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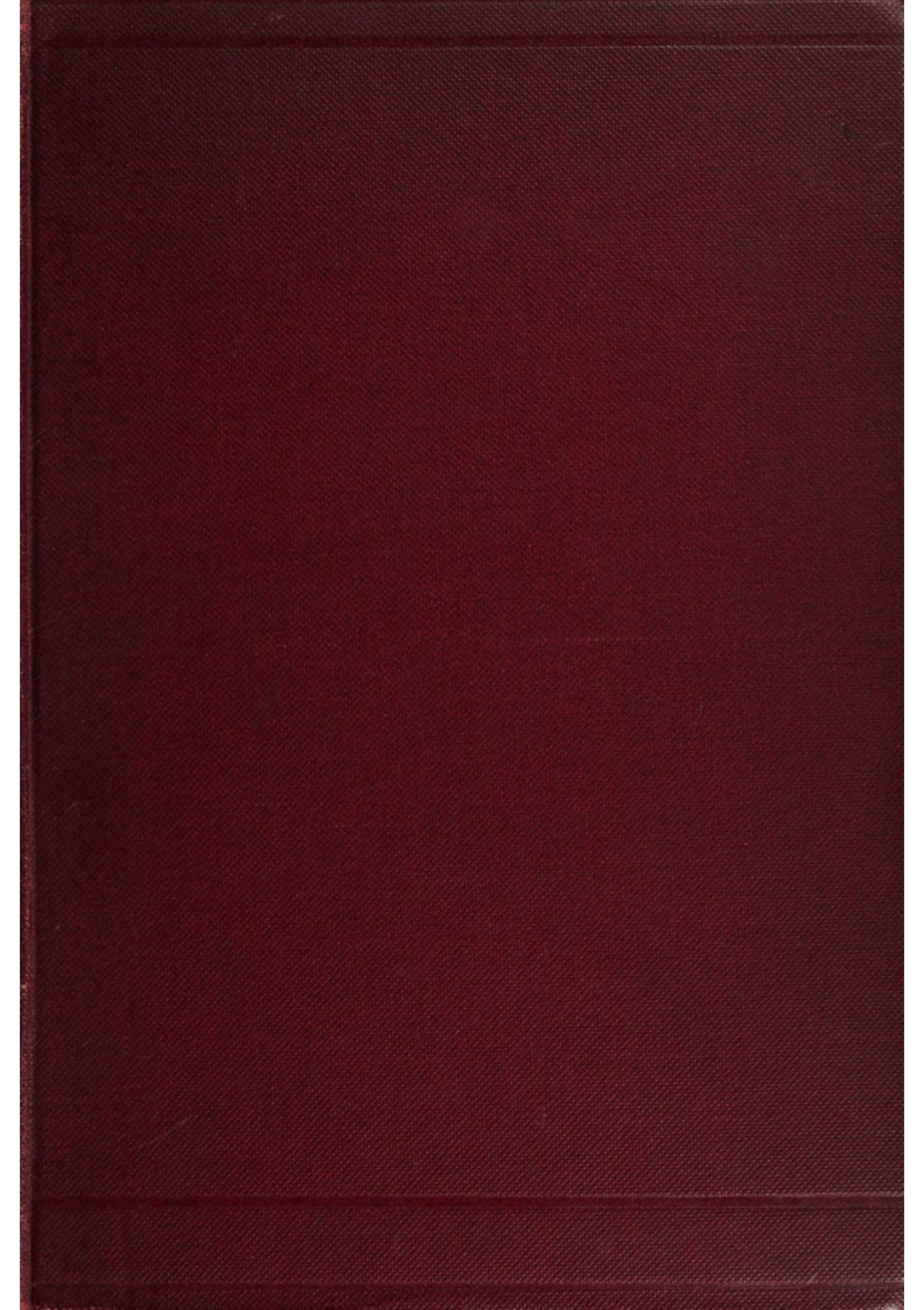
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STUDIES IN CLINICAL PSYCHIATRY





STUDIES  
IN  
CLINICAL PSYCHIATRY

BY  
LEWIS C. BRUCE, M.D., F.R.C.P.E.

London  
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## PREFACE

ADVANCES in the knowledge of mental diseases are slow, partly on account of the difficulty of the subject, and partly owing to the conditions under which these diseases are at present studied. I refer to the asylum system of treating the insane. Before a patient can be admitted to one of these institutions the disease process must be fully developed, with the result that the earlier stages can only be studied on very rare occasions. In such fully developed cases the mental aspect of the disease attracts attention by the prominence and bizarre character of the mental symptoms, and so it has happened that from distant times up to the present day the tendency to confuse the study of pathological psychology and the underlying physical disease has done much to prevent the advance of knowledge. When psychology is divorced from psychiatry, and the study of psychiatry is prosecuted along the lines of advance in general medicine, our knowledge of mental diseases cannot fail to be added to. The matter contained in the following pages is based on work so conducted: psychology is omitted; pathology, in so far as the changes in the brain cells and nerve fibres are concerned, is practically ignored, because until we know what the causes of such changes are we are none the wiser for such knowledge. The views advanced and

the evidence which is believed to support them are admittedly incomplete, but so much material had accumulated on my hands that I found it expedient to crystallise my views in type.

I am much indebted to Mr. H. J. Grierson, my colleague Dr. C. J. Shaw, and other friends, for their assistance in correcting the proofs.

*23rd May 1906.*

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PART I



## CHAPTER I

### THE PHYSICAL SYMPTOMS OF MENTAL DISEASES

THE following work is an attempt to bring the study of mental diseases into line with the more recent methods employed in the clinical study of general physical diseases.

It has always appeared to me that the study of the physical symptoms of mental diseases has been neglected, and that far too exclusive notice has been taken of the mental symptoms. The most generally accepted view of the onset of mental diseases is that they start *de novo* in disordered function or structural change in the brain cells and nerve fibres, and that the physical symptoms are secondary to and caused by the diseased condition of the brain. When this statement is tested by comparing the onset and physical symptoms in insanity with the onset and physical symptoms of diseases whose causation is beyond doubt, it will be found that very few cases of mental disease conform to such a view. In taking the history, for instance, of a case of phthisis, it is very common to find that the patient has a hereditary predisposition to tubercular disease, and we know that this hereditary predisposition does not mean that the patient has inherited tubercular disease, but that he has started life with a constitution deficient in the power of resisting the tubercle bacillus. In the same way we find among the insane that there is a strong hereditary predisposition to mental diseases, and because no distinct physical symptom can be satisfactorily demonstrated as the causal agent, ninety-nine out of a hundred alienists are willing to believe that the man with hereditary predisposition who becomes insane does so because he has



inherited what is called an unstable brain. He certainly has inherited a brain easily thrown out of gear, but in the light of what we know about other physical diseases, it is improbable that the insane man's brain becomes diseased *per se*, or through purely mental causes, such as worry or anxiety. There is a very close connection between hereditary predisposition to tubercular disease and insanity. Tubercular disease and mental disease occur commonly in different members of the same families who have either a hereditary predisposition to tubercular disease, or the neurosis, and the defective constitution which permits of the attack of the tubercle bacillus is apparently closely allied to that which favours the onset of mental disease. Two sisters of a highly neurotic family nursed their mother through a long and painful illness. The one developed phthisis, the other became melancholic. The medical man who attended these patients argued quite correctly that the anxiety, worry, and want of sleep that nursing entails had so lowered the resistive power of the one sister that she had become infected with the tubercle bacillus, and he ascribed the melancholia to the same causes, but it never seemed to occur to him that there was a physical cause, a disordering of metabolism directly due to the moral and physical strain which was the real factor in the production of the melancholia.

The proper nutrition of the body by means of regular meals of nourishing food without excesses of any kind is regarded as one of the best safeguards to the general health, and we know that so long as a man is in a well-nourished condition he is not specially liable to disease. Let the same man, however, lower his general health by deficient or improper food, or by excesses of food or drink, and he immediately lays himself open to the inroads of disease. The starved man or the drunkard is more liable to pneumonia, typhoid, or other infectious conditions than the man in a state of health. In the same way one finds that the patient liable to insanity is much less likely to suffer from an attack when his general physical health is good, than when he is thin, dyspeptic, and run down, and it is rare, indeed, to find a patient suffering from a first attack of mania or melancholia who is not in poor



condition, and who has not presented physical symptoms long before the mental symptoms brought his case into prominence. Among the poorer classes deficient food and irregular meals, and in some cases alcoholic excess, are just as much predisposing causes of insanity as they are of pneumonia, tubercle, typhoid, or allied conditions.

The effect of environment on the maintenance of health or the production of disease is also a factor recognised by the general practitioner, and there is no doubt that unhealthy environments at home or at work can act as predisposing causes of insanity by lowering the resistive powers of the body. The effect of healthy environments—fresh air, sunshine, and suitable occupation—in assisting towards the recovery of health, either in general physical disease or in insanity, is too well recognised to require further mention.

Some diseases—such as typhoid, small-pox, and allied conditions—appear to confer immunity upon the patient who has contracted them and recovered; others, such as rheumatism, gout, erysipelas, and tubercle, appear to confer no immunity, but the patient is often more liable to another attack. All mental diseases are allied to this latter class. One attack of insanity confers no immunity after recovery, but rather renders the patient liable to further attacks. The insane are particularly liable also to contract diseases of the allied class, especially tubercle, rheumatism, and erysipelas.

One of the most difficult things to obtain in public asylums is a reliable account of the onset and course of the patient's illness, because the patient is often brought by an official who knows nothing of the history but the barest facts. When a good history can be obtained of a recent acute case of mental disease, there is a striking similarity between the details of that history and the details of a history taken in any general hospital, and in the vast majority of cases physical symptoms have preceded the mental symptoms often for weeks and months.

The general appearance and expression of the face in the acute forms of insanity resemble very closely those seen in acute physical disease, and the variations from the temperature of health are just as marked in insanity as in other physical



diseases. The characteristics of the temperature in the various forms of insanity will be described in detail under each disease.

**Alimentary System.**—The alimentary system appears to be peculiarly liable to disorder in many forms of bodily disease, and it is equally if not more liable to disorder in patients suffering from insanity. Among the inmates of our public asylums carious teeth are the rule—carious teeth are also the rule among the same class outside of asylums; but there are good grounds for believing that oral sepsis is in some patients the starting-point of a condition of toxæmia which may end in insanity in those predisposed to nervous diseases. The removal of carious teeth in such cases is followed by marked improvement in health. The furred or coated tongue of the dyspeptic is nearly always to be seen in patients suffering from acute melancholia, mania, and allied conditions; and the dry, brown, cracked tongue met with in conditions of exhaustion and acute septicæmia can be seen in patients suffering from insanity which has passed into the typhoid state. The desire for food is lost in all forms of acute insanity, and when consciousness is impaired thirst is also lost, so that these patients frequently require artificial feeding. The loss of appetite in acute insanity is associated with a failure of the digestive power of the stomach juices. I have seen patients suffering from acute mania and melancholia vomit milk unchanged, although the milk had been given two hours previously. The gastric fluid of cases of acute melancholia when withdrawn by a stomach tube has no digestive power at all when tested *in vitro*, and the gastric fluid in the early stage of acute mania has little or no action when similarly tested. Persistent vomiting, flatulence, and constipation may precede or accompany any of the acute insanities.

**Hæmopoietic System.**—The work of Horseley and others has shown that the functions of the thyroid gland have important bearings on the subject of nervous diseases. It has been shown experimentally on animals, that when the gland is removed a condition similar to the disease myxœdema in man is produced. Further, it has been proved that if patients suffering from myxœdema are treated with thyroid



extract marked improvement or recovery follows. The chief mental symptom connected with myxœdema is a condition of hebetude. Certain conditions of insanity, although presenting none of the physical symptoms of myxœdema, are apparently due to either deficient thyroid secretion or alteration of secretion, for they also react in a specific manner to thyroid medication. This treatment is at present empirical, as cases suitable for the treatment cannot readily be distinguished, but I have seen the same case recover time after time from recurrent attacks of insanity under the administration of thyroid extract.

Overaction or altered secretion of the thyroid gland is also recognised as a cause of mental symptoms, varying from excessive nervousness and irritability to mania and melancholia. A similar mental condition is sometimes induced by overdosing with thyroid extract. I have several times seen acute mania supervene upon a condition of stupor as the result of large doses of thyroid extract.

As the results of comparatively recent researches, it is now known that certain infective conditions and toxæmias are followed by changes especially in the white corpuscles and serum of the blood. In many infective or toxic conditions the white corpuscles are increased in number, and this increase is chiefly in the polymorphonuclear or neutrophile leucocytes. The leucocytosis in health is below 10,000 per cubic millimetre of blood, and the percentage of polymorphonuclear leucocytes is about 70. In toxic conditions, especially of bacterial origin, the leucocytosis may rise far above 10,000 per c.mm., and is then spoken of as a hyperleucocytosis; and the polymorphonuclear percentage according to the acuteness of the toxæmia or the vigour of the reaction of the patient to the toxine is also increased and may rise above 90. The same symptom is found in many acute insanities. In acute mania the early leucocytosis may be 30,000, or even higher, per c.mm., and the polymorphonuclear percentage may be 80 or 90 according to the severity of the condition. The same observation applies to excited melancholia, katatonia, folie circulaire, some stages and complications of general paralysis, and the mental symptoms associated with epilepsy. The



leucocyte changes are more fully described under a separate chapter.

Whenever the mammalian body is infected, either naturally or by artificial means, with any toxic substance it immediately protects itself by forming an antibody to that substance, and when the toxic substance is cellular or organismal along with the antibody, there may also be formed a substance known as an agglutinine, from the fact that the serum of the infected animal has the power of agglutinating or clumping the specific organism or cellular element, causing the infection, when mixed with it in certain proportions *in vitro*. This reaction is now commonly used in the diagnosis of typhoid fever (Widal's reaction), in Malta fever, and some other diseases. In the serums of those cases of insanity which present the symptom of hyperleucocytosis one also very commonly finds various agglutinines present. For the last two years I have examined the blood in every acute case of insanity coming under my care. A small exploring needle is passed with antiseptic precautions into some prominent vein in the forearm, and from 5 to 10 c.c. of blood is then allowed to run into a flask of sterile nutrient broth. This is then incubated at 37° C. for three days and subcultures made on agar. Only two cases gave positive results. One, a case of acute mania in a typhoid state, gave a pure growth of a small streptococcus, and the other, a case of katatonia, also yielded a streptococcus somewhat larger. I have tested the serum reaction of 23 cases of mania to both these organisms in dilutions of 1 to 30, and have obtained definite agglutination of the organism obtained from the case of acute mania in 19 of these cases. The serum of 6 cases of excited melancholia gave agglutination to the same organism in 3 cases. The streptococcus obtained from the case of katatonia gave definite agglutination in 12 cases of that disease, while 6 cases failed to give the reaction. In every case control serums were also used, and no agglutination was ever obtained with them to these organisms. The serum of general paralytics, also, very commonly contains agglutinines to the bacillus coli communis, and, if the patient is maniacal, to the streptococcus of mania as well. In the case of the organisms which are known to produce agglutinines,



such as the typhoid bacillus, it is recognised that many of these organisms effect a lodgment in the tissues of the patient. There is at present no proof that this occurs in cases of mania and excited melancholia.

The following experiment, however, proves that it is not necessary for an organism to effect a lodgment in the tissues to produce agglutinines in the blood serum, provided that a sufficient quantity of the live organism passes into the alimentary canal. The serum reaction of the blood of two adult rabbits was tested to the micrococcus found in the case of mania; in dilutions of 1 to 20 and 1 to 30 there was no agglutination obtained, and the coccus grew readily in dilutions of 1 to 3 of the serum when placed in the incubator at 37° C. The animals were then sprayed with live cultures of the organism daily for a month. The rabbits licked themselves, and so infected the alimentary tract. At the end of six weeks the serum reaction was again tested, and agglutination of the micrococcus was obtained in less than half an hour in dilutions of 1 to 50 and 1 to 100. In dilutions of 1 to 3 of the serum placed in the incubator at 37° C. for 24 hours there was arrest of growth of the organism. A third rabbit then received a dose of 4 c.c. of the live broth culture by the mouth. Two hours later it was killed, and cultures made from the stomach and small intestine. The stomach was sterile, but the small intestine gave a growth of *bacillus coli communis*.

It is evident, therefore, that in the production of such an agglutinine a healthy animal has only to digest the organism in the stomach.

I have bacterially examined on two occasions the stomach contents of cases of acute mania with acute toxic symptoms two hours after food had been taken. Microscopically in films made from the fluid two varieties of cocci—one large and the other small—were seen in great numbers, and many of the large squamous cells present were full of organisms. There were also present a streptobacillus and the *bacillus subtilis*. In plate cultures made in 4" Petrie's capsules from the stomach fluid, only two colonies of organisms were to be seen at the end of 48 hours' incubation at 37° C.



It is possible, therefore, that the infection may in these cases be from the mouth, nasal passages, pharynx, or œsophagus, and that the organisms are destroyed by the gastric juices even of a case suffering severely from acute mania. There is, however, post-mortem evidence that the large intestine may be also a seat of infection. Ford Robertson has pointed out that atrophic changes in the mucous and submucous coats of the large intestine are common in all forms of acute insanity which present symptoms of toxæmia, and also in cases of general paralysis.

I have examined the red bone marrow in six cases which during life presented the symptom of hyperleucocytosis, and in every case the marrow was leucoblastic, and the cells were undergoing active mytosis.

**Circulatory System.**—Valvular lesions of the heart are more common among the insane than the sane population, and failure of competence, especially in old people, frequently seems to produce mental symptoms; but the symptoms which are most frequently to be seen in acute insanities are altered arterial tension, irregularity in force and rhythm, and also increase of pulse-rate without any temperature changes. The arterial tension in health is between 100 and 120 mm. of mercury as tested by Barnard and Hill's sphygmometer. In states of mental depression the arterial tension is raised sometimes as high as 160 to 170 mm. Hg., and in many cases of excitement the tension may be below 100 mm. Hg. Pulse changes are best seen in cases of acute melancholia, when a pulse-rate of 120 per minute may be associated with a temperature of 99° F., or even lower. At the onset of acute mania of any variety a fast, weak pulse of over 100 per minute with a subnormal temperature is one of the earliest symptoms. In cases which have passed into a condition of stupor, on the other hand, the pulse-rate may fall below 50 per minute, and the peripheral circulation is markedly impaired, the hands, feet, and ears being livid and cold, the feet being frequently œdematous.

**Respiratory System.**—In common with all conditions which produce lowered vitality the insane are liable to phthisis. Ford Robertson, McRae, and Jeffrey have recently



pointed out that in general paralysis the lungs are frequently infected with a bacillus, which when passed into the alimentary tract of rats produces a slowly advancing paralysis and death. I repeated the experiment on a goat, and the animal died more than one year after infection with paralytic symptoms.

**Integumentary System.**—Subjective sensations in the skin are not, as a rule, complained of by patients labouring under acute forms of mental disease, but irritable conditions of the skin, and especially of the scalp, must be present in many cases if one may judge by the number of patients who pull out their hair, who constantly rub the scalp, and who, whenever they get the opportunity, wash their heads. The cause of these symptoms is as difficult to explain in insanity as in ordinary conditions of disease.

A dry, harsh skin is seen in melancholia, where there is deficient excretion of the waste products of the body, just as one finds dry skins in all uræmic conditions. Drenching perspirations may occur in the acute stage of katatonia, in exhaustive conditions, and sometimes as a crisis in acute mania, closely resembling the perspirations of rheumatic fever and pneumonia. After any acute disease, desquamation may be a common sequel, and there is no exception to this rule in insanity. I have seen cases of mania during convalescence desquamate similarly to a scarlet fever convalescent. The appearance of the skin in insanity depends, as in other diseases, upon the general health of the body, and the improving colour of the skin and its freedom from undue pigmentation are always regarded as hopeful signs of recovery, especially in cases of melancholia. During the acute stage of what I designate toxic insanities—acute mania, excited melancholia, katatonia—skin rashes are frequently to be seen. These rashes may take the form of red blotches,—in some cases due to rupture of superficial capillaries,—papules, or pustules. A crop of boils occurring during the acute onset of mania is regarded as of good prognostic import.

In all acute and chronic insanities the hair tends to be dry, harsh, and brittle, and in some cases of mania assumes a more or less erect position. The nails also suffer in the general malnutrition, and may become brittle and deformed.



The transverse notch to be seen during convalescence from acute physical diseases may also be seen in patients recovering from acute attacks of insanity.

**Urinary System.**—The frequency with which urine is voided in cases of acute mental disease depends on—first, the quantity of urine being secreted; and, secondly, on the centre of consciousness in the brain being in a fit state to receive sensory impulses from the bladder. In all cases of acute melancholia little urine is voided because little urine is secreted. The act of micturition may only be performed once in the 24 hours, or in extreme cases once in 48 hours, and the amount of urine passed may be only 3 or 4 oz., or more commonly 10 or 12 oz. In acute mania, on the other hand, or any mental disease which implicates consciousness, the act of micturition may be purely reflex, and though only performed once or twice during the 24 hours, it is not due to any failure in the excretion of urine, but to failure of the sensory areas in the brain cortex to appreciate that the bladder is full. The same conditions are to be met with in ordinary physical diseases due to practically the same causes. In ordinary clinical observation the chief changes to be found in the urine of those suffering from acute mental disease are—(1) A deficient excretion of urea in conditions of mental depression; (2) The presence of albumen, especially in acute melancholia and mania, the albuminuria being of irritative origin, for numerous leucocytes can be observed by microscopic examination; (3) A deficient excretion of chlorides in certain toxic conditions.

**Reproductive System.**—It has long been recognised that the testicles and ovaries have an influence on the nutrition and development of the body and mind, and the removal of these glands in either sex before development is complete will arrest entirely the sexual mental characteristics. It is quite possible that conditions of deficient or altered secretory activity may coexist with the presence of these glands in an apparently, but not really healthy state, and this may account for the symptoms of arrest of physical and mental development so commonly seen in insanity occurring during puberty and early adolescence. Nothing definite, however, is known about



the cause of these changes, and it is purely hypothetical to ascribe them to failure in the activity of the reproductive glands. Insane women, especially adolescents, are liable to amenorrhœa, and to relapses and exacerbations in their mental symptoms during the period of menstruation. Certain forms of insanity have been ascribed to disordered ovarian and uterine function, such as "ovarian insanity" and "old maid's insanity," but there is no definite proof that they are even associated with such disorders. There is no doubt, however, that in adolescent women recovery from any form of mental disease cannot be considered as complete until the menstrual function has become regular and healthy in its occurrence. Far too much importance has been and is attached to the symptom of eroticism which frequently occurs in both sexes during periods of excitement. This symptom chiefly occurs in cases where the higher controlling centres of the brain are impaired by disease; and as man deprived of his higher centres is after all only an animal in his instincts and appetites, it is not surprising that the lower centres come more into evidence.

**Nervous System.**—The special system affected in mental diseases, and the system from the clinical study of which one might expect to obtain the most information of all, is unfortunately the one most difficult to examine, because the patient is generally unable, owing to mental impairment, to render the examiner any assistance. It is impossible in acute mental disease, where consciousness is impaired or abolished, to say whether there are any subjective sensations present; and although such patients may be completely anæsthetic to pain of all sorts, this may be accounted for by the fact that the centres of conscious sensation are not in a state to receive or interpret sensory impressions. In certain forms of delusional insanity—especially those due to alcoholic excess—sensations of pricklings, tinglings, and formication are present, and are interpreted by the patient as "electrical" agencies, "marconigrams," or the presence of vermin in the bed-clothes or dress, and it is probable that some of the unexplainable symptoms and insane conduct of those acutely insane patients may be due to similar paræsthesial sensations. In the few cases which can be examined, one frequently finds impairment of



the power to distinguish between the prick of a pin and contact with a hot test-tube, or the sensibility to cold may be lost or hyperacute, or the muscular sense may be deficient; but these symptoms are in no way associated with any one type of disease, but are dependent on the patient's mental state at the time of examination, and are therefore of no assistance in diagnosis. The sense of sight may vary enormously in the same patient in different stages of such a disease as *folie circulaire*. During the stage of excitement the patient may be able to read the smallest print without glasses, but as the stage of depression sets in the use of glasses of gradually increasing strength are necessary to enable the patient to read at all, as pointed out by Clouston. In the same way the sense of hearing is hyperacute in certain maniacal conditions, and the senses of taste and smell may lose their delicacy, or become impaired, so that the patient becomes coarse in his appetites, or the sense organs may be so disordered as to give rise to hallucinations and delusions of poisoned food, or the presence of poisonous gases in the room. Irregularity of outline or of size in the pupils is very common, but not confined to cases of general paralysis. Inequality in the size of the pupils is certainly common in various forms of insanity, especially insanity of long standing. The pupils are, as a rule, widely dilated in conditions of exhaustion, excitement, or fear, and the reflexes to both light and accommodation are sluggish. Pin-point pupils may be observed in cases of general paralysis, in conditions of excitement where there is super-added the emotion of anger or rage, and in senile dementia, where there are probably advanced vascular and fibrous changes in the brain cortex. The organic reflexes of micturition and defæcation are out of control of the higher centres in all cases where consciousness is impaired or abolished, as in conditions of acute mania, the onset of katatonia, and in cases of general paralysis during and after congestive seizures. In advanced dementia wet and dirty habits are mostly the result of the loss of the ordinary instincts of cleanliness, as such patients can be re-educated to more cleanly and human customs.

Swallowing is impaired, sometimes lost, in the last stages of general paralysis, owing to impairment of sensibility and



advanced muscular paralysis. In the resistive or stuporose stage of katatonia the passive resistiveness generally extends to the reflex of swallowing. Such patients allow saliva to accumulate in the mouth, from whence it constantly dribbles away, but the reflex is not in any way abolished, as it is the custom to feed such cases by hand.

The superficial reflexes in cases of mental disease follow the usual laws which govern their activity in nervous diseases. They are increased in cases in which the control of the cerebral centres is diminished, as in acute mania or the onset of any acute insanity which implicates consciousness. They are diminished when there is interference with the reflex loop, as in cases of alcoholic poisoning, or where there is increased cerebral inhibition, as in some states of stupor. The deep reflexes do not conform to the rule that increase accompanies impairment of consciousness, because there may be no increase of the knee-jerks in acute mania or any of the conditions which implicate consciousness. All the tendon reflexes are, as a rule, increased in general paralysis and the stuporose stage of katatonia and long-standing cases of epilepsy. They are diminished or lost in cases of alcoholic or other poisonings which interfere with the reflex loop. As a diagnostic symptom these reflexes are only of use when taken in association with other nervous symptoms in cases of general paralysis, katatonia, and chronic alcoholism. In no form of insanity is there paralysis of the voluntary muscles, with the exception of general paralysis, organic and secondary dementia, and certain forms of senile insanity. The sluggishness of movement and the disinclination to move seen so frequently in conditions of melancholia are due to a failure of energy in the volitional centres. There is, so far as can be demonstrated, no implication of the motor tract. Inco-ordination of movement, on the other hand, is common to all acute insanities, and may vary from failure of fine associated muscular movements, such as are used in games of skill, to the wild, purposeless inco-ordinate movements which are seen in acute states of mental excitement. There is a close connection between the motor and mental functions. Upon examination, it is seen that these associations of the motor and mental activities



become so complex and so inextricably blended with one another, that the conclusion is forced upon us that the motor centres underlie all conscious mental manifestations.

In the condition of sleep, where consciousness is lost and cerebral activity is relegated to the lower and reflex motor centres, the muscular tonus is lost, and the whole muscular system is relaxed. No sooner, however, does sleep give way to ordinary consciousness than the muscular tonus returns—in other words, the brain cortex sends down a steady flow of energy, and a consciousness of motor power returns with the knowledge of real existence. This is certainly a variety of motor mentalisation of which we are often keenly aware, when it is deficient in quality or quantity—when, as one terms it, we feel out of form—the muscular relaxation produces or is a part of the feeling of mental lassitude and disinclination to motor activity. On the other hand, when the subject is in vigorous health the adjustment between muscular potentiality and thought is so perfect, that the readiness and capacity for movement are represented in the very attitude of the body.

The act of voluntary attention has two motor equivalents, the one inhibitory, the other excitatory. These motor actions are complex and affect even the cardiac and respiratory reflexes. During profound attention the respirations become slower and deeper, the cardiac systole is more prolonged. Together with a generally firm state of the skeletal muscles, the muscles of expression are in characteristic action. The orbicularis muscles are in tension, and the eyes are directed by co-ordination of the ocular muscles towards the object exciting the attention. Further, the eyes, in transmitting impressions of objects to the brain, involuntarily trace the outlines of these objects, and the sensations thus originated tend to fix the impressions in the receiving cortex.

Even memory has its motor representation, as the act of remembering implies the reproduction of an image, as, for instance, where the eyes trace insensibly the outlines of words, letters, phrases, scenes and persons, and the resulting brain sensations, by stimulating the association of ideas, assist in recalling the representation of the desired subject.

Will power implies motor, sensory, and mental actions.



To think of an act is to set in nascent motion the muscles about to be actively employed, and to inhibit that act is to control the motor activities. In forming a mental judgment we have to exert attention and memory, and it has already been demonstrated that there is motor action involved in the discharge of these functions.

Speech without motor action is, of course, an impossibility, and the power of speech is furthered and accentuated by muscular movements of the body and limbs. Control by mechanical means the motor activities of an orator, and at once you obstruct his flow of thought and power of expression. Without their motor equivalents the expression of the emotions would be impossible. It has been demonstrated that in hypnotic states varying emotions can be induced in the subject by the operator, who, by placing the muscles of the subject in suitable combinations and positions, suggests the required emotion. The mental act is, in short, induced by means of the motor equivalent.

Every action and position of the healthy subject is pregnant with potential activity. The expression, the attitude, sitting or standing, the walk, convey to the observer the mental equivalent of the subject observed.

If such is the case in health, it is not wonderful that in disease, and especially in mental disease, the motor functions should suffer in proportion, and sometimes out of all proportion to the mental functions. This is, of course, a wide subject; it will, therefore, be sufficient for purposes of illustration to observe more particularly some of the motor changes which occur in melancholia. A glance at a patient, the subject of mental depression, suggests a condition of lowered vitality. The face lacks variety of expression, the facial muscles are flattened and relaxed, and, especially in the lower third of the face, pulled down, acted upon by gravity. The attitude is slouching and wanting in vigour. The walk is dragging and apathetic, and every movement is lacking in the finer co-ordinations. Although consciousness is present, the muscular sense, the feeling of self-confidence inspired by a healthy tonus of the muscular system, is defective. The attention when attracted is incapable of concentration, and the muscular



equivalents of expression vanish with the mental failure. Memory is deficient, and cannot be assisted by motor action. Speech is slow and halting, and a deaf observer could draw but little inference from the negative activity of the muscles of expression. When we come to more complex movements, such as writing, playing billiards, tennis, golf, or any act which implies fine co-ordination, we find a failure of power. Nor do the cardiac and respiratory functions escape the general lethargy. It is the exception to find a melancholic patient whose cardiac systole is not weakened or whose respirations are not shallow and feeble.

We have seen in health that in addition to motor innervation there is motor inhibition, and this power of inhibition we may also find impaired in melancholia. Involuntary muscular movements are apt to occur without apparent reason or external stimuli. It is admitted that weakened cells, in addition to giving deficient energy, can also discharge too easily, so that motion when initiated is apt to lead to dissipation of energy.

**Vasomotor and Nutritive Functions.**—Inequality of the body temperature in the axillæ, so commonly seen in cases of hemiplegia, occurs in general paralysis after congestive seizures which are more or less unilateral in distribution; but this inequality is also to be seen in cases of katatonia and melancholia, where there is no reason to suppose that one side of the body is implicated more than the other. The extremely low temperatures to be seen in mania, excited melancholia, and katatonia, after the period of onset is past, are probably due to disturbance of the vasomotor functions. Flushing of the skin, especially over the malar bones, is a precursor of mania; while pallor of the skin with lividity of the extremities is a characteristic of stupor and dementia.

The drenching perspirations seen in acute insanity are probably reflex in origin, and may in some cases accompany the emotion of fear—the fear being the outcome of terrifying hallucinations or delusions. The perspirations which occur during the crises in mania and melancholia are more probably an effort of nature to eliminate poisons. Unilateral perspiration is common in the stuporose stage of katatonia, and



comes on as a rule after taking food. I have seen such cases in which, on one side of a line drawn from the vertex to the chin, the skin was dry, on the other perspiration stood in large beady drops.

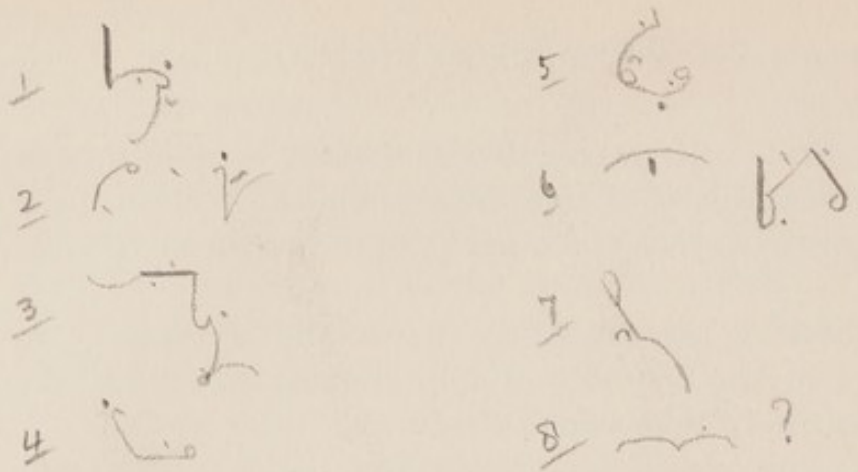
The lachrymal secretion may be totally suppressed in melancholia, and the first sign of improvement in such a case may be a return of the power to shed tears.

Excessive and modified secretion from the salivary glands has long been recognised as occurring in connection with mental diseases, but there appears to be no association between such symptoms and any special type of insanity.

The albuminuria so commonly seen during the acute stage of onset of mania and melancholia is most probably of irritative origin, but the transient glycosuria which occurs after epileptic fits and during the course of delusional conditions is probably vasomotor in origin.

The mental symptoms necessarily bulk very largely in the description of mental diseases; and in the description and diagnosis of the majority of insanities, however important the physical symptoms may be, it is impossible to make the diagnosis without reference to the mental symptoms also. On the other hand, to entirely ignore the physical symptoms, and to endeavour to make the diagnosis from mental symptoms alone, as so many alienists do, cannot be correct, because in the light of what we know of the idiosyncrasies of various brains, and the widely varying mental symptoms which can be produced by the action of a known toxine, such as alcohol, on the brains of different persons, such a method of diagnosis is fallacious. Three men, let us say, each take an overdose of alcohol; one becomes depressed and emotional, the second becomes excited, violent, and destructive, while the third passes into a state of stupor. Is it reasonable to make each of these mental states a disease, and call the one melancholia, the second mania, and the third stupor?

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## CHAPTER II

### THE MENTAL FUNCTIONS

THE two most common departures from the state of health to be noted in Mental Diseases, are conditions of morbid depression, and elevation or excitement. These states, always dependent primarily upon functional disorders of the brain cortex, are frequently aggravated by false stimuli arising in the special sense centres, particularly those of sight and hearing. A case of acute melancholia, let us say, is always depressed, but the depression is at times accentuated and associated with the emotion of fear and sometimes delusions, when vivid auditory hallucinations are added to the symptoms. On the other hand, the depression of folie circulaire is more uniform and entirely of central origin, as these patients rarely suffer from hallucinations.

**Elevation and Excitement.**—Elevation and excitement are frequently used synonymously in the description of maniacal states, but true elevation or mental exaltation is only to be seen in mental diseases uncomplicated by confusion, such as the elevated stage of folie circulaire, and is always associated with buoyancy of spirits and a feeling of well-being. Excitement, on the other hand, more correctly describes the combination of mental and physical symptoms to be met with in the early onset of any of the acute insanities, but more particularly acute mania. Excitement is frequently associated with mental confusion, and complicated by the emotion of fear, the result of hallucinations. The presence or absence of hallucinations very markedly affects the character of the mental symptoms in all acute insanities.



**Mental Confusion.**—The state known as mental confusion is a relative term, and may vary from a condition of impaired mental activity—as evidenced by a difficulty in understanding and answering simple questions—to a condition of absolute unconsciousness and lack of knowledge of position. All states of confusion, according to some authors, are of toxic origin; but this is rather too absolute a statement, as mild mental confusion is always associated with states of mental exhaustion, in which no element of toxæmia can be demonstrated, while on the other hand certain conditions of undoubted toxæmia, such as the excited stage of folie circulaire, are not complicated with any loss of consciousness. Well-marked mental confusion is to be seen in acute melancholia, continuous excitement with toxæmia, acute mania, and katatonia. In continuous excitement with toxæmia a condition of false consciousness may be present. The patient, though excited, may answer questions and remark upon the surroundings, but upon recovery it will be found that such a patient's memory is a complete blank for the period corresponding to the illness. Other patients realise the state of confusion under which they are labouring, and in their relatively lucid intervals describe their condition as a dream.

**Hallucinations** are false sense impressions which occur without normal stimuli, and may be divided into hallucinations of peripheral origin and of central origin. Peripheral hallucinations are always associated with some functional derangement or structural alteration in the sensory end-organs or sensory nerve-trunks, while those of central origin may arise from the same causes either in the sense centres or in the higher mental centres. The presence of hallucinations indicates either that the patient is suffering from toxæmia or nervous exhaustion. The study of the onset of various forms of mental disease proves that hallucinations may be one of the earliest indications of nervous disorder, and in many instances hallucinations are suffered from long before the patient is insane, and are recognised as false impressions by the sufferer. It is possible that repeated hallucinations may cause mental disease, as the brain, being entirely dependent upon sensory impressions for its state of mental health, must



be disordered by the constant reception of false stimuli. On the other hand, hallucinations may be secondary to mental disease, and in such a case do not develop until the patient has passed into a condition of insanity.

Hallucinations of hearing are undoubtedly the most common sensory disturbance met with in acute and chronic insanities, and the auditory impressions may be simple, such as whistlings, ringing of bells, rumblings, creaking, scratchings, or the sound of falling water; or they may be more complex, and consist of voices—the words being inaudible—or of voices uttering distinct words and sentences which may or may not have reference to the patient; or the hallucinations may consist of singing or musical sounds of great complexity. In some cases the auditory sensations take the form of the repetition of words being written or spoken, or even thought, but not uttered, by the patient—a condition which is spoken of by some French alienists as “the echo.”

A patient suffering from hallucinations of hearing frequently presents certain peculiarities of attitude and manner which are characteristic of the symptom. In the acute early stages of such conditions as acute melancholia, katatonia, and excitement with confusion, the patient sits in an attitude of strained attention, the eyes turn frequently to a window, door, or certain points of the wall, floor, or ceiling, with an expression suggestive of expectation. In the later and more chronic stages of acute insanity and delusional insanity the patient may hold conversations with unseen persons, or instead may suddenly launch into violent and apparently causeless abuse and rage; attacks which may subside as suddenly as they began, or may be continuous, and constitute the chief outward mental symptom of the disease.

Aural hallucinations, like all hallucinations, tend to be most pronounced at night.

Hallucinations of sight are frequently associated with hallucinations of hearing in the acute stages of toxic insanities, and they are a characteristic symptom of acute alcoholic insanities, particularly delirium tremens and any acute insanity which may be complicated by alcoholic excess.

Hallucinations of taste and smell are commonly associated

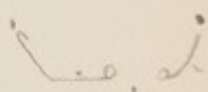


and are of rare occurrence, except in delusional insanity, particularly delusional insanity of alcoholic origin; and they are invariably the result of the alimentary disorders which so frequently complicate chronic alcoholism. Hallucinations of smell are sometimes in women associated with uterine disorders, and in men with the symptom of masturbation.

Hallucinations of common sensibility and the sensibility to heat are most frequently met with in delusional insanity, and the insanities associated with alcoholism, morphinism, and cocainism. These hallucinations take the form of tingling or pricking sensations, numbness in the extremities, pains of a burning, cutting, stabbing, or twisting character, which may be referred to the pelvic, abdominal, or thoracic organs, and give rise to the delusions of persecutions by electricity, marconigrams, hypnotism, and X-rays, or the belief that the body or clothes are crawling with vermin, or that attempts at castration, rape, and common assault are made on the patient during sleep. Patients suffering from such delusions are frequently dangerous to others, or may make attempts at self-destruction in order that they may escape from their supposed persecutors.

Illusions are sense impressions which are wrongly interpreted by the patient, and they are a connecting link between hallucinations on the one hand and delusions on the other. Illusions are very prevalent in delirium tremens, where the patient mistakes the identity of those about him, or may fondle and stroke his pillow under the belief that it is a dog, cat, or child, or may believe that the rattling of a window and the sound of the wind are shots and cries of distress. A combination of hallucinations and illusions in some instances leads to the delusion that the patient is dead, and that his own personality has disappeared, and a strange personality has been substituted. Such patients may speak of themselves always in the third person singular, while others who believe that their bodies are occupied by themselves and a second personality may use the first person plural, or different names for the dual personality in describing their symptoms and actions.

**Delusions.**—An insane delusion is defined by Clouston as “a belief in something that would be incredible to people of the same class, education, or race as the person who ex-





presses it, the belief persisting in spite of proof to the contrary, this resulting from diseased working of the brain convolutions."

On one occasion a patient was admitted into the Royal Edinburgh Asylum with certificates to the effect that he laboured under the delusion that he was pursued by the devil. The patient, a low-type uneducated Irishman, who could neither read nor write, was kept under observation, but presented no symptoms of insanity. His own account of his arrest as a lunatic was as follows. He left his home in the West of Ireland in a fishing boat and found his way by sea to Leith, where he arrived in the evening, tired and exhausted. Not being able to find a lodging he retired into a close and went to sleep. A policeman making his round, lamp in hand, discovered the patient and roused him from sleep. Whether the Irishman had never seen a policeman before or not I cannot say, but this sudden visitation proved too great a strain for his nerves, and he shouted out in terror, and being superstitious, immediately came to the conclusion that his visitor was the Evil One. This was clearly a case of a delusion, but not an insane delusion, arising through exhaustion and fatigue, the sudden apparition in strange surroundings, and the result of deficient education and inherent superstition. Delusions are subjective mental phenomena, and the character, variety, and complexity of the delusions are dependent on the type and education of the brain affected.

Two cases of delusional insanity, at present under my care in Murthly, both suffer from the same sensory disturbances. The one patient is well educated and an engineer to trade; his sensory disturbances are ascribed to electricity, phonographs, etc., and his explanations of the methods by which these forces are applied are ingenious and full of detail. The other patient is an ignorant ploughman, who can just read and write, who has spent all his days in a remote country farm, and his solitary delusion is that he is "hypnoted," and he can advance no further explanation than reiteration of the statement, "I ken that I'm hypnoted."

The majority of delusions are apparently the result of false sense impressions, and may be described as secondary, but



some, especially those which accompany states of depression, are probably primary, and are directly the outcome of the feeling of mental misery.

Delusions are usually described as fixed and fleeting according as they occur as a permanent symptom of such a disease as delusional insanity, or only as a passing phase of an acute mental state, such as acute melancholia or mania. The fixed delusions are further subdivided into delusions of persecutions or suspicion, and delusions of grandeur or pride. All forms of insanity, if they become chronic, may be complicated by delusional states, and the mere presence of a delusion or delusions is of no diagnostic import except in the one disease, delusional insanity.

**Stupor.**—The condition of stupor is defined by Clouston as “a morbid condition in which there is mental and nervous lethargy and torpor, in which impressions on the senses produce little or no outward present effect, in which the faculty of attention is or seems paralysed, in which there is no sign of originating mental power, in which the higher reflex functions of the brain are paralysed, and in which the voluntary motions are almost suspended for want of convolitional stimulus, but where the patients usually retain the power of standing, walking, masticating, and swallowing.” All authors describe stupor as a disease, and subdivide it into clinical varieties according to the mental or muscular conditions, and the result is unsatisfactory and confusing, because stupor is not a disease, but merely a symptom or stage of various diseases. To subdivide stupor into non-resistive, or cataleptic, or resistive is incorrect, because any one case of stupor may present all these muscular symptoms in the course of an attack. It is equally incorrect to subdivide the symptom into anergic and conscious or melancholic and delusional (Hayes Newington), because these types run into one another. All that can be definitely said of stupor is that it is a symptom which may complicate any acute mental disorder, that it is a definite stage of the disease known as katatonia, and that it may occur as the result of brain exhaustion from starvation, sexual excess, and repeated and severe epileptic seizures, and that it is more liable to occur during adolescence than adult life. The patient may be quite



unconscious and without memory, or fully conscious and capable of remembering everything that occurred during the attack. The muscles may be flaccid, or resistive on passive movement, or capable of being placed in fixed attitudes and postures (catalepsy), and there is general impairment of nutrition with alimentary disturbances and defective circulation, the hands and feet being cyanosed and often oedematous. All patients suffering from the condition of stupor are liable to unexpected and sudden impulses which may be destructive, homicidal, or suicidal in nature.

**Enfeeblement.** — Mental enfeeblement may be either primary or congenital in origin,—such as occurs in idiocy and imbecility,—or secondary to acute mental disease, or the natural failure of the vital powers which occurs in old age. Mental enfeeblement in the psychological sense may imply loss of judgment, will power, self-control, blunting of the moral sense, loss of power of attention and interest both in the person and environments, impairment of memory, and inability to originate new ideas and actions. The condition may be transient, as the result of ill-health or exhaustion from severe illnesses and physical and mental shocks and strain, or it may be permanent from deficient brain development, or the result of cerebral lesion either from accident, vascular changes, or acute attacks of mental disease.

**Self-control.**—Loss of self-control is characteristic of all forms of insanity, of states of delirium complicating acute physical diseases, and of the condition of brain known as senile. Normal self-control is so much a matter of race, age, the state of health, temperament, moral and physical upbringing, that it is impossible to lay down any law whereby this mental quality can be gauged, or to determine when deficiency has passed from a normal to an abnormal state, but in the majority of acute mental diseases there is no such difficulty. The patient who, suffering from terrifying hallucinations, hides under the bed, jumps out of a window, or attempts suicide, or the acutely excited patient who shouts, laughs, grimaces, sings, tears the bedding or clothes, has obviously no self-control; neither have the epileptic or alcoholic patients when they exhibit outbursts of rage on



slight or no provocation. In the earlier symptoms of certain mental and nervous diseases, such as general paralysis, failing self-control may be represented by undue irritability and emotionalism ; but as loss of sleep, worry, physical exhaustion, and bodily pain and disease may produce the same condition, the value of the symptom from a diagnostic point of view must be judged on its merits in each individual case. It would be obviously absurd to expect similar self-control from a child and an adult, or from a man in health and one pulled down by illness or fatigue ; and yet, as an early symptom of mental breakdown, irritability is of diagnostic importance and is not infrequently missed or misinterpreted. To the failure of self-control is directly due the condition of impulsiveness, which is another common mental symptom among the insane. An impulse is an action committed consciously, but without motive or forethought, and sometimes directly opposed by the will power of the individual. In some cases the impulse is suggested by hallucinations of hearing, but in others the impulse is of central origin, arising suddenly and apparently causelessly. Impulsiveness may take various forms, but the most important are the impulses to break glass and crockery and destroy clothing, the impulse to steal (*kleptomania*), and the impulses to homicide and suicide. There is at present a patient in the Murthly Asylum who will suddenly and causelessly thrust her hand through several panes of glass in rapid succession. She is not excited, she can never give any reason for her action, and the fact that she cuts her hands very badly does not deter her from repeating the action. Another patient will suddenly tear her dress into shreds, and then set to work to repair the damage. Another patient—a man—made sudden homicidal assaults sometimes with the bare hands, and sometimes with improvised weapons such as chairs. If one sat down on the same seat beside him he would shake violently all over, as if holding himself in, and say, "I would like to kill you." A female patient, who had been stuporose and apparently unconscious for a year, suddenly jumped off her seat, seized an electric lamp, broke it with her hands, and tried to swallow the fragments ; no sooner was the impulse over than the patient again lapsed into stupor. Sometimes



impulses to suicide are undoubtedly suggested to the patient. A stuporose but perfectly conscious female patient made a desperate attempt to cut her throat with a knife, after hearing a fellow-patient read from the daily paper the account of a suicide by cut-throat. The impulsive assaults so frequently made by delusional cases are not true impulses, but rather the result of insane revenge prompted by the hallucinations and persecutory delusions, and are premeditated actions. The moral sense, like the power of self-control, is largely a product of heredity and upbringing, and although the code of ethics which controls our ideas of right and wrong varies enormously in different individuals of the same class and education, and in individuals of the same family, there are certain departures from the commonly accepted standard which are regarded as disease. In the early stages of the excitement of folie circulaire and the prodromal period of general paralysis, there is very marked perversion of the moral sense, which sometimes comes on quite suddenly, and is accompanied by a complete change in appearance and character. Such patients will lie, steal, drink, and commit sexual excesses openly and shamelessly. They prefer to associate with people below them in the social scale, and are regardless of their duty to their families or society. The moral depravity, cowardice, and meanness of the victims of alcoholism and morphinism are characteristic features of these diseases. The attention to the personal appearance in the insane may vary from a tendency to dress loudly, and decorate the person with brilliantly coloured ribbons and valueless trinkets, to an entire neglect of personal cleanliness, and a filthiness of dress which to the same patient in a state of sanity would be repulsive and abhorrent.

The tendency to suicide, which is liable to occur in any form of mental disease, but which is a possibility never to be forgotten in the treatment of patients who are depressed, and particularly those who suffer from sleeplessness, in some cases occurs as an accident, in others as the result of an impulse, while in others the attempt is carefully planned and pre-arranged. Accidental suicides are those which occur during the acute onset of mental disease when the patient is in a



state of confusion or unconsciousness, and the most frequent form is precipitation from a height by jumping through a window—the patient being in a state of terror and trying to escape from terrifying hallucinations. Impulsive suicidal attempts may be prompted by suddenly presented opportunities or means of self-destruction, such as the sight of water, fire, a knife, cord, or poison. As a rule such patients are oblivious to pain at the time the action is committed. I have seen one patient try to swallow live coal, another gouged out her eye with a pair of scissors, another nearly severed the hand at the wrist with a small penknife attached to a watch chain, while another jumped into a bath of boiling water. The patients who attempt premeditated suicide not infrequently devote their attentions to only one method of destruction, and fail to avail themselves of others equally practicable. I have seen a desperately suicidal case pass a busy railway and canal, and attempt suicide by lying naked on a hill in the hope that the cold and exposure would prove fatal. As a rule, the more educated the patient the more ingenious and various are the methods adopted to attain the desired result. The only efficient prevention of such accidents is constant supervision by day and night, the patient never being allowed for any reason whatsoever to be out of sight or control of those in charge. The mere fact that a patient fears death, and thinks that his life is in danger, is no proof that such a patient will not attempt suicide. Many such patients are very suicidal, and, paradoxical as the statement may seem, commit suicide to escape the imaginary death which they fear.

**Attention.**—The faculty of attention is variously affected in those suffering from insanity. In some patients the attention is entirely subjective, being occupied by the sensations of misery, depression, or sensory disturbances, but the power of attending to what is passing around them is gone. In others the attention is wholly objective, as in the excited stage of folie circulaire, where the attention is attracted by every passing movement or sound, while in conditions of complete confusion and stupor the capacity of attention is wholly abolished. In all acute toxic conditions the attention is difficult to attract and



cannot be held, while the capacity for work or occupation is in abeyance. In such a condition as hebephrenia the chief mental symptom is the loss of power of continuous attention, and with it the power of work or even capacity for amusement, and the same symptoms are characteristic of the exhaustion which follows acute insanities, and may be the earliest indication of the onset of the mental state known as dementia.

**Volition.**—The volition or will power in mental diseases may be entirely lost, as in secondary dementia, where the lack of capacity to originate action or new ideas is more or less present in every case. In certain states of depression and confusion there is a paralysis of volition, the patient never being able to decide upon a line of conduct or course of action. Such patients cannot make up their minds to dress or undress, to go out or stay at home, and no action is so trivial but it becomes a source of doubt and indecision. In other cases the volitional disorder takes the form of extreme obstinacy and resistiveness to any suggested action, though the patient may readily and voluntarily perform such actions if they originate spontaneously.

**Memory.**—The memory for both recent and remote events is more or less impaired or entirely abolished in mental states characterised by confusion and loss of consciousness. In states of mental depression and exhaustion the faculty of memory is present, but the effort to recall events is such a labour that the patient either will not face it, or in the process rapidly becomes mentally exhausted and the mind becomes a blank. In other cases the memory is abnormally active for both recent and remote events, and this is particularly characteristic of the elevated stage of folie circulaire. A failure of memory for recent but not remote events is a symptom of senility and alcoholism, while the condition of pseudo-reminiscence is to be met with in the same states of mental impairment.

**Speech.**—The speech centre, though a motor centre, is so intimately associated with the mental processes that it is frequently implicated in the course of insanity. In states of acute excitement the speech is loud, tumultuous, and profuse in character, that is to say, as a motor centre it is exhibiting



symptoms of overaction, just as the overaction of the other motor centres is shown by restlessness and exaggerated movements of the arms, legs, and facial muscles, while on the mental side the speech is incoherent or disconnected, and the association of ideas accidental and fragmentary. The mental state is frequently reflected by the character of the speech. Patients labouring under delusions of grandeur, or a sense of exaggerated self-importance, may be pompous, garrulous, or boastful, while those who suffer from depression may speak in hardly audible whispers and on very rare occasions. In complete stupor the power of speech is either abolished or in abeyance, while in less marked stupor—particularly that of katatonia and the maniacal stages of the same disease—implication of the speech centre is demonstrated by the tendency these patients have to repeat numbers, words, or sentences in a rhythmical manner (verbigeration), or to echo the words and tone of a person addressing them (echolalia).

The tone and character of the voice during states of acute excitement is altered, harsh, and unnatural, or husky and indistinct, through dryness of the mouth and irritation of the vocal chords. Incoherence of speech in conditions of acute excitement differs from the incoherence of more chronic conditions, the one being due to confusion and a tumultuous rush of words, the other to functional or structural changes in the cortical grey matter

The following is a good example of incoherence, written verbatim, from the speech of a case of delusional insanity with delusions of grandeur, the patient believing himself to be the angel Gabriel:—"The angel Gabriel, the substance of the Queen of England, mocks the angel into amber colours in the heavens, and demanded her that she should come down and grow something to be burnt to stop the growth of her cold nakedness. The Queen would not idea the divine truth, and claimed the issue of blood to be created by her. Cardinal Wolsey received instructions being revealed to she was to receive her own breath and seed her own apron strings."

The impairment of speech so commonly met with in general paralysis and alcoholic insanity is directly due to the muscular paresis characteristic of these conditions. During



the early stages of acute insanity complicated by mental confusion it is impossible to get the patient to write. In some the acquirement is lost, while in others, if anything approaching formed letters and words is produced, these are unreadable, and merely accentuate the failure of co-ordination for fine muscular movements. In excitement without confusion (*folie circulaire*), and in the early stages of general paralysis, voluminous letter writing is a common symptom. The handwriting of many cases of delusional insanity is peculiar and eccentric, copiously underlined, or written in bold, over-elaborated detail.

**Sleep.**—No function of the body is so liable to disorder in general physical disease as the function of sleep, and it is no over-statement to say that no case of mental disease passes through an attack without some disorder or abolition of sleep. One of the earliest symptoms of acute recurrent mental attacks is sleeplessness, and the most troublesome symptom to treat in the acute stage of the disease is sleeplessness; while the return of natural sleep is often the first symptom of physical and mental improvement. Sleep may be absolutely, or apparently absolutely abolished in some cases of chronic excitement, who though under observation for years have never been actually seen asleep, or there may be a regular periodicity of sleep and sleeplessness. The most common periodicity is for the patient to sleep one night and not the next, or to sleep for two nights and miss the third, and these periodic conditions are accentuated in some cases by alterations of temperature and pulse on the day preceding or following the sleepless night. Excessive sleep is frequently seen in some stages of general paralysis, in the stupor of *katatonia* and some senile cases. Much the most common disorder in senility with mental symptoms, however, is for the patient to sleep during the day and awake to abnormal activity at night.

The causes of general sleeplessness are innumerable, but in all acute insanities complicated by this symptom the direct exciting cause is a *toxæmia*, and in the more chronic cases *toxæmia* plus a bad brain habit. In some cases the *toxine* apparently acts directly upon the higher cortical grey matter, producing a mental state incompatible with sleep, but in



others the toxine produces physical symptoms which of themselves alone are capable of preventing the condition of sleep. These symptoms are a high arterial tension and a rapid pulse-rate. The normal arterial pressure, according to Hill, is about 110 mm. Hg. when the patient is in the horizontal posture. In patients who have recovered from attacks of insanity I have found the tension to vary from 100-120 mm. Hg. Another characteristic of the arterial pressure in health is that it is always lower in the evening than in the morning. All my observations have been made with a Barnard and Hill's sphygmometer, which consists of a leather arm-piece fastened round the arm with straps. Inside the leather lies a rubber bag connected to a long rubber tube which passes out through a circular opening in the leather, and is secured to a recording tambour. A pump on the same principle as a bicycle pump is attachable to another tap on the tambour. The leather arm-piece with the enclosed rubber bag is fastened over the brachial artery. Air is then pumped into the tambour, and so to the rubber bag encircling the arm, and according to the pressure the indicator on the tambour moves round on a clock-face giving the reading in mm. Hg. The pulsations of the artery are transmitted to the indicator, which oscillates at each pulsation, and the point of maximum oscillation is regarded as indicating the arterial tension in the patient being examined. The instrument is suitable for restless patients, and further, the strap and rubber bag can be left on a patient's arm and readings made during the state of sleep.

In natural sleep it was found that the arterial tension fell some 10 mm. Hg., *i.e.* a patient who before sleep registered 110 mm. Hg., during sleep registered 100 mm. Hg. The same patient, after receiving a 3i. dose of paraldehyde, fell asleep an hour later, and the arterial tension registered 80 mm. Hg. Two hours later the pressure had risen to 95 mm. Hg., and four hours later to 100 mm. Hg.; that is to say, during the drug sleep the arterial pressure at first fell below that of normal sleep, but gradually rose again, and the drug sleep apparently passed into a condition of natural sleep.

In all cases of insanity suffering from metabolic poisoning



and sleeplessness the arterial pressure in the evening was higher than that of the morning, and during the state of sleeplessness was as high as 140-150 mm. Hg., but if sleep occurred naturally the tension always fell below 110 mm. Hg. In a similar condition sleep induced by such a drug as paraldehyde was accompanied by a fall in pressure to at least 110 mm. Hg., but rarely lower. In patients suffering from excitement a high arterial pressure was often associated with the state of sleeplessness, and in these cases also the arterial pressure always fell whether sleep occurred normally or by the use of drugs. It was found, however, that some patients suffering from excitement with sleeplessness—particularly from excited melancholia—had abnormally low arterial pressures with a rapid pulse-rate. In such cases the exhibition of say 3ii. paraldehyde raised the pressure and further excited the patient, while doses of 10 or 15 mins. of paraldehyde, or 30 grs. of sulphonal, or 20 grs. of trional, lowered the pulse-rate and produced sleep without in any way altering the arterial pressure.

## CHAPTER III

### CAUSATION OF INSANITY

ACCORDING to the most widely accepted views of the present day, the cause of all typical insanity lies in the fact that the insane patient possesses an unstable brain, which is easily thrown out of gear by the direct action of moral or physical causes upon the brain cells and nerve fibres. How such causes produce their direct action has not so far been explained. When a man who has lost his money, or a mother who has lost her child, becomes pathologically melancholic, the disease is held to be directly due to the mental effect of such losses. When a recently parturient woman becomes maniacal the disease is due to "the physiological cataclysm, the pains of labour, the excitement mental and bodily, the exhaustion, the loss of blood, septicæmia, the sudden diversion of the stream of vital energy from the uterus to the mammæ, the reflex disturbances on an unstable brain from the reproductive organs; these acting together or separately" (Clouston).

If no such outstanding causes are demonstrable the disease is then explained as "idiopathic" in origin, or, as in many of the mental states seen in adolescence, as conditions of brain involution.

There is, however, another view of the causation of the vast majority of acute insanities which is practically in its infancy, but is steadily gaining ground both in this country and abroad, and that is the toxic theory of origin. The only author who in recent times has written upon the toxic origin of mental diseases is Macpherson. He says, "The toxic basis of all forms of insanity is a presumption for which there is



fairly good foundation, but no direct proof." That was written more than ten years ago, and there is still no direct proof, and never will be, because the only satisfactory and irrefutable proof would be the experimental production of morbid mental states by the use of toxins, and that is impossible. Macpherson divides his causes of toxæmia into :—

A. Auto-intoxication as the result of—(1) Physiological instability, and (2) defective metabolism; (3) defective gland secretion; (4) auto-intoxication from the alimentary tract; (5) auto-intoxication from the liver and kidneys.

B. Intoxication from micro-organisms introduced into the system.

C. Voluntary intoxication by alcohol and drugs.

All these sources of toxins may certainly act independently of one another, but in group A it is impossible in practice to differentiate auto-intoxications arising from physiological instability, defective metabolism, auto-intoxication from the alimentary tract or auto-intoxication from defective action of the liver and kidneys. It is highly probable that they all depend on some degree of hepatic insufficiency, the liver failing to arrest or destroy toxins produced by any of these causes; and, secondly, renal insufficiency through the kidneys failing to excrete the toxins by the urine. The kidneys almost invariably present pathological changes in all forms of acute and chronic insanity, and as the earliest changes noticed are always vascular, it is probable that renal insufficiency is only a link in a chain of morbid processes of toxic origin.

The statement "micro-organisms introduced into the system" is hardly correct, because the organisms which cause certain forms of insanity are not necessarily introduced into the system, they being already there; they are, in fact, the normal inhabitants of the human body, and produce their toxic action through some disorder of the defensive mechanism of the body allowing of their excessive increase, or through a failure in the destruction of the toxins which they produce in the discharge of their functions. Their point of attack is, probably, in most cases the intestinal tract. When a patient suffering from typhoid fever becomes maniacal, it is not the toxin of the typhoid bacillus which is



the cause of the mania—the toxine of the bacillus typhosus is merely the agent which exhausts the bacterial defences of the body, and allows of attacks by the toxins of organisms habitually present in the alimentary tract. Micro-organisms are very rarely present in the blood of patients suffering from acute insanity, and when they are present they are invariably of the streptococci and staphylococci group, and these invasions are not, as is at present stated, terminal infections, because the agglutinines produced by the toxins of these organisms can be demonstrated in the blood serum of many cases of insanity which are not in a typhoid state. The micro-organisms, the toxins of which produce mental symptoms, do not attack through the skin except on rare occasions; they may attack through the lung, they certainly do attack through the intestinal tract, and therefore such toxæmias, under Macpherson's classification, should come under auto-intoxication through the intestinal tract.

Again, toxæmia following upon the abuse of alcohol is not necessarily caused by the alcohol itself. Much more frequently the alcohol, by its toxic action on the liver cells and other bacterial defences of the body, allows of the passage of the toxins of intestinal bacteria into the blood stream in such quantities as to produce a toxæmia sufficiently severe to induce brain symptoms. Here, again, it is possible in the laboratory to demonstrate by changes in the blood cells and serum that such toxins are present in the system, whereas in uncomplicated alcoholic poisoning there are no blood or serum changes.

If this explanation of the toxæmic origin of mental diseases is correct, it is easy to explain how the purely mental symptoms may vary in closely allied cases. There is, in the first place, the individual reaction to the same toxine; and, secondly, the fact that in many cases the toxæmia is a mixed toxæmia. The futility of attempting to base a classification upon mental symptoms alone is also at once apparent.

**Predisposing Causes of Insanity.**—But whatever the exciting causes of insanity may be, the chief predisposing factor is hereditary predisposition. The sane, equally with the insane, are liable to the exciting causes of insanity, and



the factor which in a man without hereditary predisposition may result in a passing malaise, will in one with sufficient hereditary predisposition produce mental disease. Hereditary predisposition should, I think, be used in its very widest sense, and include on the mental side such parental defects as extreme nervousness, eccentricity, alcoholism, hysteria, criminality, vagabondage, epilepsy, want of mental balance, and inequality in mental development, such as extreme mental brilliancy in one direction combined with deficiency in others; on the physical side, any weakness of the defences of the body against toxic and bacterial invasion.

Sound nervous structures cannot exist in unsound bodies, and one constantly sees degenerate parents, although apparently mentally sound, produce offspring still more degenerate who readily develop mental disease. The children of gouty, rheumatic, and tubercular families are more liable to mental diseases than those of healthy parents. Children of syphilitic parents are also more liable to mental diseases, especially juvenile general paralysis. It is not by any means clear in what manner hereditary predisposition produces its effect, but it is highly probable that there are two factors at work. Firstly, the potential patient starts life with an unstable nervous system; and, secondly, the natural defences of the body against metabolic and bacterial toxins are inefficient.

Hereditary predisposition has various ways of manifesting itself. So far as the extremes are concerned, the offspring may be born deficient, or, on the other hand, mental soundness may be retained until old age sets in. The brain habits of certain families seem to run in grooves, especially the tendency to suicide. There is no certainty in the transmission of mental disease; one, or even two or three generations may escape, and there is no certainty as to the form of disease transmitted—epileptics beget maniacs, and melancholics and maniacs epileptics. Alcoholism in the parents, in so far as it is an evidence of mental instability, is distinctly associated with idiocy, imbecility, epilepsy, and mental breakdown during adolescence in the offspring.

The maternal heredity is the one said to be the most



liable to be transmitted to the child, but the paternal heredity is also a strong factor, as the following family history proves. The father became insane at 70. The eldest daughter became melancholic at the climacteric. The second daughter suffered from puerperal mania at 38. The third daughter developed adolescent mania at 18, and the fourth child, a boy, was born an imbecile. The mother appeared to be a healthy woman.

According to the present asylum statistics, in from 15 to 50 per cent of the patients admitted into the District and Royal Asylums of Scotland there is an ascertained direct or collateral heredity to insanity. These are statistics of actual insanity admitted in the relatives or parents, and if one included the various other factors stated under heredity the percentage of hereditary transmissions of mental defect would be very much higher.

As further predisposing causes of mental disease, consanguineous marriages and modern civilisation are frequently mentioned. Consanguineous marriages only produce insanity in so far as they tend to emphasise bad heredity, and civilisation only in so far that men live in large centres and under unnatural conditions.

After heredity, I would call any cause a predisposing one which impairs the general vitality and lowers the general defences of the body against toxic influences, such as worry or anxiety, which are fertile causes of the disorganising of the metabolic processes of the whole body; the critical periods of adolescence, the climacteric, puerperal states, and senility; deficient or improper food supply; the abuse of alcohol and certain drugs, such as opium and cocain; faulty environments, which lead indirectly to physical breakdown; any exhausting physiological processes, such as excessive or prolonged lactation; or any exhausting pathological condition, such as chronic suppuration, malignant disease, and chronic valvular incompetence of the heart; shock and cerebral traumatisms. Lastly, faulty upbringing and moral training which facilitate the onset of mental symptoms through a deficiency of will power on the part of the patient. The more one sees of mental disease the more one is struck by the fact that the majority of



adolescents, at all events those of them who find their way into asylums, give ample evidence that in their upbringing they have never been encouraged or trained to exert their self-control. Such a statement at first sight appears to be entirely in favour of the theory of the central origin of all brain diseases, but in reality it is not so, for it is possible by the will power to suppress the action of certain toxins, such as alcohol for instance, and I have seen cases of recurrent mania in which a threatened attack was held in check by the will power until the acuteness of the toxæmia had passed off.

**Exciting Causes of Insanity.**—Of all the causes usually assigned in the official statistics of asylums not more than two, viz. lactation and alcohol, are possibly exciting causes. Excessive lactation produces a condition of exhaustion, and alcoholic excess is often followed by certain symptoms which may be directly due to alcoholic poisoning. Syphilis also is a direct cause of certain pathological mental symptoms. So far as the other assigned causes are concerned, they may act as predisposing or associated causes, but there is absolutely no proof that they are exciting. It is here that the toxic theory of the origin of insanity clashes with the preconceived notions of the older school of psychiatrists.

