DISTURBANCES OF SIGHT RESULTING FROM INJURIES WHICH DO NOT INVOLVE THE EYES.

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Introduction :

Theoretically this section should consist of two distinct parts, the first referring to secondary effects in the eyes produced by injuries of organs or tissues not primarily concerned with vision; and the second with the effects of injury on the extra-ocular parts of the visual system. Actually, however, there is very little to be said under the first heading. Loss of blood may cause serious disturbance of sight, and is the most important general bodily disturbance to be considered here. Diseases of many other organs affect the eye secondarily but, with the possible exception of those involving both of the kidneys, the same cannot be said of injuries.

Under the second heading we are chiefly concerned with injuries of the head, and these in different ways may affect the acuity of vision or cause defects in the visual fields, or disturb the harmonious conjugate action of the two eyes. Finally, hysterical disturbances of vision, associated with injury, must not be overlooked.

General Bodily Injury.

Loss of blood.

Blindness or amblyopia may result from loss of blood. Severe ocular symptoms rarely, however, follow traumatic hæmorrhage (as from war injuries) but more commonly result from intestinal or uterine hæmorrhage, and in such cases are likely to have been preceded for some time by severe secondary anæmia, due to repeated smaller losses of blood. The discs are hazy at first and later become atrophic. In some cases slight improvement of sight eventually occurs. In rare cases cerebral thrombosis may occur as a result of great loss of blood and if the posterior cerebral artery, or its branches are affected homonymous hemianopia or cerebral blindness may result. Some years ago I had under my care an artist who had narrowly escaped bleeding to death from a wound of the radial artery; after recovery from the hæmorrhage he was left with complete homonymous hemianopia, which quite disabled him from following his occupation.

Head Injuries.

Concussion.

Serious disturbances of vision may result from head injuries, whether these involve concussion, fracture of the skull, or penetrating wounds. During the last war shell explosions occasionally caused "concussion-blindness." This was an hysterical phenomenon which will be referred to later. More common in civil life as a concomitant of cerebral concussion is photophobia. Though usually moderate in degree this may be one of the most pronounced symptoms and may persist after all other symptoms of concussion have passed away. While mild photophobia is a common hysterical or neurasthenic symptom, the photophobia associated with concussion may have a basis in fine organic changes either in the retina or in the cerebrum.

Fractures of the skull.

Fractures of the skull may involve the optic nerves in several ways. Immediate blindness of an eye may result if the fracture passes through the optic

foramen and there is sufficient displacement of the bones or sufficient hæmorrhage to crush the optic nerve. The pupillary reflex to light is lost at once in the affected eye and optic atrophy is apparent in a few weeks. It sometimes happens that both eyes are affected simultaneously. Fractures through the optic foramen are particularly liable to result from severe blows or falls on the frontal prominence, and may then be accompanied by fracture of the orbital plate. In other cases, fractures of the sphenoid involve the optic foramen. Recovery of any vision is unusual. When it occurs the recovery is usually in the upper part of the field.

Intracranial Hæmorrhages.

In cases where there is no injury of the optic nerve, the hæmorrhage associated with a fracture of the base of the skull may infiltrate along the sheath of the nerve. It would perhaps be more exact to say that this occurs when a cerebral vessel has been damaged, and has given rise to sub-arachnoid hæmorrhage. In such cases papilloedema results. It may be unilateral or bilateral and is usually associated with amblyopia and restriction of the visual field. A moderate degree of recovery is the rule, but some impairment of vision remains permanently. A young man who had a fall from his bicycle was admitted to hospital unconscious and found to have a fracture of his skull. After recovery of consciousness he complained of some dimness of vision in his left eye; it was then found that he had papilloedema in the left eye with constriction of the visual field. No recovery of vision ensued during many weeks that the patient was under observation. It may be noted that a similar disturbance might occur without fracture of the skull, since it is found in cases of spontaneous sub-arachnoid hæmorrhage.

Arachnoiditis and other delayed effects.

Finally, in rare cases, vision becomes impaired as a delayed effect—several months—perhaps a year—after a head injury.

This delayed effect which has long been recognised, but not explained, is probably to be accounted for by arachnoiditis. In some instances callus may cause pressure on an optic nerve some weeks after a fracture, but more commonly the visual impairment does not begin for months. The arachnoid membrane becomes thickened and as a result of contraction of the infiltrating fibrous tissue it binds down one or both optic nerves or the chiasma to the base of the skull, and optic atrophy results from the pressure. In other cases a collection of fluid is enclosed within adherent arachnoid tissue, and this cyst may cause symptoms by pressure on the chiasma. The field disturbances in this group of cases are very variable; in some instances bi-temporal defects show that the acting lesion must be at the chiasma, but quite often a central scotoma is the first disturbance, and both eyes may be affected in this way. The discs show increasing pallor which goes on to atrophy. Blindness or severe amblyopia may ensue in the course of a few months. By freeing the optic nerves the progress of the visual disturbance may be checked and some restoration of vision brought about. Apart from operation, the outlook for preservation of useful sight is poor.

