fingers placed above Poupart's ligament came directly upon the tumour. There was very little pain. He had begun to have a little uneasiness down the course of the anterior crural nerve. He gradually became cachectic as the tumour increased and died about six months after I saw him.

In this group the tumour is in the lower abdomen, usually on one side, and, as in Cases 1 and 2, it may have the outlines of an enlarged testis with the epididymis attached. It is interesting to note that in two of the cases there was ascites, a not uncommon event in connexion with the solid abdominal tumours, particularly of the ovary.[2] The general contour of the abdomen in Case 1 suggested simple ascites and the tumour was only discovered on deep palpation. The cases are not very common. Chevassu in his recent study[3] has collected 128 cases of tumour of the testicle from recent literature, and of these ten were inguinal and five abdominal. There is not much difficulty in the diagnosis, as very often the tumour has the shape of the testicle with its epididymis. In the cases of Abel[4] and of Marion[5] the tumour occurred in hermaphrodites with the external genitalia of women. The nature of the growth was of course not suspected until operation when the uterus and ovaries were absent and sarcomatous change was found in one of the abdominal testicles. The question of prompt surgical treatment is important, as involvement of the glands may occur very early as in this case. The abdominal tumour has been removed in many instances, but great difficulty has been met with in complete extirpation, as in Case 1. The retained testis lies so close to the posterior abdominal wall that the adjacent tissues are soon involved. Considering the liability to rapid involvement of the lymph glands of the affected testicle it would seem reasonable in all cases to remove them as well as the primary tumour. It adds greatly to the seriousness of the operation, but in young persons the risk is worth taking. It was done by Roberts[6] of Philadelphia, but his patient was old and fat and the operation was secondary to a recurrence.

Two Clinical Lectures

ON

LESIONS OF THE TRIGEMINAL NERVE.

Delivered at the Royal London (Moorfields) Ophthalmic Hospital

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I.

HERPETIC AND ALLIED AFFECTIONS OF THE CORNEA.

GENTLEMEN,—Herpes zoster is a disease which shows a definite seasonal variation. Dr. H. Head,[1] who has made a special study of the condition and to whom we owe so much in the elucidation of the pathology of the disease, states that in 1896, 1897, and 1898 there was an epidemic in the middle of March; in 1897 and 1898 there was an outbreak from the middle to the end of May. Herpes ophthalmicus is merely that form of herpes zoster in which the area supplied by one or more branches of the fifth nerve is affected. I do not know whether other forms of herpes zoster were prevalent during last November, but I was particularly struck with the fact that within ten days in the middle of that month four cases of herpes ophthalmicus in the acute stage came under my observation—two at this hospital and two in private. There are corneal conditions which bear a very marked resemblance to herpes corneæ, without, however, the concomitant skin eruption. Two of these cases, almost identical in their characteristics, came under my care during February—one in this clinic and one at University College Hospital. The opportunity seemed, therefore, favourable for drawing your attention to this interesting class of cases, particularly because it occurs in batches in this peculiar manner, being uncommon in sporadic form, and also because accurate diagnosis in the early stage enables the surgeon to foretell the probability of relapses, instilling confidence into the patient by the knowledge he displays of the tedious condition which he is called upon to treat.

Almost invariably in herpes ophthalmicus the history is that of the sudden onset of an attack of "erysipelas." This is due to the swelling and redness of the affected parts and to the exact delimitation from the normal skin around. Even at this stage diagnosis is not usually very difficult. The presence of vesicles is equivocal, but the exact distribution in areas supplied by one or more branches of the ophthalmic nerve and the severe neuralgic character of the pain experienced facilitate diagnosis. Generally the ophthalmic surgeon first sees the case at a slightly later stage when the swelling and redness are subsiding and only vesicles or their scars mark out the areas affected. The branches of the fifth nerve most commonly involved are the supra-orbital, supra-trochlear, and nasal. The supra-orbital nerve supplies the skin of the forehead and scalp extending as far back as the occiput, consequently vesicles usually occur far back into the hairy scalp. The nasal nerve supplies the skin of the tip of the nose, which is therefore affected, leaving an area of normal skin on the side of the nose, since the infra-orbital nerve is seldom involved. Specially noteworthy is the absolutely unilateral limitation, the areas of inflammation never extending beyond the middle line. The branches of the ophthalmic nerve which supply sensory filaments to the globe are by no means always attacked, so that in a considerable number of cases the cornea escapes. The patient or his friends should be warned that the blebs will leave indelible scars. These characteristic, slightly depressed white cicatrices, with their very typical distribution, afford permanent evidence of a previous attack of ophthalmic herpes. Some of you may remember seeing recently in my clinic a man who came complaining of epiphora affecting the left eye. The history he gave was that the onset dated from a severe attack of inflammation six years before. He had the typical scarring of ophthalmic herpes involving the usual areas of the left side, and careful investigation left little doubt that the occlusion of the lower canaliculus which was found was due to the inflammation accompanying this attack.

