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CONSIDERATION OF THE CLASSIFICATION OF RECURRENT CONVULSIONS

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It seems odd, in this day and generation, that the question should be raised whether recurrent convulsions, usually called epilepsy, form a disease entity or are only a symptom of some underlying pathological state. The older writers, and most modern ones, believe that these convulsions form an independent disease, and any one daring to disagree with this conclusion may draw down the fire of the gods upon his head. It is not the purpose of this paper to argue the subject exhaustively, but simply to suggest a classification of recurrent convulsions into groups according to certain etiological factors and to mention the salient features of two of these groups which have especially attracted my attention.

If we accept as a disease entity practically all cases of recurrent convulsions and term them epilepsy, we, to a large extent, halt investigation, and if we treat them with sedatives only we render ourselves liable to sink deeper and deeper into the mire of therapeutic despair. Hence, while the introduction of substances like luminal may be considered a boon of the day they may, by inhibiting clinical investigation and scientific research, prove a curse of the tomorrow.

HEREDITY

Let us briefly consider the factor of heredity in epilepsy. In the study of 4,146 cases of epilepsy by Gowers,[1] Spratling[2] and Aldren Turner,[3] the hereditary factors of epilepsy, insanity, alcoholism and other nervous disorders were found to be, in Gowers 40 per cent, Spratling 56 per cent, Turner 51 per cent, or an average, let us say, of 50 per cent.

Turner, in the study of 676 cases, considered epilepsy alone as an hereditary factor in 37.2 per cent, but he included brothers and sisters. If we deduct the cases of epilepsy in brothers and sisters, because we should hardly consider them in an hereditary sense, we find epilepsy in the ancestry of these cases, including grandparents, parents and parents' collaterals, in only 26.6 per cent. Therefore, if nearly 75 per cent of epileptics are free from epileptic heredity, and if 50 per cent are free from any neuropathic heredity at all, and if we consider how many of the apparently normal individuals we meet in a day's journey have alcoholism, epilepsy, insanity and other neuropathic diseases in their ancestry, we can not be very deeply impressed with the frequency of an hereditary influence in epilepsy.

We shall have, of course, to admit that there is such a thing as hereditary epilepsy when defective brain conformation is transmitted from ancestor to descendant, but we submit that these cases, and no other cases, may be truly termed epilepsy.

CONVULSIONS

Let us now consider one aspect of convulsions. As is well known, convulsions, which in themselves are in no way distinguishable from those occurring in so-called idiopathic epilepsy, appear in such states as eclampsia, uremia, apoplexy, meningitis, cerebral injury, cerebral tumor or abscess, syphilis of the central nervous system, cerebral arteriosclerosis, encephalitis and, at times, in acute febrile conditions. Whether they occur singly, in series or are recurrent, if the cause is patent, we do not think of calling the patient epileptic. Still other convulsions are connected with hyperthyroidism, certain gastro-intestinal disturbances and obvious brain trauma which we are glad also not to term epilepsy. If these convulsions, single or recurrent, are known and acknowledged to be a symptom of some definite condition, why can we not consider all convulsions symptomatic? There is one exception to this view, the influence of which is hard to estimate: it seems reasonable to believe that repeated convulsive seizures may, and do, cause cerebral cortical cell defect of themselves until what

has been styled a convulsive habit is established.

CLASSIFICATION

It is important, therefore, that we should, by careful search, endeavor to find the basic pathological cause for every case usually termed epilepsy and so classify the individual case that, if possible, it may not be stigmatized as epileptic. It is also necessary that therapeutic measures should be directed toward the underlying condition rather than that we should administer sedatives to suppress the symptom. Most of the recurrent convulsions, ordinarily termed epilepsy, may be classified into the following groups:

1. *Hereditary Group.*—Feeble-minded heredity is believed to be the most important, with the transmission of cortical defect to the descendant. Appearance of convulsions usually from three to ten years of age.

