tachment close to the lens. Foreign body in the globe was now suspected, owing to the irritation of the eye and changes in the pupil. This was verified by a skiagraph. Later the retina greatly reattached, and a shiny foreign body could be seen. Gradually the eye became irritable, and enucleation, was performed. The pathologic examination by **Collins** showed the retina at the ora serrata torn away from the ciliary body. The foreign body must have entered the upper and outer part of the globe, passed through behind the lens to the ciliary region on the opposite side; and contraction of the vitreous was the cause of dragging towards the center of the globe, of the retina in the region of the ora serrata.

The right eye of **Wootton's** patient presented a flat, wavy membrane resembling a retina that had been torn from the ora serrata, extending from above downward, on the temporal side. The mass was possibly one of proliferating retinitis.

TOXIC AMBLYOPIAS.

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This section includes the literature from January, 1917, to May, 1918. But some instances of the harmful action of certain substances may be found under "Therapeutics," or under "Injuries." See also "Optic Nerve."

QUININ AMBLYOPIA.—**Ballantyne** presents a very complete analysis of the various theories and experiments, which have been offered in explanation of the production of quinin amaurosis. A good reference list follows. The author first details a typical case, followed by the case history of one of his patients, pointing out wherein it differed; in that, altho the patient was first seen four days after the administration of the quinin, blindness ensuing, a further period of five days elapsed before the characteristic ischemia of the disc and retinal vessels made its appearance. The paper is summarized as follows:

1. That in quinin poisoning, complete loss of vision may be found, in association with a normal condition of the fundus oculi; and that there may be a striking recovery of vision, in spite of the presence of well marked fundus changes.

2. That in all, or nearly all, cases of quinin amaurosis, ophthalmoscopic changes, such as congestion of optic nerve and retina, pallor of the disc, narrowness of the retinal vessels, and cloudy opacity of the retina, make their appearance sooner or later; but that there is no correspondence between the character or severity of these changes, and the intensity of the visual defect.

3. That the visual defect cannot, therefore, be due to such changes, but rather to a condition of the retinal elements invisible with the ophthalmoscope.

4. That this change may be induced, or aggravated, in the first place, by ischemia due to contraction of the vessels of the optic nerve and retina; but that it is, in the main, the result of a direct toxic action of quinin upon the retina itself, and that the ultimate recovery of central vision, with loss of peripheral vision, and failure of vision in twilight, suggests a selective action of the poison upon the rods.

Santos Fernandez directs attention to the disturbance of vision from quinin and paludism, as possibly being a hybrid affection. He notes, especially, cases of sudden blindness, following the exhibition of quinin for several malarial attacks. The anatomic basis is apparently total ischemia of the papilla, followed by atrophy of the optic

nerve. In such cases, the drug is naturally accused, but it is, of course, not impossible that the disease was an active factor. Does quinin ever set up this ischemia in the subject, and does its action on the plasmodium react unfavorably on the organism? It is highly important to ascertain the special affections of the eye, which result from paludism alone, and from quinin alone.

Quinin, without reference to any particular therapeutic use, is commonly accused of causing "amblyopia" and "amaurosis," which names suggest functional disturbances of temporary duration. It is conceded, however, that it is not so much heavy dosage, as idiosyncrasy, which is responsible for quinin amblyopia, etc. The entire question remains unsettled, because of lack of autopsy material, and because we are influenced, necessarily, by results of experimental work on animals; which appear to show that quinin is surely neotropic to the optic nerve, if the dose is large enough.

A soldier, aged 20 years, received by mistake, 40 grams of sulphat of quinin, instead of sulphat of magnesia. In half an hour, he lost consciousness. When he recovered, he could neither hear nor see. Examination by **Galiana** showed pale papillae, muddy edges, contracted vessels, fingers at 2 M. About three weeks later, when last seen, vision had greatly improved, but fields remained contracted. This is the only case of amblyopia by intoxication, observed by Dr. Marquez, in the Buen Suceso Hospital, among 34,000 patients seen in fourteen years.