It has already been mentioned that the cornea is not always the seat of an herpetic eruption in these cases but the most thorough examination of its condition must be invariably made. In all cases there is some conjunctival injection, and not infrequently acute conjunctivitis. In the absence of true herpes of the conjunctiva and cornea this is an epiphenomenon due to the action of organisms, particularly staphylococci, &c., derived from the inflamed skin.

The true herpetic affection of the cornea consists, as in the case of the skin, of the development of minute vesicles upon its surface. This stage is rarely seen; the walls of the vesicles, composed only of epithelium, rupture very quickly owing to the movements of the lids and usually the tiny ulcers resulting from their rupture first come under the surgeon's observation. They are generally multiple, often arranged in groups or rows. They may easily be overlooked, since in the early stages they are not infiltrated and examination is rendered difficult by the irritation and reflex blepharospasm present. They are, of course, best made manifest by staining with fluorescin.

This acute form of vesicular keratitis is much commoner in herpes febrilis than in herpes ophthalmicus; in the latter a deep infiltration of the cornea is not uncommon. The fundamental identity of the corneal affection in the two diseases is shown not only by their identity during the vesicular stage but also by the similarity of the subsequent course. Indeed, it is probable that the pathogenesis of the vesicles is the same in each case. Whether due to herpes febrilis or zoster the keratitis is characterised by its long duration and the frequency of relapses. Sometimes the minute ulcers heal rapidly, to be followed at a short interval by a fresh crop of

[2] I have reported two cases of solid tumours of the ovary in which the ascites recurred for many months, requiring repeated tapping, and in both patients the diagnosis of the solid tumour was only made on examination after tapping. Both patients recovered after the removal of the affected ovary and both are alive to-day, one 20 and the other seven years after the operation.

[3] Tumeurs du Testicule. Paris, Steinheil, 1906.

[4] Virchow's Archiv, Band cxxvi., p. 420.

[5] Annales des Maladies des Organes Génito-urinaires, tome xxiii.

[6] Annals of Surgery, 1902.

[1] Head: Allbutt's System of Medicine, vol. viii.

vesicles. In other cases the spots enlarge and fuse, a considerable area of the cornea becoming denuded of epithelium, and this area continues to increase, showing a crenated border. In other cases, again, the typical dendritic ulcer is formed. Grey striæ extend in one or more directions from the minute ulcer, grow longer and throw out lateral branches, finally breaking down. I do not think that it is correct to consider herpetic all ulcers of the cornea which have a superficial resemblance to the dendritic ulcer. In many cases they are traumatic and heal rapidly and the term "dendritic," though it describes their appearance, is best not applied to them, for it has come to have a specific meaning attached to it.

All these herpetic ulcers of the cornea are superficial and never extend in depth unless they become infected. In most cases they involve Bowman's membrane and leave permanent faint nebulæ, though the scars may be so faint as to cause little or no impairment of vision in spite of the fact that they are generally in the pupillary area. Characteristic of all forms of herpetic keratitis is the fact that the sensitiveness of the cornea is depressed. If the cornea is touched with a wisp of cotton wool there is much less response in the form of reflex closure of the lids on the affected side than on the sound side.

