The Influence and Relations of Innutrition as a Factor in Neurasthenia.

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The term 'neurasthenia', as employed in the nomenclature of disease, has acquired a significance of much wider scope than its precise meaning would suggest. The symptoms commonly present indicate derangement of diverse functions in addition to those of the nervous system, particularly disorders of digestion and circulation; and the sequent or other relation of the various factors is not always obvious, nor easy to determine.

Neurasthenia, in its wider sense, is conceived as essentially a functional disorder, in which the predominant symptoms are referred to the nervous system: if there be any underlying structural change it has not been observed, and at least stops short of the recognised forms of degeneration. In view of the intimate physiological relation between the nervous system and all functional activities, we might anticipate the actual issue that neurasthenia, in its strictest sense, merely marks a point in a vicious circle of disorders, the circumference of which may traverse any other functional system, involving such morbid action and reaction as tend to aggravate and prolong the general process of vital depression. Further, the classical symptoms of neurasthenia, such as exhaustion, restlessness, sleeplessness, irritability, irresolution, mental depression, diminished power of memory and attention, etc., are manifested in connection with many pathological conditions, some of which exhibit gross lesion outwith the nervous system: they are common in the diseases characterised by tissue wasting, and often mark the stage of convalescence after acute febrile diseases. The 'antecedent phenomena' and complications may thus be varied, yet the neurasthenia, as such, presents no clinical distinctions; it remains 'nervous exhaustion', more or less profound in proportion to the general deterioration.

Sir Clifford Allbutt[1] has emphasised the fact that "neurasthenia, in its several forms, is not only a frequent, but also a uniform and consistent malady"; and the implied rebuke was not untimely, that it is "no mere hotchpot - no limbo into which odds and ends of unconsidered neurotic trifles are to be thrust away for lack of definite affinities". The fact remains, however, that neurasthenia has an unusually wide connection with other morbid states; and while appreciating the force of this distinguished writer's protest against 'attempts to define diseases', it may be said that neurasthenia is one of the least definable, and that in no other disease is the interdependence of functions more clearly corroborated by the interdependence of their failure. In the nomenclature a group of symptoms - characteristic, but rarely isolated - has been selected for undue prominence, and is therefore apt to dominate the attention at the expense of elements of equal, if not greater import, and so to obscure the primary locus of mischief. A notable instance was recently recorded by Lockhart Mummery,[2] in which a case of 'so-called chronic neurasthenia' was found to be due to adhesions which anchored the stomach and transverse colon to a small area in the left iliac fossa.

The difficulties arising from the wide application of a limited term have been met, to some extent, by classification. Savill[3] proposes a subdivision based on 'pathological conditions', viz:- Toxaemia, Malnutrition, Fatigue, Emotional Shock and Traumatism. Allbutt[4] prefers an anatomical basis:- Cerebral, Spinal, Cardiac and Vasomotor, Gastro-intestinal, Sexual neurasthenia; though he admits that "like neurasthenia itself, these subdivisions are no water-tight compartments, but are in frequent communication one with another." Such classifications have certain advantages, but on the other hand they tend to confuse the concept of an underlying unity.

These and like considerations have suggested the question whether neurasthenia, however diverse its phases and associated errors of function, may not always be referrible in its pathology

to a determinate point, and a definite fault? I submit, as a hypothetical proposition, that it may be so referred, and that the nervous failure is related to, if it does not actually depend upon continued deficiency of stored nutrient material in the cell-body of the neuron.

The central nervous system is conceived to be a grouping, in definite arrangement, of nerve-cells and their dendritic and axonic processes. The cell-body ('neurosome': Campbell) occupies a central site except in the case of peripheral sensory neurons, and the post-ganglionic neurons of the autonomic system, in both of which the neurosome has an extra-central situation in lateral, collateral, terminal, or other ganglion. It is assumed that inter-neuronic relation is maintained by a system of synaptic junctions.

Although little is known regarding the complex activities of these structures, it is acknowledged that the neurosome or its nucleus exercises an intimate nutritional influence on the neurofibrils; and that the generation and transmission of nerve-impulses depend upon a supply of nutriment, as well as oxygen, to the neuron. In addition, therefore, to the nutriment utilised in the metabolism of repair, a further provision is necessary for special functional activity; and as the supply to the organism is intermittent, while the demands upon functional activity are practically constant, it is presumed that the nervous tissues 'possess a special function involving storage of material'.[5] The importance of proteids - stored mainly in the form of nucleo-protein - is indicated by the proportion in which they occur in the grey matter, where they constitute over fifty per cent of the total solids;[6] while certain lipoids, such as lecithin, are known to be also indispensable.

