CONCUSSION OF THE RETINA.

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SEVERAL years have elapsed since Dr. R. Berlin¹ described a series of cases in which he had observed a peculiar retinal change after the eye had been struck with a blunt object, as, for example, a stick or a stone. Under those circumstances he noticed a cloudiness of portions of the retina, not involving the retinal blood vessels. The milky appearance reached its height in twenty-four to thirty-six hours, and disappeared in two or three days. Berlin pointed out that the rapidity with which the cloudiness developed and the length of time that it persisted stood in direct relationship with the severity of the original injury. This curious condition, which Berlin called *commotio retinae*, was associated with some reduction of sight, episcleral congestion, and a difficulty in getting the pupil to dilate when atropine was dropped into the eye. Small retinal hæmorrhages were sometimes present. Berlin explained the ophthalmoscopic picture by supposing that a rupture of the choroid was followed by bleeding and oedema of the retina. This theory has recently been opposed by Denig.² That observer, as the result of experiments upon rabbits, believes that the blow upon the eyeball causes the vitreous to impinge upon the retina, to tear the internal limiting membrane, and to force the vitreous into the nerve-fibre layer. The alternate elevations and depressions thus brought about in the nerve-fibre layer of the retina are, according to Denig, the cause of the ophthalmoscopic appearances.

Since the publication of Berlin's original paper, few cases³ of *commotio retinae* have been recorded. Indeed, the retinal changes are of so fleeting a nature that an opportunity for observing them must occur comparatively seldom. This fact leads me to place upon record brief notes of a somewhat interesting case that has recently fallen under my notice.

E.S., aged 11 years. First seen on July 25th, 1899.

History.—At 6.45 P.M. on July 24th the patient was struck in the right eye with a cricket ball, made of cork and covered with rag cloth.

Present State.—Right eye: Small abrasion of the skin of the lower lid with a surrounding area of redness. Some general conjunctival congestion, with a definite ecchymosis in the ocular conjunctiva opposite the lower-outer quadrant of the cornea. Tension *minus* 1. The pupil distinctly sluggish and a trifle larger than the other one. Anterior chamber deep. A narrow line of blood clot lay at the bottom of the anterior chamber. V. $\frac{1}{2}$ (11 letters). Pupil dilates imperfectly to a mydriatic. With the mirror alone some parts of the fundus oculi were seen to be unduly white. When examined more closely with the ophthalmoscope there was found a wide but defective zone of whitish fundus situated peripherally upwards, inwards, and outwards. No such appearances could be made out in the lower part of the fundus. The retinal vessels, which lay anterior to the affected areas, showed no changes. In most places it was possible to get beyond the whitish patches so as to see the edges of the latter. These margins were irregular, and showed white, tongue-like projections running into normal fundus. Some small islands of cloudiness lay, however, beyond the area of general haziness. Around the yellow-spot region was a white radiating appearance, but no definite white mass was present in that place. Left eye: No fundus changes. V. $\frac{1}{2}$ (7 letters). Tension normal.

Treatment.—Vaseline to abrasion of skin of lid; atropine drops (2 grs. to the ounce—to each eye twice a day); rest in bed.

Progress.—July 26th. R.V. $\frac{1}{2}$; tension still rather low. The blood clot present in anterior chamber and also on anterior capsule of lens renders it difficult to see the fundus clearly; but no white patches can be made out in the fundus.

July 27th. A little blood is still present in the lower part of the anterior chamber. The parts of the retina that were milky have resumed almost their natural appearance, and the changes above mentioned are now represented merely by a faint, whitish, ill-defined stippling of the areas in question. Around the yellow spot is a system of fine radiating lines, which extend for some distance into the surrounding fundus. This is doubtless due to oedema of the retina.

July 28th. R.V. $\frac{1}{2}$ (1 letter); tension still slightly *minus*. Ecchymosis present in ocular conjunctiva, but the blood has disappeared from the anterior chamber. Pupil not so wide as that of the left eye, although atropine is being used to both. Faint cloudiness lower third of the cornea, made up of almost transparent dots, as may be seen with a +20 lens in certain positions of the eye. Fundus changes have disappeared; faint radiating lines, however, may still be seen around the yellow spot region.

July 29th. R.V. $\frac{1}{2}$ (11 letters) T—I. Pupil now as large as that of the other eye. Yellow spot region still surrounded by a wide band of fine, closely set, radiating grey lines. It may be noted that the corresponding region of the left (unaffected) eye is encircled by an ordinary oval reflex.