These types of keratitis associated with herpes febrilis are less likely to be accompanied by complications than those found in ophthalmic herpes. It is said that the cornea is more prone to be attacked in the latter group when the nasal nerve is affected than when it escapes, possibly due to the fact that the nerve-supply of the uveal tract is derived through the lenticular ganglion from a branch of this nerve. The rule is certainly not an invariable one. Iritis not infrequently occurs in ophthalmic herpes corneæ, and there may even be severe iridocyclitis, with precipitates ("k. p.") upon the back of the cornea. Indeed, it is the rule for some slight diminution in the intra-ocular pressure to be present, pointing to interference with the processes of secretion of intra-ocular fluid by the ciliary body. I do not think too great importance should be attached to this statement, for the depression in the intra-ocular tension is seldom very marked.

Allied to these typical forms of herpetic keratitis are other cases which are not vesicular but which generally follow some febrile attack and pursue a course strikingly similar to true herpes. One such case I am able to show you. About a week after a febrile attack, probably influenza, this young woman's left eye became inflamed. On examination it was found that there was a group of yellowish-grey spots of about the size of a small pin's head, slightly above the centre of the cornea. They were separated from each other by greyish areas, which on magnification with the loupe showed fine striæ. Several of the spots showed staining with fluorescein at this period. The other case, which I have already referred to as occurring at University College Hospital, was almost identical both in history and appearance.

Of the forms of keratitis previously described by other writers these cases most resemble the condition known as superficial punctate keratitis (Fuchs). They differ from most of Fuchs's cases in that these are generally bilateral, and there is no loss of substance so that they do not stain with fluorescein. In the University College case I scraped the cornea and made a film and a culture on serum. No organisms could be found in the film and the culture was sterile, possibly owing to the previous use of cocaine. Both cases were treated by cauterisation with pure carbolic acid and both are very markedly improved. The irritation and redness of the eyeball have almost disappeared but the spots are quite plainly visible, though they no longer stain. They will probably persist for many weeks or even months and it is not unlikely that recurrent attacks of irritation and possibly fresh spots may occur.

There are other allied conditions which I cannot now discuss in detail. Some cases show an extreme tendency to desquamation of the corneal epithelium, so that when the patient wakes in the morning the greater part of the corneal epithelium is torn off and great pain is experienced. There are also cases of filamentary keratitis occurring in otherwise sound eyes, which are probably similar in nature and pathology.

The pathology of these herpetic and parerpetic conditions is of much interest. As long ago as 1862 herpes zoster was attributed to lesions of the posterior root ganglia (von Bärensprung[2]). Wyss[3] (1871), Sattler[4] (1875), and Kaposi found hæmorrhage and infiltration of the Gasserian ganglion in herpes ophthalmicus. The subject was attacked and exhaustively investigated by Campbell and Head[5] in 1900. 17 cases of herpes zoster were examined at all periods, from a few days to one and a half years after the eruption. In all the acutest cases hæmorrhages into the ganglion were found, usually small, but surrounded by a considerable amount of inflammatory exudation. The ganglion cells were destroyed to a varying extent and in the later cases parts of the ganglion were markedly sclerosed. Three cases of zoster within the territory of the trigeminal were investigated and in all changes were discovered in the Gasserian ganglion.

There is therefore good evidence to believe that ophthalmic herpes is dependent upon definite lesions in the Gasserian ganglion, which is morphologically a dorsal root ganglion. It is probable that the pathology of the various types of cases referred to in this lecture is fundamentally the same, different as are the clinical features in certain details. The frequency of a febrile onset is striking, and though herpes febrilis does not manifest the same accuracy of distribution according to nerve supply that is found in herpes zoster it is yet probable that the causal lesion should be referred to the peripheral sensory nerves and most likely to their ganglia rather than to their terminations. Herpes zoster is the outcome of an intense and concentrated attack upon certain dorsal root ganglia. Herpes febrilis and the parerpetic affections of the cornea (and possibly of other parts of the body) may reasonably be regarded as a less acute, more widely diffused attack of a similar nature. In herpes zoster parts only of an individual ganglion are likely to be destroyed. In the parerpetic forms we must predicate less destruction of tissue and abolition of function, but finer localisation, often combined with wider diffusion, of partial derangement of tissue and disorder rather than demolition of function.

