

of connective tissues were seen; no granular cells; a few corpora amylacea; anterior horn cells intact.

Dr. Schultze refers to several interesting points in this case. The small volume of the whole cord, which he thinks may be a congenital condition in cases of hereditary ataxy; the recovery from ataxic symptoms whilst the lesion of the lateral parts of the cuneati in almost the whole extent of the cord remained; the paræsthesia of the left ulnar region in connexion with the severe affection of the left cuneated columns in the cervical bulb; the moderate amount of degeneration of the posterior columns in the lumbar bulb, apparently leading to no sensory phenomena in the lower limbs; but the important feature was that the initial symptoms were all recovered from for years, except the bladder weakness and the absence of the patellar reflex, in spite of persistent grave anatomical lesions, and he asks: "Does this show that the anatomical lesion exists even in the earliest stages of tabes, when the symptoms are few, and that the view that a symptom stage precedes that of true anatomical destruction is untenable." At any rate a case like this suggests to us that no instance of so-called cure of tabes should be considered as such unless, death having resulted from some cause alien to the tabes itself, the posterior portions of the cord are found healthy. In a case in which the ataxic symptoms were peculiarly prominent, the patient not being able to guide his legs at all, even with the help of sight, and with the support of an arm on each side, under the long-continued use of ergot the gait became normal, and he left the hospital able to walk straight and in a perfectly coördinate manner with closed eyes. The patient went out thinking himself cured, and returned in about a year paraplegic, and after some months died. Apart from the extension of the lesion into more anterior regions of the cord, the posterior root zones were found sclerosed in an extreme degree, and it was quite evident that during the remission of the symptoms the lesion of the cord had not improved at all.

In this case the duration of the disease was from seven to eight years, and the temporary improvement took place after four years' ailment, so that it does not directly bear upon the question of the curability of tabes in a pre-ataxic stage. But it is an instance of very complete remission of some of the gravest phenomena, with persistence of the lesion. There is no doubt that greater confidence would be felt in the curability of tabes if it were proved that this lesion is as closely connected with syphilis as some observers state. Erb considers that about 85 per cent. of such cases are specific, and Dr. Dowse, in a recent excellent paper, goes even farther when he says: "I feel sure that nearly every case of locomotor ataxy is due to syphilis, either hereditary or acquired; so much so, in fact, that it may well be called syphilitic ataxy." It is certain that a large proportion of cases of locomotor ataxy have had syphilis at some period of their life. It seems less certain that the syphilis has had much to do with the lesion, at least directly. Indirectly the subjects of previous syphilis may be more liable than others to any disorganising process; but pathological evidence shows that syphilis affects the spinal centres with a different arrangement from that which obtains in tabes, and clinical experience proves that but few such cases derive much benefit from mercurials and iodide of potassium.

The old question therefore crops up again, "Can syphilis by its toxic influence on the nervous centre induce the various phenomena that are called the pre-ataxic signs of locomotor ataxy, without sclerosis of the posterior root zones?" If this can be proved, it seems only reasonable to believe that at this stage the mischief may be checked by treatment. But take the following case: A weakly man, of forty-four years of age, finds his walking powers, which had been very good, gradually diminish for some twelve months. He contracted syphilis in a severe form, and within ten days of the first appearance of the sore he suffered from decided paresis of the lower limbs. Within three weeks he became almost paraplegic as regards motion and sensation, with partial retention of urine. Patellar tendon-reflex absent; diplopia from slight paralysis of the right external rectus. No retinal lesion; pupils acted to light, though sluggishly. Ulnar numbness in each hand; sense of hearing confused. He was placed under antisyphilitic treatment, with the effect of making every symptom worse, and materially injuring the general health. Under a long course of phosphorus and strychnine with cod-liver oil the ulnar numbness diminished, sensation returned in the lower limbs; the power of walking was re-

gained to a considerable extent, although a stick was necessary to enable the patient to walk quite straight. All the bladder symptoms vanished, and the diplopia was only observable at times in the day, when there had been much fatigue.

Is this recovery? No. The patellar tendon-reflex is still absent. Is the disease syphilitic? If so, why did not the appropriate treatment do good instead of harm, especially as in this case, if ever, the important symptoms would seem to have depended on syphilitic poisoning. Have we however, as yet, any proof that sclerosis can be recovered from? The remission of symptoms, even for several years, leads to the belief that under treatment directed to the tone of the vessels, or the nutrition of the cord, other tracts of this organ take on the duties that we consider the attributes of the posterior root zones. But during this very remission, if we may judge by pathological anatomy, the lesion is slowly, though surely, following a progressive course; and, although there is nothing impossible in the more hopeful view of cure, I would still repeat that so far published facts are wanting to prove it, whilst, on the other hand, many cases of temporary improvement of symptoms have shown eventually post mortem a steady progress of the lesion.

Clifton.

#### ON THE

### TREATMENT OF PSEUDO-MEMBRANOUS (DIPHTHERITIC) CONJUNCTIVITIS BY LOCAL APPLICATIONS OF SOLUTION OF SULPHATE OF QUININE.

