Short Notes and Clinical Cases

'CROWDING' OF EXCITATION AS THE IMMEDIATE CAUSE OF SOME EPILEPTIC FITS: A CONSIDERATION OF THE PATHOGENESIS OF EPILEPTIC FITS PRECIPITATED BY ANGER AND OF THOSE OCCURRING IN SITUATIONS OF DANGER

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PAVLOV, working with dogs, has produced evidence tending to show that the cerebral cortex may be represented as a mosaic of constantly shifting areas of excitation and areas of inhibition, each opposing the irradiation of the other. Applying his observations to man, I have tried to show that the interplay of excitation and inhibition sometimes leads to phenomena interpretable as manifestations of the 'crowding' of either of these functional states into particular cerebral areas. A few examples will show what I mean.

'Crowding' of Inhibition.—In a recent paper¹ I have referred (p. 9) to five cases of narcolopsy, reported respectively by Kinnier Wilson, Cave and Rosenthal, in which the attempt to ward off an impending sleep attack sometimes led to an attack of powerlessness. (Later I came across the same observation in Case I of Wenderowic.⁷) This phenomenon may be understood in the light of Pavlov's view that sleep (an inhibitory state) results from the irradiation of inhibition over a sufficiently large part of the cortex. When a person is falling asleep, we may infer that inhibitory processes are irradiating over his substrate of consciousness. When he tries to resist sleep, a multitude of excitatory processes come into play, opposing the spread of the inhibitory processes. The latter, deflected, so to speak, from their original goal, will invade those areas where they meet with the least opposition. Thus, in the cases cited, they were evidently deflected from the substrate of consciousness to the motility substrate. This deflection reminds one of what happens in a football game, when the player rushing the ball is crowded by his opponents, as they advance upon him, into that part of the field where he sees the least opposition.

'Crowding' of Excitation.—From a number of examples cited in a recent paper³ the following is chosen. MacCurdy has given an account of the

reaction of soldiers to their first bombardment. After remarking that the commonest reaction is fear, he said ⁵ (p. 17): ‘A less common reaction is that of excitement, accompanied even with a kind of spurious elation. The man has a tendency to make facetious remarks about the shells, to laugh at feeble witticisms, and very often feels under considerable *motor tension, there being a pressing desire to do something, to do it immediately and do it hard*’ (italics mine). I have suggested ³ (p. 346) that this motor tension may be regarded as a manifestation of crowding of excitation. The most basic (not the strongest) wish of the soldier is to run away to a spot where there are no shells. Let us, for the sake of convenience, speak of the nervous mechanism utilized in running away as the ‘running substrate.’ We may assume that when a soldier has the impulse to run away, there are, in the highest levels of his ‘running substrate,’ excitations which, figuratively speaking, clamour for expression.* Since he refrains from running away, we may assume that these excitations are opposed by stronger inhibitions. The excitations cannot vanish into nothingness; they are merely crowded away from the ‘running substrate’ into other parts of the motility substrate. Their persisting in the motility substrate may be inferred from the fact that the soldier is aware of motor tension and of ‘a pressing desire to do something, to do it immediately and do it hard.’

Whenever an impulse to action is suppressed, one may reasonably suppose that excitation is crowded away from the substrate of the act in question and is diverted into the remaining part of the motility substrate; there is, in consequence, apt to be an increase in the ‘tension’ † of this remaining part. When the suppressed impulse is strong, the increase in tension in the remainder of the motility substrate may be great. I now submit the question: Supposing a ‘discharging lesion’ to exist in some part of the motility substrate, may this increase in tension precipitate an epileptic fit?

This question may, I think, be answered affirmatively. In illustration I refer to those cases in which epileptic fits are precipitated by anger. In considering the pathogenesis of fits thus precipitated, it must be remembered that anger is usually, if not always, accompanied by aggressive impulses aimed at the person or object which has provoked it, and (what is very important) these impulses often must be suppressed. In these cases I suggest that it is not justifiable to say with certainty that the patient’s discharging

* This assumption is in accord with Hughlings Jackson’s views on the central processes correlative with movement. In a recent paper ⁴ I have presented those of his views which bear on the present discussion.

† ‘Tension’ is here used figuratively. In its literal sense, tension describes a state of muscular, not nervous, tissue. It should be emphasized that the suppression of an impulse does not always lead to an increase in the tension of the motility substrate. The excitation concomitant with the impulse is opposed by inhibition; the motility substrate, if unduly inhibitable, may succumb to this inhibition, the result being a cataplectic attack—a state in which there is *absence* of tension in the motility substrate.

lesion is set off by anger. It is possible that the decisive factor is not anger, but the suppression of powerful aggressive impulses. Concomitant with this suppression, excitation is crowded away from the substrate of a particular act (the aggressive act which would have appeased the angry man); there may be, in consequence, a 'piling up' of excitation in the remaining part of the motility substrate, and an increase in tension in this remaining part; it may be this increase in tension that causes the discharging lesion to go off.

