Staff Meeting Bulletin
Hospitals of the » » »
University of Minnesota

Diseases of the Eye

Volume XVII | Friday, May 3, 1946 | Number 21

STAFF MEETING BULLETIN
HOSPITALS OF THE . . .
UNIVERSITY OF MINNESOTA

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INDEX

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I.

UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
CALENDAR OF EVENTS
May 4 - May 10, 1946
Medical Visitors Welcome

No. 112

Saturday, May 4

7:45 - 8:50 Orthopedics Conference; Wallace H. Cole and Staff; Station 21, U. H.

9:00 - 9:50 Surgery-Roentgenology Conference; O. H. Wangensteen, L. G. Rigler, and Staff; Todd Amphitheater, U. H.

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; M-515 U. H.

9:00 - 9:50 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustis Amphitheater.

10:00 - Anatomy Seminar; A Review of Experimental Chemotherapy of Neoplastic Disease; Ruby Engstrom; 226 I. A.

Sunday, May 5

11:00 - 1:50 Obstetrics and Gynecology Grand Rounds; J. L. McKelvey and Staff; Station 44, U. H.

Monday, May 6

9:00 - 9:50 Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U. H.

9:00 - 10:50 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; Interns Quarters, U. H.

12:15 - 1:15 Obstetrics and Gynecology Journal Club; M-435, U. H.

12:30 - 1:20 Physiology Seminar; Potentialities in Histo- and Cyto-Chemistry; David Glick; 214 M. H.

12:30 - 1:20 Pathology Seminar; Experiences in World War II; N. Lufkin; 104 I. A.

8:00 - Elias P. Lyon Lecture; Enzymatic Reactions in Carbohydrate Metabolism; Prof. Carl F. Cori, Dept. of Pharmacology, Washington University, St. Louis; Natural History Museum Auditorium.

8:00 - Hennepin County Medical Society; Injuries of the Cervical Spine; Dr. Arthur G. Davis, Shriners' Hospital for Crippled Children, Philadelphia; 20th Floor Medical Arts Bldg., Minneapolis.

Tuesday, May 7

9:00 - 9:50 Roentgenology-Pediatrics Conference; L. G. Rigler, I. McQuarrie and Staff; Eustis Amphitheater, U. H.

12:30 - 1:20 Pathology Conference; Autopsies; Pathology Staff; 104 I. A.

12:30 - 2:30 School of Public Health Seminar; Experiences of the Laboratory of 26th General Hospital; Paul Kabler; Room 15 MoS.

2:00 - 3:00 Dermatology and Syphilology; H. E. Michelson and Staff; Veterans' Hospital, Bldg. III.

3:15 - 4:15 Gynecology Chart Conference; J. L. McKelvey and Staff; Station 54,UH.

3:45 - 5:00 Pediatric Staff Rounds; I. McQuarrie and Staff; W-205 U. H.

4:00 - 4:50 Surgery-Physiology Conference; Congenital Pulmonary Stenosis; Drs. Paine and Keys; Eustis Amphitheater.

5:00 - 5:50 Roentgenology Diagnosis Conference; Drs. Oscar Lipschultz and Donald Peterson; M-515 U. H.

Wednesday, May 8

8:00 - 8:50 Surgery Journal Club; O. H. Wangensteen and Staff; M-515 U. H.

9:00 - 10:50 Neuropsychiatry Seminar; Staff; Station 60 Lounge, U. H.

11:00 - 11:50 Pathology-Medicine-Surgery Conference; Carcinoma Stomach, Myocardial Infarction; E. T. Bell, C. J. Watson, O. H. Wangensteen and Staff; Todd Amphitheater, U. H.

12:30 - 1:20 Physiology Chemistry Journal Club; Staff; 116 M. H.

4:00 - 6:00 Medicine and Pediatrics Infectious Disease Rounds; W-205 O. H.

4:30 - Neurophysiology; Investigations on the Vago-Insulin System; Mrs. Carmen Casos; 113 MoS.

4:30 - *Lecture on Antibiotics--Antibiotics, Nature and Formation; Dr. Selman A. Waksman, Prof. of Microbiology, Rutgers U.; Room 15 MoS.

Thursday, May 9

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; Todd Amph., U. H.

12:15 - 1:15 Pediatrics Seminar; Irvine McQuarrie and Staff; 6th Floor Eustis.

12:30 - 1:20 Physiological Chemistry; Karl Sollner; 129 M. H.

4:30 - 5:20 Ophthalmology Ward Rounds; Erling Hansen and Staff; E-534, U. H.

4:30 - Bacteriology Seminar; 214 M. H.

5:00 - 5:50 Roentgenology Seminar; Standards in the Administration of Radiation Therapy; J. C. McKelvey; M-515 U. H.

Friday, May 10

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amph., U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221 U. H.

