The significance of heredity in ophthalmology : a Tasmanian survey / by J. Bruce Hamilton.

THE SIGNIFICANCE OF HEREDITY IN OPHTHALMOLOGY.

A TASMANIAN SURVEY.

To My Wife

THE SIGNIFICANCE OF HEREDITY IN OPHTHALMOLOGY.

A TASMANIAN SURVEY.

By

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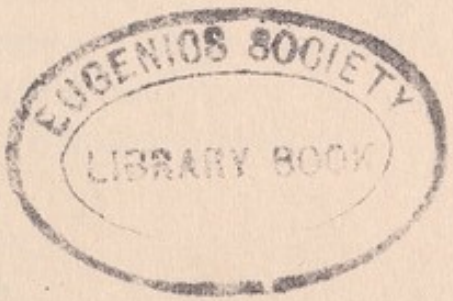

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THE SIGNIFICANCE OF HEREDITY IN OPHTHALMOLOGY.

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THE SIGNIFICANCE OF HEREDITY IN OPHTHALMOLOGY.

A TASMANIAN SURVEY.

By

J. BRUCE HAMILTON.

Part 1

1. HISTORICAL.

Although Van Diemen's Land was discovered by a Dutchman — Abel Jansen Tasman — on November 24th, 1642, it was an Englishman who formed the first settlement on the Island in 1803, and the Island has remained a British possession ever since. At first, it was entirely a penal settlement, but by 1815, free settlers had commenced to arrive, and in 1853, transportation of convicts terminated. In 1856 the Island was declared a Crown Colony and the name changed to Tasmania in memory of its discoverer; while in 1901, a Federation of Australian States and Tasmania was formed as the Commonwealth of Australia.

It appears to be a significant fact that while Colonel David Collins was founding the first permanent settlement in Tasmania, in the form of a penal settlement in July, 1804, John Cunningham Saunders was preparing to circulate to the wealthy merchants in London, his appealing letter dated October 1st, 1804, which resulted in the foundation of the world's first eye hospital, namely:— "Moorfield's Eye Hospital," in March of the following year. In St. David's Park, Hobart, stand several historic monuments, on one of which is inscribed the following epitaph:—

> "To the memory of David Collins, Esq., Lieutenant Governor of the Colony, and Lieutenant Colonel of the Royal Marine Forces.
> On the first establishment of the Colony of New South Wales, he was employed as Deputy Judge Advocate, and in the year 1803, he was entrusted by His Majesty's Government with the command of an expedition destined to form a settlement at Port Phillip on the South Coast of New Holland, which was subsequently removed to Van Diemen's Land. Under his direction as Lieutenant Governor, the site of this town was chosen, and the foundation of its building laid in 1804.
> He died here on March 28th, 1810, aged fifty-six (56) years, and this monument long projected, was erected to his memory in 1838, by direction of His Excellency, Sir John Franklin, K.C.H.K.R. Site of the first Church erected in 1810 in Van Diemen's Land. Built over the grave of Lieutenant Governor Collins whose body rested beneath the Altar."

In the present hall of the Royal London Ophthalmic (Moorfields) Hospital, stands a bust with the curt inscription:—

> "John Cunningham Saunders — Founder, MDCCCIV.
> This bust erected — March 25th, 1845."

Unlike Moorfields, the progress of Tasmania was very retarded, and it was not until 1853 that the transportation of convicts to the Island was abolished subsequently, and the Island commenced its history as a Colony of the Crown. The progress of Ophthalmology in the Island was equally retarded, and it was not until the advent of the last decade that there was a surgeon in the Island who devoted his entire attention to eye work.

Unfortunately no serious attempt has been made up to the present to record local observations on eye disease, and I am able to find in the literature only one article on "Hereditary Eye Disease in Tasmania" (Hogg, 1928).

Consequently this survey has been a pioneer work and an extremely difficult one; firstly, because naturally many of the local inhabitants are very reticent about their antecedents and resent strongly any probings into the past; and secondly, because the population is so sparse and scattered. (It is well to remember here that the total population of Tasmania is only 1/30 of that of the whole of the Commonwealth of Australia); and thirdly, relatives of deceased persons reside in many parts of Australia which are beyond the reach of an ophthalmic surgeon. As a result, most of my pedigrees to date are short, except for one or two obvious exceptions, and even these exceptions have taken much tact and time to obtain.

One has only to read Usher's Bowman Lecture of 1935 to realise with what definite pains Nettleship and his disciples have striven in an attempt to correlate their findings and to present the subject of heredity from the ocular standpoint. When I first read Julia Bell's monograph, The Treasury of Human Inheritance, Vol. 11, the Nettleship Memorial Volume, I felt that the subject had been more or less exhaustively dealt with, but there is no monograph in the Nettleship Memorial Volume on congenital cataracts, ptosis, epicanthus or squint; or coming down to the bread and butter of every oculist on refractive errors. This gap should be filled as speedily as possible.

REFERENCES.

BELL, J. (1922-32), Treasury of Human Inheritance. Vol. 11, Parts I—V.
COLLINS, T. (1929), The History and Traditions of Moorfields Eye Hospital, London.
COMMONWEALTH OF AUSTRALIA (1935), Official Year Book.
GIBLIN, R. W. (1928), The Early History of Tasmania. Vol. 1.—London.
HOGG, G. H. (1928), Med. Journal of Aust. March 24th, 1928.
SAUNDERS, J. C. (1816), Diseases of the Eye — London.
STALLARD, H. B. (1936), Personal Communication.

2. STATISTICAL.

The population of Tasmania on 30th June, 1946, was 251,064, but we have to refer to the 1933 census when the population was 227,599 to find the place of nationality. The Commonwealth Bureau of Census and Statistics has supplied me with the following vital statistics in regard to Tasmania and Australia. (See Table 1.)

Except for one hundred and four Chinese gardeners and a number of half-castes from the Strait Islands of Flinders and Cape Barren, the population of Tasmania is purely Caucasian, and almost purely British stock; so that the pedigrees to follow should and do closely correspond to those more elaborate ones worked out by Nettleship in England, and Usher in Scotland.

With an area of 26,215 square miles the Island has roughly nine inhabitants to the square mile, or one to every 70 acres. The capital, Hobart, which is in the south of the Island has a population of 70,000 and a surrounding urban population of about 50,000. In the north of the Island are roughly another 130,000 inhabitants of which 34,000 live in the city of Launceston, the chief civic centre of that part of the Island.

REFERENCES.

COMMONWEALTH OF AUSTRALIA (1935). Official Year Book. Page 514.
COMMONWEALTH BUREAU OF CENSUS (1946). Personal communication and statistics.

TABLE No. I.

VITAL STATISTICS — 1933.

Birthplace	Tasmania	Australia
Australia	214,839	5,717,229
New Zealand	1,201	45,924
Other	11	777
Total Australasia	216,051	5,763,930
Other than Australasia	11,548	865,909
Total	227,599	6,629,839

Nationality (i.e. Allegiance) — 30th June, 1933.

	Tasmania	Australia
British	227,258	6,568,916
Foreign.		
Austrian	—	409
Belgian	2	206
Bulgarian	—	212
Chinese	104	7,792
Czechoslovakian	1	424
Danish	13	1,279
Dutch	4	915
Estonian	5	838
Finnish	3	1,062
French	9	1,647
German	23	3,672
Greek	9	5,652
Hungarian	5	156
Italian	80	17,658
Japanese	—	2,084
Lithuanian	1	119
Norwegian	7	1,238
Polish	2	1,757
Portuguese	—	293
Rumanian	1	112
Russian	3	2,055
Spanish	1	596
Swedish	19	1,370
Swiss	7	952
Turkish	—	33
U.S.A.	26	2,557
Yugoslavian	1	2,286
Other	6	2,345
Total Foreign	332	60,259
Not Stated	9	664
GRAND TOTAL	227,599	6,629,839

3. INHERITANCE OF EYE DISEASE.

The questions that first arouse our attention in considering the inheritance of Eye Disease are the following:—

(1) *What eye diseases may be considered hereditary?*

The answer to this is found in Table (II).

From Ruggles Gates (1929), Duke-Elder (1934), Van Duyse (1933), Francheschette (1938) and Sorsby (1940), I have been able to tabulate the following hereditary eye diseases in alphabetical order. In this list I have marked (*) against those diseases of which I have been able to obtain pedigrees in Tasmania; while (†) indicates that isolated cases of these diseases have been examined by me, but no definite hereditary evidence obtained;

(2) *Which inherited eye diseases have been found in Tasmania?*

This is revealed in Table (II) also.

We can arrive at the answer to this question quite easily by taking three cross sections of the community, viz:—

(a) In a survey of 11,006 private patients both from the point of view of inherited disease and blindness.

(b) In a survey of 195 Pension returns and inmates of the Tasmanian Institution for the Blind.

(c) In a survey of 24 pupils of the Sight Saving School.

Of course there is some slight overlapping in these three (3) sections, but to no appreciable extent and certainly not sufficient to give an erroneous impression.

(3) *What are the ocular manifestations of inherited disease in other organs of the human body?*

The answer to this will be found in Tables (III), (IV) and (V).

The association of hereditary defects of the eye, with hereditary defects in other parts of the body is a peculiarly interesting branch of the subject under discussion, and yet it appears to have received scant appreciation. Accordingly, I append three Tables of such associated defects met in the literature, and possibly other writers could add further examples of their own. More particularly does hereditary eye disease appear to be associated with corresponding defects of the nervous and skeletal systems (See Tables III and IV), but outside these categories there are a few other associated diseases, especially the Laurence-Moon-Biedl-syndrome. (See Table V.) These will be referred to again later in this paper.

TABLE No. II.

POSSIBLE HEREDITARY EYE DISEASES.

† Albinism.
Amaurotic family idiocy.
Angiomatosis of retina (Lindau's Disease).
† Aniridia.
* Anisocoria.
† Anophthalmos.
* Angioid Streaks.
Arcus juvenilis (Embryotoxon).
* Astigmatism.
Bule Sclerotics.
† Buphthalmos.
* Cataracts, congenital (including Dystrophia Myotonica).
* Cataracts, senile.
Choroideremia.
Choroiditis — Doyne's honeycomb.
† Coloboma of eyelids.
* Coloboma of iris, lens, choroid and optic nerve.
* Colour blindness.
Correctopia.
Congenital retinal folds.
† Corneal Dystrophias.
Corneal Pigmentation (Kayser-Fleischer Ring in Wilson's hepatolenticular degeneration).
Cornea plana.
Cryptophthalmos.
Cysts of Retina (Bournville's Disease).
* Dacryocystitis.
Distichiasis.
† Ectopia Lentis.
Entropion.
† Epicanthus.
Exophthalmos (Schuller Christian Disease).
Flocculi of Iris.
Glaucoma, congenital (not buphthalmos).
* Glaucoma.
† Glioma of Retina.
Gyrate atrophy of retina and choroid.
* Hypermetropia.
† Jaw-winking (Marcus Gunn Phenomena)
* Keratoconus.
† Kerato-Conjunctivitis Sicca.
Lagophthalmos.
† Macular Degeneration — Juvenile.
* Macular Degeneration — Senile
† Megalocornea.
† Melanosis Bulbi.
Microcornea.
† Microphthalmos.
* Myopia.
Neuro-fibromatosis of Retina and Iris (Von Rechlinghausen's Disease).
† Night Blindness — stationary.
* Nystagmus.
Optic Atrophy-Congenital.
* Optic Atrophy-hereditary (Leber's Disease).
Ophthalmoplegia.
* Pterygium.
* Ptosis.
* Retinal Detachment.
* Retinitis Pigmentosa.
Retinitis Pigmentosa sine pigmento.
† Retinitis punctata albescens.
* Sarcoma of Choroid.
* Strabismus.
Tapeto — retinal degeneration (Weber).

---

* represents diseases for which pedigrees have been found in Tasmania.

† indicates that isolated cases have been examined by the author but no definite hereditary evidence has been obtained.

TABLE No. III.

HEREDITARY ABNORMALITIES OF THE NERVOUS SYSTEM. ASSOCIATED WIITH EYE DEFECTS.

	NERVOUS SYSTEM	EYE
	Amaurotic family idiocy group. (Tays-Sachs disease.)	Cherry red spot at macula and optic atrophy.
	Angiomatous cysts of the cerebellum. (Lindau's disease.)	Angiomatosis of the Retina.
	Cerebral angioma. (Sturge-Weber's Disease.)	Buphthalmos or Glaucoma.
*	Dystrophia myotonica.	Cataract.
	Familial hypertrophic neuritis (Dejerine-Sottas).	Nystagmus and abnormal pupil reactions.
	Friedreich's ataxia.	Nystagmus and optic atrophy.
	Hepato-lenticular degeneration (Wilson's disease).	Kayser-Fleischer ring of cornea.
	Hereditary cerebellar ataxia.	Optic Atrophy and ophthalmoplegia.
†	Mongolism.	Keratoconus, Lens opacities, Strabismus
	Neurofibromatosis (Von Recklinghausen's disease).	Tumours of retina and iris.
†	Tuberose sclerosis. (Bourneville's disease.)	Cyst of the Retina.

* = Pedigree of this syndrome obtained in Tasmania.

† = Isolated cases of this syndrome found in Tasmania.

TABLE No. IV.

HEREDITARY DIGITAL ANOMALIES IN RELATION TO EYE DEFECTS.

	DIGITS	EYE
†	Syndactyly (Webbed digits).	Aniridia.
†	Arachnodactyly (Spider Fingers).	Ectopia lentis (Marfans syndrome).
	Polydactyly (Supernumerary digits).	Retinitis pigmentosa.
	Brachydactyly (Short digits).	Microcornea.
	Doubling of thumbs with atrophy of terminal phalanges.	Bilateral macular coloboma.

† = Cases of this syndrome found in Tasmania.

TABLE V.

MISCELLANEOUS HEREDITARY ABNORMALITIES ASSOCIATED WITH EYE DEFECTS.

	ABNORMALITIES	EYE
	Aortic Stenosis.	Ectopia lentis.
	Dysostosis Cranio-facialis (Crouzon Disease).	Exophthalmos.
†	Eighth nerve deafness.	Retinitis pigmentosa.
	Fragilitas ossium and otosclerosis.	Blue Sclerotics.
	Gargoylism (Hurler's Disease).	Corneal Infiltration.
	Haemophilia.	Colour Blindness.
	Hare Lip.	Myopia. Microphthalmos.
	Hypertelorism (Greig).	Optic Atrophy. External Strabismus.
	Hypogenitalism and obesity (Laurence-Moon-Beidl syndrome).	Retinitis Pigmentosa.
	Osseous Xanthoma (Schuller-Christian Disease).	Exophthalmos.
	Osteitis Deformans (Paget's Disease).	Central choroidal sclerosis and Optic Atrophy.
	Oxycephaly-proper.	Optic atrophy.
	Progressive Facial Hemiatrophy (Romberg syndrome).	Keratitis.
*	Pseudo-Xanthoma Elasticum (Darier's Disease).	Angiod streaks of Retina.

* = Pedigree of this syndrome obtained in Tasmania.
† = Cases of this syndrome are found in Tasmania.

REFERENCES.

BELL, J. (1933), Trans. Ophthal. Soc. U.K., Vol. LIII, Page 42.
BELL, J. (1922-32), Treasury of Human Inheritance. Vol. II. Parts 1-5.
BERLINER, M. L. and GARTNER, S. (1940), Arch. of Ophthal. Vol. 24. No. 4, page 691.
BEST, H. (1934), Blindness and the Blind in U.S.A., N. York
BIGGS, H. H. (1919), Amer. Jl. Ophthal. Vol. XI. Page 408.
BLACKER, C. P. (1934), The Chances of Morbid Inheritance, London.
CLAUSEN, H. (1923), Zentralb. fur die gesamte Ophthal. und ihre grenzgebiete. Vol. XI, 6. Page 209.
CLAUSEN, H. (1923), Zentralb. fur die gesamte Ophthal. und irhe grenzgebiete. Vol. XI, 10. Page 417.
CLAUSEN, H. (1923), Zentralb. fur die gesamte Ophthal. und ihre grenzgebiete. Vol. XI., 11. Page 481.

COOPER, E. L. (1936), Brit. Med. Journ. April 18th. Page 793.
CURDES, F. C. and HOGAN, M. J. (1942), Arch. of Ophthal. Vol. 27. No. 4. Page 637.
CREW, F. A. (1926), Brit. Med. Jl. August 14th. Page 225.
DUKE-ELDER, W. S. (1938), Text Bk. of Ophthalmology. Vol. II. London.
DOGGART, J. H. (1937), Brit. Encyclopaedia of Med. Practice. Page 229.
FRANCESCHETTI, A. (1938), Ophthalmologica. Vol. 99. No. 3 and 4.
GATES, R. G. (1929), Heredity in Man, London.
GRONBLAD, E. (1940), Modern Trends in Ophthalmology. Page 90.
GUBB, A. (1938), Erbleinden des Auges — Leipzig.
HUDSON, A. C. (1932), Proc. Roy. Soc. Med. Vol. XXVI. Page 35.
KING, E. F. (1934), Proc. Roy. Soc. Med. Vol. XXVII. Page 298.
KISCHK, K. (1937), Arch. Fuer. Augenheilkunde. Vol. 110. Chap. XXV. Page 357.
LAWES, F. A. and HALLIDAY, J. C. (1944), Med. Journal of Aust., May 20th. Page 465.
MANN, I. C. (1933), Trans. Ophthal. Soc. U.K. Vol. LIII. Page 47.
MOORE, E. (1934), Heredity — Mainly Human, London.
MOORE R, FOSTER (1925), Medical Ophthalmology, London.
PUNNETT, R. C. (1933), Trans. Ophthal. Soc. U.K. Vol. LIII. Page 9.
RADOS, A. (1942), Arch. of Ophthal. Vol. 27. No. 3. Page 477.
SEAR, H. R. and MADDOX, J. K. (1945), Med. Journal of Aust. May 12th. Page 488.
SORSBY, A. (1934), Proc. Roy. Soc. Med. Vol. XXVII. Page 694.
SORSBY, A. (1940), Modern Trends in Ophthalmology. Page 139. London.
THANNHAUSER, C. J. (1940), Lipoidosis — New York.
TIDY, H. L. (1949), Synopsis of Medicine. London.
VAN DE HOEVE, J. (1932), Trans. Ophthal. Soc. U.K. Vol. LII. Page 380.
WHISHAW, R. (1936), Personal Communication.
WILSON, S. A. (1934), Proc. Roy. Soc. Med. Vol. XXVII. Page 297.
WILSON, S. A. (1940), Neurology. Vol. II. Page 806. London.

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4. INHERITED EYE DISEASE AND INHERITED BLINDNESS IN TASMANIA.

As previously stated, I will attempt to determine the incidence of inherited eye disease and inherited blindness in Tasmania from three sources.

A. PRIVATE PATIENTS:—

From our 1947 analysis of 11,006 private cases, classified under 240 separate diseases, which are tabulated in Table VI, I have marked all possibly inherited diseases with a dagger (†), and those inherited diseases for which I am able to obtain pedigrees in Tasmania with the usual asterisk (*). I do not for one moment wish to suggest that, for example, in chronic Dacryocystitis, all the 135 cases were inherited; any more than all the 59 cases of Ptosis were inherited, but I do suggest that these are inheritable diseases, and I attempt to show that in some of them we have obtained a pedigree or found a history of familial incidence, and that pedigrees of these conditions have been reported in other countries.

I present this table with the hope of being able to roughly assess the proportion of inherited eye disease to be found in the Australian community.

In Table VII is indicated the 148 blind found in the analysis of 11,006 cases examined, and you will there see that we have analysed all the blindness under the

scheme recommended by the Union and Countries Association for the Blind. Again, it is obvious that the whole of the 81 cases, under the heading of:— "Congenital, Hereditary, and Developmental" have not a definite history of heredity and therefore in Table VIII it will be found that 34 of these cases are included in 19 pedigrees examined by me, and further that 59 out of these 81 cases may reasonably be supposed to have been inherited. There were many other cases of blindness (in the 19 pedigrees) who have been examined by other Ophthalmologists, or whose diagnosis of blindness could easily be made from the history of the propositus.

B. BLIND PENSIONS:—

The definition of blindness throughout the world varies greatly, but I will refer briefly here to the definition employed by the Government of the Commonwealth of Australia. It is— "Loss or diminution of vision to an extent which renders the sufferer unfit for work other than that usually performed by blind workers."

With this definition as a guiding principle certification for blind pensions is made throughout Australia, and the surveys made by W. D. Counsell and myself (1937, 1939, 1940 and 1941) into the causation of blindness in Tasmania, were carried out on the principle of this definition. A second difficulty in estimating the causation of blindness, is the lack of an international classification of causes. We found when making our review that one disease might be put under several headings. A third difficulty was the incomplete data which was due to two factors:—

(a) Certification by incompetent referees.

(b) Vague and incomplete certification forms.

A fourth difficulty was that many of the aged blind received old-age pensions, and not blind pensions, and fifthly, the personal factor:— i.e. that competent referees vary in their opinion of causation.

The next cross section which I have taken to define inheritable blindness in Tasmania, is a modified table of the work of Counsell and myself in 1939, and published in the Australian Medical Journal in that year. Here in Table IX we analysed 195 cases of blindness, of which 62 belonged to hereditary diseases, and these inherited diseases have been re-analysed in Table X for clarity. You will see there are 21 cases of Congenital Cataracts, 2 Detachment of the Retina, 11 of Primary Glaucoma, 1 of Glioma, 11 of Myopia, 1 of Nystagmus, 10 of Leber's Optic Atrophy and 5 of Retinitis Pigmentosa. The inheritability of Primary Glaucoma may be questioned by some, but I have no hesitation in saying that in Tasmania (as I will show later in this paper) I have been able to obtain 9 pedigrees of Primary Glaucoma in the North and South of the Island.

C. THE SIGHT SAVING SCHOOL:—

The last cross section is but a scanty contribution of the subject under review, and yet it analyses the disease of 24 pupils attending the Sight Saving School in Hobart, in 1940. It will be seen here, except for the one case of Lamellar Cataracts, that all the children are suffering from inherited or congenital blindness, and I think it is encouraging to notice here, that there is not one case suffering from the results of Ophthalmia Neonatorum, Smallpox, or Measles, in fact, not one case of blindness from bacterial disease. There is no case of blindness from Trauma,

and only one from what might be described as general disease, namely, Lamellar Cataract. This indicates that, from the public health point of view, the eyesight of all children in Tasmania is particularly good.

It may be asked here why no reference has been made to the school at the Tasmanian Institution for the Blind and Deaf. It must be pointed out that at the end of 1939 the school was closed as only two pupils remained, both being over the age of 14 years and both suffering from congenital cataracts.

Recently the Blind class at the Tasmania Institute for the Blind and Deaf was re-opened and is attended by three children, two suffering with Corneal Opacities and one with Congenital Cataracts.

Before closing this subject of hereditary blindness, it might be permissable to return to Table X and note there that out of 195 cases, Counsell and I considered that 83 (that is 62 inherited blind, and 21 others) could have been prevented by one means or another and this would mean 43%. But I have already pointed out in my paper on the "Prevention of Blindness in Tasmania" (1940) that at least 40% of the prevailing blindness is cured by practising Oculists in Tasmania, so if a further 43% of the remaining 60% are curable it gives a possible "Prevention or Cure" figure of 66% which coincides with Best's figure of 1934, which is that at least 70% of blindness is either curable or preventable in the United States. This is a very encouraging prospect. His actual quotation is as follows:—

"Thus we find what seems to be a fair estimate, 72%, or not far off 3/4 of Blindness in the United States to be of a preventable character."

The highest possible figure in Tasmania is about 66%.

TABLE No. VI.

EYE DISEASES FOUND IN ANALYSIS OF 11,006 CASES IN TASMANIA, 1931 - 1947.

	Accommodation, paralysis of	37
†	Albinism	6
	Amblyopia, congenital	42
*	„ ex anopsia	380
	„ functional	27
	Anaesthesia of orbital nerves supra and infra	3
*	Angioid Streaks	4
	Angioma, various types	16
	Angioneurotic oedema	3
*	Aniscoria	226
†	Anisometropia	154
	Anopia, quadrant & hemi	16
†	Anophthalmos	117
	Aphakia	200
	Argyll Robertson pupil	8
	Argyrosis	11
	Atropine irritation	20
	Birth Injuries	53
	Blepharitis, simple	319
	„ ulcerative	9
	Brow, Abscess of	24
	Canaliculus, laceration of	6
	Cataract, anterior polar	6
	„ Blue dot	17
	„ complicated	78
*	„ congenital	118
	„ diabetic	52
	Cataract, glass blower	1
	„ Lamellar	8
*	„ Senile	1,050
	„ Traumatic	75
	„ X-ray	1
	Chalazion	175
	Choroid, detachment of	1
	„ rupture of	8
*	„ sarcoma of	11
†	„ stretching of	62
*	Colobomata, of choroid	11
	„ of disc	5
*	„ of iris	6
	„ of macula	2

	Choroiditis, acute	39
	„ old	176
	„ Tay's	172
*	Colour Blindness	36
	Conjunctiva, cyst of	11
	Conjunctival abrasion	5
	„ burn	17
	„ foreign bodies	71
	„ laceration	25
	„ scar	1
	Conjunctivitis, acute (organism unknown)	148
	Conjunctivitis, staphylococcal streptococcal	7
	Conjunctivitis, pneumococcal	9
	„ Koch-Weeks	9
	„ allergic	7
	Conjunctivitis, ophthalmia neonatorum	15
	Conjunctivitis, angular	50
	„ chronic	381
	Convergence Excess	112
	„ Insufficiency	516
	Corneal abrasion	137
	„ „ recurrent	5
	„ burn	25
	„ foreign bodies	193
	„ laceration	20
	„ nebula	347
	„ staphyloma	5
	„ ulcer (see dendritic)	209
	Cyclitis	27
	Cyclophoria	19
	Dacryocystitis, acute	23
*	„ chronic	112
	Dendritic ulcer	23
	Dermoid cyst of brow	2
	„ „ „ „ lid	36
	„ „ „ „ limbus	4
	„ „ „ „ orbit	2
	Disc optic, cyst of	2
	Divergence insufficiency	141
	Eale's disease	9
	Ectropion	69
	Electric Ophthalmia	3
	Embolism of central artery	4
	Endophthalmitis	3
	Enophthalmos	6
	Entropion	19
	Epicanthus	30
	Episcleritis	15
*	Esophoria	330
*	Esotropia	326
	Eserine irritation	13
*	Exophoria	636
	Exophthalmos	40
*	Exotropia	161
	Glaucoma, acute	20
*	„ chronic	125
	„ secondary	65
†	Glioma retinae	2
	Globe, contusion of	47
	„ perforation of	77
	Haemorrhage, retinal	80
	„ subconjunctival	92
	„ subhyaloid	3
	Herpes ophthalmicus	23
	Heterochromia iridis	12
	Horner's syndrome	6
	Hordeolum	265
	Hyaloid, persistent	14
*	Hypermetropia	421
*	Hypermetropic astigmatism	3,050
†	Hyperphoria	170
	Hyphaema	40
	Hypopyon	15
	Intracranial tumours	20
	Intraocular foreign bodies	23
	Iris, atrophy of	6
	„ bombé	14
	„ sphincter rupture	8
	„ perforation of	6
	„ prolapse of	17
	Iridocyclitis	77
	Iridodialysis	10
	Iritis, acute	70
	„ diabetic	5
	„ old	39
	Keratitis	61
	„ acne rosacea	11
	„ bullous	4
	„ disciform	4
	„ interstitial	37
	„ E. lagophthalmos	6
	„ marginal	80
	„ mustard gas	2
	„ neuro-paralytic	13
	„ profunda	12
	„ sicca	20
	„ striate	15
	„ superficial	21
	„ „ punctate	95
	„ vesicular	12
	Kerato-cyclitis	3
	„ globulous	1
*	Keratoconus	55
	Lachrymal fistula	3
	Lashes itinerant	14
†	Lens, dislocated	24
	Leucoma	17
	Lid, abscess of	3
	„ abrasion of	6
	„ acne rosacea of	1
	„ angioma of	2
	„ concretions	40
	„ contusions of	16
	„ coloboma of	5
	„ cysts of	5
	„ dermatitis of	82

	„ foreign bodies	10
	„ haematoma	1
	„ Herpes of	7
	„ hydroma	1
	„ laceration of	19
	„ naevus of	1
	„ papilloma of	39
	„ retention cysts	20
	„ retraction of	38
	„ wart of	16
	Macula, massive haemorrhage	2
	„ hole at	5
*	Megalocornea	10
†	Melanoma, of iris	10
†	„ of retina	61
†	Microcornea	5
†	Microphthalmia	11
	Migraine	55
	Miosis	7
*	Mixed astigmatism	1,064
	Mydriasis	26
*	Myopia	182
*	Myopic astigmatism	1,611
	Myotonic pupil	10
	Myxoedema	4
	Naevus, pigmented, various	27
	Nasal accessory sinus infection	24
	Nothing abnormal diagnosed	215
*	Nystagmus	106
*	Obstruction, nasolachrymal	73
	„ punctum and canaliculus	51
	Occlusion, post-cerebral artery	11
	Oedema of lid, insect bite	10
	Opaque nerve fibres	21
	Ophthalmia, sympathetic	5
*	Optic atrophy, Leber's	32
	„ „ primary	31
	„ „ secondary	17
	Orbital cellulitis	5
	„ foreign bodies	2
	Overaction of inferior oblique	36
	Panophthalmitis	5
	Papillitis	30
	Papilloedema	9
	Paralysis, external rectus	27
	„ facial or orbicularis	32
	„ inferior oblique	6
	„ „ rectus	1
	„ internal „	7
	„ superior „	25
	„ „ oblique	7
	„ third nerve	14
	„ 3rd and 4th nerves	7
	„ 3rd and 6th nerves	12
	Phlycten	54
	Phthisis bulbi	14
	Pinguecula	25
	Presbyopia	3,250
	Pseudoneuritis	15
*	Pterygium	160
*	Ptosis	59
	Pupillary membrane, persistent	21
	Retinae commotio	8
	Retinal arterio sclerosis	304
*	„ degeneration, senile	108
*	„ detachment	90
	Retinitis	48
	„ active	30
	„ acute	32
	„ albuminuric	13
	„ arterio sclerotic	54
	„ circinata	2
	„ diabetic	33
	„ old	123
*	„ pigmentosa	17
	„ proliferans	20
	„ senile massive exudate	27
	Retrobulbar neuritis	15
	Scleritis	23
	Siderosis bulbi	4
	Socket, contracted	2
	Stenosis, puncta and canaliculi	5
	Symblehparon	6
	Synchisis scintillans	15
	Synechiae, anterior	39
	Thrombosis of retinal vein	43
	Thyreotoxicosis	40
	Trachoma	16
	Trichiasis	95
	Ulcer rodent	11
	Uveitis	14
	Vitreous haemorrhage	46
	„ opacities	227
	„ prolapse	2
	Xanthelasma	8

† = Possible inherited diseases.

* = Tasmanian Pedigrees obtained.

TABLE No. VII.

THE CAUSES OF BINOCULAR BLINDNESS IN TASMANIA.

Private Practice

Total Cases Examined: 11,006
Total Blind: 148

Group	No. of cases
CONGENITAL, HEREDITARY AND DEVELOPMENTAL:—	**81**
1. Albinism	1
2. Amblyopia Congenital with colour blindness	2
3. Amblyopia ex Anopsia	—
4. Cataracts Congenital	11
5. Cataracts Congenital with Dislocated Lens	4
6. Cataracts Senile	15
7. Glaucoma	21
8. Keratoglobus	1
9. Macular Degeneration with Angeoid Streaks	1
10. Myopia without Detachment	3
11. Nystagmus	1
12. Optic Atrophy.	2
13. Optic Atrophy, Leber's	6
14. Refractive Errors other than Myopia	—
15. Retinal Detachment — Hereditary	3
16. Retinal Detachment — Myopic	2
17. Retinitis Pigmentosa	10
18. Retinoblastoma	1
19. Sarcoma of the Uvea	—
INFECTIOUS AND BACTERIAL	**23**
1. Ophthalmia — Neonatorum	3
2. Gonorrhea	1
3. Syphilis	3
4. Trachoma	3
5. Local Infection of the Eye Coats—(includes Keratitis)	2
6. Smallpox	—
7. Meningitis	—
8. Tuberculosis	—
9. Phylctenular Keratitis	—
10. Acute Septicaemia	—
11. Focal Sepsis (includes Iridocyclitis)	7
12. Other Infections	—
13. Optic Atrophy, Cause unknown	3
14. Leprosy	—
15. Other Lesions. Cause unknown	1
16. Whooping Cough	2
TRAUMATIC AND CHEMICAL	**13**
1. Industrial	
(a) Trauma	2
(b) Disease	—
2. Non-Industrial	
(a) Trauma	6
(b) Systemic Poisoning	—
(c) Birth Trauma	2
(d) War Trauma	—
3. Sympathetic Ophthalmia	3
GENERAL DISEASES	**31**
1. Anaemia	1
2. Vascular Diseases	11
3. Disease of the Central Nervous System	1
4. Diseases connected with Childbirth	1
5. Tumours	
(a) of the Brain	3
(b) of the Orbit	—
6. Diabetes	8
7. Nephritis	1
8. Other General Diseases	—
9. Senile Degeneration	5
10. Deficiency Diseases (Mongolism)	1

TABLE No. VIII.

HEREDITARY CAUSE OF BLINDNESS IN TASMANIA.
PEDIGREE CASES.

Disease.	Total Number of Cases.	Total Pedigree Cases.	No. of Pedigrees Concerned.
1. Albinism	1	1	1
2. Amblyopia congenital with Colour Blindness	2	2	1
3. Cataracts Congenital	11	7	3
4. Cataracts Congenital with Dislocated Lens	4	4	1
5. Glaucoma	21	6	5
6. Macular Degeneration with Angioid Streaks	1	1	1
7. Optic Atrophy, Leber's	6	5	2
8. Retinal Detachment, hereditary	3	3	1
9. Retinitis Pigmentosa	10	5	4
TOTAL	59	34	19

(See Table VII)

TABLE IX

BLINDNESS IN TASMANIA

GLOSSARY: (p)—Preventable. (h)—Hereditary. (i)—Injury. (v)—Venereal Disease.

