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FEAR AND ATAXIA

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Movements are coördinate which are accomplished with appropriate muscular effort, harmonious rhythm, regular speed, exactness in direction and accuracy in extent. To subserve the coördination of movement a complex neuro-muscular mechanism exists. This coördinating mechanism is almost coëxtensive with the nervous system. It is thus widely exposed to injury. Lesions of any part of the mechanism may temporarily or permanently derange the function of coördination and induce a state of local or general incoördination or ataxia. Hence, ataxia or incoördination complicates many nervous diseases. The incoördinating effect of a lesion depends upon its site, extent, and rapidity of development. In the disease with which ataxia or incoördination is particularly identified in popular medicine, locomotor ataxia, the vestibular, cerebellar, spinal and peripheral components of the coördinating mechanism all may be affected. The main stress falls habitually upon the spinal and peripheral components.

The initial stage of locomotor ataxia or tabes dorsalis is distinguished essentially by the absence of ataxia. Evidence of profound structural change in the regions characteristically affected

by locomotor ataxia may be present in this initial stage of outwardly perfect coördination.

Typically the onset of the ataxia is insidious; the patient notices he no longer feels the ground as before; his feet seem numb, it is as if he walked on felt; he negotiates stairs with increasing awkwardness; he has difficulty in walking in the dark; he falls into his washbasin if he closes his eyes when washing; or other indications of commencing uncertainty in movement alarm him. The condition gradually advances and manifestations at first intermittent become more nearly continuous until he can hardly take any action without some consciousness of uncertainty. Not rarely there occurs a sudden exacerbation, which the tabetic attributes to a definite incident, such as a chill, a fall, a fright, or overwork.

The ataxia may first appear in a limb where sensory loss is evident, and the increase of the ataxia may be roughly parallel to that of the sensory loss. But the severity of the ataxia does not always correspond with the severity of the sensory alterations; Frenkel and others allege that ataxia may occur without sensory loss; we may have sensory changes without ataxia (*e. g.*, in the preataxic stage). The preataxic differs from the subsequent ataxic stage only by that which the name implies, the absence of ataxia in the first and its presence in the second.

Increase of the lesions alone is often inadequate to explain the ataxia, for the ataxia sometimes begins and progresses without any apparent change having occurred in the organic lesions, and considerable sensory changes sometimes arise without any corresponding increase of incoördination.

After ataxia has developed, a return to the preataxic stage may be obtained by suitable exercise therapy. This desirable result has not been invariable nor even customary but it has been reported and I have observed several such successes. And although complete success is rare and only to be obtained in those whose ataxia either is not marked or is of recent appearance, a considerable improvement in the coördination almost inevitably results from adequate educational exercises. The exercises do not in any discernible way affect the existing structural change; they do not diminish lesions; indeed they may have no detectable objective influence other than the relief of the ataxia.

The ataxic stage may as the result of the occurrence of blind-

ness retrogress and the preataxic stage be again established. In a recent paper in which incontrovertible cases of this complete retrogression were enumerated, and other evidence was given of the benign influence of blindness upon ataxia, I suggested the following law to define the relation of blindness to ataxia:—Of two tabetics one blind, the other seeing, at any stage of the disease, other things being equal, the blind is the more liable to persist coördinate, the less liable to become ataxic.

The ataxia varies in degree according to moods and emotions: it is greater in fatigue than in freshness, in despondency than in cheerfulness, in terror than in calm and in pain than in comfort. These oscillations in the ataxia last just as long as the mental state which causes them. As the fatigue or the despondency or the fear or the pain diminishes the ataxia decreases.

As ataxia may be absent in presence of tabetic structural changes; as an increase in the extent of these structural changes is not necessarily or invariably followed by an increase in the ataxia; as without evident alteration in the structural lesions ataxia may advance—as I shall presently show; or may retrogress owing to the influence of education or blindness; and as transient mental states without detectably affecting the structural changes may markedly influence the ataxia, the ataxia is not wholly dependent upon the tabetic structural changes. It is indubitably a consequence of the tabetic lesions but neither an invariable nor a constant nor an indispensable consequence. It is a consequence which to a certain extent is indirect, and to that extent probably avoidable and certainly remediable.

