

irritants. There is some, but not very much, inflammatory action—no implication of the deep tissues; and local remedies suffice for the cure. In other cases, several parts of the body are affected at one time; the local mischief is severe; there is much heat, and itching or burning sensations, perhaps pain; the cellular tissue beneath the skin is implicated in the mischief; the discharge is not only free, but irritative to the parts around, and the general system sympathises, whilst there may be distinct ill health, dyspepsia, a gouty diathesis, and the like. In a third class of cases, occurring in young and lymphatic children, there is very free and early formation of pus, out of proportion to the degree of inflammation as compared with the last form of eczema, indicating a pyogenic habit of body well marked. There is also free crusting, and often distinct evidence of the scrofulous diathesis in the family history. Now though these now and then run the one into the other—and there are no hard and fast boundary-lines in medicine,—yet on the whole the distinctions are clear, and the terms simplex, rubrum, and impetiginodes accurately portray these clinical varieties.

Pray let us simplify, as we may do, the whole question of eczema, and especially prime down the prolific nomenclature with which it has been associated. Some authorities have managed to describe twenty and more varieties, whilst three are alone necessary. Whilst we accept them as best in harmony with clinical observation, and as indicating most conveniently appropriate treatment, we should not forget that each must have its *stages*; that these stages may, under different circumstances, be better marked at one than another time, according to the age of disease at which the physician sees it, and the intensity of its expression; and that our conception of eczema must be, not piecemeal, but one inclusive of all its stages as one whole affair. If we speak of stages, let us call them stages, and be chary of the abuse of the term variety.

Whether right or wrong in my opinions, at least it is after very considerable thought, and much canvassing in the presence of actual disease, that I have arrived at the conclusion that Willan's arrangement and general description of eczema is the best yet written; and that, developed, and but slightly modified in accordance with the progress of pathological science, it is the truest to nature. It were unaccountable that Englishmen are so ready to let the views of our illustrious countryman, who died in 1812, be misinterpreted without a single protest, and the credit of establishing a true clinical reading of eczema be taken from one of themselves, but on the ground of the ignorance which prevails in regard to what he actually wrote.

ON THE OPHTHALMOSCOPIC SIGNS OF SPINAL DISEASE.

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THE use of the ophthalmoscope as an aid in the diagnosis of encephalic disease is gaining ground quickly, and attention is now being given also to its indications in diseases of the spine.

It is on this latter subject that I propose to make a few remarks. I fear I have little to offer to the reader in the shape of positive knowledge, or, indeed, of strongly supported conjecture; but I think a review of the facts as they stand at present, though of but little permanent value, may nevertheless so direct discussion and observation as to lead to the establishment of something definite. I am quite sure that in this way we shall arrive at facts which will be valuable in diagnosis, and which will have a remarkable value in pathology. In the meantime it would be, perhaps, an error to withhold my own observations from the printer because there is much which still perplexes me, and much more which is incapable of proof, if not of probable interpretation. In the present study, as in any other, it is easy to welcome new facts which will submit themselves to a favourite hypothesis; but if we resist this first temptation,

and pursue our observations with industry and caution, we are presently made aware that the new facts are far more difficult of study than we had supposed, they turn out to be far less simple and uniform than at first they seemed, and they begin to refuse the shelter of the favourite hypothesis which appeared so convenient for them.

The facts I have to deal with here will be best discussed in the following order:—

Firstly. Do disturbances of the optic nerve and retina commonly follow spinal mischief?

Secondly. If so, then what kind of disturbances are they? And

Thirdly. What reason or reasons can we assign for their occurrence?

Firstly, then, are the accounts of disturbance of the inner eye secondary to spinal disturbance to be trusted? Of this there is little doubt: it is tolerably certain that disturbance of the optic disc and its neighbourhood is seen to follow disturbance of the spine with sufficient frequency and uniformity to establish the probability of a causal relation between the two events.

