Now, how is such an extraordinary course of the malady to be understood? Has advanced age anything to do with it? Is it due to a mixed infection—syphilis combined with the poison of influenza—in a system where malarial fever may have left its traces? It is well known that erysipelas, scarlatina, typhoid fever, when contracted during the time that the constitution is under the influence of syphilis, can make the symptoms of the latter disappear. The syphilitic symptoms vanish although the antisyphilitic treatment is stopped, in most cases to reappear after the cure of the intercurrent fever, in rare cases as it seems for ever.

[In this regard Dr. Thissen says: In 1882, while resident physician at the Luisinhospital of Aachen, I attended under Dr. Brandt's direction a gentleman who had come to Aachen for treatment suffering from secondaries. During his cure he was seized with typhoid fever, and had to be received in the hospital. When I first saw him his body was covered with syphilitic rash and his throat was ulcerated. The antisyphilitic treatment was stopped at once when typhoid fever was ascertained, and the patient was treated by antipyretics and cold baths. I was struck to see the syphilitic symptoms vanish almost like magic, never to reappear.]

Perhaps we may express our theory in this way. On the one hand we have erysipelas, scarlatina, typhoid fever causing the syphilitic symptoms to disappear. On the other hand there is some other complicating organic virus influencing the syphilitic one so as to give its action a turn highly different from the rule and in a mischievous direction. In fact, it seems difficult otherwise to explain our case. It will be remembered that at the last International Congress of Dermatology in London, when Tarnowsky exhibited his excellent wax models of cases of what is called malignant syphilis, the question was discussed whether such cases might not be the consequence of more than one organic virus, for instance, the septic virus acting together with the syphilitic virus, as has been frequently observed in a similar way in diphtheria. The latter opinion was maintained in the discussion by Dr. Brandis, who stated that not a small number of such observations had fallen under his observation during his long practice. He also pointed out that according to his experience what is called malignant syphilis always, or at least generally, occurs in the early stage of the malady, and that it is accompanied by high fever, which very often bears the character of pyæmic fever. We may repeat that our case seems to support such views.

The second point of importance to which we should like to draw attention is the efficacy of energetic specific treatment. It was only too natural that it should have come late, very late indeed—not before seven months after the infection—syphilitic symptoms having been undiagnosed all that time. Whatever could have been done in order to cure malarial fever—namely, quinine and change of climate—had been tried in vain, and the syphilitic virus was allowed to do all the mischief before it was recognised. The first specific treatment—potassium iodide and grey powder—showed its good effect almost immediately; however, these remedies proved insufficient afterwards, and the disease went on in its course of destruction. One year after infection tracheotomy was necessary. At last the patient was sent to Aachen, where he underwent the inunction cure for more than two months, which in combination with the use of the Aachen waters is well known to be one of the strongest, if not the most powerful, antidote to the syphilitic poison.

Lastly, we may be allowed to add a few remarks regarding the local treatment of the laryngeal complaint. Although a piece of necrotic cartilage had been previously expectorated, from which fact it was almost certain that more necrotic cartilage had to come away, the swelling of the laryngeal mucous membrane rendered examination of the larynx very difficult; and it took Dr. Schmidthuyssen some time before he discovered where the necrotic part was located. Another difficulty was to prevent the latter from slipping down into the trachea. For that purpose Dr. Schmidthuyssen used an instrument the shape of which was that of a hook. The handle is short and concave for the thumb of the left hand, by which, when the longer part of the instrument is introduced through the tracheal fistula into the trachea, it is held *in situ*. The longer part is also a little concave, and blunt at the end. The latter part of it is sieve-like, so as to allow free respiration

below, while it leaves sufficient room above for a small, sharp hook, which is passed through the tracheal fistula into the larynx in order to loosen the necrotic piece. A fit of coughing facilitated the elimination after several attempts with a sharp hook. Six days later another necrotic piece was removed through the mouth. It was most gratifying to observe how soon after the operation all swelling subsided, so that the patient could be left without cannula, and also how very little remains of the complaint could be traced on the patient's return to Aachen this year.

A NOTE ON THE MICROSCOPIC APPEARANCES OF THE SPINAL CORD IN TETANUS.

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THE importance of the examination of the spinal cord in tetanus has for long been recognised, and many careful and exhaustive observations as to its microscopic appearances have been put on record. But these observations by no means homologue one another, and, indeed, the variety of changes described as occurring in the cord in this disease is very considerable. The relation, too, of many of these changes to one another is difficult of interpretation; and the conclusion that presents itself to one's mind is that the essential changes to be found in the cord in tetanus have not yet been clearly and satisfactorily laid down. The subject is one of great interest, and I think I need urge no excuse for adding to its literature the record of three cases I recently investigated.