The function of coördination as it is habitually exercised is a reflex and an automatic function. We do not consciously determine the relaxation of the antagonists; or the order, the strength and the relative rate of the contractions in the several groups of muscles which may be involved in a single movement.

A reflex does not alter from moment to moment. Its constancy and infallibility are among its chief characteristics. Neither does the effectivity of a group of reflexes vary without cause. Hence, to explain the variations in ataxia which result from education, mood and blindness we must postulate the existence in our coördinating power of some other potential component besides the reflex structural mechanism which has been established by physiologists and which by some, such as Egger, is held to be

the sole mechanism. The influence of this other component may vary from hour to hour according to the mood, environment, comfort, and physical state of the patient. This influence may be enhanced by educational exercises; and is greater in the blind than in the seeing.

The only influence which is capable of such variation, is subject to mood, to environmental and to physical conditions and is greater in the blind than in the seeing, and in the trained than in the untrained, is a psychic influence. The coördinating power must have a psychic component. The function of the automatic reflex coördinating mechanism must be capable of being enhanced or diminished by psychic power.

The value of any factor in a symptom is almost impossible to estimate unless the influence of that factor can be isolated. The onset of ataxia is usually too gradual to permit us to define the influence of the psychic component. I thought that by taking cases of atypical onset of ataxia and by ascertaining to which factor the atypical onset was due, I could more clearly establish the determinants of ataxia. The particular cases I shall discuss here are instances in which the ataxia began not insidiously as is its wont, but suddenly, at or very soon after the occurrence to which the ataxia was attributed. The ataxia in these cases began either violently or, if mildly, increased with unusual rapidity.

I have seen three cases in which tabetics unhesitatingly attributed their ataxia to a minor accident. Physicians to whose attention I have brought this subject have so frequently been able to furnish similar examples from their personal practice that I am certain this mode of onset of ataxia definitely exists.

These accidents were so slight that they could not possibly, under normal circumstances, have affected motor stability. No bones were broken. The greatest lesion of continuity was a skin abrasion. So the effect was altogether disproportionate to the physical injury, was altogether inexplicable as the result of the physical injury alone; was, indeed, greater than could be produced by any physical injury.

The minor accident was a slip on a greasy pavement, a fall down a few steps, a stumble over a small obstacle. I have under my care at present a gentleman who is suing a Traction Company because his ataxia began almost immediately after he fell down a subway stair. Such traumatic ataxia is common in the courts.

Not rarely the litigants and experts confound the onset of ataxia with the onset of the tabes.

The ataxia in the three appeared suddenly, at a definite ascertainable moment. This moment may be suspected in a case such as the patient who, at present, is suing the Traction Company. I have not been able to verify or to disprove his statement; but in the other cases I have got such corroborative evidence from relatives and friends as removed my doubt that ataxia did develop suddenly in these patients; that there was a moment which we may call the "*ataxic moment*" before which the movements were apparently normal and after which they were more or less chaotic, and incoördinate.

What was the relation of the accident to the ataxia? Ataxia we have already shown begins typically as a slight uncertainty of movement and increases by imperceptible stages. Here ataxia appeared synchronously with or immediately after the accident in each case. The ataxia not only appeared suddenly but in one case was of acute degree from the onset and in the others developed with fulminating rapidity. In the absence of any ascertainable cause except the accident we must attribute to the accident both the atypical onset and the bizarre degree of the ataxia. Had there been no accident, the ataxia would not have appeared when it did, but would have later developed with its usual insidiousness.

In those cases I found on first examination, in one, absence of one knee jerk, in two, of both; in two, complete, in one incomplete Argyll-Robertson pupils; in all three some hypotonia and some sensory loss;—typical signs of syphilis and locomotor ataxia.

