Manual elevation secures a very pleasing effect and it is proposed to do a fascia lata graft to complete the plastic repair by elevating the canthus.

The left eye was next operated on in June, 1942, this time by a pedicle graft from the upper lid inserted into the lower lid. The normal outer third of the lower lid with its eyelashes has not been removed, the fibrous band and notch only being excised. The result is fairly satisfactory and there seems no need in this case to resort to transplantation of cartilage.

This case seems to be the least severe of the three cases under review.

I am obliged to Dr. Alan Booth for the following report on the X-ray appearances:—"The zygomata of the patient are very poorly developed and can be seen to come to a point in the view of the base of the skull. The poor development is also seen in the 'nose-chin' view."

UNILATERAL INVOLVEMENT OF THE OPTIC NERVE IN HEAD INJURIES

BY

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CASES of traumatic optic atrophy are to be found in every surgical ward. Those types of accident most frequently producing it are motor accidents; falls or blows on the head received in industry; and, in these days, war injuries; for example, blunt injuries sustained in air attacks from falling timber and masonry.

The lesion is uni-ocular and therefore prechiasmal.

The degree of violence may be so great that the patient suffers a fractured skull and lies unconscious for some weeks, or may be so slight that the patient is only momentarily dazed, with perhaps no external signs of violence at all. A remarkably simple injury was that in Praun's case in which optic atrophy resulted from a knock on the eyebrow with a potato!

Twenty-two patients were investigated by us in the Tennent Institute, but the records of two were omitted as one was complicated by a meningitis, the other by a positive Wassermann reaction. In some of the twenty cases remaining, the records were incomplete owing to the fact that an ophthalmic surgeon had not been summoned at an early period.

The clinical picture is a simple one. Soon after the accident each patient complained of a reduction of vision in one of his eyes. Seven of our cases were found to have no light perception; two had light perception only; three could count "fingers" at one

metre; and eight possessed a visual acuity of 6/18 Snellen, or less. The pupils of the affected eyes reacted to light directly, more or less according to the degree of damage to the nerve fibres; while in all of them the consensual reaction remained. In the cases showing partial optic atrophy, central vision was depressed, and in two there was a contraction of the field with an-insular scotoma.

Externally, apart from the pupillary anomaly mentioned, nothing could be seen. Internally, on the other hand, the nerve head sooner or later revealed a pathological state. Fuchs says the abnormality may not become apparent for as long as eight weeks. According to Parsons the atrophy reveals itself in from fourteen to twenty-one days. In our cases the average length of time was seventeen days; the shortest four days.

The earliest phenomenon (it is well known) observed ophthalmoscopically is pallor of the disc, in whole, or in part. The papilla retains its sharp border, the pigment and scleral rings being unchanged, and the lamina cribrosa in many cases easily discernible. All this was well seen in our own cases.

It is difficult to understand why the pallor of the disc occurs at all. One would expect in simple interruption of the conductive power of the nerve fibres, say by experimental section, that the resulting degeneration would advance up the afferent tract to the brain away from the site of the injury, and that those parts of the neurones nourished by the unharmed ganglion cells lying in the retina would not degenerate at all. Yet pallor of the papilla is observed. Probably there are additional factors in the colour change, for example, an accompanying gliosis, or an atresia of the little vessels supplying the disc, which would produce an ischaemia. We have seen cases where the entire disc had the appearance of a uniform bluish-white atrophy, although we knew from clinical evidence that some of the visual fibres were still functioning; so we can say that the picture does not depend entirely upon a degeneration of the optical fibres.

Leinfelder (1940), experimenting with cats and monkeys, made section through the optic tract *above* the chiasma. After ten days there was no sign of any degeneration of the axis cylinders in the nerve below that point. After seventy-five days there was a minor degree of atrophy only. On the other hand, section made *below* the chiasma, nearer the retina, in the region of the canal, led to complete degeneration of the axis cylinders throughout the entire nerve after the minimum period of time, *and both above and below the site of the lesion*. The atrophy, therefore, has been proved to advance in both directions, with the further important points that the nearer the injury is to the ganglion cells the earlier and greater will be the retrograde degeneration as observed at the optic disc.

The other change observed ophthalmoscopically is that occurring in the vessels. This change is restricted to the arteries, the veins being unaffected. Eleven of our twenty cases showed arterial changes, the time of their first onset ranging from one to six months. First of all, those tiny central vessels seen tracing a course across the pink background of the disc and fundus began to disappear. It was not until some weeks later that the larger arteries were seen to be affected. They had become narrower and straighter than in the sound eye, having changed imperceptibly. In a few cases only, we noticed variations in calibre.

