yielded negative results each time. There was no history of gonorrhoea, and no evidence of involvement of the genito-urinary tract. Patient had posterior synechiae and deposits of uveal pigment on the anterior lens-capsule of each eye. There was no history of " rheumatism " having ever occurred in his life before, and there was no evidence of his having had malaria.

Conclusions.

(1) Patients suffering from an infection by *B. dysenteriae* (Shiga) may occasionally develop anterior uveitis as a result of this infection, as pointed out by Morax[1].

(2) This ocular affection may, or may not, be accompanied by articular manifestations.

(3) The ocular affections would appear to occur most frequently about one calendar month after the first signs of involvement of the bowel, but may occur as early as the twelfth day (Case 2).

(4) The articulo-ocular syndrome corresponds exactly to that occurring in another affection of a mucous tract, viz., gonorrhoea, as pointed out by Garrod.

A CASE OF SIDEROSIS AFFECTING THE INNERVATION OF THE PUPIL

BY

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DILATATION of the pupil following siderosis has been noted before; and Vossius, in a paper on Siderosis Bulbi, read before the Heidelberg Ophthalmological Society in August, 1901 (see *Ophthalmic Review*, Vol. XXI, p. 223), ascribed the dilation of the pupil to a chemical irritation of the sympathetic fibres in the iris. But the following case (which was treated at the Royal London Ophthalmic Hospital, while I was House Surgeon and temporary Curator in 1917, under the care of Mr. J. Herbert Parsons, who has kindly given me permission to publish my observations) appears to show that iron salts injure the endings of the 3rd nerve in the iris rather than stimulate those of the sympathetic; and that the dilatation is due to the prevention of constrictor impulses reaching the iris through the 3rd nerve. Indeed, in this case I have found that the sympathetic nerve endings are injured by iron salts quite as much as those of the 3rd nerve, and that the dilatator pupillae loses its function; so much so that some time after removal of the piece of

1. *British Jl. of Ophthal.*, March, 1917, pp. 186.

iron causing the siderosis, when the constrictor pupillae begins again to respond to light and atropin, the dilatator pupillae is still paralysed and does not respond to cocain.

J. P. was admitted on January 16, 1917. There was a foreign body in the left eye, but there was no visible scar on the cornea or sclerotic, and no definite history of the eye having been hit by a piece of metal. His occupation was that of a tin-smith and plate worker, and he has worked with all kinds of metal plates (copper, tin, zinc, aluminium, iron and brass). The main feature in the history was the fact that at the beginning of August, 1916, the sight of the right eye gradually became slightly misty at night, but by day was good. There had never been any redness. About September 7, 1916, the sight was mistier and some friend noticed that the left pupil was much bigger than the right. His doctor (Dr. Robson) noticed that the left pupil was immobile and inactive to light. He was treated at Maidstone Hospital for six weeks, and then as the eye became painful, he attended Moorfields as an out-patient in November, 1916. In January, 1917, the left eye was quiet and showed no injection or "keratitis punctata." The left pupil was two-thirds dilated and was inactive to light both directly and consensually. There was a kind of faint mottled appearance over the anterior surface of lens and some vitreous haze, also floating vitreous opacities in the extreme periphery below and a black mass on the retina which might be a foreign body. A skiagram showed the presence of a foreign body, when the eye was submitted to the X-rays on January 16, 1917, situated 11 mm. below and 10 mm. deep to centre of cornea and 2 mm. to temporal side. This suggested that the foreign body was embedded in the retina or sclerotic below, or even that it was almost through the sclerotic. I attempted to extract the foreign body with the Haab magnet on three occasions, viz., January 17, 18, and 20, 1917, but without result. On the first occasion I made a conjunctival flap in the lower fornix so as to get as far back as possible and pull the foreign body forward if it was outside the globe.

He was discharged on January 20, 1917, with instructions to return when the new Mellinger magnet was installed. Accordingly he was re-admitted on February 6, 1917. There was very little change in the eye except that the iris stroma was beginning to atrophy and that there was more very fine deposit (suggesting siderosis) all over the front of the lens capsule, and perhaps more vitreous haze. Vision now was only 6/18 partly (unaided) or with + 0·5 cyl. ax. hor.=6/12. The pupil was still dilated, and quite inactive to light. On February 7, 1917, I tried the Mellinger magnet, without much expectation of success, but at last with the largest rod I succeeded in bringing a moderate-sized piece of iron forward into the anterior chamber from behind the iris below. The foreign body was removed through a keratome incision at

the limbus above, and the eye healed up without any adverse circumstance. He was discharged on February 17, when the vision with $\frac{-0{\cdot}75 \text{ sph.}}{+0{\cdot}5 \text{ cyl. ax. hor.}}$ was 6/9, the eye being under the influence of eserin.

