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THE PROBLEM OF JUVENILE BEHAVIOUR DISORDERS IN CHRONIC EPIDEMIC ENCEPHALITIS. *

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The manifestations of chronic epidemic encephalitis are so varied and, in individual cases, so liable to change in nature with the passage of time that it is almost impossible to put cases satisfactorily in a permanent classification. Nevertheless, cases can be classified at any one time into broad groups, each with a prominent and characteristic feature. The largest of these is undoubtedly the Parkinsonian group, but one of the most outstanding is that of behaviour disorders in children. In contrast to the knowledge only too readily gained of the clinical picture presented by these latter cases is the obscurity of the morbidity processes producing the disorders. It is the problem of the precise nature of these morbid processes with which it is proposed to deal in this communication. The incidence of the psychological changes in general and the behaviour disorders in particular in children attacked by epidemic encephalitis will first be shown; the clinical features of the behaviour disorders, with three illustrative cases, will then be described and, finally, theories of pathogenesis quoted and discussed.

INCIDENCE.

Grossman, of 89 cases, found over 50 per cent. with mental abnormalities, and Hall, commenting on these figures, said that slight changes were probably present in many more. Wimmer stated that of 25 children attacked 11 died, two were untraced, and the remaining 12 all showed pronounced psychological changes. Shrubsall, examining 119 children nine months after the acute attack, found 44 with conduct changes. Of 67 cases under our own observation 26 show behaviour disorders; 23, pure Parkinsonism; nine, a mixture of these two; five, serious mental retardation (only one definitely feeble-minded); and five are mentally normal. These figures indicate that of children attacked by epidemic encephalitis over 70 per cent. of survivors show some psychological changes and more than a third show behaviour disorders.

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CLINICAL FEATURES.

According to Wimmer, of all cases of behaviour disorder changes in morals and character stamp the picture, revealing curious uniformity in different patients. Psychical changes may occur at the outset of the disease, but in most cases they develop at a later period. The following is Wimmer’s list of characteristic conduct changes: restlessness, garrulosity, meddlesomeness, wide-awake yet erratic attention, excessive curiosity, moods of foolish mirthfulness, irritability, queer manners, desire to tease, querulousness, marked emotional irritability with uncontrollable outbursts of anger, scolding and the use of abusive language, propensity to destructiveness, biting nails, auto-mutilations, smearing objects with faces and urine, wilful spitting or micturition on surrounding persons or objects, violence, attempts at murder, attempts at setting fire to hospital beds, cruelty towards other children or animals, truancy from school, vagrancy, begging, dishonesty, mendacities, denunciations and pilferings; very commonly precocious eroticism, associated sometimes with premature puberty leading to masturbation, obscene conduct, exhibitionism, attempts on adult females or offences to small girls. The children, says Wimmer, become backward at school, perhaps primarily on account of their lack of mental perseverance. Any of the above disturbances may be presented in a mixed type with a degree of Parkinsonism, though not very frequently. He adds that it is doubtful whether actual defects of intelligence are present in the majority of cases and that some may be of the ‘ nocturnal ’ type, i.e., the patients’ behaviour is normal during the daytime, but becomes disordered at a fixed hour in the evening and continues so during the night. Any of the above conduct changes may be associated with signs of organic or visceral disease, e.g., tics, respiratory troubles, etc.

The above description covers exactly the picture presented by our 26 cases. The following points may be added.

Misbehaviour, whether immediately following the acute attack or appearing only after some months, develops to a maximum fairly rapidly, and then runs a steady course for years, not progressing or retrogressing, except when in some cases Parkinsonism supervenes and, when pronounced, abolishes it. The cases may continue in a mixed state but behaviour disorders do not occur in severe, and are usually lessened by mild, Parkinsonism. The children show at frequent intervals any of the forms of abnormal behaviour mentioned above. A severe case may be continually misbehaved, a mild case remain normal except for occasional outbursts. In all but the mildest cases the patients have in common a liability to bouts of anger and fighting, and a general restlessness with secondary inability to sit still for long or concentrate in school. Attempts at mental concentration make their restlessness and behaviour worse, whilst occupations not demanding much sustained concentration usually improve them for the time. However, they are fundamentally uninfluenced by environmental
changes, the effect of discipline, etc., being quite superficial. Very mild cases may appear normal under the latter, but relapse when the patients return home.

