Advantages of moderate amounts and disadvantages of high amounts of magnification, *e.g.*, in telescopic systems. Effects on brightness on observer's interpupillary distance, constriction of light pencils, and disturbances by vitreous and other opacities.

Technique of use. Table of corrections for ametropia.

MR. LINDSAY REA,

**"The Use of Heparin in Thrombosis."**

*Heparin.* Its discovery—its composition—its anti-coagulating activity—its use in blood transfusion—its use in surgery—leading to its use in ophthalmology.

*Cases* cited.

*Suggested dosage* and urgency.

During the Congress the usual trade exhibition was held and Messrs. Hamblin were so good as to organize a large exhibit of topical appliances for war-time measures.

# ABSTRACTS

## MISCELLANEOUS

(1) **Yudkin, Arthur M., and Geer, Harriet A. (New Haven, Conn.)—An investigation of experimental cataracts in the albino rat. Clinical implications.** *Arch. of Ophthal.*, January, 1940.

(1) **Yudkin** and **Geer's** albino rats consistently developed cataract when fed on a diet containing 35 per cent. of galactose. At the end of the fourth to seventh day slight engorgement of the ciliary vessels was present, then vacuoles, filled with fluid, developed beneath the lens capsule and at the end of 24—30 days fully developed cataract could be observed. During this period the animals maintained normal growth and health. When the amount of galactose was reduced to 25 per cent. lens opacities developed in only two-thirds of the rats, when reduced to 15 per cent., no definite ocular change was found. Raising the proportion of protein (casein) in the diet from 15 per cent. up to 35 per cent. delayed the onset of ocular changes by about two days. Decreasing the proportion of protein in the diet to 5 per cent., made the rats more susceptible to galactose and it was found that cataract developed when only 15 per cent. of this sugar was incorporated in the diet. Even when the proportion was reduced to 10 per cent. on a low protein diet,

cataracts developed in some of the rats. Cystine in large amounts (up to 4 per cent. of the diet) seemed to be efficacious in preventing the development of galactose cataract though it led to nutmeg changes in the liver with fatty infiltration. The effect of large amounts of riboflavin in preventing galactose cataract was negative. Administration of di-nitro phenol (without galactose) failed to produce cataract. Removal of galactose from the diet of 14 rats which had developed mature cataracts resulted in clearing of the edges of the lenses, with retention of nuclear opacity.

When commenting on the cause of galactose cataract, the authors quote Bellows and Rosner's observation that this sugar decreases the permeability of "the fresh beef lens" and so may interfere with its nutrition. The clinical applications of this work are not outstanding, but Yudkin's concluding sentence is worth quoting:— The patient with incipient cataract should be placed on "a well balanced diet, supplemented by vitamin B complex and lemon juice."

F. A. W-N.

(2) **Gifford, Sanford R., and Cushman, Beulah (Chicago).— Certain retinopathies due to changes in the lamina vitrea.** *Arch. of Ophthal.*, January, 1940.

(2) **Gifford** and **Cushman** remark that it is not generally known that closely packed aggregations of hyaline bodies in the macular region may cause more or less serious disturbance of vision in some middle aged and elderly people. That this is brought about by mechanical pressure on the rods and cones, resulting finally in their destruction, was shown by histological examination carried out by Treacher Collins. The shiny crystals found in some cases are due to deposits of calcium carbonate in the hyaline bodies. Clinically, the first symptom is metamorphopsia followed by the development of a central scotoma. In one case examined by Fuchs where characteristic colloid bodies were present without visual impairment, the pigment epithelium was found to be histologically intact over the hyaline deposits and the retina undamaged. The authors consider that "typical central disk-shaped retinopathy" (? synonymous with what is called central senile exudative retinitis) may have its origin in changes in the lamina vitrea. Several observers have examined such cases histologically and have found degenerative changes in Bruch's membrane such as thickening and the formation of fissures, with in some cases, vessels growing through them; rupture of these gives rise to haemorrhages in the central disc of connective tissue. The authors describe the histological examination of a case which ophthalmoscopically showed a greyish brown mass in the outer part of the macular area, and had a central scotoma. The mass consisted of thickened retina undergoing cystic degeneration and a layer of closely packed fibrillar spindle cells between

the retina and the lamina vitrea, much of which was formed by proliferation of the pigment epithelium. The lamina vitrea varied in thickness, being extremely thin in places and showing one defect through which passed a vessel lined only with epithelium. The authors consider that it is not always necessary for complete defects in the membrane to be present in order to produce "central disk-shaped retinopathy" and suggest that in some cases, an altered permeability is present which allows fluid from the chorio-capillaris to collect beneath the pigment epithelium bringing about its proliferation and the formation of a plaque. Cracks in Bruch's membrane may also cause the appearance of angioid streaks.

F. A. W-N.

(3) **Lucic, Hugo (Baltimore).—Sensitization of rabbits to uveal tissue by the synergic action of staphylotoxin.** *Arch. of Ophthal.*, September, 1939.

(3) **Lucic's** object in this work was :—

(1) To produce hypersensitiveness to uveal tissue in rabbits.
(2) To show that histologically the reaction was similar to that in man. And
(3) To compare the reactions occurring in the eyes of sensitised and non-sensitised animals after intra-ocular injection of uveal pigment.

