of symptoms, all of which are present in this case—symptoms which may be found independently in other morbid conditions, but which experience proves are never found in combination in any other disease. These symptoms are three—namely, loss of certain sensations, while others are retained; the presence of paralysis, with wasting of muscles; and trophic changes in the skin and other structures. Of these the most characteristic symptom, taken by itself, is the partial loss of sensibility—the "dissociated anaesthesia" of Charcot. Normally, we are conscious of three kinds of sensation in the skin—sensation of pain, of touch, and of temperature. In most conditions of impaired sensation all these kinds of sensibility are lost in about the same proportion, but in syringomyelia it is otherwise. In this disease the sense of touch is generally normal in every respect, but the sense of pain and the power of distinguishing hot and cold objects in contact with the skin are more or less completely lost. This peculiar dissociation of sensation is not easy to explain; but in view of the pathological anatomy of syringomyelia, its occurrence lends colour to the hypothesis that the paths of the different kinds of sensation in the cord are not the same, and that sensations of pain and of temperature pass through the grey matter. The distribution of the anaesthesia corresponds to the segments of the spinal cord affected, and does not, as in the case of peripheral neuritis, correspond to the distribution of the sensory nerves. In consequence of the loss of sensibility serious injuries may be unnoticed by the patient, and to this some of the trophic changes are doubtless due.

The distribution and nature of the muscular paralysis are such as we might infer from what we know of the morbid anatomy. If the lesion is in the cervical enlargement, there will be paralysis, with wasting of the muscles of the upper extremity, corresponding to the spinal segments involved, and the muscles of the trunk and even of the lower extremity may suffer in succession. More common is it to find the lower limbs indirectly involved from pressure on the pyramidal tracts, there being no wasting, but spasticity of the muscles with exaggerated reflexes. A case of this kind might readily be mistaken for progressive muscular atrophy. When, however, as in the present case, the lesion is chiefly located in the lumbar enlargement, there is simply more or less paralysis of the muscles, with atrophy, loss of reflexes, and degradation or abolition of electrical response.

The nutritional disturbances in the limbs are of various kinds and may affect any of their structures. In the present case the smaller toes are much deformed, apparently from shortening of the flexor tendons and disorganization of some of the joints, there is an indolent ulcer on the sole of the foot, reminding us of perforating ulcer, there are multiple ulcers on the leg, and a purpuric eruption on the feet. The stiffness and flexion of the fingers is, I feel sure, one of the manifestations of the spinal cord affection, though I do not find that such a condition has been previously described in this disease. A painless disorganization of the larger joints, like that described by Charcot in locomotor ataxy, has been met with in some cases, and painless fractures have been described. The present case is exceptional in the advanced age of the patient, as well as in the locality affected. Though cases are occasionally seen late in life, the great majority of those recorded have been in patients under 40.

The progress of the disease is commonly, as in the present case, exceedingly slow. It may spread upwards to the medulla oblongata and so prove fatal, but as a rule it does not directly tend to shorten life.

The treatment must be symptomatic and palliative, since we have no means of influencing the processes at work in the spinal cord. It is thus on the same lines as the treatment of the commoner degenerations of the spinal cord. The absence of pain calls for vigilance on the part of the practitioner, since the patient, feeling no discomfort, may take no notice of more or less serious injuries, with the result that troublesome ulceration may arise and be the source of various forms of septic infection.

BEQUESTS TO MEDICAL CHARITIES.—Under the will of the late Mr. Charles William Curtis, of Kearsney Abbey, near Dover, which has now been proved, the Brompton Consumption Hospital, the Cambridge Asylum, the Convalescent Home at Walton-on-the-Nase, the Victoria Hospital for Children, the Dover Hospital, the Margate Seaside Hospital, the Ramsgate Seaman's Infirmary, King's College Hospital, and the Savernake Cottage Hospital, each receive a sum of £100.