		1. Receiving Pensions ..	(a) Blind—Reliable Clinical Information	(b) Blind—Unreliable	(b) Not now blind—Reliable Clinical Information ..	2. Not Receiving Pensions	(a) Blind—Reliable Clinical Information	(b) Blind—Unreliable Information ..	3. Totals 1a and 2a. Blind—Reliable Clinical Information	4. Totals—1b and 2b. Blind—Unreliable Clinical Information ..	5. GRAND TOTALS—Totals—1a and 2a, 1b and 2b .. ..
Anophthalmos	? Cause	2	2						2		2
Cataract-Cong	(h-p)	16	15		1	6	4	2	19	2	21
Cataract-Sen	(p)	3	—	3		4	2	2	2	5	7
Cataract-Traum.	(1)	1	1						1		1
Choroiditis	? Cause	2	2						2		2
Detach. Ret. (Hered)	(h-p)	1	1			1	1		2		2
Detach. Ret. (Idiop)		1	1						1		1
Eales' Disease		1	1						1		1
Glaucoma. Primary	(p-h)	6	5	1		5	5		10	1	11
Glioma	(h)	—	—			1	1		1		1
Injury	(i)	13	7	6		4		4	7	10	17
Injury Optic Atrophy	(i)	1	1			1	1		2		2
Injury Phthisis Bulbi	(i)	2	2						2		3
Injury Symp. Ophth.	(p-i)	5	5						5		5
Interstitial Keratitis	(p-v)	3	3						3		3
Iriodocyclitis	? Cause	10	9	1		4	4		13	1	14
Leucoma	? Cause	2	—	2						2	2
Myopia	(h)	9	4	5		2	2		6	5	11
Neuroretinitis	? Cause	2	—	1		1	1		1	1	2
Nystagmus Cong.	(p-h)	1	1		1				1		1
Ophthalmia Neonatorum	(p-v)	12	7	5					7	5	12
Optic Atrophy	? Cause	8	2	6		3	1	2	3	8	11
Optic Atrophy Lebers	(p-h)	9	8	1		1	1		9	1	10
Optic Post Neuritic		3	2	1					2	1	3
Optic Atrophy-Tabetic	(p-v)	2	2			2	2		4		4
Whooping Cough		1	1						1		1
Retinitis Pigmentosa	(p-h)	5	4	1					4	1	5
Thrombosis Central Vein		0	0	20		2	2		2		2
Trachoma	(p)	2	1	1					1	1	2
Unclassified		18		18		19		19			37
Totals		141	87	52	2	56	27	29	114	81	195
Preventible	(p)	65	52	12	1	19	15	4	67	16	83
Hereditary	(h)	47	38	8	1	16	14	2	52	10	62
Injury	(i)	22	16	6		5	1	4	17	10	27
Venereal Disease	(v)	17	12	5		2	2	0	14	5	19

TABLE No. X.

CAUSES OF HEREDITARY BLINDNESS IN TASMANIA IN 1938. (Hamilton and Counsell), Pensions List.

TOTAL PENSIONERS — 195.

Disease	Cataract Congenital	Detachment of Retina	Glaucoma Primary	Glioma	Myopia	Nystagmus	Leber's Optic Atrophy	Retinitis Pigmentosa
1. RECEIVING PENSIONS:	16	1	6	0	9	1	9	5
(a) Reliable Clinical Information	15	1	5	0	4	1	8	4
(b) Unreliable Clinical Information	0	0	1	0	5	0	1	1
(c) Not now Blind	1	0	0	0	0	0	0	0
2. NOT RECEIVING PENSIONS:	6	1	5	1	2	0	1	0
(a) Reliable Clinical Information	4	1	5	1	2	0	1	0
(b) Unreliable Clinical Information	2	0	0	0	0	0	0	0
3. Total:— 1a and 2a.	19	2	10	1	6	1	9	4
4. Total:— 1b and 2b.	2	0	1	0	5	0	1	1
5. GRAND TOTAL:—	21	2	11	1	11	1	10	5

TABLE No. XI.

DISEASES OF 24 PUPILS AT HOBART SIGHT SAVING SCHOOL, 1940.

DISEASES	NO. OF CASES OF EACH DISEASE
Cataract — Anterior Cortical	1
Cataract — Congenital	2
Cataracts — Lamellar	1
Hypermetropic Astigmatism	5
Lenticonus — Posterior	1
Mixed Astigmatism	2
Myopia	3
Myopic Astigmatism	7
Retinal Detachment — Inherited	1
Retinitis Pigmentosa	1

REFERENCES.

BEST, H. (1934), Blindness and the Blind in U.S.A. 2nd Edition, New York.

HAMILTON, J. B. and COUNSELL, W. D. (1937), The Cause and Prevention of Blindness in Tasmania. A Preliminary Survey. Report of the National Health and Medical Research Council. Appendix 7.

HAMILTON, J. B. and COUNSELL, W. D. (1939), The Cause and Prevention of Blindness in Tasmania. A Supplementary Report. Aust. Med. Journ. March, 1939.

HAMILTON, J. B. (1940), Blindness in Tasmania. Trans. Ophthal. Soc. of Aust. Vol. 2. Page 37.

HAMILTON, J. B. (1941), Tasmanian Blindness. Trans. Ophthal. Soc. of Aust. Vol. 3. Page 60.

LEAGUE OF NATIONS (1929), Welfare of the Blind.

MACCALLAN, A. F. (1935), Brit. Jl. Ophthal. Vol. XIX. Page 338.

MARQUES, Prof. (1933), XIV Concillium Ophthal. Hispania. T.111 2-3. Page 106.

PREVENTION OF BLINDNESS COMMITTEE OF THE UNIONS OF COUNTIES ASSOCIATION FOR THE BLIND (1933), Report on Hereditary Blindness, London.

5. SOCIOLOGICAL PROBLEMS.

The Tasmanian Institute for the Blind and Deaf, has the following objectives in regard to the blind and deaf in the Island of Tasmania:—

1. To promote the education, industrial training, employment and general advancement in the life of the blind and deaf and their social economics and spiritual welfare.
2. To undertake measures for the saving of sight or hearing, and the correction of defects of speech or hearing.

3. To provide buildings, premises, equipment, materials and the facilities for any such purpose as aforesaid.
4. To co-operate with any person or organisation having any similar aims for any of the purposed aforesaid.

The Institution is administered by a Board of Management on which sit representatives of the State Government, the Braille Writer's Association, the Hobart Women's Auxiliary Committee and Subscribers. It has entire control of all sociological problems relating to the blind, and the prevention of blindness, but the education of the blind and partially sighted is in the hands of the Tasmanian Government Department of Education.

The sociological problems relating to the sufferers from hereditary eye diseases cover an even wider field, and include the normal sighted, the partially sighted and the blind. I propose dealing with these problems under the following headings:—

1. Education
2. Employment
3. Welfare
4. Prevention

and to pay special attention to the work undertaken in Tasmania in these directions.

6. EDUCATION.

Children with hereditary eye defects naturally fall into five classes as far as educational facilities are concerned.

1. Normal sighted — non progressive to 6/12 vision.
2. Normal sighted with progressive lesions — e.g. Myopia.
3. Partially sighted — non progressive lesions — e.g. Nystagmus. 6/18 to 6/36 vision.
4. Partially sighted — progressive lesion — e.g. Retinitis pigmentosa.
5. Blind — 6/60 or less vision.

The education of all the above five groups may be divided into three epochs:—

(a) Primary education in institutions or special schools.

(b) Higher education.

(c) Home tuition.

TYPES OF DEFECTIVE VISION:—

1. Normal Sighted. The main duty of the ophthalmic surgeon is to ascertain that the condition is non-progressive, and the Report of the Committee of Enquiry into the Problems Relating to Partially Sighted Children (Crawley, 1934), stresses in more than one of its pages, the necessity for the half-yearly examination of the partially sighted, and I feel this provision should be extended to the normal sighted known to have inherited eye defects, for the above reason at least. These normal sighted children can be educated along ordinary lines so long as their condition is non-progressive, but should they show signs of deterioration, then the ophthalmic surgeon in charge of the case or school, must recommend the transfer to Group (2). On the other hand, hypermetropes, after adequate correction and continuous use of their glasses, may show a marked improvement in visual acuity, which necessitates the surgeon transferring these cases from Group (2) back to Group (1).

2. *Normal Sighted with progressive lesions.* In this group, which chiefly includes the myope, the opinion of an ophthalmic surgeon of experience is absolutely essential in deciding the merits of each case. The "Crawley Report on Partially Sighted Children" lays down on Page (23) criteria for the special education of myopes. At the same time, on Pages (32) and (33), the Report stresses the fact that once the myopia has ceased to increase more than ½ dioptre per annum, and provided the vision and fundus changes are satisfactory, such children should be redrafted to ordinary schools, (Group 1.). On the other hand, if the malady is rapidly progressive, the child may be forced to enter a special school in order to complete its tuition. On Page (35), the necessity for a bi-annual examination by ophthalmic surgeons of all scholars of partially sighted classes is stressed.

3. *Partially Sighted—non-progressive.* Group (3) are stationary pupils in the partially sighted class, and their education should run along uninterrupted lines.

4. *Partially Sighted — progressive.* Group (4) is a difficult one, and just when they should be transferred from the partially sighted class to the Blind Institution must again be decided by the ophthalmic surgeon at his regular periodic examinations. The "Crawley Report" (1934) on Page (25) recommends the education of these patients in "partially sighted" schools as long as practicable. It also devotes the whole of Chapter V to weighing the pros and cons of segregation versus non-segregation of the partially sighted, and after a full survey of the merits of each, concludes the discussion in favour of the partially sighted not in schools for such, but in special classes in ordinary schools. The final paragraph of the chapter is worthy of quotation:—

"After balancing the advantages of the segregation and non-segregation systems, and recognising fully the difficulties which must be overcome, and the prejudices which must be broken down, the Committee recommends that the education of partially sighted children should be conducted where possible in special classes attached to, and forming an integral part of, the ordinary school. This decision has only been arrived at after studying this system in America, where it is used extensively, and at Liverpool where it has been found most satisfactory."

Myer (1930), in his survey of Sight Saving classes in the public schools of the United States of America, details some of the standards of admission to such classes in America. Variation in the different States is enormous, and the National Society for the Prevention of Blindness has given the following guide for finding potential sight saving class (not school) pupils, but they stress emphatically that all cases must be considered individually.

(1) General Statement, children having a visual acuity of 20/70 (Snellen 6/12) or less in the better eye after proper refraction. In addition the following are recommended as potential candidates:—

   (a) Children in elementary schools having four or more dioptres of Myopia.

   (b) Inactive, subsiding (or progressive) cases, such as interstitial or phlyctenular Keratitis, optic neuritis, trachoma, etc., in which some irritation may be present provided the approval of the attending physician is given.

(2) All cases must be considered individually.

(3) Any child who, in the opinion of the ophthalmologist, would benefit by assignment to a sight saving class, subject to suggestion for treatment and training from such ophthalmologist, and the acceptance of the educational authorities having charge of such classes. (In this category come children with amblyopia ex anopsia undergoing occlusion of the better eye.)

(4) It is assumed that all children assigned to sight saving classes have average normal mentality.

5. *Blind.* Although Group (5) appears straight-forward, complaints have arisen in the past, due to failure of the necessary authorities to have all candidates for Blind Schools examined by an ophthalmic surgeon before admission. The "Crawley Report" (1934), describes the education of the partially sighted in the blind institution as "indefensible" — not only does such an education stigmatise the partially sighted, but also it is quite inadequate for their needs in after-school life.

EPOCHS OF EDUCATION:—

(a) Primary Education in Institutions—Schools for blind children have been instituted in practically every country, but in some they have been incorporated with classes for the deaf also. This, to some extent, is unfortunate, as each defect has entirely different problems, and Best (1934), points out that, as in many institutions the blind are in the minority, their education is subordinate to that of the deaf. But for financial reasons in smaller states and countries this combination is unavoidable though not ideal. It must be remembered that Best (1934), states that it costs eight times as much to educate each blind child in a residential institution as to teach each sighted child in a day school. For transport reasons most blind schools are residential, but the ideal methods of tuition are day schools, which can only function satisfactorily in larger cities. Further, schools for the blind should, as far as possible, be treated as educational rather than charitable institutions, for, to stigmatise the education of the blind as charity is not in accord with the trend of modern thought. Education nevertheless, should be compulsory for at least 11 years, i.e. from 5-16 years of age; and if higher education is desirable and desired, it can be undertaken on a voluntary basis. Schools for the partially sighted, on the other hand, should be built in close proximity to large primary schools so that the visually defective child may have constant playtime contact with the normally sighted.

(b) Higher Education.—This group contains those who, through special adaptability, qualify for higher education. This can, at first, be undertaken at High schools, and later at a university. The number of blind, able to pass the necessary examination even with the aid of a sighted coach and Braille writer, is extremely limited.

(c) Home Tuition.—This group is usually carried out by the Welfare (or field) Officers, and is almost totally confined to adults whose blindness has supervened after school life and who wish to learn Braille.

LOCAL CONDITIONS.

In the Commonwealth of Australia there are six States, and in each State capital, including Hobart, a blind school is situated, primarily for the education of blind children, but also catering for a limited number of partially sighted. In no part of the Commonwealth has any attempt to organise separate schools for the partially sighted or myopes been made except Tasmania, Victoria and New South

Wales. This has been verified by letters addressed to each blind school within the Commonwealth.

The Sight Saving School in Hobart, which was opened in March, 1940, is now filled to capacity. The success of the School is most encouraging, and parents and children in Hobart are delighted with it. Our thanks should be extended to the former Director of Education in Tasmania, Mr. G. V. Brooks, C.B.E., to whose foresight is due the opening of the first Sight Saving School in Australia.

The planning of the Sight Saving School in Hobart was done by Mr. V. A. Coronel, A.R.A.I.A., A.R.V.I.A., of the Architectural Branch of the Tasmanian Public Works Department. When an extension of the school is required, and it is now obviously necessary, another classroom can be built on to the right hand side of the present one allowing for equivalent adequate lighting and wall space. Behind the school building, at some 30 ft. distance, is a large enclosed sun-room, where the children can congregate on suitable days for oral lessons and dramatisation and also to partake of refreshments. To the right of the Sight Saving School is the main Elizabeth Street State School, which is the largest of its kind in Hobart, and this proximity allows for playtime and out-of-school contact between both types of pupils. A feature of the playground of the new Sight Saving School is the elimination of obstacles and this has been carried out by levelling and paving with concrete blocks. Certain areas have been left unpaved so as to encourage horticultural pursuits in those children whose condition allows for such activity.

Special attention has been paid to the classroom lighting. The aspect and construction has allowed for a maximum of natural illumination, but when this fails below 15 foot candles a photostatic cell automatically switches on the row of indirect electric lights nearest to the main windows. A second row of indirect lights furthest from the windows are left constantly burning. Further, the hyloplate consists of 3 movable boards which are also directly illuminated from inset lights in the ceiling and it is interesting to see how successful primrose boards with blue chalks have proved. This innovation arose in England and is predicted to increase the teaching efficiency at least 10% and also conveys a remarkably cheerful atmosphere in the classroom.

The residential difficulties of country pupils attending the Sight Saving School have been overcome by the Tasmanian Institute for The Blind and Deaf, accommodating these partially sighted pupils during term and providing for their escort to and from the school.

It might be mentioned here that the routine examination of the eyesight of the school children of Tasmania is at present carried out by the school nurses, and the survey mentioned above proves conclusively that many children with poor eyesight attending school have never received attention from an oculist or an optician. Both the lack of appropriate treatment and the lack of expert medical supervision is to be regretted greatly.

In 1906, compulsory schooling for the blind and deaf in Tasmania was introduced by legislation, and 7-16 years defined as the appropriate span of tuition of those so affected. In 1938, the span of compulsory education was extended to include the years of 5-16 and this can only result in better educational results.

Higher education is also in force in Tasmania. One boy aged 15 years is at present attending the State High School, while another young man, having matricu-

lated, has finished his arts course at the University of Tasmania, and is now teaching sighted children for the Education Department of Tasmania. The High School education is free, and the University of Tasmania waived the collection of fees in the individual case mentioned.

Home tuition, which is carried out extensively by the Welfare Officers in Hobart, Launceston and Latrobe and by adults who have been blind from childhood, is proving most satisfactory.

REFERENCES.

AUSTRALIAN BLIND INSTITUTIONS (1945), Personal Communications.

BARRETT, J. (1938), Med. Journ. Aust. Aug. 13th. Page 242.

BEST, H. (1934), Blindness and the Blind in U.S.A. 2nd Ed. New York.

CRAWLEY, R. H. (1934), Report of the Committee of Enquiry into Problems Relating to Partially Sighted Children.

FLANAGAN, C. A. (1934), Sight Saving Review. Vol. IV. No. 3.

HAMILTON, J. B. (1940), Trans. Ophth. Soc. Aust. Vol. II. Page 43.

HATHAWAY, W. (1937), Sight Saving Review. Vol. VII. No. 2., June.

LEAGUE OF NATIONS (1929), Report on the Welfare of the Blind.

MYER, E. T. (1930), A Survey of the Sight Saving Classes in the Public Schools of U.S.A., New York.

NATIONAL INSTITUTE FOR THE BLIND (1932), A Handbook on the Deaf Blind. Bulletin No. 4, London.

RODIN, F. H. (1929), Calif. & West. Med. Vol. XXX.

TASMANIAN INSTITUTION FOR THE BLIND (1945), Personal Communication.

TASMANIAN INSTITUTION FOR THE BLIND (1945), Annual Report.

7. EMPLOYMENT.

However disabling most hereditary eye diseases may be, those which cause blindness are paramount in importance, for it is common knowledge that guided by our special senses, our existence depends say 4/5 on eye sight, and 1/5 on the other senses. I have therefore no doubt in writing that the blind are at least 80% handicapped, and when Best (1934), assures us that only 7% of the blind in the United States of America are self supporting, and the League of Nations (1929) reports that 65% of the blind are unemployable, I feel this figure of 80% may be too low. To my mind the greatest significance of heredity in ophthalmology lies in the question:— Does or does not each hereditary disease cause blindness?

Persons suffering from hereditary eye diseases should be classified as regards employment, under four headings:—

1. Normal Sighted.
2. Myopes.
3. Partially Sighted.
4. Blind.

and will be dealt with in this order, with special references to conditions pertaining thereto in Tasmania.

1. Normal Sighted. Except in rare instances (such as the colour blind), these people can avail themselves of any offered occupation. The modern use of red and green signal lights precludes the colour blind from aerial, marine and transport pursuits, and certain branches of the textile industry.

2. Myopes. Nothing has been done for myopes in regard to employment, either in Tasmania or in Australia, but when a move is made in this direction within the Commonwealth, the investigations of the Board of Education Committee on the Partially Sighted (Crawley Report, 1934), should not be lost sight of. The Committee of Enquiry found that the myopes tutored in special classes deteriorated more rapidly in after school years than their fellow myopes tutored in ordinary schools, and they suggest that it appears important that "dioptre saving" must be continued in after school years to be effective. Consequently occupations should be found for myopes which do not entail lengthy periods of close work, or much bending or straining.

3. Partially Sighted. In England some organised attempt has been made to find suitable employment for these people after they leave the sight saving classes, but so far the result is disappointing. In Australia and Tasmania no attempt has been made to place these people in suitable occupations such as distributing trades, farm work, gardening, canvassing and domestic duties. Consequently they find the most remunerative occupation of which they are capable, without respect to suitability. This is most regrettable and certainly needs rectifying. In the United States, the Department of the Interior (1937), issued a Bulletin setting out occupations for handicapped adolescents in day schools and these suggestions included the visually handicapped.

4. Blind. The employment for the blind has been a most difficult problem ever since Valentine Hauy instituted it in Paris in 1748. There appear to be two difficulties. First, to find suitable employment, and secondly to dispose of the articles manufactured at a profitable rate; at the same time it must be remembered that, on account of mental, physical or temperamental disabiliies, at least 65% of the blind are unemployable (League of Nations Report, 1929). Best (1934), estimates that only 7.7% of the blind in the United States of America are self-supporting. Since the 1914-18 war many countries have attempted to find fresh employment for sightless workers, and, in many instances, blind men and women have been incorporated in sighted teams working in industrial concerns of various types. For instance, in England (League of Nations Report, 1929), 5 girls work in a team at Messrs. Cadbury, Fry and Pascall's factories packing chocolate; and another five at Messrs. Wells Ltd., on tin toys and certain processes in soap factories have been declared suitable for blind workers. In Germany, this branch was developed to an extraordinary degree and 218 occupations found among sighted workers. On the other hand, it is only natural that many employers have avoided utilising the blind in their factories on account of the fear of accidents, and resulting consequences under the various Workers' Compensation Acts. In Tasmania no attempt has been made in this direction, nor have the potentialities of subcontracting (N.I.B. Bulletin, No. 1), been investigated to any extent.

In Australia the occupations of the blind are almost entirely confined to broom making, mat making, piano tuning and music. In Tasmania there is one workshop at the Tasmanian Institution for the Blind where brush and mat making are the predominant industries. To-day, 1947, 19 blind men and 1 blind woman are employed there and the articles manufactured are sold to the forces. Each blind operative, besides receiving a blind pension of £2/2/6 per week from the Commonwealth Government, has his or her wages augmented by the Institution up to the basic wage.

Besides this, the Welfare Officers of the Institution, in Hobart, Launceston and Latrobe, instruct the blind women in cooking, towel hemming, tatting, knitting, crochet and bead work, from the sale of which they make a few shillings profit each week.

Music and piano tuning also find employment for a few blind men, but the advent of the radio has greatly reduced the number of pianos requiring tuning; and the advent of the talkies has thrown a great many professional musicians out of employment. This is most unfortunate as these two occupations are probably the most remunerative available for the blind. It should again be stressed here the scope of employment for the blind is extremely limited, and an enquiry into possible new trades for the blind is being carried out in Australia at the moment. Gardening has not been systematically attempted in Tasmania, but has been taught to several blind children and adults, in view of the satisfactory results obtained since 1920 by the Guild of Blind Gardeners of Great Britain (N.I.B. Bulletin, No. 3). Massage courses have not been instituted in Tasmania (and therefore are not available for the blind), although they have proved a very great success in London.

REFERENCES.

BEST, H. (1934), Blindness and the Blind in U.S.A. 2nd Ed., New York.

CRAWLEY, R. H. (1934), Report of the Committee of Enquiry into the Problems Relating to Partially Sighted Children.

LEAGUE OF NATIONS (1929), Report on the Welfare of the Blind.

MERRILL, E. B. (1936), Sight Saving Review. Vol. VI. No. 3. Sept.

NATIONAL INSTITUTE FOR THE BLIND. Basket Making for the Blind. Bulletin No. 5. London.

NATIONAL INSTITUTE FOR THE BLIND. Employment of the Blind. Bulletin No. 1. London.

NATIONAL INSTITUTE FOR THE BLIND. Gardening for the Blind. Bulletin No. 3. London.

NATIONAL INSTITUTE FOR THE BLIND. (1940), The Training of the Blind for Professions. London.

O'TOOLE, C. E. (1936), Sight Saving Review. Vol. VI. No. 2. June.

TASMANIAN BLIND INSTITUTION (1946), Annual Report, 1946.

UNITED STATES DEPARTMENT OF THE INTERIOR. (1937), Occupational Experiences for Handicapped Adolescents in Day Schools. Bulletin No. 30.

8. WELFARE.

The welfare of the blind in Tasmania is undertaken by the Tasmanian Institute for the Blind and Deaf, there being active branches of the welfare department in Hobart, Launceston and Latrobe.

It is arranged for three age groups under the following headings:—

1. Children of pre-school age.
2. Children from 5-16 years.
3. Adult Blind:—
   (a) Employable,
   (b) Unemployable and Necessitous.

Naturally these include those suffering from the various forms of hereditary blindness.

1. Children of pre-school age are visited by the Welfare Officers as occasion arises, and instructions are given to parents along orthodox lines. When necessary they are sent to the nursery at the Royal Victorian Institution for the Blind in Melbourne.

2. Children from 5-16 years:— These are educated at the residential school attached to the Institution, and education is compulsory from 5-16. Like the public schools, three terms a year are arranged and the children return to their homes for the vacations.

3. Adult Blind:— In 1947 there were 185 persons receiving blind pensions in the State of Tasmania, and, of these, 19 men and 1 woman were employed in the factory attached to the Institution, where brush and mat making are undertaken. The remainder were unemployable to a large extent, or were engaged in minor pursuits such as cooking and sewing. This is comparable to the figures for England and Wales, where about 70% of the blind are unemployed owing to age, mental weakness, or other defects.

Those not attending the factory are placed under the care of the Welfare Officers and their assistants, but to date there is no residential home either for old men or old women, as it has been found more satisfactory to find homes for them in the homes of the sighted. Best (1934), considers that such special homes should only be instituted as a last resource and, on the whole, are objectionable.

As far as I am able to estimate, the blind in Tasmania are costing the Commonwealth Government about £19,000 per annum, which is at the rate of 1/1d. per head of population per annum. On the other hand, Patrick (1939), estimated that in England the blind cost the local authorities under the Blind Pensions Act of 1922-1938, £1,861,932 which is at the rate of 10d. per head of population per annum.

Looking at this from another aspect, the 185 (104 men, 81 women) registered blind in Tasmania (June 1947) cost the Commonwealth Government more than £19,000 per annum. This means that on each registered blind person in Tasmania is expended about £98 per annum. On the other hand in England on the 74,494 registered blind was expended £26 per head per annum. This is explicable by the fact that there is approximately 1 blind person in every 1,700 inhabitants in Tasmania, while in England there is approximately 1 blind person in every 600. It would therefore appear that in Tasmania with proportionately a third as much blindness as in England and expending slightly more per head of population, the blind receive more than three times the assistance that they receive in England.

The facilities provided for the adult blind either by the State or Commonwealth Governments, State Municipalities, the Tasmanian Institute for the Blind and Deaf or other sources are:—

(a) Free tram passes by the City councils of Hobart and Launceston.

(b) Free railway passes by the Government. These allow each blind person and guide to travel for the fare of one. In special instances free passes for the blind travelling alone are issued.

(c) Free theatre tickets by theatrical and motion picture companies.

(d) Free radio licences by the Commonwealth Government.

(e) Radio sets either free or at cost price by the Institution for the Blind.

(f) Pensions have been paid by the Commonwealth Government since 1910. These are now at the rate of 42/6 a week, (£110/10/- per annum), for all blind adults over 16 years of age, whose total income from earnings and other sources does not exceed £260 per annum. Re-examination is seldom undertaken, so that undoubtedly certain pensioners who have received benefit from treatment still retain their pensions despite the fact that they are able to earn a livelihood.

(g) Augmentation of wages and pensions by the Tasmanian Institute has been mentioned under employment. It suffices to say that most operatives receive in all an amount equivalent to the basic wage pertaining in Tasmania.

(h) Retiring allowances by the Tasmanian Institution:— On a blind operative reaching the age of 65 (men), 60 (women), he or she is retired from the factory and the following payments are made weekly: married men, £2/5/-; unmarried men, £1/15/-; married or unmarried women, 15/-. These payments are made from Institution funds towards the blind person's upkeep, in order to supplement the pension of 42/6 a week.

(i) A Medical Union supervised by the Tasmanian Institute:— All blind pensioners by contributing 8/- per annum to the fund receive all medical consultations and all medicines free—also dental attention at cost price.

(j) Braille Writer's Association:— Members of this Association which works under the auspices of the Institute, take responsibility for all transcribing, importation of literature, training of Braille writers, supervision of Braille library, and other activities.

(k) Braille Mutual Progress Society also under the aegis of the Institute takes responsibility for weekly social gatherings which include lectures, card evenings, play readings and dances. Also visits to museums have been experimented with.

(l) Library and Talking books:— The former is controlled by the Braille Writer's Association and financed by the Institute. Free postage of books is permitted by the Commonwealth Government. Many of the talking books have been purchased by the Institute and distributed to the blind. A talking book library has been inaugurated and is controlled by the Hobart Welfare Officer.

From the above account it will be seen that every effort is being made in Tasmania to cope with the problems appertaining to the Welfare of the Blind, and the Northern, North Western and Hobart Welfare Officers now have orders to contact school teachers throughout the Island, and through them, the parents of the children with defective eyesight. These children are taken to an honorary oculist of the Institute both in Hobart and Launceston where treatment is carried out and educational advice given. This work, as I have indicated, has recently been extended to the North-West coast of Tasmania.

REFERENCES.

BEST, H. (1934), Blindness and the Blind in U.S.A. Page 460. 2nd Ed., New York.

CRAWLEY, R. H. (1934), Report of Committee of Enquiry into Problems Relating to Partially Sighted Children.

LEAGUE OF NATIONS REPORT (1929), The Welfare of the Blind.

NATIONAL INSTITUTE FOR THE BLIND. Handbook on the Deaf Blind. Bulletin No. 4. London.

NATIONAL INSTITUTE FOR THE BLIND. Care of the Blind Baby. Bulletin No. 6. London.
NATIONAL INSTITUTE FOR THE BLIND. Museums and the Blind. Bulletin No. 2. London.
PATRICK, J. (1939), National Institute for the Blind. Information Leaflet. No. 3.
SUTTON, H. (1935), Med. Journal Austral. April 6. Page 417.
TASMANIAN INSTITUTE FOR THE BLIND. (1945), Annual Report.

9. PREVENTION OF HEREDITARY DISEASE.

Blindness throughout the world has lately so concerned social workers and Governments, that International Associations for the Prevention of Blindness and also against Trachoma have been set up, as well as much legislation passed to check its apparent increase. As a result of this legislation, blindness from smallpox and ophthalmia neonatorum has greatly declined, while the ravages of Trachoma have been slightly checked; but little attempt has been made to check the enormous disability of hereditary eye disease probably because of the lack of reliable data. In Tasmania I find 25-30% of the blind are suffering from hereditary diseases of the eye, while Miles Bickerton (1934), estimated that 24% of the blind of Great Britain suffer from hereditary eye disease, and in 1937 he found that nearly half the 69 children in Wavertree School for the Blind, suffered from hereditary or developmental defects. He suggests possible remedies. Who, on reading this list of remedies can honestly suggest that more drastic education is not long overdue? Bickerton asserts that our present outlook is inhuman—with which I agree—but the difficulty is to persuade the public that reform is urgently necessary. Most men and women approach the subject of heredity in one of two ways, according to Eldon Moore (1934), either as "a mysterious tricky affair responsible for all those queer things in men, which cannot be explained otherwise, or else it is looked upon as a sort of fantastic demon whose existence all nice minded people should ignore." And Eldon Moore continues:— "Actually the hereditary mechanism is the most important part of all living things." I feel that few have grasped the truth of this last sentence.

Counsell and Hamilton (1939) reiterated the following recommendation made to the National Health and Medical Research Council in 1937:—

That, as at least 40% of the blindness in Tasmania (and probably in the rest of the Commonwealth of Australia) is preventable, active measures should be taken by the Commonwealth Government to lessen its incidence. We propose that the following measures be adopted:—

(a) That voluntary sterilization should be available for carriers of hereditary eye disease, and that consanguineous marriages should be entirely prohibited.

(b) That arrangements should be made for voluntary pre-marital certification.

(c) That only certified persons should be allowed to use explosives.

(d) That the eyes of artisans in certain industries should receive compulsory protection.

(e) That the midwive's regulations should compel the notification of any abnormal condition in the eyes of infants under the age of twenty-eight days, and should incorporate precise instructions for the adequate prophylaxis of ophthalmia neonatorum.

(f) That all pregnant women should be compelled to undergo adequate ante-natal supervision.

(g) That existing Commonwealth regulations should be more rigidly enforced to prohibit the landing of immigrants with signs of active or latent trachoma, and that trachoma should be a notifiable disease throughout the Commonwealth.

(h) That only registered medical practitioners should be permitted to treat diseases of the eye, including refractive errors in childhood.

(i) That sight saving classes be established in every city in the Commonwealth with a school population of over 5,000.

(j) That the routine examinations of the eyes of school children should be carried out by the school medical officers at least once yearly, and that there should be provision for expert treatment and adequate follow-up.

(k) That the services of an ophthalmic surgeon should be available to the inmates of all charitable institutions.

(l) That the registration of the blind should be compulsory.

(m) That the Commonwealth Government should adopt provisionally the model form of certification recommended by the English Committee for the Prevention of Blindness (Evans, 1936), until a standard British form is provided.

(n) That the certification of blindness should only be permitted by medical practitioners possessing special knowledge in ophthalmology.

At the end of our paper we also emphasised the following five points:—

First — The necessity for adequate and complete certification forms filled in by reputable oculists.

Second — Increased propaganda on eye disease and failing vision especially in adults passed middle life.

Third — Increased sight saving facilities, including schools.

Fourth — More rigorous treatment of all veneral diseases.

Fifth — That no pension be issued to patients with curable blindness.

Franceschetti, before the 1935 meeting of the International Association for the Prevention of Blindness, suggested the following measures for diminishing the incidence of hereditary blindness:—

(a) Collection of precise and complete statistics.

(b) Training of physicians especially ophthalmologists, in genetics and the education of authorities and the public.

(c) Extension of facilities for pre-marital consultation, and the general introduction of pre-marital certificates.

(d) Increased use of social workers.

(e) Decrease of consanguineous marriages.

(f) Decrease of the transmission of hereditary eye disease, by making sterilisation available to the patient.

No one can fail to agree with every one of the above suggestions and they are constantly kept before the Prevention of Blindness Committee of the Ophthalmological Society of Australia.

Let us discuss each of these suggestions, especially the last suggestion of sterilisation, for its effects would be more far reaching than any of the others.

(*a*) *Collection of precise and complete statistics:*— With regard to this recommendation, the Report of the Prevention of Blindness (1936), repeatedly stresses the fact that uniform certification of Blindness is essential before dependable statistics can be compiled. It suggest four improvements in this direction, namely:—

(1) A universal form for examination and report on all blind persons admitted to the register, and it published in detail the form recommended by the Ministry of Health (1933), in their circular No. 1,353.

