suspect organic disease. This suspicion becomes almost a certainty that one is dealing with a case of disseminated sclerosis if, as the story is unfolded, other transient disorders, like numbness and clumsiness of a limb, or hesitancy of micturition, are found to have occurred even after intervals of many years of good health, in spite of a complaint from the relatives that the patient has become more emotional than before. This symptom in itself is significant in such a story, for it is common for patients with disseminated sclerosis to laugh or cry too easily, and has undoubtedly an organic basis. To stress the meaning of a history of this kind is perhaps to underline the obvious, if it is supported by the presence of physical signs; but experience has shown that its diagnostic importance is not much diminished by the absence of physical abnormalities. They will almost certainly appear later.

Physical Signs

Coming to the physical signs of neurotic ailments, we can put to one side the exaggerated reactions to fear, at the same time remembering that they may be indications of mild hyperthyroidism, which is often mistaken for a psychoneurosis. It is sometimes difficult, or impossible, to say which is primary—the glandular disorder or the anxiety state. Long-continued emotional stress seems to stimulate thyroidal activity, and it may be said with some reason that the naturally over-anxious person has also, as a part of his heritage, an abnormally active thyroid gland. This association probably determines the onset of illness in many women who break down at the climacteric. The major part of the symptomatology of the so-called menopausal neurosis as well as the physical signs are those with which we are familiar at earlier periods of life in cases of mild hyperthyroidism. The neurotic physical signs which will be particularly discussed are those of hysteria. From the point of view of physical examination the diagnosis of hysteria rests on: (1) the absence of signs of organic disease; and (2) the presence of positive signs of hysteria.

Certain manifestations are invariably organic in origin. Of these the most important are dysphasia with or without dysgraphia, papilloedema and optic atrophy, nystagmus with the slow movement towards the rest position and the quick movement towards the object of fixation, absence of vestibular reactions, local muscular atrophy with alteration in the electrical excitability coming on within a few weeks of the onset of paralysis, inequality of the abdominal reflexes or the tendon jerks or abolition of the latter (the abdominal reflexes are difficult to obtain in people with lax abdominal walls), and a true extensor plantar reflex.

Many sensory abnormalities are of equal value. Some only will be mentioned, and others will be referred to later. Of defects in the visual fields, central scotoma for red and green, with reduction of visual acuity and homonymous or bitemporal field defects, especially if quadrantic, are important; so also is pain which consistently is in the distribution of spinal posterior roots, all the more if it is associated with cutaneous tenderness or sensory loss. There are other pains, such as those of *tic douloureux* and the lightning pain of *tabes*, and the neuralgias of the head and face, due to sinus infection, which have features of their own. Certain varieties of sensory dissociation occur only from organic lesions. Obvious examples are the analgesia and thermo-anaesthesia of segmental distribution with preserva-

tion of tactile sensibility in syringomyelia and other central lesions of the spinal cord, and loss of postural sensibility with ataxia, diminished vibratory sense, and astereognosis as the predominant sensory defects from posterior column lesions.

To the unwary, however, some of these physical signs may appear to be present, whereas they are the result of faulty methods of examination or a lack of familiarity with normal variations. Thus, in testing the tendon jerks, care must be taken that the two limbs which are being compared are in the same position and the muscles relaxed. In regard to the plantar reflex, most stress should be laid on the response obtained from the outer edge of the sole, which is the focus of the receptive field for the reflex. With slight lesions of the pyramidal tract the abnormal reaction may be obtained only from this part, and stimulation of the rest of the sole may yield the normal downward movement of the toes. In doubtful cases help is usually obtained by observation of the reaction of the rest of the limb to plantar stimulation. In normal people who are not over-sensitive, the flexor plantar response is often accompanied by a quick contraction of the muscles in front of the thigh, whereas if the reflex is organically abnormal, whether the toes move up or fail to move, the limb flexors tend to contract. This contraction can be best appreciated in the hamstrings by feeling their tendons tighten. If then the toe response is different on the two sides, observation of the effects of the response on the quadriceps and hamstrings in each limb is often helpful. If the patient is over-sensitive, and especially if the stimulus is painful and applied roughly without due warning, the reaction in the limb is likely to be a sudden flexion or withdrawal of the limb, and the toes may either extend or flex. This is largely a voluntary response.