2. *Chronic Toxicosis or Infectious Group.*—Rare. Toxic hyperthyroidism is an example. Appearance of convulsions at any age.

3. *Birth Trauma Group.*—Skull or brain injury may be obscure or manifest. Appearance of convulsions usually first three years of life.

4. *Jacksonian Group.*—Due to post-birth cerebral trauma. Appearance of convulsions at any age.

5. *Intracranial Growth Group.*—Due to slow growing neoplasms which are usually unrecognized until late. Sometimes revealed by radiographic evidence of pressure on the skull, especially local evidence. Appearance of convulsions at any age.

6. *Hemorrhagic Group.*—Rare. Due to small unrecognized hemorrhage or repeated small hemorrhages in the cortex. Usually evidence of atheromatous degeneration elsewhere. Appearance of convulsions usually during adult life.

7. *Cerebral Lues Group.*—a. Congenital. Appearance of convulsions during childhood.

b. Acquired. Appearance of convulsions during adult life.

8. *Cerebral Arteriosclerotic Group.*—Appearance of convulsions usually after middle age.

9. *Hypopituitary Group.* — Appearance of convulsions during adolescence.

10. *Variable Vascular Tension Group.*—Low grade or latent nephritis can usually be found. Appearance of convulsions during middle age.

It is thus seen that, according to this classification, the age of onset of the convulsions is of considerable importance. It is to the brief consideration of the last two groups of this classification that your indulgence is asked.

RECURRENT CONVULSIONS DUE TO HYPOPITUITARISM

Convulsions in connection with pituitary lesions were described by Cushing previous to a report I made of a few cases in 1914. In this and subsequent studies it was found that convulsions may occur which are believed to be due to certain states of under-secretion of the pituitary gland. These cases may be either static, the convulsions appearing in individuals who were chronically hypopituitary before puberty,* or transitional, occurring in those who do not show previous evidence of hypopituitarism, but who, during adolescence, have a sudden let down in pituitary secretion due, frequently, to illness or injury. To make the diagnosis of hypopituitary recurrent convulsions three essentials must be borne in mind: First, the convulsions must make their appearance during the period of adolescence. Second, the case must present clinical evidence of hypopituitarism. Third, radiographic evidence of sella turcica change consisting of a small, or encorached upon sella showing approximating or bridging clinoid processes, or massive posterior clinoid processes, or roughened or irregular dorsum sellae, must be present.

Analyzing these cases we find that in nine of them no attacks have occurred for three years or more. Cases 2, 6, 8 and 12 have been taken off of all medication and the attacks have not returned. Two other cases seemed definitely controlled by pituitary feeding, four cases were improved to some extent, one case was not improved, but had a bad family history, one case was lost track of.

*It seems that convulsions are more apt to occur in the mild and moderate cases rather than in the very pronounced cases, i. e., those termed adiposis genitalis congenita.

TABLE OF STATIC OR CHRONIC HYPOPITUITARY CONVULSIONS. (COMPARISON OF RESULTS IN 1919 AND 1921.)