At present the locality of storage can only be a matter of inference, but authoritative opinion seems to point to the nerve-cell. It is noteworthy that the cytoplasm of the neurosome contains the chromatophile bodies known as Nissl's granules, the main

constituent of which is an iron-containing nucleo-protein "named by Marinesco 'kinetoplasm' to express the idea that it forms a source of energy to the cell."[7] With reference to the substance of these granules Halliburton[8] says "it can hardly be denied that, forming as it does so large a proportion of the cell-contents, and made of a material in which nuclein forms an important constituent, it is intimately related to the nutritional condition of the neuron." This accords with the changes observed in primary degeneration - first a 'disintegration of the chromatophile substance of the protoplasm', to be followed in progressive cases by 'disintegration of the fibrils, degeneration of the nucleus, and death of the whole element'.[9] It is significant also that "if the alteration does not proceed further than simple chromatolysis it may be recovered from."[10]

Mott[11] cites Golgi as having shown that the dendrons serve a nutritive function by absorbing the necessary products from the lymph space in which they lie, and in a foot-note remarks that 'according to Ramon y Cajal and Lenhossek, if the dendrites do represent agents of nutrition, they would only act by increasing the surface of absorption of the cell to which they belong'. As the distribution of the Nissl bodies is confined to the hyaloplasm of the nerve-cell and its continuation in the dendritic trunk, the evidence points definitely to the neurosome as the storehouse of nutrient material.

But we are not without evidence that these bodies serve a purpose beyond the mere nutrition of the neuron: it is admitted, indeed that "the integrity of the nucleus, and of the fibrils between which the Nissl substance lies, is much more important to the actual vitality of the cell."[12] It has been observed that 'massive discharge of impulses' in epilepsy has been followed by the disappearance of the granules, which had undergone chromatolysis;[13] and though the exact relation of the nerve-storm to this change in the chromatoplasm is regarded as open to doubt, the phenomena are at least

coincident. In a recent review of the subject of the neuron, Campbell[14] expressed the opinion that even under physiological conditions the reserve of nutrient material, though never entirely exhausted, may be drawn upon to an extent involving the disappearance of Nissl's granules; and he stated, further, that "there is evidence that the neurosome and dendrites are continually elaborating from the circumambient plasma substances which are used up in the activities of the ceptors". This is but a re-statement of the view previously held to have been 'definitely proved';[15] and it seems to find confirmation in Halliburton's reference to observations on the bee, which showed that after a hard day's work the quantity of chromatophilic material was much reduced.[16] The latter fact is cited as bearing on 'the influence of fatigue in producing chromatolysis': but if the chromatophilic material may be regarded as representing potential energy, to be expressed kinetically in bodily and mental work, I suggest that by carrying the sequent relation a step further we arrive at a possible basis of neurasthenia, viz:- the influence of chromatolysis in producing exhaustion.

It must be confessed, of course, that no theory relative to metabolism, especially when applied to the nervous system, can be other than tentative while the nature and functions of internal secretions, and even of the cerebro-spinal fluid itself, remain to a great extent unexplored.

The following experiment was undertaken with the object of observing the changes, if any, produced in the appearance of the contents of the neurosome by total privation of proteids:-

Five rabbits of the same age - two months - were fed for seven days on a full diet of crushed oats (cooked) and milk. The first was killed two hours after the last protein feed, and the brain and lumbar cord removed for examination. The remaining four were thereafter restricted in diet to a small quantity of potato daily; and were killed in rotation at intervals of twenty-four hours.

The brain and spinal cord were hardened by immersion for fourteen days in a solution of perchloride of mercury in absolute alcohol, and embedded in paraffin for section. In view of the affinity of the chromatoplasm of the nerve cell-body for basic dyes, two series of sections of each structure were stained: one by Lenhossek's method (using thionin blue, saturated solution; 5 minutes immersion; dehydrated in acetone); and the other with haematoxylin and eosin (dehydrated in alcohol). A third series was stained by the Giemsa method. All were cleared in oil of cloves, and mounted in Canada balsam.

I have appended impressionist drawings of the larger cell-bodies from the anterior cornua of the spinal cord, in which I have attempted to indicate the different values in the staining; also photo-micrographs of the same class of cell-body in the haematoxylin series.

