DISTURBANCES OF SOMATIC FUNCTIONS IN CATATONIA WITH A PERIODIC COURSE, AND THEIR COMPENSATION.*

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MY problem is to give you a short review of some investigations we have undertaken at Dikemark Mental Hospital on the disturbances of somatic functions in catatonia with a periodic course (1).

This small group, which was first defined by Kraepelin (1908) (2), embraces scarcely more than 2 to 3% of schizophrenics. It is characterized psychopathologically and with regard to its course as follows.

The onset of the illness occurs in most cases before the age of 20, rarely at the ages of 14 to 15. After slight indications of alteration in conduct for a few days, a state of confusion or excitement suddenly sets in. This clears up and repeats itself after a varying interval. The restlessness can increase rapidly, so that the patient, even after a few days, reaches the severest degree of excitement and violence. In other cases the condition shows itself only in increased irritability, affective lability, over-activity and talkativeness. The body-weight falls rapidly, 4 to 8 lb., in the course of 24 hours after the onset of restlessness. The excitement often only lasts a few days, or weeks, rarely a few months. The intervals between the phases of excitement are as a rule of somewhat longer duration. The patient usually becomes quiet just as rapidly as he became excited, even although one notices towards the end of the attack a slight diminution in the excitement and confusion. Suddenly, one day, the patient is completely controlled in his behaviour but remarkably quiet and indifferent. He appears dull and apathetic and as a rule does not attain any insight into his condition, notwithstanding the fact that he may remember many details of his excited state, which he thinks has been quite harmless and conditioned by his surroundings. He now believes himself to be quite cured and is persistently asking for his discharge. His bodily health rapidly improves, though the weight during the interval may often be lower than during the normal healthy period. After some years a prolonged period may occur during which symptoms remain in abeyance. In other cases the attacks increase in duration and number year after year ; in yet other cases the attacks may become completely regular and continue for decades. Very rarely a quotidian type may develop. It is important that as a rule a condition sets in

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characterized by marked impairment of all mental faculties, with poverty of ideation, lack of critical faculty, emotional flattening and mannerisms. Two-thirds of these patients are females, in whom the periodicity from the sexual point of view may predispose them to this particular course of the illness.

Kraepelin initially included this form of the disease in the manic-depressive group, but later found that it resembled dementia præcox far more than mania, on account of the dementia that develops, and because of its more or less mechanical automatic nature and the poverty of ideation by which the excited phase is characterized.

What has been said about the catatonic states of excitement showing periodicity is also valid for the somewhat rarer cases of periodic catatonic stupor. It is easy to understand why this particular group raised the hope that, by careful investigations, disturbances of function in the metabolic sphere might be elicited from one phase to another ; especially as there occur sudden changes of phase in the mental state concurrent with numerous disturbances of somatic function, both during the periodic excitement and the periodic stupor, with changes in the pupils, pulse, blood-pressure, muscle tone and secretions. The goal we had before us when in 1925 we started these investigations was exactly the same as Goodall mentioned in a lecture—patiently and quietly to observe how the human organism behaves when it is entering and leaving this pathological phase.

I am indebted to W. Bjerknes, Professor of Physics and Aerodynamics at Oslo University, with regard to choice of method. As a young student I used to see his method of attacking his special problem of establishing relationships in weather phenomena, relationships as capricious and apparently incalculable as those of the so-called schizophrenic process. He applied a kinetic method of registration from hour to hour or from day to day, not only of a single factor such as barometric pressure or temperature, but of as many factors as it was possible to register in an objective manner. Similarly in an idiopathic or functional illness with unknown pathogenesis this kinetic method of registration of a number of representative functions seems to be the most appropriate. I am also indebted to H. A. Cotton, who will be known to this Association from his pioneer work on the significance of focal infection in endogenous psychoses. During a stay at Trenton I was able to gain a personal impression of the significance of chronic infection, at any rate in several cases. Without prejudicing the question of the role of infection in the causation of psychoses generally, I found that during the first year of my investigation, when focal infection in patients was either defectively eradicated or not at all, registration of metabolism repeatedly showed that chronic infection can interfere with the endogenous process conditioned by the illness and vitiate the results, so that its true characteristics are not recognizable.

METHOD.

Our method has been briefly the following :

1. *Eradication of focal sepsis.*—Registration of temperature and pulse throughout weeks and months, examination of the blood picture, clinical examination of the nose, throat, ears and teeth, X-ray examination of teeth and sinuses, and investigation of the gastro-intestinal tract, the thorough removal of focal sepsis, which

should be done in stages and in relation to the patient's physical condition. The effects of chronic infection will, as a rule, disappear during the course of a few weeks, frequently, however, only after months following detoxication, and its cure shows itself in the stabilization of the blood picture, pulse and temperature, with a certain and definite rhythm of both.