Like the question of the pallor of the disc, it is not known why these vessel changes occur. Two probabilities come to mind. On the one hand, it may simply be the result of a post-degenerative gliosis. This we consider the more likely. Wolff points out in his text book that the optic nerve, especially the head, is extremely rich in neuroglia. It is known too that glial fibres fill out the meshwork of the septa of the atrophic optic nerve in *tabes dorsalis* (Stargardt). Abelsdorff (1928) says that neuroglia always proliferates when nerve fibres denegerate, just as connective tissue proliferates, a statement supported by Behr. The assumption, then, is that as the glia proliferates the neighbouring vessels will be drawn upon and pressed upon, and their lumina slowly occluded. Such effects on the septal and central vessels would be extremely difficult to observe in a day to day examination, and would account for the lack of any obvious changes.

On the other hand, these vessel changes may be vasomotor in origin. Duke-Elder (Text-Book, Vol. II, p. 672) believes that the efferent fibres of the nerve are vasomotor in function; although Leinfelder, using Bodian's silver stain, could find no evidence of their presence. We incline to the view that they exist as the slenderest fibres of the nerve (Duke-Elder), although according to Ingvar they run in the periphery of the nerve (Ingvar, quoted by Wolff). Parsons too suggests that they are fairly numerous. It may well be, therefore, that these elusive fibres run as a solitary bundle, a state of affairs more characteristic of the central nervous system—the pupillary fibres, for example, have been shown to run as a dorso-lateral bundle (Henschen). If the fibres were indiscriminately mingled, then in a lesion of the nerve it is likely that they would be damaged to an extent equal with the visual and pupillary, thus producing a degree of *dilatation* of one or all of the central vessels, a feature which never occurs. We think it more probable, however, that these fibres run as a solitary bundle. If that be so then a lesion in a neighbouring part of the nerve might cause a secondary irritation of the efferent fibres, producing thus a *contraction* of the vessels, similar to that which

occurs. Whatever the cause may be, there can be little certainty of its origin until more is known of the intimate nature of the optic nerve fibres.

As regards lesions of the retina, we discovered none in any of our cases, a finding which is almost generally accepted. A few contradictory cases are noted, however. Favory (1931), for instance, describes two cases showing papilloedema and peripapillary haemorrhages in subjects put under observation about two weeks after the injury. He says these lesions are due to compression of the central retinal vein at its exit from the nerve by an intervaginal haematoma, but notes no evidence in support.

The picture we have just described, then, a round pale, clearly outlined disc with narrowing of the arteries, is that typical of a so-called simple or primary atrophy.

We have given above the clinical picture of a lesion whose pathology is not clearly known. Many people have drawn conclusions with regard to it from post-mortem cases. But, and we would like to emphasize this point, the conditions found in fatal cases are not strictly relevant. In most of them the patients die without recovering consciousness, and the state of their vision and fields of vision must be a matter of conjecture, based on the state of the pupillary light reactions. For our present purpose, therefore, we have to reject a good deal of the current literature on the subject. We have based our own observations entirely on surviving cases, results having been obtained as a result of clinical and X-ray investigations.

It is a well-known fact that X-ray photography of the optic canals presents certain difficulties and is open to fallacious interpretation. The fact that no fewer than twenty-seven different methods have been devised suggests that, so far, none is completely satisfactory. After some experience of the different methods, using single plates, we adopted the method of stereoscopic photography. The following technique was used :—

X-RAY TECHNIQUE FOR STEREOSCOPIC EXAMINATION OF OPTIC FORAMEN AND CANAL

Anode—film distance ...	28 ins.
K.V. ...	72
M.A. ...	150
Time ...	3/10th sec.
Tube Shift ...	2 cms. each side.
Size of films used ...	6½ ins. x 4¾ ins.

The head is placed with nose and forehead toward the film. The head is rotated through 35° and 40° to right and left sides in turn. The chin is then allowed to make contact with film support so that the radiographic base line is at an angle of 30° and the side

of the face being examined is parallel to the film. The nose, eye-brow, zygomatic bone and chin should be in contact with the film support.

Centre $2\frac{1}{2}$ ins. above and behind the opposite external auditory meatus. The stereoscopic films are taken one with a right shift of 2 cms. and the other with a left shift of 2 cms. from the centring point.

A large enough cone must be used to cover each film.

