this and in all other parts of physiology Great Britain is holding her own. But no science, least of all those on which medicine is based, belongs to one country, and neither we nor any other nation can claim to occupy a pre-eminent position in physiology such as might have been granted us three centuries ago as the result of Harvey's epoch-making discovery.

OCULAR MANIFESTATIONS OF LESIONS OF THE FIFTH CRANIAL NERVE*

BY

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The Gasserian ganglion is the trophic centre for all the sensory branches of the trigeminal nerve, the most important somatic sensory nerve of the body. It corresponds with a posterior root ganglion of a spinal nerve, and, like it, lies outside the central nervous system. From each cell body in the ganglion a single fibre arises which bifurcates, sending one branch centrally to the nucleus in the medulla and one to be distributed to the tissues, and any pathological process in these cells may be referred to the corresponding point of its peripheral distribution.

The ganglion may thus be involved by the spread of inflammation from middle-ear disease, producing unilateral headache; it may be involved in a basic meningitis, or a tumour of the cerebello-pontine angle, or it may be implicated in inflammatory or neoplastic processes in any part of its peripheral course. Further, there can be no doubt that the trigeminal is of great importance in the production of headaches, and indeed Ivy Mackenzie goes so far as to say that the "physiological pathology of headaches may be reduced to a question of the irritation of the trigeminal nucleus in the medulla."

REFERRED PAIN AND IRRADIATION

In many respects the eye is the most convenient organ in the body for the examination and observation during life of diseased tissues, such, for example, as the blood vessels, the optic nerve, or the iris, all of which can be examined in a way that is possible with no other tissues, and, further, it lends itself to the examination of the phenomenon of referred pain. By referred pain is meant pain which is referred to a tissue which has no reason to produce it. It occurs as a result of excessive afferent stimuli ascending to the nucleus of the nerve from a diseased peripheral structure, and there irradiating so as to stimulate neighbouring nerve cells, the fibres of which supply the area which is the subject of the referred pain. On the question of irradiation Sherrington says, "If, by appropriate stimulation of the skin of the foot, the ordinary flexion reflex of the hind limb of the dog be evoked, the extent of the reflex increases with the increase in the intensity of the stimulus."

There is an exceedingly free interconnexion between any afferent channel and the congeries of arcs of the whole central nervous system. It is not surprising, therefore, that reflex reaction should spread; the extreme degree of irradiation may be seen after strychnine administration, for it is generally accredited that centripetal impulses passed in by any afferent nerve will then excite reflex discharge over the efferent channels of the whole nervous system. Further, it seems that limited irradiation tends to spread along certain paths with greater facility than

along others, and this difference is probably referable to the difference in the facility of conduction at different synapses.

Referred pain is, on good grounds, believed to be an expression of irradiation in the nucleus of the nerve. Thus pain resulting from an inflamed iris will at first be referred to the iris. If, however, the inflammation in this structure becomes more severe or prolonged, so that the afferent impulses from it are augmented, a spread will occur within the fifth nucleus to immediately adjacent cells, so that pain now will be referred to the side of the nose or orbital margin, and, with further increase, the side of the face may share the extension of pain, or the meninges may be involved and cause headache, or vomiting may ensue from spread to the vagus centre.

There are two anatomical factors which facilitate the spread of the stimuli within the fifth nucleus and from one nucleus to the other. First, individual fibres of the primary neuron ascending and descending give off collaterals at different levels, and so constitute a bond of integration between different levels in the nucleus, and, secondly, there is a free communication between the two trigeminal nuclei, so that a close intercommunicating link is provided which associates together all the diverse tissues from which afferent impulses are directed to this nucleus.

Thus it comes about that a severe or prolonged stimulus may overflow the cells which are in immediate organic continuity with the inflamed tissues and involve first one, and later the opposite nucleus, till pain is referred to the whole territory of each nerve, and, overflowing further, may spread widely and cause more distant and diffuse manifestations.