2. *Kinetic registration.*—During one or two months in which the patient is becoming adjusted to the new somatic conditions following defocalization, he is being trained to the new conditions necessary for metabolic investigation. He is put to bed in a single room, and receives a diet of definite and constant composition. This consists of milk, cream, eggs, sugar, Bemax, lemon, salt. The portion for 24 hours is divided into three main meals. He is taught, as far as possible, to relax completely during the measurement of oxygen consumption, and to save his fæces and every drop of urine. When this training has succeeded, as it usually does succeed even in the most excited or stuporous case, the actual research begins. A daily note on the mental condition of the patient is made, and a seismograph placed under the bed registers his movements day and night. *The food* is estimated daily for total nitrogen. *The urine* is daily examined for total quantity, specific gravity, total nitrogen, urea, ammonia, creatinine, titratable acidity, sulphates, phosphates, and chlorides. *The fæces* are estimated for total nitrogen. *The blood* investigations include blood-count and morphological picture three times a week: alkali reserve, total nitrogen and non-protein nitrogen twice a week. *The basal metabolism* is measured daily by the assessment of oxygen intake and carbon dioxide output over a known period.

Thus, in my opinion, the fundamental principles of technique are (i) complete removal and cure of all infection, (ii) kinetic registration of a number of representative physiological functions.

During the last twelve to thirteen years we have examined, with these methods, the metabolism of altogether 33 patients; these have all been male, in order to avoid disturbances due to menstruation. Of these there are 24 periodic catatonics, 6 paranoids and 3 belonging to other conditions. The periodic catatonic patients we have hitherto investigated seem to fall into two main groups according to their *mode of reaction:*

(i) The first group comprises the well-defined periodic catatonic patients, of whom some have shown phases of catatonic excitement, others phases of stupor. There is a total of 10 of this type between the ages of 14 and 52, who altogether have been investigated for a total of 3,500 days. The characteristic feature of the mode of reaction in this group seems to be that stupor or excitement begins suddenly and is pronounced, and that awakening from the stupor of the transition from excitement to a quiet phase sets in suddenly and is well defined. The changes in metabolism are synchronous with the changes in mental phase, and with one another, and show the same direction in vegetative regulation (syntonia). This mode of reaction I have therefore termed *synchronous-syntonic.* (Fig. 1.)

(ii) In the second group of 14 patients, who were examined for a total of 1,900 days, the transition to stupor or excitement is gradual. They pass little by little into a condition of mild stupor or moderate excitement and emerge slowly from these phases, but consciousness never becomes very clear nor are they very active mentally. These therefore do not strictly conform to Kraepelin's

group, but can be regarded as borderline cases. At their best they are apathetic, torpid, without interest or initiative. A large number of our catatonics show a course such as this. The periodic deviations in patients of this group are often

