CASE V.

S. C., aged 22, single, a seamstress, was admitted into the Manchester Royal Infirmary under Dr. (afterwards Sir) William Roberts, July 22nd, 1867, suffering from what was then called syphilitic rheumatism. After a course of alkaline carbonates and potassium iodide, these were on August 7th discontinued and the syrup of sarsaparilla in full doses was substituted.

	st. lb.	lb.
Weight on Aug. 7th, 1867	6 2½	
" Aug. 14th "	6 7½	a gain in the week of 4½

On August 15th the patient left the hospital at her own request.

CASE VI.

J. P., aged 49, married, a fustian-cutter, was admitted into the Manchester Royal Infirmary under the care of Dr. H. Simpson, July 29th, 1867, suffering from syphilitic cachexia, with a livid papular eruption and evidences of chronic peritonitis. He was at first treated with iron, but on August 9th syr. sarsae was substituted for the iron. He was at that time taking a milk and beef-tea diet; this he continued until August 20th, when he was ordered an ordinary meat diet.

	st. lb.	Gain per Week.
Weight on Aug. 10th, 1867	6 8	
" Aug. 17th "	6 12½	4½ lb. on sarsaparilla only
" Aug. 24th "	7 2	2½ lb. " "
" Aug. 31st "	7 4½	2½ lb. " "
" Sept. 7th "	7 6½	1½ lb. " "
Total gain in four weeks, 12½ lb.		= an average of a little over 3 lb. per week.

The greatest gain having been during the week when the patient was on a milk and beef-tea diet seems to show that the gain cannot entirely be accounted for by the diet.

CASE VII.

F. B., aged 25, married (but separated from her husband), a dressmaker, was admitted into the Manchester Royal Infirmary under Dr. T. H. Watts, February 3rd, 1868, suffering from syphilitic cachexia with periosteal symptoms. On her admission she was ordered a mixture containing 5-gr. doses of potassium iodide. On March 11th, syr. sarsae was substituted for this, but on the 19th, for some reason not stated in my notes, the sarsaparilla was discontinued and the potassium iodide resumed.

	st. lb.	lb.
Weight on Feb. 14th	6 13½	
" Feb. 21st "	7 3	a gain in the week of 3½

CASE VIII.

W. M., aged 29, single, a collier, was admitted into the Manchester Royal Infirmary, under the care of Dr. T. H. Watts, February 10th, 1868, suffering from syphilitic ulceration of the pharynx, said to be of five months' duration. On February 12th he was ordered syr. sarsae, 4 fluid oz., three times a day, without other medicine.

	st. lb.	lb.
Weight on Feb. 13th	8 6½	
" Feb. 20th "	8 9	a gain in the week of 2½

CASE IX.

J. D., aged 25, a widower, by occupation a wheelwright, was admitted into the Manchester Royal Infirmary under the care of Dr. T. H. Watts, March 6th, 1868, suffering from large painful swellings along the front of both tibiae and caries of the bones of the nose. He was ordered potassium iodide in doses of 5 gr., the quantity to be gradually increased. On March 28th he was taking 35 gr. per diem. On April 4th he was taking 45 gr. daily. He was now ordered syr. sarsae in addition, and continued to take it until April 25th.

	st. lb.	Gain per Week.
Weight on April 4th, 1868.	8 1	
" April 11th "	8 7¾	6¾ lb. on pot. iod. and sarsap.
" April 18th "	8 12½	4½ lb. " "
" April 25th "	9 0¾	2½ lb. " "
" May 2nd "	9 3	2½ lb. on pot. iod. without sarsap.
" May 9th "	9 4½	1½ lb. " "

On May 11th he became an out-patient. For two weeks he was, owing to an oversight, entirely without medicine. On May 23rd he was ordered 10-gr. doses of pot. iodid. with iron.

	st. lb.	lb.
Weight on May 16th	9 3½	a loss in the week of 0½
" May 23rd "	9 3½	gain " 0½
" June 2nd "	9 3	loss " 0½
" June 9th "	9 5½	gain " 2½
" June 16th "	9 6½	" 1
" June 30th "	9 7	gain in 2 weeks of 0½

The following and last case is one in which improvement was greater when treated with potass. iodid. alone than when treated with sarsaparilla alone:

CASE X.

M. J., aged 27, married, was admitted into the Manchester Royal Infirmary, under the care of Dr. (afterwards Sir) William Roberts, April 17th, 1868, suffering from a syphilitic ulceration at the os uteri and at the posterior commissure of the vulva, a livid scaly eruption, intense headache, with tenderness of scalp (the pain being worse at night), and slight delirium. The potassium iodide ordered on admission was supplemented on April 25th by 4 fluid oz. of syr. sarsae, three times a day. On May 1st the sarsaparilla began to be given alone, but on the 7th it was for some reason discontinued, and the potass. iodid. resumed.

	st. lb.	lb.
Weight on April 17th, 1868	7 14	
" " 24th "	7 3½	gain, 2 lb. (pot. iod.)
" May 1st "	7 6	gain, 2½ lb. (pot. iod. and sarsap.)
" " 8th "	7 6½	gain, 0½ lb. (sarsap. only)
" " 15th "	7 10	gain, 3½ lb. (pot. iod. only)
" " 22nd "	7 12	gain, 2 lb. (pot. iod. only)

I realize that these notes are not full enough or numerous enough to be of any very great value, but few and scanty as they are, they seem to me to be worth placing on record. The Manchester series of observations was watched at the time with considerable interest, not unmixed with a wholesome scientific scepticism, both by the resident and visiting staff. Those of my readers who are familiar with the atmosphere of a hospital common-room will well understand this. Nowhere have doubtful questions and "tall" stories to bear the brunt of a more unmerciful scrutiny, and this Leeds story of mine had to share the usual fate. I have had no opportunity since of following up these observations save in the most desultory manner. But I was pleased to know that sarsaparilla still holds its own at Leeds. I was also glad to be reminded that the credit of introducing the preparation at the Leeds Infirmary was due to that fine old surgeon (who was also lecturer on midwifery), Samuel Smith, in whose praise Professor Allbutt says not one word too much.