As I have previously stated, the present and most widely held view of the origin of the majority of insanities is that the disease originates in the cells and fibres of the brain cortex, and that the physical symptoms which may be noted in such conditions are directly the result of the pathological working of the brain cortex. If this view is to be accepted in its entirety, then, the brain must be regarded as exempt from the effects of the perturbations of the rest of the body. In the ordinary teaching of medicine, we are taught when investigating a case of disease to look for the cause. When a patient suffers from pain after taking food the general practitioner does not say, "This patient has inherited a stomach whose secretory cells are very easily thrown out of gear, and they have therefore become diseased *de novo*, and are not acting properly"; he would rather say, "This patient is evidently liable to gastric disturbance, but what is the extrinsic cause of this attack?" Such a view of



the onset of insanity takes no cognisance of physical symptoms which frequently precede the onset of the mental symptoms, and does not explain why, when recovery occurs, physical improvement commonly precedes mental improvement.

The toxic theory of origin postulates that there is a physical cause underlying the symptoms of acute insanity, and that the brain symptoms are secondary to the physical symptoms. The clinical symptoms alone in many cases suggest that a condition of toxæmia is present, and as the brain cells and fibres are most easily affected by substances conveyed to them by the blood and lymph which nourish them, it is reasonable to suppose that their disordered working is due to some toxic substance conveyed to them by the blood. When, further, it can be demonstrated that in some varieties of acute insanity there are distinct evidences of metabolic poisoning, in others a hyperleucocytosis with the presence of bacterial agglutinines in the serum, together with integumentary and kidney symptoms, which would indicate that the excretory organs are endeavouring to throw off some toxic substances, there are grounds for the belief that toxic influences are at work in the causation of acute mental disease. There is also evidence that mental diseases are often rather disorders of function than of structure. I have seen a patient who had suffered from acute mania for three months, and who was acutely maniacal at 10 A.M., quite clear and sensible at 12 o'clock mid-day. And the effect any acute intercurrent disease which raises the polymorphonuclear leucocytosis has, in cutting short mental symptoms, is recognised by all who have had any experience of the treatment of mental diseases.

Broadly speaking, the exciting causes of insanity seem to me to divide themselves into two large classes, the non-toxic and the toxic.

1. **Non-Toxic Causes.**—The chief non-toxic cause is probably conditions of physical exhaustion. When the bodily condition and strength are reduced to a low ebb, the brain cells and brain functions of necessity suffer with the rest of the body. If the patient has hereditary predisposition he will suffer from brain symptoms the more readily.

As further causes of insanity of non-toxic origin I would



mention brain injuries, the result of hæmorrhage or traumatism, anæmia of the brain, due to an impoverished condition of the blood, cardiac disease, or narrowing of the lumen of the blood-vessels, and, lastly, sudden deprivation of the special senses of sight or hearing.

**2. Toxic Causes.**—The toxins which act as exciting causes are: (1) Toxines of metabolic origin, where there are evidences of grave nutritional disorder—a breaking-up of the fluids in the body, and a deficient discharge of the waste products by the skin, kidneys, and intestines. (2) Toxines of bacterial origin which produce a condition of hyperleucocytosis, bacterial agglutinines in the blood serum, a deficient excretion of chlorides in the urine, and sometimes irritative rashes on the skin. The organisms which produce these toxic conditions are probably normal inhabitants of the human body, which become virulent through failure of the defences of the body against organismal toxins. The weakness of these defences being, as previously pointed out, probably as much a part of nervous heredity as an unstable brain. It is possible that the toxins of the usual bacterial inhabitants of the human body, particularly the alimentary tract, serve some physiological purpose, possibly a nervous stimulant, as I have noticed that the blood serum of persons who are extremely nervous and excitable, but not insane, often contains abundant agglutinines (alexines) to such organisms as the staphylococci. I have seen conditions of mental confusion and excitement, which were impossible to distinguish from acute mania, arise as the result of acute poisoning from the staphylococcus aureus, streptococcus pyogenus, and the diplococcus pneumoniae. (3) Toxines of drug origin, such as morphia, cocain, chloral, and more rarely alcohol.

It is quite possible and very common to have conditions of mixed causation. An exhaustive condition may pass into a condition of bacterial toxæmia, and then as a rule the disease is fatal; or metabolic and bacterial symptoms may be associated and produce mixed symptoms, and very rarely, indeed, is such a drug toxæmia as alcoholism a pure uncomplicated state, for alcoholic excess is a potent factor in breaking down the bacterial defences of the body.

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In conclusion, I would point out that the brain is an extremely delicate and complicated organ, and that as in health no two brains act alike, it is not to be expected, therefore, that in disease, and even under the influence of the same toxine, any two brains will give exactly the same mental reaction. Further, brain cells acquire bad habits more readily than good ones, and the ordinary periodicities of alternating energy and rest are often much exaggerated as the result of undue stimulation on the one hand and the natural reaction of exhaustion on the other.



## CHAPTER IV

### CLASSIFICATION OF INSANITY

THERE are certain terms of nomenclature which have been used in the description of mental diseases ever since any knowledge of such conditions existed, and although some of these terms, such as "mania" and "melancholia," are at present used to designate either a disease or a symptom, and are therefore incorrect and lead to confusion, it is impossible for comparative purposes to bring forward a new classification without referring to the presently accepted ones. The present classifications are generally admitted to be unscientific, inaccurate, and confusing, whether regarded from the symptomatological or clinical standpoint. No two writers seem to be quite at one in their descriptions of mania, probably because patients suffering from widely different mental diseases may present symptoms of mental excitement, and for the time being are regarded as labouring under "mania." Excitement with confusion, excitement without confusion (the elevated stage of folie circulaire), and the excitement which at times complicates katatonia and hebephrenia, are all symptomatologically states of mania, but the underlying disease processes differ from one another both in the mental and physical symptoms. Further, the physical symptoms which accompany excitement with confusion, while most frequently associated with maniacal excitement, may also be associated with states of depression or fear.

In the same way, if the presently accepted varieties of melancholia are examined physically, it will be found that while some present symptoms of pure metabolic disorder, others are associated with more severe toxæmias, and in these the symptom



of hyperleucocytosis is always present. The mental symptoms in both forms may be very similar, but the physical symptoms at once demonstrate the fact that we have to deal with two different diseases. With few exceptions, the present so-called clinical classification is even more confusing than the symptomatological. Puerperal insanity is elevated to the position of a disease, whereas a little practical ward work will clearly demonstrate that almost any variety of mental disease may be the sequel of the puerperal state. The term adolescent insanity includes such widely differing diseases as excitement with confusion (mania), katatonia, hebephrenia, and delusional insanity (paranoia); while the physical and mental symptoms of insanity occurring at the climacteric and senile periods, and at present designated as climacteric and senile insanities, do not differ from the physical and mental symptoms of similar diseases occurring at other periods of life. In other words, the symptomatological classification is based on the outstanding mental symptoms, while the physical symptoms have been ignored, and in the clinical classification a predisposing has been mistaken for an exciting cause. In the following classification I have partly retained some of the existing nomenclature, and regrouped the affections broadly under insanities of non-toxic origin and insanities of toxic origin. This classification is not put forward as final or complete, but merely as a working basis.

#### CLASSIFICATION :—I. Insanities of Non-Toxic Origin

1. **Exhaustive Insanity.**—A collection of physical and mental symptoms which are fairly constant, and occur as the result of some physical or mental strain, as the sequelæ of exhausting diseases such as typhoid, influenza, phthisis, etc., and is most typically seen as the result of prolonged lactation.

2. **Insanity the result of gross brain lesion or traumatism.**

3. **Insanity resulting from brain anæmia.**

4. **Insanity resulting from deprivation of the special senses, particularly of sight and hearing.**

5. **Insanity from mental or physical shock.**



## II. Insanities of Toxic Origin

*Group I.*—Insanities the result of toxins of metabolic origin.

1. **Acute Melancholia.**—A mental disease in which there is always depression, confusion, and vivid hallucinations associated with physical symptoms of malnutrition and evidences of deficient excretion of the waste products of the body. The acute stage is of short duration, and is followed by recovery, or more commonly by a subacute stage of indefinite duration, in which the patient is always depressed, but may present a variety of mental symptoms, and in which the condition of malnutrition persists together with deficient excretion of the waste products of the body. The symptom of hyperleucocytosis is never present.

2. **Insanity associated with deficient, excessive, or altered secretion of the thyroid gland.**

3. **A variety of puerperal insanity.**

4. **Delusional Insanity.**—A definite disease process which in its later stages is characterised by fixed delusions, but the early symptoms are very similar to those of acute melancholia. The disease is at present described as progressive systematised insanity, mania of persecution and grandeur, megalomania, and paranoia.

5. **Chronic Metabolic Toxæmia** (premature senility, chronic brain atrophy).

*Group II.*—Insanities in which there is evidence of bacterial toxins being present in the blood, in which there is always a hyperleucocytosis, and in which bacterial agglutinins are frequently present in the serum.

1. **Excited Melancholia.**—A mental disease in which there is depression, with constant noise and restlessness, but without marked mental confusion, with the symptom of hyperleucocytosis always present. Partial recoveries followed by exacerbations are very common, and the disease, if not recovered from, may terminate in a state of fixed delusion associated with chronic malnutrition. The condition rarely passes on to typical dementia.

2. **Maniacal Excitement with Confusion** (Acute Mania).—A type of mental disease very variable in the intensity of the mental symptoms, but nearly always associated with



maniacal excitement and confusion, with the symptom of hyperleucocytosis always present, and generally persistent even after recovery. The disease may be continuous or recurrent, and may occur at any period of life. Complete or partial recovery is common generally within three months from the onset of the symptoms, but one attack always predisposes to a second. Cases which do not recover generally become delusional, chronically restless, noisy, and destructive, and tend to terminate sooner or later in dementia. This disease is never associated with alternate periods of excitement and depression, although a condition of exhaustive stupor with confusion may follow the maniacal attack.

3. **Folie circulaire.**—Maniacal excitement or mental depression without confusion — Kræpelin's manio-depressive insanity—and which probably includes the simple mania and melancholia of most authors. A mental disease in which there may be either recurrent attacks of states of depression or states of excitement, or the states of depression and excitement may regularly alternate. In neither state is there typical mental confusion. The symptom of hyperleucocytosis is always present during the stage of excitement, but disappears on recovery. The stage of depression is frequently associated with a hyperleucocytosis, which is most marked in those cases in which there are alternate attacks of excitement and depression. The disease is apt to recur, attack following attack, but rarely, if ever, terminates in dementia.

4. **Katatonia.**—A disease which most frequently occurs during adolescence, and is characterised by a stage of acute onset, in which fear, impulsive actions, confusion, and vivid hallucinations are the chief mental symptoms. Hyperleucocytosis is always present, and in many cases the voluntary muscles are thrown into a state of rigidity or spasm of very variable duration. The acute stage is followed by a stage of stupor without loss of consciousness, and the onset of stupor is associated with a very marked hyperleucocytosis. The voluntary muscles pass into a state of resistiveness to passive movements, and in many cases efforts at voluntary movement are difficult, awkward, or even impossible, owing to counter-stimuli or irritability of the opposing muscles. The stage of



stupor may terminate in recovery or dementia, or the patient may pass into a stage of excitement, and then recover or become hopelessly demented. Until the disease terminates in dementia or recovery, the symptom of hyperleucocytosis is always more or less persistent. If the patient relapses, the disease may again pass through all the stages, but not uncommonly patients who have suffered from one attack of katatonia and recovered, if they relapse, present mental symptoms of excitement which, without a history of the first attack, are confusing and difficult to diagnose.

5. **Hebephrenia.**—A disease which occurs during adolescence, and is characterised on the physical side by delayed or arrested physical development, by recurrent attacks of hyperleucocytosis and alterations in the pulse and temperature, and on the mental side by steadily progressive enfeeblement, incapacity for occupation, with occasional outbursts of excitement. In 80 per cent of the cases affected the termination is hopeless dementia.

*Group III.*—Insanities the result of alcoholic and drug toxines.

1. **Delirium tremens.**
2. **Alcoholic mania.**
3. **Chronic alcoholic insanity.**
4. **Morphinism.**
5. **Cocainism.**
6. **Chronic poisoning by chloral, ether, chloroform, mercury, iodoform, lead, carbon bisulphide, paraldehyde, Indian hemp, and thyroid.**

*Nervous Diseases frequently complicated by Mental Disease—*

1. **Epilepsy.**
2. **General paralysis.**
3. **Dipsomania.**

*States of Mental Enfeeblement—*

1. **Idiocy and imbecility.**
2. **The higher imbeciles.**
3. **Secondary dementia.**
4. **Organic dementia.**

## PART II





## CHAPTER V

### INSANITIES OF NON-TOXIC ORIGIN

1. **Insanity of Exhaustion.**—The insanity of exhaustion is a disease of middle or later middle life, when the reparative powers of the body naturally commence to fail, and this is especially true of men. In women one may see the disease earlier as the result of prolonged lactation.

Hereditary predisposition is a prominent cause, as are also nervous diseases and a neurotic temperament. The exciting causes are many, and include conditions which exhaust the physical powers.

The onset of the illness is slow, and long before mental symptoms manifest themselves the decline of physical energy, accompanied by indigestion, constipation, anæmia, deficient circulation, a tendency to catarrhal conditions, nervousness, irritability, and sleeplessness, have been noticed by the patient and the patient's friends. When mental symptoms appear they are always of the nature of depression, not infrequently associated with delusions of suspicion. Upon examining such a patient one is at once struck by the appearance of malnutrition, the pallor and unhealthy condition of the skin, the lack-lustre eyes, the exhausted—often confused—expression, and the listless attitude. The temperature is, as a rule, subnormal. In the alimentary system the digestive power is deficient. The patient complains of weight, pain, or distension after taking food. The bowels are usually confined. There is always anæmia—both a deficiency of hæmoglobin and red cells. The leucocytosis is never raised, and there is always a deficient polymorphonuclear percentage. The blood is thin



and watery, and coagulation is delayed. The heart's action is weak, and functional murmurs are commonly to be heard over the mitral and aortic areas. The skin is pale and has a dirty yellow appearance. Profuse sweating may be induced as the result of slight nervous shocks, such as the sudden banging of a door. The urine is excreted in fair quantity, is generally pale in colour but shows no other abnormality. There need not necessarily be any disorder of the sensory or motor functions. The pupils are generally dilated, but react to light and accommodation. In severe cases there may be œdema of the feet and ankles. Mentally there is always more or less depression and confusion. Any attempt at conversation or the simplest mental work quickly leads to exhaustion. There is no loss of knowledge of position. Hallucinations may or may not be present. Delusions are not a necessary symptom, but when present they are always of the nature of suspicions of poisoning, of electricity and unseen agencies, and of people talking about and laughing at the patient. Loss of self-control may show itself in little outbursts of temper at trifles, irritability, obstinacy, and causeless weeping. The power of attention is deficient. The memory is accurate for recent and past events. Speech is lacking in vivacity and character. There is never incoherence. Often the comprehension of language is a labour to the patient. Sleep is always deficient. The prognosis is largely influenced by the age of the patient and the cause of the physical exhaustion; the younger the patient the better it is. Lactational cases generally recover; those which follow influenza, fevers, general infective conditions, and prolonged physical and mental strain, are slow in convalescence, and frequently pass into a chronic condition of ill-health. The diagnosis depends upon the exhausted physical condition and the absence of any symptoms of toxæmia. Treatment consists in prolonged rest in bed, careful attention to the diet, and general tonic treatment.

The following is a typical example of a case of exhaustive insanity:—

Mrs. S., aged 38, was admitted into the asylum suffering from delusions of suspicion regarding her neighbours and certain



unknown persons. Her illness, taking into account all the physical symptoms, had lasted for four years.

*History.*—No hereditary predisposition was admitted, but the patient was undoubtedly a neurotic woman. She had a comfortable home, and never had suffered from any previous mental attacks.

Four years ago the patient's baby died. She had nursed the child for ten months, and had become much run down and emaciated. Immediately after this she took influenza and was sharply ill. Upon recovering from the influenza she felt nervous and suspicious of those about her. Two years ago she began to imagine that her neighbours were speaking about her, and as she had a "prickling" sensation in her skin she thought some one was "playing electricity" upon her. She was at this time sleepless and dyspeptic. Her husband changed his house, and for a time the change did good; but the patient never had a chance of resting, as she had three young children to look after and the housework to do besides. The old delusions of suspicion and electricity returned, and she was certified and sent to the asylum.

On admission she was a thin, weakly, ill-nourished woman, sallow and anæmic, and obviously depressed. She had an exhausted, confused appearance. She took her food, but complained of a feeling of weight and uneasiness after eating. Her bowels were confined.

A blood examination gave the following results:—Hæmoglobin, 45 per cent; red cells, 3,500,000; leucocytes, 9000; polymorphonuclears, 47 per cent; small lymphocytes, 23 per cent; large lymphocytes, 28 per cent; eosinophiles, 0; mast cells, 2 per cent. Her arterial tension was 140 mm. Hg., and her pulse was 80. She complained of an occasional "prickling" sensation in her skin. The skin was as a rule dry, but every now and then she had drenching perspirations. She was menstruating upon admission. In her nervous system there was no abnormality except the subjective sensation of "prickling," and her voluntary movements were sluggish. Mentally she was depressed and confused. She stated that she had difficulty in understanding what was said to her. She further stated that she thought her neighbours laughed at and talked about her, and that she thought the "prickling" sensation in her skin must be electricity. Some time prior to admission she had jumped out of a window, and she told me this was due to a voice she heard telling her to do this thing, and for the time being she lost her head and tried to escape from this unseen terror. She took no interest in her personal appearance, and she told me that she only wished to rest, that she had no desire to work or talk or occupy herself in any way. Her memory appeared good, but it was evident she had difficulty in recalling recent events.

The morning following admission she had a temperature of



99° F., and she was very irritable and difficult to manage, refusing food and throwing herself about in bed. In the evening her temperature was 98·6°. The following day her temperature was normal, and she was quiet and reasonable. She was kept in bed for two months, fed very carefully at first with milk and farinaceous foods, and later put upon ordinary full diet. She was given tonics of quinine, arsenic, nux vomica, and dilute acids, which were frequently changed. At the end of five months she had gained a stone in weight, and she was discharged recovered. At no period during her illness did she present any symptoms of toxæmia.

Now here was a patient who exhibited a variety of mental symptoms. She was depressed and delusional, at one time obstinate, resistive, and troublesome, and yet there can be little doubt but that her mental symptoms were only one of the evidences of an exhausted physical condition.

**2. Insanity the Result of Gross Brain Lesions and Traumatisms.**—The brain lesions most apt to be followed by mental breakdown are those which affect the motor regions, particularly lesion of the left frontal lobe, implicating Broca's convolution, and are in the majority of instances of vascular origin. The mental symptoms may come on immediately after the brain lesion is acquired, especially if there is aphasia, or they may be delayed for some months. The early symptoms somewhat resemble mania, and may be very acute. They are probably due to cerebral irritation from the extravasated blood, to impairment of cortical nutrition, and to the absorption of some of the products of the breaking-down red blood corpuscles. These acute symptoms rarely last more than a few days, and are followed by a condition of childishness, emotionalism, extreme irritability, and outbursts of causeless rage. These later symptoms may also come on gradually as the result of brain lesion without any acute period of onset. There are no physical symptoms except those of paralysis corresponding to the areas of the brain affected.

G. W., male, aged 51, was admitted suffering from right-sided hemiplegia, with aphasia and excitement.

*History.*—He has been a steady, industrious, hard-working man, who rarely indulged in alcohol. Three nights previous to admission he went to bed complaining of headache and dizziness, and awoke



in the morning paralysed in the right arm and aphasic. In the evening his temperature rose to 100° F., and he appeared confused and talked incoherently. He was restless and delirious all night. In the morning it was noticed that the right leg was also paretic. The second night of his illness the delirium gradually passed into mania, during which he was very noisy and difficult to manage.

On admission he was a big, well-made man, with tortuous atheromatous arteries. The right arm was completely paralysed, but the right leg was capable of some movement. The patient was quite aphasic. Mentally he was confused, restless, and so far as he could be, violent, kicking and striking at those about him. He did not apparently understand what was said to him, and every now and then gave vent to inarticulate cries, evidently attempts at speech. He was put to bed, and kept as quiet as possible, sleep being obtained when necessary by means of chloral.

A week after admission he was no longer maniacal, but sullen, morose, and irritable, and he was with difficulty coaxed to take sufficient food. In a month he was able to move about with assistance. He seemed to live in a chronic state of irritability, and when spoken to would turn savagely upon his addresser and snarl, "You're a liar"; any further friendly overtures were invariably met by a volley of "You're a liar" crescendo. These words, "You're a liar," and "damn" were the only four I ever heard him utter. He used to try to read, but one could never ascertain if he understood what he was reading. For the greater part of the day he would sit in the hospital gazing into vacancy or limping about the ward. Sometimes he had crying turns, when he howled and sobbed like a child, apparently without cause. He did not recognise his relatives when they came to see him, or if he recognised them he very evidently did not desire to see them. His general nutrition failed very much in spite of the fact that his appetite was good. He died of pneumonia two years after admission.

Traumatisms of the brain have been accredited with producing all sorts of mental diseases, such as acute mania and general paralysis. They may produce such conditions, but I know of no cases in which the examination of the patient has been sufficiently complete to exclude toxæmia. Epilepsy, however, is a condition which certainly does follow brain injuries, and as a result of the epileptic discharges one finds symptoms of mental irritability, impulsiveness, and morbidly violent temper, and the pernicious effects of alcohol on such brains in producing wild, furious attacks of transient mania



while the alcohol is circulating in the blood stream are well recognised. As the result of exhaustion from repeated epileptic discharges, also, one finds stuporose conditions, total loss of memory, and loss of the power of continuous mental application.

**3. Traumatic Insanity.** — Kraft-Ebing divides insanity which follows head injuries into—

(1) Those in which the insanity immediately follows the accident.

(2) Those cases in which a change of disposition and character follows the accident, these symptoms frequently terminating in an attack of insanity.

(3) Those cases in which insanity occurs months or years after the accident.

In the first class the mental symptoms are those of a toxæmia. In all probability the condition is one of metabolic disorder. The patient is confused, suffers from hallucinations of sight and hearing and fleeting delusions. The speech is incoherent, and the general appearance that of delirium. The physical symptoms are those of shock. The expression is anxious, the temperature subnormal, the pulse slow and soft, the mouth dry, and the breathing shallow.

In the second class the accident has usually produced injury of the cerebral cortex, either by hæmorrhage, depression of the bone, or resulting localised inflammatory lesions. The earlier mental symptoms are irritability, lack of interest, incapacity for work, failure of memory and suspiciousness. These earlier symptoms may be followed by acute maniacal attacks, very similar to epileptic mania, and often complicated by epilepsy.

In the third class one could place at least 50 per cent of the patients admitted into asylums. It is very rarely, indeed, that the friends of an insane patient cannot remember some head injury which in their opinion is responsible for the mental attack. Such head injuries may be and are the starting-point of epilepsy, especially if the patient takes alcohol freely; but every other case of insanity following long after an injury which I have been able to examine was either hereditarily predisposed to mental disease, or suffered from



insanity which it was reasonable to suppose arose from causes quite apart from the accident.

A. G., a male, aged 39, was admitted in February 1900, suffering from acute excitement. The patient was by occupation a carter, married, and the father of five children, all healthy and living. Six months previously he was kicked upon the head by a horse, and was unconscious for some hours. A week after the accident, however, he was back at his work. His wife did not notice any difference in his conduct, beyond the facts that he was more irritable than usual, and that either he was drinking more alcohol, or the alcohol which he habitually took affected him more easily, as he came home frequently the worse of liquor.

One week before admission he was found lying unconscious beside his cart and breathing heavily. He was carried home and put to bed, where he gradually regained consciousness, but was confused. The same night he became excited, wandered about the house, and apparently suffered from hallucinations of hearing and sight. He was kept at home for the rest of the week, but finally became so violent and dangerous that he was removed to the asylum.

On admission he proved to be a strong, well-developed man. His temperature was 100° F., his pulse 90, full, and strong, and his leucocytosis 16,000 per c.mm. Mentally he was quite unconscious and wildly excited. He was put to bed, his bowels cleared out by a saline purge, and in three days he was quiet and sensible. No depression or scar was found over the cranium. His leucocytosis had now fallen below 10,000 per c.mm. One week later, while sitting in the ward, he suffered from a severe epileptic attack, which left him confused, but no maniacal attack followed. He was put on full doses of potassium bromide for three months, and as he never had another epileptic attack and appeared to be quite sane, his friends took him home. Six months later he was found dead by the roadside. In this case probably the accident, plus the alcohol, combined to produce an explosive condition of the brain cortex.

A. C., aged 40, a coachman, was admitted, suffering from excitement, with extravagant delusions. His friends were most positive that his illness dated from a carriage accident in which he received head injuries, and which had occurred twelve months previously. On examination he proved to be a case of general paralysis, with a well-authenticated history of having acquired syphilis six years prior to the accident.

W. H., a male, aged 67, was admitted in July 1885, suffering from complete change of character and mental enfeeblement. Two of his children have been insane and treated in the asylum. Two



months before admission, while working on the railway, he was knocked down and badly concussed, being unconscious for more than an hour. For a week he was confused and depressed. Then he became sleepless, very irritable, and later noisy at night.

On admission he was thin and poorly nourished. There was some impairment of sensibility to pain over the left side of the body, with paresis of the muscles on the same side. The pupils were irregular and unequal, the left being the larger. Both reacted to light and accommodation. Mentally he was confused, understood very little of what was said to him, and his memory for recent and remote events was very defective.

He lived for three months after admission. During this period he became profoundly demented, very dirty in his habits, and was at times excited, and at others listless and dull. He also suffered from several epileptic seizures.

At the post-mortem examination no lesion was found in the brain, but there was marked general atrophy, with hyperplasia of the fibrous tissue in both the grey and white matter.

Insanity, the result of intracranial tumours, is of rare occurrence. Impairment of the mental faculties, such as loss of memory, irritability, altered character and habits, occur to some extent in every case, but very rarely pass into states of excitement or depression. In the later stages, when the intracranial pressure is increased, the mental symptoms resemble those of stupor or coma, but are then usually associated with physical symptoms of paralysis, etc.

F. B., female, aged 19, was admitted as a case of catalepsy.

*History.*—About two weeks previously her friends noticed that the patient was very easily irritated. Her memory was affected; she made mistakes in her work, such as writing the same letter twice, giving wrong change, and forgetting orders received. Then she left work altogether, took to bed, and refused food. She vomited a good deal. When she did get out of bed she postured and attitudinised, and when she walked she strutted about with her head in the air and her shoulders held back.

On admission she appeared to be demented. Her pupils were dilated, but equal and reacted to light. Optic neuritis was well marked in both eyes. She never spoke except in monosyllables, and did not appear to understand what was said to her. There was no paralysis. A fortnight after admission she died suddenly. At the post-mortem examination a glioma was found which implicated the left optic thalamus.



F. C., female, aged 18, was admitted, suffering from paralysis of the right side of the body.

*History.*—The symptoms in this case had only lasted a week prior to admission. The patient first became irritable, forgetful, and unable to work. Then paralysis of the right leg came on quite suddenly, and this was followed by paralysis of the right arm and right side of the face. This paralysis was not persistent. The facial paralysis would some days pass off altogether.

On admission she looked dazed and confused. Her gait was dragging on the right side, there was also some paresis of the right arm, and some drawing of the face to the left. There was slight optic neuritis in the left eye. She understood what was said to her, but never spoke. She seemed to have no memory. She was put on full doses of potassium iodide and mercury, and in the course of a month the paralytic symptoms had passed off and the optic neuritis had diminished. She still remained speechless, although she knew what was said to her, and was not aphasic. Her mental reactions were very slow. She was discharged in this state, but completed her recovery at home. This was diagnosed as probably a case of tubercular tumour.

D. F., male, aged 19, was admitted, suffering from excitement and grandiose ideas.

*History.*—About a week before admission the patient left his work and sat about the house doing nothing. Then he became sleepless and restless, and he seemed to have no memory.

On admission he was a well-nourished, muscular man. There were absolutely no physical symptoms beyond optic neuritis in both eyes and sugar in the urine. His gait was stilted, and he carried himself in a peculiar manner. Mentally he was rather talkative, and had an abnormally high opinion of himself and his capacity for work. He had practically no memory.

For the next three weeks he was sometimes stuporose, sometimes elevated and talkative. The optic neuritis steadily increased, and his gait became more stilted.

He gradually became more and more stuporose, and was removed by his friends.

As the result of slowly advancing cerebral destruction, either through softening following interference with the blood supply, or from a gradually progressive degeneration of the brain cells from causes at present unexplainable, one meets occasionally with a train of mental symptoms which are characteristic of the condition, and do not differ from those of profound secondary dementia.



C. M., female, aged 55, was admitted into the asylum on August 29, 1904. She had been ill for two years.

*History.*—No hereditary predisposition to brain disease admitted by the friends. The patient had led a quiet, industrious life as a dressmaker. She had never had any previous mental attacks. Two years prior to admission it was noticed that her memory began to fail; then she gradually lost the power of speech. At first there was only a difficulty in finding suitable words to express her meaning, but finally she could not express herself at all. A year later she had an attack of excitement, when she screamed and struggled. This lasted for about one week. Six months after she began to suffer from attacks of "fainting," when she would suddenly fall to the ground without warning or cry, and get up at once. Her sister stated that during these faints she did not apparently lose consciousness. These attacks came on in the morning immediately after the patient had risen from bed, and as soon as she had had something to eat the attacks ceased. She lost control of the functions of the bladder and rectum about a year after becoming ill. She took food well. She had no intelligence. She did not know her own sister, nor did she recognise her surroundings. She had no affection or interest in her friends, and she had no memory. Her speech was babbling and unintelligible. She did not understand what was said to her, and could not read or write. Her sister states that speech, reading, writing, and memory all failed gradually and together, but the power of writing was the last to entirely disappear. She slept well.

*State on Admission.*—She is a well-developed woman, but is now bent, with marked spinal curvature to the right. This "stoop" has developed since the illness commenced. Her expression is without life, interest, or intelligence. She wanders aimlessly about the ward, knocking over tables and chairs as if she did not see them. So far as can be ascertained there is no disease of the alimentary, circulatory, or respiratory systems. Her leucocytosis is 8700 per c.mm., with a polymorphonuclear percentage of 60.

Her skin is soft and greasy. No urine could be obtained for examination.

*Nervous System.*—She appears to feel pain and heat. She sees, but how much cannot be tested. The left pupil is larger than the right, both are regular in outline, and react to light and apparently to accommodation. There is no paralysis of the orbital muscles and no evident nystagmus. Hearing, taste, and smell cannot be tested. She has no control over the reflexes of micturition and defæcation, but there is no paralysis. Her knee-jerks are exaggerated. She has power of movement in all her limbs, but the movements are lacking in purpose and grace. She shuffles



when she walks like a senile person, and her arm movements are awkward. She is muscularly very strong, and struggles violently if arrested in her aimless wanderings.

*Mental Functions.*—She is absolutely without intelligence, and she cannot be got to comprehend anything. Her attention cannot be attracted. She has no wants and no interests. She swallows her food when it is put into her mouth, but she would die of starvation unless some one brought food to her and fed her. She sleeps well at night. For two months the patient remained in this condition, but gradually lost the restlessness which impelled her to move about the ward, and she was kept in bed. For six weeks she practically slept day and night, gradually became weaker, and finally died without presenting any symptoms of acute intercurrent disease.

4. **Brain Anæmia.**—The mental symptoms which accompany diseases which produce narrowing of the lumen of the cerebral capillaries may, of course, be due to the irritant or toxine which is the cause of the vascular lesion acting directly upon the brain cells. Such conditions are common and are described elsewhere. But it is reasonable to suppose that arterial changes which may produce anæmia of the cerebral cortex may also produce mental symptoms. These mental symptoms take the form of loss of intelligence and incapacity for mental work, delusions of suspicion and unseen agency, associated with disorders of common sensibility. Physically there is always high arterial tension, but the heart's action is not quickened or irregular in force or rhythm. There is no increase of leucocytosis, and there is no evidence that metabolic waste products accumulate in the body of the patient. In practice I do not think it is possible to distinguish such cases from those in which there is no doubt that chronic metabolic disorders are directly responsible for the mental and physical symptoms.

5. **Insanity resulting from Deprivation of the Special Senses.**—Of the five special senses, those of sight and hearing are undoubtedly the most important in the symptomatology of mental disease. It is by means of these senses that we keep in touch with our environments, and adapt ourselves to the constantly changing conditions under which we live and work. A man may lose his sense of smell or taste, and no one is



cognisant of the fact; but the loss of sight or hearing immediately places the loser at a disadvantage with his fellow-men. It is not surprising, therefore, that in persons predisposed to mental instability the loss of either of these senses is sometimes followed by mental symptoms. The man who cannot hear is cut off from brain stimuli which are necessary for healthy mental life, and in addition is constantly endeavouring to understand, by the use of his remaining senses, what is being said about him. In this he largely fails, or arrives at false conclusions. In the same way the loss of sight produces a sense of helplessness, loss of the sense of position and the true knowledge of surrounding actions. The remaining senses as a result acquire new activity. There is, therefore, probably an over-stimulation from the senses which remain, and deprivation of stimulation from the sense which has been lost. This disproportion of brain stimuli is, probably, a negligible quantity in the development of the mental symptoms; rather it is the sense of helplessness and loss of touch with the environments which, in the first place, make the blind or deaf man suspicious, these suspicions gradually assuming definite form and becoming delusions. In the few cases of insanity from deprivation of the senses of sight or hearing which I have seen, the patients were, without exception, labouring under delusions of suspicion. The deaf man who sees his neighbours laughing first thinks they are laughing and talking about him, and the morbid process easily passes into a certainty. In the same way the blind patient hears movements which he does not see, and is first suspicious that actions are taking place of which he is to be kept in ignorance; and, finally, these suspicions assume the definite opinion that he is being robbed, that strangers habitually use his house, and that various impediments are placed in his way so that he by falling over them may injure himself. In no case have I ever seen any physical symptoms associated with such mental symptoms.

The treatment of such a case is moral and sympathetic. Gain the confidence of your patient, and the symptoms lose their acute character. Want of tact, sympathy, and straightforward conduct on the part of the friends or guardians will at once light up again all the old suspicions.

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Insanity directly the result of mental shock—that is to say, following immediately upon the receiving of the shock—is stated to occur; but as I have never seen such a condition I cannot describe it. If it does occur, it is highly improbable that it is toxic in origin, therefore I have merely mentioned this class of case under the non-toxic group of mental conditions.



## CHAPTER VI

### INSANITIES OF TOXIC ORIGIN

#### GROUP I.—INSANITIES THE RESULT OF TOXINES OF METABOLIC ORIGIN

**METABOLIC** toxæmia is certainly not a scientific term, because the toxines themselves are unknown. The most important metabolic toxines are probably the preurea bodies, and, to a lesser extent, the ethereal sulphates. In certain kidney conditions deficient excretion of the waste products is accompanied by well-recognised physical symptoms, such as high arterial tension, quickened pulse-rate, and anæmia.

When one finds somewhat similar physical symptoms in some acute mental diseases, associated with deficient excretion of urine and urea; and, further, when one finds these diseased conditions suddenly passing off with a coincident increase of the excretion of urine and urea, it may be presumed that the mental symptoms and the diminished or increased excretion of urine and urea have some definite relationship to one another. In all acute forms of insanity there are evidences of metabolic disorder, but the type of insanity in which metabolic toxæmia is outstanding, and is probably the chief cause of the disease, is acute melancholia.

**Acute Melancholia.**—At the present time all forms of melancholia are regarded as varieties of one disease, and classified under the heading "States of Mental Depression." Some authors go so far as to say that melancholia is merely an exaggeration, a pathological phase of the physiological condition of melancholy, and state that the difference between

melancholy the physiological state, and melancholia the pathological condition, is that in melancholy the cause and effect are in proportion to one another, while in melancholia the effect is out of proportion to the cause.

When one comes to study the physical symptoms of the various states of melancholia, it will be found that while in one patient there are disturbances of the alimentary, circulatory, and excretory systems, with impairment of consciousness, vivid hallucinations, and loss of self-control, in another there is very little physical disturbance, but pure mental depression, without impairment of consciousness, without hallucinations, and without loss of self-control. In other cases, again, an examination of the blood reveals the fact that one patient is passing through a period of severe toxæmia, while in another a blood examination gives no clue to the condition. With such clinical evidence there can be but two conclusions: firstly, that melancholy and melancholia have nothing in common; and, secondly, that there are at present several diseased conditions classified under "States of Mental Depression."

Acute melancholia is a definite physical disease with definite physical symptoms, and it is a disease of adult life and the decline of life. Typical acute melancholia is not characteristic of adolescence, and when it does occur in adolescence it is evidence of bad heredity, and is liable to recur and eventually end in a chronic delusional state. The chief predisposing cause is hereditary predisposition, and after that come any wearing-out physical and mental conditions, such as overwork, anxiety, sick-nursing entailing worry and deprivation of sleep. These latter might also be called exciting causes, in so far as they produce disorder of the metabolic processes of the body, which is the direct cause of the condition. The onset of the disease is slow and insidious; indefinite symptoms of illness, gastric disturbances, loss of body-weight, and sleeplessness are often present for weeks and months before mental symptoms are noticed.

The subjects of acute melancholia are not necessarily of poor physique, but by the time mental symptoms manifest themselves they are thin and poorly nourished. The general appearance and expression are those of depression, sometimes



with confusion and fear. The temperature is often slightly raised in the evening.

For purposes of description the disease is best divided into an acute stage of onset and a subacute stage, which may lead to recovery or prolonged illness. In the acute stage one finds the following symptoms:—

The tongue is furred and foul. There is no desire for food, but excessive thirst is often present. The digestive power of the stomach juices is gone. I examined the stomach contents of six cases of this disease with regard to the digestive power. A test breakfast was given, and the contents of the stomach withdrawn at the end of an hour. The contents were filtered, and 8 c.c. of the filtered fluid were placed in test-tubes with a measured quantity of coagulated egg albumen. A control experiment with artificial digestive fluid was used in every case. The tubes were placed in the incubator at 37° C.; at the end of ten hours the stomach fluid from the cases of melancholia had not acted upon the egg albumen, while the control fluid had acted vigorously. The motor power of the stomach was also observed to be weak.

The bowels are constipated, and the stools abnormally offensive.

In a pure uncomplicated case there is no marked leucocytosis.

The pulse is rapid—90 to 120 per minute—and is generally irregular in force and rhythm. The arterial tension is high—140 to 180 mm. Hg. The skin is hot and dry. The urine is scanty, and passed at long intervals. The excretion of urea is deficient, and a trace of albumen is often present. The failure in the urinary excretion is one of the marked symptoms of the disease. In health the urine excreted is in proportion to the fluid ingested, but in acute melancholia, although the patient may be taking two to three pints of fluid in the twenty-four hours, I have seen so low an excretion as 2 oz. of urine for the same period. What becomes of this fluid? It is not excreted by the skin or by the lungs. One is forced to the conclusion that there is a breaking up of water in the system, and this is further borne out by the fact that it has been noted in those cases which have died of the



disease that the tissues post-mortem were abnormally dry. The excretion of urea is on an average below 200 grs. for the twenty-four hours, but varies in proportion to the age, weight, and sex of the patient.

*Nervous System.*—The sensibility to heat, touch, and pain is frequently diminished. The special senses of sight and hearing, so far as their discharge of function is concerned, are not implicated, but both taste and smell may be disordered. The organic reflexes of micturition and defæcation remain under control. The skin and tendon reflexes are sometimes exaggerated. Voluntary muscular movements are sluggish, and the powers of fine co-ordination are impaired.

The nutritive function is markedly disordered. One of the patients under observation lost 9 lbs. in seven days.

*Mental Symptoms.*—The mental symptoms vary in intensity; in some cases they are quite mild, while in others they are very acute indeed. In a case of average severity there is depression, which is markedly accentuated if unpleasant hallucinations are present. Intelligence is always affected. There is confusion of thought, with more or less loss of the knowledge of position. Vivid hallucinations, chiefly of hearing, are common, and as a result there may be delusions expressed, but, as a rule, the mental confusion masks the delusions, which become more noticeable in the subacute stage of the disease. There is complete loss of the sense of personal neatness and order, no attention being paid to clothing or personal appearance.

Loss of self-control shows itself in restlessness and impulsive movements, such as sudden accessions of fear, purely reflex in character, and directly due to unpleasant hallucinations. Causeless weeping and fear of personal injury are due to the same cause.

The whole attention of the patient is self-centred and subjective. The memory cannot be tested, and speech, though not incoherent, is jerky and spasmodic. As a rule, the patient understands language both spoken and written. Sleeplessness is a constant symptom. Put shortly, the mental symptoms are those of confusion plus hallucinations of hearing, and possibly of sight, which markedly affect the patient's conduct.



Such symptoms are by no means confined to this one type of insanity. This acute stage may last for one, two, or even three weeks, and the patient in rare cases may pass directly into a stage of convalescence and recovery, or more commonly the symptoms, both mental and physical, gradually become less acute, and the patient passes into a state which may be called the subacute stage of the disease.

In the subacute stage there is no longer the restlessness or distress of the acute period. The tongue cleans first at the tip and edges; the appetite for food returns; the digestive power of the gastric juice, at first weak, becomes more active, and there is less tendency to constipation. There is no hyperleucocytosis upon recovery, and no agglutinines have so far been noted in the blood serum. The pulse is regular, softer, from 70 to 80 beats per minute, and the arterial tension falls to 120-130 mm. Hg. The skin becomes moister. Sometimes there is profuse perspiration, as in the crisis of pneumonia. The urinary excretion becomes more abundant, often rising well above 50 oz. for the twenty-four hours, and the excretion of urea is increased in some cases to 700 or 800 grs. for the twenty-four hours, and this is particularly noticeable in cases which pass by crises into comparative convalescence.

The voluntary movements become more natural. Mentally there is less confusion, and if hallucinations exist they do not markedly affect the patient's conduct; in other words, there is some return of the power of self-control. The attention is more readily attracted, and the patient is no longer so absolutely self-centred as in the acute stage. If any strain is placed upon the powers of attention, however, the patient soon shows signs of mental exhaustion.

The memory of what has happened in the acute stage is imperfect, or absolutely nil, but there is memory of previous events. Speech is slow, and sometimes difficult, but spoken and written language are now more easily understood. The power of sleep also returns. The whole appearance of the patient recovering from acute metabolic toxæmia is that of a person who has recently passed through an exhausting physical disease.



The diagnosis depends upon the clinical symptoms: The disordered alimentary tract, the rapid pulse, the high arterial tension, the deficient excretion of urine and urea, and the absence of hyperleucocytosis. Mentally, the symptom of depression is the only constant one; all the other mental symptoms which may occur are common to all conditions of acute toxæmia.

*Terminations.*—Acute melancholia may terminate in rapid recovery by crises during the acute stage, or by gradual recovery during the subacute stage, or the patient may pass into a condition of chronic depression with symptoms of nutritive failure. The patient then is thin and unhealthy in appearance. The digestive powers are weak, and the appetite is poor, while the bowels are obstinately constipated. The pulse is weak and thready, but often of high tension, and the extremities tend to become cold and blue. The lungs are liable to be attacked by tubercule, the skin is dry and pigmented, and the hair is brittle and lacks lustre. The urinary excretion is very irregular, one day deficient, the next copious, and the excretion of urea is markedly irregular; there is want of tone in the nervous system; the general sensibility is impaired. The movements are sluggish, and lack purpose and energy.

Mentally there are depression, apathy, and often a delusion or delusions.

The patient may die, especially in the acute stage, through passing into a condition of typhoid exhaustion, or accidentally, as the result of suicide. It should never be forgotten that in the acute stage impulsive actions may result in suicide, and in the subacute and chronic conditions all depressed patients are liable to commit suicide. In some the tendency is very strong, perhaps to the exclusion of all other symptoms; in others, apparently, absent, but it is a factor to be constantly kept in view.

*Prognosis.*—In middle age or adult life the prognosis is good, and the patient should recover within six months. In old age and the decline of life the prognosis is not so favourable, and the subacute stage is apt to be prolonged. If recovery does not occur within a year the case is not neces-



sarily incurable, but the prognosis is much more grave. If acute melancholia occurs during adolescence the disease is likely to recur; and repeated attacks of melancholia, or a condition which closely simulates acute melancholia, are often the prelude to a condition of delusional insanity.

*Treatment.*—In the acute stage of the disease rest in bed is imperative, with efficient nursing, both by night and day. The state of the alimentary tract, and especially the failure of digestive power, indicate that the diet should be as simple as possible. The very best food is milk, which can be assimilated with the minimum of digestive power. Further, there is a demand for water, not only to meet the apparent destruction of water in the body, but also in order to act as a solvent and mechanical agent to assist in the excretion of solids. The sudden rise in the excretion of urea as the patient passes from the acute to the subacute stage points to the fact that there is during the acute stage an actual accumulation of waste products in the body, and it is necessary during treatment to flush the tissues to prevent such accumulation. The dietary, therefore, should conform to the following conditions:—

1. That the alimentary tract must be saved all unnecessary labour.
2. That the excretory functions must be assisted.

The only diet which meets these conditions is a purely milk one. While the acute symptoms last the patient should receive three pints of milk in the twenty-four hours, given in small quantities, and frequently. It is well to dilute the milk with aerated water, and also to give hot milk and water, as this assists the action of the skin. At least five pints of fluid should be given in the twenty-four hours. The bowels are best relieved by large saline enemata.

Under this treatment, especially if the disease is treated early, there is rapid and marked improvement. The function of sleep returns, the pulse-rate and arterial tension fall, there is a free and copious discharge of urine and urea, and the skin becomes soft and moist. As the appetite returns additions may be made to the dietary with caution. A sudden return to an ordinary full dietary is certain to be followed by a



relapse. At first absolutely purin-free foods should be used, such as bread or toast, butter, sugar, eggs, potatoes, and rice, and these should be substituted gradually for the milk. Later on very dilute tea, fish, and white flesh may be added.

The two most useful drugs during the acute stage are paraldehyde for sleeplessness, and citrate of potassium in 20-40 grain doses thrice daily to assist the urinary excretion. Paraldehyde is the best drug to use for the sleeplessness of acute melancholia, as in this disease the sleeplessness is due to high arterial tension. In observations made at Murthly it was found that a 3ii. dose of paraldehyde given to a sleepless melancholic with a tension of 140 mm. Hg., in producing sleep reduced the tension to 110 mm. Hg. Paraldehyde should not be given every night. My rule is to let two nights pass, and if sleep has not been obtained, a 3ii. dose is given on the third night. This gives nature a chance of re-establishing the sleep habit, whereas if paraldehyde is given every night one runs a risk of establishing a paraldehyde habit instead. Sulphonal should never be given as a hypnotic in acute melancholia. Experimentally it has been shown that sulphonal interferes with the elaboration of nitrogen into urea.<sup>1</sup>

After-treatment should consist of tonics, particularly quinine, dilute acids, and strychnine plus very gentle and gradually extended exercise. It is well to bear in mind that a state of metabolic poisoning may complicate or be complicated by other forms of toxæmia.

There are cases of metabolic toxæmia which might be called simple melancholia, so mild are the symptoms presented, and such modified symptoms may precede and terminate acute attacks, or the disease may never pass beyond the prodromal stage so far as the mental symptoms are concerned. This form of simple melancholia is, however, a different disease from typical simple melancholia, which latter is a disease characterised by pure mental depression without confusion or impairment of the intellect, and which lacks the symptoms of metabolic disorder,

<sup>1</sup> "On a Method of Estimating the Interference with the Hepatic Metabolism produced by Drugs," Noel Paton and J. Eason, *Journal of Physiol.*, vol. xxvi. p. 166, 1901.



and is only a stage of a more complicated disease known as "folie circulaire."

The following cases are examples of the symptoms to be met with in metabolic toxæmia:—

C. A., female, aged 28, by occupation a domestic servant, was admitted suffering from depression. The illness had lasted a few days.

*History.*—Her sister is insane; the patient is of correct and steady habits, but has already suffered from two previous attacks of melancholia. A few days ago she became sleepless, depressed, and refused food.

*State on Admission.*—She is a well-nourished, well-developed girl, depressed in appearance and attitude, and her complexion is muddy. Temperature 98.4°. Her tongue is furred and her bowels confined. She has no desire for food. Her leucocytosis is 13,700, with a polymorphonuclear percentage of 69. Her pulse is 90 per minute, the arterial tension being 130 mm. Hg. Her skin is greasy. During the first twenty-four hours after admission she excreted 12 oz. of urine, which contained 190 grains of urea. During the same period she had ingested 60 oz. of fluid.

*Nervous System.*—She complained of no subjective sensations. Her sensibility to heat and cold, touch and pain, is present, but conduction of sensation is delayed. Her pupils are medium, and react to light and accommodation. Her muscles are well developed. Her organic reflexes of micturition and defæcation are under control. Her skin reflexes are slightly exaggerated, and her tendon reflexes are present.

*Mental Functions.*—She is depressed and slightly confused, but does not suffer from hallucinations. Her memory is dulled. She will not speak, but answers in monosyllables, and evidently dislikes being spoken to. Her sleep is deficient. The first night after admission she slept four hours, and the second night she did not sleep at all. On the second day after admission she was placed on fluid diet. That evening her temperature rose to 99.8° F., and she was sweating profusely. A week after admission she was much improved; she was sleeping fairly well. Her tongue was clean, and she volunteered the remark that she felt much better. Her excretion of urine had now risen to 33 oz. in the twenty-four hours, and the urea excreted was 364 grains for the same period. She lost 2 lbs. in weight during the week. A full ordinary diet was given on the tenth day after admission, but the patient immediately became worse. The temperature rose a little at night, and in three days she was depressed and confused again. Fluid diet



was recommenced the following day, and continued for the four succeeding days. She again improved, the urinary excretion being 32 oz., and the urea 460 grains at the end of that period. Two weeks later she was allowed out of bed, at first for only one or two hours at a time. She made a good recovery, and was discharged within two months of admission.

The dry pigmented skin, the deficient excretion of urine and urea, the slightly febrile temperature unaccompanied by a hyperleucocytosis, combined with depression and slight confusion, are typical of a mild attack of metabolic toxæmia.

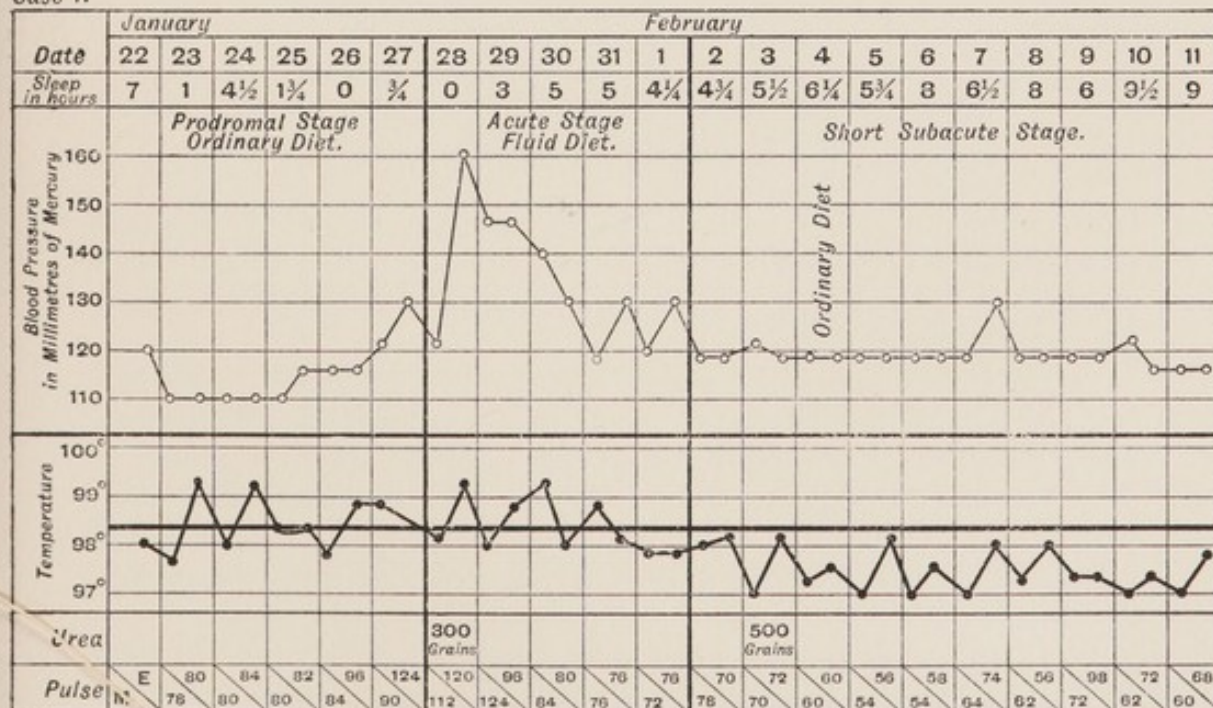
The leucocytosis for the first three days after admission is as follows:—

| Leucocytosis. | Polymorphs. | Lymphocytes. | Mononuclears. | Eosins. |
|---------------|-------------|--------------|---------------|---------|
| 13,700        | 69          | 20           | 10            | 1       |
| 10,300        | 61          | 30           | 8             | 1       |
| 9,711         | 63          | 31           | 5             | 1       |

Charts 1, 2, 3, 4, showing the temperature, pulse, arterial tension, and excretion of urea, are typical of acute melancholia.

CASE 1.—The patient was a female, aged 34 years. The chart

Case 1.



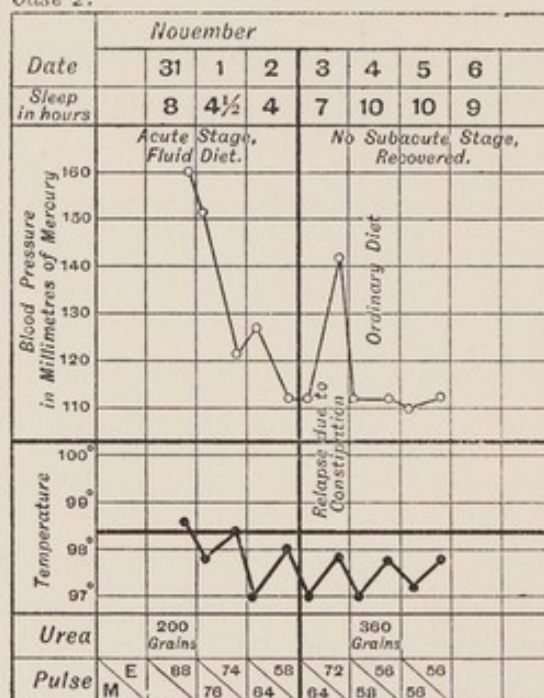
is interesting, because it shows the attack from the commencement. The temperature became febrile on January 23rd, but the tension did not rise until the 28th, when the symptoms became very acute.



Under fluid diet, treatment, and rest in bed, all the symptoms had subsided by February 1st. On the nights of January 29th, 30th, and 31st, sleep was obtained by the administration of two drachms of paraldehyde. She made a good recovery. Weight is rapidly lost during the onset of the disease, and this patient lost 9 lbs. in seven days though she was on ordinary diet.

CASE 2.—The patient was a female, aged 64 years. The attack was treated early—within a week of onset. The acute symptoms

Case 2.

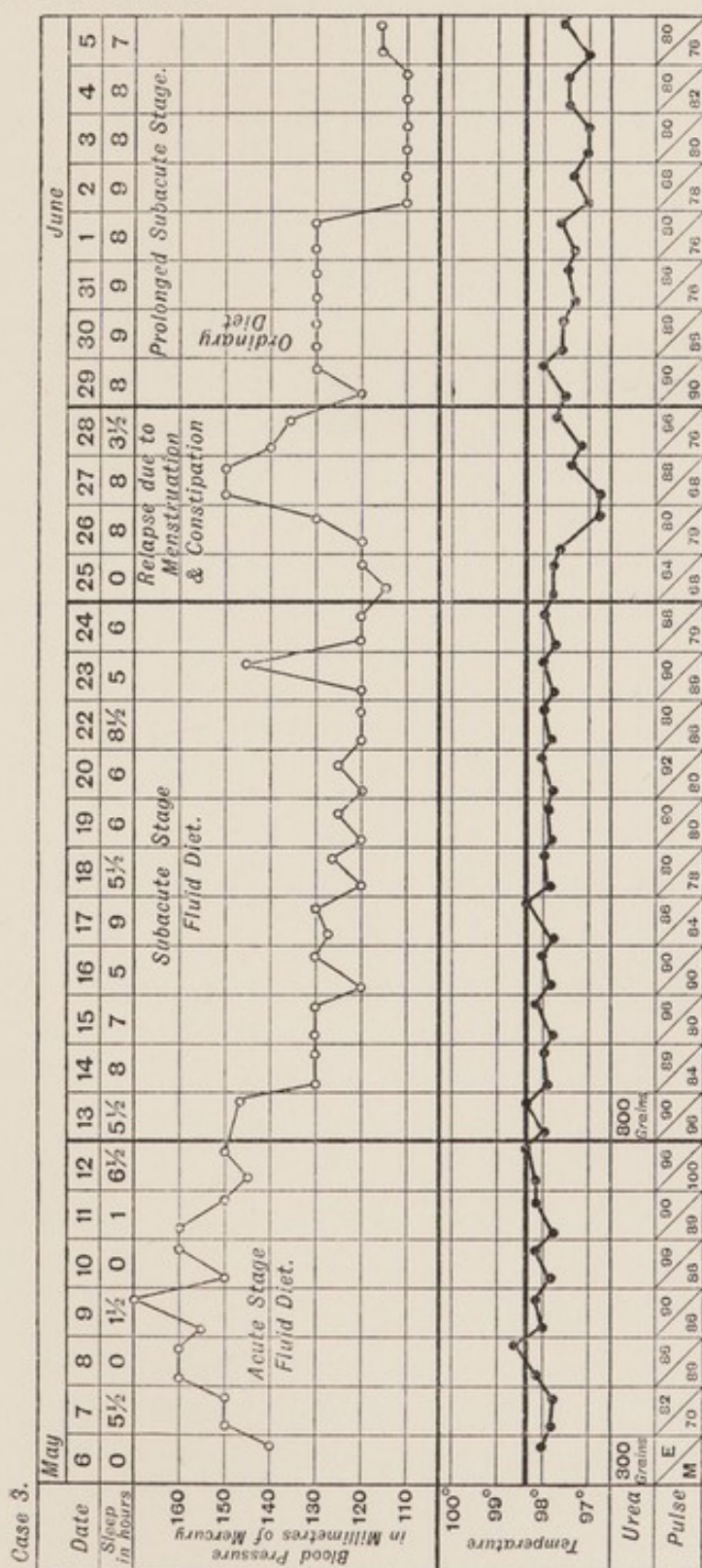


lasted only three days. Sleep was obtained by the administration of 2 drachms of paraldehyde on the nights of October 31st and November 1st and 2nd. There was practically no subacute stage. The patient recovered.

CASE 3.—The patient was a female, aged 42 years. She had been ill for one month prior to admission to hospital. The acute stage lasted for seven days. Sleep was obtained upon the nights of May 7th and 9th by the administration of 2 drachms of paraldehyde. The subacute stage was complicated by a threatened relapse, and was prolonged, but the patient has now completely recovered.

CASE 4.—The patient was a man, aged 34 years. He had been ill for two months prior to admission. The acute stage lasted for fifteen days. The onset of the subacute stage was gradual. The patient made a good recovery. In the acute stage sleep was obtained upon the nights of February 22nd, 25th, and 28th by the administration of 2 drachms of paraldehyde.

The following case is a good example of a very acute case of metabolic toxæmia:—

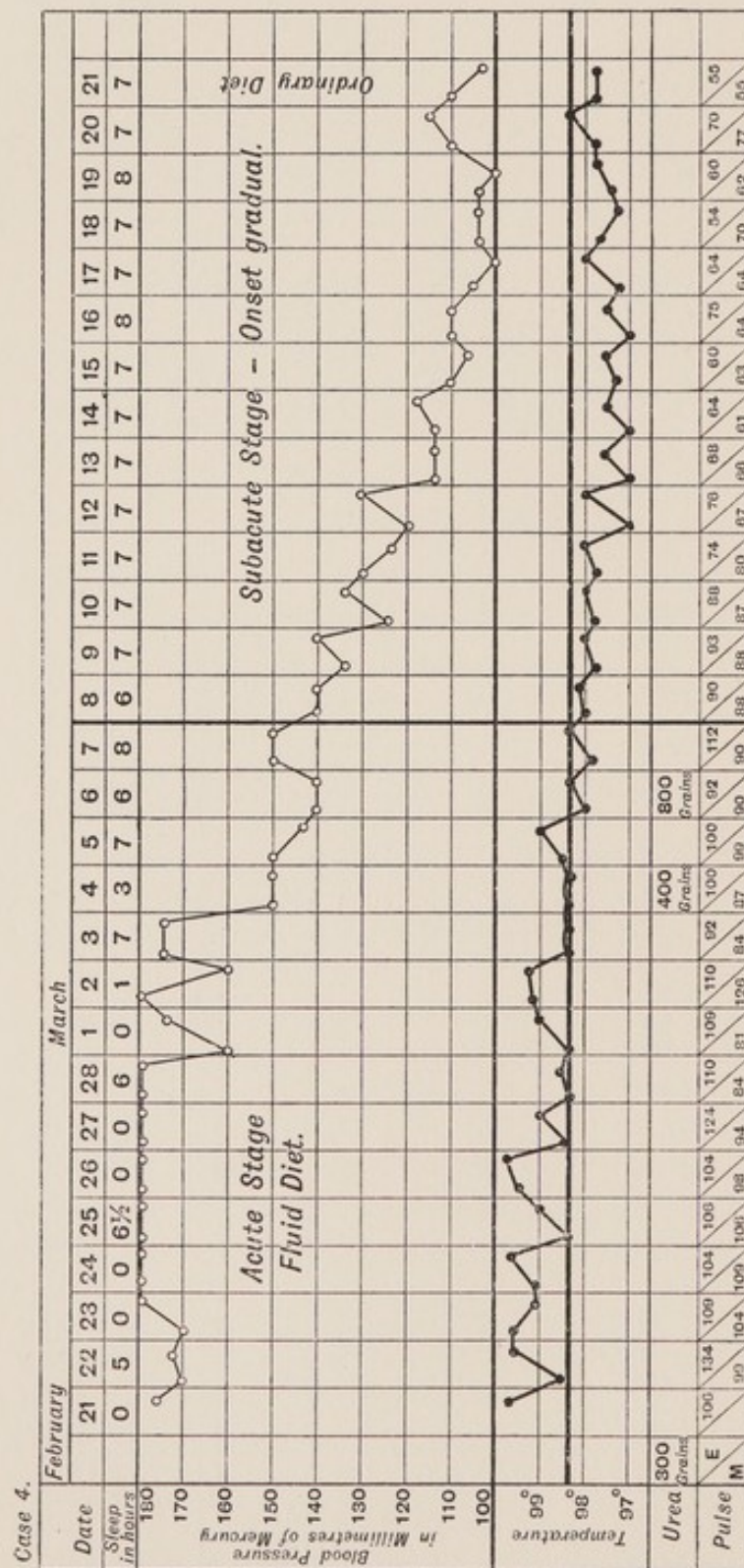


See page 74.

Mrs. J., aged 42, married, housewife, was admitted on 10th



August suffering from depression and mental confusion. She has been ill one week.



See page 74.

*History.*—No hereditary predisposition to insanity was admitted. She has been of steady habits, but recently has had a good deal of



domestic worry. She has not suffered previously from mental disease. Her illness is attributed to nursing. She nursed her last child for six months, and her physical health suffered, and she became depressed a week ago.

*State on Admission.*—She is thin and in poor condition, but is well developed. She looks very confused, and lies in bed staring vacantly about her. Temperature 99.6° F. Her tongue is furred, she has no desire for food, and the bowels are obstinately confined. Her leucocytosis is 11,000 per c.mm., with a polymorphonuclear percentage of 66. Her heart's action is rapid, her pulse being 84, and the arterial tension 150 mm. Hg. Her skin is dry, rather yellow and parchment-like. 12 oz. of urine were excreted in the first twenty-four hours, and 150 grains of urea were excreted for the same period.

*Nervous System.*—She complains of no subjective sensations. Her sensibility to touch, heat, and pain is slightly impaired. The pupils are widely dilated, but react to light. The organic reflex of micturition is not under control, as she passes urine in bed unconsciously. The skin and tendon reflexes are present, and are not exaggerated. She is slightly resistive to passive movements.

Mentally there is depression with confusion. She suffers from distressing hallucinations, and at times becomes terrified, and screams and struggles to get under the bed or out of the window. She does not know where she is. The power of self-control is lost, and she is impulsive in response to her hallucinations. She has no power of attention, and her memory cannot be tested. Her speech is clear, but often quite incoherent, and she never clearly understands what is said to her. During the attacks of terror and excitement she understands nothing, and becomes absolutely incoherent. For the past two nights she only slept four hours.

She was put on fluid diet on the second day after admission, but had to be fed by the nasal tube.

For the first five days she was acutely ill, the temperature was slightly febrile, the pulse-rate rarely below 90 per minute, and she was fed artificially. The tongue became brown and cracked, the teeth and lips covered with sordes, and the patient passed urine and fæces under her—the urine being voided at long intervals. The mental symptoms were unusually acute. There were complete loss of consciousness and constant restlessness. During the whole illness, however, the leucocytosis never rose above 11,000, nor the polymorphonuclear percentage above 70. Sleep returned on the evening of the fifth day. The arterial tension fell to 125 mm. Hg., and the patient took food voluntarily and was clean in her habits. The urinary secretion rose to 20 oz. for the twenty-four hours, and the urea to 260 grs. Mentally there was still confusion, but she



now understood what was said to her. On 19th August, ten days after admission, she excreted 24 oz. of urine and 390 grains of urea. She made a good recovery, and was discharged within two months of admission.

The treatment throughout the first three weeks consisted of an exclusively fluid diet of milk on which the patient actually put on weight. Large enemata of warm normal saline fluid were given every third day, and on two occasions  $\frac{3}{4}$ ii. doses of paraldehyde were given to produce sleep. Prolonged warm baths were given on two occasions during the first five days. The first appeared to give relief, and the patient slept for one and a half hours, but the second produced no beneficial action.

**Myxœdematous Insanity.**—The mental symptoms which result from failure in the secretion of the thyroid gland are slowness in the mental processes and a lack of initiative. The condition is one which closely resembles the state of secondary dementia, and the diagnosis depends upon the presence of the physical symptoms of myxœdema. According to Macpherson, delusions and hallucinations occur in about half of the cases, while mania or melancholia, with delusions of suspicion and self-accusation, occur in an equal number. It is highly probable that such symptoms occurring in cases of myxœdema are accidental complications. The treatment consists in administering thyroid extract in very small doses, the patient being rigorously confined to bed during the early days of treatment.

An excessive or altered thyroid secretion, such as occurs in exophthalmic goitre, produces a condition of excessive nervousness, loss of self-control, and excitability. According to Savage, mental symptoms associated with exophthalmic goitre are very variable, and may include depression, restlessness, incoherence, destructiveness, and sleeplessness, and are frequently associated with vomiting, diarrhoea, and altered appetite. The same symptoms may follow the administration of thyroid extract in large doses. I have seen one case of myxœdema which, under excessive doses of the drug, passed into a typical condition of acute mania with a marked hyperleucocytosis. Where insanity is associated with exophthalmic goitre I know of no treatment which is of any use, but possibly treatment with antithyroid serum may in the future be worth a trial.



Some cases of puerperal insanity are said to result from metabolic poisons arising from uterine involution, and more probably from disordered lacteal secretion, but so far I have had no opportunity of examining such cases.

Many cases of delusional insanity undoubtedly commence as conditions of acute metabolic poisoning indistinguishable from acute melancholia, and examples of this variety of mental disease are described under delusional insanity.

**Delusional Insanity.**—The term delusional insanity is used in this chapter to denote a variety or type of mental disease which is variously described as progressive systematised insanity, mania of persecution and grandeur, monomanias of persecution, unseen agency, and grandeur, megalomania, and, on the Continent and in America, paranoia.

There appears to be some doubt in this country as to whether paranoia and delusional insanity are the same disease.

Magnan has divided systematised delusional insanity into three stages—the stage of onset, the stage of persecutory delusions, the stage of grandiose delusions—but these divisions are based on mental symptoms only, which alone are no basis for a diagnosis in any one known form of insanity.

Many forms of insanity, particularly excitement with confusion, tend to become complicated by delusions if the patient does not recover, but the delusions in such cases are merely indications of chronicity and mental impairment, and there is always the history of the case, and in many instances physical symptoms, which help to differentiate such a patient from a case of true delusional insanity. Syphilis and chronic alcoholism also tend to produce mental conditions indistinguishable from delusional insanity, and the majority of authors describe such conditions under the terms syphilitic and alcoholic insanity.