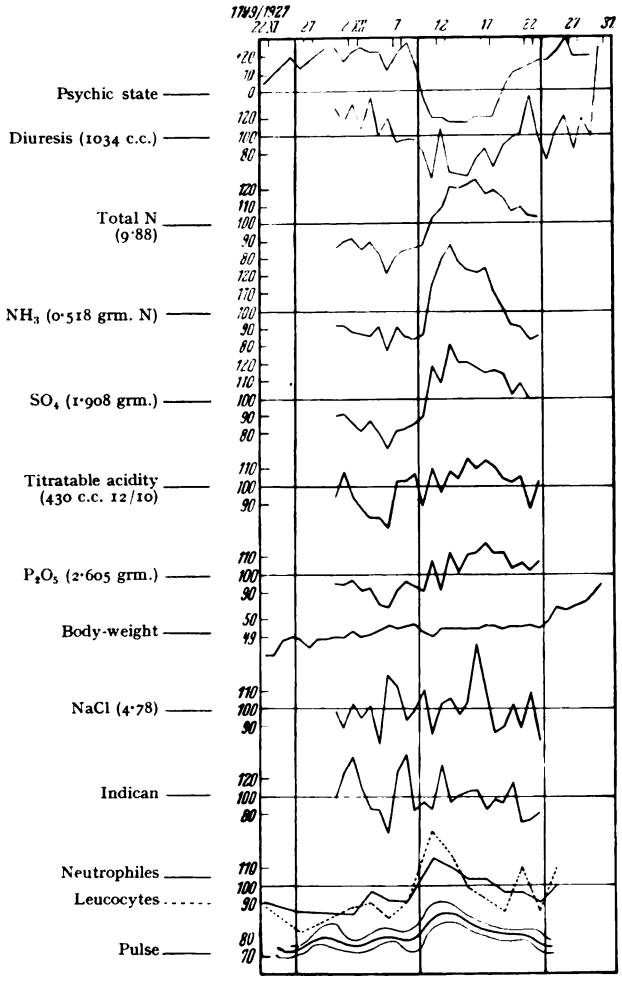

FIG. 1.—Periodic catatonic stupor. Boy, aged 14. Psychic state, diuresis, total nitrogen, ammonia, sulphates, titratable acidity, phosphates, body-weight, chloride of sodium, indican, count of neutrophiles, leucocytes, and pulse in one wakeful and one stupor period. Except the curves for psychic state, body-weight and pulse-count, the curves show the aberration from the main value in per cent. of the total period.

so ill defined that they are easily overlooked if one does not register the pulse, temperature, weight and mental state daily. What characterizes the pathophysiological conditions which are expressed in the metabolic changes are the split asynchronous and asyntonic mode of reaction. The alterations of the

various functions set in, not as in the previous group simultaneously and in the same direction, but singly and often in opposite directions, i.e., *asynchronous—asyntonic*.

In the following, I intend to give only a short description of the disturbance of function in patients who belong to the synchronous-syntonic group inasmuch

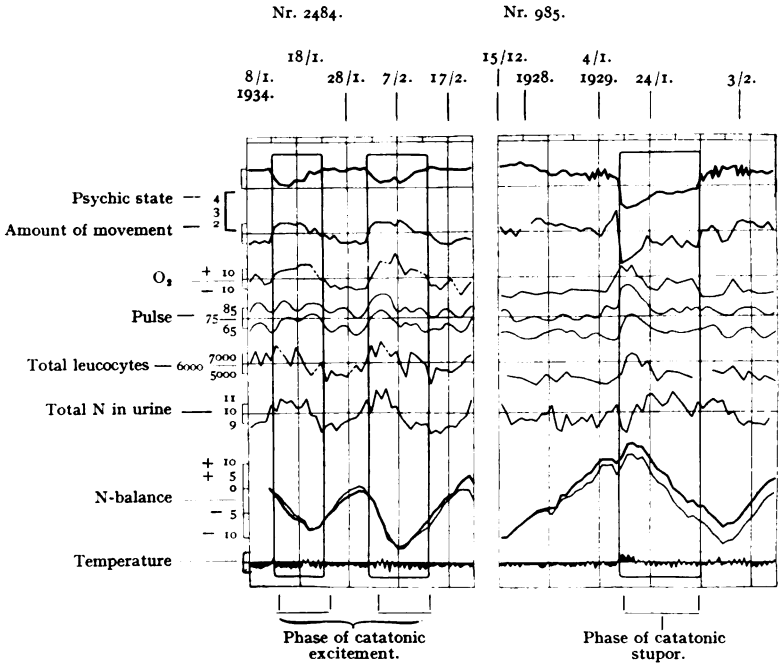

FIG. 2.—Collocation between the psychic and somatic features during periodic catatonic excitement and periodic catatonic stupor (to the left two periods of excitement, to the right 1 period of stupor). During the catatonic excitement the ability of concentration, C, scarcely decreases to the level of stupor, and the amount of movement, M, is increased. During the catatonic stupor C falls down far below the level of stupor, and after a short period of pre-stuporous excitement M drops nearly to akinesia. In contrast to this the course of O_2, pulse-rate, leucocytes, total N excretion in the urine, and the N-balance curve are very much the same in both cases.

as the typical morbid changes are here shown more clearly. In this group, the mode of reaction shows that the patho-physiological changes in patients with catatonic excitement correspond in the main with the findings in patients suffering from periodic catatonic stupor. (Fig. 2.) It is therefore not necessary to mention separately the course in these two conditions. I shall thus designate either the excited or stuporose phase as the *reaction phase*.

This group, with the synchronous-syntonic mode of reaction, shows from closer investigation two different *types of course* which I will, for the time being,

call *type A* and *type C* respectively. (The designating letter B has been reserved for a possible type not yet discovered.)

