PERIODIC CATATONIA.

By J. S. Bonar Lindsay, M.B., Ch.B.(N.Z.).
(From the Division of Mental Hygiene, Health Department, N.Z.)

Kraepelin (10) in 1908 first defined a group within his classification of schizophrenia which he described as periodic catatonia. The clinical features of this type may be briefly summarized. The onset, usually between 14 and 20 years, is frequently characterized by a period of erratic conduct followed by an interval of stupor, confusion or excitement, which thereafter undergoes periodic remissions and relapses. The periodicity is remarkable in the rapidity with which all changes occur. In the periods of remission the patient is free of catatonia and associated symptom-complex (sympatheticotonia), but is still dull, apathetic and withdrawn; he often recalls events from the reactive phase, but without insight. The patient is still schizophrenic, with emotional flattening and poverty of ideation, and remains so throughout. In the reactive phase the picture is one of catatonic stupor or excitement, with clinical evidence of sympathetic over-activity.

The most clearly defined cases are those in which there is a synchronicity in catatonia and sympathetic function; in these the change in state is more rapid, the catatonia more profound. Gjessing (7) calls these the synchronous-syntonic group. Other less typical cases are described as asynchronous-asyntonic. The sympathetic changes synchronous with the onset of catatonia are those of adrenal-orthosympathetic over-activity. Increases in the pulse from 70 to 100–120 occur in a few hours. The blood pressure may likewise increase by over 40 mm. Hg. There is pallor, cold clammy extremities, mydriasis, fleeting anisocoria, and, during stupor, a fine tremor and retention of urine. Even when the patient is immobile night and day there is an increase in B.M.R. Urinary output and weight decrease and the viscosity of the blood increases.

Gjessing (7) has studied this group of patients, recording many variables and body-constants over 3,500 "patient-days." He charted the following: psychic state, urinary output, specific gravity and constituents (ammonia, titratable acidity, chlorides, phosphates, indican, sulphate, and nitrogen output), oxygen intake, carbon dioxide excretion, respiratory quotient, alkali reserve, haemoglobin, white cell count, blood sugar, plasma nitrogen, body temperature, weight, and pulse rate. He also made estimations of the nitrogen balance.

As a result of this momentous amount of work, a relationship has been established between the nitrogen balance and the periodicity. This further resolves itself into two types which Gjessing calls types A and C. In type A the patient increases his nitrogen store to a peak, and shortly after excretion starts, catatonia develops, only to disappear when the store has been eliminated. In type C the catatonia starts at the end of the period of excretion and persists

https://doi.org/10.1192/bjp.94.396.590 Published online by Cambridge University Press

throughout the building up of the store of nitrogen. Gjessing was also able to demonstrate that the electrocardiogram shows a flat T1 during nitrogen retention irrespective of the mental state. The non-protein nitrogen in the blood follows the nitrogen periodicity. A rise in the blood sugar is seen in both types at the onset of the catatonia and orthosympathetic phenomena, but in type C the level remains above normal in the quiescent periods, in type A below normal.

It can be seen from a study of type C that the increased metabolism of the reactive phase is not the cause of the nitrogen swings, for here there is retention during activity. Stokes (16) has reported a case with characteristic metabolic studies in which later it was possible to demonstrate periodic catatonia, not with phasic changes, but with a steady accumulation of nitrogen.

It may be stated, therefore, that in periodic catatonia the mental state is not the direct cause of the known biochemical changes, nor are the latter the immediate cause of the former.

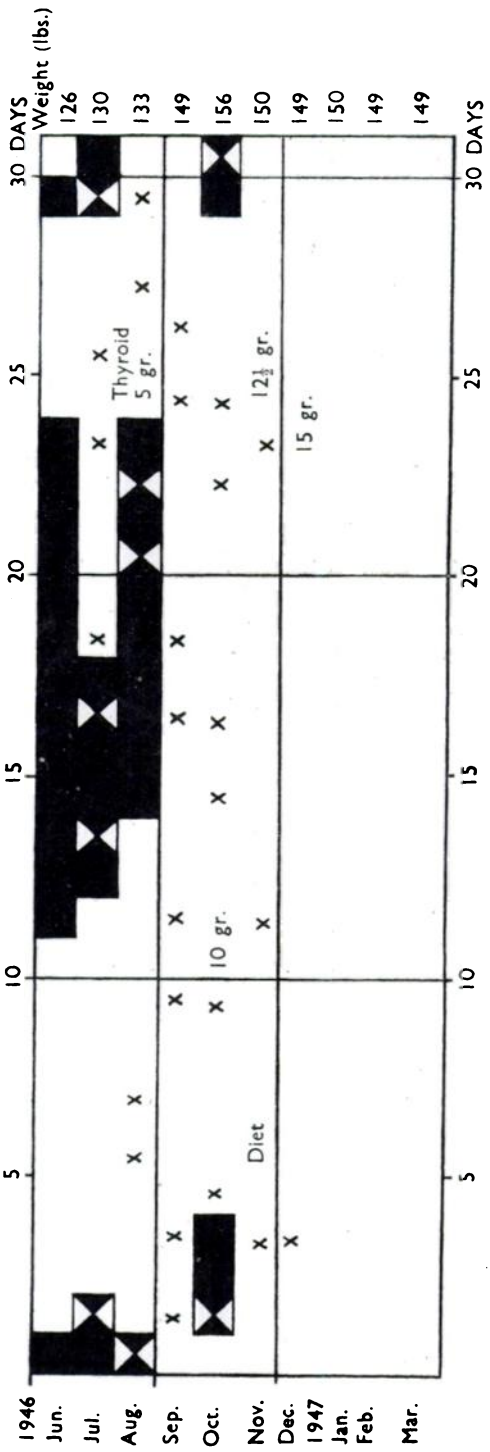
The two cases that are reported below have been studied over a long period. Only one body constant has been recorded, that of body weight. As this may reflect the total metabolism of the individual better than a record of the individual parts thereof, it may be deemed relevant to the study of periodic catatonia.