There are, however, good grounds for the belief that the diseases variously described as progressive systematised insanity, mania of persecution, paranoia, etc., are not different diseases, but a similar disease process, whether arising through unknown causes or as a sequel of chronic alcoholism or syphilis, whether the onset is definite or indefinite, and whether occurring during adolescence or early adult life.



*Cause.*—The universally accepted predisposing cause is hereditary predisposition to mental disease which occurs in over 50 per cent of the cases affected. As to the exciting causes nothing is known beyond the fact that syphilis and long-continued alcoholic excess can produce the disease. In many of the cases the onset is gradual and unnoticed, in others the disease commences with acute symptoms very similar to those of acute metabolic poisoning, from which the patient may recover only to relapse later and pass into a chronic delusional state; and so frequently has this occurred in my own experience, that I incline to the belief that in many cases there may be an acute onset which may pass unnoticed, or may not be connected with the subsequent mental aberration, especially as months or even years may elapse before the delusional state becomes sufficiently developed to attract attention.

The disease occurs as frequently in men as in women, and, unless caused by alcohol or syphilis, commences during early adult life or in adolescence. But whether it occurs in adult life or adolescence it is essentially a disease of failure of nutrition, with vague symptoms of malaise and sensory disturbances, which sooner or later develop into definite hallucinations and delusions always at first of a disagreeable nature.

The histories of the cases which are said to have developed insidiously and gradually must be accepted with reserve, as the patient has often been ill for years when notice is first drawn to the condition, and the transient stage of acute onset has already been forgotten, or was never regarded by the friends as more than an accidental occurrence, in no way associated with brain disease or the subsequent delusional state. In the cases in which the acute onset is observed it is possible to trace the development of the delusional state. The acute attack is generally of the nature of acute melancholia, but with distinct delusions. The patient's digestive system is out of order and there is no desire for food. The temperature may be febrile, and the pulse fast, with a high arterial tension. The urine is excreted in small amount, frequently contains albumen, and there is a deficiency in the excretion of both



chlorides and urea. The leucocytosis is never raised throughout the whole course of the disease. Mentally there is depression with more or less confusion. Hallucinations, particularly of hearing, are always present, and associated with them may be delusions of a persecutory nature. There is loss of self-control, the patient being frightened and apprehensive. The power of attention is in abeyance and the memory untrustworthy, and although the patients may understand what is said to them they are too deeply engrossed with their own sensory disturbances to enter into conversation. Sleep is always abolished.

Under rest in bed and a simple dietary these acute symptoms quickly subside, and very frequently the patient is discharged apparently recovered, but this does not occur in every case. Some patients may have these attacks repeated several times before passing into the delusional state, the most marked symptoms of the later attacks being that the hallucinations persist in the periods of remission. In other cases, again, one attack is apparently sufficient to start the disease process, but more than a year may elapse before the delusional state becomes apparent. On the other hand, the patient may at once pass into delusional insanity.

The delusional state commences very frequently with hallucinations, generally of hearing, but in well-marked cases all the special senses become later involved. There are, however, cases in which hallucinations cannot be detected. At first the patient recognises that the hallucinations are not real impressions, but as judgment weakens they tend to affect the conduct and give rise to the various fixed delusions which are characteristic of this stage of the disease. The physical symptoms are now quite definite. The patient is thin and sallow, and has a furtive, suspicious, insane appearance. The saliva is sometimes secreted in copious quantities, and is thin and watery in character. There is always more or less gastric disorder, such as furred tongue, foul breath, and pain and fulness after taking food. The leucocytosis is below 10,000 per c.mm., and so far no agglutinines have ever been demonstrated in the blood serum. The heart's action is weak and irritable, and there is a tendency to cardiac failure. The



pulse is as a rule slow, but becomes rapid under slight provocation. The arterial tension is about 120-130 mm. Hg. The lungs may become infected with tubercle, but acute phthisis is an unusual complication. The urine is often excreted in large amount, sometimes due to the fact that these patients drink large quantities of water, but even when under observation the amount of urine excreted is above the average. There is also an increased excretion of urea, chlorides, and purin nitrogen. The skin is dry, sallow, and scurfy. The temperature chart every now and then shows slight unexplainable rises of temperature with an increased pulse-rate, and the patient may complain of indefinite symptoms of malaise, and during these attacks the percentage of purin nitrogen in the urine is increased. There are frequently sensory disturbances of the nature of pricking, tingling, and formication of the skin, which are ascribed to the use of electricity, X-rays, etc., employed by some person or persons, known or unknown, who are in league against the patient. Sensibility to heat, touch, and pain are not markedly if at all impaired. The pupils may be unequal, but react to light and accommodation. The special senses of taste and smell are quite commonly disordered. There is no implication of the organic reflexes, of the skin, or tendon reflexes, or of voluntary movement or co-ordination. The mental state is variable. Sometimes the patient is depressed, sometimes sulky, sometimes irritable and truculent. There is no confusion and no loss of knowledge of position. In typical cases hallucinations are always present. The patient may simply hear voices abusing him and making accusations against him, or his thoughts may be repeated from the floor, ceiling, or wall before they are uttered, or a letter in process of being written may be repeated word by word. The disordered senses of taste and smell suggest that there is poison in the food, or that foul gases are being blown into the room, while the sensory disturbances are explained as electricity, marconigrams, hypnotism, etc. Many patients have visceral sensations which are described as twisting, tearing, gnawing, or cutting, and give rise to further delusions of persecution. Sometimes these persecutions are ascribed to the malign influence of some known person, or some sect or order.



Associated with these sensory disorders and the persecutory delusions to which they give rise there are delusions of a different character, which suggest impairment of judgment, and a failure on the part of the patient to appreciate the relative value of position and environment. These delusions take the form of unpractical schemes and improbable expectations: one patient has discovered a panacea for all ailments, and expects daily a letter appointing him to a professorship; another has discovered perpetual motion, and spends all his time making models which never work; while a third is equally certain that he has discovered the secret of making gold out of earth and stones, and fills his pockets full of rubbish wherewith to carry on his experiments. They are all hopeful, and when their plans miscarry it is entirely due to the interference of their persecutors. If there is much depression, the delusions resulting from the sensory disturbances may be of a distinctly melancholic type, often of a religious nature, and such patients may be suicidal. Self-control is not lost in so far that the patient cannot control his actions, and though such patients are apt to make assaults under the influence of their delusions, these assaults are not of the nature of impulses, and the patient is cognisant of his actions, and lays his plans carefully beforehand. The power of attention is good, and the patient can talk sensibly and collectedly, and see reason on any subject except his own particular delusions. If there is no alcoholic complication the memory is not impaired. Speech is often voluble and forcible, but again many patients are taciturn and obstinately silent. Incoherence is only seen occasionally, and is most liable to occur in adolescent cases. Sleep is deficient, the patient being more disturbed by the sensory disturbances at night than during the day.

This state of physical and mental symptoms may last for years, the patient varying somewhat from day to day, or at regular intervals—in women particularly at the menstrual periods—so that the patient who is agreeable to-day may to-morrow be abusive and angry.

Magnan's third stage, or the stage of delusions of grandeur, does not necessarily appear in every case. The transition from the stage of persecution and suspicion to the false sense of



well-being associated with delusions of grandeur is a gradual one, although on one occasion the change appeared to take place suddenly at night. According to the patient's own account he heard in the night the voice of God telling him he was Emmanuel, and he at once realised the reason for all the persecution to which he had been subjected, and he awoke to a new sense of his duties in life.

So far as I have been able to observe, the stage of grandiose ideas is marked by the same physical symptoms which characterise the stage of persecution, and the change is apparently a purely mental one. The gait often becomes peculiar and mincing in character, and there is a tendency to be fantastic in dress and manner.

*Prognosis.*—The prognosis is hopeless as regards mental recovery, but some of these cases improve sufficiently to live in quiet country places. There is no risk to life, and as a rule little mental enfeeblement except in a few of the adolescent cases, who deteriorate rapidly in mind and become demented and incoherent.

*Treatment.*—In the acute stage, which simulates or is a form of acute metabolic poisoning, rest in bed with a fluid diet is the best treatment, but for the later stages characterised by fixed delusions nothing can be done beyond ordinary attention to the bodily health.

S. H., male, aged 54, was admitted to the Perth District Asylum, a confirmed case of delusional insanity of at least nine years' standing. The patient, who was by trade an engineer, had led a dissolute life, being much addicted to alcohol. No history regarding the onset of the illness could be obtained, and all that was known about the patient was that he had been in American Asylums for more than eight years, and upon his first acquaintance with these institutions he was apparently a fully developed case of delusional insanity.

On admission he was a stout, well-developed man. His expression was slightly furtive and suspicious, and his facial muscles were tremulous when he was speaking. No abnormality could be detected in the physical condition. He complained of vague sensory disturbances in the abdomen. His sensibility to touch, heat, and pain was acute. The pupils were unequal, the left being the larger, but both reacted readily to light and accommodation. The special senses were unaffected. Both the



superficial and deep reflexes were present and active, and there was no paresis or inco-ordination of the voluntary muscles. The muscles of the whole body, but particularly of the face and arms, showed coarse tremors, which became much exaggerated when the patient became excited. On the mental side there was neither depression, elevation, nor confusion. He gave a clear account of his past life and past experiences, admitted that he had drunk excessively, especially when living abroad, and upon arrival in America some nine years ago he had to seek police protection from the Jews, who were persecuting him. These persecutions, he went on to complain, consisted of marconigrams, by which his persecutors read his thoughts before they were uttered, tried to steal his ideas so that they might profit by them, and at night by the same means they tore and twisted his inside, and it is obvious that their purpose was to kill him. To draw attention to his case he on one occasion assaulted a fellow-patient while in America, and he has threatened to do so in this asylum, especially in the mornings, when he is most irritable.

There is, however, another side to his character. He is in his own belief an inventor whose discoveries are largely the cause of his being a "marked man." He believes that he has discovered perpetual motion, and he has some half-dozen schemes which are certain of success if some one would only take them up. He is a clever workman and his devices are ingenious; but although an educated man and an engineer by trade, he is unable to see the fallacies of the schemes he explains—fallacies which are apparent to men who understand nothing about mechanics or engineering.

The case of S. H. is an example of delusional insanity the result of or complicated by long-continued alcoholic excess.

D. J., male, aged 31, was admitted suffering from depression. No history of hereditary predisposition to insanity could be obtained. The patient had during his youth led rather a wild life, but for the last few years had been quite steady.

The present illness commenced a few weeks ago, when he became sleepless and depressed, imagined his fellow-workmen were tampering with his horses, drugging his whisky, laughing and staring at him and criticising him adversely. Beyond slight alimentary disorders and diminution in the excretion of urine and urea, no abnormality was detected physically. He suffered from hallucinations and also delusions of suspicion, and slept very badly. Under rest in bed and fluid dietary he improved rapidly, so that in two weeks he was out working in the garden, and in less than three months he was discharged apparently recovered; but he was always



a taciturn, incommunicative man and was unsociable. He was re-admitted sixteen months later suffering from well-marked delusional insanity. In appearance he was pale, sallow, and thin. He took food fairly well, but lost weight steadily. Every now and then his temperature rose suddenly without obvious cause. His heart's action was feeble but regular. He excreted large quantities of urine, with an excess of urea and chlorides. Mentally he was suspicious and irritable. He apparently suffered from hallucinations and delusions based on some sensory disturbances, but he refused to speak or answer questions when the subject turned on his mental symptoms. At times he made statements such as "There is a 'dead set' against me." Two or three months later his delusions became more pronounced, and he directed them against certain individuals. These delusions were rather peculiar. One was that certain people sat on his moustache, and to prevent this he borrowed a pair of scissors and cut his moustaches off. For the next three years there was steadily advancing mental deterioration; the patient became quite incoherent and was unable to express himself so as to be easily understood. It is now four years since the period of delusional insanity commenced, and so far as I can ascertain the patient has no delusions of a grandiose nature.

The following case of M. L. is of interest on account of the universal implication of all the special senses associated with delusions of persecution:—

The patient, a female, had two attacks of depression at the age of 30, in one of which she attempted suicide by poisoning. She recovered from the first attack, but after the second developed vivid hallucinations of hearing. She heard voices abusing her and accusing her of immoral conduct. Either coincidently or soon after the commencement of auditory hallucinations, hallucinations of all the special senses set in. Electricity was played on her at night, her food was poisoned, noxious gases emanated from the floor, and these were associated with delusions such as that her life was in danger, that the doctors experimented with her to see how much electricity she could stand, that she got human flesh to eat. She frequently saw paragraphs in the papers which referred to her case, and when she saw two people talking and laughing, they were always discussing her and devising new methods of torture. She was often noisy, abusive, and violent, but at other times was quite friendly. She read the papers, knew what was going on, and was a useful and intelligent ward worker. In the earlier stages of the disease her



memory was good, and if she made a promise she kept it ; but now, after ten years of illness, her memory is failing and her word is not to be depended upon. Physically she suffers from sleeplessness, chronic indigestion, and recurrent attacks of mild anginous pains.

**Chronic Metabolic Toxæmia—Premature Senility.**—In the chapter dealing with non-toxic insanities I referred to mental symptoms the result of anæmia of the brain directly due to arterial changes. In the majority of instances these arterial changes are due to general constitutional disorders which produce or are a part of a condition of toxæmia producing changes identical with those described in the arterial system in chronic Bright's disease.

Bevan Lewis describes these cases as premature senility, chronic brain atrophy, or cerebral Bright's disease.

Pathologically there can be no doubt of the changes which occur in the minute arterioles of the body, and especially in those of the cerebral cortex, the change, according to Lewis, being a thickening of the muscular tunics of the small vessels.

The disease is never seen before middle life, and if there is such a disease as "climacteric insanity," then cerebral atrophy may be described as that disease, so frequently does it occur at or about the climacteric.

The onset is always gradual, and is associated with gastric derangement, indigestion, constipation, loss of appetite, especially in the morning. These symptoms may last for months. The appearance of the patient alters ; the skin assumes a dull pallor, partly referable to anæmia, but also in part to some structural or functional alteration, whereby the subcutaneous tissues become œdematous. The pulse is small and of very high tension, generally between 140 and 180 mm. Hg. There is no increase of the leucocytes, but there is deficiency in the quantity of red blood corpuscles and in the percentage of hæmoglobin. There is always a deficient excretion of urine and the solids of the urine.

Long before mental symptoms are present the patient may suffer from well-marked nervous symptoms. One of the earliest is attacks of palpitation, and these may be associated with severe headaches, vertigo, and a feeling of dryness or



prickling in the skin. The mental symptoms associated with these physical disorders are always those of depression with hallucinations and delusions.

It is extremely common to find that these patients have previously suffered from a mental attack, and that that attack was invariably one of depression. Chronic cerebral atrophy is the chronic form of metabolic poisoning; sometimes occurring as the sequel to an acute attack of acute melancholia, at other times as a more chronic process, while at still others it appears to be associated with long-continued indulgence in alcohol. Bevan Lewis regards the majority of his cases as due directly or indirectly to chronic alcoholism, but I have seen very typical cases in which the abuse of alcohol as a causative factor could be excluded. The abuse of alcohol is an associated symptom in a large proportion of the cases. In some the alcohol is taken to relieve the physical discomfort of the early gastric disorders; in others the alcohol undoubtedly produces metabolic disorder by interfering with the functions of the intestines, liver, and kidneys. Whatever may be the cause of the earlier physical symptoms, there is no doubt that by the time mental symptoms develop there is abundant proof that the body is suffering from an accumulation of waste products, and the symptoms, both mental and physical, only differ in degree from those of chronic melancholia.

J. M., female, aged 50, was admitted suffering from depression with suicidal tendencies.

*History.*—She is the mother of four children, who are alive and healthy. She has taken alcohol all her life, but apparently not to any excess. Up to a year ago she enjoyed good health, when she complained of headaches and giddiness. This went on to depression, and she had to be certified and removed to an asylum. The symptoms of this acute first attack were those of acute metabolic poisoning, and they lasted for one month, but she was not considered fit for discharge until she had been under care for two months.

The present attack commenced three weeks ago with gastric disorder and sleeplessness.

*State on Admission.*—She was a well-developed woman, but thin and obviously run down in health. Her pulse was 76 per minute, small, with an arterial tension of 150 mm. Hg. Only 10 oz.



urine were excreted for the first forty-eight hours, and only 160 grs. of urea for the same period; a trace of albumen was also present. Mentally she was confused and depressed. She suffered from hallucinations of hearing, and at times became frightened. She expressed the delusion that her children were dead, and that she had heard them murdered just outside the window of the ward. Her memory was markedly impaired. She was placed on a simple dietary of milk and milk puddings, and given large quantities of fluids between meals. Under this treatment she improved, but the improvement was not maintained. She was with difficulty persuaded at times to take sufficient food; this was partly due to gastric derangement and partly due to delusions of suspicion that the food was poisoned. The urinary excretion was always deficient, and she always ingested more albumen than the amount of urea excreted, yet she never gained weight. Six months after admission she steadily began to lose weight. Mentally she was more confused and took no interest in her surroundings. She became so weak that she had to be confined to bed, and finally died apparently of exhaustion.

Post-mortem, it was found that the stomach and duodenum presented all the appearances of chronic catarrh, and there was great thinning of the walls of the large intestine. The right kidney was contracted and fibrous. The brain was atrophied, with an increase of fibrous tissue in both the grey and white matter.

J. U., male, aged 48, was admitted suffering from delusions regarding his food.

*History.*—He had always been a steady and industrious man until a year ago, when he began to suffer from indigestion. He complained of nausea in the morning, and often had eructation after taking food. Latterly he has been depressed and peculiar in manner, finally refusing to take any food which he had not prepared by himself.

On admission he was thin, sallow, and anæmic, and looked at least sixty years of age. His skin was oedematous, and his eyes had a sunken look in consequence. He took food readily, but appeared to have little appetite. The excretion of urine was scanty, of low specific gravity, and rarely contained more than 200 grs. of urea for the twenty-four hours, notwithstanding the fact that he injected sufficient albumen to excrete 300 grs. of urea per day. His heart's action was slow, and the pulse firm and wiry. His leucocytosis was always below 10,000 per c.mm., with a polymorphonuclear percentage of 56.

Mentally he was confused and suspicious. He spoke very little, and was silent regarding his own case. He undoubtedly



suffered from hallucinations of hearing, but they did not, so far as could be judged, affect his conduct. His memory was impaired for both recent and remote events. He was listless and without initiative, and gave the impression of being slightly demented. Under treatment he improved somewhat, and the progress of the disease was apparently arrested ; but he remains, so far as his mental state is concerned, a case of mild dementia.

## CHAPTER VII

### TOXIC INSANITIES—*continued*

#### GROUP II.—INSANITIES IN WHICH THERE IS EVIDENCE OF BACTERIAL TOXÆMIA

##### EXCITED MELANCHOLIA

INSANITIES complicated or caused by bacterial toxins are much more serious conditions than insanities of purely exhaustive or metabolic origin. The invariable symptom common to the whole group of bacterial insanities is the presence of a hyperleucocytosis with in the early stages a high percentage of polymorphonuclear leucocytes, and wherever such a symptom can be demonstrated the case will almost certainly prove to be one of the diseases described under the present group of toxic insanities. The mental symptoms associated with this physical symptom are various and by no means diagnostic, but excitement, associated with mental confusion, depression, or elevation is very frequently present, and is by far the most characteristic form of mental derangement.

Another common group symptom is the fact that patients who suffer from these toxæmias are liable to relapse. So far as my observations go, they indicate that such patients very rarely make perfect recoveries. There are, long after all mental symptoms have vanished, persistent evidences of toxæmia to be demonstrated by examination of the blood; and although many such individual patients may never suffer from a second attack, or years may elapse before they do so,



there are unfortunately a large majority who relapse again and again.

In order to bring my clinical descriptions into line with the commonly accepted symptomatological classification of the present day, I describe the various diseases under the names at present in use.

**Excited Melancholia.**—Excited melancholia is at present always regarded as a type or variety of melancholia. Superficially there is some resemblance, but when one comes to examine the physical symptoms, there is no doubt that excited melancholia and acute melancholia are two separate and distinct diseases.

Excited melancholia is a disease prone to occur in adult life and during the decline of life. In women it is frequently seen during the menopause. Heredity is not, in my experience, a frequent predisposing cause, nor is the disease commonly caused by excesses in food or drink. Long-continued anxiety and worry, together with the natural failure of the vital powers as life declines, seem to be the chief predisposing factors. The exciting cause in every case is a condition of toxæmia almost certainly bacterial. The point of attack and the cause of attack are at present unknown, but it is probable that the point of attack is the intestine, and that some of the saphrophitic bacteria of the body become virulent and are the direct cause of the toxæmia, the toxine having a disposition to attack the nervous tissues. The history of a typical onset is as follows: The patient, after having stood much mental and physical strain, loses weight, becomes anæmic, dyspeptic, and sleepless. There is a lethargy of action both physical and mental which the patient notices, and for which medical advice is frequently sought. Gradually the patient becomes restless, unable to settle to any occupation, and unable to rest. The pulse is fast, the temperature is sometimes slightly elevated, especially in the evening, and as the disease develops the condition passes into acute excitement with depression. The patient is always thin and badly nourished. The appearance and expression of the face indicate misery, anxiety, and often fear. The tongue is furred; there is a disinclination for food or drink, and in severe cases artificial

CHART No. 1.

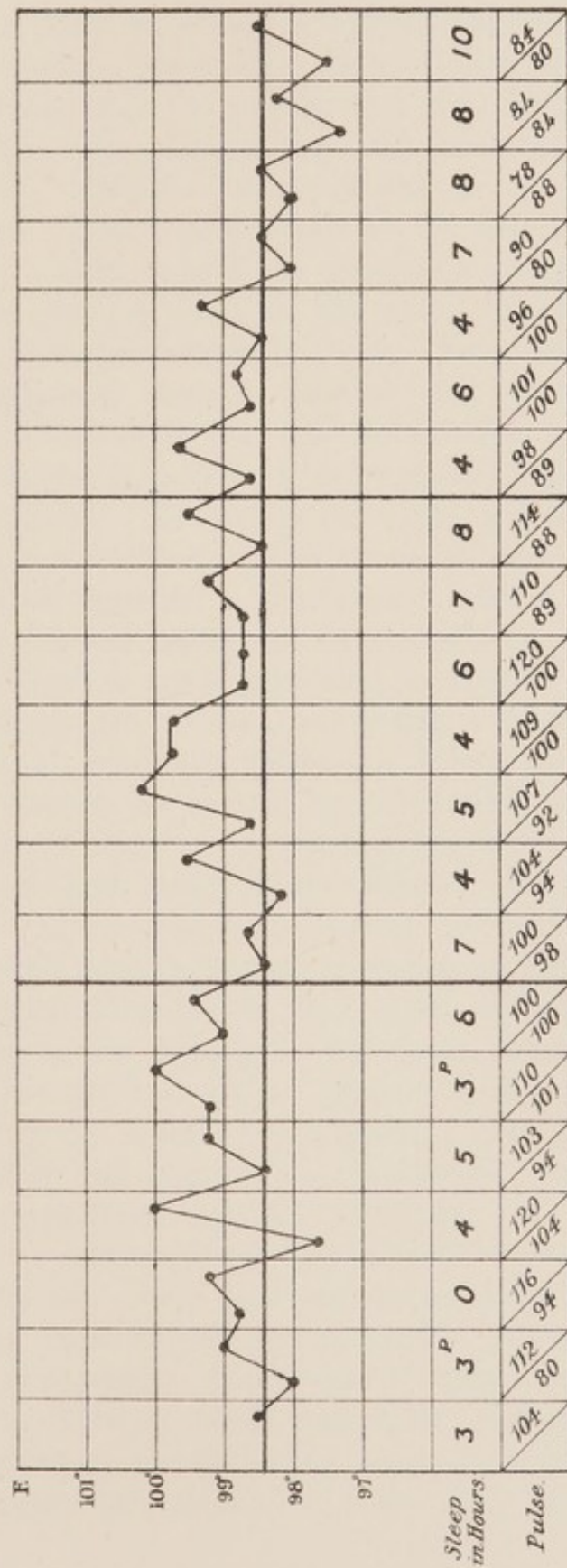


CHART showing the Temperature, Pulse, and Sleep in hours of a case of Excited Melancholia during the first three weeks after admission. P = paraldehyde, 5ii.





feeding may be necessary. Flatulence, with noisy eructation, is common. The bowels are confined. Anæmia is always present, and there is always a hyperleucocytosis, with a high polymorphonuclear percentage. Agglutinines, similar to those found in cases of acute mania, may also be present in the blood serum. The pulse is fast and weak, and the arterial tension is low. Many of these patients are prone to recurrent erysipelatous attacks, particularly of the face, and sometimes these attacks seem to take the place of a mental attack. In other cases papules form on the skin. These the patient picks, and so causes unsightly sores. The urine is passed in fair quantity, and there is no deficiency or excess of urea. Flushings of heat and cold and formication of the skin are often complained of, and there is frequently loss of sensibility to heat and pain. The pupils are equal, and react readily to light and accommodation. The senses of taste and smell are, however, often impaired. The organic reflexes of micturition and defæcation are rarely out of control, as in this disease mental confusion is not a prominent symptom. The skin and tendon reflexes are present and active. While there is no impairment of voluntary movement, irritability of the motor cortex is present, as shown by restlessness and slight in-co-ordination in the finer movements, especially of the muscles of the hands and face. These muscles also show fibrillary tremors. The outstanding mental symptoms are acute depression and misery, without confusion or loss of consciousness. If mental confusion does occur it is transient and corresponds to the height of the acute attack. Hallucinations of sight and hearing are uncommon in typical cases, nor are delusions a necessary symptom. Loss of self-control is evidenced by constant restlessness, noisy declamation, and sometimes suicidal attempts. All the patient's attention is introspective, but objective attention can be readily obtained; there is, however, no capacity for occupation. The memory is not markedly affected either for recent or past events. The power of speech and the comprehension of languages, either spoken or written, are not lost. Sleep is interfered with during acute attacks, and is often defective after the attack is over. The chief differences



between acute melancholia and excited melancholia are as follows :—

|                   | Acute Melancholia.   | Excited Melancholia.   |
|-------------------|--|--|
| Physical—         |  |  |
| (a) Alimentary .  | Marked disturbance.  | Less disturbance.  |
| (b) Hæmopoietic . | Moderate leucocytosis.   | Hyperleucocytosis.   |
| (c) Urinary .     | Excretion of urine and urea deficient.                               | No abnormality detected.   |
| Mental . . . .    | Mental confusion with vivid hallucinations which affect the conduct. | Little or no mental confusion. Hallucinations not necessarily present. |

Excited melancholia is a long-continued, intractable, exhausting disease. The patient lives in a state of constant restlessness, wandering aimlessly about, twisting or rubbing the hands, picking the face, hair, or clothes, often moaning or ejaculating in a rhythmical manner. The patients are generally thin and unhealthy-looking, and require constant attention, particularly at meals, to see that they take a sufficient quantity of food. The condition may last for years. The prognosis is bad. Partial recoveries and constant relapses are, however, very common.

*Treatment.*—During the acute stage rest in bed with simple dietary, not necessarily fluid, is essential. After the acute symptoms have subsided, a course of tonics, with fattening food and very mild exercise, does good. Sun baths or keeping the patient in bed in the open air all day often improve the physical condition. Some cases, especially elderly people, do well on small doses of Tinct. opii or nepenthe given three or four times a day, the dose being constantly diminished as the sedative effect is gained. If moderate doses of opium do not produce the desired effect in a few days, it is advisable to discontinue the drug, otherwise the opium habit may be established. In very acute cases sulphonal in small doses (10 grs. thrice daily) is a very useful sedative, and when 30 grs. of Potass. bromide is added to each dose the sedative effect is much enhanced. Unfortunately both sulphonal and the bromides tend to disorder the stomach and lower the powers of nutrition.



The following case illustrates an acute attack of excited melancholia which terminated fatally :—

A. A., a married woman, aged 59, was admitted suffering from restlessness and depression. Her illness was of three months' duration upon admission.

*History.*—No hereditary predisposition was admitted. The patient was a woman of steady habits until during the last two months, when she began to take alcohol. She had been a healthy woman and had never suffered from any nervous disorder. As the result of domestic worry the patient ran down in health and lost flesh. Three months ago she became restless, neglected her work, left her home and refused to return to her husband. On admission she was thin and looked very ill. She was, however, well developed. Her skin was sallow and dry. She appeared both frightened and depressed. Temperature 98° F. Her tongue was furred, her bowels obstinately confined, and she was fed with difficulty. Her leucocytosis was 10,000, with a polymorphonuclear percentage of 87. Her pulse was quick, 90 per minute, weak and irregular. Her skin was very dry and she bruised easily. There was a large quantity of albumen in the urine and some blood. There was considerable anæsthesia to pain and heat. She was constantly in movement, picking at the bed-clothes and muttering to herself. There was great depression but no loss of consciousness; she knew where she was and spoke rationally when questioned. At times she appeared to have hallucinations of sight and hearing, and sometimes she mistook the identity of those about her. Her attention was largely subjective, but could be easily attracted. Her memory was slightly impaired. She suffered from absolute loss of sleep. A week after admission she was much worse, and became acutely restless and excited. Her pulse was 120, weak and thready; her tongue was dry and furred, and her teeth and lips were covered with sordes. Her temperature in the evening was 101° F. An erysipelatous inflammation then developed over the left elbow-joint and a large bursal abscess formed a week later. The abscess was opened, but next day abscesses formed in the large joints of the left foot and hand. The next day the left knee-joint became inflamed and suppurated. All the abscesses were freely opened, 10 c.c. doses of polyvalent streptococcus serum were given by rectal injection at intervals of three hours. The temperature, which had risen to 103° F., fell to normal, but a week later she had a rigor in the evening and presented symptoms of fluid in the left pleural cavity, and her temperature again rose to 103° F. Diarrhœa now set in, and she died the following morning. At the post-mortem examination both pleural cavities were found filled with pus,



cultures from which yielded a pure growth of the staphylococcus aureus pyogenes. Throughout the attack she suffered from a hyperleucocytosis with a high polymorphonuclear percentage.

Case No. 2 is a more typical one, which terminated in partial recovery after an illness lasting two years:—

A. C., female, aged 39, single, by occupation a mill-worker, was admitted in January 1900 suffering from typical excited melancholia. Her illness had lasted two months upon admission.

*History.*—The patient was the youngest child of a family of eleven. No member of the family had suffered from nervous disease. The patient was a woman of industrious steady habits. She had never suffered from any previous nervous complaint. Two months ago the patient's mother died, and in addition to having had the strain of sick-nursing, with its attendant anxiety and want of sleep, there occurred a family quarrel after the mother's death, when the patient was accused by the rest of the family of having appropriated for her own use certain articles of value which the mother had been known to possess. This charge the patient felt very keenly. She became moody and sleepless, left her work, refused her food, and constantly wandered about the house, paying absolutely no attention to her personal appearance. She finally passed into a condition which necessitated removal from home.

*State on Admission.*—She was a woman of average height and development, but very thin and badly nourished. Her skin was drawn and parchment-like, and her appearance and expression indicated a condition of great mental anxiety. Temperature 99° F. Her tongue was thickly furred, she had little appetite, and complained of discomfort after taking food, and she suffered from constipation. No abnormality was detected in the thyroid, spleen, or lymphatic glands. She was anæmic, the red blood corpuscles numbering 3,000,000 per c.mm., and the percentage of hæmoglobin was 50. The leucocytes were 19,000 per c.mm., with a polymorphonuclear percentage of 80. Her heart's action was rapid, irregular, and weak. Pulse 99 per minute. No abnormalities were detected in the lungs. Her skin was dry, rather pigmented, and there were a few inflamed papules over the forehead, neck, back, and chest. The hair was dry, brittle, and untidy in appearance.

She excreted 40 oz. of urine during the first twenty-four hours after admission, and 400 grains of urea. No albumen or other abnormality was detected. Her menstruation was irregular. The sensibility to both heat and pain was impaired. Her pupils were dilated, but reacted readily to light and accommodation. Hearing,



taste, and smell, so far as they could be examined, showed no impairment of function. The skin and tendon reflexes were present and active. There was excessive muscular movement, the patient constantly wringing the hands and picking at the hair, face, and bed-clothes, while the movements were slightly inco-ordinate and the hands very tremulous. She was acutely depressed without being able to give any reason for her depression. She was perfectly clear in mind and conscious, answered questions readily, and recognised that she was ill. She suffered from neither hallucinations nor delusions. Her self-control was deficient in so far that she gave way to her depression, but she was not impulsive. Her attention was largely subjective, but could be easily attracted, and she observed all that was going on in the ward. Her articulation was weak but her speech coherent, and she understood what was said to her, and could read and write when asked to do so. She suffered from almost complete sleeplessness.

She was put to bed and placed on a simple dietary of milk, very weak tea, toast and butter, milk puddings and eggs. Her bowels were relieved by large enemata of normal saline solution, and a draught of 2 drachms of paraldehyde was given every third night if the patient was sleepless. In three weeks she had considerably improved and was got out of bed for a short period daily. At the end of two months she was taking very light exercise, but she tired easily. Three months after admission she was visited by her friends, who renewed their inquiries for the missing property which had belonged to the deceased mother, and there was a scene. That night the patient did not sleep. The following morning her pulse was 90 per minute. She was very restless and depressed, and the following night was also sleepless. Next morning her temperature was  $99.4^{\circ}$  F. and she was much worse mentally. Her leucocytosis was 30,000, with a polymorphonuclear percentage of 90. The blood, which had been examined at frequent intervals since admission, always showed a hyperleucocytosis, but the polymorphonuclear percentage had fallen to 60 after the first week of observation. This second attack lasted five months, and she improved most rapidly when the weather permitted her to sit out in bed in the fresh air and sunshine. Nutrition continued very poor, and neither after the first nor second attack had the patient gained more than 5 lbs. in weight. She constantly suffered from dyspepsia, with pain and eructation after food, and she never felt hungry. In November of 1900 she had another attack, the physical and mental symptoms of which were very acute for a fortnight, and she did not regain the lost ground until April of 1901. During the whole of this time she still had a hyperleucocytosis with a varying percentage of polymorphonuclear leucocytes. During the later part of 1901



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she slowly gained 10 lbs. in weight, and was finally discharged relieved in March 1902.

As the patient's serum gave a definite agglutinative reaction to a small coccus obtained from the blood of a case of acute mania, a polyvalent streptococcus serum was frequently given during acute attacks, but no benefit resulted from this treatment.

## CHAPTER VIII

### TOXIC INSANITIES—*continued*

#### MANIACAL EXCITEMENT WITH CONFUSION (ACUTE MANIA)

THE word Mania is probably the most elastic term in psychiatric nomenclature, and is undoubtedly used at the present day to describe widely different conditions of disease, such as the excitement of Folie Circulaire, Katatonia, and Hebephrenia. The word Mania practically describes one outstanding symptom—that of excitement; but, in practice, the word is also used to designate a distinct disease which is further subdivided into varieties such as acute mania, delirious mania, remittent mania, recurrent mania, adolescent mania. The more I see of the condition of acute toxæmia associated with maniacal excitement, the more I am convinced that the present subdivisions of the disease known as Mania are misleading. The physical symptoms alone point to the fact that the disease is the same whether it lasts for a fortnight or a lifetime, whether it recurs every month or six weeks, or never recurs at all, whether it occurs in youth or old age, whether the symptoms are so acute as to deserve the term delirious, or so slight as to be called sub-acute.

The duration, severity, or recurrence of the disease are merely phases of individual resistance to the toxæmia which causes it. These views are the result of work done during the last three years, and further research may again modify them, as I am convinced that our knowledge of maniacal states is incomplete and confused.



Regarded from the mental aspect, and to some degree separated by physical symptoms also, I believe there are two types of Mania. Firstly, a condition of excitement associated during the early stages with complete loss of consciousness, with hallucinations and illusions, with complete loss of the powers of attention and memory, with incoherence of speech and loss of comprehension of language spoken or written; while, on the physical side, there are evidences of very acute toxæmia. This disease will be described under the term *Acute Mania or Excitement with Confusion*.

The second type is a condition of excitement without confusion, but rather associated with a hyperacute consciousness, without hallucinations or delusions. The powers of attention are not lost, but wander loosely from subject to subject. The memory is often very acute. The speech, though rambling, disjointed, and inconsequent, is not, in itself, incoherent. The patient readily understands spoken or written language, and, although the writing may be fantastic in style and in composition, the power of writing is not lost.

Physically, the symptoms of toxæmia are much less severe. This type is described under *Folie Circulaire or Excitement without Confusion*.

Mania with confusion, if it lasts for any length of time, is liable to be mistaken for the excitement of folie circulaire, as in course of time consciousness returns with some capacity for attention and observation; and the disease, viewed from the mental side, is in many cases not dissimilar from the allied condition of excitement without confusion.

The term typhoid mania or delirious mania is, in my opinion, a misnomer, as I have seen all the toxic insanities, whether of metabolic or bacterial origin, terminate in typhoid or delirious mania, an almost invariably fatal complication.

Simple mania is also a misleading term, as it applies equally to the very early symptoms of the disease, which terminates in mania with complete loss of consciousness, and to a degree of the mania of the folie circulaire type.

**Acute Mania.**—The condition of excitement with confusion associated with evidences of bacterial toxæmia has certain common physical symptoms, whether the disease is





CHART No. 2.

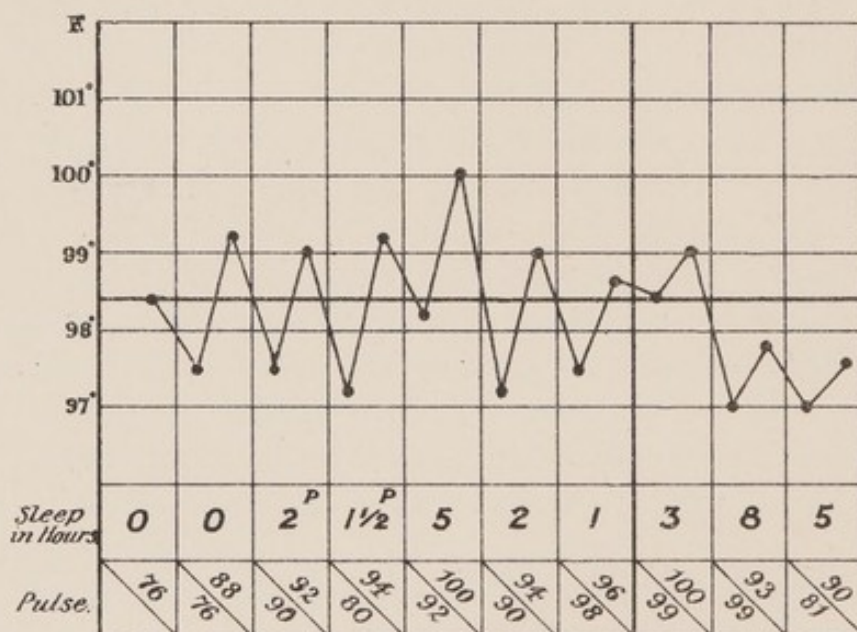


CHART showing the Temperature, Pulse, and Sleep in hours in a case of Acute Mania during the first 10 days after admission. P=paraldehyde, 3ii.

continuous, recurrent, or remittent, or whether the disease occurs in youth, adult life, or old age; and, for purposes of description, it is probably best first to describe the disease as it occurs typically in the adult where the symptoms are continuous, although it is by no means uncommon to find the same condition in adolescents.

The onset of the disease is, as a rule, gradual. The patient feels out of sorts, is generally sleepless, restless, and unable to attend to business or to concentrate the mind upon any work for any length of time. Unaccountable dislikes to near relatives or friends often occur. Headaches and neuralgias are complained of, but the physical symptom which is frequently present is dyspepsia with flatulence. The maniacal attack itself may be sudden or gradual in onset, or even, in a few cases, suppressed; the patient, instead of passing into acute mania, becoming irritable, moody, confused, and finally delusional. In the history of the patient hereditary predisposition to mental disease is found in at least 50 per cent of the cases. Secondly, it is not uncommon to find that the patient has exceeded in alcohol shortly before the onset of the illness. In such a case alcoholism is an early symptom, not a cause, of the disease. It is also common to find that the patient has passed recently through a period of intense mental worry or anxiety, often with bodily privation or unhealthy environments. In women, child-birth is a frequent cause. Some condition, in short, has occurred which has lowered the natural resistive powers of the patient.

The patients who suffer from this type of disease are by no means weaklings. In the great majority of typical cases the patients are well developed, though they may show want of condition. The face is drawn and pale, the eyes bright and staring, with widely dilated pupils. The temperature varies according to the stage of the disease; at the onset it is irregularly febrile; but after the first or second week the temperature shows a tendency to become subnormal, and thereafter throughout the attack, even after apparent recovery, the temperature runs at about one degree below the normal, and often the difference between the morning and evening temperatures may be so marked as to



give the impression that the patient is passing through a mild septicæmic attack. Another peculiarity of the temperature is that it may be paradoxical, *i.e.* higher in the morning than in the evening. The paradoxical temperature is often associated with a paradoxical pulse-rate. The paradoxical temperature is not, however, confined to patients suffering from acute excitement.

In the early days of the attack the alimentary tract is much disordered. The teeth and lips become covered with sordes and the tongue is furred and foul. There is little desire for food, but thirst is generally present. I examined the stomach contents in two cases of acute mania at the onset of the disease, and in both the digestive power was almost nil. At a later period of the disease, although still maniacal, I found the digestive power of the gastric juice of these same two patients very active. These observations are quite in keeping with what one observes in the appetite of these patients. At the onset of the disease it is with great difficulty that the nurse can induce her patient to take a sufficient quantity of milk, and injudicious feeding often produces vomiting. In a fortnight or three weeks the same patient will eat ravenously and digest almost anything.

The circulatory system is but little disturbed. The pulse at the onset may be rapid and a little irregular. It rarely exceeds 100 beats per minute. It is not nearly so fast as the pulse of acute melancholia, for instance. When a patient has suffered from a long attack of mania there is a tendency to heart weakness and failure.

The rate of breathing is slightly increased in bouts of excitement. Beyond a tendency to slight attacks of lobular pneumonia, which give rise to little local or general disturbance and are thus frequently missed, the respiratory system presents no peculiarities.

The skin is generally dry, but the palms of the hands and the soles of the feet may perspire freely. The hair is dry and brittle. Evanescent rashes and diffuse pustular eruptions are not uncommon.

The urine is scanty and high coloured at the onset of the attack, and there is an excessive output of the nitrogenous





The Chlorides ingested in the Daily Dietary and the Chlorides excreted in the Daily Excretion of Urine, in grammes.

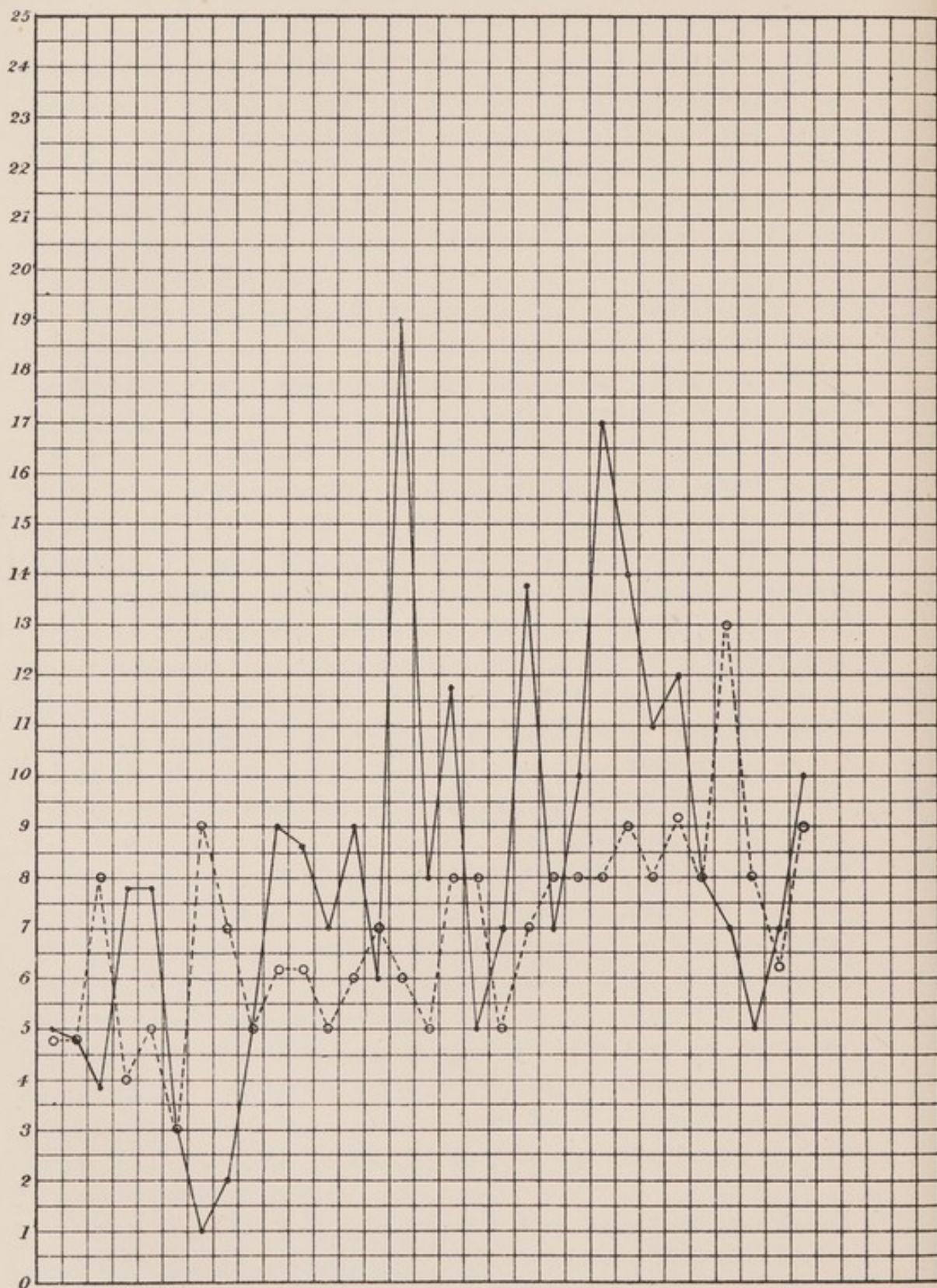


Chart showing the Chlorides ingested and excreted in a case of Acute Mania occurring in a female aged 36. The rise in the excretion of chlorides coincided with an improvement in the mental and physical symptoms.

Chlorides ingested are represented ○----○----○

Chlorides excreted are represented ●——●——●



waste products of the body. Later in the disease the secretion of urine is abundant.

A daily estimation of the chlorides shows that during the early stages the excretion is diminished. As improvement sets in the excretion of chlorides increases, and this increase is sometimes sudden, the rise being in proportion to the amount of sodium chloride ingested during the period of the acute symptoms. This sudden rise of chloride excretion, which resembles that which takes place in pneumonia at the crisis, is not, in my experience, necessarily followed by any sudden change for the better, but it is another link in the chain of evidence that acute mania is a toxic disease in which the chlorides are retained for some purpose of the bodily economy, just as they are in other known bacterial conditions.

Erotic conduct and speech are common in both men and women. The menstrual function is never suppressed in women as it is in some other forms of mental disease, but it may be irregular.

There is general loss of sensibility to heat and pain, but the sense of touch is acute. The pupils are widely dilated, but react to light and accommodation and also to emotional conditions—for instance, in a fit of anger the pupils may contract strongly. All the special senses except taste are acute,—according to Macpherson, hyperacute. The sense of taste is frequently disordered. Unless the patient is very ill the organic reflexes of micturition and defæcation are kept under will power. The skin reflexes are slightly increased. The tendon reflexes, so far as I have been able to examine them, are not increased.

There is no paralysis or weakness of the voluntary muscles. The inco-ordination of movement, most strikingly exhibited by the facial muscles, is of central origin.

The mental symptoms are—loss of self-control, characterised by mental excitement and motor restlessness, loss of consciousness and knowledge of position, loss of the power of attention, incoherence of speech, and sleeplessness.

Hallucinations of both sight and hearing are the rule often associated with delusions. Macpherson states that “hallucinations are rarely present, but may be simulated by



illusions of the hyperacute senses"; this applies to excitement of the folie circulaire type only.

The muscles, especially of the face and upper limbs, may show fine fibrillary tremors.

Probably the most interesting observations, as throwing light on the etiology of the disease, are those which have been made by my assistants and myself upon the leucocytosis, which is present in every case of "acute continuous mania" which we have examined. We are not by any means the first workers in this field. Macphail, Krypsiakiewicz, Kroumb-miller, and others have examined the leucocytes in mania and other mental conditions. I am bound to admit that our observations do not bear out all the results of these earlier workers. I can only explain this discrepancy by the fact that these observers were misled by making isolated observations. We, on the other hand, made continuous observations on each patient for weeks and months. Further, we have not confined our observations to one type of disease, but have examined every acute case admitted to the asylum, so that we have been able to compare our results in different diseases. We have also made frequent control examinations on healthy subjects. In recording these observations, I start on the hypothesis that a leucocytosis between 6000 and 10,000 per c.mm. of blood is what one might expect to find in health. The blood was taken from the lobe of the ear; as nearly as possible at the same hour every day just before the mid-day meal. The numerical counts were made with Thoma Zeiss' hæmocyto-meter, according to the method of Coles, and never less than 40 fields were counted at each examination. The differential counts were made from films stained by various methods, and never less than 200 leucocytes were counted to obtain the various percentages.

The following were the results obtained. If the case was observed early in the acute stage in every case there was a leucocytosis of from 18,000 to 20,000 per c.mm., and sometimes the leucocytosis was even higher. The percentage of the polymorphonuclear cells was never below 70 per cent. This state of affairs did not last for many days, and then it was noticed that the leucocytosis fell sometimes as low as



10,000 per c.mm., but never lower. More generally the leucocytosis was 14,000 or 15,000 per c.mm. Along with this change the polymorphonuclear cells fell to 60 per cent, or even lower, but they never in any case came near 70 per cent. There was a corresponding rise in lymphocytes. This stage in most of the cases lasted for weeks, in others for months. The leucocytosis varied a good deal, corresponding to exacerbations of the disease. A slight increase of excitement was accompanied by a rise in the leucocytosis to perhaps 17,000 or 18,000, but the percentage of polymorphonuclear cells rarely rose above 60 per cent. A further change noticed during this period was the occurrence of eosinophiles, which were sometimes so numerous as to constitute a mild eosinophilia of 3 to 5 per cent. An eosinophilia did not, however, occur in every case. Whenever distinct mental improvement set in the leucocytosis again rose, and along with this rise the percentage of polymorphonuclear cells rose, sometimes above 80 per cent, always above 70 per cent. As recovery became complete the percentage of polymorphonuclear cells gradually fell until it averaged somewhere between 60 and 70 per cent, but the most interesting thing of all was the fact that in many cases the leucocytosis persisted for weeks and months after complete recovery. It is highly probable that this leucocytosis persists indefinitely, and that it is a protective leucocytosis. If these observations are correct they prove that acute continuous mania is an acute infective disease, and that when recovery takes place a condition of immunity is established.

The persistent leucocytosis would indicate that although there is apparent recovery, the cause of the disease is still present in the body of the patient, and is only kept in check by the high leucocytosis maintaining an efficient immunity.

In the vast majority of patients who do not recover the leucocytosis tends to fall, and the percentage of polymorphonuclear leucocytes may fall below 40 or even 30. Quite recently, however, I have seen an adolescent male patient recover whose leucocytosis after the first few weeks of his illness remained persistently below 10,000 per c.mm. of



blood, and, on one occasion, the polymorphonuclear percentage was 29. In this case the illness was prolonged, the symptoms were subacute in character, and the patient presented some symptoms of brain damage. He was dull, lethargic, and sleepy, and he became fat and œdematous in appearance.

*Course of the Disease.*—The majority of patients who recover do so within six months of the onset of the symptoms. Sometimes recovery sets in suddenly, but, as a rule, this is an unfavourable termination. More generally the acute symptoms lessen in severity, sleep returns, and the patient begins to gain in weight, but often presents a bloated, puffy appearance very similar to that seen in patients who have recovered from prolonged septic conditions; there are transient periods of sanity most frequently to be noted in the morning, after a good night's sleep. These periods become more and more prolonged until, with occasional loss of self-control, the greater part of the twenty-four hours is passed in quiet and sanity. This finally passes into complete recovery, with, however, a persistent hyperleucocytosis.

On the other hand, the acute symptoms may abate, but, instead of recovering, the patient passes into a state of chronic restlessness. Consciousness returns and the patient is cognisant of all that passes, but there is no return of self-control and little capacity for occupation. Many patients in this condition become mischievous, destroying clothes and articles of furniture, occasionally passing into causeless paroxysms of anger or apparent joy, in which they may be impulsively violent or erotic. This state of chronic excitement may last for years until partial dementia arrests the more troublesome symptoms, and the patient may be capable of unskilled occupation under supervision.

A very small proportion of the cases die in the acute stage by passing into the condition known as delirious or typhoid mania; the symptoms of which are, delirious excitement, which passes into a stage of exhaustion, in which the patient lies helpless, tossing in bed and picking the bed-clothes. The lips and tongue become dry and cracked, the teeth are covered with sordes, and the urine and fæces are passed unconsciously. The patient rapidly emaciates, and



diarrhœa, with hypostatic congestion of the lungs, generally terminates life in the matter of a few days.

*Treatment.*—Rest in bed, efficient nursing both by night and day; a fluid diet, consisting of milk, strained broth, meat juice (if necessary artificially digested) given in small quantities every two or three hours, together with a liberal allowance of stimulants, is the best treatment for all cases in the early stages of the disease. Marked benefit follows the use of large normal saline enemata, and, where the patient is too ill to retain such enemata, subcutaneous injection of from one to two pints of normal saline solution produces equally good results. The continuous hot bath at 100° F. will produce quiet and sleep when hypnotics fail, but the effect is not lasting. Sleep must be obtained at least every third night by hypnotics if necessary, and paraldehyde in large doses, even so large a dose as 5 drachms, is, in my experience, by far the most certain hypnotic. All hypnotics, however, in the treatment of excitement are bad, as they tend to derange the stomach and still further lower the self-control of the patient; further, they stupefy the patient, and the habits in consequence become wet and dirty.

The following case illustrates the features of acute continuous excitement and toxæmia, and, further, is interesting as showing a recurrence of the disease which ended fatally in the typhoid state:—

J. L., aged 36, single woman, by occupation a laundress, was admitted in June 1900, suffering from acute excitement, which had lasted for six days upon admission.

*History.*—A brother had been insane. She was a woman of steady, industrious habits. Her attack commenced some four months ago with an attack of gastritis, from which she failed to make a satisfactory recovery. She suffered from pain after taking food, and attacks of vomiting often followed by depression. She became thin and sallow, left her situation, and could settle to no work. Finally, she became sleepless, and then symptoms of excitement developed.

*State on Admission.*—She was a big, strong, well-developed woman, but in very poor condition. Her expression was startled and unnatural, and her eyes staring. Her temperature was 99° F.

Her tongue was foul and coated, she refused solid foods, but drank fluids greedily. The thyroid was slightly enlarged, but no abnormality was detected in the spleen or lymphatic glands. The



leucocytosis was 20,000 per c.mm. of blood, with a polymorphonuclear percentage of 81. The heart's action was irregular, the pulse 80, and the arterial tension 140 mm. Hg. The skin was dry on admission, but subsequently she perspired very freely. The hair was harsh and semi-erect. She excreted 40 oz. of urine in twenty-four hours, which was high-coloured, with a dense deposit of urates. The excretion of urea was 550 grs. No other abnormality was detected. There was no amenorrhœa. The patient appeared to be oblivious to pain or heat. The pupils were widely dilated and reacted to light. Hearing, taste, and smell could not be examined.

She was clean in her habits; both skin and tendon reflexes were present. Voluntary movements were excessive and quite incoordinate.

Mentally she was excited, confused, and oblivious to her surroundings. Hallucinations both of sight and of hearing were present, and she mistook the identity of those about her. Self-control was abolished, and with it the power of attention. Speech was absolutely incoherent; she could not understand what was said to her, and was apparently unable to write. The function of sleep was abolished.

For the first three weeks she was confined to bed, and fed largely on milk and potash water. The excitement was partially controlled by warm baths, which were generally followed by periods of natural sleep, and occasionally a 2-drachm dose of paraldehyde was given at night. It was always noticed, however, that on the day following the exhibition of paraldehyde the patient was irritable, and more maniacal. At the end of the three weeks the patient was bromidised, and for a week all the mental symptoms were masked, and she was kept dry and clean with difficulty. As soon as the effect of the bromide passed off the condition of mania returned. From this time until October 1901 she was in a condition of chronic excitement, noisy, impulsive, mischievous, destructive, and sleeping very irregularly. Towards the end of October she had lucid intervals, generally in the early morning; sleep returned, and the appetite, which had been excellent throughout, with the exception of the first three weeks of her illness, became enormous, and the patient rapidly laid on weight. During November the periods of sanity became more prolonged and more frequent, until by the end of the month recovery was apparently complete. Examinations of the blood, however, still showed a hyperleucocytosis with a polymorphonuclear percentage between 60 and 70. The temperature also was subnormal, being often below 97° F. in the morning. By the end of December 1901 she was discharged, and she returned to work.

She was readmitted in July 1904, suffering from excitement which had lasted fourteen days upon admission. No history of this



attack could be obtained beyond the facts that the illness came on rather suddenly, with a complete change of appearance and character, and that sleep was lost. She then became confused, obstinate, and dirty in her habits.

*State on Admission.*—The patient was gaunt and thin, having lost 2 stone in weight since her discharge in 1901. Her appearance was one of exhaustion, and she lay in bed tossing restlessly about and muttering incoherently. Temperature 98·8° F.

Her tongue was dry and furred, her lips and teeth coated with sordes; there was no desire for food or drink, and the bowels were obstinately constipated. The leucocytosis was 11,000, with a polymorphonuclear percentage of 75. Her pulse was 90 and weak. The skin was dry and harsh. Pus was present in the urine, which was withdrawn by catheter as the patient passed urine and fæces under her. She was menstruating upon admission. Examination of the nervous system was impossible, but it was noted that she was anæsthetic to pain, that her pupils were equal, slightly dilated, and reacted very sluggishly to light, and that she had no control over the reflexes of micturition or defæcation.

Mentally she was quite unconscious, and lay in a state of muttering restless delirium.

She was fed frequently with peptonised milk, beef-tea thickened with oat flour, and 2 oz. of whisky every four hours. Saline enemata could not be retained, so normal saline solution was given subcutaneously in the loose tissue of the flanks. No treatment seemed to benefit the condition, the exhaustion became more pronounced, the leucocytosis rose to 30,000, with a polymorphonuclear percentage of over 80, and the pulse became fast, irregular, and weak. On the ninth day after admission the temperature rose to 104, diarrhoea set in, and the patient died apparently of pure exhaustion. A thorough post-mortem examination was made, but no cause for death could be ascertained. Bacterial cultures made from all the organs were sterile with exception of those from the bone-marrow and lungs, which gave a pure growth of a coccus which resembled in staining reaction and growth characteristics the *Staphylococcus aureus*.

Dr. Ford Robertson, of the Scottish Asylums Laboratory, kindly made a microscopic examination of the organs, and his report was as follows:—

“The stomach stained with Hæmotoxylin and Eosine showed some slight chronic catarrhal proliferation of the epithelium of the mucosa. In the ileum (Hæmotoxylin and Eosine) the mucosa was narrowed, the villi were diminished in number, fibroid



and atrophied. There was great increase of interglandular fibrous tissue, and the bases of many of the glands were in consequence widely separated from each other, while the submucosa showed fibrous thickening. In his opinion the condition was one of severe chronic atrophic catarrh. There were marked fatty changes in the epithelial cells of the liver."

*Brain.*—Parietal cortex (Methyl-Violet).

"The blood-vessels were everywhere distended with blood. The nerve-cells throughout were affected by marked chromatolysis, but very many of them showed a more advanced lesion consisting in disintegration of the nucleus. Many of the axis-cylinders of the medullated nerve-fibres presented a marked affinity for the stain, and were at the same time swollen and granular, evidently a state of acute primary degeneration."

This condition of excitement with symptoms of toxæmia in the adult, instead of being continuous, may be irregularly remittent or intermittent, or possibly a better description is recurrent. A large number of the continuous cases show remitting tendencies, the patient every now and then improving somewhat and again relapsing. The recurrent cases, on the other hand, have distinct periods of sanity between attacks, and the attacks themselves are of limited duration. The difference between the continuous and the recurrent cases is that in the continuous cases immunity is slowly established, and, once established, may possibly last for years or a lifetime; while in the recurrent cases immunity is more rapidly established but is of short duration. This difference of reaction may be explained by variations in the resistive power of the patients, or on the ground that different toxins are responsible for periods of prolonged or short immunity.

The following case is an example of the disease in a recurrent form:—

H. R., female, aged 46, a single woman, was admitted in April 1902, suffering from hallucinations of hearing.

*History.*—So far as could be ascertained there was no insane heredity, but the patient looked nervous; and a sister was also unduly excitable. She was a woman of good habits, industrious and sober.

The illness began about a year ago, when she changed in character, became obstinate, and was suspicious of her friends. Later, hallucinations of hearing became a prominent symptom, and



she refused her food, saying it was poisoned. For the last three or four days she had been impulsive and sleepless.

*State on Admission.*—She was a sparely-built, very nervous-looking woman, and badly nourished. She looked confused, and for the most part lay in bed, refusing to answer questions; but every now and then, evidently in response to hallucinations of hearing, she became impulsive and struggled violently. Temperature 98° F.

Her tongue was furred and she refused food and required to be hand-fed.

The thyroid was slightly enlarged. The leucocytosis was 15,000 per c.mm., with a polymorphonuclear percentage of 73. The pulse was 98 and rather irregular, and the skin was abnormally dry. No abnormality could be detected in the urinary or nervous systems. Mentally, she was confused with outbursts of excitement which were evidently caused by hallucinations of hearing. Her self-control was deficient and she was impulsive in her movements, suddenly jumping out of bed and running to the windows or door. Her attention could not be attracted and she did not appear to understand what was said to her. For the first two nights after admission she did not sleep.

She was put to bed and fed on fluids, and in three weeks was apparently quite well, but she still had a hyperleucocytosis. The next attack commenced in June, when she began to worry her fellow-patients regarding the state of their souls, and she lectured them unceasingly on religious matters. A week later she was maniacal, suffering from furred tongue, loss of appetite, constipation, and her pulse was over 100 per minute. In this attack, which lasted for three weeks, she was much more demonstrative and noisy. She remained fairly well but peculiar until September, when she took to her bed and refused to get up to take her food; was irritable and obstinate. Her pulse was fast and her leucocytosis over 20,000, and she suffered from sleeplessness. This attack only lasted for one week and was followed by apparent recovery. During December she was depressed for ten days, but the attack passed off without excitement. During 1903 she had four typical attacks of acute excitement—all commencing with religious emotionalism and much talking. The blood was examined daily from 9th October until 26th December, during which period she had two attacks. It was noticed that just prior to an attack the leucocytosis fell, and the polymorphonuclear percentage often fell below 50. As the excitement developed the leucocytosis rose, sometimes above 30,000; this, again, was followed by a fall, to be succeeded by a rise of both leucocytosis and polymorphonuclear percentage as recovery set in. Early in December another attack threatened, when the patient developed facial erysipelas, with a hyperleucocytosis of 28,000 and



a polymorphonuclear percentage of 89. The temperature was febrile for four days. At the end of that time she was again quite clear mentally, and remained so well that she was discharged early in 1904.

Throughout the various attacks the temperature never rose above normal, but there was a distinct variation of temperature during and also between attacks. As the illness came on the temperature rose from subnormal to normal, and remained about 98° F. during the period of excitement; a sudden drop in temperature again to subnormal always preceded recovery.

Cases of recurrent mania might be described as conditions of continuous toxæmia with recurrent attacks of mental excitement with confusion.

The same symptoms as those already described as occurring in the adult occur during adolescence, and are at present described as one of the varieties of adolescent insanity. Regarded from either the physical or mental standpoint, I fail to see any difference in the disease, whether it occurs during adult life or adolescence. In the adult the excitement tends to be of the continuous type and recovery takes place more slowly. In adolescent patients the excitement is as frequently recurrent as continuous and recovery is more rapid.

In the recurrent form of the disease in adolescent patients the symptoms are as follows:—Each maniacal attack is of short duration—a few days to two or three weeks at the very outside—and between attacks the patient is apparently quite well. The attacks invariably set in with gastric disturbance, the pulse becomes rapid, the arterial tension rises; the temperature may rise to 99° F., but rarely goes above 100° F. Sleeplessness is a constant symptom. Self-control is lost rather suddenly as a rule, and the patient becomes acutely maniacal. The mania is of a type which might be termed delirious, as little impression is left on the patient's mind after the attack is past as to what has happened. The pupils, as a rule, are widely dilated, the tendon and skin reflexes are exaggerated, and the skeletal muscles present jerking movements and fine fibrillary tremors. As the attack wears off, the patient shows signs of exhaustion. The temperature falls to subnormal, and occasionally is paradoxical. The pulse-rate falls, sleep



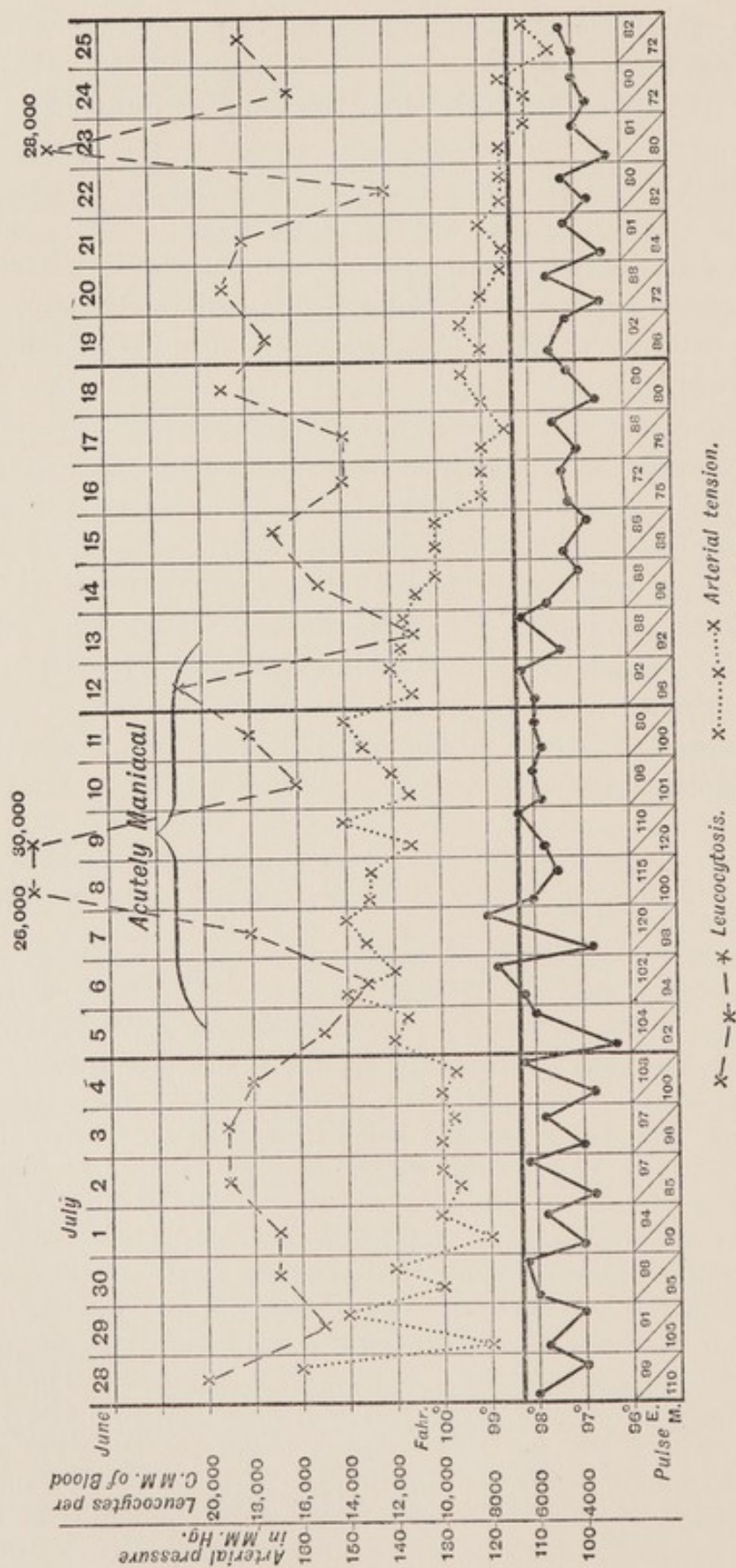
returns, the patient takes food greedily, and in a few days is outwardly recovered. In the periods between the attacks the patient is apparently healthy, the only symptoms being a persistently high leucocytosis and a rather low temperature, which every now and then becomes paradoxical. The attacks come on irregularly; in women sometimes at the menstrual periods, but not necessarily so. In this form of disease the menstruation in women may be irregular, but rarely suppressed, as it is in other types of insanity occurring during adolescence. A recovering patient gains weight; the attacks come on at longer intervals, and are shorter and less severe. If recovery does not set in, each attack seems to leave some damage behind, until finally, even in the intervals between attacks, the patient is obviously insane. Even when dementia sets in there are recurrent periods of excitement with intervals of quiet. The patients were all well developed and no satisfactory exciting cause was ever detected. Heredity undoubtedly is the predisposing cause.