Type A.—In patients with this type of course there occurs at the beginning of the stupor or excited phase the following changes, which have much in common with Cannon's emergency reaction, i.e., a sudden rise in pulse-rate and blood-pressure (even if the stupor sets in during sleep, as we have found by a special method of seismographic recording). (Fig. 3.) The pulse may increase during the course of a few minutes or a few hours from 60 to 70 up to 110 or 120, and the systolic blood-pressure from 100 to 140, although the patient may be

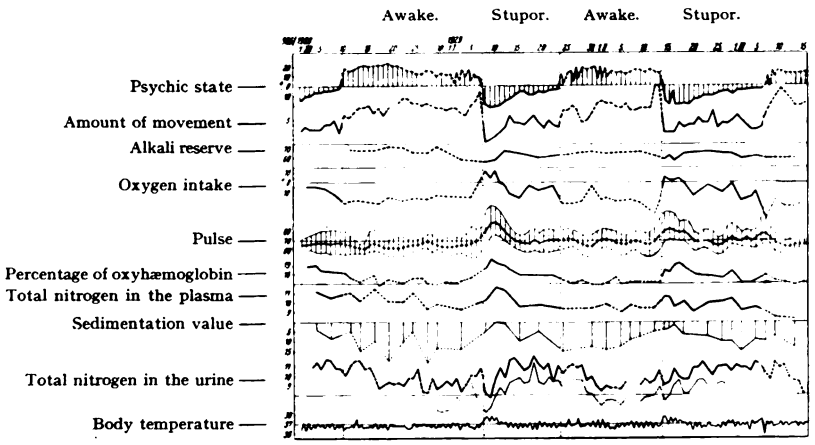

FIG. 3.—Periodic catatonic stupor. Male, aged 32. The figure shows two periods with state of awakeness and state of stupor. Psychic state, amount of movements (measured with seismograph), alkali reserve, oxygen intake, pulse-rate, percentage of oxyhæmoglobin, total nitrogen in the plasma, sedimentation rate (enlarged 2·5 times), total nitrogen in the urine, and body temperature.

resting quietly in bed. Simultaneously an adrenal-sympathetic impulse is felt in practically the whole vegetative field. There is pallor, with cold and clammy extremities, mydriasis, fleeting anisocoria, and in stupor as a rule a more or less pronounced muscular rigidity, fine tremor and retention of urine. These reactions, which in both types begin suddenly, give the impression of being *reactions to an irritant*, and we may summarize these disturbances of function in the term "*irritation symptom-complex*". At the beginning, particularly of stupor during the first few days, there is a certain lability in the vegetative field before the vegetative symptoms become more lasting and definitely sympatheticotonic. That the oxygen consumption increases in catatonic excitement is not surprising, but it is also increased in catatonic stupor, where the patient's seismogram shows that he has lain in bed motionless night and day. A sharp rise in the hæmoglobin content indicates the increased viscosity of the blood. Diuresis diminishes as well as body-weight. On the other hand the

blood sugar, both fasting and after glucose administration, is high, and reaches its highest levels during one of the first days after the beginning of stupor or excitement. Subsequently it diminishes gradually day after day, and during the free interval it is lower than normal.

If we now examine the total excretion of nitrogen in the urine and the nitrogen content of the fæces and of the food daily, so that we can draw a curve of the nitrogen balance, it appears that the excretion of total nitrogen with the same constant diet is lowered during the interval and increased in the stuporose or excited phase. (Fig. 4.) Less nitrogen is excreted than is assimilated in the stupor-free (or quiet) period, and the daily balance-sheet shows a positive

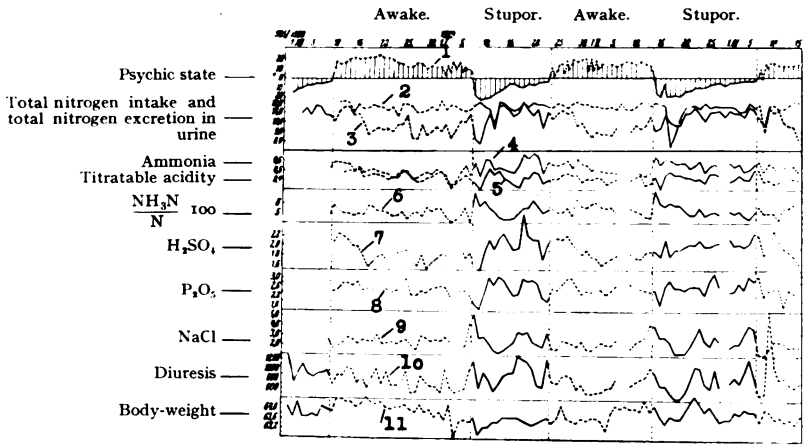

FIG. 4.—The same patient in the same periods as in Fig. 3. 1, Psychic state. 2, Total nitrogen intake. 3, Total nitrogen excretion in the urine. 4, Ammonia. 5, Titratable acidity. 6, Ammonia in percentage of nitrogen. 7, Sulphates. 8, Phosphates. 9, sodium chloride in the urine. 10, Diuresis. 11, Body-weight.

nitrogen balance. (Fig. 5.) The patient increases his store of nitrogen most probably in the form of " deposit nitrogen " (Sandiford and Boothby) until he reaches the threshold characteristic for him, usually between 15 and 25 grm. N. When this maximum has been reached stupor or excitement sets in. In a young patient of 14 stupor began the same day that he reached his maximum, and the transition from the waking state to stupor took about ten hours. With increasing age and duration of illness it seems that stupor or excitement only occurs some time—one or two days—after the excretion has begun. This circumstance is of importance, because it shows that it is not the excitement which causes the excessive excretion.