Ethylhydrocuprein Amblyopia.—A lady, aged 40 years, to whom 4 grams of ethylhydrocuprein in 0.20 gram doses, every two hours, was administered for pneumonia, developed tinnitus, later complete deafness, and just before the last dose, total amaurosis. **Lorant** observed the pupils moderately dilated and fixed, papillae pale, contracted vessels, arterial pulse on pressure on globe, and in the left eye, small, circumscribed hemorrhages.

Schreiber reports three cases of visual disturbance, due to internal use of ethylhydrocuprein. A doctor, aged 54 years, ill with pneumonia, received four 0.3 gram doses of quinin hydrochlorid, fourteen 0.25 gram doses of ethylhydrocuprein and one tablet of digipuratum, three times daily. On the fifth or sixth day, amaurosis developed, which disappeared in ten hours. Two months later night blindness developed. There was no optic atrophy, altho the blood vessels were narrowed. The second patient was a woman, aged 57 years, who had received 1.75 grams of ethylhydrocuprein. Optic atrophy, narrow vessels, good visual acuity, but narrow fields ensued. The third patient, a woman, ill with pneumonia, showed conditions as found in the second patient.

Schiötz reviews, in detail, ethylhydrocuprein and its drawbacks. He also reports a case of transient blindness, in a sailor, aged 48 years. While the vision is reduced, it was not enough so to incapacitate him. He was color blind, and had restricted fields. **Van der Hoeve** and **Mansholt** report of a patient with pneumonia, on an exclusive milk diet, who was given, in moderate doses, a total of 4 grams of optochin. He became totally blind. Vision partially returned, but with paralysis of accommodation, atrophy of the optic disc, and sclerotic changes in the vessels. The vascular changes did not develop until several weeks after the first symptoms, but they continued a progressive course, while the nerve changes seemed to show a tendency to retrogress.

Out of thirty cases of pneumonia, in which ethylhydrocuprein had been given, **Pollnow** saw no after effects in 25 cases, but disturbances of sight and hearing in 5 cases. In 2 cases, there were slight, quickly transient, visual disturbances; in one, tinnitus of the ears, and two, serious troubles in the eye. Both of the last were quite blind at first. One patient later regained approximately normal conditions, but only a very slight improvement was obtained in the other. They received 0.25 grams of ethylhydrocuprein every four hours.

In the latter case, amaurosis set in on the third day, and gradually began to improve after several days. Now, both papillae are very pale, the vessels tortuous, the arteries very small, some obliterated, some with white sheathes. The retinas were edematous over a large area about the papilla, with a red spot in the fovea. Vision of each eye, fingers at from 1 to 1½ meters. Visual fields were concentrically contracted to a high degree. Blue and red are the only colors recognized of large objects. Diagnosis, neuritic atrophy, secondary to disease of the optic nerve, and retina, with edema and vascular disease.

In the other case, the symptoms and the changes in the fundus are similar, but the vision has improved to 4/4. It appeared to the writer to be a typical quinin poisoning, in which probably, circulatory disturbances play the chief part. On account of the greatly varying susceptibility to quinin, ethylhydrocuprein should be given in very careful doses.

Feilchenfeld observed a man, aged 20 years, who was given 5 grams of ethylhydrocuprein in the course of thirty hours, for a beginning pneumonia. After one day, there was loss of hearing, and a day later, poor vision, which within another twenty-four hours had become almost complete blindness. Both symptoms receded, under the employment of large doses of sodium iodid, and tincture of strophanthus, but at the end of two months, there was a permanent injury, manifested by hemeralopia, flickering before the eyes, annoying subjective scotoma, concentric narrowing of the visual field, and of color sensation in both eyes. The employment of ethylhydrocuprein, internally, should, therefore, begin with smaller doses; and since the disturbances of hearing always appear first, their occurrence should always be the signal for immediate cessation of the drug.

Nitrophenol Amblyopia. — Three observations are published by **Sollier** and **Jousset** of soldiers, employed in the use of nitrophenol in the manufacture of high explosives. All suffered from symptoms closely allied to those of a chronic retrobulbar neuritis. The first had early paralysis of the brachial plexus with a slow atrophy of the deltoid. The ophthalmic examination gave a visual acuity of 5/10, accommodation paralysis, green blindness and a concentrically contracted field. There was slight edema of the nerve head.