CASE 1.—The patient was born in 1919. His father died of melancholia in 1928. His earlier life can be briefly summarized as follows. He attended school till 15 years old, with two years' post-primary education. The subsequent year he spent as an office boy, and during the next year he had three jobs. During this period he became progressively more retiring, self-conscious and "absent minded," and often day-dreaming.

He was first admitted in July, 1936, after three weeks' excited schizophrenic behaviour. During the first two months he showed two periods of excitement with fairly rapid onset. He asserted that he was Edward VIII, and that he was to marry his sister, and make her Queen of England. This fluctuating course persisted for eight months, with his weight varying from 9 st. 7 lb. to 8 st. 6 lb. His weight then increased to 11 st. 5 lb. and at the same time his mental state improved, so that after five months in a parole villa he was released on probation and later discharged recovered.

For nearly two years after his release he worked in the country. In July, 1939, he was arrested for petty theft and readmitted. For four months he was periodically restless, noisy, erratic, and abusive, with at times signs of the catatonic stupor, later so pronounced. During this four months his weight rose from 9 st. 8 lb. to 11 st. 6 lb. At this stage he again improved, and after 8 months was discharged recovered. The record of his weight is of interest at this period. One month before his discharge his weight was 12 st. 4 lb., at the time of his discharge 11 st. 2 lb. Three days later he returned for admission as a voluntary boarder as a precautionary measure on account of some restlessness. His weight was 10 st. 13 lb. He relapsed and showed periodic catatonic behaviour a week later. He was formally committed three months later after he attempted to commit suicide, prompted by the bad thought he got from the Devil. For two years (1941 and 1942) he showed a continuation of the periodicity of catatonic phenomena, with quiescent intervals during which he was well enough mentally to work with a party. His weight through these two years varied from 9 st. 6 lb. to 10 st. 11 lb. The next three years showed a progressive worsening in the degree and length of the stupors. In the quiescent periods he was dull, apathetic and indifferent, but able to do routine tasks under supervision and care for person and clothes. In a stupor he lay curled up, immobile, mute, indifferent to personal habits and resisting all attention. The maximum recorded weights over these three years are 9 st. 12 lb., 9 st. 5 lb., 8 st. 11 lb., and correspond to the progressive increase in length and depth of stupor.

https://doi.org/10.1192/bjp.94.396.590 Published online by Cambridge University Press

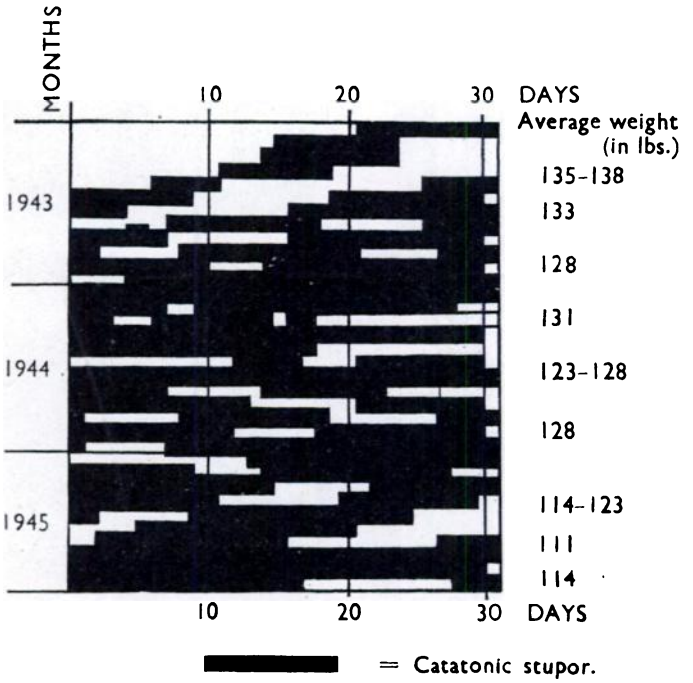


CASE I.—Diagrammatic representation of the progress of treatment.

