slightly injected and there was a quite obvious crop of keratic precipitates in the lower part of the cornea. The vision was roughly 6/18 and the tension not raised, there were no synechiae present. After having dilated the pupil it was difficult to get a clear view of the fundus; the disc appeared normal but the lower branch of the central vein was markedly dilated. The field showed a large quadrantic defect which ran in to the blind spot. We judged that there was a patch of choroiditis somewhere and finding that pus was exuding from a sinus in connexion with one of the teeth in his upper jaw we suggested this as a possible cause, advised him to see his dentist without delay and promised to communicate with our doctor friend. Then it was that he began to cross-examine us about his optic nerve. We did not realize the reason until two days later when another doctor rang us up and explained that he had previously sent the patient to his own oculist who had diagnosed optic neuritis and had urged institutional treatment. The doctor seemed quite hurt: "so-and-so says he's got optic neuritis, you say he hasn't, which am I to believe?" This was a ticklish question but after a little talk it was settled that the patient should be returned to the first oculist and that we should meet him there in consultation. Of course the man was late for his appointment and while waiting my colleague told me that he had not found the precipitates and I confessed to not having seen a patch of choroiditis, though I had suspected it. Both precipitates and a choroidal patch were quite obvious when he at length arrived. When, some time after, we met our colleague we asked what had happened to this man. "Oh," said he, "he went off to see a third man, one of our clinical assistants at the hospital, and he said he could find nothing the matter with the eyes at all."

When the seniors make an error how shall we blame the clinical assistants?

ABSTRACTS

MEDICAL OPHTHALMOLOGY

(1) **Good, P. (Oak Park, Illinois). — Choked discs in lead encephalopathy.** *Amer. Jl. Ophthal.*, Vol. XXIV, p. 794, 1941.

(1) **Good** states that lead poisoning usually occurs in children 1 to 3 years of age and the chief source is paint on cribs and toys. In some cases nipple shields have been blamed. A blood concentration of lead below 0·03 mg. per 100 c.c. is normal and over 0·06

mg. per 100 c.c. is abnormal. A clinical feature of diagnostic value in children is the X-ray evidence of dense bands in the growth line of long bones, where lead is deposited. Encephalitis and associated papilloedema are other clinical signs in children. In adults basophilic stippling of the red cells and lead lines in the gums are more frequent findings.

The cerebro-spinal fluid pressure may be as high as 700 mm. of water and in infants the cranial sutures may be separated.

The author quotes a case of a child, aged 2 years, with bilateral papilloedema who underwent sub-occipital craniotomy. The cerebro-spinal fluid contained lead and 0·1 mg. was found in 703 c.c. of urine. Spectographic examination of the blood showed 0·2 mg. of lead per 100 c.c. Bands of increased density were seen at the ends of the diaphyses on X-ray examination.

The other case described by the author had a medulloblastoma of the cerebellum in addition to lead poisoning.

H. B. STALLARD.

(2) **Watkins, C., Wagener, H. P., and Brown, R. W. (Rochester, Minnesota).—Cerebral symptoms accompanied by choked optic discs in types of blood dyscrasia.** *Amer. Jl. Ophthal.*, Vol. XXIV, p. 1374, 1941.

(2) **Watkins, Wagener** and **Brown** write that haemorrhage into the nervous system in the course of thrombocytopenic purpura has long been recognised. The haemorrhages may be large, small, single or multiple and may occur sub-dural, in the meninges, intra-ventricular, intra-cerebral and into the spinal cord and thus may cause mental confusion, transient coma, paralyses, aphasia, hemianopia, diminished hearing and convulsions.

Ocular complications consist of retinal haemorrhage, bilateral papilloedema and bilateral external rectus paralysis. The authors describe three cases of thrombocytopenic purpura in which bilateral papilloedema was present without any demonstrable evidence of an intracranial pathological lesion and in none could it be proved that intra-meningeal and intra-cerebral bleeding had occurred.

The fourth case reported by these authors was that of a man, aged 50, with recurrent gastric haemorrhage from a large ulcer. He had bilateral papilloedema and post-mortem examination revealed no intracranial lesion. They suggest that the mechanism of papilloedema was the same in these four cases, a local reaction of the optic nerve tissues to anoxaemia from loss of blood. They quote some experimental work on animals to support this view and comment that papilloedema is also seen in scurvy, haemophilia and haemolytic icterus with severe secondary anaemia.