I have myself examined, from this point of view, 30 well-marked cases of *spinal injury*, and in 8 of these I found secondary disturbance within the eye. Of these cases, 17 were severe injuries which proved fatal within a few weeks, and in none of these did changes appear in the eye; the remaining 13 cases were of chronic spinal disease following accidents of less severity, and it was amongst these 13 that I discovered the 8 cases of sympathetic disorder in the eye. Of *acute myelitis* I have examined 5 cases, and in 1 only did eye disorder supervene. This remarkable case was of very long duration, and was followed by partial recovery; in it sympathetic disorder of the eye came on many weeks (twelve or thirteen weeks at least) after the subsidence of the acuter symptoms. The myelitis in all these 5 cases was in the lower dorsal or lumbar region. Of *chronic degenerations of the cord*, exclusive of locomotor ataxy, I have records of 9 cases. In 5 of these marked changes in the eye appeared.* In *locomotor ataxy* the occurrence of affection of the optic nerve is so well known that I think it scarcely worth while to sum up my notes of this disease. I wish now to make use of the three following conclusions in particular which my researches seem to indicate:—

1. That changes at the back of the eye do not infrequently follow spinal disease.

2. That these changes do not become established in the cases which run a short course, but they slowly supervene in the course of weeks or months in more chronic cases.

3. That in spinal disease arising from injury, the higher the seat of the injury the sooner are there changes in the eye. Of this last conclusion I have satisfied myself after a careful observation of well-marked cases. I have found that the optic changes follow injury to the spine more rapidly if, for example, the injury be in the upper cervical region than if it be in the lower cervical or in the upper dorsal region. One of the best-marked cases of eye disorder with spinal injury that has occurred in my own practice was in a man who had suffered an injury to the spine in the region of the atlas and third cervical vertebra. The injury was set up by a sudden twist of the head backwards and to the left. In him changes at the back of the eye appeared very quickly and decisively. The patient is still living, but it was clear from the symptoms that the lower cervical and upper dorsal regions were unaffected.

Having seen, then, that there are changes in the eye symptomatic of spinal disease, our second inquiry is, Of what kind are these changes? Confining ourselves to the optic nerve and the retina with their vessels, and omitting all reference to injection of the conjunctiva, or the state of the pupil, what kind of changes are dependent upon disturbance of the spine? I find that they may be well classified under two heads:—(1) Simple or primary atrophy of the optic nerve, sometimes accompanied at first by that slight hyperæmia and inactive proliferation which make up the state I have called chronic neuritis. This sort of change I have never found as a result of spinal injuries, but I have often met with it in chronic degeneration of the cord and in locomotor ataxy. (2) A somewhat characteristic hyperæmic change which I have not seen in chronic degeneration or in

* The above numbers refer only to cases which I have watched and noted carefully during a long period. A very much larger number of cases have come before me in a more or less passing way.

locomotor ataxy, but in cases of injury to the spine only. The retinal arteries do not dilate, but become indistinguishable; while the veins begin to swell and become somewhat dark and tortuous. The disc then becomes uniformly reddened, and its borders are lost, the redness or pinkness commencing with increased fine vascularity at the inner border, and which thence invades the white centre and the rest, so that the disc is obscured or its situation known only by the convergence of the vessels.* In many cases, rather than redness, I have observed a delicate pink—pink which sometimes passes into a daffodil colour. In one case, in particular—a railway accident,—which I examined in consultation with my friend and colleague, Mr. Teale, this daffodil colour of the whole field was very curious; no disc was to be distinguished, but the dark vessels stood out in beautiful relief.† The other eye presented the more common appearances of hyperæmia and serous effusion, with slight swelling. It is to be remarked that this state is generally or always of long duration; it passes very slowly up to its full development, and then shows a disposition to end in resolution rather than in atrophy. In those cases which I have been able to watch diligently for many months the pinkness seems slowly to have receded, leaving an indistinct but not very abnormal disc behind. Sometimes the sight suffers a good deal in these cases, sometimes but little or scarcely at all. I have never seen true optic neuritis with active proliferation as a sequel of spinal disease.