Case	Average Frequency of Grand Mal	Result of Pituitary Feeding 1919	Remarks Made in 1919	State of Cases in 1921	Remarks 1921
1	1 a month	2 in 18 months	Marked improvement	No attacks for over 3 years	Recovered
2	1 a month	None in 3 years	Cured	No attacks for 5 years	Recovered
3	1 every 6 weeks	None in 14 months	Attacks ceased	Went 3½ years without an attack; then took medicine irregularly. Now has occasional attack.	Apparently controlled with pituitary extract
4	1 every 2 months	1 every 2 months	Not improved	Took anterior lobe irregularly; then whole gland regularly and no attacks for 3 years.	Recovered
5	1 a month	3 in 18 months	Marked improvement	1 or 2 attacks a year	Improved greatly
6	2 to 3 a week	None in over 3 years	Cured	No attacks for over 6 years	Recovered
7	No grand petit daily	None in over 3 years	Cured	No attacks for over 5 years	Recovered
8	No grand, petit many a day	None in over 3 years	Cured	No attacks for over 5 years	Recovered
9	3 a year	None for over a year	Prospects of cure	Several attacks a year. One epileptic sister and two feeble-minded brothers	No improvement
10	1 to 2 a week	1 a month	Improvement	1 attack a month; stopped pituitary	Some improvement
11	3 or 4 a year	None for 2 years	Prospects of cure	No attacks for over 4 years	Recovered
12	1 every 6 weeks	1 in 2 years	Prospects of cure	No attacks for over 4 years	Recovered
13	3 a year	None in over a year	Took medicine irregularly	About 3 a year. Took pituitary irregularly	Some improvement
14	1 a week	1 in 2 months	Marked improvement	1 slight attack in 2½ years. This while off pituitary temporarily	Controlled
15	1 every 6 weeks	1 every 3 months	Improvement	Lost track of entirely	
16	1 a month	None for over 2 years	Apparent cure	No attacks for over 4 years	Recovered
17	1 every 2 months	None in 6 months	Marked improvement	Attacks much less frequent; patient died.	Improved at first while on pituary, then did not

Of the eleven transitional cases reported in 1919[4] the results were not nearly so striking, but it was found that they did not take the pituitary substance as regularly. However, definite improvement occurred in eight of the eleven cases.

VARIABLE VASCULAR TENSION RECURRENT CONVULSIONS

Recurrent convulsions which make their appearance during middle life, or, at times, somewhat later, should not be classified under the general head of epilepsy. Recognizable uremia, cerebral syphilis and cerebral arteriosclerosis may be the cause. After eliminating these and other conditions there still seems to be left a small group of cases which appear to have latent cardiorenal disturbance with variable vascular tension as the underlying basis. In these cases, when the blood pressure becomes stable, even though considerably elevated, the attacks may spontaneously cease. Cases thought to belong to this group were observed in 12 of 500 cases with the general diagnosis of epilepsy.

An outline of this group is as follows: The family and birth history are negative and there is no previous history of epilepsy. During middle life or somewhat after, usually between the age of forty and sixty, the patient has a convulsion, which is repeated from time to time with irregular periodicity. The urine may, or may not, show a slight amount of albumen and a few casts. There is usually a slight increase in blood urea and urea nitrogen. Repeated blood pressure estimations reveal a blood pressure which is variable. Careful examination may demonstrate early signs of cardiovascular disease and at times focal infections may be observed, but the patient, as a rule, considers himself in good health except for the convulsions. The convulsions may be of the grand or petit mal type, or both, but the individual grand mal attacks are of somewhat longer duration than those observed in the young patient. Treatment directed toward blood pressure stabilization influences the attacks beneficially.

Of the twelve cases, seven were males and five females. The age of onset of convulsions was from forty to sixty years, eight of them being between forty and fifty. The urine showed some albumen or casts, or both, in nine of the cases. The greatest blood pressure variability noted between visits was seventy points. Three recent cases tested had moderately increased blood urea and urea nitrogen. Nine cases had grand mal attacks, two grand and petit mal attacks and only one petit attacks. The length of the individual convulsion was from five minutes to one hour, usually about fifteen minutes. The frequency of the attacks varied from three a day to six a year. As far as could be ascertained three of the cases ceased spontaneously and have not had an attack for over three years. All but one have markedly improved when the blood pressure variability became more stationary. The attacks usually extend over a period of several years. The Wassermann test was negative in all cases.

CONCLUSIONS

In conclusion it might be said that there seems to be a tendency not to consider carefully enough the etiology of so-called epilepsy; that the age of onset of the attacks has frequently a bearing on the diagnosis of the causative factor; that recurrent convulsions should be considered a symptom rather than a disease entity; that most of the cases may be placed under certain groups which form an etiological classification and that the recurrent convulsions falling into two of these groups, the hypopituitary group and the variable vascular tension group, appear to form rather definite syndromes which have not been generally recognized as such.

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