I submit that the sections (which are sent herewith) support the contention that the neurosome is the seat of protein storage; and that while any considerable expenditure of nerve-energy has been shown to involve chromatolysis, and a relative diminution of stored nucleo-protein, the replenishment of the chromatoplasm depends upon the provision and digestion of proteids; and the same relative diminution attends ordinary nervous activity when the replenishment is in abeyance.

The possible bearing of these observations on the etiology and pathology of neurasthenia is kept in view in the remarks which follow.

Note:- Thirty microscope-slides are sent in with this essay.

The most common complication of neurasthenia is digestive disorder in one or other of its many forms. Nucleo-protein is essential to cell-nutrition and activity: but between the native proteins of the ingesta and the nucleo-protein available for the nutrition and activity of the neuron there intervene a series of vital and chemical processes, on the efficiency of which the nervous system depends for nourishment, and which, in turn, depend upon the nerve centres for their efficiency. Gastro-intestinal mobility and secretion, as well as the adequate production and catalytic action of enzymes are readily influenced, not only by the nature and mechanical preparation of the ingesta, but also by innervation, and reflex adjustment of the circulation. When these correlated functions are considered in detail, it is not surprising that digestion should be liable to disturbance from a great variety of apparently trivial causes, or that derangement may often be so slight as to escape notice until its persistent action has produced a train of effects gross enough to be felt and recognised.

Assuming an adequate supply of nourishment, functional efficiency in digestion, and an unimpaired power of absorption and synthesis in the nerve-cell, one may ask 'at what point is the healthy circle most likely to break down?' It is obviously in the digestive arc that it is most exposed to interruption; and it is here more than elsewhere that lesser functional errors would be likely to become established as habitual. The circle is doubtless liable to initial interruption at any point; but so far as the nervous system is concerned it is better protected against direct attack; it claims and receives more care; slight departures from health are more readily felt, and more impressive; and the agents of disturbance that might act directly are in detail less numerous, and in nature less palpable than those to which the digestive system is constantly exposed.

I have had opportunity of studying 73 cases of neurasthenia in

which the diagnosis was confirmed by others, and in each of which there was noted some degree of malnutrition, and some derangement of digestion to which it was thought the malnutrition was mainly due. Savill[17] investigated 102 cases 'associated in some way with symptoms of gastric disorder', with the object of ascertaining the order of sequence. From his results it would appear that the onset of the digestive trouble may precede the neurasthenia by terms ranging from months to years (74 cases); that the conditions may begin 'about the same time' (13 cases); or that the neurasthenia may appear first (15 cases). As these were selected from a consecutive series of 157 cases it is to be presumed that in the remaining 55 no 'symptoms of gastric disorder' were observed. There is the objection to such statistics that their accuracy would depend on the patient's recollection and powers of observation, which are not always trustworthy. Some degree of disturbance may be present long before it is noticed. The outstanding fact with regard to my own series is that neurasthenia and gastric derangement, with general innutrition, were associated when the patient came under observation. I believe that this association is characteristic of a large majority of cases, and that the nervous exhaustion is not only subsequent to digestive disturbance, but usually follows as a consequence.

In the latest edition of his 'System of Medicine' Allbutt[18] repeats, in effect, a former statement[19] that "many of our patients have not fallen into neurasthenia by way of innutrition, but by stress of work or of care, or by the effects of heredity acting in a constitution otherwise healthy". Probably so! Just as there are many factors, including even 'heredity' or what it stands for, so there are doubtless many avenues of entrance. But if it is meant by the example cited that neurasthenia may be 'attended with good physical health'; that, as Oppenheim[20] observes, "the general nutrition need not suffer in the slightest"; in short, that neurasthenia can exist for an indefinite time without nutrition becoming involved, I can only

say that either I have not seen such cases, or have been unable to identify them. And it must be difficult to account for them if it be conceded that nervous exhaustion, which is the one feature common to all cases, implies that for some reason protein is not available. While muscular energy may be derived from carbo-hydrates and fats, tissue repair and nerve-energy are so dependent on the proteids that in the absence of supply from other sources the tissues themselves will be to some extent consumed. It would appear, therefore, that although nutrition may not suffer immediately, it cannot be hoped that it should escape indefinitely, no matter how the state of exhaustion has been reached.