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Two years ago I recorded several cases of true diphtheritic ophthalmia that had been successfully treated, so far as concerned the integrity of the cornea, by local applications of sulphate of quinine (THE LANCET, 1880, vol. i., pp. 125, 282). Since then I have similarly treated four other cases, and though the cornea remained wholly intact in only one of these, they all recovered with useful sight, and with, at worst, but slight nebulæ. To the quinine I ascribe the credit of saving the cornea in these cases from total destruction. Three of the four cases occurred in men and one in a woman. Two began as purulent conjunctivitis of gonorrhœal origin, and two were pseudo-membranous from the first. The former, a man and a woman, were admitted with advanced purulent conjunctivitis of the right eye, the left being free. In both instances, within a few days of admission, and while the inflammation of the right conjunctiva was subsiding, the *left*, in spite of protection by Buller's shield, became the seat of adherent pseudo-membranes. In neither did much pseudo-membrane appear on the right conjunctiva. As regards the two cases that were primarily pseudo-membranous, it is noteworthy that both occurred in men engaged in handling animal carcasses, one being a porter in Smithfield meat-market, the other a slaughterman in the country. No direct contagion from pharyngeal diphtheria or other similar disease could be traced in either of the cases. The meat-carrier thought he had acquired his disease from contact with an acrid "sweat" on some diseased American pork he had been carrying. The slaughterman did not connect his illness with his occupation, though he volunteered the statement that he had often killed diseased animals, and that but few calves have perfectly healthy livers, "not one in ten," most of them containing larger or smaller collections of putrid "matter."

I need not relate the details of the cases; they were all treated in the wards of the Royal London Ophthalmic Hospital.

As soon as the nature of the disease was definitely recognised, all other treatment, if any, was stopped, and quinine lotion, containing four grains of sulphate of quinine, with a minimum of dilute sulphuric acid to an ounce of water, was alone employed. As far as possible the diseased surfaces were kept constantly bathed with the

solution, the conjunctival sac being converted, as it were, into a trough holding the quinine lotion. A bowl of the solution was also placed within reach of the patient, who washed the eye frequently and kept a well-soaked compress constantly applied in the intervals. Besides these applications by the patient and by the nurse, the house-surgeon visited each case three or four times a day. On these occasions the lids were everted and the conjunctival sac thoroughly cleansed with the quinine lotion. The superficial disintegrated portions of the exudations were then gently removed with wet lint, care being taken not to aggravate the inflammation by rough handling or by rude attempts to tear off the pseudo-membrane. Usually, the quinine lotion was iced. In two cases the local application of powdered sulphate of quinine was tried at first, or sulphate of quinine rubbed up with an equal part of calomel; but, in addition to causing great pain, the powder did not appear to be so beneficial as the quinine in solution, and was therefore soon abandoned.

The virtues of the quinine are, I believe, specific in the diphtheritic exudation, and manifest themselves by checking or controlling the inflammatory process and the limiting the inflammatory neoplasms. Whatever may be the influence of the minute organisms that are said by some writers to be essential factors in producing diphtheria, it is certain that in diphtheritic ophthalmia the cornea is destroyed by the degree of the inflammation and the amount of concurrent exudation. The bacteria or vibrios or micrococci (for there is a hopeless disagreement as to which are the noxious organisms) do not destroy the cornea; nor, on the other hand, do strong applications of carbolic acid, which is, we are told, so fatal to "germs," arrest the progress of the pernicious pseudo-membrane in time to save the cornea. Three of the cases to which I have referred were at first treated by frequent ablutions with a five per cent. solution of carbolic acid, and in every instance the pseudo-membranes rapidly spread under these applications, whereas they were immediately controlled by the quinine lotion. Personally, I have a strong impression that the carbolic acid did harm, by aggravating the inflammatory process, and thereby increasing the amount of exudation. But, as formerly remarked, quinine not only checks the inflammation, but it is likewise detergent and antiseptic. (See THE LANCET, 1880, vol. i., p. 282 *et seq.*)

In my previous communications I stated briefly the reasons that led me to employ quinine in cases of true diphtheritic ophthalmia. With these reasons I am still content. Indeed I would now even emphasise them, as I would also emphasise the fundamental difference between the pellicular membrane of ordinary purulent ophthalmia and the parenchymatous pseudo-membrane of diphtheritic conjunctivitis. Extended experience has satisfied me that the two forms are essentially distinct and separate in their pathological as well as in their clinical relations. The pellicular form is amenable to simple treatment, and need not give much anxiety, whereas the pseudo-membranous is terrible in its ravages and appalling in its possibilities. Not a little harm may be done by confounding these two diseases. An application of nitrate of silver to the pseudo-membranes of diphtheritic ophthalmia increases the damage, whereas judicious applications speedily cure the membranous variety of purulent ophthalmia. The difference is appreciable from the earliest to the latest stages. Even when the pseudo-membrane is thin and detachable, its removal does not expose a swollen and vascular mucosa, but a smooth, pale surface often of a dull leaden hue. Later on the difference is still more marked; the membranous form only affects the epithelial layers of the mucous membrane, whereas the pseudo-membranes always invade the deeper texture, and heal by cicatrization. In all the cases I have diagnosed as diphtheritic conjunctivitis cicatrization was always pronounced.

