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A DISCUSSION ON ATAXIA, AND THE DISEASES OF WHICH IT IS A SYMPTOM.

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PHYSIOLOGY AND PATHOLOGY OF ATAXIA.

IN a discussion on ataxia or inco-ordination (which I take it will mainly refer to inco-ordination of movements), it may be well to begin by briefly considering what is meant by co-ordination. In nearly all movements, even in very simple ones, more than one muscle is engaged. Thus, for the simple act of clenching the fist, apparently performed by the long flexors of the fingers, there is required a simultaneous contraction of the extensors of the wrist. Other simple movements, such as flexion and extension of the ankle, raising and lowering the eyeballs, etc., are obtained, not by the direct traction of one muscle, but as a resultant of the oblique traction of two or more. Now, the majority of our movements are much more complicated than these, and consequently require a more complex association of muscular actions; and for the proper performance of such movements not only must a proper set of muscles act, but each must act at a proper time, for a proper length of time, and with a proper force. To this harmony in the elementary forces which produce a movement the term "co-ordination" is applied, and when the harmony is imperfect then there is inco-ordination, or, which is the same thing, ataxia.

When the desired effect of muscular action is a movement, the disability produced by such want of harmony is called "motor ataxia." But muscular co-operation may be required not for movements only, but also to retain a fixed position against external forces, and a similar imperfection in this respect is called "static ataxia."

Does this definition maintain the distinction usually made between ataxia and paralysis? Dr. Mercier,[1] whom I have followed, puts the point in this way. Co-ordination consists in the preservation of a due ratio between the muscular forces (their strength, time, and duration of action); so that when this ratio fails there is inco-ordination. But if all the muscles concerned act insufficiently, their relative strength being nevertheless preserved, then there is weakness or paralysis. This is clear enough, so far as concerns paralysis of a movement. But paralysis of an isolated muscle may upset the harmony of a movement. Thus a man with palsy of the extensors of his toes, in walking lifts his knees inordinately high. We might call him ataxic, but incorrectly, for co-ordination, I take it, is essentially a function of the central nervous system; whereas, in this case, the paralysed muscles are cut off by disease of the lowest nervous segment from any and every combination; and we can no more blame the nerve centres than we could impugn the skill of a pianist if some of the keys in his instrument were dumb.

Nevertheless, in cases of central nervous disease, we must not be surprised if the distinction between paralysis and ataxia is sometimes difficult to maintain. For, if the higher motor centres, as Dr. Hughlings Jackson teaches us, represent not muscles but movements, then the selection and combination of muscles is as much a part of their function as is mere stimulation of muscles, and failure of such centres might conceivably be evidenced by ataxia as well as by paralysis of movement.

Again, it may be thought advisable to extend the term inco-ordination so that it shall include cases not only of faulty adjustment between the muscles concerned in one movement, but also between the several movements of a series; or, again, of faulty adjustment between the sensory stimulus which starts a movement and the motor response which follows—as, for instance, in the maintenance of equilibrium by muscles which act in response to afferent impulses from the semicircular canals and elsewhere.

If co-ordination be produced by the action of the central nervous system, where are the "centres" for this process situated, and what is the mechanism on which they act? We do not certainly know. But perhaps we may hazard some such statement as this: There is no one "co-ordinating centre" for all and every kind of movement, ready to put this finishing touch, as it were, on motor stimuli of every grade and variety. Nay, rather, if every movement, from the simplest to the most complicated, requires co-ordination, we must suppose that this function is closely connected with the action of every motor centre, from those of a low grade to those of the highest grade. Ataxia might then be caused by disease at very various levels, and might conceivably manifest itself in very various ways. But, curiously enough, the disease of which it is most characteristic—namely, tabes—is a disease that affects white fibres rather than grey centres, and it is round this question—namely, the production of ataxia by disease of conducting fibres—that the battle of theories has raged.

There are two main theories.[2] First the sensory—or better the reflex—theory of Leyden. On this view the due selection and control of muscular action takes place in response to afferent impulses from sensory organs. The initiation of a movement may be voluntary, but the muscular adjustment, once learnt, is reflex. For as we learn complicated movements by laborious attention to the incoming stream of sensations—from eyes, from semicircular canals, from skin, joints, and (above all) from muscles, so (it is supposed) when the movement has become automatic, these stimuli still remain as guides to the central co-ordinating mechanism. And ataxia may arise when disease of afferent fibres blocks, delays, or perverts these guiding impressions.

Against this theory Erb has urged the following objections, among others: that sometimes in cases of marked ataxia there is no discoverable loss of sensation, and conversely that total loss of sensation has been observed without ataxia. He adopts, therefore, a second or motor theory. The block, he thinks, cannot (as represented in the reflex theory) be on the afferent side of the co-ordinating centre, it must therefore be on the efferent side. Co-ordination of movement once learnt by the higher centres is carried on, he believes, without the guidance of sensation, and "co-ordinating impulses" are transmitted from the centres down special fibres which are distinct from the motor fibres and lie probably in the posterior columns. Disease of these fibres causes the ataxia of tabes. It is obvious, however, that the existence of these fibres is quite hypothetical, and perhaps we might add unlikely. Neither theory, then, is quite free from objections; it may suffice to have briefly stated the two.

A third view must be mentioned[3] (which appears to be an almost inevitable modification of the motor theory) namely, that the ataxia of tabes is a cerebral symptom, and has nothing to do with the spinal condition. It is based, I believe, on some researches of Jendrassik into the condition of the cortex cerebri in tabes.

THE CLINICAL FEATURES OF ATAXIA.

It is one thing to define inco-ordination verbally and from a physiological standpoint, another to recognise ataxic movements when we see them. Perhaps this ought not to be so; but I imagine that here, as often, the clinical description came first, the physiological definition afterwards.

Let us confine ourselves to the limbs and trunk, and consider the upper limbs first. In moderate ataxia there is a

[1] *Brain*, vol. vi, p. 79.

[2] See Erb on Diseases of the Spinal Cord in Ziemssen's *Cyclopædia of Medicine*, vol. 13, p. 87 *et seq.* (American trans.).

[3] Sydney Kuh, Pathology of Locomotor Ataxia, *Med. News*, March 3rd, 1894.

clumsiness and want of precision about the finer movements, rendering difficult or impossible such actions as threading a needle, writing, fingering a musical instrument, or such handicrafts as involve delicate manipulation, or even such an action as fastening a button. In picking up a pin the hand wavers uncertainly, goes wide of the object, and the fingers fumble in grasping it. There is difficulty in bringing the finger tip rapidly to a given point, say to the tip of the nose. All these difficulties are much magnified when the eyes are closed. Nevertheless, the muscular strength as tested by the dynamometer, or by resistance to passive movements, may be normal. I need hardly stay to contrast verbally this affection of movement with others such as chorea, athetosis, the rhythmical tremors of paralysis agitans, the irregular oscillations of disseminated sclerosis, etc., from the majority of which it is easily distinguished. In the upper limbs ataxia of advanced degree, such as to render them altogether useless, is certainly rare. On the other hand, the fineness of muscular adjustment which is requisite for many hand and finger movements renders the early stages of ataxia more easy to appreciate in them.

Now the movements of the lower limbs may be examined in a somewhat similar way, that is, by making the patient, while sitting or lying down, touch a given point with his toe, touch his knee with the opposite heel, describe figures with his foot, etc.—both with his eyes open and with his eyes shut. But that which is of real importance to him, and that in which ataxia best shows itself, is the functions of the lower limbs in standing and walking, for which their muscles have to be co-ordinated not only with each other, but with those of the trunk. Two different types of ataxic gait are commonly recognised:

1. That of tabes.
2. That of cerebellar disease.

In advanced ataxia due to tabes the patient as he walks (if he still can walk) leans forward with his eyes fixed on his feet; he keeps his feet apart, and, as he advances either foot, is apt to fling it forward and outward in a jerky way to an unnecessary height and with unnecessary force, and bring it back to the ground with a stamp. He is said to "walk like a cock." He can only stand still with his feet wide apart, so as to secure a wide basis of support, and while he looks at his feet. As he stands, the tendons round the ankle-joints twitch spasmodically from his efforts in balancing, just as in a normal person who is standing on one leg.

In the cerebellar type of ataxia the patient walks like a drunken man. He swerves from the direct line of march, and reels as if about to fall to one side or the other; there is the same tendency to keep the feet apart, but none of the flourishing and stamping characteristic of tabes. High authorities maintain, as we shall presently see, that the cerebellar gait is not an instance of ataxia at all, but for the purpose of a discussion it is, perhaps, best to include it.

In the earlier stages of tabes, the contrast with cerebellar disease is not nearly so great, because there is then no flourishing of the feet, and all that is recognisable may be an uncertainty in the movements of the legs, which is increased when the patient rises to begin walking, when he turns, when he stops suddenly, when he tries to walk backwards, and, above all, when his eyes are closed. Again, in standing, the patient may manage fairly even with his feet together; but if he shuts his eyes, swaying movements of the trunk begin which threaten to upset him. This last test, well known as "Romberg's symptom," is considered by most physicians to be evidence (and, indeed, the most delicate test) of the presence of ataxia. Co-existing with it is found, in most cases at least, more or less anæsthesia of the feet or legs. It is therefore, *pro tanto*, an argument in favour of the sensory theory of ataxia. The impressions from his feet failing him, the patient substitutes those from his eyes, and when these also are cut off he falls. Erb indeed says[4] that this staggering with the eyes shut is not due to inco-ordination in the strict sense, but is only evidence of loss of that sensory control which is necessary to equilibrium, for he draws a distinction between processes which serve to maintain the equilibrium and processes of co-ordination. He admits, however, that true ataxia may be increased by closing the eyes. A curious observation has been made in this connection, of which I know no sufficient explanation, that ataxic patients who are completely blind from optic atrophy may stagger when they close their eyes.

[4] *Op. cit.* at pp. 92 foll.

Ataxia as a Symptom.

It remains to say something of the diseases of which ataxia is a symptom. Some of these are so well known that I need only mention them; of some others which are rarer I will mention the main characteristics as briefly as I can:

The following diseases of which ataxia is a symptom are enumerated:
- Diseases of cerebral hemispheres (posthemiplegic ataxia, etc.).
- Diseases of cerebellum.
- Diseases of corpora quadrigemina.
- Diseases of spinal cord:
  - (1) Disseminated sclerosis and other non-systematic lesions.
  - (2) Systematic diseases of posterior columns—for example, tabes, ataxic paraplegia.
  - (3) Diseases of posterior spinal nerve roots (Hughes Bennett).
  - (4) Diseases of peripheral nerves.
- Certain other rare diseases:
  - (*a*) So-called "acute ataxia" or false disseminated sclerosis.
  - (*b*) "Hereditary ataxia" of Friedreich.
  - (*c*) "Hereditary ataxia" of Sanger Brown.
  - (*d*) "Hereditary ataxia" of Déjerine and Sottar.

In disease of the cerebral hemispheres ataxia is an occasional but not a common symptom. I remember a man who, after a sort of apoplectic fit, became, not hemiplegic, but ataxic in one arm. Dr. Gowers has recorded cases of posthemiplegic inco-ordination, in one of which he found, *post mortem*, an old cicatrix in the optic thalamus.[5] Children with defective cerebral development may walk in an ataxic way, like a normal infant that is learning to walk. In connection with cerebral disease, also, we may note the possibility of a hysterical ataxia.

Ataxia from cerebellar disease usually shows itself in the reeling gait which I endeavoured to contrast with the gait of advanced tabes. Now Dr. Hughlings Jackson has for some years maintained that this reel is due, not to ataxia as commonly understood, but to paralysis affecting the muscles of the back.[6] The back muscles being weak, the trunk tends to fall over and the legs have to run after it to prop it up. And recently he has strongly substantiated his view by the publication of a case of cerebellar cyst in which the back muscles were demonstrably weak.[7] So that facts go to demonstrate the truth of this ingenious theory. Nevertheless, there remains a certain residuum of facts which suggest that this may not be the whole truth, to wit:

1. That in some few cases of cerebellar disease the movements of the feet are irregular and stamping as in tabes.[8]
2. That sometimes the arms are ataxic too.[9]
3. That in other diseases where the back muscles are weak, such as pseudo-hypertrophic paralysis, the gait is different from that of cerebellar disease.

We may say that there are at least three possible views with respect to cerebellar ataxia:

1. The simple and precise view propounded by Dr. Hughlings Jackson, that paresis of the back muscles is the essential factor, and that the staggering is complementary to it, being an attempt on the part of the legs to correct the weakness of the back.
2. That, apart from paralysis of either set of muscles, the muscles of the back do not act in proper unison with those of the legs (inco-ordination in the precise sense of the term).
3. That the fault of adjustment lies, not so much between muscles or sets of muscles as between impressions received from the sensory organs of equilibrium (semicircular canals, etc.), and the motor response which they should normally evoke (inco-ordination in a less precise sense). On this theory cerebellar staggering would be analogous to aural vertigo. But, whatever the explanation, the fact remains that this reeling gait is an early and a common, though, perhaps, not an invariable, symptom of disease of the cerebellum, and, some would add, usually of its middle lobe.

Disease of the corpora quadrigemina or their neighbourhood may be characterised by a similar gait. This is not surprising considering their proximity to the cerebellum and

[5] *Medico-Chirurgical Transactions*, vol. lix, p. 31.
[6] British Medical Journal, 1880. vol. i, p. 194.
[7] British Medical Journal, 1894, February 24th (Hughlings Jackson and Risien Russell).
[8] *Brain*, vol. xv, p. 461.
[9] *Neurol. Centralbl.*, 1890, p. 468, and 1884, p. 58.

to the strands which connect it with other parts. According to Nothnagel[10], ataxia of the cerebellar type, occurring early in the course of an illness and followed by an ophthalmoplegia which involves both eyes, but in an unsymmetrical and incomplete fashion, is characteristic of disease of these parts.

In connection with the discussion of these points I may mention a rare form of disease known sometimes as "acute ataxy" sometimes as "false disseminated sclerosis." Leyden distinguishes two forms of acute ataxia,[11] the one (which I omit for the present) due to peripheral neuritis, the other of somewhat uncertain origin, but probably due to small disseminated lesions in the pons, medulla, and cord.

This affection comes on in connection with acute febrile diseases, and mostly during the early stages of them. The onset is acute and usually masked by severe cerebral symptoms such as delirium, coma, and the like. When these subside the patient is unable to speak, or at any rate to speak properly; and as he becomes less completely bedridden, impairment in the movement of the limbs is noticed. Accurate observations are in most instances of a later date, and they show:

1. That the speech is monotonous, somewhat nasal, and laboured, each word and syllable being separated and brought out with effort.

2. An affection of the limbs. The gait, usually described as ataxic, is sometimes simply unsteady and not like that of advanced tabes; but sometimes it is stamping. In a few cases there has been weakness and tendency to rigidity rather than inco-ordination; or indeed the gait may be normal. The movements of the hands are sometimes described as fumbling and ataxic; sometimes there is tremor of the hands. It would appear, therefore, that the speech affection is a more invariable symptom than the affection of the limbs; it is also a more persistent one. Consequently the name "acute ataxia" does not appear a very good one, any more than the name "false disseminated sclerosis." Sensation is normal, and the knee-jerks are exaggerated, or at least retained, not abolished as in tabes. Lesions not unlike those of disseminated sclerosis have been found in the medulla and cord of one such case;[12] but more probably it began as a disseminated inflammation; for the disease is not progressive like ordinary disseminated sclerosis, but tends towards recovery.[13]

Passing to diseases of the spinal cord, we may first notice that the symptom of ataxia may occur in some of those where the lesion is not limited to any one system of fibres, such as incomplete transverse myelitis, meningo-myelitis of a mild type, disseminated myelitis[14], disseminated sclerosis, syringomyelia, etc. But it is most common in diseases characterised by systemic degeneration of the posterior columns. Of these diseases tabes is the chief. About the ataxia of tabes I need say little more than I have done, save perhaps this:

1. That it is generally a late symptom; there may be a long "pre-ataxic stage," lasting even for many years. During this stage, however, the diagnosis of tabes may be made, from the combined presence of three main symptoms—(*a*) Lightning pains, (*b*) absence of knee-jerk, (*c*) reflex iridoplegia.