(2) A Universal Definition of Blindness, and it quotes the Ministry of Health's circular No. 1,353, which includes such a definition.

(3) The certification of the blind should be undertaken only by a—"Medical practitioner with special experience in ophthalmology," and it quotes the Ministry of Health's definition of such a term in their circular No. 1,353.

(4) The collection of such statistics by a select sub-committee authorised by the Ministry of Health, and the Board of Education is essential. Authority to form such a Committee and to tabulate the statistical findings has been given to the Prevention of Blindness Committee.

Within the Commonwealth of Australia, (which includes Tasmania in its certification laws), there is absolutely no attempt to compile reliable statistics of the causes of blindness. No form other than that for invalid pensions in general is provided; no detailed definition of blindness has been formulated, and no statistics are kept of the certified blind. Further, the services of experienced ophthalmologists to certify the blind are not obligatory, but the Commonwealth Pensions Department is now being urged to take the necessary steps to have this rectified.

(*b*) *Training of physicians in eugenics, and the education of authorities and the public:*— The Prevention of Blindness Report (1936), is emphatic that the training of medical men in ophthalmology is inadequate, and recommends the close attention of the General Medical Council to this fact. The training of medical practitioners, and the education of authorities and the public in general in genetics, has been advocated for many years by the Eugenic Society in London, but has enlisted little support. Obviously man does not wish to face facts where reproduction of his species is concerned.

(*c*) *Extension of facilities for pre-marital consultation and introduction of pre-marital certificates:*— Like sterilisation to be discussed in detail later, this recommendation will obviously receive the wholehearted disapproval of the populace. Yet a stand must be made sooner or later to combat the evil results of hereditary disease. But just where to start concerns every social worker. I think it was Bernard Shaw who said, "First choose your parents." Men and women should not be allowed to mate with less respect to eugenics than is shown in breeding domestic animals. Every couple intending to marry should be asked to appear before a tribunal, presenting their pedigrees for scrutiny and approval, before marriage is allowed.

Legislation of this type was in vogue in Rumania, but in no other country has it been attempted up to the present. Those rejected should be advised of the reasons, and suggestions made for treatment. If the reasons for rejection are hereditary disease, then means should be taken to prevent rejected persons from breeding.

The Leicester Institution for the Blind (1939), in England, refuses to employ two blind individuals who are married to one another, unless a medical certificate is produced to say that the marriage will be childless.

(*d*) *Increased use of Social Workers*:— In this regard much excellent work has been done in the past, but there is gigantic scope for enlargement in the near future, especially in regard to out-patients work in special hospitals, and general hospitals with large ophthalmic departments.

(*e*) *Decrease in consanguineous marriages:*— Later in this work, where consanguinity is discussed in conjunction with nystagmus, optic atrophy and retinitis pigmentosa, it should convince the most ardent sceptic that consanguineous marriages are inadvisable, and should be legislated against at once. Best (1934), considers that—"the proportion of the blind with blind relatives is over three times as great amongst those whose parents are cousins as it is among the blind as a whole." Surely this is sufficient!

(*f*) *Sterilisation:*— At the end of 1933 the following countries throughout the world had passed sterilisation laws:—

26 States of the United States of America principally in the West. (24 of these States had compulsory laws and 2 have voluntary.)

2 States of Canada, both of which have voluntary laws.

1 Canton of Switzerland.

Germany, where sterilisation was compulsory.

Denmark with a voluntary law.

Except in the case of Germany, none of the above mentioned countries enacted laws on sterilisation to deal with hereditary blindness, but in the main deal with mental cases and feeblemindedness.

None of the six States of Australia has made any move in the direction of laws for human sterilisation, with the exception of Tasmania, but an inquiry into the subject under discussion should be a matter for investigation by the Commonwealth Government and not by States individually.

The Broche Report on Sterilisation (1933) was very guarded in its recommendations, and suggested very limited legislation to deal with the propagation of the unfit. For many reasons it was opposed to compulsory sterilisation, but strongly advocated voluntary sterilisation, not only of the mentally unfit, which was its limited reference, but also certain forms of hereditary blindness, deafness, haemophilia, and brachydactyly. This, I feel certain, will never be satisfactorily accomplished unless the physical results are expounded to the public in clear, straightforward terms.

Hereditary diseases of the eye have always interested eugenists particularly in view of the certainty of diagnosis in experienced hands. The Broche Report of the Committee on Sterilisation (1933), found its enquiry into hereditary mental deficiency much embarrassed by the fact that there were so many degrees of mental deficiency, and so many borderline cases. This is not so with hereditary eye diseases which are clear cut, and well recognised diseases. Yet throughout the whole Broche Report (1933), there is only one practical instance mentioned (Page 43), of the sterilisation of the hereditary blind. In view of the fact that so much has

been done to prevent blindness in other directions, surely this new avenue of prevention should be utilised without delay.

On the subject of the legality of sterilisation, the above reports are quite emphatic. Legality of therapeutic sterilisation is not disputed, but the legality of eugenic sterilisation in the present state of the Law of England is couched in the following terms on Page 6, "but in practice it seems to be almost universally accepted that sterilisation is illegal, and involves the surgeon in the risk of legal proceedings, even though the full consent of the patient be obtained." On the other hand, Beasley (1935), discussing the legality of eugenic sterilisation of the unfit in Western Australia, asserts: "Sterilisation performed at the request of a patient who understands the nature and result of the operation, and freely consents thereto, does not constitute a disablement within the meaning of Section 294 of the Western Australian Criminal Code, and does not involve in criminal responsibility the surgeon who performs the operation."

H. C. Lewis, barrister-at-law, in a private communication with reference to the laws of Tasmania in regard to eugenic sterilisation, is of the opinion that it is inadvisable in this Island under the existing laws.

As clear thinking men and women with inherited defects have commenced to ask for sterilisation, as has been the case in Tasmania, then the time is ripe, more than ripe, for the Government of this Commonwealth to take the matter up, and have the proper legislation for voluntary sterilisation enacted without delay, to include not only the mentally unfit, but those suffering from all inherited diseases.

If these laws were enacted there is a possibility of stamping out over half the preventable blindness in Tasmania—a most pleasing prospect.

LOCAL CONDITIONS:—

The Tasmanian Committee for the Prevention of Blindness and Deafness has now been functioning intermittently for over eight years, and consists of two educationalists, one ophthalmologist, one aurist, one Braille writer, one accountant and one politician. It had many meetings prior to 1941, including conferences with the Director of Public Health, the Superintendent of the Lunatic Asylum, the Chairman of the Mental Deficiencies Board regarding voluntary sterilisation. It has participated in the launching of the Sight Saving School, arranged with the Hydro-Electric Commission for better public and private building lighting, and has drawn the attention of the authorities to the need for better ante-natal care for mothers. It has conferred with the Ophthalmological Society of Australia regarding a Commonwealth Scheme of Prevention, and has now issued brochures on the Prevention of Blindness to authorative citizens throughout the State.

REFERENCES.

ANDERSON, J. R. (1939), Med. Journ. Aust. November 14th. Page 680.

BEASLEY, F. R. (1935), Med. Journ. Aust. March 9th. Page 293.

BEST, H. (1934), Blindness and the Blind in U.S.A. 2nd Ed. New York.

BICKERTON, J. M. (1934), Brit. Med. Journ. January 20th. Page 93.

BROCK, (1934), Report of the Departmental Committee on Sterilisation, Ministry of Health. Great Britain.

COMMONWEALTH OF AUSTRALIA (1935), Official Year Book. Page 514.

COMMONWEALTH BUREAU OF CENSUS AND STATISTICS (1940), Private Communication.

CRISP, H. R. (1935), Med. Journ. Austral. March 9th. Page 303.
DEPARTMENT OF PUBLIC HEALTH. Children's Eyesight. Hobart.
FRANCESCHETTI, A. (1935), Report of the International Association for Prevention of Blindness. Page 14.
FRANCESCHETTI, A. (1938), Ophthalmologica. Vol. 97. Page 203.
HAMILTON, J. B. (1940), Trans. Ophth. Soc. Austral. Vol. 11. Page 37.
LEICESTER INSTITUTION FOR THE BLIND (1939), Regulations. (Abstract.)
MINISTRY OF HEALTH (1939), Circular 1,621. London.
MINISTRY OF HEALTH (1938), Circular 1,681. London.
McWHAE, D. M. (1935), Med. Journ. Austral. March 9th. Page 298.
MOORE, E. (1934), Heredity — Mainly Human. London.
SHARP, S. G. K. (1939), New Beacon. July 15th. Page 182. (Abstract.)
STANDING COMMITTEE FOR THE PREVENTION OF BLINDNESS OF THE UNION OF COUNTIES ASSOCIATION FOR THE BLIND (1936), Report on the Prevention of Blindness.
STERILISATION OF THE UNFIT (1935), Discussion. Med. Journ. Aust. March 9th. Page 318.
SWAB, C. M. (1934), Nebraska Med. Journ. Vol. XIX. pp. 184-187.
TASMANIAN INSTITUTION FOR THE BLIND (1945), Personal Communication.
THOMPSON, E. J. T. (1935), Med. Journ. Austral. March 9th. Page 301.

10. MODE OF TRANSMISSION.

It is not within the scope of this paper to attempt to expound the problems of Mendelian characters in regard to Ophthalmology, but rather to decide whether or not hereditary eye disease in Tasmania conforms to these established laws. It is only to be expected in view of the fact that most of the original members of these pedigrees were born within the British Isles that these laws should hold in Tasmania.

Since 1900, Mendel's laws have become widely appreciated, so that patients affected with hereditary diseases are constantly seeking advice from their medical practitioners. Thus it behoves all medical men, and especially ophthalmologists, to be conversant with the various types of transmission, and I therefore propose to give a brief survey of the behaviour of Mendelian and sex-linked characteristics, for guidance when discussing the pedigrees later.

DOMINANT TRANSMISSION.

1. If both parents are affected:—
   (a) DD. x D(R). All children affected.
   (b) DD. x DD. All children affected.
   (c) D(R). x D(R). $\frac{3}{4}$ children affected, $\frac{1}{4}$ normal.
2. If one parent is affected:—
   (a) DD. x RR. All children affected.
   (b) D(R). x RR. $\frac{1}{2}$ children affected, $\frac{1}{2}$ normal.

RECESSIVE TRANSMISSION.

1. If both parents are affected:—
   RR. x RR. All children affected.
2. If one parent is affected:—
   (a) DD. x RR. All children normal.
   (b) D(R). x RR. $\frac{1}{2}$ children affected, $\frac{1}{2}$ normal.

SEX LINKED TRANSMISSIONS.

The disease may be dominant, or recessive — generally recessive.

A. If the Disease is Recessive:—

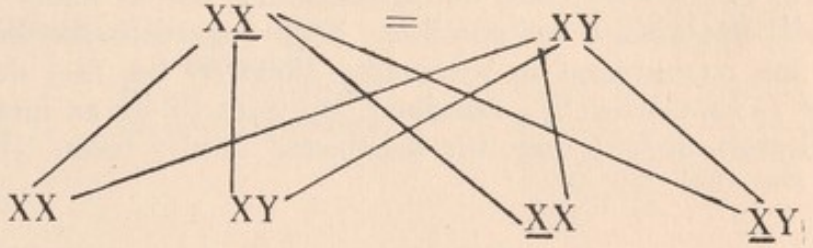
1. The males are affected more frequently than the females.
2. Females are of three kinds.
   (a) Completely normal and not a carrier = XX.
   (b) Completely normal, but a carrier X$\underline{X}$
   (c) Affected $\underline{X}\underline{X}$.
3. Males are of two kinds.
   (a) Completely normal = XY.
   (b) Affected $\underline{X}$Y.

Note:— Males cannot be carriers in the sense of (2b).

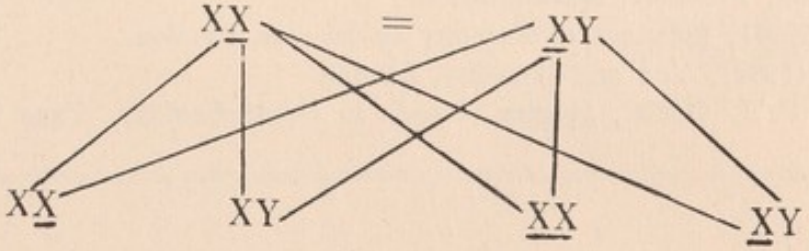
Examples of recessive sex linkage:—

1. A female of class (2a) married to an affected male (3b) will produce normal children. All the daughters will be carriers (2b) and will transmit the disease to half their sons.

2. A carrier female (2b), married to an unaffected male (3a), will transmit the diseases to half the sons. Half the daughters will be carriers (2b).



3. A carrier female (2b), married to an affected male (3b), will have half the daughters affected (2c), and the other half carriers (2b). Half the sons will be affected (3b), and half will be normal.



4. An affected female (2c), married to a normal male (3a), will hand on the disease to all the sons (3b), and all the daughters will be carriers (2b), and will hand on the disease to half their sons.

B. If the Disease is Dominant:—

Females—

(a) Diseased XX
(b) Diseased XX
(c) Normal XX

Males—

(a) Diseased XY
(b) Normal XY

Joseph Pearson, D.Sc. (1940), who has been most generous with his advice, drew my attention to three important facts in assessing human pedigrees.

1. The information given by patients may be intentionally or unintentionally unreliable.
2. The fertilizing spermatozoa may not be that of the woman's husband.
3. Some diseases at least appear to be caused by more than one gene.
4. Mutations must not be lost sight of, and are, comparatively speaking, common in some diseases, such as amaurotic family idiocy.

Later, when I comment on each individual pedigree which I am reporting, I shall endeavour to define the mode of transmission, but as many of the pedigreees are short, this will not always be possible, With regard to the brevity of some of the pedigrees, I am encouraged in presenting them by the fact that the Bureau of Human Heredity (115 Gower St., London), appears to be as interested in shorter as in longer pedigrees, and appeals for unselected family trees. (Brit. Med. Journ., 1936.)

REFERENCES.

BLACKER, C. P. (1934), The Chances of Morbid Inheritance, London.
BRIT. MED. JOURN. (1936), Vol. 1. Page 1,259.
DAWSON, Lord (1935), Brit. Med. Journ. Nov. 2. Page 829.
GATES, R. G. (1929), Heredity in Man, London.
PEARSON, J. (1940), Personal communication.
PENROSE, L. S. (1934), Influence of Heredity on Disease, London.
SCHEINFELD, A. (1939), You and Heredity, London.
WAARDENBURG, P. J. (1940), Modern Trends in Ophthalmology. Page 101.

THE SIGNIFICANCE OF HEREDITY IN OPHTHALMOLOGY.

A TASMANIAN SURVEY

By

J. BRUCE HAMILTON

Part 2.

TABLE No. XII.

LIST OF PEDIGREES.

This table indicates the inherited eye diseases so far found in Tasmania, with the pedigree numbers against each disease. There are 18 diseases and 111 pedigrees.

DISEASE	PEDIGREE NUMBERS
ANGIOID STREAKS	No. 151.
CATARACTS — CONGENITAL	Nos. 6, 7, 8, 9, 18.
CATARACTS — Dystrophia Myotonica	No. 10.
CATARACTS — SENILE	Nos. 87, 163, 217.
COLOBOMA OF THE IRIS	No. 142.
DACRYOCYSTITIS	Nos. 71, 107, 135, 143, 144, 145, 150, 176.
GLAUCOMA	Nos. 11 (with 113), 13, 14, 77, 91, 103, 108, 179, 203.
KERATOCONUS	Nos. 15, 16, 222.
MACULAR DEGENERATION—Senile pigmentary	No. 118.
NYSTAGMUS	Nos. 4, 27, 28, 29, 30.
OPTIC ATROPHY — LEBER'S	Nos. 31, 33, 34, 88, 221.
PTERYGIUM	Nos. 35, 141.
PTOSIS	Nos. 36, 215, 216, 232.
REFRACTIVE ERRORS—Hypermetropic Astigmatism	Nos. 5, 69, 91, 92, 96, 129.
Mixed Astigmatism	Nos. 1, 2.
Myopic Astigmatism	Nos. 20, 21, 22, 23, 25, 55, 63, 78, 95, 101, 124, 125, 140, 146, 148, 158, 162, 168, 169, 173, 180, 210, 213.
RETINAL DETACHMENT	No. 37.
RETINITIS PIGMENTOSA	Nos. 38, 39, 40, 41, 190.
SARCOMA OF THE CHOROID	No. 89.
STRABISMUS:—	
Esophoria	Nos. 42, 198, 200.
Exophoria	Nos. 44, 45, 79, 106, 152, 164, 166, 174.
Esotropia	Nos. 46, 47, 49, 50, 51, 52, 157, 177, 184, 191, 226, 229.
Exotropia	No. 204.
Hyperphoria	No. 43.

TABLE No. XIII.

This table is the key to the signs used throughout the pedigrees and will need to be referred to in rare instances only.


Albinism.


Albuminuric Retinitis.


Allergy.


Amblyopia.


Angioid Streaks.


Aniridia.


Anisocoria.


Anisometropia.


Aphakia.


Astigmatism, Hyper.


Astigmatism, Mixed.


Astigmatism, Myopic.


Binocular Blind.


Born With One Hand.


Brain Tumour.


Cataract, Blue Dot.


Cataract, Complicated.


Cataract, Congenital.


Cataract, Diabetic.


Cataract, Senile.


Cataract, Traumatic.


Cavernous Atrophy of Disc.

Cerebral Tumour.


Mentally Defective.


Microphthalmia.


Migraine.


Mirror Writer.


Monocular Blind.


Myopia.


Nystagmus.


Ocular Palsy.


Ophthalmia, at Birth.


Optic Atrophy.


Overaction Inferior Oblique.


Papilloedema.


Papilloma of Lid.


Paralysis of Accommodation.


Paralysis External Rectus.


Paralysis Inferior Rectus.


Paralysis Superior Rectus.

Periph. Cystic Ret. Degeneration.

Periph. Pigmentary Ret. Degeneration.

Phlycten.

Pigmentary Degen. of Macula.

Presbyopia.

Pseudo-Glioma.


Choroidal Stretching.


Choroiditis.


Choroido-retinal-degeneration.


Colour Blindness.


Convergence Excess


Convergence Insufficiency


Cyclophoria.


Cystic Disease of the Lungs.


Dacryocystitis.


Dead


Deafness


Defective Sight


Detached Retina.


Diabetes.


Dislocated Lens.


Divergence Insufficiency.


Epicanthus.


Epiphora.


Esophoria.


Esotropia.


Exophoria.


Exotropia.


Glaucoma.


Heterchromic Cyclitis.


Propositus.


Pseudo-Xanthoma Elasticum.


Pterygium.


Reattached Detachment of the Retina.


Retinal Haemorrhage.


Retinal Arterio-Sclerosis.


Retinal Cysts.


Retinitis.


Retinitis Pigmentosa.


Retinitis Proliferans.


Retinitis Punctata Albescens.


Retrobulbar Neuritis.


Sarcoma of the Choroid.


Sec. Optic Atrophy.


Senile Ectropion.


Senile Exud. Retinitis.

Spasm of Accommodation.

Strabismus.

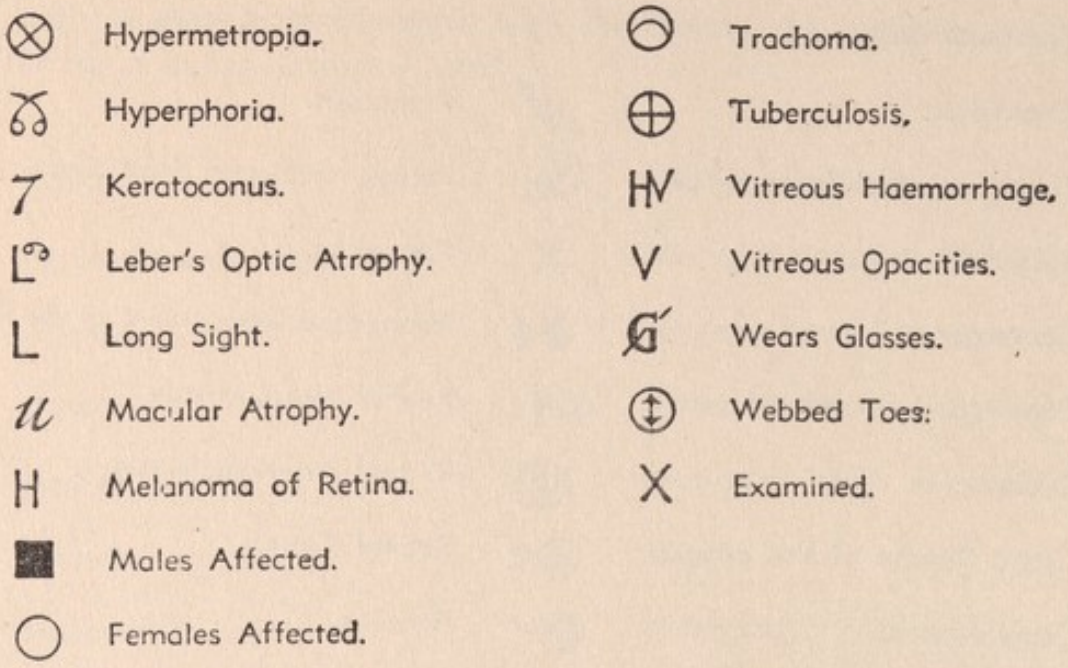
Tay's Choroiditis.

Third Nerve Paralysis.

Thrombosis of Retinal Vein.

Tobacco Amblyopia.

TABLE No. XIII (Continued)



I intend to take the eighteen diseases in alphabetical order and, by doing so, will commence with the least satisfactory of them all . . .

ANGIOID STREAKS.

(Gronblad-Strandberg Syndrome)

I am encouraged to report this pedigree (No. 151) by a remark of Batten (1931), that bilateral vascular choroiditis without angioid streaks may appear in one member of a family, some other members of which exhibit typical angioid streaks. Also by Duke Elder's (1940) remark that the condition has a "sporadic familial tendency, the lesion being transmitted as recessive hereditary characteristic." Both these comments appear to apply to my pedigree (No. 151) under review.

CONSANGUINITY.

Gronblad (1938) reported two cases resulting from a consanguineous marriage, but there was no consanguinity in my pedigree—nor in a second non-Tasmanian one which I possess. That there is a decided family and doubtful hereditary incidence is apparent, because in the non-Tasmanian pedigree (which I am not reporting) appear four brothers with the disease and now I hear of a nephew in New Zealand who has also become affected.

CONCURRENT ABNORMALITIES.

Gronblad (1938) shows clearly that Angioid Streaks, Pseudo Xanthoma Elasticum (Darier) and Osteitis Deformans (Paget's Disease), are closely related entities, and this has been confirmed by Batten (1931), Terry (1934), Benedict and Montgomery (1936), and Duke Elder (1940), but it appears from Batten (1931) and from pedigree No. 151, that central exudative retinitis may also be a feature.

SEX INCIDENCE.

In the 10 cases reported by Batten (1931), 6 were males and 4 were females, but in my pedigree No. 151, the 3 cases were females, while, on the other hand, in the non-Tasmanian pedigree (not to be reported) the five cases affected were males.

COMMENTS ON PEDIGREES.

Pedigree No. 151.

11/2 Female, aged 86 years, reported with defective vision. Her visual acuity was reduced to: Right vision 4/36; Left vision 3/36, and through both sclerosed lenses could be seen extensive patches of senile exudative choroiditis, the limits of which could not be well defined. She had no refractive error. Her skin was not examined.

111/1 Daughter of 11/2 seen at the age of 48 years; complaining of defective sight of two years' duration. Her vision with myopic and presbyopic correction was: Right vision 6/36, and J.8; left, 6/18 and J.5. She had a large absolute central scotoma right and left for 1/330 white. One year later her visual acuity was: Right vision equals Fingers at one metre; Left vision equals 6/60 with correction.

Her fundi showed old pigmented choroiditis at both maculae with angioid streaks right and left. Her skin exhibited typical pseudo-xanthoma elasticum.

111/2 Female aged 52 years. Eyesight, chiefly left, failing for 15 months. Vision with myopic and presbyopic correction is:: Right vision 6/18 + J.4; left vision 6/60.

Both fundi exhibit active senile massive exudative retinitis without angioid streaks. Her skin reveals no abnormality.

REFERENCES.

BATTEN, R. (1931), Brit. Jl. Ophthal. Vol. XV. Page 279.
BENEDICT and MONTGOMERY (1936), Brit. Jl. Ophthal. Vol. XX. Page 551.
DUKE ELDER, W. S. (1940), Textbook of Ophthalmology. Vol. 111. Page 2,413. London.
GRONBLAD, E. (1938), Arch. Ophthal. Vol. XIX. No. 1. Page 1.
GRONBLAD, E. (1940), Modern Trends in Ophthal. Page 92. London.
HAGEDOORN, A. (1939), Arch. Ophthal. Vol. XXI. No. 5. Page 746. Arch. Ophthal. Vol. XXI. No. 6. Page 935.
LAW, F. W. (1938), Trans. Ophthal. Soc. U.K. Vol. LVIII. Part 1. Page 191.
TERRY, T. L. (1934), Trans. Amer. Ophthal. Soc. Vol. XXXII. Page 555. (Abstract)

CATARACTS — CONGENITAL.

According to Macklin (1927), it is estimated in Canada that 13 per cent. of the pupils in institutions for the blind are suffering from hereditary cataract. The proportion in Tasmania in 1937 was much higher than this; three (3) of the six (6) blind pupils in the Tasmanian Institution for the Blind were suffering from cataract. On the other hand, Best (1934), considers that 13.7 per cent. of blindness in U.S.A. is due to cataract, but in Tasmania (1937) I found 20.45 per cent. of the blind, of whom reliable clinical information is obtainable, suffering from this disease. This figure appears to me to be astounding, especially in view of the fact

that of all hereditary eye diseases causing blindness, "cataract" should be the most responsive to treatment. I can account for this high percentage in Tasmania in the following manner:—

(1) *Late operative interference*:— This is exemplified in Pedigree No. 7, where one father (IV/15) refused to allow his children to be operated on until they had developed nystagmus and strabismus with amblyopia. In other cases, operation has not been considered, due to ignorance or isolation of the parents.

(2) *Incomplete operative interference*:— This is exemplified in Pedigree No. 6, where (1V/5) had a right needling done many years ago, but did not report for glasses, which eventually gave him 6/12 partly and J.4. Fortunately, he has submitted his daughter, aged one and a half years, for operation and the visual result was good.

(3) *Conservative Medical Advice*:— The unsatisfactory results that have sometimes followed discission of the crystalline lens has often deterred ophthalmologists from advising operative interference.

(4) The Commonwealth Government does not insist on applicants for blind pensions undergoing expert examination and treatment before being accepted. It is left to the applicant's choice between blindness with pension, or sight without pension, and, unfortunately, many choose the former course. This, undoubtedly, should be remedied at once.

SEX INCIDENCE.

In my 5 pedigrees (Nos. 6, 7, 8, 9 and 18) there are 19 affected males and 27 affected females, giving a percentage of 41.3 and 58.7 respectively. In pedigree 7 there are 5 members whose sex has not definitely been determined. Taking the pedigrees reported by Bishop Harman (1909), Macklin (1927), Galloway (1930), Khan (1926), Knapp (1926), and Dunforth (1914), I have found the sex incidence as 55 males and 63 females, giving a percentage of 46.6 and 53.4 respectively, which conforms wth the predominance of females shown above.

AGE OF ONSET.

Loy (1936) quotes Nettleship's 6 laws pertaining to the occurrence of cataracts in females, and the fifth law states that "the age of onset is approximately the same in persons of the same generation." In my five pedigrees, as far as I am able to ascertain, every case was apparent from infancy with the exception of (111/23) of pedgree 8, where the opacities are only partially developed at puberty.

TYPE OF OPACITY.

According to Clapp (1934), congenital cataracts may take 10 partial forms, and, of course, one complete form, but nowhere in his book can be found a discussion on the hereditary nature of these opacities.

My longest pedigree (No. 7), exhibited zonular opacities almost uniformly, with occasional dislocation of the lens, and in one case at least, the lens had been practically absorbed. In pedigree No. 6 of the cases examined, 2 were of coralliform type, while in pedigree No. 8 it was impossible to ascertain if uniformity existed or not.

I have used the word "zonular" in reference to pedigree No. 7 intentionally, as I feel the word "lamellar" should be left for those cataracts following infantile tetany.

TYPE OF INHERITANCE.

In pedigree No. 7 the defect is possibly dominant in transmission, while in the other 4 pedigrees the number of affected individuals is too small to make even a conjecture.

ASSOCIATED DEFECTS.

Feeblemindedness. There is no evidence of this in any of the 5 pedigrees. A small number of the affected individuals are somewhat retarded mentally and occasionally temperamental, but in no case could I classify them as feebleminded.

Dystrophia Myotonica. The one pedigree (No. 10) of this disease is described separately to avoid any possible confusion.

Endocrine Disturbance. There is no evidence of these in my Tasmanian pedigree.

Rubella in Pregnancy. This can be definitely excluded in all cases. In the majority of patients the cataracts were present before this new disease arose. (Gregg, 1941, 1944, 1945, Swan, 1943-1944.) The earliest case of congenital defect in the infant resulting from Rubella contracted by the mother during pregnancy, occurred in Tasmania in 1929.

Toxoplasma of Pregnancy. Too little is as yet known about the relation of toxoplasma to congenital cataracts to express an opinion (Koch, 1943; Robertson and Hamilton, 1946); but the matter should certainly be kept in mind.

Treatment. Operative interference in the form of repeated needling has been singularly successful in these Tasmanians with one exception. When I say successful, I refer to anatomical,, but not the functional results. All the poor functional results were due to late operation, and this stresses the fact that early operation is essential if good visual acuity is to be expected. To obtain this the public must be educated and its confidence gained.

COMMENTS ON PEDIGREES.

Pedigree No. 6. This is an example of coralliform congenital cataract.

111/6 Female, age unknown, has cataracts but has not yet been examined.

1V/5 Male, aged 26 years, has had his right cataract needled after puberty, with a very fair visual result; while his left eye exhibits a typical coralliform cataract.

1V/10 and 13. Females, aged 22 and 17 years respectively, have been examined by an ophthalmic surgeon and certified for pension purposes as blind from congenital cataract.

V/3 Female, aged 12 years, has typical coralliform cataract in a very advanced state right and left, with nystagmus. Both cataracts have been needled, and the visual result is good.

V/6 Male, age unknown, has cataracts but has not been examined by me.

V/10 Male, aged 8 years, has congenital cataracts and this has been confirmed by one of my ophthalmic colleagues.

Pedigree No. 7. This pedigree is one of zonular cataracts.

111/1 Male, first examined at age of 23 years with nystagmus. Both eyes needled for congenital cataract 8 years previously and left sight lost from cyclitis. At age of 44, right capsulotomy followed by retinal detachment.

111/2 Female, first examined at age of 62 years. History of poor sight for 40 years at least, accompanied by nystagmus and left esotropia. She had mature right black cataract dislocated up and in. Left shrunken cataractous lens was dislocated up and out. Right intracapsular extraction performed with 6/36 + J.4 visual result.

111/8 Female, is one of the propositii. She was examined at the age of 48 years. Twenty-five years previously her right lens had been removed by operation, with good operative but poor visual results, the latter due to amblyopia. In her left eye besides her cataract she had gross keratoconus and nystagmus.

1V/1 Female, seen at the age of 9 years with right and left adherent leucomata. Bilateral iridectomies revealed immature cataracts of unspecified nature. She also has nystagmus and squint.

1V/3 Male, examined at 12 years. Then had posterior polar cataracts with nystagmus. Right eye needled and result unknown. He also has strabismus.

1V/5 Male, age unknown, has been examined for pension purposes, and certified blind from congenital cataracts.

1V/9 Male, examined at age of 9 years, then had nystagmus, right internal strabismus and bilateral posterior polar cataracts. Right eye needled with corrected result of 3/60.

1V/18 Female, seen at the age of 10 years. Nystagmus with right central and left post cortical lens opacity. Both lenses needled with resulting corrected vision of 3/36 right and 6/60 left.

1V/21 Male, examined at age of 36 years. Nystagmus and alternating strabismus with bilateral congenital cataract.

1V/25 Female, had opaque lenses right and left when examined by me at age of 42 years. Both irides were tremulous, and the left lens obviously dislocated. There was marked nystagmus. I extracted the right lens by the intracapsular method of Sinclair, and, although the anatomical result is good, acquired amblyopia prevents a good visual one. Nevertheless the vision was materially improved by the operation, until a retinal detachment developed.

V/12 Male, seen at age of 46. Dense bilateral cataracts. Vision improved with + 10.0 right and left to counting fingers at 3 feet in each eye. He has dislocated lens and, presumably, uses his aphakic area.

V/15 Female, seen at age of 37 years. Dense bilateral congenital cataracts—left is dislocated up and in. There is a good reflex through left aphakic gap with corrected vision to 4/60. Nystagmus is marked ←operation is always refused by patient. This patient was also a propositas.

V/36 Male, aged 23 years, has had both zonular cataracts needled by me, and his squint straightened. But nystagmus and amblyopia, (both congenital, and ex anopsia) prohibit a good visual result, yet he is considerably improved by the operative interference and can use an ordinary typewriter with ease. This patient was also a propositas.

V/37 Male, aged 21 years. Has also congenital cataracts, nystagmus and squint. Both lenses are markedly dislocated, so that through the aphakic gaps with correction, he is able to read 5/60 with both together. In view of the post-operative visual result of his brothers, I feel that no operative interference would improve the present visual acuity.

V/40 Male, aged 16 years. Had dense zonular cataracts right and left, with right convergent concomitant strabismus, and gross nystagmus. He has had both lenses needled and his squint straightened by me three years ago, and is now pursuing a High School education. His visual result is poor due to amblyopia.

V/42 Female, aged 12 years. Had only thick capsules right and left, although she had had no previous operations. A capsulotomy was done on each eye by me four years ago, and with correction she is able to see 6/36 right and left, and J.10 right, and J.16 left with bifocals. I think V/42 certainly indicates that had these 5 siblings been operated on in infancy, the visual results would have been more satisfactory.