In each of these cases the cord was fixed and hardened in formol for twenty-four hours, and then placed in alcohol till ready for use. Sections were stained with methyl blue, with fuchsin and with congo red, all according to Nissl's method.¹ Two other spinal cords, considered as perfectly healthy, were fixed and stained in a precisely similar manner, and used as standards of comparison.

The first case was a boy aged 12. It was a typical case of tetanus supervening upon a septic wound of the subcutaneous tissue of the thigh, and the whole duration was not more than twenty-four hours from the onset of the convulsions. To the naked eye the necropsy was practically negative, and if the spinal cord differed from the normal it was in the presence of a slight hyperæmia of its posterior surface. The tetani bacillus was cultivated from the wound both before and after death. No antitoxin was administered, as the patient died a short time after admission to the hospital.

Sections from five levels of the cord, from three levels of the medulla, and from two of the pons were examined microscopically. All these showed a marked dilatation of the vessels, and this was more noticeable in the grey matter than in the white. In nearly all the sections minute hæmorrhages were to be made out. The hyperæmia and hæmorrhages, however, seemed less intense in the pons, medulla, and upper cervical than in the lumbar region of the cord. No inflammatory corpuscles were to be seen, and the only exudation present was the red blood corpuscles of the hæmorrhages. The nuclei of the neuroglia were not increased unless round the central canal, where they were collected in considerable numbers. There was no sclerosis in any of the white columns. The ganglion cells were well formed, showing no deficiency in processes, and in almost all nucleus and nucleolus were well defined. Very few of the cells contained any considerable amount of pigment. Their plasma, however, was different from that seen in the sections of the normal cords. Instead of the triangular-shaped collections of deeper stained granules in the more faintly stained ground-work of the cell the whole plasma had a more or less uniform staining and showed none of this differentiation of its substance. It was only in the upper parts of the medulla and in the pons that the ganglion cells in this respect presented a normal appearance.

The second case, a man, aged 45, was successfully treated by means of antitoxin. He subsequently developed exfoliative dermatitis, to which he succumbed after five days' illness, and fifteen days after the onset of the tetanoid con-

vulsions. No attempt was made to cultivate the bacillus of tetanus, but the diagnosis seemed undoubted. At the *post-mortem* examination considerable congestion of the posterior surface of the cord was noted.

Sections from several portions of the cervical, dorsal, and lumbar regions were examined. No hæmorrhages could be seen in any of these, and the dilatation of the vessels, which was most marked in the lumbar region, was not nearly so prominent as in Case I. Although there was a certain degree of hyperæmia, it was not much more than is seen in a normal cord fixed in formol. No inflammatory corpuscles were present in the neighbourhood of the vessels, and there was no sclerosis of any of the white fibres. Many nuclei in this case also were collected round the central canal. The ganglion cells presented an appearance similar to that in the last case. They were well formed; many of them contained a large amount of yellow pigment, but in none were the various elements of the plasma defined, the whole of it presenting a more or less uniform, almost opaque, appearance.

The third case was a child, aged 4. Here the convulsions lasted for twenty-four hours, and seemed quite unaffected by an injection of antitoxin given four and a-half hours before death. In this case also the membranes of the cord were, at the *post-mortem* examination, found much congested. It is further to be noted that the lower lobe of the left lung was in the condition of red hepatisation. The bacillus tetani was cultivated from the wound.

As in the last case various portions of the cord were examined microscopically. In this one, however, the appearances were practically normal. The vessels seemed dilated, but not so much as in Case II, and really not more so than in the normal cords which were preserved and stained in a similar manner. There were no hæmorrhages and no increase of neuroglia corpuscles unless round the central canal. The cells lining the central canal, however, seemed to have undergone some proliferation, and the canal itself had a wider lumen than is usually seen. The ganglion cells were perfectly normal, their plasma showing very distinctly the differentiation into deeper stained "triangles" and the lighter coloured ground-substance.