10:30 - 12:20 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Otolaryngology Department; U. H.

11:50 - 1:15 University of Minnesota Hospitals General Staff Meeting; Treatment of Wound Infections with Special Reference to Secondary Osteomyelitis; Edward Evans; New Powell Hall Addition Amphitheater.

1:00 - 2:00 Dermatologic Allergy; Dr. Stepan Epstein; W-312 U. H.

2:00 - 3:20 Dermatology and Syphilology; Presentation of Selected Cases of the Week; H. E. Michelson and Staff; W-312 U. H.

1:30 - 2:20 Roentgenology-Neurosurgery Conference; H. O. Peterson, W. T. Peyton, and Staff; Todd Amphitheater, U. H.

8:00 - *Lecture on Antibiotics--Production and Activity of Antibiotics; Dr. Selman A. Waksman; Room 15 MoS.

*In addition to these lectures there will be a number of informal seminars to be announced in the Daily.

II. DEVELOPMENTAL ABNORMALITIES AND DISEASES OF THE EYE

G. H. Dolmage

In the summer of 1940 there occurred in Australia an epidemic of German measles, which was characterized by its severity and the presence of associated complications. Early the following year an unusual number of cases of congenital cataract were diagnosed in Sidney. Gregg[1] became interested in the matter, studied these cases, and realized that something more than the law of averages was at work. He went to the bottom of the matter, and in time detected the fact that many of these children with cataracts had been born of women who had had rubella early in pregnancy. Additional investigation was made by him and associated workers, and the following facts were brought to light:

1. 78 cases of congenital cataract were found; of these, 62 were bilateral.

2. All layers of the cataractous lenses were involved except the outer, and the defects were considered as having originated early in embryonic life.

3. The babies were generally small, ill-nourished, and feeding problems. 44 of them had associated heart lesions.

4. 68 of these 78 children had been born of mothers who had had German measles, in most cases in the first or second month of pregnancy. Of the other 10 mothers, 7 could not give a satisfactory history; 1 had had some form of kidney disease; 2 had definitely not had measles.

Quite naturally, this report aroused the scientific interest of medical men, and case reports with rather similar findings began to appear in medical writings. Prendergast[2], for example, has reported the result of a sruvey conducted in 1944 among California pediatricians, obstetricians and ophthalmologists. His findings are these:

1. 80 cases of congenital cataract were reported. It is likely that there were some duplications.

2. 10 women were known to have had rubella during the first trimester of pregnancy.

3. Of these 10 women, 5 gave birth to children with congenital cataracts.

There are many earnest and thorough workers who are not convinced that there is cause-and-effect relationship between the two phenomena of measles during early pregnancy, and congenital lens defects. Be that as it may, one of the results of the interest displayed in this matter has been to focus the attention of many doctors on the complex matter of developmental anomalies, abnormalities and diseases of the eye. This is the subject for discussion today; as such, the paper is in great part an abstract of the exhaustive works of Mann[3] and Duke-Elder[4].

Before one becomes involved in the actual matter of eye defects, it would be well to review as briefly as is consistent with an understanding of the subject, certain salient embryological points, insofar as they deal with the eye and its related structures. It will be recalled that early in intrauterine life, two evaginations appear from the forebrain, which become known as the optic vesicles. By about the 4 mm. stage, these are found attached to the brain wall by constricted structures called the optic stalks. Each vesicle becomes invaginated when rapid marginal growth occurs, and the optic cup is formed. This invaginated layer becomes the nervous part of the retina; the outer part of the cup against which the invaginated part rests becomes the pigmented part of the retina. While these changes are taking place, the mesoderm that surrounds the cup is becoming transformed into the fibrous sclera and the vascular uvea.

This original invagination of the optic cup does not stop at the point where it joins the optic stalk, but continues back at one point as a groove

in the inferior surface of the stalk. As a result, there occurs a notching of the optic cup, and the development of a furrow-like groove along the stalk known as the choroid or fetal fissure. During the seventh week of intra-uterine life this fissure will close, with the result that there is formed a tube within a tube.

In the meantime, the surface ectoderm overlying the optic vesicle has thickened into the lens plate. This likewise invaginates, and in so doing becomes the lens vesicle. The vesicle frees itself from the parent ectoderm, and comes to occupy the concavity of the optic cup. The cells of the inner wall of the lens vesicle proliferate, obliterate the original cavity, and form the early transparent lens fibers. Later, the lens will grow when additional fibers are formed by cells lying in the region of the equator of the lens.