We have already observed that the resistance to the spread of impulses may be greatly reduced by strychnine, so as to allow an efferent impulse almost free run of the whole central nervous system, and so also it would seem that the irradiation of painful afferent stimuli may depend not only upon the strength of such stimuli but also upon the condition of the brain tissues which receive them, and thus we may well believe that such factors as endogenous or exogenous toxins, of which we hear so much, and, I venture to think, too much, constitutional debility, whatever its source, or inherited instability of the nervous system, may be factors which are important in the dissemination of referred pain.

HERPES ZOSTER OPHTHALMICUS

It is probably unnecessary to remark that herpes cornea, with which dendritic and stellate corneal ulcers are closely allied, is connected by scarcely more than name with herpes zoster ophthalmicus.

The close relation which exists between herpes zoster and varicella is well established, and Le Feuvre¹ urges that "shingles should be recognized as an infectious disease, and one, under certain unknown conditions, likely to become a starting point for a chicken-pox epidemic." Until Jonathan Hutchinson's classical account of herpes ophthalmicus in 1866-7,² this disease had been confused with erysipelas, a mistake that is not quite unknown to-day.

In this connexion Hutchinson describes what, for this association, we may call an interesting domestic occurrence. In 1866 a plate representing herpes zoster of the head and face, which had been presented to the New Sydenham Society, excited a little amusement among some of the members. "Who," they said, "had seen the disease in such a part? Surely it was unnecessary to depict so rare and far-fetched a complaint." We may judge how little the disease was recognized. Hutchinson goes on to say, "Curiously, some one of the Council (Dr. Markham, who at this time was editor of our Association's *Journal*), who had had his joke on the

* Read in opening a discussion in the Section of Ophthalmology at the Centenary Meeting of the British Medical Association, London, 1932.

occasion, was himself, last week, pronounced by his friend Dr. Stewart (another of the unbelieving ones) to be a splendid specimen of that very disease we were joking about a month ago at the Council of the Sydenham Society."³ Hutchinson adds, "Herpes zoster of the head and face, however, is no joke to the patient."

In the same way that the clinical aspect of herpes ophthalmicus was elucidated by Hutchinson, so also the pathology of the disease was placed on a secure basis by the work of Head and Campbell,⁴ amongst whose cases were two in which the Gasserian ganglion was affected. They showed that the ganglion was in a condition of acute inflammation with profuse haemorrhages into it and its capsule. With respect to the changes in the ganglion, they showed that they depended upon the stage at which it was examined, and the severity of the disease. If, they say, the patient has died with the eruption still out, the affected ganglion will be found to be in a condition of profound inflammation. In the centre of the haemorrhagic areas the ganglion cells are absolutely destroyed. The greater the severity of the eruption, and the profounder the scarring, the more certainly will change be found in the ganglion. On the other hand, if the disease has not been severe, the inflammation may pass and leave no recognizable change in the ganglion. Thus, in one case, when the eruption was scanty and inflammation of the skin not severe, no change could be found in the ganglion 139 days afterwards. The degeneration is not limited to the ganglion cells, but spreads along the peripheral nerves and can be traced to the fine branches which pass upwards to the skin, and, in a severe case, the degenerated nerves are replaced by fibrous tissues: thus sclerosis was present in one of Head and Campbell's cases thirty days after the attack, and is, of course, permanent.

These pathological findings are in accord with clinical findings in the cornea, for in many mild cases no discoverable impairment of sensation of it occurs, whilst in severe cases loss of sensation may be sufficient to lead to the development of neuro-paralytic keratitis, though this is rare.

ITS CLINICAL ASPECTS

Herpes zoster appears to be an acute specific disease, some product of which shows a selective activity for the ganglion cells; but, somewhat surprisingly, an eruption which is indistinguishable from it may also be produced by so strictly local an exciting agent as a trauma. General paralysis and tabes dorsalis predispose to its occurrence: de Schweinitz has seen it produced during the war by anti-typoid inoculation, and arsenic is reputed to cause it. It starts with a prodromal period with fever, three or four days before the rash appears; and shows so close a parallel with anterior poliomyelitis that it has been suggested that posterior poliomyelitis would be an appropriate name for it.