Many of our patients are physically normal, though, as mentioned above, nine show a mild degree of Parkinsonism. Ocular abnormalities, tics, and nocturnal insomnia are common. There is one case of moderate dystrophia adiposogenitalis but none of premature puberty.

The patients never suffer from a gross defect in intelligence. In nearly all the parents say with certainty that no falling off in general intelligence was apparent to them. Anderson states that the children "do not suffer serious mental defect and that conversely severe impairment is not accompanied by behaviour disorders in any marked degree," and she adds that a noteworthy characteristic is that all have excellent memories. Also, Marshall has found that the general intelligence is not appreciably affected and that it is quite exceptional to find an intelligence quotient below 85. A recent investigation by Dawson and Conn in Glasgow showed that all children attacked by encephalitis showed some small falling off in response to intelligence tests compared with controls. The mental ratio of 974 non-encephalitic patients averaged 90-53 and of 46 encephalitic patients 84-63. This not very greatly reduced response was common to all encephalitic children besides those with conduct changes. These investigators conclude as follows: "Whatever may be the ultimate explanation of the moral and emotional instability it does not appear to be due to intellectual degeneration." Of our patients a number of the most marked cases of behaviour disorder give a normal response to intelligence tests of their appropriate age and none shows serious retardation.

All the forms of misbehaviour are of a primitive and instinctive type of reaction and their immediate cause appears to be a slavery of the children to primitive and instinctive tendencies. These tendencies, which are present but much more under intelligent control in the normal child, seem to have their normal affective accompaniment so greatly exaggerated that obedience to them, in spite of the child's intelligence, is compulsory. The pleasure of satisfying them is so seductive and the pain of obstructing them so unbearable that sensual, crude and instinctive forms of behaviour have to be indulged in. There seems a general increase in the affective side of the child's life; and in a child whose behaviour is so much more than in an adult immediately determined by instinctive tendencies and responses, whose power of intelligent reflection and control is so undeveloped, and who is normally so ready to satisfy its appetites, to blaze into anger or dissolve into tears, it is easy to see how an increase in affective response to stimuli, whether mental or physical, pleasurable or painful, can exaggerate primitive and instinctive tendencies and produce their omnipotence.

The children realise the atrociousness of their actions, often showing spontaneous regret for them and evidence of trying to control themselves, weeping or giving signs of the most genuine repentance and complaining that
they "could not help it." Thus Anderson says: "... they appear to be acutely conscious of the impulse to do wrong, but are quite unable to control it." They frequently express their inability to hold themselves in check.

A patient of Anderson's made this remark: "I am a bad boy. I know I am a bad boy, but I cannot help it." Another patient of mine (Case 2) on sitting down at a table pushed a porcelain vessel out of her reach for fear she might have an uncontrollable impulse to smash it and, having spat fiercely at me, burst into tears and would not be consoled, blaming herself bitterly. Another patient said he often had a feeling that he must go into his garden and hurt his dog.

The great exaggeration in these patients of primitive and instinctive tendencies and their consequent slavery to them accounts for their general restless-ness and inability to concentrate on intellectual work, although they often wish to and are distressed because they cannot (Cases 2 and 3), for they continually succumb from minute to minute to fresh impulses. Advice, admonition and punishment, though the patient receives them with genuine penitence and promises of amendment, are without effect, for primitive tendencies are naturally uninfluenced, and, as soon as these arise again, the patients relapse.

Tics have been mentioned above as occurring in cases of behaviour disorders and occasionally (Case 3) some of the actual disorders of behaviour are of a tic-like nature, i.e., the same piece of misbehaviour of short duration is repeated in identical form over and over again for no apparent reason. Kinnier Wilson has emphasized the importance in the causation of tics of a seeking by the patients for a pleasurable sensation and of their susceptibility to impressions heavily charged with pleasurable feeling-tone." This would explain their presence in these encephalitic children whom the disease has made so susceptible to states of pleasure or displeasure.

ILLUSTRATIVE CASES.