What conclusion, then, is to be drawn from the examination of these three cords? Two of the cases died within twenty-four hours. The cord of the former (Case I) showed a well-marked hyperæmia with hæmorrhages, as well as an alteration in its ganglion cells. The other (Case III) we may consider as being practically normal. The remaining one (Case II) also presented a hyperæmia, though in a much less degree. Its ganglion cells, too, showed changes. But it is to be remembered that this case lived for about a fortnight after recovery from the tetanus, and it is therefore impossible to say to what extent the cord may have recovered in that time.

What is the meaning of the hyperæmia—let us say in two of the cases—and the absence of it in the third? A hyperæmia of the cord seems to be the microscopic appearance most constantly met with in the cases of tetanus hitherto reported. But is this not just the same hyperæmia as is seen more less in all the tissues of the body in this disease? And may it not therefore be considered simply as a congestion resulting from spasm of the muscles of respiration? The absence of the hyperæmia in the third case is difficult to explain. Possibly the child died from syncope rather than from asphyxia, and the congestion in the left lung, too, may have lessened that of the spinal cord. Be this as it may, the hyperæmia of the spinal cord in tetanus is, I take it, to be regarded as a result of spasm in the muscles of respiration, and not as being directly due to the poison of tetanus. And it is to be remembered that a hyperæmia of the spinal cord is found in most deaths from convulsions whatever be their nature. In considering the pathology of tetanus then, we cannot attach any special importance to the hyperæmia so often met with in the spinal cord.

Besides this dilatation of the vessels, signs of irritation have been found in the presence of leucocytes round these vessels, and in what have been thought to be fluid effusions, causing a disintegration of the neighbouring nervous tissue. But as these leucocytes and fluid effusions are by no means seen in all the cases reported—they were certainly absent in our three cases—we cannot give them much consideration.

The same applies in even a greater degree to the yellow pigment sometimes present in the ganglion cells in large amount, and sometimes found to be almost entirely wanting, for this variation I find occurs in the ganglion cells of cords considered as being quite healthy.

Coming, then, to the changes we noted in the plasma of the ganglion cells of our first two cases, we must consider what these changes mean, and if they have any bearing on the pathology of tetanus. We noted that in Cases I and II the plasma of the ganglion cells no longer showed its normal differentiation, but stained with a uniform intensity throughout. It might be urged that this appearance was merely the result of the method of staining. I do not think this can be so, for very many sections from all the cases were examined. Those from the same cord were stained at different times with different stains and in different degrees of intensity. Yet in all the sections from the first two cases the plasma stained with uniformity throughout, while in the third case and in the two normal cords examined the ganglion cells showed the normal differentiation of their substance most clearly.

Now this change in the ganglion cells of the first two cases is of considerable interest in view of the results obtained by Marinesco² on injecting guinea-pigs with the tetanus toxin. He found on examining these animals after death not only hæmorrhages into the grey matter of the cord, but also alterations in the ganglion cells such as I have just described. He found these cells had become smaller than normal, taking the form of a tip-cat in place of being polygonal. The cell plasma was granular and did not show the differentiation on staining in the way the normal cells do. The cell nucleus was altered only in the cases where the convulsions had lasted for some considerable time, and then the nucleus was noted to be ill-defined and to stain more intensely than usual. In cases of chronic tetanus a degeneration of the medullated nerve fibres could be made out.

What explanation then is to be given of this altered appearance in the ganglion cells found by Marinesco in his guinea-pigs, and by me in two of the cases we are at present considering? The only suggestion I can offer is that these cells are in a state of commencing degeneration. It has been shown³ that when a nerve fibre is cut its corresponding ganglion cell degenerates, owing presumably to loss of function. The first change to be observed in this process of degeneration is a disappearance of the deeper stained "triangles" of the plasma, these seeming to become dissolved in their lighter coloured matrix. This is exactly the appearance presented by the ganglion cells in our first two cases, and the suggestion therefore which comes to one's mind is that these cells were commencing to undergo degeneration. What exactly this degeneration is due to I cannot pretend to know. In all probability the tetanus toxin acts as a poison on the ganglion cell and so causes its death. But it may be that the toxin is only a powerful stimulant, and that the degeneration of the cell is due to over-use, just in the same way as certain other cells in the body atrophy from over-use. It might be contended that the ganglion cells in Case II should have recovered their normal appearance in the fortnight the patient lived after the tetanoid convulsions ceased. But it is to be remembered that during much of this time the tissues generally were contending with another and very severe disease. The conditions therefore were not those favourable to regeneration of cells in any part of the body.

A difficulty remains in there being no degeneration of the ganglion cells in Case III. To this I can give no explanation.