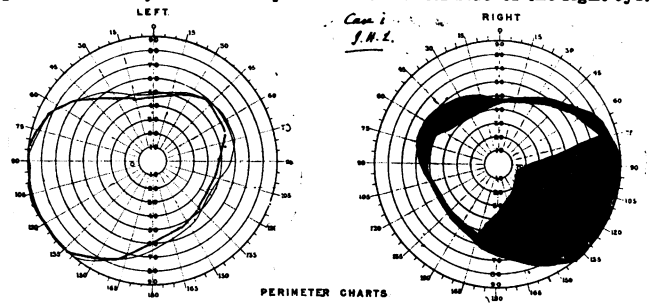
INDIRECT INJURIES OF THE OPTIC NERVE.*

BY J. JAMESON EVANS, M.D.BIRM., F.R.C.S.ENG.,
Honorary Surgeon to the Birmingham and Midland Eye Hospital.

CASES of loss of vision in one or both eyes following blows or falls on the head have been recorded by several observers. As a rule the loss of sight is complete in or one both eyes, and though Nettleship says it may be incomplete I can find no detailed record of such cases. Apart from their apparent rarity the following cases are of interest in that they seem to show that a special type of incomplete unilateral blindness is apt to result from blows in the region of the external angular process of the frontal bone.

CASE I.

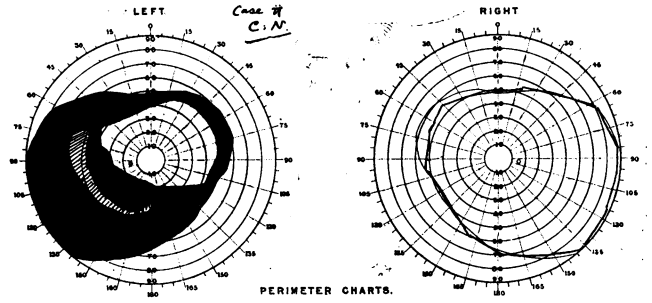
J.H.L. was thrown from his bicycle whilst quietly riding in town. In falling his head struck the curbstone. The blow was not forcible enough to cause anything beyond a momentary dizziness and a bruise of the outer part of the right eyebrow, and he was able to continue his ride home. He consulted me ten days later owing to the fact that he had noticed that the sight in the right eye had not been quite so good since the accident. I found the following conditions on examination: Right eyelids slightly ecchymosed and a small triangular patch of subconjunctive ecchymosis on the outer side of the right eye.



The right pupil was slightly larger than the left, and not quite so active to light. The fundus showed nothing abnormal, beyond, perhaps, a slight fullness of the veins and arteries. There was 3° of hyperphoria. R.V.=$\frac{6}{18}$ not improved by glasses. Colour vision was slightly impaired—all colours, though recognizable, appearing paler than they did with the left eye. The greater part of the right field of vision was wanting on the temporal side, and there was a slight contraction (about 10°) on the nasal side (vide chart). The left eye was normal. Field full. L.V. = $\frac{6}{6}$. A year later the visual fields were practically the same. The right optic disc was decidedly pale, and the retinal arteries were slightly contracted. R.V. = $\frac{6}{9}$; L.V. = $\frac{6}{6}$.

CASE II.

C. N. was thrown off his bicycle whilst racing. He was unconscious for twenty minutes, and a wound over the outer part of the left orbital margin had to be stitched up and the eye bandaged. Eleven days later, when the eye was uncovered, the patient found that the sight of the left eye was defective. Three weeks after the accident I found the following conditions: Over the external angular process on the left side was a



scar which indicated the site of the wound. The left eye was normal to all appearances except for a slight dilatation and sluggishness of the pupil. The greater part of the temporal field was lost, and there was a slight contraction on the nasal side (see chart). L.V.=$\frac{6}{9}$. The right eye was normal except for a pre-existing myopic astigmatism. Visual field full.

$$\text{R.V.}=\frac{6}{6}\ \bar{c}\ \frac{-7\text{ D. sph.}}{-1.5\text{ D. cyl }150^\circ}$$

A year later the left optic disc was markedly pale and the retinal arteries were contracted. The visual field was as above, except that there was a comma-shaped area in the blind temporal field, in which the test object could be indifferently seen (shaded area in chart). Possibly this had been overlooked in the previous examination. L V.=$\frac{6}{12}$.