https://doi.org/10.1192/bjp.94.396.590 Published online by Cambridge University Press

The minimum weight recorded was in June, 1945, 7 st. 13 lb., near the onset of the longest stupor recorded in his files—78 days. Towards the end of this stupor the toes showed marked " Raynaud's " phenomena, which progressed till they were nearly gangrenous. With the termination of the stupor these improved spontaneously. This observation was presumed to be an index of the intensity of the orthosympathetic over-activity. The pulse rate was frequently up to 130 in a stupor and between 70 and 82 in quiescent phase. Axillary temperatures were recorded up to 99·2° F. in a stupor.

After several more stupors in 1946 of up to 20 days' duration, with a body weight between 8 st. 4 lb. and 9 st. 2 lb., active treatment was instituted in July, 1946. E.C.T. was first given. Resulting from this was an increase in weight over two months of 7 lb. Stupors appeared to recur, but each recurrence remitted after



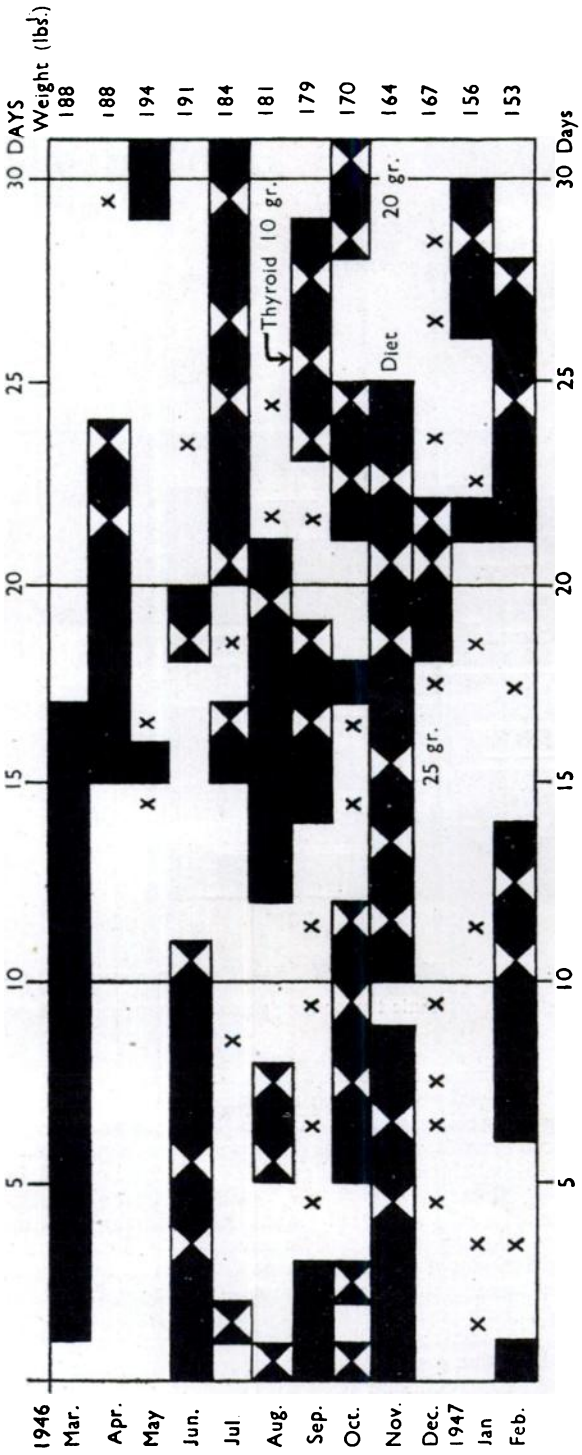
CASE 1.—Periodicity of the catatonic stupor for 34 months (represented diagrammatically).

two E.C.T. In the next two months he was given thyroid extract (5 gr. per diem, increased to 10 gr. after six weeks), with also approximately the same number of E.C.T. as in the previous two months. His weight increased by 23 lb. in the two months and he had only two short periods of stupor—five days in all. Body weight at the end of this two-month period was 11 st. 2 lb. He remained free of periodic catatonia.

Further treatment was continued. According to the recommendation of Gjessing, he received a low protein diet (qualitative). The thyroid extract was increased to 12½ gr., then 15 gr. per diem and the E.C.T. tapered off. In this quiescent phase the basal metabolic rate was — 7 per cent. on thyroid extract 15 gr. per day. With the increase in thyroid dosage his weight dropped to a steady level between 10 st. 5 lb. and 10 st. 9 lb. In the next six months he remained at a fair institutional level, he was still dull and apathetic, and still had nocturnal enureses. The thyroid dose remained at 15 gr./day and his weight 11 st. to 11 st. 8 lb.