The nitrogen accumulation which has been stored up in the interval is now eliminated during the stuporose or excited phase. Where the elimination takes place rapidly the patient is finished with it whilst the excitement persists, and as soon as the bottom has been reached, so to speak, nitrogen retention sets in

even if the patient is still excited. This lasts during a free interval until the previously mentioned maximum has been reached, and excitement sets in again. In another case the elimination is continued for a portion of the interval, after which retention again begins. This shows that the retention is not dependent on the physical or mental excitement. The retention may begin during the excitement, and continues even if the patient has become perfectly quiet. What characterizes this group is that stupor or excitement sets in shortly after the nitrogen store has reached its *maximal* value.

Type C.—The type of course seen in these cases shows on the whole the same picture as far as stupor or excitement are concerned. (Fig. 6.) The excitement

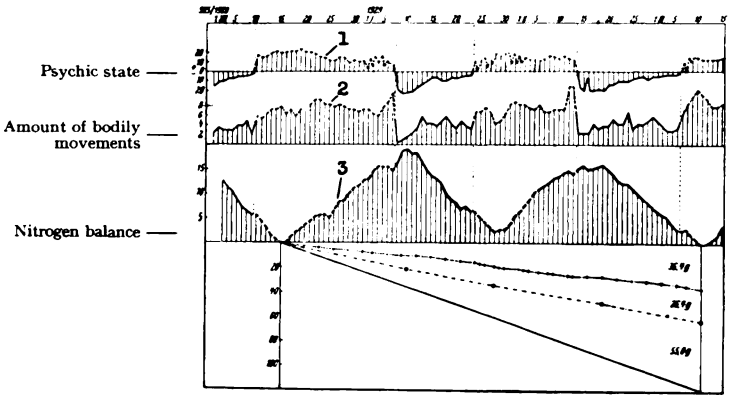

FIG. 5.—1, Psychic state. 2, Amount of bodily movements (measured with seismograph). 3, Nitrogen balance curve in the same period as in Figs. 3 and 4. As the figure shows, stupor sets in whenever the amount of retained nitrogen is about 15 grm. Probably due to the vegetative revolution, the over-excretion of nitrogen and the onset of the emptying of the storage of nitrogen is delayed for some few days.

begins with an increase of pulse-rate and blood-pressure, a steep rise of oxygen consumption, an increase which is now entirely conditioned by the motor unrest. These functions show more lability than in the " A " type. The *blood sugar* is also increased. During the quiescent interval the latter is abnormally high in this group. It increases still more during the first period of excitement, but falls in the latter half of the excited phase considerably below the values in the quiescent interval. As soon as the excitement is over, the blood-sugar values increase to the interval value again.

This type of case has the same tendency to N-retention and compensatory N-over-excretion as the previous A type, but in this C type, stupor or excitement sets in shortly before the termination of the negative N-balance, i.e., just before the *lowest point* in the trough of the wave. We therefore get the astonishing relationship that the reactive phase (stupor or excitement) of the disease in all its essentials coincides with a nitrogen retention. The quiescent interval begins when the store has reached its maximum. This type of course shows more

distinctly that the negative nitrogen balance does not arise on account of excitement and that the increased catabolism is not due to increased muscular contraction.

What more particularly distinguishes this last group clinically from the previous A type is a higher degree of mental and vegetative lability during the whole of the excited phase, and it seems as if the adrenal-sympathetic impulse has greater difficulty in asserting itself during a simultaneous nitrogen retention than during nitrogen excretion.