The Lumleian Lectures

ON

TABES DORSALIS.

DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS

By DAVID FERRIER, M.D., F.R.S.,

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LECTURE II.*

In health the centrifugalized deposit from the cerebro-spinal fluid contains scarcely any cellular elements beyond an occasional endothelial cell and perhaps one to three lymphocytes in a field with a magnification of 400 diameters.

In certain diseases the cellular elements are present in excess. In acute microbic affections of the cerebro-spinal meninges, as by staphylococcus, pneumococcus, and *Diplococcus intracellularis*, a leucocytosis occurs, in which the leucocytes are of the polynuclear type. On the other hand, in certain chronic affections of the meninges, syphilitic, tuberculous, etc., an excess of leucocytes also occurs. These are of the mononuclear type; in other words, there is a lymphocytosis. It has been shown by Vidal, Sicard, Ravaut, and others—and their observations have been confirmed by my own and Purves Stewart's investigations—that in tabes, as well as in general paralysis of the insane, from the earliest to the latest stages there is a marked lymphocytosis of the cerebro-spinal fluid. I have in several instances relied on this lymphocytosis as establishing the diagnosis of general paralysis before there were any abnormalities of the pupils or other physical signs characteristic of the disease. In a case of tabes or general paralysis, instead of two or three, as in the normal state, one may count a hundred, or even several hundred, lymphocytes in the field of the microscope. At first sight this lymphocytosis would seem to confirm the views of those who ascribe the disease to a syphilitic affection of the meninges, either generally, or of Nageotte's meningeal root sheath, or of the posterior lymphatic system of the spinal cord as maintained by Marie and Guillain. But lymphocytosis occurs in other affections in which meningitis, syphilitic or otherwise, plays no part. Thus it has been found in Landry's paralysis, in the sub-acute combined degeneration of pernicious anaemia (Purves Stewart), and in herpes zoster (Sicard). Lymphocytosis cannot therefore be regarded as pathognomonic of meningeal inflammation, though no doubt meningeal inflammation would accentuate it. But the most powerful argument against the syphilo-meningeal origin of the

* The lecture was illustrated by lantern slides.

lymphocytosis in tabes and general paralysis is the fact that it does not yield in the slightest degree to the most energetic antisyphilitic treatment (Milian, Purves Stewart). In active syphilitic lesions of the nervous system, however, the lymphocytosis that may be present tends to disappear in the course of treatment. In a case under the care of Dr. Purves Stewart, the lymphocytes had decreased after a month's antisppecific treatment from 52.6 to 16.1 per field, and the symptoms had become greatly ameliorated.

Having thus passed in review the different theories which have been advanced in reference to the pathogeny of the tabetic degeneration, we have seen that there is not one which is not open to several more or less serious objections.

Instead of attempting to be more precise than the facts permit, I am inclined to adopt the hypothesis of Thomas and Hauser, that the essential lesion of tabes is a dystrophy, similar to that induced by certain toxic agents, affecting the sensory protoneurone as a whole, and manifesting itself in degeneration both of the peripheral and central terminations, of which the intramedullary are the more vulnerable, and are usually the earliest to exhibit anatomical change. The process, however, is not confined to the spinal protoneurone, but may affect among others the optic, the sympathetic, and certain motor neurones.

And here I would express, in concurrence with Fournier, Mott, and many other neuro-pathologists of the present day, my belief in the essential pathological identity of tabes and general paralysis. They are, in my opinion, merely different aspects of the same polymorphic disease. One cannot say that they are the same disease so far as their clinical manifestations and course are concerned, any more than one would say that tuberculous meningitis and pulmonary consumption are the same. But the morbid process underlying both is identical.

Both are tabetic or wasting affections—of the sensory protoneurone in the one case, and of the cortical neurones in the other. The essential etiological factor is, as we shall see, the same, and the date of development of the two affections after the primary infection corresponds. No blind or crude primary meningeal inflammation could selectively attack only such regions—the fronto-parietal by preference—as exhibit the atrophic cortex and adherent lepto-meninges characteristic of general paralysis.

The primary lesion in general paralysis, as in tabes, is a dystrophy of the neurones and their connexions; the sclerosis, thickening of the membranes and walls of the blood vessels, being only a secondary result of the parenchymatous degeneration.

Not infrequently tabes and general paralysis are combined, or succeed each other, in the same individual. Clinically, some of the characteristic signs, notably the Argyll-Robertson pupil, are common to both, and in both there is the same cerebro-spinal lymphocytosis.

It would, in my opinion, conduce to a truer conception of the pathology of these affections if we were to call them both by the generic name "tabes," classifying them specifically as Cerebral Tabes (general paralysis), Spinal Tabes (tabes dorsalis, locomotor ataxy), and Cerebro-spinal Tabes (so-called tabo-paralysis), according as the disease affects the brain, or spinal cord, or both conjointly.

ETIOLOGY OF TABES.