H. B. STALLARD.

(3) **Covitz, E. E. (Boston).—Exophthalmos.** *Amer. Jl. Ophthal.*, Vol. XXIV, p. 1423, 1941.

(3) **Covitz** comments that exophthalmos is uncommon as the first and earliest sign of thyroid disease. In one of his cases this preceded thyrotoxicosis by 1 year and 8 months, in another by 6 months, and in another by 8 months. Exophthalmos occurs in 75 per cent. of cases of thyrotoxicosis and is more prominent in the young.

The author discusses the theories about the aetiology of exophthalmos. He comments that a toxic thyroid may spontaneously enter a phase of remission.

H. B. STALLARD.

(4) **Klemme, R. M. (Saint Louis).—Oculogyric crises.** *Amer. Jl. Ophthal.*, Vol. XXIV, p. 1000, 1941.

(4) **Klemme** comments that oculogyric crises are a late result of epidemic encephalitis. He quotes Hall's figures of the incidence of 15 to 20 per cent. of 384 post encephalitic Parkinsonians. This phenomenon may also occur in neurosyphilis, brain tumour, multiple sclerosis, hysterical attacks, arterio-sclerotic Parkinsonism and some psychic disorders. Gliosis of the corpus striatum, the substantia nigra and their connecting tracts has been suggested as the site of the lesion.

Among therapeutic agents which have been tried are stramonium, benzedrine sulphate, hyoscine and atropine sulphate. The author describes his operation of pre-motor cortical excision at the junction of the first and second frontal convolutions, adjacent to the anterior edge of the motor cortex and passing to the depth of the sulcus. He has operated on 130 patients and states that oculogyric spasms ceased after operation. He found that typical oculogyric attacks could be produced by cortical faradic stimulation at the site of operation described above.

H. B. STALLARD.

(5) **Bab, W. (San Francisco, California).—Allergy of the eye.** *Amer. Jl. Ophthal.*, Vol. XXIV, p. 759, 1941.

(5) **Bab** discusses the nature of allergy, the clinical and experimental facts associated with such a disorder and the imperfections in our present knowledge about its aetiology. He states that skin tests, at best, can only furnish evidence corroborative of the history. He comments that most hypersensitive persons are nervous, have a labile and unstable character and a psychopathic constitution. Excitement is known to precipitate some allergic manifestations.

The author writes that it is possible that contact allergy plays a major role in superficial eye conditions such as allergy of the cornea,

conjunctiva and eyelids. The main feature in ocular allergy is the preponderance of a subjective hypersensitiveness; the complaints of the patient do not correspond to the objective findings.

In discussing treatment the author considers that the results of desensitization are often unsatisfactory. He recommends calcium in small or medium doses by mouth and is impressed by the value of a salt-free diet in some cases. He believes that epinephrine in eye-drops and injected subcutaneously seldom alleviates symptoms, and if so is only of transient value, the condition later becoming worse.

H. B. STALLARD.

(6) **Charlin, C. (Santiago, Chile).—The toxic syndrome of ocular tuberculosis.** *Amer. Jl. Ophthal.*, Vol. XXIV, p. 1392, 1941.

(6) **Charlin** stresses the importance of a careful investigation into the general condition of a patient, suffering from an ocular lesion suspected of being tuberculous, before tuberculin injections are begun, He states that any departure from the normal concerning appetite, sleep, energy, weight and temperature is a signal for caution. Disturbances of these functions, and in women associated dysmenorrhoea, constitute what the author terms a toxic syndrome. He quotes four cases where the symptoms and signs of this syndrome were improved by tuberculin injections. The dose is not recorded and the tuberculosis aetiology is conjectural in some of the cases reported.

H. B. STALLARD.

(7) **Thygeson, P., and Stone, W. (New York).—The treatment of inclusion conjunctivitis with sulphathiazole ointment.** *Jl. Amer. Med. Assoc.*, Vol. CXIX, V, p. 407.

(7) The value of sulphanilamide by oral administration in cases of inclusion conjunctivitis in infants and adults has been claimed by **Thygeson** in previous reports. It was found that infants responded in the first days of treatment and were usually cured within a week. In adults the papillary type of the disease responded equally rapidly, but in cases with predominant follicular hypertrophy several weeks were required for the conjunctiva to return completely to normal. The inclusion bodies characteristic of the disease could not be found after the first few days of treatment. There were no recurrences in cases in which the treatment was continued six days or longer. Local therapy with the same drug was ineffective.