A superficially similar reaction is common in severe spastic paralysis of organic origin, but there are important differences. In the organic case, as the pin is being drawn along the sole from the back to the front of the foot, jerky flexion movements associated with extension of the toes commonly occur before the limb becomes vigorously flexed. Sometimes, however, violent withdrawal is almost immediate. Observation of the after-response is then of value. In the organic case, if the spasticity is more extensor than flexor, the limb, after a brief delay, is vigorously thrust out with the muscles in strong contraction, the toes being usually extended. Should the spasticity be predominantly in the flexor muscles of the limb, the subsequent reaction to withdrawal is slower and jerky. Once these reactions have been seen they can again be easily recognized. Usually, however, the response to stimulation of the sole is not the only available physical sign for diagnosis. The tendon reflexes, the reaction of the muscles, if rigid, to passive stretching and to voluntary efforts, are all of the greatest value. The final conclusion will rest on the whole available evidence.

The other source of fallacy lies in the want of recognition of normal variations—for example, the confusion between the haziness of the optic disk in a hypermetropic eye and papilloedema, and between a large physiological cup and optic atrophy; so also is apparent weakness of one side of the face and tongue the result of natural asymmetry of the facial bones.

Physical Signs of Hysteria

A psychoneurosis is the outcome of an attempt to solve a mental conflict. The imperfect and irrational solution results in illness which, as in anxiety states, still leaves the patient with fears and obsessions. In hysteria, however, the development of a physical disability, such as paralysis or blindness, is, as a rule, completely satisfy-

ing, and with its appearance worry vanishes. "Belle indifference" has long been known to be characteristic of the simple hysteric whose anxiety returns if he is compelled by the suggestions from a personality he believes to be stronger than his own, or by sudden emotional shock, to give up his fears and obsessions. The cure of hysteria, therefore, demands not only the disappearance by suggestion and explanation of physical disabilities, but also a reasonable adjustment, satisfying to the patient, of the conflict which led to their appearance. Hysterical symptoms may, however, and frequently do, form an incidental part of other psychoneurotic illnesses, and psychotic states, such as depression and schizophrenia, as well as gross physical illnesses, are often not free from them.

Hysterical manifestations are mental in origin, and are determined by the individual's idea of what constitutes paralysis, loss of sensibility, or a fit. They do not, therefore, conform to the rules of pathological physiology and anatomy. It is this discrepancy that enables the physician to differentiate between signs of hysterical and those of organic origin.

Hysterical Paralysis

Almost the whole body, or one side of it, or a limb, or only the peripheral part of it may be affected. Paralysis limited to the adductors of the vocal cords, resulting in aphonia, is common. The distribution and form of the paralysis is determined by suggestion frequently provided by a physical illness or accident, which in itself is often trivial. Thus laryngitis may lead to aphonia, or a slight wound to the hand to paralysis of this part. The factor which determines the perpetuation of disability in hysterical form is a state of anxiety with consequent unhappiness, or a fear of the future, after the physical illness is over, and it may be that the patient unconsciously welcomes disablement so that he need not continue with disagreeable tasks or can attract sympathy, while at the same time the necessities of life, flavoured with luxuries, can be provided for him by others.