Up to 1876, when Fournier first pointed out the great frequency of antecedent syphilitic infection in tabes and indicated their probable causal relationship, tabes was most commonly attributed to sexual excess, or the complex of injurious influences associated with exposure to cold. The supposed causal relationship between sexual excess and tabes was probably only a *damnsa hereditas* from the tabes dorsalis or sexual neurasthenia of the Hippocratic writers.

Sexual excess, like all other debilitating influences, may be placed among the exciting causes, and many tabetics have probably exhausted themselves in this way; but the influence of sexual excess in the causation of tabes has been greatly over-estimated, and if that were the only cause the disease would certainly be much more common than it is.

Similarly as regards cold or damp cold, which Romberg thought, and which is still regarded by v. Leyden as one of the principal causes of tabes, this view is probably founded

more on the patient's belief that his tabetic pains are rheumatic in character, and that he must have caught a chill, slept in a damp bed, or the like, than on any more substantial basis. When we consider how often, when our etiology is at fault—not only in respect of diseases of the nervous system, but many others—it has been the custom to fall back upon exposure to cold as the probable cause, afterwards proved insufficient, we may without undue depreciation call it the last refuge of destitute etiology. Like sexual excess, exposure to cold and damp would without doubt have an injurious influence, and probably excite or intensify tabetic symptoms already incipient, so that the patient might have some grounds for ascribing his malady to this cause. But that exposure to cold by itself is a sufficient cause of tabes is, in my opinion, a mere vague assertion, supported by no accurate data and at variance with one's daily clinical experience. A history of exposure to cold is not found more frequently in tabes than in many other affections of the nervous system.

Fournier's views as to the causal relationship between syphilis and tabes were at first received with scepticism, not to say strong opposition, in many quarters; and the frequency of antecedent syphilitic infection in the personal history of the tabetic was not regarded as more than might be found in other forms of disease of the nervous system. But Fournier's views soon gained powerful support, among the first by Gowers in this country and by Erb in Germany; and Erb, who at one time (1878) was a strenuous opponent, is now perhaps the most eminent advocate of the syphilitic origin of tabes. It is now so generally admitted by neuro-pathologists, with some few notable exceptions, such as v. Leyden, that syphilis is the principal cause of tabes, that it seems unnecessary to quote the data on which this opinion is based. But as the elimination of cause and effect depends for the present mainly on the statistical method, and as this is liable to many fallacies depending on the personal equation of the observers and the material dealt with, it will be sufficient to state the results obtained by Erb, whose probably unequalled experience, and laborious inquiries, carried out with systematic uniformity, invest his inductions with exceptional value. In the most recent of his numerous contributions on this question, Erb states that among 1,100 cases of tabes occurring in his own practice among men of the better class, and therefore better witnesses than those living among poor and squalid surroundings, 89.45 per cent., or practically 90 per cent., had had chancre or syphilis; and only 10.54 per cent., or practically 10 per cent., had not been ostensibly infected. But of these, in only 3 per cent. was it impossible to find any ground for suspecting an antecedent syphilitic infection.

Now contrast these statistics, obtained from 1,100 tabetics, with those of 10,000 cases, also of men of the better class, of all varieties of disease, excluding tabes. Of these, only 21.5 per cent. had had syphilis, as against 89.5 per cent. among the tabetics, that is, that syphilis was found 4.5 times more frequently among tabetics than among other patients. Similar investigations among 138 tabetic men of the lower orders give a somewhat lower percentage of antecedent syphilis, namely, 77.2 per cent.; but among 1,300 non-tabetics only 6.54 had had syphilis, as against 93.46 non-infected.

Among women, who are less liable to tabes than men, and those of the better class less so than those of the lower orders, it is for various reasons much more difficult to determine the question of syphilitic infection. But Erb found that among 15 tabetic women of the better class, at least 86.7 per cent., and probably 93.3 per cent., had been infected. Among 16 women of the lower orders, however, syphilis was probable in only 68.7 per cent., and unproved in 31.3 per cent.

Taken altogether, a statistical inquiry among 1,321 cases—namely, 1,100 men of the better class, gives 89.5 infected; among 158 men of the lower class, 77.2 per cent.; and among 63 women, 80 per cent. as probably infected. Essentially the same is true of general paralysis.

So far for mere statistics, the general result being that in about 10 per cent. of the cases syphilis is unproved. These 10 per cent., however, constitute the pivot round which the essential question turns, whether tabes, like diphtheria or tuberculosis, is always due to a specific

infection, or whether it may result from some other cause. This is not a mere academic question, but one of far-reaching practical importance. Can we in the present state of our knowledge give a definite, or approximately definite, answer to it?

We may, perhaps, do so by taking other facts into consideration besides mere percentages.

It has been established, and is generally recognized, that tabes frequently follows syphilitic infection of a mild type, and therefore more likely to escape notice, particularly among those in whom personal cleanliness is not a cardinal virtue. And syphilis ignota or occulta is familiar to syphilologists and dermatologists.

Some noteworthy investigations on this point have been made by Hirschl, the results of which are worth quoting. He found that, of 63 patients suffering from gummatous affections of indubitable syphilitic origin, in only 54 per cent. was there a certain history of syphilis; in 9.5 per cent. it was probable; while in 36.6 per cent. no evidence, other than the patient's actual state, was forthcoming of syphilis, hereditary or acquired. Practically the same results were obtained by him in reference to general paralysis.

Similar statistics to those obtained by him in reference to syphilis and tabes have been quoted by Gowers as having been arrived at by Dr. Pernet, of University College Hospital, in reference to syphilitic affections of the skin. Syphilis could only be proved in 80 per cent. of the cases.