The second case had anesthesia and paralysis in both feet. Acuity of 2/10, with central scotomata for green and yellow, and contracted field and accommodation paralysis, with normal pupils. The third had a bilateral papillitis, with acuity of 1/20, in the right eye, and 3/10, in the left.

The authors have fifteen analogous cases, but cannot trace the point of entrance of the poison. Perhaps the nitrites, being vasodilators, might be counteracted by the use of a vasoconstrictor. It is well to warn the workers, so that they may be on their guard, and have them use masks, gloves, and hand disinfection. At the first symptoms, the worker should be sent to a specialist, who will decide upon the advisability of giving up the position in the powder industry.

Paint Amblyopia.—**Besenbauch** alludes to the frequency of untoward effects of the volatile principles in paints, as experienced by painters. He relates a case in which the nature of the fumes was obscure. The patient suffered an acute hallucinatory confusion and optic neuritis. Benzol could be excluded, but the manufacturers admitted the use of a distillate of Borneo petroleum known as sanzazol. This statement was confirmed by an analysis. The toxic substance was also identified as putrol, a substitute for turpentine, known for its disagreeable odor. The paint, a light green, was used on ships, especially for decorating small spaces, in which ventilation was poor. Numerous cases of poisoning had been reported, and a single case is given in detail. The victim made an uneventful recovery. To prevent such accidents, painters are warned not to converse, whistle, etc., while painting. The greatest possible ventilation should be secured, a portable ventilator being

available. The shift should be only three hours long, with an hour's intermission, to be spent on deck. The paint, pronounced relatively nontoxic, was not condemned.

Wood Alcohol Amblyopia.—There was no demonstrable ophthalmoscopic cause in **Risley's** patient, who doubtless suffered with a sudden blindness, due to wood alcohol. A young Italian, with his friends, was in the habit of using a beverage made of alcohol, water, sugar, and "orsa rossa." The nature of the latter was not ascertained. **Golovin** has written, regarding blindness after the use of wood alcohol, and the other substitutes for vodka.

A male, aged 33 years, presented by **Strickler,** may have suffered a toxic amblyopia, due to tobacco or alcohol, although the history and findings were not conclusive. Vision was reduced to counting. fingers at three feet, color sense fair for green, red or blue. There was no true scotoma. The temporal side of the disc was pale, the nasal side appeared swollen.

Lead Amblyopia. — Five or six months previous to consulting **Leoz,** a mining engineer noticed weakness in his sight, which increased until he had to give up his work. On clear, sunny days he was unable to see where he was going. Distant vision was rather better than near, a dark spot and a halo somewhat less dark, obscuring close objects. Luetic, alcoholic and tobacco history was negative. The paralysis of the extensors of the fingers confirmed the diagnosis of saturnin amblyopia. The fundi were slightly congested, and the papillae of a reddish color. Definite scotomata were observed. Following the use of iodides, laxatives, and sulphur baths, a cure was effected within two years. For practical reasons the author advises scientifically studying the special causes of toxic amblyopias. If lead intoxication provokes a nephritis, and this in turn a retinitis, the amblyopia would be albuminuric and nothing more.

THE OPTIC NERVE.

Charles P. Small, M. D.

CHICAGO.

This review covers the literature relating to the optic nerve from January, 1917, to May, 1918.

Anatomy and Histology. — Impresssed by the variety to be observed in the amount of *cupping* in cases of glaucoma, **Fuchs** has written an exhaustive paper accompanied with forty-eight drawings and microphotographs, to show that the difference might be largely due to variation in the anatomy of the lamina cribrosa. He says that the picture of excavation is first formed by the weakening and subsequent disappearance of the glial trabeculæ. The connective tissue trabeculæ follow later. If this occurs early, cupping is deep, while there is still good sight and but little alteration in the nerve head. If, however, it occurs late, then, while the cupping remains shallow, and is mainly due to atrophy of the nerve, the sight rapidly diminishes. He concludes that the usual process in raised pressure consists in a sclerosis and thickening of the lamina, probably as a result of the greater load it has to bear. This may either remain as a permanent change, or resorption may follow under the continued pressure.

That *hemorrhage* into the optic nerve sheath, vitreous, and retina not infrequently follows fracture of the skull is evident from the review of the litera-