The following case is very typical of recurrent excitement in an adolescent:—

L. E., female, aged 20, was admitted in a state of acute excitement on 20th June 1900. She had been maniacal for one day upon admission.

*History.*—There was predisposition to mental disease, a sister being insane. She was a girl of good habits, but somewhat obstinate, selfish, and wilful. Her home was a comfortable one, and no cause could be suggested for the illness, which was said to have come on suddenly. The patient herself, however, when recovered, stated that she had felt ill for months and had contemplated suicide. She was a well-developed, fairly well-nourished girl. The mental symptoms were very acute excitement with confusion and absolute loss of knowledge of position. No observations could be made beyond a blood examination which gave a hyperleucocytosis of 30,000, and the temperature was 99° F. Rest in bed, a liberal fluid dietary with a 2-drachm dose of paraldehyde to induce sleep, was the treatment adopted. Three days after admission all the symptoms had subsided, and the patient was conscious, but her mind was a blank for the days during which the mania lasted. Following the period of excitement there was a period of exhaustion with mild confusion which lasted for another week. When this passed off the patient was to outward





appearance recovered, but the leucocytosis never fell below 15,000 per c.mm. during the period which preceded the next attack, which came on early in July. The first symptom noticed was that the patient became flushed and that the conjunctivæ were injected; the pulse was over 100 and of high tension, and she felt disinclined to take food. She was put to bed and placed on fluid diet, but after a sleepless night the patient again passed into acute excitement with a temperature of 99° F. Just prior to the onset of the attack, the leucocytosis fell to 13,000 per c.mm.—the lowest count recorded since admission—but rose again to 30,000 during the attack. This attack lasted for seven days, and at its termination the temperature fell to subnormal, and the patient menstruated two days later. Then followed five weeks of apparent recovery, with a subnormal temperature and persistent hyperleucocytosis, when attack number three commenced with the usual symptoms of sleeplessness, quick pulse, flushing of the face, and injection of the conjunctivæ. The patient was again put to bed, placed on fluid diet, and 10 c.c. polyvalent antistreptococcus serum was given by the mouth. The effect of the serum was to reduce the pulse by ten beats per minute and the temperature 1° F., and the maniacal attack was postponed for four days. This attack only lasted two days, and was again followed by menstruation. Thirteen days later another attack threatened. The patient was placed in bed and 10 c.c. doses of polyvalent antistreptococcus serum were given by the mouth for three days, and there was no maniacal attack and she again menstruated. The following week another attack threatened, and was treated by serum and passed off harmlessly. During the succeeding eight weeks three attacks threatened and were treated by serum; none of them developed, and, after that, the patient made an apparently perfect recovery. The only other treatment adopted in this case was cold baths and tonics in the intervals between attacks. Two years after discharge this patient's blood was examined on several occasions, and the leucocytosis was never found to be below 17,000 per c.mm.

The accompanying chart shows the temperature, pulse, leucocytosis, and arterial tension in a period of four weeks during which she had one maniacal attack.

Two male adolescents presented identically the same sequence of physical and mental symptoms, and both made excellent recoveries, but neither of them was benefited by the use of polyvalent antistreptococcus serum.

The following case is a typical example of the type of continuous excitement with toxæmia met with in adolescence, and is interesting because of the history of onset:—



L. S., female, aged 25, by occupation a seamstress, was admitted 10th March 1900, suffering from mental excitement of seven days' duration.

*History.*—Hereditary predisposition to mental disease was denied by the parents, who both looked strong and healthy. She was an intelligent, well-educated, and industrious girl, whose occupation was, however, sedentary; but she had never suffered from any illness of note until the attack developed. Some three months ago she suffered greatly from toothache and had several alveolar abscesses. She became thin, dyspeptic, and sleepless, and maniacal symptoms developed suddenly.

On admission she was very pale and anæmic, of fair height and development, but very thin. Her expression was strained and unnatural, and the temperature  $98.6^{\circ}$  F. Her tongue was dry and furred, she had not one sound tooth—many being mere septic stumps—and she refused all food. The leucocytosis was 18,000, with a polymorphonuclear percentage of 74. The pulse was 110 and the arterial tension 125 mm. Hg. The skin was greasy and sallow. During the first twenty-four hours after admission she passed 17 oz. of urine, in which there was a heavy deposit of mixed urates but no other abnormality. The urea for the twenty-four hours was 350 grs.

The sensibility to pain was much diminished all over the body. The pupils were widely dilated, but reacted to light. There was no impairment of the organic reflexes of micturition and defæcation. Voluntary movement was excessive and inco-ordinate.

Mentally she was excited, confused, and oblivious to her surroundings. She had hallucinations of both sight and hearing, to judge by her actions. She was absolutely incoherent in speech, and could not be induced to read or write.

During the first week after admission her condition grew worse; she was frequently wet and dirty, tore her bedding, and spat at any one who came near her. She required to be hand-fed with fluid food. Sleep was obtained every third night by 2-drachm doses of paraldehyde, as the continuous warm bath produced no beneficial effect. Two weeks after admission she was more manageable, and large saline enemata ( $2\frac{1}{2}$  pints) were given every second day. As the result of this treatment she began to sleep better, but the excitement and destructive habits continued for another month. Early in May, all the decayed teeth were extracted. Her pulse at this date was rarely below 90 per minute, and at irregular intervals the temperature was febrile. The leucocytosis never fell below 16,000, nor the polymorphonuclear percentage below 70. By June she was sleeping well and rapidly gaining weight, but she was still restless, incoherent, and confused.



During July and August she was more confused than excited, and still gaining weight. Her expression was dull, heavy, and unintelligent. During September and October she was able to work, but, if conversed with for any length of time, she became incoherent and confused. By the end of November, however, she appeared to be recovered and went home early in December, having gained 3 stone in weight since her admission in March. After recovery, her mind was a complete blank from prior to her admission up to September, when she was first able to occupy herself. I have little doubt that in this case the carious teeth were the starting-point of the toxæmia which caused the condition of mental excitement.

**Puerperal Mania.**—As a sequel to the puerperal state one meets with every variety of mental disease—depression, excitement, katatonia, delusional and exhausted conditions. All the cases suffering from excitement which I have been able to examine presented very marked symptoms of toxæmia, and the disease did not differ in any way from acute continuous mania with confusion. In some of these cases there can be no doubt that the source of the toxæmia is a septic uterine cavity; but in others there is no evidence that this is so; further, many of these patients upon recovery present the symptoms of hyperleucocytosis, noticed in cases of recovery after acute mania not complicated by the puerperal state, and these patients are liable to relapse. The outstanding fact, however, in these cases of excitement following the puerperal state is the acuteness of the symptoms both mental and physical. On the mental side puerperal patients are very apt to be impulsive and suicidal, and on the physical side, symptoms of exhaustion set in with great rapidity.

The treatment consists in feeding frequently with small quantities of fluid nourishment (if necessary, artificially digested), and the free use of alcoholic stimulants. If there is uterine tenderness or fœtid or suppressed lochia, vaginal douches of some mild hot antiseptic should be given at least twice daily. According to Clouston the prognosis in puerperal mania is good—over 70 per cent recovering—and he also states that the tendency to relapse is much less than in other forms of mental disease. So far as my limited observations go, I am inclined to the belief that recovery from excitement with



toxæmia complicating the puerperal state is of two types, one complete and the other incomplete. Those in which the patient makes a complete recovery, without any evidences of permanent toxæmia, are probably those in whom the uterus was the seat of septic processes: when the source of toxæmia is removed there is no tendency to relapse. On the other hand, the patients who present persistent hyperleucocytosis after recovery must have some other source of toxæmia akin to that which causes excitement with toxæmia of non-puerperal origin, and these patients are liable to relapse.

In conclusion, I see no necessity for the term Puerperal Insanity, as no one type of symptoms, either mental or physical, follows the puerperal state when the patient becomes insane. According to Clouston, insanity complicating the puerperium forms 5 per cent of all the cases of insanity among women, and this is not to be wondered at when one considers the exhaustion and pain of labour, the loss of blood, and the liability to septic infection; but, beyond the septic infection, all the other concomitants of labour are merely predisposing causes, which act by producing exhaustion and lowering the resistive powers of the patient.

## CHAPTER IX

### TOXIC INSANITIES—*continued*

#### EXCITEMENT WITHOUT CONFUSION (FOLIE CIRCULAIRE)

THE type of Mania in which consciousness is not lost but only impaired, and in which the senses are frequently hyperacute, is almost invariably associated with the disease described as Folie Circulaire, in which the excitement may be either preceded or followed by a state of pure depression. Kraepelin describes this great class of diseases under the term of manic-depressive insanity. It is certainly a fact that symptoms of depression and even exhaustive stupor are common in patients suffering from the type of mania which I have already described, but the excitement and depression of the type now about to be described differ in both mental and physical symptoms from those of excitement with confusion. On the other hand, both types have certain symptoms in common which will be discussed later. Folie Circulaire as a distinct disease was first described by the French writers Falret and Baillarger, and is now included under periodic or recurrent insanities.

The excitement of the folie circulaire type may be prolonged or of very short duration. It may recur at frequent intervals, or the periods between attacks may extend over several years; but whatever its duration, the physical and mental symptoms are the same, and are liable to be complicated by attacks of depression. This is, however, a rule with many exceptions; for in cases of undoubted folie circulaire I have seen attacks of excitement which were not



followed by depression, and attacks of depression which were not followed by excitement: in other words, a stage of the disease may be suppressed or so transient as to escape observation.

Excitement of the "folie circulaire" type is a disease which commences in adolescence or early adult life, and rarely originates during the decline of life.

Hereditary predisposition to insanity is a predisposing cause in over 50 per cent of the cases affected, while moral or physical causes do not appear to play an important rôle in the causation of the disease. Whatever the history of the onset may be, the condition of the patient at the onset of the symptoms does not suggest physical break-down. A common history is that the patient has changed in character and disposition—is sometimes depressed or elevated for short periods. Finally, the elevated periods become more pronounced, and characterised by excitement and restlessness, and this passes into a state of continuous excitement, generally with complete loss of sleep. On examination the patient is usually stout and well nourished, and not necessarily neurotic in appearance. The face is flushed, the eye bright, the expression unnatural but not unintelligent. The temperature is as a rule elevated. In a case recently admitted the temperature was 101° F.

In a recent acute case the alimentary tract may be disordered, and there may be no desire for food or drink; but, on the other hand, especially if it is not the first attack, the appetite may be gross and perverted, the patient having a craving for stimulants and condiments.

The leucocytosis in the early stages is not so high as in excitement with confusion; but the percentage of polymorphonuclear leucocytes in a first attack may be well above 70. Where there are recurrent attacks the leucocytosis at first falls, and the percentage of polymorphonuclear leucocytes also falls, just prior to the onset of the excitement. As the excitement increases the leucocytosis gradually rises, culminates at the height of the excitement, and then gradually falls to normal. The characteristic of the leucocytosis during the acute stage of a first attack, however, is its irregularity and its tendency to fall as the excitement subsides.

CHART No. 3.

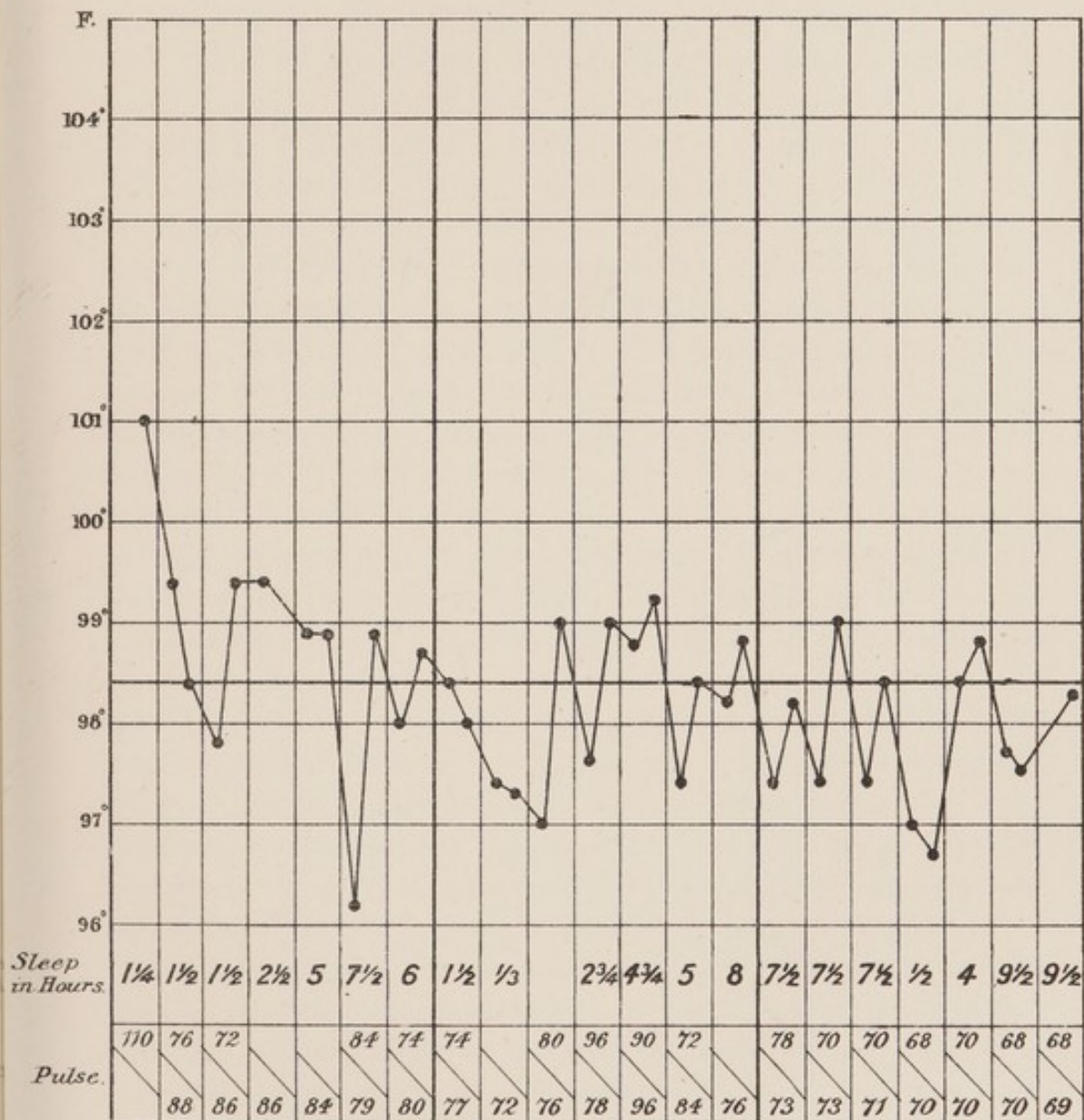


CHART showing the Temperature, Pulse-Rate, and Sleep in Hours in the excited stage of Folie Circulaire.





The pulse is not unduly fast, generally between 70 and 80 per minute, and the arterial tension is low.

The skin is moist, greasy, and often offensive. The urine is concentrated, of high specific gravity, with a heavy deposit of urates, and a trace of albumen or sugar is sometimes present. The chlorides are excreted irregularly, but there is never any marked failure of excretion. Indoxyl is frequently present, especially if the patient is constipated. As a rule the patient is too excited to notice or express subjective sensations if they are present, but the sensibility to touch, heat, and pain is acute. All the special senses are hyperacute. The pupils are dilated and react to light and accommodation. The organic reflexes of micturition and defæcation are under control, but the patient may be mischievously dirty in habits, especially if not kept under continuous observation. The skin and tendon reflexes are active, and the movements of the voluntary muscles excessive and slightly inco-ordinate. The mental state is one of pure excitement or elevation without confusion, hallucinations, or delusions; although, as Macpherson has pointed out, the hyperacute state of the special senses may simulate hallucinations. Loss of self-control is shown by extravagant but purposeful movement, noise, and mischievous conduct, rather than by impulsiveness. The moral sense and sense of decency are deficient, the speech and conduct being commonly lewd, coarse, and mischievous. In a patient who has recurrent attacks the faculty for remembering and mentioning scandal and unpleasant facts about those with whom he is brought in contact is a marked characteristic of the mental state. The attention is attracted by every passing word, sound, or movement, and quickly diverted by fresh stimuli. In acute first attacks the memory for recent and past events may be in abeyance, but in a recurrent case the memory is often phenomenal, such patients repeating with ease long passages of poetry or prose. The speech, though perhaps inconsequent and irrelevant, is not incoherent, and the power of writing and reading is not lost, though the writing is often fantastic and insane in character. Sleep is deficient throughout the attack, and often abolished during the height of the excitement.

The course of the disease is the same whether the attack



lasts a year or a week. The patient lives in a state of restless mental and physical activity, easily pleased and as easily annoyed. Preoccupied with the fancy of the moment, quarrelsome, noisy, vindictive, mischievous, or amorous as the mood dictates; and not too scrupulous in action, personal cleanliness, or in his dealings with those who are brought in contact with him. The restlessness gradually lessens, sleep returns, and the physical condition, which is lowered by the excitement, begins to improve, and this passes into apparent sanity. The treatment in such cases is unsatisfactory. If it is a first attack, prolonged rest in bed, if possible, lessens the acute symptoms. If the appetite is deficient in the early stages, the food is best given in small quantities at frequent intervals, but where the appetite is good a full general dietary is necessary, as the patient loses weight rapidly if the symptoms are acute. Where the excitement is associated with much noise and sleeplessness, small doses of sulphonal (10 to 15 grs.) given thrice daily tend to lessen it, and do not appear to interfere with the digestion. Otherwise treatment must be general, and consists in exercise in the open air, with, if possible, some occupation, and attention to the physical health. During the period of acute excitement, these patients seem to be oblivious to heat, cold, and much personal discomfort.

The period of depression which may follow or precede the period of excitement is characterised by a complete change in the physical state of the patient. The tongue becomes furred and flabby, the appetite capricious, and the bowels constipated. The leucocytosis is often high, but there is no marked rise in the polymorphonuclear percentage. The heart's action is weak, the pulse shabby and slow, but the arterial tension tends to rise. The extremities are cold, sometimes cyanosed, and in cases which tend to stupor may become oedematous. The skin is still greasy, and the palms of the hands and soles of the feet are sodden. No change occurs in the urine so far as I have been able to observe, but urine is evacuated at long intervals. The patient often complains of vague pains and physical discomfort. There is no loss of sensibility to heat, touch, or pain, but the senses are no longer hyperacute—rather the reverse. The skin and tendon



reflexes may be exaggerated or dulled, and there is no desire for unnecessary movement of any sort. The mental state is one of pure depression, without confusion, hallucinations, or delusions; although I have seen several cases who became hypochondriacal and complained of imaginary diseases so persistently as almost to be classified as delusional. The restlessness, noise, and rapid alternation of mood in the excited stage is changed into apathy and inertia sometimes so complete as to simulate dementia; and the patient may be sleepless. The course of the depression in prolonged cases is not marked by any sudden change. The patient often becomes fat and flabby, and the return to normal health or a subsequent attack of excitement is gradual.

The treatment for this stage is, I am convinced, rest in bed; and the washing out of the large intestine with normal saline enemata, twice a week, seems to give mental and physical relief, particularly in the way of producing sleep.

A. A., female, aged 47, was admitted in April 1902 suffering from excitement, which was of seven days' duration upon admission.

*History.*—A brother and sister of the patient had suffered from insanity. She was always a woman of good habits, and had lived a healthy country life up to one month ago, when she contracted influenza. After the influenza she became restless and sleepless, refused her food, and changed in her appearance and manner. About a week ago she became excited and elevated, and gave as her reason for refusing food that the food was poisoned. On admission she was a big, strong, well-nourished woman. Her face was flushed, her eyes widely opened, and her expression unnatural. Temperature  $98.6^{\circ}$  F. Her tongue was furred, she refused food, and the bowels were costive. The leucocytosis was 16,000 per c.mm., and the polymorphonuclear percentage 84. The heart's action was regular, the pulse 84, and the arterial tension 140 mm. Hg. The skin was moist and greasy, and the smell from the perspiration very offensive. The urine was high coloured, and contained a heavy deposit of urates on cooling. Sensibility to touch, heat, and pain was acute. The pupils were dilated, but reacted to light and accommodation. The senses of hearing, taste, and smell could not be tested.

There was no loss of control of the organic reflexes of micturition or defæcation. The skin and tendon reflexes were active. There was no paralysis and no inco-ordination. Mentally the symptoms were those of pure excitement without confusion and



without hallucinations. Her attention was attracted by any chance sound, word, or action, and she was noisy, erotic, and shameless in speech and conduct. She understood perfectly all that was said to her, but her memory could not be tested. She continued in a state of excitement during May and the early part of June. The temperature was irregularly febrile, there was often a hyperleucocytosis with a high polymorphonuclear percentage, and she slept very badly. She was fed largely on fluids, and frequently required sedatives, such as sulphonal, as she was mischievously impulsive and violent, and was so alert to her surroundings that she took advantage of members of the staff whom she knew were not able to control her. In the middle of June she began to sleep, and there was a cessation of mental symptoms until early in July, when she relapsed, and was again excited for a month. During August and September she was apparently recovered, but her expression was heavy and flat, and she had often a hyperleucocytosis. In October she began to complain of pains in the back and urinary discomfort, but no cause could be detected for these symptoms. She complained of pain after taking food, and often vomited large quantities of mucus. She passed into a condition of depression, her temperature fell, she was fed with difficulty, and her bowels were very constipated. The leucocytosis was often as high as 20,000 and 30,000 per c.mm., but the polymorphonuclear percentage rarely exceeded 60. The heart's action was weak and slow, the hands and feet cold and cyanosed, and the body temperature was subnormal. The skin was greasy and clammy. Urine was voided in fair quantity, but at long intervals, but there was no deficiency in the excretion of urea.

She complained of vague pains. She was not sensitive to pain, but felt draughts and cold acutely. The pupils were rather contracted, but reacted sluggishly to light and accommodation, and there was some impairment of sight. The special senses of hearing, taste, and smell were not affected.

The organic reflexes of micturition and defæcation were under control, and the skin and tendon reflexes were still active. There was no paresis, but a distinct disinclination to exertion of any sort. Mentally the state was one of pure depression without confusion, hallucinations, or delusions. She spoke seldom, and took no interest in herself or her surroundings; but her memory was good, and she remembered having been admitted, and that she was excited. She slept very little—three or four hours a night. She continued in this state of depression, with one or two slight intermissions, during the months of November and December 1902, and January, February, March of 1903. During April she was



brighter, but during May and June all the symptoms of depression returned. Then she began to lay on weight and took food greedily ; but the expression of her face was listless and unintelligent, and she seemed to lack initiative. This state of subacute depression lasted until October 1904, when she was discharged.

The excited period lasted in all for about three months, then for two months she was apparently recovered. This was followed by depression, which lasted for about twelve months, and was succeeded by apparent recovery.

Although this patient has so far completed only one cycle of the disease, the excitement and depression were both characteristic of the physical and mental symptoms of folie circulaire.

T. F., male, had his first attack of mental disease at the age of 39. There was a history of direct hereditary predisposition to mental disease, and two brothers had also been treated in asylums.

The first attack came on during 1898, and lasted for six months. The mental symptoms were at first those of pure depression, without hallucinations or confusion, but it is recorded that for the last two months of his residence he was talkative, forward, inquisitive, interfering in matters which did not concern him, and discussed his private affairs openly and foolishly with casual acquaintances. The first cycle of the disease consisted, then, of four months of depression and two months of excitement.

The next attack commenced six months later with mild depression, which was quickly followed by excitement. The depression was not longer continued than a fortnight, but the excitement lasted for nearly three years. The onset of the excitement was quite typical. At first the patient became a little talkative and boastful of past performances. Then he became insolent and domineering, had many quarrels with attendants and fellow-patients, and he wrote constantly to the authorities making complaints and charges against the management of the Institution. His memory was excellent, and as he remembered all the little bits of scandal he had heard during his first residence in the Asylum, he took a great delight in repeating these facts before the interested persons, and was quick to perceive a successful effort on his part. Then he lost his self-control completely, became noisy, abusive, and absolutely sleepless. When very excited and angry he was incoherent, but throughout the whole attack consciousness and knowledge of position were never lost. At one time he had apparently delusions of identity, as he believed that some of the attendants were "M'Gregors"—a name for which he had a great aversion ; but to the best of my knowledge he never suffered from hallucinations except once when, under the influence of bromide,



he believed "gases" were blown into his room at night. Throughout the attack he took food well, but he lost 28 lbs. in weight during the first three months of the illness. This loss was regained as the excitement gradually passed off. Treatment in this case was confined to the use of bromides and sulphonal, as the patient was too violent and hostile to allow of any attempts at lavage of the intestine, etc., being made.

The second intermission lasted, like the first, for six months, when he was again admitted in a state of depression. He looked fat and well-nourished, but the skin was pale and pigmented. His attitude was listless, and his temperature 97° F. He took his food mechanically, but had no appetite, and his bowels were obstinately constipated. His leucocytosis was invariably high, but the increase was not in the polymorphonuclear cells but in the large lymphocytes and unicellular leucocytes. His heart's action was slow, the pulse full, and the arterial tension 140 mm. Hg. The skin was pale, pasty, and greasy; the urine 28 oz. for the first twenty-four hours, sp. gr. 1022. There was a heavy deposit of urates, and 400 grs. of urea were excreted. He complained of no sensory disturbances, and his common sensibility was not impaired. The pupils were dilated and sluggish in reaction to light and accommodation. Hearing, taste, and smell were unaffected. The organic reflexes were under control, and the skin and tendon reflexes active. Voluntary movement was sluggish, and to attempt the slightest manual work was a great labour to him.

Mentally he was depressed. There was little or no confusion. He did not suffer from hallucinations or delusions. He was quite self-controlled, and his memory was good. He took no interest in anything, and apparently had no power of initiative. For the most part he lay in bed, only rousing up at meal-times. His sleep was very irregular and deficient. During this attack he was treated by lavage of the intestine, large quantities of fluid by the mouth, and an occasional saline purgative. He slept better under this treatment, but I cannot say that the course of the disease was in any way altered by it, as the attack lasted for six months. The excited attack which was expected to follow the depression never came off, and the patient was finally discharged. The leucocytosis was high throughout the six months of depression, and he still presented this symptom upon discharge.

J. A. is an example of a short cycle. He has had recurrent attacks of excitement since adolescence, and there is hereditary predisposition to insanity in his family. The attack commences with depression associated with "rheumatic" pains, and very rarely lasts more than one day or at most two days. During the



depressed stage the leucocytosis may be as high as 17,000 to 18,000 per c.mm. of blood. As the excitement sets in the leucocytosis falls, but rises and culminates with the excitement, to fall again to normal. The excited period lasts from three weeks to six weeks, and is characterised by a complete change in manner, habits, and appearance. He becomes forward and abnormally inquisitive, his habits—usually tidy—are now the reverse, and often filthy. His appearance is suggestive of a man in alcohol. His temperature usually rises in the evenings to 99° or 100° F. His tongue is furred and he suffers from thirst. The heart's action is fast, and the arterial tension is 130 or 140 mm. Hg. The skin is moist, greasy, and offensive; the urine scanty and high coloured, and the sexual instincts somewhat accentuated. He has no sensory disturbances, and no impairment of sensibility. The pupils are dilated, but react readily to light and accommodation. Sight is very acute, and the patient can read without glasses, whereas, between attacks, he uses glasses. There is no loss of control over the organic reflexes. The skin and tendon reflexes are active. Voluntary movement is rather excessive, and never inco-ordinate.

*Mentally.*—He is elevated but not confused, and he never suffers from hallucinations or delusions. He is mischievous, sometimes destructive, always wanting in moral sense and attention to personal appearance. His memory is excellent, and he can quote chapters of Scripture on the slightest provocation, and all the events of past attacks seem to be vividly reproduced in each recurring one. The speech is inconsequent but not incoherent, and the tone of the voice is changed, being harsh and grating. In the intervals between attacks, which sometimes only last a few weeks, and at other times many months, there are no symptoms beyond an occasional hyperleucocytosis.

A. J., female, whose mental attacks commenced during adolescence, the first attack following a period of stress and worry caused by sick-nursing. No hereditary predisposition to insanity was acknowledged by the friends. The patient is a well-developed woman, whose attacks of excitement and depression cannot be said to constitute one cycle, but rather alternate with one another. The last attack I saw began on 1st July and was over by 12th July: this was the stage of depression. She was discharged, and readmitted in January of the following year, this time suffering from excitement, which lasted a fortnight, and the patient was again discharged. She had one complete cycle of symptoms prior to the one I was able to observe, and there was a period of four months between the period of depression and excitement. The record of the period of depression was as follows:—On admission



she was noted to be a tall, well-developed, well-nourished woman. Temperature  $98.2^{\circ}$  F. Her tongue was furred, and she had no appetite for food. The leucocytosis was 13,500 per c.mm., with a polymorphonuclear percentage of 67.5. The pulse was 90, irregular and irritable, and the arterial tension was 130 mm. Hg. The urine was scanty, loaded with urates, and there was a trace of albumen.

Sensibility to heat, touch, and pain was hyperacute. The pupils were dilated, and reacted to light and accommodation. The organic reflexes were under control. The skin and tendon reflexes were exaggerated and very rapid in reaction to stimuli, and there was no inco-ordination or loss of muscular power.

Mentally she was depressed and appeared confused, but, after recovery, she had a clear and accurate memory of all that had been said and done in the ward during her illness. She had no delusions and there were no hallucinations. Her memory was good, but she soon became exhausted if asked many questions, and speaking was evidently a labour. She slept irregularly, but was never restless.

After admission her leucocytosis gradually rose to 19,000 per c.mm., but there was no rise in the polymorphonuclear percentage. Her pulse became slower, her tendon and skin reflexes less exaggerated and active. Her temperature throughout the attack was irregular, but only once above  $98.4^{\circ}$  F. Twelve days after admission she rather suddenly improved, and said she felt quite well; thereafter she made a good recovery.

The treatment adopted was rest in bed, and the diet was chiefly milk and potash water for the first few days; the bowels were cleared by enemata.

When readmitted in January of the following year, she was well-nourished, but flushed and excited in appearance. The temperature was  $98.6^{\circ}$  F. The tongue was covered with a thick fur, and she suffered from constant eructation of gas. The leucocytosis was 13,500 per c.mm., the polymorphonuclear percentage 60. The pulse was 90, and the arterial tension 150 mm. Hg. The skin was dry. During the first twenty-four hours after admission 37 oz. of urine were excreted. The urine was pale, sp. gr. 1004, and contained 340 grs. of urea. No abnormal constituents were present. The nervous symptoms were similar to those noted during the period of depression.

Mentally she was excited and talkative, and not in the least confused. There were no hallucinations of the special senses, but hearing was abnormally acute. She scolded every one, and had many grievances against the nurses who had looked after her during previous attacks. Her attention was easily attracted and



as easily diverted. Her memory was good. Her speech was clear and coherent, but inconsequent. For the first eleven nights after admission she only slept eight and a half hours. On the twelfth night she slept seven hours, and thereafter slept well and made a rapid recovery. The leucocytosis during this period was never higher than 18,000 per c.mm., but the polymorphonuclear percentage was often above 70, and once above 80.

The treatment adopted was rest in bed; the food was milk diluted with potash water given in small quantities every two hours.

In reviewing these two types of excitement, the question arises, are they two distinct diseases or are they the same disease modified by individual resistive power or by differences in the toxins causing or complicating the disease? The answers to these questions cannot in the present state of knowledge be given, but there are certain facts and observations which throw some light upon their causation and course. Both excitement with confusion and excitement without confusion are undoubtedly toxic conditions, both present the symptoms of hyperleucocytosis, but whereas the one has a persistent and prolonged hyperleucocytosis, in the other the hyperleucocytosis is irregular and not persistent.

As recorded in an earlier chapter, I isolated from the blood of a case of excitement with confusion a pure culture of a small coccus, and, on testing the serum of this patient, I found that the serum in a dilution of 1-30 agglutinated this organism in less than an hour. Control serums in dilutions of 1 in 10, 1 in 20, and 1 in 30 never agglutinated this organism even after so long a period as twelve hours. I therefore tested the agglutinative power of the serum from every acute case admitted, and I found that in dilutions of from 1-20 to 1-30 the serum of cases suffering from excitement with confusion very frequently agglutinated this organism; sometimes agglutination was not complete for six hours, but against this is the fact that the control serums never produced the same action even in twelve hours or longer. Another fact noticed was that although a case might fail to agglutinate the organism in the early days of the illness, later in the disease or after recovery the same case had a specific agglutinine to this organism present in



the serum. Out of 20 cases so examined, 14 (or 70 per cent) gave definite reaction at some period of the illness, and 6 never gave a reaction at any time. In the same way it was found that the serum of cases of folie circulaire sometimes contained the same agglutinine, and the presence of the agglutinine in the blood varied according to the stage of the disease, being present in the period of excitement and sometimes absent during the stage of depression. Out of 14 cases of folie circulaire examined, 11 (or 71.4 per cent) gave definite reaction at some period of the disease, and 3 failed to give a reaction. These facts point strongly to the conclusion that the toxæmia in the two forms of excitement is of very similar origin. Quite recently I have been able to demonstrate another fact in connection with the resistive power of patients suffering from mania with confusion, which is, that some of these cases in the acute stage are unable to form agglutinines, while later in the disease agglutinines are readily formed. It occurred to me that by the subcutaneous injection of broth cultures of the organism it might be possible to raise the immunity of the patient and so cut short the attack. The organism was grown in broth for a month at 37° C., killed by chloroform vapour, and from 5-10 c.c. were then injected subcutaneously. A recent acute case with a deficient leucocytosis so treated reacted very slightly to the injections and no agglutinine was formed in the serum; while a chronic case, on the other hand, reacted vigorously and produced agglutinines in a fortnight after injection, so that the serum in dilution of 1-100 gave complete reaction in less than 30 minutes. It was noticed in this case that after the injection the eosinophile leucocytes increased and were often as high as 10-12 per cent; an interesting fact when it is remembered that a mild eosinophilia is generally present in the blood of these cases in the period succeeding the acute stage of the disease. This attempt at treatment was discontinued as no mental improvement resulted, and, so far, I have not tried it in excitement of the folie circulaire type. In normal serum there is present a substance of the nature of an agglutinine to some strains of the staphylococcus aureus, so that in dilutions of 1-10 the



organism is strongly agglutinated in 30 minutes. One of these strains of *staphylococcus aureus* I isolated from the bone marrow of a case of excitement with confusion which died in a typhoid state. On testing the serum of patients suffering from both forms of excitement, I found that they failed to agglutinate the organism in dilutions of 1-10, while out of 20 controls not one failed to agglutinate in a very marked manner. The serum, therefore, of these patients is deficient in some substance present in the serum of healthy persons.

It has often been noticed that acute intercurrent disease of the nature of boils, erysipelas, etc., frequently cuts short an attack of excitement, and I endeavoured to reproduce such a physical condition by injecting pure turpentine subcutaneously into the flank. The changes produced chiefly affect the leucocytes and are as follows:—From 12 to 24 hours after the injection the leucocytosis is unaffected, or may actually fall, but thereafter there is a distinct rise which culminates 48 hours after injection. Coincidentally with the hyperleucocytosis in acute cases there is almost invariably some mental improvement; but absolute and complete recovery, as the result of the treatment, occurs in only 10 per cent of the cases so treated. If cessation of excitement occurs in cases of *folie circulaire*, it does not follow that the depressed period is suppressed. The artificially stimulated leucocyte reaction appears to arrest or control the toxic condition alone, but does not affect the usual course of the disease. In chronic conditions the injection of terebene, beyond demonstrating that the leucocyte-producing power of the patient is either exhausted or disordered, produces no beneficial action.

The use of thyroid extract in the treatment of mental diseases is treated of elsewhere, but a short statement of its action on the two types of excitement under review is not out of place. In addition to the action of the drug on the circulatory, integumentary, and alimentary systems, there is also to be noted its action on the leucocytosis. In whatever form of mental disease thyroid extract may be given, it always reduces the leucocytosis and the percentage of the



polymorphonuclear leucocytes. As recovery in excitement with loss of consciousness apparently depends on an increase of the leucocytosis, it is not surprising to find that the exhibition of thyroid extract during the period of excitement is harmful and increases the severity of the mental symptoms. The same rule applies to the excitement of the folie circulaire type. If, however, the excited stage of folie circulaire can be anticipated and the drug then exhibited, it frequently cuts short and aborts the attack. It will be remembered that in the depression either preceding or following the excited stage of folie circulaire the leucocytosis is high, and the effect of administering thyroid at this stage is to lower the leucocytosis. The drug has to be used with caution, as excessive or prolonged use may precipitate the excited stage instead of aborting it.

So far as these observations go, they tend to prove that the state of toxæmia and serum reaction in the two types of excitement are very similar. The effect of artificially stimulating the leucocytosis by terebene injections is similar in both conditions in so far that in a small percentage of the cases it cuts short the excitement; but in cases of folie circulaire the depressed stage may, and usually does, follow just as if the excited stage had run its usual course—pointing to the fact that there is some other condition underlying the excited and depressed stages of the disease. The effect of thyroid medication also suggests that there is some deeper and unknown cause which permits of toxæmia in the disease known as folie circulaire. Excitement with confusion is, in all probability, a purely toxic condition; but folie circulaire is a more serious disease, and may be, as suggested by Macpherson, a neurosis liable to recurrent toxic complications. There is some support of this view in the fact that the types of excitement which complicate known neuroses such as epilepsy and general paralysis are very commonly of the folie circulaire type.

## CHAPTER X

### TOXIC INSANITIES—*continued*

#### KATATONIA (*κατατείνω*, I stretch tightly)

THE disease called katatonia, so called on account of the symptom of muscular spasm or rigidity which is present during certain stages of the disease, was first described and named by Kahlbaum in 1874. More recently the disease has been described by Kraepelin as one of the three varieties of Dementia Præcox—a group of diseases of adolescence which according to Kraepelin may present the three varieties of Katatonia, Hebephrenia, and Paranoia. Kraepelin's description of the disease does not tally in the sequence of symptoms with that of Kahlbaum, but on comparing the descriptions of these two authors there can be no doubt that they are both describing similar symptoms, only differing in detail. Of the two descriptions Kraepelin's is the most classical. Of late years many monographs have been published on the subject of "Dementia Præcox," with its three varieties "Katatonia," "Hebephrenia," and "Paranoia." Many of these publications consist of confused and unsatisfactory descriptions, and it is little wonder that most psychologists in this country refuse to admit the existence of these clinical entities. The term "dementia præcox" is unsatisfactory and unscientific. Some authors have gone so far as to include all adolescent disorders of mind under the term, their descriptions being based, mainly, on mental symptoms.

Katatonia is not a "recurrent insanity." The disease passes through at least three stages, and relapses in any of



these stages are common, but I have never seen the disease recur, in the ordinary sense of that word. I therefore prefer to describe katatonia as a distinct clinical disease without any reference to the supposed allied states of hebephrenia or paranoia.

Katatonia is commonly a disease of the developmental period of life, but I have seen typical cases occur in adult life, so that adolescence is not a necessary predisposing cause. It attacks women more commonly than men.

Hereditary predisposition to insanity is present in over 50 per cent of the cases, and is the most frequent predisposing cause, but I have known the disease to follow child-birth, scarlet fever, intemperate and vicious habits, mental and moral shocks, mental and physical strain, and in fact any factor which lowers the physical condition of the patient. In every case the history is one of gradual and insidious onset, with loss of energy and failure of nutrition. Hallucinations of hearing of a distressing nature are the earliest mental symptoms to manifest themselves, and these hallucinations lead sooner or later to impulsive actions, delusions, and complete loss of self-control. Physically many of the adolescent cases are poorly developed at the time the mental symptoms become pronounced. Every case I have seen was thin and badly nourished.

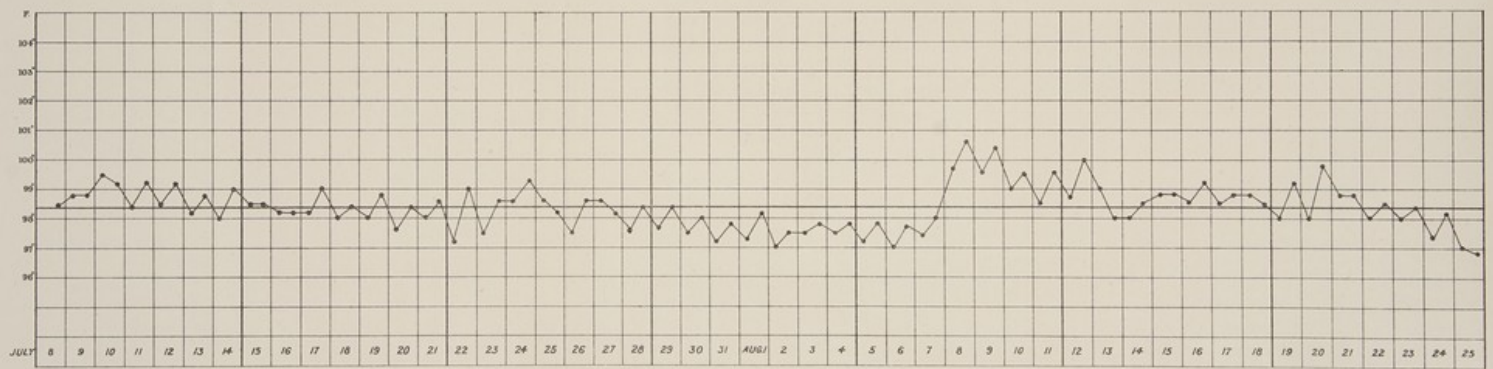
For convenience of description the disease may be divided into (1) The Stage of Onset; (2) The Stage of Stupor; (3) The Stage of Excitement.

The symptoms of the stage of onset are as follows:—The alimentary system is invariably disordered; there is loss of appetite, and frequently vomiting after food. The heart's action is rapid, irritable, irregular, and intermittent. The arterial pressure gradually rises as the symptoms become more acute. The skin is frequently drenched with perspiration, while blotchy and pustular rashes are present in about 50 per cent of the cases. No abnormality, beyond transient albuminuria, was noted in the urine. Menstruation is always suppressed in the female cases. The sensibility to touch, heat, and pain is always dulled. The pupils are dilated and sluggish in their reaction to light. The special senses of sight and hearing are





CHART No. 4.



TEMPERATURE CHART in a case of Katatonía. The febrile attack which commenced during the fifth week after admission was followed by the stage of stupor.

not affected so far as outward impressions are concerned, but taste and smell are often completely perverted. The organic reflexes of micturition and defæcation are not under control, and the patients are usually wet and dirty in their habits, unless constantly attended to and watched. The skin and tendon reflexes are exaggerated. At uncertain intervals the voluntary muscles pass into a state of katatonic spasm, which lasts, variably, in different cases, for a few minutes or hours. The mental state is essentially one of confusion, with vivid and distressing auditory hallucinations, which affect conduct and frequently lead to paroxysms of terror, during which the patient shouts, struggles, or perhaps tries to jump through a window, run out of the door, or hide under the bed. What are called attempts at suicide in this stage are always the frenzied efforts of a terrified patient to escape from some unknown danger suggested by the auditory hallucinations. In the intervals between such attacks the patient may lie for hours with closed eyes, apparently oblivious to all surroundings. In other cases there may be brief periods of apparent sanity, in which, however, the patient is always confused, and there is no power of attention to conversation, and no memory for recent events. Sleep is abolished or deficient. The temperature is irregular, rarely febrile, but often paradoxical. In typical cases, at the end of the acute stage, there is a distinct febrile attack which ushers in the stage of stupor, but not infrequently this symptom is wanting. The leucocyte counts show a moderate persistent hyperleucocytosis, the increase being chiefly in the polymorphonuclear and large mononuclear cells. At the termination of the acute stage, and just prior to the onset of stupor, there is a sharp rise in the leucocytes, the increase being chiefly in the polymorphonuclear cells. The acute stage may last from one day to six weeks, the average duration being three or four weeks.

The second, or stuporose stage of the disease, comes on immediately after the febrile attack, or, when this symptom is wanting, after the high leucocytosis which terminates the acute stage. The physical symptoms of the stuporose stage are very characteristic. The alimentary tract is still disordered and the bowels constipated. The heart's action is



weak and slow, and the arterial tension falls. The extremities are blue and cold, and the feet and hands become œdematous. The temperature is subnormal, with occasional rises to 99° and 100° F. The skin often desquamates in small, branny scales, is generally greasy, giving to the face a "varnished" appearance. Amenorrhœa is a constant symptom in women. It is impossible to test the sensory functions, but the special senses are quite active, as patients in this stage are aware of all that passes around them. There is a tendency to retain the urine and fæces, the patient resisting these organic reflexes. The skin reflexes are active, but the tendon reflexes cannot be elicited on account of muscular resistance. The voluntary muscles are thrown into resistance by any attempts at passive movements.

The mental state is one of semi-conscious stupor often complicated by delusions. Impulsive movements and actions, often suicidal, curious attitudes, mutism, rhythmical movements, rhythmical repetition of words, letters, names, numbers, etc., sudden outbursts of excitement—as sudden in termination as in onset—and obstinate resistance to any attempts at movements or assistance in the way of feeding, dressing, and undressing occur, in some degree, in every case. The function of sleep returns, and is often excessive. The condition of the leucocytosis is as follows:—Immediately after the onset of the stupor the leucocytes may fall as low as 8000 per c.mm. of blood, but immediately rise again, averaging between 12,000 and 16,000 per c.mm. The percentage of polymorphonuclear cells falls to about 60, the lymphocytes increase, and a transient eosinophilia occurs in every case. In favourable cases which recover without passing into the third stage, or stage of excitement, the percentage of polymorphonuclear cells never falls below 60. In cases which are not going to recover the leucocytosis, early in the stage of stupor, falls to below 10,000 per c.mm., the polymorphonuclear percentage falls below 50, and I have seen it as low as 28.

Prognosis in a case of katatonia is always difficult, but so long as the leucocytosis and polymorphonuclear percentage is maintained there is good hope of recovery.

The stage of stupor is of very uncertain duration; in



some cases it may last a few weeks, while in others it may last several months, or even years. The prognosis in long-continued stupor is bad.

The third stage—that of excitement—may follow immediately upon the stage of stupor, or the patient may apparently recover after the stupor, and then relapse, even after so long a period as two or three years. There is always a certain amount of confusion and impairment of memory, delusions and hallucinations are more pronounced, and frequently affect the conduct. The excitement is not continuous, but is liable to remissions and exacerbations, and is frequently associated with stereotyped rhythmical motor disturbances, which evidence themselves by aimless “sentry-go” walking, or walking in circles, movements of the hands, and irregular action of the facial muscles. The condition is difficult to diagnose without the history of a previous stage of stupor. The older the patient the less tendency is there for the disease to terminate in marked dementia, but the majority of adolescent cases, unfortunately, do not recover. According to Kraepelin, out of every 100 cases 13 recover, 27 make partial recoveries, and 60 become more or less demented.

Bacterial examination of the blood was made in 10 cases during the acute stage. The method adopted was to run 3, 4, or 5 c.c. of blood into 200 c.c. of sterile broth, by means of an exploring needle passed into any prominent vein in the forearm. Seven of the flasks were sterile, two were accidentally contaminated, but one, obtained from a case which had passed into a typhoid state, presented a pure culture of a short streptococcus. The patient recovered from the typhoid state, and passed into stupor, and the blood serum was found to agglutinate the organism in a dilution of 1-30 in two hours.

I have tested the serum reaction of 20 cases of katatonia to this organism, and obtained agglutination in 15 cases, or 75 per cent. The reaction was always slow, sometimes taking 6 hours, but 20 control serums failed to give reaction in 12 hours or longer. I have also tested the serum reaction to this organism in 120 cases of other forms of insanity. Twelve gave agglutination, the remaining 108



failing to give reaction. The cases which gave reaction were three cases of folie circulaire, two of excited melancholia, two of alcoholic mania, two of mania with confusion in adolescent cases, one of hebephrenia, one unclassified case transferred from another asylum, one senile case whose symptoms, though not typical, approached more closely those of katatonia than any other known form of mental disease. It is probable, therefore, that the agglutinine frequently present in the blood of patients suffering from katatonia is a specific agglutinine.

The treatment of katatonia is unsatisfactory. During the acute stage rest in bed, with a dietary of milk, eggs, toast, butter, and weak tea, and the free use of normal saline infusions, either subcutaneously or by the rectum, give a certain amount of relief. Hypnotics should be avoided, if possible, but when necessary, paraldehyde in two or three drachm doses is the best, and should only be employed every third night. If death occurs during the acute stage the cause is usually exhaustion, with evidences of severe toxæmia.

During the stage of stupor the treatment should be general. Fresh air, tonics, a full dietary, and patience. If exercise is taken it must be voluntary, and not forced. When the patient shows signs of recovery hot baths, with mustard added, seem to hasten the process. If, however, this treatment raises the temperature above normal, or unduly quickens the pulse-rate, then it should be at once discontinued.

During the period of excitement tonics, especially iron and quinine, are indicated, and if the restlessness is so great that the patient becomes exhausted, 10 grains of sulphonal thrice daily and rest in bed often produce a good result.

Serum treatment has so far produced no action. A goat was immunised to the streptococcus obtained from the case of katatonia, and the serum was injected subcutaneously, in 12 c.c. doses daily for a week, into two cases in the stage of stupor, and one case in the acute stage. All the patients suffered from urticaria and local erythema round the point of injection, but otherwise the result was nil. Two stuporose cases were then treated by 10 c.c. doses administered by the mouth. The only result noticed was that the temperature in both



cases fell to below 97° F. during the period of administration. One acute case was treated with doses of 80 and 140 c.c. by oral administration. In this patient also the temperature fell below 97° F., but no other change was observed beyond the fact that the patient developed urticaria. Five stuporose cases were treated by injections of broth cultures of the streptococcus killed by heat at 60° C. All gained weight and developed agglutinines to the organism, but were otherwise unchanged. One acute case was treated also by subcutaneous injections of broth culture, and the treatment appeared to give temporary relief, but at the end of six weeks the period of stupor set in. It is worth noting that this case failed to produce a satisfactory agglutinine.

Thyroid treatment never improves the condition, and often aggravates all the symptoms, stuporose patients waking up to a condition of restless, impulsive excitement.

Rabbits experimentally infected with the streptococcus, either intravenously or by the alimentary tract, developed a condition of malaise with irregular temperature, increased skin reflexes, and mental hebetude. The disease tended to terminate naturally in about six weeks, and a condition of immunity was established to the organism.

As the result of my observations, I believe katatonia to be an acute toxic disease, with a definite course and onset, in which the symptoms vary according to the resistive power of the patient, but in which the following diagnostic symptoms are generally present:—A prodromal period of gradual onset, which leads into the period of acute onset, with aural hallucinations, mental confusion, paroxysms of fear, impulsive actions, katatonic spasm of the muscles, and a hyperleucocytosis which, at the termination of the acute stage, indicates a virulent toxæmia. In the second stage a condition of stupor without loss of consciousness, with muscular resistiveness to passive movements, and this stage may or may not pass into a stage of excitement.

The following case illustrates a mild attack of katatonia, which terminated in recovery:—

J. J., female, aged 20, was admitted on 12th August 1902. She had been ill for two months upon admission.



*History.*—No hereditary predisposition to insanity was known. The patient was the youngest child of a large family, all the other members being strong and healthy. About two months previous to admission she became listless and depressed, left her work, and moped about at home. Then she developed hallucinations, as the result of which she announced that she was Satan, and became somewhat impulsive, running out of the house and hiding herself.

On admission she was a healthy-looking girl, but undeveloped for her age and in poor condition. Temperature 97° F. Her tongue was furred, her teeth carious, the bowels constipated, and she refused food. The leucocytosis was 11,000, with a polymorphonuclear percentage of 75.5. The pulse was 80 and weak, and there was cyanosis of the hands and feet. The skin was darkly pigmented, with a pustular rash over the arms and legs. Nothing abnormal was found in the urine. She was suffering from amenorrhœa. Sensibility to touch and heat was impaired over the arms and legs. The pupils were widely dilated, the right being larger than the left. The reaction to light and accommodation was present, but sluggish. The sense of taste was impaired, as strychnine was mistaken for sugar. The organic reflexes of micturition and defæcation were under control. The skin and tendon reflexes were exaggerated. There was no failure of muscular power, and no inco-ordination, rigidity, or tremor.

Mentally she was slightly confused; suffered from hallucinations of hearing, the voices telling her she was "Satan." Her attention was difficult to attract, and she refused to enter into conversation, merely reiterating that she was "bad, bad Satan."

She slept fairly well. During the next few days she was often restless and impulsive; her pulse became rapid, and the leucocytosis gradually rose to 15,000 per c.mm. She had two very transient attacks of muscular rigidity. On the morning of the eleventh day after admission the temperature rose to 99.4° F., and the pulse was 104. In the evening the temperature was 100.6°, and the pulse 96. No cause could be detected to account for the state of fever. The leucocytosis was 32,000, with a polymorphonuclear percentage of 86. The following morning she was stuporose, but not markedly resistive, although she required to be fed by hand, and refused to speak or move. The leucocytosis was now 13,000, and the polymorphonuclear percentage 74. For the next two months she was stuporose and resistive to passive movements, absolutely mute, but alive to what was passing around her. During December she was dressed each day and got out of doors. On two occasions she stripped herself of her clothes and stood motion-



less in the ward, refusing absolutely to speak or give any reason for her conduct. By the end of December she was improving, and by January 1903 she was apparently recovered. She was kept under observation until April 1903. The treatment adopted during the acute stage was rest in bed, fluid diet, chiefly milk, and, as stupor passed off, hot mustard baths. During the whole illness the leucocytosis never fell below 12,000 per c.mm., and the polymorphonuclear percentage only once fell below 60.

C. M., female, aged 27, was admitted in July 1901 in a state of excitement and confusion.

*History.*—There was no known hereditary predisposition to insanity, but the patient had suffered from some form of mental disorder at the age of 20. The present attack commenced some three months prior to admission, when the patient, a laundress, left her work, was depressed and peculiar in conduct, lost all interest in her personal appearance, and became sleepless. The acute symptoms, consisting of hallucinations both of sight and hearing, associated with restlessness, came on five days prior to admission. On admission she was noted as a big, strong woman of youthful appearance for her age, and in a poor state of nutrition. Temperature 98° F. Her tongue was furred, the teeth carious, and the desire for food and drink was deficient; the bowels were confined. The heart's action was weak, the pulse 90, and the arterial tension 130 mm. Hg. The leucocytosis was 9000 per c.mm., with a polymorphonuclear percentage of 72. The skin was moist and clammy, and at times she perspired freely. Nothing abnormal was noted in the urine. She menstruated once just after admission, and then ceased to menstruate until dementia set in. There seemed to be no impairment of sensibility. The pupils were semi-dilated, and reacted to light and accommodation. The special senses of taste and smell could not be tested. The organic reflexes were not under control, and the patient was frequently wet. There was no paralysis, no inco-ordination of movement, but the muscles of the hand, forearm, and face were very tremulous. No rigidity of the muscles was noted in the acute stage. Mentally the patient was frightened, confused, and suffering from acute and vivid hallucinations of sight and hearing, under the influence of which she had wild attacks of terror, when she tried to climb out of the windows, or screamed and crept to the bottom of the bed. During these attacks the skin streamed with perspiration, which was offensive. In the intervals between attacks she lay quiet, with the eyes shut, muttering to herself, and apparently oblivious to her surroundings. For the first three nights she did not sleep, and there were frequent attacks of terror. She took a fair quantity



of fluid food, and large saline enemata were given daily. The condition remained unchanged for three weeks, during which there were one or two very transient periods of sanity, when she asked where she was and answered questions. Then a febrile attack set in, with persistent vomiting of food; the leucocytosis rose to 28,000, and the polymorphonuclear cells to 80 per cent. The fever lasted a week, during which the mental and physical symptoms were acute. On the fifth day of the fever she passed into a condition of stupor with muscular rigidity. The circulation now became weak, the arterial tension low, and the feet and hands livid. The skin, especially over the face, desquamated, and later assumed a varnished, drawn appearance, and the face was expressionless. For the next nine months she was stuporose, dirty in her habits, requiring to be hand-fed and dressed and undressed. At times she retained her urine until the bladder was distended. Sometimes she spent hours swaying her body backwards and forwards in bed; at others she sat rigid, with the eyes tightly shut, and during these attacks she became cataleptic, and would remain in grotesque and uncomfortable attitudes for long periods. The temperature was uniformly subnormal, and the leucocytosis rarely rose above 12,000 per c.mm., while the polymorphonuclear percentage averaged 50. During the tenth month of stupor she improved sufficiently to work in the laundry, and she continued to work for four months, when she again became stuporose. On 15th September 1902 she became excited, walking restlessly up and down the ward. At times she was mischievous, annoying her fellow-patients, breaking dishes and windows, and tearing her clothes. Confusion was well marked throughout the period of excitement, which lasted until January 1903, when she again returned to work in the laundry. After the period of excitement the patient gained weight rapidly, became apathetic and listless, and passed into confirmed dementia. She is now a fat, healthy-looking dement, who is capable of working under supervision. In this case the acute stage lasted about four weeks, the stupor, with a slight intermission, for fourteen months, and the excitement for about four months; but the excitement so gradually passed into dementia that it was impossible to say when excitement ended and dementia was complete. During the later period of stupor and the stage of excitement the leucocytosis was irregular, but the polymorphonuclear percentage was frequently 30 and 40.

J. D., male, aged 59, an engine-driver, was admitted in January 1903 in a state of depression which had lasted seven days. A brother and uncle had suffered from insanity.

*History.*—He had had one previous attack thirteen years ago,



the nature of which was unknown. He was a man of intemperate habits, and the present attack commenced three months ago, when the patient was dismissed from his situation for being drunk. He became dull, sleepless, and disinclined for food, and lost nearly two stone in weight. During these last seven days he had evidently suffered from hallucinations of hearing, as he was constantly starting up, saying that he heard engines "whistling," and he had not slept for many nights. On admission he was a big, powerful, well-built man, but emaciated. His expression was one of fear and confusion; the temperature 99° F. The tongue was glazed and dry, the conjunctivæ were jaundiced, there was marked distaste for food of any sort, and the bowels were confined. The heart's action was irregular and tumultuous, and the pulse 120 per minute. The skin was moist, and he perspired freely, the perspiration being sour in odour. The leucocytosis was 11,000, and the polymorphonuclear percentage 80. The urine could not be collected for examination, as the patient was constantly wet. The sensibility to touch, heat, and pain was markedly diminished; the pupils were dilated and sluggish in reaction to light. Taste, smell, and hearing could not be tested. The organic reflexes of micturition and defæcation were not under control. The skin and tendon reflexes were increased. There was no paralysis or inco-ordination, but the muscles of the face, lips, tongue, arms, and hands were very tremulous. Mentally he was frightened, suspicious, and confused. There were hallucinations of both sight and hearing, and he had some vague delusion that his wife and children were in danger of being killed. He had repeated attacks of wild, frenzied excitement, in some of which he required the subcutaneous injection of hyoscine to control him. These attacks were evidently due to fear, as he was evidently terrified, and trying to escape from some imaginary danger. For three weeks he remained in this condition and slept very badly. The temperature was often slightly raised. He had repeated attacks of retention of urine, and was confused and oblivious to his surroundings. On the twenty-second day after admission he passed into a state of muscular rigidity, the eyes were tightly closed, and his arms, legs, and trunk so rigid that the body was "board-like." This condition gradually passed into stupor, with resistance to passive movements. During the attack of rigidity the leucocytosis rose to 40,000 per c.mm., and the polymorphonuclear percentage to 91. This condition of stupor barely lasted a fortnight, when he became excited and impulsive, laboured under the delusion that his wife and children had been murdered, and he was often dangerous to the officials in consequence. He was very confused, and quite irrational and incoherent in conversation, and his sleep was irregular. During the period of stupor he slept



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almost continuously night and day. The condition of excitement has lasted for over two years, and, though the patient is less confused, there are evidences of enfeeblement now setting in. The leucocytosis is low, and the polymorphonuclear percentage is often below 50.

## CHAPTER XI

### TOXIC INSANITIES—*continued*

#### HEBEPHRENIA ( $\eta\beta\eta$ , puberty; $\phi\rho\acute{\eta}\nu$ , the mind)

THE word hebephrenia, which means the mind of puberty, was first used by Hecker and Kahlbaum to describe certain pathological mental symptoms which occur at puberty or immediately after. Kraepelin now includes hebephrenia as one of the subgroups of the diseases called by him *dementia præcox*, the other two divisions of the same diseases being called katatonia and paranoia. In this country hebephrenia is not recognised as a distinct disease. Clouston describes cases of it in his adolescent insanities. Macpherson does not mention it at all, and its existence is denied by many writers. The symptoms are regarded as the result of premature brain involution, and the disease terminates in dementia in the majority of cases.

Where there is no uniformity of knowledge there can be no uniformity of description, and the disease which I hereafter describe as hebephrenia is a disease of adolescence rather than puberty, in which the symptoms, both mental and physical, are for the most part subacute, but run a definite course, and in which, although there is so far no evidence of metabolic toxæmia, the symptom of hyperleucocytosis is so frequently present as to justify its inclusion in Insanities of Bacterial Origin.

The disease as I have seen it is quite distinct from katatonia on the one hand and paranoia on the other, and I see no need to classify it under that most unscientific term



*dementia præcox*, merely because it occurs in adolescence and frequently terminates in dementia.

While katatonia is most frequently seen in adolescence, but may occur in adult life or even later, hebephrenia is essentially a disease of early adolescence, and never occurs in the adult. According to Kahlbaum the disease is more prevalent in females than in males, and hereditary predisposition to insanity is present in over 50 per cent of the cases. The symptoms in their onset are always gradual and indefinite. The patient, who has more frequently than not been intelligent or even precocious, changes in character. Peculiarities of conduct, which may vary from eccentricities to undoubted mental aberration, develop, and there is a loss of capacity for sustained attention, and consequently a loss of power for work. The patient wanders aimlessly about, and may become dissolute or mischievous, but as a rule such cases shun the society of their fellows and lead a solitary existence, and may be frequently seen giggling and muttering when unobserved. The relatives at this stage fail to recognise that the conduct of the patient is the onset of disease, and the treatment adopted is either spiritual exhortation or physical repression, under which the deficient self-control of the patient is apt to show itself by irritability and violence often with little provocation. As the disease advances intolerance of control is a marked symptom, and in the worst cases delusions develop early. Every case of hebephrenia I have seen showed arrest of physical development, *i.e.* a patient twenty years of age looked much more like fifteen or sixteen, and such patients carry this preternaturally youthful appearance throughout life. The general appearance is that of a physical and mental degenerate. The expression of the face is furtive, and lacking in vivacity and intelligence. The attitude may be peculiar and the movements awkward, and there is no desire for active exercise or such amusements as one would expect to interest a healthy individual of the same age. The temperature is for long periods unaffected, but during the occurrence of acute symptoms, and every now and then during the course of the disease, periods occur which may be regarded as relapses, when the patient becomes sleepless, and perhaps restless, depressed,





CHART No. 5.

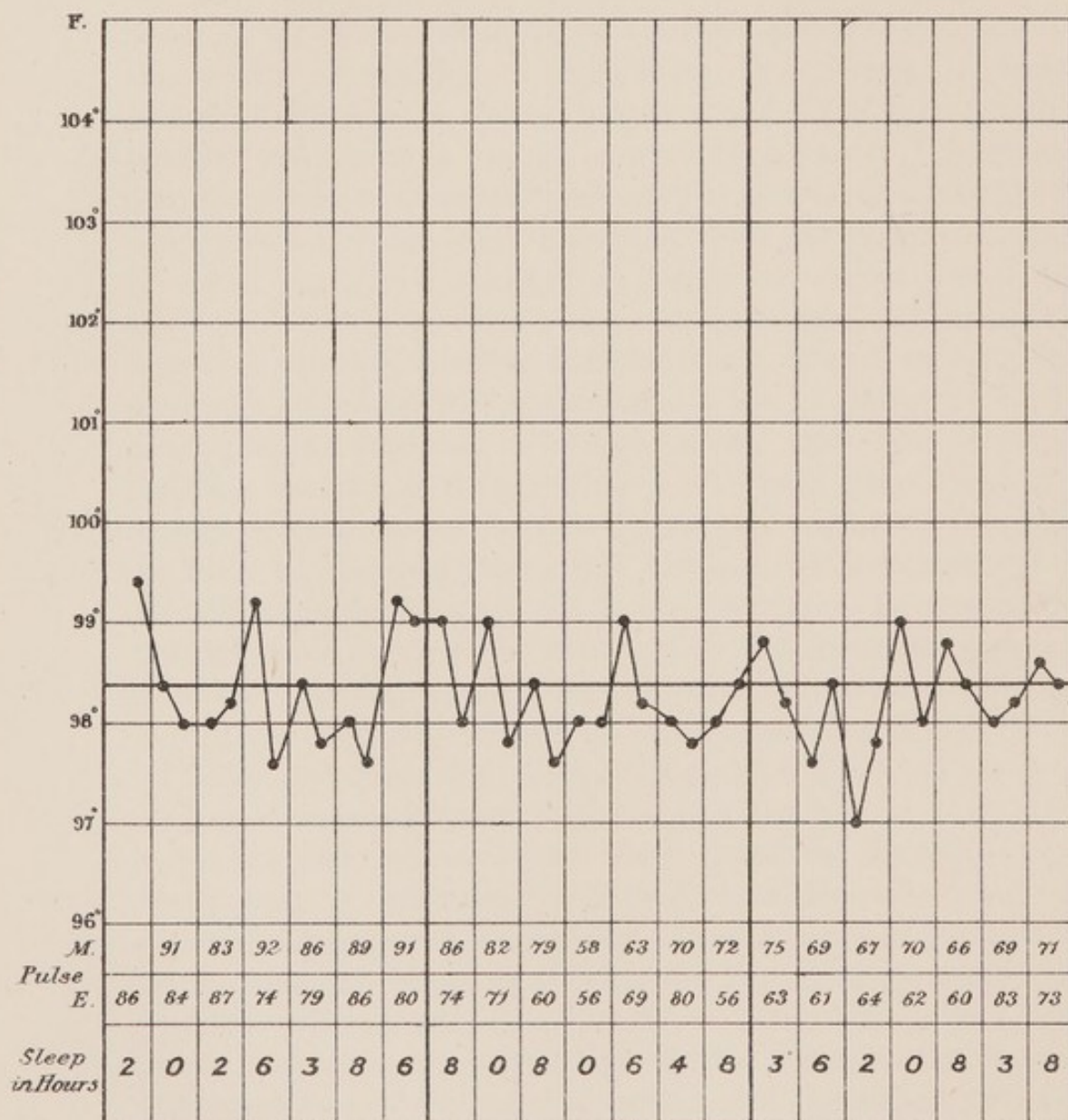


CHART showing the Temperature, Pulse-Rate, and Sleep in Hours in the early stages of a case of Hebeephrenia.

or troublesome. During such periods the temperature rises slightly, 99°-100° F., generally in the evening, and may continue thus for one, two, or three weeks.

The alimentary tract presents no characteristic symptoms even during acute attacks. The leucocytosis is generally about 12,000 to 14,000 per c.mm., but in some cases a marked hyperleucocytosis occurs without any corresponding mental or physical change, and such a hyperleucocytosis is rarely associated with a high percentage of polymorphonuclear cells, the increase being chiefly in the mononuclear and large lymphocyte cells, which may rise to 20 or 30 per cent. I have only seen one case recover, and in that case the leucocytosis fell below 10,000 per c.mm. on recovery.