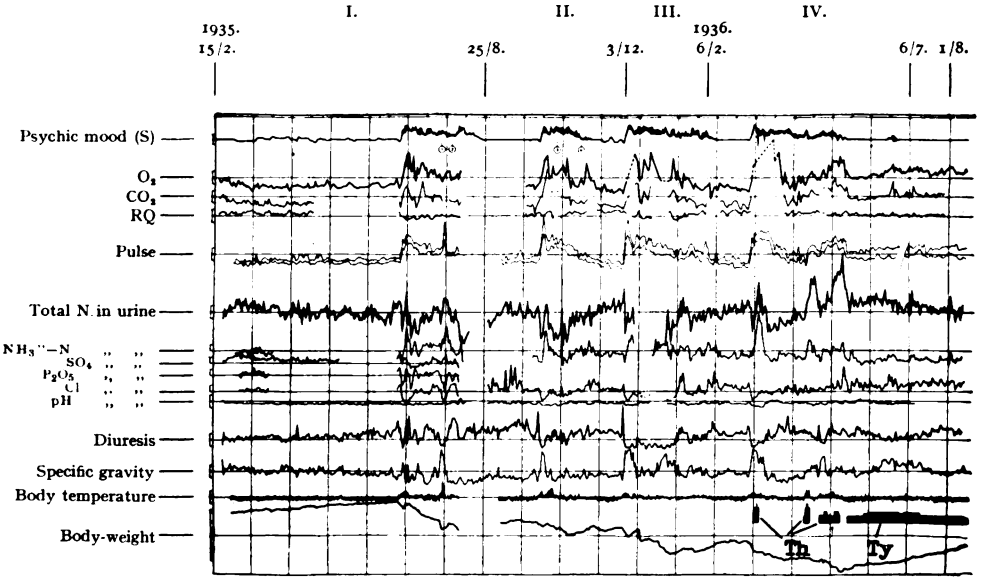

FIG. 6.—Periodic catatonic excitement. Type of course, C. No. 1928, C. J. L—, male, aged 58. The mood (S), O_2 intake, CO_2 excretion, R.Q., pulse-rate, total N, "NH_3"-N, SO_4, P_2O_5, Cl and pH in urine, diuresis, specific gravity, body temperature and weight, in the period 15.1.35 to 1.8.36. Observe the reduced total N-excretion in the urine during the excited phases. After the three first spontaneous excited periods (I–III) the patient in the fourth got (intra-muscular) thyrotropic hormone (Th., instead of thyroxin and with the same effect, afterwards dried thyroid substance (Ty).

This astonishing relationship (i.e., that stupor or excitement can begin in two different places—at the beginning or end of the negative N-balance) might well give reason for doubting the correctness of the analyses. We have, however, 3 certain cases of the C type and 7 of the A type, and our results are so uniform that they cannot be fortuitous. In addition the results are supported by a number of other independent investigations, of which I propose to mention a few. In type A, the *non-protein nitrogen of the blood* decreases during the whole of the interval, and reaches its lowest value with the maximum positive nitrogen balance (beginning of stupor or excitement). It then increases until it reaches the initial value at the beginning of the interval. In type C the non-protein

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nitrogen increases during the whole interval, and reaches its highest value at the beginning of stupor or excitement, and thereafter falls, to reach its original value at the beginning of the quiescent period. The non-protein nitrogen curve is thus opposite in the two types, and evidently only dependent on the nitrogen balance curve, but in a reverse manner. The *electro-cardiogram* taken during the retention phase shows the same characteristics as in mild anoxæmia and myxœdema with a completely flat T_i. During the phase of over-excretion a well-developed T_i occurs. A few days after the over-excretion has begun the electro-cardiogram is again normal; this holds for both A and C-type cases.

The values of the *impedance angle* also show a phasic displacement with the various phases of nitrogen balance. In the C type the highest negative values occur in the excited phase, not in the quiescent interval, indicating a resemblance to the hypothyroid state in this reactive phase.

The essential metabolic changes in periodic catatonia are therefore: (i) *periodic variation in nitrogen balance* with alternating phases of retention and over-excretion; (ii) the occurrrence of a reaction syndrome, which I have called the *irritation symptom-complex*, which shows many similarities with an adrenal-sympathetic impulse, and in which the mental state is abnormal. In addition, there are a number of somatic features contrasting with those of the quiescent interval; (iii) this *reaction syndrome* sets in partly at the *beginning*, partly at the *end* of the negative phase of nitrogen balance.

I will not attempt any further explanation than to state that it must be considered as possible that at these two times of maximum retention and maximum excretion, i.e., at the time of change of phase in a varying nitrogen balance, one or more toxic substances are formed which act, through the blood-stream, on the vegetative and myostatic centres of the diencephalon. Such a hypothetical substance occurring at both these times might reasonably be connected with *disturbances of protein metabolism*, and may possibly be a biogenic amine or a protein breakdown product of unknown or atypical kind. These may act as irritants in a similar manner to the action of foreign protein injected parenterally.