With regard to age and sex incidence: Lodge,⁵ in twenty cases, found the average age to be 55, and Elliott, in twenty-one cases, 41. Hutchinson, however, reports a case in a child aged 4, and I have seen it at the age of 6. There seems a general consensus of opinion that it takes a more severe form in the aged. Elliott's cases showed a marked preponderance of females: this sex preponderance, however, is not borne out by Lodge's or Hutchinson's cases. Recurrences are rare; Head and Campbell find only four second attacks among four hundred cases.

The first local manifestation of the disease is pain, of a burning or lancinating character, over the area, which three or four days later will be the seat of the rash. The skin phenomena are: Hyperalgesia with increased temperature, erythraemia, vesiculation, rupture of the vesicles, cicatrization, and anaesthesia. In some cases pain and irritation are severe and persist, and some degree of anaesthesia is usual. The ophthalmic division of the fifth gives a branch to the meninges before it enters the superior orbital fissure, involvement of which may cause intense headache and vomiting, and so meningitis may be simu-

lated. The secretory fibres to the lachrymal gland come from the facial nerve, and so secretion of tears is not affected. The naso-ciliary nerve supplies a large area of the mucosa of the septum and the outer wall of the nose, and involvement of these branches is a common and unpleasant complication—it occurred in two-thirds of Elliott's cases.

The most important complications of herpes ophthalmicus, however, are those which involve the eye, and, were it not for these, the disease might be dismissed as an unpleasant and rather painful self-limited one. The ocular complications are: corneal affections, iridocyclitis, ocular palsies, optic atrophy.

From a general survey it would seem that intraocular complications occur in about 60 per cent. of cases, and Burton Chance⁶ points out that they usually come on after the eruption has passed its acme. Hutchinson first made the observation that the inside of the eye became involved only when the naso-ciliary nerve was affected, as evidenced by a rash on the side of the nose. Bowman joined issue on this point and thought there was no connexion between them, and apparently as a consequence, at a later date, Hutchinson says, "My rule ought perhaps to be modified a little. It would, I think, be quite safe to assert that the eye scarcely ever suffers much when the nose is not affected, and the severity of the eruption in the one part is usually in direct relation with the severity of the inflammation of the other."

The occurrence of corneal involvement in herpes is referred to by Mackenzie in the third edition of his famous book. The cornea becomes the seat of patchy interstitial infiltration, from which permanent opacities are common: occasionally the surface epithelium is raised to form vesicles, and so small ulcers follow. Some degree of permanently impaired sensitivity of the cornea is common, and occasionally is so complete as to lead to the development of neuro-paralytic keratitis.

Iridocyclitis is frequent, with or without corneal involvement, and may result in the development of glaucoma; on the other hand a lowered tension often occurs. F. A. Juler reports such a case⁷ where it was so low as -3, and the cornea was completely anaesthetic.

ASSOCIATED PARALYSIS OF NERVES

The first report of paralysis of one of the ocular nerves seems to be by Hutchinson in 1864.⁸

The paralysis—complete third nerve—cleared completely in a few weeks, and five years later Hutchinson says the pupil reacted normally and no trace of ptosis remained. R. B. Metz⁹ collected fifty cases of ocular palsies complicating herpes, and the third nerve, either partial or complete, was involved in 74 per cent. of them. He remarks that recovery occurred in most. J. Ramsay Hunt¹⁰ collected 158 cases of paralysis in herpes zoster of the whole body, and of them eighteen affected the third nerve.

Another important complication is optic atrophy; the first example I have come across of this is by Bowman,¹¹ in which, three weeks after the appearance of the rash, the left eye was quite blind from optic atrophy. In this case there was no paralysis of ocular muscles, and the distribution of the rash seems to have been a little unusual. It is described as being on the left half of the face, as low as the lobe of the ear, but not involving the ear, to the left temporal region, and a short distance above the brow. In one of Hutchinson's cases in which optic atrophy occurred he states that the stress of the disease had fallen on the nasal and trochlear nerves.