The conclusion that there exists a definite relation between neurasthenia and faulty metabolism, usually referrible to digestive error, is arrived at also when the subject is considered from the point of view of treatment. In the system suggested by Weir-Mitchell[21] the aim was to induce improved nutrition, without particular attention to the nervous failure. In the result it has been found that by providing a full supply of suitable nourishment, and aiding its assimilation by massage, the digestive functions respond, and an anabolic process is initiated. The element of complete rest is an aid to securing the fullest cumulative value in nutrition; and isolation remains the only condition of treatment addressed specially to the nervous features of the case. Where the diagnosis is correct the patient usually recovers: where the treatment is skilfully applied, and fails, there is reason on that ground alone to suspect some graver mischief than the term 'neurasthenia' should be held to embrace.

The cases of my series were treated on the lines indicated, and I append a graphic record of the improvement in somatic nutrition so far as it was measurable by increased weight. In all of these the sense of exhaustion, and the distressing nervous symptoms associated with it, diminished in proportion - not always constant - to the

gain in weight, the result being naturally most striking in those cases where the initial weight was much below the normal standard, and a greater increase was therefore possible. Relapse occurred in 19 cases, the interval of health ranging between 9 months and 7 years. Of the remaining 54 cases, 9 showed a slight loss of weight on relaxation of the strict regimen, though the health did not appear to suffer; the improvement was fully maintained in 17; and in 28 there was further increment. In one case (Male, age 42) relapse occurred after six years of good health and active business life. The patient was again restored to vigour by a second course of treatment, and on each occasion the gain in weight exceeded 28 lbs. The relapse had gradually supervened upon reversion to a former habit of spending long days in a small, ill-ventilated office, which entailed also irregular and insufficient meals. In only one case was I permitted to obtain photographs (appended); and though the difference they show is not so impressive as in Lauder Brunton's familiar illustration,[22] yet it is remarkable, and not least so in the happier state of mind indicated by the facial expression. In one of the cases where the gain under treatment did not exceed 7 lbs. the subsequent history was instructive. The patient (Female, age 50) had resided for ten years in a Spanish colony in the Pacific. During the last three years of that period she had suffered from persistent, profuse diarrhoea, and returned to England with the object of seeking medical advice. Her weight was 72 lbs.; and although after 42 days under treatment it was still only 79 lbs., the diarrhoea had ceased, and the lost appetite was returning. At this stage she went on a prolonged visit to relatives in the country, who undertook to carry out a prescribed course of generous nourishment. When I saw her again, after an interval of 18 weeks, I failed for a moment to recognise her: she now weighed 122 lbs. - a further gain of 43 lbs.: total gain 50 lbs. This was a striking example of the fact that subject to reasonable care a constructive process, once started, tends to be maintained, and to be concurrent with

increase of energy.

If the word 'neurasthenia' be interpreted literally, rather than regarded as a mere class-name restricted to cases which conform more or less closely to a 'type', the more common etiological factors may be classified in general terms, with reference only to the protein-storage functions of the neurosome:-

1. Any conditions entailing total or partial privation of nutriment. Such conditions arise in circumstances where food is not obtainable in sufficient quantity, or if provided is deficient in proximate principles, especially proteids. The effects are equivalent to those of deficiency in quantity or quality of the blood-supply, and are recorded, with many acute examples, in Surgeon Parke's 'Experiences'[23] It is acknowledged that though in inanition or starvation the 'master organs' are the last to suffer material loss, and in this respect suffer the least, their functional efficiency is very soon involved. In all cases of anaemia, whether primary or secondary, as well as in chlorosis, the effect on the nerve centres of the diminished protein capacity of the blood amounts practically to some degree of privation; and this is an aspect worthy of notice even in such toxic anaemia as results from the action of Pb., Hg., and As., or the virus of syphilis or malaria, over and above any specific effect on the nerve structures themselves.

2. Any conditions involving a demand upon stored nutriment in excess of the supply.

The chief of these conditions is STRESS, manifested in an endless variety of forms in the industrial activities, commercial competitions, and domestic harassments of modern life. To the direct action of overwork and pressure must be added the malign influence of confinement in workshop and factory, wearisome monotony of occupation, insufficient time for meals, overcrowded and insanitary dwellings, disturbed or insufficient sleep, lack of relaxation and repose, anxiety as to 'ways and means', and the numberless other enervating

circumstances under which the mass of the people pass their lives. In the same category may be placed the cases attributed to sexual and other excesses. The observations of Sherrington[24] - that reflex arc conduction both depends largely on the supply of oxygen, and involves considerable fatigability - greatly illumine the relation of stress and its correlative factors to nervous exhaustion.