Granting the accuracy of our views as to the site of the lesion there remains the extremely difficult task of explaining its manifestations. We are accustomed to regard sensory nerves as conveying only afferent impulses and it is difficult to imagine how even an inflammatory block in the course of the nerve can produce the pathological changes in structure at its distant terminations. The trophic control of the nerve fibres themselves by the dorsal root ganglion is a well-established fact. The trophic control of the tissues supplied by the fibres, early invoked to account for the phenomena of neuropathic keratitis, &c., is doubtful, if indeed it may not be said to have been disproved. Several cases of neuropathic keratitis following extirpation of the Gasserian ganglion for trigeminal neuralgia have come under my observation. If suitable precautions are taken during and after the operation this complication is infrequent, a fact which goes far to disprove the trophic theory. It is impossible to enter into a discussion of the experimental investigations upon the subject here. A careful examination of the facts derived from this source leads me to agree with the conclusions arrived at by Wilbrand and Sänger.[6] It is probable that neuropathic keratitis is due neither to abrogation of trophic influence nor to this cause assisted by loss of sensation and the invasion of pathogenic organisms. There is reason to believe that in all those cases in which the cornea becomes ulcerated after division of the fifth nerve the cut ends of the nerve are subjected to some abnormal irritation, either by blood clot, pus, or other agent. This view fits in well with the manifestations of herpes and parerpetic affections. If, then, there is undue irritation in the course of the nerve, as for example at the ganglion, how are we to explain the distant effects? They must be due either to the transmissisn of impulses along the nerve in the reverse direction to the normal afferent impulses or to the transmission of deleterious agents along the nerve.

There is some experimental evidence of the transmission of impulses along sensory nerves in the opposite direction to that of the normal impulses. In 1876 Stricker showed that vaso-dilatation of the hind limb of the dog occurred when the peripheral ends of the divided posterior roots of the sixth and seventh lumbar nerves were stimulated. In 1900 Bayliss,[7] contrary to his expectation, confirmed these results

[2] Von Bärensprung: Annales de Charité, vol. xi.
[3] Wyss: Archiv für Heilkunde, vol. xii.
[4] Sattler: Berliner Klinische Wochenschrift, 1875.
[5] Campbell and Head: Brain, 1900.
[6] Wilbrand and Sänger: Die Neurologie des Auges, vol. ii., 1901.
[7] Bayliss: Journal of Physiology, vols. xxvi. and xxviii.

and subsequently adduced other examples of a similar nature. He called these efferent impulses occurring in afferent nerves *antidromic impulses*. It is probable that further investigations from this point of view would elicit facts of interest and importance in the pathology of herpetic and parerpetic conditions.

The time at my disposal does not permit me to enter into the question of the treatment of these affections, nor to discuss more fully the allied forms of neuropathic keratitis.

[Since this lecture was delivered the obvious bearing of the researches of Mr. W. M. Bayliss upon Dr. Head's work has been the subject of experiment by them and the results of their researches are embodied in a further paper by Dr. Head. See Head and Thompson, *Brain*, vol. xxix., No. 116, 1906 (just published).—J. H. P.]

II.

NEUROPARALYTIC KERATITIS.

GENTLEMEN,—In my last clinical lecture I brought forward some cases of herpetic and parerpetic conditions of the cornea. I propose now to deal with neuroparalytic keratitis, paying special attention to the pathology of the disease in so far as this can be elucidated at the present time. The time is indeed not unsuitable for such a discussion, since researches of the very greatest importance upon the physiology and pathology of the sensory nervous system have recently been published in this country. It is advisable often to review old facts in the light of new experiences; it is imperative always to remember that the eye is but a member of a complex organism.

Neuroparalytic keratitis is a comparatively rare disease; it accompanies paralysis or paresis of the trigeminal nerve or of its ophthalmic branch, but is by no means present in all such cases. Of late years this fact has been conspicuously demonstrated by the many cases of extirpation of the Gasserian ganglion for trigeminal neuralgia at the hands of Krause, Sir Victor Horsley, and other surgeons. If due care is exercised only a very small proportion of these cases develop corneal complications. The disease is indeed the more mysterious on this account, for in most cases it is impossible to discover any adequate cause for it. Other morbid conditions which lead to paralysis of the fifth nerve or its ophthalmic branch may induce neuropathic keratitis. The seat of the lesion may be in the sphenoidal fissure or cavernous sinus, peripheral to the ganglion, involving it, or proximal to it; it may even be in the brain involving the central connexions of the nerve. The operative removal of the Gasserian ganglion, however, is best suited to provide evidence of the true *rationale* of the disease, for it has all that accuracy of localisation which pertains to an experiment and is so often open to doubt in the manifestations of disease.