It would be interesting to ascertain whether optic atrophy was associated with some regular distribution of the rash. Other cases of this serious complication are

reported by Adams, in a man aged 35, by Paton, in a woman aged 67, and by Veasey,¹² in a case in which sight was lost on the seventh day of the disease. Sinclair showed a case, in a woman aged 57, at the last Congress of the Ophthalmological Society of the United Kingdom at Edinburgh. I have not come across this complication, and all the reported cases seem to have been seen only when optic atrophy was already present.

During an attack in which the cornea is affected it is not rare for nodules to appear in the sclerotic, due, it is presumed, to implication of the ciliary nerves.

Mayou¹³ reports two cases in which this seemed to have occurred. G. W. Thompson¹⁴ reports four more. He describes the appearance as small, sharply circumscribed, reddish nodules, somewhat flat and bossy, like episcleritis, the centre being translucent: they follow the corneal troubles, and persist after them. They clear up completely without disintegration or ulceration, leaving a small brown or slatey-blue discoloration of the sclera with the overlying conjunctiva adherent. Four other cases are carefully reported by Penman.¹⁵

PATHOLOGY

The pathology of herpes zoster raises the question of the occurrence of antidromic impulses.

In 1901 Bayliss produced dilatation of the vessels of the hind limb by stimulation of the dorsal roots of certain lumbar nerves, and believed that the passage of the stimuli was by sensory components of the nerve and due to their capacity for antidromic conduction: he expressed the view that afferent fibres bifurcate at the periphery, one branch going to sensory nerve endings, and one to the musculature of the arterioles. Sherrington says, "Trophic influence, in a direction the contrary of ordinary functional relationship and connexion, is very obscure. Herpes zoster seems to be a case of influence, so to speak, against the stream." It will be seen that he gives a very guarded acquiescence to the occurrence of such antidromic impulses, though such are not universally accepted.

The sequence of events in herpes zoster would seem clear: First, an acute infection producing an acute inflammation of the Gasserian ganglion, followed by pain referred along the fibres of the affected cells, and, four days later, the appearance of the rash. Ivy Mackenzie ascribes patches of vaso-dilation about the face and conjunctiva, associated with headaches, to antidromic impulses passing down the fifth nerve, and suggests that the inflammatory processes of herpes zoster are due to similar, but intensified, impulses. The connexion between the skin lesion and the nerve cannot be doubted, and if we are disposed to question the occurrence of antidromic impulses, may we not suppose that the products of the breakdown of the nerve fibres in the skin are sufficient to cause the eruption, or that the products of the breakdown of the ganglion cells themselves are conveyed along the nerve fibres to their skin terminations, and there produce the skin lesion; and in this connexion we may remark that there is ample proof that the toxins of tetanus and herpes febrilis may travel to the central nervous system by either motor, sensory, or sympathetic nerves; that is, the route may be antidromic—Goodpasture believes it is by way of the axis cylinders. I do not think the fact that herpes seldom follows injection of the ganglion with alcohol negatives these suggestions.

THE CORNEAL NERVE FIBRES

The posterior parts of the sclera are supplied directly from the ciliary nerves. Anteriorly the ciliary nerves form a plexus, from which a large number of fibres are distributed to the front part of the sclera behind the sclero-corneal junction, and from here some run forward into

the cornea, and some backward to the sclerotic itself. The corneal nerves enter this structure at about the middle third of its thickness. The fibres form an anastomosis at the limbus, from which sixty to seventy branches run into the cornea, usually losing their medullary sheaths as they enter it.

The appearance of the nerve fibres in the cornea has been made familiar by the advent of the slit-lamp. They are easily seen, but I was not clear as to how frequently they were visible. I therefore made observations, and found that, out of 130 eyes, I was able to identify nerve fibres in 115 of them. This is so large a proportion that I have little doubt that it was for want of pertinacity that I did not see them in all, and I expect it may be said that they can always be found.