The third and most difficult inquiry now remains: What is the reason of the occurrence of these symptomatic changes—what are the processes which, following the changes in the spine and preceding the changes in the eye, link the two events together in the chain of causation? One answer has been lately offered to this question by a distinguished physiologist, Mr. Wharton Jones. His argument is, that when the cord is injured, the sympathetic nerve or its origins are involved; and that, as the sympathetic nerves govern bloodvessels and bloodvessels govern nutrition, therefore the changes in the nutrition of the eye are due to irritation of the sympathetic, which cuts off arterial blood from the optic nerve, or to the palsy of it, which deluges the nerve with blood. Mr. Jones, indeed, speaks as if dilatation of the arteries at and about the disc were a matter of direct observation. I can only say, after some experience with the ophthalmoscope in cerebro-spinal diseases, that this phenomenon has hitherto escaped me. A really satisfactory explanation of the concurrence of spinal and ophthalmic disorders cannot, in truth, be given until a far greater number of observers have been at work, and until careful autopsies have been made in such cases, with minute examination of the nervous tracts and centres. Meanwhile no doubt we must deal more or less in conjecture. The objections to the sympathetic-nerve theory, however, are manifold. The theory is not a new one, and in my lectures on the ophthalmoscopic symptoms of cerebro-spinal diseases I have already pointed out objections which seem fatal to it. In the first place, to call up the sympathetic system is to call up too potent an agency for the pressing difficulty. Are we to suppose that the irritated sympathetic causes the destruction of all connected parts; or that it starves the optic nerve by preference, while it leaves all other parts in its district unaffected? Or can a palsied sympathetic be the ruin of the optic disc, when its effects are unseen in the pupil, unseen in the conjunctiva, unseen in the ear and cheek? Again, it is a matter of verified observation, in numerous cases in which there have been most obvious signs of a palsied sympathetic in the ear, face, and outer eye, that in these very cases the back of the eye has been found unchanged. Such is the teaching of Dr. Wm. Ogle's case read before the Medical and Chirurgical Society on the 23rd March, 1869; and I have an equally instructive case under my own care at present. In this little boy, a blow upon the nape has set up "strumous" mischief in the cervical portion of the spinal column, with consequent palsy of the arms and legs. During the last few weeks, owing, no doubt, to a lateral extension of the mischief, the left sympathetic in the neck has also become involved, and we have the well-known signs in the left face—namely, narrowed

palpebral aperture, injected conjunctiva, undilatable pupil, flushed cheek and ear, and temperature of the cheek ranging from 5° to 9° above the right cheek, except during a febrile access, when this difference ceases or is diminished. Now in this patient the symptoms of concurrent disorder of the optic nerve and retina were observed in both eyes many weeks before the affection of the cervical sympathetic occurred; the changes in the eye were of the second kind mentioned above—namely, hyperæmia with serous exudation; and there has been no change in the left disc, or in either disc, since the affection of the sympathetic.

Again, atrophy of the optic nerve, with or without chronic neuritis, is very different in its onset and in its course from the injected fundus; it shows a very different process, and it accompanies very different kinds of spinal disease: the presumption therefore is that one explanation will not serve for the two sets of phenomena. Moreover the atrophy, sometimes with chronic neuritis, is commonly met with in locomotor ataxy, and in degenerative conditions of the cord like unto it; but the part of the cord affected in these cases is often far away from the connexions of the cervical sympathetic; and we know, in addition, that while in locomotor ataxy the degeneration destroys the posterior roots, yet it invariably leaves their ganglia whole. These objections, not only taken together but also taken singly, are at least considerable, and in my opinion are fatal to a belief in the sympathetic nerve as the cause of those secondary disorders of the eye which we are discussing.

It is less easy to undertake to say what are the causes of disorder of the optic nerve and retina in spinal affections; I shall try, however, to find out in what direction the facts themselves seem to lie.

It is clear, first of all, that we have to do with two distinct kinds of consecutive disorder, and it is probable that they arise from distinct causes. Again, these changes are not peculiar to cases of spinal disease, but they are seen in encephalic disorders also; and, in default of evidence to the contrary, we must assume that their causation is identical or similar in the two cases. Now, this kind of hyperæmia, with serous exudation, when occurring in encephalic disorders, is, so far as my experience goes, very commonly associated with meningitis of the base; while atrophy or chronic neuritis is either not associated with meningitis, or, if associated with it, is clearly due to other causes—in particular, to disease of the encephalic vessels, to degeneration of the optic fibres or centres, to disseminate sclerosis, or to severance of the continuity of the encephalic optic fibres by pressure, local neuritis, and the like. Hence my former supposition, that the two kinds of change have different causes, is supported by my experience of their causation when dependent on encephalic conditions. Again, as I have said, this hyperæmic state seems to be less a destruction of the nerve than a protracted interference with its vascularity, and this state occurs rather with injuries of the spine than with chronic degenerations of the cord. In these latter cases, when any changes occur they appear not to be of the nature of a transient interference, but of an essential destruction. These facts seem to support the foregoing: injuries to the spine are very commonly followed by meningitis of a subacute character, while slow degenerations of the cord itself are either unattended by meningitis, or the meningitis is a mere local thickening not likely to spread.