2. That even in cases of otherwise typical tabes there may exist alongside of the ataxic a certain amount of positive motor weakness.

"Ataxic Paraplegia."

Ataxic paraplegia is a combination of ataxia with loss of power. It is exceptional in simple tabes, but characterises an allied group of spinal cases. Here there is found, *post mortem*, degeneration both of the lateral columns and posterior columns of the cord, and during life an association of such symptoms as we are accustomed to refer to either of these lesions. Thus the gait is of a mixed character, partly characterised by unsteadiness and irregularity of movement, partly by dragging of the feet due to a loss of motor power and a tendency to rigidity. Into the further symptomatology (which I must own is rather variable) I must not now enter. Probably the disease differs essentially from true tabes, and not least in the conditions of its origin. Thus syphilis is said to be a far less frequent antecedent here than in tabes.

Again, combined degeneration of the posterior and lateral columns may constitute the spinal part (and this is in some instances the predominant part) of paralytic dementia. It may originate in hereditary defects, as in Friedreich's disease. It may occur in connection with pernicious anæmia.[15]

Furthermore, ataxia may be produced by lesions external to the spinal cord. Hughes Bennett has published a case in which tabes was simulated by disease of the posterior nerve roots[16], Déjerine and others have showed that it may be simulated by peripheral neuritis.[17] Such facts support the view that afferent stimuli have much to do with co-ordination of movement. When a peripheral neuritis assumes the form of pseudo-tabes, instead of its more common guise of a neuritic paraplegia, then, just as in true tabes, there is ataxia with loss of knee-jerk; but usually the reaction of the pupils to light is normal, the onset of the disease has been more rapid, and the history reveals some antecedent condition favourable to a neuritis. Such antecedents are toxic influences (alcohol particularly, and metallic poisons such as arsenic), glycosuria, acute diseases, especially diphtheria, and, more rarely, tubercle, rheumatism, gout, or syphilis. The diagnosis of this pseudo-tabes from true tabes is of practical importance, because the one disease often gets well, the other rarely, if ever.

Family Ataxia.

Having thus seen that ataxia may result from disease in very various parts of the nervous system, let us leave the anatomical lines, and consider some forms of ataxia which have in common this clinical characteristic that they run in families.

First, the hereditary ataxia described by Friedreich. This is often ranked as a variety of tabes, but the differences are well marked. The characters common to both diseases are (1) ataxia beginning in the lower limbs, and progressing slowly upwards; (2) loss of tendon reflex, in the great majority of cases at any rate. The differences are as follows: Cases of tabes are usually solitary; Friedreich's disease occurs in several members of a family, sometimes in several generations. Tabes is a disease of middle life, and of men rather than of women; Friedreich's disease begins in childhood, at puberty, or in early adult life; and attacks females and males indiscriminately. In tabes the onset of ataxia is preceded or accompanied by numerous other symptoms; by lightning pains and characteristic affections of the pupil almost always; by transient paralysis of the eye-muscles or other parts, bladder troubles, optic atrophy, and affections of sensation—commonly; by visceral crises, perforating ulcers, disease of bones and joints—sometimes. But in Friedreich's disease there are none of these things; sensation is normal, there are no pains, at least during the early stages. On the other hand, the disease has symptoms of its own, for as the ataxia progresses the speech may be involved; it becomes slurred, drawling, and indistinct, and, later, nystagmus may appear. There may develop also actual motor paresis of the legs with some amount of rigidity; and deformities such as club-foot, contractions of the toes, curvature of the spine. Frequently there is a sort of static ataxia, that is to say, a general shakiness or "wobbling" of the head, trunk, and limbs; sometimes this condition is indistinguishable from a mild chorea. The patient has often a stunted figure and semi-idiotic face. The gait has more often the reeling cerebellar type than the foot flourish of tabes. What is the anatomical basis of this disease? It has been said (Hammond, Senator[18]) disease of the cerebellum. This view lacks confirmation *post mortem*. The result of such examinations as we have seems to be that the whole nervous system is small, the cerebellum included, but not more than

[10] *Brain*, xii, 21.

[11] *Zeitschrift f. klin. Med.*, 18, Nos. 5 and 6 (abstract in *Neur. Cent.*, 1891, p. 270). This does not include the rare instances of true tabes in which the symptoms develop acutely.

[12] Ebstein, *Deutsch. Archiv f. klin. Med.*, vols. 9 and 10.

[13] References on this disease are Whipham and Myers, *Trans. Clin. Soc.*, vol. 19, p. 164; Dawson Williams, *Medico-Chir. Trans.*, vol. 75; Westphal, *Arch. f. Psychiatrie*, vol. 3, p. 376, and vol. 9, p. 173. Jaccoud, *Clinical Lectures*, 1886.

[14] Dreschfeld, BRITISH MEDICAL JOURNAL, 1894, vol. 1, p. 1176.

[15] Bowman, *Brain*, 1894, p. 198.

[16] *Transactions of the Clinical Society* for 1885.

[17] *Arch. de Phys.*, 1884. *Arch. de Méd. Expér.*, 1889. *Sem. Méd.*, April, 1893.

[18] *Berl. klin. Woch.*, 1893, No. 21; and 1894, No. 22.

other parts. Upon this general wasting, or (it might be more correctly said) want of growth, there is engrafted distinct degeneration, first and principally of the posterior columns; secondly, and in a less intense degree, of the lateral columns.

A second form of "hereditary ataxia" was described by Sanger Brown in 1892.[19] He collected observations of no fewer than twenty-one cases from a single stock, six of which he observed himself. Here, as in Friedreich's disease, there is a family affection, characterised by ataxia of cerebellar type and without affection of sensation, beginning in the legs and progressing slowly upwards. In two other respects there is an apparent similarity, namely, that speech is affected and that there are involuntary movements. I should suspect, however, from the description given that these symptoms differ somewhat from those of Friedreich's disease, the speech being more explosive in character, the movements more spasmodic. At any rate the similarities end here. The differences are: that the age at onset may be much later, sometimes over 30 or 40 years of age; that the tendon reflexes are retained or exaggerated, not annulled; that there are no distortions of toes or feet, nor spinal curvature. The eye symptoms are different: thus nystagmus appears to be generally absent, but there is a peculiar affection of the levators of the lids (ptosis when at rest, an overaction causing staring when the lids are opened), sometimes ophthalmoplegia externa, sometimes optic atrophy. Sanger Brown's picture of an advanced case represents, I think, something different from Friedreich's disease. He writes:

> There is permanent contraction of the legs. When the patient attempts to talk the tongue appears to move in every conceivable position without being protruded; the face undergoes various inco-ordinate movements; the head is bent forward and moved from side to side; there is protrusion of the chin; and the arms are moved backward and forward. The movements are highly suggestive of chorea, but less rapid.....There is almost complete ptosis. There is scarcely any action of the external ocular muscles.....There is complete and typical atrophy of the optic nerves.

Sanger Brown had no opportunities for any necropsy; but if we may reckon under the same type three cases (brothers) published by Nonne,[20] we find that the results of an examination were as follows: General smallness, apparently from defective development of all the nervous centres; no spinal degeneration, but optic nerve atrophy, and atrophy of the large nerve fibres in the spinal nerve roots.

Nonne, though speaking cautiously, appears inclined to refer the symptoms to cerebellar atrophy, and I notice that Professor Marie speaks unreservedly of this class of case as "hereditary cerebellar ataxy."[21] I should classify them as a different type from Friedreich's disease, but under the same natural order. Both types exhibit as a common element a congenital defect in the structure of the central nervous system, evidenced *post mortem* by its smallness; but the actual points in this weak citadel which degeneration chooses for attack may well be different.

I will conclude by mentioning a rare form of hereditary ataxia described by Déjerine and Sottar,[22] in which the peripheral nerves appear to be the part mainly attacked. The clinical account they give of these two cases (a brother and a sister) suggests a veritable hybrid; some symptoms are those of peroneal muscular atrophy (itself a "family disease"), some are those of Friedreich's disease, some those of ordinary tabes. In one of these cases they found *post mortem* an interstitial neuritis, beginning apparently in the peripheral nerves, and spreading up along the nerve trunks to their spinal roots; there was degeneration of the ascending tracts in the posterior columns, but no more than was, in the author's opinion, secondary to disease of the nerve roots.

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I wish in my remarks to touch mainly on the types of ataxia one meets with in the insane, to indicate briefly their character, and try to correlate them with the results of pathological investigation. I shall first touch on cases which, although they exhibit an ataxic or unsteady gait, yet show other symptoms which so vastly preponderate that the ataxic disturbance is to be looked upon as but a minor part and out come of a vast cerebral change that is going on. In some mental or brain diseases in which there is profound reduction of higher consciousness, with inability to fix the attention, and in which the patient fumbles and stumbles about his room in an automatic stage or a condition of semi-delirium, there is sufficient ataxia and unsteadiness to make him fall frequently to the ground, and these are the patients who, therefore, in our asylums show a tendency to severe bruises about their legs, hips, etc., from frequent slight falls. In these the element of weakness (adynamia) does enter to a certain extent; but what one has to bear in mind is the grave and profound nutritive cerebral disturbance taking place, the rambling or weak and incoherent utterance—when they do speak at all—the restless and fidgety movements of the hands, the furred tongue and slight febrile temperature, and the similar restless fumbling and stumbling about the room during all this time. This may occur in certain phases of general paralysis, in cases of alcoholism during the stages when there is much cerebral breaking down associated with a chronic delirious or somnambulistic stage, in senile recurrent attacks of mania (this not frequently), and in many other of the insanities.

In all these cases there is much brain breakdown going on, much nutritive disturbance, and, when the attack is over, these patients may exhibit much mental reduction, loss of memory of their previous condition, and considerable bodily and cerebral fatigue. They are a striking group of cases as showing how, with the profound nutritive disturbance going on in the brain, there may be a low semi-conscious life during which restlessness, loss of memory and attention, and overaction (irritation) of lower cerebral centres, will produce among other symptoms a restlessness combined with ataxia. I take it that in these cases we have weakness and also lack of co-ordination, the nutritive disturbance in the kinæsthetic area being the cause of the ataxia. *Per contra*, compare cases of stupor where all restlessness or delirium is absent, where the mental functions can find no outward expression, and where there is no ataxia, the patient being able to stand still or, if induced, to walk without ataxia. I think the contrast is a striking one.

Coming now to the group of ataxia from *spinal* diseases, I shall only refer to three of the insanities in which ataxia may be present. These are: general paralysis, with early or predominant spinal symptoms; chronic alcoholism; and cases of true tabes passing on to general paralysis.

Dr. Ormerod states that there are cases of tabes with a pre-ataxic stage—that is, loss of knee-jerk and reflex iridoplegia, and that these are distinctly rare. In our asylums we certainly—not uncommonly—get such cases. Many of these are cases of general paralysis. I can recall several such cases to mind in my own experience. Such patients have absent knee-jerks, the pupils are unequal, they react to accommodation, but not (or very feebly) to light; and some of the articulatory or early mental symptoms of general paralysis may also be present. In other words, there are cases of general paralysis with tabetic or pseudo-tabetic onset. I shall say "pseudo-tabetic" onset rather to distinguish them from cases of true tabes, for sometimes one can do so. The further development of these pseudo-tabetic cases is interesting, for they may take take one of two main pathways, *a* and *b*.

In (*a*) one type, the tabetic symptoms get intensified, and distinct ataxia in gait is developed. They further get lightning pains and allied sensations, including girdle and other sensations. On testing with a pin head or pin point there is diminution of cutaneous sensibility to touch and pain—irregularly distributed about the lower legs—sometimes erroneous localisation of the touch or prick in the opposite limb (allocheiria). The knee-jerk remains absent, never returns, and the ataxia of gait is intensified, and remains so while the disease progresses.

In the other type (*b*) the pre-ataxic symptoms disappear, having never attained further development; but now a group of cerebral symptoms appear on the scene. The patient gets moderate or frequent attacks of epileptiform convulsions with more permanent and deeper brain damage than in ordinary epileptic or epileptiform attacks, and with these there is a slight or moderate rise of temperature, much flush-

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[19] *Brain*, vol. xv, p. 250.
[20] *Archiv f. Psychiatrie*, vol. xx, p. 283.
[21] *Semaine Médicale*, 1893, No. 56.
[22] *Mémoires de la Société de Biologie*, Series 9, vol. v.

ing and sweating, and great throbbing of vessels in the face and head, slight or moderate pareses of the hands or legs, which after a time are recovered from. These attacks recur, and, as the result of their recurrence, they produce distinct spinal, or rather bulbo-spinal effects. Amongst the latter are a group of effects which simulate closely the lesions of lateral sclerosis. The knee-jerk, which was absent, now returns, and is even exaggerated; the pseudo-tabetic sensory troubles are abolished; he gets ankle-clonus, the former ataxic gait, with its wide basis of support in locomotion, is replaced by a normal gait, in which, however, a tendency to spasm of the calf muscles now appears; the gait becomes spastic. With the progress of the cortical disintegration accompanying each "seizure" or series of seizures, the spinal symptoms get more and more like those in spastic paraplegia, the spastic condition coming on first, and the plegia or paraplegia following later on.

There is (*c*) yet another class of cases in general paralysis in which we can find no ataxic stage, but in which the cerebral symptoms are the first and earliest; in which seizures with loss of consciousness rapidly develop, ending in lateral sclerosis with excess of knee-jerk and spastic gait. In these there is no previous loss of knee-jerk, wide gait, or early spinal sensory disturbances. It is especially in cases of alcoholic general paralysis, that is general paralysis in which alcohol plays a prominent part in causation, that this type is present. We might perhaps put these in a separate group under the heading chronic alcoholism, with foci of brain-softening, followed by descending lateral scleroses.

I now come to true tabes. This may occur in the insane apart from brain disease, but the close connection between tabes, alcoholism, and general paralysis, must never be overlooked. It may be that tabes is one of the system diseases, which in the growth of our civilisation mark the milestones, so to speak, on the way to insanity. But however that may be it is a disease by itself. In it the lesion is one of the posterior root nerve-cell of the spinal cord, and especially of its central process; its origin is therefore properly extra-spinal; it involves the ganglion cells which, embryologically are developed from the "Zwischenstrange" of His, and whose processes grow the one outwards to the surface, etc.; the other inward to the spinal cord. It is a system disease of these cells, and we must, with Gowers, Westphal, and others, look upon it as a primary nervous disease—a true neuropathy. It is different from those spinal diseases secondary to perivascular scleroses, to repeated vascular disturbances and endarteritis, or to cortical destructions.

It would be wise to establish a distinction between true tabes and all these other forms (pseudo-tabes), including those secondary to cerebral disease. In the spinal cord substance we may have endarteritis or periarteritis, and these two go together to a certain extent. In cases of the pseudo-tabes of alcoholism or of general paralysis it is frequent to find a sclerosis in the non-nervous tissues of Goll's column affecting the pia mater, the connective tissue, septa, and the adventitial sheaths of the vessels. Whatever may be the cause of these—whether a direct effect of alcohol or not on the vascular and connective tissues—these produce other effects (secondary) on the intrinsic nerve tracts within the posterior column. Examination of the posterior nerve roots outside the cord may in early cases show no lesion. I have had five such cases in which I have examined the spinal cord; and within the cord substance lies the disease, the tracts and foci of scleroses. This has two effects—namely, (*a*) nutritive and (*b*) pressure effects on the intraspinal tracts. The nutritive disturbance is due to the endarteritis, with lessening or obliteration of the lumina of the small vessels; the pressure effects are due to the perivascular sclerosis and coalescing of these sclerotic patches. These changes have their seat of election in the posterior column, and as a result they produce destruction and degeneration of nerve fibres there. The posterior part of Goll's column, and a group of fibres near the posterior commissure are most affected. In the mild and recoverable pre-ataxic stage these lesions cannot be advanced, and the spinal disturbances are to be looked upon as due to commencing changes, vascular engorgement, and exudation, owing to the early effects of the alcohol or other agent, but their disappearance in certain cases is a proof that the lesion has not progressed further, or produced much actual destruction of nerve tissue.