The heart's action is regular, and the pulse between 60 and 70 beats per minute, except during exacerbations, when the pulse becomes faster and irregular, but there is little change in the arterial tension.

The lungs are liable to tubercular infection. The skin is sometimes dry and harsh, sometimes moist and greasy. The urine is excreted in fair quantity, and there is no diminution in the excretion of urea or chlorides.

Menstruation is suppressed or irregular in women, and the male patients are generally masturbators.

There is in some cases slight anæsthesia to pain. The pupils incline to dilatation, but react to light and accommodation. Hearing, taste, and smell are apparently unaffected.

The organic reflexes of micturition and defæcation are under control. The skin and tendon reflexes are not increased, and there is no paresis or coarse inco-ordination of the voluntary muscles. Prior to an exacerbation or relapse the power of nutrition fails, and the patient rapidly loses weight.

*Mental Symptoms.*—The mental symptoms are various: sometimes the patient is dull and suspicious, but not confused, sometimes slightly elevated and excited. Hallucinations may be entirely absent in the early stages of the disease, but if present they are usually hallucinations of hearing. Some patients suffer from delusions, but delusions are not an outstanding or necessary symptom. There are always some



enfeeblement and irritability. There is little or no affection for relatives or friends. There is deficient power of attention, and little capacity for occupation. The power of initiative is impaired, and a typical case is content to sit day after day absolutely without interest or occupation of any sort. The memory in the earlier stages is good for recent events, but later there is marked impairment. The speech is often hesitating and jerky, and the power of coherently carrying on a conversation is gradually lost as the disease progresses. Sleep is always abolished during the periods when the temperature is febrile, and in some cases there is a curious diurnal variation, the patient sleeping one night, but not the next, or sleeping two nights and not sleeping on the third. As the disease progresses the power of attention and mental application becomes further impaired and finally abolished. Hallucinations of hearing frequently develop and lead to delusions and impulsive conduct—assaults, tearing of clothes, breaking of glass and crockery, and general restlessness; this phase gradually passes into complete dementia.

*Prognosis*—According to Kraepelin 5 per cent recover, 15 per cent are so far relieved as to be able to live at home, but are mentally enfeebled, the remaining 80 per cent become hopelessly demented.

The prognosis is to some extent indicated by the development and physique of the patient. Those who recover are generally the better developed of the cases, and during recovery there is marked improvement in development.

*Causation*.—Absolutely nothing is known regarding the causation of hebephrenia. There are practically no physical symptoms beyond the deficient development and the slight hyperleucocytosis, which differs from the leucocytosis of excitement with confusion in the fact that the polymorphonuclear cells are rarely above 70 per cent. No agglutinines have so far been discovered in the serum, but in common with many other forms of insanity, cases of hebephrenia may be deficient in the normal agglutinines or alexines which act upon some strains of staphylococcus aureus and are present in a state of health.

*Treatment*.—The treatment must run on general lines.



During the acute exacerbations the patient should be confined to bed, but for the rest, regular hours, light steady work if possible, routine discipline, tonics, and fattening, non-stimulating food produce the best results.

The following are clinical examples of the disease :—

J. D., male, aged 20, was admitted in March 1903 suffering from mild excitement and sleeplessness. There was distinct hereditary predisposition, the father having been insane. He was a lad of good habits, steady and intelligent, but at the age of 17 he had suffered from a mental attack which was treated at home. The present illness commenced three weeks ago with sleeplessness and change of habits and character. The patient left his work, wandered aimlessly about, often stayed for days at a time in bed, and was constantly sniggering and talking to himself.

On admission he was thin and poorly developed. There was no growth of hair on the face, and he had a very boyish appearance. His expression was foolish and fatuous, and he sat up in bed chattering and laughing to himself. The temperature was  $99.4^{\circ}$  F., and for the next three weeks the temperature was frequently febrile, always in the morning. There were no gastric symptoms, and the patient took food well. His leucocytosis was 12,000 per c.mm. with a polymorphonuclear percentage of 62. For the next three weeks the leucocytosis varied between 12,000 and 15,000, and the highest percentage of polymorphonuclear cells recorded was 69. The heart's action was a little irregular, and the pulse-rate was generally faster in the morning than in the evening, and varied between 91 and 74 for the first week. No abnormality was detected in the respiratory, integumentary, or urinary systems.

*Nervous System.*—There was no impairment of sensibility to touch, heat, or pain. The pupils were semidilated, but reacted to light and accommodation. The senses of sight, hearing, taste, and smell were all acute. The organic reflexes of micturition and defæcation were under control. The skin and tendon reflexes were present and active, and there was no impairment of voluntary muscular movement. Mentally he was slightly elevated, but not in the least confused. He knew where he was, and noticed all that passed in the ward. There were no evidences of hallucinations or delusions, but his appearance suggested enfeeblement, and his constant giggling was abnormal.

When first admitted he was restless, and several times got out of bed, but he was quiet and biddable when the nurse told him to go back to bed again. He had sufficient self-control to stop giggling and chattering during a medical visit, and he was never impulsive. His attention, however, was deficient, he could not settle down to



read, and he could not carry on a conversation. His memory for recent and past events was good. The speech was clear and crisp, but boyish in tone, and he understood both written and spoken language.

For the first three nights after admission he slept only four hours, but after that sleep gradually returned. He was kept in bed for six weeks, and was fed on a full dietary supplemented with milk and eggs, but no butcher meat. At the end of three weeks he was much improved, and by the end of six weeks he had gained a stone in weight. During the next six weeks, when he was up and working in the garden, he developed enormously—his voice became deeper and more manly, his moustache and whiskers grew rapidly, and his muscular development was good. He never took any interest in games, and was rather cowardly at cricket. He was discharged three months after admission, recovered. This case is of interest, as the patient was seen early in the attack, and demonstrates how physical improvement goes hand in hand with mental recovery.

J. H., female, aged 23, was admitted in April 1902, because her friends were unable to manage her at home. She had been unable to follow her occupation as a domestic servant for a year prior to admission. Her mother was a nervous, eccentric woman. The patient had been a bright, intelligent girl of good habits, and an excellent servant until a year ago, when she became peculiar in manner, and eccentric and unreliable in the discharge of her work. At home she was irritable, restless, and unable to work. She became abnormally shy and avoided the society of her relatives. She was often noticed going through automatic movements with her hands, and about three weeks ago she apparently developed hallucinations and delusions, as she thought that people were chasing her.

On admission she was a feeble, flabby, anæmic girl below middle height, and very imperfectly developed for her age. She had a furtive, shy manner, never looked one in the face, and generally sat with her chin tucked into her chest. She often sniggered in a silly, causeless way and refused to speak. The temperature was 98° F.

Beyond suppression of menstruation there was no physical abnormality detected. The leucocytosis was 13,700 per c.mm. and the polymorphonuclear percentage 53, but the large unicellular cells were 12·5.

*Nervous System.*—She complained of no subjective sensations, and so far as could be detected, sensibility to touch, heat, and pain were acute. The pupils were dilated, but reacted readily to light and accommodation. The special senses were unaffected. There was diminution of the skin and tendon reflexes, and the voluntary



movements were awkward. Mentally she was foolish and enfeebled rather than depressed. She expressed no delusions, and apparently did not suffer from hallucinations. She was quite unable to occupy herself in any way either by sewing, knitting, or reading, and she took no interest in her surroundings. When she answered questions she spoke in whispers, and it was impossible to test her powers of memory. On admission she slept seven or eight hours each night. Beyond the fact that she gained in weight there was no change in her condition until March 1903, when she gradually began to lose weight again. Attempts were made to occupy her in the laundry, but without success. During December 1903 she complained of feeling sick, but her tongue was clean, and she took food fairly well, but at times required to be hand-fed. Her pulse became fast—over 90 per minute—and she perspired freely. Then she became restless and sleepless, and suffered from hallucinations of hearing. During this exacerbation of symptoms, which lasted from Dec. 1903 to March 1904, the temperature was irregularly febrile, the pulse fast, and the leucocytosis varied between 13,000 and 15,000 per c.mm., the polymorphonuclear percentage being between 50 and 60, but on two occasions the polymorphonuclear percentage rose once to 70 per cent and once to 80 per cent. On recovering from this relapse she again began to gain weight, and in May 1904 she menstruated for the first time since admission. During this menstrual period she first developed impulsiveness, when she made sudden and unprovoked attacks upon the staff and fellow-patients, stating as her reason for these assaults that people were calling her names. These impulsive attacks came on suddenly and without warning. For the remainder of 1904 she was always impulsive during menstrual periods, but the acuteness of the attacks gradually lessened, and she is now—May 1905—much demented, and only a little restless at the menstrual periods.



## CHAPTER XII

### TOXIC INSANITIES—*continued*

#### GROUP III.—INSANITIES THE RESULT OF ALCOHOLIC AND DRUG TOXINES

As is well known, alcohol taken in excess by an individual in health produces temporary mental aberration which commences with excitement, confusion of ideas, and motor inco-ordination, and may terminate in stupor, with complete loss of muscular power. Such excess in an individual of sound nervous constitution produces no visible permanent result, unless the excess is repeated at very frequent intervals extending over long periods. It is different, however, when the alcohol drinker possesses an unstable brain cortex, either as the result of hereditary predisposition, previous brain disease, or brain traumatism. In such cases the quantity of alcohol which may produce acute mental symptoms is often small in proportion to the acuteness of the symptoms.

H. H., an old soldier, was constantly being admitted to the asylum in a state of acute excitement, the result generally of one or, at most, two glasses of whisky. He was discharged from the service because of this tendency to violence, which invariably came on if he took alcohol. He had only suffered from these attacks since receiving a severe head injury. On one occasion he was brought to the asylum in a cab, the glass in the windows of which was broken, and the floor was burst out. It took the united efforts of two powerful inspectors of poor, who were with him, to keep the patient in the cab. Two hours after admission he was quite sane, but confused, and without any memory of what had recently happened.

In a great many cases of what are wrongly called alcoholic insanity the alcoholism is only a predisposing cause of the disease, and in a still larger proportion of cases it is only a symptom.

**Delirium Tremens.**—Delirium tremens is probably the most common form of mental derangement associated with alcoholic excess. The onset of the symptoms, however, suggests that the disease is not due directly to alcoholic poisoning, but to nutritional disturbances brought about by the changes which excessive alcoholic indulgence produces in the functions of the stomach, intestines, and liver. A patient may suffer from delirium tremens who has never at any time been intoxicated, but who has, nevertheless, partaken freely and frequently of alcoholic beverages. In such a case the disease is usually a complication of surgical shock, or injury, or pneumonia. Or again, the patient may develop the condition weeks after the use of alcohol has been entirely discontinued. The most marked early symptoms of the disease are those of alimentary disorder, loss of appetite, abdominal pain, and sometimes persistent vomiting, associated with sleeplessness. There is impairment of the sense of taste, and the skeletal muscles are very tremulous. The mental symptoms generally come on suddenly during the evening or night. The most prominent symptoms are hallucinations of sight and hearing, associated with confusion and illusions. The visual hallucinations are particularly vivid, and consist of processions of animals, such as rats, serpents, cats, and dogs, often of a red colour, which pass along the walls or ceiling, or sometimes collect upon or round the bed. Or the visions may take the form of demons, goblins, or fairies, which perform fantastic dances or make hideous grimaces. The aural hallucinations are frequently unorganised, and consist of rumbling noises, ringing of bells, scrapings and scratchings in the walls, but they may take the form of screams, yells of pain, and taunting voices. These hallucinations, as a rule, terrify the patient, and may lead to violent strugglings and efforts to escape from the imagined danger. True delusions are not common, the false beliefs being more of the nature of illusions; the patient misinterprets sense impressions, *e.g.* he may imagine the ward



attendants are devils, that the chairs in the room are animals, or that noises in the vicinity are those of people forcing an entrance, etc. The attention is attracted with difficulty, and cannot be held. Speech is rambling and incoherent. Where the memory can be tested past events are sometimes remembered, but there is no memory for recent occurrences. Sleep is always abolished. The most marked physical symptoms are, loss of the desire for food, a dry, glazed tongue, and constipation of the bowels. There is said to be a moderately high leucocytosis. The heart's action is weak, fast, and irregular. The skin is sallow, sometimes jaundiced, and there is a tendency to profuse perspiration. Urine is passed in small quantities, is high coloured, and frequently contains albumen and tube casts. There is often loss of common sensibility to pain and marked tremors of the voluntary muscles.

The acute symptoms may last from a few days to three weeks, but are usually over in three or four days. The first symptom of recovery is the natural return of sleep and desire for food. The subsequent return to mental health is gradual. Death occurs in about 5 per cent of the cases, and is due to exhaustion, hypostatic congestion of the lungs, or epileptic convulsions.

*Treatment.*—It is essential that the patient, from the earliest onset of the symptoms of the disease, should be fed frequently and with small quantities of fluid food. Milk mixed with potash water is probably the simplest and best dietary, and in the very earliest stages of the attack a full dose of chloral and bromide, by producing sleep, may abort the attack. Where the disease has fully developed all the usual hypnotics, such as chloral, opium, sulphonal, and trional, must be used with caution, and only as a last resort. Paraldehyde should be especially avoided, as it frequently increases the excitement. The insomnia is more safely treated by continued hot baths (100° F.), with cold applications to the head, and the use of bromides. Where there is a tendency to collapse and exhaustion, hypodermic injections of strychnine, combined with subcutaneous injections of from 1 to 2 pints of warm normal saline solution, is the best treatment. The patient should



never be allowed out of sight or control of the nurse, as there is often a tendency to suicidal and impulsive acts.

Recovery is as a rule complete, but one attack renders the patient more liable to a second; recurrent attacks usually result in brain damage and moral deterioration.

**Chronic Alcoholic Insanity.**—Chronic alcoholic insanity is a disease characterised by a gradual physical and mental impairment. It occurs with equal frequency both in men and women, and there is always a history of prolonged alcoholic indulgence, but not necessarily of gross intemperance. The onset of the condition is usually gradual, or it may be complicated by attacks of delirium tremens, maniacal excitement, or alcoholic convulsions. The patient has usually a bloated, unhealthy appearance with congestion of the venules of the face. The conjunctivæ may be bloodshot or slightly jaundiced. The expression is heavy, morose, stupid, and lacking in vivacity.

There are evidences of chronic gastric disorders, such as loss of appetite, especially in the morning, with pain and discomfort after taking food. There never is a hyperleucocytosis. Valvular heart lesions and weak peripheral circulation are commonly present. Albumen and sugar are frequently to be found in the urine. There is a tendency to attacks of boils and carbuncles, an evidence of lowered tissue resistance.

Subjective sensations may or may not be present, but hypersensibility to pain is a common symptom. The pupils may be unequal, and their reaction to light and accommodation somewhat impaired, but not lost. The skin and tendon reflexes are in some cases abolished, but in others much exaggerated. The voluntary movements are clumsy and difficult, especially for fine co-ordinated actions, the gait awkward and shuffling, and the muscles—particularly of the tongue, face, and hands—are tremulous. Mentally the impairment is more of a moral than intellectual character. There is no confusion, and no loss of knowledge of position, except in those cases which go on to dementia. Delusions when present are of the nature of morbid suspiciousness of the actions of others, and if the patient is married insane jealousy is a common symptom, and may lead to homicidal attacks or attempts at suicide. Irritability and ungovernable outbursts of rage are the usual



evidences of loss of self-control, and are most frequently indulged in at home and suppressed before strangers. Not unfrequently the patient is obtrusively polite, or foolishly emotional and maudlin with little cause. The affection for near relatives is lost early. There is a tendency to theft, a disregard for truth and honourable dealings both in business and in the actions of everyday life. The personal appearance is neglected, and there is no sense of self-respect. The will-power is deficient, and the capacity for useful occupation is fitful or lost. The memory is not necessarily affected early in the disease, but sooner or later the memory for recent events is impaired, and in those cases which go on to dementia there may be and usually is complete amnesia. The speech may be thick and indistinct, and the handwriting tremulous, with imperfectly formed letters.

Some of these cases develop typical delusional insanity, others are liable to attacks of excitement without confusion, during which they express delusions of a grandiose or boastful nature, and are sometimes difficult to diagnose from general paralysis. Others become demented and suffer from peripheral neuritis, complete amnesia, and pseudo-reminiscence. This later condition is now described as Korsakow's disease. Chronic alcoholics not infrequently suffer from epilepsy.

*Treatment.*—The patient must be removed from the temptation of alcoholic indulgence, and the general health improved by regular hours, exercise which excites mental interest, and the use of such tonics as iron, quinine, strychnine, and arsenic. If mental deterioration is marked treatment does very little good, but if the case is seen early, and the patient is genuinely desirous of helping himself, the use of hypodermic injections of atropine combined with strychnine should always be tried. The exhibition of atropine and strychnine is entirely empirical, but the beneficial results are undoubted in many of the cases so treated.

The method of exhibition is as follows:—The patient, who is usually suffering from gastric irritability and constipation, is put to bed and given a dose of calomel. The following morning an injection of atropine gr.  $\frac{1}{100}$  with strychnine gr.  $\frac{1}{100}$  is given after breakfast, say at 9 o'clock. A similar



injection is given again at 3 o'clock, and a third at 9 o'clock. Combined with the injections it is usual to give cinchona in the form of a mixture.

This mixture should be given before each meal thrice daily. This treatment must be persevered in for at least a month, and the dose of atropine may with advantage be increased to gr.  $\frac{1}{80}$  after the first week. The treatment produces dryness of the mouth and thirst, and the patient should be allowed to satisfy this thirst freely, alcoholic beverages being of course excluded from the liquids so taken. The diet should at first consist largely of milk, certainly so long as there are any symptoms of gastric irritation. Whenever the appetite improves, the patient should be got out of bed and encouraged to take exercise. Mere routine exercise does very little good unless the patient is mentally interested at the same time. The diet should be increased, and consist largely of bread, toast, butter, eggs, rice, fresh vegetables, and fish. Practically no butcher meat should be allowed, and if more nitrogen is required cheese is probably the best substitute. In successful cases the craving for alcohol is lost, and many permanent recoveries are now on record. The diseases called alcoholic delusional insanity and alcoholic mania are simply delusional insanity and mania, in which alcohol has acted as a predisposing factor in the causation of these diseases, and they do not differ in mental or physical symptoms from the diseases elsewhere described as delusional insanity and states of excitement with or without confusion. The sudden, short, violent outbursts of excitement which occur in certain individuals as the result of often quite moderate doses of alcohol, is known as "Mania a potu," and usually occur in those who have suffered from severe head injuries and sunstroke.

**Morphinism.**—The disease known as chronic morphinism is usually confined to the better classes, and is more frequently to be met with in men than women. In the majority of cases the habit is acquired through the use of opium or morphia as an analgesic, but as immunity is established, larger and more frequent doses of the drug are taken to produce the desired effect, until finally as much as 50 or 60 grains of morphia may be taken daily. The victims of the habit are



either of neurotic constitution, or may have fallen into a state of ill health as the result of pain and sleeplessness from disease, or from excessive mental worry and anxiety. The rapidity with which the symptoms develop depends largely upon the resistive power of the patient and the quantities of the drug employed, and may vary from a few months to several years.

The prolonged use of the drug affects primarily the general metabolism. The patient loses weight, and becomes sallow and unhealthy in appearance. The mouth is dry owing to deficient salivary secretion, and this generally leads to thirst; as a result chronic morphinism is often associated with chronic alcoholism. The pulse is slow, irregular in tension and rhythm, and there is a tendency to attacks of syncope and palpitation. The skin is dry and loose, but the patients are also liable to profuse attacks of perspiration. The urine frequently contains albumen. There is loss of sexual appetite combined with impotence, and female patients generally suffer from amenorrhœa. There are usually vague sensory disturbances, chiefly referred to the thoracic and abdominal organs, and there is hyperæsthesia to pain and touch. The pupils are contracted, and their reaction to light and accommodation impaired, while the patient may complain of defects of vision. The superficial and deep reflexes are, as a rule, exaggerated, and the power of the voluntary muscles is much impaired.

Mentally the chief symptom is depression, unless the patient is seen immediately after taking a dose of the drug. Confusion, hallucinations, and delusions are not as a rule present, but there is an impairment of will power, a loss of the moral sense, neglect of the personal appearance, and impairment of the memory. The patient sleeps irregularly, and is frequently troubled by dreams and visions. Some of these patients eventually develop symptoms of delusional insanity which rapidly progresses to dementia.

If the drug is suddenly discontinued the "deprivation" symptoms of morphinism set in, and closely resemble those of delirium tremens. The patient becomes anxious and restless, suffers from hallucinations of sight and hearing, and may



pass into a state of excitement, associated with fear and loss of consciousness. There is complete anorexia, often vomiting, profuse perspiration and salivation, with abdominal pains and diarrhoea. Sensibility to touch and pain is hyperacute over the whole body, and the patient complains of great weakness and discomfort. Death may occur from collapse or syncope. After the acute mental symptoms have passed off these feelings of discomfort and exhaustion persist often for weeks.

*Treatment.*—Treatment consists in depriving the patient of the use of the drug, and this may be done suddenly or gradually. The sudden withdrawal is almost certain to be followed by the symptoms of deprivation mentioned above. In the gradual withdrawal of the drug the quantity which the patient has been in the habit of taking is divided by 2, and given at fixed times during the twenty-four hours for the first two days. The dose can then be reduced to one grain for the twenty-four hours, and this quantity is given for four or five days, when the drug can be finally discontinued. Whichever method is adopted the patient always suffers considerable distress, and in some cases symptoms of collapse, with diarrhoea and delirium, may set in. The patient should be fed at frequent intervals with milk, eggs, meat-juice, egg-flip, etc., and where collapse is threatened diffusible stimulants, such as alcohol, aromatic spirits of ammonia, or hypodermic injections of strychnine, should be employed. The restlessness and discomfort are much allayed by the administration of the bromides.

Another method of treatment advocated by, and highly recommended by MacLeod of Hong-Kong, is to reduce the dose of the drug gradually, and at the same time rapidly bromidise the patient. The bromide is given every four hours, and the dose is increased until the patient is in a state of stupor, when the drug is discontinued. The patient remains in this state of stupor for about a week, and has to be very carefully fed with fluids. The danger of the treatment lies in the fact that as all the reflexes are impaired food may readily pass into the trachea and produce pneumonia.

The after-treatment consists in the use of tonics, cold baths, regular exercise, and constant supervision to prevent



the patient resuming the habit, an occurrence which is, unfortunately, too frequent. No patients are more unsatisfactory to treat than alcoholics and morphomaniacs.

**Cocainism.**—The cocain habit occurs most frequently in those who habitually handle the drug, such as chemists, and the usual methods of self-administration are either by insufflation, by the hypodermic syringe, or by the mouth.

The nervous disturbances are similar to those of chronic alcoholism and morphinism, but there is one hallucination which is peculiar to the cocain habit, the feeling that something is creeping under the skin, and this is particularly referred to the fingers and palms. Obstinate insomnia is almost invariably present. Physically there are disturbances of nutrition, but without gastro-intestinal derangement, so that although the patient suffers from no loss of appetite, weight is steadily lost.

If the habit is complicated by mental disease the symptoms are invariably those of delusional insanity, with delusions of persecution and suspicion.

**Treatment.**—The drug should be gradually withdrawn, and the withdrawal is rarely attended by such acute symptoms or distress as in the case of morphia. The insomnia is best treated by large doses of bromide and paraldehyde, and in some cases warm baths do good. The diet should be simple, given frequently and in small quantities, while tonics—such as iron, quinine, and strychnine—are useful in the later stages. The patient should be constantly supervised to prevent access to the drug. The prognosis is as a rule unfavourable, as these patients readily relapse.

**Chloral Habit.**—The long-continued use of chloral hydrate produces certain well-marked physical and mental symptoms. The patient looks pale and unhealthy, the eyeball has an irritable, inflamed appearance, and the patient is often loquacious, and complains of singing sounds and a feeling of emptiness in the head. Later there are symptoms of depression with gastric derangement, and a failure of muscular power which may go on to paresis.

**Treatment.**—The drug must be discontinued, and a long course of iron, quinine, and arsenic given to raise the general health and reduce anæmia.



**Ether and Chloroform Habit.**—The symptoms arising from the prolonged use of these drugs are those of physical and mental deterioration, and resemble the symptoms of chronic alcoholism. The ether is usually drunk while the chloroform is inhaled. A single dose of chloroform is sometimes followed in neurotic subjects by states of excitement, with hallucinations of hearing, and in others by delusions, as the result of which the patient may make accusations of assault, etc., against those who were concerned with the giving of the anæsthetic.

**Paraldehyde Habit.**—The prolonged use of paraldehyde as a hypnotic produces a habit so that sleep cannot be obtained without recourse to the drug. It increases the tendency to hallucinations of hearing, if such already exist, or it may actually produce vivid auditory hallucinations.

**Mercury and Lead Poisoning.**—The accumulation of the salts of mercury and lead in the system, as the result of chronic poisoning, produces states of mental depression with delusions. Physically there are paresis and tremors of the voluntary muscles, associated with neuralgic pains, and in lead poisoning severe headaches are a common symptom.

**Iodoform Poisoning.**—The use of iodoform as a surgical application occasionally produces a condition of low muttering delirium, with tremors of the voluntary muscles. The cases I have seen were delirious for about three weeks, and made perfect recoveries.

**Carbon-Bisulphide Poisoning.**—Occurs amongst rubber workers, and the symptoms are those of delusional insanity, with vivid hallucinations of hearing, the physical symptoms being marked anæmia and malnutrition. The disease is usually recovered from, but runs a chronic course, and may last for more than a year. The treatment is general: tonics, such as iron and arsenic, being especially indicated. After recovery the patient should not return to the rubber works, as a relapse is almost certain to take place.

**Thyroid Poisoning.**—The prolonged or excessive use of thyroid extract in nervous cases may result in attacks of excitement, without confusion, which are transient in character and pass off when the drug is discontinued.



## CHAPTER XIII

### NERVOUS DISEASES FREQUENTLY COMPLICATED BY MENTAL DISEASE

#### EPILEPSY

EPILEPSY is a nervous disease characterised by a sudden discharge of nervous energy from the grey matter of the cerebral cortex, this discharge occurring without normal stimulus (Gowers).

Under normal conditions the grey matter of the cerebral cortex is constantly producing energy, which is ever at such a tension that it can be suddenly released upon the application of appropriate stimulus. The energy so liberated discharges itself by associated nerve tracts which in a state of health are selected by the will power. The more frequently nervous energy passes by these tracts the more easily is it discharged, and the more readily does it pass, until what were at first complicated co-ordinate movements demanding mental concentration for their accomplishment, become practically reflex actions; in other words, a brain habit has been established. This rule, which applies to all healthy brain action, applies with equal force to diseased action, and once the brain cortex has discharged nerve energy without normal stimuli, a similar discharge is liable to recur again and again. "The recurrence of attacks is one great feature of epilepsy, and the recurrence is in great measure the result of the previous attacks. Every fit is, in part at least, the result of those which have preceded it, and in part a cause of those which follow it. Hence the immediate cause of the first fit must be regarded as the cause

of the disease, although the essential cause is the disposition, that is, the state of nutrition, which makes such a discharge possible" (Gowers).

*Causation.*—Hereditary predisposition to nervous diseases is present in over 50 per cent of the patients suffering from epilepsy.

*Age.*—According to Gowers, one-eighth of the cases begin during the first three years of life. More than one-quarter begin during the first ten years, and a half in the second ten years, that is to say, three-quarters of the cases of epilepsy begin before 20 years of age, and the earlier the commencement of the disease the more marked is the heredity.

The disease affects men and women in about equal proportion.

The exciting causes of the disease may be stated as:—

(1) The result of a brain habit following the occurrence of infantile convulsions.

(2) As the result of cerebral injuries.

(3) As a sequel of scarlet fever.

(4) As the result of sudden mental shocks, such as excessive fear.

(5) As the result of mental anxiety and strain.

*Symptoms.*—Epileptic attacks are divided into the severe fit or *grand mal*, the lesser fit or *petit mal*, and the form commencing with local spasm called "Jacksonian epilepsy," after Dr. Hughlings Jackson who first described the condition.

In the *grand mal* the patient falls unconscious, and the muscles of the body pass into a state of tonic contraction which temporarily arrests respiration, and may cause a forcible evacuation of the bladder contents. The patient becomes blue and livid, and the arterial tension is in a few cases so great as to cause rupture of the superficial capillaries. This state of tonic spasm lasts for about 30 seconds, and is followed by a stage of clonic spasm in which the muscles move in violent jerks; the tongue is often shot out of the mouth and bitten, and the patient foams or froths at the mouth. This stage lasts from one to two minutes, and is succeeded by a stage of exhaustion, with stertorous breathing, which gradually passes into sleep. After recovery from a fit



the patient is generally confused and dull, and may complain of muscular pain and stiffness.

The *petit mal* in its typical form is characterised by a temporary arrest of consciousness without visible muscular spasm, in which the patient may or may not fall to the ground. The forms of both the major and the minor seizures vary enormously in different patients, and even in the same patient. In the seizure of "Jacksonian" epilepsy the muscular spasm commences locally in a hand, foot, finger, or toe, without loss of consciousness, and the spasm may be localised to the part primarily affected. More commonly the spasm tends to spread so as to implicate first one and then both sides of the body, and when the discharge becomes thus general, consciousness is lost. The local spasm is associated with local instability in some portion of the cerebral cortex, which may or may not be due to demonstrable local lesion.

The *grand mal* is commonly preceded by a warning or "aura," which may be a sensory disturbance implicating any of the special senses, or may be visceral, the attack being heralded by some sensation referred to the throat, the heart, or stomach.

Epilepsy, in common with chorea, disseminated sclerosis, paralysis agitans, general paralysis, is liable to be complicated by mental disease, and it is incorrect to use such a term as Epileptic Insanity, as the insanity associated with epilepsy is merely an accident. Many sufferers from epilepsy go through life without developing mental symptoms, beyond being at times unduly irritable, impulsive, passionate, and easily stirred to emotionalism, particularly of a religious nature. If, however, a patient suffering from epilepsy becomes insane, the most common mental complication is excitement, with or without confusion. The predisposing cause of the mental disease is undoubtedly the inherent or acquired brain deficiency which permits of the sudden discharges of nervous energy which constitute epilepsy. Such additional factors as alcoholic excess, mental shocks, and anxiety, also bulk largely as causes of the mental complication. Once an epileptic patient has suffered from the most transient attack of mental disease, there is always a liability to recurrence, and each succeeding attack in-



creases the tendency to recurrence and to the reproduction again and again of the same mental symptoms. The earlier mental attacks are most commonly short, sharp bouts of excitement, from which the patient completely recovers, but after the attacks have recurred there is less and less tendency for the patient to regain complete mental equilibrium, and, finally, the condition passes into a chronic state of mental unsoundness characterised by delusions, hallucinations, loss of self-control, and impulsive actions which may be homicidal or suicidal in nature. The moral sense often becomes impaired, and the epileptic insane are often untruthful and vindictive. Religious emotionalism is a strongly developed characteristic, and is a symptom of unstable brain control, as the patient when most religious is most liable to outbursts of frenzied rage and virulent abuse. The sexual appetite is abnormally active, and may take the form of self-abuse, while many patients are gluttonous and given to alcoholic excesses. There is always more or less mental enfeeblement, which may pass into dementia of a most degraded type.

The mental symptoms may precede or follow the epileptic seizures, or the fit may be represented by a mental explosion. The symptoms preceding a fit may be pure demonstrative excitement, accompanied by much noise, shouting, and singing, or the patient may be morose, irritable, or quarrelsome. Symptoms which follow a fit are always those of excitement, often furious and violent in character. When the fit is represented by a mental outburst the attack is sudden, violent, short, and apparently causeless. Consciousness is always lost, and the patient has no memory of actions committed during the attack. This condition is known as "Larvated Epilepsy." I have seen such sudden mental attacks follow attacks of *petit mal* on several occasions. Insane epileptic patients who suffer from recurrent attacks of acute excitement frequently develop symptoms of descending degeneration in the lateral columns of the cord. I have seen this symptom develop in a few months in some acute cases where the patients suffered very seldom from epileptic attacks, but very frequently from sudden maniacal attacks.

All sufferers from epilepsy, both sane and insane, are liable to what is called Automatism, which usually follows a fit. The



patient may simply lose consciousness for a time, but proceed with his work as if nothing had happened, but on recovery there remains a distinct blank in his memory for which he is unable to account. Or the patient may unconsciously perform some unusual action of which he is afterwards quite unconscious. One patient after each attack of *grand mal* used to get up and run round the ward, turning each door-handle in succession. He always followed the same course, and always ended in the same place, after which he lay down and went to sleep. If interfered with in the automatic action he became violent and dangerous. Another patient had a habit of walking slowly into the middle of the ward, where he first micturated, and then undressed himself and folded his clothes neatly on the floor. Consciousness invariably returned when he had completed this action, and he then looked surprised and confused, proceeded to re-dress, and sat down on a seat.

Another patient in the post-epileptic state had, after a fit, to be placed in seclusion, as he invariably assaulted the first person he encountered immediately the seizure had passed off; and undoubtedly many criminal actions may be committed during the state of epileptic automatism. Clouston's Convulsive Melancholia is a condition of excited melancholia which begins as such, and becomes complicated by epilepsy.

*Treatment.*—The treatment of epilepsy and the complicating insanity should be regarded from different standpoints, although the treatment which benefits the explosive tendency has an undoubted effect upon the character and recurrence of the mental complications.

The diet should be simple, fattening, and non-stimulating, and moderate in quantity. Animal food, tea, coffee, and tobacco should be either withheld or given very sparingly. The bowels should be relieved at intervals by saline purgatives, and the patient encouraged to take large quantities of water. Medicinally the only drugs of any benefit are the bromides, particularly bromide of potassium. The usual dose of the bromides is 30 grs. given thrice daily after food. This treatment should be continued without intermission for long periods.



Richet and Toulouse have introduced a very ingenious modification of the bromide treatment, based upon the theory that privation of chlorides produces increased attraction of the cells for similar salts—bromides—and therefore intensifies their action. About 14 grms. (210 grs.) of chloride of sodium are consumed daily by normal persons. Richet and Toulouse reduced the quantity of chloride of sodium consumed by their patients to 2 grms. (30 grains) per day. The diet they used was as follows:—Milk, 35 oz.; butcher meat, 10 oz.; potatoes, 10 oz.; flour, 7 oz.; sugar  $1\frac{3}{4}$  oz.; coffee, 154 grs.; butter,  $1\frac{1}{2}$  oz.

Bromide of sodium in daily doses of about 50 grs. was given to nine patients. On ordinary diet an average of 9.54 fits occurred in 10 days, but in the case of the patients on the diet described the average fell to 1.16, a diminution of 87 per cent. The value of the treatment was confirmed by the fact that the attacks returned when ordinary diet was resumed, although the bromide treatment was continued. Dr. Toulouse thinks that possibly good results may be obtained in a simpler manner—by removing salt from the ordinary dietary, and taking into account the quantity contained in the bread.

It is useful to combine with each dose of the bromides 2-5 mins. of liquor arsenicalis, or 2-5 grs. of salol, which tend to diminish the symptoms of bromism, the most characteristic of which are acneous eruptions on the forehead, face, and back, alimentary disorders, and mental lethargy. When the symptoms of bromism are troublesome, biborate of soda, in doses of from 5 to 10 grs. thrice daily, combined with 5 mins. of the liquor arsenicalis, may be tried, and is effective in some cases. Tincture of opium, belladonna, Indian hemp, nitroglycerine, and erythrol tetranitrate are all useful, particularly in the treatment of minor attacks in young persons. When a distinct sensory aura occurs in an extremity the subsequent fit may be arrested by tying a ligature tightly round the limb. Quite recently Ceni has adopted the device of withdrawing blood from the vein of one epileptic by means of an exploring needle, and injecting the serum so obtained either into the same or another patient; his theory being that some antibody



is formed in the serum after withdrawal from the body. My assistant, Dr. Peebles, tried this method in six cases. One patient distinctly benefited, but immediately the treatment was discontinued the seizures returned. Another patient, who at first acted as a serum producer, was free from fits during the time he was bled, which lasted for some two months. As soon as bleeding was discontinued he again suffered from seizures, which in his case occurred daily and nightly. The same patient, upon being injected with serum taken from another patient, was not in the least benefited. The remaining four cases were not affected either by injections or bleeding. The maniacal and impulsive mental attacks are best treated by quiet and rest in bed. If excitement comes on slowly and culminates in an acute mental attack, either with or without a fit, the administration of a minim of croton oil, or 20 grs. of calomel, frequently arrests the attack; and this treatment is useful when the patient is robust, given to over-eating, and only suffers from occasional attacks. Where the mental symptoms follow a fit or succession of fits, a full dose of chloral and bromide, by producing sleep, may also arrest the attack.

The status epilepticus is a condition of toxicity plus exhaustion which follows a succession of fits. At first each seizure is distinct from the one which follows it, but as the patient becomes exhausted the fits tend to run into one another, and the patient passes into a condition of deep unconsciousness with stertorous breathing. The skin is bathed in perspiration, the pupils are dilated, and do not react to light, urine and fæces are passed involuntarily, and the temperature is pyretic. Death frequently follows with hypostatic congestion of the lungs. I have never seen any treatment benefit this complication. The accidents to which epileptic patients are particularly liable are, injuries received in the fall which follows the loss of consciousness, choking which may occur if the patient takes a fit during the course of a meal, and suffocation, resulting from the patient turning over upon the face in the period of unconsciousness succeeding a fit.

*Pathology of Epilepsy.*—The general physical symptoms of patients suffering from epilepsy, independently of mental





CHART No. 6.

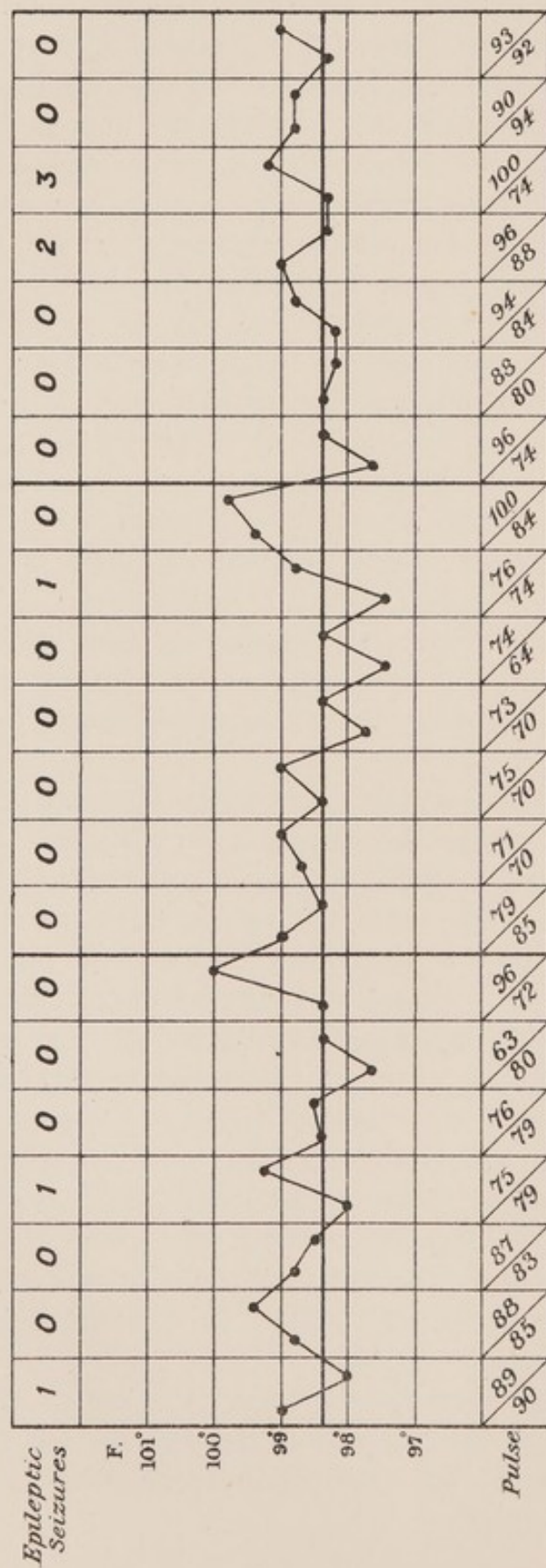


CHART showing the Pulse and Temperature in a case of Epilepsy.

complications, suggest that the disease is toxic in origin. The temperature of epileptic patients is irregular, and liable to become febrile at intervals, independently of any outward bodily or mental symptoms. The pulse is liable to the same irregularities, and may be unduly fast or slow, irregular in force and rhythm, and also intermittent. The leucocytosis is frequently raised, and an increase occurs in the polymorphonuclear cells, but the epileptics whose blood I have examined were all insane, and it is probable that the toxines producing the mental complications were the cause of the hyperleucocytosis in these cases. Several foreign workers have recorded that the urine of epileptic patients withdrawn during a fit, if injected into the venous circulation of animals, will produce convulsions. Haig regards the disease as due to uric acid poisoning, and has demonstrated the fact, which has been confirmed by other workers, that the excretion of uric acid in the urine falls prior to a fit, and there is a rise of excretion after the fit. Krainsky has injected defibrinated blood, withdrawn from epileptic patients during the course of a fit, into the venous circulation of rabbits, and true epileptic fits resulted, and he regards the toxic agent as carbamic acid and ammonia, both of which are present in excess in the blood of epileptic patients before, during, and after an epileptic attack.

No known macroscopic or microscopic changes in the nervous tissues are characteristic of epilepsy with insanity. The cranial bones are frequently thickened over the vault, and this thickening is sometimes associated with the development of large irregular bosses of bone, or of small excrescences on the inner aspect; the arachnoid may be thickened or opaque; there is often felting of the subpial layer of the neuroglia, and the nerve cells may show some chromatolysis—all changes which are common to long-continued insanity.

G. A., male, aged 18, was admitted, suffering from acute excitement. His father was a confirmed drunkard, and the patient developed epilepsy at the age of 14 years. The patient was a steady and industrious worker until a week ago, when after an epileptic seizure he became confused, and then excited and violent.

On admission he was a well-made and well-nourished lad.



His appetite was good, and he was inclined to eat too much. The urine contained albumen and a heavy deposit of urates. The skin and tendon reflexes were exaggerated. Mentally he was slightly confused, irritable, and complaining, and his memory was impaired.

Rest in bed, a saline purge, and a moderate dietary, with 30 grs. of potassium bromide thrice daily, improved his mental and physical condition enormously. Within a fortnight he was working in the garden. Three weeks later he again became querulous and quarrelsome, and after having quarrelled with either an attendant or fellow-patient, he was unscrupulously vindictive, trumping up all sorts of evil reports and charges against these individuals; and as he was constantly interfering with the affairs of others, he was always in trouble. Between his bouts of quarrelling he devoted himself to reading his Bible, and occasionally singing hymns and psalms with great fervour. During this irritable stage he affected the more soothing forms of sacred music, such as "Lead Kindly Light," but as the excitement increased he much preferred something with a bit of go in it, his favourites being "Onward Christian Soldiers" and "Hold the Fort." During this stage his pulse became fast, and his temperature frequently rose in the evenings to 99° F., or even higher. Sometimes the excitement stopped here, but most frequently a maniacal attack, lasting for three or four days, was the sequel to each attack of irritability. The epileptic seizures were of the nature of *grand mal*, preceded by what he called "sensations," occurring most frequently when the patient was having two or three epileptic attacks during the day. These sensations were probably of the nature of sensory discharges, and some of them appeared to be attacks of *petit mal*, as the patient became confused and sometimes passed into a state of automatism. Sometimes the epilepsy heralded a mental attack; sometimes the mental attack took the place of the epileptic seizures, but in whatever way the mental attack commenced, coincidently there were evidences of toxæmia, the pulse became fast, the temperature febrile, and the leucocytosis rose often to 30,000 and 40,000 per c.mm. of blood, while the polymorphonuclear percentage was always above 70. After the bout of excitement the patient was quite clear as to all that had passed during his attack, with the exception of the period when he was acutely excited. On two occasions, instead of having maniacal attacks, the patient passed into stupor with catalepsy and loss of consciousness, which lasted for about a week. During the stuporose period there was no leucocytosis and no signs of toxæmia, the temperature and pulse being below normal. After recovery from a stuporose attack the patient was irritable and uncomfortable, until either a series of fits or a maniacal



attack came on, after which he passed again into comparative sanity.

Treatment with the bromides often combined with liquor arsenicalis reduced the frequency of the maniacal attacks, but the patient became more enfeebled mentally, and there was a stiffness and awkwardness in his gait.

J. Mc., male, aged 23, was admitted with the diagnosis that he was suffering from cerebral tumour. No history was obtainable beyond the fact that the patient had made a suicidal attempt, and was thus brought under notice. He was a weak, nervous-looking lad, with ptosis of the left eyelid. Beyond the fact that his digestion was feeble, and that he had a hyperleucocytosis, no physical symptoms were to be noticed. There was no impairment of sensibility, and the special senses were all acute. The fundus of the left eye seemed to be slightly congested. The gait was slightly dragging. The superficial and deep reflexes were exaggerated, and there was slurring of speech.

Mentally he was confused and dull, and he gave the impression of suffering from brain exhaustion.

A few days after admission he brightened up and talked freely about himself, and there can be no doubt that he had no memory of his attempt at suicide. A little later he became restless and incoherent, and then impulsive. He twice sprang out of bed and pushed his head through a pane of glass before he could be restrained, and his dragging gait entirely disappeared when he was making one of his impulsive rushes. This impulsive stage lasted for four weeks, and terminated with an attack of asthma. Following the asthma he was fairly quiet, but confused, for the next two weeks, and still suffered from ptosis of the left eye, slurring of speech, and dragging gait. Then he made another impulsive suicidal attempt, and was under constant supervision day and night for a month, and during this attack he had two epileptic seizures, and the acute symptoms terminated with a second asthmatic attack. The patient was then transferred to a neighbouring asylum, where he made an excellent recovery under ordinary treatment with potassium bromide.

J. M., a male, aged 52, was admitted in a state of wild mania. His face was flushed, the eyes congested, and his tongue and lips were so dry that he could hardly articulate. He appeared to be quite unconscious of his surroundings, and often struck out savagely at anyone standing near him. His history was that he was found by two policemen in this maniacal state on the public road, and nothing further was known about him. The morning after admission he was quite sane, but slightly confused. He told me



that he was an ex-policeman, and that he had the previous day been walking from Dundee to Perth on the lookout for work, and that he remembered reaching a certain point in the road, but from that time until he found himself in the hospital ward at Murthly he had no memory. That evening his pulse was 101, and the temperature rose to 99.4° F., and he looked flushed and the eyes were congested. Suddenly, at 11 o'clock at night, he again passed into very acute mania, and required an injection of hyoscine to control him, but in the morning he was again apparently sane. He denied ever having had epilepsy, and thought his illness was due to his having been short of food and in straitened circumstances for some weeks. He suffered from several of these short, violent, and sudden outbursts during his first month in the asylum, and then one night he had a series of three epileptic fits. He was then put on 30 grs. of potassium bromide thrice daily, and for the remainder of his residence he was free from the sudden mental outbursts, but he was irritable and quarrelsome and discontented. He was at Murthly for eight months, and had during that time only five epileptic seizures, but within six months of admission he presented most marked spastic gait and symptoms of descending degeneration in the crossed pyramidal tracts.

## CHAPTER XIV

### NERVOUS DISEASES FREQUENTLY COMPLICATED BY MENTAL DISEASE—*continued*

#### GENERAL PARALYSIS; DIPSOMANIA

GENERAL Paralysis is a disease characterised by symptoms of progressive degeneration of the central nervous system, more particularly of the motor centres, in which remissions and arrest of progress of the symptoms are common and characteristic, which is liable to be complicated by epileptiform seizures commonly spoken of as congestive attacks, and which is always associated with progressive mental enfeeblement. The disease is almost invariably fatal, the patient either dying as the result of congestive seizures, or from intercurrent diseases such as pneumonia or phthisis, or as the result of extreme emaciation and complete paralysis. Apparent recoveries do very occasionally occur, though this is denied by the majority of alienists. Patients suffering from general paralysis are liable to attacks of mental disease, which may be of any known form of insanity—excitement with or without confusion, melancholia, delusional states, katatonia, and folie circulaire, and it cannot be too strongly stated that any such mental affection constitutes an additional disease superimposed on the primary one. The general paralytic who suffers from excitement, with or without confusion, is suffering from a double toxæmia: firstly, the toxæmia which causes the neurosis; and, secondly, the toxæmia which causes the excitement. The mental symptoms may come on so early in the disease as to suggest that the condition commenced as insanity, and that



the nervous disease developed as a sequel, whereas the mental symptoms have only masked the physical symptoms of general paralysis and led to a mistaken diagnosis. So rarely does the disease occur uncomplicated by mental symptoms, and so frequently does it, apparently, commence with mental symptoms, which are described by most alienists as characteristic of general paralysis, that it is an accepted belief that general paralysis is a mental disease, and the failure to realise the fact that the nervous disease and the insanity are two separate conditions, due to different causes, and only accidentally associated, has led to confusion in description and arrest in the advance of knowledge.

Two cases demonstrating these facts may as well be stated here :—

CASE A.—A male, aged 42, was admitted in a state of acute excitement. The physical and mental symptoms suggested excitement with confusion, but there were in addition pupillary changes which suggested general paralysis. The patient had been working up to a month prior to admission, and a change in facial expression and in character were the first symptoms noticed in his illness. On examination it was found that he had a leucocytosis of 20,000 with a polymorphonuclear percentage of 70. Now, in the first stage of uncomplicated general paralysis the leucocytosis is rarely above 12,000 or 13,000. Further, the serum contained an agglutinine to the organism isolated from the case of excitement mentioned in a previous chapter; while the serum of uncomplicated cases of general paralysis does not contain this agglutinine. I injected subcutaneously into this patient .5 c.c. of streptococcus pyogenes vaccine. In response to the injection the leucocytosis rose to 40,000 per c.mm. and the polymorphonuclear percentage to 85. Coincidentally the excitement subsided and never returned. The patient now presented well-marked physical symptoms of general paralysis, passed quietly through the various stages of the disease, and died two years later without any further mental symptoms than enfeeblement and gradual loss of memory.

CASE B.—A female, aged 40, was transferred to Murthly from another asylum. She was diagnosed as a case of "acute mania," and the general paralytic symptoms had been missed because they were masked by the excitement. Here again the leucocytosis was abnormally high for general paralysis, and the agglutinine so common in the serum of cases of excitement was also present. An injection of .5 c.c. of streptococcus pyogenes vaccine raised the



leucocytosis and arrested the mania, and the general paralytic symptoms were then self evident. This patient had several recurrent attacks of excitement, and died early of pneumonia. The diagnosis of general paralysis was verified *post-mortem*. In both these cases artificial stimulation of the leucocytosis arrested the mania, but did not affect the course of the graver and underlying condition, general paralysis.

*Causation.*—General paralysis is essentially a disease associated with the artificial and strenuous life of cities, as it is practically unknown in country districts, and attacks men much more frequently than women. It may occur in adolescence, adult life, or old age, but by far the majority of cases affected are in the prime of life, between the ages of thirty-five and forty-five. Hereditary predisposition to insanity is said to occur in only 10 per cent of the cases, and sufferers from the disease are as a class well developed and vigorous in both mind and body. Acquired syphilis is regarded by many as the exciting cause, by others as only a predisposing cause; but whether it be the exciting or the predisposing cause of the disease, there can be no doubt that between 70 and 90 per cent of general paralytics have suffered from syphilis. Syphilis of itself, however, is not generally believed to be a direct cause; it apparently requires some additional factor, such as alcoholism, sexual excesses, great bodily or mental stress, worry, influenza, lead poisoning, or head injury, to permit of the syphilitic or parasyphilitic toxine starting the process of degeneration known as general paralysis. None of these causes by themselves appear to be capable of originating the disease unless the patient is predisposed by syphilitic infection. Every now and then, however, cases occur in which syphilis can be excluded, and in which no satisfactory cause for the disease can be advanced, and such cases suggest that there must be some other common exciting cause not yet demonstrated, and that syphilis and the other above-named factors only strongly predispose to the disease. The statement that general paralytics can be reinfected with syphilis is against the hypothesis that syphilis is an exciting cause. The course and symptoms of general paralysis suggest a toxæmia. This is generally admitted, and the intoxication



is regarded as an autointoxication of syphilitic or parasymphilitic origin. There is, however, a certain amount of clinical, experimental, and pathological evidence which supports the contention that the toxæmia is of bacterial origin, and that syphilis, alcoholism, worry, etc., are only active in so far that they break down the resistance of the patient and allow of bacterial attack.

The clinical evidence is, firstly, that the temperature is characteristic of a toxæmia. In the first stage of general paralysis uncomplicated by acute mental disturbance the temperature is irregular, the evening temperature often rising to 99° or 100° F., and falling in the morning. Sometimes the swing of the temperature is decidedly remittent in character. In the second stage the febrile temperature is recurrent every two, three, or four weeks, and in the last stage very irregular. If the general paralysis be complicated by acute mental disease the temperature is not characteristic; it is apt to be higher in the first stage and subnormal in the second, more particularly if the mental complication has been acute excitement. Secondly, disturbances of the alimentary system, which often appear early in the disease, and are characterised by attacks of diarrhœa, jaundice, biliousness, and capricious appetite, are suggestive of a subacute toxæmia. Thirdly, the leucocytosis is irregular in the first stage, with a high polymorphonuclear percentage, regularly recurrent in the second, corresponding to the rises of temperature, etc., very irregular in the last stages, and characterised by a low polymorphonuclear percentage. It is thus typical of a bacterial infection which gradually exhausts the leucocyte-producing power of the bone marrow. The blood serum also is deficient in some substance present in the serum of healthy persons, which is capable of agglutinating certain strains of *staphylococcus aureus pyogenes*, and it occasionally contains an agglutinine to the diphtheroid organism of Ford Robertson, M'Rae, and Jeffrey. Further, the frequency with which general paralytics become affected with tubercle indicates a low bacterial resistive power. Lastly, the steady advance of the physical symptoms, the tremor of the muscles, the frequency with which the terminations of the sensory and



motor nerves are implicated early in the disease, all point to some diffusible toxine carried by the blood stream, which is capable of attacking the nervous system either centrally or at its periphery.

The experimental evidence consists in the fact that the serum of a case of general paralysis in a state of remission when injected into an early progressive case arrests the disease. I have seen this occur four times within the last five years, and three of these patients are now earning their own livelihood. This result can only be explained on the supposition that the serum of some cases while in a stage of remission contains an immune body. Further, severe attacks of erysipelas, carbuncle, pneumonia, or abscesses induced by the subcutaneous injection of terebene, will often arrest indefinitely the progress of the physical symptoms. I actively immunised one patient to the diphtheroid organism of Ford Robertson, M'Rae, and Jeffrey, and completely arrested the progress of the disease, the patient being finally discharged, but he died six months later of phthisis.

Ford Robertson, by feeding rats with a growth of the diphtheroid organism, has produced a fatal disease which in some respects is allied to the symptoms of general paralysis in man. More recently I infected a goat with the same organism. Broth cultures of the organism were injected subcutaneously, but the animal by licking the point of injection infected itself through the alimentary tract. Months after the last injection the animal became paretic in gait and rapidly emaciated. Finally, it suffered from two seizures very similar in appearance to congestive seizures, and died in a third. The diphtheroid bacillus was reobtained from a culture made from the œsophagus, and the pathological changes in the brain were very similar to those found in cases of general paralysis in man.

The pathological evidence which points to a toxine circulating in the blood as the direct cause of the disease is the fact first recorded by Angiolella, and more recently by Ford Robertson, that the earliest pathological changes commence in the vascular system, and it is now known that these lesions are not confined to the vessels of the nervous



system, but attack equally non-nervous organs. The frequency with which the diphtheroid bacillus has been isolated by Ford Robertson, M'Rae, and Jeffrey from the bronchi, tonsils, œsophagus, and intestines of cases dying from general paralysis, is of interest when taken along with the results of the above experiments on animals. Whatever may be the source of toxine in the early stages of the disease, there is no doubt that in the terminal stages the toxæmia is that of a mixed infection, probably gaining access to the body through ulcerated patches in the intestinal tract.

The disease may attack the nervous system either centrally or peripherally, and quite a number of cases occur accompanied by *tabes dorsalis*, the diseased process here attacking the cord before the brain. For purposes of description general paralysis is divided into four stages—the prodromal, the first stage, the second stage, and the third stage. There is no sharp line of demarcation, however, between these stages, the one passing gradually into the other.

*Prodromata.*—General paralysis may commence without warning, but often there are more or less definite symptoms which precede the actual development of the physical symptoms characteristic of the disease. The patient changes in habits, becomes either restless and unusually energetic, or torpid and sluggish. Alcoholic and sexual excesses are common symptoms, and are wrongly regarded as exciting causes. There is often a distinct history of attacks of biliousness, diarrhoea, or constipation, with fitful appetite, vague neuralgic pains, and numbness in the extremities, sometimes associated with sudden and transient changes in vision, hearing, taste, and smell. Passing attacks of aphasia, paresis of the ocular muscles causing double vision, paralysis of a hand, arm, or lower limb, are common and very suggestive of the disease. One patient of mine had transient paretic attacks without loss of consciousness for seven years prior to the onset of the characteristic symptoms, and whenever such symptoms occur they should suggest general paralysis.

The mental symptoms are a general impairment of the more specialised powers of the brain, a loss of memory and of the power of attention, associated with impairment of judgment





CHART No. 7.

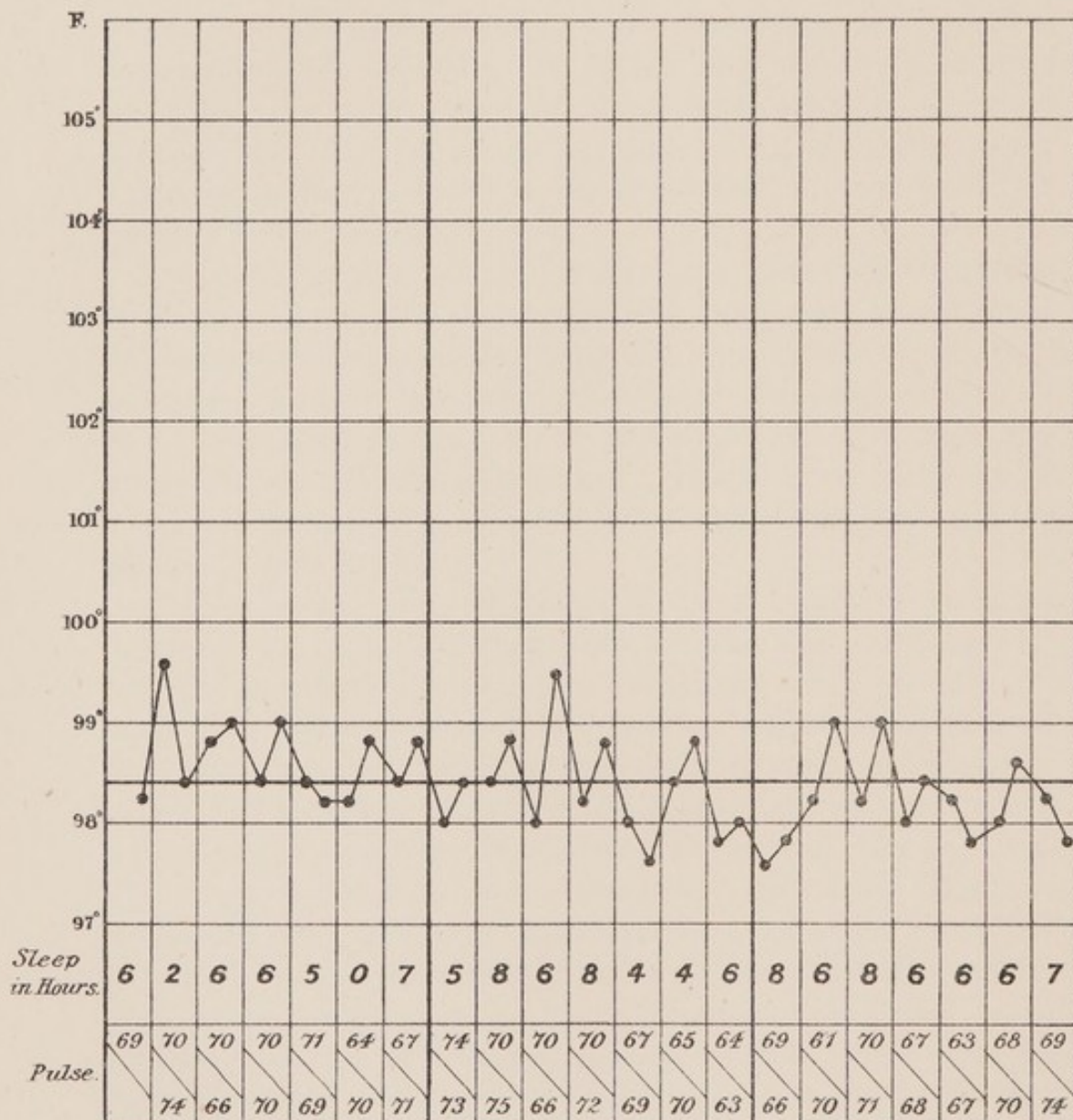


CHART showing the Temperature and Pulse-Rate in a case of General Paralysis towards the end of the first stage of the disease.

and will power, the patient being easily led into foolish and absurd actions and speculations. There is generally loss of self-control, showing itself in irritability or violent temper, emotionalism, a loss of the sense of honour, honesty, and morality, which may lead the patient to commit theft or more criminal offences. Attacks of cardiac failure and mild congestive attacks, generally mistaken for epileptic seizures, may occur very early in the disease.

More commonly general paralysis begins suddenly with marked mental or motor symptoms, the mental symptoms being generally those of excitement or depression, and the motor being evidenced by a congestive attack or some form of paresis. Whatever the manner of the onset the physical symptoms are soon evident, and the nature of the disease recognisable. As general paralysis is a nervous and not a mental disease, the physical symptoms of the various stages are described by themselves.

*First Stage.*—In the first stage there is generally some flattening of the facial muscles so that the expression alters. The temperature is commonly 99° F., or even higher in the evening. Sometimes there are distinct gastric symptoms associated with obstinate constipation, but gastric symptoms may be wanting, and the patient apparently enjoys good health so far as the appetite is concerned. The leucocytosis is irregular, and the polymorphonuclear percentage is frequently above 70. The heart's action is quick and irritable, and the arterial tension is high. The urinary excretion may be excessive, but as a rule little abnormality is to be noted. In males the sexual instincts are commonly very active or in abeyance. Sensory disturbances are not characteristic of the first stage, but a diminution of sensibility to pain is present in about 50 per cent of the cases. The senses of sight, hearing, taste, or smell may present abnormalities, but they are not usually detected so early in the disease. The pupils are generally unequal in size, and frequently irregular in outline, being oval or somewhat sinuous. The inequality of the pupils varies from day to day, the right pupil being one day larger than the left, and the next day *vice versa*. The reaction of the pupils may be defective to both light and

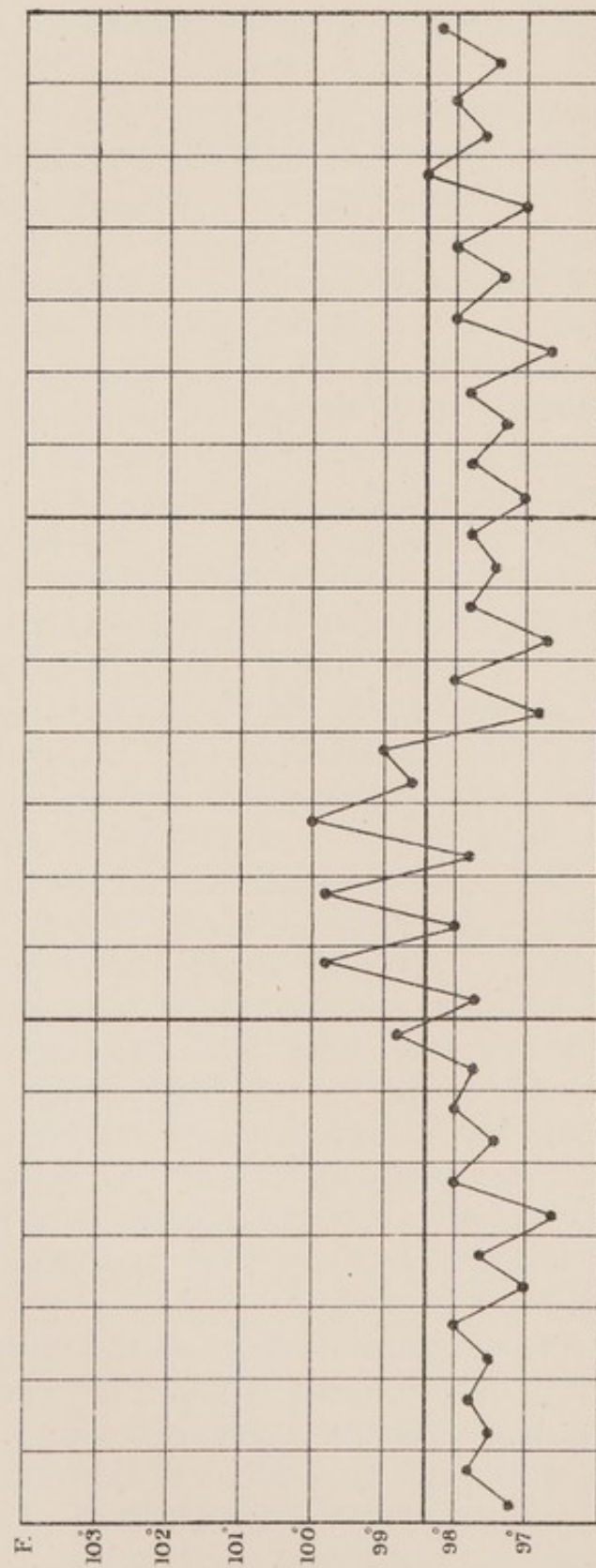


accommodation, or they may present the Argyll Robertson symptom, in which the reaction to light is lost, but the reaction to accommodation is present. The pupils may be contracted to pin points, or they may be widely dilated. Sometimes one pupil only is deficient in reaction, while its fellow may react to both light and accommodation. The pupillary symptoms vary so enormously in different cases, that it is only possible to say generally that inequality in size, irregularity in outline, and some defect of reaction to either light or accommodation, are common symptoms of general paralysis. The organic reflexes are rarely affected. The skin reflexes are exaggerated, and the tendon reflexes are markedly increased, except in cases complicated by chronic alcoholic poisoning or locomotor ataxia. The power of voluntary movement is always impaired. This impairment of muscular function is not confined to any one group of muscles, but is general throughout the body. It is most marked in the muscles of the face, tongue, hands, and lower limbs. The impairment of the facial muscles shows itself in loss of expression, the lines and folds of the face being rounded or flattened. Paresis of the tongue and lips leads to difficulty in speech; sometimes the speech is ataxic, more commonly it is slow, monotonous, and paretic, with slurring of the labials. Certain words are pronounced with difficulty, such as "hippopotamus," "Royal Artillery," "West Register Street." The movements of the tongue are often inco-ordinate, and the tongue, lips, and facial muscles show well-marked tremors, particularly if the patient be excited, angry, or fatigued. The muscular power of the arms and hands may be impaired, and the finer co-ordinated movements, such as writing, are commonly affected. The writing may be shaky and lacking in power, words or syllables are omitted, and the successive lines of a written page diverge or converge in an irregular manner. The muscles of the forearm, hands, and fingers are always tremulous, particularly after exertion. The gait is sometimes dragging, sometimes spastic, but the patient always has difficulty in walking along a straight line, in placing one foot in front of the other, and particularly in turning, when the gait is noticed to be lurching or swaying. Many patients





CHART No. 8.



cannot stand with the heels together and the eyes shut, and very few can raise themselves on the tiptoes with the eyes shut. Myotatic irritability of the muscles is very commonly present. During the first stage the patient loses weight, and the function of sleep is deficient.