The central artery of the retina courses along the groove in the optic stalk to the back of the optic cup, which it enters through the fetal fissure; it then becomes known as the hyaloid artery. A bulbar swelling forms in the region of what will be the disc, from which appear buds that grow into the nerve fiber layer of the retina. These buds will in time become canaliculized. They gradually extend anteriorly to reach the ora serrata. The main part of the hyaloid artery continues forward through the vitreous cavity to reach the posterior surface of the lens, where it breaks up into a capillary network called the posterior part of the tunica vasculosa lentis. Capillaries from this network pass forward between the equator of the lens and the rim of the optic cup, to anastomose with annular vessels that have come from the hyaloid artery, and which run around the rim of the cup. Still other branches from this posterior capillary network form the lateral part of the posterior portion of the tunica vasculosa lentis. Later, vessels will pass from the annular vessel over the anterior surface of the lens as the anterior part of the tunica vasculosa lentis. The central part of this anterior lens structure becomes known as the pupillary membrane, which consists of vascular loops arranged in three tiers of arcades. At about seven or eight months, the central part of this vascular structure will begin to atrophy. The atrophic process extends peripherally to the first arcade, which persists as the permanent lesser circle of the iris.

The iris is formed by the forward growth of the margin of the optic cup. The inner layer develops from the part caeca of the retina, which is the inner, non-nervous zone near the rim of the cup. The outer layer is formed from an extension of the pigmented layer of the retina. These two layers blend. The pupillary muscles are formed from the pigment epithelial layers.

Fibers growing from neuroblasts in the nervous or inner layer of the retina form the optic nerve. These fibers pass toward the optic stalk and grow through the substance of the stalk back to the brain.

The cornea contains both ectodermal and mesodermal elements. The surface epithelium comes from surface ectoderm. The stroma is thought to be formed by the ingrowth of mesoderm into a pre-existing fibrillar material derived from the lens vesicle and surface ectoderm.

The eyelids develop as mesoblastic, epithelium-covered folds, which appear at the 16 mm. stage. They extend gradually over the eye from above and below to meet in the midline, where they become adherent to each other for a time. During this period of adherence, the glands, muscle fibers near the margin, and the tarsal plates are differentiated. The epithelial adhesions break down by the end of the fifth month, and the palpebral fissure results.

With this brief embryological background, we pass to another introductory matter: the classification of abnormalities of the eye according to type. We find that there are two principal types, with subdivisions:

I. Simple arrest of development or growth. As a result of this, we have present in the final stages of development a condition that would have been normal at an ear-

lier age. Example: if the optic vesicle fails to bud off the forebrain, anophthalmos results.

2. Abnormal growth. This is a pure aberration.

   A. Simple overgrowth. Example: the myelin sheath that normally develops forward around the optic nerve fibers to the lamina cribrosa may extend on to the retina.

   B. Abnormal development. Here we have a condition that does not resemble the condition of the eye in any normal stage. Example: corneal opacity.

   C. Arrest of development, followed by normal development. Example: microphthalmos with cyst formation. The eye in size resembles the eye that is normal at an earlier period; it is abnormal in that it has a thin-walled cyst attached to it.

An additional preliminary point for discussion is the matter of the several growth periods. As far as the eye is concerned, there are nine of these. Their appreciation enables us to understand better the time at which an abnormality developed; also, we have a better knowledge of just what factor or factors could have caused the defect to appear. These growth periods are as follows:

1. The optic pit developes into the optic vesicle, and the lens plate forms. This period ends at about 3-4 weeks.

2. The lens pit and vesicle appear. The optic cup is formed. This period ends at the end of 4 weeks.

3. The fetal fissure closes. The lens separates from the surface. The primary lens fibers form. Retinal differentiation begins. The tunica vasculosa lentis begins. This period ends at the sixth week.

4. Secondary lens fibers begin to form. The tunica vasculosa lentis has been formed. Ectodermal layers of the iris begin. Period ends at 3 months.

5. The central retinal artery, ciliary muscle, pupillary muscles, sclera, ciliary body, and outer layer of the choroid appear. The posterior vascular capsule of the lens begins to retrogress. Period ends at fourth month.

6. The pupillary membrane retrogresses. Medullation of optic nerve begins. The period ends at the seventh month.

7. The hyaloid artery disappears. The medullation of the optic nerve reaches the lamina cribrosa. This period ends at the ninth month.

8. In the neo-natal period, 4 to 6 months after birth, the macula differentiates.

9. In the post-natal period, there is formation of the secondary lens fibers, and growth of the entire eye. This period ends at about the age of 25 years.

The earliest periods are the shortest, and potentially the most dangerous, as gross abnormalities arise then. Once the organs have established themselves, and growth is occurring in an orderly manner, the abnormalities that develop are never so obvious.

At this point in our discussion, it seems advisable to state the various factors that can bring about eye defects. Briefly, their general classification is as follows:

1. Germinal factors. These may be found in the sperm, the ovum, or both. They account for familial and hereditary defects either present or not apparent at birth. The pernicious factor is present in the chromosomes. Abnormalities due to germinal factors have these characteristics: they are likely to be present in several children of a parent, or in several generations; usually both eyes are affected.