V/43 Male, aged 7 years, also had dislocated zonular cataracts, nystagmus and concomitant strabismus. Three years ago both lenses were needled, and his squint straightened by me, but the visual improvement is only slight owing to amblyopia.

V1/1 Female, seen at the age of 16 years. Nystagmus and dense central lens opacity right and left. Both cataracts needled with resulting corrected vision of 6/60 each eye. Divergent strabismus present.

V1/12 Female, seen at the age of 5 years. Blind since birth. Both cataracts needled with satisfactory operative result. Exact vision not obtainable yet.

V11/1 Female, seen at the age of 9 months. Nystagmus with dense central cataracts right and left. Parents refuse operation.

Pedigree No. 8.

111/23 Male, aged 14 years. He complained of defective sight and had marked anterior and early posterior cortical opacities in both eyes, but the vision with correction in each eye is still 6/12. I had been at a loss to account for the condition of 111/36, until 111/23 appeared.

111/36 Female, aged 11 years, has had cataracts since birth and was operated on when 5 months of age in both eyes without result; is now an inmate of a blind institution, with gross nystagmus, shrunken lenses and secondary glaucoma.

Pedigree No. 9.

11/1 Male, age unknown, is a blind pensioner, certified by an ophthalmic surgeon as having congenital cataracts. The type is not stated, but his vision is less than 6/60 in each eye.

11/11 Female, examined at age of 16 years. Right adherent leucoma with dense lens opacities. Left globe staphylomatous.

111/1 Female, aged 19 years, has been examined by me, and has gross post-cortical lens opacities, so that right and left vision is reduced to hand movements. To date she has refused all operative interference. She is the propositus.

IV/1 Female, aged one year, is not affected. She is the elder daughter of III/1.

Pedigree No. 18. This is a pedigree of inherited cataracts (zonular) very much resembling those found after infantile tetany but without the riders.

II/2 It is believed that this female had congenital cataracts.

III/1 Female, aged 42 years, bilateral cataracts. This patient, with correction, can read 6/18 + J.1 in her better (left) eye. Her right vision, with correction, is 6/60. Her right and left refractive error was hypermetropic astigmatism.

III/4 Female, aged 33 years. Sister of III/1.—Bilateral cataracts. She has, with correction, 6/24 + J.4, right and left eyes. Her right and left refractive error was simple hypermetropic astigmatism.

III/12 Male, aged 47 years. Has fine bilateral zonular cataract with normal vision with correction in right and left eyes. The right refractive error is mixed astigmatism while the left refractive error is hypermetropic astigmatism.

III/13 Female, aged 43 years. Has high myopic astigmatism only. Was wife of III/12.

III/17 Female, aged 32 years. Had congenital cataract herself, but these have now been removed successfully. She has married a male related to this pedigree, who is himself free from the disease.

III/18 Female, aged 29 years. Gross bilateral zonular cataracts and high myopic astigmatism. Corrected vision in each eye is 6/9 + J.1. Her right and left refractive error is compound myopic astigmatism.

IV/6 Male, aged 23 years. Zonular cataract right and left with high myopic astigmatism. Vision corrects to 6/24 + J.4 right and left.

IV/7 Female, aged 16 years. No cataracts but high myopic astigmatism.

IV/9 Female, aged 12 years. Zonular cataracts with myopic astigmatism right and left. Vision is 6/9 + J.1 right and left eyes with correction.

REFERENCES.

BEST, H. (1934), Blindness and the Blind in U.S.A. 2nd Edition. New York.
BUTLER, T. H. (1927), An Illustrated Guide to the Slit-Lamp. London.
CLAPP, C. A. (1934), Cataract. Its Etiology and Treatment. London.
DANFORTH, G. H. (1914), Amer. Jl. Ophthal. Vol. XXXI. Page 161.
FOSTER, J. (1933), Brit. Jl. Ophthal. Vol. XVII. No. 7. Page 408.
GALLOWAY, R. M. (1930), Brit. Med. Jl. January 25. Page 149.
GREGG, N. M. (1941), Transaction of Ophthal. Soc. of Aust. Vol. III. Page 35.
GREGG, N. M. (1944), Transactions of Ophthal. Soc. of Aust. Vol. IV. Page 119.
GREGG, N. M. (1945), Med. Jl. of Aust. Vol. 1. No. 13. Page 313.
HARMAN, N. B. (1909), Trans. Ophthal. Soc. U.K. Vol. XXIX. Page 101.
KAHN, W. A. (1926), Brit. Jl. Ophthal. Vol. X. Page 387.
KNAPP, F. N. (1926), Amer. Jl. Ophthal. Vol. IX. Page 683.
KOBY, F. E. (1930), Slit-Lamp Microscopy of the Living Eye. 2nd Edition. Page 233. London.
KOCH, F. P. L. (1943), Arch. of Ophthal. Vol. XXIX. No. 1. Page 1.
MACKLIN, M. T. (1927), Canad. Med. Assoc. Jl. Vol. XVII. Page 940.
NETTLESHIP, E. (1909), Trans. of Ophthal. Soc. of the U.K. Vol. XXIX. Page 189.

ROBERTSON, G. and HAMILTON, J. B. (1946), Trans. Ophthal. Soc. of Aust. Vol. V. Page 85.

SWAN, C. (1943), Med. Jl. of Aust. Vol. 11. Page 201.

SWAN, C. (1944), Med. Jl. of Aust. Vol. 1. Page 409.

VINSONHALER, F. and COSGROVE, K. W. (1936), Arch. Ophthal. Vol. XV. No. 2. Page 222.

VON BAHR, G. (1936), Studies on the Aetiology and Pathogenesis of Cataracta Zonularis (Uppsala).

CATARACT WITH DYSTROPHIA MYOTONICA.

In 1911, Greenfield reported the first cases of cataract associated with dystrophia myotonica, and in 1924, Adie pointed out that cataract occurred alone in many individuals in a dystrophia myotonica pedigree. Vogt, in 1921, and Goulden in 1928, gave a lengthy description of the slit-lamp appearance, so typical of the disease, and many cases of successful extraction have been reported (Souter, 1933). Rouquier and Christian (1935), consider the neurological lesion to be in the diencephalon and the mesencephalon, but no satisfactory explanation has been given for the accompanying cataracts.

Comment on Pedigree No. 10.—I have but one pedigree (No. 10) to report, and that one exhibits cataract in the father (1/1, while three children, 11/8, 11/13, and 11/15 have dystrophia myotonica and cataracts.

1/1 I can make little comment on 1/1 who has not been examined by me, but has had a recent cataract extraction.

11/8 Female, aged 41 years, had her left cataract extracted four years ago at the age of 37 years, and her right lens exhibits an advanced, but typical stage, of myotonic cataract. She has obvious signs of muscle wasting and the typical myotonic grip.

11/13 Male, aged 40 years, brother to 11/8, has loss of power in both hands and also has lens opacities in the anterior cortex of both lenses, typical of dystrophia myotonica.

11/15 Male, aged 29 years, another brother, conformed physically to an historic case reported by Caughey (1933), viz:—

"He is short, bald, speaking through his nose, looking forty at the age of twenty-five."

As a matter of fact, he was 29 when he came under observation and had typical posterior star-shaped lens opacities in the cortex of both lenses. He had muscular weakness and a myotonic grip. Both cataracts were removed and at present he has 6/4 and J.1 left and 6/6 + J.1 right vision with aphakic correction.

REFERENCES.

ALLEN, J. H. and BARER, C. G. (1940), Arch. of Ophthal. Vol. 24. No. 5. Page 867.

BUSCHLE, W. (1943), Arch. of Ophthal. Vol. 30. No. 6. Page 751.

CAUGHEY, J. E. (1933), Proc. Roy. Soc. Med. Vol. XXVI. No. 7. Page 848.

KOBY, F. E. (1930), Slit-Lamp Microscopy of the Living Eye. 2nd Edition. Page 292. London.

ROUGIER, A. and CHRISTIAN, J. (1935), Brit. Med. Jl. April 18, 1936. Page 65. (Abstract).

SOUTER, W. C. (1933), Trans. Ophthal. Soc. U.K. Vol. LIII. Page 73.

CATARACTS — SENILE.

The fact that inherited defects can appear in any decade of life is beyond dispute. That they may manifest themselves in the sixth and seventh decades has been clearly shown in such eye diseases as glaucoma, angioid streaks and pigmentary macula degeneration. To these we may now add a fourth, namely senile cataracts. But, like the three previously mentioned diseases, the pedigrees of senile cataract take a whole life-time to acquire, for the disease usually does not appear in each generation till about the age of seventy. Nettleship (1906) has pointed out that there was an element of anticipation in each succeeding generation, but that is not the case in every pedigree. Also in these senile inherited diseases, many members of each pedigree die before their three score years and ten, which adds to the difficulty of the tedious task of recording.

It might be well before discussing the pedigrees to reiterate Nettleship's six rules pertaining to the occurrence of inherited cataracts.

(1) Descent is direct. No generations are skipped.

(2) If all children in one family have cataract, the liability of the next generation is increased.

(3) Transmission from like sex to like sex is most common.

(4) Cataract tends to appear earlier in life in succeeding generations.

(5) The age of onset is approximately the same in persons of the same generation.

(6) The families with cataract are not affected as to fertility, health and longevity.

COMMENTS ON PEDIGREES.

Pedigree No. 87.—That at least four members of generation II had senile cataracts is fairly certain, although I only have the propositus' statement of this. But, as she is an intelligent woman, I think we can take her statement as a reliable estimate.

III/2 Male, aged 68 years. Had a right senile cataract extracted by me, and has advanced post cortical lens changes in his left eye.

III/5 Male, aged 64 years. Is a patient of a colleague. He reports bilateral senile cataracts of an advanced nature, the left being ready for extraction.

III/7 Female, aged 52 years. Had bilateral senile cataracts extracted by me at this age, and after eight years still has 6/4 vision in each eye with aphakic correction. She is the propositus.

III/19 Male, aged 58, bilateral senile cataracts. Right cataract extracted, with corrected vision of 6/4 + J.1.

Pedigree No. 163.

III/4 Female, aged 67 years, who is the propositus, had bilateral senile cataracts.

III/6 Female. Had, at the age of 63 years, a right senile cataract extracted by me. A left mature senile cataract remained untouched.

The propositus, who is reliable, is certain that both her uncle, II/1, and her cousin, III/1, had senile cataracts, too.

Pedigree No. 217.

II/3 Male. Had a right senile cataract extracted at the age of 78 years. His left cataract is, as yet, immature.

II/4 Male. Had bilateral immature cataracts at the age of 77.

II/5 Male. Had left senile cataract extracted at the age of 72 years. His right cataract is still immature.

III/1 Male, age unknown. Has senile cataract. This patient has been examined by an ophthalmic surgeon.

III/2 Female, aged 60. Bilateral incipient cataracts.

REFERENCES.

BLACKER, C. P. (1934), The Chances of Morbid Inheritance, London.
BELLOWS, J. G., & CHINN. H. (1941), Arch. of Ophthal. Vol. 26. Page 1,066.
NETTLESHIP, E. (1906), Royal London Ophthalmic Hospital Reports. Vol. XVI. Page 389.
VINSONHALER, F., & COSGROVE, R. W. (1936), Arch. of Ophthal. Vol. XV. Page 222.

COLOBOMA OF THE IRIS AND CHOROID.

Coloboma of the Iris and Choroid are fairly common but seldom recorded abnormalities. It is an established fact that they may be unilateral or bilateral. Further, both iris and choroid may simultaneously be involved, but sometimes only the iris or the choroid are affected, and this fact is, as yet, unexplainable. It has also been noted that the coloboma does not always occur below at the site of the foetal cleft, but if the iris and choroid are both involved, then the defect always lies downward, or downward and outward.

CONCURRENT DEFECTS.

Julia Bell (1932) in the Treasury of Human Inheritance, Volume II, part 5, page 476, exhibits a table of defects observed in association with coloboma of the iris in the same individual. It will be noticed how frequent is its association with coloboma of the choroid and cataract. In my own pedigree (No. 142), the mother (III/4) has a coloboma of the choroid in one eye, and the eldest daughter (IV/1) has coloboma of the iris and choroid in both eyes with cataracts, while the youngest child (IV/5) has coloboma of the iris and choroid in one eye with bilateral cataracts. The mother (III/4) also had an ocular torticollis with an overactive inferior oblique on the left side, but I do not think this has any relation to her coloboma of the choroid.

INHERITANCE.

Macklin (1927) says the inheritance is dominant, but Julia Bell (1932) does not commit herself, regarding the mode of inheritance. In two of her reported pedigrees consanguinity appears to have re-introduced the disease, for in her pedigree No. 1,156, two first cousins married and one of their grand-daughters had coloboma of the iris, while in pedigree No. 1,173 there was again consanguinity in an affected sibship, with the result that a daughter had coloboma of the iris. In my pedigree No. 142, it will be noted that the grandparents of III/4 on her father's side were first cousins. It would be very interesting to examine II/1 and II/2, to see if either has an unsuspected coloboma of the choroid.

SEX INCIDENCE.

Julia Bell (1932) points out that in the 111 individuals which she was able to collect, there was an equal number of males and females, exactly as she found in her survey of aniridia. My Tasmanian cases are far too few to dogmatise on this point.

COMMENTS ON PEDIGREE No. 142.

This pedigree is of extreme interest in showing just how supposition may mislead one. Before the pedigree was worked out, I was of the opinion that, as IV/5 had a coloboma of the iris and webbed toes, and as three of his paternal relatives had webbed toes also, the coloboma of the iris of IV/1 and IV/5 were possibly inherited from the paternal side of the family, and that the webbed toes and coloboma were concurrent congenital defects. As I have pointed out in Table IV on Hereditary Digital Abnormalities in relation to Eye Effects, Syndactyly often accompanies the congenital defect of aniridia, but I could not find any mention in the literature of the relationship of coloboma of the iris and choroid to Syndactyly except that Sorsby (1935) connects congenital coloboma of the macula with skeletal abnormalities including Syndactyly. It was only recently, when III/4 was examined, that I found she had quite unsuspected unilateral coloboma of the choroid. Therefore, my assumption that the syndactyly and coloboma of the iris and choroid were associated defects was obviously wrong. I shall now report my three cases in full:—

III/4 Female, aged 45 years. Has ocular torticollis with overaction of the inferior oblique, and a small coloboma of the choroid at 6 o'clock about 4 discs diameter from the ora serrata and three disc diameters in diameter.

Right vision with correction = 6/4.
Left vision with correction = 6/4.

IV/1 Female, aged 25 years. This patient has a coloboma of the iris and large coloboma of the choroid below in both eyes. She further exhibits an anterior polar cataract in the left eye, lens opacities at the site of both coloboma of the irides and central embryonic cataracts in both lenses as well.

Right vision with correction = 6/18.
Left vision with correction = 6/24.

IV/5 Male, aged 12 years. Has left coloboma of the iris below and a large coloboma of the choroid in the same eye. Persistent hyaloid arteries in both eyes and embryonic cataracts in both lenses.

Right vision with correction = 6/4.
Left vision with correction = 6/18.

He also has webbed toes on the left foot, the first and second toes being adherent.

REFERENCES.

BELL, JULIA (1932), Treasury of Human Inheritance. Vol. II. Page 472.
ESKELUND, V. (1938), Structural Variations of the Human Iris and Their Heredity. London.
MACKLIN, M. T. (1935), Can. Med. Assn. Jl. Vol. XVII. Page 937.
SORSBY, A. (1935), Brit. Jl. of Ophthal. Vol. XIX. Page 65.
WARDALE, J. D. (1930), Trans. Ophthal. Soc. of U.K. Vol. L. Page 613.

DACRYOCYSTITIS.

The hereditary nature of obstruction of the lacrimal apparatus with or without ultimate infection, has been debated in the medical literature for many years. Traquair (1940) was able to record a family tree of 17 persons, 13 of whom were afflicted with dacryocystitis. For the past 10 years I have been endeavouring to collect pedigrees of dacryocystitis, but I have seldom been able to detect more than two affected members in each tree. Pedigree 144 is a sibship of four children, the second, third and fourth of whom had dacryocystitis; but this sibship is indeed an exception.

Of my 250 cases of lacrimal apparatus obstruction or infection (see Table XIV), I have been able to find 15 inherited cases, which gives a six per cent. incidence as against 11 per cent. found by Traquair (1940). Dr. F. Phillips has been good enough to allow me to examine his records and I find 101 cases of lacrimal obstruction or infection, including seven cases with an hereditary history, giving a seven per cent. incidence. This figure closely resembles mine. He also has two sisters under six months, with lacrimal obstruction.

SEX INCIDENCE.

I thought it would be wise to tabulate my analysis of 250 cases, and I do so in Table XIV. It will be seen here that in the 28 cases under the age of 12 months, there were 14 males and 14 females affected, while in those over 12 months there were 52 males and 170 females affected. Traquair (1940) pointed out that in babies, the males approximately equalled the females, while in later life, there were four females to one male involved. My ratio for over 12 months was three females to one male.

If we now take the total 15 inherited cases, we find 10 females to five males, but under 12 months there were six cases, four of which were males. In the over 12 months group, there were nine cases, eight of which were females. Of course, these figures are too small for any conclusion, but if we analyse Dr. F. Phillips' seven cases, we find six females and one male; one male and two females being under 12 months.

ASSOCIATED DEFECTS.

The relation of Rubella in the pregnant mother to congenital cataract in the offspring has recently been stressed by Gregg (1941 and 1945) and Swan (1943). But the relation of infantile dacryocystitis to Rubella in the pregnant mother has not been so carefully worked out. In pedigree 176, while III/1 and the aunt (III/3) both had chronic obstruction of the nasolacrimal duct, the baby (IV/4), born of a mother who contracted rubella in the first month of pregnancy, had bilateral cataracts and chronic left dacryocystitis. Gregg and others (1945), in a report on "The Occurrence of Congenital Defects in Children following maternal Rubella in Pregnancy," mention that nasolacrimal duct stenosis may occur, but they do not consider this of any significance. Nevertheless, it is a point to be kept in mind if a further epidemic ensues.

COMMENTS ON PEDIGREES.

Pedigree No. 71.

II/2 Male, aged 67 years.
1932—History of watery eyes for years — both sacs syringed and freely patent. Puncta dilated.
1936—Eyes still watery, but both sacs still freely patent.
1938—Condition unchanged.

II/3 Female, aged 61 years.
1931—No history of epiphora.
1933—No history of epiphora.
1935—No history of epiphora.
1937—Right eye watering for 3 days. Sacs syringed.
Right nasolacrimal duct completely blocked.
Left nasolacrimal duct partially patent.

IV/1 Male, aged 12 weeks.
1934—Left eye discharging and watery since birth. Under general anaesthesia left nasolacrimal duct found to be blocked. Probes passed with immediate cure.

Pedigree No. 107.

I/1 Female, aged 58 years. At age of 39 years had right lacrimal sac excised on account of discharge from that eye. Since then right eye comfortable. Reported for refraction.

III/1 Male aged 14 weeks. Left eye watery since birth and discharging. Treated with Guttae Mercurochrome 1% with spontaneous recovery.

Pedigree No. 135.

II/3 Male, aged 71 years. Eyes watery for two years. Has had left canaliculus slit. Constriction in left lower canaliculus at distal end, dilated with restoration of patency of left lacrimal apparatus.

IV/10 Female, aged 11 weeks. Left eye watering and discharging since birth. Left sac infected due to block in nasolacrimal duct. Left sac probed and syringed under general anaesthesia. Cure immediate.

Pedigree No. 143.

I/2 Female, aged 38 years. Left eye watering for seven years. Acute left dacryocystitis followed by excision of lacrimal sac.

II/1 Female, aged 17 years.
1933—Left eye discharging for 12 months. Right sac freely patent. Left sac partially patent with some mucous regurgitation. Condition treated by syringing.
1939—Acute left dacryocystitis.

Pedigree No. 144. This is a sibship of four children. The second and third had one eye watering and discharging for six months when it resolved spontaneously.

II/4 Female, aged 8 months. Right tear sac blocked for seven months. Right sac probed and syringed twice, under general anaesthesia, with good results. Stricture was in nasolacrimal duct.

Pedigree No. 145.

II/3 Dead, but propositus assures me that this patient had a unilateral epiphora.

III/4 Female, aged 65 years.
1937—Left dacryocystectomy for chronic dacryocystitis.
1945—Right dacryocystectomy for mucocele.

Pedigree No. 150.

I/2 Propositus assures me that her maternal grandmother had unilateral epiphora.

IV/1 Male, aged 4 months. Left eye watering and discharging since birth. Under general anaesthesia left sac found infected and left nasolacrimal duct occluded. Probed and syringed with immediate cure.

Pedigree No. 176.

III/1 Female, aged 45 years. Both lacrimal sacs excised for chronic dacryocystitis.

III/3 Female, aged 42 years. Right eye watering for two months. Sacs syringed and partial nasolacrimal duct obstruction found. Cured by syringing.

IV/4 Male, aged 3 days. Mother had Rubella during first month of pregnancy. Child has mature bilateral cataracts and left chronic dacryocystitis.

REFERENCES.

GREGG, N. M. (1941), Trans. of Ophthal., Society of Aust. Vol. III. Page 35.

GREGG, N. M., *et al* (1945). Med. Journal of Aust., July 28th. Page 122.

PETERS, R. (1923), Kiln, Monatsbl. F. Augenh. Vol. 71. Page 726.

POLJACK, B, and POPOUA, F. (Abstract) (1929), Vrac. Graz. No. 17/18. Page 2,291.

SCHEICHLER, L. (Abstract) (1925).

SWAN, C., *et al* (1943), Med. Journal of Aust. Sept. 11th. Page 201.

TRAQUAIR (1940), Trans. of Ophthal., Society of United Kingdom. Vol. LX. Page 127.

TABLE XIV.

LACRIMAL OBSTRUCTION

	Stenosis of Puncta or Canaliculii	Obstruction of Naso-lacrimal Duct.	Acute Dacryo-cystitis.	Chronic Dacryo-cystitis.	TOTALS
1. Total No. of Cases ..	48	74	23	105	250
2. Cases under 12 months	3	3	2	20	28
3. Cases over 12 months	45	71	21	85	222
4. Sex Ratio in (1)—					
Male	15	18	29	4	66
Female	33	56	19	76	184
5. Sex Ratio in (2)—					
Male	2	2	1	9	14
Female	1	1	1	11	14
6. Sex Ratio in (3)—					
Male	13	16	3	20	52
Female	32	55	18	65	170
7. Inherited Cases in (1)	2	3	2	8	15
8. Inherited Cases in (2)	nil	1	nil	5	6
9. Inherited Cases in (3)	2	2	2	3	9
10. Sex Ratio in (7)—					
Male	1	nil	nil	4	5
Female	1	3	2	4	10
11. Sex Ratio in (8)—					
Male	nil	nil	nil	4	4
Female	nil	1	nil	1	2
12. Sex Ratio in (9)—					
Male	1	nil	nil	nil	1
Female	1	2	2	3	8

Percentages of Inherited Cases = 6%.

GLAUCOMA — CHRONIC IN ADULTS

Increased intra-ocular tension both in its acute and chronic form is relatively rare in Tasmania, and especially in its acute form. When chronic glaucoma does occur in the residents, it is surprising to find that a very marked proportion are not native born Australians. Out of 9,980 private case records during the past 14½ years (i.e. January, 1931, to May, 1945), I can find the following cases of increased tension:—

Acute Glaucoma	14
Chronic Secondary Glaucoma	64
Chronic Primary Glaucoma	113

If an analysis is made of my 9,980 cases, then the percentage of chronic primary glaucoma amongst these cases is 1.13 per cent., which compares favourably with the statistics quoted by Julia Bell (1932) which ranged from 0.4 per cent. in Germany to 2.73 per cent. in Italy; and estimates from almost a million European cases make an average percentage of 0.97 per cent.

Of these 113 cases of chronic primary glaucoma, 64 were native born Australians, 23 were born in the British Isles, and the birthplaces of 26 are unknown, which, in percentages, is 56.63 per cent., 20.45 per cent. and 23 per cent., respectively.

Now, according to the 1933 census, 95 per cent. of the population of Tasmania were born within the Commonwealth, and 5 per cent. outside the Commonwealth, so that one would have expected to find the percentage of glaucoma identical. Presuming 5 per cent. of the population are not native born, then 499 of the above 9,980 cases should have been born outside Australia, and 9,481 of the cases born within Australia. Of these, approximately 499 cases, no less than 23 have glaucoma — a surprisingly high percentage of 4.61, while of the 9,481 Australian-born cases, only 64 have glaucoma—a surprisingly low percentage of approximately 0.681. I am unable to account for these figures, and intend reviewing them at a later date, in order to confirm the startling disproportions. If we conclude that "worry" is a predisposing cause of glaucoma, then a faint ray of truth may be revealed. The recent emigrants have undoubtedly been subjected to far more anxiety than their Tasmanian brothers and sisters, who, by reason of the prolonged isolation of Tasmania and the isolated position of their residences, were prohibited reasonable contact with the outside world until 1925. Since the advent of radio in 1925, and the world-wide telephone communication in 1936, this isolation, to a great extent, has been banished, and it may be interesting to see if the glaucoma figures will rise in the next half of this century (see Table XV).

HEREDITARY INFLUENCES.

Of my 113 cases of chronic primary glaucoma there are only 14 hereditary cases, the remainder of the cases in my nine pedigrees being obtained from outside sources, so that amongst my own patients the hereditary factor is extremely low. Bell (1932) complains of the paucity of reliable records of hereditary glaucoma, and finds only three pedigrees reported by Nettleship, and one by Usher, and was able to collect only 68 reported pedigrees in the literature.

SEX INCIDENCE OF HEREDITARY GLAUCOMA.

Taking the nine hereditary pedigrees I find in a total of 14 cases examined by me, there are eight males (57.2 per cent.) and six females (42.8 per cent.) in all. Whereas Bell's figures (1932) are 99 males to 97 females, Zorab (1932), on the other hand, in surveying six pedigrees, found 25 males and 10 females affected; while Frank Kamenetzki (1925) recorded a pedigree in which every affected member was a male. The total hereditary cases of glaucoma reported in Tasmania are 28, of which 17 (60.7 per cent.) are males and 11 (39.3 per cent.) are females. This again confirms the higher male incidence.

So that there appears to be no doubt that males are more prone to be affected than females, when the disease is hereditary. Conversely, of the 99 non-hereditary cases seen by me since 1930, 54 were females (54.5 per cent.), and 45 males (45.5

per cent.), which gives a greater predominance of females, and conforms to Rimpler's statistics — quoted by Frank Kamenetzki — of 2,021 cases of glaucoma in which 1,150 (56.9 per cent.) were females, and 871 (43.1 per cent.) were males.

AGE OF ONSET.

I can give no useful information as to this. Pedigree 11, which was collected long before I anticipated writing this paper, might have helped considerably, but these details are wanting, unfortunately, and quite possibly the propositus would not have been able to supply them. Taking the age of onset of the 14 cases which I have examined, it varies from 32 to 88, and appears to show some anticipation as indicated by Lawford (1905).

MODE OF TRANSMISSION.

Although Macklin (1927) considers the transmission of hereditary glaucoma dominant, yet my few pedigrees prove that this is not always so by any means, and even so great an authority as Nettleship did not feel disposed to commit himself on the mode of transmission.

Frank Kamenetzki (1925) reports one pedigree in which all the afflicted members are males, and the mode of transmission was a recessive sex-linked one.

CONCURRENT ANOMALIES.

In the very interesting pedigree of Frank Kamenetzki (1925) the individuals affected with glaucoma had an accompanying atrophy of the iris stroma. Bell (1932) states, "So far as I can judge, cases of hereditary glaucoma appear in otherwise healthy stock, and very few examples of associated hereditary diseases or anomalies are found amongst the affected individuals." My few cases exhibit no peculiar anomalies.

TREATMENT.

There appears to be little variation from the orthodox treatment in the hereditary cases. Frank Kamenetzki (1925) found iridectomies difficult in his cases owing to the iris atrophy, while James (1927) reports satisfactory results from trephining. Stokes (1940) considers any fistulizing operation gives good results if performed early in the disease. Personally, I have found no indication in my few cases to depart from routine procedures.

COMMENTS ON PEDIGREES.

Pedigree No. 11. These are two separate pedigrees but they have now been combined and will be taken as one. The reason for the decision is plain. There has been intermarriage between the two families and as the result of this marriage there are four female siblings.

III/11 Male, aged 85 years, unable to read. Left absolute glaucoma with early rise in tension in right eye. Although there are advanced anterior cortical changes in both lenses, yet the fundi can be clearly seen. Both discs are grey but not deeply cupped, and it is possible that this is a case of cavernous atrophy of the discs. The loss of right central vision bears this out.

Right vision with correction = 6/36.
Left Vision — Perception of light with loss of projection.
Fields of vision: Right. Normal to 1° white.
Left. Small temporal island to 4° white.

III/16 Female, aged 70 years. Sight failing for six years. Four years ago right eye operated on for glaucoma. Now has right absolute glaucoma with perception of light only. Left eye has gross loss of field, but corrects to 6/24.

III/18 Male, aged 66 years. Operated on for bilateral glaucoma by iridectomy at age of 53 years. Bilateral absolute glaucoma resulted.

Pedigree No. 13.

III/3 Male, aged 88 years, was under treatment by me for cavernous atrophy of the optic discs, and raised tension — central vision has been lost in one eye, but the fields of vision are normal to 1° white.

IV/27 Female, had advanced glaucoma right and left at the age of 37 years. Four other members of the family examined are free from this defect, but all four of these exhibited gross astigmatism in both eyes.

Pedigree No. 14. There are three sibs. II/4 is dead, but his sisters (II/1 and II/3), at the age of 89 and 86, respectively, have advanced chronic glaucoma in both eyes, and they did not report for treatment until aged 86 and 84, respectively. One significant fact about these old ladies is that when each reported (not under miotics), the tension in both eyes was 20 mm. Hg. (Schiotz), i.e., the four eyes had similar normal tension, and yet the four fields were greatly, and typically, constricted; and the optic discs grossly cupped.

Pedigree No. 77.

II/3 Male, aged 78 years. Bilateral early glaucoma — both eyes trephined with excellent results.

III/1 Male, aged 65 years. Left chronic glaucoma just prior to death.

Pedigree No. 91.

III/3 Female, aged 69 years. Myopic Astigmatism.
Right Vision with correction = 6/5.
Left Vision with correction = 6/5.
Fundi — Moderate Retinal Arterio Sclerosis.

III/5 Female, aged 66 years. Hypermetropic Astigmatism and Esophoria.
Right Vision with correction = 6/9.
Left Vision with correction = 6/5.
Fundi — Normal (dilated).
Right and Left Lens Sclerosis.

III/7 Female, aged 73 years. Myopic Astigmatism and Glaucoma.
Right Vision with correction = 6/4.
Left Vision with correction = 6/4.
Fundi — Right Vitreous opacities ++
Early cupping of the optic disc.
One small cyst on disc.
Tay's Choroiditis.
Left—Vitreous opacities ++
Optic disc normal.
Fields — Right One degree white shows partial loss of upper nasal field and early loss of lower nasal field.
Left—One degree white, normal.

IV/1 Male, aged 65 years. Myopic Astigmatism and Chronic Glaucoma.
Right Vision with correction = 6/3.
Left Vision with correction = 6/3.
Fundi — Early cupping of both optic discs.
Fields — Normal to one degree white.
Scotomotry baring of the left blind spot to 5 mm. white at 2 metres.
Operation; right and left eyes decompressed satisfactorily.

IV/2 Female, aged 56 years. Hypermetropic Astigmatism.
Right Vision with correction = 6/3.
Left Vision with correction = 6/3.
Fundi are normal (dilated).

IV/3 Female, aged 56 years. Myopic Astigmatism and Exophoria.
Right Vision with correction = 6/3.
Left Vision with correction = 6/3.
Fundi are normal (dilated).

IV/4 Female, aged 50 years. Myopic Astigmatism, and vitreous opacities.
1931—Right Vision with correction = 6/4.
Left Vision with correction = 6/4.
Fundi—Right—Normal.
Left—Optic disc appears pathologically cupped. Fields of vision to 1° white normal.
1938—Unchanged. Fields and vision normal.
1945—Unchanged. Fields and vision normal.

IV/5 Male, aged 43 years. Mixed Astigmatism and Esophoria.
Right Vision with correction = 6/3.
Left Vision with correction = 6/3.
Fundi are normal (dilated).

IV/6 Female, aged 34 years. Hypermetropic Astigmatism.
Right Vision with correction = 6/4.
Left Vision with correction = 6/4.
Fundi are normal (dilated).

IV/7 Male, aged 42 years. Hypermetropic Astigmatism and Esophoria.
Right Vision with correction = 6/4.
Left Vision with correction = 6/4.
Fundi are normal (dilated).

IV/8 Female, aged 36 years. Mixed Astigmatism.
Right Vision with correction = 6/4.
Left Vision with correction = 6/4.
Fundi are normal (dilated).

IV/9 Male, aged 40 years. Hypermetropic Astigmatism.
Right Vision with correction = 6/4.
Left Vision with correction = 6/4.
Fundi are normal (dilated).

IV/10 Female, aged 37 years. Mixed Astigmatism.
Right Vision with correction = 6/4.
Fundi are normal (dilated). Left vision with correction = 6/4.

IV/11 Female, aged 42 years. Hypermetropic Astigmatism.
Right Vision with correction = 6/4.
Left Vision with correction = 6/4.
Fundi are normal (dilated).

IV/12 Male, aged 43 years. Hypermetropic Astigmatism.
Right Vision with correction = 6/4.
Left Vision with correction = 6/4.
Fundi are normal (dilated).

IV/13 Male, aged 40 years. Refractive error unknown.

IV/14 Female, aged 47 years. Myopic Astigmatism.
Right Vision with correction = 6/4.
Left Vision with correction = 6/4.
Fundi are normal (dilated).

IV/15 Male, aged 36 years. Hypermetropia.
Right Vision with correction = 6/4.
Left Vision with correction = 6/4.
Fundi are normal (dilated).