CASE 1. Female, age 15. Before the onset of encephalitis the patient was in every way quite normal, well-behaved, well-liked, fairly good at school and quite obedient. There was no neuropathic history. In February, 1922, at the age of nine years, she had a typical acute attack of epidemic encephalitis, with diplopia and lethargy which persisted for a month ("she slept 23 hours a day"). In the course of another month this slowly passed away and she seemed quite normal. For six months she continued quite well and then started quite suddenly quarrelling with other children at school or in the street. She grew very inattentive and developed a violent temper, which was extremely easily aroused and during which she would strike her mother fiercely. All this grew worse and worse until she became completely unmanageable at home. If any of her desires were thwarted, e.g., if she were not given her meals directly she wanted them, she would fly at her mother.

Since admission in 1926 she has shown considerable disorder of conduct. She is continually interfering with other children's occupations; she will issue orders to them, try to 'boss' them, and a fight will ensue. Her very violent, easily aroused temper produces fierce fights. When her interference with the other children is prevented by anyone, she will attack the latter, striking her and trying to seize her by the throat. Her interference with other children is governed by a curious maternal or protective
feeling for them, which leads her occasionally to order the nurses to carry out this or that treatment for them. Her life and actions in the hospital seem largely governed by this feeling. She fought with ‘maniacal’ temper one morning to get at a child from whose eyes she wished to remove a bandage. Her violent outbursts of temper, fighting, or defiance last only a few moments and are of daily occurrence. After them she cries with genuine repentance and expresses her sorrow and her inability to control herself. Her insight is very clear. She realises her uncontrollable desire to interfere with the other children, and after an outburst will say: ‘I am ashamed of myself. I went mad. I did not know what I was doing.’ However, in addition she will often make excuses and try to justify herself. She is often quite pleasant and normal for short periods, though minor delinquencies, e.g., thefts of food and lying to gain advantages, are very constant. The ward staff make this comment on her: ‘She seems to lose all control and then does not mind what she does to anybody, regardless of consequences.’

Punishment, reproof and appeal, though productive of tearful response and promises of good behaviour at the time, produce absolutely no effect in the long run. Since her admission, in spite of discipline and occupation, her behaviour disorder has shown no improvement. She has a very voracious appetite.

Physical development is normal. There is no sexual misbehaviour or premature puberty. There is moderate nocturnal restlessness, but no diurnal lethargy. Her intelligence appears unimpaired. In the words of her parents: ‘No loss whatever noticed in her intelligence, judgment or memory.’ In 1926 at the age of 13 her mental age was 12+1, ‘but attention tires readily.’ Physically: pupils, right larger than left. Both sluggish to light and accommodation; marked coarse nystagmus in all directions; visual acuity normal. No extra-ocular paresis, but frequent transient diplopia. Otherwise normal; no trace of Parkinsonism or visceral involvement.

**Case 2.** Female, age 13. Before her illness the patient was a ‘sweet-tempered, bright and happy child.’ She had been very clever at school and there was no neuropathic history. In March, 1924, at the age of nine, she had an acute attack of epidemic encephalitis of sudden onset, with headache, fever, weakness of limbs, delirium and complete insomnia for three days and nights. Then for a fortnight she remained in bed, very drowsy, sleeping for ‘hours and hours.’ She then got up but continued to suffer from great nocturnal restlessness and diurnal lethargy. Five months later she seemed to have recovered and to be almost herself again. For one year she remained at school, never missing a day. Then she began to get very drowsy again during the day, ‘had no life,’ and did not want to go out at all. She slept all night and on rising would fall asleep over her breakfast, go to school and promptly fall asleep as soon as she got there, not being able to do her work. She developed at the same time frequent bouts of ‘deep-breathing’ and had ‘queer turns’ during which she would go quite rigid for a few moments. Toesillectomy was performed in hospital. Immediately following the operation her condition became much worse. She started habits of spitting at and pinching other people, grinding her teeth, and used to cry a great deal (October, 1925). For the next four months, until admission to hospital, she was unmanageable at home, not only because of these impulsive actions, but because of other abnormal conduct, such as taking articles of her parents out of the house and giving them away.