2. Mechanical factors. Abnormal pressure on or trauma to the fetus is thought to cause some defects.

3. Transplacental factors. It is believed that toxins, hormones, drugs, virus organisms and bacteria can pass through the placenta from the mother to the fetus, with injurious effect. The mother may or may not be similarly affected. For example, a child may be born with an active corneal ulcer when the mother has variola; the infection here is through the bloodstream.

In general, we must confess our ignorance as to the primary cause of many developmental defects.

It is now time to pass to specific abnormalities and diseases of the eye. Because of their multitude and the time limitation imposed upon today's discussion, many of them will have to be dismissed with their mention. Some of those of more general interest and importance will be discussed in more detail.

A. Deformities of the orbit.

1. Failure of the orbit to develop.

2. Oxycephaly. Because of the shallow orbits, the eyes protrude. Poor visual acuity may be present because of associated optic atrophy.

3. Hypertelorism. The orbits in this condition retain their primitive lateral position.

B. Abnormalities affecting the eye as a whole.

1. Anophthalmia. The development of the entire forebrain may be suppressed or abnormal. Or the optic pits may fail to deepen and to form an outgrowth from the forebrain. In this condition, certain parts of the eye that develop without stimulus from the optic vesicle may be present (eyelids, lacrimal apparatus, sclera, conjunctival sac, cornea, extraocular muscles).

2. Cyclopia. There may be a single median eye; or the two eyes may be in contact with each other; or they may be in various stages of fusion. In its fullest development, the condition is accompanied by fusion of the superior maxillae, absent nasal cavity, union or fusion of the two orbits, and a small proboscis or snout located about the fused orbit.

3. Cystic eye. This results when development becomes arrested after the optic vesicle has appeared, but before it has become completely invaginated.

4. Microphthalmia with cysts.

5. Buphthalmia. Abnormalities in the region of the angle of the anterior chamber may obstruct the drainage of the intra-ocular fluid. As a result, the intra-ocular pressure rises and congenital glaucoma results. The entire eye enlarges, as at this stage the coats of the eye are sufficiently elastic to permit stretching. The causes are regarded as being one or more of these factors:

   a. Strands of trabecular tissue stretching across the angle, blocking drainage.

   b. Adherence of the peripheral part of the iris to the cornea.

   c. Absence of the canal of Schlemm.

   Buphthalmos is usually bilateral. There is a marked hereditary influence.

6. Typical colobomata of the iris, choroid and retina. The term coloboma means "curtailed." It refers to any notch, gap, hole or fissure, congenital or acquired. The typical cases show defects that lie near the fetal fissure, and are thought to be due to

failure of that cleft to close properly. These run down and slightly inwards around the eye. With the ophthalmoscope, the examiner sees a white area in the fundus. If the condition involves the periphery, there will be seen a gap in the ciliary body and the iris.

The factor of heredity is definite in this condition; the deformity is usually present as a dominant characteristic.

C. Lids, lacrimal apparatus and orbital contents.

1. Ablepharia, microblepharon, lagophthalmia. If there is complete failure of the lids to develop, as in ablepharia, the corneal epithelium undergoes metaplasia into skin, and we see the skin from the forehead passing directly down into that of the cheek. In lagophthalmia, the lids are so short that they meet over the eye only with difficulty.

2. Coloboma of the lids. Congenital notches may be seen along the margins, associated with the absence of lashes or glandular structures at the involved points. The condition is non-hereditary; it is often associated with other alterations of the surface structures of the head and face; it may be due to the pressure of amniotic bands, to amniotic adhesions, or to localized failure of adhesions of the lid folds.

3. Epicanthus. This is a fold of skin, continuous with the skin of the bridge of the nose, that extends over and partially hides the inner canthus. The patient has a flat bridge of the nose and poorly developed eyebrows. Epicanthus may be due to a simple arrest of development. It may be seen combined with ptosis.

4. Abnormalities of lid margins. The principal of these are distichiasis, congenital alopecia, and white or piebald cilia. In distichiasis, the Meibomian glands are replaced by a second row of lashes. In congenital alopecia, the hair of the entire body, as well as the lashes, is absent. In the white or piebald cilia cases, a localized streak of white hair on the scalp is continued down through the eyebrows and lashes.

5. Extra-ocular muscle anomalies. Developmental paralyses are due to central nervous system lesions, or defects in the muscles themselves. An important example of this is the retraction syndrome, in which we have paresis of the internal rectus muscle combined with fibrosis of the external rectus; as a result, adduction of the eye is weak, and accompanied by retraction of the eye into the orbit and partial closure of the lids.