Characteristics of Hysterical Paralysis

It is often selective. All voluntary movement in the affected part may appear to be impossible, but there are no reflex signs of organic significance or wasting, unless the paralysis has lasted for many months. The paralysis may, however, be incomplete. In such cases two forms of reaction may be observed. The first is that the patient is unable to use certain muscles for one voluntary movement, while he is able to employ the same muscles for all other voluntary movements for which they are required. The following examples are illustrative. The hysterical aphonic talks in a whisper but can usually cough to command, equally a voluntary act. So also the act of writing is difficult or impossible in writer's cramp, although the hand is as good as ever for all other movements. That the paralysis represents an idea of disability is again shown by the case of the sergeant who, after fighting successfully for three years, quite naturally became tired of it, but discipline and a sense of duty compelled him to struggle on in spite of his desire to escape. During a bombing attack he was slightly wounded on the forearm, and soon after became unable to grasp a bomb or a rifle with his right hand, but never lost the power to use it for other purposes. The paralysis, which incapacitated him from fighting but for nothing else, enabled him to get the rest he required with preservation of his self-esteem.

Again, in the form of hysterical paralysis known as *astasia-abasia*, the inability is confined to standing and walking, for in bed all voluntary movements of the lower limbs can usually be well carried out. The same incon-

sistency can be observed in cases of ataxia. Thus, although the patient staggers in walking and sways when standing, especially if asked to do so with closed eyes, so long as these actions are in his mind, he will, nevertheless, stand steadily if his attention is directed to the examination of his pupillary reactions.

The second form of reaction consists of the display of excessive effort, which is manifest not only in a general way by clenched teeth and hands and of violent muscular exertion of the non-affected portion of the body as if the patient were trying to show that he was doing his best, but also by strong contraction of the opposing muscles at the joint of the affected limb which he is asked to move. Either no movement results, or it is accomplished in jerks due to irregular relaxation of the muscles opposed to the prime movers. These two modes of behaviour are diagnostic of hysteria, and, if recognized, cannot lead to confusion in diagnosis.

Passive movement of a paralysed limb in hysteria may evoke no resistance, or resistance of a diagnostic kind may be encountered. It is then proportionate to the effort expended by the physician in the sense that the more he tries to move the joint so the resistance steadily increases. This reaction, which is due to muscular spasm brought about by the unconscious or, more truly, the partially conscious efforts of the patient to resist, can easily be differentiated from the two common varieties of organic muscular rigidity. These are due to pyramidal and extra-pyramidal motor lesions. When one attempts to bend the paralysed knee of a patient with spastic hemiplegia, the so-called clasp-knife rigidity is encountered. At first the movement is easy, then resistance is suddenly encountered, but if pressure is maintained it gives, although not completely, and the rest of the movement can be carried out fairly easily and with diminishing effort. In Parkinsonism the tonic reaction of the affected muscles is different. It is displayed throughout the whole movement at the joint, but is jerky, hence the term "cogwheel" rigidity. Its jerky character can, as a rule, in slight cases of Parkinsonism, be most easily detected at the wrist, but it should be remembered that the hand may have to be moved in flexion and extension several times before it appears.

The various contractures met with in hysteria are the result of muscular spasm, which increases when attempts are made to overcome the abnormal posture either by the patient or the physician. Furthermore, postures of the limbs always differ, at least in detail, from those of organic spastic states, and not uncommonly the hysterical contracture is limited to a hand or foot. That hysterical contracture is due to active spasm is well shown in cases in which there is apparent weakness of one half of the tongue. The tongue, say, is protruded to the right, but when withdrawn into the mouth it still deviates to the same side, whereas if the unilateral paralysis had been organic, retraction would inevitably cause deviation to the opposite side. So, also, hysterical diplopia with convergent squint is the result of voluntary or semi-voluntary contraction of the internal recti.

No manifestation of hysteria can be recognized so quickly as the manifold disorders of gait. One or both limbs may be affected. The patient may drag the paralysed limb along behind him with the foot firmly scraping along the floor, or will ostentatiously, in his jerky and irregular progress, clutch at objects in his way that would give him support. The observer who may be helping him to walk is soon aware that the patient is trying less to support himself than to sink to the ground, quite unlike the sincere efforts of the paraplegic whose disability is of organic origin. Hysterical helplessness in walking is all the more striking if, as may be the case, movements of the lower limbs are well carried out in bed.