CASE 2.—This patient was born in 1902. Until 1937 he had an excellent record. He was a university graduate, and a good athlete, though shy in his manner and bearing. There was no family history of instability. The onset of the psychosis was sudden and he was admitted in April, 1937. When he was first examined

https://doi.org/10.1192/bjp.94.396.590 Published online by Cambridge University Press

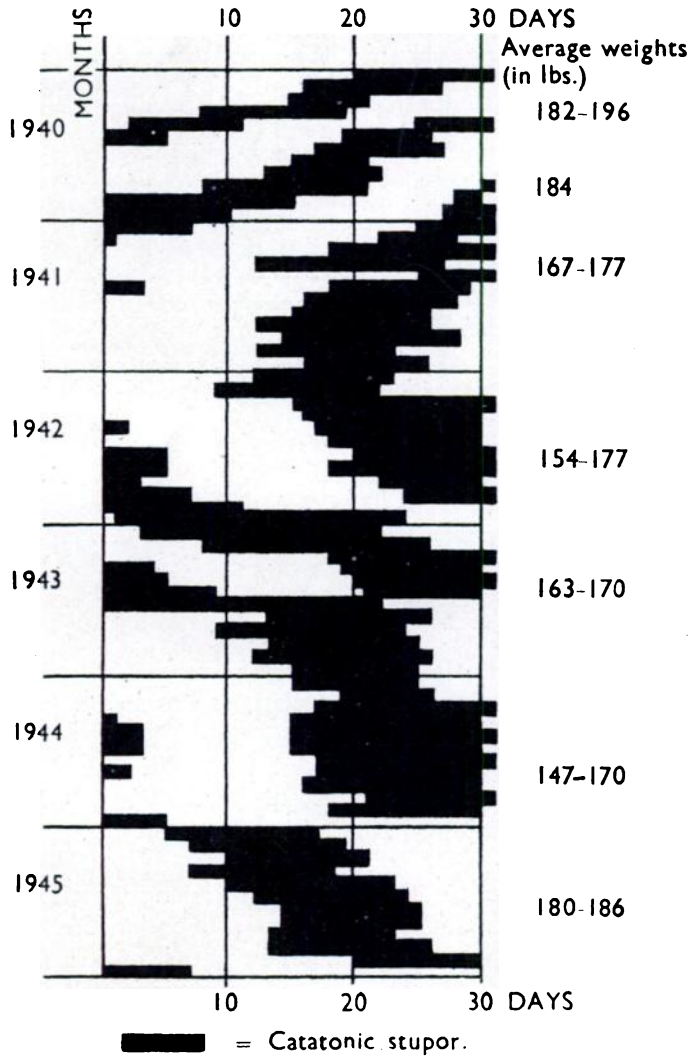


CASE 2.—Diagrammatic representation of the progress of treatment-symbols used are the same as in Case 1.

https://doi.org/10.1192/bjp.94.396.590 Published online by Cambridge University Press

he was usually co-operative, though at times vague and hesitant. On other occasions his speech was rambling, with intervals of silence, loss of train of thoughts and tears for no apparent reason.

" It's dawning on me now, I'm St. John, John of Runnymede, King John. . . It's being revealed to me I'm Sir Percival, the knight who saw the Holy



CASE 2.—Periodicity of the catatonic stupor for 72 months diagrammatically represented.

Grail. . . . You're the fellow—, the engineer, I'm the protector of the poor and I've been betrayed. . . . No! I haven't been betrayed." He denied hallucination, but stated " It's all within me." This type of conversation and his schizophrenic behaviour has persisted in his quiescent phases ever since.

Shortly after admission he became restless, more confused, fragmentary in speech, and neglected his bodily habits. He rapidly changed into a marked catatonic stupor, lying curled up on his back, staring at the ceiling for hours with negativistic and impulsive behaviour if disturbed. This period of catatonia remitted, but was followed by another in three weeks. In this second stupor he had to be cathe-

https://doi.org/10.1192/bjp.94.396.590 Published online by Cambridge University Press

terized for retention. For the next eight years and six months the periodicity of the catatonia was regular, and in this period of 102 months he had 116 periods of stupor. The duration, sequences, and body weights over six years can be seen in the graph.

In January, 1946, he was subjected to a full investigation of his urinary tract following further episodes of retention. No lesion was found, nor would such be likely to be found if the retention were due to orthosympathetic over-action as postulated. In the next three months he had three periods of stupor of 12 to 14 days' duration, with a sequence similar to those immediately preceding it. In the catatonic phase the pulse varied from 96 to 118 and the axillary temperature reached as high as 99·4° F. In the quiescent phase the pulse was recorded between 70 and 80.