*Second Stage.*—In the second stage of the disease the facial muscles become further involved, the face is expressionless even when the patient is pleased or angry, and the skin often has a pasty, puffy look suggestive of kidney mischief. The temperature chart, if the case is an uncomplicated one, shows mild recurring febrile attacks every two, three, or four weeks. The appetite is capricious, excessive, sometimes perverted, the breath is offensive, the tongue often furred, and particularly towards the end of this stage attacks of diarrhoea alternating with constipation are not uncommon. The leucocytosis follows the curve of the temperature chart, but the polymorphonuclear percentage rarely exceeds 70. The heart's action is slow and the pulse full. The arterial tension is high, and attacks of cardiac failure may occur especially after meals, or while taking exercise. The skin is unhealthy in appearance, greasy and œdematous, and the hair is dry and harsh. Urine is often excreted in considerable quantities, and the excretion of urea and chlorides may be increased. There may be loss of sexual appetite and impotence. There is now distinct impairment of common sensibility especially to heat and pain. Any of the special senses may be affected. Homonymous hemianopsia is common after congestion seizures, but is frequently unobserved. The sense of taste is said to be impaired. I have seen patients drink quinine and water under the impression that the fluid was whisky, but such cases are rare. The sense of smell to pungent substances, such as ammonia, is certainly impaired very frequently. The organic reflexes, especially of micturition, may now become affected, the patient suffering from incontinence or retention of urine. All the muscles affected in the first stage show advancing paralysis, the tongue is flabby, and through lying heavily in the mouth becomes indented by the teeth, and the movements of the tongue are more ataxic and inco-ordinate. The lips are pendulous and exhibit well-marked tremors during

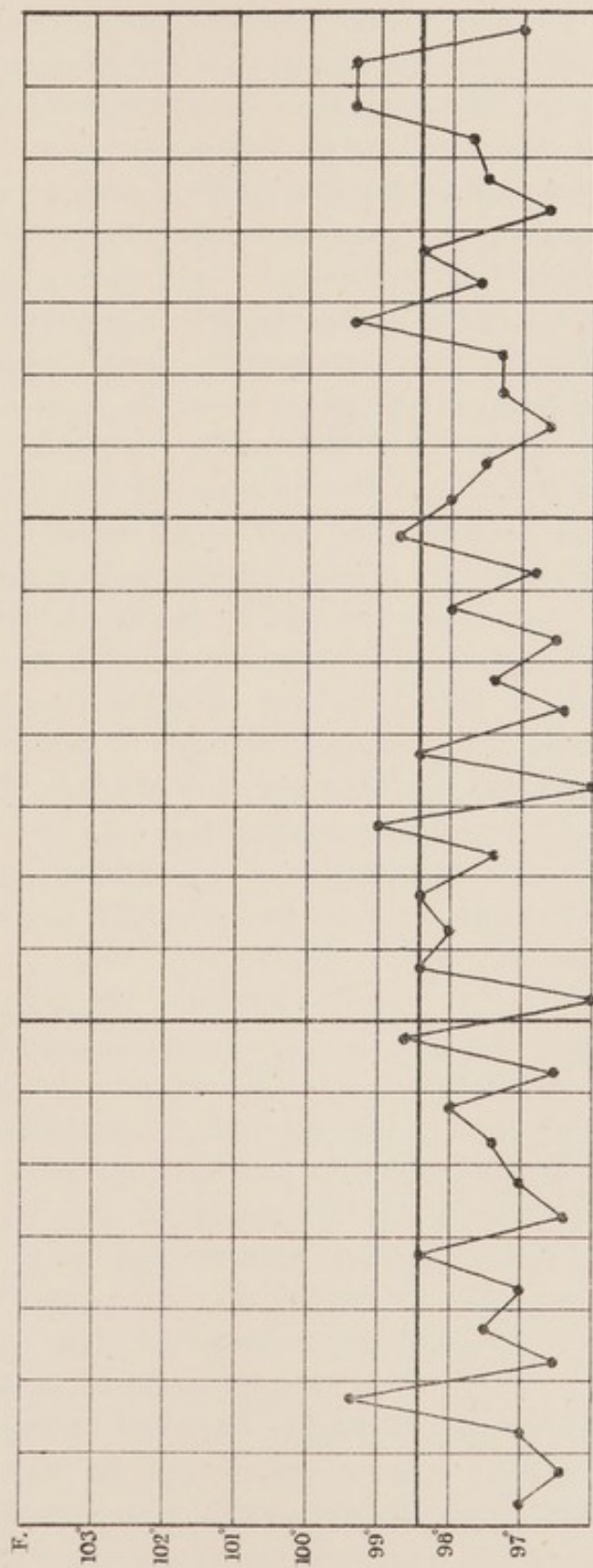


speech, and these tremors spread all over the facial muscles; speech is more difficult, slow, slurred, and paretic, while the tone of the voice is altered through paresis of the laryngeal muscles. The writing is more tremulous, and the character of the handwriting alters, the letters and words being often written in a large, round, childish hand. There is distinct weakness of muscular power, with inco-ordination in both the upper and lower limbs, in some cases more pronounced on one side, in others confined to one limb. The gait is uncertain, often swaying, and the patient walks with a broad base; turning is always associated with marked efforts to retain the balance, and when moving, the patient often falls forward upon the face. During the second stage the patient gains weight, but the nutrition of the body is not that of health, the tissues being loose and flabby.

*Third Stage.*—In the third stage the progressive nature of the paralysis is most apparent, and is associated with rapid emaciation and muscular wasting.

The face is thin, drawn, and expressionless. The temperature is irregularly febrile. The appetite is often good, but there is a distinct tendency to diarrhoea and vomiting. The leucocytosis becomes very irregular, independently of the changes in the body temperature, and the polymorphonuclear percentage falls as low as 30 and 40. The heart's action is feeble, and the pulse irregular in rhythm and force. Cyanosis and oedema of the extremities are common. The lungs are apt to become infected with tubercle. The skin is yellow and parchment-like, and there is a tendency to the formation of boils, carbuncles, and bed-sores. Boils and carbuncles, in fact, may occur early in the second stage. The urine presents no special abnormality. Common sensibility is almost lost. Sight, hearing, taste, and smell are all more or less affected. The organic reflex of swallowing, though sometimes affected in the second stage, is now generally implicated, and choking attacks may be of frequent occurrence during the taking of food. This complication is due in part to loss of sensibility in the pharynx, and in part to paralysis of the muscles. The urine and faeces are commonly voided without consciousness, and the patient is kept clean and dry with difficulty.

CHART No. 9.



TEMPERATURE CHART in the 3rd stage of a case of General Paralysis showing the irregular Temperature.





Paralysis of the muscles may advance so far that movement is difficult and often impossible. The power of speech is often abolished, the patient cannot perform the most simple actions such as feeding himself, and any effort of any kind is attended by exhaustion. All muscular movements are inco-ordinate and associated with marked tremors; walking becomes an impossibility, and unless the disease is terminated by accident, congestive seizures, or pneumonia, the patient dies from exhaustion. Emaciation and muscular wasting are marked features of the last stages of the disease.

*Complications.*—Hæmatoma auris, though not confined to any one form of insanity, is particularly liable to occur in general paralysis. The condition arises sometimes as the result of direct violence, but often no cause can be traced.

The tissues about the helix become inflamed and livid, and a fluctuating swelling forms, which may be circumscribed or involve the whole of the ear cartilage. The swelling may increase rapidly to the size of a hen's egg and then rupture, or it may gradually resolve before a distinct fluctuating tumour forms. Whether rupture takes place or not the ear gradually shrivels, and the cartilage becomes deformed. The contents of the cyst consist of altered blood-clot, which is sometimes gelatinous in appearance. The treatment for the condition is blistering as soon as signs of inflammation are detected. The cyst should never be opened unless the contents become purulent.

*Fractures.*—The bones in general paralysis undergo a degenerative change whereby the elasticity is impaired, and the bones become brittle and liable to fracture. The cartilages, especially of the ribs and sternum, become ossified, and this, associated with brittleness in the ribs themselves, leads more frequently to their fracture than any other bones in the body. At the same time the power of repair of such injuries is vigorous, and fractures unite just as readily in general paralysis as in healthy persons.

*Congestive Seizures.*—Epileptiform or apoplectiform seizures may occur at any stage of the disease. They may be the first and only symptoms, the true condition being only recognised *post-mortem*, or they may precede the other physical



symptoms for years, or may so aggravate them that the nature of the disease is only recognised after a severe congestive attack, or they may be delayed and occur during the later stages of the disease, and then frequently terminate life. In their simplest form they may simulate attacks of cardiac failure or attacks of *petit mal*, or, again, they may simulate true epilepsy. The type of seizure in general paralysis is the apoplectiform or congestive attack. The attack is sometimes preceded by restlessness and excitement, or a rise of temperature, and may come on gradually or suddenly. Consciousness is generally lost, and the symptoms take the form of chronic convulsions often restricted to the head, a limb, or one side of the body. The convulsions may last for several hours or even days, the skin streams with perspiration, the temperature of the body rises, there may be involuntary evacuation of the bladder and rectum, but retention of urine is a very common sequel. After recovery from the seizure there is always some paralysis which is transient in character, but all the physical symptoms of the disease are accentuated as the result of such attacks, and a succession of congestion seizures is a common termination of the disease. I have seen death follow a congestion seizure in which symptoms of suffocation and rapid heart's action were the only symptoms.

The treatment of congestive seizures is purely expectant. The patient should not be fed, as food is liable to pass into the air passages and cause pneumonia or gangrene of the lung. Rectal injections of large doses of chloral, 30 grs., have been advocated, but as a rule they are not retained, and I never saw any benefit result from such treatment. The patient should be sparingly clad, kept free from draughts and noise, and the heels may be protected by bird-nest pads, as they are apt to become gangrenous through prolonged pressure. The bladder should be relieved by catheter, and the bowels evacuated by large enemata.

*Trophic Changes.*—In addition to hæmatoma auris, bed-sores, perforating ulcer of the foot, gangrene of the heels and points of the toes, are all liable to occur in the terminal stages of the disease, and particularly after congestive seizures.



*Mental Symptoms.*—As on the physical side progressive paralysis and wasting of the muscles is the only symptom universally present in every case, so on the mental, a gradually advancing enfeeblement ending in dementia is the only constant symptom present. More commonly, however, the disease is complicated by such marked mental aberration, that there is an almost universal belief that general paralysis is always associated with insanity. The mental symptoms may take the form of pure elevation or depression without confusion, acute excitement, or depression, with confusion and vivid hallucinations, and all the physical symptoms associated with these mental states. Twice I have seen general paralytics develop all the symptoms of katatonic stupor, and give the same serum reaction as typical cases of katatonia, and symptoms resembling those of "folie circulaire" have been recorded by several writers.

The mental symptoms which have been always regarded as typical of the disease, however, are elevation with delusions of grandeur.

In the first stage of the disease the patient often gives the appearance of overflowing energy and self-satisfaction. There is mental confusion and lack of judgment, in so far that the patient sees nothing incongruous in wildly improbable statements and delusions, but there is no loss of consciousness, and no loss of knowledge of position. Hallucinations are rarely associated with this condition of elevation, which finds its expression in grandiose ideas and delusions and expansive good-humour. Loss of self-control is shown by irritability over trifles and undue emotionalism. There is a tendency to fantastic decorations of the dress, blunting of the moral sense, particularly as regards the sanctity of property, the patient often stealing useless articles without any attempt at concealment. The attention is easily diverted, and there is no real capacity for occupation. The memory is untrustworthy. The speech is voluble, but not incoherent, and the patient readily understands spoken or written language, and the false sense of well-being not infrequently takes the form of voluminous writing of poetry or imaginary business letters. Sometimes such patients sleep well, while in others sleep is obtained in



short snatches at irregular intervals, or is actually deficient. Night terrors are not infrequently seen early in general paralysis. The whole character of such mental symptoms is that of simple excitement of the folie circulaire type.

As the physical symptoms of the second stage of the disease develop the mental symptoms alter. The patient is no longer so energetic and restless, and there is distinct blunting of the general intelligence. The special senses are less acute, and there is therefore less response to the environments. Delusions, similar to those of the first stage, persist and may be sufficiently extravagant, but they no longer interest and absorb the patient so markedly as previously. The tendency to theft is exaggerated, and takes the form of collecting rubbish. The personal appearance is neglected; the patient is apathetic, facile, and listless, and the volubility, writing, and endless attempts at occupation of the first stage are diminished, because the advancing paresis renders speech and writing difficult, and the increasing mental enfeeblement curtails the power of originating new channels of activity. The function of sleep is variable, sometimes excessive, sometimes diminished.

The mental symptoms of the second stage pass insensibly into those of the third. The patient becomes more and more demented, occasionally expressing fragments of grandiose delusions, but all the interest in life and all the human instincts are gradually lost. Congestive seizures frequently hasten the process of mental destruction, but not always. I have seen patients wake up after congestive seizures to some of their former activity. One patient, who was dull and uninteresting in the second stage, after congestive seizures gave vivid accounts of visits to heaven and the important positions he there occupied. He always described heaven as a grand place; "the streets are paved with gold, lighted by electricity, while vaccination is done upon the premises."

The duration of general paralysis is very variable. I have seen a case die in six weeks from the first onset of acute symptoms, while cases are on record which lived for ten and even twenty years. As a rule the disease is fatal in from two to three years. The duration depends largely upon the



character and causation of remissions. If, in the early stages of the disease, the patient suffers from some acute intercurrent disease, such as pneumonia or erysipelas, the remission which follows may be of very long duration, and the advance of the disease may be arrested for months or years.

The diagnosis between general paralysis and other forms of mental disorder is not always easy, and mistakes are made by the most experienced alienists. Chronic alcoholism may simulate general paralysis so closely that a differential diagnosis is impossible until the patient is under treatment for some months. Lead, cocaine, and chloral, by their toxic action, produce nervous symptoms which resemble those of general paralysis very closely, and general paralysis cannot be excluded until the effect of treatment has been observed. Lastly, it is well to remember that disseminated sclerosis may give rise to mental and physical symptoms which may very well be mistaken for those of general paralysis.

*Treatment.*—Unfortunately there is no specific treatment for general paralysis, and the question which most frequently arises is, "Shall the patient be treated in an asylum or at home?" Home treatment is quite suitable for quiet demented cases, but for those exhibiting acute symptoms asylum treatment is almost a necessity; if, however, the patient can afford it, removal from home to some quiet country quarters, under the care of good attendants, may be as efficient. It should always be borne in mind that men suffering from the disease can beget children, and it is extremely undesirable for the sake of the progeny that such an accident should occur.

The diet should be fattening and non-stimulating in the first stage, and alcohol should be entirely withheld or given in very small quantities. Patients suffering from the disease are, as a rule, easily affected by alcohol. Sedatives, especially sulphonal and the bromides, given in small doses (sulphonal 10 grs., pot. brom. 30 grs.) three or four times a day lessen the restlessness and excitement if the mental symptoms are acute. The bowels should be regulated by laxatives if there is any tendency to constipation. In the second stage of the disease the diet may require especial preparation if the patient shows a tendency to bolt food. Tobacco, which may be allowed



in the earlier stages, may require to be discontinued later, as sickness and heart failure may occur from a very moderate use of this luxury. Sleeplessness is not infrequently due to cold feet, and attention to this symptom often makes all the difference to the comfort and quietness of the patient at night. The treatment of congestive seizures has already been described.

In the last stages of the disease the patient must be carefully fed with soft food to prevent choking, and the usual nursing precautions must be taken to prevent the formation of bed-sores.

Anti-syphilitic treatment is rarely of any benefit, but it should be given a trial in every case. Of late the treatment of tabes dorsalis and general paralysis by mercurial injections has been much advocated by French alienists. A solution suitable for such a method of treatment can be readily prepared as follows:—5 grs. of benzoate of mercury are dissolved in 10 drachms of sterilised normal saline solution, and made fresh every third day; 1 c.c. of this solution, which equals about  $\frac{1}{7}$  gr. of the benzoate, is sufficient to start with, but the dose may be rapidly pushed to 3 and 4 c.c. per day. The injections are continued for three weeks or a month, and cause little local discomfort. My personal experience has not been sufficient to enable me to give an opinion as to the value of this method of treatment.

If a specific remedy is ever discovered for general paralysis it will be of the nature of an anti-toxine, but before such an advance is possible our knowledge of the toxine which is the direct cause of the disease must be definite and exact.

A. R., male, aged 48, was sent to the asylum from the neighbouring infirmary, to which he had been admitted as the result of a carriage accident. Prior to the accident his wife affirmed that she had seen nothing wrong with the patient, but in the infirmary he had been at times delirious and restless at night, and from the physical symptoms alone he was diagnosed as a case of general paralysis.

The patient, who in his earlier years had acquired syphilis, had, so far as was known, always been a temperate, steady man. On admission he presented no physical symptoms beyond a slightly febrile temperature. His pupils were, however, unequal, irregular



in outline, and failed to react to light. The muscles of the hands and forearms were very tremulous. The facial expression was "flattened," and his facial muscles when he spoke twitched irregularly, and his tongue was very tremulous. His speech was slightly slurred, especially when he tried to talk rapidly. The tendon reflexes were markedly exaggerated, his gait was swaying, particularly when he turned, and he could not stand with the heels together when his eyes were shut. Mentally he was enfeebled and emotional over trifles, but he expressed no delusions. He knew where he was, but was perfectly unconcerned, and his memory was rather hazy about both recent and past events. He lived for three years in this condition, becoming more and more paralysed and demented, and finally died in a congestive seizure.

A. F., a male, aged 39, was admitted suffering from acute depression. He was a brick-maker to trade, and a good workman up to three months before his admission. He had, so far as could be ascertained, no hereditary predisposition to mental disease, and his habits of life were fairly steady. His wife first noticed that he lost his appetite and became sleepless. His memory also failed him, as once or twice he proceeded out to his work on a Sunday. He often complained of feeling ill; his lapses of memory worried him, and, as a consequence, so his wife thought, he became depressed.

On admission he was very thin and poorly nourished. He groaned and asked for poison, but could give no satisfactory replies to questions regarding his symptoms and illness. Nothing was detected physically beyond inequality of the pupils and a certain flattening of the facial muscles. Mentally he was depressed, confused, and unable to realise his position. A week after admission he attempted suicide by swallowing matches, but these were quickly ejected by the use of emetics and a stomach pump. Whether as a result of the thorough cleansing of the stomach during the use of the stomach pump, or from some other unknown cause, from that day he improved both mentally and physically. He took food greedily, and within a month had laid on a stone in weight. The diagnosis of general paralysis was now, however, evident. The pupils, the speech, the gait, and reflexes were all typical of the disease, of which he died within two years after admission.

A. F., male, aged 43, was admitted in a state of acute delirious mania. A dyer to trade, he had been peculiar, irritable, and unsteady in his habits for more than a year, and the attacks of mania came on quite suddenly. Twenty years before this illness the patient contracted syphilis.



On admission he was thin and anæmic. His temperature was 100° F. He refused all solid food, but drank greedily. His pulse was 120, irregular and intermittent. His urine was scanty, high coloured, loaded with urates, and contained albumen. Microscopically many leucocytes and some granular tube casts were observed.

He was too restless and violent to be examined thoroughly, but it was noticed that his tendon reflexes were exaggerated, and that his muscles were very tremulous.

Mentally he was wildly excited, greatly confused, noisy and incoherent in speech, and, for the first night, sleepless.

His leucocytosis was 25,000 per c.mm. with a polymorphonuclear percentage of 92. A week after admission he developed pneumonia, which ultimately sent his leucocytosis up to 45,000 with a polymorphonuclear percentage of 95, and coincidentally his maniacal symptoms subsided. He was now suspicious, irritable, and obstinate. He smelled all his food to ascertain if it contained poison; he refused to allow any blood observations to be made, and refused to enter into conversation.

One month later, when he got up out of bed, it was noticed that his gait was weak and dragging; he could not stand with his heels together and his eyes shut. Both pupils were irregular in outline, and the left was larger than the right. The left pupil was fixed to both light and accommodation, but the right pupil reacted normally. His speech was indistinct, and he especially stumbled over such test words as "hippopotamus," "West Register Street." He now took food well, and rapidly grew fat, and it was then noticed that his facial muscles were flattened. . . . A few months later he developed delusions of grandeur chiefly regarding wealth, but he never mentioned these unless questioned. He gradually became fat and demented, in which condition he now remains six years after admission.

A. C., aged 30, was sent to the asylum from prison, where he was serving a sentence of one month for theft. Nothing was known about the patient beyond the fact that in broad daylight he walked up to a shop door and stole a cloth cape. For this offence he was arrested and sentenced. During his term of imprisonment he took to singing at night and destroying his clothes, and was judged insane by the prison surgeon and transferred to the asylum.

On admission there was no doubt as to the diagnosis. His expression, gait, and physical symptoms were typical. He was quite demented and foolish, had no memory for past or recent events, and was quite unconcerned regarding his present position or future prospects. A year later he died in a congestive attack.



J. R., aged 40, was sent to the asylum, as he was found wandering aimlessly about the country-side. He was a salmon fisher, and had been more or less intemperate in his habits for years. His friends had noticed during the year prior to his admission that he acted strangely. For instance, he began to transplant all the plants and vegetables in his neighbour's garden into his own. On another occasion he entered a farmyard and carried off two ducks, having first wrung their necks. These performances were put down to drink. Latterly his memory was impaired, as he frequently forgot to return home, and apparently slept anywhere, begging or stealing food.

On admission he was a tall, well-made man, somewhat thin and haggard in appearance. His temperature was irregularly febrile. He took enormous quantities of food, and never appeared to be satisfied. The nervous symptoms of general paralysis were well marked. Mentally he was extremely good-humoured and demented. He expressed extravagant delusions of wealth and power. Everything he possessed was superb. His dog retrieved otters daily. His horse was the fastest in the country-side. He always went about at home "in a kilt, with silver buckles on his shoes, and a gold watch the size of a turnip in his pocket." He was so facile that he accepted any delusion suggested to him, but his memory was so fugitive that he rarely repeated the same delusion in the same way twice running. At first after admission he grew fat. Then he had a congestive seizure, after which he suffered from retention of urine. On recovering from this attack he lost weight, and finally became bedridden and much emaciated. He lost control of his bladder and rectum, and being a heavy man, bed-sores threatened over the heels and sacrum. Swallowing was impaired, and he frequently choked while being fed. Probably owing to some such accident he developed a pneumonia which proved fatal one year after admission.

*Pathology.*—The earliest changes probably consist in proliferative and degenerative changes in the walls of the capillaries, not only in the cerebral cortex, but also *in the general capillaries of the body*. This alteration of the capillary walls interferes with the nutritive changes between the blood and the tissues.

The pia-arachnoid is thickened and milky, particularly over the convexity of the brain. The cortical nerve cells show signs of pigmentary degeneration, and the neuroglia cells are hypertrophied, especially in the first layer of the cortex and in the white matter.



The later changes consist in marked general atrophy of the brain, dilatation of the ventricles and the subarachnoid and subdural spaces. The cortex on section is irregularly congested, narrowed, and of a bluish tint, while the striation is indistinct. There are also granulations on the walls of the ventricles.

The pia-arachnoid, in addition to being milky, opaque, and thickened, becomes adherent to the underlying cortex at the summits of the convolutions, so that on stripping the pia, portions of the cortex are torn off with it, giving a lacerated, "mouse-eaten" appearance. The cortical nerve cells show marked pigmentary degeneration. The neuroglia becomes hypertrophied throughout the cortex, but most markedly in the first layer, and also in the white matter. The medullated nerve fibres of the brain are degenerated, and the peripheral and cranial nerves show similar changes. The peri-vascular lymph spaces are packed with leucocytes and débris, while the larger intra-cerebral vessels often present clustering of leucocytes in and around their walls.

**Dipsomania.**—Dipsomania is a nervous disease characterised by a periodic craving for stimulants. The physical state underlying the disease is suggestive of *folie circulaire*. The onset of an attack is characterised by headaches, malaise, and sleeplessness, and this is followed by a period of restlessness, a craving for stimulants and narcotics, and sometimes by sexual excesses. If the attack be long continued it is apt to be complicated by excitement and by delusions of a somewhat grandiose nature. The attacks are irregularly periodic, and between the attacks the patient may have full self-control. If the attacks be frequent, however, brain damage invariably sets in, and is characterised very frequently by a complete blunting of the moral sense and loss of memory. Physically the patient may present symptoms of alcoholic degeneration of the muscular and nervous system.

**Treatment.**—Between attacks the patient should lead so far as possible a physiological life, as attacks are induced by any cause which lowers the general health, such as over-work, mental strain, or anxiety. When attacks threaten, a non-stimulating dietary combined with tonics, particularly strychnine,

nine, iron, and arseniate of soda, is the best treatment. Full doses of bromides are probably the best hypnotic for the insomnia which usually accompanies the earlier stages of the attack.

Cases of dipsomania have been recorded as being benefited by hypodermic injections of atropine and strychnine. This form of treatment has been described under the treatment of chronic alcoholism.



## CHAPTER XV

### STATES OF MENTAL ENFEEBLEMENT

#### IDIOCY AND IMBECILITY; THE HIGHER IMBECILES; DEMENTIA

STATES of mental enfeeblement are always the result of failure of development or structural changes in the cortical grey matter of the brain. If the enfeeblement is due to failure of development or brain damage occurring in early life, it is spoken of as idiocy or imbecility. If it occurs as the sequel to acute brain disease, or the result of old age or gross brain lesion, it is spoken of as secondary dementia, senile dementia, or organic dementia.

**Idiocy and Imbecility.**—Ireland (*Idiocy and Imbecility*, by W. W. Ireland, M.D.) defines idiocy and dementia as follows: "Idiocy is a mental deficiency or extreme stupidity, depending upon malnutrition or disease of the nervous centres, occurring either before birth or before the evolution of the mental faculties in childhood. Imbecility is generally used to denote a less decided degree of mental incapacity." He further classifies idiocy into—

1. Genetous, or the variety in which the child is born deficient, and in which the arrest or failure of development has occurred in utero.

2. Eclampsic, or those whose brains have been injured by convulsions occurring during dentition.

3. Epileptic, where the brain is injured by the early onset of epileptic convulsions.

- 4, 5, and 6. Paralytic, traumatic, and inflammatory, where the brain injury is caused by vascular lesions, traumatisms, or

inflammatory conditions, secondary to middle ear disease or ulceration of the throat.

7. Microcephalic, where the head measures less than 17 inches in circumference. The causes of this condition are obscure, for although in a considerable number of microcephalic idiots the sutures of the skull have been found closed, there are cases equally numerous in whom the sutures remain open.

8. Hydrocephalic, where there is enormous enlargement and deformity of the head.

9. Idiocy from deprivation of the senses, where from lack of external stimuli the mental faculties have failed to develop.

10. Cretinism, a condition of dwarfishness associated with failure of development of the thyroid gland, occurring in the children of races subject to the condition known as goitre.

Other writers describe the states of idiocy and imbecility according to the degree of mental impairment, and divide them as follow :—

1. Imbecility or Idiocy of the First Degree.—A condition in which the failure of mental power may be so slight, that it only becomes apparent when the child commences to be educated, but generally becomes more marked as the age of puberty is approached. Such children remain childish and lack the power of application. They are deficient in self-control, impulsive, and passionate, with deficient moral sense, and deficient control of the normal appetites and instincts. The incapacity to learn is sometimes associated with an abnormal memory for dates, numbers, or trivial details. Physically there is little or no deformity, but the expression is often stupid and vacant. They are liable to gastric and intestinal disorders, their circulation is weak, and the general resistance of the body to tubercle and infective disorders is low.

2. Idiocy of the Second Degree.—The mental impairment is observable early in life, and the function of speech, the instincts of cleanliness, the acquirement of the simplest habits of civilised life, develop very slowly, and never reach complete development. Physically there is always deformity of the body—hydrocephaly, microcephaly, and peculiar head and face formations being common. They are liable to gastric



and intestinal disorders, unhealthy action of the skin, and tubercular infections. They are lacking in general sensibility, and the sense of sight is often deficient, associated frequently with squinting and astigmatism. The voluntary movements are clumsy and inco-ordinate, and the nutritional activity of the body fails early in life.

3. Idiocy of the Third Degree.—In these cases the mental faculties may be said to be practically non-existent; they have no memory, no power of speech, and no knowledge of position. The only instinct developed is the desire for food and drink, but even this instinct is incapable of stimulating a mental reaction unless the food is actually in view of the patient. The facial expression is vacant, the features unformed and ill-balanced, while the skin is thick, puckered, and dry. There is great loss of sensibility to touch and pain; the special senses cannot be tested, but sight and hearing are generally deficient. The organic reflexes of micturition and defæcation act reflexly, and the voluntary movements are inco-ordinate, awkward, spasmodic or choreic, and sometimes greatly impaired by various forms of paralysis.

Idiocy of the first and second degree is liable to be complicated by attacks of mental excitement, impulsiveness, and deficient control of the sexual appetite. All cases of idiocy are liable to convulsions and epilepsy.

**The Higher Imbeciles.**—Just as there are certain children mentally defective at birth, who are termed imbeciles or idiots, so there is a large class of persons who, though not sufficiently defective to be termed imbeciles, are in reality only a degree removed from imbecility. In this class I include what are termed cases of moral insanity, vagrants, and habitual criminals. Moral insanity is a euphonious but undefinable mental disorder. It presupposes that all men, if normally constituted, should be born with a moral sense, a fact which is open to argument. Uncivilised races of mankind, if judged by the standard of civilisation, may be regarded in many respects as having no moral sense, while among civilised races the moral standard varies enormously in individuals of the same class, and still more in individuals of different classes of society. There are, however in all civilised communities recognised



standards of conduct, and any habitual departures from such standards are regarded as abnormal. Such departures from the normal do not by any means all tend towards wrong-doing, because it will be found in everyday life that the man or woman who is, let us say, abnormally religious, is just as much a departure from the normal as the man or woman who habitually lies, steals, or drinks. Both have a common deficiency, a lack of mental balance, but whereas the one is not a danger to society and passes unnoticed, the other, being a nuisance, attracts notice. It is not within the scope of this chapter to discuss the responsibility of the criminal, but to demonstrate that individuals exist who either through deficient or arrested development, or as the result of disease, are below the mental standard of the general community in their capacity for conforming to the laws of society. So far as our knowledge goes, many of these cases are apparently born deficient, while others are most certainly the possessors of brains damaged by disease. The first are, as a class, intellectually higher than the second. Many of them possess good average brain power, but lack mental balance.

A. C., a female, aged 25, had acted as a nurse in two hospitals. She was musical, well educated, intelligent, and apparently trustworthy, but she had to leave both her appointments because she appropriated all sorts of articles, many of which could be of no use to her, while of others she either made no use or did not try to conceal. She evinced no shame upon discovery, merely affirming that she had no intention of stealing, having only taken the articles for temporary use. As her collection of stolen goods contained forks, spoons, cups, dishes, handkerchiefs, collars, odd cuffs, aprons, etc., she could only be regarded as a case of kleptomania. So far as could be ascertained she had never suffered from mental disease.

C. B., a female, aged 20, one of a family of five children, never at any period of her existence showed any affection for her parents, brothers, or sisters. As she grew older she resented any interference on the part of her parents, and matters came to such a pass that she was finally certified and placed in an asylum. Careful examination of the patient elicited only one physical symptom, and that was an occasional hyperleucocytosis which occurred irregularly and at long intervals. Mentally she was decidedly below average intelligence, but beyond being childishly petulant



and prone to attacks of sulkiness and irritability, I never saw any marked symptoms of mental disease.

C. F., a male, aged 20, showed marked mental ability as a student, but his capacity for shameless, causeless lying, and inventing scandal, was such that his relatives in despair gave him up as hopeless, and he finally found his way to South Africa, where all trace of him disappeared.

A. C. was as a boy fairly intelligent. While studying as a student in Edinburgh he suddenly left home, and was next heard of in Australia. He eked out a miserable existence, sometimes as a billiard-marker, at others as boots in an hotel, hanger on at a general store, etc., but he never stayed in any situation for more than a few weeks. He finally came home, was induced by his friends to enter a private asylum as a voluntary patient, where he appeared to be quite satisfied with his position.

Many habitual criminals are of this type, but some of them, at least, are the result of early brain disease.

M. D., a male, aged 41, has spent the greater part of the last ten years either in the asylum, poorhouse, or prison. He never works by any chance. He drinks whenever an opportunity presents itself, and he is constantly in trouble on account of petty thefts. His mental symptoms are so indefinite that he is very rarely certified, but upon his last admission to the asylum we found that he presented all the physical symptoms of mania without confusion. On looking up his earlier history it was ascertained that he had suffered from an attack of acute mania during adolescence. On admission he was insubordinate, insolent, and liable to sudden outbursts of anger on slight provocation. These acute symptoms gradually subside in the course of a few weeks, but at his best he was a notorious liar, a chronic grumbler, and habitual thief.

It should also be remembered that moral depravity may be the earliest symptom of acute brain disease, particularly general paralysis and mania of the folie circulaire type. In such cases, however, the onset of mental and physical symptoms clear up the diagnosis.

**Dementia.**—The condition of dementia or mindlessness is usually divided into acute or primary dementia and secondary dementia. Acute or primary dementia is by some authors regarded as stupor, and by others as true dementia arising without apparent cause. It is highly improbable that



such a condition exists. All states of dementia, whether they occur as the result of acute mental disease, or apparently as a primary disease, are the result of structural changes in the cortical grey matter of the brain, and the causes which in one brain will produce first an attack of acute excitement which ends in dementia, will in another, more feeble in resistive power, produce dementia without a previous stage of excitement. It is quite misleading to confound dementia and stupor, because dementia means a terminal incurable condition, while stupor is only a stage of other diseases and is frequently recovered from. I therefore describe dementia as an outward representation of a hopelessly damaged brain cortex—a result, not of the acute mental symptoms, but of the toxins causing these symptoms, which the resistive powers of the patient have failed to neutralise. Dementia, being the result of brain damage caused by toxins, may occur after such diseases as typhoid or typhus fevers, just as readily as after acute mental disease. Clouston has pointed out that the most typical secondary dementia is that which occurs after adolescent insanity, and it is just in adolescent insane patients that one meets with mental diseases complicated or caused by the most severe toxæmias. The acuteness and duration of the mental symptoms, apart from the severity of the toxæmia, have nothing to do with the onset of the mental state which we call dementia. Dementia will occur in patients who have never presented acute mental symptoms, while, on the other hand, cases occur where repeated and acute mental attacks are recovered from and do not produce dementia. The character of the toxæmia has also a great deal to do with the production of the condition. In cases where the toxins are purely metabolic, such as acute melancholia, typical dementia is never seen. In delusional insanity, certainly in part a metabolic condition, dementia is very slow in onset, and uncharacteristic in symptoms even after years of illness. In folie circulaire, where the toxæmia is intermittent, dementia is of rare occurrence. On the other hand, where one can demonstrate symptoms of acute continuous toxæmia of bacterial origin, such as occur in acute excitement with confusion and katatonia, particularly in adolescent subjects, we have con-



ditions which, together with hebephrenia, fill our asylum wards with demented who constitute two-thirds of their population. The case which terminates in complete dementia has generally a hereditary predisposition to mental or nervous disease. This inherited weakness must mean something more than mere brain instability. It also means a constitution incapable of dealing effectively with toxæmias, which, although not dangerous to life, are fatal to the delicate mechanism of the cortical matter of the brain. This failure of reaction we can partly demonstrate and compare with analogous conditions of disease which are known to be infective in origin.

As a typical case of disease terminating in dementia, I describe a case of acute mania in a male adolescent:—

A. A., aged 21, was admitted in a state of acute excitement with confusion, exhibiting all the physical and mental symptoms of the disease previously described. On admission his leucocytosis was 20,000 per c.mm. with a polymorphonuclear percentage of 75. This, unfortunately, did not continue. In a week his leucocytes had fallen to 12,000 and the polymorphonuclear percentage to 60. A subcutaneous injection of terebene failed to stimulate a leucocytosis for more than one day, and the polymorphonuclear cells only rose to 65 per cent. A month after admission his leucocytosis was rarely above 9000 per c.mm., and the polymorphonuclear cells had fallen as low as 50 per cent. He became persistently wet and dirty in his habits, destructive and impulsive in his conduct, and incapable of any form of occupation. Six months after admission he became fat, bloated, and sleepy, and had every appearance of permanent mental weakness. The serum of this patient on admission contained no demonstrable agglutinines, and injection of broth cultures of the coccus obtained from the case of acute mania failed to stimulate the production of either agglutinines or immune body.

Compare the case of A. A. with B. B., a female aged 41, who also suffered from acute excitement. On admission her leucocytosis was 28,000 per c.mm., and the polymorphonuclear percentage 85. At the present date, three years after admission, she still has a leucocytosis often rising to 16,000 and 17,000 per c.mm., with a polymorphonuclear percentage nearer 60 than 50. On admission her serum contained marked agglutinines to the micrococcus isolated from the



case of mania, and two years after admission one injection of 5 c.c. of a broth culture of the organism immediately stimulated the formation of agglutinines, but no efficient immune body. Although this patient has been acutely excited for three years she shows no signs of dementia. Both these patients suffered from the same disease, and probably the same toxæmia; the one becomes demented, and showed by his leucocytosis and inability to form agglutinines and immune body a failure of reaction to the toxine. The other after three years' illness is not demented, and the leucocytosis and the capacity to form agglutinines and, to some extent, immune body are still unimpaired.

The same remarks apply to katatonia. If the leucocytosis keeps up, and especially if the polymorphonuclear percentage remains about 70, the chances of recovery are excellent; if the leucocytosis falls, and the percentage of polymorphonuclear cells falls below 50, the onset of dementia is, so far as my experience goes, a certainty. In the one class of case the resistive power is good and eventually neutralises the toxins, in the other the toxins overcome the resistive power, and brain damage is the result. There is an analogy between these observations and our knowledge of similar changes in known infective conditions. In pneumonia, for instance, if the leucocytosis fails to rise before the crisis the chances of recovery are very poor. Here the toxæmia causes physical death, in acute mental diseases the toxæmia causes mental death.

The symptoms of dementia are very variable in degree in different subjects; in one patient there may be only slowness of mental reaction and lack of initiative. Another is incapable of attending to himself in any way, and may require to be dressed, undressed, fed, washed, and attended to like an infant, and whose power of memory is so deficient that he cannot tell you his name or where he is. All cases of dementia, however, have certain physical and mental symptoms in common. In appearance they are deficient in general activity; the carriage is slouching, and the gait shuffling and lacking in purpose. The facial expression is dull and listless, and the muscles of expression are slow in action, and prob-



ably partially paralysed through disuse. The body temperature is fully 1° F. below the normal. The appetite is good, often excessive, and the food may be bolted ravenously without mastication. Perverted appetite is quite common, some patients eating filth, stones, cloth, or cinders unless constantly supervised. Many demented, and in fact the majority of the chronic insane, drink large quantities of water. Saliva, of a thin, watery consistence, is excreted in large quantities, and as the reflex act of swallowing is often impaired, the mouth first fills with saliva, which then dribbles down the chin and on to the clothes. There is generally anaemia, and the leucocyte counts are low, with a polymorphonuclear percentage as low as 30, with an excessive percentage of small lymphocytes. The leucocytosis, however, depends largely on the physical state of the patient. Many demented are liable to recurrent subacute attacks of mental disease—in other words, to recurrent attacks of toxæmia—and during such attacks the leucocytosis may rise to 15,000 or 16,000 per c.mm., but there is no marked increase of the polymorphonuclear percentage. The heart's action is weak, and the peripheral circulation sluggish, so that lividity of the lips, ears, hands, and feet are common, and the feet and ankles in addition tend to be oedematous. The breathing is shallow, and the lungs readily become infected with tubercle. The skin is dull, dry, and earthy in appearance, and the palms of the hands and the soles of the feet are always moist and sodden. The hair is hard and wiry, and the nails brittle and coarse. The urine is frequently passed in large quantity, is pale in colour, and of low specific gravity. The large quantity of urine excreted is generally due to the habit of excessive water-drinking.

There is impairment of sensibility to touch, heat, and pain. Sensations are recorded slowly, and there may be dulling of all or any of the special senses. The organic reflexes of micturition and defæcation in the worst cases act reflexly, but there is a weakening of the reflexes in every case, so that there is a tendency for these patients to become wet and dirty in habits, especially at night. The voluntary movements are sluggish and deficient in power, and there is



a lack of co-ordination for fine associated actions. Vaso-motor disturbances are evidenced by the excessive salivary secretion and the rapidity with which demented patients become blue, livid, and cold if the atmospheric temperature suddenly falls. Mentally there is always loss of general intelligence and interest in life; many patients do not know where they are, and would not understand if they were told. Hallucinations and delusions may be present. The habits are frequently degraded, filthy, and destructive, the patients tearing their clothes and destroying furniture without any apparent cause. All demented are deficient in control, and many suffer from periodic outbursts of excitement, often with vivid hallucinations and impulsive conduct. The emotions are blunted in some, lost in others, who never exhibit joy or sorrow, affection or hatred, but many are irritable. There is impairment of the moral sense, and loss of the sense of self-respect, of decency, and shame, and there is also neglect of the personal appearance. The power of attention is much impaired. There is no real capacity for work, and the loss of the power of originating new mental or motor processes is a symptom which is never absent in any case. The memory is weak or entirely absent, and speech may be indistinct, incoherent, or abolished. Very few demented patients voluntarily enter into conversation. Sleep is generally excessive.

The majority of demented patients, however, are capable of some re-education and training even when the condition is very pronounced, so that with assistance and supervision they become clean and tidy in habits, and capable of performing routine or mechanical duties.

The capacity for re-education is well demonstrated by the following case of A. B., a female patient, who was admitted after having been demented for 18 years. She had suffered from acute mania during adolescence, thereafter became demented, and was taken home by her relatives. She had lived for these 18 years in a remote Highland valley tended by her mother. On admission she was absolutely incapable of attending to herself in any way. She had to be fed, dressed, and undressed, and she passed urine and faeces as she lay. She had lost the power of speech, and apparently did not understand what was said to her. Within three months she was cleanly in her habits, able to knit a



stocking, and capable of feeding herself with a spoon. She never learned to dress or undress without assistance, and she never spoke, but gradually understood simple orders, such as being told to go to dinner or to bed.

Mild cases of dementia are frequently capable of doing simple work, but the discharge of their duties is not marked by an intelligent appreciation of the reason for doing the work.

J. M. assists the gardener, and can stoke a fire and water the plants. When summer comes round he is told to water a certain border of rose bushes every night at 6 o'clock. This order he readily remembers and executes, but I have seen him watering the plants in a deluge of rain, and after it had been raining incessantly all day so that the ground was like a marsh. It never occurred to him that on such an evening watering was unnecessary.

J. C. works in the smithy, and is very willing to assist in any operation there in progress. If told to work the forge-bellows he will do so for an hour, regardless of the fact that the furnace is out or deficient in fuel.

T. Y. has been trained to be moderately clean and orderly. The only work he can perform is to wheel a barrow, but someone must be there to fill the barrow, to tell him where to go, and again he must be directed where the contents are to be emptied. Without such orders he would stand for hours beside his barrow whether it was empty or full.

T. S. cannot be taught to be clean or tidy, or to do any work. He sits all day huddled up on a seat, picking his clothes or the furniture, and everything he picks up he eats—stones, leaves, grass, the cuffs of his shirt and coat, nothing comes amiss to him. He does not know his own name or where he is, and if asked a question he grunts, or makes some unintelligible reply. He does not know his own bed in the dormitory, nor can he dress or undress. At meal times he eats voraciously, and steals all the food he can from neighbouring plates. His thieving habits often lead to protests from his fellows, who strike him, but he pays no attention to blows or threats, and never returns the assault. He is constantly wet and dirty unless attended to, and has to be under supervision at night for the same reason. This patient originally suffered from an acute attack of katatonia in adolescence.

*Pathology of Secondary Dementia.*—There is generally

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marked atrophy of the brain, with some thickening of the soft membranes. The neuroglia of the first layer is often increased. The cortical nerve cells are diminished in number, and those remaining frequently show degenerative changes with stunting of the apical processes.



## CHAPTER XVI

### THE CLINICAL PATHOLOGY OF MENTAL DISEASES

ALTHOUGH the histological pathology of the nervous system of patients dying insane has received much attention during the last twenty years, although improved methods and apparatus have done much to assist this line of inquiry, and although many additions have been made to our knowledge of the microscopical changes which occur in the brain cells and nerve fibres of the victims of insanity, our knowledge of the causes of these changes remains as obscure as it was before histological pathology reached its present condition.

We must, therefore, direct our attention to some other method of inquiry in the hope of attaining more fruitful results. I am convinced that the best method to this end is the clinical study of mental diseases supplemented by clinical pathology and laboratory observations.

The following notes, taken from time to time as the result of clinical and laboratory observations, are published, not because they are considered complete or convincing, but because I believe they demonstrate that there lies a fruitful field of research in the study of the physical symptoms of mental disease.

*Temperature.*—Let any one take a series of temperature charts compiled in the hospital ward of a modern asylum—that is to say, charts of patients treated in bed as ordinary cases of physical disease—it will be found that certain characteristics of the temperature curves are departures from the state of health. Some are febrile, others subnormal, some remittent, others intermittent, while again many are irregular,—

one day febrile, the next subnormal, and every now and then paradoxical, that is to say, higher in the morning than in the evening. But from a pathological point of view the two most striking features in the temperatures of insane patients are, firstly, that the febrile reaction is very rarely in proportion to the obvious severity of the disease; and, secondly, that very frequently one finds no febrile temperature combined with a high polymorphonuclear hyperleucocytosis. In other words, with every clinical evidence of a virulent toxæmia there is no corresponding febrile reaction.

*Bacteria of the Mouth and Stomach.*—As an associated symptom of these toxic states, there is the rapid accumulation of sordes on the lips, teeth, and tongue. These sordes are composed chiefly of inspissated mucus and saliva, swarming with bacteria of the streptococcus group. These streptococci, when cultivated in artificial media, are very virulent to rabbits when injected intravenously. Doses so small as .1 c.c. prove fatal in three or four hours. Now it is known that the streptococci which can be grown from the buccal saliva of a healthy man are not particularly virulent when injected into the lower animals. In the same way the gastric disorders of insane patients are in some cases associated with an excess of bacteria in the stomach contents. In a case recently examined, the gastric contents yielded a pure growth of a streptococcus which when cultivated in nutrient broth and injected intravenously in .1 c.c. doses into rabbits proved fatal in from one to three hours.

*Coagulation of the Blood.*—Systematic and long-continued blood examinations of the insane have yielded a rich harvest of facts, many of which are at present unexplainable. For instance, the coagulation time of the blood is much delayed in states of acute mania and katatonia. Especially is this the case in the stage of katatonic stupor, where coagulation may be delayed for twenty or thirty minutes.

*Hæmolysis.*—Again, I have seen the blood serum of a case of acute mania with confusion hæmolyse the washed red blood corpuscles of a case of general paralysis when mixed *in vitro* in the proportion of one part of red blood corpuscles to three parts of serum. The same serum mixed in the same pro-



portion with control red blood corpuscles did not alter them in the least in six hours; while control serum mixed in the same way with the red corpuscles of the general paralytic also had no action. One could cite many similar reactions,—certainly departures from health,—but to no purpose, as at present our knowledge is not equal to the task of explaining such reactions.

*Leucocytosis.*—In the light of modern advances in knowledge, however, the observations made on leucocytosis in mental diseases have a distinctly practical bearing, as it is now recognised that certain diseased conditions are always associated with alterations in the number of leucocytes per c.mm. of blood, and by differences in the percentage of the various cell forms recognised as leucocytes. In making the numerical counts I have throughout my observations used Coles's method of counting by fields, not squares, on a Thoma Zeiss' hæmocyte counting slide, and never less than forty fields were counted for each observation. For the differential or percentage counts I used films fixed and stained with Louis Jenner's stain and examined with an oil-immersion lens. In examining films so stained it is not necessary to use a cover-glass unless the films are to be kept. All that is necessary is to let the film, after staining, dry thoroughly in the air—(centrifuging the films saves time, as they are thus dried in one or two minutes)—and then examine directly by placing the oil upon the stained film. Two hundred leucocytes were counted in each film to obtain the differential percentages. The blood for these observations was always taken at the same hour every day, and in no case were results based upon a single observation. The blood was systematically examined from day to day in each patient, in some cases for six months at a time.

For purposes of description I have divided these observations into :—

1. Leucocyte counts in Non-Toxic Insanities.
2. Leucocyte counts in Insanities due to Metabolic Toxæmias.
3. Leucocyte counts in Insanities due to Bacterial Toxæmias.



4. Leucocyte counts in Insanities the result of Alcohol or Drug Toxæmias.

5. Leucocyte counts in Epilepsy, General Paralysis, and Dipsomania.

6. Leucocyte counts in States of Mental Enfeeblement.

In the differential counts the following varieties of leucocytes were recognised:—

1. The multinucleated cells with neutrophile granules, commonly spoken of as polymorphonuclear leucocytes, or shortly as polymorphs.

2. The Small Lymphocyte. A cell about the size of a red blood corpuscle, but often distinctly larger. The nucleus of this cell stains deeply, and occupies the greater part of the cell-body, the surrounding protoplasm being scanty or absent. With Louis Jenner's stain the protoplasm frequently shows coarse blue granules.

3. The Large Lymphocyte. A cell distinctly larger than the Small Lymphocyte, with a distinct and complete ring of protoplasm surrounding the nucleus, which stains less darkly than the nucleus of the small lymphocyte. The body protoplasm always shows blue granules, sometimes faint, at other times large, dense, and deeply stained.

4. The Hyaline or Mononuclear Leucocyte. A large cell whose protoplasm stains faintly grey or blue, with a rounded or lobed nucleus, often eccentric in position.

5. The Eosinophile Leucocyte. A bi-nucleated or multi-nucleated cell with large eosinophile granules.

6. The Mastzellen Leucocyte or Mast Cell. A cell with a single, lobed, or double nucleus, the surrounding protoplasm containing large violet granules.

For purposes of comparison I have made control counts from the blood of various members of the staff. I found in these control cases that the leucocytes per c.mm. of blood varied from 5000 to 9000 in young healthy men, and from 6000 to 13,000 in women. Several of the women were anæmic.

The average percentage of the different forms of cells in the control men were as follows: Polymorphonuclear, 60 to 70 per cent; small lymphocytes, 20 to 30 per cent; large



lymphocytes, 4.5 to 8 per cent; unicellular, 3 to 13.5 per cent; eosinophiles, 1 to 2 per cent; and in the control women, polymorphonuclear, 50 to 65 per cent; small lymphocytes, 25 to 30 per cent; large lymphocytes, 2 to 9 per cent; unicellulars, 5 to 8 per cent; eosinophiles, 2 to 4 per cent. Mast cells occurred occasionally, and a percentage of 1 to 2 was met with in the control bloods.

The leucocyte counts in non-toxic conditions do not call for comment. The leucocytosis is rarely above 10,000 per c.mm. of blood; the percentage of polymorphonuclear cells is as a rule well below 70 in men, and 60 in women, while the lymphocytes, both small and large, are proportionately increased. If in the course of such a case the symptom of hyperleucocytosis occurs and persists, then the case is no longer a pure case of non-toxic insanity.

In mental states associated with uncomplicated metabolic poisoning I have never found hyperleucocytosis. In the last 12 patients examined suffering from acute melancholia the leucocytosis only twice passed 12,000, and the polymorphonuclear percentage did not reach 70. The lymphocytes are always increased in number, and there never is an eosinophilia during any period of the disease. In several cases the mast cells have reached 2 per cent. Recovery or relapse does not seem to affect the leucocytosis in any way. I have only had one opportunity of examining the blood in a case of acute metabolic poisoning which passed into a typhoid state and died. In this patient the leucocytosis was never above 10,000 per c.mm. for the first week of the illness; then suddenly abdominal symptoms set in, the abdomen became tympanitic, the bowels were obstinately constipated, and the patient vomited incessantly. Large enemata unloaded the large intestine and reduced the tympanites and vomiting. The patient, however, passed into a typhoid state and died two days later. Coincidentally with the onset of the abdominal symptoms the leucocytosis rose to 45,000 per c.mm. and the percentage of polymorphonuclear leucocytes to 94. At the post-mortem examination no lesion sufficient to cause death was discovered. A pure growth of *bacillus coli communis* was obtained from cultures made from the liver, kidneys, spleen, and bone marrow.



It is, however, by no means uncommon to find cases of undoubted metabolic mischief presenting every now and then a transient hyperleucocytosis with a rise in the polymorphonuclear percentage, while others without ever presenting the symptom of hyperleucocytosis have an unusually high polymorphonuclear percentage.

In both these types of cases I believe there is some complicating toxæmia, but they differ from the cases of bacterial toxæmia in the fact that on recovery the symptom of hyperleucocytosis and high polymorphonuclear percentage disappears, and recovery is not marked by an eosinophilia, which is a very common symptom in all bacterial conditions.

Cases of metabolic toxæmia, which eventually develop into cases of systematised delusional insanity, do not differ in their leucocytosis, in the earlier stages of the disease, from cases of acute melancholia. Some present little or no divergence from the leucocytosis of health, others have transient hyperleucocytosis, while others again have an abnormally high polymorphonuclear percentage. When delusional insanity finally sets in the leucocytosis presents no peculiarities.

In all insanities in which there is some evidence of toxæmia of bacterial origin, there is always a marked hyperleucocytosis at some period of the disease.

At the onset of excited melancholia and excitement with confusion (acute mania), the symptom of hyperleucocytosis with a high polymorphonuclear percentage is never absent, and eosinophiles are rarely present. Leucocytosis of 20,000, 30,000, or 40,000 per c.mm. are the rule during the period of the early acute symptoms. When the acute symptoms subside the leucocytosis falls, sometimes suddenly, sometimes gradually, and with it the polymorphonuclear percentage falls also. This fall is again succeeded by a rise which averages from 15,000 to 18,000 per c.mm. of blood, with a polymorphonuclear percentage of 60 to 70 per cent. In patients who make rapid recoveries this second rise of the leucocytosis is marked, continues long after recovery is complete, and is accompanied by a rise in the eosinophile leucocytes.

In the more chronic cases, which, however, eventually



recover, there is always present some hyperleucocytosis, and the polymorphonuclear percentage is rarely below 60 per cent in men and 50 per cent in women. A very common feature of the leucocytosis of such cases is the occurrence of an eosinophilia which not infrequently rises to 20 per cent or even higher. Many of the polymorphonuclear cells in such cases also take on eosinophile characteristics, especially in the size and colour of their granules. This is an interesting observation, because I have noticed that patients who had been injected with bacterial vaccines, and who reacted to the injections, and in some instances formed agglutinines, also presented this symptom of eosinophilia within a few days of receiving the injection. If the leucocytosis falls instead of rising after the acute stage is past the prognosis is bad, and especially bad if the polymorphonuclear percentage falls below 50 per cent. Many such cases pass into chronic insanity or become demented. There are, of course, some exceptions to this rule, but they are few. One male adolescent made a recovery from acute mania with confusion, notwithstanding the fact that his leucocytes rarely rose above 10,000 per c.mm., and on some occasions his polymorphonuclear percentage fell below 40. His illness was prolonged, however, lasting in all eight months. For a long time he presented symptoms of dementia, but when, finally, he did improve his leucocytosis showed an eosinophilia as high as 20 per cent. Another adolescent male, recently discharged, at one time threatened to become demented. His leucocytosis was below 10,000, and his polymorphonuclear percentage sometimes below 50, but as he improved his polymorphonuclear percentage gradually rose to nearly 70, although the leucocytosis was generally below 10,000, and he also had a marked eosinophilia. So far I have mentioned excited melancholia and acute mania with confusion together, because the leucocyte changes are very similar. In cases which recover the leucocytosis is high and of good quality, that is to say, the polymorphonuclear cells are present in relatively good proportion. In the case of those patients who become chronic, or who succumb mentally to the toxæmia, the leucocytosis is low and of poor quality; but they differ in this respect, that the leucocytosis of excited melancholia is



more irregular than that of mania with confusion, and there is less tendency for a failure of the production of the polymorphonuclear element. In other words, the toxæmia of excited melancholia has less tendency to produce dementia than the toxæmia of acute mania with confusion. In recurrent attacks of either excited melancholia or mania with confusion, the leucocytosis tends to fall just before the onset of acute symptoms, and the polymorphonuclear percentage falls with it. If the attacks recur the reaction to each subsequent attack becomes less and less marked. The polymorphonuclear percentage always falls far below the average of 70 for men and 60 for women, and coincidentally with this fall, the case may cease to be recurrent and pass into a subacute continuous state of excitement, with slight exacerbations corresponding to slight variations of the chronically persistent toxæmia. In folie circulaire, or manio-depressive insanity, one finds quite a different leucocytosis. If the disease commences with excitement (mania without confusion), and the attack is a first attack, the leucocytosis is not necessarily high, but it is irregular, and the polymorphonuclear percentage is always above 70, and as the excitement subsides the leucocytosis always falls with it. If, as very frequently happens, depression follows the excitement, this stage is characterised by recurrent attacks of hyperleucocytosis with a relatively low polymorphonuclear percentage, and very frequently a decided eosinophilia which may last for two or three weeks. There is, however, no persistent hyperleucocytosis when the patient recovers. If the attack commences with depression the leucocytosis is often well marked, but the polymorphonuclear percentage is rarely above the average of 60 or 70. When excitement supervenes on the depression, there is, generally, first a fall in the quantity and quality of the leucocytes, followed by a rise which culminates with the excitement and then falls to normal. In other words, the toxæmia of manio-depressive insanity is not persistent.

The leucocytosis of katatonia is most conveniently divided as follows:—

(a) The leucocytosis which occurs during the acute stage of onset.



(b) The leucocytosis which occurs during the period of stupor.

(c) The leucocytosis which occurs when the patient is recovering.

(d) The leucocytosis of katatonic mania.

During the acute period of onset, which rarely lasts more than three weeks, there may be a hyperleucocytosis of over 20,000 per c.mm., with a polymorphonuclear percentage of 70 or 80. Or the leucocytosis may be as low as 12,000 or 14,000 per c.mm., with an average polymorphonuclear percentage. In typical cases this period of onset terminates with a sudden and marked hyperleucocytosis—in one case a hyperleucocytosis of 68,000 per c.mm. was recorded—and the polymorphonuclear percentage may rise to 90 or even higher. Immediately after this, the patient passes into stupor, when coincidentally the leucocytosis falls, often below 10,000, and the polymorphonuclear percentage to 60. During the period of stupor the leucocytosis rarely rises above 15,000 to 16,000 per c.mm., but in cases which eventually recover the polymorphonuclear percentage remains well above 65 in both men and women. The prognosis is bad in cases in which the polymorphonuclear percentage falls below 50. At a varying period after the stuporose stage sets in—sometimes weeks, sometimes months—the percentage of eosinophiles rises, and this occurrence of an eosinophilia, which rarely exceeds 15 per cent, is a hopeful sign of improvement. If recovery takes place the leucocytosis may rise, but the polymorphonuclear percentage always rises generally above the average of health. The eosinophilia always disappears upon recovery, but a slight hyperleucocytosis without any increase of the polymorphonuclear percentage is often present in patients discharged recovered. Patients who do not recover, but become demented, always present a low leucocytosis and a low polymorphonuclear percentage. It frequently happens that a recovered katatonic patient relapses, and the mental symptoms simulate mania, or in other cases the state of stupor passes into the state of mania without any intervening lucid period. In such states of katatonic mania the leucocytosis is irregular, and not marked by any increase of polymorphonuclear or eosinophile cells.



In hebephrenia the leucocytosis is generally about 12,000 to 14,000 per c.mm. of blood, but every now and then a marked hyperleucocytosis may occur, rising as high as 30,000 per c.mm. The polymorphonuclear percentage is not always high, and there often is an increase in the hyaline and large lymphocyte cells, which may rise to 20 or 30 per cent. I have never seen an eosinophilia beyond 3 or 4 per cent occur in such cases. If, as rarely happens, recovery occurs, the leucocytosis always falls below 10,000 per c.mm. of blood.

In cases of general paralysis the leucocytosis depends largely upon the type of mental disease which complicates the nervous disorder. If the patient be maniacal, then the leucocytosis is that of mania. If the patient be depressed, then the leucocytosis conforms more to that of the uncomplicated disease, that is to say, of the patient who merely becomes progressively weak-minded and paralysed. Speaking generally, the first stage of general paralysis always shows a more or less irregular hyperleucocytosis, with irregular increases in the polymorphonuclear percentage. Transient eosinophilias are common. In the second stage the leucocytosis may be still irregular, but if the patient suffers from recurrent febrile attacks every two, three, or four weeks, then the leucocytosis follows the temperature curve rising with the fever. Recurrent increases of the eosinophile cells to 5, 6, 7 or 8 per cent occur for several days just about the decline of the fever. In the third stage the leucocytosis becomes markedly irregular—one day below 10,000 per c.mm. of blood and the next 30,000. The polymorphonuclear percentage is generally low and the lymphocytes are greatly increased, and may outnumber the polymorphonuclear cells. If a patient recovers or enters upon a marked remission, equivalent to a recovery, the leucocytosis invariably falls below 10,000 per c.mm. of blood, but the polymorphonuclear percentage is generally very low, *i.e.* between 40 and 50.

In patients who are both epileptic and insane there is always a hyperleucocytosis, which is most marked in recent cases and much less so in chronic.

Two cases of "masked" epilepsy and one case of "con-



vulsive" melancholia also presented the symptom of marked hyperleucocytosis. In the only case of dipsomania in which I was able to make continuous blood examinations, and who was also maniacal, there was a hyperleucocytosis which disappeared upon recovery.

In insanities the result of alcohol and drug toxæmias, so far as my observations go, there never is a hyperleucocytosis, but these observations do not include cases of delirium tremens, as during the past four years I have not had a case admitted into the Perth District Asylum.

In idiots and imbeciles there is no hyperleucocytosis unless the patient presents some symptoms of toxic insanity such as mania or katatonia.

In secondary dementia slight rises of the leucocytosis to 15,000 or 16,000 per c.mm. are not uncommon, but as a rule the polymorphonuclear percentage is low.

In all insanities in which there are evidences of bacterial toxæmia there are certain features in the leucocytes which are common to the group:—

1. During acute attacks, when the leucocytosis is high and the polymorphonuclear percentage is above 70, the polymorphonuclear leucocytes are large, very granular, and deeply stained, and the nuclei may be subdivided into five or six lobes.

2. As the acute stage passes off, the polymorphonuclear cells become much less granular, and there is less subdivision of the nuclei. Very frequently an eosinophilia occurs, and coincidentally the granules of the polymorphonuclear cells appear to have a special affinity for the eosine dye.

3. In patients who recover there is evidence that the leucocyte-producing tissues are active, and especially the tissues which produce the polymorphonuclear cells. Such patients react vigorously to the subcutaneous injection of such an irritant as terebene.

4. In patients who do not recover there is evidence that the power of producing polymorphonuclear leucocytes is impaired or exhausted. Such cases do not react to subcutaneous injections of terebene or any other irritant. There is no exception to the rule that chronic cases of insanity of this class have a deficient polymorphonuclear percentage.



*Agglutinines.*—I have elsewhere described, under the chapters on katatonia and mania, the presence of agglutinines in the serum of patients suffering from these diseases. These agglutinines appear to be specific agglutinines to varieties of streptococci isolated respectively from the blood of a case of katatonia and the blood of a case of acute mania with confusion. Rabbits injected subcutaneously or fed with these organisms readily form specific agglutinines which agglutinate these organisms and no others. Adult rabbits, either fed or inoculated subcutaneously, intraperitoneally or intravenously, with the streptococcus obtained from the case of mania, present no symptoms beyond a rise of temperature, but rabbits of two or three days old, when inoculated intraperitoneally, are apt in the course of from three weeks to two months to develop paralysis of the hind legs, bladder, and rectum. A specific agglutinine to the organism is present in the blood, but the organism itself has never been recovered from the blood or cerebro-spinal fluid.

Adult rabbits inoculated intravenously with the streptococcus obtained from the case of katatonia, or fed with the organism, develop in a few days a febrile temperature with a state of hebetude which lasts for about six weeks. Specific agglutinines to the organism develop in the blood serum, but the organism has never been recovered from the infected animals. Injections of 2 c.c. of a broth culture of the same organism injected subperitoneally into rabbits two or three days old, retard their growth and development, and occasionally produce paralysis of the hind-legs, bladder, and rectum. I have inoculated 5 cases of acute mania with confusion with vaccine made from the streptococcus obtained from the case of mania, and also one case of excited melancholia. The inoculations were made weekly for six weeks. At the end of that time the serum was found to rapidly agglutinate the organism *in vitro* in dilutions of 1 in 100 in four of the cases of mania, but the fifth case of mania, and the case of excited melancholia, formed no agglutinines, and none of the cases formed immune body. That is to say, when one volume of the organism was mixed *in vitro* with three volumes of the serum of the patient, and placed in the incubator for four hours, and then inoculated



on agar tubes and incubated, the organism grew within twenty-four hours. Control serums were used in every observation, and very frequently they retarded the growth of the organism, and in two cases killed the organism, no growth appearing on the agar tubes after 48 hours' incubation. None of the patients appeared to benefit by the vaccine injections, and the treatment was discontinued, but the observation suggested that the serum of these patients did not possess some substance which is present in healthy serum, and that, further, their capacity to form immune body was impaired.

In following up this clue, I found that the serum of various members of the staff had the property of agglutinating certain strains of staphylococcus aureus when mixed *in vitro* in dilutions of 1 in 10. The staphylococci used in the observations were obtained from the bone marrow of a patient suffering from acute mania with confusion, who died in a typhoid state, and from the bone marrow of a patient suffering from mania without confusion, who died from phthisis and exhaustion. Out of twenty-four control sera so examined it was found that twenty-one agglutinated the organism in less than one hour, while the serums of forty-seven patients suffering from various forms of insanity did not possess this power in the same proportion, and sometimes failed to agglutinate even after six hours.

The cases examined were as follows:—

Four cases of acute melancholia, who all agglutinated in  $1\frac{1}{2}$  hours.

Four cases of delusional insanity, who agglutinated in from 1 to 2 hours.

One case of excited melancholia, who did not agglutinate in 6 hours.

Eight cases of acute mania with confusion, who agglutinated in from 3 to 6 hours, but the action was rarely complete. Two of these cases on recovery agglutinated in one hour.

Four cases of acute mania without confusion, who agglutinated in from 1 to 6 hours.

Eight cases of katatonia, two of whom agglutinated in one hour; the remaining six agglutinated in from 2 to 6 hours, sometimes very imperfectly.



Six cases of hebephrenia,—one case agglutinated in one hour, the remaining five either failed to agglutinate, or took from 2 to 6 hours. Six cases of general paralysis, who all agglutinated in from 1 to 6 hours. Six cases of epilepsy with insanity—all chronic—agglutinated in from 1 to 6 hours.

*Immune Bodies.*—No observations were made upon the opsonic indices of these cases, nor upon their capacity for destroying the staphylococcus *in vitro*, but the results, such as they are, suggest that the serum of the insane, particularly those suffering from insanities complicated or caused by bacterial toxines, are deficient in some protective substance which is present in the serum of healthy persons. The incapacity of general paralytics as a class to form antibodies was demonstrated by injecting four patients with vaccines made from the bacillus of Ford Robertson, M'Rae, and Jeffrey. One of these patients formed immune body and agglutinines as tested *in vitro*; the remaining three did not. The one who formed immune body improved so much that he was discharged, but died six months later from phthisis.

To further ascertain the capacity of cases of general paralysis to form immune body or allied substances, I injected three cases of the disease with red blood corpuscles taken from healthy persons and washed in normal saline solution. During the first fortnight six injections were given. Each injection consisted of .25 c.c. of red blood corpuscles in 3 c.c. normal saline solution. A week after the last injection these patients' sera were examined *in vitro*, one volume of washed red blood corpuscles being mixed with three volumes of the serum, and placed in the incubator for one hour. No hæmolysis occurred, but one case agglutinated the corpuscles. Two weeks later one large injection was given in each case, consisting of 1 c.c. of washed red blood corpuscles in 5 c.c. of normal saline solution. Another week was allowed to elapse, and the serum was again examined, but without any result.

It is possible that in these cases an insufficient quantity of washed red blood corpuscles was given to stimulate the production of hæmolysines, but judging from the rapidity with which man and the lower animals react to single injections of



bacterial vaccines, one would expect that some reaction should have been capable of demonstration *in vitro*, if the patients had any capacity for forming antibodies. The result of the observation appears to me to further emphasise the fact, that the onset of these diseases may depend largely upon the failure of the defences of the body against the action of the ordinary saprophytic bacteria.

*Blood Pressure.*—The clinical pathology of the circulatory system—the high arterial tension of acute metabolic toxæmia, the lividity and œdema of stuporose and exhaustive states—has been mentioned in the chapters describing the disease conditions in which these symptoms most frequently occur.

*Phthisis.*—With regard to the respiratory system it has already been pointed out that the insane are particularly liable to contract phthisis.