Hysterical Sensory Disorders

Loss of sensibility in hysteria differs fundamentally from that due to organic causes. In the first place, it fails to conform to anatomical distributions—central, root, or peripheral. Thus, cutaneous analgesia in a limb tends to stop short at obvious landmarks such as the wrist, ankle, or knee. If it extends to the groin in front it is bounded behind by the fold of the buttock, and does not include the lower sacral areas. If the whole upper limb is analgesic the upper border encircles the shoulder-joint, a distribution which would be impossible in organic states. So also the front of the trunk may be insensitive while the back is intact. Secondly, the boundary of the analgesic area is sharply defined. Polyneuritis and subacute combined degeneration give rise to cutaneous sensory loss encircling the lower limbs or trunk, but the analgesic areas correspond to peripheral or segmental sensory distributions and their upper borders are never abrupt.

In organic states examples of sharp delimitation between regions of normal sensibility and sensory loss are few and are well explained on anatomical and physiological grounds. When, for example, there is analgesia over one half of the body, the line of demarcation between the areas of abnormal and normal sensibility is sharp. But should there, in addition, be absence of vibratory sense in the limbs and abdomen, on one side, a vibrating fork can still be appreciated by the chest, especially if it is placed near the sternum, since the chest is a sounding-box. In complete hysterical hemianaesthesia, however, appreciation of vibration may well be abolished on one half of the chest as well as cutaneous sensibility.

When there is complete loss of sensation below a level on one or both sides of the body, if the sensory loss is organic its upper border will have segmental or root characteristics, whereas in hysteria it will not. For example, the upper border of analgesia on the trunk, when organic, is never strictly transverse, but follows well-known wavy lines, whereas in hysteria it does not. Lastly, because of the budding out of the limbs, certain sensory root and segmental areas become contiguous with others from which they are separated by those which correspond to intervening roots or segments, so that the sensory overlap is absent. When this happens the borders of sensory loss are sharply defined. These regions of strict delimitation are especially between the outer and inner aspects of the upper arms, the anterior and posterior aspects of the thighs, and the sacral saddle-shaped areas in the buttocks; on the trunk, a similar abrupt transition is found roughly at the level of the second ribs in front and the spines of scapulae behind, where the dermatome of the fourth cervical segment marches with that of the second thoracic.

A third characteristic of hysterical sensory loss is that it can be varied and abolished by suggestion. A fourth is that the inevitable, indirect consequences of organic sensory loss may be absent in hysteria. For example, ataxia of the lower limbs in polyneuritis, tabes, or Friedrich's ataxia is due to loss of postural sensibility from lesions of peripheral nerves, sensory roots, or posterior columns, and is naturally worse in the dark and if the patient's eyes are covered. In carrying out Romberg's test in the hysteric, however, the response obtained will, as has already been mentioned, depend upon whether or not he understands that his steadiness or some other function is under investigation. Hysterical loss of vision illustrates the same principle. Blindness may appear to be complete; but, even so, the pupillary reactions to light are brisk, the fundi are clear, and the patient usually succeeds in avoiding objects when he attempts to walk about. Spiral fields of vision have long been known to indicate hysteria, and doubtless are the result of sugges-

tion in performance of the test. Unilateral blindness and deafness may occur in association with complete hemianaesthesia. The psychogenic origin of the deafness as in cases of bilateral deafness can be recognized by evoking nystagmus by rotation of the body, and the blinking reflex by a sudden noise.