E.C.T. was started in April, 1946, and after an initial favourable response (the predicted stupor was represented by one day of erratic conduct) associated with a gain in weight of 6 lb. he relapsed. For the next four months the stupors recurred more frequently, but each could usually be interrupted by two or three E.C.T. The weight dropped 15 lb. Thyroid administration was then started (10 gr. per diem of thyroid extract), and this made the situation worse. The stupors remained as frequent, but no longer responded to E.C.T., so that he had six stupors in eight weeks with only 14 days in the quiescent phases. His weight dropped in this two-month period a further 15 lb.

Temporary success was gained by increasing the thyroid extract to 20 gr. and then 25 gr. per diem. He had one four-day stupor in 54 days. His weight did not fall, though he was for this period on a low protein diet (qualitative). Regular E.C.T. was continued once or twice a week.

A relapse followed, associated with another fall in weight of 13 lb. He changed little in the next six months. Weekly or fortnightly E.C.T. was continued to interrupt the stupors. Their frequency was thus still increased. The body weight remained at 12 st. 6 lb. approximately for most of this period. Thyroid was discontinued after the period recorded in the graph.

Comment.

These two cases represent examples of the least common type of periodic catatonia. Two-thirds of the cases are females and excitement is commoner than stupor.

The course of the illness in these two patients is different. Cases with a clockwork regularity over decades are recognized, and others with progressive intensity of the syndrome over years with or without remissions are also identified.

The first case conforms to all the criteria, including the therapeutic test with thyroid for periodic catatonia. Apart from the therapeutic failure in the second case it likewise appears to be indubitably a case of periodic catatonia. The fact that the underlying process is schizophrenic is well shown in the second case with manifest schizophrenic symptoms present in all the quiescent phases.

It has been possible to follow the body weights of these two patients over the period of illness and the variations have been recorded. The first case shows several facts of interest. The patient has never been free of catatonic behaviour of a periodic nature with a body weight of less than 11 stone (approximately) before treatment. With thyroid he achieved this at a level of 10½ st. The lower his weight has been, the longer have been the periods of catatonia, and *vice versa*. His most profound stupor was associated with the most marked sympathetic phenomena. In the second case some less marked correlation appears to exist between the body weight and the length of the stupor. The

https://doi.org/10.1192/bjp.94.396.590 Published online by Cambridge University Press

observation of interest in this case is in the therapeutic failure. The patient over four months with E.C.T. alone lost 15 lb., in the next two months with thyroid and E.C.T. another 15 lb. The catatonia was worse. The first patient gained 7 and 23 lb. on a similar sequence, and was free of catatonic behaviour.

Studying this body weight constant in the reported cases, there appears to be a relationship between body weight and the symptoms. Periodic catatonia does not remit unless there is a gain in weight. Gjessing's (7) case C. J. L— shows a more frequent and longer catatonia with a lower body weight. Stokes' (16) case shows similar changes: thus the ratio between duration of catatonia to quiescent phase was 2 to 1, with the weight between 8 to 9 stone, but between 9 st. to 9½ st. the ratio was 6 to 5. These figures are only approximations in view of the small scale of the reproduced graphs. Later phases in Stokes' case appeared to have an ill-defined end to the catatonic phases with the weight over 10 st., suggesting a further lessening of the degree of stupor. There is no doubt that gross anatomical lesions of the hypothalamic nuclei can result in alterations in weight and metabolism. The body weight has been used tentatively as an index of the ability of the hypothalamic centres to control a normal internal environment.

Discussion.

Much suggestive work has been done that throws light on the synchronous catatonic orthosympathetic phenomena. De Jong (5), after many experimental studies, considers that catatonia is a "general" reaction form of the C.N.S. which, like symptomatic epilepsy, may have many causes, and likewise represents a non-localized motor discharge syndrome of the C.N.S. The sympathetic phenomena accompanying catatonia he attributes to the overdischarge of the centres. Experimental lesions in the posterior hypothalamus have produced hypokinesis and akinesis, and Bailey (2) has demonstrated a cataleptic element with these lesions. On the other hand, cortical stimulation is facilitated by simultaneous hypothalamic stimulation. Affective (rage) reactions are demonstrated in stimulation of the intermediate zone, and orthosympathetic overactivity dominates from posterior hypothalamic lesions. Similar reactions are induced in decorticate animals from very ordinary stimulation. Cairns (4) has reported a case of third ventricular cyst with akinesis and mutism. Likewise Furtado (6) has noted a similarly placed cyst with catatonia. A case of pituitary cachexia with catatonia was recorded by Stocks (15). Cases of hypothalamic lesions with excitement have been reviewed by Alpers (1). A few reports are available dealing with paroxysmal autonomic discharges, either as a diencephalic autonomic epilepsy (Penfield (13)) or post-encephalitic autonomic crises (Oller (11), Ostow (12)). The case reported by Ostow (12) showed post-encephalitic crises with cataleptic manifestations, such as akinesia, rigidity, and mutism, with concomitant autonomic reactions consisting of diaphoresis, rapid pulse, fever and hypertension. Oller's (11) case showed hyperkinetic reactions and aggressiveness, associated with an autonomic discharge. Thus the clinical picture is like the periodic catatonia-orthosympa-