One may therefore say with justice that the syphilitic origin of tabes and general paralysis is statistically just as well proved as that of gummatous affection of the skin.

Apart from juvenile tabes, which is always syphilitic, hereditary or acquired, and which affects both sexes in the same proportion, tabes occurs most commonly among men of from 30 to 40 years of age, and on the average from seven to ten years after the primary syphilitic affection. There are, however, exceptions to this rule, and I have seen it develop in a man of 70, some forty to fifty years after infection with syphilis; and there is a case on record (Berger) of a man of 74 becoming tabetic after syphilitic infection at the age of 70.

Tabes at from 30 to 40 is a significant harvest of the wild oats sown some ten years previously, and the classes who sow wild oats most freely are those who reap tabes most abundantly. Townspeople furnish more cases of tabes than those who live in the country; and great centres of population with their greater risks and temptations more than the smaller. Tabes and general paralysis are rare among priests, and of these only such as have had syphilis, or exposed themselves to it (Bouchard).

Tabes is less common among women of the better class than among those of the lower—doubtless, as Moebius has suggested, owing to the fact that men of the better class less frequently marry while still suffering from infective syphilis.

But conjugal tabes is not uncommon, and most neurologists have seen examples. Among others, Mott has given several interesting illustrations. Almost without exception there has been syphilitic infection (90 to 96 per cent., Hudovernig) on one side or the other; usually the husband has infected the wife. Nonne has related a very remarkable family history. A child of 5 was infected with syphilis through sleeping with a syphilitic bedfellow. At 18 he became tabetic. The child infected the mother, and the mother the father. Both these became tabetic. In another family, described by Souques (quoted by Erb), the father was syphilitic and became a general paralytic; the mother became tabetic, and two young daughters also showed signs of tabes—four in one family. Many similar cases might be quoted in detail, but it is unnecessary to multiply them. Syphilis, acquired or hereditary, was the common factor in all.

And the question has been mooted (Lavalle, Moebius, Erb) whether there may not be a form of the syphilitic virus falling with special incidence on the spinal and cerebral centres, and leading in due course to tabes or general paralysis. Thus Brosius relates the remarkable history of seven glass-blowers who were infected in the mouth from the same source in 1891. All were treated for syphilis and dismissed as cured after a few weeks of anti-specific treatment. In 1902, that is, after eleven years, five of them came under his observation again. One was tabetic; the second had signs of general paralysis; the

third was already a marked general paralytic; the fourth had Argyll-Robertson pupils, and indications of so-called tabo-paralysis; while the fifth at the time of examination was still in good health. The other two had not been traced. The only common factor in all these cases was the syphilitic infection.

It has been shown by Krafft-Ebing that general paralytics are insusceptible to inoculation by the syphilitic virus. This is a negative fact of great significance and of much more importance than if the converse were true, for reinfection is always a possibility. Whether the same holds in respect to tabetics has not, so far as I know, been yet demonstrated, but it is more than probable. Who has ever seen a tabetic with a primary sore, or even with secondary symptoms? A considerable number, both during life and after death, exhibit tertiary manifestations, or traces of antecedent syphilitic lesions. But these date from the infection which preceded the tabes. Yet it cannot be said that tabetics do not run the risk of reinfection, for it is notorious that in the pre-ataxic stage tabetics frequently exhibit excessive erotic propensities, and expose themselves freely to the possibility of acquiring syphilis.

Female children apart, tabetic *virgines intactae* are *rarae aves*. They are, however, not unknown. Of two of whom I have read, the one (reported by Nonne) was a tabetic girl of 20, infected in infancy by her mother suckling a syphilitic foster-child at the same time; the other (described by Erb) a virgin of 36, who some time previously had had a chancre on her lip.

One might multiply arguments in favour of the causal relation between syphilis and tabes, but they are unnecessary. For those above related, singly and collectively, leave in my opinion little room for doubt that tabes and general paralysis are in all cases syphilitic, and that tabes *per se* is as much a proof of syphilis as a gumma of the skin. Those who may be still unconvinced, and seek a more positive sign, will probably have to wait till in some pathological laboratory a monkey will be exhibited, inoculated with a virus of syphilitic origin (a product perhaps of the *Spirochaeta pallida*), showing Argyll-Robertson pupils and loss of knee-jerks; and the question of the syphilitic origin of tabes will be solved by the walk of a tabetic chimpanzee.

It has been urged by v. Leyden and Goldscheider as an argument against the syphilitic origin of tabes, that the disease is rare among prostitutes, among whom nevertheless syphilis is rife. But it must be remembered that tabes is less common among women than men; and one must also take into account the fact that as tabes is a late manifestation of syphilis, it is not to be expected among those who are still young enough to ply their wretched calling. This accords with the statistics of Kron, who, though he found no case of tabes among 148 young prostitutes, yet among 36 over 25 years of age found 5—a large percentage enough.

Similarly it has been contended that in certain countries and among certain races tabes is rare, though syphilis is common. Thus it has been asserted that in Bosnia and Herzegovina, in Japan, in Abyssinia, among the Arabs, and among negroes tabes does not occur. But more accurate investigations have proved this assertion to be unfounded. For tabes is common enough in Japan (Nose); it is not unknown in Bosnia and Herzegovina; it is as common in Abyssinia as in Vienna (v. Halban), and it certainly occurs among negroes (Collins, Hecht).

It may not be so common among primitive races as among those more advanced in civilization and all that this connotes, but this is not the point. The fact that tabes only occurs where infection with syphilis is possible is the essential point in this relation.