*Abstracted from a paper read before the Central Division, Birmingham Branch, British Medical Association, January, 1905.

CASE III.

(For notes of this case I am indebted to Mr. Lloyd-Owen.)

E. S. was thrown off his bicycle and received a slight wound in the region of the left external angular process. He was unconscious for about an hour. Three months later his left vision was little more than perception of light, and that only on the nasal side. The temporal field was lost up to the point of fixation. The optic disc was atrophic, and the retinal arteries smaller than those on the right side.

The right eye was normal and field of vision full.

CASE IV.

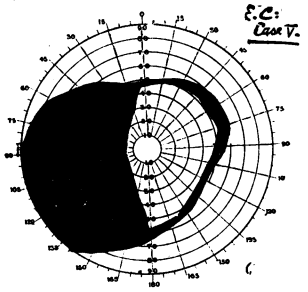
H. A. fell off his bicycle and struck his right temple. Six months later his condition was as follows:

Right eye: Atrophy of the optic nerve, with some narrowing of the retinal arteries. Visual field contracted to within 10° of the fixation point on the temporal side. R.V. = $\frac{6}{6}$ $\bar{c}$ + .50 D.

Left eye: Normal; field of vision full. L.V. = $\frac{6}{6}$ $\bar{c}$ + .50 D.

CASE V.

E. C., a miner, was struck by a "coal tub" on the left side of forehead and face one month before his admission to the Birmingham Eye Hospital. There was no loss of consciousness; there was some bleeding from each nostril, and the left eye was hidden by swollen and ecchymosed eyelids. Four days later, when the swelling had subsided and he was able to open the lids, he found that the sight of the left eye was defective. On admission there was a large scar involving the outer third of the left eyebrow, and with two other smaller scars, one at the junction of the outer and middle third of the inferior orbital margin and the other on the left side of the bridge of the nose, showed the extent of the injury. The left pupil was very slightly larger than the right, and its reaction to light was very sluggish, but its associated and consensual reflexes were good. There was no interference with the motility of the eye. The optic disc was pale, the pallor being more decided on the temporal side. The temporal field was lost up to within 10 deg. of the point of fixation (vide chart). L.V. = Fingers at 2 metres. The



right pupil was somewhat dilated (about 10 mm. in dull daylight). The direct and associated reflexes were brisk, but the consensual reflex was deficient. The visual field was normal. R.V. = $\frac{6}{6}$. This case being a recent one, is still under observation.

The sequence of events in these cases was as follows:

1. A more or less severe blow in the region of the external angular process of the frontal bone.
2. Sudden impairment of vision on the side of injury.
3. Loss of the greater part of the temporal field of vision on the same side.
4. Absence of ophthalmoscopic changes for the first few weeks, followed by atrophy of the nerve head on the injured side.
5. Central vision may be almost completely restored, but the limitation in the field of vision remains practically and permanently the same.

The nature of the lesion in these cases is very problematical.

The following may be mentioned as possible causes of such changes:

1. Fracture extending through the optic foramen.
2. Laceration of the nerve by a detached and displaced anterior clinoid process or some other spicule of bone.
3. Haemorrhage into the sheath or substance of the optic nerve.
4. A limited contusion of the optic nerve.