Fits

It is sometimes difficult, if an attack has not been observed by a competent witness, to decide whether a fit is epileptic or hysterical, especially if there is no history of injury during a fall, incontinence, or tongue-biting. All these features, which are common in epilepsy, may at the same time be absent. If the physician or a trained nurse has observed an attack it is usually easy to arrive at a correct diagnosis. The hysterical seizure is characteristically a dramatic performance with arching of the limbs and trunk, violent purposive movements, and often shouting and articulate speech. The patient may become cyanosed from holding his breath, but his eyelids are usually tightly shut and resist efforts to open them, and his pupils react normally to light. Furthermore, after the attacks the tendon-jerks and the plantar responses are invariably normal. Difficulties may, however, arise if the physician is unacquainted with less recognized forms of epileptiform attacks. Thus, in so-called tonic fits due to sudden disturbance of function of the upper part of the brain stem from organic lesions such as tumour or tuberculous meningitis, there is opisthotonus and the limbs are rigid, the lower in extension with pointing of the feet and toes and the upper flexed or extended; but the eyes are open, the pupils are usually inactive to light, and incontinence may occur. Certain minor seizures, especially if unassociated with loss of consciousness, may present more difficulty. Thus in "myoclonus epilepsy" as the result of sudden muscular contraction the patient, although conscious, may fall down, or, if an upper limb is the part affected, an object held in the hand may be thrown across the room. So also in cases of "inhibition epilepsy," in which muscles of the limb are suddenly relaxed, falling may occur without apparent loss of consciousness. Again, involuntary clonic movements are often limited to a limb or one side of the face in Jacksonian attacks associated with focal cerebral lesions, but physical signs are to be found in the affected part at least for a time after the seizure. The necessity for routine examination of the optic disks is recognized but is not always applied.

Hysterical amnesias and fugues may occasionally be difficult to differentiate from automatism following petit mal attacks, but a careful history is usually decisive. Moreover, the hysteric in a fugue is not inclined to empty his bladder, as in the classical case of Trousseau's Judge, remove his clothes in public, or be homicidal; in fact, his actions usually pass unnoticed. A chronic epileptic is often an emotional subject, and may show hysterical symptoms from time to time like other unstable persons. Nevertheless, there is little justification for the term "hystero-epilepsy."

This leads to the last point to which I would draw your attention. Emotionalism does not necessarily mean that the ill person has not some organic illness. It is perhaps equally unnecessary to say that states of anxiety may act as precipitating causes of physical disturbances, such as migraine, intracranial hypertension headaches, and occasionally epileptic attacks, as well as aggravating enfeeblement of organic origin. But it is less generally recognized that emotionalism may in turn be due to proven structural disease of the brain. Emotional facility, with an undue tendency to laugh or cry, is one of the commonest manifestations of disseminated sclerosis, a disease in which the transient character of the early disorders of function is apt to lead the unwary into making a wrong diagnosis.

All the more so if at the time of examination no physical signs are discovered. A similar emotionalism occurs also in pseudo-bulbar palsy, progressive bulbar palsy, chronic alcoholism, dementia paralytica, and occasionally with cerebral tumours, but by the time it appears, clear indications of structural damage can usually be found.

Lastly, the apathy of Parkinsonism, which is often more apparent than real, may be wrongly interpreted as psychogenic. But quite apart from the unmistakable physical abnormalities, even in early cases, which are now common knowledge, the lack of emotional reaction should in itself lead one to suspect its organic basis.

In conclusion, while the analysis of symptoms and signs is of the utmost value in the subject under discussion, the investigation of the behaviour of the individual as a whole in the past as well as in the present is the basis upon which diagnosis has often to rest. Let us not forget, too, that a neurotic may develop organic troubles like other people, and also that a person may become mentally unstable as the result of anxieties consequent upon physical disabilities. In this respect, minor head injuries ought especially to be remembered.

DIFFERENTIAL DIAGNOSIS OF ORGANIC AND FUNCTIONAL NERVOUS DISORDERS *

BY

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The differential diagnosis between functional and organic nervous disease is obviously a wide subject. At the outset an exact definition is needed as to what constitutes functional nervous disease. Broadly, this may include all nervous manifestations in which no evidence of pathological changes in the nervous system has been demonstrated. Cases of dysfunction without demonstrable organic lesion may therefore be classed among the functional cases, and disturbances resulting from psychological stresses in the highest psychic level will also need to be included. The symptoms of the diseases commonly known as the neuroses or psychoneuroses will, of course, come under review, as will some of the psychoses. While some psychoses, such as general paralysis, have a very evident organic basis, others represent derangement of function, and little objective organic evidence may be obtainable.