If tabes can be shown to exist, or to have existed, in any land or people among whom syphilis has never entered, and who have never had communication with any outside syphilized world, the exclusively syphilitic origin of tabes will have to be abandoned, but not till then.

The relative infrequency of tabes among those who have suffered from syphilis—it has been variously calculated at from 1 to 5 per cent. of the whole—is no argument against the syphilitic origin of tabes. For there are many affections due to syphilis, such as aortitis, hepatitis and orchitis, which are relatively rare. And as Erb remarks, the fact that perhaps only 1 in 10,000 of those infected

with tubercle suffers from Addison's disease does not disprove the tuberculous nature of the affection of the suprarenal capsules. But the smallness of the percentage, as well as other considerations, suggest that, in addition to syphilis, there may be some predisposing or other co-operating causes, determining the incidence of the syphilitic virus on the nervous system, and the development of tabes either of the brain or spinal cord or both.

Heredity, as such, plays no part in the etiology of tabes, though, as we have seen, juvenile tabes indicates a syphilitic inheritance.

Mention has already been made of sexual excess and cold, to which great, but as we have seen, undue prominence has been assigned by certain pathologists. The same may be said of neuropathic inheritance, to which great importance was attached by Charcot. It is not more frequent in tabes than in other forms of nervous disease. Some facts, however, suggest that certain families are specially prone to develop tabes: and several instances might be quoted of more than one member of the same family becoming either tabetic or general paralytics after acquiring syphilis.

Certain malformations, or developmental anomalies, such as heteropia spinalis, have been recorded by several observers (Mott, Orr and Rows, Rebizzi) in the spinal cord of those affected by tabes or general paralysis. All these have, no doubt, their significance, but they are by no means constant nor are they even common.

The view that the substratum of tabes lies in some defect of development, or vice of inheritance, which predisposes to premature decay of the essential elements of a highly specialized tissue, and reversion to those of a lower type, so that certain individuals are, as it were, predestined to tabes, reaches its culmination in Benedikt's burlesque, "*Tabicus nascitur: non fit!*" No one who has had experience of tabes in all ranks, and has seen among its victims numbers of men of the most healthy stock and vigorous physique, can regard it otherwise than a gross libel to speak of tabetics in general as being "connective-tissue individuals" (*Bindegewebs-individuen*) or "degenerates" in any true sense of the term.

Among other exciting causes of tabes, injuries have been blamed, and many cases of so-called traumatic tabes have been put on record. There is no doubt that in certain instances—perhaps 5 per cent.—tabetic symptoms have first made themselves manifest after injuries of the spine or other parts of the body, and the limb injured has not infrequently been the primary, or principal, seat of the characteristic pains; but a critical examination of such cases by Hitzig and others has failed to substantiate the efficacy of trauma as a cause of tabes independently of antecedent syphilitic affection.

Of greater importance, perhaps, than any of the above-mentioned exciting or co-operating causes of the tabetic degeneration is the influence of over-exertion or of over-exertion in debilitated states of the system. The principle underlying these degenerations is that the normal equilibrium between waste and repair which characterizes health is disturbed, and that in consequence of the process of waste exceeding that of repair the neurons, especially those most engaged, tend to break down and show dystrophic change. Edinger and Helbing have found that in rats artificially rendered anaemic, which have been compelled to work in a treadmill, or, by other device, to engage in long-continued muscular exertion, the posterior roots and posterior columns of the spinal cord exhibit degenerations similar to those of tabes.

These facts are suggestive, and appear to harmonize well with the views of those who regard fatigue and over-exertion as the most important causes of tabes. As co-operative causes certainly, but not apart from syphilis; for the same degree of fatigue and over-exertion which may excite tabes in one in whom the soil is already prepared by syphilis, may be borne by thousands of others without material damage.

Edinger's "compensation theory" (*Ersatz-theorie*) seems also to afford a plausible explanation of the incidence of the affection in the parts most subjected to continuous strain, the most common being the sensory apparatus concerned in co-ordinated locomotion and maintenance of equilibrium. Occasionally, however, as in cervical tabes, the arms, for similar reasons, are more especially affected; and the pupils and optic nerves would

in like manner probably suffer more readily than any of the other organs of special sense.

So also muscular atrophies would be more prone to occur in the muscles which are most taxed, similar to the occupation-muscular atrophies of blacksmiths, railway porters, and the like. Probably the same principle may serve to explain the incidence of the tabetic degeneration in one case on the brain, and in another on the spinal cord. Raymond has related an interesting narrative of two brothers, similarly constituted by inheritance, who both became infected by syphilis about the same age. The one who lived a fast life and indulged in sexual excess became a tabetic; the other who exhausted his brain in the struggle for existence, became a general paralytic. Facts like these are of eloquent significance. The sensory neurons and posterior columns of the spinal cord appear to be specially vulnerable to certain toxic agents. Whether this can be explained on evolutionary or developmental principles; or more mechanically by peculiarities in the lymph circulation in the posterior roots and posterior columns; or by an elective affinity of certain poisons for special structures, such as nicotine for the cells of the sympathetic ganglia, is a question for the future. But degenerations similar to those of tabes have been shown to occur from poisons introduced from without or generated within, as well as in the course of cerebral tumours, in some more obscure manner.

In the great epidemic of ergotism which occurred in Hesse in 1880 Tuzek observed many cases of so-called ergotin tabes, in which, following the preliminary acute gastric and other constitutional disturbances, paraesthesiae and ataxic symptoms set in, sometimes several months after the ingestion of the poison. Occasionally there was a tendency to relapse, even when no further use was made of the poisonous grain in the dietary. These ataxic and sensory symptoms were found to coincide with degenerative changes in the posterior roots and posterior columns similar to those of tabes, differing only in their acute course and in the absence of sclerotic shrinkage. Lissauer's zones, however, remained intact, and there were no pupillary symptoms.