Since the virus of inclusion conjunctivitis is known to attack epithelium only and its superficial layers most concentratedly (thereby differing in toto from trachoma), oral therapy would appear to have no advantage over local therapy other than that concerned

with the maintainance of a constant therapeutic concentration of the drug.

The local use of 5 per cent. sulphathiazole sodium ointment six times daily was effective in causing rapid healing in 11 out of 15 cases of inclusion conjunctivitis in infants, children and adults.

A. F. MACCALLAN.

(8) **Gifford, Sanford R. (Chicago).—Position of muscles after operation for strabismus.** *Arch. of Ophthal.*, March, 1942.

(8) After quoting the opinions of various authorities on this subject, **Gifford** proceeds to give his experiences in 24 cases where re-operation was carried out by himself in order to straighten the eyes after a previously unsuccessful operation. Details of each case are given and the general findings are as follows.

In divergence following tenotomy of the internal rectus, which was often 50 or more degrees in amount, the attachment of the internal rectus to the sclera might be found as far back as 17mm. from the limbus, and successful results attended its reattachment, combined with recession of the external rectus. In cases where recession of the internal rectus had produced an over-correction, the defect was probably due to extra adhesions occurring between the muscle and sclera behind the line to which it had been recessed. The latter was usually at or near the location of the scleral sutures. After resection and tucking operations which had produced an insufficient effect, adhesions were found between the muscle and the globe far behind the original insertion, possibly due to necrosis of the fibres or to clot formation among them. In a case of resection and advancement, the adhesion was along the line of the original insertion. Two cases of unsuccessful myotomy of the inferior oblique were found to owe their failure to the occurrence of reattachment of the muscle. The author therefore advises the removal of at least 4 mm. of muscle fibres to produce a permanent effect. In investigating some cases of diplopia following detachment operations, it was found that diathermy may cause attachment of muscles to the sclera whether or not they have been resected.

F. A. W-N.

(9) **Verhoeff, F. H. (Boston).—Retinitis pigmentosa with widespread gliosis—so-called choroideremia.** *Arch. of Ophthal.*, April, 1942.

(9) In this paper, **Verhoeff** makes the interesting suggestion that many cases in which the diagnosis "choroideremia" is made are really cases of retinitis pigmentosa in which the choroid is intact but hidden from view by a deep seated retinal opacity, resulting

from gliosis. In support of this, he mentions a case histologically examined by him in 1931. In it there were places in the fundus where there was much pigment, with deeper seated gliosis behind it which hid the choroidal vessels. He also mentions cases of choroideremia examined clinically, in which careful ophthalmoscopic examination disclosed evidence of retinitis pigmentosa and in which the history was also suggestive of the same disease.

F. A. W-N.

(10) **Cordes, Frederick C. and Hogan, Michael J. (San Francisco).—Dysostosis multiplex (Hurler's disease: lipochondrodysplasia: gargoylism).** *Arch. of Ophthal.*, April, 1942.

(10) This is a rare disease of congenital origin, and **Cordes** and **Hogan** record 5 cases. It is characterised by chondrodystrophic skeletal changes and deposition of a lipoid-like substance in many of the body tissues. The typical picture comprises dwarfism, deformity of the limbs, enlargement of liver and spleen, with a large head, widely separated orbits, a short neck, kyphosis and a protuberant belly. In many cases, a corneal haze is present. The disease was first described by Hunter in 1917 at the Royal Society of Medicine, but the corneal complications were not noted until described by Hurler in 1919. Although familial, the disease is not usually inherited. In the 50 cases reported up to date, no cause has been found for it.

The first signs usually appear towards the end of the first year of life when enlargement of the head and kyphosis are seen. Some degree of umbilical hernia develops in all cases and there are characteristic deformities of the hands and feet. So far as ocular findings are concerned, a distinctive haziness of the cornea occurs in over 75 per cent. of cases, in 16 it appeared before the age of 2 years and in two instances, was present at birth. It is always bilateral and originates in the deeper layers of the stroma in the pupillary area, spreading gradually to involve the whole cornea except the epithelium and the endothelium. With the slit-lamp the opacity can be resolved into uniformly distributed tiny grey or yellowish grey dots. In a number of cases the corneae are larger than normal, simulating buphthalmos. Histologically, lipoid granules have been found in the cornea.

Commenting on their cases, the authors state that Dysostosis Multiplex is to be classed among the lipoidoses, but differs from the other forms (Tay-Sachs', Neimann-Pick's, Hand-Schuller-Christian's and Gaucher's diseases) in having additional bony changes not apparently due to lipoidosis.

F. A. W-N.