We know from the work of Minor, of Moscow, and others, that slight lacerations of the lymph and vascular capillaries of the cord may occur after severe accidents to the spinal column. But such changes are not confined to the root zones and to the dorsal columns. Such changes cannot produce only the signs which were discovered in these tabetics. And such changes occur after severe accidents, not after the ludicrously trivial occurrences which were in these three cases dignified by memory as accidents mainly because the ataxia then appeared.

The trivial accident could not possibly have caused structural changes in the nervous system. A widespread, latent, unsuspected neuro-muscular defect of a tabetic nature must have existed before the accident.

Before the inciting injury, the nervous lesions, whatever their incoordinating tendency, were yet inadequate to produce ataxia; any potentiality for ataxia they possessed *was* latent and *remained* latent until the accident in which ataxia was made manifest.

The slight accidents which established ataxia were trips, stumbles, and falls. In those cases in which trivial injuries produced sudden ataxia, the mechanism which was directly affected was that of progression.

As these accidents were too trivial to produce organic lesions, and as their effect far exceeded that of severe mutilation, the petty mishap, the momentary interference with progression, must have profoundly influenced the psychic component of coördination in these tabetics to cause the onset of ataxia. The mishap acted as an electric spark acts in an explosive gaseous mixture; it produced a psychic explosion which precipitated the patient from coördinate movement into ataxia. The psychic component of coördination before or at the moment of the mishap must have been on the verge of disorganization.

If the fall be the dominant factor, then other things being equal, those tabetics most liable to fall must be the most ataxic. Those tabetics most liable to fall are the blind. They, in their blindness, stumble more than any preataxic tabetic. Yet ataxics, when they become blind, may become less ataxic; and blind tabetics are less prone to develop ataxia than seeing tabetics. Hence, we must conclude that the nature of the injury is not the dominant factor. The importance of the injury may partly lie in its nature (an interference with progression) but it mainly lies in its power to reinforce the incoordinating tendency of the mental state which at the moment of the accident exists. The effect of the mishap in these cases is not unlike that of a foreign body dropped into an over-saturated solution; the ataxia is initiated by the mishap just as crystallization is by the foreign body.

An elderly gentleman formerly under my charge in hospital, three years ago decided, not without misgiving, to trust himself for the first time in his life for a short holiday among the perils of the Continent. He was visiting a cave somewhere in the Low Countries. A steep staircase led into this cave, which was lit by electricity. The patient told me he did not like the escapade at all: to go under the earth alone with foreigners seemed to him in the highest degree hazardous. Suddenly all the lights went out.

The darkness was absolute. His worse fears seemed about to be realized. He stretched out a panic-stricken foot for the next step, and felt—nothing. Then he collapsed, was gently carried out of the cave, and tended in the local hospital. He had completely lost his power of walking. When I saw him two weeks later, the “paralysis” which had struck him, was an incapacitating ataxia and nothing more. He had marked ataxia, no knee jerks, Argyll-Robertson pupils, and other classical signs of tabes.

Another, whose case I have twice demonstrated at the Post Graduate Medical School, who was sent to me by Dr. Vincent Williams, and who was formerly a medical student, told me that he consulted a doctor for pain in the stomach, accompanied by vomiting, having no relation to food, and occurring in inexplicable spells. He swallowed many drugs during many days. He then, six years ago, had transient double vision. Next, about four years ago, sudden pains in his legs appeared which another doctor called rheumatic. He visited a third doctor a year later who, for the first time, associated the word “shooting” with the pain. There arose in the patient’s memory a medical tag twenty years old, and he asked if “shooting pains” were not typical of locomotor ataxia. The doctor agreed that they were. “Have I locomotor ataxia?” “I don’t know, I am no nerve specialist.” The patient who had entered walking well, now greatly perturbed, with much difficulty reeled home. Two weeks later he could not walk without the aid of a stick. His ataxia thereafter swiftly incapacitated him wholly.