After admission to hospital in February, 1926, the patient’s behaviour and mental condition were reported as normal, except that she was apt to hang her tongue out frequently (a tic) and had bouts of noisy tachypnoea when excited. She ate and slept well and was good at school. Seven months after admission a change gradually appeared. She became constantly restless, more pert than usual to adults, was incessantly grinding her teeth when unoccupied, and developed numerous impulsive actions. She would suddenly tear her clothes, or smack someone in the face, or, if nobody were near, smack herself on the head, and she took to blowing at people who went near her and attempting
to spit at them, and was constantly spitting when walking about. She was quite conscious of her sins and ashamed and tearful on being spoken to about them, but said, "I can't help it," or "something makes me do it," and added that it "helped" her to smack someone and that it gave her the same satisfaction to smack her own head if no one else was present. She began to smash chinaware whenever she could, and to tear bed-clothes (making one sudden rip). She appeared to improve slightly after a quinsey 13 months after admission, showing more powers of control. However, two months later she was as bad as ever, developing much crying, frequently throwing her meals on the floor or fire, and even getting out of bed at night and pinching and biting another child who was asleep. Many uncontrollable actions then took place daily. She jumped suddenly out of bed and hurled a marble locker-top through a window (she said she felt dizzy and did not know what she had done until she saw the damage); she struck without warning or reason a nurse who was bathing her; picked up a chair and broke a large window; hurled a chamber through a ward window; hit a stooping person hard on the head, etc. She would implore not to be made to sit near windows. "in case I smash something." Eighteen months after admission she developed a strong desire to have her head covered day and night, as it "stopped her from spitting." She was very fond of school but attendance was often impossible, as suddenly she would bang on her desk for a few moments with her fists and then rapidly fly at and strike another girl or the teacher. It would be over in a few moments, and she would then cry: "It's no good. I can't stick it any longer."

During her stay in hospital (up to the present day) she has remained impulsive and uncontrollable. She is never normal in behaviour for a single day. Her intelligence is normal. A recent Binet-Simon test shows her at least up to her age-standard, and similar test done at the age of nine years gave her mental age as 11½. Physical development is normal. There was at one time some mutual masturbation with another girl. There is no nocturnal restlessness. During the last four months she has shown the development of slowly advancing Parkinsonism. She has a slight Parkinsonian mask, slight cog-wheel rigidity in the legs, and a somewhat Parkinsonian attitude and gait.

**Case 3.** Female, age 13. Prior to the onset of encephalitis the child was described as "very bright," quite natural, well-behaved, with no bad temper, and with no "tricks or habits." She was in good physical health, slept well and was of normal intelligence, was not a nervous child, and displayed no neurotic signs in infancy. She had an acute attack of epidemic encephalitis in March, 1924, with fever, diplopia and inversion of sleep rhythm. She was in bed four days only and, immediately after she got up, a change of character was noticed which grew rapidly more marked and was fully developed within the first month. She became quarrelsome and disobedient, screaming with passion when reprimanded, and in addition became restless and developed tics. She suffered from dribbling (? over-salivation) and bouts of noisy breathing when excited. She became abnormally affectionate, her parents describing her displays of over- afectation as "sickening." No diminution in intelligence was noticed by the parents, who said that she was "if anything a bit quicker."

The patient was admitted to hospital in October, 1926. There was a faint trace of Parkinsonism in her attitude and gait. She was quarrelsome and easily upset and was very restless, being unable to sit or stand in any one position for more than a few moments. She showed a violent temper when she could not get her own way, would fight readily, and displayed an uncontrollable impulse to pinch and slap anyone who came near, which she would obey constantly even if she were hit back in return. Bouts of noisy tachypnoea would come on at any time, particularly when she was excited, but were commonest at night, when she was very restless and slow in going to sleep. She had an uncontrollable tendency to behave in an over- afectative way. When she was not pinch ing or slapping them, she would like to sit with her arm round one of the other patients and longed for caresses. She tried to behave in the same way to the nursing staff. They described her
as liking to be petted and "craving for sympathy." She was quite conscious of her abnormal behaviour, regretted it and said that she could not help it. She could only be kept out of mischief by being put to bed. She showed signs of trying hard to maintain self-control. When reproved for over-affection she said, "I can’t help it," and when reproved for other actions said, "I can’t help it. I try to be good but I feel I must do it. I will try to stop it." She became keen on school and did comparatively well, trying very hard. Sometimes she would say, "Can I stay upstairs by myself until schooltime? I feel I am going to fight." Four months after admission slight Parkinsonian bradyphrenia with easy fatigability began to develop. Seven months after admission her power of control was definitely greater and not so much discipline was required. She has been trying hard and steadily improving ever since, but her Parkinsonism has been slowly advancing.