In default of a series of autopsies, then, we seem to be led towards the conjecture that hyperæmia of the back of the eye, following injury to the spine, is probably dependent upon a greater or less extension of the meningeal irritation up to the base of the brain. Now, have we any reason to suppose that spinal meningitis does creep up into the encephalon? We have: for, setting aside the curious head symptoms such patients often present, here the actual demonstration of autopsy comes to aid us. It is tolerably well known to careful pathologists that encephalic meningitis is a very common accompaniment of spinal meningitis. I am glad to be able to enlist Mr. Wharton Jones on my own side in this, who makes the same statement himself on the authorities of Ollivier and Abercrombie. It is scarcely needful to point out that if this explanation of an ascending meningitis be the correct one, it accords with my observation, stated above, that, in general, the higher the injury to the spine the sooner the affection of the eye.

Finally, we have learnt, from our experience of encephalic diseases, to attribute atrophy of the discs to severance of the

* I need not say that the disc has in reality no borders, but only apparent borders, which are readily blotted out by any loss of transparency in the retina.

† I think these colours are due to the loss of retinal transparency and the blending of its acquired reflections with those of the coats behind it. The state is, I believe, an incomplete or a receding ischæmia papillæ.

optic nerve-fibres, to sclerosis in patches, or to travelling degenerations, rather than to meningitis. Very commonly it is due to what, for brevity's sake, we may call Wallerism, from the well-known experiments of Waller upon the travelling degenerations of nervous fibres. Now, as I have said, atrophy of the discs is seen, not in injuries of the spine, but in slow degeneration of the cord—in cases, that is, where meningitis is usually absent or inactive; and it is seen most frequently by far in that degeneration of the cord called sclerosis of the posterior columns.

Supposing, therefore, that atrophy of the disc in encephalic disease is due generally to travelling degeneration, this supposition, in the case of spinal optic atrophy also, is supported by the remarkable fact that it occurs especially with degeneration of that part of the cord—the posterior columns—which tends to travel towards the encephalon, and not towards the periphery. Thus we are led to conjecture that the optic atrophy is, in some obscure way, the result of the propagation of the destructive change upwards; and that in all cases of degeneration of the cord accompanied by atrophy of the optic nerves the degeneration includes the posterior columns, and climbs upward by means of these. Can we get any farther? Perhaps we may venture upon another step in the dark. If wasting of the posterior columns creeps upwards, it creeps up to the cerebellum. Now we know that diseases of the cerebellum are very commonly associated with atrophy of the optic nerves; and this, as one or two autopsies appear to show, seems frequently to be by means of the processus cerebelli ad testes. It is certain, at any rate, that optic atrophy occurs not only with tumours of the cerebellum, but with mere wasting diseases of that organ; so that creeping degeneration, and not pressure alone, may be the agent of destruction.

Finally, I think it not unlikely that the curious tendency seen in some states of the nervous centres to sclerotic degeneration in patches may in many cases account for symptomatic nerve-atrophy. It may be that in these states the optic nerves, on account of their vascularity and rich connective tissue, are always among the first portions to suffer, and to suffer independently of the affection of other parts—independently, that is, of any mechanical or physiological connexion, and in virtue only of an affinity in structure.

There are sad gaps, I know only too well, in these reasonings; and gaps there must be until many careful autopsies have been made, and the parts microscopically examined from the present point of view. I am disposed to think, however, that the considerations I have ventured to place before the profession, though in many parts conjectural, are yet not without interest, and not without promise.*

Leeds, Jan. 1870.

ON

THE VALUE OF PERINEAL EXTENSION IN FRACTURES OF THE FEMUR.