A word as regards the post-cerebral symptoms—that is, spastic following paraplegic conditions. Westphal, Mickle, Bevan Lewis, and others have shown that here we have diffuse and scattered degenerations in the pyramidal tract—especially its posterior part—associated in some way either consecutively or simultaneously with the breaking down in the cerebral cortex (Rolandic areas). The spastic ataxias in such are all secondary to brain mischief, and must be differentiated from the primary spinal lesions—that is, intrinsic cord affections, whether alcoholic, tabetic, or general paralytic, in which the nerve structures suffer in a different way, and probably in sequence to lesions of vascular and non-nervous elements. Our pathological and clinical material in the West Riding Asylum is the basis on which I found the above contribution, with the view of drawing more attention to the study of these nervous lesions in the insanities.

III.—H. Waldo, M.D.Aberd.,
Physician to the Bristol Royal Infirmary.

Dr. Waldo said that ataxia appeared to be a symptom of many organic diseases of brain and spinal cord, as well as of the peripheral nerves, and also occurred in hysteria. He touched upon its presence in disseminated sclerosis, ataxic paraplegia, cerebellar tumours, hysteria, and in tabes, and related a case of tabes with retention of knee-jerks, with the presence of other characteristic symptoms of the disease. Dr. Waldo gave short notes of a case (which he demonstrated to the meeting) of early tabes, in a man, aged 48, with Charcot's disease of the knee-joint and perforating ulcer of the foot.

IV.—Dawson Williams, M.D.,
Assistant Physician to the Children's Hospital, Shadwell.

Dr. Dawson Williams said he thought that the profession would be very much indebted to Dr. Ormerod for the admirable classification of cases of ataxia which he had worked out in his introductory remarks. The need for such classification was very great, as there could be no doubt that great confusion existed as to the true pathology of cases presenting ataxia as their most prominent, or one of their most prominent, symptoms. Dr. Williams referred to the occasional occurrence after all, or almost all, the acute specific diseases of a widespread nervous disorder, which presented in many instances the symptom of ataxy. In the acute stage these cases might present a complex of symptoms which very closely resembled the condition of a patient with advanced locomotor ataxy. Mistakes in diagnosis had actually occurred in post-diphtherial ataxy. The majority of the patients who survived the acute attack no doubt recovered completely, but in some a chronic condition remained, which resembled in its symptoms disseminated sclerosis. He added that it was interesting to observe that after an acute infectious disease, such as diphtheria, the ataxy was of acute type, whereas after a chronic specific disease, syphilis, we got a chronic progressive disorder, the typical form of locomotor ataxy.

V.—William Gordon, M.A., M.B.Cantab.,
Physician Devon and Exeter Hospital.

It is no paradox to say that it is wonderful that whilst still we know so little about inco-ordination, we already know so much. It is wonderful because the subject is so vast, embracing almost all we know about the nervous system, and so difficult—difficult not only anatomically in unravelling the tangled systems of fibres and cells, but also physiologically in tracing the course of sensory tracts which plunge into relay after relay of nerve cells on their path from the periphery. But difficult as is the task of the physiologist, he is, I think, doing more than the clinician to bridge the gulf which separates them. On the clinical side there is, it seems to me, a lack of classification. Dr. Ormerod has given us a most able pathological classification. Would it not be well to classify the cases, as far as possible clinically, according to the probable part of the mechanism involved, and clinically to try to trace the distinctions between the ataxies of each group? Thus we might have (A) *afferent* ataxies, including

(1) those due to cutting off of afferent impulses, as in tabes; and (2) those due to the passage up of abnormal impulses, as in Menière's disease. (B) We may perhaps have *efferent* ataxies. If Charcot's surmise as to the cause of tremor in insular sclerosis is correct, may the same cause not account for the ataxy, which seems merely a gross exaggeration of the tremor? (C) We should also have *central* ataxies, and these might differ according to whether: (1) Groups of nerve cells were unequally weakened, of which we may, perhaps, take cerebellar ataxy as an example, following Dr. Hughlings Jackson's view that the cerebellum especially subserves the trunk muscles, and that these are weak because the cerebellum is damaged. (2) Conducting tracts between nerve cells might be altered so as to conduct either too sluggishly or too easily. With respect to too easy conduction, I should like to refer to a common and marked form of ataxy, that of chorea. If we watch a choreic patient whom we have told to perform a simple movement of the wrist and fingers we find the act carried out with great disorder. It has been said that the coexisting involuntary movement hampers the voluntary. I think there is more than this. The attempt at voluntary movement excites the disorderly and harassing involuntary movements, and it excites these mainly in parts where cortical representation is close to that of the voluntary movement itself. Thus a movement of the hand and wrist is very apt to cause all sorts of disorderly movements, not only in other muscles of the hand and wrist, but in the elbow and shoulder. Again; tell a child nearly well from chorea to hold the hands over the head. The fingers and thumb may be steady until she is told to put the tongue out, when the tongue movement sets up movement in the fingers and thumb. The ataxy of chorea seems due to a too easy spread of motor impulses from centre to centre along the cortex.

VI.—J. MICHELL CLARKE, M.A., M.D.Cantab.,
Physician and Pathologist Bristol General Hospital.

DR. MICHELL CLARKE made a few brief remarks on the points of difference between Friedreich's disease, tabes dorsalis, and ataxic paraplegia, and observed that the term "hereditary" in the strict sense of the word as implying the direct ancestors of the patient were similarly affected, was in many cases a misnomer. He showed a case of Friedreich's disease with retention of the knee-jerk, but without increase of the deep reflexes or spastic rigidity; the brother of this patient suffered from similar symptoms, and had died recently from a cerebellar tumour (see below). He also showed another case of Friedreich's disease, and a case of tabes dorsalis of some eight years' duration, affecting chiefly the upper part of the cord, in which the knee-jerks were present, and there was slight nystagmus in the lateral positions of the eyeballs.

A CASE OF FRIEDREICH'S DISEASE OR HEREDITARY ATAXY, WITH NECROPSY.

By J. MICHELL CLARKE, M.A., M.D.Cantab., M.R.C.P.Lond.,
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HISTORICAL.

THE elder brother of the patient whose case I am about to describe is still living, aged 37, and the disease in him is very slowly progressive. He is now not able to walk or stand without support of some kind, and the movements of "static ataxia" are more marked than they were in 1889. His symptoms are precisely similar to those of the patient the subject of this paper, and I need not therefore give them in detail. He still shows the very unusual feature in Friedreich's disease that the knee-jerks are lively, perhaps brisker than normal, as was the case in his younger brother; at the same time there is no ankle clonus, no excess of the deep reflexes in the arms, and no muscular rigidity or spasm.

[I took the opportunity of exhibiting this patient after the discussion on ataxia opened by Dr. Ormerod at the Bristol meeting of the British Medical Association, and he was considered by those present to be a typical instance of Friedreich's disease.]

The following is a copy of the notes taken in 1889:[1]

The father of these patients, a butcher, A., suffered from heart disease and dropsy, and died in an asylum, apparently of melancholia. His parents died of old age, his father living to be 96; one of his sisters committed suicide; family healthy. The patient's mother, B., is still living. Her father's father died at the age of 99, and his mother at the age of 101. Her father lost all his hair before he was 30 years old, and died of diabetes; her mother died at the climacteric period. There were two other sons and another daughter, all of whom survive. A. and B. had fourteen children—(1) Georgina, aged 37, healthy; (2) Albert, aged 35 (one of the patients); (3) George (drowned); (4) Isaac (died of croup); (5) a son, aged 25 (examined and found healthy); (6) William, and (7) Mary Ann (both died, at the age of 5 months, of "weakness"); (8) Lizzie, aged 21 (examined and found healthy); (9) a daughter (died at the age of 1 month, overlaid); (10) Caroline, aged 18 (one of the patients); (11) Isaac (died, at the age of 3 years, of croup); (12) John, aged 14 (examined and found healthy); (13) Isaac, aged 11 (one of the patients); (14) a child prematurely born at seven months (died in three days). The father was a heavy drinker; no evidence of syphilis could be obtained. The mother is a strong, healthy woman, and does hard work. Has never suffered from fits, from any nervous affection, or any severe illness. She was carefully examined as to state of reflexes, etc., and found to be normal in every respect.

CASE II.—I. A., aged 11, is an intelligent boy, and looks healthy. Since the age of 4 his mother considers he has been "restless," and that this restlessness increases. He often falls down when walking, and runs in a zigzag way—that is, from side to side, like a drunken man. He has had no fits or bad illness. As he sits he lets his head fall back, or from side to side, and keeps his mouth open; this gives him a stupid appearance; at the same time there are rocking movements of the head and trunk. Standing with feet together there is well-marked static ataxia; movements of tendons on dorsum of feet, and slight movements of shoulders, head, and arms, whilst the feet are shifted a little from time to time; when his attention is occupied—as by counting fifty—these movements become more marked. He stands by choice with his feet apart. None of the movements at all resemble those of chorea. With his eyes shut there is more general unsteadiness and decided increase in the swaying movements of the trunk and head. There are occasionally slight emotional, not twitching, movements about the mouth. There is slight nystagmus on extreme convergence of the eyes. The pupils are equal, and act well to light and accommodation. Sensation and muscular sense normal, muscles firm, and strength fair. Special senses normal. Speech good. The tendon reflexes in the upper limbs were not obtained. The knee-jerk on the left side was about normal; that on the right diminished, but present. Plantar and other superficial reflexes normal. His gait is a little unsteady, and he is quite unable to stand on one leg. The movements of the upper limbs show some ataxy, which becomes more marked when the eyes are shut.

Since the above report, which was made in 1889, the patient was under my observation from time to time, and became progressively, though slowly, worse. When seen in March, 1892, he was decidedly worse; though he managed to get about, he often fell down in walking. He walked with a staggering gait, swaying from side to side, and stood with legs wide apart. He complained at this time of giddiness, but not of headache or vomiting. The optic discs were healthy. The pupils were equal, and reacted well; there was very slight nystagmus in lateral positions of the eyeballs. No paralysis of any cranial nerve. Shortly before this he had had a mild attack of influenza, and three days afterwards some sort of fit, in which there was momentary loss of consciousness. He was easily tired, and often felt faint. He breathed through the mouth, with snoring, rather irregular respiration. He was never still, there being constant irregular movements of the limbs, and twitching of the angles of the mouth. The knee-jerks were normal; there was no muscular rigidity nor spastic symptoms.

In February, 1893, I admitted him into the hospital for purposes of better observation. We then learnt that he had suffered, at intervals of about a month, ever since he could remember, from attacks of sickness and headache coming on after meals, and for three or four years previously from attacks of sharp shooting pains in the upper part of the right thigh.

At this time he had grown in height (aged 15), and was a lanky lad with badly developed musculature, but with no sign of rigidity or muscular wasting. His expression was dull and heavy, but he was intelligent and bright and cheerful, helping in the work of the ward so far as he was able. The respiration was of the type described above. He had no difficulty in swallowing. His speech was indistinct, somewhat nasal and hesitating, jerky and irregular, but not scanning.

The patient was never still, there being constantly slight, irregular, somewhat jerky movements of the limbs and of the muscles of the head and trunk. He protruded his tongue straight, but it was tremulous, especially at the tip. There was a high arch to the palate.

[1] *Lancet*, vol. i, 1889.

Gait unsteady, the feet raised somewhat high, and the right foot turning inwards. He did not walk straight, but staggered occasionally to one or the other side. The gait was much more ataxic if he tried to take long steps, and he then threw his arms about to balance himself. On standing with his feet together swaying movements from before backwards, and also occasionally from side to side, occurred. These were only slightly marked if the feet were kept wide apart, and were much exaggerated when the eyes were closed.

Arms.—He could bring the tips of his fingers together with outstretched arms, and could touch the tip of his nose with his forefinger, but these movements were uncertain and ataxic, markedly so when carried out slowly or with the eyes closed. He could pick up a pin fairly easily with the aid of sight, but without it only with great difficulty.

Eyes.—On steady fixation of an object slow lateral movements of the eyes occurred, and occasionally a from side-to-side movement of the head. There was nystagmus in extreme lateral position of the eyeballs; no squint; no diplopia. Pupils equal, and reacted well to light and accommodation.

Sensation everywhere normal to touch, pain, and temperature. No delay in perception of sensation; sense of position of limbs, and of movement (muscular force exerted) appeared to be normal. No affection of special senses. There was an absence of expression about his face, which gave him a silly appearance, not justified by the quite average degree of intelligence he possessed.

Reflexes.—Superficial reflexes were all present and normal. The knee-jerks were normal; elbow-jerks present; wrist-jerks not obtained; no ankle-clonus. No interference with micturition or defæcation.

Spine.—No tenderness over spinal column or irregularity of spinal processes; slight lateral curvature to right in mid-dorsal, to left in lumbar region.

Feet.—The feet were hollow and shortened, presenting the deformity described in cases of Friedreich's disease.

It is important to note that there was at this time no optic neuritis, no headache, vomiting, or other symptom of intra-cranial tumour, and that the symptoms present were precisely those noted in 1889 in more aggravated form.

I heard that he was getting rather more rapidly worse during the summer, and that whilst in the country at the end of July he was seized with vomiting of cerebral type, headache, inability to stand or walk, and some convulsive seizures of doubtful nature. He was admitted to the hospital on August 5th, 1893, a few days before I returned from my holiday. I saw him on August 14th. He was then very dull and heavy, but could be roused to answer questions. He complained of intense pain in his head, and constantly vomited. There was intense optic neuritis in both eyes. He lay constantly on his left side, with his legs flexed; he seemed unable to lie in any other position, and when placed on his back or right side, turned round, apparently involuntarily, on to his left again. The eyes were sometimes directed to the left, but this was not constant. There appeared to be some paresis of the right sixth nerve. The knee-jerks were probably very faintly present, but this was doubtful. He could neither stand nor walk, and there was incontinence of urine and fæces. He became rapidly worse, passing gradually into a state of coma, which was profound for two or three days before his death on August 21st. The knee-jerks were entirely lost for some days before death.

From the 17th to the 21st the respiration became extraordinarily slow and irregular, the rate being only 7, 8, or sometimes 10 respirations to the minute. He sometimes paused eight to ten seconds between two respirations. The pulse-rate varied from 72 to 92. On August 17th he suffered from attacks of marked opisthotonic spasm, the body resting only on the head and heels, and this opisthotonos increased in intensity, remaining constant for about twenty-four hours before death. I should have said that on admission there was marked retraction of the head and neck and rigidity of the posterior muscles of the neck. The urine contained no albumen.

Necropsy.

At the necropsy thirty-three hours after death, the body was emaciated. The lungs were congested, especially their bases and posterior borders, and the bronchi full of mucus The heart was flabby and uncontracted, the right cavities full of dark clot. The thoracic and abdominal viscera were otherwise normal. The skullcap was thick: the membranes of the brain were healthy, and there was no sign of meningitis. The longitudinal sinus was empty. The vessels on the convexity of the brain were empty, with an absence of cerebro-spinal fluid; the surface of the brain was dry. On section, the cerebrum was somewhat soft and bloodless, but otherwise normal; cortex cerebri appeared healthy. The cranial nerves appeared to be soft, which, together with the slight softness of the cerebral white matter, was attributed to the *post-mortem* examination taking place thirty-three hours after death in very hot weather.

Springing from the under surface of the right half of the cerebellum was a round, somewhat lobulated, tumour, which lay beneath the cerebellum by the side of the pons and upper part of medulla. The neighbouring parts were pushed aside, but not destroyed by the growth; the right half of the cerebellum slightly upwards, the pons to the left. The tumour was of soft consistence; in appearance resembling the white matter of the brain, and distinctly demarcated from the surrounding cerebellar tissues. On microscopical examination it proved to be a small round-celled sarcoma. The aqueduct of Sylvius was greatly, the third and lateral ventricles slightly, distended and full of cerebro-spinal fluid.