Somewhat similar degenerations, but less strictly confined to the posterior columns, have been observed in pellagra, endemic in certain districts, especially in Northern Italy, and due to the consumption of poisonous maize. The degenerations in this case affect not only the posterior columns, particularly that of Goll, but also the antero-lateral and pyramidal tracts. The exact nature and localization of these degenerations are, however, not as yet precisely determined.

So also in lathyrism, caused by the seeds of the *Lathyrus sativus*, degenerations occur in the spinal cord; and a large number of inorganic poisons, which specially cause peripheral neuritis, also occasionally lead to degeneration of the posterior roots and posterior columns, such as alcohol, lead, arsenic, and some others.

Many of the organisms which are the cause of certain epidemic and endemic diseases, such as those of diphtheria, beri-beri, and lepra, generate toxins which not only induce multiple neuritis, but also in some instances degeneration in the posterior roots and in their intramedullary processes and collaterals. Whether these degenerations are primary or secondary to the peripheral neuritic processes, or whether the two are simultaneous, is a question on which it is impossible at present to pronounce definitely. Further—and perhaps of greater significance—in reference to the pathogeny of tabes, it has been shown that in certain cachectic states of the system such as pernicious anaemia (Lichtheim, Minnich, etc.), and diabetes (Fischer, Williamson), probably by the autochthonous development of toxins, degenerations occur in the spinal cord, especially in the intramedullary processes of the posterior roots, and tabetic-like symptoms ensue in the form of paraesthesiae, pains, and ataxic disorders; but however much these toxic affections resemble tabes, they are in reality only pseudo-tabetic, and none of them conforms in its clinical history, course, and termination to tabes properly so-called. Though weeks, or even months, may elapse before the poisons introduced into the system, or engendered in it by the specific organisms, produce their deleterious effect on the nerves and nerve-centres, and though relapses may occur, there is a direct continuity between the introduction of the poison and the development of the symptoms, which

run their course, and cease when the virus is exhausted. In none of them do years elapse between the original infection and the commencement of the symptoms, and none has the progressive character of true tabes. In none of them do the children suffer like the juvenile tabetics, "who have their teeth set on edge because their fathers have eaten the sour grapes" of syphilis.

The similarity, however, between the degenerations caused by these poisons and those of tabes, strongly support the hypothesis, originally advanced by Strümpell, that the tabetic degeneration is due to the action of a toxin of syphilitic origin. The tabetic degeneration is, as we have seen, a parenchymatous dystrophy, and has none of the features usually considered characteristic of tertiary syphilitic lesions. There are no cellular infiltrations; no specific vascular or gummatous changes; and it does not yield to the usual antispecific remedies. Hence it has been customary to term the lesions of tabes parasymphilitic (Fournier) or metasyphilitic (Moebius) manifestations. It may be said, however, that this is a mere *petitio principii* and that we have no certain criterion of what constitutes a syphilitic lesion. The *Spirochaeta pallida* has not yet been shown to be present in tertiary syphilis. But, in any case, it would be incorrect to term a state of abiotrophy (Gowers), or defective nutrition, a positive lesion. For this reason Lesser's term, quartary syphilis, however appropriate to certain late manifestations of syphilis in the liver and other organs, is not strictly applicable to a condition in which parenchymatous dystrophy precedes the development of the sclerotic process.

The most probable pathogeny of the tabetic degeneration, therefore, is that it is the result of a toxin generated or conditioned by the syphilitic virus. But this is at present a pure hypothesis, and one which has to surmount great difficulties. Even if, like the diphtherial neurotoxin, it may be the product of the action of the specific organism on the proteids of the body, or set free in the elaboration of protective antitoxins, that it should take so long to develop, or lie dormant for so many years before exhibiting any signs of activity, is unlike anything that we know of organismal toxins. There is no parasymphilitic or metasyphilitic cerebro-spinal lymphocytosis as such, apart from the commencement of the tabetic process, and this is a fact of great importance. Dr. Purves Stewart, who has made investigations for me on this point, found that, in a series of 12 cases, the average number of lymphocytes in the centrifuge deposit of the cerebro-spinal fluid in secondary syphilis was 2.7, while in tertiary syphilis it was 0.9, or not more than the normal. The tertiary cases with active cutaneous lesions in progress had more lymphocytes than the others, but even these were less than the average of the secondary cases. The lowest count in tabes, however, was 45. Most were between 80 and 100, while the highest was 316 in the same relative field.

Even if we were to consider it possible that there might be long years of apparently normal health, in spite of the presence of some malignant toxin in the system, we have to reckon with the fact that the tabetic process, once begun, is essentially of a progressive nature, and postulates a more or less continuous generation of the poison.

In this relation mention may be made of the researches of Ford Robertson, McRae, and Shennan. These observers have found a bacillus closely resembling the Klebs-Loeffler bacillus of diphtheria in cultures from the walls of the respiratory, gastro-intestinal, and genito-urinary tracts of patients dying from general paralysis, and from the genito-urinary tracts of general paralytics and tabetics during life. Rats fed on broth cultures of this diphtheroid bacillus became ill after some weeks, and, when killed, showed in addition to signs of gastro-enteritis acute degenerative changes in the cortex cerebri resembling to some extent those of general paralysis, as well as degenerative changes in the cells of the spinal cord. These observations are of much interest, but a definite causal relationship between this bacillus and tabes or general paralysis is far from being established. For the same bacillus was found by them in the genito-urinary tract in 10 out of 14 patients suffering from forms of insanity other than general paralysis.