It will be seen that the branchings are dichotomous, forming narrow angles, for the most part of from 15 to 30 degrees and seldom so wide as 45 degrees, the angles themselves sometimes being bridged by fibres which, it seems, have run into one branch a short distance and have then retraced their course to reach the other branch.

Head and Campbell demonstrated degeneration in nerve twigs as far as the skin in the cases of herpes, but were not able to demonstrate degenerated fibres in the actual portion of the skin examined. I wondered whether it would be possible, by means of the slit-lamp, to be sure of the absence of nerve fibres in the cornea, or of any demonstrable change in them, either in herpes zoster or in neuro-paralytic keratitis. As is to be expected, they are visible in some cases of zoster, and, whilst in several I did not see them, I did not feel able to say that they were absent. After injection of the Gasserian ganglion with alcohol I have been able to find nerve fibres in the cornea up to two and a half years afterwards, and have failed to identify any abnormality in their appearance. A source of difficulty is the fact that in such cases the transparency of the cornea is so often impaired, but it is a point which should be explored further.

It is evident that a corneal section must divide the nerve fibres of supply to an area of the cornea.

I have made observations on 37 cases of cataract in an endeavour to discover how soon the sensitivity of the part of the cornea involved in the section is restored; the time of observation varied from forty-eight hours after the operation to thirteen years. It is obvious that at first a demilune of the cornea has its nerve fibres severed, and that consequently this portion will be anaesthetic. The restoration of sensation will be found to occur from below up, and seems to vary a good deal in its progress. The first evidence will be found in the form of hypersensitivity at the angle of the demilune; the earliest date I have a note of this is the sixteenth day after section. Sensitivity now spreads upwards gradually, but I have no note where it was complete at the end of six months—the earliest time where I thought this might be said to be the case was ten months. On the other hand I have notes of cases where, after 1 year, 1½ years, 2½ years, 3 years, and 4 years, sensation still was a little impaired, and after much longer periods the line of section of the cornea and just below it will be found to be a little dull, and I doubt whether full sensation is ever restored to this area.

In this connexion I should like to refer to Tudor Thomas's beautiful work and, in particular, to his paper to the Royal Society last year, in which he made observations on the return of sensitiveness in corneal grafts. He showed that, of 29 successful grafts, 21 became sensitive, and 8 did not. The main points that he brings out are that, where the graft became vascularized, sensitivity was recovered and the graft was in great measure opaque; thus he says, "Of 29 grafts, 21 became sensitive and 8 were insensitive. Not one of the sensitive grafts was clear, and in most cases the opacity was pronounced; each sensitive graft was found to have become

vascularized." And again, "Of 7 that remained insensitive, 5 were clear, 1 was opaque, and 1 was partially opaque, after an average time of thirteen months."

The margins of the graft become sensitive before the centre, which is in accord with my findings and with what would be expected.

NEURO-PARALYTIC KERATITIS

Leslie Paton, with characteristic industry, discusses seven possible explanations of the development of neuro-paralytic keratitis,¹⁶ and comes to the conclusion that no complete and satisfactory explanation is forthcoming. I do not propose to discuss an eighth possible explanation.

The chief controversy ranges round the question of the existence of trophic nerves, the destruction of which is responsible for the ulceration. Sherrington says, "Every nerve and nerve centre that can influence the proper and specific activity of a tissue possesses in so far a trophic influence on that tissue. If by trophic nerve be understood no more than this, all nerves are trophic nerves. That, on the other hand, any nerve directly modifies the nutrition of a tissue independently of inducing vascular changes in it, or influencing those particular metamorphoses of material for which the tissue is the machine specifically adapted, there is no proof." Duke-Elder suggests, in his recent excellent book, that the formation of histamine in the tissues is under the control of impulses passing antidromically down the fifth nerve; when therefore this control is eliminated the changes in the blood vessels and capillaries of the tissues, the so-called triple response of Lewis, no longer take place, and the so-called trophic changes in the tissues develop as a consequence.