The results of our cases we give below in tabular form :—

TABLE A

Name	V.A. on last examination	V.F. defect	Type of film	Traumatic interference with optic canal	Associated injuries of head
*1. Robert G.	6/18	Superior hemianopia	Straight Stereo.	No No	Bruises over malar bone. Cut over eye-brow, unconscious on admission. Head-on injury
*2. Thos. McD.	No P.L.	—	Stereo.	No	Bruise at inner nasal angle. Head-on injury
*3. Jas. McQ.	P.L.	—	Straight	No	Fracture of infra-orbital margin centrally. Unconscious on admission. Head-on injury.
4. Walter W.	6/60	Upper outer quadrant	Stereo.	No	Fracture side of skull. Unconscious for short while only
5. William J. (age 19)	6/18	Peripheral contraction	Stereo.	Yes	Fracture orbital margin up and out. Lacerated wound above the eye
6. William J. (age 36)	Fingers	Upper nasal quadrant. Scotoma insulare present	Stereo.	Yes	Bruises on nose and side of head. Unconscious for a few minutes
7. Robt. H.	6/12	Scotoma insulare in upper nasal quadrant	—	—	Fractured skull
8. Alex. B.	Fingers	Peripheral contraction	Straight	Yes	Fracture parieto-occipital region. Concussion.
9. Cissie B.	1/60	Peripheral contraction	Stereo.	Yes	Bruises on temple

TABLE A—continued

Name	V. A. on last examination	V. F. defect	Type of film	Traumatic interference with optic canal	Associated injuries of head
10. Andrew S.	5/60	Quadrantic upper outer	Straight Stereo.	No No	No bruising or scarring. Line of violence unknown
11. Mima K.	6/36	Peripheral contraction	Stereo.	Yes	Fracture fronto-parietal region
12. Wm. F. ...	No P.L.	—	Stereo.	Yes	Lacerated wound side of eyebrow
13. Joseph K.	No P.L.	—	—	—	Fractured frontal bone. Shocked on admission, slight cerebral irritation, 2 months later relapsed and died. P.M. refused
14. John S. ...	No P.L.	—	Straight	No	Unconscious for a few minutes
15. Wm. O'D.	6/36	Peripheral contraction	Straight	No	Bruises on brow. Semi-comatose for a few hours
16. Robt. P.	Fingers	Peripheral contraction	Stereo.	Yes	Fracture of frontal and middle fossa. Wounds on temple
17. Rosetta H.	No P.L.	—	Stereo.	Yes	Superficial scratches. Shock
18. Thos. McT.	No P.L.	—	Straight Stereo.	No Yes	Fractured frontal. Unconscious for some days
19. Stanley M.	No P.L.	—	Stereo.	Yes	Wounds on temple. Unconscious for 14 days
20. Robert T.	P.L.	—	—	—	Wound left temple

* In the majority of our cases—for the most part apparently affected in the canalicular part of the nerve—pallor of the disc was observed in about seventeen days. The nearer the lesion is to the papilla, the earlier is the pallor noticed (Leinfelder), so that in those cases showing pallor in about seven days (e.g. Cases No. 1, 2, 3, Table A), the lesion may be said to lie anterior to the canal and nearer to the disc than in the others. The explanation of this very probably depends upon the line of the force applied to the head, for it is obvious that a blow whose direction corresponds with the long axis of the canal (head-on injury); would be less likely to damage it than a blow striking it at an angle. So far as we can ascertain, this would appear to account for the lack of canalicular damage and the early onset of pallor in Cases 1, 2 and 3, for as far as we can ascertain, these three, unlike the others, were of the "head-on" type.

The X-ray reports were supplied by Dr. Scott Park, Senior Radiologist to the Western Infirmary.

Right Eye—Fracture.

Left Eye—Normal

FIG. 1.

FIG. 2.

Left Eye—Straight film, reported within normal.

Left Eye—Fracture.

Right Eye—Normal.

FIG. 3.

Right Eye—Fracture.

Left Eye—Normal.

Fig. 4.

Three of these cases tabulated above, typical of the condition, and each showing traumatism of the bony canal, we note at greater length, giving the stereoscopic pictures:

Case 19.—Stanley M., aged 15 years. Fig. 1.

This little boy was knocked off his bicycle by a motor lorry, and was struck on the right eyebrow and temple. He was unconscious for fourteen days. He had superficial wounds, but no skull fracture. On recovering consciousness he discovered that he had lost the sight of his right eye.

Examination three months after the accident: R.V.=No P.L. L.V.=6/5. O.E.R. Complete atrophy of the nerve. Narrowing of the arteries. X-ray report: Appearances suggest a fracture inferiorly of the right optic canal (Scott Park).

Case 18.—Thomas McT., aged 28 years. Figs. 2 and 3.

The patient was admitted to a Birkenhead Hospital having fallen on to the dock from his ship. He was unconscious for several days with a fractured frontal bone. He has not been able to see with his left eye since.