From literature and from report I could multiply these examples considerably but I think that what they teach is so obvious that further instances are unnecessary for the purpose of this paper.

The tourist volunteered that he was much perturbed from the moment he began to descend into the cave. Indeed, he was led to take his trip to Europe because he felt completely “run down” and nervous. The whilom medical student stated that he had steadily grown more worried as his illness progressed. He thought that locomotor ataxia was associated with shooting pains, that it affected walking and that it was incurable. He was literally staggered by the suspicion that he had this much-dreaded disease. His power to walk perfectly had survived unimpaired through five years of his malady. A correct diagnosis was more pernicious in its immediate effects than five years of mistaken therapy.

Why should the slight psychic influences of a fall, or a fright, prove efficacious in precipitating ataxia? The tabetic fears a thousand times a day. Before the ataxic moment, when the incriminated shock was experienced, many such doubtless occurred, but none was followed by ataxia.

Here, we must again recognize the great importance of the psychic state at the moment of the injury. *Upon the mental state at the moment of the injury depends the degree of the shock and the power of the shock to induce ataxia.*

The sole aspect of the mental state which is of importance to the result, so far as we are at present concerned, is that which is vaguely termed cerebral control. We may imagine every brain at any given moment to possess a certain definite degree of this power to control its experiences so far as such experiences affect the ordinary cerebral functions. This control as regards any given function must depend upon the degree to which that function is established. The degree to which the function is established must vary according to the biological capacity of the individual brain, the nutritional influences exerted upon the brain, and the amount of education in the function which the brain has received. Toxic influences, such as tobacco and alcohol, and disease processes, such as engender syphilitic neurasthenia and general paralysis, will affect the control of the function either in a general or in a special way according as the function is a generalized or a localized brain function and according as the pernicious influences act generally or locally.

The loss of control due to actual cerebral disease tends to increase so long as the action of the disease-producing agent persists.

Tabes progresses in pain and disability which maintain the patient in a constant state of psychic stress in all his relations with the external world. There tends thus to be a progressive loss of cerebral control as the mental strain of enduring crises, pains, ataxia, etc., increases. The cerebral control tends constantly to vary throughout the course of the disease, according as the daily strain of living with tabes temporarily increases or diminishes.

The effectiveness of the actual cerebral control in tabes tends constantly to diminish. At a given moment it is equal to the biological cerebral control increased by the amount of control derived

from education, and diminished by adverse nutritional influences, by cerebral disease processes and by the stress of the tabes.

In using the term cerebral control I mean not to imply that the control is consciously exerted. It may be. But the lessening of ataxia during favorable mental states such as freshness, cheerfulness, and calmness and the increase of ataxia during adverse mental states are merely phenomena of the mental states.

Nor do I mean the term to imply the mode of action of the cerebral influence. Cerebral influence may possibly be exerted in a positive manner upon the sensitivity of the coördinating mechanism; improvement resulting from psychic causes may actually represent enhancement of the reflex coördinating mechanism. But the cerebral influence may possibly be exerted in a negative manner; cerebral influence may diminish reflex coördinating power and improvement may be due to a freeing of the automatic coördinating centers from disconcerting cerebral action; or there may be different kinds of cerebral influence, some diminishing and others increasing coördination.

To avoid misconception we shall group the cerebral with the automatic or reflex coördinating influences and discuss them together as coördinating power. In tabes dorsalis certain characteristic structural morbid changes in the peripheral, spinal, cerebellar and vestibular elements of the reflex automatic mechanism occur; and changes in the psychic component of coördinating power also arise. We may regard these changes, in so far as they diminish coördination, as a constellation of ataxia complexes, among which we may distinguish two essential groups, a physical and a psychic.