On March 3, 1917, when he came to the hospital as an out-patient, there was no light reflex of the left pupil, but it contracted well with eserin. Vision was 6/9 partly (unaided). I prescribed eserin drops ¼ per cent. b.d., for his left eye till March 24, when they were to be discontinued. On April 4 he came to the hospital again. The left pupil now reacted slightly but definitely to light, both directly and consensually, though the response was much more certain in the upper half than the lower. In a dim light the diameter of the pupil was 6·5 mm., but it would not dilate with cocain. The front of the lens still had a finely mottled appearance, and there was still marked vitreous haze. Vision with +0·5 cyl. ax. hor. was 6/6 (slowly) but was said to be misty. On April 28 (eserin having been used for three weeks, but discontinued three days before) the left pupil reacted definitely to light all round its circumference. The size of the pupil on this date was as follows :—

	Right Eye.	Left Eye.
In dim light	7·5 mm. ...	5·5 mm.
In strong light	2·5 mm. ...	3·0 mm.
After cocain in good light	10·0 mm. ...	5·5 mm.

L.V. was 6/6, improved by +0·5 cyl. ax. hor.

Eserin was not prescribed again and when he came up as an out-patient on June 30 the left pupil reacted to light almost as well as the right, but there was a noticeable difference in the briskness of the response. The left pupil, on the other hand, did not dilate nearly as well in a dim light as the right. The following observations were made :—

	Right Eye.	Left Eye.
In dim light	7·0 mm. ...	5·5 mm.
In strong light	2·5 mm. ...	2·5 mm.
After cocain in dim light ...	11·0 mm. ...	6·0 mm.
After cocain in strong light	4·0 mm. ...	4·0 mm.

After homatropin and cocain in the left eye the pupil was 8·5 mm. in diameter and there could be seen five or six deep yellow rusty spots on the front of the lens capsule just uncovered by the margin of the dilated pupil on the temporal side. The front of the lens capsule as a whole was certainly freer from the mottled appearance of a very fine deposit which had always been present before: but there was still some over the upper third of the lens. The large yellow spots were joined up by a line of much finer ones. I am certain that these rusty spots were not present last February when the pupil was widely dilated, and I hazard the suggestion

that they have been formed by an aggregation of microscopic siderotic particles on the front of the lens capsule which are apparently clearing away in this manner. The colour of the left iris was a kind of greyish-brown, while the right iris was a light yellowish-grey. There was still marked vitreous

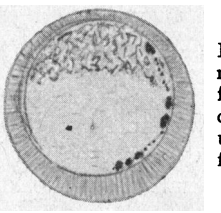

Diagram of the left pupil of J.P. on June 30, 1917, after instilling 2 per cent. homatropin and cocain.

haze and the outlines of the optic disc were indistinct but I could not see any definite vitreous opacities, even at the periphery below. Vision was 6/6 (unaided) improved by +0·5 cyl. ax. hor. but he said that everything looked misty. He could read J.1 slowly but correctly at a distance of 10 inches with correction for his presbyopia. He was aged 46. The field of vision was markedly restricted in all directions, only extending to 50° on the outer side, 40° below, 30° on inner side and above.

The interest of this case is, I think, considerable; illustrating, as it does, *first*, the success of the Mellinger magnet where the Haab had failed; *secondly*, the recovery of 6/6 vision after siderosis had developed; *thirdly*, the toxic effect of iron salts on the neuromuscular mechanism of the pupil, viz.: marked degeneration or injury of function of both the sympathetic and 3rd nerve fibres ending in the iris; *fourthly*, the recovery of function of the 3rd nerve fibres before those of the sympathetic. If both sets of fibres were degenerated this would suggest that regeneration has taken place more quickly in the 3rd nerve fibres because the nerve cells from which they spring are in the ciliary ganglion. On the other hand, as Parsons and Henderson showed (*Ophthalmic Review*, XXVII, p. 325, 1908), the endings of the sympathetic in the iris seem easily affected by adverse conditions, for they found that cocain produced no effect on the pupil of a rabbit after the cervical sympathetic in the neck below the superior cervical ganglion had been cut. It will be interesting to note if the power of reacting to cocain returns to the pupil in the next twelve months.