August 1st. R.V. $\frac{1}{2}$; (Tn.) A small ecchymosis still present in the ocular conjunctiva on the outer side of the cornea. No blood in anterior chamber; no corneal cloudiness. Radiating appearance still present around yellow spot of fundus.

August 6th. R.V. $\frac{1}{2}$; L.V. $\frac{1}{2}$; (Tn.)

August 12th. Vision unaltered. Radiating lines still present around yellow-spot region of affected eye.

September 5th. The right pupil rather larger than its fellow, but no break in the continuity of the edge of the iris can be discovered to account for this. The action, both to light and to accommodation, of the pupils is equal. The radiating lines formerly present around the yellow spot of the right eye have been replaced by an ordinary oval reflex, like that present in the other fundus. Tn.; R.V. $\frac{1}{2}$ (1 letter), L.V. $\frac{1}{2}$ (1 letter); No. 7 Jaeger read easily.

September 7th. Under atropine. R.V. = $\frac{1}{2}$ + 1.5 D. Sph. = $\frac{1}{2}$. L.V. = $\frac{1}{2}$ + 1.0 D. Sph. = $\frac{1}{2}$.

REFERENCES.

¹ *Klin. Monatsbl. f. Augen.*, xi, 1873, p. 42. ² *Arch. of Ophthalmology*, xxvi, 1897, p. 377. ³ Two good illustrations of the condition are given in Haab's *Atlas und Grundriss der Ophthalmoskopie*, an English edition of which has lately been brought out by Mr. Ernest Clarke.

CARCINOMA OF THE ASCENDING COLON AND ITS TREATMENT,

WITH THE RECORD OF TWO CASES.

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THE comparative rarity of malignant disease of the ascending colon causes the publication of every case to be a matter of some interest, and it is on this account that I venture to record the two following cases.

It is a fact that while the cæcum is almost as frequently attacked by carcinoma as is the sigmoid flexure, yet the ascending colon is very much less often affected by the same disease than the descending colon, and only slightly more frequently than the transverse colon with its hepatic and splenic flexures. These general statistics are, however, from the records of published cases, and they may not be so accurate if all the cases that occur were tabulated, but the main statement may be taken as true that the ascending colon is one of the less common sites in which malignant disease begins. In these remarks I have purposely excluded the rectum, which is undoubtedly the region where carcinoma most usually attacks the bowel.

There does not appear to be any very clear reason why the ascending colon, and to a minor degree the descending colon, should be to such an extent exempt from the appearance of a cancerous growth in its walls.

The blood supply of the cæcum is derived from what may be taken as a terminal branch of the superior mesenteric artery, the ileo-colic, but the ascending colon, transverse colon, and descending colon, are supplied by vessels which freely anastomose with one another, the two former by branches from the superior mesenteric, and the last by a branch from the inferior mesenteric. The sigmoid colon with the rectum are on the other hand nourished by arteries which again are of the nature of terminal vessels. Whether such a disposition of the arterial supply has any connection with the determination of the deposit of carcinoma, it is impossible to say, but it may be a factor in the causation of the disease.

It is also significant that the cæcum, the sigmoid, and the rectum are all dependent parts of the intestinal tract, and this, in addition may be another slight factor.

The lymphatics of the ascending colon pass to the meso-colic lymphatic glands, lying between the layers of the meso-colon, and sending their efferent vessels to the glands placed in the proximity of the superior mesenteric vessels. This fact renders it extremely difficult or well-nigh impossible to eradicate the whole of the disease, when once these glands have become infected, in a case of carcinoma of the ascending colon.

The peritoneal investment of this portion of the bowel is, in the majority of instances, but a partial one. It is, moreover, usually less complete even than that of the descending colon, thus rendering the ascending colon as a rule more fixed than the corresponding portion of gut on the left side.

In about 26 per cent. of subjects there is a true meso-colon on the right side. The length of the ascending colon is on the average eight inches, while its calibre is larger than that of the descending colon. Its posterior relations are found to be the right kidney, above and externally, the second portion of the duodenum, internally, and the quadratus lumborum, at the lower part. The right colic artery enters it from behind, about the middle of its length. The right colic vein joins the superior mesenteric vein, and thus reaches the portal trunk. The position of the three longitu-