The cerebellum, greater part of the cerebrum, pons, medulla, and cord were hardened in Müller's fluid.

On making a series of vertical sections through the medulla and pons, from before backwards, the tumour was found to be largest at about the mid-point of the cerebellum (antero-posteriorly). It here measured $1\frac{3}{4}$ inch in its horizontal and $2\frac{1}{4}$ inches in its vertical diameters. The flocculus and lobules on the under surface of the right half of cerebellum were invaded and destroyed by the tumour, the central white matter on this side was much flattened, compressed, and partly destroyed; the lower part of the pons was pushed to the left and tilted so that its left side lay higher than the right. In transverse sections the posterior fibres of the middle peduncle appeared to be fewer in number on the right than on the left side, especially those in the superior part of the pons.

Microscopic sections showed that except at this part the fibres of the middle peduncle were about the same on both sides. Both medulla and lower part of pons were slightly distorted by pressure, the right side being a little flattened, the left elongated in the vertical direction. The only striking change here was a total atrophy on the right side of the flattened band of white fibres that runs in a longitudinal direction in the floor of the fourth ventricle, dorsal, and superficial to the nucleus of the twelfth nerve. The restiform body was normal on both sides; arcuate fibres and nerve nuclei normal. In the lower part of the medulla the nucleus of the cuneate column appeared to be deficient. The pyramidal tract was healthy throughout the pons and medulla. In the pons, the fillet, both lateral and mesial, the brachium conjunctivum, and posterior longitudinal bundles, were all noted as normal.

Sections of the cerebellar cortex were healthy, both as regards grey and white matter, but on the right side the vessels were everywhere distended.

In the region of the basal ganglia sections taken through the internal capsule showed nothing abnormal. Sections of the motor area of the cortex, and from the frontal and parietal lobes, were healthy. It was specially noted that the large pyramidal cells of the the motor cortex were well developed and normal in appearance.

The above sections were stained by Weigert's method for all parts, in addition for cerebellum by eosin and logwood, and the Biondi-Heidenhain triple stain, and for cerebrum by aniline blue-black. The sections of the cord were stained (after hardening in Müller) by Weigert's stain, Pal-Weigert, borax carmine, Biondi-Heidenhain triple stain, and by aniline blue-black. Some were embedded in celloidin, others not.

First, even allowing for the fact that the patient was aged 15, the cord appeared to be small.

The following measurements were taken after hardening, the transverse diameter being given first:

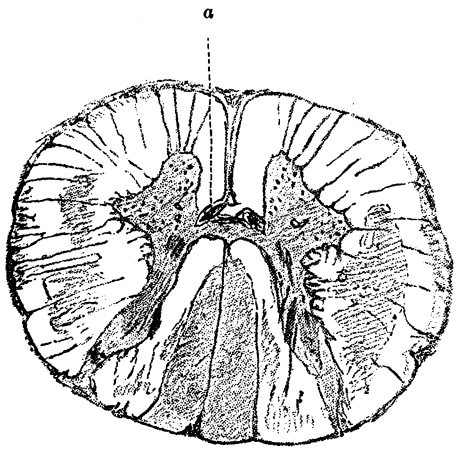


Fig. 1.

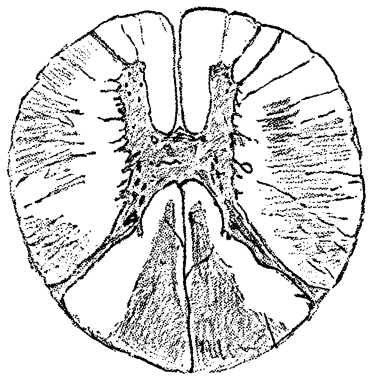


Fig. 2.

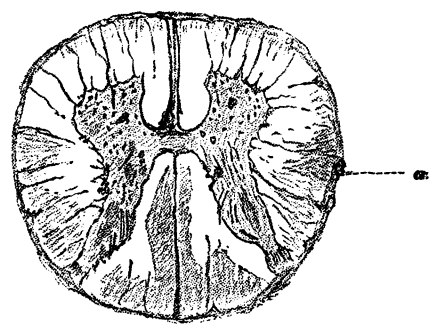


Fig. 3.

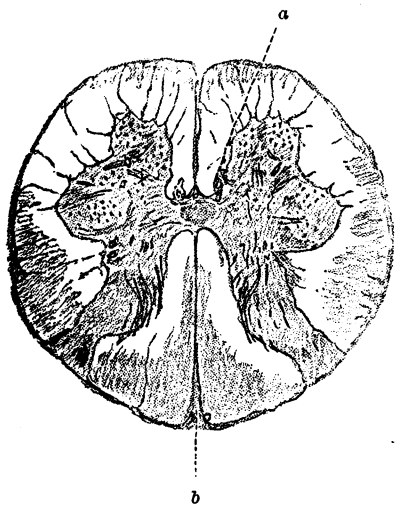
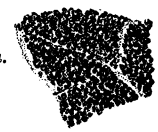


Fig. 4

Fig. 6a.



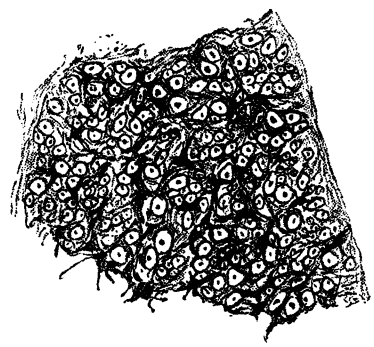


Fig. 5.

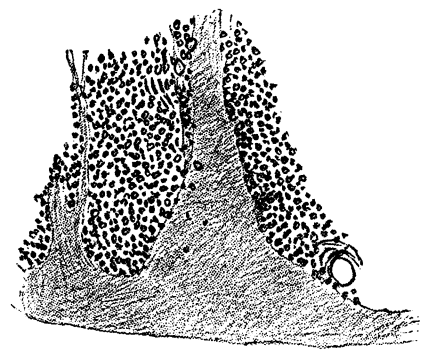


Fig. 6b.

EXPLANATION OF FIGURES.—Figs. 1, 2, 3, 4 are $7\frac{1}{2}$ times the natural size. Fig. 1 from cervical region. Fig 2. from mid-dorsal region. Fig. 3 from junction of dorsal and lumbar regions. Fig. 4 from lower lumbar region. The seat and extent of the degenerated areas is indicated by the shading, the depth of which roughly represents the degree of degeneration. Fig. 1 (*a*) large vessel. Fig. 3 (*a*) thickened pia mater with large vessel with thickened walls. Fig. 4 (*a*) large vessel; (*b*) widening out of posterior fissure, filled by homogeneous tissue. Fig. 5 × about 250 times. Zeiss apochromatic. 4 mm, compens. oc. 4. From anterior column in dorsal region. To show the richness in neuroglial tissue of this part of the cord, which contained no degenerated fibres, and appeared otherwise healthy. Fig. 6 (*a*, *b*) × Zeiss, A. Oc. 3. Fig. 6 (*b*) from margin of posterior fissure to represent as closely as possible the average degree of degeneration present in this cord in the posterior columns (and lateral columns where these were most affected) and number of nerve fibres remaining, for comparison with Fig. 6*a*, in which the number of fibres in a healthy part of the cord is shown.

	Cord in my Case.		Normal Cord.*
Mid-cervical ...	... 11 × 8 mm.	...	14 × $9\frac{1}{2}$ mm.
Lower ,, ...	... 9 × $7\frac{1}{2}$ mm.	...	11 × 8 mm.
Mid-dorsal ...	... $6\frac{1}{2}$ × 6 mm.	...	9 × $8\frac{1}{2}$ mm.
Lower ,, ...	... 7 × $6\frac{1}{2}$ mm.	...	$9\frac{1}{2}$ × $7\frac{1}{2}$ mm.
Upper lumbar ...	... 7 × $6\frac{1}{2}$ mm.	...	9 × 8 mm.
Mid ,, ...	... $7\frac{3}{4}$ × $7\frac{1}{4}$ mm.	...	10 × 9 mm.
Lower ,, ...	... $7\frac{1}{2}$ × 7 mm.	...	$9\frac{1}{2}$ × $8\frac{1}{2}$ mm.

* For the above figures of the healthy cord I am indebted to MM. P. Blocq and Georges Marinesco, *Sur l'Anatomie Pathologique de la Maladie de Friedreich*. Paris. 1890.

In the fresh cord no lesion whatever could be detected. and after hardening in Müller there only appeared indefinite slight change in Goll's column. In the sections stained by Weigert's and Pal-Weigert's methods the following changes were observed. I may premise that there was nowhere complete and absolute degeneration of any tract but that the change was partial or diffuse:

Anterior roots everywhere healthy.

Posterior Roots.—Degenerated fibres present in many places but only few in number, most obvious in mid-cervical and mid-lumbar regions. These roots were normal in the lower lumbar and sacral regions.

Posterior Columns.—More or less degeneration of Goll's column was present throughout the cord, most marked and extensive in the cervical region; it chiefly affected the posterior part and centre of the column and did not reach up to the posterior commissure; it was also well marked at the dorso-lumbar junction. In some parts of the dorsal region degeneration did not affect parts of Goll's column alongside of the posterior fissure, but left a narrow elongated tract of healthy fibres.

In the upper lumbar region degeneration did not extend so far forwards in Goll's column, and affected a narrow strip of the most posterior part of the postero-external column along the periphery of the cord as far as the internal posterior root zone; below this degeneration in the posterior column affected the posterior part only of both Goll's and Burdach's columns. In the mid-dorsal region both columns were also similarly affected. In the lumbar and dorso-lumbar regions the posterior fissure was widened out and occupied by a wedge-shaped area (apex forwards) of non-nervous tissue. Burdach's column was slightly affected in its middle part in the cervical region. In one section in the lower cervical region the internal posterior root zone was slightly affected; in all others Lissaner's tract with the entrance of the posterior root fibres was quite intact. There was an excessive number of very fine fibres in the posterior columns. Just above the decussation of

the pyramids there was slight degeneration in the cuneate nucleus, and (?) in the nucleus of the slender column.

Lateral Columns.

In the cervical region, middle and lower parts, is a small ill-defined crescentic area of diffuse degeneration just external to the tip of the lateral horn, and on the surface of the cord, opposite the lateral horns, there is in some sections a small wedge-shaped area from which nerve fibres are absent, and over which the pia mater is thickened. There is well-marked diffuse degeneration in the crossed pyramidal tracts, and in a small area just external to the entrance of the posterior roots. The direct cerebellar tract is however healthy. In many cervical sections there is a little degeneration running round the surface of the cord, and in the mid-dorsal and dorso-lumbar regions this marginal area is damaged by thickened pia mater and its ingrowths into the cord. The direct lateral cerebellar tract is slightly affected in places in the latter regions; but though there is a general excess of structureless intervening substance its fibres are conspicuously large and healthy.

In the dorso-lumbar and lumbar region there is on the margin of the cord just anterior to the crossed pyramidal tracts a small wedge-shaped area (base of wedge outwards) of diffuse degeneration, and in many sections there is, in addition, an indentation of the cord filled with structureless material, with thickened pia mater over it containing large vessels. The crossed pyramidal tracts show evident degeneration, especially marked in the lower lumbar region, but everywhere diffuse and partial, and nowhere absolute. There is also a small patch of degeneration just external to, but not involving, the entrance of the posterior root.

In the sacral region there is slight degeneration in the crossed pyramidal tract and in the marginal area just in front of it. The vessels which pass through the lateral columns, and the processes extending into them from the pia mater, are more conspicuous than normal in most sections.

The anterior columns appeared normal in these preparations, except for slight degeneration in the most anterior part on the surface of the cord in the lumbar region only. In the middle and lower lumbar regions there appeared to be a paucity of fibres running through the posterior horns from the posterior roots, and in one section only the fine fibres which enter and those which run longitudinally close to Clarke's column on its inner aspect appeared deficient, but in all the other sections these sets of fibres were normal, as was also the band of fibres which enter the posterior horn from its inner side, the longitudinally running bundles at the posterior extremity of tubercle of Rolando, the posterior commissure, and the fibres which leave the grey matter for the lateral columns.

There was a very large number, probably abnormally so, of very fine fibres in the lateral columns, as in the cord generally. In sections stained in aniline-blue-black, carmine, etc., the cells of the anterior cornu, of the lateral horn, and of Clarke's column appeared healthy in all respects.

There appeared to be an excessive amount of neuroglial tissue in proportion to the nervous elements, most marked in the anterior and lateral columns. In the anterior columns in carmine-stained sections this was most obvious, there being in them a large number of very large neuroglial cells between the fibres. In places there was much homogeneous or structureless substance between the nerve fibres, and this condition, so marked on the marginal zone of the cord, has been already alluded to. In places there was a peculiar arrangement round the vessels of fibres arranged concentrically or in whorls, and this peculiarity has been already described by Déjerine. The vessels generally showed thickened walls, chiefly due to the adventitial coat, and most marked in those of the pia mater, but also in those of the grey matter, perhaps especially in the cervical region, and in one place in Clarke's column.

The changes in the pia mater, correlated as they were to underlying changes in the cord, have been already mentioned. It remains to be added that the central canal was blocked throughout the whole length of the cord to just below the decussation, where it became patent, with the products of desquamated cells. Sections of the median, ulnar, sciatic, internal popliteal, and peroneal nerves were made, and showed these nerves to be normal.

Commentary.

To sum up, the morbid changes affected chiefly the posterior columns, next the lateral, with involvement especially of the margin of the cord, with thickening in the pia mater and in the walls of the vessels, and probably with some general excess of neuroglia, and small size generally of the cord. The changes were diffuse, and nowhere had produced complete degeneration, and varied much at different levels. They correspond to those found in the cases of this disease previously reported, but are slighter in degree; in fact, the symptoms during life appear to have been more severe than the changes in the cord would have led one to expect. It must, however, be remembered that though the morbid alterations in single sections were not severe enough to lead to absolute destruction of any one tract of fibres, yet the cord is involved throughout its whole extent.

An interesting feature of the case is the complication of it by the growth of a cerebellar tumour; unfortunately, I did not see the patient until near the fatal termination. The tumour was of very rapid growth, and gave rise to such marked symptoms as to leave no doubt as to the diagnosis; so that the symptoms due to the tumour could be distinctly recognised as a fresh addition to those of Friedreich's disease.

Lastly, the disease in this case was not hereditary, in the sense that either parents or grandparents suffered from it; all that can be said is that there is a marked neuropathic tendency in the family. I have, also, under observation two brothers from another family affected with this disease, one of whom was shown at the Bristol meeting. In these cases there is also no history of any other member of the family having been affected, but two of the mother's sisters died of diabetes, and a father's brother committed suicide. There is no evidence of a syphilitic taint.

DIRECT INTRODUCTION OF URIC ACID INTO THE BODY; ITS BEARING ON THE PREVENTION AND TREATMENT OF DISEASE.

By Alexander Haig, M.A., D.M.Oxon., F.R.C.P.Lond.,
Physician to the Metropolitan Hospital and the Royal Hospital for Children and Women.

As I have previously pointed out in the *Journal of Physiology* (vol. xv, p. 167) and in my book,[1] uric acid may be introduced into the body, and its physiological effects observed like those of mercury, belladonna, nux vomica, or any other drug, by the simple process of swallowing it. In judging of its effects, however, the laws which govern its solubility in the blood must be taken into account; and it is by neglecting to do this that previous experimenters have, I believe, fallen into serious error. Thus, when 2 or 3 grains of uric acid are swallowed along with the food they pass little by little into the blood along with the other products of digestion; but unless the alkalinity of the blood is very high, unless it is thus in an exceptionally good condition for holding urates in solution, they will not remain in the blood stream more than a moment or two, but will be caught up and temporarily deposited in the liver, spleen, and other organs, in which they may generally be found to be present in considerable quantity.