It has by some been objected that the term *diphtheritic conjunctivitis* is a misnomer because the disease is not usually attended by grave constitutional symptoms, and because the pseudo-membranes do not spread to other parts. To these criticisms I would answer, first, that even pharyngeal diphtheria is often unattended by much constitutional disturbance; and, second, that the conditions of the conjunctival mucous membrane are little favourable to the extension of the disease down the nasal duct. There are many reasons for believing that diphtheria is primarily a local disease, and that the constitutional infection occurs secondarily. The poisoning in pharyngeal diphtheria is due partly to the inspiration of the noxious gases generated in the putrefying pseudo-membrane, and partly to the ingestion

of the detritus from these decayed masses. In the case of diphtheritic ophthalmia, on the contrary, the disease is shut out from the system by the conjunctiva and underlying tissues, and all the discharge escapes externally, so that the risks of constitutional infection are few, though not altogether absent. Of the nine cases that I have recognised as genuine diphtheritic conjunctivitis two (infants) died within three weeks, one of what was called "inflammation of the lungs," the other of pharyngeal diphtheria. Five had more or less extensive adherent pseudo-membranes inside the mouth or in the pharynx; and most of the patients showed extreme, almost characteristic, pallor, accompanied with general malaise. As regards the extension of the pseudo-membranes from the conjunctiva down the nasal duct, it may be said that if the diphtheritic pseudo-membrane rarely spreads from the nasal cavity up the nasal duct into the conjunctival sac, it is not surprising that it should so seldom travel in the opposite direction. The canaliculi and the nasal ducts may roughly be compared to funnels with the mouths downwards; hence it is easier for inflammation to extend from the nasal cavity to the conjunctiva than from the conjunctiva to the nose. The canaliculi are normally too small to allow the pseudo-membrane to pass through them, and very little swelling of the mucous membrane would obliterate them altogether. It does, nevertheless, sometimes happen, as I have just hinted, that even when the diphtheritic process begins in the eye it shows itself subsequently in other parts. This actually occurred in the meat-carrier referred to above. Four days after his admission into the hospital, and after the conjunctival mucous membranes showed appreciable improvement, he complained of soreness of the throat. Within twenty-four hours his tongue, roof of the mouth, and inside of the cheeks and lips were covered with an adherent dirty coating, which could not be rubbed or pulled off. The pharyngeal mucous membrane was red and swollen, and soon became completely covered with a thick pseudo-membrane, exactly resembling that on the conjunctiva. He eventually made a good recovery, and left the hospital with perfectly clear corneæ, though his palpebral conjunctivæ were contracted by dense cicatrices. Furthermore, it should be borne in mind that the pseudo-membrane of diphtheria usually spreads more quickly by contiguity of tissue than by continuity. This is especially noticeable in pharyngeal diphtheritis where, say, one tonsil becomes affected, then the contiguous portion of the uvula, and then the intervening arch.<sup>1</sup> So in the conjunctiva two opposing portions of ocular and palpebral conjunctiva become affected long before the pseudo-membrane has time to travel round by the oculo-palpebral fold.

One word of explanation may be necessary—namely, that all four cases were of a very severe type. In one case (gonorrhœal) the conjunctival sac looked as if it had been filled with dirty tallow, except opposite the cornea, which was of a turbid sea-green colour. In the case where the pseudo-membrane was least in amount and extent (the slaughterman) the cornea suffered most. After the pseudo-membrane had disappeared a superficial ulceration spread over the greater part of the cornea, but did not cause much opacity.

I must, in conclusion, thank Mr. Burnham, Dr. Webster Fox, Mr. Milles, and Dr. Fitzgerald, past and present house-surgeons, for the intelligent and sympathetic manner in which they carried out my instructions and conducted the various observations.

<sup>1</sup> For a detailed description of the mode of extension of the diphtheritic pseudo-membrane in the pharynx, see Trousseau's *Clinique Médicale*, 5th edition, vol. i., p. 433; or the New Sydenham Society's Translation, vol. ii., p. 472.

**REWARDS FOR MEDICAL MEN IN CHINA.**—The following paragraph appeared in the *Daily News* on the 4th inst.: "The late Empress of China having recovered from her former serious illness, some half-dozen surgeons, chosen by governors of provinces and sent to Peking, according to Imperial instructions, to attend upon her Majesty, have been rewarded by various appointments. One, it is announced, is to be made a taotai, or intendant of circuit, another a prefect, another a district magistrate, and so forth. 'This,' says the *Shanghai Courier*, 'is very much as if after the recovery of the Prince of Wales from his historic illness Sir William Jenner had been made a county court judge and Sir W. Gull a stipendiary magistrate.'"