IV/16 Female, aged 39 years. Mixed Astigmatism.
Right Vision with correction = 6/4.
Left Vision with correction = 6/4.
Fundi are normal (dilated).

V/3 Female, aged 16 years. Myopic Astigmatism.
Right Vision with correction = 6/4.
Left Vision with correction = 6/4.
Fundi are normal (dilated).

V/4 Male, aged 12 years.
Refractive error unknown.

V/5 Female, aged 11 years. Hypermetropia.
Right Vision with correction = 6/5.
Left Vision with correction = 6/4.
Fundi are normal (dilated).

V/6 Male, aged 15 years. Hypermetropic Astigmatism.
Right Vision with correction = 6/4.
Left Vision with correction = 6/4.
Fundi are normal (dilated).

V/8 Female, aged 16 years. Hypermetropic Astigmatism and Esophoria.
Right Vision with correction = 6/5.
Left Vision with correction = 6/5.
Fundi are normal (dilated).

V/10 Male, aged 8 years. Hypermetropic Astigmatism and Esophoria.
Right Vision with correction = 6/5.
Left Vision with correction = 6/5.
Fundi are normal (dilated).

V/20 Male, aged 11 years. Myopic Astigmatism.
Right Vision with correction = 6/4.
Left Vision with correction = 6/4.
Fundi are normal (dilated).

V/25 Male, aged 3 years and 6 months. Esotropia.
No refractive error.
Fundi are normal (dilated).

TABLE XV.

INCIDENCE OF HEREDITARY GLAUCOMA IN TASMANIA FROM JANUARY 1st, 1930, TO MAY 21st, 1945.

NUMBER OF CASES EXAMINED = 9,980.

CHRONIC PRIMARY GLAUCOMA—		113	= 1.1	per cent.
Born in Australia	64		= 56.63	„ „
Born outside Australia	23		= 20.45	„ „
Birth-place unknown	26		= 23	„ „
Males	51		= 45.13	„ „
Females	62		= 54.87	„ „
Hereditary Cases amongst above—		14	= 12.39	„ „
Males	8		= 57.14	„ „
Females	6		= 42.86	„ „
Non-hereditary Cases amongst above—		99	= 87.61	„ „
Males	45		= 46.46	„ „
Females	54		= 53.54	„ „
No. of hereditary reported in Tasmania—		28		
Males	17		= 60.7	„ „
Females	11		= 39.3	„ „
Percentage of native-born Australians in population			= 95	„ „
Percentage of non-native born Australians in population			= 5	„ „
Approximate number of my 9,980 cases born in Australia			= 9,481	
Approximate number of my 9,980 cases born outside Australia			= 499	
Percentage of glaucoma amongst 9,481 presumed Australian-born cases			= 0.68	„ „
Percentage of glaucoma amongst 499 assumed non-Australian born cases			= 4.61	„ „

Pedigree No. 103. This pedigree has been given me by Dr. J. L. R. Carter, of Launceston. It is of male first cousins (III/1 and III/3) who developed glaucoma in their seventh decade. Both cases have had decompression operations.

Pedigree No. 108.

III/1 Male, age unknown, examined by Dr. J. L. R. Carter, of Launceston, and has absolute glaucoma in both eyes.

III/7 Male, aged 63 years. Right eye operated on for glaucoma at age of 59 years. Four years later developed left glaucoma for which operation was performed.

IV/1 Female, aged 38 years. Has had operation for left chronic glaucoma six years previously. Now has bilateral raised tension with partial left superior temporal venous thrombosis.

Pedigree No. 179.

II/1 Female, aged 55 years. Has left amblyopia ex anopsia and right chronic glaucoma with marked right field loss.

II/3 Male, aged 61 years. Left Herpes Zoster Ophthalmicus complicated by acute glaucoma in left eye. This responded to medical treatment.

Pedigree No. 203.

I/2 Female, aged 90 years. Had central senile choroiditis but not glaucoma.

II/7 Male, aged 68 years. Chronic glaucoma in both eyes with left field loss.

II/8 Female, aged 53 years. Wife of II/7. Acute bilateral glaucoma.

II/9 Male, aged 58 years. Chronic glaucoma both eyes with right field loss. Both eyes decompressed.

II/11 Male, aged 56 years. Raised tension both eyes without perimetric or scotometric loss.

REFERENCES.

ACKERMAN, W.G., and ALLEN, T. D. (1942), Illinois Med. Jl. Vol. 82. Page 295.
ALLEN, T. D., and ACKERMAN, W. G. (1942), Arch. of Ophth. Vol. 27. Page 139.
ANDERSON, J. R. (1935), Contribution to the Study of Congenital Glaucoma. Melbourne.
ANDERSON, J. R. (1939), Hydrophthalmia. Cambridge.
BARKAN, O. (1942), Am. Jour. Ophthal. Vol. 25. Page 552.
BELL, J. (1932), Treasury of Human Inheritance. Vol. II. Part V. Page 448. Cambridge.
BIRO, I. (1939), Ophthalmologica. Vol. 98. Page 43.
BRIGGS, A. (1939), Brit. Jour. of Ophthal. Vol. XXIII. Page 649.
COMMONWEALTH BUREAU OF CENSUS AND STATISTICS (1936), Private Communication.
COUNSELL, W. D. (1936), Personal Communication.
ELLIOT, R. H. (1922), A Treatise on Glaucoma. 2nd Edition. London.
FRANK KAMENETZKI, S. G. (1925), Klin—Monatsbl. f. Augenheilk. Vol. LXXIV. Page 133.
JAMES, R. R. (1927), Brit. Jl. Ophthal. Vol. XI. Page 438.
LAWFORD, J. B. (1905), Royal London Ophth. Hosp. Reports. Vol. 17. Page 57.
MACKLIN, M. J. (1927), Canad. Med. Assoc. Jl. Vol. XVII. Page 422.
STOKES, W. H. (1940), Arch. of Ophthal. Vol. 24. Page 885.
ZORAB, A. (1932), Trans. Ophthal. Soc., U.K. Vol. LII. Page 446.

KERATOCONUS.

Although Keratoconus, like Retinitis Pigmentosa, is an hereditary disease, yet many sporadic cases, in which there appears to be no hereditary influence whatsoever, are found in routine work. In 9,980 private cases (mentioned under glaucoma and pterygium), I find 43 cases in all of keratoconus—making a case incidence of 0.43 per cent. Of these 43 cases, eight can definitely be said to be hereditary (pedigrees 15 and 16), while three others are strongly familial in incidence (pedigree 222). Of these 43 cases, 36 came from Southern Tasmania where the proportion of city and country dwellers is equal. Yet of these 36 patients, 16 came from the city and 21 from the country.

ASSOCIATED DEFECTS.

Fleischer's Ring. This was present in only one of the 75 eyes affected, and was not accompanied by opacity of the cornea.

Apical Corneal Opacities. These were present in 12 of the 75 affected eyes, and in three eyes they required surgical treatment.

MODE OF TRANSMISSION.

According to Franceschetti (1930) keratoconus is transmitted both as a dominant and recessive. In my two pedigrees (15 and 16), we must undoubtedly presume that the mode of transmission was recessive.

SEX INCIDENCE.

Of these 43 patients, 12 were males and 31 were females, while the two hereditary pedigrees (15 and 16) show only five females and three males affected. As I am unable to obtain a copy of Jaonsch and Stahl's articles, I cannot compare and contrast these figures with statistics from other countries.

CONCURRENT DISEASES.

Mongolian Idiocy. Two of these 43 patients were Mongolian Idiots, with advanced conical cornea in three eyes, which needed and received operative interference.

Thyroid Dysfunction. Of the 43 patients, 18 were generally examined. Eleven showed no trace of endocrine dysfunction, three were subthyroid, and four hyperthyroid. Knapp (1924) found low basal metabolic rates in two out of six patients.

TREATMENT.

Contact Lenses. These were prescribed in one case and four other cases had their visual acuity tested with them. In all five cases the result was highly satisfactory. But a problem, which needs further investigation and discussion, is the possibility of retarding the progress of the corneal bulging by ordering contact lenses early in the disease.

Miotics and Hormone Therapy. Although I have given both these prolonged trial, I frankly admit that I have seen very little benefit derived from their use. In one case I thought "Hormotone" retarded the progress.

Vitamin D Therapy. This was tried in six cases, and I think at the present juncture is the most scientific of all imperical therapy. This assertion is supported by Knapp (1939) on clinical grounds.

Surgical Interference. This was necessary on three eyes, and the electric cautery was applied to the apex of the cones until perforation resulted, after the manner of Knapp (1929). In one case the anterior chamber took three weeks to reform, but the ultimate results in all were satisfactory. Surgical interference should be reserved for the most advanced cases when the central corneal nebula is marked and the apex of the cone acting as a foreign body between the lid margins.

COMMENTS ON PEDIGREES.

Pedigree No. 15 — In this pedigree we have a record of 7 affected members through three generations. Others have been examined and treated chiefly for high myopic astigmatism.

II/1 Male, aged 30 years. Right and left keratoconus with right vision correcting to 6/12 and left vision to 6/18.

III/6 Male, aged 30 years. Early bilateral keratoconus. Right vision with correction = 6/12. Left vision with correction = 6/12.

III/8 Female, aged 34 years. Gross keratoconus right and left without corneal scarring. Right and left vision improves to 6/12 with high oblique concave cylinders. She is also the propositus.

III/12 Female, aged 40 years. Gross bilateral keratoconus and myopic astigmatism. Right and left vision correcting to 6/18.

III/14 Male, aged 24 years. Early bilateral keratoconus and myopic astigmatism. Right and left vision correcting to 6/12.

III/17 Male, aged 19 years. Moderate bilateral keratoconus. Right vision correcting to 6/9 and left vision to 6/5.

IV/5 Female, aged 10 years. Early right keratoconus, correcting to 6/9.

Pedigree No. 16.

III/7 Female, aged 22 years. Bilateral keratoconus with right linear corneal opacities. Right vision with correction stood at 6/60, but with a contact lens improved to 6/24. Left vision with correction reached 6/12.

III/17 Female, aged 20 years. Marked bilateral keratoconus with right vision correcting to 6/18 and left vision to 6/12.

III/21 Male, aged 15½ years. Early right and marked left keratoconus. Right vision corrects to 6/12 and left vision to 6/9.

It will be noted that while III/17 and III/21 are both cousins of III/7, they are not related to one another.

Pedigree No. 222.—This is merely familial and not inherited keratoconus, but involves three children in a sibship of four.

II/1 Male, aged 25 years. Patient of Dr. F. Phillips. Bilateral keratoconus with right vision correcting to 6/12 and left vision correcting to 6/9.

II/3 Male, aged 24 years. Patient of Dr. F. Phillips. Bilateral and progressive keratoconus correcting to 6/24 in each eye. Contact lenses are being fitted.

II/6 Male, aged 19 years. Bilateral keratoconus with right vision correcting to 6/9 and left vision to 6/5.

REFERENCES.

FRANCESCHETTI, B. (1930), Kurze, Hand. D. Ophthal. Page 731. Berlin.
HOMEWOOD, J. (1935), Personal communication.
KNAPP, A. (1929), Arch. Ophthal. Vol. II. Page 658.
KNAPP, A. (1939), Am. Jl. of Ophthal. Vol. 22. Page 289.
KOBY, F. E. (1930), Slip Lamp Microscopy of the Living Eye. London.
SANDER, P. (1931), Brit. Jl. Ophthal. Vol. XV. Page 23.

MACULAR DEGENERATION — SENILE PIGMENTARY.

That macular degeneration of one type or another may occur at any time from infancy to senility, has been pointed out clearly by numerous authors for many years, but more recently by Sorsby (1934) and Waardenburg (1936). Sorsby (1934) in his paper divides macular degeneration into four types:—

(1) Infantile.
(2) Adult.
(3) Pre-senile.
(4) Senile.

but the first three are not pertinent to my pedigree (118) and so in this section of my paper, I am only dealing with type (4), namely senile macular degeneration of a pigmentary type.

As as senile disease, pigmentary macular degeneration is rare, and in a 14 year survey in Tasmania in which I examined a total of 9,980 cases, I found 25 cases of senile macular degeneration. Watching some of the cases over a period of years, they appear to be progressive, and their progress is after this fashion. There is often in the pre-senile stage, a mild pigmentary or cystic degeneration of the periphery of the retina accompanied by a few Tay's spots about the macula. Sometimes a small melanoma, about half a disc diameter and situated two or three discs diameter from the optic disc is present. Later pigmentary and atrophic changes commence at the macula, with a lowering of central vision, and this is usually accompanied by retinal arteriosclerosis which ultimately produces scattered haemorrhages in the fundi particularly in the peri-macular area.

In pedigree (118) which I am about to report, no member developed the final stage of this degeneration, namely pigmentary macular degeneration with haemorrhages of the fundi, but as I said previously, I have seen such a stage in Tasmania, Behr (1921) reports a male, aged 65 years, with exactly the same final condition.

In view of the above statements, I am rather reluctant to entitle this section of my monograph Senile Pigmentary Retinal Degeneration because the process of degeneration is much wider and more prolonged. The process begins as a pre-senile pigmentary change sometimes of a true melanotic type accompanied by excrescences from the elastic lamina. Only as a final stage does the macula actually degenerate and this is usually accompanied by retinal vascular changes. Furthermore, in the accompanying paragraph on defects, I will draw attention to the 8th nerve deafness in this pedigree, which is also a degenerative process.

MODE OF TRANSMISSION.

Waardenburg (1935) is of the opinion that the mode of transmission of senile macular degeneration is extremely doubtful as many members of the families do not live sufficiently long to develop degeneration. In my pedigree there are only 3 fully affected members and in each one the disease presented itself past the age of 70 years. In all, 72 cases were examined in this pedigree (including those in pedigree 69). Of these 72 examinations 8 cases were over 70 years of age.

ACCOMPANYING DEFECTS.

(1) *Night Blindness.*—One of the accompanying defects in this pedigree is partial stationary night blindness, which affected 3 of the males, namely II/3, III/28

and IV/40, none of whom are affected with macular degeneration. Stationary Night Blindness was fully dealt with by Nettleship (1907), and Behr (1920) draws attention to the fact that Nyctolopia and colour blindness, while common in the senile forms, may even occur in the infantile, adult and pre-senile types of macular degeneration. Sorsby (1940) also comments on concurrent night blindness.

(2) *Refractive Errors.*—In pedigree 118, which is quite considerable in length, the whole family, almost without exception, exhibited a small hypermetropic astigmatism of no great consequence and there is absolutely no association of the senile macular degeneration with myopia.

(3) *Deafness.*—There are, in this pedigree, cases of 8th nerve deafness, usually of a mild type. Its onset is approximately at 45 years and usually at 70 years a hearing aid is required. There are 7 cases to note in this pedigree and in 3 cases at least the diagnosis of senile degeneration of the 8th nerve has been confirmed by a reputable aurist. When one remembers the incidence of 8th nerve deafness in relation to adolescent retinitis pigmentosa, it is not at all surprising to find an 8th nerve deafness in a pedigree of senile pigmentary retinal degeneration such as pedigree 118. In fact the relationship of 2nd and 8th cranial nerve lesions is a subject which has not been so far fully explored.

COMMENTS ON PEDIGREE 118.

III/1 Female, aged 74 years. Hypermetropia.
1931:—
Right Vision, with correction = 6/12.
Left Vision, with correction = 6/5.
Fundi. Right—Pigmentary Senile Macular Degeneration.
Left—Fundus normal.
1932:—
Right Vision, with correction = 6/18.
Left Vision, with correction = 6/5.
Fundi. Right—Macular Degeneration unchanged. Optic disc grey and atrophic.
Left—Early Senile Pigmentary Degeneration of the Macula. Optic disc grey.
1934:—
Right Vision, with correction = 6/18.
Left Vision, with correction = 6/5.
Fundi. Unchanged, except for exudate appearing around the Macular Degeneration in both eyes.
1936:—
Right vision, with correction = 6/60.
Left vision, with correction = 6/9.
Fundi. Right — Unchanged.
Left — Increase in senile pigmentary degeneration.

III/4 Male, aged 83. Myopic astigmatism and cataract.
Right vision, with correction = 6/4.
Left vision, with correction = 6/9.
Fundi. Right—Tay's spots about macula. Marked retinal arteriosclerosis.
Left—Exudate below disc. Marked retinal arteriosclerosis.

III/6 Female, aged 73 years. Hypermetropia.
1931:—
Right vision with correction = 6/9.
Left vision with correction = less than 6/60.
Fundi. Right — Marked peripheral pigmentary retinal degeneration with moderate retinal arteriosclerosis. Marked macula atrophy with pigmentary disturbance.
Left — Marked peripheral pigmentary retinal degeneration. Marked pigmentary degeneration of the macula with moderate retinal arterio sclerosis.
Fields of vision — Both fields show concentric constriction of a moderate degree and the left field as well exhibits a marked central scotoma.
1932:—
Right vision with correction = 6/9.
Left vision with correction = less than 6/60.
Fundi. Right — No change in degeneration but optic disc now rather pale.
Left — No change in degeneration but optic disc also looks rather pale.

III/9 Male, aged 75 years. Hypermetropic astigmatism and senile cataract.
1935:—
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Right and left moderate retinal arteriosclerosis with peripheral pigmentary retinal degeneration.

III/19 Male, aged 63 years. Hypermetropic astigmatism.
Right and left vision = 6/4.
Fundi. Normal (dilated).

III/20 Female, aged 74 years. Senile cataracts and mixed astigmatism.
Right and left vision = 6/9.
Fundi. Normal (dilated).

III/22 Female, aged 70 years. Senile cataracts and hypermetropic astigmatism.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Tay's spots in both fundi and especially in the right macula.

III/23 Male, aged 74 years. Hypermetropic astigmatism.
Right and left vision with correction = 6/4.
Fundi. Moderate retinal arteriosclerosis.

III/28 Male, aged 63 years. Hypermetropic astigmatism.
Right and left vision = 6/5 with correction.
Fundi. Normal (dilated).
Has partial night blindness and inner ear deafness.

III/29 Female, aged 70 years. Hypermetropic astigmatism and senile cataracts.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. One small retinal haemorrhage in each fundus.

III/30 Male, 42 years. Hypermetropic astigmatism.
Right and left vision with correction = 6/5.
Fundi. Normal. Has middle ear deafness.

III/36 Female, aged 61 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).
This patient has inner ear deafness.

III/38 Female, aged 63 years. Hypermetropic astigmatism.
Right vision with correction = 6/6.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/50 Female, aged 47 years. Hypermetropic astigmatism.
1914:—
Right vision with correction = 6/6.
Left vision with correction = 6/6.
Fundi. Normal (dilated).
1939:—
Right vision with correction = 6/5.
Left vision with correction = 6/9.
Fundi. Subretinal exudate about maculae, with central retinal pigmentary degeneration. Retina appears raised at the maculae, especially left.

III/65 Male, aged 47 years. Hypermetropia.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Normal (dilated).
NOTE:—It will be seen that the descendants of III/1 are not recorded in this pedigree. These are recorded in a separate pedigree (No. 69), under Refractive Errors. There is no pigmentary macular degeneration in pedigree No. 69 so far, although 37 have been examined.

IV/2 Female, aged 37 years. Hypermetropic astigmatism.
Right vision with correction = 6/3.
Left vision with correction = 6/3.
Fundi. Normal (dilated).

IV/3 Female, aged 49 years. Hypermetropic astigmatism.
Right vision with correction = 6/6.
Left vision with correction = 6/6.
Fundi. Normal (dilated).

IV/4 Female, aged 47 years. Hypermetropic astigmatism.
Right vision with correction = 6/3.
Left vision with correction = 6/3.
Fundi. Normal (dilated).

IV/5 Female, aged 51 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Right — Normal (dilated).
Left — Opaque nerve fibres down and out from disc.

IV/10 Female, aged 50 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/14 Male, aged 47 years.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Both show slight retinal arteriosclerosis. No pigmentary degeneration.

IV/15 Female, aged 49 years. Hypermetropic astigmatism.
Right vision with correction = 6/12.
Left vision with correction = 6/4.
Right eye has heterochromic cyclitis with complicated cataract.
Fundi. Both fundi normal.

IV/19 Female, aged 26 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/20 Male, aged 30 years. Hypermetropic astigmatism.
Right vision with correction = 6/3.
Left vision with correction = 6/3.
Fundi. Normal (dilated).

IV/21 Female, aged 24 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (undilated).

IV/23 Male, aged 20 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/24 Male, aged 23 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/25 Female, aged 21 years. Myopic astigmatism.
Right vision with correction = 6/3.
Left vision with correction = 6/3.
Fundi. Normal (dilated).

IV/28 Male, aged 52 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).
This patient has inner ear deafness.

IV/29 Female, aged 52 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/38 Female, aged 40 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).
This patient has paralysis of accommodation.

IV/40 Male, aged 44 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).
This patient has inner ear deafness.

IV/41 Female, aged 39 years. Hypermetropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/42 Female, aged 34 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/43 Male, aged 32 years. Hypermetropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/60 Female, aged 31 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/61 Male, aged 28 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

V/5 Female, aged 12 years. Hypermetropic astigmatism and Enophorid.
Right and left vision with correction = 6/4.
Fundi. Normal (dilated).

V/6 Male, aged 8 years. Hypermetropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

V/8 Female, aged 22 years. Myopia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

V/9 Male, aged 40 years. Hypermetropia.
Right vision with correction = 6/3.
Left vision with correction = 6/3.
Fundi. Normal (dilated).

V/12 Male, aged 9 years. Hypermetropic astigmatism.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Normal (dilated).

V/20 Female, aged 9 years. No error recorded.
Right vision without correction = 6/5.
Left vision without correction = 6/4.
Fundi. Normal (undilated).

V/22 Female, aged 5 months. Treated for epiphora—right.

REFERENCES.

BEHR, C. (1920), Monatsblatter fur Augenheilkunde, 1920. Bd. LXV. Page 465.
BEHR, C. (1921), Monatsblatter fur Augenheilkunde, 1921. Bd. LXVII. Page 551.
LLOYD, R. I. (1935), Trans. American Ophthalm. Society. Vol. 33. Page 146.
LLOYD, R. I. (1935), American Journal of Ophthalmology. Vol. 26. Page 499.
NETTLESHIP, E. (1907), Trans. of the Ophthalmol. Soc. of the U.K. Vol. XXVII. Page 269.
SORSBY, A. (1934), Trans. of the Ophthal. Soc. of U.K. Vol. LIV. Page 160.
SORSBY, A. (1940), The Brit. Jl. of Ophthal. XXIV. Page 469.
TREE, M. (1937), The Brit. Jl. of Ophthal. Vol. XXI. Page 65.
WAARDENBURG, P. J. (1935), Manuscript received on July 18th.
WAARDENBURG, P. J. (1936), Genetica 18. Page 38/46. (Abstract).

NYSTAGMUS — CONSANGUINITY.

The spasmodic precipitation of hereditary eye disease had always remained a mystery to me until I began to collect pedigrees for this paper. Then the pernicious influence of consanguinity became apparent, and will be referred to again under retinitis pigmentosa.

In my pedigrees on nystagmus, consanguinity is evident both in pedigrees Nos. 4 and 30. It will be seen in pedigree No. 4 that, in generation III, two first cousins married in a family in which hereditary nystagmus was recessive, and, as the result, three of the four sibs of this union developed the disease in a gross form. In the case of pedigree No. 30, repeated consanguinity precipitated the first case of nystagmus in a perfectly healthy family. Many branches of this latter family are personally known and have been examined by me; and it is surprising, with the frequent intermarrying which has taken place, that more members have not exhibited congenital defects in one form or another.

MODE OF TRANSMISSION.

Hereditary nystagmus may be transmitted in one of two ways:—

(a) Ambisexual group, where either parent transmits it to both sexes.

(b) Recessive male limited group, transmitted by unaffected females to their male offsprings.

According to Knighton (1929) and Holm (1927) the second method of transmission is rare.

SEX INCIDENCE.

In my five pedigrees, the sex incidence was nine males to five females—*i.e.*, 61 per cent. males. I therefore, took 4 available reported pedigrees at random, and found that the total sex incidence in these was 42 males to 13 females; *i.e.*, 76.36 per cent. males. This is an interesting comparison, and I think suggests that the sex incidence in Tasmania conforms closely to that in other parts of the world.

CONCURRENT DEFECTS.

Albinism. Partial albinism is most apparent in nearly all cases of congenital nystagmus, and pedigrees 4 and 29 exhibit this tendency most markedly.

Head Nodding. This has been reported by Cox (1936) and many other authors, but I have not found a case of this in my pedigrees. It must not, however, be confused with lateral rotation of the head to one side, and both eyes to the other, which position many patients take up in order to steady their eyes, and improve their visual acuity. Case III/81 of pedigree 4, had developed this habit to a marked degree. Nor must this head nodding be confused with spasmus nutans — a totally different clinical entity.

Astigmatism. This is a most common feature as indicated by Holm (1927), and Niccol (1915), and its correction offers one hope in the alleviation of this disease. Frank (1936) states it is only treatment of any account.

Amblyopia. This varies considerably in members of the same pedigree. Pedigree No. 27 has three affected members, I/1 and II/2 having 6/9 or better vision with correction; while II/1 would not improve above 6/24 in each eye. Similarly, in Pedigree No. 4, IV/21 would not improve to 6/9, partly with astigmatic correction. It is interesting to note here, that Usher in 1912 (quoted by Niccol) reported a case of congenital nystagmus in which "a microscopic examination of the retina showed that there was no proper fovea and a few ganglion cells were present in this region. There was no evidence of lack of retinal pigment."

Stammering. Although none of the affected members of Pedigree No. 4 had concurrent stammering, yet two brothers of III/81 had this defect in a marked degree.

TREATMENT.

Except for correction of refractive errors, and especially astigmatism, there is little treatment availing. But I must stress here that careful retinoscopy combined with careful mydriatic and post-mydriatic subjective testing will frequently produce quite promising results, and due compensation for extra care taken. Orthoptic training, which has been mooted on occasions appears to hold out definite hope of improvement. III/81 has responded rapidly to it.

COMMENTS ON PEDIGREES.

Pedigree No. 4. This is a complex pedigree and I shall only refer to the right-hand section. III/60 and III/63 have divergent squints and amblyopia.

III/81 Female, aged 13 years. The most recent member under observation; has marked nystagmus, with mixed astigmatism, the correction of which improves her vision from 6/24 to 6/12 in each eye.

III/79 and III/82 — Males, no nystagmus, but stammering.

IV/21 Male, aged 19 years. Has high myopic astigmatism and nystagmus with only 6/60 vision in each eye. His brother and sister have not been examined but I believe they also have grossly defective sight.

IV/22 and IV/24 are believed to be affected, but they have not been examined.

IV/25 Male, aged 24 years. Although he has no nystagmus, he has congenital amblyopia of his right eye, without any lateral deviation, and only five dioptres of vertical deviation.

IV/29 Female, has not been examined, but is believed to be affected.

Pedigree No. 27.

I/1 The father, aged 42, has nystagmus, and high myopic astigmatism, the correction of which produces 6/6 in either eye. Also has 30° of alternating divergent concomitant strabismus, while both fundi are absolutely normal.

II/1 Male, aged 12 years. Has nystagmus and hypermetropic astigmatism. His vision will not improve above 6/24 in either eye.
Fundi. Normal.

II/2 Male, aged 10 years, on the other hand is practically emmetropic, and has 6/9 in each eye, despite his nystagmus.
Fundi. Normal.

II/4 Female, aged 10 years. Has nystagmus and myopic astigmatism, also slight exophoria.
Vision correction to 6/12 in each eye.

With regard to the nystagmus in this family, I/1 and II/2, who have good central vision, only have nystagmus when both eyes are deviated to one side or the other; while II/1 and II/4, with poor central vision, have their nystagmus most marked when looking straight ahead.

Pedigree No. 28. This is possibly a sex-linked transmission.

I/1 Male, aged 37 years. No refractive error or muscle imbalance and no nystagmus.

I/2 Female, aged 36 years. Has fine horizontal nystagmus and pure myopia.
Right vision with correction = 6/9.
Left vision with correction = 6/9.

II/1 Male, aged 9 years. Has fine horizontal nystagmus and hypermetropic astigmatism.
Right vision with correction = 6/9.
Left vision with correction = 6/9.

Pedigree No. 29.

III/2 Male, reported by the propositus to have nystagmus.

IV/18 Male, aged 10 years. Is an albino with nystagmus and mixed astigmatism. The nystagmus is more marked on looking laterally, and both macula exhibit fine pigmentary stiffling in contrast to the pale periphery of the retina.

IV/19 Male, aged 14 years. Nystagmus and myopic astigmatism.
Right vision with correction = 6/12.
Left vision with correction = 6/12.
Fundi. Normal (dilated).
This boy is a patient of Dr. F. Phillips.

Pedigree No. 30. This has been included to show how repeated consanguinity will precipitate congenital nystagmus.

IV/2 Female, aged 47 years. Is almost emmotropic, and shows no nystagmus.
Fundi. Normal (dilated).

V/1 Female, aged 22 years. As well as nystagmus, has high myopic astigmatism, and will not improve beyond 6/24 in either eye.
Fundi. Normal (dilated).

REFERENCES.

COX, R. A. (1936), Arch. Ophthal. Vol. XV. No. 6. Page 1,032.
DOGGART, J. H. (1938), Clinical Journal. July, 1938.
FRANCESCHETTI, B. (1930), Kurze, Hanb. D. Ophthal. Page 686. Berlin.
FRANK, T. (1936), Med. Jl. Austral. July 11. Page 66.
HOLM, E. (1927), Acta Ophthal. Vol. IV. Page 20.
KNIGHTON, W. S. (1929), Arch. Ophthal. Vol. II. No. 4. Page 437.
NICCOL, W. (1915), The Ophthalmoscope. Vol. XIII. Page 224.

OPTIC ATROPHY — HEREDITARY (Leber's).

Although previous mention had been made of this disease in the literature, it was first described in detail, with pedigrees, by Leber in 1871, since when considerable research has been done in many countries. Consequently, Julia Bell was able, in 1931, to publish 237 pedigrees, two of which were recorded by Hogg, from Tasmania, and are included (with his permission) with my pedigrees. To Hogg's two pedigrees (Nos. 33 and 34) I am able to add three further Tasmanian pedigrees. I propose to discuss the five Tasmanian pedigrees together under the following headings, but have nothing new to add. So much has been written about Leber's disease, and so little new elucidated, that I have found this section of my work the most unsatisfactory to write.

SEX INCIDENCE.

At once it became apparent that males greatly predominate in all five Tasmanian pedigrees, and the percentage of males affected is given in the following figures:—

Pedigree No.	Males	Females	Percentage of Males
31	22	0	100%
33	25	2	92.55%
34	3	1	75%
88	22	4	84.61%
221	4	0	100%
Totals:—	77	7	91.66%

This is certainly a higher rate than Julia Bell found in European and Japanese pedigrees, which were respectively 84.8 per cent. and 59.1 per cent. of males.

AGE OF ONSET.

Hogg, when describing his two pedigrees (Nos. 33 and 34), gives no indication of the age of onset in cases examined by him.

In my three pedigrees, the following is the recorded age of onset:—

In Pedigree 31 — II/19 at 33 years, III/44 at 27 years, IV/7 at 40 years, and IV/45 at 25 years.

In Pedigree 88 — III/13 at 37 years, IV/8 at 47 years, IV/12 at 43 years, IV/14 at 32 years, IV/31 at 41 years, V/I at 26 years and V/26 at 19 years.

In Pedigree 221—II/3 at 36 years, and III/7 at 15 years.

CAUSATION.

In none of the cases was there any suspicion of pituitary dysfunction, nor were the plotted visual fields in the slightest degree suggestive of this condition. Dr. Hogg reports that his X-ray findings of the sella turcica were negative in all cases. Gowers, in 1904, declared that the disease was an abiotrophy (Swab, 1934). I feel certain he came nearer the truth than any previous worker, and research, during the subsequent 41 years, has not been able categorically to contradict his assertion. If Gowers is right — and there seems little doubt that he is — then the correct line of treatment is prophylactic; that is, the prevention of breeding by all carrier females.

MODE OF TRANSMISSION.

In the five Tasmanian pedigrees there is no variation in transmission from those quoted from England and Scotland. In every case the transmission was through an unaffected female who frequently has affected brothers; and in no case did an affected male or female appear to transmit the disease, as was found in the pedigree of congenital optic atrophy reported by Thompson (1935). The disease in Tasmania is therefore of the sex-linked recessive type of Lossen. (Macklin, 1927.)

There is no history on consanguinity in any of the five Tasmanian pedigrees, and Julia Bell (1931) records consanguinity in only 18 of her 237 pedigrees; so that this factor does not appear to be a very important one. Nevertheless, Russell (1931) reported a pedigree in which a consanguineous marriage appears to have precipitated the disease in three brothers, in an otherwise clean stock, with no affected members in previous generations.

TREATMENT.

Kuhn (1931) reported surgical exposure of the chiasma of a male with symptoms of four weeks' duration, no abnormality was detected, and no visual improvement resulted, a further proof that to date the only available treatment is prophylactic.

MEDICO-LEGAL ASPECT.

That patients deliberately conceal the inherited factor in this disease has been revealed clearly by Schelzer (1937). He reported four in one family who attributed their loss of vision to some mild trauma. One of my cases attempted to obtain compensation for his optic atrophy, and for weeks denied that any other members of his family were similarly affected. Eventually the deception was detected and compensation naturally refused.

COMMENTS ON PEDIGREES.

Pedigree No. 31. I consider this pedigree accurate and fairly complete, as it was first given me by III/44, and later verified by IV/7. As many of the family are living in New Zealand, and some in Fiji, as well as many on the continent of Australia, it has been impossible to examine the majority of this affected. V/30 A male, who lives in Fiji, and is in his early twenties, is the most recent to become affected.

II/18 Male, aged 33 years. Myopic astigmatism.
Right and left vision with correction = 6/6.
Fundi. Normal.

III/44 Male, aged 59 years. History of bilateral loss of sight at age of 27 years.
Right vision with correction = 6/12.
Left vision = hand movements.
Bilateral primary optic atrophy.