At the present time physical development is normal, there is no premature puberty, and she is not yet menstruating. Apart from a definite degree of bradyphrenia her intelligence is normal. There is a slight Parkinsonian mask, excessive salivation, an attitude of flexion, and a typical, though not severely marked, gait with little arm-swinging. There is an occasional tremor of the right arm, but muscle tone is normal in the limbs, and there is no hypothalamic involvement.

THEORIES OF PATHOGENESIS.

The theories that have been put forward are very varied. Some of them may here be mentioned.

Marshall considers that the patients suffer from psychomotor excitement similar to that occurring during an attack of acute mania of the manic-depressive type; Mapother, that their restlessness does not resemble acute mania, but that the symptoms represent a definite regression primarily due to affection of some part of the brain underlying foresight; Shrubsall, that the condition is due to a persistence of the mental upset of the acute stage of the illness as a habit or a psychoneurosis. Leahy and Sands vaguely believe that the cause is a chronic irritation and stimulation of nerve-cells by the resolving infection; Potts, that it is a chronic toxæmia, and Cloake, a depression of the highest cortical functions in particular, by a general toxi-infective state partly resembling the acute effects of a neurotoxin like alcohol. The latter also suggests 'psychic trauma,' such as putting the patient in a bath against his will.

These theories either are based on an interpretation of the clinical features with which we do not agree, or are too vague, making no satisfactory attempt to explain the distinctive pathology that must produce such distinctive signs. Thus we can find no evidence for regarding the condition as one of acute mania, functional nervous disease, or general chronic toxæmia. Occurring in an encephalitis, running such an unfluctuating course so similar in every case, and uninfluenced by the environment, the condition would seem to be due to an organic lesion of the brain. But we think that there are grounds for being more definite than the above theories about the latter. We have suggested above that the key to the abnormal behaviour is an increase in the affective accompaniment of primitive and instinctive tendencies, and we think that it is possible to indicate the organic lesion responsible for this.
We suggest that the organic lesion is an encephalitic one producing a loss of cortical inhibition over, or chronic excitation of, the optic thalami. The thalamus is the seat of affective life and the effect of loss of cortical inhibition over it by a destructive lesion producing thalamic release is well known. A similar effect could be produced by a chronic encephalitic focus producing chronic irritation and excitation of it. By either of these means an exaltation of the affective function of the thalamus would be produced. The affective response of the organism to any stimuli, mental or physical, would be increased and the condition that we consider exists in the encephalitic children with behaviour disorders would be produced. Their instinctive tendencies would have their normal affective accompaniments exaggerated and their dominance as suggested above would be produced. Our suggestion is supported by the known predilection of the encephalitic virus for the thalamo-striatal level on the one hand, and by the fact that owing to the preservation of memory and intelligence there can be no serious involvement of the cortex on the other. If it is agreed that behaviour disorders represent a primary disorder of affect of organic origin the lesion must be thalamic, with one very hypothetical exception mentioned below, as the thalamus is the centre of affect.

Further support for the theory that the lesion exalts thalamic function is forthcoming in that probably the thalamus in man still has some responsibility for the origin in itself of crude, primitive, instinctive behaviour. As McDougall says: "It has been shown that instinctive actions of the mammalia are chiefly dependent on the basal ganglia of the brain (especially the optic thalamus)." McDougall's long list of instincts is not accepted by all psychologists, but he says: "... there is good evidence that each of the principal forms of instinctive activity is especially dependent upon a particular small mass of nervous tissue within the basal ganglia." Each patient with behaviour disorder certainly displays favourite forms of primitive conduct; one is predominantly over-affectionate, another aggressive and self-assertive, and so on. Perhaps with the furtherance of knowledge the lesions may be localisable in a particular part of the thalamic region from the clinical signs.

There are, of course, no gross physical signs of thalamic release in the cases of behaviour disorder, though compared with normal controls they all show much greater emotional reaction to pin-prick, deep-pressure pain, etc. A comparison of Case 3 with a case of unilateral thalamic syndrome described by Head and Holmes is interesting. The latter patient confessed that he had become more amorous since the attack: "I crave to place my right hand on the soft skin of a woman. It's my right hand that wants the consolation. I seem to crave for sympathy on my right side." What would his behaviour have been like if his lesion had been bilateral?