Penetrating wounds.

Penetrating wounds of the skull and brain, such as gun-shot or shrapnel wounds, if they affect vision, most commonly give rise to partial or complete homonymous hemianopia. The extent and situation of the defects depends naturally on the site of the intracranial lesion. Blindness may result from a horizontal transverse wound through the temple, the optic nerves or chiasma being destroyed. In a case in which a piece of shrapnel had come to rest on the floor of the skull to the left of and slightly behind the pituitary fossa, the patient had complete right homonymous

hemianopia, and it seemed most likely that this was due to division of the left optic tract.

In cases of injury of the cerebral hemispheres by penetrating missiles the optic radiation may be damaged, and complete or partial hemianopia result in consequence. The optic radiation enters the hemisphere on its medial side and takes up a position just behind the posterior end of the internal capsule: from there its fibres spread out fanwise, but in two main divisions. The upper division traverses the parietal and occipital lobes to reach the upper lip of the calcarine fissure, while the lower sweeps forward and downward into the temporal lobe and thence backward through the occipital to the lower lip of the calcarine fissure. Thus the radiation as a whole is deeply placed in the hemisphere and injuries which involve it are likely to cause extensive damage to the hemisphere and so are usually fatal. Moreover, the radiation for a great part of its course lies in contact with the wall of some part of the lateral ventricle, and as many of the wounds we are considering are infected, those which involve the radiation, if not immediately fatal, often lead to fatal results within a few days from ventricular infection. If the patient survives, homonymous hemianopia is likely to be associated with hemiplegia and partial hemi-anæsthesia.

In cases of wounds of the occipital lobe the visual cortex around the calcarine fissure may be injured or destroyed without much damage to other cerebral structures, and so the proportion of cases of survival is greater. Wounds of the occipital lobe give rise to homonymous defects and these may occasionally be of limited extent. Some interesting examples from his experience in the last great war were given by Holmes in his Montgomery lectures (1919). Injury of the tip of one occipital pole gave rise to homonymous paracental scotomata bounded by the vertical meridian of the field. More extensive injuries gave rise to larger homonymous defects and the periphery of the half field was evidently associated with the anterior part of the calcarine cortex.

Other curious disabilities resulting from wounds, such as word blindness and loss of spatial perception have been recorded, but are rarities which need not be discussed here, except to say that they usually indicate a cortical lesion in the lower part of the left parietal lobe (angular and supra-marginal gyri). A loss of visual "topognosis" (recognition of a known place and ability to find one's way about in it) has been considered to result from damage to the visual commissural fibres in the posterior part (splenium) of the corpus callosum.

Cerebral abscess.

Small penetrating wounds or graze wounds of the skull often lead to cerebral abscesses and these may in turn produce visual disturbances either in consequence of papilloedema or by interference with some part of the optic radiation.

Hysterical Disorders of Vision.

Hysterical blindness or amblyopia may occur in association with many kinds of trauma, but especially those involving explosions or flashes, e.g. the explosion of a shell in front of a soldier's face at a time when he has been well protected by a trench, and has not received serious bodily injury. The first essential in the diagnosis of such a condition is to make quite sure that there are no signs of physical injury of the eyes. The pupil reflexes remain normal and some dim vision is frequently retained. Blepharospasm may be pronounced and it is important to make sure that there is no sand or grit in the conjunctivæ. In the last war these cases were treated by hypnotism and suggestion with considerable success. Where the blindness is of one eye only it is usually found that the

patient with both eyes open does not see in the corresponding half of the visual field. In fact, he may be able to see over a wider field with only one eye open than with both. This may point the way to convincing treatment by methods of persuasion and suggestion.

Hysterical symptoms in general are likely to get worse the more they are taken notice of, and in a case of hysterical amblyopia the visual field frequently diminishes as the examination goes on, so that a spiral or helicoid chart is obtained. While this is usual in cases of hysterical amblyopia, it may also be of diagnostic help in cases of doubtful hysterical disorders of other kinds.

Disorders of the Ocular Musculature.

In addition to the signs and symptoms arising from injuries which interfere with vision, valuable information may be obtained by examination of the musculature of the eye. The six extra-ocular muscles share between them the services of the third, fourth, and sixth cranial nerves, while the intra-ocular musculature is controlled by the third nerve, stimulation of which contracts the pupil, and by branches from the cervical sympathetic, stimulation of which dilates the pupil. The upper lid is also supplied by the same two nerves. Pupil signs are easily observed and are of considerable importance in head injuries. They will therefore be considered first. Their importance is shown from the observation of Blakeslee (*Arch. Ophthalm.* 566. Nov. 1929) who states that they occurred in 65 per cent. of all skull fractures observed by him, and of this number, 40 per cent. died, whereas of the 22 per cent. of cases without any eye signs, only 8 per cent. died.