Three months after the accident he was examined in the Tennent Institute. R.V.=6/6. L.V.=No P.L. O.E.L. Simple optic atrophy. Complete pallor. Narrowing of the vessels very marked. X-ray report (straight film, fig. 2): The left optic foramen appears to be within the normal, the rim being complete (Scott Park). We were not satisfied and had a stereoscopic film taken. X-ray report (stereo, fig. 3): Compared with the previous examination the left optic foramen appears considerably flattened and has almost certainly been fractured (Scott Park).

Case 17.—Mrs. Rosetta H., aged 71 years. Fig 4.

This old lady received a glancing blow on the head from falling timber in the March air-raid on Glasgow, and had a very narrow escape. She was rescued in a dazed condition. The day following she discovered she was quite blind in the right eye. No skull fracture found.

Examination three months after injury: R.V.=No P.L. L.V.=6/12 with glasses. O.E.R. White atrophic disc. Some variation in calibre of the vessels, especially the lower temporals. X-ray report: The foramen on the right side is greatly distorted, and this is presumably the result of trauma (Scott Park).

On summarising the X-ray findings of these three cases, and the others in Table A, we get the following figures:—

In 7 straight films, 6 intact canals, 1 traumatised canal.

In 13 stereo films, 4 intact canals, 9 traumatised canals.

The difference, even in this small series, is considerable. Straight X-rays must obviously be rated as unreliable, and findings based on them treated with reserve. It appears fairly certain

in fact that part at least of the cause of the atrophy lies in injury to the optic nerve by displacement of the bony wall of the optic canal. This of course is not a new idea, but modern radiographic technique makes it more possible for the radiologist to provide evidence of the traumatism.

Callan (1891) and Rawling (1904) were amongst the first to voice the opinion that the optic canal acts as a *locus minoris resistentiae*—so that a blow on any part of the head will expend its major force there. Rollet and Levy (1930), by radiographic investigation—jumping forward almost a half century—came to their conclusion that total loss of vision was always caused by a fracture of the canal, although as our own investigations differ (e.g., see Case 2, Table A) we cannot agree with this. In a recent communication, F. Ridley (1942) quotes J. W. Aldren Turner, who has records of 12 cases of injury to the optic nerve in 450 cases of head injury. In all twelve cases stereoscopic X-ray photos showed no fracture of the optic foramen. The controversy may depend here upon the period of examination. Rollet, Levy and Paufigue preferred to make their investigations early, before the fissures filled up. In our own cases, this was done wherever possible, but in many cases, by force of circumstance, the films were taken later. The fissures when present had by this time filled up, the callus having ossified. The important point is, however, that the views of Levy, Rollet and Paufigue support those of Callan and Rawling.

Traumatism of the bony canal, however, is not necessarily the whole story in injury of the nerve, although it appears to be a large part of it. Few fractures, if any, ever occur without some associated haemorrhage. We must therefore consider the part played by the latter.

The optic nerve is similar in structure to the white matter of the brain. The axis cylinders have no sheaths and there are no cells of Schwann, so that there can be no regeneration. And yet the truth is that many of these cases show improved vision and fields. This can only mean one thing—the nerve fibres have not been destroyed after all, but their conductivity in some degree only temporarily suspended. Such a state of affairs could be due to an outflow of blood, the improvement which follows resulting from its absorption.

Like the rest of the brain the optic nerve is surrounded by the three meningeal sheaths, which form a *cul de sac* just short of the bulb. By reason of their extreme vascularity they are of much surgical importance. Of the three, the pia is the most vascular, and when one realises that from it countless septa pass into the nerve substance, partitioning it off into bundles, each system with its blood supply, and that haemorrhage is the most common sequel

to any head injury, bleeding into the sheath spaces or into the nerve substance itself would seem certain to occur.

Kuhnt believes that in head injuries there is frequently a rupture of the small veins crossing the intervaginal space to communicate with the central ophthalmic vein. Ballantyne (1939), too, is of the opinion that in most cases there is a rupture or occlusion of the nutrient vessels of the nerve as they cross the sheath spaces. This might be caused, he says, by changes of tension in the parts at the moment of injury, or by subsequent haemorrhage into the space. In support of this, Pallin (1934) described the presence of blood in the sheaths, tracking forwards from a sub-dural haemorrhage. But whether or not such a thing actually occurs in non-fatal cases no one is in a position to say.