IV/7 Male, aged 62 years. Vision in both eyes failed at 40 years.
Right and left vision, less than 6/60ths now, even with correction.
Has bilateral primary optic atrophy with nystagmus.

IV/45 Male, aged 40 years. Lost sight at age of 25 years.
Right vision = 6/24.
Left vision = counting fingers at three feet.
Vision not improved with lenses. Bilateral primary optic atrophy.

IV/47, IV/51, IV/52, IV/58: Propositus assures me that these individuals have, or had, the disease.

Pedigrees Nos. 33 and 34. These are published by kind permission of Dr. G. H. Hogg, of Launceston, Tasmania; but I am unable to give any details at all. Dr. Hogg, in a private communication, regrets that he has no details available (other than those given when he published the pedigrees in the Medical Journal of Australia, on March 24th, 1928), except that the members came from the northern end of this island, and are unlikely to be included in my complete pedigree (No. 31).

Pedigree No. 88.

III/13 Male, aged 39 years. Bilateral optic atrophy for years.
Right and left vision is counting fingers at two inches.

IV/7 Male, aged 48 years. Patient of Dr. J. L. R. Carter, of Launceston.
Bilateral optic atrophy with reduction of right and left vision to perception of light.

IV/8 Male, aged 47 years. Sight failed in both eyes two months ago.
Right and left vision equals hand movements.
Both optic discs pale.

IV/12 Male, aged 43 years. Sight failed at age of 43 years.
Right vision = 1/60.
Left vision = 1/60.
Bilateral optic atrophy.

IV/14 Male, aged 33 years. Bilateral optic atrophy for one year.
Both discs pale.
No record of vision.

IV/31 Male, aged 41 years. Sight failed at age of 41 years. Bilateral optic atrophy.
Right vision = counting fingers at two feet.
Left vision = counting fingers at three feet.
This patient was examined by the late Dr. Lindsay Miller.

V/1 Male, aged 32 years. Sight in both eyes failed six years ago.
Right and left vision less than 6/60.
Primary optic atrophy right and left.

V/26 Male, aged 39 years. Sight failed 20 years ago.
Right and left vision reduced to counting fingers at 12 inches.
Bilateral primary optic atrophy.

Pedigree No. 221.

II/3 Male, aged 46 years. Sight failed ten years ago.
Right and left vision reduced to shadows.
Has bilateral primary optic atrophy.

III/7 Male, aged 45 years. Bilateral failure of vision at age of 15 years.
Right and left vision = 3/60.
Has bilateral primary optic atrophy. He is the propositus.

REFERENCES.

BEDELL, A. J. (1934), Amer. Jl. Ophthal. Vol. XVII. No. 3. Page 195.
BELL, J. (1931), Treasury of Human Inheritance. Vol. II. Part IV. Cambridge.
CORDES, F. C. (1933), Trans. Amer. Ophthal. Soc. Vol. XXXI. Page 289.
HOGG, G. H. (1928), Med. Jl. Austral. March 24. Page 372.
HOGG, G. H. (1936), Personal Communication.
KUHN, A. S. (1931), Arch. of Ophthal. March (abstract).
MACKLIN, M. T. (1927), Canad. Med. Assoc. Jl. Vol. XVII. Page 1,339.
RUSSELL, W. R. (1931), Trans. Ophthal. Soc., U.K. Vol. LI. Page 187.
SCHMELZER, H. (1937), Arch. of Ophthal. Vol. 137. Page 216.
SWAB, C. M. (1934), Nebraska Med. Jl. Vol. XIX. Page 184.
THOMPSON, A. H. (1935), Proc. Roy. Soc. Med. Vol. XXVIII. Page 1,415.
USHER, C. H. (1927), Brit. Journ. of Ophthal. Vol. II. Page 417.

PTERYGIUM.

Tasmania, which is famous throughout the Antipodes for its delightful climate, is practically free from progressive pterygium, trachoma, hyperkeratosis and rodent ulcer of the lids. During the past fourteen and a half years, out of 9,980 private case records, I have found the following statistics:—

Pterygia	141 cases	=	1.41%
Trachoma (old)	12 „	=	0.12%
Trachoma (active)	4 „	=	0.04%
Rodent Ulcer of the Lids	11 „	=	0.1%

Of these 141 cases of pterygium, 27 cases have had to be dealt with surgically, so that it is evident that pterygium per se is a disease of little consequence in Tasmania, although on the Continent of Australia it is a constant source of concern.

Of these 141 cases I can find an hereditary influence in only 4 cases — two brothers (whose maternal parent suffered from the same condition), and a mother and daughter.

SEX INCIDENCE.

Reference to hereditary pterygium in the literature is very scanty. Macklin (1927) reports two pedigrees from the literature — one of three affected males and five affected females in four generations; and another of ten affected males and two affected females in six generations.

My two pedigrees consist of two males and three females, and this predominance of females is not at present explained.

MODE OF TRANSMISSION.

Macklin (1927) considers the transmission as dominant, being transmitted by both parents to offsprings of either sex. I, personally, have found no evidence of this.

COMMENTS ON PEDIGREES.

Pedigree No. 35.

I/2 Female — is dead, but definitely had a pterygium which her sons think was on her right eye.

II/1 Male, aged 25 years. Right nasal pterygium, which was marsupialised.

II/2 Male, aged 23 years. Right nasal pterygium which was marsupialised.

Pedigree No. 141.

I/1 Female, aged 67 years. Right anophthalmos from absolute glaucoma. Left chronic dacryocystitis, left nasal pterygium, left chronic glaucoma with complicated cataract.

II/3 Female, aged 34 years. Early bilateral nasal pterygia.

REFERENCES.

GATES, R. R. (1929), Heredity in Man. London.
MACKLIN, M. T. (1927), Canad. Med. Assoc. Jl. Vol. XVII. Page 698.

PTOSIS — HEREDITARY.

In the Transactions of the Ophthalmological Society of Australia, Volume 1, page 84, I reported 33 cases of birth injuries to the eyes and annexa. Included in these 33 cases are four patients with ptosis, three of whom had been delivered by forceps. So, in reviewing our cases of hereditary ptosis, it is very necessary to be cautious and to avoid including in the pedigrees any case whose condition may have been caused by birth trauma. Unfortunately, in my four pedigrees about to be reviewed, only one member in each has been examined by me. Nevertheless, my cross questioning of the propositi has left me in no doubt that the condition is hereditary in each family tree. On the other hand, as pointed out by Faulkner (1939), hereditary ptosis may not occur in some pedigrees until adult life, and therefore some members of a pedigree may die before the onset of this condition.

SEX INCIDENCE.

According to Macklin (1927), the sex incidence is about equal, taking all reported pedigrees collectively, but in my four pedigrees, 14 males have been affected and only seven females.

MODE OF TRANSMISSION.

Briggs (1919), Usher (1925), and Macklin (1927), all assert that the mode of transmission is dominant, but only one of my pedigrees confirms this; namely, No. 215.

AGE OF ONSET.

Pedigree No. 232 confirms Faulkner's (1939) statement that the onset in certain pedigrees may be during adult life. As far as I am able to ascertain from the propositus, the age of onset of the six persons in this pedigree was approximately between 40 and 50 years.

ASSOCIATED DEFECTS.

Epicanthus. Usher (1925) and Clausen (1923) surveyed the literature on hereditary epicanthus with ptosis — they found them frequently reported together, and although ptosis occurs often without epicanthus, yet they could only find one instance of hereditary epicanthus per se.

High Refractive Error, Nystagmus, and Paralysis of Superior Rectus. These also have been frequently reported in pedigrees of hereditary ptosis. Usher (1925) reports that in 18 eyes examined, only two had normal vision. One of my examined cases had a high refractive error, and an affected superior rectus.

Squint with Amblyopia ex Anopsia. This likewise is a fairly frequent concurrent sign, and was present in one of my cases, in the form of a convergent strabismus of 20° and partial amblyopia in the left eye.

External Ophthalmoplegia. This has been reported by Faulkner (1939) as accompanying ptosis — both conditions commencing in adult life in his pedigree.

PATHOLOGY.

According to Briggs (1919), the defective action of the levator may be due to five causes:—

(a) Defective development.
(b) Adhesion to the superior rectus.
(c) Abnormal insertion.
(d) Replacement by connective tissue.
(e) Entire absence.

but in my series I have had no biopsy material to examine and so cannot comment personally.

TREATMENT.

In view of this pathology, to attempt a routine advancement of the levator appears to be futile, and therefore some other muscle must be attached to the lid and used to elevate it. This can be carried out by utilising the superior rectus as in the classical Motais operation. The more recent operation of Greeves (1933) and of Lexer (Armstrong, 1940) utilise the frontales, as does the classical operation of Hess. Personally, I find Snellen's sutures produce an equally satisfactory result if allowed to cut their own tracks upwards.

COMMENTS ON PEDIGREES.

Pedigree No. 36.

II/1 Male, supposed to be affected, but this has not been confirmed.

III/7 Male, not examined but confirmed. Patient has only a slight degree of bilateral ptosis.

III/8 Female, aged 45 years. Examined. No ptosis.

IV/3 Male, aged 33 years. Examined. No ptosis.

IV/4 Female, aged 34 years. Examined. No ptosis.

IV/18 Male, not examined, but propositus reports that he is affected.

V/1 Male, aged 5½ years. Marked bilateral ptosis with head tilting and contraction of both frontales. Marked defective movement of right globe up, and up and in. Left convergent concomitant strabismus of 20° with six dioptres of left hyperphoria.

An interesting point in this child is: Having corrected a horizontal muscle error by exercises and operation, I attempted to correct his vertical error with six prism dioptres, combined with his hypermetropic lenses. The wearing of this prism increased his vertical error, so that without the prism this stood at twelve prism dioptres, while with it the vertical error was stationary at six prism dioptres. Apparently he has an abnormal vertical correspondence of six prism dioptres, an unusual but not unreported condition.

Pedigree No. 215.

II/1, II/3, II/5, III/3 and III/5 are reported by the propositus to be affected.

III/7 Male, aged 22 years. Bilateral congenital ptosis of a moderate degree.

III/9, III/15 are also supposed to be affected.

Pedigree No. 216.

II/9 Male, aged 30 years. Has moderate bilateral ptosis, and assures me that his deceased daughter and his living son inherited his lid condition.

Pedigree No. 232.

III/3 Female, aged 51 years. Assures me that in all the affected members (I/2, II/1, II/2, II/3 and III/6) of her family, ptosis came on in middle life. She, herself, was born in 1894, and has given me photographs taken of herself at varying intervals from 1914 to 1945. At the age of 26 years she certainly had wide palpebral apertures, but by 39 years of age there was definite evidence of bilateral ptosis. In 1945 her palpebral apertures are reduced to 3 mm. and her vision is obscured by the ptosis.

REFERENCES.

ARMSTRONG, T. M. (1940), Trans. of Ophthal. Soc. of Aust. Vol. II. Page 84.

BRIGGS, H. H. (1919), Amer. Jl. Ophthal. Vol. II. Page 408.

CLAUSEN, W. (1923), Zentralbl. f. die. ges. Ophthal. und ihre Grenzgebiete. Vol. XI. No. 11. Page 481.

FAULKNER, S. H. (1939), Brit. Med. Jl. Oct. 28th. Page 854.

GREEVES, R. A. (1933), Brit. Jl. Ophthal. Vol. XVII. No. 12. Page 741.

HAMILTON, J. B. (1939), Trans. Ophthal. Soc. Aust. Vol. I. Page 84.

MACKLIN, M. T. (1927), Canad. Med. Assoc. Jl. Vol. XVII. Page 55.

McILROY, J. H. (1929), Proc. Roy. Soc. Med. Vol. XXIII. Page 285.

USHER, C. H. (1925), Ann. Eugenics. Vol. I. Page 128.

USHER, C. H. (1935), Trans. Ophthal. Soc., U.K. Vol. LV. Page 164.

REFRACTIVE ERRORS.

I have divided refractive errors into three classes — Hypermetropic Astigmatism, Mixed Astigmatism and Myopic Astigmatism. In the first group six pedigrees are reported; in the second group two, and in the third group 23 pedigrees are reported. These pedigrees have been compiled from an examination of 9,980 patients over a 15 year period, of which no less than 7,047 showed a refractive error. Of these 7,047, 3,393 showed hypermetropic astigmatism or hypermetropia; 1,212 showed mixed astigmatism and 2,242 showed myopic astigmatism, or myopia. In other words, 33.96% of my total 9,980 cases showed hypermetropic astigmatism or hypermetropia, 12.1% showed mixed astigmatism, and 24.49% showed myopic astigmatism or myopia. From these figures we see that there is a definite predominance on the hypermetropic side.

TABLE XVI.

REFRACTIVE ERRORS.

Total Patients Examined in Past Fifteen Years 9,980
Total Cases of Refractive Errors Found 7,047

	Cases Examined	Percentage of Total	Hereditary Cases Found	Percentage of Total
Hypermetropic Astigmatism or Hypermetropia	3,393	33.96	301	3.0
Mixed Astigmatism	1,212	12.14	90	0.9
Myopic Astigmatism or Myopia	2,242	22.46	629	6.3

Now let us turn to the percentage of hereditary refractive errors. We find that there were 301 cases of inherited hypermetropia or hypermetropic astigmatism, showing a 3.0 percentage. In mixed astigmatism there were 90 cases, giving a 0.9 percentage. In myopic astigmatism and myopia there were 629 cases, or 6.3%. These are indeed interesting figures, because they show that in the first group of hypermetropia or hypermetropic astigmatism only one in every thirteen cases shows an hereditary tendency, and mixed astigmatism is the same, but when we come to myopia and myopic astigmatism the figure rises immediately to one in four.

If we look at these figures in another light, we see that out of a total of 31 pedigrees collected, no less than 23 are of myopic astigmatism, giving a percentage of 74.2. Now, if we take the total number of cases of hereditary refractive errors, namely one thousand, we find that there are 629 cases of myopic astigmatism or myopia, giving a percentage of 62. Statistics such as these are of somewhat doubtful importance, taking into consideration the old adage that one straw does not show which way the wind is blowing.

MODE OF TRANSMISSION.

It is obvious that myopia and myopic astigmatism are far more frequently inherited than hypermetropic astigmatism or mixed astigmatism. Holm (1925) considered the mode on transmission of myopia may be either dominant or

recessive, and this is borne out in my 23 pedigrees, although most other authors consider it recessive only in transmission. Astigmatism, according to Duke Elder (1934) and Ruggles Gates (1929), is undoubtedly hereditary in many pedigrees, and is usually dominant in transmission. They state that not only is the degree of astigmatism maintained, but also the axis in most pedigrees. With astigmatism manifesting itself in at least 3/4 of ophthalmic cases, the surgeon ultimately regards it as a necessary evil, and gives it scant consideration. Nevertheless, I feel that if ophthalmologists working in confined areas (as I do) would pay some attention to the hereditary aspect and report their findings, they would be agreeably surprised to find that astigmatism is not as boring as they had thought. Although in my mind, refractive errors are definitely inherited, such authors as Levinsohn (1934) and Franceschetti (1930), seriously doubt the hereditary factor, while Roden (1933) tries to explain all cases on a mechanical or pathological basis. At least 6.3% of my 2,442 myopes gave an hereditary history.

COMMENTS ON PEDIGREES.

Hypermetropic Astigmatism.

Pedigree No. 5. This is of special interest, as a member of the first family, in which there are at least six members who have compound myopic astigmatism, married a member of the second family, of which at least three had compound hypermetropic astigmatism. The result has been five sibs, the first being affected with hypermetropia, the second with hypermetropic astigmatism in both eyes, the third has hypermetropia, the fourth has not been examined and the fifth has myopia.

I/5 Female, aged 74 years. Myopic astigmatism.
Right vision = less than 6/60 with correction.
Left vision = 6/6 with correction.
Fundi. Right — Degeneration of the macula.
Left — Normal (dilated).

I/7 Female, aged 58 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/1 Male, age unknown. Refractive error unknown.
Right vision with correction = 6/8.
Left vision with correction = 6/18.

II/3 Male, aged 62 years. Hypermetropic astigmatism.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Retinal arteriosclerosis.

II/4 Female, aged 56 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/7 Male, aged 58 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/8 Female, aged 58 years. Mixed astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi (dilated). Left Tay's choroiditis.

II/9 Female, aged 48 years. Myopic astigmatism.
Right vision with correction = 6/18.
Left vision with correction = 6/9.
Fundi. Both appear normal, but are obscured by lens sclerosis.

II/11 Male, aged 42 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/12 Female, aged 42 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/14 Female, aged 41 years. Myopic astigmatism.
Right vision with correction = 6/6.
Left vision with correction = 6/6.
Fundi. Normal (dilated). Early peripheral lens opacities.

II/15 Female, aged 51 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Peripheral pigmentary retinal degeneration.

II/16 Male, aged 68 years. No refractive error.

II/20 Female, aged 50 years. Myopic astigmatism and anisocoria.
Right vision with correction = 6/9.
Left vision with correction = 6/3.
Fundi. Left — Normal. Early lens striae.
Right — Normal. Marked lens striae.

II/21 Male, aged 51 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Tay's choroiditis.

II/22 Female, aged 29 years. Myopic astigmatism.
Right vision with correction = 6/6.
Left vision with correction = 6/4.
Fundi. Normal (undilated).

II/23 Female, aged 32 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/27 Female, aged 36 years. Mixed astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/31 Female, aged 55 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Both eyes. Peripheral pigmentary retinal degeneration. Tay's choroiditis.

II/33 Female, aged 48 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated). Early peripheral lens striae.

II/38 Male, aged 46 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/3 Male, aged 24 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Early retinal arterio sclerosis.

III/4 Female, aged 28 years. Hypermetropic astigmatism.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Normal (dilated).

III/8 Female, aged 19 years. Hypermetropic astigmatism and exophoria.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/16 Male, aged 21 years. Hypermetropic astigmatism.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Normal (dilated).

III/28 Male, aged 21 years. Hypermetropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/29 Female, aged 26 years. Hypermetropic astigmatism and exophoria.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/30 Female, aged 14 years. Hypermetropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/32 Male, aged 14 years. Myopic astigmatism, and exophoria.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Normal (dilated).

III/33 Male, aged 3 years. Refractive error unknown.

III/35 Male, aged 8 years. Myopic astigmatism, anisocoria, amblyopia ex anopsia, exophoria.
Right vision with correction = 6/9.
Left vision with correction = 6/24.
Fundi. Normal (dilated).

III/37 Female, aged 6 years and 6 months. Hypermetropia, overaction both inferior obliques, esophoria.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Normal (dilated).

Pedigree No. 69. The most interesting point about this pedigree is that II/4 has hypermetropic astigmatism and marked esophoria, while her niece III/22, not only has hypermetropic astigmatism but has an esotropia.

I/1 Female, aged 74 years. Hypermetropic astigmatism and glaucoma.
Right vision with correction = 6/12.
Left vision with correction = 6/5.
Fundi. Pigmentary macula degeneration (dilated).

II/3 Male, aged 62 years. No refractive error.
Right vision without glasses = 6/6.
Left vision without glasses = 6/3.
Fundi. Normal (undilated).

II/4 Female, aged 61 years. Hypermetropic astigmatism and esophoria.
Right vision with correction = 6/6.
Left vision with correction = 6/4.
Fundi. Normal (dilated). Marked post cortical lens opacities right and left.

II/5 Male, aged 58 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Peripheral lens striae.

II/6 Female, aged 54 years. Hypermetropic astigmatism.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Normal (dilated).

II/7 Male, aged 48 years. Right eye removed at age of 11 years, following perforating injury.
Left eye — hypermetropic astigmatism.
Left vision with correction = 6/3.
Left fundus — Normal (dilated). Early post cortical lens opacities.

II/8 Propositus assures me that this individual has the disease.

II/9 Female, aged 37 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated). Right and left lens sclerosis.

II/11 Male, aged 41 years. Hypermetropic astigmatism and divergence insufficiency.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/12 Female, aged 33 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/13 Female, aged 53 years. Mixed astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Mild retinal arteriosclerosis.

II/16 Female, aged 43 years. Hypermetropic astigmatism and divergence insufficiency.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/17 Female, aged 50 years. Mixed astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated). Peripheral lens striae.

II/18 Male, aged 62 years. Mixed astigmatism.
Right vision with correction = 6/18.
Left vision with correction = 6/12.
Fundi. Peripheral retinal degeneration. Gross posterior cortical cataracts.

II/19 Female, aged 54 years. Hypermetropic astigmatism.
Right vision with correction = 6/5.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/1 Female, aged 37 years. Mixed astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/2 Male, aged 27 years. Myopic astigmatism.
No refractive error.
Fundi. Normal (dilated).

III/3 Male, aged 24 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/4 Female, aged 26 years. Mixed astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/5 Female, aged 29 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/6 Male, aged 28 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/7 Female, aged 24 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/8 Female, aged 20 years. Hypermetropic astigmatism.
Right vision with correction = 6/3.
Left vision with correction = 6/3.
Fundi. Normal (dilated). Persistent hyaloid artery.

III/9 Female, aged 14 years. Hypermetropic astigmatism and convergence excess.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/11 Male, aged 19 years. Myopic astigmatism, colour blindness.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Right — Shows small congenital cyst on optic disc.
Left — Normal.

HI/16 Female, aged 9 years. Hypermetropic astigmatism.
Right vision with correction = 6/3.
Left vision with correction = 6/3.
Fundi. Normal (dilated).

III/19 Female, aged 14 years. Hypermetropia.
Right vision with correction = 6/4.
Left vision with correction = 6/5.
Fundi. Normal (dilated).

III/21 Male, aged 14 years.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
No record of refraction or fundi.

III/22 Female, aged 10 years. Hypermetropic astigmatism.
Esotropia.
Right amblyopia ex anopsia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/23 Female, aged 8 years. Orbital cellulitis.
No record of refraction.

III/24 Male, aged 19 years. Optic atrophy, possibly traumatic.
Right vision with correction = less than 6/60.
Left vision with correction = less than 6/60.
Fundi. Primary optic atrophy.

III/25 Male, aged 20 years. Myopic astigmatism and esotropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/26 Female, aged 17 years. Mixed astigmatism and esotropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/26A Female, aged 24 years. Hypermetropic astigmatism.
Right vision with correction = 6/3.
Left vision with correction = 6/3.
Fundi. Normal (dilated).

III/34 Male, aged 6 years. Hypermetropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/1 Female, aged 4 years and 6 months.
No record of refractive error.

IV/3 Male, aged 13 weeks. Epiphora.
No record of refractive error.

IV/10 Male, aged 10 months.
Congenital nystagmus.

Pedigree No. 91.

III/4 Female, aged 69 years. Myopic astigmatism.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Moderate retinal arteriosclerosis.

III/6 Female, aged 66 years. Hypermetropic astigmatism and esophoria.
Right vision with correction = 6/9.
Left vision with correction = 6/5.
Fundi. Normal (dilated).
Right and left lens sclerosis.

III/8 Female, aged 73 years. Myopic astigmatism and chronic glaucoma.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Right — Vitreous opacities ++
Early cupping of the optic disc.
One small cyst on disc.
Tay's choroiditis.
Left — Vitreous opacities ++
Optic disc normal.
Fields. Right — 1 degree white shows partial loss of upper nasal field and early loss of lower nasal field.
Left — 1 degree white, normal.

IV/1 Male, aged 65 years. Myopic astigmatism and chronic glaucoma.
Right vision with correction = 6/3.
Left vision with correction = 6/3.
Fundi. Early cupping of both optic discs.
Perimetry. Normal to 1 degree white.
Scotomotry. Baring of the left blind spot to 5 mm. white at 2 metres.

IV/2 Female, aged 56 years. Hypermetropic astigmatism.
Right vision with correction = 6/3.
Left vision with correction = 6/3.
Fundi. Normal (dilated).

IV/3 Female, aged 56 years. Myopic astigmatism and exophoria.
Right vision with correction = 6/3.
Left vision with correction = 6/3.
Fundi. Normal (dilated).

IV/4 Female, aged 50 years. Myopic astigmatism and vitreous opacities.
1931:—
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Right — Normal.
Left — Optic disc appears pathologically cupped.
Fields of vision to 1° white normal.
1938:—Unchanged.
1945:—Unchanged.

IV/5 Male, aged 43 years. Mixed astigmatism and esophoria.
Right vision with correction = 6/3.
Left vision with correction = 6/3.
Fundi. Normal (dilated).

IV/6 Female, aged 34 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/7 Male, aged 42 years. Hypermetropic astigmatism and esophoria.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/8 Female, aged 36 years. Mixed astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/9 Male, aged 40 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/10 Female, aged 37 years. Mixed astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/11 Female, aged 42 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/12 Male, aged 43 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/13 Female, aged 47 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/14 Refractive error unknown.

IV/15 Male, aged 36 years. Hypermetropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/16 Female, aged 39 years. Mixed astigmatism and paralysis of accommodation.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

V/3 Female, aged 16 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

V/4 Male, aged 12 years.
Refractive error unknown.

V/5 Female, aged 11 years. Hypermetropia.
Right vision with correction = 6/5.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

V/6 Male, aged 15 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

V/7 Female, aged 16 years. Hypermetropic astigmatism, and esophoria.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Normal (dilated).

V/8 Male, aged 8 years. Hypermetropic astigmatism, and esophoria.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Normal (dilated).

V/9 Male, aged 8 years. Hypermetropia and esophoria.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi (dilated).

V/10 Female, aged 7 years. Hypermetropia.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi (dilated).

V/18 Male, aged 11 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

V/21 Male, aged 3 years and 6 months. Esotropia.
No refractive error.
Fundi. Normal (dilated).

Pedigree No. 92. This pedigree consists of two families both suffering from hypermetropic astigmatism. The resulting sibs of the marriage of a female in one family to a male in the other, are two females with marked hypermetropic astigmatism in all four eyes and the younger of the two also has an exophoria.

I/3 Female, aged 68 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

I/5 Female, aged 66 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Pigmentary degeneration in right macula periphery. Left fundus normal.

I/7 Female, aged 50 years. Hypermetropia.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Normal (dilated).

II/1 Female, aged 50 years. Hypermetropic astigmatism.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Normal (dilated).

II/5 Female, aged 27 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/6 Female, aged 50 years. Hypermetropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/1 Male, aged 20 years. Hypermetropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
No record of condition of fundi.

III/2 Female, aged 10 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/3 Female, aged 6 years. Hypermetropic astigmatism and exophoria.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

Pedigree No. 96. It is interesting to note in this pedigree that, while the paternal grandmother of the twins had a very high hypermetropic astigmatism, two paternal aunts had a low degree, while the twins had a high degree almost corresponding in amount to the grandmother's.

I/2 Female, aged 80 years. Hypermetropic astigmatism.
Right vision with correction = 6/9.
Left vision with correction = 6/12.
Fundi. Optic discs normal.

II/1 Female, aged 50 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Retinal arteriosclerosis and senile cataracts (dilated).

II/4 Female, aged 47 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/1 Male, aged 3 years. Hypermetropia and right esotropia.
Vision not ascertained.
Fundi. Normal (dilated).

III/2 Male, aged 3 years. Hypermetropia and alternating esotropia.
Vision not ascertained.
Fundi. Normal (dilated).

Pedigree No. 129.

II/1 Female, aged 80 years. Hypermetropic astigmatism.
Right vision with correction = 6/12
Left vision with correction = 6/6.
Fundi. No record.

II/3 Male, aged 73 years. Hypermetropic astigmatism.
Right vision with correction = 6/60.
Left vision with correction = 6/6.
Fundi. Right — Thrombosis of sup. temporal vein.
Left — Marked retinal arteriosclerosis.

II/4 Female, aged 70 years. Hypermetropic astigmatism.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Early macula degeneration right and left.

II/6 Male, aged 65 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/7 Female, aged 50 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/12 Female, aged 58 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/13 Male, aged 54 years. Mixed astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/1 Male, aged 41 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Moderate retinal arteriosclerosis, right and left.

III/2 Female, aged 40 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/3 Female, aged 21 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/4 Male, aged 33 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/7 Male, aged 24 years. Hypermetropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/1 Female, aged 6 years. Myopia.
Right vision with correction = 6/5.
Left vision with correction = 6/5.

IV/3 Female, aged 10 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/4 Male, aged 11 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

Mixed Astigmatism.

Pedigree No. 1.

I/1 Male, aged 49 years. High mixed astigmatism.
Right vision with correction = 6/18.
Left vision with correction = 6/9.
Fundi. Normal (dilated).

I/2 Female, aged 54 years. Myopic astigmatism, and cataracts.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/2 Male, aged 15 years. High mixed astigmatism and esophoria.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Pseudo-neuritis right and left.

II/4 Female, aged 16 years. Right high mixed astigmatism.
Left eye — emmetropic.
Right vision with correction = 6/12.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

Pedigree No. 2.

I/2 Female, aged 54 years. Hypermetropic astigmatism, and cataracts.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/1 Female, aged 37 years. High mixed astigmatism.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Normal (dilated).

II/3 Male, aged 34 years. Right eye — Emmetropic.
Left eye — High mixed astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/9.
Fundi. Normal (dilated).

II/7 Male, aged 27 years. Right Eye — Anopthalmos due to injury.
Left eye — Hypermetropic astigmatism.
Left vision with correction = 6/9.
Fundi. Normal (dilated).

II/9 Male, aged 27 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/14 Female, aged 27 years. Anisometropia.
Right — High myopic astigmatism and amblyopia.
Left — Moderate hypermetropic astigmatism.
Right vision with correction = 6/5.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/7 Female, aged 8 years 6 months. High mixed astigmatism.
Right vision with correction = 6/9.
Left vision with correction = 6/9.
Fundi. Normal (dilated).

III/8 Female, aged 6 years, 6 months. High mixed astigmatism.
Right vision with correction = 6/18.
Left vision with correction = 6/18.
Fundi. Normal (dilated).

III/9 Male, aged 7 years. Right — Moderate hypermetropic astigmatism.
Left — Hypermetropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

Myopic Astigmatism.

Pedigree No. 20.

I/1 Female, aged 76 years. Myopic astigmatism and retinal degeneration. Has not been examined, but the propositus assures me she has very defective eyesight and refuses treatment.

II/1 Female, aged 44 years. High myopic astigmatism.
1934:—
Right vision with correction = 6/12.
Left vision with correction = 6/12.
Fundi. Marked choroidal stretching involving the macula.
1939:—
Right vision with correction = 6/12.
Left vision with correction = 6/60.
Fundi. Right — Unchanged.
Left — Extensive detachment of the retina with multiple holes, re-attached by diathermy.

II/3 Female, aged 36 years. Hypermetropic astigmatism and anisometropia.
Left amblyopia ex anopsia.
Overaction of right inferior oblique.
Esophoria and right hyperphoria.
Right vision with correction = 6/5.
Left vision with correction = 6/18.
Fundi. Normal (dilated).

II/4 Male, aged 39 years. Myopic astigmatism and retinal detachment with albinism.
1933:—
Right vision = hand movements.
Left vision = 6/18 with correction.
Fundi. Right — Large upper temporal detachment with horseshoe-shaped hole re-attached by diathermy.
Left — Normal.
1934:—
Recurrence of right detachment in upper temporal quadrant.
Re-attached by diathermy and no further recurrence.
1937:—
Left retinal detachment in upper temporal region with multiple holes.
Operated on by diathermy with re-attachment.
1945:—
Right vision with correction = 6/18.
Left vision with correction = 6/36.

Fundi. Right — Normal except for operation scars.
Left — Normal except for recent haemorrhage at the left macula.

II/8 Male, aged 34 years. Mixed astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/12 Male, aged 24 years. Myopic astigmatism, anisometropia and amblyopia.
Right vision with correction = 6/5.
Left vision with correction = 6/18.
Fundi. Normal (dilated).

II/13 Female, aged 28 years. Emmetropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
No record of fundi except optic discs are normal.

III/5 Female, aged 9 years. Mixed astigmatism and anisometropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/7 Female, aged 5 years.
Right vision with correction = 6/6.
Left vision with correction = 6/6.
Refraction not recorded.

Pedigree No. 21.

III/1 Male, aged 91 years. Right myopic astigmatism. Left hypermetropic astigmatism.
Right vision with correction = 6/36.
Left vision with correction = 6/24.
Fundi. Normal. Gross lens changes.

III/9 Male, aged 85 years. Myopic astigmatism and paralysis of external rectus.
Right vision with correction = 6/5.
Left vision with correction = 6/4.
Fundi. Cavernous atrophy of both optic discs.

III/11 Female, aged 82 years. Myopic astigmatism.
Right vision with correction = 6/18.
Left vision with correction = shadows.
Fundi. Gross myopic stretching of both fundi involving the maculae. Gross lens changes.

III/14 Male, aged 80 years. Hypermetropic astigmatism.
Right vision with correction = 6/12.
Left vision with correction = 6/6.
Fundi. Cavernous atrophy of both optic discs. Right and left lens sclerosis.

III/15 Female, aged 79 years. Myopic astigmatism.
Right vision with correction = 6/6.
Left vision with correction = 6/5.
Fundi. Normal (dilated).

III/16 Male, aged 67 years. Hypermetropia. Left old traumatic retinitis.
Right vision with correction = 6/9.
Left vision = hand movements.
Right fundus (undilated). Normal.
Left fundus (undilated). Old retinitis involving macula.

IV/3 Female, aged 47 years. Hypermetropia. Epileptic.
Right vision with correction = 6/9.
Left vision with correction = 6/9.
Fundi. Normal (dilated).

IV/4 Female, aged 48 years. Hypermetropic astigmatism.
Right vision with correction = 6/9.
Left vision with correction = 6/9.
Fundi. Normal (dilated).

IV/6 Male, aged 57 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Early pigmentary macular degeneration.

IV/7 Female, aged 55 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Pigmentary macular degeneration. Retinal arteriosclerosis.

IV/10 Male, aged 57 years. Right and left myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/11 Female, aged 58 years. Hypermetropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/13 Male, aged 70 years. Mixed astigmatism, glaucoma and senile cataracts.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).
Fields. Slight loss to 1° white.