A possible but very hypothetical cause of a disorder of affect producing a disorder of conduct is involvement of the hypothalamus, which occurs quite often in encephalitis, i.e., a lesion affecting the hypothalamic visceral centres.
The James-Lange theory is discredited but the following sequence in the generation of emotion has been suggested by Herrick. An intelligent process, i.e., a process of cortical association, occurs and involves the thalamic neurones and so gains an affective colouring. The hypothalamic visceral centres are activated and the usual visceral accompaniments of emotion take place. Afferent impulses from the sites of visceral change then ascend to the thalamus and reinforce the original emotion. Herrick calls this a "back-stroke" action. An involvement of the hypothalamus might disturb this "back-stroke" action. Might a disorder of affect and so of conduct thus result? Worster-Drought has recorded a case of moral imbecility associated with dyspituitarism apart from epidemic encephalitis.

Finally, cases of behaviour disorder may be contrasted with cases showing the bradyphrenia of the Parkinsonian syndrome. The bradyphrenia, something apart from the mere appearance of mental inertia due to the physical state, is characterised by a good intelligence with a subjective state of mental inertia and loss of interest or 'feelings.' It is worth while suggesting that this condition is produced by a cutting out of the mental life of much of the normal susceptibility to affective states and tendency towards instinctive forms of response which together make up much of the 'animal spirits,' spontaneity, and joie de vivre of a normal person. That is, it is suggested that bradyphrenia is the exact opposite to the mental state of cases with behaviour disorder and is due to a depression of thalamic function. A chronic irritative lesion of the thalamus might produce behaviour disorder and, when eventually the lesion becomes paretic and the thalamus succumbs, the condition might pass into bradyphrenia. On the physical side the general hyperkinesia of the cases of behaviour disorder can be contrasted with the bradykinesia of Parkinsonism.

SUMMARY.

The clinical features of juvenile behaviour disorders in chronic epidemic encephalitis are described, with three illustrative cases. The children have a preserved intelligence and clear insight into their condition, realising the error of their conduct. The latter is compulsive and consists of actions of a crude, primitive and instinctive type. Its compulsiveness is regarded as being due to an overpowering increase in the pleasure that normally comes of obeying instinctive tendencies and in the displeasure that normally comes of obstructing them. The liability of a general increase in the affective response to all stimuli to produce this effect in the child, where instinctive tendencies are normally so prominent, is pointed out.

The pathology is organic and is an encephalitic process, causing exaltation of thalamic function either by thalamic release through loss of cortical inhibition or by chronic irritation and excitation. This is supported by (1) the known incidence of the encephalitic virus on the thalamo-striatal level, and by (2) the
preservation of memory and intelligence indicating that the cortex cannot seriously be involved. Also, the thalamus probably has some action in the origination, in itself, of primitive and instinctive behaviour.

There is a possibility that a hypothalamic lesion may produce affective and consequently conduct disorder.

The mental condition in cases of behaviour disorder is contrasted with bradyphrenia, which, it is suggested, is due to depression of thalamic function.

The observations in this paper were made in the Post-Encephalitis Lethargica Unit of the Metropolitan Asylums Board, and I have to acknowledge with thanks the permission of the Medical Superintendent of the Northern Hospital to publish details of the cases.

REFERENCES.

Auden, Jour. Ment. Sci., 1925, lxxi, 647.
Dawson and Conn, Arch. Dis. Childhd., 1926, 1, 357.
Duncan, Brain, 1924, xlvii, 76.
Ebaugh and Rhein, Amer. Jour. Psychol., April, 1924.
Gellatly, Lancet, 1923, i, 1213.
Head and Holmes, Brain, 1911, xxxiv, 109.
Jelliffe, Post-Encephalitic Respiratory Disorders, New York, 1927.
Kirby and Davis, Arch. Neur. and Psychiat., May, 1921.
Leahy and Sands, Jour. Amer. Med. Assoc., 1921, lxvi, 373.
Paterson and Spence, Lancet, 1921, ii, 491.
Werner, Zeits. f. d. g. Neurol. u. Psychiat., 1927, cvii, 231.