In most cases in which neuroparalytic keratitis follows extirpation of the ganglion it commences during the first days after the operation. There are a few cases in which corneal anæsthesia due to disease has resulted in this form of keratitis only after an interval of months or years (up to four years). In a typical example the cornea becomes dull and the epithelium is thrown off, first at the centre, then more and more peripherally, until eventually there is only a narrow rim, from two to three millimetres broad at the margin. There can be little doubt that this vulnerability of the epithelium is the characteristic feature of the disease. The subsequent increase in the opacification, development of hypopyon, perforation, &c., are attributable to the invasion of the substantia propria by pyogenic organisms, now rendered possible by the removal of the first line of defence. The rapidity of the destruction of the deeper layers of the cornea, which is frequently noticeable, indicates that the resistance of the tissues is diminished but affords no indubitable evidence that this is due to the affection of the nerve. There is, of course, no pain, but there is conjunctival and often ciliary injection. The absence of lacrymation is due to the block in the afferent limb of the reflex arc and has no bearing upon the question of the true secreto-motor supply of the gland, whether derived from the fifth or seventh nerve.

It is not my intention to attempt to discuss in detail the *pros* and *cons* of the rival theories of neuroparalytic keratitis. This has been done exhaustively by Wilbrand and Sänger.[8] After enumerating these theories I propose to point out the bearing of recent researches on the afferent nervous system upon the question and to elaborate the theory which in my opinion best explains the facts of the case. The *trophic theory*, enunciated by Magendie (1824) ascribed the disease to the abolition of special trophic nerve fibres running in the nerve. These fibres under normal conditions control the nutrition of the cornea. Similar fibres were supposed to run in other nerves, their destruction accounting for the development of bedsores and other "trophic" lesions. It is noteworthy that Gaule opposed the theory on the ground that no centrifugal fibres were distributed to the cornea, but the chief fact which militated against it was the possibility of warding off the disease by protecting the eye from external injury (Snellen, Senftleben). Hence arose the *trophic-traumatic theory*, which asserted the necessity of injury in addition to the diminution in resistance of the tissues brought about by the destruction of the trophic fibres. The *vaso-motor theory* was brought forward by Schiff (1867), attributing the inflammation to dilatation of the blood-vessels owing to paralysis of the vaso-motor nerves. In a later modification the *vaso-motor-traumatic theory* admitted the influence of external injury. Theories which eliminated trophic influence entirely were the pure *traumatic theory*, which ascribed the unusual effect of injury to the insensibility of the cornea; the *xerotic theory*, which invoked drying of the cornea, due to diminished blinking, &c.; and the *mycotic theory*, which attributed the disease to bacterial invasion.

It will be observed that simple division of the fifth nerve affects the cornea in many ways. The afferent sensory impulses are blocked, so that anæsthesia and analgesia result. Hence slight injuries, foreign bodies, &c., are liable to pass unnoticed and produce deleterious effects. Accompanying the anæsthesia is the abolition of the reflex secretion of tears. In lower animals there is also diminution in the frequency of blinking, absent in higher animals, except in bilateral paralysis, owing to the synergic activity of the two orbiculares. The vaso-motor element is itself complex. Vaso-constrictor fibres for the eye, derived from the cervical sympathetic, run near the Gasserian ganglion and join certain of the branches of the nerve. Their division results in dilatation of the vessels. It is probable that vaso-dilator fibres for some parts of the eye run in the trigeminal itself. Their division results in vaso-constriction. It is difficult to define the relative importance, if any, of these conflicting factors. That the bacterial element is of importance in the later stages of neuroparalytic keratitis cannot be doubted, but it is unlikely that it explains the desquamation of the epithelium which is the characteristic feature of the disease and is known to be caused only by such organisms (e.g., gonococcus) as are almost certainly absent.