The association of so-called functional elements with organic lesions raises an even wider issue. Cases in which no functional element was apparent before an organic lesion developed bring up the whole question as to the relation between these groups, and as to how far they can be justifiably separated. The attempt may be difficult, as may be judged from the fact that a leading neurologist stated, in discussing hysteria, that there were few organic symptoms which he had not seen reproduced in that disease. Functional disorder of the nervous system may disturb any bodily system and lead to confusion with diseases other than those of the nervous system, and separating the functional incapacity in a given lesion from that due to organic disease may be very difficult. In a short paper it is obviously impossible to deal with all aspects, but I shall endeavour to show the importance of examining the problem as a whole, and of not neglecting the psychological approach.

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Consideration of the person's whole make-up and circumstances often renders the diagnosis clear when individual symptoms may be very puzzling. Recognition of a hypochondriacal attitude of mind is of great importance in the valuation of symptoms. This abnormal concentration on the individual's own body represents a psychological abnormality, whether it is present as a simple neurosis or whether associated with a depressive or paranoid psychosis.

Neurasthenia

As I am approaching the subject rather from the functional point of view, I shall first consider the different aspects of the neuroses. While hysteria and its subsidiary, the traumatic neurosis, perhaps provide the principal problem, neurasthenia and anxiety neurosis, and the various mixed types, also need consideration. By neurasthenia I mean the exhaustion neurosis, in which the patient shows marked mental and physical fatigability.

The individual is usually a limp, sparsely nourished, unhealthy-looking person, unable to concentrate, finding all exertion, mental or physical, too great. If attempting to perform a physical act he rapidly tires, and he becomes increasingly inefficient when faced with a mental task. Special tests prove this to be a feature of a genuine case. Such a state of affairs exists to some extent after any great and debilitating illness, but the conditions persist in the neurasthenic long after the recuperation would have taken place in the normal individual.

The presence of anaemia, tuberculosis, or any chronic associated factor must of course be eliminated, and then arises the question of some chronic toxæmia. The teeth and intestines fall under suspicion, sometimes rightly, but at times drastic measures in regard to these are adopted without avail. Sometimes the case is one of incipient melancholia, the loss of interest and disinclination to exertion being a stage in the depressive phase of a cyclothymic or manic-depressive psychosis. What is at first regarded as a post-influenzal debility, and then as neurasthenia, may ultimately prove to be the early stage of a depressive psychosis. Usually, however, the associated depression, as opposed to a limp apathy, differentiates between them. Too much listless apathy and indifference to external circumstances would, on the other hand, arouse the fear that the case was really one of schizophrenia, but the pure neurasthenic is not given to fantasy, "day dreaming," and other features of the praecox case. Although the fatigability of the muscles in myasthenia gravis to some extent suggests neurasthenia, it is not common to confuse the condition. The facial muscles are usually quite mobile in neurasthenia, and there is no paralysis of groups of muscles such as the orbicularis palpebrarum or palatal muscles. The acute symptoms of encephalitis lethargica are hardly likely to be confused with neurasthenia. The dulling and immobility of mind of the encephalitic might be confused with a neurasthenic or depressive individual, but the mask-like rigidity and salivation so common in the former are quite uncharacteristic of the latter. The dull immobility of the praecox syndrome, possibly associated with salivation, is far more likely to be confused with the effects of the encephalitis lethargica, and confusion has at times arisen. The encephalitic, however inhibited, is more fully alert, given time to express himself, while the praecox is actually confused, and no amount of time allowed for expression will improve matters. The mannerisms and wrinkled forehead of the praecox are usually distinctly different from the mask-like face of the encephalitic. Mentally, variability and inconsequence, or perhaps stupor, will contrast with the slow, inhibited endeavour to find expression. This slow,