One of the earliest supporters of the view that bleeding into the sheath—dissociated from any bony lesion—causes the defect was Pringle (1917). Operating upon two cases he evacuated blood from the sheath, unfortunately without benefit to the patient. Krönlein and Davies had a similar experience. Garrow, however, recalls two unpublished cases of his own. "In neither" (he writes in a personal letter) "was blood found in the orbit; the optic nerve sheath was normal in colour and in size; that is, there was no distension in either case. An incision was made through the sheath with quite negative results—no blood or other effusion. In both cases optic atrophy and blindness followed."

Pringle's work we find highly interesting, if not completely convincing. Of his series of one hundred and thirty-six cases examined post-mortem, he found ten with intravaginal bleeding on which to base his theory (not a very high figure). None of these fatal cases regained consciousness before death, and so the visual acuity and the extent of the field of vision could only be surmised. The value of Pringle's work, however, lies in the fact that in two cases which survived injury, and manifested a unilateral loss of vision, it was possible to demonstrate the presence of blood within the sheaths of the optic nerve. Pringle's view was that pressure of the effused blood was the cause of injury to the optic nerve. Unfortunately there was no possibility of obtaining any histological evidence in the operated subjects. The primary cause in that event might have been found to lie *within* and not *without* the nerve. To illustrate this, Belloni (1937) has described a case in which the patient was taken suddenly ill and died of a purulent meningitis seven days after he received his injury. The post-mortem examination revealed a fracture of the right parietal bone (the patient had complained of diminished visual acuity of the right eye) which extended into the middle fossa. But there was no fracture of the canal, and although there was blood in the vaginal spaces there was also a triangular area

of disintegrated nerve fibres and blood vessels in the nerve stalk itself, the base of the triangle being at the periphery, the point in its core.

The position, then, is as follows. In some cases the presence of blood within the sheaths has been demonstrated (Pringle), although in others no blood has been found (Garrow). The operations of Lillie and Adson (1934), showing the formation of early callus around the intracanalicular part of the nerve, without any intravaginal bleeding, make it appear likely that our X-ray appearances are quite correctly interpreted. One cannot therefore explain the whole cycle of events on the grounds of haemorrhage alone, or of fracture alone. Each or both are possibilities. Indeed in view of the improvement in some of our cases, showing (by X-rays) involvement of the canal, we can be fairly certain that the two events, fracture and haemorrhage, occur together. In a smaller proportion of cases nevertheless it seems that the entire picture is due to haemorrhage—as, for example, the occurrence of the quadrant defect in case No. 10, which would be readily explained if we suppose a parenchymatous extravasation of blood. In this type of case, the defect suggests the destruction of an isolated nerve bundle, while in those peripherally contracted, the field defects suggest a diffuseness of the lesion. In neither type, however, did we find any correlation between the field defects and the bony distortions.

Clinical and X-ray investigation, therefore, in cases of unilateral involvement of the optic nerve in head injuries elicits the following possible explanations, occurring singly or collectively:—

1. Subvaginal haemorrhage—with pressure on, or tearing of, the nutrient vessels of the optic nerve.
2. Intraneural haemorrhage.
3. Fracture, with perhaps tearing of, or pressure on, the nervous tissue.

The obvious parallel with cerebral lesions proper requires no comment.

Summarising:—

1. Twenty cases of unilateral atrophy due to head injury, none of which was fatal, were investigated.
2. The clinical picture is that of a primary atrophy, but why the disc changes its colour, and the arteries their form, is not clearly understood.
3. Current literature, with theories based on post-mortem findings, is not completely relevant. Our results have been obtained as a result of clinical and X-ray investigations only.

4. Stereoscopic photographs in nine out of thirteen cases revealed a distorted foramen on the side of the defective eye. This distortion was reported at its least definite to be "almost certainly traumatic."

5. There is a school of thought which considers that this frequent occurrence of traumatism of the canal has a mechanical basis, that the canal is, in fact, "locus minoris resistentiae." It is thought, however, that there will be no traumatism of the canal where the blow is delivered in the same direction as its long axis.

6. Clinical investigation suggests that in addition to the bony lesion, associated haemorrhage from nutrient or septal vessels, or both, plays a large part in many of the cases.

7. Haemorrhage alone may result, and such cases would of course have a slightly better prognosis.

8. As the defect cannot be recognised with certainty at an early stage, and as the possibility of parenchymatous bleeding cannot be excluded, effective treatment appears to be impossible.

We would like to thank Professor Ballantyne and Dr. Loewenstein of the Tennent Institute for their encouragement and guidance; Dr. Scott Park and Miss Hendry of the Radiology Department, Western Infirmary, for the stereoscopic pictures; and Dr. Garrow of the Royal Infirmary for notes on his two operation cases.

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