I believe it is usual to ascribe unilateral amaurosis resulting from blows on the head to fracture involving the optic foramen. It is hardly conceivable that such a fracture could have occurred in the cases recorded here. The injuries were all comparatively slight, and, judging from some experiments which I have made on the cadaver, quite inadequate to produce a fracture of the optic ring. Notwithstanding the high percentage of fractures involving the optic foramen recorded by von Hölder and others, I am convinced that it is exceptional to find such fractures in the pathological museums in England. Fracture of the ring would presumably cause blindness by laceration of the fibres by the displaced bone or loss of function in the nerve by pressure of displaced bone or callus. In either case the interference with the nerve function would be greater and more variable than that recorded in the above cases. Further, it is not likely that the ophthalmic artery would escape injury in cases of fracture of the optic ring, and evidence of disturbed circulation would probably be visible in the eye and orbit. A fractured and displaced anterior clinoid process would be more likely to lacerate or compress the temporal rather than the nasal fibres, and perhaps more likely to affect one of the optic tracts than one of the optic nerves.

Haemorrhage into the sheath or substance of the optic nerve is a very likely cause of complete amaurosis following blows on the head. In two cases of extensive fractures of the skull examined by Uhthoff haemorrhagic neuroretinitis was found ophthalmoscopically, and *post mortem* the nerve sheaths were found distended with blood, though there was no fracture of the bony optic foramen. Such a haemorrhage would not remain localized to one side of the nerve, and, should the amaurosis not be complete and permanent, the signs and symptoms would vary as the blood became absorbed and the undestroyed fibres resumed their function.

We are therefore forced to the conclusion that, in the cases recorded here, the lesion must be one of limited contusion of the nasal fibres of the optic nerve by *contrecoup*. The nerve on the side of the injury is driven against the inner boundary of the optic foramen, whilst the nerve on the opposite side is driven against the outer wall of its foramen. That the nerve on the opposite side does not get bruised is in the main due to the protection given to it by the opthalmic artery which winds round its outer side from below.

Assuming such an immediate cause for the symptoms mentioned, the course and prognosis of the cases can easily be deduced. A certain number of fibres are permanently destroyed (functionally) at the time of the accident. The more centrally-placed fibres are not so extensively damaged and partly recover their function, as shown by the improvement in central vision. The atrophy of the nerve is apparently not progressive and no increase in the limitation of the field need be feared.

Generally speaking, we may expect a considerable improvement of central vision in those cases in which there is a fair central vision immediately after the accident, but little or no change for the better or for worse in the field of vision.

These cases should be treated on the plan adopted for concussion of the brain—that is, rest and quiet in a darkened room, light diet, and aperients for a week or two, and avoidance of work and mental excitement for a further period of two or three weeks.

BIBLIOGRAPHY.

Nettleship: *Ophthalmic Review*, vol. xiv, p. 97. Von Hölder: *Graefe's Archiv*, Band vi. and Norris and Oliver's *System of Diseases of the Eye*, vol. iii, p. 16. Uhthoff, *Ophthalmic Review*, vol. xxi, p. 222.

THE SUBSTITUTE FEEDING IN INFANTS.*

BY T. H. SANDERSON-WELLS, M.D.LOND., M.R.C.S.ENG.,

Physician to the Princess Christian Hospital for Women and Children, Weymouth.

THE diet of all young mammals is milk, and the constituents of which all milks are composed, though varying in percentage strength, are practically the same. Milk is an emulsion of fat containing solids in solution, and is composed of proteid, fats, sugars, salts, and water. It is sterile, and has of course the temperature of the human body. Its average composition varies only within narrow limits, and is readily determined.

Any food, therefore, we prescribe to take the place of the natural mother's milk should as nearly as possible have its composition and properties. Any foods not conforming to this standard must be rejected.

The composition of human milk may be taken to be as follows:

1. Proteid, 1.5 per cent.:
   Composed of:
   (*a*) Lactalbumen (0.8 to 1 per cent.), soluble form of albumen not curdled by acids coagulating 165° F.
   (*b*) Caseinogen (0.4 per cent.), insoluble form of albumen; forms a thick curd with rennin and acids; not coagulated by heat.
   (*c*) Lactoglobulin, soluble, only a trace.
   (*d*) Nitrogenous extractives.

* Read before the West Hants and Dorset Branch of the British Medical Association at Blandford on May 17th, 1905.