If the effectivity of the ataxia complexes exceeds the coördinating power we get ataxia. The measure of the liability to ataxia at any moment, what we may call the coefficient of ataxia, is the ratio of the ataxic complexes to the actual coördinating power at the moment.

$$\text{Coefficient of ataxia} = \frac{\text{Ataxic complexes}}{\text{Actual coördinating power}}.$$

In the cases we have just cited the onset of ataxia, the coefficient ataxia was at the ataxic moment suddenly, by accident or shock, increased above unity. In those in which a slight accident, a slight fall, precipitated the ataxia, we may conclude that immediately prior to the fall their coefficient of ataxia was unus-

pectedly near unity, so near that the slight increase in the psychic group of the ataxic complexes resulting from the accident, or shock, was sufficient to raise the coefficient above unity. Otherwise ataxia could not have resulted. In all previous accidents or shocks which these patients sustained, the coefficient of ataxia was safely below the *ataxic threshold*, so that the increase of the ratio which then resulted was inadequate to exert a visible effect upon coördination. So long as the denominator of this formula is greater than the numerator, adequate repression of the ataxic tendency must prevail.

In addition to the incoördinating influence of fear in tabes another effect on movement is often seen, a paralyzing effect. Some tabetics when seated dread to stand, when standing dread to walk, when walking are disturbed to a ridiculous extent by trivial obstacles. One whom I demonstrated in June at the Post Graduate Medical School, and who is still a patient there, could walk excellently in my office even without a stick, yet he began his effort before the class by insisting punctiliously upon the arrangement of his surroundings—moving a chair and lifting a piece of paper from the floor,—by adjusting his garments, by rubbing his moist hands, and by screwing himself up for a great endeavor. After two or three reeling steps he collapsed in a heap opposite an imperceptible crack on the floor. Supported by my little finger, which he rigidly clutched, he circled freely round the amphitheatre. Asked to stand with his eyes shut he swayed and fell, or collapsed without swaying. This patient told me that in these circumstances he could not remember how to move so that when I urged him to walk he could only collapse upon the floor. His disability vanished the moment he was reassured even by the slight support of my little finger, an aid altogether disproportionate to its effect.

It is perhaps not out of place here to refer to the analogous states which sometimes arise in sleep. With the limited misty consciousness peculiar to disturbed slumber, sometimes an inability to move associated with intense fear arises. The sleeper knows the cause of the fear and realizes that if he can only move even a toe, the fear will vanish; but the struggle to attain that movement is none the less frightful. This tabetic said he knew his fears were groundless but none the less he could not move except with aid. With the slightest aid his fear vanished just as the terror does when the sleeper can move.

In another form of *pavor nocturnus* a feeling of falling down precipices into bottomless pits arises. An analogous state occurred in this man when he attempted to move unaided and in public. With the slightest aid he could move freely, so a faulty reflex mechanism was not the root of his trouble; nor was faulty association of kinesthetic images to blame. The trouble seemed to be in a higher sphere, perhaps in the sphere where perception of posture lies, the sphere which has been shown by Head and Holmes to be situated probably in the cerebral cortex.

The ataxia in this person was preceded by nervousness, irritability and sleeplessness for fourteen months; during that time he was seen by several physicians and correctly deemed by them to be suffering from syphilitic neurasthenia.

An interesting corollary to this case is another at present under my care at the Neurological Hospital, Blackwell's Island. He refuses to walk and when coerced shuffles slowly and extremely cautiously along supporting himself on both sides and collapsing or threatening to collapse at frequent intervals. He is a taboparetic, blind in one eye, with laryngeal crises and he has been ill only since June. He has slight but evident ataxia.

In both of these it is not the coördinating power alone which is affected but the whole psychic mechanism of progression; and with this affection is a certain degree of general mental deterioration.

Besides these extravagant effects lesser but distinct motor results of fear are evident in the great majority of tabetics. All tabetics tend to be more ataxic in public; at the change of surface and of level at street crossings; in traffic, the open, and the dark; and under the stress of emotions and the influence of depressing weather conditions. No matter how carefully reëducated by Frenkel's method a tabetic may be he can only exceptionally be induced to discard his stick. And the attitude of the ataxic—his inappropriate wide base, his curious mixture of exaggerated and restricted movement, his rigid maintenance of the ill-judged muscular contractions which he thinks are indispensable to balancing,—is, in part, an expression of the fear and tension in which he lives.