Time does not allow me to describe a number of conditions which strictly belong to a description of functional disturbances, such as alterations of acid—base equilibrium, water balance and creatine-creatinine metabolism. These functions are affected both by nitrogen balance and by the active phase (stupor or excitement) and together form a secondary *interference symptom-complex*, dependent on the relation between the reactive phase and the nitrogen balance phase.

An Attempt at the Compensation of Metabolic Disturbances.

In 1929 we noticed in a case under observation that the stupor period occurred every time the patient's nitrogen store had reached a figure of about 15 grm. N. This led us to investigate whether this nitrogen retention or nitrogen

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storage was causally related to the excitement or the stupor syndrome. Our hypothesis was somewhat as follows : the filling up of the store to its maximum is a condition for the formation of a toxic substance, which reaches the blood-stream and calls forth a central irritation, at any rate in the vegetative centres, with a stupor or an excitement as a reaction. It was therefore reasonable to suppose that if the store could be emptied and could remain empty, the syndrome of stupor or excitement should not occur.

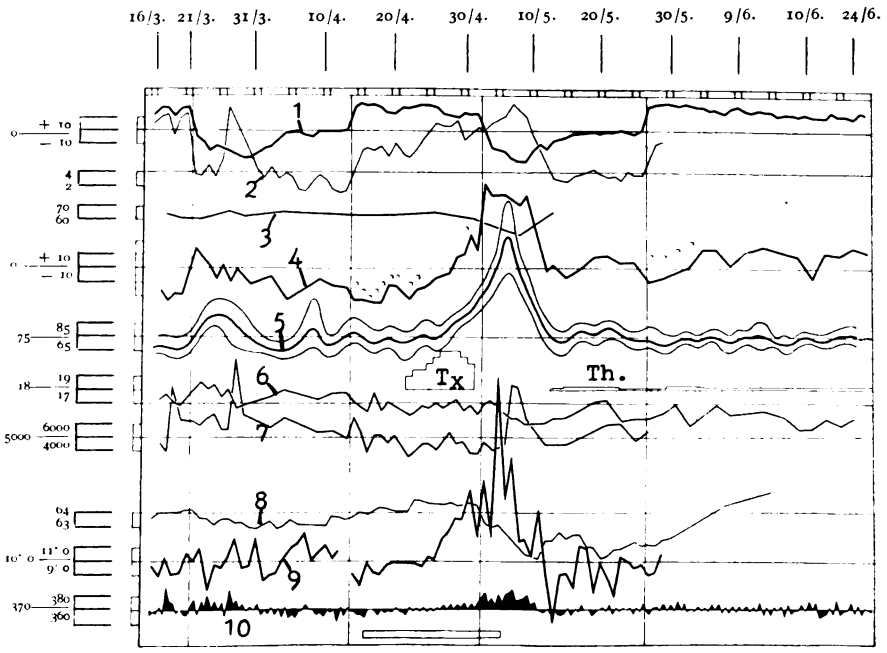

FIG. 7.—Periodic catatonic stupor. Type of course, A. No. 986, N. D. H— (the same patient as in Figs. 3, 4 and 5). Thyroxin (intramuscular medication) begins about 10 days before the beginning of the stupor phase is expected. 1, Psychic state. 2, Actogram (amount of movement). 3, Alkali reserve. 4, Oxygen consumption. 5, Pulse-rate. 6, Oxyhæmoglobin percentage. 7, Total white count. 8, Body-weight. 9, Total N in urine. 10, Temperature. Tx.= thyroxin. Th.= dried thyroid substance.

Thus we started using thyroxin in such large doses as to bring about a definite negative nitrogen balance (Fig. 7). In our first case a total of 44 mgrm. of thyroxin was given over a period of 8 days, and as a result the rate of nitrogen excretion rose to about 200% of the average. The pulse-rate increased to 180 with threatening collapse, but this was only of temporary duration and disappeared after a few days. When the pulse-rate had decreased to about 90 the patient received dried thyroid so that the oxygen consumption was stabilized at 10–15% above normal. The patient came out of stupor about 24 days after

completing thyroxin treatment, but whilst still on thyroid. The stupor period had then lasted just as long as the previous ones. The surprise, however, came as one month after another elapsed without any stupor phase, whereas the patient during the previous six to seven years had had stupor phases every month. The patient not only became stupor-free and accessible as in his previous intervals, but after a few months completely free of symptoms, with good insight and social feeling; he was keen and attentive to the work he was given. After discharge he took up nursing and X-ray technique, and since qualification has been appointed to our hospital as assistant in the X-ray and operating departments.