IV/14 Male, aged 51 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/15 Female, aged 42 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/18 Female, aged 52 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/22 Female, aged 60 years. Presbyopia
Right vision = 6/9.
Left vision = 6/9.
Not refracted.

IV/23 Female, aged 58 years. Myopic astigmatism.
Right vision with correction = 6/3.
Left vision with correction = 6/3.
Fundi. Normal (dilated).

IV/24 Female, aged 58 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/25 Female, aged 53 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/31 Male, 62 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/32 Female, aged 46 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated). Gross vitreous opacities.

IV/34 Male, aged 49 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/35 Female, aged 43 years. Hypermetropic astigmatism.
Right vision with correction = 6/6.
Left vision with correction = 6/6.
Fundi. Normal (dilated). Gross lens changes.

IV/36 Male, aged 43 years. Myopia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/37 Female, aged 56 years. Hypermetropia and esophoria.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/40 Female, aged 51 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/43 Male, aged 52 years. No refractive error.
Fundi. Right — Normal.
Left — Old retinitis.

V/3 Male, aged 20 years.
Right vision = 6/6.
Left vision = 6/6.
Not refracted.

V/5 Female, aged 35 years. Mixed astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/6.
Fundi. Early degeneration of both maculae.

V/9 Female, aged 35 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

V/18 Male, aged 23 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

V/19 Female, aged 20 years. Ocular palsy due to trauma.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

V/20 Female, aged 30 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

V/22 Female, aged 17 years. Hypermetropic astigmatism, and esophoria.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

V/24 Female, aged 16 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

V/26 Female, aged 10 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

V/28 Male, aged 22 years. Mixed astigmatism and esophoria.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

V/29 Female, aged 24 years. Hypermetropia and esophoria.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

V/33 Male, aged 29 years. Mixed astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

V/39 Female, aged 17 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

V/41 Male, aged 23 years. Atrophy of both maculae.
Right vision with correction = 2/60.
Left vision with correction = 6/60.
No refractive error.

V/43 Male, aged 23 years. Hypermetropic astigmatism and esophoria.

V/50 Male, aged 31 years.
Right vision = 6/4.
Left vision = 6/4.
Not refracted.

V/51 Female, aged 14 years. Myopia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

V/54 Female, aged 14 years. Myopia.
Right vision with correction = 6/3.
Left vision with correction = 6/3.
Fundi. Normal (dilated).

VI/18 Male, aged 7 years. Hypermetropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

VI/22 Male, aged 10 years. Hypermetropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

Pedigree No. 22.

III/1 Male, aged 49 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/1 Propositus assures me that this male has short sight.

IV/2 Female, aged 21 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/7 Female, aged 20 years. Myopic astigmatism and exophoria.
Right vision with correction = 6/6.
Left vision with correction = 6/6.
Fundi. Normal (dilated).

Pedigree No. 23.

II/3 Male, aged 59 years. Myopic astigmatism and disseminated choroiditis.
Right vision = hand movements.
Left vision with correction = 6/9.
Fundi. Right — Gross disseminated choroiditis.
Left — Normal.

III/3 Male, aged 17 years. Myopic astigmatism.
Right vision with correction = 6/9.
Left vision with correction = 6/9.
Fundi. Normal (dilated).

Pedigree No. 25.

I/1 Male, aged 52 years. Presbyopia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/3 Female, aged 31 years. Myopic astigmatism and anisometropia.
Right vision with correction = 6/9.
Left vision with correction = 6/3.
Fundi. Normal (dilated).

II/4 Male, aged 31 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated),

II/7 Male, aged 28 years. Myopic astigmatism.
Right vision with correction = 6/5.
Left vision with correction = 6/9.
Fundi. Normal (dilated).

II/9 Female, aged 14 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

Pedigree No. 55.

II/8 Male, aged 69 years. Myopic astigmatism.
Right vision with correction = 6/9.
Left vision with correction = 6/5.
Fundi. Vitreous opacities, right greater than left.

II/9 Female, aged 75 years. Hypermetropic astigmatism.
Right vision with correction = 6/9.
Left vision with correction = 6/6.
Fundi. Retinal arteriosclerosis with arteriosclerotic retinitis.

II/11 Male, aged 74 years. Not examined, but known to have defective eyesight due to shortsightedness.

III/5 Male, aged 39 years. No refractive error.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/6 Female, aged 43 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/7 Female, aged 36 years. Myopic astigmatism and exophoria.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/9 Female, aged 34 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/13 Male, aged 36 years.
Known to have defective eyesight due to shortsightedness.

III/15 Female, aged 19 years. High myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

Pedigree No. 63.

I/4 Female, aged 84 years. Mixed astigmatism.
Right vision with correction = 6/12.
Left vision with correction = 6/12.
Fundi. Normal (dilated), but marked lens changes.

II/2 Female, aged 73 years. Myopic astigmatism.
Right vision with correction = 6/60.
Left vision with correction = 6/12.
Fundi. Right — Gross stretching involving the macula. Gross lens changes.
Left — Normal, early lens changes.

IV/4 Female, aged 8 years. Myopic astigmatism. Exophoria.
Right vision with correction = 6/6.
Left vision with correction = 6/6.
Fundi. Normal (dilated).

Pedigree No. 78.

II/6 Female, aged 66 years. Mixed astigmatism.
Right vision with correction = 6/9.
Left vision with correction = 6/12.
Fundi. Tay's choroiditis, marked lens changes.

II/9 Male, aged 80 years. Hypermetropic astigmatism.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Normal (dilated). Early lens changes.

III/8 Female, aged 37 years. Myopic astigmatism, and mixed astigmatism.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Normal (dilated).

III/12 Male, aged 35 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/13 Female, aged 32 years. Myopic astigmatism, anisometropia and amblyopia ex anopsia.
Right vision with correction = 6/4.
Left vision with correction = 6/12.
Fundi. Normal (dilated). Left marked lens opacity.

III/14 Male, aged 51 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated). Early lens changes.

III/17 Male, aged 46 years. Myopia.
Right vision with correction = 6/6.
Left vision with correction = 6/6.
No record of fundi.

IV/3 Female, aged 14 years. Myopic astigmatism.
Right vision with correction = 6/5.
Left vision with correction = 6/9.
Fundi. Normal (dilated).

IV/5 Female, aged 10 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

Pedigree No. 95.

II/8 Female, aged 43 years. Retrobulbar neuritis following measles. No refractive error.
Fundi otherwise normal.

III/1 Female, aged 15 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/2 Male, aged 12½ years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/3 Male, aged 6½ years. Myopia.
Right vision with correction = 6/6.
Left vision with correction = 6/6.
Fundi. Normal (dilated).

III/8 Female, aged 17 years. Right — Myopia.
Left — Hypermetropia.
Anisometropia.
Amblyopia ex anopsia.
Right vision with correction = less than 6/60.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

Pedigree No. 101.

II/8 Female, aged 63 years. Right — Mixed astigmatism.
Left — Myopic astigmatism.
Anisometropia.
Right vision with correction = 6/9.
Left vision with correction = 6/12.
Fundi. Right — Gross vitreous opacities.
Left — Early macula stretching with moderate vitreous opacities.

II/9 Male, aged 64 years. Mixed astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated). Marked left lens changes.

III/8 Male, aged 24 years. Gross hypermetropic astigmatism.
Right vision with correction = 6/9.
Left vision with correction = 6/6.
Fundi. Normal (dilated).

III/9 Female, aged 18 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/3 Female, aged 4 years. Convergent concomitant strabismus.
Vision and refraction not ascertained.

Pedigree No. 124.

II/1 Male, aged 50 years. Myopic astigmatism and post cortical cataracts, right and left.
Right vision with correction = 6/9.
Left vision with correction = 6/12.
Fundi. Normal (dilated).

II/2 Female, aged 41 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/1 Male, aged 10 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/2 Female, aged 6 years. Myopic astigmatism and exophoria.
Right vision with correction = 6/3.
Left vision with correction = 6/3.
Fundi. Normal (dilated).

Pedigree No. 125.

II/1 Female, aged 81 years. Myopic astigmatism.
Right vision with correction = 6/6.
Left vision with correction = 6/5.
Fundi. Gross lens sclerosis right and left.

II/5 Female, aged 61 years. Myopic astigmatism, optic atrophy, and macula degeneration.
Right vision with correction = 6/36.
Left vision with correction = perception of light.
Fundi (dilated). Secondary optic atrophy right and left and right macula degeneration.

II/6 Female, aged 52 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/7 Male, aged 54 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/10 Female, aged 71 years. Myopic astigmatism.
Right vision with correction = 6/9.
Left vision with correction = 6/12.
Fundi. Normal. Gross posterior cortical lens opacities.

II/11 Female, aged 69 years. High myopic astigmatism.
Right vision with correction = 6/18.
Left vision with correction = 6/18.
Fundi. Normal. Gross vitreous opacities and lens sclerosis.

III/4 Female, aged 27 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/6 Female, aged 38 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/7 Female, aged 33 years. Mixed astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/10 Female, aged 21 years. Hypermetropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/12 Male, aged 45 years. Mixed astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Moderate retinal arteriosclerosis.

III/14 Female, aged 51 years. Hypermetropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/15 Female, aged 51 years. Hypermetropic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/16 Female, aged 32 years. Mixed astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/1 Female, aged 9½ years. Myopic astigmatism.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Normal (dilated).

Pedigree No. 140.

III/2 Female, aged 42 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/8 Female, aged 25 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/9 Male, aged 24 years. Mixed astigmatism and exophoria.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/10 Female, aged 19 years. Myopic astigmatism and exophoria.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

Pedigree No. 146.

II/2 Female, aged 63 years. Myopic astigmatism and senile cataracts.
Right vision with correction = 6/9.
Left vision with correction = 6/9.
Fundi. Marked retinal arteriosclerosis both eyes.

II/7 Female, known to have worn monocle for myopia.

III/4 Female, aged 36 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/5 Male, aged 22 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/6 Female, aged 21 years. High hypermetropic astigmatism in right eye.
Left eye — Emmetropic.
Right vision with correction = 6/9.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/7 Known to wear glasses for myopia.

III/12 Male, aged 30 years. Myopic astigmatism.
Right vision with correction = 6/5.
Left vision with correction = 6/4.
Fundi (dilated). Some old left retinitis.

IV/4 Female, aged 7 years. Myopic astigmatism.
Right vision with correction = 6/12.
Left vision with correction = 6/12.
Fundi. Normal (dilated).

IV/5 Female, aged 5½ years. Myopic astigmatism.
Right vision with correction = 6/9.
Left vision with correction = 6/9.
Fundi. Normal (dilated).

IV/7 Female, aged 3 years. Right hypermetropic astigmatism. Left hypermetropia.
Right vision with correction = 6/12.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

IV/8 Female, aged 7 years. No record of refraction.
Right vision without correction = 6/6.
Left vision without correction = 6/9.

IV/9 Female, aged 5 years. Right myopia and left emmetropia.
Right vision with correction = 6/24.
Left vision with correction = 6/6.
Fundi. Normal (dilated).

Pedigree No. 148.

I/1 Male, aged 74 years. Myopic astigmatism and senile cataracts.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Early vitreous opacities.

II/1 Female, aged 47 years. Myopic astigmatism and exophoria.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/2 Male, aged 37 years. Left hypermetropic astigmatism. Right sarcoma of the choroid.
Left vision with correction = 6/4.
Left fundus. Normal (dilated).

II/3 Male, aged 43 years. Myopic astigmatism.
Right vision with correction = 6/5.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/1 Female, aged 17 years. Right myopic astigmatism, left emmetropia and exophoria.
Right vision with correction = 6/18.
Left vision with correction = 6/3.
Fundi. Right myopic disc. Left fundus normal.

III/6 Male, 8 years. Emmetropia.
Right vision with correction = 6/3.
Left vision with correction = 6/3.
Fundi. Normal (dilated).

III/8 Male, aged 10 years. Myopic astigmatism.
Right vision with correction = 6/9.
Left vision with correction = 6/9.
Fundi. Normal (dilated).

Pedigree No. 158.

II/1 Female, aged 78 years. Hypermetropic astigmatism and retinitis.
Right vision with correction less than 6/60.
Left vision with correction = 6/18.
Fundi. Haemorrhagic retinitis and senile cataracts.

II/2 Female, aged 77 years. Myopic astigmatism and anisometropia.
Right vision with correction = 6/9.
Left vision with correction = 6/36.
Fundi. Normal, gross lens changes right and left.

II/3 Male, aged 84 years. Hypermetropia.
Right vision with correction = 6/9.
Left vision with correction = 6/12.
Fundi. Normal. Gross lens changes.

III/4 Male, aged 64 years. Myopia.
Right vision with correction = 6/4.
Left vision with correction = 6/12.
Fundi. Normal. Gross left vitreous opacities.

III/6 Female, aged 57 years. Myopic astigmatism, exophoria and cataracts.
Right vision with correction = 6/4.
Left vision with correction = hand movements.
Left visual loss due to vitreous haemorrhage.
Fundus. Right — Normal.
Left — Not seen.

IV/5 Female, aged 20 years. Hypermetropic astigmatism and exophoria.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Normal (dilated).

IV/6 Male, aged 30 years. Myopic astigmatism.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Normal (dilated).

IV/8 Female, aged 18 years. Myopic astigmatism and alternating esotropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

V/1 Female, aged 6 years. Myopic astigmatism and anisometropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

V/6 Female, aged 2 years. No details of refraction.

Pedigree No. 162.

II/1 Female, aged 49 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/2 Female, aged 50 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/1 Male, aged 17 years. Myopic astigmatism and anisometropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/2 Female, aged 13 years. Myopic astigmatism and anisometropia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/4 Female, aged 2½ years. No record of refraction.

Pedigree No. 168.

I/2 Female, aged 64 years. Myopic astigmatism.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Normal (dilated). Left senile lens changes.

II/1 Female, aged 29 years. Myopic astigmatism and esophoria.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/2 Female, aged 32 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/3 Male, aged 22 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

Pedigree No. 169.

I/1 Male, aged 83 years. Myopic astigmatism and retinal haemorrhage.
Right vision with correction = 6/9.
Left vision with correction = 6/60.
Fundi. Right — Retinal arteriosclerosis.
Left — Thrombosis of the central vein.

I/2 Female, aged 69 years. Right hypermetropic astigmatism.
Left myopic astigmatism.
Right vision with correction = 6/3.
Left vision with correction = 6/36.
Fundi. Right — Gross retinal arteriosclerosis.
Left — Gross lens changes, fundus not discernable.

II/1 Female, aged 49 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/2 Male, aged 52 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/3 Male, aged 43 years. Myopic astigmatism, and exophoria.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. No record of condition.

III/1 Female, aged 19 years. High myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/3 Female, aged 14 years. Moderate myopic astigmatism.
Right vision with correction = 6/6.
Left vision with correction = 6/6.
Fundi. Normal (dilated).

III/4 Male, aged 8 years. No record of refractive error but has exophoria.
Right vision with correction = 6/5.
Left vision with correction = 6/4.
Fundi. No record.

III/6 Male, aged 8 years. No refractive error recorded. Exophoria.

Pedigree No. 173.

I/1 Male, aged 58 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated). Early senile lens changes.

I/2 Female, aged 40 years. High myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/2 Female, aged 16 years. High myopic astigmatism, and convergence excess.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

Pedigree No. 180. I have very reliable information that the patients not examined by me in this pedigree are short sighted, and I consider it sufficiently correct to warrant inclusion below.

I/1 Male, aged 70 years. Defective vision due to short sight.
Details unknown.

II/1 Male, aged 38 years. High myopic astigmatism and esophoria.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/2 Female, aged 36 years. Mixed astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/3 Male, age unknown. Has defective sight due to myopia.
Details unknown.

II/7 Male, age unknown. Has defective sight due to myopia.
Details unknown.

III/1 Male, aged 8 years. Moderate myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/2 Male, aged 11 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

Pedigree No. 210.

II/3 Male, aged 49 years. High myopic astigmatism.
Right vision with correction = 6/5.
Left vision with correction = 6/5.
Fundi. Right — Normal.
Left — Choroidal stretching.

III/1 Female, aged 16 years. Myopic astigmatism, nystagmus and pseudo-glioma cataract and choroiditis.
Right vision with correction = no perception of light.
Left vision with correction = 6/18.
Right — No fundus details. Left — Old choroiditis.

III/2 Female, aged 17 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/3 Female, aged 20 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

III/4 Female, aged 15 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

Pedigree No. 213.

II/1 Male, aged 57 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

II/2 Female, aged 64 years. High myopic astigmatism.
Right vision with correction = 6/18.
Left vision with correction = 6/18.
Fundi. Normal (dilated). Gross lens changes.

III/1 Female, aged 17 years. Myopia.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. No record.

III/2 Male, aged 17 years. Myopic astigmatism.
Right vision with correction = 6/4.
Left vision with correction = 6/4.
Fundi. Normal (dilated).

REFERENCES.

BEST, H. (1934), Blindness and the Blind in U.S.A. 2nd Edit. New York.

BLACKER, C. P. (1934), Chances of Morbid Inheritance. London.

GATES, R. R. (1929), Hereditary in Man. London.

FRANCESCHETTI, B. (1930), Kurze. Handb. D. Ophthal. Page 720. Berlin.

HOLM, E. (1925), Acta. Ophthal. Vol. III. Page 335.

LAW, F. W. (1934), Trans. Ophthal. Soc., U.K. Vol. LIV. Page 281.

LEVENSOHN, G. (1934), Acta. Ophthal. Vol. XII. Page 362.

PASCAL, J. I. (1935), Arch. Ophthal. Vol. XIV. Page 624.

RODEN, F. H. (1933), Arch. Ophthal. Vol. XI. Page 264.

SHUGG, A. W. (1935), Personal communication.

SORSBY, A. (1928), Brit. Jl. Ophthal. Vol. XII. Page 197.

SORSBY, A. (1935), Brit. Jl. Ophthal. Vol. XIX. Page 216.

WALKER, J. S. P. (1932), Brit. Jl. Ophthal. Vol. XVI. Page 485.

WILSON, J. A. (1935), Brit. Jl. Ophthal. Vol. XIX. Page 613.

RETINAL DETACHMENT.

This heading should, perhaps, have been better designated "JUVENILE HEREDITARY RETINAL DETACHMENT," because all the cases about to be reported occurred in children before puberty. Before I discuss the cases, I first want to review our current knowledge of polycystic disease of the kidneys and congenital cystic disease of the lungs, because I consider both diseases have a bearing on the subject under review.

Firstly, about polycystic disease of the kidneys, liver and pancreas. To the surgeon, this is a disease which frequently comes under his knife, but I fear that the majority do not realise that, at least in the case of polycystic disease of the kidneys, the condition is almost invariably inherited. Further, it seems to be inherited in two forms — polycystic disease of the kidneys, which is present at birth, and a senile form which appears after the age of 40, and from which the patient dies of uraemia. .

The infantile form in some cases produces so large a tumour as to obstruct delivery, while the senile form often exhibits large abdominal tumours just prior to the patient's death. May I refer you to two papers, the first appearing in the Quarterly Journal of Medicine, 1925, and written by a famous Australian, Sir H. W. B. Cairns, now Nuffield Professor of Surgery at Oxford University. He pointed out in a very clear and concise way, giving pedigrees, that polycystic disease of the kidneys is a definitely inherited condition.

This paper was followed the succeeding year by one from W. Gordon Sears, in Guy's Hospital Report. He believes that in 30% of the cases of polycystic disease of the kidneys, the liver is also affected, and he quotes cases in which the pancreas is involved, too. Sears dwells considerably on the hereditary nature of the disease and states that males and females are alike affected and both may transmit the disease. He further stresses that other congenital anomalies may be associated with polycystic disease of the kidneys; firstly, those associated with the urinary apparatus; secondly, malformation of the genital organs; thirdly, visceral malformations; and, fourthly, other abnormalities including the meningocele, hydrocephalus, polydactylism, webbed fingers and toes, and clubbed feet. He quotes Lussato as saying that 71% of the cases of polycystic disease of the kidneys are associated with other congenital abnormalities, half of which are outside the urinary tract.

Cairns remarks in his review of cases from East London that, at least, seven of the affected individuals were myopic, but as many of the others were not subjected to routine ophthalmic examination, it is likely that this figure is understated. Personally, I think that this myopia is quite coincidental, and has no relation to the inheritance of the polycystic disease. His patients appear to have suffered from two inherited diseases — polycystic disease of the kidneys and inherited myopia.

CONGENITAL CYSTIC DISEASE OF THE LUNGS.

In the light of the above, let us consider the cyst of the lung: The most masterly interpretation of congenital cystic disease of the lungs was given by Sellors in 1938. He divides these diseases into four categories, as follows:—

(1) Balloon cysts.
(2) Solitary cysts.
(3) Multiple cysts of the gross "Bubble" type.
(4) Cystic disease of the "Berry" type.

To the "balloon cyst" he attributes spontaneous pneumothorax. To the "solitary cyst" he attributes haemoptysis not due to tuberculosis. To the "bubble type," lung abscess; and to the "berry type," the honeycomb lung or, in other words, congenital bronchiectasis. But Sellers will not commit himself that congenital cystic disease of the lungs is associated with congenital disease in other parts of the body, but he is of the opinion that this will ultimately be confirmed as a fact. However, he makes no reference to the inherited nature of congenital cystic disease of the lungs, nor does Richardson (1943).

So, from this preamble we are confidently able to say that, at least, polycystic disease of the kidneys is definitely inherited.

INHERITED DETACHMENT OF THE RETINA.

Now let us turn to the title of this paper, Inherited Retinal Detachment. That detachments of the retina are inherited there is no doubt, and Richner (1936) gave a complete survey of the literature on the subject, with special reference to senile inherited detachments of the retina. In the 32 pedigrees of spontaneous detachment of the retina which he was able to collect, he found accompanying these cases other congenital defects of the eye, and he further establishes, beyond doubt, that spontaneous senile detachment of the retina is due to secondary cystic degeneration of the retina, which is in itself inherited.

Richner confined his remarks to the senile form of inherited retinal detachment, but this review particularly concerns infantile cystic degeneration of the retina accompanied by spontaneous detachment. The preceding preamble has been written entirely to try and throw some light on the strange behaviour of infantile detachments and the stranger appearance of their condition.

Let me digress for a moment and quote from Weve (1938). In an article of his discussing congenital retinal folds in relation to other congenital eye diseases he states, "As Carl Heyl points out in his recent book (Ueber Retinitin Unbekannten Ursprungs, 1937), 'we must abandon mistaken opinion about the origin of the pseudo-glioma.' In his opinion a great majority of the so-called pseudo-gliomas are congenital abnormalities." I want to stress this point particularly because, in the case histories which I am going to discuss later, this point is of paramount importance. I, now, have pleasure in presenting a family with congenital cystic disease of the retina, and retinal detachment accompanied by lesions which we would previously have called pseudo-glioma.

SURVEY OF PEDIGREE THIRTY-SEVEN.

The most interesting feature of the whole family in Pedigree 37 is this — that the first child, whom I saw in 1932, has, in the year 1945, developed congenital cystic disease of the lung of such severity that she is unable to pursue any occupation. To my knowledge, this is the only member of the whole pedigree whose chest shows cystic disease at all, and, although at least four other members of the pedigree, who have retinal detachments, have undergone a chest X-ray, no abnormalities have been found to date.

It appears on the surface, and I feel quite certain that it will stand the test of time, that congenital cystic disease of the retina, in its infantile form, may be associated with congenital cystic disease of the lungs as part of a generalised cystic process through the body.

If I now refer you to the article of Bruce (1939) on congenital cystic disease of the lungs, it will be found that he stresses that these cysts of the lungs often become infected, and another point he mentions is that the congenital cysts of the lungs may rupture.

Now let us take the first of these two points; that the congenital cysts of the lungs may become infected. I consider the condition found in the fundi of some of the forthcoming patients to be retinal cysts, which have become infected just as congenital cysts of the lungs become infected. For example, III/19 of Pedigree 37, had pseudo-glioma of both eyes; while III/24 had pseudo-glioma in the left eye with aniridia. To my mind, these pseudo-gliomata are really infected congenital retinal cysts.

The second point Bruce made was that some of the congenital cysts of the lung rupture. I am of the opinion that congenital retinal cysts rupture also. In fact, one of the patients whom I am reporting, namely, III/19, although she had bilateral detachments for at least ten years, to-day has no detachment in one eye. The only interpretation I can make is that the retinal cysts which she had, have ruptured and the retina has re-attached itself.

Further to this point, some years ago, I saw another child not included in this pedigree, with unilateral detachment of the retina. Recently, she came to see me again, and although she has had no treatment, her retina had completely re-attached itself. (I might mention that her second eye is highly hypermetropic.) She has not reached puberty yet, and I should think we might call this case also an infantile cystic detachment of the retina, and possibly analogous to the case in my pedigree which has been caused by retinal cysts and ultimately healed by their rupture.

Let us now survey the whole Pedigree 37 analytically, and in so doing I refer you to Table XVII, where the whole analysis is tabulated. First of all, there are ten patients examined, a total of eighteen eyes. Of these ten patients, six were refracted, making a total of ten eyes refracted, and everyone of them showed hypertropia or hypermetropic astigmatism, so that I think we can rule out a relationship to myopia with certainty. One of the parents was very positive that she could tell when her children's eyes were "going wrong" from the fact that the colour of the eyes changed. I was never able to verify this fact, nor was I able to tie the parent down to a clear explanation of exactly what she meant. Personally, I was unable to note any iris colour change, even in the cases of monocular detachment.

Of the ten patients examined, five had visible retinal detachments, and a total of seven eyes were involved. These might be listed under four headings:—

1. Those retinal detachments which were pseudo-gliomatous.
2. Those associated with retinitis proliferans.
3. Those in which the detachment re-attached itself.
4. Those retinal detachments without special features.

Two further cases have monocular cataracts with loss of projection, and I consider undoubtedly they have a retinal detachment too.

Pseudo-glioma occurred in two patients, with a total of three eyes involved. One of these three eyes had a detachment which ultimately re-attached itself.

Patient III/19 had a bilateral pseudo-glioma, while patient III/24 had a unilateral pseudo-glioma. The nature of the other eye of III/24 might have been pseudo-gliomatous, but this was not apparent as the patient had previously undergone an operation for re-attachment without result. Patient III/22 had retinitis proliferans accompanied by retinal detachment, but the condition did not have a pseudo-gliomatous appearance.

With regard to complicated cataracts, two patients with three eyes affected, developed complicated cataracts after their detachments had appeared, but two other patients, namely III/26 and III/33, were not seen until they had developed unilateral cataracts in one eye, with loss of projection. We can rightly presume that a detachment was present behind the cataracts. To my mind there is no doubt.

Peripheral choroido-retinitis was present in four patients unilaterally. This is an interesting fact. One eye also had an associated retinal cyst. The other three had choroido-retinitis without the cyst formation, but, in at least three of these four eyes, the condition was active and, I should say, progressive.

Patient III/24 exhibited a partial aniridia of one eye. There was an almost complete aniridia of the mesoderm with loss of half the ectoderm. The condition on inspection was from centre outwards as follows:—

A very dilated pupil; a wide ring of iris pigment followed by a narrow ring of iris stroma. The relation of this aniridia to the other defects accompanying the pedigree is not yet explained. It has been pointed out, however, by Richner (1936) that inherited retinal detachment is often accompanied by other inherited eye conditions.

COMMENTS ON PEDIGREE THIRTY-SEVEN.

This pedigree has so many unusual features that I intend to report it in full.

II/14 Female, aged 36 years, 1939.
Right vision = 6/4 + J1.
Left vision = 6/4 + J1.
Small compound hypermetropic astigmatic error in both eyes.
Fundi. Normal.

III/19 Female, aged 7 years.
1932. History of failing vision in both eyes for four months. Pneumonia and empyema three years previously, followed by chronic bronchitis.
Right vision = counting fingers at 12″.
Left vision = 5/60.
Fundi (dilated)—
Right — Gross vitreous opacities. Large retinal detachment below, in colour resembling a pseudo-glioma. No hole was seen.
Left — Large detachment below with solid portion down and in, resembling a pseudo-glioma.
General Examination—
Chest X-ray — Bronchiectasis.
Wassermann Reaction — doubtful positive, but on repetition was negative.

1935.
Right vision = counting fingers at 6".
Left vision = counting fingers at 3 feet.
Fundi (dilated)—
Right — Complete retinal detachment. No hole was seen.
Left — Extensive detachment below and much old choroiditis above. No hole was seen.
General Examination—
Wassermann and Kline reactions — negative.
1940.
Right vision = hand movements.
Left vision = counting fingers at 12".
Fundi. Unchanged.
1945.
Right vision = doubtful perception of light.
Left vision = hand movement with accurate projection.
Fundi. Right — Immature cataract. Faint reflex. No fundus details.
Left — Marked disseminated retinitis impregnated with crystals. No detachment present.
Left field of vision to 4° white is reduced to within 5° of fixation.
General Examination—
Chest X-ray — Congenital cystic disease of the left lung. The lower half of this lung is almost solid and shows multiple cavities with many fluid levels.

III/22 Female, aged 13 years, 1945.
History of pneumonia two years ago. Failing vision for past year.
Right vision = 1/60.
Left vision = 6/12.
Fundi (dilated). Right — Extensive retinitis proliferans above with very extensive detachment below. No hole seen.
Left — Early peripheral pigmentary retinal degeneration at four and twelve o'clock.
General Examination—
Sputum — No tubercle baccilli.
Chest X-ray — Negative.
Mantoux — Negative.
Kline — Negative.
Wassermann — ±.

III/23 Male, aged 11 years. Patient of Dr. J. L. R. Carter.
1945.
Has compound hypermetropic astigmatism.
Right vision = 6/5.
Left vision = 6/6.
Fundi. Right — Normal.
Left — Large patch of congenital choroiditis down and out from optic disc.
1946.
Sudden loss of sight in right eye.
Fundus. Right — Extension detachment of the retina in the lower half of the right eye.

III/24 Male, aged 12 years.

1932 history. Left sight failed four years ago and right sight 18 months ago. Has had three Gonin operations on right eye without improved vision.

Right vision = shadows.

Left vision = no perception of light.

Fundi (dilated). Right — Complete retinal detachment with large hole at 7.30 o'clock, about two disc diameters from ora serrata.

Left — Complicated cataract obscuring fundus details. Partial aniridia, that is almost complete aniridia of the mesoderm with loss of half the ectoderm. The condition on inspection was from centre outwards:— A very dilated pupil: a wide ring of iris pigment followed by a narrow ring of iris stroma.

1935.

Right eye — Commencing iris bombe.

Left eye — Mature cataract. Commencing transverse corneal degeneration. Aniridia unchanged. Shrunken opaque lens dislocated above. Complete opaque retinal detachment impregnated with glistening crystals.

General Examination—

Wasserman — Negative.

Kline — Negative.

Had cataract operation right eye, August, 1939. No improvement of vision.

Right vision = perception of light.

Left vision = no perception of light.

R.E. — Multiple anterior synechise.
Thick capsule. No reflex.

L.E. — Cornea completely opaque.

1945.

Chest X-ray — Negative.

III/26 Male, aged 12 years. 1935.

History — Left sight failed two years ago.

Right vision = 6/12 with + 3.0 = 6/5 + J1.

Left vision = Perception of light. No projection.

Fundi. Right — Normal.
Left — Hypermature cataract.

General Examination—

Wassermann — Negative.

Kline — Negative.

1945.

Right vision with $\frac{+\ 3.25}{+0.25\ \text{ax.}\ 30.} = 6/6 + J1.$

Left vision = no perception of light.

Fundi (dilated). Right — Some fresh exudate in nasal periphery at 3 o'clock, but no cyst seen.
Left — Hypermature cataract.
General Examination—
Chest X-ray — Negative.

III/28 Female, aged 11 years. Patient of Dr. J. L. R. Carter, from 1936 onwards.
Has compound hypermetropic astigmatism with normal fundi.

III/31 Male, aged 15 years. Patient of Dr. J. L. R. Carter.
Right vision = 6/4.
Left vision = 3/60.
Fundi. Right — Normal.
Left — Extensive detachment of retina in the lower nasal quadrant with hole. Vitreous opacities.
Chest X-ray — The lung fields appear to be within normal limits. Search was made for any cystic condition.

III/33 Male, aged 11 years. 1945.
Right vision = no perception of light. Divergent 15°.
Left vision = 6/6 + J1. Hypermetropic astigmatism.
Fundi (dilated)—
Right — Mature cataract.
Left — Some recent exudate surrounding a small cyst in retina at 3 o'clock.
General Examination—
Chest X-ray — Clear.

III/34 Male, aged 8 years. 1945.
Right vision = 6/4 under mydriatic with + 2.25 = 6/4.
Left vision = 6/4 under mydriatic with + 2.25 = 6/4.
Fundi were normal.
Refraction — Bilateral hypermetropia.

REFERENCES.

Congenital Cystic Disease of the Kidneys, Liver and Pancreas.
CAIRNS, H. W. B. (1925), Quart. Jl. of Med. Vol. XVIII. Page 359.
CAMPBELL, G. C. (1929), Brit. Med. Jl. Oct. Page 716.
LATIMER, E. O. (1923/27), Trans. Chicago Path. Soc. Vol. XII. Page 353.
SEAR, W. G. (1926), Guy's Hospital Report. Vol. LXXVI. Page 31.

Congenital Cystic Disease of the Lungs.
BRUCE, T. (1939), Acta Medica, Scandinavia. Vol. 102. Page 295.
ILLINGSWORTH, R. S. (1938), Proc. Royal Soc. Med. Vol. 32. Page 602.
RICHARDSON, J. S. (1943), Jl. of R.A.M.C. April. Page 213.
SELLORS, T. H. (1938/9), Congenital Cystic Disease of the Lungs, Tubercle. Vol. 20. Page 49/71, page 114/136.

Detachment of the Retina.
ANDERSON, J. R. (1931), Detachment of the Retina, Cambridge.
ARRUGA, H. (1933), XIV Concilium Ophthal. 1933. Hispana T. 11/1 (abstract).
BOGATSCH, G. (1911), Klin. Manatsbl. f. Augenheilk. Vol. XLIX. Page 431.
BRIT. MED. JL. (1940), Bilateral Detached Retina. June 15th.
CARTER, J. L. R. (1945), Personal communication.