The effect of fear upon tabetic symptoms cannot be overestimated. We saw from the last two cases that the psychic mechanism of progression may be affected in its entirety by fear. We

saw from the cases of the tourist and the former medical student, that upon the component of this mechanism which specially subserves coördination, the result of fear may mainly fall. We further saw from the cases in which slight accidents affecting the mechanism of progression caused ataxia that the state of this mechanism of progression at the moment of the accident determined the result.

Head and Holmes suggested that detection of change of posture is effected by discrimination between successive postural schemata which are elaborated from muscular sense and cognate impressions.

We know that an isolated movement cannot occur; that every movement of a limb is but a maximum regional expression of a general muscular change. I think we are therefore not unjustified in assuming that by the postural schemata or rather by the unconscious immediate precursors of these schemata all movements are controlled. If the schemata be imperfect, movement is imperfect. This imperfection of movement may be localized to the limb from which the fault in the schemata arises, *e. g.*, ataxia occurring in a single limb. Sensory impressions from the whole musculature tend to modify, to correct, and to mitigate pathological impressions arising from any one region so that the schemata may retain an efficient degree of functional activity in spite of local morbid changes. The unconscious precursor of the schema and its motor expression together constitute the psychic mechanism of progression. Influences which diminish cerebral activity, such as fear and depressed mental states, affect the whole mechanism of progression, lessen the functional activity of the whole mechanism, and thus accentuate the weakness of any enfeebled component of it. Hence, ataxia is more manifest in tabetics under such adverse conditions. Similarly, influences which increase cerebral activity enhance the functional power of the whole mechanism and tend to mask the ataxia.

I have shown that tabetics are ataxic in part at least because of preëxisting affective states.

From the infallibility with which emotion such as fear, anger, grief, etc., engenders an increase in the ataxia of an ataxic tabetic, one is tempted to regard *ataxia in them as equivalent to an emotional expression.*

Tabes is a disease associated with lightning pains, neuralgias,

crises, bladder trouble, impotence, perhaps diplopia, incipient blindness, commencing deafness; and, above all, ataxia. This group of symptoms has a tremendous influence in establishing and perpetuating a state of anxiety, of mental stress, of fear. The many perils to which the ataxic is exposed tend steadily to become more and more harrassing. Fear complexes form, which, as they become elaborated, tend more and more immediately to succeed one another and to produce an unrelieved state of perturbation.

Two elements, therefore, participate in the ordinary progress of ataxia, the increase in the fear complexes which result from the tabetic symptoms, and the advance of the incoördinating lesions. When the ataxia advances markedly at certain definite periods synchronously with bouts of pain, vomiting, or, other malign influences, it is the psychic not the physical ataxic complex which is increased.

Daily variations in the degree of ataxia due to transient changes in fatigue, depression, nourishment, etc., are to be attributed to oscillation in the effectivity of this psychic complex.

The influence of the morbid mental state, which is directly due to the existence of the tabes, is, therefore, one of the most profound in diminishing the coördinating power.

James states that "the bodily changes follow directly the perception of the exciting fact and that our feeling of the same changes as they occur is the emotion." Tabetics fear because they are ataxic. And tabetics are ataxic because they fear.

The mental mechanism of progression I hope later to treat more fully. Here my essential purposes have been to emphasize the importance of psychic factors in producing coördination and among these factors, *particularly to emphasize the influence of fear upon ataxia*. Frenkel and others have rendered invaluable service to therapy by teaching us the power of educational exercises to relieve ataxia. In a recent paper I established the beneficent influence of blindness upon ataxia. In my next article I will describe a method of curing ataxia which is based upon the knowledge of the effects of educational exercises, blindness and fear upon ataxia; a method which I have found of great service in curing ataxia; and which I hope in the hands of others will prove equally efficacious.