Further, uric acid itself interferes with its own solubility in the blood, for it, just like nitrous or any other acid and many salts of the mineral acids, has the power of converting the normal phosphates of the blood, which are good solvents of uric acid, into acid phosphates, which are not solvents of uric acid at all.[2] Hence, if you give 3 grains of uric acid three times a day and watch the urine for the day, you will find that you have probably not in the least increased the excretion of uric acid and you may have diminished it, for the uric acid you introduced not only did not remain in the blood but it prevented any other uric acid from remaining there. (Fig. 1.) But if, before you gave the uric acid, you had

[1] *Uric Acid as a Factor in the Causation of Disease*, Churchill, London, 2nd Edition, 1894.

[2] *Uric Acid*, p. 31 and elsewhere. Hence the acid phosphates are tonics and stimulants, and have been used as such in hay fever: see *Medical News* February 24th, 1894.

taken care, by the administration of alkalies or otherwise, to get the blood into a favourable condition for holding urates in solution, then you would find that your administration did increase the excretion of uric acid in the urine and that you had produced all the physiological and pathological signs, which I have elsewhere fully described, of excess of uric acid in the blood.

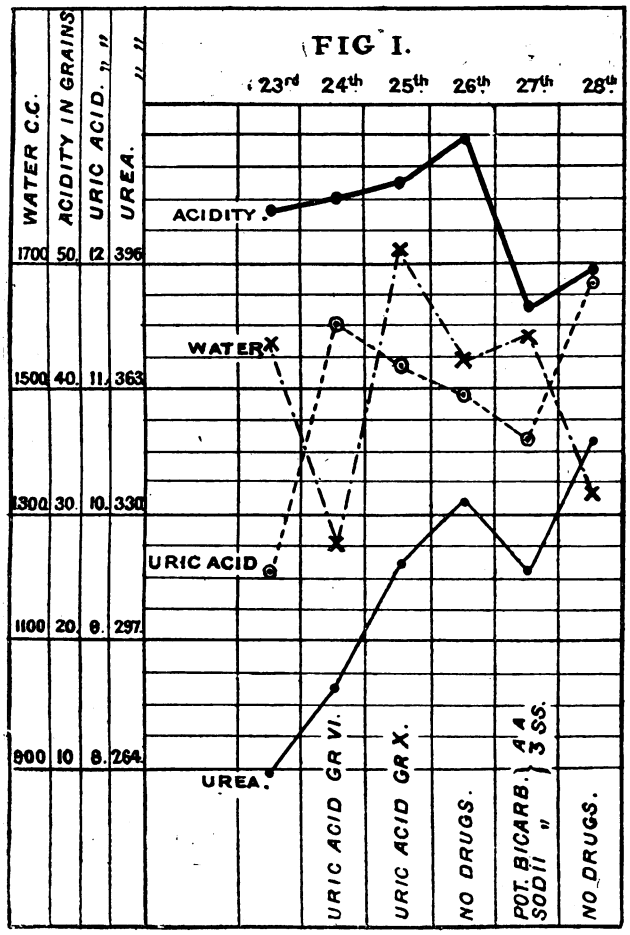


Fig. 1.—Effects of swallowing uric acid. It causes a rise of urea and acidity, and a rise of uric acid, especially on the 24th. But the rise of uric acid would have been much greater if the rise of acidity had not interfered with its excretion.

N.B.—In those on ordinary diet whose urea and acidity would be higher than mine the consequent interference with solubility and excretion would be greater, and the uric acid might not be in excess in the blood and urine for two or three days after it was swallowed.

Similarly those experimenters who have injected uric acid into the blood have paid no attention to its solubility, and have concluded, because they produced no symptoms, that uric acid in solution in the blood is not toxic. The truth is, I believe, that the uric acid they introduced produced no symptoms because it remained in the blood only a few seconds; and if they had bled the animals to death, as I have done, a few minutes after the injection they would have found almost no uric acid in the blood.

On the other hand, by observing the laws which govern its solubility, any of those who care to do it can bring as much uric acid as they like into the blood (Figs. II. and IV.), and they will then find that they have produced most of the signs and symptoms which I have described, such as high arterial tension, headache, mental depression, with scanty secretion of water from the lungs, kidneys, and other glands, and that these will be sufficiently severe to leave on their minds no doubt as to the toxicity of uric acid when in solution with alkalies in the blood. I would remind you also in this connection that uric acid may be present in the blood in combination with salicylic acid, or the normal phosphates in large quantity, as shown by a large excretion in the urine, without producing headache, mental depression, or high tension pulse to any marked extent. So far as I know, it is only the combination of uric acid with alkalies that produces the symptoms I have been led to ascribe to uric acid.

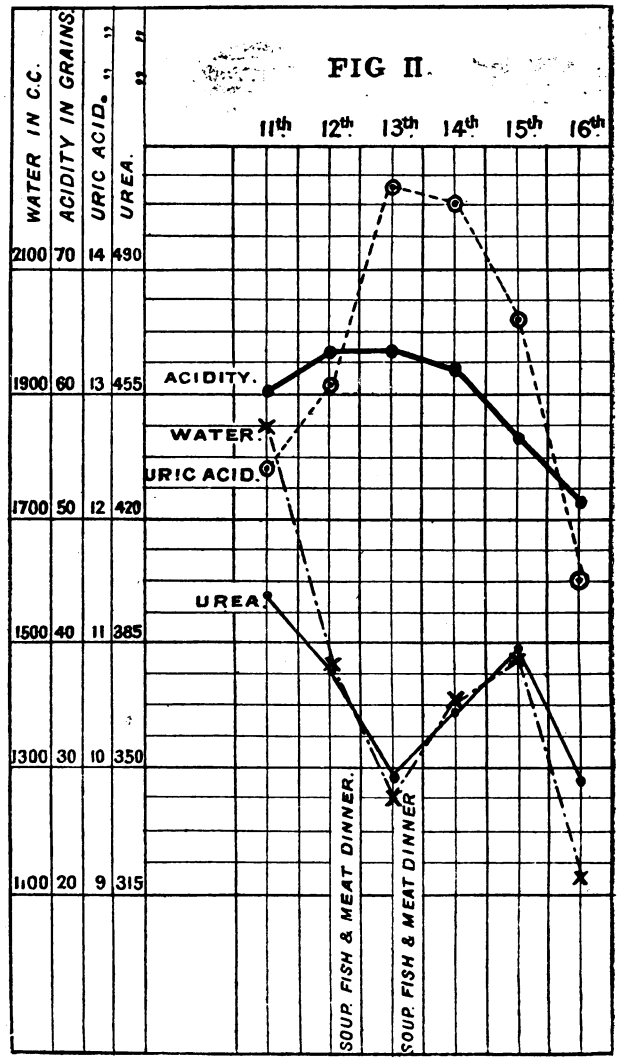


Fig. 2.—Effects of taking soup, fish, and meat. It causes a slight rise of acidity and a rise of uric acid, coming down again on the fourth and fifth day.

This is a point which I am still investigating, and not without hopes that I may some day be able to explain it; but I mention it here merely to prevent your attaching importance to injection experiments, which really prove nothing at all. It is easy to inject uric acid into the blood stream of an animal, but it is difficult, or impossible, to ensure its remaining there.

Not to go into details which can be found in my book, it may be said, speaking generally, that the solubility of uric acid in the blood is favoured by everything that increases the alkalinity of the blood, and hindered by everything that diminishes the alkalinity of the blood, and that uric acid accumulates in the body because it is insoluble, while the soluble urea is practically excreted just as it is formed.

Now, if you take any body of which you happen to be making a *post-mortem* examination and estimate the percentage of uric acid in samples of its various tissues, you will

probably be able to calculate that the body as a whole (though there are no visible urates anywhere) has contained several hundred grains of uric acid.[3] And I say, for reasons you will find in my book, that it contains all this uric acid because during the years of its life the excretion of uric acid has to this extent fallen short of the formation of uric acid *plus* the introduction of uric acid.

For it follows from my previous reasoning that all the uric acid contained in the foods ingested did find its way as uric acid into the body and blood, that is, was introduced.

Those who (as I have pointed out) urged that uric acid could not be introduced into the body in this way, reasoned in ignorance of the laws which govern the solubility of uric acid in the blood, and so reasoned wrong; and the rise of urea, which they thought to be due to the conversion of uric acid, has another and a simpler explanation.[4]

Suppose, then, that we find in such a body 500 grains of uric acid (of course those having visible deposits in their joints or urinary passages will have much more than this), the problem I wish specially to consider in this paper is what share in producing this accumulation has the direct introduction of uric acid had; or, in other words, how much less uric acid might this body have contained to-day if, during the last twenty or thirty years its owner had been careful never to introduce unnecessary uric acid?

I believe that at the present day a great deal of uric acid is often unnecessarily and thoughtlessly introduced into the body, while very slight modifications of diet rules might serve to exclude several grains a day, and this, taken over weeks, months, and years, might just make the difference between health and disease.

Let us see how much uric acid may be introduced in a single meal consisting of soup, fish, meat, with a little meat extract in some savoury dish.[5] Say a man has:

				Grain.
8 oz. soup	containing	0.020	per cent. uric acid	=0.70
2 oz. fish	,,	0.030	,, ,,	=0.26
3 oz. meat	,,	0.040	,, ,,	=0.52
½ drachm meat extract	,,	0.800	,, ,,	=0.24
				1.72

giving a total introduction of uric acid for this one meal of 1.7 gr. (See Fig. II.) Let us say that in his animal food for the rest of the day he introduces half this amount of uric acid in addition, and that in tea, coffee, or cocoa he may introduce 1 grain more, we get 1.7+0.85+1.0=3.55 gr. as his total daily introduction. And, when we further consider that hospital beef-tea may contain as much as 0.098 per cent., while veal, lamb, kidney, and liver all contain a considerably larger percentage of uric acid than that I have reckoned from above, it is easy to see that under many circumstances the daily introduction of uric acid may be much more than 3.5 gr. But of this introduction it is obvious that a considerable quantity is unnecessary, for, without materially reducing the animal food, we may cut out at once the soup, the meat extract, and the vegetable alkaloid—all these things being, like uric acid itself, stimulants, no doubt, but almost devoid of nourishment—and this at once reduces the daily introduction of uric acid to 1.6 gr., or less than half the original quantity.

Let us say that a man in this way introduces 3 gr. of uric acid a day; this will amount to upwards of 1,000 gr. in a year, and more than 20,000 gr., or about 3 lbs., of uric acid in twenty years, so that we thus get enough to account probably for all the uric acid that has ever been found in a human body. But uric acid thus introduced into the body may not only stay there, but at the time of its introduction it absolutely interferes—as shown above—with the excretion of the uric acid which has been previously introduced or formed in the body.

It is easy for anyone to satisfy himself on this point by taking a little uric acid, say two or three grains, three times a day for one day. He will find that it produces a stimulant effect with feelings of strength, well-being, and mental activity, and with this there is a diminished excretion of uric acid in the urine and a rise of urea.

[3] *Uric Acid*, 2nd Edition, pp. 46 and 377.
[4] *Journal of Physiology*, previous reference, p. 171.
[5] The percentages are taken from the figures given in my book, 2nd edition, p. 355.

Now the experimenters who gave uric acid appear to have considered that this rise of urea was due to the conversion of uric acid into urea, and that the uric acid which did not appear in the urine was accounted for by the urea which did appear; but if they had watched a few days longer they would have seen that the uric acid they gave did appear in the urine, and they could also have found out that a similar rise of urea is caused by every drug that clears the blood of uric acid. Thus acids which introduce no nitrogen into the body (as citric or sulphuric) will, if they clear the blood of uric acid stimulate circulation and metabolism throughout the body and produce a precisely similar rise of urea.[6]

Now, as I have elsewhere pointed out, everything that diminishes the excretion of uric acid and clears the blood of it produces the same stimulating effect and the same feelings, so that mercury, iron, zinc, copper, silver, acids and acid salts, as the sulphates, all produce these effects.

With regard to the sulphates, their use may, as I have shown[7] produce an attack of gout, and I have elsewhere pointed out that Lord Byron is reported to have recorded their effects on the spirits in the following words: "Talk of champagne, there's nothing cheers your spirits like a dose of Epsom salts." A remark I have myself often verified, and it runs parallel with the similar effects of acid phosphates above referred to.

Therefore the first action of uric acid is that of a stimulant, and this is due to the fact that it clears the blood of uric acid and diminishes the excretion in the urine. I may remark in passing, as I have already done elsewhere, that this is the probable explanation of the stimulant effect of soup, beef-tea, and meat extracts, as well as of the chief effects of thyroid extract, which has as its first effect a diminished excretion of uric acid going with general stimulation of function and nutrition (Fig. III). Though no doubt in all these cases the effect of the uric acid contained is increased by the similar effects of the phosphates, sulphates, and other salts, which act in the way I have pointed out, and of other nitrogenous compounds which act like uric acid in diminishing the alkalinity of the blood, and increasing the acidity of the urine; but, as I have said, anyone can produce similar effects on himself by taking uric acid alone.

Later on, when the uric acid thus introduced comes to be present in excess in the blood and urine, general depression of mind and metabolism take the place of the previous stimulation, and the picture is reversed; but this does not concern us just now, though it accounts, I think, for the somewhat serious troubles that thyroid treatment may produce later on. We see then that uric acid thus introduced day after day may not only remain and accumulate in the body, but will also block the excretion of other uric acid previously introduced or formed in the body; and we may thus, I believe, answer the question which we asked above, somewhat as follows:

If a man had by care in diet during twenty years or more of his life introduced only a few hundred grains of uric acid in place of 3 lbs. of it, he would not only have had less uric acid from which to accumulate a store, but he would have been able to excrete more freely and completely that uric acid which he himself formed.

I will only say therefore, in conclusion, that it is possible to diminish very greatly the daily introduction of uric acid into the body without in any way diminishing the income of those substances on which the nutrition of the body depends. (See Fig. IV.)

There is almost no nourishment in soup, beef-tea, meat extracts, and vegetable alkaloids; and I venture to say that the nutrition of the body will not suffer in the least if they be entirely omitted

No doubt uric acid in these and other forms is, at the time it is taken, a stimulant; but this stimulation may be obtained from other substances, and is I think dearly purchased at the cost of large accumulations of uric acid in the body. Thus acids, such as citric and others, contained in many fruits, and products prepared from them, will produce a similar stimulation of nutrition and metabolism, and though they no doubt temporarily interfere with the excretion of

[6] *Journal of Physiology and Uric Acid.* Second Edition.
[7] *St. Bartholomew's Hospital Reports*, vol. 24, p. 217, and *Medico-Chirurgical Transactions*, vol. 72, p. 402.

uric acid, they do nothing to increase the quantity introduced.

The craving for fruit acids in warm weather is, I think, perfectly natural and physiological, as something is required to replace the acids lost in perspiration; and it is far better to indulge it to a moderate extent than to supply what is required from effete nitrogen in the form of uric acid and its

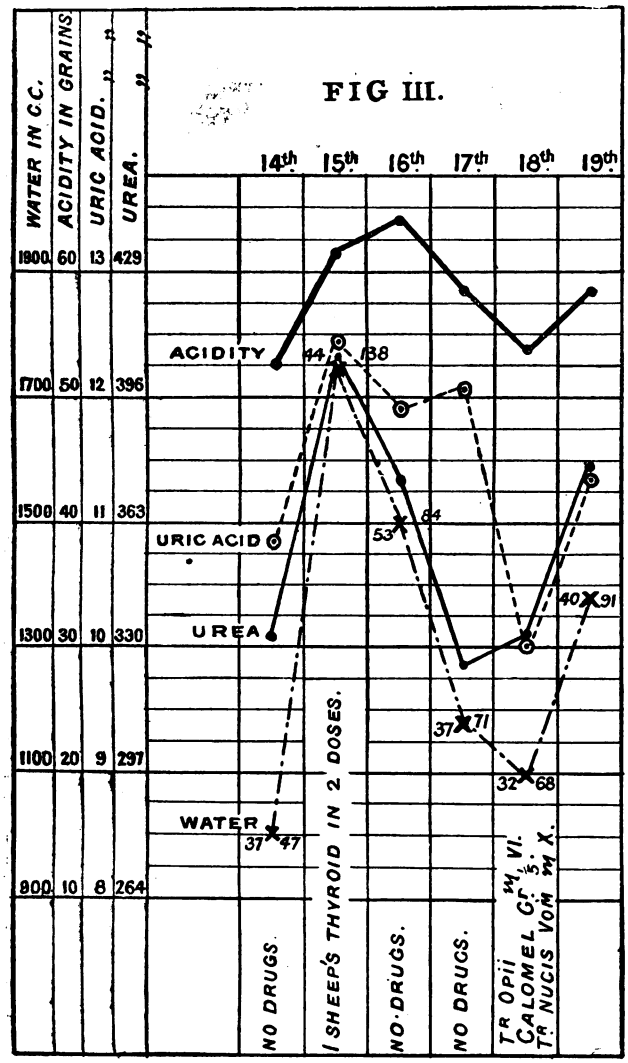


Fig. 3.—Effects of swallowing a sheep's thyroid. It causes a rise of urea and acidity on the 15th, followed by a *plus* excretion of uric acid on 16th and 17th. Uric acid was relatively less on 15th than on 14th.