3. Any conditions tending to vitiate the quality of the nutriment supplied.

Imperfect digestion may entail imperfect protein cleavage; while the food itself may prove to be the vehicle of toxins. This class of 'causes', however, is practically inseparable from the following:

4. Any conditions so affecting the neurosome directly that it is rendered incapable of performing its functions of storage and delivery.

It may be assumed that any such failure originates in some interference with the synthetic power of the cell, due probably to toxic influence, to which nerve structures are especially sensitive. Even if we exclude the toxins of specific diseases such as diphtheria and influenza, the effects of which are often graver than mere functional disturbance, there remain toxic products more insidious in origin if less virulent in action, such as those of faecal decomposition. The internal secretion of the liver, by its power of converting the amido-antecedents of urea into that much less toxic compound, greatly diminishes the risk of autointoxication from the products of tissue metabolism, and doubtless affords some protection here also, though the blood-stream is still exposed to direct invasion by way of the middle and inferior haemorrhoidal veins. Lane[25] recently called attention to 'chronic intestinal stasis' as a precursor of autointoxication; and as evidence of the influence of the toxins on the nervous system he cited the case of a patient who had been 'confined to bed for many months, having neither the capacity nor desire to stand or walk; and whose mental condition was

such that she was regarded by many as an imbecile', who became 'a happy, active, intelligent woman within a few weeks of the removal of the large bowel'. F.W.Brook,[26] in an investigation of 45 cases of neurasthenia, demonstrated the presence in the intestine, in more than half the number, of bacteria 'which had acquired pathogenic properties'; the evidence of infection being obtained by serological tests, though not indicated by culture. In the streptococcal cases (S. pyogenes longus) 'pyorrhoea was either present, or a definite history of it could be obtained'.

It has been suggested that true neurasthenia is always of autotoxic origin; and although this view is not justified by experience it is true that a large proportion of cases are due to autointoxication, especially where oral sepsis is either excessive or associated with deficiency of free hydrochloric acid in the gastric juice. The effects are probably aggravated by a superadded intoxication due to impaired metabolism, or imperfect elimination of waste products; though as Allbutt[27] says "the state seems too enduring, too ingrained to depend upon the accumulation of the fatigue products investigated by Mosso, Michael Foster, and others".

That the neurasthenia of autotoxic origin should exhibit less profound involvement of the nerve centres than that due to the specific toxins of certain acute diseases points to a difference in degree, rather than in essential pathology. It is true that certain bacterial toxins, such as those of diphtheria and tetanus, appear to be selective of particular sites for their more specific action; and that toxaemia in general may be marked by delirium, mental torpor, and occasional paralyses. These profound effects, however, are not inconsistent with the more direct route of invasion when the portal circulation is evaded, and with the consequent larger dose, and more rapid action which then stand in the way of acquired tolerance. I have known many cases in which extreme toxic asthenia followed early in the wake of invasion by the tonsillar path. In the

stage of convalescence, and in the absence of structural change, the nervous condition in such cases is not distinguishable from so-called neurasthenia.

5. Any conditions so affecting other portions of the neuron that it is rendered incapable of utilizing the store of nutrient material. It has been shown[28] that certain poisons, such as chloroform, ether, and strychnine, exert a profound influence on reflex arc conduction as compared with nerve trunk conduction. With reference to this fact Campbell[29] suggests that though certain poisons may produce a specific effect on particular portions of the neuron, their influence on nerve function is the result mainly of direct action on the ceptors and mittors. This speculation offers a plausible explanation of the role of the drug habit in the etiology of neurasthenia.

6. Any combination of these conditions.

There is, in fact, no combination that is not possible; and no single cause, even if it be primary as to time or importance, remains long the sole cause in operation.

There remain those cases of traumatic and emotional origin in which shock, often without actual injury, seems to be the determining factor; but in which also the mode of operation by which shock or trauma may be related to the subsequent course of events is as yet a mystery. These cases do not usually show the sharp line of demarcation seen in those of 'idiopathic' origin. They form a mixed category; and though neurasthenia is a frequent enough sequel, it is often complicated by the intrusion of hysteria and hypochondriasis. So far as the neurasthenia is concerned, its onset is rarely immediate: it is significant that as a rule there intervenes a variable period between "the incidence of shock and the first serious manifestation of symptoms, very often between it and the first symptom".[30] Horsley finds this period to vary between one and several weeks; and according to Oppenheim[31] it may be measured by days, weeks, or months.