https://doi.org/10.1192/bjp.94.396.590 Published online by Cambridge University Press

theticotonia of periodic catatonics, apart from the time course of the disturbance. This difference can be correlated with the fundamental disease-process responsible for the discharge. In post-encephalitic states episodic manifestations are common, and are comparable in duration with the disturbances in the cases quoted. The relationship of periodic catatonia to schizophrenia is discussed later.

It is in catatonic schizophrenia particularly that physical signs are most evident. These signs are of sympathetic nervous function disorder and are not those of recognized endocrine disorders. On the other hand, paranoid states with a paucity of such signs show a relatively high incidence of endocrine tumours at autopsy (Pollak (14)). From these considerations it is suggested that the catatonic sympathetic phenomena of catatonic schizophrenia are a definite somatic syndrome associated with schizophrenia, and that this syndrome is a reaction pattern of the C.N.S., probably hypothalamic in origin. The possible significance of the fact that the sympathetic phenomena appear synchronously and in a pure culture of orthosympathetic overactivity in periodic catatonia will be examined later.

The other *sine qua non* of periodic catatonia is the basic schizophrenic illness. In the quiescent periods the schizophrenic nature is usually apparent. In the reactive phases they are obvious catatonic schizophrenics. Referring briefly to the two reported cases, it is suggested that the difference in the course between the two is related to the degree of the basic schizophrenia. The first case with remissions showed only mild symptoms during the quiet phase, but the second with a relentless periodic course was markedly schizophrenic. Further life history studies of these patients are required to confirm or refute this suggestion.

Following Stokes' (16) demonstration that the periodicity could continue without phasic nitrogen swings it becomes necessary to classify this latter with other secondary phenomena. The treatment for periodic catatonia advocated by Gjessing (7) is the administration of thyroid hormone. This was devised to eliminate the nitrogen storage. The pharmacological response following thyroid administration is an increase in metabolic rate. A decrease in body weight and elimination of nitrogen are probably secondary results of this increase. The marked increase in body weight that follows thyroid administration in periodic catatonia (favourable response only—Case 1) is difficult to fit into accepted ideas. It would seem more logical, however, to explain the therapeutic response in terms of the more fundamental action. In the two cases reported, the favourable response to treatment only occurred in the case with a weight increase during thyroid administration. It is suggested that a thyroid-hypothalamus interrelationship is involved and that, as the hypothalamic control of the internal environment improves, in a favourably responding case, the body weight is approximated to the mean for height and build.

Theories to date to explain the nature of the illness appear to be two in number. The intoxication hypothesis, particularly amine intoxication, has been repeatedly suggested as a cause of many types of mental illness, and Gjessing (7) suggested it as a possibility in periodic catatonia.

https://doi.org/10.1192/bjp.94.396.590 Published online by Cambridge University Press

The weight of evidence is against any such amine intoxication hypothesis, even though certain amines can cause mental symptoms, catatonia and sympathicomimetic changes, e.g. 3-4-5-trimethoxy or 3-4-dimethoxy derivatives of phenylethylamine. It has also been shown that some secondary carbinamines inhibit brain respiration in dilutions approaching *in vivo*. Jantz (9) has shown that metabolic changes can occur after 3-4-5-trimethoxy-phenylethylamine similar to those in schizophrenia. Stokes (16) has shown in his reported case periodicity of mental state, at first synchronous with the phasic nitrogen changes, and then later independent of these metabolic changes. The nitrogen changes are not, therefore, the immediate cause of the mental changes. The changes reported are those of total protein metabolism and not just amine excretion. Gjessing (7) noted the occurrence of supernumerary phases after the thyroid administration had produced considerable nitrogen loss.

A second possible hypothesis is posed as a question by Stokes (17): "Is there a rhythmic upset of endocrine function more fundamental that the overt periodicity?" The overt periodicity is one of central autonomic regulation and catatonia. It is unlikely that there is a more fundamental rhythm than the central autonomic cycles except from higher centres controlling the hypothalamus.

It is proposed to examine the periodicity of central autonomic regulation in more detail, but before so doing it is advisable to note briefly some observations on the maintenance of homeostasis in schizophrenia.