In the previous lecture it was shown, chiefly on the evidence brought forward by Head and Campbell, that the causal lesion in herpes ophthalmicus is situated in the Gasserian ganglion. In the absence of known efferent channels in the parts of the fifth nerve under consideration it was necessary to have recourse to antidromic impulses travelling centrifugally along the "afferent" fibres, basing the explanation upon the proved occurrence of such impulses under certain conditions by Bayliss.[9] Head has now carried his researches upon the sensory nervous system a vast stride forwards. He has chosen his coadjutors, Rivers, Sherren, and Thompson,[10] with the same acumen that he has displayed in devising the details of the research, for they are severally specially qualified to attack the problems allotted to them. Commencing with an investigation of lesions of the peripheral nerves, Head has arrived at the conclusion that three types of sensibility may be distinguished, called respectively deep, protopathic, and epicritic, and that the corresponding impulses travel to the central nervous system by three distinct sets of fibres. Deep sensibility persists after destruction of all cutaneous afferent fibres. The impulses travel in motor nerves (thus confirming previous work of Sherrington) as far as the separation of the anterior and posterior spinal roots, when they pass into the posterior roots and join the rest of the afferent system. This form of sensibility includes deep pressure, movements of muscles, localisation of pressure, and recognition of the extent and direction of passive movements in joints. Loss of protopathic sensibility abolishes cutaneous

[8] Wilbrand and Sänger: Die Neurologie des Auges, vol. ii., 1901.

[9] Bayliss: Journal of Physiology, vols. xxvi., xxviii.

[10] Head, Rivers, and Sherren: Brain, vol. xxviii., No. 110, 1905. Head and Sherren: Brain, vol. xxviii., No. 110, 1905. Head and Thompson: Brain, vol. xxix., No. 116, 1906.

pain, sensations of heat above 45° C., and sensations of cold below 20° C. Loss of epicritic sensibility abolishes recognition of light touch, discrimination of compass points, appreciation of differences in size, and discrimination of intermediate degrees of temperature from 25° to 40° C. After division of a sensory nerve protopathic sensibility is restored in about six weeks if the ends of the nerve are placed in apposition. Cutaneous sores which may have occurred in the area affected from injury, &c., show little or no tendency to heal until protopathic sensibility is restored, when healing rapidly ensues. Epicritic sensibility requires much longer for restoration and the time varies with the position of the affected part. The areas of protopathic and epicritic sensibility are not identical. In any peripheral nerve the distribution of the protopathic fibres usually overlaps greatly the area supplied by the fibres of the adjacent nerves, whilst the distribution of the epicritic fibres in the larger peripheral nerves, such as the median and ulnar, overlaps only slightly. It is found for certain peripheral nerves or nerve groups—such as the median, ulnar, or pre-axial nerves of the arm—that each of these forms a unit of the epicritic system, whilst the protopathic unit must be sought in one or more posterior nerve roots, for the nearer the lesion is situated to the posterior roots the more extensive and definite is the loss of protopathic sensibility. Similarly the more nearly the injury divides one of the nerve groups the more definite and extensive is the epicritic loss.

Lesions of the spinal cord show that there is a complete redistribution of afferent impulses, so that peripheral impulses are transmuted into those of the secondary level of the afferent nervous system. This transmutation and recombination take place on the same side as that by which the impulses enter the cord. The secondary paths for sensory impulses then cross with greater or less rapidity, so that ultimately all except those subserving the sense of passive position and movement and tactile discrimination have passed to the opposite side within the limits of the spinal cord. Even these sensory impulses cross after reaching the nuclei of the posterior columns. At the same time, within the cord afferent impulses become separated into sensory and non-sensory. Of the latter many pass up in the secondary system of the direct cerebellar tract to reach the cerebellum.

It has been mentioned that skin lesions in an analgesic area show little or no tendency to heal until restoration of protopathic sensibility has taken place. There is further evidence that these peripheral lesions are in some manner under the control of the protopathic system. Head has previously shown that there is a close correspondence between the distribution of the tenderness caused by irritation through visceral disease of the segments within the cord and the areas marked out on the skin by the eruption of herpes zoster. He has also shown that herpes zoster is due in most cases to acute inflammation of a posterior root ganglion, whereas the tender areas in visceral disease are due to irritation of intramedullary segments. But, though all the cells and the fibres peculiar to them must be affected by the profound inflammation of the ganglion, one system only, so far as we know, can produce antidromic effects upon the skin, and these are the fibres shown by Bayliss to have their cells of origin in the ganglion of the posterior root. Head and Bayliss have now shown that these antidromic fibres are capable of excitation in the divided nerve of a cat five weeks after it has been reunited to the central nervous system. Hence, it may be considered proved that the power of producing changes in the skin of the periphery is a function of fibres which run in the protopathic system.