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It is well known that in many cases of fracture of the shaft of the thigh-bone it is difficult to maintain or to keep up accurate coaptation of the broken ends of the femur, in consequence of the rotation outwards of the upper fragments by means of the gemelli, obturators, and other external rotators of the thigh, and the tilting upwards by the action of the psoas and iliacus muscles, no longer balanced by the opposing weight of the limb. Extension, too, can best be obtained only indirectly by means of the perineal band. It becomes a question how far it is judicious to keep up daily-renewed extension of the limb by this means.

Great difference of opinion appears to exist on this subject, as far as I have been able to judge from the remarks made by the surgeons whom I have had the opportunity of consulting on the subject. Some proceed at once to extend the

limb and tighten the perineal band whenever they suspect the slightest shortening, and this perhaps repeated daily, with all the force that is necessary to rectify the displacement, until the patient, becoming chafed by the pressure of the splint and bandages, pleads for some less severe treatment. Is not such disturbance of the limb unnecessary, and more likely to aggravate the mischief which it is desired to overcome? The muscles, if undisturbed, will, in a day or two after the injury, return to a quiet, relaxed state, very favourable for promoting the union of the broken bones in good position. Any disturbance is sure to excite violent contraction of the muscles and consequent shortening of the limb. Extension must disturb the processes of union, for the muscles are at once excited to contract, to oppose all attempts at interfering with the steady progress towards consolidation of the broken bones. When the muscles are thus kept at a state of tension any slackening of the bandage must lead to further shortening of the limb. The perineal band is of great service for keeping up the extension which was first applied, and for supporting the splint. Probably the long splint, folded in a sheet passed round the limb, the free edge of which is fastened along the outside of the splint with pins, will keep up a more uniform and equable pressure and extension of the limb. By carefully watching the patient, any loosening of the perineal bandage may be prevented by gathering up the slack, without injuriously disturbing the limb. It is no doubt of great importance to keep up a uniform tension of the perineal band. In fractures of the thigh, with firm perineal extension the patients, especially children, are almost sure to tilt the pelvis and the rest of the body over to that side where they can lie most at their ease, and this must seriously interfere with the accurate coaptation of the broken surfaces. The two following cases occurred in the practice of a friend, who kindly gave me the opportunity of carefully watching the patients throughout, and by whose permission I am now enabled to publish the cases in proof of my statement. In Case 1, notwithstanding the daily and vigorous use of extension, the limb, at first a quarter of an inch shorter, was found to be as much as an inch shorter than the other leg a fortnight after the receipt of the injury, when, the thigh and buttock becoming chafed, extension was discontinued, the perineal band being kept moderately tight. The bone became firmly united, with about a quarter of an inch of shortening, three weeks after.

CASE 1. *Fractured femur, treated by firm perineal extension.* M. L.—, aged nine, was run over by a light spring cart on Jan. 13th, 1869, and the right femur was fractured about the centre of the shaft. The limb was put up and extended upon the long splint, but with a quarter of an inch of shortening. On the 16th the limb was measured, but there was no shortening. On the 26th there was an inch of shortening, although extension had regularly been applied, and that with considerable force and frequency. On Feb. 1st, extension of the limb was suspended, in consequence of the groin and perineum becoming chafed, but the perineal band was kept firm. She was dismissed, cured, six weeks after, with a quarter of an inch of shortening.

Perineal extension by means of pulleys or weights is in some cases of great service, especially where the fracture is oblique. Case 2 is one where manual extension was applied for some time daily, but without gaining any permanent advantage. A seven-pound weight was applied with rack and pulley, and the bone became firmly united, with half an inch of shortening.

CASE 2. *Oblique fracture of femur, treated by firm extension with the long splint and pulley.*—E. S.—, aged eleven, was knocked down by a cart and run over. When first visited by her medical attendant, she was found to have sustained a fracture of the right femur, about the centre of the shaft. The limb was extended upon a long outside splint and perineal band, and manual extension was had recourse to daily, but without gaining any permanent advantage, there being still $1\frac{1}{2}$ in. of shortening. Three weeks after receipt of injury, the limb was put up afresh, and extension made by a pulley fixed at the foot of the bed, with a 7 lb. weight attached. She was dismissed with firm union of fracture, and only $\frac{1}{2}$ in. of shortening.

Great George-street, Westminster, Jan. 1870.

* I have erased a paragraph referring to paralysis of accommodation as an occasional result of accident to the nervous centres. I believe it occurs; but I have not yet seen many cases of it.