N.B.—*Plus* excretion of uric acid is shown by its height above urea.

congeners of the xanthin group, which may remain in the body, and cause terrible damage in years to come.

Precisely the same argument applies to the use of caffeine for the cure of a uric acid headache; no doubt it may cure the headache, so would a few grains of uric acid itself; but they cure it by increasing the store of uric acid in the body, and this, if persisted in, will bring disaster at no distant date.

It follows from this that whatever other diet treatment may be necessary for the prevention or cure of uric acid disease, it is of the utmost importance never to introduce into the body a single grain of uric acid that can be left outside it; for that grain of uric acid, if introduced, may not only remain and help to form a store in the body, but it may also prevent the excretion of some of the uric acid which is already in the body.

Lastly, I would again call your attention to the fact that, bearing in mind the conditions which affect its solubility in the blood stream, the physiological and pathological effects of uric acid can be demonstrated with ease and certainty by its direct administration; and indeed this is a corollary of what I have just said about diet and caffeine, and have elsewhere demonstrated as to their effects.[8] So that the direct

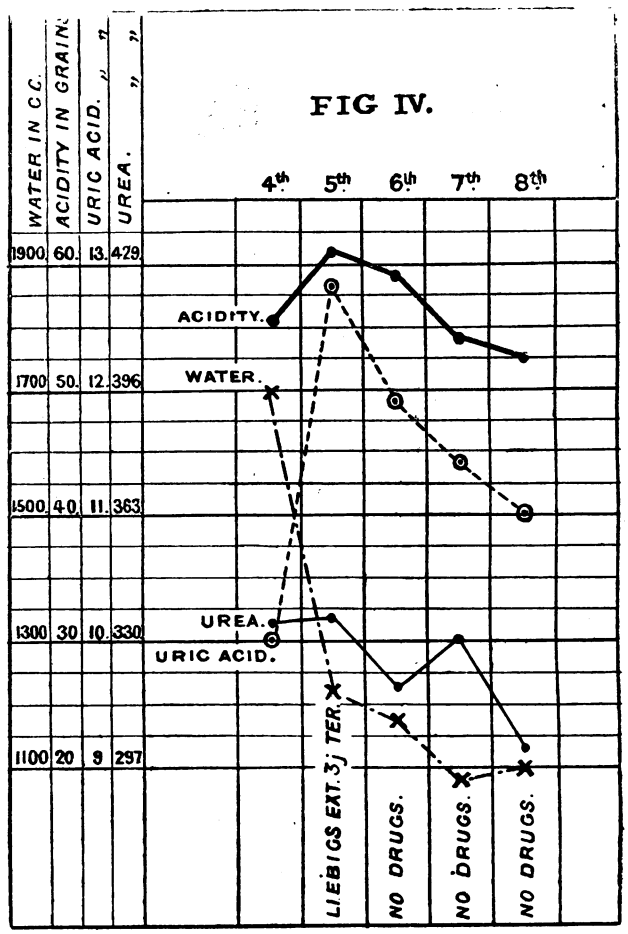


Fig. 4.—Effects of swallowing Liebig's extract of meat. It causes a slight rise of urea and acidity, and a marked rise of uric acid. Note the relation of uric acid and water in illustration of the law that they vary inversely.

introduction of uric acid into the body not only plays an important *rôle* in the causation of disease, but can be employed at any time to demonstrate the pathological effects of an excess of uric acid in the body and blood.

PRACTICAL HINTS FOR INVALIDS ON THE MAINTENANCE OF HEALTH IN THE CLIMATE OF EGYPT.

WITH SOME SUGGESTIONS TO MEDICAL MEN AS TO THEIR SELECTION OF CASES SUITABLE FOR RESIDENCE THERE.

By ARTHUR J. M. BENTLEY, M.D.,
Cairo.

IN offering the few remarks which I am about to make, I must apologise for any shortcomings on the score of originality. Having spent, however, three winters in Egypt in the neighbourhood of Cairo, at a health resort situate on the edge of the desert at the foot of the Great Pyramid of Gizeh, much frequented by invalids, I have thought that my experi-

[8] BRITISH MEDICAL JOURNAL, ii, 1893, p. 673; July 21st, 1894, p. 113.

ences might prove of service to such members of my profession as may contemplate sending patients there.

"The good effects of a complete climatic change upon certain forms of disease have long been an established fact." But in order to obtain the full benefit from a residence abroad not only must cases suitable for each particular climate be selected, but proper directions for the maintenance of health under the changed circumstances of life must be given.

The proper choice of a climate is one of vital importance to the invalid; and nothing can be more opposite in climate, as regards phthisis for example, than the air of a crowded town like Cairo, and the pure desert air of Mena or Helouan, or the almost absolutely dry air of Luxor or Assouan; or "between the Delta, which is under the influence of numerous surfaces of water and cultivated ground, and Upper Egypt, which is under the influence of the two deserts."

Every climate has its disadvantages as well as its advantages. The climate of Egypt in the winter months may be summed up as dry and tonic; its advantages consist in a moderately uniform warm diurnal temperature, in an almost complete absence of rain, and in a bright sunshine mostly all through the winter months, allowing every facility for an outdoor open life. To such as possess sufficient energy and are inclined to take advantage of the invigorating exercises of riding and driving, especially the former, ample opportunities are afforded, whilst to those of a more robust nature excellent sport will be found available all through the winter. There is also complete absence at the different health resorts of fog and of excessive cold, while outside the large towns there is an absolutely pure desert air. The hotels are luxurious and comfortable, and the food supply good. Vegetables and fish are abundant, and fruit unrivalled.

There is besides a complete absence of typhoid at all the health resorts excepting Cairo itself, and there there were only three or four cases amongst the English hotel residents during the whole of last winter, any other which may have developed in visitors after they had left Egypt being probably acquired in Alexandria, where the water should not be drunk unless under the advice of residents.

Malaria is also unknown at Mena, Luxor, or Assouan, so that the windows of the sleeping apartments, if facing south, can safely be left open, thus ensuring complete ventilation, except in some chest cases where chill of any kind may be injurious.

The drawbacks to a winter sojourn in Egypt, and in fact anywhere out of England, are:

1. The injurious habit Egypt shares unfortunately with the Riviera, that invalids do not consult the doctor until they are attacked by serious illness, which they mostly might have escaped from if they had been guided from the beginning by a judicious physician.

2. The ignorance of most invalids as to the nature of the climate to which they resort, and apparently the want of a full appreciation of the real object for which they go abroad, that is the restoration of their health.

3. The opportunities which exist for sight-seeing and social gatherings, with their attendant evils, over-fatigue, overcrowded rooms, and late hours.

4. Its occasional cold high winds and at times unreliable weather.

5. The sudden and sometimes marked fluctuations of temperature between day and night, sunshine and shade.

6. Its hot winds, which commence in February and blow for about two days at a time, accompanied by fine particles of sand suspended in the air (Khamseen).

It is to the end of lessening or obviating altogether some of these disadvantages that my paper is chiefly directed.

It is a mistake for invalids to take a long railway journey without a break somewhere *en route*. Travelling by train is in itself a rapid change of climate, and is to most people exciting, and if any inflammatory mischief is present it will probably be aggravated. Before starting strict attention should be given to the state of the bowels, and during the journey as careful a dietary as possible should be taken both as to quantity and quality, and rest for a while should be ensured if inflammatory symptoms arise *en route*. Much injury is sometimes done through want of attention to these details.

The risks and discomforts to which all newcomers are exposed from a want of knowledge of the vicissitudes of the climate and of the comparatively cold winds that blow more or less in December and January are at times trying but easily avoidable. The diurnal temperature of Egypt is very uniform, but the sudden fall at sunset of several degrees, reaching its climax at about 4 A.M., is felt by invalids, whose sensitiveness to cold is at all times marked, if they are foolish enough to expose themselves to it. All invalids, therefore, should go indoors an hour before sunset, and should not venture out for two hours after sunrise.

Again, nothing is more noticeable than the great difference there is between sunshine and shade. This disadvantage is, however, easily avoided by invalids wearing as warm clothing in Egypt as they do in the autumn or winter at home, the air being so dry that any oppressiveness from woollen clothing is not felt there; also by their carrying always a light coat or wrap, and a silk handkerchief for the neck to be put on when going from sunshine into the shade, while in the house after a walk they should not remove their overcoats at once. Cummerbunds, or a roll of fine flannel round the abdomen, should be worn by all as a preventive of diarrhœa arising from chills, and a heavy or fur-lined coat should always be used when driving; all rooms facing the north should be provided with fireplaces, and a fire kept burning in December and January, when the weather is cold or unsettled. Want of care in avoiding chill may lead to diarrhœa, dysentry, or pneumonia, or to a fresh access of inflammation in a quiescent chronic condition of lung disease. Care is also necessary at times in women to avoid the possible chance of a chill, a case having come to my knowledge last winter where severe local peritonitis followed exposure to cold in this way after a ride.

The direct rays of the sun, if not tempered by the use of an umbrella, or suitable head protection, or smoked spectacles, more especially in March and April, are often injurious, leading to congestions, headaches, and feverishness. I have so often seen invalids sit or lie in the direct rays of the sun while, at the same time, a cold wind was blowing over them, that I am induced to lay stress on the folly and possible danger of this.

Now it is especially to these conditions that the invalid is exposed on going up and coming down the Nile, the days being warm, the wind keen, while the cold after sunset is intense. The warmest clothing and wraps are therefore necessary on the voyage. Draughts which are unavoidable should, as far as possible, be guarded against, even, if necessary, by the invalid remaining for most of the time in the saloon or in his cabin, and on no account should he go on deck after sunset; over-fatigue in any form and long donkey-rides to tombs and other distant places of interest should be scrupulously declined by the consumptive, labouring more or less, as he does, under physical debility. Such persons should not attempt to take part in the amusements and occupations of the strong, but cheerfully acquiesce in their enforced idleness and inaction, for by doing so they very materially advance their chances of ultimate recovery.

The tour made by the regular tourist boat of Messrs. T. T. Cook and Son, each of which carries an experienced physician, and is a floating hotel of comfort and luxury, is well suited to the robust pleasure seeker, but is not the best way for the invalid to reach Luxor. The post boat, being quicker and more direct, is for him by far the best, if he is unable to afford the expense of a dahabyeh on the Nile for the winter.

The hot wind of Khamseen commences in February, and blows for a day or two at a time, but only very occasionally till the middle of April. It has a depressing effect on some, while it is agreeable and invigorating to others. During its continuance the air is devoid of all moisture, and is more or less charged with electricity, while it is, at the same time, full of minute particles and in a state of suspension, causing irritation of the eyes and throat; it has, however, more an irritating effect on the temper than an injurious one on the health. It is less disagreeable to many than the colder winds of December and January, and in cases of phthisis with cavities it has a positive drying up and healing influence. Its goods effects, also, in rheumatism and some cases of asthma is marked. Its discomforts are entirely obviated by the

invalid remaining indoors till it has blown itself out, which it generally does in about two days.

As a rule invalids leave Egypt too soon in the spring and undo in many cases the good effects of their winter sojourn by encountering the cold variable weather of the spring in Europe. Let me warn them against this and in place of hurrying off as soon as the first Khamseen blows to remain quietly in Egypt till the end of April, and if they find the neighbourhood of Cairo too hot and enervating there is Ramleh, on the sea coast near Alexandria, to go to, where excellent hotel accommodation can now be obtained and where they will run no risk of encountering the chills of an early spring.

The neighbourhood of Alexandria, owing to the nearness of the sea, has a more uniform temperature and has advantages in autumn, spring, and summer. (Hermann Weber).

Every facility exists in Cairo, Mena, and Luxor for obtaining every medical comfort and good nursing. On this score there is little left to be desired. Invalids, however, who require constant attention are strongly recommended to bring with them their own nurse or valet.

With these few imperfect observations, which apply more or less to all southern health resorts, and to the robust as well as to the invalid, I conclude with the sincere hope that if acted on they may verify by their results the old saying that "prevention is better than cure."

THE NATURAL MINERAL WATERS OF BUXTON: THEIR INDICATIONS AND MODES OF APPLICATION.

By Samuel Hyde, M.D.,
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The natural medicinal waters, to which Buxton mainly owes its reputation as a health resort, rise by several springs or "sources," but the principal springs are situated near the Crescent, at the western end of that building. It is from the edge of the limestone, which meets the millstone grit at the lower level of the town, that the Crescent springs rise through various fissures to the surface; and there can be no doubt, from the existence of several similar springs and other evidence, that the larger "sources" found in the Crescent are derived from the limestone formation upon which the higher portion of Buxton stands. The temperature of the water as it issues from the earth at the Crescent source is 82° F. The water has a slight bluish tinge; it is perfectly clear, tasteless, and remarkably buoyant. The chief peculiarity of the water is the considerable quantity of free nitrogen gas with which it is charged, the amount being greater than that in any other known mineral water. The waters of Gastein and Wildbad resemble the Buxton water in this respect, but in a less degree.

Bearing in mind the relatively small proportions of the several salts contained in the Buxton water, it is evident that, whatever the effects may be which are produced by them, they must be traced not so much to the individual action of each, but rather to their united action upon the organism. As to the precise part played by the nitrogen gas contained in the Buxton water, much has been written and a great deal has been said which is merely of a conjectural character and has no really scientific basis. It is a question which has up to the present time received no satisfactory answer. Nor am I prepared on this occasion to propound any new hypothesis where so many have already failed. But, having made this confession, it must not be supposed that such waters as those now under review contain nothing of a special character which, if properly understood, would account for the special influences they are alleged to exert over many diseased conditions.

No one who, like myself, has observed during many years the truly remarkable curative effects constantly produced before our eyes—effects which it would be an affectation to ascribe to mere change of air and surroundings—can for one moment doubt their reality. In saying this, I have referred chiefly to my own personal experience; but if additional testimony be desired as to the curative effects of these waters, it will be found in the records of the Devonshire Hospital at Buxton, which accommodates over 300 in-patients, and yearly confers incalculable benefits upon numerous poor sufferers from rheumatic and other painful affections. In this institution, out of 2,559 persons treated as in-patients during the past year, no fewer than 2,398 were discharged as "improved."

Indications for the Use of the Buxton Waters.

The use of these waters is indicated in the following diseases:

Rheumatism.—Acute rheumatism, subacute rheumatism, chronic articular rheumatism (rheumatoid arthritis of Garrod or the rheumatic gout of Fuller), muscular rheumatism, arthritis deformans, or chronic progressive articular rheumatism, gonorrhœal rheumatism.

Gout.—In acute and chronic gout and the many protean forms of that disease the Buxton waters are efficacious. In ordinary lithæmia and latent gout the waters promote the elimination of the lithic acid from the system.

Neuralgic Affections.—Sciatica, lumbago, and intercostal and brachial neuralgia are greatly benefited by a course of the waters.

Paralysis and Spinal Disease.—In local forms of paralysis, especially from metallic poisoning, as, for instance, lead, and in writer's cramp; in facial paralysis and diphtheritic paralysis; and in hemiplegia when of not too long standing. I have also seen some remarkably good results in locomotor ataxy.