It is hardly possible to account for the small class of acute cases in which the symptoms immediately follow the injury, on the ground of any direct interference with nutrition; although it is conceivable that by dislocation of the delicate synaptic junctions the store of nutriment might become suddenly inaccessible, and practically useless. But it is otherwise with regard to the cases in which the onset of symptoms is delayed. It is recognised that 'a psychic influence enters largely into the activity of gastric secretion'[32]; and that states of mind marked by anxiety, worry, and morbid introspection, especially if associated with insomnia, exercise considerable influence on somatic nutrition. The general effect is probably secondary to derangement of the digestive functions, indicated always by anorexia; and frequently, as Horsley observes, by acute[33] or atonic[34] dyspepsia, which may lead to gastric dilatation and fermentation.[35] In these cases the habit of introspection is almost inevitable, since the attention is naturally focussed upon the injury, or the source of a violent emotion. It would, indeed, be extraordinary if in the circumstances the patient should escape the "reflective appreciation of the defect of capacity", even if there should not be obtruded "the sense of baffled purpose, of waning hope, and too often of failing resources" which Allbutt[36] specifies as conspiring to mental gloom. The psychic state itself may be submerged in the patient's consciousness by the current of subsequent events; but its action is still revealed in the failure of nutrition, and the vicious circle thus initiated.

The same explanation applies to those cases in which long-continued pain eventuates in physical and nervous break-down.

It is not without significance that in cases of neurasthenia of traumatic origin, as in those attributed to any other cause, the best results are obtained by cutting the vicious circle at the link of impaired nutrition.

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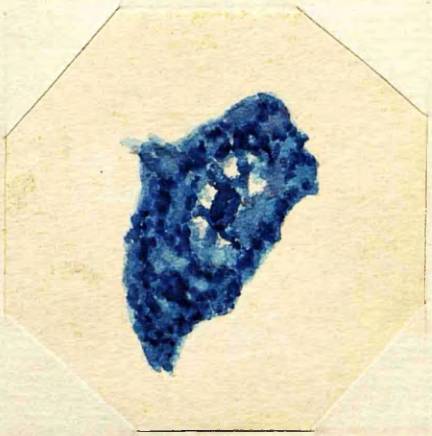
As I have not been able to obtain Prof. Sherrington's pamphlet, the references to his work are made on the authority of Dr.H.Campbell, from citations in Lancet, 3/8/12, pp.326,327

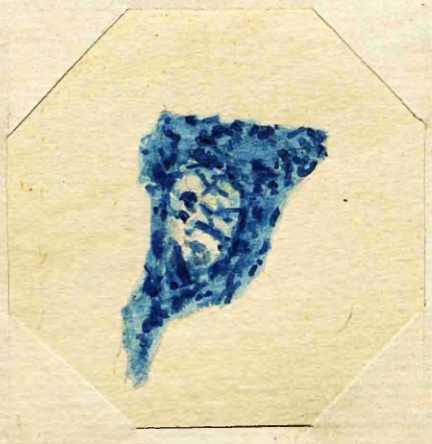
Cells of spinal cord (anterior cornu). Stained thionin blue.

Rabbit 1.

Rabbit 2.



Rabbit 3.



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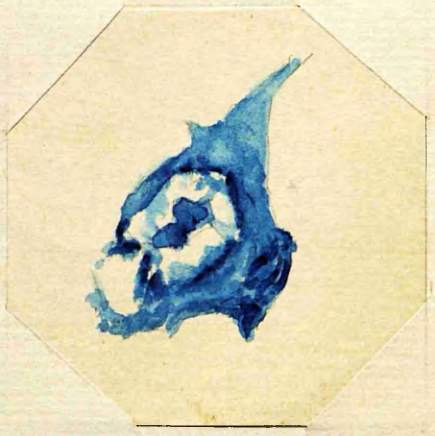