6. Lacrimal apparatus. The lacrimal passages may fail to develop by the normal process of canalization of the line of buried epithelium formed when the maxillary process overlaps the lateral nasal process. There may be accessory puncta, abnormally-shaped puncta, or fistulae of the lacrimal sac.

D. Conjunctiva. When the lid folds fail to form, the conjunctiva will be absent. In other cases it may show partial metaplasia, taking on the character of moist skin, and revealing the presence of hair follicles and sebaceous glands. It may show pigmentation. There may be abnormal folds and bands. It may contain tumors: dermo-lipomata, angiomata, lymphangiomata. It may be inflamed, or reveal the presence of a pustule or ulcer; this infection is thought to come by means of the placental bloodstream.

E. Sclera.

1. Blue sclera. This is strongly hereditary, and is likely to be associated with other abnormal-

ities in the supporting tissues of the body, such as multiple fractures or dislocations; deafness also is often present. The sclera is abnormally translucent, with the result that the uveal pigment shows through. It may be due to developmental arrest, since young tissues show greater translucency than do older structures.

2. Congenital posterior ectasia. The scleral condensation starts anteriorly, and reaches the posterior pole relatively late in development. It is understandable that if this condensation were to be delayed, a normal intraocular pressure might cause the posterior pole to stretch. This may be a factor in the development of myopia.

3. Miscellaneous: cysts; melanosis; presence of cartilage.

F. Cornea. This structure during its development never normally contains blood vessels. During part of its history, the cornea is not protected by the lids, but remains in direct contact with the amniotic fluid; thus it may be influenced by abnormal amniotic factors.

1. Absence of cornea. If the lid folds fail to form, the cornea remains exposed, and undergoes metaplasia into fibrous tissue covered by ordinary skin.

2. Lack of normal transparency. The causes of this are said to be numerous: imperfect differentiation of Descemet's membrane and endothelium; contact between the lens and the cornea; abnormal development of the stroma; the union of the cornea with strands of the pupillary membrane; intrauterine infection; birth injuries (as the rupture of Descemet's membrane).

3. Abnormalities of size.

a. Microcornea. The cornea in this condition is less than 10 or 11 mm. in diameter. Usually the entire anterior part of the eye is small; the posterior segment is normal. Vision is normal. There is a tendency for this eye to develop glaucoma. The condition is caused by an arrest of development after the 5th month of intrauterine life.

b. Megalocornea. This must not be confused with buphthalmos, in which the entire eye is enlarged from increased intra-ocular pressure. In megalocornea the diameter of the cornea is from 13 to 18 mm. The condition is usually bilateral, and associated with enlargement of the anterior segment of the eye. It seems to be hereditary, usually transmitted through females. It is regarded as being due primarily to a developmental overgrowth.

4. Abnormalities of curvature. The cornea may be flat, having about the same curvature as the sclera. It may be vertically oval in the horizontal or vertical meridian. It may be conical, due to central thinning and yielding.

5. Miscellaneous. There may be pigment cells near the limbus. An arcus juvelilis may be present. The cornea may show the effects of inflammation: the child with congenital syphilis may have an active or inactive interstitial keratitis at birth; the smallpox virus may infect him in utero, and cause him to show at birth the presence of an active or healed corneal lesion.

G. Abnormalities of the anterior chamber. This chamber may be absent if the structures derived from the surface ectoderm, as the cornea and lens, fail to develop. The abnormalities already discussed under the subject of buphthalmos may be present. There

may be tiny pigmented bodies found in the anterior chamber.

H. Iris and pupil. Two primary embryonic tissues are involved in the development of the iris: from the mesoderm, the stroma and pupillary membrane form; from the neural ectoderm of the margin of the optic cup, we have developed the two layers of pigment epithelium and their derivatives, the sphincter and dilator muscle.

1. Aniridia. This term is not quite correct, as the iris is present in an extremely rudimentary condition. It is clinically absent, however, as the short stump is hidden behind the corneo-scleral junction. The influence of heredity is extremely strong in this condition; the anomaly behaves as a dominant characteristic. There are many theories as to its cause, the best of which credits its appearance as being the result of a failure of development of the retinal epithelium, or of an aberrant development of the vascular mesoderm. It is likely to occur in association with poor vision, nystagmus, an absent fovea, cataract, microphakia, and ectopia lentis. The condition is usually bilateral.

2. Coloboma of iris. In this condition, there is a partial or total absence of a sector of the iris. In typical cases, this is secondary to a defect of the optic cup, in which as associated coloboma of the choroid and retina will be seen. The defect is usually single, triangular in shape, and may or may not involve the entire thickness of the iris. It may be seen as a notch at the pupillary margin, as a hole in the substance of the iris, or as a defect at the ciliary border (iridodiastasis). There is a strong hereditary influence; the deformity is transmitted as a dominant characteristic. There are two possible causes: (a) A localized failure of a portion of the ectodermal margin of the optic cup. (b) A persistent vascularized strand belonging to the fibrovascular capsule of the lens prevents the rim of the optic cup from growing forward at this point.