Burky in 1933 discovered a staphylococcus which produced an exotoxin, lethal for rabbits. By repeated intra-cutaneous injections of the toxin, into a rabbit, active immunity was produced, but at the same time there developed hypersensitiveness to the broth in which the organism was grown. Other substances could be substituted for the broth with the production of hypersensitiveness to them. The author therefore used this method in sensitising rabbits to uveal tissue, and inoculated a mixture of this tissue and the staphylococcal toxin intra-cutaneously four times at weekly intervals. As a control, a second group of rabbits received inoculations of uveal tissues only, and a third group inoculations of uveal tissues and killed bacillus subtilis. At the end of the sensitising period, the animals were tested with injections of stock uveal pigment. Tissue from the injected area was removed two weeks later for histological examination. Of twenty-two rabbits receiving inoculations of bovine uveal pigment and staphylococcal toxin, six showed histological reactions characteristic of hypersensitivity. Of eleven animals which received inoculations of rabbit uveal tissue alone, none showed any reactions and the same was true of ten animals which received inoculations of uveal tissue and bacillus subtilis, and of ten which received bovine uveal tissue alone. Two of the rabbits which gave positive reactions were subsequently tested with simultaneous injections of rabbit, swine and autogenous

uveal pigment, and in both there was a uniformly positive reaction to all three injections, thus demonstrating the organ specific antigenicity of uveal pigment.

Intra-ocular injection of uveal pigment in three hypersensitive animals, combined in one of them with injury in the form of a ragged iridectomy in the fellow eye, failed to produce a histological picture greatly different from that observed in control animals similarly treated. The histological picture did not simulate that commonly observed in sympathetic ophthalmitis.

F. A. W-N.

(4) **Berg, F. (Upsala).—Corneal astigmatism and total astigmatism. (Hornhautastigmatismus und Totalastigmatismus).** *Acta Ophthal.*, Vol. XVII, p. 137, 1939.

(4) **Berg** finds that the curvature of the anterior corneal surface is not the exclusive factor in corneal astigmatism. In combination with the astigmatism induced by the posterior surface, astigmatism against the rule 0·1–0·2 D. results, and this does not account for the total astigmatism, which is largely lenticular in origin. The oblique incidence of the visual line in the horizontal meridian of the cornea is responsible for a variable amount of corneal astigmatism against the rule though not more than 0·01 D. in most cases.

ARNOLD SORSBY.

(5) **Bedell, Arthur J. (Albany, N.Y.).—Traumatic retinal angiopathy.** *Arch. of Ophthal.*, September, 1939.

(5) The first account of this relatively unknown condition was probably that given by Purtscher at Heidelburg in 1910. **Bedell** in this paper describes three cases. The first, a man aged 21 years, sustained fractures of the lumbar vertebrae as the result of a motor car accident: in the right eye, the entire posterior pole consisted of an immense milky white swelling which obscured the disc and covered the retinal vessels. There were many superficial haemorrhages. A similar condition was present in the left fundus. Three months after the accident there were still some remnants of the cloudy opacity and a few haemorrhages. The vision of the right eye had recovered to 6/6 however, but that of the left is not recorded because the eye was amblyopic. The second case had sustained a crushing injury of the chest and when seen, a few hours after the accident, was found to have several radiating cloud-like plaques near the discs. Retinal haemorrhages were found four days later. The right eye recovered 6/6 vision, but the left developed optic atrophy and did not see better than 6/30. The third case had an injury on the right side of the head and developed fluffy white exudates in the right retina, round the disc with haemorrhages, the

left eye being unaffected. Sixteen days after the injury the exudate was flatter but there were many fresh haemorrhages. The disc gradually became pale and vision nine months after the accident had gone down to 6/120. The condition of traumatic retinal angiopathy is diagnosed from commotio retinae by the unevenness of the swelling and the recurrent retinal haemorrhages in the former, and from blockage of the central retinal artery by the same features. The cause of the condition is unknown, but it appears possible that the cloudiness develops in the posterior layers of the vitreous rather than in the anterior layers of the retina since there is no evident relation between the amount of exudate and the visual end result.

F. A. W-N.

## BOOK NOTICES

**Hydrophthalmia or Congenital Glaucoma.** By J. RINGLAND ANDERSON. Pp. 377, 116 Illustrations. Cambridge University Press. 1939. Price, 25/-.

As Sir John Parsons says in his foreword, this volume is a much more extensive treatise than its forerunner published in 1897 by Dr. Edmund le Gros, and will long remain authoritative. No better raison d'être could be given for its appearance than the opening sentences of the introduction which give an account of a conversation between an ophthalmic surgeon and the father of a child who is blind from congenital glaucoma. The material of the book is derived from an exhaustive survey of the literature, from personal experiences of the author, and from the results of a questionnaire sent to oculists in many different countries. The author justifies his use of the term hydrophthalmia in preference to buphthalmia by saying that the phrase "eye of the bull" does not suggest the failing vision or the raised tension which are essential features of the condition.

The eight chapters into which the book is divided deal successively with general aetiology; differential diagnosis; structure, development and comparative anatomy of the involved tissues; pathology; pathogenesis; treatment; prognosis and finally general reflections. At the end of the book are three comprehensive tables of analyses of specimens of the disease and at the end of each chapter is a short summary of its contents.

It is not possible in a short review to do justice to the many admirable features of this book. To the practising ophthalmic surgeon, the chapter of greatest interest will probably be that which is devoted to treatment. Many and various are the operations performed, and although trephining is in most general use, Herbert's