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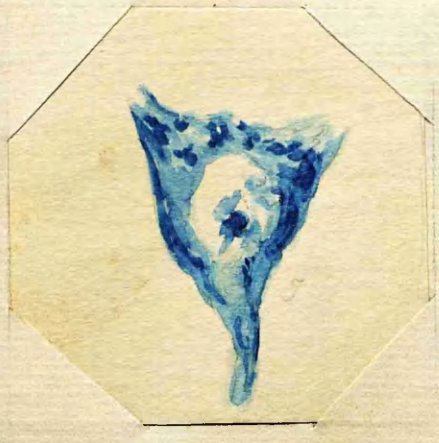
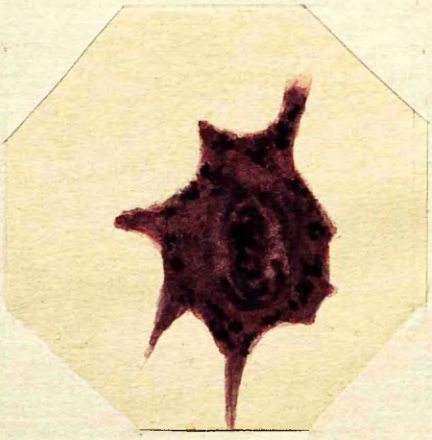


Cells of spinal cord (anterior cornu). Haematoxylin and eosin.

Rabbit 1.

Rabbit 2.



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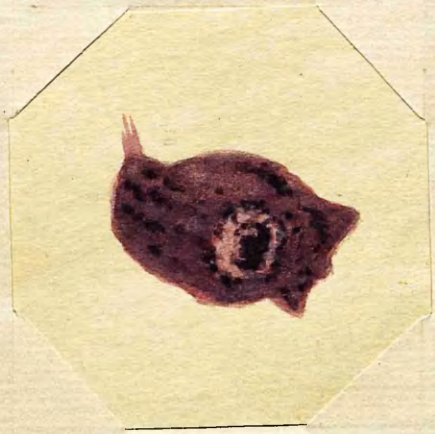
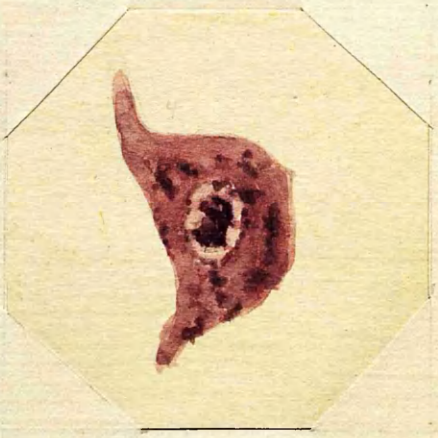


Rabbit 4.



Rabbit 5.

Cells of spinal cord (anterior cornu). Haematoxylin and eosin.

Rabbit 1.

Rabbit 2.

I regret that the negatives were not correctly exposed to show the cell-contents definitely; and as they cannot be replaced within the time-limit I must withdraw this page.