Anæmia, Malaria, etc.—Especially where these diseases are complicated with rheumatic or gouty symptoms the Buxton waters are of great use. This is often seen in the case of officers and others who have resided in malarial districts abroad, and who, on returning to this country, contract rheumatism. The characteristics of this form of rheumatism are very striking, the malarial cachexia modifying the course and symptoms of the disease in an unmistakable manner.

Diseases of the Skin.—In psoriasis and eczema, particularly when of gouty or rheumatic origin, the waters afford very satisfactory results.

Diseases of Women.—Irregular and painful menstruation, leucorrhœa, chronic ovaritis, neuralgia of the ovaries, ulcerations, catarrh, and sterility frequently derive much benefit from the Buxton bath.

Throat Affections.—In chronic laryngitis, pharyngitis, and clergyman's sore throat the waters sometimes do much good.

Surgical Diseases.—In many surgical affections the Buxton thermal waters are of great service. Conditions such as those following fractures and dislocations, sprains of joints and such like injuries, together with surgical diseases of a constitutional character, as, for instance, hip-joint disease, caries and necrosis of bone, white swellings, chronic synovitis, tumours, etc., are benefited by these waters.

Modes of Application of the Buxton Waters.

These are divided into internal, external, and the combined methods of using the waters.

The internal use consists in drinking from 6 to 10 ounces of the water two or three times a-day between meals, followed by gentle exercise for about half an hour. As a rule, no unpleasant sensations are experienced, but sometimes the drinking of the water is followed by slight giddiness, probably due to an over-stimulating effect on the cerebral circulation, and when this occurs it is an indication to reduce the quantity imbibed or to stop the water altogether for a few days. In some cases I find the addition of milk to the water renders it tolerable where otherwise it could not be taken. The water should not be drunk within a short time before taking a bath, but may be taken immediately after bathing. The chief observable action of the water is upon the kidneys and the skin, especially the former, free diuresis and frequent micturition being common effects during the drinking of the waters.

The external use of the waters is carried out either at the natural temperature of 82° F., or at artificially raised temperatures of from 86° to 100° F. The natural temperature being so low, the water finds its most useful and widest application in the hotter kinds of baths. The forms of baths in vogue at Buxton include swimming and plunge baths, lounge

or reclining baths, douche, needle, spray, shower, wave, and sitz baths, massage douche baths.

The immersion baths are given in the form of "full," "three-quarter," and "half" baths according to the exigencies of the case. Many patients can take partial baths who could not bear a full bath. In such cases the body is immersed to the specified height, and the un-immersed portions are laved with the water. The time usually occupied in the hotter baths varies from 4 to 15 minutes. About 8 is the average time. For the natural bath the time ranges from 2 to 10 minutes.

The best time for taking the baths is half an hour or an hour before breakfast. If the patient is too weak for this, the next best time is about an hour before the midday meal. Contrary to the usual practice of taking a brisk walk after an ordinary bath, the patient is advised not to walk much soon after the hotter forms of the Buxton bath, unless he has a difficulty in getting up a reaction, which is very rarely the case; but to return quietly to his lodgings, and there to rest for an hour or so, either in an easy chair or reclining on a couch. After this he is at liberty to take outdoor exercise.

It is a good plan to take one or two doses of aperient medicine (a saline is the best) before beginning to use the waters. This practice in most cases requires repeating several times during the visit, as more or less constipation is apt to follow the change to the tonic and bracing air of Buxton. The number of baths and the duration of the course will vary according to each case. As a rule not more than four or five baths should be taken in one week, and the course usually occupies from three to six weeks.

Many cases present themselves for treatment at this spa in which the external use of the waters is contraindicated, but who derive much benefit from the internal use of the waters alone. It may, however, be stated that as a rule the combined system of drinking the waters and the application of baths is carried out in the vast majority of cases. Generally speaking, whether the water be drunk or used in the form of a bath there are no unpleasant subjective sensations experienced by the patient. In some cases, however, disagreeable sensations are experienced by bathers, but when such is the case it may be assumed almost with certainty that there is some more or less serious organic disease connected with either the heart or large blood vessels, the lungs, or the brain. To a person in an ordinarily sound state of health the bath is perfectly safe, and there is no ground for that fear which has been excited by some writers. Having said this much, however, it is necessary to warn invalids against the pernicious practice of drinking these waters and taking the baths without proper medical advice. By such a course I have seen very frequently, not only direct injury result, but much valuable time and money wasted by the patient either taking the wrong kind of bath for his case or taking too many or too few of the right ones. It should also be mentioned that at the Buxton spa free use is made of the numerous valuable adjuncts to modern spa practice. Amongst the adjuncts to internal treatment strict and regular diet occupies an important position. Drugs are also often brought into requisition, especially simple purgatives, cholagogues, and hepatic stimulants. Dry massage, shampooing, electricity, active and passive mechanical exercises, counter-irritation, dry and wet packing, hot air and vapour baths, steam douching, are all used more or less frequently.

Before concluding it is necessary to say a few words as to the diseases and conditions in which the use of the Buxton waters and baths are contraindicated:

Cardiac affections, including valvular disease, hypertrophy, dilatation, fatty degeneration, and angina pectoris.

Diseases of blood vessels, such as atheroma, aneurysm, phlebitis, and varicose veins.

Renal diseases, acute and chronic, excepting such as are associated with the gouty diathesis.

Bladder and urethral affections in the form of cystitis, catarrh, prostatitis, and calculi.

Pulmonary diseases, including phthisis, asthma, emphysema, and acute bronchitis.

Gastric troubles, including acute dyspepsia, catarrh, ulceration, hæmatemesis, and diarrhœa.

Cerebral congestion, hyperæmia, tumours, tuberculous meningitis, apoplexy, and epilepsy.

Cancer and malignant disease.

These and many other diseases which the above enumeration will suggest are inappropriate and often positively contraindicate the use of the Buxton mineral waters either internally or externally, although in many instances much benefit accrues from a residence in the Buxton climate. To these may also be added certain general conditions of the patient, such as extreme emaciation, muscular atrophy, and nervous exhaustion.

NOTES ON DIABETES TREATED WITH EXTRACT AND BY GRAFTS OF SHEEP'S PANCREAS.

By P. WATSON WILLIAMS, M.D.Lond.,
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No disease has received a larger share of attention at the hands of clinicians, pathologists, and chemists in the last fifty years than has diabetes, while the ample literature and the very diverse opinions that have been advanced by the most able observers in regard to its pathology and treatment testify to the difficulties which surround us when we seek to explain the problems involved. Certain facts recently come to our knowledge seem at first sight to justify very important conclusions as to the real nature of diabetes, and to indicate fresh lines of treatment as at least affording some chance of increased control over this disease.

It would serve no useful purpose were I to attempt to review and to reconcile the results of recent investigations in the region of difficulty; they are well known to all physicians who are acquainted with current medical literature, and have proved a source of perplexity to many who are better able than I am to speak on the subject, but as the question of the treatment of pancreatic diabetes by means of the pancreas or extracts is a matter of special interest, and more especially as the possibility of treating pancreatic diabetes by means of grafts of pancreas is likely to receive further attention in the immediate future, I think it is my duty to place on record the results in a case unsuccessfully treated by myself, and to indicate what my unfortunate experience may teach us to avoid.

Before relating my own brief experiences I would call to mind the two interesting cases of diabetes resulting from obstruction in the pancreatic duct, reported by Freylan[1] which support the conclusions of Minkowski based on his recent investigations in the diabetes resulting from destruction of the pancreas in dogs. In Freylan's first case, that of a man, aged 35, who was passing 70 ounces of urine containing 3 per cent. of sugar, death was immediately due to tuberculous lung disease. The pancreas was atrophied and no trace of parenchyma was observable in microscopic sections. The pancreatic duct was completely occluded by concretions of carbonate of lime. His second case was that of a woman, passing 70 ounces of urine containing 8 per cent. of glucose. Death resulted from kidney disease, but the pancreatic duct was found blocked with calcareous detritus and a definite calculus was found in the gland itself, which only contained a few isolated nodules, the remainder being atrophied.

On the other hand we cannot overlook the result of Williamson's investigations of the pancreas in 14 cases of diabetes, which showed that in 50 per cent. the pancreas was normal in structure, rendering it evident that we must guard against attributing a too important position to the pancreas as a factor in diabetes mellitus.

CASE I.—C. T., aged 15, was admitted to the Bristol Royal Infirmary on July 31st, 1893. He was in good health up to three weeks before admission, when he began to complain of thirst, and was noticed by his parents to be wasting rapidly and getting extremely weak. Patellar tendon reflexes absent; no fatty stools. On admission he was weak, rapidly wasting; weight 5 st. 9 lb. 4 oz.; skin dry; average daily amount of urine 77 oz., containing 3,212 grains of sugar and 2¾ per cent. of urea. Daily amount of fluid drunk 7 pints. The case was so grave that I did not consider I was justified in waiting more than two days on a strict diet, modified by allowing brown bread, before commencing active treatment, for the amount of sugar had risen to 9 per cent. in 78 oz. On August 3rd, therefore, he had an absolutely restricted diet with gluten bread. From this day he took freshly minced pancreas for seven days, then 1 drachm freshly prepared liquid extract of pancreas three times daily with his meals, and had a drachm every time he took any food at all in the intervals, so that he never had any food without pancreatic extract. This was continued for four days more, the urine, the amount

[1] *Berl. klin. Woch.*, No. 6, 1893.

of sugar and polydipsia all steadily diminishing, till on August 11th he passed only 56 oz., with 4.16 per cent. of glucose. Then for four days he had 3 drachms of the extract hypodermically daily (except on the first of these four days, when he had 2 drachms), with practically no alteration in the symptoms. Then the pancreatic extract was again resumed by the mouth as before. By this time the elimination of sugar amounted to about 1,188 grains daily—that is, was reduced to nearly one-third of what it had been; the urine passed was about 50 oz. daily, and the polydipsia correspondingly improved. The diet and administration of pancreatic extract remaining unaltered, he was now ordered 1½ grain of codeine daily. In seven days the sugar averaged 456 grains and urine 50 oz. daily. The pancreatic extract was now left off, August 27th, as I believed that the reduction of sugar in the first instance was due to the strict dietary, and in the second to codeine. No other alteration was made, but within two days the amount of sugar nearly trebled, in four days it had risen from an average of 1.9 per cent. to 7.1. But after simply resuming the pancreatic extract it remained about the same, and only fell slightly on changing the gluten bread for Clarke's starchless biscuits. In the meanwhile he gained 5 lbs., but had again lost 4 lbs. in weight.

On September 18th the codeine was increased to 2⅔ grs. daily but without benefit. Without further alteration the freshly-prepared glycerine pancreatic extract by the mouth was discontinued on September 20th, and 1 drachm daily of pancreatic extract prepared in Paris by Brown-Séquard and d'Arsonval's process was given hypodermically for eleven days. The amount of sugar eliminated rather increased, and here the treatment by pancreatic extract was left off. The boy was, in spite of a very fair trial of pancreatic extract both by the mouth and under the skin, combined with strictest regimen and both without codeine and subsequently with 2⅔ grs. daily, practically in the same position as on admission. He was passing 8.2 per cent. of sugar in 66 ozs. of urine daily.

On October 6th the codeine was increased to 4 grains a day. In six days' time he began to be a little sleepy in the daytime although getting up every day. There was a slight diminution in the amount of sugar eliminated (6.2 per cent. in about 60 ozs. of urine).

On October 14th I obtained a supply of freshly-prepared orchitic fluid obtained from a young bull by d'Arsonval's process by my friend, Dr. Pagan Lowe, of Bath, and without any other alteration in the conditions, the patient had 1 drachm daily injected hypodermically, and as he felt so much brighter and better this was continued, and although the amount of sugar passed was at first increased, it again decreased to about 5.5 per cent. in 55 ozs.

October 26th. The codeine discontinued, but the amount of sugar so greatly increased that in twelve days I felt it advisable to give morphine (¼ grain daily at first, subsequently less).

At first the effect of the morphine was very satisfactory, but about December 17th it appeared to be losing its power of controlling the diabetes, and once more the glucose rose to 8.3 per cent. in 80 ozs. of urine. The advisability of resorting to grafts of sheep's pancreas had occurred to me, and my medical colleagues, who had kindly seen this case with me frequently during the time the patient was under my care, agreed with me that the case was one which offered some chance of benefit from such a procedure, seeing that laboratory investigations of Minkowski and others appeared to lend some hope that the patient might be favourably modified.

I had previously visited a neighbouring slaughterhouse in order to familiarise myself with the best method of rapidly extracting the pancreas from a sheep. By suspending the carcass by the hinder extremities one had only to open the abdomen and allow the intestines and stomach to fall down, and the pancreas was exposed and readily removed.

On December 20th my surgical colleague, Mr. Harsant, placed the patient under chloroform, while I extracted the pancreas with strict aseptic precautions from a freshly-slaughtered sheep, so that by the time the patient was anæsthetised the pancreas was at hand, and three pieces each of the size of a Brazil nut had been grafted into the subcutaneous tissues of the breast and abdomen, and the operation completed within twenty minutes of the death of the sheep. The patient was under the anæsthetic a quarter of an hour. The wounds were dressed antiseptically. He passed 116 ounces of urine containing 7.4 per cent. of sugar in the next twenty-four hours.

On December 22nd 95 ounces of urine containing 3.12 per cent. of sugar. The incisions appeared perfectly healthy, and Mr. Harsant considered that they were healing by first intention. But the patient felt very depressed and weaker and drowsy, and there was every indication of coma supervening. The urine yielded a more decided acetone reaction, although this had been noticed in a less degree for some weeks. With a view to prevent the onset of coma bicarbonate of potash was administered, and an aperient ordered.

On the 23rd he died comatose. Large doses of citrate of potash by the mouth, combined with 30 grains of bicarbonate of soda, given hypodermically in three doses had had no effect whatever. Oxygen inhalations and oxygen water had also been tried.

At the necropsy on the following day the heart and lungs were found to be normal, but the liver and kidneys were enlarged and fatty. The pancreas only weighed half an ounce in its capsule. It was small, shrivelled in appearance, and seemed to be little else than fibrous tissue. Examination of sections of the pancreas showed that almost all the secreting structure had disappeared, and that the gland consisted of little else but fibrous stroma. The grafts had apparently failed to become united with the tissues around, and though the incisions in the skin had apparently united, numerous micrococci were found on microscopical examination. But the necropsy was made sixteen hours after death from diabetic coma.

I think that the considerable number of cases of diabetes that have now been recorded as having been treated with pancreatic extract by the mouth have shown that practically nothing can be expected from such treatment. In the case of T. C., the method received a very fair trial, the freshly-prepared extract being given first every time the patient took any food whatever, and subsequently by cutaneous injection.

Of course, in some respects this was not a typical case of pancreatic diabetes, inasmuch as the amount of urine was not very excessive, and there were no fatty stools; but I think the history of the case and the *post-mortem* examination of the pancreas leave no doubt that it was rightly considered to be pancreatic diabetes, and one that presented all the conditions that might lead one to hope for a beneficial result from successful grafting of pancreas, if anything can be hoped for in this direction at all. Failure was possibly due to obtaining the graft from a sheep that had been killed by bleeding, and I fear may have been rendered more likely from the administration of the anæsthetic. If ever I felt justified again in resorting to pancreatic grafts in a similar case, I should obtain them from a living animal anæsthetised and dispense with the anæsthetic altogether.

CASE I.—T. C. Average amount of sugar passed in twenty-four hours: On admission, first two days, 3,212.1 grains.

August 11th, after four days of strict dieting and pancreatic extract by the mouth, 978.8 grains.

August 15th and 16th, after four days' strict dieting and pancreatic extract hypodermically, 983.8 grains.

August 18th and 19th, after the pancreatic extract was resumed by the mouth, other conditions the same, 1,188 grains.

August 26th, after seven days with pancreatic extract by the mouth, strict diet, and codeine (1½ gr. daily), 456 grains.