I can therefore draw no conclusion from the examination of these three cases, especially seeing the third is a negation to the other two. The cases, nevertheless, seem worth reporting, if it only be to draw attention to the importance of noting the minute changes which may be found in the plasma of the spinal ganglion cells. And I may add that for this purpose Nissl's method of staining is, as far as I know, the only one of any use.

In conclusion, I wish to acknowledge the kindness of Dr. Lindsay Steven, by whose permission I publish Case I. The sections were cut in his laboratory; these he has examined, and he verifies my observations. I am also much indebted to Dr. Workman for the other two cords, and for permission to publish the results of their investigation.

REFERENCES.

¹ Colman, *Section-cutting and Staining*, p. 101; Lee, *Microtomists' Vademecum*, p. 380. ² Marinesco, communicated to the Société de Biologie, reported in *Gaz. Méd. de Paris*, July 11th, 1896. ³ Ballet, *Le Progrès Médical*, May 16th and June 27th, 1896.

CERTAIN POINTS IN THE CAUSATION AND TREATMENT OF SPINAL CURVATURE.

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II.

In my previous paper on this subject¹ I referred at some length to a cause of lateral curvature which in 1895² I had briefly mentioned under the name "amesial pelvis." One of the illustrations given (Fig. 2) was that of a young girl who

Fig. 5.—Scoliosis with pelvis amesial to right. Note.—The edge of the plate is in the same parallel; the white line of tape has inadvertently been cut away.

Fig. 6.—Girl with slight scoliosis, pelvis amesial. Note.—In this figure the perpendicular has been drawn a little too much to the right.

manifested this condition in exceptional severity; but it must not be supposed that amesiality in somewhat lesser degree is rare. On the contrary, it is very common as a cause of lateral curvature and of failure in such treatment as is directed to the spine only, ignoring the malposture of the lower parts of the body. To those, therefore, who have to do with spinal deformities the matter is of great importance, but has escaped notice because the figure is rarely uncovered lower than the level of the hips, the rectitude of lower limbs and pelvis being taken for granted—that is, the middle of the latter, most clearly indicated in external marking by the intergluteal fold, is assumed to lie in the middle line, while the lower limbs, or rather the centre of the space between them, is considered to be perpendicular. When about ten years ago my then newly-invented scoliometer³ revealed that herein lay a noteworthy fallacy, I instituted a more extensive series of observations, and took, as opportunity offered, photographs of patients of various age and sex. A very restricted number of them are reproduced here, but they suffice to show the condition insisted on. It is, however, first necessary to say how a true horizontal,

the white line by the patient's heels, was obtained. A piece of tape was secured to the floor, and the centre marked by a carpet pin, at each side of which even lengths—say two-foot—were accurately measured. The middle of a cord was held at the centre of the camera lens; here a knot was tied, from which absolutely equal lengths of the string were cut and the ends secured to the two-foot marks by driving carpet pins through string and tape into the floor. Thus was procured an exact isosceles triangle, the apex of which was at the lens, while the mid-point of the base was between the patient's heels. An imaginary line therefore drawn from the centre of the lens to this mid-point of the base formed with the tape exact right angles. Furthermore, in order to avoid error arising from minuteness of image two of the figures (5 and 6) were enlarged in printing. On these 9-inch images perpendiculars were drawn by means of an accurate steel square, and from these points photographic negatives were taken. The perpendiculars on the other two negatives were cut with a sharp knife through the gelatine of the plate, but their accuracy was verified also on enlarged images.

Fig. 7.—Child with markedly amesial pelvis and incipient scoliosis.

Fig. 8.—Amesial pelvis. Mode of investigation.

In estimating the variation it must be remembered that these figures are $3\frac{1}{2}$ inches long. The man stands 5 ft. 10 $\frac{1}{2}$ in., the girl 5 ft. 10 in., so that $\frac{1}{4}$ inch on the plate represents 1 inch on the body, or $\frac{1}{8}$ inch about the aberration in these two illustrations means $1\frac{1}{2}$ inch of real displacement. Moreover, if the rima natium is $1\frac{1}{2}$ inch from the perpendicular there is that amount of pelvis too much on the one side and the same too little on the other, therefore that bone as a whole lies $2\frac{1}{2}$ inches out of the straight.

My room is so arranged that when I have fully explained how my patient is to stand I leave until all is prepared. I then enter from behind, expose the plate, and quit the room again while garments are resumed, without ever going in front of the patient; but save in the case of young children and for investigation sake entire nudity is unnecessary.