NOTE.—I had written this before Dr. Ford Robertson had delivered his Morisonian Lectures on The Pathology of General Paralysis, now in course of publication, and I am

indebted to him for allowing me to read the MS. of those as yet unpublished, and to examine some of his microscopical preparations. He holds that general paralysis is the result of a bacterial toxic infection from the respiratory and alimentary tracts, owing to the impairment of the resisting power of the organism by syphilis, alcoholism, or the excessive use of nitrogenous foods. The bacillus has the cultural and morphological features, and reaction to Neisser's stain, of the Klebs-Loeffler bacillus, of which it is an attenuated or more probably a special form, to which he gives the name—*Bacillus paralyticans*. The bacillus is polymorphic; and among other characters which distinguish it from the xerosis bacillus, and Hoffmann's bacillus, is its power of forming acid when cultivated in glucose broth.

The leucocytes of the blood of the general paralytic, he maintains, have a greater dissolving power over these bacilli than those of normal blood, and the serum is also more lysogenic.

These diphtheroid bacilli, more or less altered by lysogenic action, have been found by him and his colleagues in the catarrhal pneumonic foci of general paralytics, in the adventitious spaces of the cerebral vessels, and in the meshes of the pia arachnoid. They have also been found in the blood, in the centrifuge of the cerebro-spinal fluid, and in the urine of the general paralytics, especially during congestive attacks. From these fluids cultures of the bacilli have been obtained, when sufficient time has been allowed to destroy the phagocytic or lysogenic action of the blood. Rats infected or fed with cultures of these bacilli showed changes in the cerebral cortex suggestive of those of general paralysis, and similar appearances were also found in a goat which had ingested them.

He considers that in general paralysis there is a life and death struggle between the leucocytes and the diphtheroid bacilli, in which the leucocytes succumb. There is a bacillary invasion. Most of those which reach the blood are digested, but many escape from the circulation, either through the capillaries of the kidneys, or through the walls of the cerebral blood vessels into the adventitious lymph channels. The further disintegration of the bacilli in these lymph channels gives rise to a local toxic action in addition to the general toxæmia. The special cause of the cord lesion in tabes is probably the absorption of toxins from some peripheral septic focus such as the bladder.

Interesting as these views are, there are many difficulties in the way of their acceptance as a satisfactory pathogeny of tabes, cerebral or spinal. Eyre and Flashman, as the result of examination during life of the throats of large numbers of patients suffering from different forms of insanity, as well as of the tissues of a considerable number of cases *post mortem*, could not determine any greater incidence of diphtheroid organisms in the throats of the insane than of the sane, and found no evidence that *B. diphtheriae* is more common in the throats of general paralytics than of other insane patients. Of 10 cases of general paralysis examined *post mortem*, diphtheroid organisms were found in the respiratory tract in 4, and the same number in 26 cases of other forms of insanity. They were unable to trace any causal connexion between *B. diphtheriae* and general paralysis of the insane.

I have the authority of Dr. Bulloch, one of our eminent bacteriologists, for the following remarks: Diphtheroid organisms are ubiquitous both in health and disease. They may be found in the intestines, in the genito-urinary passages, in the throat; in the nasal secretions, and elsewhere in perfectly normal people, as well as in catarrhal conditions of the respiratory tracts. The characters given by Dr. Ford Robertson are not sufficient to distinguish his *Bacillus paralyticans* from other forms of diphtheroid organisms. The formation of acid in glucose broth is not special to the *Bacillus paralyticans*, for this is possessed in different degrees by various other diphtheroids.

If diphtheroid bacilli can be found in the blood films of living general paralytics, then general paralysis must be regarded not as a toxæmia, but as a form of septicaemia, ranking with relapsing fever as regards the dissemination of the organisms. If diphtheroid bacilli exist in the blood or cerebro-spinal fluid of general paralysis, they should be capable of cultivation at once without waiting for the cessation of any supposed bactericidal action, for no lysogenic action of the serum for bacilli of this group has been proved to exist.

Specimens of the cerebro-spinal fluid, blood, and urine of several cases of tabes and general paralysis have been cultivated for me by Dr. Emery of King's College Hospital, and by Dr. Bulloch, and have proved absolutely sterile. Dr. Ford Robertson emphasizes the fact that he finds bacilli particularly after so-called congestive attacks. It is not improbable, therefore, that the bacilli which he has found so largely diffused throughout the tissues in general paralysis are only the result of a terminal invasion.

In order to establish any causal relation between the *Bacillus paralyticans* and tabes, it would be necessary to show that a toxin prepared from these bacilli can produce the symptoms and *post-mortem* appearances characteristic of tabes, cerebral or spinal. Such evidence Dr. Robertson has not produced. The experiments on rats related by him are not convincing, for it has been found by other bacteriologists that rats are almost immune to the most virulent form of the Klebs-Loeffler

bacillus. In presence of these and other considerations, we may, therefore, say that the diphtheroid-bacillary origin of the tabetic toxin is at least not proven.

In the absence of any living organism capable of generating the tabetic toxin, it may be that the syphilitic virus under certain conditions so affects some viscous or gland that in time it develops, and continues to elaborate, some toxic internal secretion which exerts its noxious influence on the nervous system.