August 31st, after four days under same conditions, except that the pancreatic extract was discontinued, 1,872 grains.

September 31st, after the conditions of August 26th had been resumed for eighteen days, 1,733 grains.

CASE II.—W. H., male, aged 47, foreman at chemical works, began to suffer from thirst about February, 1892, and soon after noticed his loss of flesh and polyuria, and was told he had diabetes. In November, when I took him into the house, he had lost 2 stone in weight and felt excessively weak. He was then passing 65 ounces of urine containing about 1,245 grains of sugar daily. After two days' restricted diet (with brown bread allowed), and while taking small doses of phosphorus and arsenic the amount of urine was slightly increased, and he passed 1,800 grains of sugar per diem. Again, after taking the same diet, but taking 1 grain of codeine daily for four days the amount of urine continued to increase, averaging 88 ounces, with 1,920 grains of sugar. His general condition *in statu quo*. The same conditions as regards diet and treatment were persisted in for twenty-nine days, during which period the amount of sugar passed had fallen as low as 152 grains daily, the average amount of urine remaining stationary, but towards the conclusion of the twenty-nine days the urine rose to 90 ounces daily, with 1,742 grains of sugar. At this point, that is, January 12th, 1893, I gave him mxl of pancreatic extract (prepared by extracting fresh pancreas with glycerine) hypodermically. The following day a drachm of the extract by the mouth, then mxl hypodermically, and again, with a day's interval, a fluid drachm by the mouth. There was no amelioration in the quantity of sugar passed. The same treatment was continued for another four days, with 1½ grain of codeine (instead of 1 grain) daily, resulted in no improvement as regarded the elimination of sugar, but he felt better and stronger in himself. The pancreatic extract was discontinued as useless, but the patient, after many months of treatment by codeine and restricted diet, completely recovered and resumed his ordinary diet without any treatment at all, and the urine has been free from sugar for a year.

MODERN HYDROPATHY: ITS RELATION TO GENERAL THERAPEUTICS.

BY C. J. WHITBY, M.D.Cantab.,
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THE object of this paper, namely, to stimulate in some degree the growing interest of clinicians in the possibilities of hydrotherapy, will perhaps be best served by presenting as tersely as may be a few salient considerations, theoretical and practical, gleaned almost at random from the mass of available material. It was by the experiments and writings of Dr. Winternitz, of Vienna, that the claims of this method to rank as a genuine scientific procedure were finally established. The publication of his compendium in 1879 did for the *rationale* of the "water cure" what the sensational career of the Silesian peasant, Priessnitz, at Gräfenberg (who died in 1852 worth several millions) had achieved for its popularity.

The recently-published investigations of Sir George Johnson reaffirm and abundantly demonstrate the vast importance of vasomotor influences, as expressed by changes in the capacity of the systemic and pulmonary arterioles, in determining many apparently very diverse pathological conditions. This idea of the predominance of vasomotor influences might almost be called the key to the *modus operandi* of hydrotherapy, for it is the special facility with which they enable us to modify the vascular conditions obtaining in disease to which the efficacy of hydriatic procedures is mainly attri-

butable. Such other elements of utility as the direct effects of thermal and concomitant mechanical impressions upon innervation, local and general temperature, the heart's action, muscular tone, and the digestive, nutritive, and metabolic processes, are, though doubtless of considerable, yet, in so far as they are divorced from the vasomotor effects of the same procedures, for the most part of subordinate, importance.

Although, of course, apart from the question of mechanical impressions, the therapeutic value of water used externally depends largely upon the means it affords of bringing various and exactly determinate degrees of temperature to bear upon parts or the whole of the body, the important sedative and narcotic properties of baths of indifferent temperatures must not be overlooked. Thus a simple warm or tepid bath taken at bedtime will cure some cases of insomnia. These effects are plausibly explained by Heymann and Krebs as being due to saturation of the sensory nerve endings (partly by imbibition, partly through suspended evaporation), and consequent diminution of conductivity.

By the action of thermal stimuli upon innervation, irritability may be raised or depressed, in some instances abolished even, at will (1) at the point of application, (2) in the central organ, (3) by reflection in other parts—for example, the muscular layer or the underlying viscera. The interesting researches of Dr. Head, recently communicated to the Neurological Society, tend to establish the fact (long suspected by clinical observers) that definite cutaneous areas, areas that do not overlap, are connected by nerves of temperature and common sensation with particular spinal segments, and thence, through the distribution of the sympathetic, with particular internal organs. And besides these direct and primary paths of reflection, there are probably many distinct parts of the organism which have positive or negative neural or vasomotorial relations to each other—for example, the feet and the head, and, according to Winternitz, the hands and the organs of respiration. The aim of the hydrotherapist (amply justified by the results of experience) is to utilise these and similar physiological data in the task of modifying by appropriate thermal and mechanical stimuli the metabolic and functional conditions of diseased tissues and organs.

Hardly less important than the distinction between the effects on the superficial and deep-lying tissues of a given thermal stimulus at the point of application is that between the effects produced at situations respectively central and peripheral to it. Generally speaking, a certain antagonism or negative relation between the superficial and deeper, as also between the peripheral and central, primary effects of moderate stimuli may be anticipated, examples of which are, in the first instance, the reflex acceleration of muscular circulation and metabolism, which is a prominent effect of cold cutaneous applications, and, in the second, the fact that the application of ice poultices to the arm near the elbow, while it induces contraction of the radial and ulnar arteries and a fall of temperature in the hand, causes precisely opposite effects in the axilla. There are probably few exceptions to the rule (verified by Schlikoff through experiments on rabbits and by Winternitz with plethysmographic observations as to the circulatory effects of partial baths of various temperatures), that local contraction or dilatation of arterioles, however induced, is accompanied and followed by contrary behaviour on the part of vessels elsewhere.

By the prolonged action of a given thermal stimulus the protective influence of vasomotor accommodation is overborne and a general thermal effect produced, but even the surface temperature of a living part is never quite equalised to that of the thermal medium. Experiments made by Schlikoff and Esmarch prove, however, that the very cavities of bones, as also the deep-lying organs of the trunk, may be cooled or heated by external applications. And to modify the temperature of the body or a part of the body is to modify in the same degree its circulatory. and thereby its nutritive and functional activities. Thus the outcome of a series of hydriatic operations individually to be characterised as a mere abstraction of heat may be, not a loss, but a gain of body weight, showing that over and above the inevitable stimulation of destructive processes there has been a still more effectual stimulation of constructive metabolism.

It is hardly necessary to insist upon the value of cold baths in the treatment of hyperpyrexia, but it is too frequently forgotten that, properly administered, they and similar hydriatic operations constitute a true antipyretic, not a mere antithermal procedure. In other words, over and above the physical abstraction of heat, diminution of its production may by their means be effected. In view of this fact, and also of the encouraging results of early hydropathic interference in fever cases, as contrasted with the depressing effects of antipyretic drugs, it is, I think, to be regretted that in private and hospital practice such interference is usually reserved for extreme cases and resorted to only after the febrile condition has been for some time in evidence. The following rules, if borne in mind by the practitioner who employs this method, will enable him to deal with such cases more effectually and with greater precision than is commonly imagined.

In the first place, it is to be remembered that the more suddenly heat is withdrawn from the body, and the more intensely the cutaneous nerves are stimulated by refrigeration, so much the more rapidly will the reactionary rise of temperature ensue. Now the heat abstraction caused by efficient wet packing being quite a gradual and the concomitant stimulation a mild one, the onset of reaction after a pack or series of packs (for several brief ones may be given in immediate succession) will be correspondingly slow. Another means of avoiding that stimulation of thermogenesis (due to reflex muscular hyperæmia) which in extreme cases takes the form of rigor, is the maintenance throughout the heat abstracting process of cutaneous vascular dilatation. Hence the antipyretic efficiency of the dripping sheet (that is, friction under a sheet drenched at intervals with cold or cool water), for this dilatation is best induced and maintained by the mechanical stimulation of active friction, which should also and for the same reason be employed during the administration of the ordinary cooling bath. It is asserted that the rapidity of heat loss may vary as much as 60 per cent. downwards, and more than 90 per cent. upwards, in accordance with the condition of the cutaneous vessels. Hence the simultaneous employment of friction will enable us to dispense with baths of very low temperature, the use of which may frequently be undesirable in these cases. Repeated packings, followed by cooling baths, have been found to reduce pyrexia more permanently than baths alone. The onset of reaction is also deferred by the application, during the intervals of more vigorous treatment, of cooling truncal compresses, that is, wet towels or narrow sheets wrapped round the body, with a light covering sufficient to interfere but slightly with evaporation, rewetted as often as may be necessary.

The above principles of antipyresis may profitably be contrasted with the hydropathic treatment of obesity, in extreme cases a true pathological condition. Here our main object is to stimulate combustion of non-nitrogenous constituents of the body, and, since physiology teaches us that such combustion is an invariable accompaniment of the phenomena of muscular activity, our object will be secured by whatever promotes the exaltation of muscular tone and function. Since the reflex effects produced in the muscular layer by external thermal stimuli are proportional to the intensity of the stimulation of the cutaneous nerves, the reduction of fat by a given application of cold will be proportional not so much to the actual as to the apparent or felt refrigeration. Hence we shall endeavour to increase the sensibility of the cutaneous nerves before submitting them to the process of refrigeration, and thus is explained the efficacy in cases of obesity of comparatively brief Turkish baths, followed by cold or cool needle, spray, plunge, or shallow baths, even without shampooing. Moderate diaphoresis is useful in such cases as an aid to the elimination of the increased waste products; but the popular notion that the longer the sweating process is borne the better the result is a mischievous fallacy.

The Turkish bath (in spite of a widespread impression to the contrary effect) is fairly described in the words of an authority as "pre-eminently the bath for the weak and disabled." In cases of general atrophy and asthenia it is (as proved by its effects upon the appetite and body-weight) one of the best restoratives at our disposal, and its cautious administration is by no means contraindicated by the

existence of any and every degree of even organic disease of the heart.

In asthenic cases exposure for from fifteen to thirty minutes to a temperature of 115° to 120° F. in the tepidarium—the thermometer hanging three feet from the floor level—will usually induce mild but sufficient diaphoresis without distress of any kind, and may be followed by some appropriate ablution, any intense thermal or mechanical stimulation being, of course, out of the question. Even this modified form of bath is probably inadmissible in severe cases of aortic regurgitation, compound valvular lesions, dilatation from adherent pericardium, or fatty degeneration, but in simple cases of aortic and mitral stenosis and mitral regurgitation it is often beneficial in many ways.

An important subject, to which a mere cursory reference is alone possible at present, is that of the properties and uses of the so-called excitant or heating compresses—cold, wet compresses covered in such a way as to preclude evaporation. The powerful influence of their continued application, in promoting the resolution of chronic inflammations and catarrhs, the absorption of old and hardened exudations, and the relief of various neuralgic and spastic affections, is one of the best-ascertained facts, and its precise *modus operandi* one of the most interesting problems of hydrotherapy. Fortunately, however, the fact itself of the efficacy in question of this apparently trifling procedure is one which can be readily tested by any practitioner.

A few words must be devoted to the subject of the methodical drinking of cold water. As collated and in part discovered by Winternitz, the effects of this procedure may be briefly summarised as follows:

1. Immediately after the drinking of cold water the pulse-rate is lowered, probably by stimulation of the pneumogastric fibres, but soon returns to the normal.

2. The temperature falls in the stomach and also, to a noticeable degree, in the rectum, a fact which may find clinical application in inflammatory conditions of the pelvic organs; conversely, cold enemata reduce the temperature of the stomach.

3. Catabolic processes are stimulated, and there is true diuresis, the excretion of urea, as also of the other urinary constituents, being markedly increased.

4. Peristalsis and the portal circulation are stimulated, and also (in consequence, perhaps, of the latter effect) the excretion of bile.

5. Böcker has attributed to water drinking an improvement in the respiratory qualities of the red blood corpuscles.

Most of the above effects are more marked when the water taken is imbibed gradually, rather than in larger quantities at a time but with longer intervals. The value of pure water as a diuretic is perhaps hardly yet sufficiently appreciated. Dropsical effusions may sometimes be relieved by its free administration, alternated with periods of nearly complete abstention. I would suggest also that in view of the proofs advanced by Dr. Haig of the tendency of alkalies to induce chronic uric-acidæmia, their routine administration in cases of gout and rheumatism, except, perhaps, in the hope of dissolving actual calculi, is hardly justifiable, and in pure water we possess at least one available substitute. Its power of increasing tissue waste indicates water drinking as a remedy for obesity.

What place, then, are we to assign to hydriatic procedures as a class in the modern therapeutic armamentarium? Obviously their scope is far wider than that of any individual drug. The modern renascence of hydrotherapy was contemporaneous with the final close of the reign of authority and tradition in the healing art and the dawn of the epoch of science. A wave of therapeutic pessimism had swept over Europe, and sectarian hydropathy was the outcome of an instinctive return to first principles, a feeling that it would be necessary to begin over again and to build upon surer foundations. But Priessnitzian hydropathy was no less arbitrary and empirical than the system it pretended to supersede, and it was not until the attention of scientific observers had been secured that the extravagant claims of fanaticism were replaced by the sober assertions of reason. In France this was the case almost from the first, but it occurred later in Germany and England. Meanwhile the new science of pharmacology had stolen into being, and it began to be perceived that the so-called rival systems were not necessarily rivals at all, but complementary factors of the one true method, their apparent antagonism being due to an imperfect understanding of their respective functions. In practice there can be no doubt that the benefits of really appropriate medication are augmented by the simultaneous employment of hydriatic procedures.

To such other elements of rational treatment as are constituted by massage, medical gymnastics, the scientific regulation of diet, aëro-therapeutics, and the various electrical methods, the natural relationship of hydrotherapy is sufficiently obvious. Its amalgamation with these has already proceeded far, and is destined, probably, to proceed much further in course of the construction of that concrete and organised system which we dimly apprehend as the gaol of therapeutic endeavour.

ON THE NATURE AND SIGNIFICANCE OF THE CRESCENTIC AND FLAGELLATED BODIES IN MALARIAL BLOOD.

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IN malarial blood certain forms of a parasitic organism are invariably present, singly or in association. The bodies referred to consist principally of a pale hyaline substance with or without granules of dark pigment, and are (1) minute nucleated bodies, or spores, which are free in the blood; (2) small epi- or intracorpuscular bodies, presumed to be these spores which have become attached to or have attacked the red blood corpuscles; (3) larger intracorpuscular, pigmented, amœboid bodies; (4) sporulating intracorpuscular forms, known as "corps en rosace," or "rosette bodies"; (5) the last mentioned outside the blood corpuscles, breaking up and becoming resolved into the first mentioned or free spores; (6) intermediate forms which serve to connect these types and suggest, if not prove, that together they form a complete vital cycle.

The presence of this parasite in the blood precedes and accompanies a special type of fever; the cycle of its development coincides in the main with the cycle of the fever; and it disappears from the blood with spontaneous recovery from or cure of the fever.

The intravenous injection of blood containing this parasite into a healthy individual is followed by the multiplication of the parasite in the blood of the person inoculated, and also by the occurrence in him of the characteristic fever. On these grounds the conclusion that malarial fever is caused by this organism is justified, if not absolutely proved.

Unless in the unnatural way by direct transfusion of blood, the malaria organism and malarial disease are not directly communicable; that is to say, the malaria parasite cannot be acquired by simple proximity to or by contact with an infected individual.

Apart from communication by transfusion of blood, the malaria organism and malarial diseases can be acquired only indirectly either through the air, the water, by food, or by other unknown way.

Malaria can be acquired, and its germ is therefore present, in places where there are few or no inhabitants, and where human beings rarely reside or pass through.

These propositions are all of them fully established facts.

Considering the frequency of the presence of the malaria organism in the human body, that in malarial countries at one time or another almost every individual harbours it in his blood, that it manifestly flourishes and progagates there, it is unreasonable to suppose that its presence in the human blood is a purely accidental circumstance in the sense that it is an organism which has strayed from its proper habitat, or to suppose that being in the blood, as regards itself, it is in