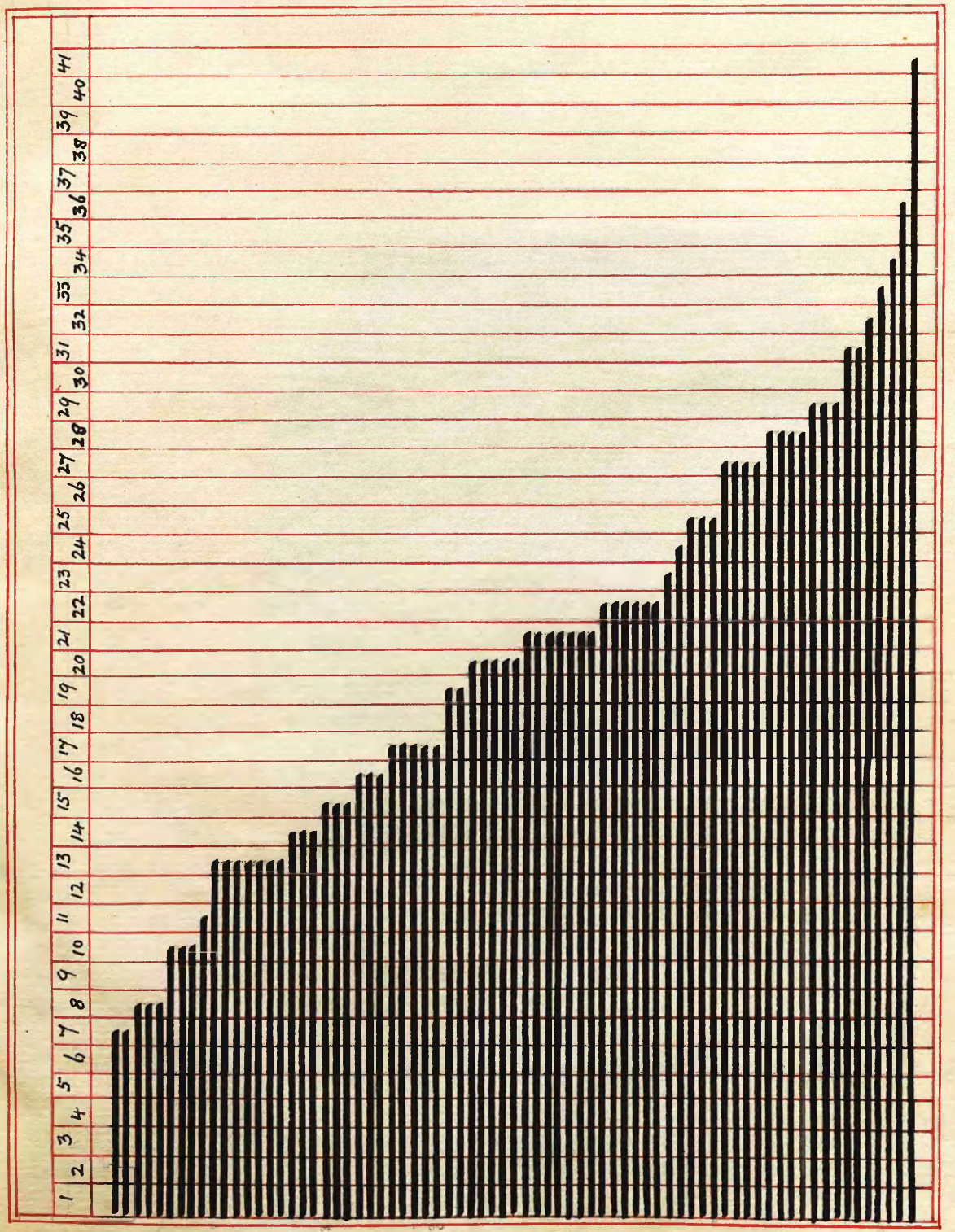
A Rambaud Ahad.

Rabbit 3.

Rabbit 4.

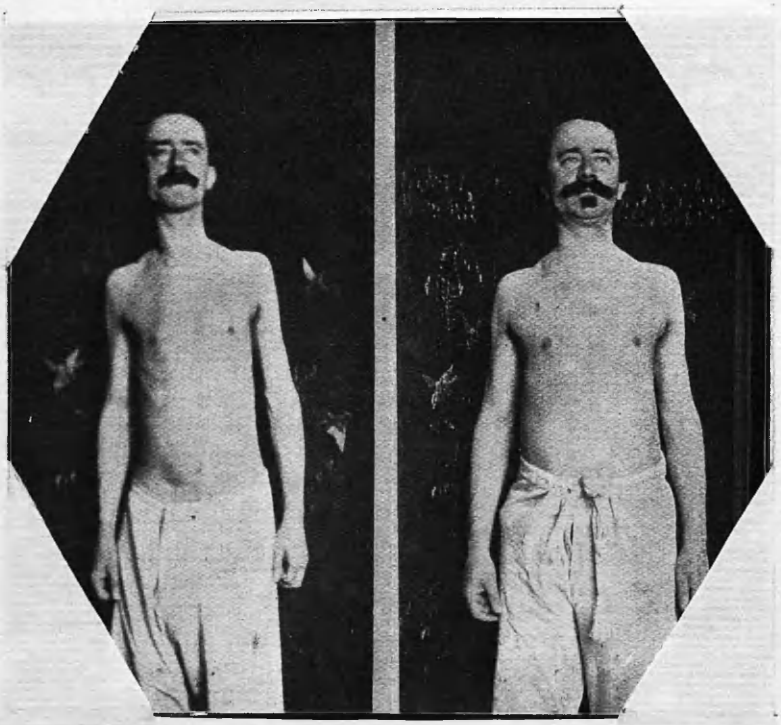
Rabbit 5.

Analysis of 73 cases of neurasthenia, with reference to the gain in weight after treatment for 42 days.



Case of neurasthenia treated by rest, full diet, and massage.

M.M.F. aet. 38



Photograph taken on first day of treatment. Weight: 109 lbs.	Photograph taken on forty-second day of treatment. Weight: 142 lbs.

Total gain in six weeks: 33,lbs.