In assessing the significance of pupil changes, there are several fallacies which have to be ruled out. When the pupils are unequal in size the condition is in very rare cases congenital, much more commonly it may be due to a blow on the eye (e.g. from a cricket ball, perhaps years previously) which has caused rupture of the sphincter and permanent dilatation of the pupil. Old iritis with adhesions of the iris to the lens may cause the affected eye to have a smaller pupil than its fellow, with impaired reaction to light, while in chronic glaucoma, the pupil of the affected eye is larger than its fellow—if the disease is not binocular—and again it may not react to light. The Argyll Robertson pupil is too well known to need more than mention, but the possibility of its presence should be borne in mind in examining cases, as should also the previous use of mydriatic or myotic drops or drugs (e.g. atropine, eserine). In order to determine whether pupil inequality is due to a lesion of the third cranial nerve or of the sympathetic, a drop of 5 per cent. cocaine should be instilled into each eye, and the pupils re-examined in half an hour. If the case be one of sympathetic paralysis in one eye, the inequality will be more marked owing to the failure of the pupil to dilate in the affected eye. In third nerve paresis, the inequality will be, if anything, less marked. If cocaine is not available, a similar result can be obtained by examining the patient in a partly darkened room, but the effect is not so striking. In examining pupil reactions, diffuse light—e.g. daylight or the light from an ordinary torch—should be employed, because focussed light, such as the narrow beam which is produced by an electric ophthalmoscope, may fall on a portion of the retina which is outside the pupillo-motor area and so fail to elicit a reaction. In addition to testing the direct reactions to light and accommodation-convergence, the consensual reactions must be looked for too.

Pupil signs usually develop simultaneously with the injury, but may take 24 hours or longer to become manifest. In some cases they vary their appearance from hour to hour, thus, according to Manley (*Penn. Med. Jl.* vol. 36 July 1933

p. 758) slight disturbance of a semi-stuporose patient may alter the size of either of the pupils.

So far as general causes are concerned, dilatation of the pupil is brought about by fear, alcohol, shock and syncope. Local causes, apart from direct eye injury, will include a paralysing lesion of the third nerve, and an irritative lesion of the cervical sympathetic in any part of its course. More widespread lesions may also cause dilatation of the pupil, especially extradural or subdural hæmorrhage, when it is usually the pupil on the affected side which is dilated. From the prognostic standpoint it is worth noting, that of patients who have sustained a head injury and show widely dilated pupils which do not react to light or dark, 95 per cent. die; whereas of those who show widely dilated pupils which react to light, only 30 per cent. prove to have sustained a fatal injury (Blakeslee). It is uncommon in head injuries to meet with contracted pupils which are inactive to light, but the sign when it occurs is an important one. If it is not due to sympathetic irritation, produced say by injury of the roots of the cervico-sympathetic or brachial plexes, it frequently indicates a basilar hæmorrhage which is often fatal. Affection of the extrinsic eye muscles—apart from direct trauma to the orbit—is not so common as one would expect in head injuries and was present in only 6 per cent. of the cases reported in Blakeslee's survey. The severity of the affection varied from slight weakness to complete paralysis of the affected muscles and the same was true of any ptosis which might be present.

The localising value of extra-ocular muscle palsies is variable, being most marked when it is seen in association with other paralyses. Neurologists are well acquainted with four syndromes of this type, one of which (Weber) may be briefly alluded to here. It consists in paralysis of one-third nerve with hemiplegia of the opposite side of the body, and occurs in connection with a lesion at the upper end of the pons or lower end of the crus, which involves the roots of the third nerve and the pyramidal fibres. Paralysis of the external rectus without other neurological signs might be due to a basal fracture involving the apex of the petrous portion of the temporal bone, but is more commonly due to any lesion which may tend to press the brain stem downwards into the foramen magnum. Wolff has pointed out that the sixth cranial nerve in its course through the skull has to pass over the sharp upper border of the petrous temporal at almost a right angle. If, therefore, the brain stem is displaced downwards, the nerve is pulled against this sharp edge with resulting interruption of conduction of impulses along it, and therefore paralysis of the external rectus.

A few words should be said about the methods employed to detect paralysis of the extra-ocular muscles. If the patient is comatose, all that can be done is to note the relative positions of the two eyes. If he is able to co-operate, however, he should be asked to follow the examiner's finger or a light with his eyes, into the nine cardinal directions of gaze (up, down, up and to the right, down and to the right, etc.) when any deviation of one or other eye can be noted. He should then be given a red glass to hold in front of the right eye and be asked to follow a light in the same nine directions and state the relative positions of the red and white images. From his answers, a "diplopia chart" can be constructed which will be helpful in working out the muscle or muscles affected.

In conclusion, two fairly common sources of error should be mentioned. One is the outward deviation which occurs, in the course of time, to a blind or an unused eye and might lead to a diagnosis of paralysis of the internal rectus. The other is the presence of a concomitant squint, or the deviation caused by an unsuccessful operation for this condition.