3. Persistent pupillary membrane. The failure of part of the pupillary membrane to atrophy is very common. In rare cases, the entire membrane persists, with circulating blood seen in the vessels. Not quite so rare is the complete persistence of the membrane without the presence of circulating blood. More commonly, there is incomplete persistence of the vascular structure. The condition is not hereditary.

4. Albinism. This is a congenital deficiency of pigmentation, not confined to the iris. The eye shows a deficiency of retinal pigmentary epithelium with pink or red pupils, translucent iris, defective vision, nystagmus, intolerance to light, high refractive error (usually myopic astigmatism). The poor vision is due to a deficiency of retinal pigment, which is necessary for proper functioning of the rods and cones, and to the dazzling that results from the entrance of an excessive amount of light through the translucent outer coats of the eye. The condition is hereditary.

5. Miscellaneous.

a. Corectopia. The normal pupil is located slightly down and in from the center of the iris. In corectopia it is displaced much farther from the center, usually up and out.

b. Slit-like pupil.

c. Polycoria. The presence of several pupils; rare.

d. Holes in the iris.
e. Absence of dilator or sphincter muscle.
f. Ectropion of the pigment border of the pupil.
g. Entropion of the pupillary border.
h. Congenital cysts of the iris, These may be derived from surface epithelium, or from the neural epithelium of the iris.
i. Congenital anterior synechiae, bands that run from the pupillary border to the cornea.

I. Lens.

1. Congenital aphakia. The lens normally is formed from the surface epithelium in response to a stimulus from the optic vesicle. The condition of congenital aphakia is very rare; in fact it may not occur. The aphakia seen in some of these cases may be due to early degeneration of the lens structure, with secondary absorption.

2. Coloboma. This is fairly common. The defect is usually down and slightly in or out, and is likely to be associated with a defect in the zonule in this region.

3. Microphakia. In this condition there is a mal-development of the entire lens system. The zonule is weak, and does not exert the normal pull on the lens; as a result, the lens assumes a somewhat spherical shape.

4. Lenticonus. Either the anterior or the posterior surface of the lens shows a conical projection.

5. Cataract. There are a variety of forms of congenital cataract. At no stage in its development is the lens normally anything but transparent; thus all congenital opacities are aberrations and never arrests. The cataracts do not necessarily have to be present at birth; they may appear in infancy, adolescence, or even in adult life. Some of the forms are these:

a. Congenital capsular cataract. Here the opacity is in the capsular epithelium.

b. Anterior polar capsulolenticular cataract. These are relatively common. A white plaque is seen, often associated with attached strands of pupillary membrane.

c. Posterior polar capsulolenticular cataract. Usually the opacity is a persistence of the hyaloid artery, or of the posterior fibrovascular sheath of the lens.

d. Lamellar or zonular cataract. This is an opacity involving one layer or zone of the lens. It is seen as hundreds of tiny opaque white dots arranged in one or more concentric zones of the lens. It is associated with other disorders, such as hypoplasia of tooth enamel and thickening of the ends of the ribs. It may be due to a temporary upset of the parathyroid-calcium metabolism.

e. Congenital Morgagnian cataract. The nucleus is intact, but the capsule is filled with a milky fluid. It is probably due to a metabolic disturbance near the end of fetal life.

6. Ectopia lentis. This condition of congenital dislocation is usually bilateral and symmetrical. The lens is most commonly displaced upward. Often there is marked familial and hereditary tendencies. The cause is not known, but one theory has it that the dislocation results from a primary abnormality of the zonule.

Rados[6] reviewed many of the cases of Marfan's syndrome in 1942. He believes that we are dealing with a symptom complex grouped around the central finding of extreme slenderness of the extremities. He believes that genes located near each other within one chromosome are damaged together; in one case the bones of the extremities may be involved along with the eyes, while in another, the extremities are affected along with the heart. In 204 cases of arachnodactyly studied by Rados, he found dislocation of the lens present in 126, other ocular defects in 39, and heart lesions in 65. Miscellaneous abnormalities are poorly developed muscular tissue, thin chests, winged scapulae, abnormal spinal curvatures.

J. Ciliary body. The principal anomalies of this structure are colobomata and displacement of the ciliary processes.

K. Abnormalities of the fundus.

1. Disc and optic nerve.

a. Crescents. These are not to be confused with the temporal crescents seen in myopia. They are usually found below the disc, and are due to failure of the pigmented outer layer of the optic cup to reach the insertion of the optic stalk. The nuclear layers also fail to reach this area, so we have only the nerve fiber layer placed between the lower edge of the disc and the lower edge of the nerve.

b. Situs inversus. Here the vessels pass toward the nasal instead of the temporal side of the disc.

c. Colobomata and pits in the disc.

d. Pseudo-neuritis. Normally at the 17-mm. stage, a small, conical mass of neuroepithelial cells (Bergmeister's papilla) gets cut off from the inner layer of the optic cup at the disc by the nerve fibers that pass at this point. This mass normally hypertrophies, then atrophies. If atrophy is deficient, and the glial tissue fills up the cup and disc, a condition resembling the picture of papilledema will be seen; this is known as pseudoneuritis.