Hoskins (8) and co-workers have studied over years many body constants and variables in schizophrenia. They come to the following conclusions: The functional level of some body constants is set at a lower level than normal. Subject to external influences (e.g. metabolic stimulants) these levels show lesser responses than normals. Despite this, however, the range over which they function in maintaining homeostasis is greater than normal. The "physiological withdrawal" of schizophrenics (decreased response to external factors) is associated with a decreased stability of the control of the internal environment. In periodic catatonia there is an increased lability in the control of various body constants. It might be deduced that such defective control would tend to magnify the amplitude of the phases of nitrogen balance. Also, studies of the nitrogen balance in normals, for example with menstrual cycles, would show a lesser variation than periodic catatonia. Quantitative data on the premenstrual nitrogen retention do not appear to be available in this country.

To provide a hypothesis for an illness with a periodic course it is necessary to consider the mechanism both for the onset and for the remission.

The only features that clearly separate periodic catatonia from catatonic schizophrenia are the orthosympathetic phenomena and the periodicity. They are otherwise clinically indistinguishable. Therefore if some characteristic mechanism can be formulated to explain the remissions, it would be reasonable to attribute the onset of periodic catatonia to the factors concerned in the genesis of the catatonia in non-periodic cases. The invariable association of schizophrenia with both the periodic and non-periodic type suggests an explanation of the onset.

https://doi.org/10.1192/bjp.94.396.590 Published online by Cambridge University Press

In the studies of the onset of schizophrenia Boisen (3) and others distinguish three methods of reacting during the development of the psychosis—the drift into a psychotic state, the delusional misinterpretation, and the panic reaction. This third type is one in which "the individual is likely, in Malamud's phrase, to become aware that he is tasting forbidden fruit and to sense the danger in which he stands and to become fearful of it" (Hoskins (8)). The disturbances may take the form of either stuporousness or agitation. It is this type of reaction that occurs in patients later described as catatonic. To quote Hoskins further (8), "primarily his reaction seems to represent a manful if misguided attempt to solve his inner problems."

This type of panic onset can further be orientated into accepted neural mechanisms. The usual response to a "fear-panic" situation is one of orthosympathetic over-activity, with hyperkinetic, or akinetic and cataleptic patterns of motor activity. The peculiar nature of the pattern in a panic situation reflects the various past experiences, habits, and intelligence of the normal, whereas the kinetic quality of the response is the result of hypothalamic stimulation activity. Likewise, in catatonic schizophrenia, the detailed pattern of the catatonia reflects the disordered processes of the psychosis, whereas the quality of the kinetic element and the orthosympathetic over-activity are due to hypothalamic activity. As indicated above, the hypothalamic control in schizophrenia is more labile than in normals. Applying this concept to periodic catatonia, a most striking similarity is demonstrated in the intense orthosympathetic activity—an emergency or fear reaction in almost pure culture. As Gjessing (7) described, these phenomena are syntonic and synchronous with catatonia in periodic catatonia. The hypothalamic response is integrated towards one end. Thus it is assumed that if the hypothalamic response to a panic situation is integrated it produces some amelioration in the schizophrenia and a period of recovery from catatonia-orthosympatheticotonia. If the cause remains, i.e. the schizophrenia, relapses will occur. The liability to relapse will depend on the intensity and persistence of the schizophrenic process, and the stability of the level of homeostatic control. The more defective the homeostasis the more marked the periodic catatonia.

On the other hand, where the hypothalamic response is asyntonic—i.e. not integrated to one purpose—and asynchronous, periodicity, as such, is irregular, gradual in onset and remission, and may be absent over long periods.

The more common cases of catatonic schizophrenia with a very irregular time course show no co-ordination or integration of the hypothalamic response. Thus both orthosympathetic and parasympathetic over-activity may coexist and may bear no temporal or other relation with the kinetic variation. This failure of integration in a central nervous reaction leads to its persistence, in the asynchronous and asyntonic form. In the acute catatonic schizophrenic reactions, the favourable response is possibly due to remission of the basic acute schizophrenic reaction. In some cases it might be attributed to the integration of the panic reaction, which appears to be fairly definite, in many of these acute reactions.

Improvement in the schizophrenia is likewise absent with defective integration of this hypothalamic neural pattern. The periodic improvement of

https://doi.org/10.1192/bjp.94.396.590 Published online by Cambridge University Press

the psychosis in periodic catatonia is of considerable interest, but it is not proposed to deal further with hypothetical aspects of the interrelationship between psychosis and hypothalamus, especially as regards E.C.T., insulin therapy and leucotomy. From a consideration of the therapeutic and spontaneous remissions it might be deduced that a possible mechanism for the remission would involve the increased metabolism of thyroid administration and orthosympatheticotonia respectively. Whether this is so or not, it would appear likely that the *locus operandi* is the hypothalamus.