An instructive illustration of the cases under discussion was reported by Marsh.⁶ The patient, a soldier, had an epileptic fit (it happened to be his first) under circumstances about to be described. He was 'an excellent soldier of the infantry, an Armenian with our army, who had difficulties with the first sergeant of his company. The sergeant, of German parentage, possessed many racial characteristics. Although a fine specimen of physical manhood and an excellent American soldier, his very appearance was odious to the Armenian, who had lost his wife and children in the massacres of his native land. Because of these differences, the Armenian was detailed to the officers' mess for duty, where he would be less apt to come in contact with the first sergeant. Some time later, however, their quarrel was renewed, when the first sergeant, in language which betrayed not a long separation from that of the Fatherland, proceeded to give the Armenian a lecture on proper respect to the first sergeant of the company. Angered to the breaking-point, the Armenian would have crushed him had he dared to violate the laws of military discipline; but, inhibited, he faced his aggressor until in emotional exhaustion he fell unconscious, and the emotion went on to an abnormal expression in a typical grand mal attack.'

We now pass to another type of case, different in detail but similar in principle to the foregoing. I refer to those cases in which epileptic attacks occur in dangerous situations. I suggest that the occurrence of a fit in a dangerous situation is probably not always a mere coincidence; conceivably it may sometimes result from suppression of the impulse to escape the danger. To take an example, let us consider the case of an epileptic who has a fit while working on a scaffold. Knowing of his malady, he cannot but feel uneasy while on the scaffold, and must have a strong impulse to return to safety. Suppressing this impulse means that there is crowding of excitation from certain motor areas, a 'piling up' of excitation and an increase in 'tension' in the remainder of the motility substrate, which in turn may cause the discharging lesion to go off.

Generalizing from these and similar instances, I suggest that when an epileptic fit supervenes on the suppression of a strong impulse, we may hypothesize the following sequence of events as a possible explanation of the fit: (1) Excitation is crowded away from the substrate of the act the impulse to which is being suppressed; (2) there is a 'piling up' of excitation, hence an increase in 'tension' in the remainder of the motility substrate; (3) as a direct result of this increase in tension, the discharging lesion goes off.

I do not believe that crowding of excitation can *of itself* account for a fit. I adhere to Hughlings Jackson’s view that without a discharging lesion there can be no epilepsy.

The cases of epilepsy discussed in this article may be contrasted with two groups of cases. (1) They may be contrasted with those cases of narcolepsy alluded to in the second paragraph of this article, in which the attempt to ward off an impending sleep attack led to an attack of powerlessness. Viewing these cases of narcolepsy, we may say they are cases in which there is undue *inhibitability* of parts of the brain, and in which some of these parts succumb to *inhibition* in consequence of crowding of *inhibition*. By contrast, viewing the cases of epilepsy, we may say they are cases in which there is undue *excitability* or dischargeability of a part of the brain (a discharging lesion), and in which this part succumbs to *excitation* (producing a fit) in consequence of crowding of *excitation*. These two groups therefore are, in Jackson’s term, corresponding opposites. (2) They may be contrasted with those cases in which cataplexy supervenes on the attempt to suppress a strong impulse. Thus, while in some cases an epileptic fit occurs when the patient is made angry (there being suppression of the impulse to aggressive movement), in others these circumstances provoke an attack of cataplexy.⁴ Again, while in some cases an epileptic fit occurs when the patient is in a position of peril (there being suppression of the impulse to escape to safety), in others these circumstances provoke an attack of cataplexy. (Several such cases, reported by Hilpert, Spiller and Kinnier Wilson, are cited on p. 127 of my recent paper.²) The attempt to suppress an impulse means that there is a ‘struggle’ between excitation and inhibition. The symptoms that follow such an attempt may result either from the crowding of excitation into certain parts of the brain (as in the cases of epilepsy referred to) or from the overflow of inhibition into certain parts of the brain (as in the cases of narcolepsy referred to).

SUMMARY

Epileptic fits are sometimes provoked by anger, under circumstances necessitating the suppression of aggressive impulses, as in a case reported by Marsh, in which a fit occurred when the patient, a soldier, was goaded into impotent fury by an overbearing superior officer. In these cases it is not obligatory to ascribe the setting off of the discharging lesion to anger. It is possible that the decisive factor in causing the lesion to go off is not anger but the suppression of the aggressive impulses which accompany anger. During the effort to suppress these aggressive impulses there is ‘crowding’ of excitation from the substrate of certain aggressive movements into the remaining part of the motility substrate; this may lead to a ‘piling up’ of excitation, and consequently to an increase in ‘tension’ in this remaining part; this in turn, it seems possible, may cause the discharging lesion to go off.

The same conclusion applies to cases in which epileptic fits occur in

dangerous situations. Granting the possibility that some of these cases may represent a mere coincidence, we may, speaking of the remaining cases, say that the patient is aware of his danger, has an impulse to escape to safety, and (for any reason whatever) suppresses this impulse. There follows the sequence of events described in the preceding paragraph.

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