Whatever may be the truth in this regard, there are certain facts that are clear.

Anaesthesia of the cornea alone will not produce ulceration, for, if the cornea be completely protected by a tarsorrhaphy, it does not develop. Further, after it has developed it is surprising how quickly healing takes place if tarsorrhaphy be done. Again, although, as has been said, a corneal section causes anaesthesia, I have not seen an ulcer arise on this account, though it is well known that exposure without anaesthesia readily produces ulceration. The patient who lies in a semi-conscious condition with the lids only partly closed, so that the lower part of the cornea remains exposed, is liable to ulceration of the exposed area. The patient with prominent eyes from Graves's disease or an animal with prominent eyes, such as the Pekinese dog, is predisposed to corneal ulceration from exposure. No doubt the insensitive cornea leads to a reduced reflex secretion of tears and frequency of blinking, and so provides an additional factor. My belief is that, when the ganglion is destroyed, the lids should at the same time be sewn together. I do not forget that, with care, no serious involvement will take place in a good many instances, and where it does occur, in spite of bandaging, I believe it is either because the patient's cornea comes into contact with the dressing, which, not being felt, is not resented, or that the patient rubs his eye. Nevertheless, tarsorrhaphy is so easily done—if done neatly it will not show the slightest deformity of the lids after separation—and is so certain in its results that I believe it should be done as a routine. My practice is to make raw the juxtaposed edges of the lids for about a quarter of an inch and a little to the outer side of the middle, avoiding the lash-bearing margin, and to join them by two silk stitches. I believe the bridge should be maintained for six months, but even after this period ulceration may take place on separation, and compel a resuturing.

I should now like to refer shortly to a special type of ulceration in which I believe a neuropathic element exists. Cases of it have been reported by Messrs. Neame, Balfour Barrow, Herbert Fisher, de Courcy, and myself.¹⁷ During the war we were all familiar with the curious milky-white

coagulated appearance of the conjunctiva which resulted from "mustard gas" poisoning. Now, many years after exposure to this gas, one still sees a condition which counterfeits very closely the appearance that was produced by the gas at the time of exposure. There is the same milky-white appearance of the conjunctiva, over which vessels run, producing a marbled effect. These vessels are in great measure characteristic, in that they are of a very irregular calibre, being now narrow, and now becoming widened out into broader, irregular channels, only again to narrow down. In some cases no further lesion occurs, but in some the cornea becomes ulcerated, and this may occur for the first time ten or twelve years after exposure to the gas. Ulcers extend deeply into the interstitial tissue, separating its layers, destroying much tissue, sometimes producing hypopyon, showing little tendency to heal, and leading finally to extensive and dense nebulae. A striking feature is the mild degree of the symptoms produced; pain, lachrymation, and photophobia being conspicuous by their almost complete absence. That these ulcers are due to the gassing no one who has seen them will doubt, and it seems to me that the clinical course they run is suggestive of a neuropathic cause for them—a suggestion which gains support from the fact that tarsorrhaphy helps markedly in their healing.

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The eye receives its sensory innervation by the first or ophthalmic division of the fifth cranial nerve. Afferent impulses pass this route to the Gasserian ganglion, thence by the central root to the pons. Here some of the fibres end in the chief sensory nucleus, but a mass of fibres descends through the pons and medulla to the upper cervical segments of the spinal cord. These form the so-called ascending or spinal root of the fifth nerve, and make connexions with a nucleus—the substantia gelatinosa of Rolando, which extends in a corresponding manner from the pons to the upper cervical cord. From the sensory nuclei a second afferent neuron arises which decussates to form the trigeminal fillet, passing on the lateral aspect of the mesial fillet, and so to the optic thalamus, whence a third afferent neuron passes via the internal capsule and corona radiata to the cortex.

The fifth nerve fibres are thus exposed to injury over a very wide area; they may be damaged in their supra-nuclear path, at the level of the nuclei, and in their peripheral distribution. A constant result of damage to

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