It is necessary to examine the contributions of previous authors in terms of this hypothesis. If it is now applied to Gjessing's (7) types A and C it is possible that the following is valid. In type A where the periodic phenomena are most marked it is likely that the nitrogen balance will follow the periodicity in a uniform direction, i.e. almost co-temporaneously with the increased metabolism, the nitrogen balance will fall. In type C, with less intense periodic phenomena, it is possible that the reversal of nitrogen balance phases is an expression of the asynchronicity and asyntonicity, which in the common type of catatonic schizophrenic is a predominant feature in the somatic syndrome.

If the degree of disturbance of the control of homeostasis is reduced, improvement will result, but still dependent on the intensity of the schizophrenic process. With a normally controlled internal environment the body constants will tend to return to their mean range of values. At some stage of improvement it is suggested that some factors (such as nitrogen balance for example), which in marked disturbance follows the periodicity closely, may become divorced from this periodicity and move in a uniform direction towards a more stable and safe level (this is to explain Stokes's (16) case with catatonia and no nitrogen swings, but with increased body weight, and nitrogen accumulation).

Conclusion.

Briefly stated, the hypothesis here advanced states that the kinetic and sympathetic phenomena of catatonic schizophrenia are of hypothalamic origin. These phenomena are a specific neural reaction pattern to the " panic " onset of the psychosis. If this pattern is integrated it serves to restore the normality of the hypothalamic centres with some improvement in psychosis *eodem tempore*. This is the case in periodic catatonia. In non-periodic catatonia the specific hypothalamic pattern is disintegrated. The efficacy of the reaction depends on its degree of integration, on the degree of causal schizophrenia, and on the level of hypothalamic control of the internal environment at that particular time.

Summary.

1. A review of the condition of periodic catatonia is made.

2. Two cases are reported.

3. An analytical study is conducted into the nature of the symptoms of the disease.

4. A hypothesis is put forward about the nature of catatonia.

https://doi.org/10.1192/bjp.94.396.590 Published online by Cambridge University Press

ACKNOWLEDGMENT.

My thanks are due to Dr. J. Russell, Director of the Division of Mental Hygiene, Dr. B. D. Hart, Medical Superintendent of Porirua Mental Hospital, and Dr. R. W. Medlicott, Medical Superintendent of Ashburn Hall.

LITERATURE.

(1) ALPERS, B. J. (1940), " Personality and Emotional Disorders Associated with Hypothalamic Lesions," *Res. Publ. Assn. Nerv. Ment. Disease*, **20**, 725.
(2) BAILEY, P., *et al.* (1945), " Effects of Lesion of the Periaqueductal Gray Matter in Cats and Monkeys," *Arch. Neurol. and Psychiat.*, **53**, 325.
(3) BOISEN, A. T. (1936), *The Exploration of the Inner World.* New York: Willett, Clark & Co., Chicago.
(4) CAIRNS, H., *et al.* (1941), " Akinetic Mutism with an Epidermoid Cyst of the Third Ventricle," *Brain*, **64**, 273.
(5) DE JONG, H. H. (1945), *Experimental Catatonia.* Baltimore: The Williams & Wilkins Co.
(6) FURTADO, D., *et al.* (1946), " Catatonia from Diencephalic Compression," *Revue Neurol.*, **78**, 449.
(7) GJESSING, R. (1938), " Disturbances of Somatic Function in Catatonia with a Periodic Course and Their Compensation," *J. Ment. Sci.*, **84**, 608.
(8) HOSKINS, R. G. (1946), *The Biology of Schizophrenia.* New York: W. W. Norton & Co.
(9) JANTZ, H. (1941), " Metabolic Changes during Mescal Intoxication in Man and Animals; Similarity to Metabolic Changes in Schizophrenia," *Ztschr. f. d. ges. Neurol. u. Psychiat.*, **171**, 28.
(10) KRAEPELIN, E. (1913), *Psychiatrie.* Eighth ed. **3**, 806.
(11) OLLER, C. I. (1946), " Paroxysmal Autonomic Discharges in the Post-encephalitic State," *Arch. Neurol. and Psychiat.*, **55**, 388.
(12) OSTOW, M. (1943), " Recurrent Autonomic Phenomena Associated with Exacerbation of Post-encephalitic Parkinsonism," *ibid.*, **50**, 342.
(13) PENFIELD, W. (1929), " Diencephalic Autonomic Epilepsy," *ibid.*, **22**, 358.
(14) POLLAK, O. J. (1944), " Post-mortem Studies in Mental Patients," *Amer. J. Clin. Pathol.*, **14**, 289.
(15) STOCKS, J. W. (1930), " A Case of Pituitary Cachexia," *Lancet*, **2**, 349.
(16) STOKES, A. B. (1941), " Metabolic Investigations in Periodic Catatonia," *Proc. R. Soc. Med.*, **34**, 733.
(17) *Idem* (1939), " Somatic Research in Periodic Catatonia," *J. Neurol. and Psychiat.*, **2**, 243.

https://doi.org/10.1192/bjp.94.396.590 Published online by Cambridge University Press