2. Nerve fibers.

a. Opaque or medullated nerve fibers. Normally, the process of medullation of the optic nerve starts centrally, and by birth has reached to a point just behind the lamina cribrosa, where the process stops. In certain cases medullation begins again after birth at the top of the disc, and spreads over the fundus for varying distances. These medullated fibers have a feathery, silken appearance when seen with the ophthalmoscope, as they are more opaque than non-medullated fibers. They are usually of no clinical importance; they may be mistaken by the inexperienced for pathological processes, however.

3. Anomalies of vascular elements.

a. Persistence of hyaloid artery. This is a fairly common finding. Normally the artery ruptures at about the seventh month of intrauterine life, with the two ends curling up and retracting. There are several grades of anomalous persistence: 1. The entire artery may be seen extending from the disc to the lens. It may or may not be patent. 2. The pos-

terior end may remain attached to the disc and extend into the vitreous, where it moves around with movement of the eye. 3. The anterior end may remain attached to the posterior surface of the lens.

b. Pre-retinal vascular loops. These may be either arterial or venous, with single or multiple loops.

c. Cilioretinal artery. This is a branch of the short posterior ciliary arteries. Instead of passing into the choroid, it appears at the disc, usually on the temporal side, and supplies part of the retina.

d. Persistence of vascular sheath of lens. A fibrous membrane may be formed behind the lens, bearing some resemblance to a retinoblastoma. Or the anterior part of the vascular sheath may persist, and form the persistent pupillary membrane, which has already been discussed.

4. Abnormalities of the retina and choroid.

a. Melanosis and congenital pigmentation. There may be a generalized deposition of pigment, in which case the condition is known as melanosis; the fundus then appears even darker than that of the negro. Or the pigmentation may be localized.

b. Albinism. This has been discussed under the section on the iris.

c. Colobomata. This likewise has already been discussed.

d. Retinitis pigmentosa. This is a familial condition, characterized by night blindness, contraction of the visual field, narrow retinal arteries, waxy-appearing disc, and patches of pigmentation in the periphery arranged in the form of bone corpuscles. It is seen in children born of consanguineous marriages; it also occurs in dominant, recessive, and sex-linked recessive forms.

e. Laurence-Moon-Biedl syndrome. This is characterized by pigmentary degeneration of the retina, mental retardation, hypogenitalism, obesity, and polydactyly.

f. Choroideremia. In this condition the choroid is absent in part or in its entirety. With the ophthalmoscope, the part of the fundus involved appears as a bright, glistoning white surface.

g. Macular dystrophies. There are a number of degenerations of the macular region that are characterized by stippling and pigmentary changes. They may be present at birth or occur shortly after, in which case they are referred to as amaurotic family idiocy; cases of this nature are associated with cerebral disturbances, as the name implies. They may make themselves known during childhood, when they are known as familial cerebromacular degeneration; both visual and cerebral findings are present. If the dystrophy occurs during adolescence, it is known as juvenile macular degeneration; the brain may not be damaged in this form.

In addition to the stippling and pigmentary changes noticed in the macular region, the following findings are likely to be present: secondary optic atrophy, narrow retinal arteries, and

retinal atrophy.

h. Glioma or neuroblastoma. Virchow thought that this malignant tumor arose from pre-existing glial cells in the retina; hence the term glioma. We now know that the lesion arises largely in one or another of the nuclear layers and not in the ganglion cell layers. There are two principal types of this form of tumor.

(1) Neuroepithelioma. This is the differentiated and less malignant form. Microscopic examination shows large columnar cells with round nuclei arranged in a radial manner around a central cavity, so as to form a rosette.

(2) Retinoblastoma. This is a highly malignant form. It is less differentiated than the other type. Under the microscope, the tissue is seen to be loaded with small closely-packed cells containing large nuclei and scanty protoplasm.

The factor of heredity is very important. Many workers have reported the high familial incidence. One series of cases showed the neoplasm to be present in four successive generations. Rados[7] and others state that the tendency to inheritance may be both dominant and recessive.