Without discussing the bearing of these researches upon the afferent impulses in the trigeminal nerve and their redistribution within the central nervous system, for which the material is indeed as yet insufficient,[11] it is clear that they have an important bearing on the pathology of neuroparalytic keratitis. Whether such so-called trophic lesions can be caused by the abolition of antidromic impulses alone—i.e., whether antidromic trophic impulses exist as a physiological phenomenon—is a question which may be set aside for the present, though the evidence in favour of such impulses is undoubtedly increased. What seems to be certain is that abnormal stimulation of the protopathic fibres in or near the ganglion produces antidromic impulses which have a deleterious effect upon the nutrition of the peripheral organs. The researches, in fact, bring forward strong evidence in support of a theory already suggested by Wilbrand and Sänger—viz., that neuroparalytic keratitis is due to irritation of the distal end of the cut or diseased trigeminal nerve. This theory explains better than any other all the diverse clinical facts which have accumulated. It explains the absence of keratitis in those cases in which it does not occur. Neuroparalytic keratitis may occur in association with retained corneal sensibility. Here the afferent tract is still open, but an irritative lesion has set up abnormal antidromic impulses in the protopathic system. There may be hyperæsthesia of the cornea with keratitis. Here not only is the afferent tract open but it is subject to abnormal stimulation either at the periphery or at the site of the lesion, and abnormal antidromic impulses are also set up. There may be anæsthesia dolorosa. This is due to irritation of the proximal end of the cut or diseased nerve, whilst antidromic impulses are set up in the distal section.

In conclusion I may perhaps be permitted to express the admiration which we must all feel for the brilliant results which have been obtained by Dr. Head and his fellow workers from their extremely laborious researches.

[11] Much material for such a discussion has been collected by Dr. Morrison Davies from an exhaustive investigation upon 50 cases from which the Gasserian ganglion had been removed. The paper will shortly be published in *Brain*.

TORSION OF THE TESTIS.*

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THE following note is the outcome of the examination by us of nine cases of torsion of the testis that have come under our immediate notice. Three of these were under our care and the other six we are able to publish by the kindness of the members of the surgical staff of the London Hospital. Four of the specimens are now in the museum of the London Hospital Medical College.

Etiology.—Age.—Torsion of the spermatic cord may occur at any age, Taylor [1] reporting a case at birth, Howse [2] one at 48 years, and Lexer [3] a case in a man of 60 years. In our nine cases the youngest was four months old and the oldest 24 years, the average age being $12\frac{1}{2}$th years. In 23 cases taken without selection from the literature, 17 occurred in patients before the age of 20 years and eight before the age of ten years. The condition is therefore most common in young subjects.

Side.—In 40 cases taken from the literature the right testis was affected in 22 cases and the left in 18, giving a slight predominance to the right side; the predominance of undescended testis is also on the right side.

Position of the testis.—The earlier cases of torsion of the cord were chiefly noted in the partially descended testis, but later many cases have been reported in the fully descended organ. In our nine cases four occurred in the fully descended testis, four in the inguinal testis, and in the other the testis could, and did, easily ascend into the inguinal canal. In connexion with the last case it is interesting to note the ease with which the testes of the young can be made to ascend into the canal by contraction of the cremaster—a condition described by Corner [4] as "moveable testis" and which he believes to be due to the presence of a long mesorchium. In the 23 cases the testis was fully descended in nine cases and in 12 cases was in the inguinal canal. The position in the other two cases was not stated. Stiles [5] has described a case of torsion in an abdominal testis, but the organ was the seat of a malignant growth. There had been five attacks of pain, vomiting, and frequency of micturition before the testis was removed.

Cause.—The predisposing cause is a congenital abnormality of the attachment of the testis to the spermatic cord, with, perhaps, a voluminous condition of the tunica vaginalis and will be more fully referred to under the pathological anatomy. The exciting cause is not certainly known.

* The superior figures are references to the bibliography at the end of the article.