Since 1929 we have had occasion to examine several of these periodic catatonics, and have introduced the same compensation with thyroxin and thyroid as mentioned above. The experience we have gained has shown us that in those cases where there is a distinct periodicity in nitrogen balance, and where stupor or excitement sets in at the beginning or end of the negative phase of nitrogen balance, we can stop the mental and physical disturbances by a thorough emptying of the nitrogen store with thyroxin, and thereafter prevent retention by suitable doses of thyroid.

It must be emphasized that in this small, carefully selected group of cases it is possible by these means to *compensate a functional deficiency* but *not* to effect a *cure*. If retention is not prevented the nitrogen store is filled again during the course of weeks or months, and suddenly the catatonic stupor or excitement sets in as before and is repeated in series. However we have cases in which, after using the smallest effective dose of thyroid for some time, it appears as if the patient's own thyroid gland has been influenced and has been able to take over the entire work. As a result, without medication and on ordinary diet, the patient has been free from attacks for years. In young and recent cases this result is easier to attain than in older and more chronic cases, but even here the *possibility* of functional breakdown remains, and in that sense the patient is not cured. It can further be said that without thyroid medication in these cases remissions would not have occurred.

It is to be understood that after having seen the effect of thyroid administration in nitrogen retention, we have also applied this method of treatment in patients in whom we have not been able to carry out these thorough metabolic investigations. As a guide during the treatment we have only had a short daily description of the patient's mental condition, pulse and rectal temperature morning and evening, and body weight. In patients with a periodic catatonia, the active phases show themselves by a distinct increase of pulse-rate and temperature, together with diminution in weight, especially during the first few days of stupor or excitement. After following a number of these phases without active interference we have then, 8 to 10 days before the next active phase was due, given the patient thyroxin intramuscularly. We begin, for example, with 2 mgrm. on the first day and increase by 1 to 2 mgrm. every day for 8 to 10 days,

so that finally we reach a dose of 40 to 50 mgrm. Sugar or glucose about 150 mgrm. given daily in lemon squash. As long as the pulse is quite regular it is of little significance if it increases to 120 or 140. Should it become irregular, however, the thyroxin is immediately discontinued. When the pulse has again fallen to about 80 or 90 we give the patient thyroid gland in order to maintain the pulse at this level for a few weeks or months. In those cases where we had determined the basal metabolism during the quiescent period we tried to keep this at +10 to +15%. As a rule at this level of basal metabolism nitrogen retention is prevented. We have, nevertheless, also had a few cases where there has been a dissociation between the catabolic and the proteolytic response, and where an increase of the oxygen consumption as mentioned above has not been sufficient to prevent a renewed nitrogen retention.

The therapeutic effect in periodic catatonia with pronounced periodicity would appear to be that new phases do not occur, provided the maximum thyroxin action sets in at the beginning of the phase of negative nitrogen balance. In that type of case where stupor sets in at the beginning of the negative phase (Type A), the thyroxin should be given during the last 8 to 10 days before stupor or excitement begins. In type C, where stupor or excitement sets in at the end of the negative balance, thyroxin medication should begin shortly before the patient has become quiet or stupor-free. I mention this because a medication in the wrong phase as a rule leads to the occurrence of supernumerary phases of stupor or excitement, two or three in number after the principal phase has ceased.

This treatment has the great drawback that there is no *definite* method of controlling nitrogen level or anticipating nitrogen retention, other than by daily nitrogen estimations. However, since the treatment is only effective in this particular group of catatonia, where periodic disturbance of nitrogen balance can be shown by such estimations, the objection does not hold.

The *treatment with thyroid is not new*. It is a special pleasure to me that I have had occasion to mention these researches in your country where, already for many years, a number of workers have, in possibly a wider field, suggested the therapeutic value of large doses of thyroid (Leeper 1900, Dawson 1923, Molony and Minski 1927), and where Pickworth by examining the thyroids of dementia præcox cases found that the iodine content of these was half the normal. The therapeutic effect of thyroid has also been reported from other countries (Berkeley, Follies 1908, Hoskins and Sleeper 1929 and 1930). The latter, in a group of 130 dementia præcox cases, found a pronounced thyroid deficiency in 18, and achieved important results in 14 of them by thyroid medication. We, in our method, have used thyroxin, which seems to have a much less deleterious effect on the cardiovascular system. Its use in depleting the deposit protein in periodic catatonics is probably original.

The subsequent thyroid medication is analogous to the use of the maintenance dose of insulin in diabetes. We determine the smallest dose of thyroid which, in addition to the patient's own thyroid, is capable of keeping him free from nitrogen retention, month after month and year after year. It is of special importance to have clear indications for this therapy. In the instance of cases in the particular group of periodic catatonia, while they cannot be cured in the strict sense of the word, they can be rendered symptom-free.

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