Case No. I. K.D., a female child, was born on March 31, 1944. Her father had had one eye removed at the age of 16 months for glioma, and the second at the age of 4 years. In Jan. 1945 the mother noticed that the girl seemed to have difficulty in seeing, so a physician was consulted. He found large binocular intraocular tumors, apparently gliomas. She was referred to the University of Minnesota Hospital, where the diagnosis was confirmed. The posterior portion of each fundus was filled with a whitish mass, and there were areas of retinal separation. X-ray examination revealed the presence of calcified areas in the eyes. Both eyes were enucleated on Feb. 13, 1945.

Case No. II: M.D., a sister of the girl whose history has been recounted above, was born on Aug. 28, 1945. At the age of 2 months she was found to have a small, rounded tumor mass in the region of the left disc. At $3\frac{1}{2}$ months, this tumor had doubled in size. She was admitted to the University of Minnesota Hospitals, where the left eye was enucleated on Jan. 24, 1946. At the time of her operation, the other eye was examined under anesthesia, and was found to be normal. She has been examined at frequent intervals since the enucleation, and has recently been found to have a similar lesion in the right fundus.

There are many other developmental abnormalities and defects that one might take up, but they cannot be considered at this time. The number that have already been discussed enable one to see how varied are the manifestations of nature's errors. The secrets of some of these abnormalities we know; others we shall probably know in the future; still others may possibly always escape solution. The surprising thing is that in a structure as complex as the eye, anomalies and abnormalities do not appear more often than they do.

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III. GOSSIP

Next week is Orthopaedic Week at the Center for Continuation Study. Dr. Arthur George Davis of Erie, Pa., will be here May 6 and 7, 1946, to discuss "Injuries of the Spine". His special contribution is overlooked: injuries of the cervical spine, which he will present at the Hennepin County Medical Society, May 8 and 9, 1946. Walter P. Blount of Milwaukee will discuss "Disorders of the Hip Joint" and on the same days Emil D. Hauser, of Chicago, will tell of his investigations of "Disorders of the Feet". Doctor Hauser is a former Minnesotan, whose book on the feet has become a must. On May 7 and 9 special trips will be made to the Bone and Joint Services at the Veterans' Hospital, Shriners, and Gillette. The official spelling, I am told, is Orthopaedic from the Greek, to distinguish it from the Latin origin of a similar term (ped.). Following this week will be Neurosurgery Week, and guests will include Tracy Putnam of New York and Ted Erickson of Wisconsin, a former Minnesotan who has distinguished himself by his "Studies on Epilepsy."..The Ramsey County Medical Society gave a rousing welcome to its former service men in a party and dinner at the St. Paul Athletic Club, Tuesday, April 30. The Ramsey County Medical Society is the most friendly, cooperative, organized medical group in the country, bar none. I would guess that every doctor in town was there and no holds were barred. This same cooperative attitude is evident at the Ancker Hospital where some of our most active teaching for veterans is being done...Selman A. Waksman, Ph.D., Sc.D., Professor of Microbiology, Rutgers University will give the first annual Duluth Clinic Lecture series. This is a special gift from the Duluth Clinic for the purpose of inviting a distinguished clinician or scholar to the campus to spend a week with the staff and students. Dr. Waksman's lectures will be "Antibiotics, Nature and Formation," Wednesday, May 8, 4:30 p.m.; "Production and Activity of Antibiotics," Friday, May 10 at 8 o'clock; and "The Story of Streptomycin," Monday, May 13 at 4:30, Room 15, Medical Sciences Bldg.... The Annual Meeting of the Minnesota State Medical Association will be held May 20, 21 and 22, 1946 in the St. Paul Civic Auditorium. All staff members are invited to attend....Harry A. Wilmer, M.D. has written another best seller called "Corky the Killer," a story of syphilis. The introduction is by Paul A. O'Leary, M.D., Mayo Clinic, and forwards are by Joseph Moore, M.D., and Kendall Emerson, M.D. It is published by the American Social Hygiene Association, and dedicated to Dr. Richard E. Scarmon, University of Minnesota. The story is written in the same vein as "Huber the Tuber" with excellent illustrations by the author. It is scientifically accurate and should be of great interest to inquiring persons. Like its mate in tuberculosis, it can be read with great profit by medical men. Harry A. Wilmer is to be congratulated for his contributions to Health Education....Philip K. Allen, M.D., Dermatologist, Minnesotan, announces the association of Walter R. Nickel, M.D., Minnesotan, with him in practice in Suite 706, Medical Dental Bldg., San Diego, Calif... ..Charles E. Rea, M.D., announces his return from medical service and the reopening of offices at 917 Lowry Medical Arts Bldg., St. Paul 2, Minnesota. Charlie, looking more sleek than ever, still retains some of the mysterious air which he cultivated while stationed in Oak Ridge, Tennessee in charge of the hospital..... Most unusual announcement card came from Springfield, Massachusetts, where Walter W. Williams, Clinical geneticist, announces his return from the service in the United States Navy, and the resumption of his practice in the diagnosis and treatment of male and female infertility....