*Urine.*—The observations which I have been able to make on the urinary excretion of cases of mental disease are by no means complete, but several interesting facts have been noted. The examination of the urine includes a record of the weight of each patient in lbs., which, multiplied by three, gives the amount of urea in grains which ought to be excreted daily. This again multiplied by three gives the quantity of albumen in grains which such a patient requires in the daily dietary (Haig). The urine in each case examined was collected and examined daily for weeks on end. The daily excretion of urea, chlorides, and purin nitrogen was estimated, and at the same time the amount of chlorides and albumen in the dietary was estimated daily. Where the percentage of purin nitrogen was estimated the dietary was always purin free, *i.e.* flesh, fish, tea, coffee, and oatmeal were omitted from the diet.

It is impossible in such a work as this to describe the results of the urine observations in detail. It seems to me more profitable to record and compare the differences in the urinary excretion in the two main classes of mental disease—metabolic and bacterial toxæmias.

The outstanding feature of metabolic disorder is the failure of the elimination of the waste nitrogenous products of the body. As I regard acute melancholia as a typical metabolic disorder, and as all the cases of acute melancholia examined closely



resemble one another, I will describe the observations made on a typical case:—

C. M., female, aged 31, weight 92 lbs., who ought therefore to excrete 276 grs. of urea per day, and required 828 grs. of albumen per day to supply this nitrogenous excretion. For the first three days after admission she ingested per day sufficient albumen to excrete 260 grs. of urea, but she only excreted 138 grs. During the same period she ingested 53 oz. of fluid and 2·5 grms. of sodium chloride per day, and she excreted 19 oz. of fluid and 1·8 grms. of sodium chloride. On the fourth day after admission her urine suddenly rose to 54 oz. for the twenty-four hours, and she excreted 432 grs. of urea and 3·5 grms. of sodium chloride. This was followed by marked mental and physical improvement, which continued until the sixth day after admission, when being very hungry she ingested 1080 grs. of albumen in her dietary—being 250 grs. above what she required. This was followed by a relapse. Gradually, however, the excretion of urea rose again, until, during the third week after admission, the albumen ingested equalled 350 grs. of urea per day, and the excretion rose to 300 grs. per day, while the ingestion and excretion of sodium chloride were practically equal. The daily fluid ingested during this week equalled 66 oz. against an excretion of 50 oz. per day.

In early cases of delusional insanity one frequently finds the same changes.

A. C., female, aged 32, weight 113 lbs.,—who ought therefore to excrete 339 grs. of urea, and required 1017 grs. of albumen per day,—when first admitted, excreted 21 oz. of urine against 58 oz. of fluid ingested. The albumen taken during the first week after admission was equal to a daily excretion of 260 grs. of urea; the actual excretion was only 119 grs. The ingestion of nitrogen was always in excess of the excretion in this case, although the actual output of urine gradually increased. She is now, one year after admission, a typical case of delusional insanity, and her weight has only increased by 4 lbs.

At a later stage of delusional insanity it is not uncommon to find an excessive excretion of urea and chlorides associated with a loss of body weight.

The urine of patients suffering from metabolic disorders is frequently turbid, the turbidity being due to micro-organisms.

In some cases the organisms are actually voided with the urine, in others the urine, in spite of being collected in



sterilised vessels, readily becomes contaminated. This must be due to some peculiarity in the urine, as although, let us say, six urines are being collected at the same time, five will be clear, and the sixth, from a case of metabolic disorder, is turbid and swarms with organisms. The organisms present are generally streptococci which are non-virulent to animals; very frequently also a bacillus is present, which is also non-virulent to the lower animals.

In bacterial toxæmias, on the other hand, one finds quite a different excretion. In the early acute stage of the disease the albumen ingested is not in proportion to the urea excreted, and there is also a deficient excretion of chlorides. Then follows a stage when the albumen ingested is in excess of the urea excreted, and, finally, the albumen ingested and the urea excreted practically balance. During the first period there is loss of weight, followed by a gain in weight during the second period, and in the last stage the weight is stationary. All cases of acute mania with confusion follow this rule.

J. M., male, aged 22, weight 128 lbs., who ought therefore to excrete 384 grs. of urea per day, and required 1152 grs. of albumen per day. For the first 7 days after admission he ingested 2.5 grms. of sodium chloride per day and excreted 1.8 grms. The daily albumen ingested was equal to an excretion of 208 grs. of urea, but he excreted 475 grs. His weight could not be taken on account of excitement, but he undoubtedly lost weight. Six months later, when improving, the sodium chloride ingested was 32 grms. against 30 excreted. The albumen ingested was equal to a urea excretion of 440 grs. per day, while the actual excretion was 367 grs. These observations lasted seven days, and during the month his weight increased from 135 lbs. to 152 lbs. A month later, when practically convalescent, the albumen ingested was equal to an excretion of 421 grs. of urea per day, and the actual excretion was 425 grs. These observations also lasted a week, and the weight for the month only increased by 2 lbs.

All cases of acute mania with confusion examined, conform to the results recorded in the case of J. M. In cases of excitement without confusion, on the other hand, there appears to be no great difference between the ingestion and excretion of chlorides and nitrogen, but the opportunities for confirming this observation have been wanting in the admissions to the

Perth District Asylum during the period when these observations were made. The observations on katatonia and general paralysis are not yet completed. Cases of hebephrenia present no peculiarities in the urinary excretion.

The observations on the percentage of purin nitrogen are only conclusive in so far that all cases of delusional insanity, either in the early or later stages, frequently have a percentage of purin nitrogen above 0·20, and particularly if the symptoms are acute. The placing of such patients on purin-free diet invariably lowers the percentage of purin nitrogen, and frequently improves the mental and physical health. During the acute early stages of acute mania, and throughout the course of general paralysis, there is also a tendency to an excess of purin nitrogen in the urine.

Indoxyl-sulphuric acid is present only in small quantities in normal urine, but it is frequently increased in cases of acute insanity. Indoxyl-sulphuric acid is derived from indol, a product of the decomposition of albumen in the intestines. After absorption the indol is oxidised to indoxyl, and combining with the sulphuric acid of the urine is excreted as indoxyl-sulphuric acid. The presence of large amounts of indoxyl in the urine is evidence of intestinal putrefaction and constipation. The most marked indoxyl reactions are met with in states of metabolic toxæmia, but it also occurs in states of acute excitement.



## CHAPTER XVII

### GENERAL TREATMENT

THE ideal treatment of mental diseases ought to depend upon the early recognition of the physical symptoms which constitute the earliest onset of these maladies, but which, unfortunately, in our present state of knowledge, are so indefinite and ill-defined as to be of little assistance in diagnosis. In the majority of cases the patients, if they do not seek medical advice, are ignorant of their real danger, and are ready to attribute their symptoms to some trivial and passing condition of malaise. There are others, however, who realise that their illness is mental in character, and who have no hesitation in saying so when they consult a medical man. A third and most unsatisfactory class of patient will not admit being ill until the friends notice an alteration in appearance and manner, and consult a medical man.

Speaking generally, every case of incipient insanity on a first attack presents some symptoms of nutritional failure. The patient looks ill, the skin is dry and harsh, sometimes jaundiced, and there is loss of body weight. In addition there is generally some disorder of the alimentary tract, such as loss of appetite with increased thirst, with discomfort, flatulence, and eructation after taking food. The bowels are usually confined, and the fæces abnormally offensive. Anæmia is commonly present, and it is highly probable that in certain toxic conditions a hyperleucocytosis precedes the onset of mental symptoms, but my own opportunities for observation on this point have been too limited to permit of a more definite statement.

If, in addition to such symptoms, one finds irritable cardiac action, particularly a rapid, irregular, hard pulse at night, associated with sleeplessness, nervousness, and mental irritability, the probabilities are that the patient, if predisposed, will develop mental symptoms.

The treatment for such a condition is mental and physical rest combined with a course of tonics, of which quinine, dilute acids, nux vomica, arsenic, and iron are the most serviceable. If there are any symptoms of increased arterial tension with increased pulse-rate, rest in bed is imperative, and the dietary should be purin-free, and consist largely of milk, milk puddings, and crisply toasted bread, while the patient should be encouraged to take at least two pints of water in the twenty-four hours between meals. Tea, coffee, and butcher meat should be prohibited. The large intestine should be unloaded by enemata of normal saline solution, which may be repeated every second day if necessary. Under such treatment the more acute and unpleasant symptoms rapidly subside, but a complete rest for at least a couple of months should be insisted upon.

It is highly probable that the commencement of many mental diseases can be referred back to nutritional failure in early life, and that what are called nervous children are really suffering from obscure alimentary disturbances which interfere with their development and growth, a state of affairs which is evidenced on the physical side by puny bodies, and on the mental by nervousness, morbid emotionalism, precociousness, and disorders of sleep. Such children in later life must be extremely liable to mental disorders. All such symptoms in nervous children which I have seen could be referred to disorders of the alimentary and circulatory symptoms, very frequently accentuated by foolish treatment on the part of the parents or guardians. A boy of seven years of age who was recently under my care, whose father had suffered from mental disease in adolescence, had suddenly developed attacks of apparently maniacal excitement immediately after being put to bed. The child was well developed and well nourished, but spoilt and petted. I saw one of his attacks, which came on within a minute of his falling asleep. He sat up in bed,



screamed, kicked, tore at the sheets, and attempted to bite his grandmother, who tried to pacify him. He appeared to be unconscious, because when roughly shaken he awoke and looked confused. On inquiry I found that he had tea at supper because he would not drink milk, and I also noticed that during the attack his pulse was small, thready, and weak, and that his extremities were cold. I stopped the tea at supper-time, gave him two-minim doses of tinct. digitalis half an hour before bedtime for a week, in order to raise the arterial pressure, and the attacks entirely ceased.

Another patient, a girl of 10, who was thin, anæmic, and of pinched appearance, suffered from night terrors and sleeplessness after trivial causes of excitement. Like all such children her appetite was capricious, and she had a preference for butcher meat. She had no inclination to romp and play like other children, but preferred to read and was abnormally studious. A mistake in her lessons threw her into such a state of grief that she was sure to have a sleepless night. She was ordered a non-stimulating dietary from which butcher meat, tea, and coffee were excluded; she was given a teaspoonful of pure petroleum thrice daily after food, taken away from school for six months, and encouraged to follow outdoor pursuits. In a year the change in her condition was such that one would not have recognised her as the same child. Petroleum in some way acts as an antiseptic in such cases, probably as a mechanical agent coating the alimentary tract, and preventing constipation. It has been proved that none is absorbed, but that as much is passed per rectum as is taken by the mouth.

Adolescent cases, also, who show nervous tendencies, must be treated on practically the same lines. There is no doubt that broadening the intellectual interests and encouraging hobbies are valuable adjuncts to treatment, but in every case it is the physical and not the mental symptoms which most urgently call for prompt and intelligent investigation and attention.

The treatment of mental diseases which have fully developed is the treatment of physical symptoms of disease. To talk of ministering to the mind diseased is to talk clap-trap



and nonsense. Mental diseases, like other physical disorders, are best dealt with on the lines of common sense and general medicine.

It is very rarely indeed that hyperpyrexia in mental diseases calls for active interference on the part of the physician. When such interference is necessary it generally occurs in cases of puerperal mischief, and in those rarely-occurring cerebral manifestations of rheumatism called cerebral rheumatism. In both conditions cold sponging or the cold bath are the proper remedies to apply. It occasionally happens, however, that subnormal temperatures may occur in stuporose and exhaustive conditions, and may call for treatment in the form of applying artificially the stimulant of heat, which the deficient oxidising power of the patients' tissues fails to supply naturally.

Sudden drops of temperature, from normal or febrile to subnormal, occurring in any acute toxic condition, such as acute excitement with confusion, acute melancholia or katonias, are always suggestive of the onset of a typhoid state, and call for prompt treatment in the shape of stimulants, hot enemata, or the subcutaneous injection of warm normal saline solution.

In all forms of early acute mental disease, disorders of the alimentary tract are almost invariably present, and must be treated. Carious teeth are a source of continuous toxæmia, and must be removed as early as possible. The effect of removing carious teeth is in many cases a marked improvement in the general health.

In acute toxic and exhaustive conditions the accumulation of sordes on the lips, teeth, and tongue can to some extent be alleviated by the use of mild antiseptic mouth washes applied by means of cotton-wool swabs. One of the most efficient washes for this purpose is made as follows: *Acidi borici*,  $\bar{3}$ i.; lemon juice,  $\bar{3}$ i.; glycerine,  $\bar{3}$ ii.; water to one pint.

In all acute insanities the appetite is diminished, and the attempt to force large quantities of food on such a patient is not only bad treatment, but must be actually harmful. As I have demonstrated elsewhere, the loss of desire for food is merely an indication that the digestive power of the stomach



is wholly or partially in abeyance. Where such a symptom exists, the physician should be content when he can induce the patient to take from two to three pints of milk, diluted with aerated waters, in the twenty-four hours, and this should be given in small quantities and frequently. Some patients, however, either cannot take milk, or dislike it. Sometimes the difficulty can be got over by heating equal quantities of milk and water, and slightly sweetening it. In others the milk can be offered in the form of very weak milk-tea. If artificial forced feeding is necessary, digested milk or liquid custards should always be employed. Food can be artificially administered either by the œsophageal or nasal tube. Where the œsophageal tube is used the nurse should provide: an œsophageal tube, with funnel, which has been thoroughly washed with hot water, and which should lie coiled in a large basin; a gag, the levers of which are protected by a wrapping of soft cloth, which should be sewn into position; a small jug of cold water; a jug containing the quantity and quality of food ordered; a small vessel containing glycerine or Castile soap solution; a sheet to cover the bed-clothes and prevent soiling of the bed linen. The tube having been lubricated with glycerine, is passed by the operator along the floor of the mouth, and down the œsophagus for 17 or 18 inches, when the eructation of gases generally indicates that the stomach has been entered. As soon as the gas ceases to escape from the funnel a small quantity of water is poured down to ascertain that the tube is clear, and when that is satisfactorily demonstrated, the liquid food should be given as rapidly as possible. The operator should always ascertain for himself before giving the meal that the food is not too hot. The patient during the operation should be in the semirecumbent posture, but as soon as the food has been given the tube, having been first flushed again with water, is rapidly and gently withdrawn, and the patient placed in the recumbent position for at least five minutes to prevent vomiting.

When the nasal tube is employed the nurse provides: a number 12 or 13 rubber nasal catheter with a funnel; a small jug of water; the fluid food; the sheet to protect the bed-clothes; and either glycerine or soap emulsion to lubricate the



catheter. The operator stands on the right-hand side of the patient, who should be sitting up or in a semirecumbent posture. The tube, having been lubricated, should be passed along the floor of the nasal passage. Difficulty in passing the tube may arise through the nasal septum being deflected to the one side or the other, or through the passage being obstructed with dried mucus. When the tube has been passed as far as the pharynx the operator should watch for the reflex action of swallowing, when the tube can be readily passed into the œsophagus and so into the stomach. If the tube is passed without watching for the act of swallowing, the point of the tube is frequently deflected forwards and comes out at the mouth, or is curled up in the pharynx and causes choking. In the majority of cases this accident gives rise to coughing, choking, and lividity of the face, but in some stuporose conditions, and in general paralysis, it is well to remember that the tube can be passed into the trachea without causing the patient any inconvenience. In such cases the only safeguard is to listen, to ascertain that air does not pass by the tube during inspiration and expiration. When it is ascertained that the tube is in the stomach, a small quantity of cold water is poured into the funnel to demonstrate that the lumen is patent. The food is then given, the tube again flushed with plain water and withdrawn, being the while pinched firmly by the thumb and finger of the right hand, to prevent any of the contents escaping during withdrawal and possibly gaining access to the trachea.

After the early stage of an acute insanity has passed the diet should be varied in character and increased in quantity, according to the type of the case. In acute melancholia, for instance, the return of the patient to an ordinary diet should be very gradual; in acute excitement with confusion, on the other hand, where the appetite is frequently voracious, a full and nutritious dietary can be given with benefit. Lavage of the stomach with dilute permanganate of potash solution often gives great relief in all the acute toxic insanities, and I believe that the stomach should always be washed out, as a routine practice, where the breath is foetid and the desire for food is in abeyance. In all acute insanities the patient should be



encouraged to drink large quantities of diluent fluids, not only to alleviate the thirst which is so commonly present, but to flush the tissues and assist the skin, kidneys, and intestines in throwing off deleterious substances. The state of the bowels should always be ascertained, and constipation treated by large normal saline enemata. Whenever indoxyl is present in large amount in the urine there is always constipation and a loaded large intestine. Lavage of the large intestine by means of normal saline enemata, given by means of a douche-can, removes all traces of indoxyl from the urine in twenty-four hours. If after such treatment indoxyl is still present in the urine, the large intestine has not been efficiently emptied of its contents. Lavage of the large intestine is a most useful method of alleviating the symptoms in acute melancholia, acute excitement with confusion, and katatonia. The use of these large enemata, consisting of from two to three pints of normal saline solution, and given every second day for three or four weeks at a time, does not produce constipation after they have been discontinued. The patient, during the administration of the enema, should be placed on the back, with the hips slightly elevated and the body inclined to the right side. The douche-can should be raised gradually, so as to allow the fluid to gravitate slowly into the large intestine.

The anæmia, which complicates acute and chronic insanities, calls for no special treatment beyond attention to the general health and the administration of iron and arsenic. By far the most valuable stimulants to the production of hæmoglobin and red blood corpuscles are sunshine and fresh air. Again and again I have seen a patient, who had failed to benefit by the administration of tonics, rapidly improve when the weather permitted of the bed being taken out day after day to the hospital garden.

The hyperleucocytosis so generally present in cases of insanity of toxic origin, together with the specific agglutinines which have been noted in the blood serum, suggest that serum therapy may eventually become of value in the treatment of some varieties of mental disease. A few cases of excitement with confusion have certainly benefited by the exhibition of antistreptococcus serum (polyvant), but the results of treat-



ment with antibodies and vaccines made from the streptococci obtained from the cases of mania and katatonia have been practically nil. Nor is this surprising when one considers that the only known efficient antitoxine at present in use is antidiphtheritic serum, and it is only of value if given early in the disease. In mental diseases, before the serum can be used, the disease process has lasted for weeks and often longer. Another argument against the possible use of antiserums is this. The bacterial toxæmias of insanity are chronic, and indicate that the real disease lies deeper than the mere toxæmia, the disease being rather a failure of the patient to form antibodies. Elsewhere I have shown that some cases of toxic insanity are unable to form antibodies when artificially infected, and if this constitutional failure be general, as I believe it is, then treatment must be directed towards stimulating the cell formation which can form antibodies. Whatever other cell tissues may enter into the process of immunisation, it is certain that the leucocytes take a very active part in producing a state of immunity. It has long been recognised that acute intercurrent disease often abruptly terminates states of mental disease. In all such cases which I have been able to observe the intercurrent disease produced a hyperleucocytosis, and acting upon this knowledge, I stimulated artificially the leucocyte production in cases of acute mania by the subcutaneous injection of 2 c.c. of terebene under the skin of the flank. In a certain number of cases this treatment was of great benefit, particularly in post-puerperal conditions; but in others it failed notwithstanding the fact that the leucocyte reaction was excellent in quantity as tested by the microscope, but our knowledge is not sufficiently advanced to test the quality or immunity-producing power of the cells. According to Erlich's theory of immunity, there are two substances in immune serum, the immune body and the complement. So far as my observations go, the complement is always present in the sera of the acutely insane, but the immune body is either absent or deficient, and it is this incapacity of the cells to produce immune body which is practically the root of the disease process. I have endeavoured to stimulate leucocytosis by the injection of cinnamic acid,



nucleic acid, and bacterial vaccines of various sorts, but without any marked or permanent result. If immune serum is used—and antistreptococcus serum is always worthy of a trial in acute mania with confusion—then the serum must be given in a large dose.

The disorders of the circulatory system which chiefly require treatment in acute insanities are rapidity of the pulse and high arterial tension. In all cases of acute insanity the pulse-rate is quickened and often irregular and intermittent, while in acute metabolic conditions the arterial tension is invariably raised. Wherever such symptoms are present rest in bed is indicated, and a raised arterial tension can be quickly lowered by placing the patient on a purely fluid diet consisting of milk and aerated water. Erythrol-tetra-nitrate in  $\frac{1}{2}$ -gr. doses will also reduce the tension within half an hour after administration, and continue to act for two or three hours. Given in this way at bedtime it is often a valuable drug in sleeplessness, particularly the sleeplessness of old age. Unfortunately the patient soon becomes used to the drug, and even increased doses do not produce the required effect. Opium also produces a beneficial action in acute melancholia by reducing the arterial tension. Continuous hot baths at 100° F., given for an hour or longer, do not, in my experience, affect arterial tension, but they do reduce the pulse-rate very markedly in conditions of excitement. In stuporose and exhaustive states the circulation is always sluggish, and the extremities become cold, livid, and oedematous. Wherever oedema is present the patient should be kept in bed, and if the bodily temperature be markedly subnormal, then artificial heat must be supplied. Cardiac failure is a common termination of life in patients who suffer from chronic insanity, particularly chronic excitement. Phthisis, which is very prevalent among the inmates of asylums, is liable to attack all cases of chronic insanity whether they are treated in or out of asylums, and calls for no special mention in the way of treatment.

Special attention should be paid to the condition and functional activity of the skin in all acute insanities. Where the skin is dry and harsh, increasing the fluids in the dietary and occasional tepid baths often produce a healthy action,



especially if the body of the patient be briskly rubbed down after bathing with a rough towel or flesh gloves. In many cases of acute excitement, and also in katatonia, the skin becomes covered with a greasy, sour-smelling perspiration which swarms with micro-organisms, especially staphylococci. Daily warm baths, to which some antiseptic or deodoriser has been added, relieve the condition at least temporarily.

Any mental condition in which consciousness is clouded is certain to be complicated by urinary troubles. Either the patient passes urine unconsciously and reflexly, or there is retention of urine with overflow of the bladder. Attention on the part of the nurse to a large extent removes the former condition, and for retention a hot sitz-bath should always be tried before a catheter is resorted to, except in cases of general paralysis after or during a congestive attack. Mild attacks of cystitis are common during the course of all acute insanities, but rarely require any further treatment than diluent drinks, hot hip-baths, and the administration of urotropine in 5-gr. doses. The deficient excretion of urine and urea, which forms such a marked symptom in acute metabolic states, is treated by attention to the diet and increasing the fluids in the food as described under the treatment of acute melancholia.

The habit of masturbation, a complication which may occur in all forms of insanity and in both sexes, can to some extent be prevented by constant supervision, especially in acute cases. In adolescent women mechanical restraint of the hands may sometimes be necessary and beneficial, but the most efficient treatment is to raise the general health of the body and lower the blood pressure by diet, etc. Masturbators are always most addicted to the habit when the physical health is much below par. Vaginal discharges occurring in cases of chronic mania, or in patients after an attack of acute excitement, with or without loss of consciousness, are frequently due to the presence of some foreign body which the patient has introduced into the vagina.

For ordinary conditions of mental excitement, efficient nursing and rest in bed are sufficient to meet the necessities of the symptoms in nine cases out of ten. The excitement may be further benefited by the use of the continuous bath and the



application of cold to the head. Sometimes, however, the patient may be so violent that there is danger of injury both to the patient and to those in charge. In such a case the injection of hyoscine  $\frac{1}{200}$  to  $\frac{1}{70}$  of a gr. may be absolutely necessary. If, as very rarely happens, the extreme violence and excitement be continuous, then recourse must be had to continuous chemical restraint in the form of the bromides and sulphonal. To obtain the best results from these drugs they should be given together in small doses and frequently, so as just to produce the required result without completely stupefying the patient. Macpherson recommends a combination of the bromides and chloral in the treatment of such cases. When sulphonal, which is a dangerous drug, is given in too large doses, the tendon reflexes are abolished, the pupils dilate slightly, and react sluggishly to light. Often it produces sickness, and in a few cases the urine becomes smoky and contains hæmatoporphyrin. Where the consciousness is already clouded, the administration of sulphonal affects the organic reflexes of micturition and defæcation, and the patient becomes wet and dirty in habits. The same symptoms follow the excessive use of the bromides, only they do not produce hæmatoporphyrinurea. Opium is not, in my experience, a satisfactory drug to use for continuous excitement, except in patients suffering from excited melancholia who are past middle life. In such cases the drug is often very useful, and the dose can be gradually reduced. In other cases, again, the quantity of the drug has to be constantly increased to produce the desired action, and if this occurs, the opium should be at once discontinued, otherwise the opium habit is contracted. The recurrent attacks of maniacal excitement, which occur in patients suffering from epilepsy, can sometimes be aborted by putting the patient to bed, and giving a full dose of chloral hydrate and potassium bromide. Others, again, do better under free purgation with croton oil or the administration of a large dose of calomel.

The restlessness and excitement of all chronic cases of insanity, which, I believe, is largely a result of bad brain habit, is best treated by exercise, regular occupation, and the use of a non-stimulating but fattening dietary.



Sleeplessness has been mentioned elsewhere as an omnipresent symptom in all forms of mental disease, and the statement made that in all acute insanities the direct exciting cause is a toxæmia, and in chronic cases a toxæmia plus a bad brain habit. The toxæmia may either act directly on the cortical grey matter of the brain, or indirectly disorder it by producing physical symptoms which of themselves alone are capable of preventing sleep. The outward manifestations of these physical disorders are a high arterial tension and a rapid pulse-rate.

In patients suffering from sleeplessness with high arterial tension and a rapid pulse-rate, it is absolutely necessary to treat these symptoms if one expects the patient to sleep. As these patients are chiefly suffering from metabolic poisoning, every effort must be made to assist elimination of the waste products of the body. The bowels must be cleared out by enemata, and the skin and kidneys stimulated to action by the free administration of fluids. If drugs be necessary,  $\frac{1}{2}$  or 1 gr. dose of erythrol-tetra-nitrate may first be tried, as its action is to reduce arterial pressure. If that fails, then paraldehyde in from  $\text{Zii.}$  to  $\text{Ziii.}$  doses is by far the best drug. Sulphonal and trional should be avoided, as they interfere with the metabolic processes of the body. In patients suffering from excitement with high arterial tension, a bath of from one to two hours at a temperature of  $100^{\circ}$  F. is often successful. If drugs be necessary, again paraldehyde is the best hypnotic. In excitement with sleeplessness, where the arterial tension is low, a hot bath often does good, but if drugs be used, then paraldehyde should be avoided, and sulphonal 30 grs., trional 20 grs., veronal 20 grs., or chloral hydrate used instead. Where sleeplessness is apparently due to a fast, irritable pulse, 30-gr. doses of potassium bromide given at bedtime frequently quiet the pulse, and sleep follows.

Opium, although it lowers the arterial pressure, does not favour elimination; it is, however, a certain hypnotic, and in elderly patients with symptoms of excitement with depression, it is sometimes the only drug of any value in the treatment of the obstinate sleeplessness which complicates these conditions. The more one sees of the effect of drugs used in the treatment



of sleeplessness in insanity, the more disinclined one is to use them. The sleep so obtained is often unrefreshing, and the after-effects of the drugs unpleasant. Paraldehyde being eliminated largely by the lungs for twelve hours after administration, is offensive to the patient, and reduces the appetite by producing a sensation of nausea. Sulphonal and trional, being motor depressants, leave behind them a feeling of lethargy and fatigue, besides interfering with the metabolic processes of the body. Veronal, a new hypnotic, often has the same effect, while chloral hydrate tends to weaken the heart's action and disorders the alimentary tract. Long-continued or excessive administration of the bromides undoubtedly lowers the resistive power of the patient to tubercular infection. In the sleeplessness of old age, alcohol, in the shape of hot toddy, is often a safe and efficient hypnotic. During the last four years I have been struck by the fact how rarely one requires to give hypnotics in the treatment of acute cases of insanity, and how much better they do without them. Warmth to the extremities, hot drinks, and attention to the general physical symptoms already mentioned, prove efficacious in the majority of cases. When drugs are necessary, then a full dose of the hypnotic considered in each case the most suitable is administered, and the dose is not repeated for at least one night; far more frequently two nights are allowed to elapse.

#### THYROID TREATMENT OF INSANITY

Thyroid extract in the treatment of insanity was first used by myself in 1892, because I had noticed that the exhibition of thyroid extract in the treatment of myxœdema produced a condition of fever. Psychiatric literature is full of the records of cases which made good recoveries after suffering from febrile conditions, and I believed that the mild fever excited by the administration of thyroid extract might prove equally beneficial. The patients during treatment were confined to bed, and the extract was given in doses ranging from 45 grs. to 60 grs. per day for seven or eight days continuously. The pharmacological action of the drug when so exhibited is as follows:—In from



two to three days after administration the temperature becomes subfebrile, rarely rising above 100° F., and the duration of the fever is limited to three or four days, even though the drug is continuously administered. After administration has been discontinued it is usual for the temperature to again become slightly febrile for one or two days. Changes in the atmospheric temperature readily affect the body temperature of patients either under treatment, or who have quite recently been under treatment. For instance, if several patients are under treatment at the same time, and the weather becomes warm and close, the temperatures of all these patients will rise simultaneously. The tongue becomes furred, the appetite diminishes, and there is usually thirst. Nausea and vomiting are not unusual complications, and are more readily induced if the dietary contains indigestible substances. The red blood corpuscles and hæmoglobin are both slightly diminished, and the leucocytosis steadily falls, there being a marked decrease of the polymorphonuclear cells, which may fall below 40 per cent. The heart's action becomes rapid and then irregular, and the arterial tension falls as much as 10 or 20 mm. Hg.

The respiratory system is not markedly affected, but if the patient suffers from latent phthisis a course of the drug is almost certain to light up active disease, and for this reason it should not be given where the patient is phthisical. In large doses thyroid extract does not act as a diuretic. In fifteen cases in which the urine was examined there was neither increased nor diminished excretion of urine, but there was a slight rise in the excretion of urea.

In many instances the voluntary muscles presented symptoms of overaction; fine fibrillary muscular tremors were observed in the tongue, lips, facial muscles, and limbs. The facial and lingual tremors resemble those seen in general paralysis.

Flushings of the skin, profuse perspirations, and rapid loss of body weight were very constant symptoms. During convalescence a rapid gain in weight was the rule. Headaches of more or less severity were frequently complained of, and were accompanied by pains in the muscles and joints. These symptoms were relieved by the administration of mild purgatives.



The mental effect of the drug is very various. Some patients become depressed, others emotional, while some become maniacal. The explanation of these varying effects is, I believe, that a course of thyroid frequently reproduces for a short time the earlier acute symptoms of the mental disease from which the patient originally suffered.

Is thyroid a direct brain stimulant? Every alienist has noticed, and many have commented upon, the improvement observable even in demented cases, during some acute intercurrent disease. The patient becomes talkative, takes an interest in his surroundings, and there may be some amelioration of bad habits, etc. In many instances these symptoms are particularly noticeable in patients during thyroid feeding. A patient under treatment had been insane for at least nine months. He appeared to be somewhat demented on admission, and had steadily become more confused and foolish. Three days after thyroid treatment was commenced, he became more lively in appearance, answered questions sharply, and proved clearly that during his illness his cortical cells had been receiving and retaining impressions from without. On the fourth day he was singing and obviously elated, and although he spoke fairly sensibly and denied several delusions expressed on admission, he was still very insane and in a state of mania. Thyroid in this case appeared to act as a cortical stimulant. On another occasion there were three cases of mania under treatment whose acute symptoms had been replaced for several months by those of secondary stupor or approaching dementia. They all during treatment again became maniacal. One passed from this induced mania into convalescence and recovery. The other two relapsed to their former condition. Here again the symptoms of cortical excitation were obvious, and in each case quite out of proportion to the febrile condition induced. I have seen at least a dozen cases with symptoms as acute as those quoted, and the impression they made on my mind was that thyroid is a direct brain stimulant. On the other hand the lowering of the leucocytosis may permit of the toxæmia, which, as I have shown elsewhere, is continuous in cases of mania, temporarily gaining the upper hand.

Does the ingested thyroid extract supply some material to



the body which the gland is supplying in deficient quantity? The period of physiological activity of the thyroid gland is different in the two sexes. In males the thyroid attains its full development at adolescence and then atrophies. During puberty and early adolescence in males the thyroid gland frequently becomes enlarged as if functional activity was then increased. In females the thyroid attains full development after menstruation is thoroughly established, and it appears to remain functionally active when any call is made on the bodily economy up to the period of the grand climacteric, when the gland gradually atrophies. In women the thyroid plays a special part in the metabolism of the sexual organs, and I have frequently seen enlargement of the thyroid in puerperal, lactational, and climacteric cases, so it is probable that this enlargement corresponds to increased functional activity at these periods.

Thyroid extract administered continuously in either small or large doses is a dangerous drug if due precautions are not taken. If the patient be not confined to bed there is an undoubted risk of heart failure. The stomach readily becomes disordered if butcher meat and vegetables be not excluded from the dietary. If the patient be the subject of phthisis, then the administration of the drug produces an exacerbation of that disease. Occasionally a patient, instead of becoming feverish and perspiring freely, passes into a curious toxic state. The temperature becomes subnormal, the skin dry and harsh, and the pulse thready and quick. A large hot drink and packing with hot bottles invariably relieve this condition.

To what class of case is the treatment applicable? It is only applicable to those patients whose symptoms are not acute. A patient who, after an attack of acute mania, passes into a state of stupor, and under suitable treatment does not tend to recover, but remains stuporose, is a typical case for thyroid treatment. The same remark applies to cases of melancholia where the symptoms remain too long subacute. In acute and recent disease thyroid is too much of a cortical excitant to be beneficial. In such cases the brain is in a state of hyperexcitability, and the administration of thyroid tends to aggravate rather than alleviate the condition. This,



at least, has been my experience in the treatment of cases of recent and acute disease. The drug is therefore especially indicated in all cases of secondary stupor. Thyroid treatment has definitely shown us that, in our present state of knowledge, the diagnosis between stupor and dementia is often imperfect. There are well-recognised clinical varieties of stupor which cannot be mistaken, just as typical dementia cannot be mistaken. There are, however, many half-way cases where it is impossible to say where stupor ends and dementia begins. Now stupor is curable while dementia is incurable. The administration of thyroid in such a case is generally diagnostic. The stuporose patient wakens up, the demented patient does not, though there may be slight temporary improvement. During the actual administration of the drug, and period of fever and malaise, many cases show undeniable improvement, in some an actual recovery, in others a steady return or awakening of the mental faculties, which culminates in recovery during the period of reaction following treatment. In others the effect of treatment is not noticeable until the period of reaction is well advanced.

Case:- A.L. Melancholia. Duration of Illness:- 2 years.

Result:- Recovered.

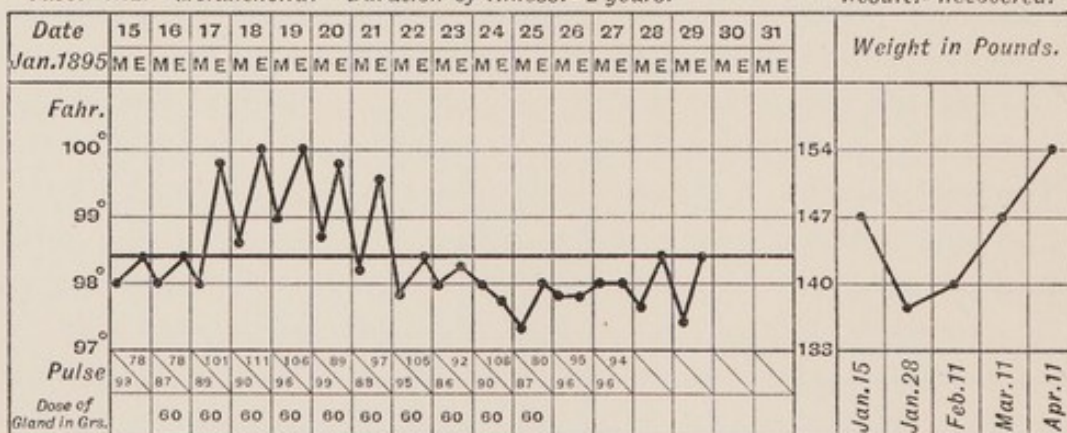
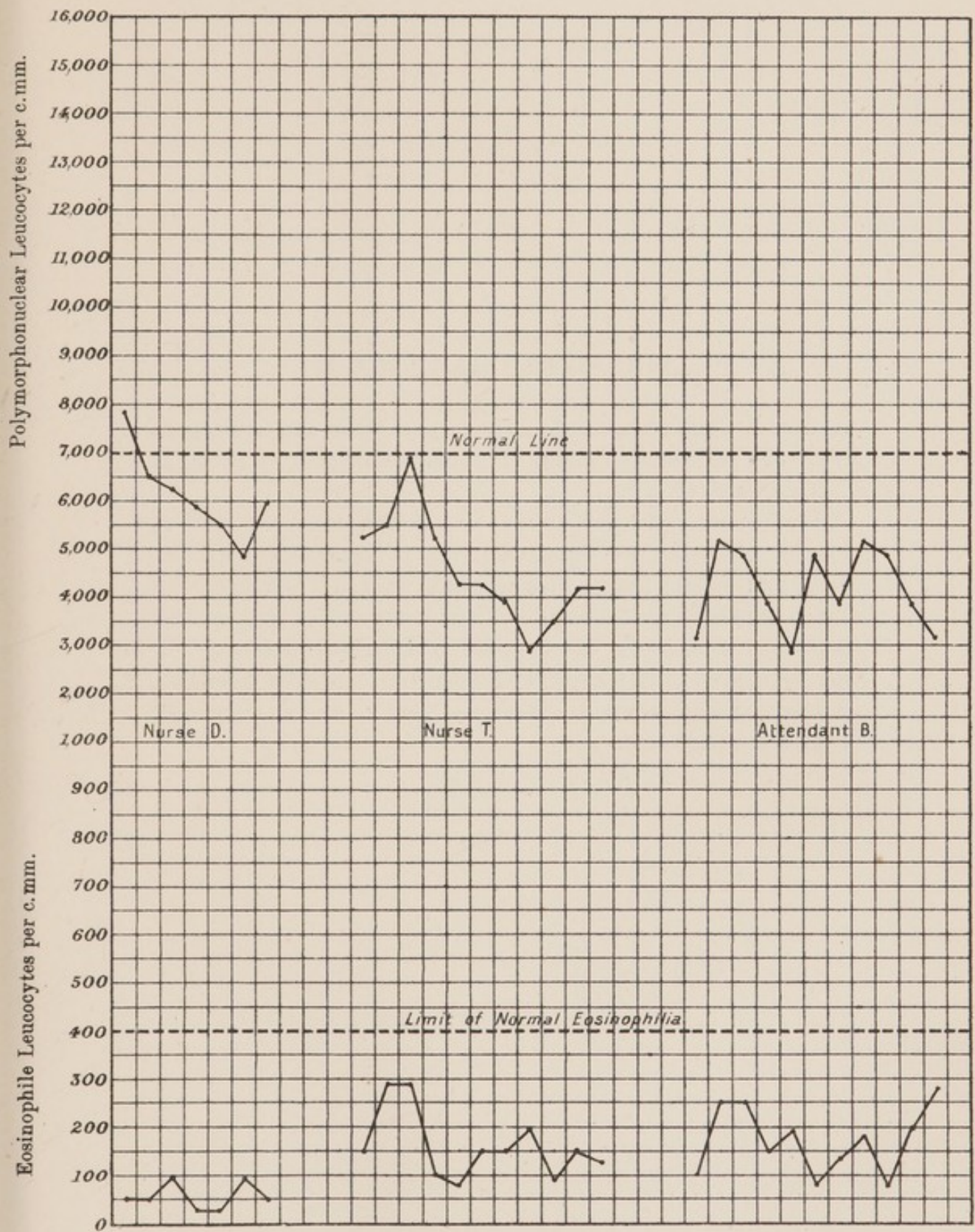


Chart showing Typical Temperature, Pulse, and Duration of Treatment. The loss of weight and subsequent gain in weight are also shown diagrammatically.



CHART No. 1.



Control Leucocyte Chart showing the Leucocytosis in members of the Staff.

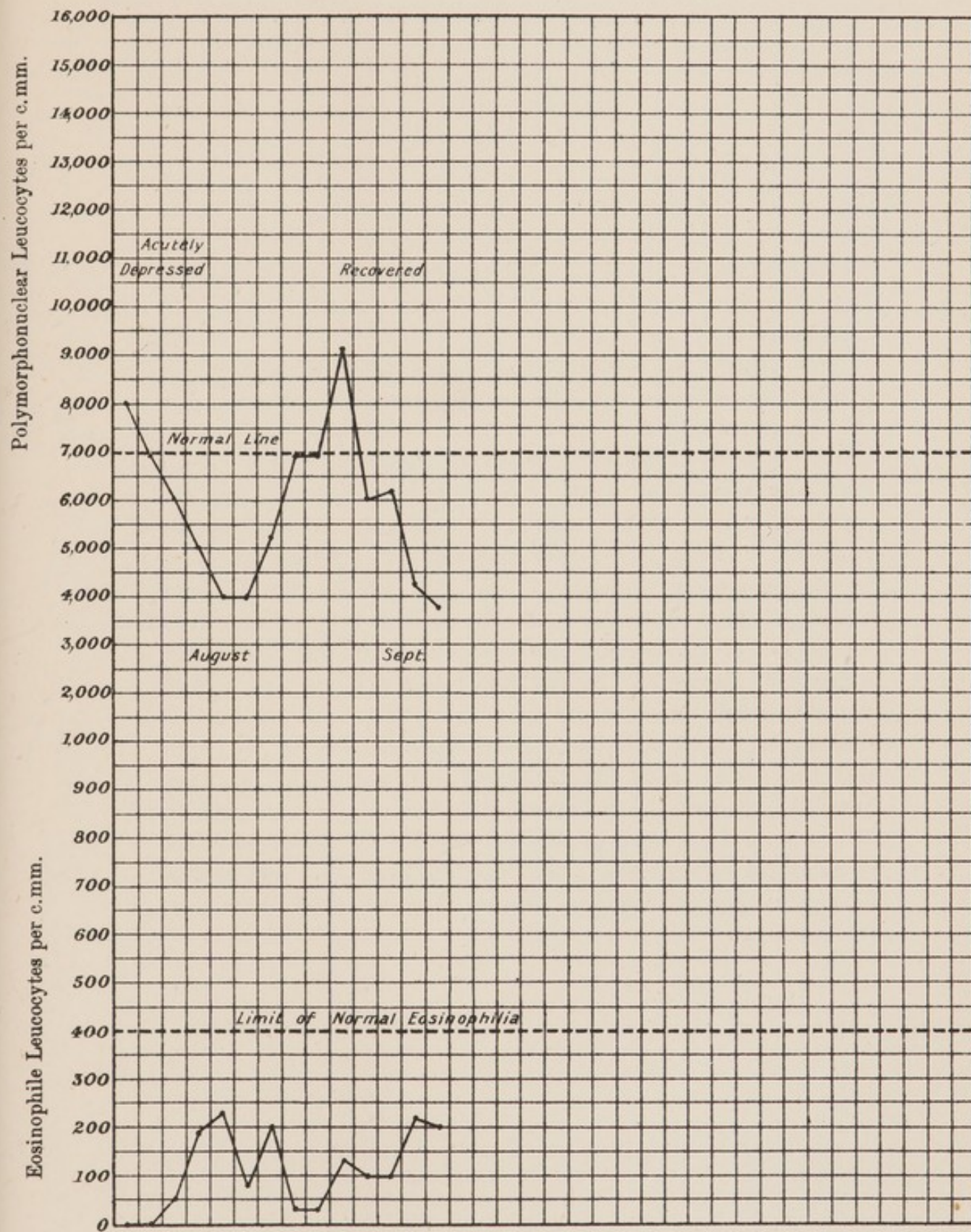


## LEUCOCYTE CHART NO. II.

LEUCOCYTE Chart showing the Polymorphonuclear and Eosinophile Leucocytes per c.mm. in a case of Acute Melancholia occurring in a female, aged 42.

The patient, who had shown mental symptoms for a week prior to admission, was in a state of acute depression with confusion, and suffered from hallucinations of sight and hearing. This acute stage lasted for five days. Then followed five days of confusion and exhaustion, which passed into complete recovery. It will be noticed that the polymorphonuclear leucocytes were only slightly increased on two occasions, and that there was no increase of the eosinophile leucocytes.

CHART No. II.



A Case of Acute Melancholia which recovered.

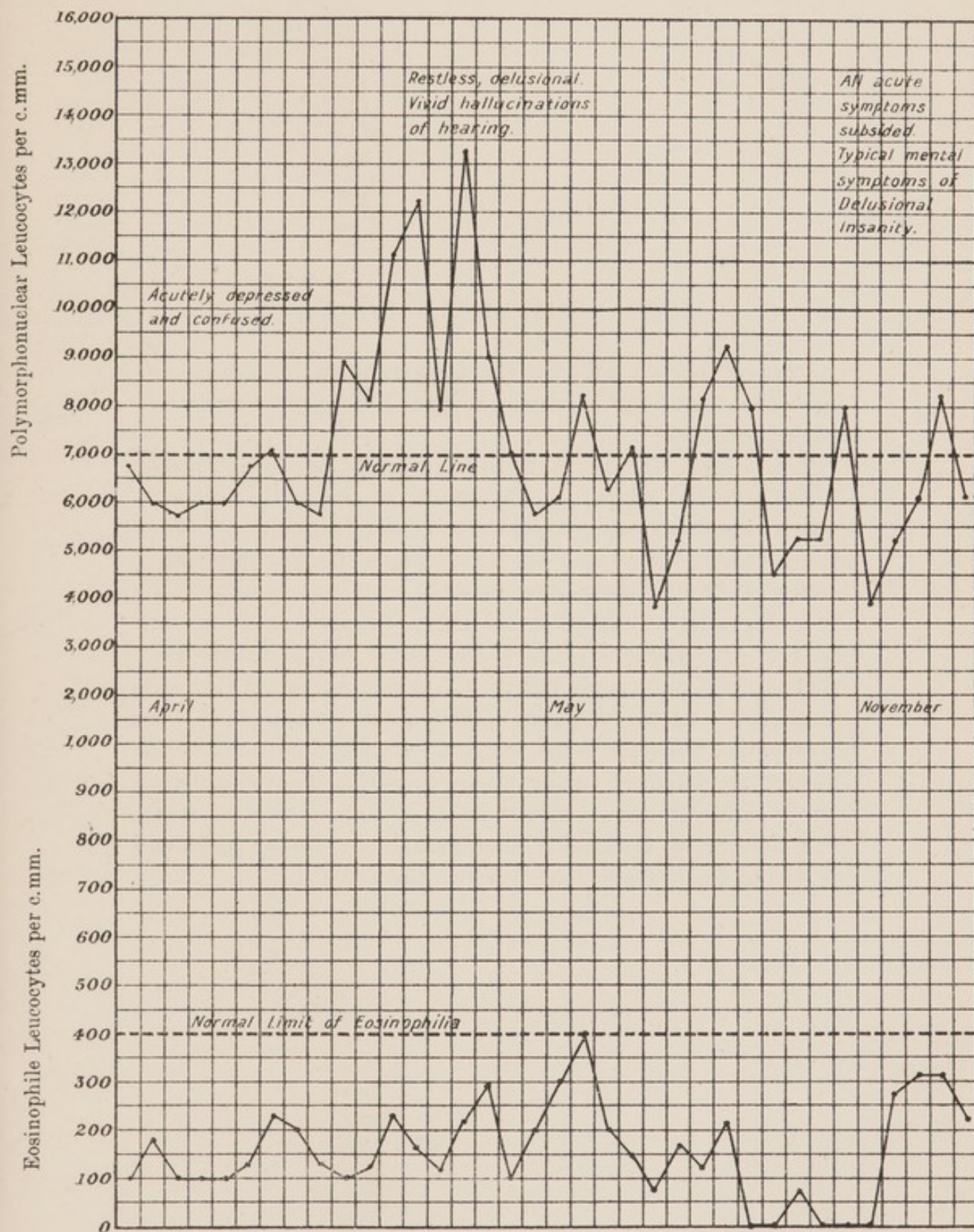


### LEUCOCYTE CHART NO. III.

LEUCOCYTE Chart showing the Polymorphonuclear and Eosinophile Leucocytes per c.mm. in a case of Delusional Insanity occurring in a female aged 51.

The patient had suffered from depression with delusions of suspicion for some three months prior to admission. On admission she presented all the physical and mental symptoms of an attack of acute melancholia, and these symptoms lasted during the greater part of April. Towards the end of April the confusion passed off, the patient slept better, and the appetite improved. During the acutely depressed and confused stage the polymorphonuclear leucocytosis was low, but as the confusion passed off there was a distinct rise in the polymorphonuclear leucocytes, unaccompanied, however, by any increase of the eosinophile leucocytes. During May, June, and July the patient was delusional; she thought that the nurses were wearing her clothes, and she affirmed that many of the ornaments in the wards had been stolen from her house. These delusions of suspicion, however, gradually gave way to delusions of grandeur, and by November her appearance, gait, and speech were all characteristic of delusions of grandeur. She now believes that the asylum belongs to her, and she also believes that the other patients are her boarders.

CHART No. III.



A Case of Delusional Insanity which had a commencement in a state resembling acute melancholia.

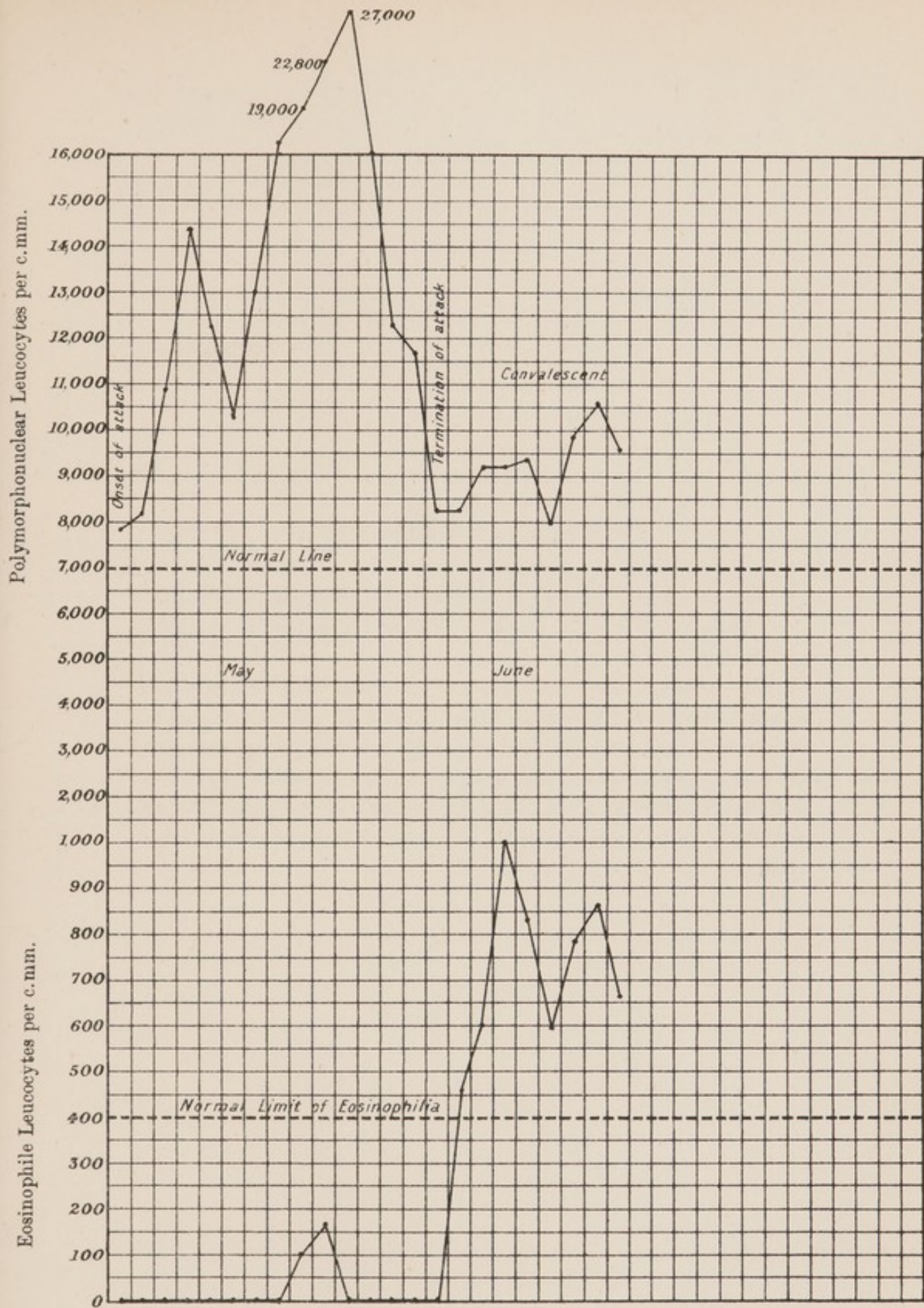


## LEUCOCYTE CHART NO. IV.

LEUCOCYTE Chart showing the Polymorphonuclear and Eosinophile Leucocytes per c.mm. in a case of Excited Melancholia occurring in a female aged 39.

The patient, who had been admitted some months previously, had had several relapses. The attack, of which the leucocyte chart is an index, commenced early in May, culminated during the middle of May, and then gradually subsided, the patient passing into a state of comparative sanity.

At the onset of the attack the polymorphonuclear leucocytosis was low, but quickly rose above the normal line, and there was a marked hyperleucocytosis during the whole period of the acute symptoms. During the same period there were practically no eosinophile leucocytes to be found in the blood films examined. During the period of convalescence the polymorphonuclear leucocytosis gradually rose again, but the most marked increase was to be noticed in the eosinophile leucocytosis,—a symptom which will be noticed to recur again and again in these charts.



A Case of Excited Melancholia in a female aged 39.

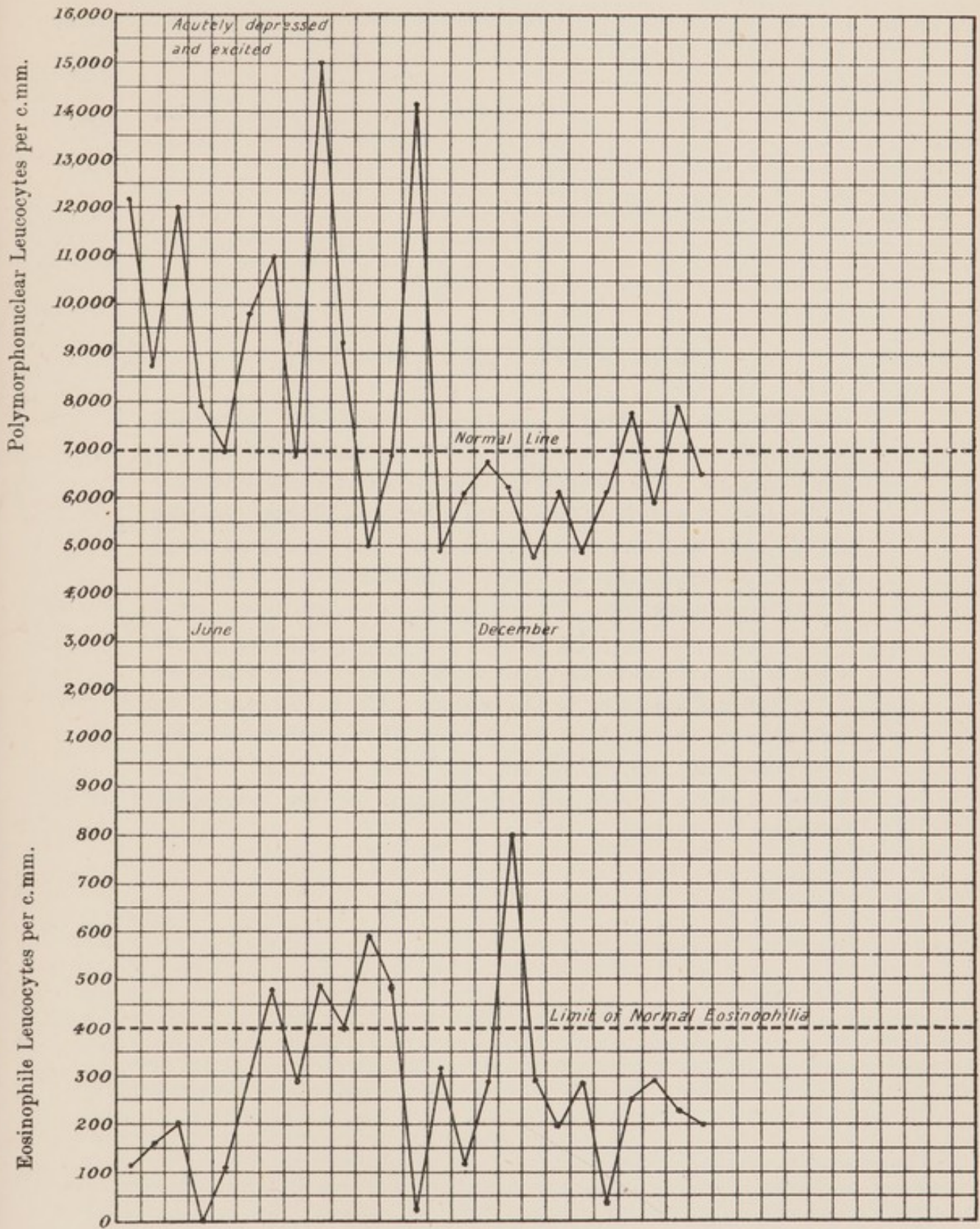


### LEUCOCYTE CHART NO. V.

LEUCOCYTE Chart showing the Polymorphonuclear and Eosinophile Leucocytes per c.mm. in a case of Excited Melancholia occurring in a female aged 63.

The patient, who was admitted in June, had suffered from mental symptoms for two months. The high polymorphonuclear leucocytosis gradually subsided. During July the patient presented the usual physical and mental symptoms characteristic of chronicity, and the leucocytosis taken again in December of the same year was uniformly low. There was no increase of the eosinophile leucocytes.

CHART No. V.



A Case of Excited Melancholia in a female aged 63, which passed into a state of chronic restlessness and mal-nutrition.



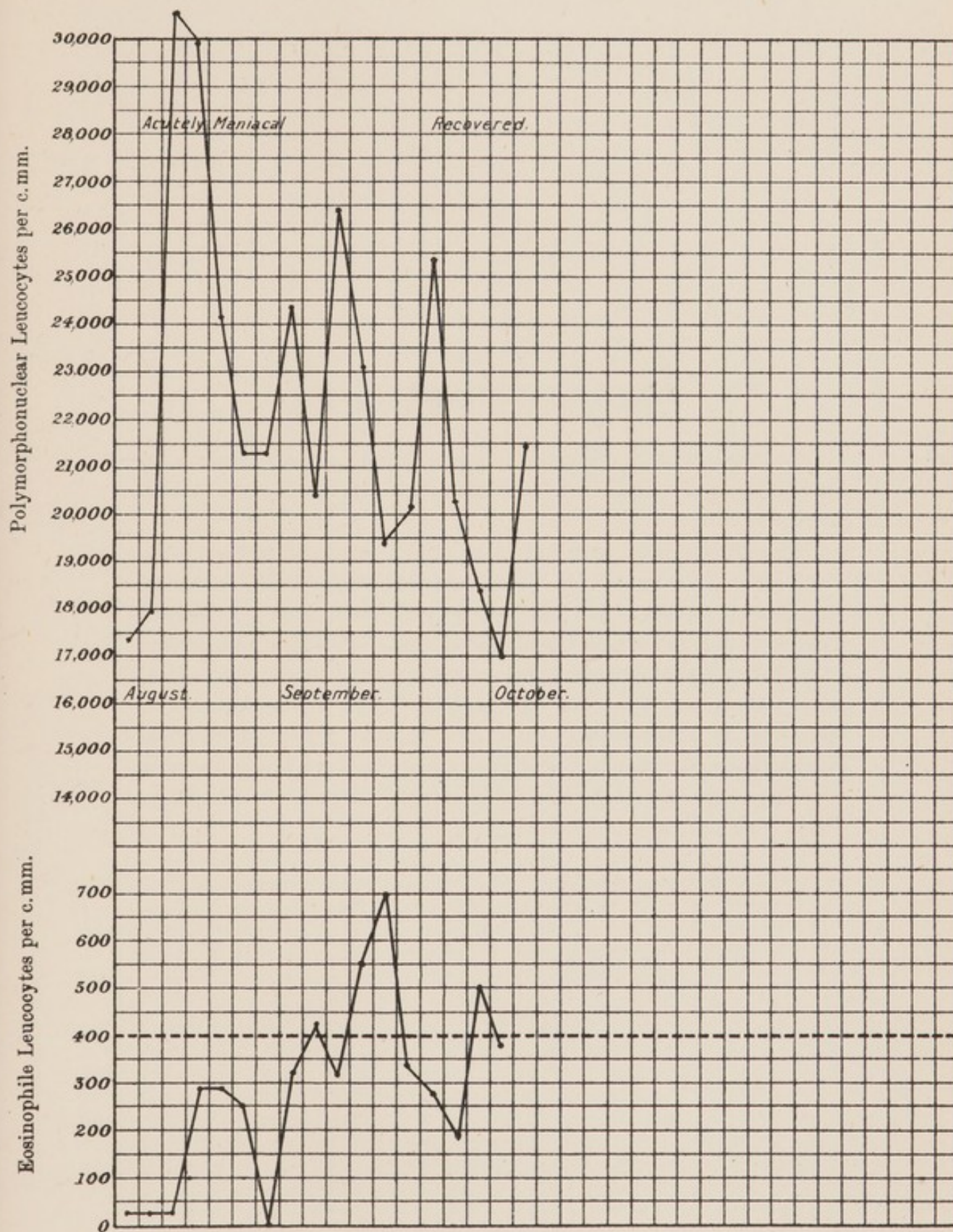
## LEUCOCYTE CHART NO. VI.

LEUCOCYTE Chart showing the Polymorphonuclear and Eosinophile Leucocytosis per c.mm. in a case of Acute Mania occurring in a female aged 29. The exciting cause of the attack was childbirth.

The acute mental symptoms lasted a week, and during the latter half of August and the whole of September the patient was to all outward appearances sane and perfectly recovered.

The hyperleucocytosis in the polymorphonuclear leucocytes is most marked, not only during the period corresponding to the period of the acute mental symptoms, but throughout the remainder of the patients' residence in the asylum. Up to the date of her discharge in the middle of October the polymorphonuclear leucocytosis only once fell as low as 17,000 per c.mm. During September there was a decided increase in the eosinophile leucocytes.

CHART No. VI.



A Case of very Acute Mania (puerperal) which made an excellent recovery.



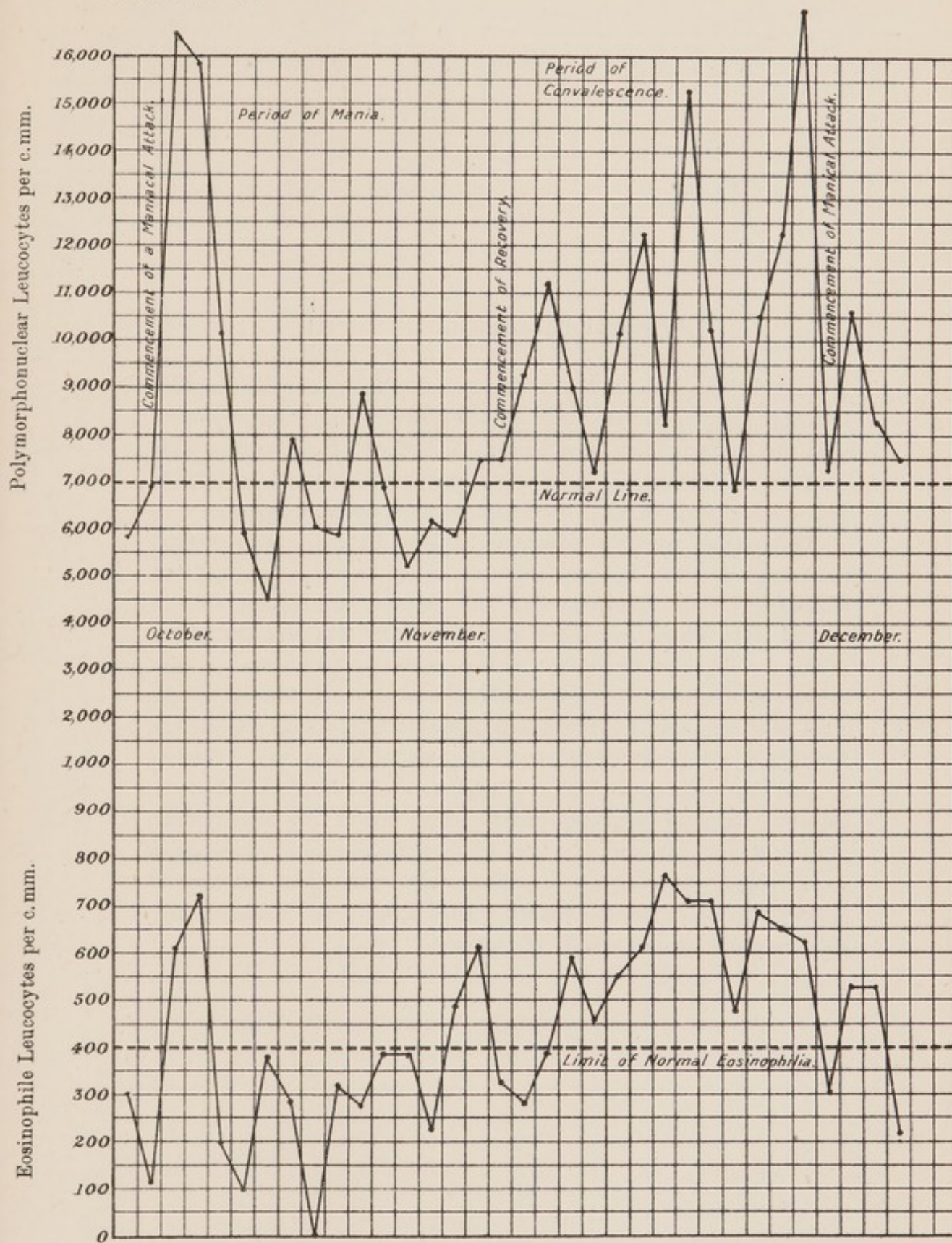
### LEUCOCYTE CHART NO. VII.

LEUCOCYTE Chart showing the Polymorphonuclear and Eosinophile Leucocytes per c.mm. in a case of Recurrent Mania in a female aged 45.

The patient had as a rule one attack every six weeks or two months. The attack of which the leucocyte chart is shown commenced with a fall in the polymorphonuclear leucocytes, followed by a rise which lasted three days. For the next twelve days the polymorphonuclear cells were low, and during this period the patient was confused and excited. The commencement of recovery is accompanied by a rise in the polymorphonuclear leucocytosis, and also by a decided increase in the eosinophile cells.

The commencement of another relapse which set in a fortnight later is also shown in the chart.

CHART No. VII.



A case of Recurrent Mania.