In view of the brilliant therapeutic triumphs of the present day, it is not, I think, too optimistic to hope that, with the progress of bio-chemistry, we may yet discover the source of the tabetic toxin, its nature, and the means of neutralizing it and staying its ravages. We cannot expect to repair damage already done, for intramedullary degeneration is irreparable, but we may at least succeed in arresting the disease. This not infrequently happens spontaneously; and the therapeutic measures last employed are apt to get the credit of the happy result. But beyond palliation, or the temporary relief of urgent symptoms, no remedy, chemical or physical, has as yet been discovered which materially influences the morbid process. Antispecific treatment may be of service against possible coincident tertiary lesions; or may even cure pseudo-tabetic syphilitic symptoms; but certainly ordinary anti-specific treatment exerts no beneficial influence on true tabetic degeneration, and may, if carried to extremes, prove positively harmful. If, however, we believe, as I think we must, that the syphilitic virus is the *sine qua non* in the etiology of tabes, it ought to be our endeavour to combat this with all the resources at our command.

The comparative mildness of the primary constitutional symptoms in those who ultimately become tabetic, would almost seem to indicate that when the syphilitic virus expends itself in severe primary and secondary manifestations there is less tendency to the subsequent development of the subtle poison which proves so disastrous to the nervous system.

This mildness of the primary symptoms without doubt leads in many cases to insufficient treatment. Even the most efficient antispecific treatment, however, may fail to prevent the subsequent development of tabes in certain cases. But the facts recently brought by Fournier before the Académie de Médecine in reference to the relation between general paralysis and syphilis and its treatment are worthy of careful consideration and probation.

Fournier states that of 79 cases of general paralysis coming under his own observation, in which he was able to obtain precise data as to the character and duration of the treatment to which they had been subjected, only 5 per cent. had been treated with thoroughness; while in at least 80 per cent. the antispecific treatment had been carried out in a more or less perfunctory manner. It is enough, he says, to treat constitutional syphilis for weeks or months only. Fournier contends that mercurial treatment should be administered for at least two years continuously, and intermittently for several years longer. "Treat syphilis well, and you will have few cases of general paralysis," he says. The same would hold true of spinal tabes. These views deserve the special consideration of those who have most opportunities of treating syphilis in its earlier stages. If they are substantiated, tabes both cerebral and spinal will become one of the rarer, instead of one of the most common, and, it is to be feared, increasing forms of organic nervous disease.

[The bibliography will be given at end of third lecture.]

SCHOOL HYGIENE.—On the initiative of Dr. César Roux the sanitary card has been introduced into the municipal schools of Nice. Each pupil is provided with a small book, in which are recorded all particulars as to his age, domicile, place and date of birth, successful vaccination, weight, height, chest girth, and aptitude for sports. The booklet also contains elementary notions of hygiene, advice for the correction of wrong positions in reading and writing, and on the dangers of alcohol and tobacco. A card attached to the book is handed to the medical inspector, who is thus enabled to advise the parents of the child as to the care to be exercised in his bringing-up. A special advantage of the sanitary card is that, by means of the observations recorded thereon, children whom the teacher might otherwise punish for idleness or misbehaviour are shown to be suffering from adenoid growths, nervous troubles, or hereditary taint of one kind or another.

MEMORANDA: MEDICAL, SURGICAL, OBSTETRICAL.

FETUS ANENCEPHALUS: TWO CASES WITHIN THREE MONTHS.

It is very unusual to meet with two cases of anencephalic monsters in private practice within three months, and

therefore I think it worth while to forward photographs of two instances of this type of monstrosity which came under my observation last year within that limited period. The smaller fetus was born prematurely at the sixth month; the larger

specimen nearly caused the mother's death. After consultation with two other practitioners premature labour was induced at the eighth month

owing to very severe albuminuria and general dropsy. The patient made an excellent and speedy recovery, and was able to attend to her duties in ten days. In the

larger specimen there was a complete absence of the posterior bones of the skull, and the spinal column was widely open down to about the seventh dorsal vertebra.

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CALCIUM SALTS IN CHILBLAINS.

THE name "erythema pernio" at once helps us to form some idea of the nature of the disease. It is associated with such general disorders as urticaria and erythema nodosum on the one hand, and with lupus erythematosus on the other. On both sides, therefore, it is associated with general diseases due to some poison which has the effect of producing characteristic local lesions.

Yet, notwithstanding such associations, it is usual to consider chilblains a purely local affection, aggravated, undoubtedly, or contributed to, by such general conditions as a "weak circulation" or "want of arterial tone." Based on such a pathology, the treatment has been to give cod-liver oil, iron, digitalis, and suchlike drugs internally, the external treatment being satisfied with painting iodine "till they break," and then applying fomentations.

The close association of chilblains with such general diseases as those above mentioned, and their appearance in persons who have tuberculous tendencies, suggested to me that the cause must be one affecting the body generally, but depending on climatic conditions for the characteristic manifestation. Cardiac and arterial tonics have been in the majority of cases so singularly unsuccessful, that I thought it advisable to administer drugs which would alter the character or quality of the blood, especially as regards its exudation properties.

Chilblains are particularly prone to occur in certain classes of the community, such as shop girls who tend to neglect themselves, and children in orphanages, and institutions for the deaf and dumb. The neglect referred to in connexion with shop girls shows its results very often in the form of anaemia, again helping to emphasize the fact that it is to the blood that attention should be directed.

The drug which seemed to me the most appropriate was calcium chloride, experiments having proved that the coagulability of the blood was increased by its administration. In haemophilia and in purpura this drug has been found useful, and I determined to try its effect on chilblains. I gave it in 10 to 15 gr. doses with liquorice