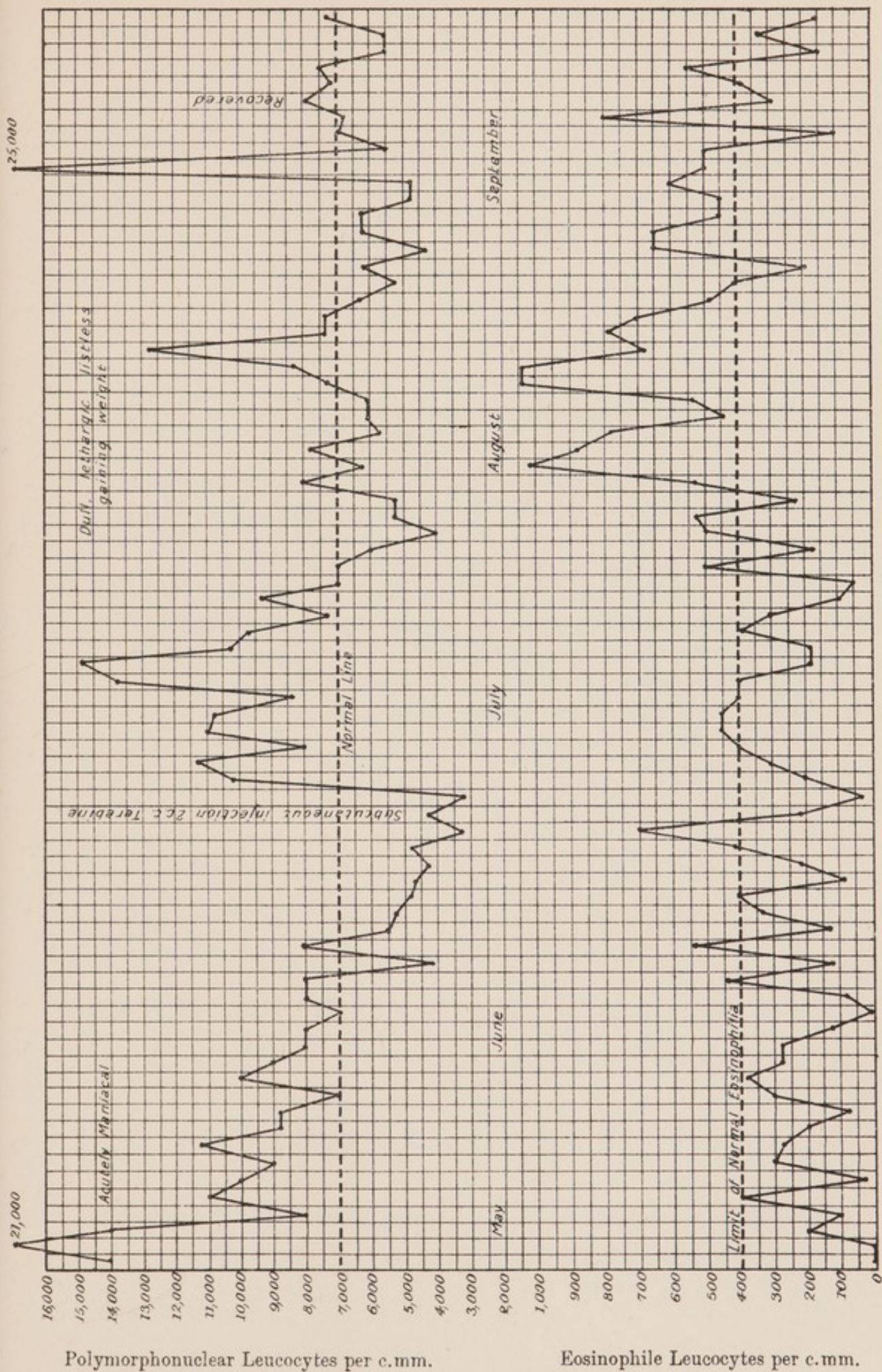
### LEUCOCYTE CHART NO. VIII.

LEUCOCYTE Chart showing the Polymorphonuclear and Eosinophile Leucocytes per c.mm. in a case of Acute Mania occurring in a male aged 22. The chart also shows the effect of a subcutaneous injection of terebene in stimulating the production of leucocytes. The acute symptoms had lasted for two days upon admission.

The patient, who was admitted in May, showed a well-marked polymorphonuclear hyperleucocytosis. By the end of June the leucocytosis had subsided, and the patient had passed into a mental state which suggested chronicity; he was wet and dirty in his habits, and often destructive. On the 30th June a subcutaneous injection of 2 c.c. terebene was given in the left flank. This was followed by a marked increase of polymorphonuclear leucocytes and an improvement in the mental symptoms. During August the patient was dull, confused, and somewhat incoherent in speech. He slept probably twenty hours out of the twenty-four. During this month it will be noticed that the eosinophile leucocytes were greatly increased; simultaneously with this increase of eosinophile leucocytes the patient gained weight, and made a fairly good recovery by the middle of October.



CHART No. VIII.



A Case of Acute Mania in a male, aged 22, who threatened to become demented, but eventually made a fairly good recovery.

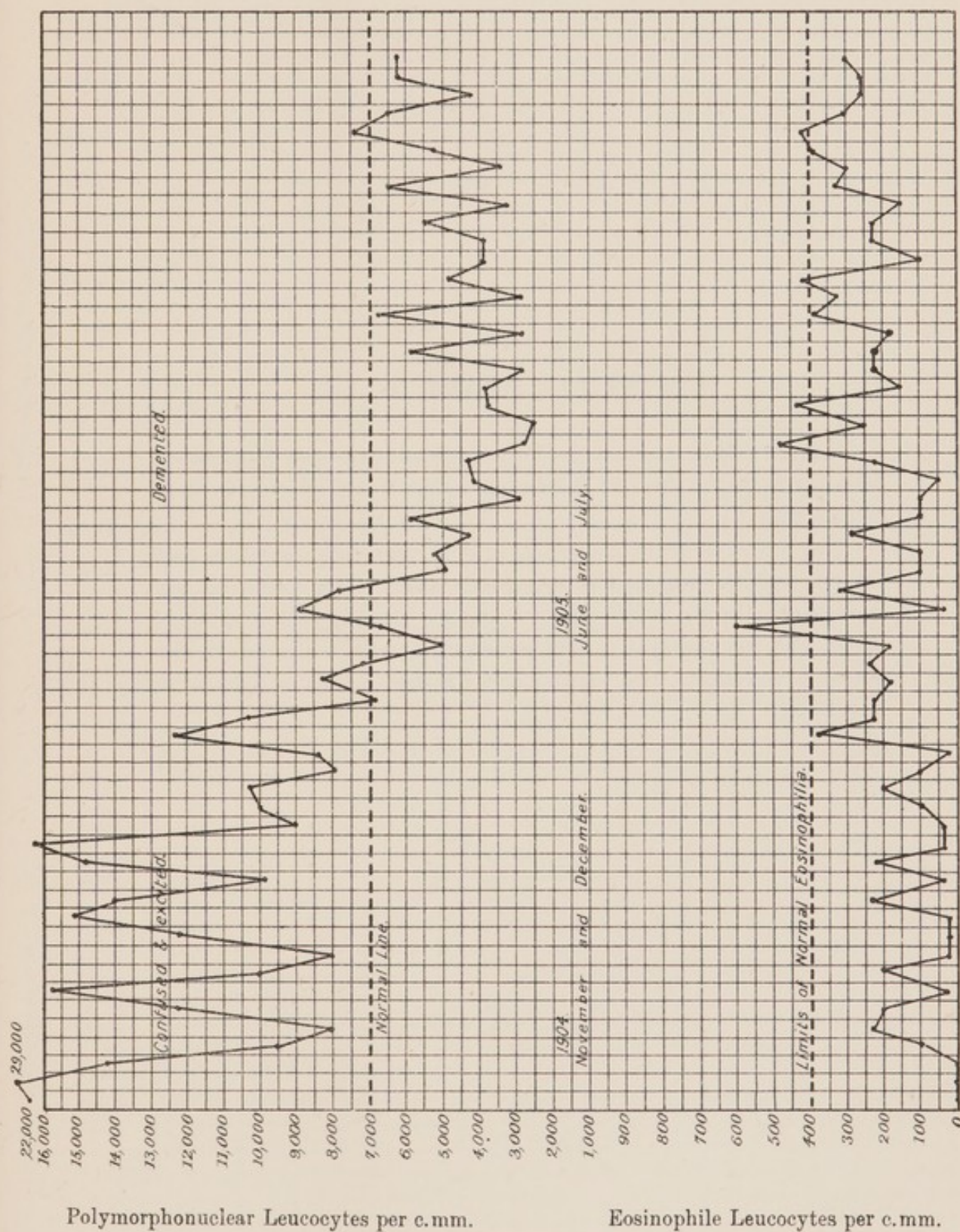


### LEUCOCYTE CHART NO. IX.

LEUCOCYTE Chart showing the Polymorphonuclear and Eosinophile Leucocytes per c.mm. in a case of Acute Mania occurring in a female aged 41.

The patient was admitted in November 1904, having been acutely ill for two days. The polymorphonuclear leucocytosis was well maintained for the first eight months after admission, and the patient improved, but very slowly. Then the leucocytosis fell, and the patient remains in a state of mild confusion with every now and then a slight lighting up of acute symptoms which take the form of hallucinations of hearing. There has been no increase of the eosinophile cells.

CHART No. IX.



A Case of Acute Mania which took nearly a year to become demented.

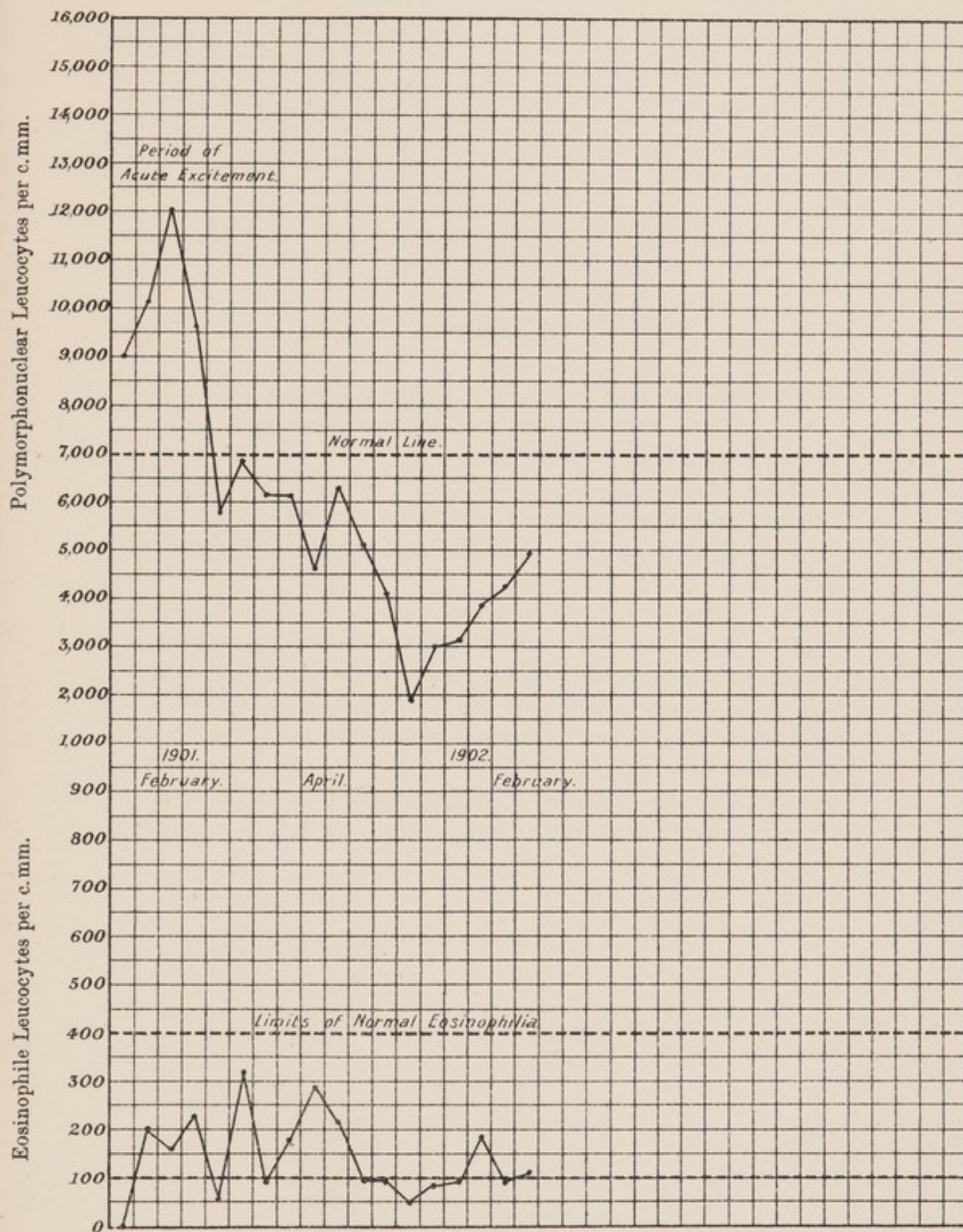


### LEUCOCYTE CHART NO. X.

LEUCOCYTE Chart showing the Polymorphonuclear and Eosinophile Leucocytes per c.mm. in a case of Acute Mania occurring in a female aged 41.

The period of acute excitement was of very short duration, and for one day the patient was apparently sane. The following day confusion returned again, the leucocytosis steadily fell, and there never was any increase in the eosinophile cells. The chart also shows a record of the leucocytosis for one month a year after admission.

CHART No. X.



A Case of Acute Mania, which rapidly passed into a hopeless condition.



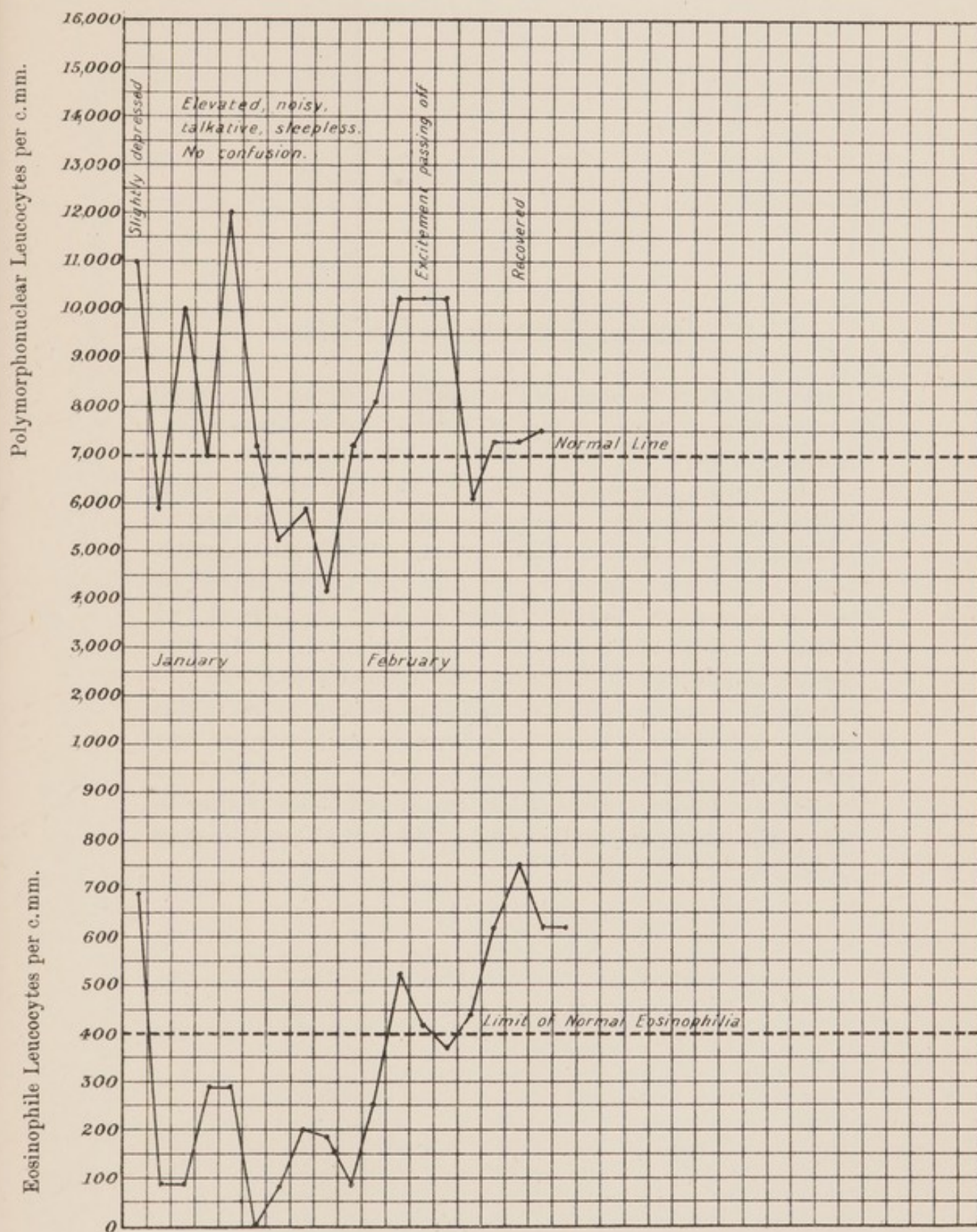
## LEUCOCYTE CHART NO. XI.

LEUCOCYTE Chart showing the Polymorphonuclear and Eosinophile Leucocytes per c.mm. in a case of Recurrent Excitement without confusion occurring in a male aged 56.

The patient first developed the disease in adolescence, and had suffered from repeated attacks of excitement ever since. The attacks occurred at irregular intervals, but rarely lasted longer than six weeks.

The leucocytosis corresponds very closely to that of the chart representing the leucocytosis of a recurrent case of acute mania; there is the same rise of the polymorphonuclear leucocytes, with a subsequent fall succeeded by a rise as the excitement passed off but between attacks the leucocytosis rarely rises above the normal line. The eosinophile leucocytes were low during the period of excitement, but they became markedly increased during the period of convalescence and recovery.

CHART No. XI.



A Case of Recurrent Excitement without Confusion.

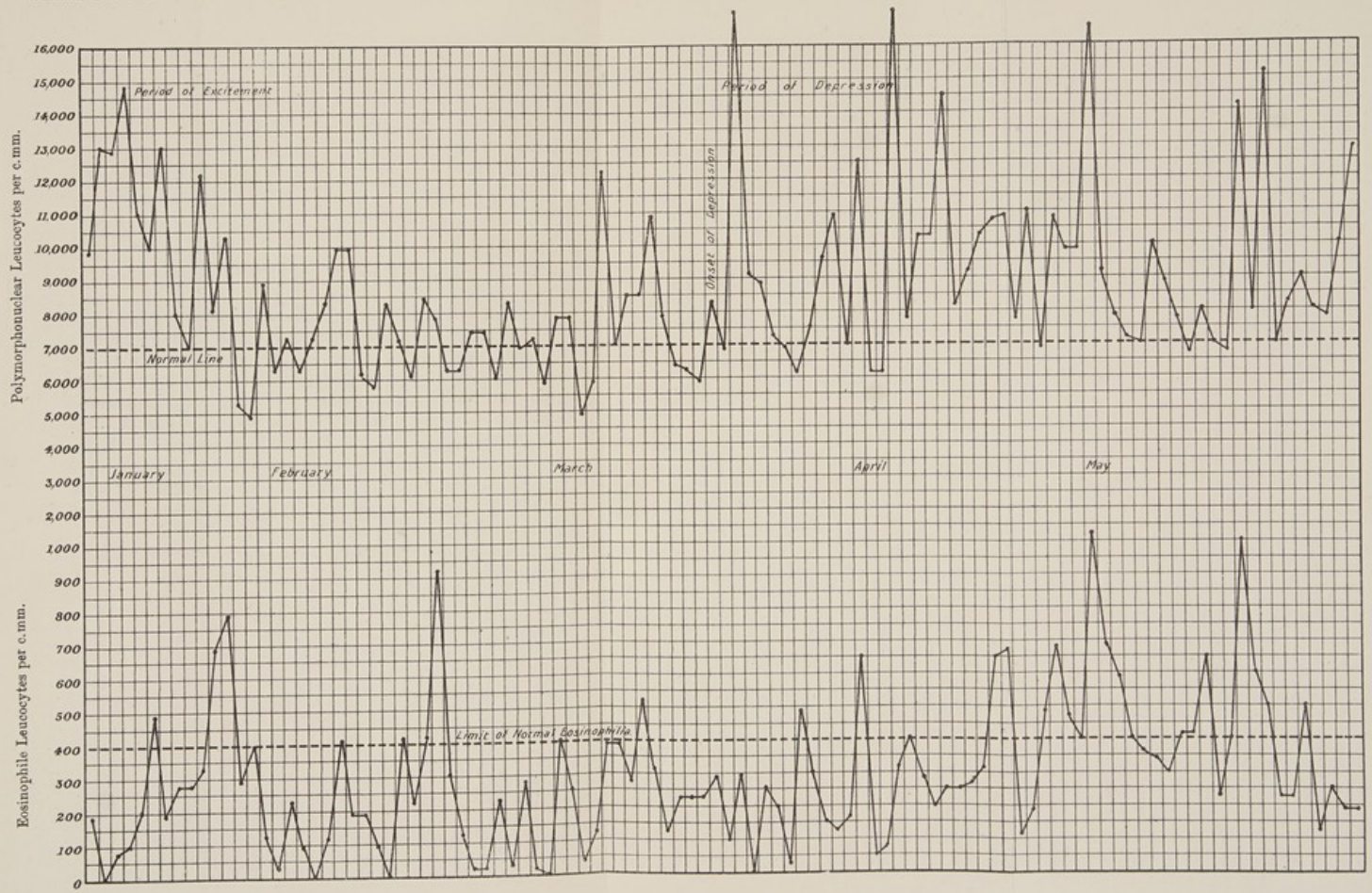


## LEUCOCYTE CHART NO. XII.

LEUCOCYTE Chart showing the Polymorphonuclear and Eosinophile Leucocytes per c.mm. in a case of Folie Circulaire occurring in a female aged 39.

The patient, who was admitted in January 1904, was acutely excited for two months. The polymorphonuclear leucocytosis was at first high, but subsided as the acuteness of the mental symptoms passed off. During March she was dull and without energy, and laid on a stone in weight. Early in April she suffered from œdema of the feet and hands, and passed into a state of depression. The polymorphonuclear leucocytes rose simultaneously with these symptoms. During April, May, and June she had several of these attacks, each of which lasted for a few days, and corresponding to each attack of depression there was a rise of polymorphonuclear leucocytes. During the end of May and the beginning of June the eosinophile leucocytes also increased. The patient was discharged in October.

CHART No. XII.



A Case of Folie Circulaire.



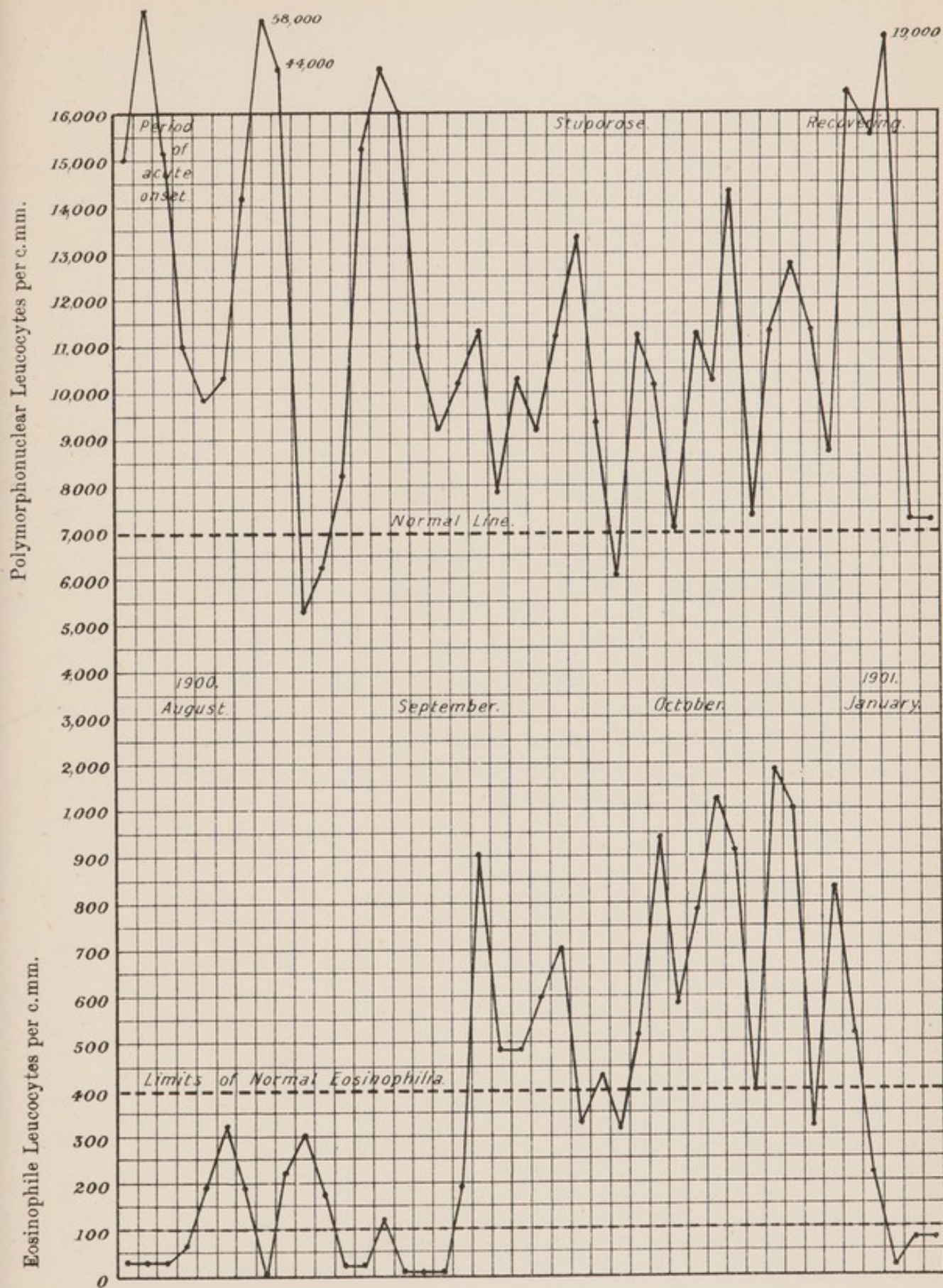
### LEUCOCYTE CHART NO. XIII.

LEUCOCYTE Chart showing the Polymorphonuclear and Eosinophile Leucocytes in a case of Katatonia occurring in a female aged 20.

The disease passed through an acute stage lasting seven days. The polymorphonuclear leucocytes then suddenly rose to 58,000 per c.mm., and on the following day to 44,000 per c.mm., and then dropped to 5000. The patient passed into a state of stupor. During the stuporose stage, which lasted during September, October, November, and December, the polymorphonuclear leucocytes were uniformly high and the eosinophile cells markedly increased. Upon recovery taking place in January 1901 the polymorphonuclear leucocytes again rose.

Compare this chart with that of the case of katatonia which did not recover.

CHART No. XIII.



A Case of Katatonia which recovered.

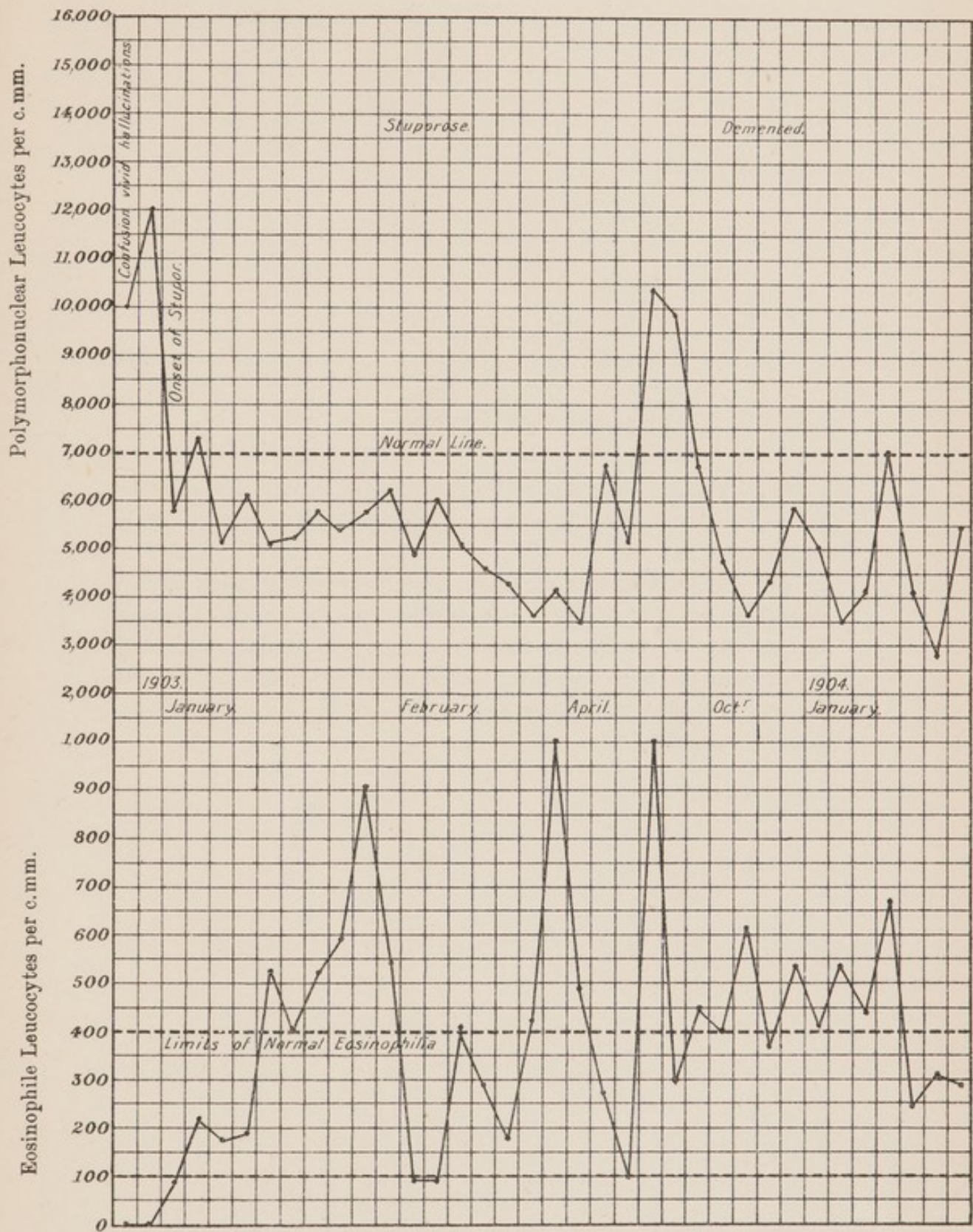


### LEUCOCYTE CHART NO. XIV.

LEUCOCYTE Chart showing the Polymorphonuclear and Eosinophile Leucocytes per c.mm. in a case of Katatonia occurring in a male aged 25.

The patient had been ill some three weeks upon admission, and almost immediately passed into a state of stupor. The polymorphonuclear leucocytosis, which rose slightly just before the onset of stupor, thereafter fell and practically never rose again. The patient passed from stupor into dementia. In this case also there is a marked rise of eosinophile cells.

CHART No. XIV.



A Case of Katatonia, which became demented.

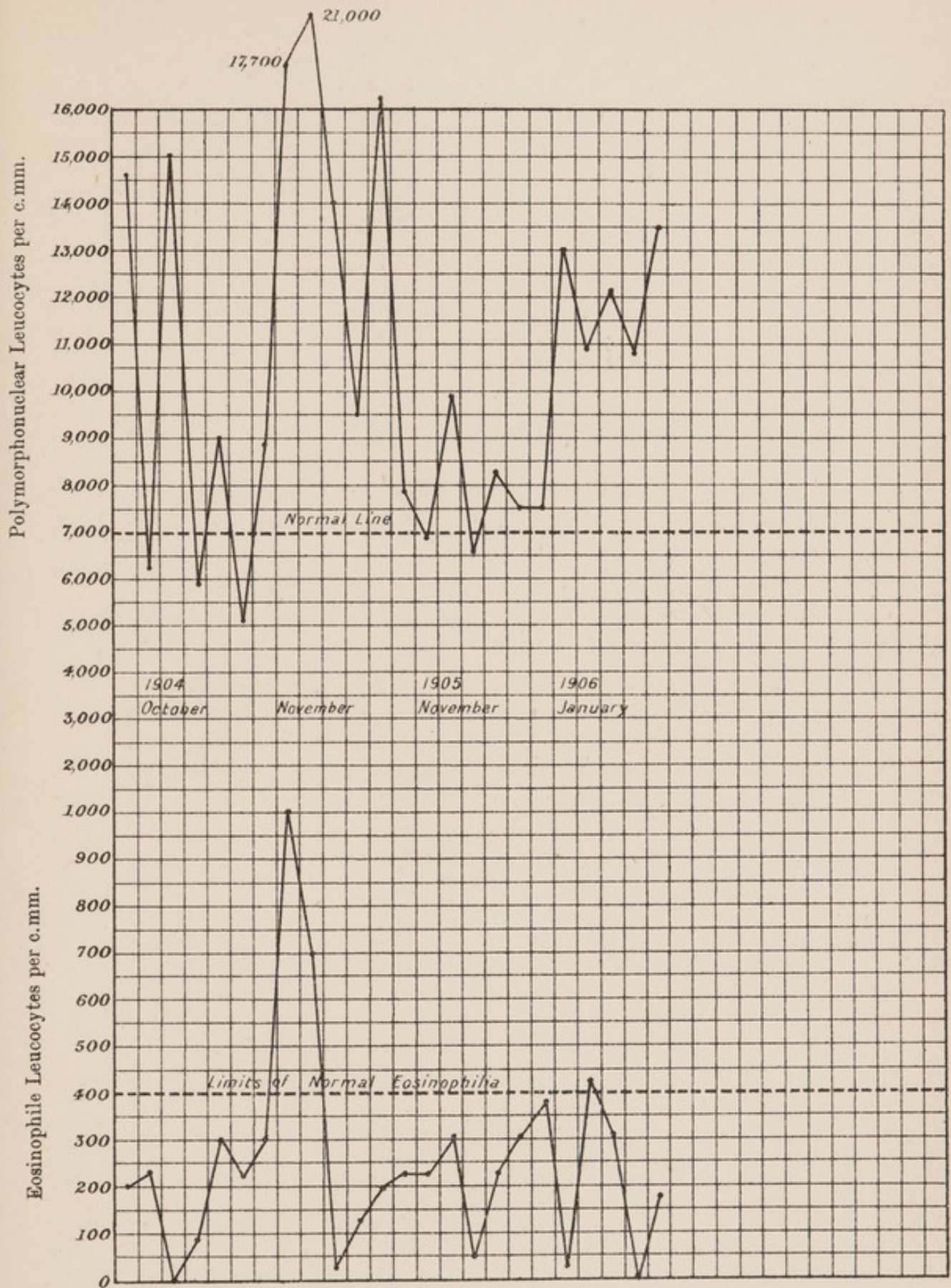


### LEUCOCYTE CHART NO. XV.

LEUCOCYTE Chart showing the Polymorphonuclear and Eosinophile Leucocytes per c.mm. in a case of Hebeephrenia occurring in a female aged 25.

The patient, who was admitted in July 1904, had been ill for several years. It will be noticed that the polymorphonuclear leucocytosis is constantly above the limits of health, and that the eosinophile cells only rose temporarily on one occasion. The patient is constantly restless and unable to apply herself to any form of occupation.

CHART No. XV.



A Case of Hebeephrenia in a female aged 25.

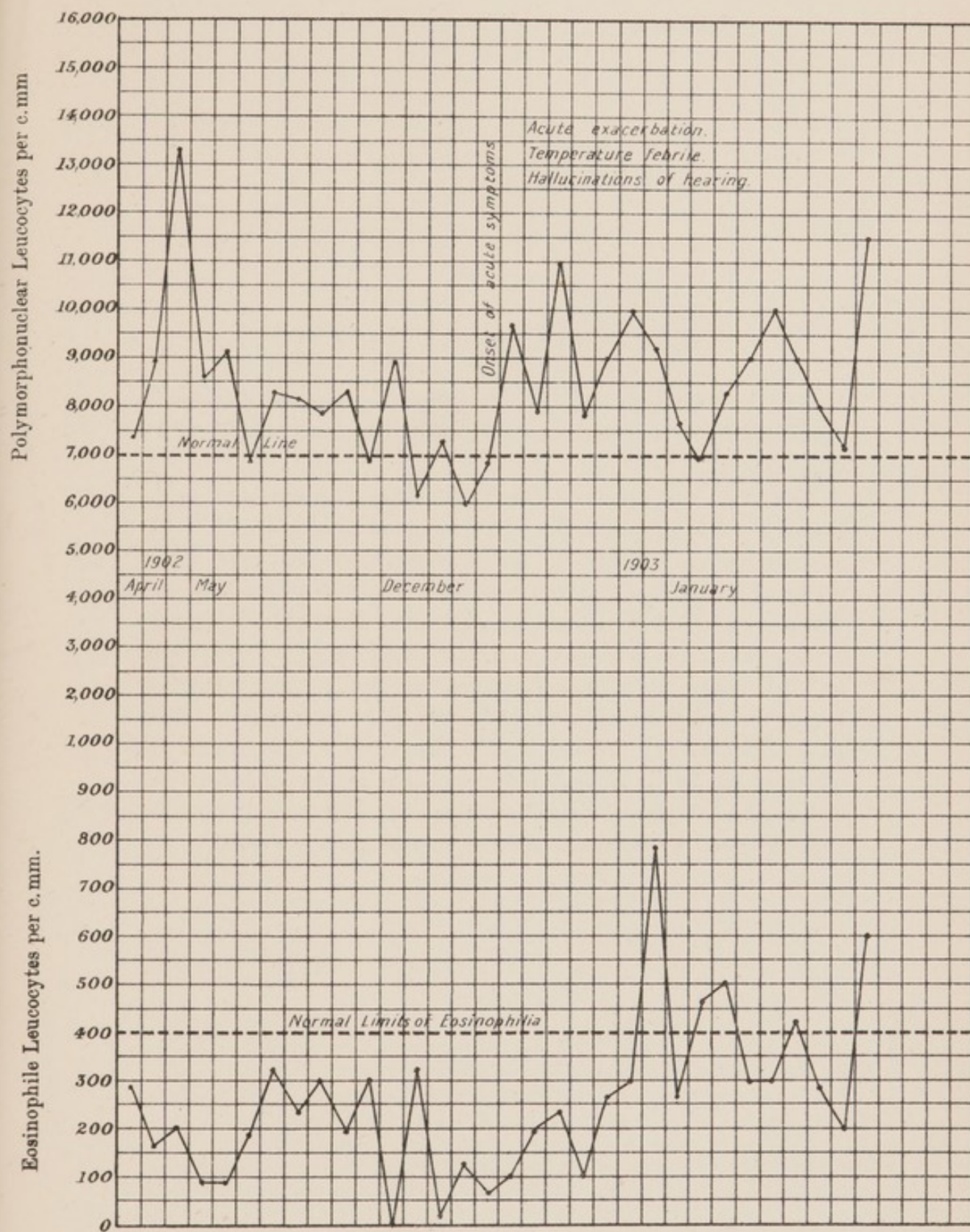


### LEUCOCYTE CHART NO. XVI.

LEUCOCYTE Chart showing the Polymorphonuclear and Eosinophile Leucocytes per c.mm. in a case of Hebephrenia occurring in a female aged 19.

The patient had presented mental symptoms for more than a year upon admission, and the first portion of the chart, taken during April, May, and December 1902, represents a period of the disease which was not characterised outwardly by acute symptoms, either mental or physical. At the end of December the temperature rose, and the patient suffered from hallucinations of hearing and was also confused. During this attack the polymorphonuclear and eosinophile leucocytosis was decidedly increased, the rise of the eosinophile cells being subsequent to that of the polymorphonuclear, and corresponds to the gradual passing off of the acute mental symptoms.

CHART No. XVI.



A Case of Hebephrenia in a female aged 19.

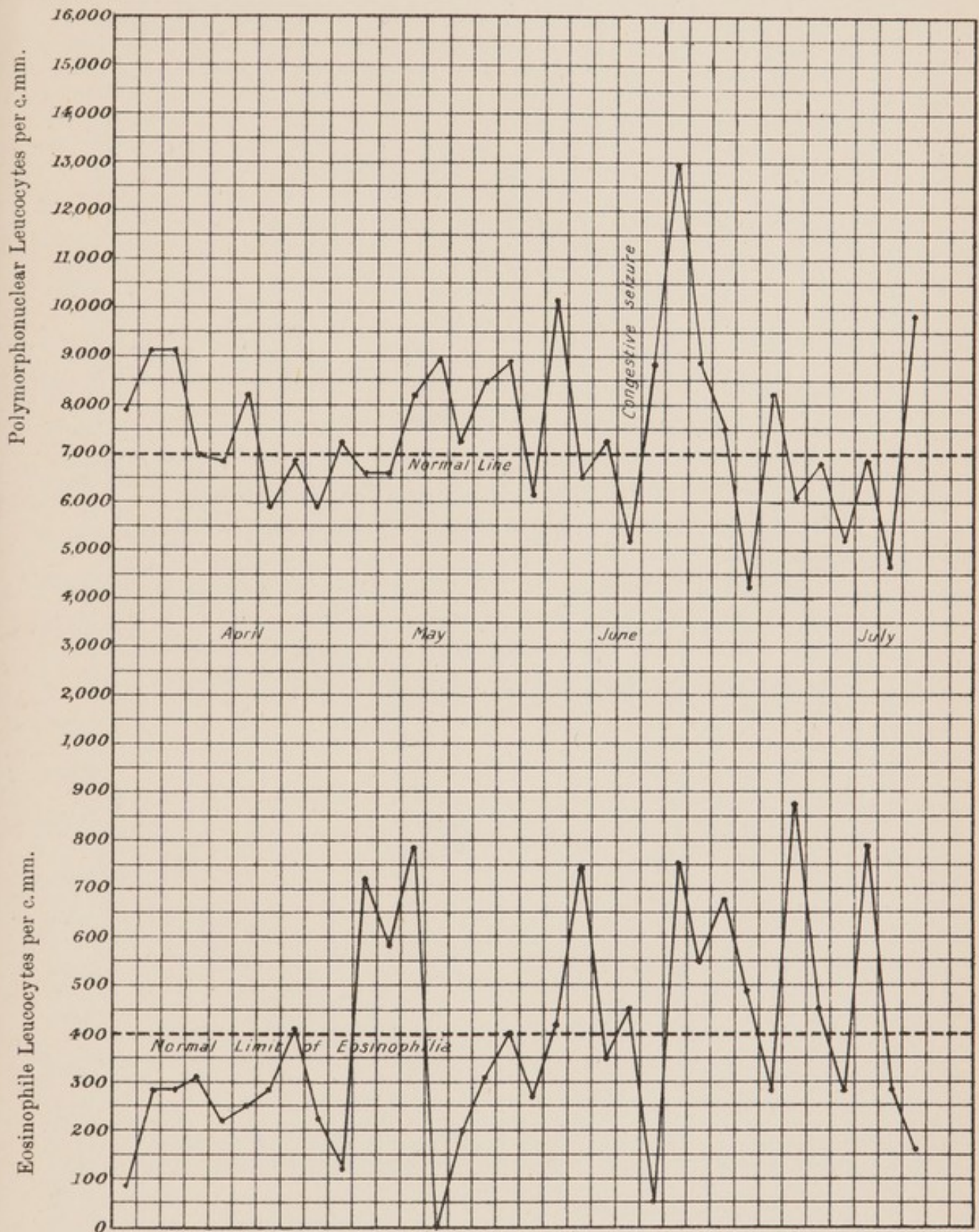


## LEUCOCYTE CHART NO. XVII.

LEUCOCYTE Chart showing the Polymorphonuclear and Eosinophile Leucocytes per c.mm. in a case of General Paralysis occurring in a male aged 43.

The chart corresponds to the stage of the disease known as the end of the first stage and the commencement of the second. The slight hyperleucocytosis in the polymorphonuclear cells which occurred during April corresponds to a slight febrile attack, and this was succeeded by a transient rise in the eosinophile leucocytes. During May there was again a febrile attack, which is represented on the chart by a second increase of the polymorphonuclear leucocytes, and again the eosinophile cells show an increase. Early in June the patient suffered from a congestive seizure, which was accompanied by a decided polymorphonuclear hyperleucocytosis, and during the period when the patient was convalescing from the congestive seizure the eosinophile leucocytes again increased.

CHART No. XVII.



A Case of General Paralysis uncomplicated with mental disease.

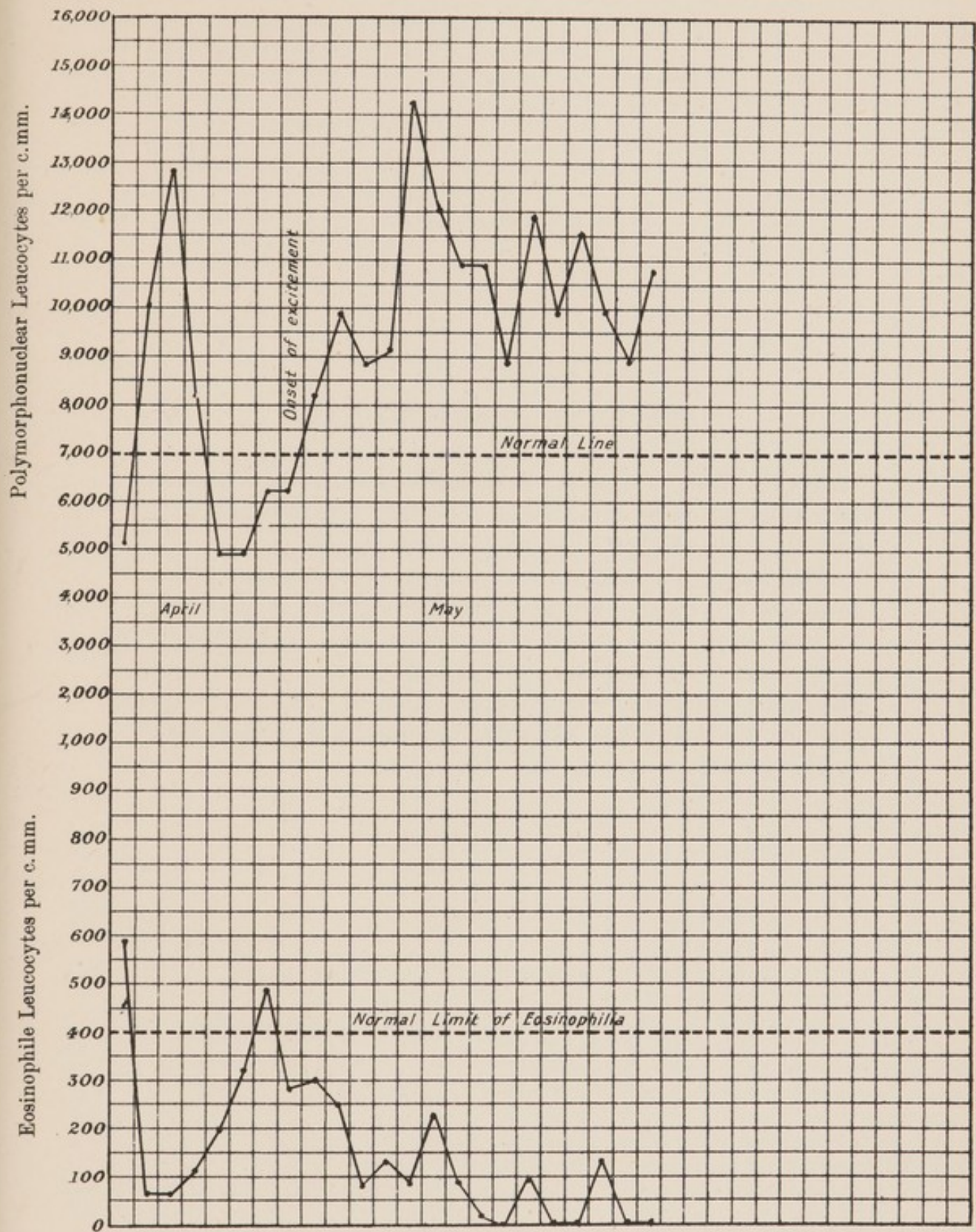


### LEUCOCYTE CHART NO. XVIII.

LEUCOCYTE Chart showing the Polymorphonuclear and Eosinophile Leucocytes per c.mm. in a case of General Paralysis occurring in a male aged 43.

The chart is interesting, as it corresponds to the onset of an attack of acute mania in what is called the first stage of the disease. During the attack of mania the polymorphonuclear leucocytes were increased, and the eosinophile leucocytes steadily fell to zero.

CHART No. XVIII.



A Case of General Paralysis in a male aged 43. The disease was in the first stage, and complicated by excitement with confusion.

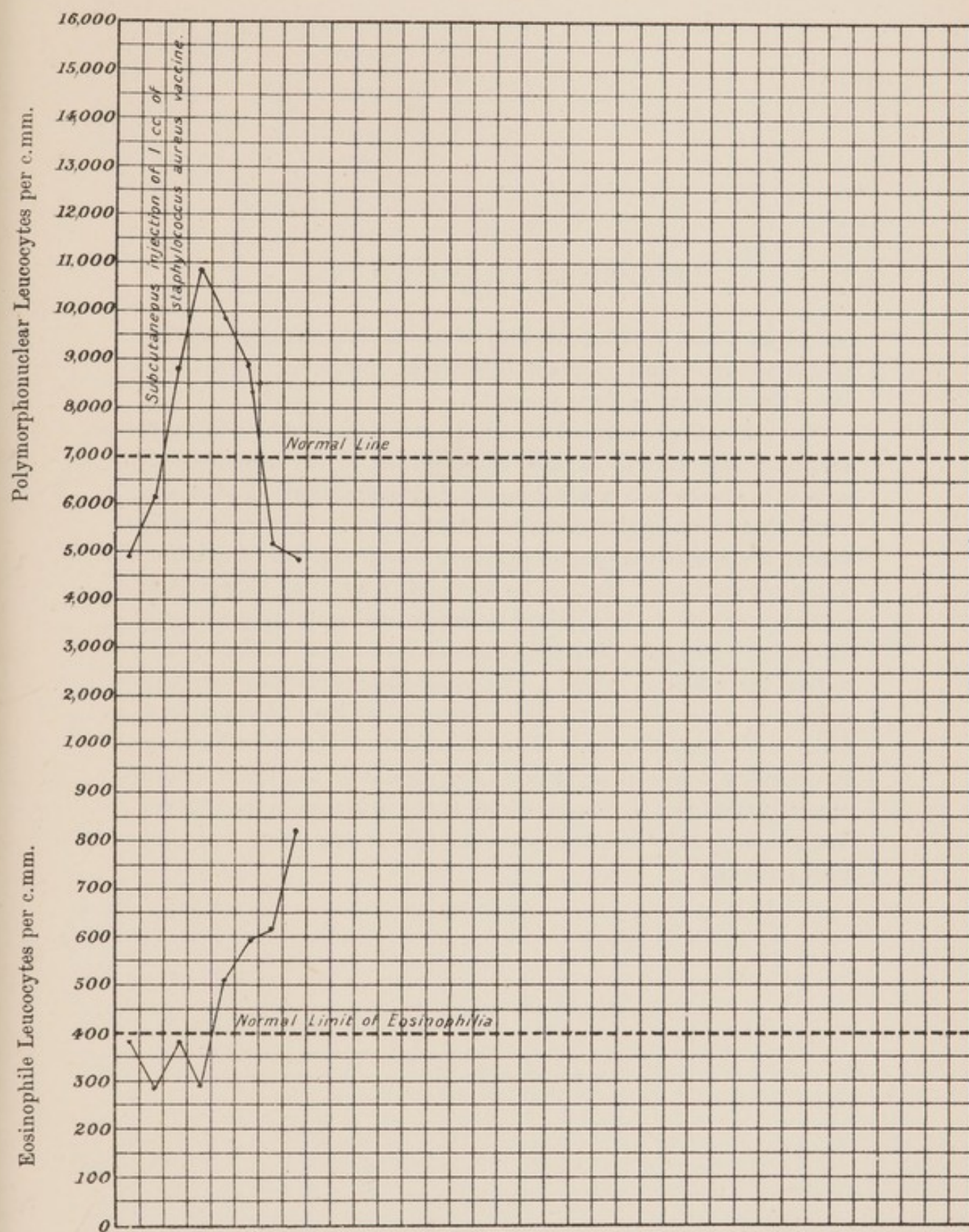


### LEUCOCYTE CHART NO. XIX.

LEUCOCYTE Chart showing the Polymorphonuclear and Eosinophile Leucocytes per c.mm. in a case of Secondary Dementia in a male aged 46, as the result of injecting a bacterial vaccine.

The patient, who suffered from sycosis menti, was injected with a 1 c.c. dose of a vaccine prepared from the organism isolated from the local lesion. The strength of the vaccine was 15,000,000 cocci to the c.mm. For four days following the injection the polymorphonuclear leucocytes were increased, and two days after the injection the eosinophile leucocytes increased, and reached their maximum six days after the date of injection.

CHART No. XIX.



A Case of mild Secondary Dementia, showing the rise of Eosinophile Leucocytes which followed an injection of bacterial vaccine.

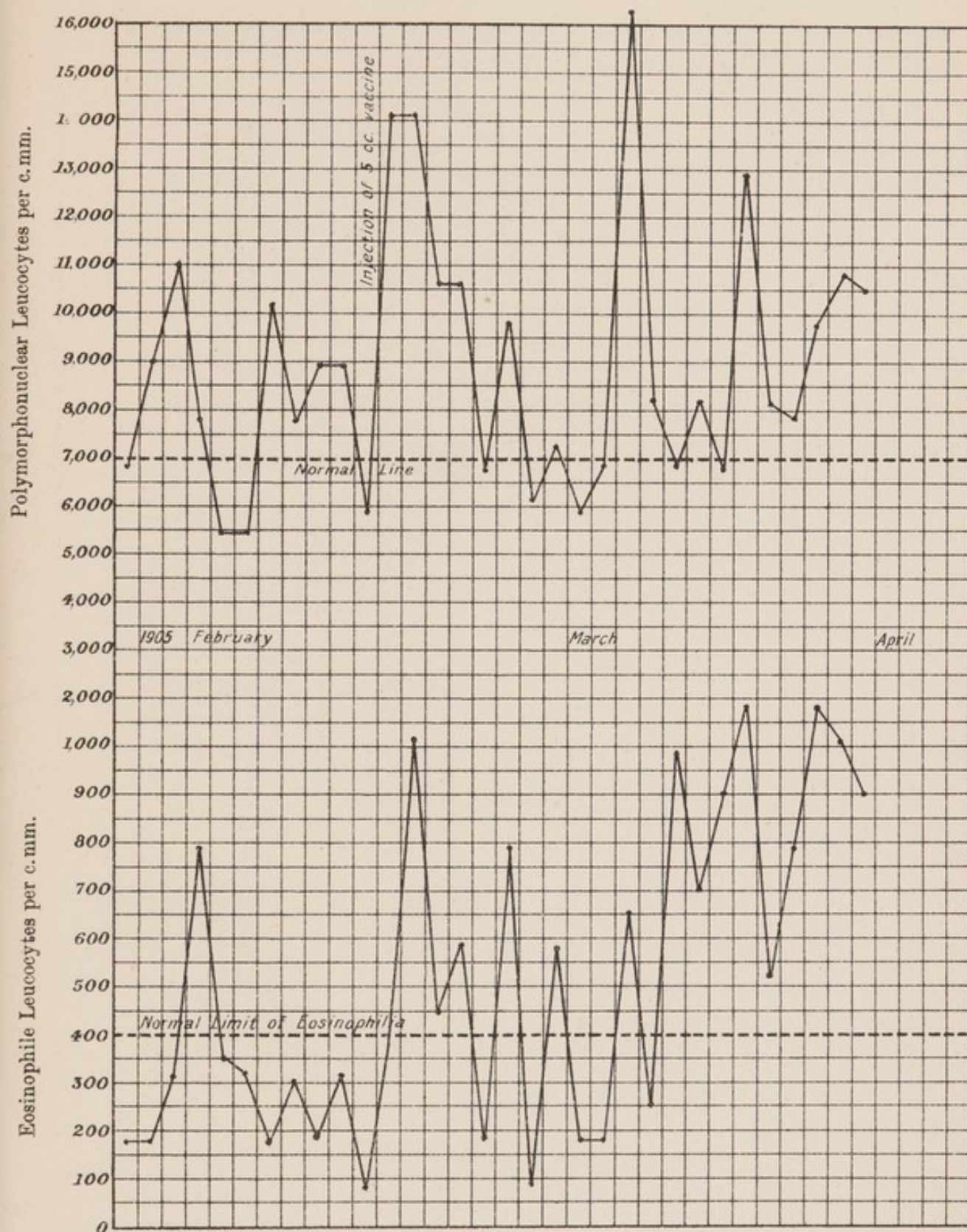


## LEUCOCYTE CHART NO. XX.

LEUCOCYTE Chart showing the Polymorphonuclear and Eosinophile Leucocytes per c.mm. in a case of Chronic Mania which had been under observation for three years.

The patient, who showed no symptoms of dementia, had upon admission agglutinines in the blood serum to the streptococcus isolated from the case of acute mania, but in February 1905 it was noted that these agglutinines had disappeared. An injection of vaccine made from the streptococcus was therefore given to ascertain if the production of immune body would in any way improve the mental condition. The injection was immediately followed by a marked increase in the polymorphonuclear leucocytes, which persisted during the whole of March. The eosinophile leucocytes were also increased immediately after the injection, but they increased in a much more marked manner towards the end of March, and during this period a powerful agglutinine was present in the blood serum. Mentally there was no improvement, but it was noticed that for the first two days after the injection the patient was more maniacal than formerly.

CHART No. XX.



A Case of Chronic Mania showing the reaction to an injection of vaccine.



CHART No. XXI.

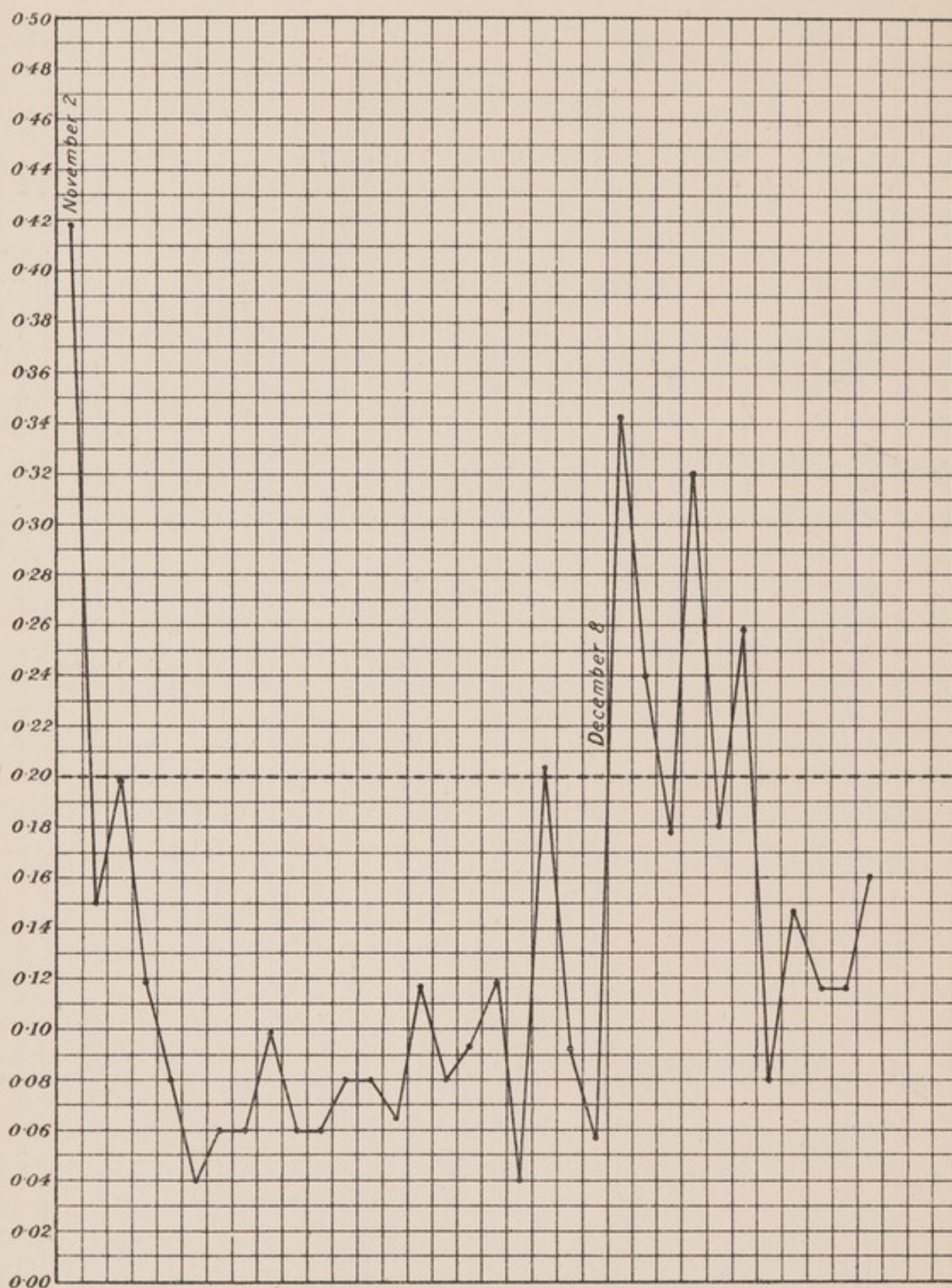


Chart showing the percentage of Pure Nitrogen in the Urine of a case of Delusional Insanity occurring in a male aged 27. The diet was purin free from the 2nd November until the 8th December, after that date the diet was an ordinary one.



CHART No. XXII.

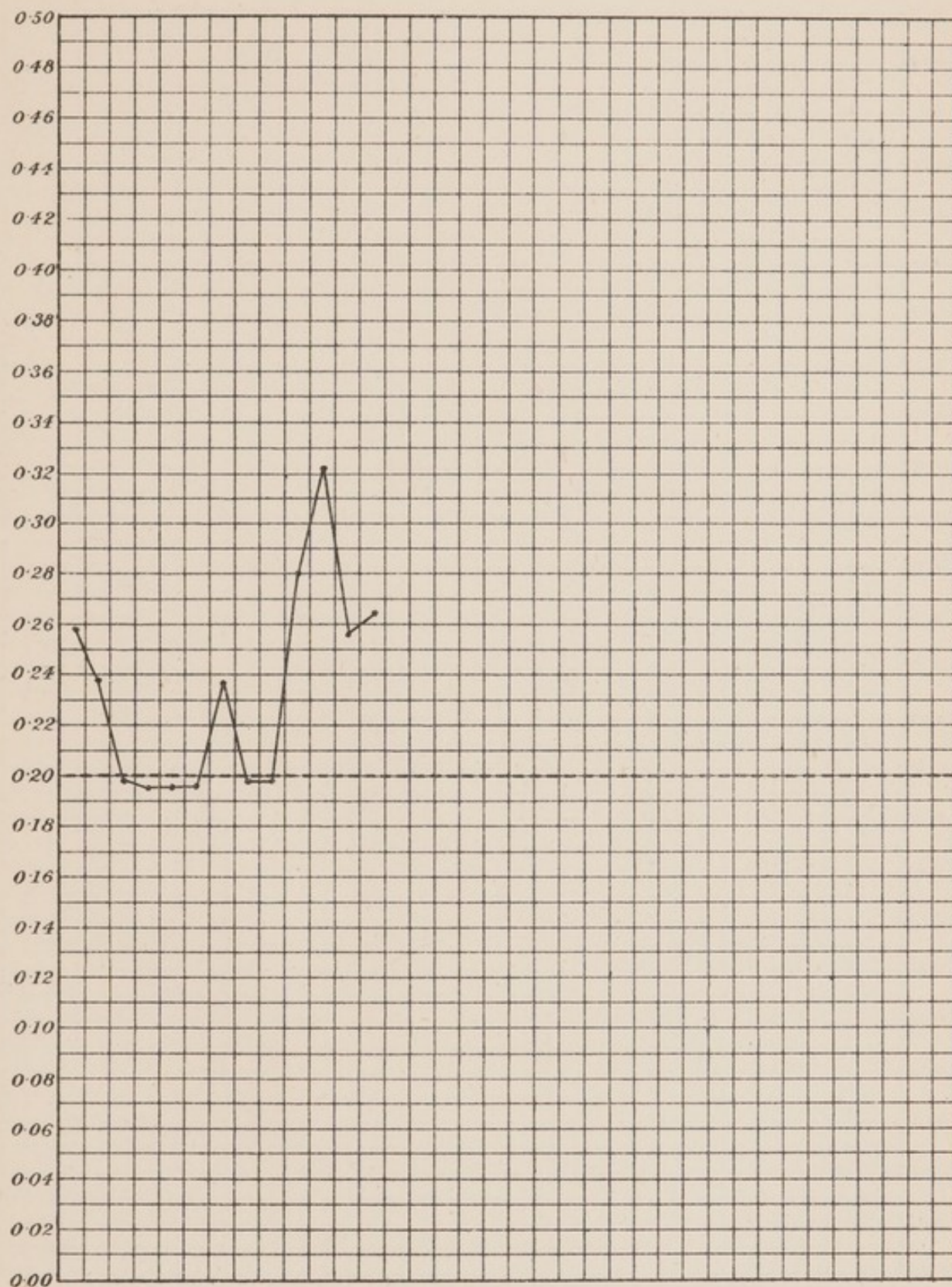


Chart showing the percentage of Purin Nitrogen in the Urine of a case of Delusional Insanity, occurring in a male aged 37. The primary fall shows the effect of putting the patient on purin free diet; the rise towards the end of the chart is the result of adding tea to the purin free diet.



CHART No. XXIII.

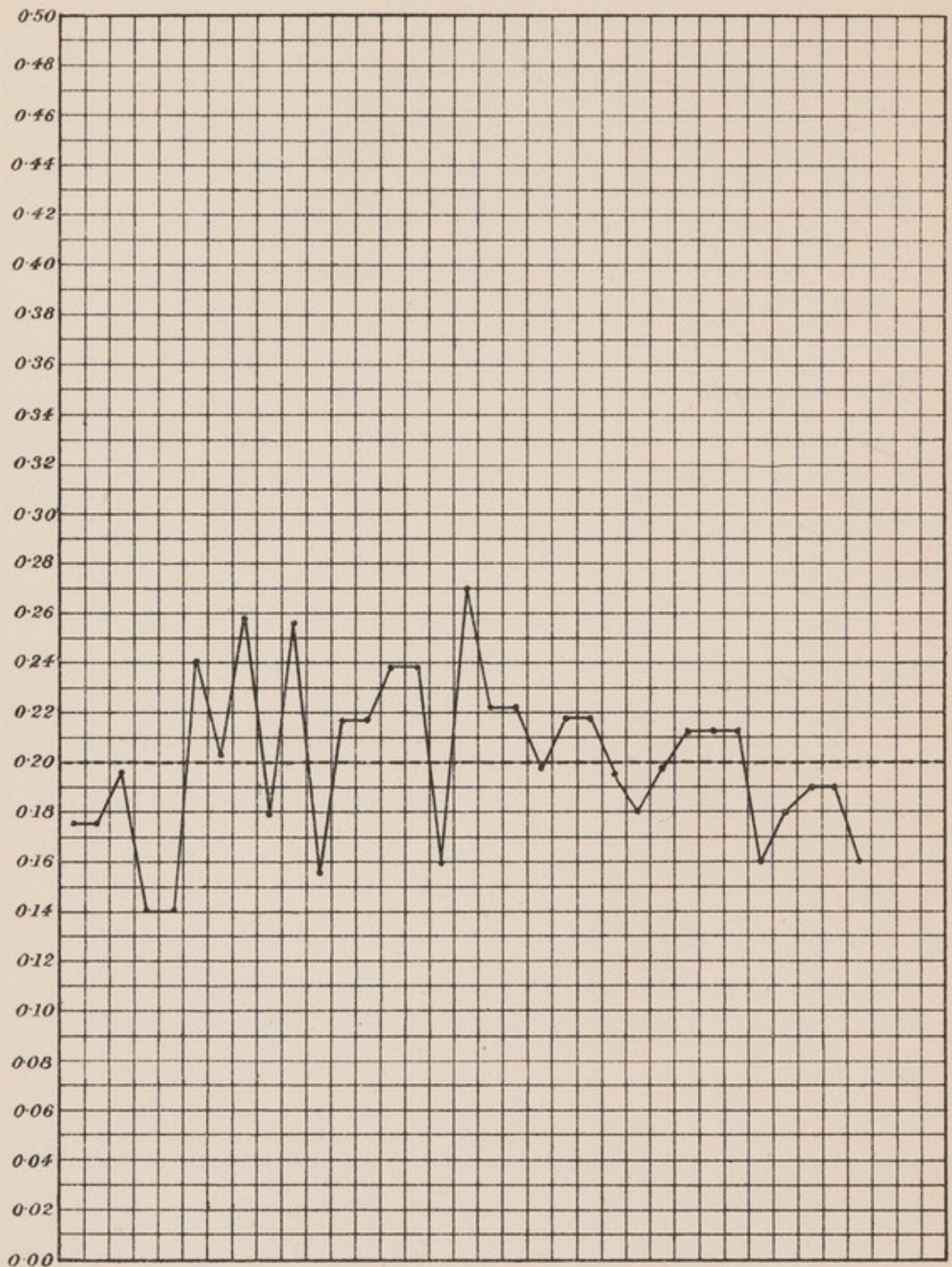


Chart showing the percentage of Purin Nitrogen in the Urine of a case of Acute Mania occurring in a male aged 25. The chart corresponds to the period of acute maniacal symptoms. (Purin free diet.)



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