CLAUSEN, W. (1923), Zentrab. F. die Ges. Ophth. und thre Grenz. Vol. LXL. No. 11, Page 481.

DE ROTH, A. (1939), Arch. of Ophth. Vol. XXII. Page 809.

GRAEFE-SAEMISCH-HESS (1916), Hand. d. ges. Augenheilk. 2nd edit. Bd. VII/2 Kap. XA/z.

HOGG, G. H. (1936), Personal communication.

MACKLIN, M. T. (1927), Can. Med. Assoc. Jl. Vol. XVII. Page 1,336.

MANN, I. C. (1928), Development of the Human Eye, London.

MARSHALL, J. C. (1936), Detachment of the Retina, London.

RICHNER, H. (1936), A. V. Graefe's Arch. f. Ophthal. Vol. CXXXV. Page 49.

SALZMANN, M. (1921), Wien. Med. Wochenschr. Vol. LXXL. Page 1,082.

SCHMELZER, H. (1929), Arch. f. Augenheilk. Vol. C-cl. Page 268.

VOGELSANG (1937), Klin. Mon. Zur. Augen. Vol. XCVIII. Page 251.

WEVE, H. (1936), Arch. f. Augenkeilkunde. Page 371.

WEVE, H. (1938), Brit. Jl. of Ophthal. Vol. XXII. Page 456.

ZUR NUDDEN (1936), Klin. Mon. Fur. Augen. Vol. XCVII. Page 236.

Aniridia.

BELL, J. (1932), Treasury of Human Inheritance. Vol. II. Part V. Cambridge.

CROLL, L. J. (1929), Arch. of Ophth. Vol. II. No. 6. Page 699.

MACKLIN, M. T. (1927), Can. Med. Assoc. Jl. Vol. XVII. Page 937.

TABLE XVII.

	Patients Examined	Eyes Examined
TOTAL	10	18
REFRACTED	6	10
HYPERTROPIA	6	10
RETINAL DETACHMENT	5	7
with		
(a) Pseudo-glioma	2	3
(b) Retinitis Proliferans	1	1
(c) Detachment Re-attached	1	1
COMPLICATED CATARACTS—		
With detachments behind	2	3
UNILATERAL COMPLICATED CATARACTS—		
With presumed detachment behind	2	2
PERIPHERAL CHOROIDO-RETINITIS	4	4
(1) With retinal cyst	1	1
(2) Without retinal cyst	3	3
(a) Inactive	1	1
(b) Active	3	3
ANIRIDIA	1	1

RETINITIS PIGMENTOSA.

It is obvious that the elucidation of the nature of retinitis pigmentosa immediately succeeded the invention of the ophthalmoscope in 1851, for, while the symptoms were somewhat typical, the actual diagnosis rested with the fundus examination. In 1853 the first case was described by van Trigh at Utrecht, and about the same time von Graefe described a case in Berlin but the credit of classifying all available information must remain with Nettleship. (Bell, 1922.)

Under the above heading, Nettleship included the following six diseases as belonging to one group of hereditary defects of sight:—

(1) Retinitis pigmentosa.
(2) Retinitis pigmentosa sine pigmento.
(3) Retinitis punctata albescens.
(4) Gyrate atrophy of the choroid and retina.
(5) Stationary night blindness.
(6) Choroideremia.

Julia Bell (1922) finds no inclination to depart from this classification, and gives her reasons, while Usher in his Bowman Lecture (1935), also gives ample pedigrees to substantiate this contention.

In my investigation of hereditary eye diseases in Tasmania during the past 15 years, I have been able to find only 17 fresh cases of retinitis pigmentosa. In not one case could I find an hereditary history, but in nine cases — two in each of three families and three in another — there was a familial history, and these are depicted in pedigrees 38, 39, 40 and 190. In one family (pedigree 38) the parents were first cousins, but I was able to examine only the female member as the male member refused to submit to any examination. The female member in this family also refused any treatment except change of glasses.

CONCURRENT ANOMALIES.

Ring scotoma. In none of the 34 eyes examined was there a complete ring scotoma, but in three eyes there was a suggestion of one, though nothing more.

Wassermann reaction. This was done in seven cases out of 17, but all seven tests were negative. One of these seven cases had a Kahn test, and two had a Kline test, but these were negative also.

Deafness. Three out of 17 patients complained of deafness — and two of these occurred in Pedigree 39. The elder sister in Pedigree 39 was examined by Dr. Hiller (1933), who reported a bilateral nerve type of deafness. The third case was also examined by Dr. Hiller (1945), who reported chronic otitis media.

Obesity, hypogenitalism, polydactylism, or mental diseases (Laurence-Moon-Biedl-Syndrome)-Sorsby (1932)-Savin (1935)-did not appear in any of the 17 cases, neither was there evidence of hyperactivity of the sympathetic system such as Reynaud's disease or chilblains. In view of the number of new pedigrees of this syndrome, reported since Sorsby's article on Laurence in 1932, I have asked my medical and surgical colleagues in Tasmania to draw my attention to any cases they might see, but without result.

PATHOLOGICAL EXAMINATION.

None of the 34 eyes examined has been submitted to pathological examination, but I feel that little new would have been revealed by such examination, as the signs and symptoms of the disease conform very closely to those reported from England, and to those seen by the author while in London. Therefore I have nothing to add to the theories of causation of this disease, except that the theory of vaso-constriction of the retinal vessels does not appear to hold good in the light of recent surgical treatment. (See treatment.)

CONSANGUINITY.

This was apparent in only one case out of seventeen; *i.e.*, 5.88%, compared with Julia Bell's (1922) estimation of 27.2% in 817 cases, but I shall say more of this under economic significance.

SEX INCIDENCE.

Of the 17 cases examined, no less than 12 were females, giving an incidence of 70.5%, but Bell (1922) found a predominance of males in her series. Dickson and Mitchell (1927) report a predominance of females, but quote no figures or references to substantiate this.

MODE OF TRANSMISSION.

Usher (1935) in his Bowman lecture describes retinitis pigmentosa as a "Mendelian recessive through a few large pedigrees with many affected individuals showing predominance." He also gives a pedigree of sex-linked retinitis pigmentosa. Allan (1937) discusses the severity of retinitis pigmentosa in relation to the mode of inheritance, and points out that cases belonging to a dominant transmission are not so severely affected as those belonging to a recessive transmission, and he explains it as follows:—"When an uncommon condition is inherited as a unit dominant trait, those affected will all be heterozygous for the trait; that is, the trait will be conditioned by a single defective autosomal gene, derived from one parent. But when such a trait is recessive, all those showing it will be homozygous for it; that is, the trait will be conditioned by both members of the pair of defective genes, one derived from each parent."

My 17 cases show no evidence whatever of being hereditary — all pedigrees given (38, 39, 40 and 190) being familial in type. Dr. G. H. Hogg (1936), of Launceston, Tasmania, in a personal communication, says that he is of the opinion that there are several cases of retinitis pigmentosa related to one another in Northern Tasmania, but that he has not tabulated them to date, and he is unable to give me details.

ECONOMIC SIGNIFICANCE.

On closely examining the first 250 pedigrees of this disease given by Julia Bell (1932), I find the following facts:—

Consanguinity — 95 pedigrees.

No consanguinity — 102 pedigrees.

No record of consanguinity — 53 pedigrees.

Total of accurate records — 197 pedigrees.

Percentage of consanguinity in 197 accurate records = 48.5%.

And so it appears that in 197 pedigrees 48.5% could have been prevented by stricter laws controlling consanguinity, as at the present time there is no dependable treatment for this progressive and disabling malady, the obvious mode of prevention lies in revised legislation.

J. B. S. Haldane (1936) quotes Usher's cases when asserting that 27 per cent. of retinitis pigmentosa cases are due to consanguinous marriages; and even at this figure legislation is urgently required. One may be here permitted to again quote Lord Byron:—

> "Marrying their cousins, nay their aunts and nieces,
> Which always spoils the breed, if it increases."
>
> Don Juan, Canto 1.

EDUCATION.

This should be pursued along normal lines with regular ophthalmological examinations (Report of the Committee on Partially Sighted Children, 1934), until such time as the ophthalmic surgeon considers admission to a sight-saving class or blind institution advisable. But the above Report stresses the necessity of repeated and close observation of the vision, fields and fundi of each case. From the onset these patients should never be allowed abroad alone between sunset and sunrise, as their disability renders them unsafe in busy traffic at night.

TREATMENT.

Barrett (1934) and Royle (1930 and 1932) both report retardation of this disease from cervical sympathectomy, while Meighan (1935) and Walsh (1935) each report three failures from the same operation. In the discussion which followed this paper by Meighan, Hepburn expressed the opinion that early cases might derive more benefit from the operation — a point stressed by Barrett (1934) and Kerr (1935). I have had one patient in this series of 17 operated on the right side only. She was a very early case, aged ten years, with an incomplete ring scotoma in the right field. I watched the case for three years to confirm the fact that it was progressive, and when the pigment commenced to take on a definite bone-corpuscle formation, and when the field at the same time began to show early scotoma, I advised operation.

The operation produced a right Horner's syndrome, but I could not detect any increase in the calibre of her right retinal vessels nor could Walsh (1935) in his three cases. After ten years both fields of vision show slow progressive deterioration, and both fundi increase in pigmentation. The deterioration was greater on the side of the operation, which was disappointing.

To date I have not tried acetylchlorine on any patient with retinitis pigmentosa.

ALLIED DISEASES.

With regard to the other diseases of this group, namely, retinitis pigmentosa sine pigmento, retinitis punctata albescens, gyrate atrophy of choroid and retina, stationary night blindness, and choroideremia. Two cases of retinitis punctata albescens combined with retinitis pigmentosa were seen in this series of 17 cases, and there was also one separate case of retinitis pigmentosa sine pigmento. So few cases of allied diseases are only to be expected, as, in the British Isles, with a population 200 times that of Tasmania, cases of these diseases are of very rare occurrence. (Milner, 1932.)

COMMENTS ON PEDIGREES.

Pedigree No. 38.

I/1 Male, aged 18 years. Not affected.

II/1 Female, aged 23 years. Typical case with temporal island in right field, and moderate myopic astigmatism. Parents were first cousins.

II/5 Male, supposed to be affected but not examined.

Pedigree No. 39.

II/2 Female, aged 73 years. Right colloidal degeneration. Left — Blind from long standing perforating injury.

III/1 Female, aged 50 years. Fields reduced to fixation combined with complicated cataracts. Bilateral vision reduced to hand movements. Nerve deafness.

III/6 Female, aged 43 years. Fields reduced to fixation. Left eye still 6/18 with myopic astigmatism corrected. Chronic otitis media.

IV/6 Female, aged 10 years. Myopic astigmatism.
Fundi. Normal (dilated).

Pedigree No. 40.

III/1 Male, not examined. Supposed to be affected but no details.

III/5 Female, aged 43 years. Fields reduced to 10° right and left.
Complicated cataracts. Right and left vision with correction = 6/12.

III/6 Supposed to be affected but now dead.

Pedigree 41. Dr. Carter, of Launceston, was good enough to send me this pedigree. Every member has been examined by him and his comments are as follows:—

I/1 Male, aged 46 years. Normal.

I/2 Female, aged 44 years. A typical variety of retinitis pigmentosa. Slight field changes.

II/1 Male, aged 21 years. Typical case of retinitis pigmentosa.

II/2 Normal.

II/3 Female, aged 16 years. Very advanced case. Fields reduced to 10° from fixation.

II/4 Normal.

II/5 Female, aged 5 years. Typical advanced case. Fields contracted to 30° from fixation.

II/6 Normal.

Pedigree No. 190.

III/3 Female, aged 30 years. Blind at birth. Right and left vision equals hand movements. Right fundus shows gross retinitis pigmentosa combined with retinitis punctata albescens. Left eye shows gross keratoconus with gross corneal opacities, through which retinitis can just be detected.

III/6 Male, aged 14 years. Blind since birth. Right and left vision equals perception of light. Gross bilateral keratoconus with gross bilateral corneal opacities. Bilateral retinitis just seen, but exact type not defined.

This combination of retinitis pigmentosa and keratoconus is, I feel, most unusual and awaits elucidation.

REFERENCES.

ALLAN, W. (1937), Arch. of Ophthal. Vol. XVIII. Page 938.
BEDDELL, A. J. (1936), Brit. Med. Jl. No. 3,944. Page 296.
BELL, J. (1922), Treasury of Human Inheritance. Vol. II. Part I. Cambridge.
CARTER, J. L. R. (1945), Personal communication.
CRAWLEY, R. H. (1934), Report of enquiry into Problems of Partially-sighted Children.
DICKSON and MITCHELL (1927), Supplement to Med. Jl. (Aust.). Dec. 3rd. Page 462.
HALDANE, J. B. S. (1936), Brit. Med. Jl. Feb. 8th. Page 281.
HILLER, B. (1945), Personal communication.
HOGG, G. H. (1936), Personal communication.
MACKLIN, M. T. (1927), Cand. Med. Ass. Jl. Vol. XVII. Page 1,341.
MILNER, J. G. (1932), Brit. Jl. Ophthal. Vol. XVI. Page 418.
RIDLEY, F., and SORSBY, A. (1940), Modern Trends in Ophthalmology. London.
SORSBY, A. (1934), Trans. Ophthal. Soc. of U.K. Vol. LIV. Page 160.
USHER, C. H. (1935), Trans. Ophthal. Soc., U.K. Vol. LV. Page 164.

Treatment
BARRETT, J. (1934), Trans. Aust. Med. Congress. Page 206.
CAMPBELL, GLEN (1931), Personal communication (Vancouver, Canada).
KERR, H. H. (1935), Amer. Jl. of Surg. Vol. XXVIII. Page 364 (abstract).
MEIGHAN, S. S. (1931), Trans. Ophthal. Soc., U.K. Vol. LI. Page 124.
MEIGHAN, S. S. (1935), Trans. Ophthal. Soc., U.K. Vol. LV. Page 93.
ROYLE, N. D. (1930), Med. Jl. Aust. Sept. 13th. Page 364.
ROYLE, N. D. 1932), Med. Jl. Aust. July 23rd. Page 111.
WALSH, F. B. (1935), Arch. of Ophthal. Vol. XIV. Page 699.

Laurence-Moon-Biedl Syndrome.
GRIFFITHS, G. M. (1938), Jl. of Neurology and Psychiatry. Vol. 1. No. 1.
PATON, L. (1936), Proc. Royal. Soc. Med. Vol. XXIX. Page 751.
SAVIN, L. H. (1935), Brit. Jl. Ophthal. Vol. XIX. Page 597.
SORSBY, A. (1932), Brit. Jl. Ophthal. Vol. XVI. Page 727.

SARCOMA OF THE CHOROID.

R. T. Davenport (1927) has shown very clearly that over a 55 year period at the Royal London Ophthalmic Hospital (1871-1925) there were 2 per 10,000 cases of sarcoma of the uvea, and, during those 55 years, the incidence was slightly decreasing rather than increasing.

In Tasmania during the last 15 years (1930-1945) I have seen 11 cases of sarcoma of the choroid confirmed by microscopic section, and, in another there was a possible sarcoma of the ciliary body, but owing to the objection of the patient's relatives to further examination, a final diagnosis was never made. Therefore I must confine my remarks to 11 cases of undoubted sarcoma of the choroid.

GEOGRAPHICAL DISTRIBUTION.

During this 15 year period I have seen 9,980 private patients, so that the incidence, as far as I am concerned, is 11.2 per 10,000 patients, which is almost six times higher than the incidence found by Lawford and Collins (1891), Devereaux Marshall (1903) and Davenport (1927b). There is no doubt that this case incidence appears alarmingly high, but even were I to eliminate three patients, who were referred to me by other ophthalmic surgeons for a second opinion, my total would be eight cases in 9,980 examinations. This makes an incidence of 8.1 cases in 10,000, which is still more than four times as high as the figures from the Royal London Ophthalmic Hospital. If we survey Deveraux Marshall's figures, we find that 20 of his 58 cases arose from the ciliary body, and not from the choroid, but in every one of my patients the sarcoma arose in the choroid alone. Therefore, if all cases of sarcoma of the ciliary body are removed from Deveraux Marshall's figures, it makes his incidence in the vicinity of 1.3 cases of sarcoma of the choroid in 10,000 examinations, as against my second figure of 8.1 per 10,000.

Though this comparison gives no explanation, it does support H. M. Traquair's contention that an investigation of the geographical distribution of eye diseases would reveal some most interesting results, and it even appears that the distribution of choroidal sarcoma in Tasmania varies greatly. J. R. L. Carter informs me in a personal communication that, in the past ten years in Launceston (where he is the only ophthalmic surgeon), he has seen only two such cases. During that period he has seen 10,455 patients with eye diseases. This brings the Launceston incidence to 1.94 cases per 10,000 examinations, which resembles the London figure closely. (See Table XVIII.)

Why there should be such a vast difference between the figures for Southern and Northern Tasmania is quite inexplicable. The populations of the North and South of this island are roughly the same; namely, 120,000 north and south of a line dividing the island horizontally. We do know that the type of thyrotoxicosis seen in Southern Tasmania has very different features to the type seen in Northern Tasmania, but I know of no figures to indicate that malignant disease (for instance in the breast, stomach or uterus) varies in these two districts.

SEX INCIDENCE.

It is pointed out by Davenport (1927b) that over a 55 year period in London, there was a predominance of females over males with sarcoma, and, in his last 35 cases, no less than 65.6% were females. On the other hand, in my cases, exclusively from Southern Tasmania, no less than eight were males and three were females, giving a male percentage of 73%, so that here again we find that the sex incidence of sarcoma of the choroid in the Antipodes, or at least in Southern Tasmania, varies greatly from that found in London. But, if we turn to Dr. Carter's Launceston figures, we find two females only, which is again an inexplicable figure. (See Table XIX.)

AGE OF ONSET.

Again, if we take the 55 year period in England; namely, 1871 to 1925, the average age of onset of sarcoma of the uveal tract was 51 years, while in Tasmania the figure for my 11 cases over a 15 year period stands at 53. My two youngest patients were a male and a female, both 31 years, with a pathological diagnosis of spindle cell sarcoma. My oldest case was a female aged 74 years.

CERTAIN ALLIED CONDITIONS.

(a) *Glioma of the Retina.*

During the last 15 years I have seen one case of glioma of the retina, although I have seen another patient whose eye was removed in 1930 for this condition, but I did not actually see the eye at the time of the first examination. That is a case incidence of one in 9,980 examinations. (See Table XX.)

(b) *Metastatic Carcinoma of the Uvea.*

I have not encountered any metastatic carcinomas of the uveal tract, although, since the report of cases in 1932 and 1933 by Foster Moore and Stallard, I have diligently searched for them.

(c) *Sarcoma of the Iris.*

Only one case has been seen by me in the past fifteen years.

(d) *Pigmented Naevi of the Conjunctiva and Lids.*

Although I have records of 24 cases of pigmented naevi of these sites, I have no evidence that any of them became malignant. On the other hand, I have seen no conjunctival or lid malignancies (other than rodent ulcers of the lid) during the past 15 years.

(e) *Melanoma of the Iris.*

I have only ten cases to report, and, so far as I know, none were progressive. All had been present for many years when I saw them.

(f) *Melanoma of the Choroid.*

Doherty, quoted by Terry (1939), estimates that 25% of patients with ocular melanosis ultimately show malignant choroidal tumour. On the other hand, Juler and Law (1936) quote Francois as saying that, in only five cases out of his 110 collected cases of ocular melanosis, was there an appearance of malignancy. Coates, quoted by Collins (1926), found melanotic sarcoma in seven of 26 cases of ocular melanosis.

In one of my eleven cases of choroidal sarcoma, it appears as if this choroidal sarcoma arose from a melanoma, especially as the patient had bilateral ocular melanosis, and his brother also has a choroidal melanoma. I have records of 44 examples of choroidal melanoma in my 9,980 cases but, to my knowledge, none has become malignant.

INHERITANCE.

The part which inheritance plays in this condition seems still to be somewhat obscure, but Davernport (1927a) has reported a most extensive pedigree. I have only one pedigree to report, namely, No. 89, in my Tasmanian series. The interesting part about Pedigree No. 89 is the fact that in the past 15 years (1930-45) I have seen only three females in Southern Tasmania with choroidal sarcoma, and two of these are included in this pedigree. Both cases exhibited pigmented tumours, and, like Davernport's pedigree, the disease appeared earlier in the second than the first generation. In fact, in Davernport's pedigree, the anticipation by the fourth generation had become so extreme that the tumour in one instance appeared at 19 years of age, and in the second instance at 29 years.

COMMENTS ON PEDIGREES.

Pedigree No. 89.

III/1 Female, aged 74 years.
Large fungating mass protruding between left lids. Mass was involving whole of globe, and was deeply pigmented. Exenteration of orbit was performed and radium applied. Patient died five months later from secondaries.
Pathological section: Tumour was melanotic sarcoma. Cells were large, spindle shape, and very heavily pigmented.

III/10 Male, aged 69 years. With normal fundi and distant vision.

III/11 Female, aged 52 years. Fundi. Normal (dilated).

IV/23 Female, aged 31 years. Sarcoma of the choroid (left). Left eye enucleated. Patient died six years later from secondaries in her liver. Pathological section: Pigmented spindle cell sarcoma. Small mass of tumour cells in vein within sclerotic. No visible extension of the tumour to the outside of the sclerotic.

IV/24 Male, aged 45 years. Right and left vision = 6/4. Has shallow right retinal detachment from 7-8 in periphery.

V/7 Female, aged 6 years. Fundi and vision normal.

V/10 Male, aged 7½ years. Vision normal. Fundi not examined.

TABLE XVIII.

GEOGRAPHICAL INCIDENCE.

	Northern Tasmania	Southern Tasmania	England
No. cases seen	10,455	9,980	?
No. cases sarcoma	2	11	?
Cases per 10,000	1.94	11.2	2

TABLE XIX.

SEX INCIDENCE.

	Northern Tasmania	Southern Tasmania	England
No. cases sarcoma	2	11	35
Males %	0%	73%	34.4%
Females %	100%	27%	65.6%

TABLE XX.

TASMANIAN ALLIED CONDITIONS.

DISEASE—	No. Cases
Glioma retinae	1
Metastatic carcinomata of the uvea	0
Sarcoma of the Iris	1
Pigmented naevi of the conjunctiva and lids	24
Melanomata of the Iris.	10
Melanomata of the choroid	44

REFERENCES.

CARTER, J. R. L. (1945), Personal communication.
COLLINS, E. T. (1926), Trans. of Ophthal. Soc. of U.K. Vol. XLVI. Page 86.
DAVENPORT, R. C. (1927a), Brit. Jl. of Ophthal. Vol. XI. Page 443.
DAVENPORT, R. C. (1927b), Brit. Jl. of Ophthal. Vol. XI. Page 609.
DUNNINGTON, J. H. (1938), Arch. of Ophthal. Vol. XX. No. 3. Page 359.
DVORAK-THEOBOLD, G. (1937), Arch. of Ophthal. Vol. XVIII. No. 6. Page 871.
HALL, G. S. (1940), Quart. Jl. of Med. Vol. IX. No. 33. Page 1.
HAMILTON, J. B. (1940), Jl. of the Old Sydney Hospitaller's Club. Dec., 1940. Page 16.
HAMILTON, J. B. (1948), Trans. Ophth. Soc. of New Zealand. Sup. to N.Z. Med. Jourl. Page 28.
JULER, F. A., and LAW, F. W. (1936), Trans. Ophthal. Soc of U.K. Vol. LVI. Page 121.
MARSHALL, T. D. (1903), Royal London Ophthal. Hosp. Reports. Vol. XV. Page 51.
MOORE, R. F., and STALLARD, H. B. (1932), Brit. Jl. of Ophthal. Vol. XVI. Page 532.
RONNE, H. (1936), Trans. Ophthal. Soc. of U.K. Vol. LVI. Page 270.
STALLARD, H B. (1933), Proc. Roy. Soc. Med. Vol. XXVI. Part II. Page 1,043.
TERRY, T. L. (1939), Arch. of Ophthal. Vol. XXII. No. 6. Page 989.
THIEL, R. (1939), Die Bosartigen Geschwulste des Auges und seiner Umgebung, Stuttgart.

STRABISMUS.

There is no doubt that strabismus stands pre-eminent as a cause of monocular blindness. The loss of monocular vision in itself is not so very devastating, nor is the loss of binocular fusion, but with only one eye functioning, all squinters are in a precarious position, and any injury or disease to the non-squinting eye may lead to blindness. To the lay mind the cosmetic effect is paramount, but to ophthalmologists, the acquired amblyopia is the main issue, or, at least, it should be. Claude Worth's name must be honoured, for he constantly urged that permanent amblyopia ex anopsia could be avoided if taken in hand before the patient reached the age of seven. Gradually the old practice of allowing squinters to grow out of their squints has passed away, and parents are now bringing their children to oculists at a much earlier age. Even in the past 15 years a great awakening has occurred in the parents of Tasmania, so that some bring their children for advice before they

are twelve months old. I feel that this has been largely brought about by the introduction of orthoptic exercises, for, as Parsons (1936) says, "It (orthoptic training) has one overwhelming argument in its favour, *viz.*, that when it is successful, it cures the squint." Pugh (1936) and Travers (1936) substantiate this view. Most parents are loath to submit their young offspring to operations, but will submit them to orthoptic training almost indefinitely if they observe even slow progress. It appears, therefore, that amblyopia ex anopsia in adults will have almost vanished in the next generation, and thus another milestone in the prevention of blindness will have been passed.

HEREDITARY FACTOR AND MODE OF TRANSMISSION.

With the increase in patients seeking early advice, the hereditary nature of strabismus has become more apparent (Mayou, 1935), and, in many families, a history of cousins, suffering from the same complaint, can be frequently elicited. In practically every instance the mode of transmission is recessive, nor does there appear to be any sex linkage. Czelletzer, quoted by Travers (1936), asserts that the transmission depends on two recessive Mendelian factors. Worth (1929) found amongst 1,373 cases of squint a history of squint in either parent, grand parent, brother or sister of the patient in no less than 711; *i.e.*, about 51 per cent., while Franceschetti (1930) believes that heredity plays but a small part in the causation of concomitant squint. In Tasmania, out of 1,502 cases of heterotropia and heterophoria, I found 223 cases with an hereditary history. This gives only a 15% incidence, which is much lower than Worth's figures.

SEX INCIDENCE.

There seems to be a preponderance of females in both the heterotropias and heterophorias in my pedigrees. In the cases of hereditary heterotropias I find 55 females and 45 males; *i.e.*, 55%, and 45% respectively. While in my cases of hereditary heterophorias I find 77 females and 46 males; *i.e.*, 62.7%, and 37.3% respectively. In the available literature I am unable to find any reference to this factor.

TREATMENT.

There is nothing specific in the treatment of hereditary squints, but Lyle and Jackson (1940) consider "a strong family history of squint makes the prognosis less good." Personally, I feel that if one of the parents themselves have a squint, then the parental understanding and co-operation is more satisfactory, which is a great help to the oculist in every branch of his work. (Hamilton, 1936, 1940, 1941.)

Relation of strabismus to concurrent refractive errors. It should be noted throughout the comments on the following pedigrees that, in those recording esophoria, exophoria and exotropia, the expected corresponding refractive error is by no means always exhibited. In fact, often the reverse is found, so that, at least, in inherited strabismus, there is no constant relation between the refractive error and the muscle error.

COMMENTS ON PEDIGREES.

Esophoria.

Pedigree No. 42.

I/2 Female, aged 54 years. Hypermetropia, esophoria.

II/3 Female, aged 31 years. Hypermetropic astigmatism and esophoria.

Pedigree No. 198.

I/2 Female, aged 35 years. Myopic astigmatism and esophoria.

II/1 Female, aged 6 years. Hypermetropia and esophoria.

Pedigree No. 200.

I/1 Male, aged 52 years. Mixed astigmatism, hypermetropic astigmatism, anisometropia and esophoria.

I/2 Female, aged 44 years. Not affected with esophoria, but has myopic astigmatism.

II/1 Male, aged 12 years. Myopia, anisometropia and esophoria.

Exophoria.

Pedigree No. 44.

I/2 Female, aged 55 years. Hypermetropic astigmatism and exophoria.

II/4 Female, aged 16 years. Myopic astigmatism and exophoria.

Pedigree No. 45.

II/1 Male, aged 57 years. Myopic astigmatism and exophoria.

II/2 Female, aged 45 years. Mixed astigmatism and right hyperphoria.

III/3 Female, aged 16 years. Myopic astigmatism, exophoria and right hyperphoria.

Pedigree No. 79.

II/4 Female, aged 61 years. Myopic astigmatism, exophoria and left hyperphoria.

III/7 Female, aged 27 years. Myopic astigmatism, exophoria and left hyperphoria.

III/8 Female, aged 23 years. Myopic astigmatism and exophoria.

III/10 Female, aged 11 years. Myopic astigmatism and exophoria.

Pedigree No. 106.

I/1 Female, aged 67 years. Hypermetropic astigmatism and exophoria.

II/1 Female, aged 49 years. Hypermetropic astigmatism and exophoria.

Pedigree No. 152.

II/10 Female, aged 49 years. Hypermetropic astigmatism and exophoria.

III/19 Male, aged 50 years. No refractive error, exophoria.

III/21 Female, aged 48 years. Mixed astigmatism and exophoria.

IV/1 Male, aged 16 years. Not refracted, exophoria.

IV/9 Female, aged 19 years. No refractive error, exophoria and right hyperphoria.

IV/12 Female, aged 16 years. Not refracted, exophoria.

IV/13 Male, aged 11 years. Not refracted, exophoria.

IV/16 Female, aged 22 years. Not refracted, exophoria.

Pedigree No. 164.

I/3 Female, aged 30 years. No refractive error, exophoria and hyperphoria.

II/1 Male, aged 21 years. Hypermetropic astigmatism, exophoria.

II/2 Female, aged 18 years. Myopic astigmatism, exophoria with keratoconus.

Pedigree No. 166.

II/1 Female, aged 78 years. Hypermetropic astigmatism and exotropia. This patient has monocular blindness due to choroiditis.

III/1 Female, aged 52 years. Hypermetropic astigmatism and exophoria. This patient was the propositus.

III/2 Female, reported by propositus to be affected.

III/4 Male, aged 53 years. No refractive error, exophoria.

Pedigree No. 174.

III/1 Male, aged 59 years. Mixed astigmatism, exophoria.

III/2 Female, aged 53 years. Hypermetropic astigmatism and exophoria.

III/5 Male, aged 48 years. Hypermetropic astigmatism and exophoria.

III/9 Male, aged 47 years. No refractive error or exophoria.

IV/1 Female, aged 24 years. No refractive error, exophoria.

IV/7 Male, aged 40 years. Not refracted, exophoria.

IV/9 Female, aged 36 years. Mixed astigmatism and exophoria.

IV/13 Female, aged 38 years. No refractive error, exophoria.

V/4 Female, aged 2 years. Myopia and exophoria.

Esotropia.

Pedigree No. 46.

I/1 Female, aged 57 years. Hypermetropic astigmatism, right esotropia and amblyopia ex anopsia.

II/1 Female, aged 34 years. Hypermetropic astigmatism, right esotropia and amblyopia ex anopsia.

Pedigree 47.

III/31 Female, aged 29 years. Hypermetropic astigmatism, right esotropia and amblyopia ex anopsia.

IV/1 Male, aged 6 years. Hypermetropic astigmatism, right esotropia and amblyopia ex anopsia.

IV/10 Female, aged 5 years. Hypermetropic astigmatism, left esotropia and amblyopia ex anopsia.

IV/13 Female, aged 7 years. Hypermetropic astigmatism, esotropia and amblyopia ex anopsia.

Pedigree No. 49.

IV/1 Male, aged 6 years. Hypermetropic astigmatism, left esotropia, amblyopia ex anopsia.

IV/2 Male, aged 2 years. Hypermetropic astigmatism, esotropia and amblyopia ex anopsia.

Pedigree No. 50.

III/10 Female, aged 6 years. Hypermetropic astigmatism, left esotropia, amblyopia ex anopsia.

III/11 Male, aged 6 years. Hypermetropia, alternating esotropia.

Pedigree No. 51.

III/1 Female, aged 5 years. Hypermetropic astigmatism, left esotropia, amblyopia ex anopsia.

III/5 Male, aged 5 years. Hypermetropic astigmatism, right esotropia, amblyopia ex anopsia.

Pedigree No. 52.

III/8 Female, aged 41 years. Hypermetropic astigmatism and esotropia.

IV/1 Male, aged 7 years. Hypermetropic astigmatism and left esotropia with amblyopia ex anopsia.

IV/2 Female, aged 3 years. Hypermetropic astigmatism, left esotropia, amblyopia ex anopsia.

Pedigree No. 157.

III/8 Male, aged 10 years. Hypermetropic astigmatism, left esotropia, amblyopia ex anopsia.

III/14 Male, aged 14 years. Hypermetropic astigmatism, left esotropia, amblyopia ex anopsia.

Pedigree No. 177.

II/3 Male, aged 29 years. Hypermetropic astigmatism, left esotropia, amblyopia ex anopsia.

III/5 Female, aged 2 years. Hypermetropic astigmatism, left esotropia.

III/7 Male, aged 3 years. Hypermetropia, left esotropia, amblyopia ex anopsia.

Pedigree No. 184.

III/4 Male, aged 2 years. Hypermetropic astigmatism, right esotropia, amblyopia ex anopsia.

III/5 Male, aged 5 years. Hypermetropia, left esotropia, amblyopia ex anopsia.

III/12 Female, propositus, reports esotropia which has been confirmed by an oculist.

Pedigree No. 191.

III/1 Male, aged 10 years. Hypermetropic astigmatism, left esotropia, amblyopia ex anopsia.

III/2 Female, aged 11 years. Right hypermetropia, left myopic astigmatism and esotropia.

Pedigree No. 226.

III/1 Female, aged 12 years. Hypermetropic astigmatism, esophoria.

III/4 Female, aged 5 years. Hypermetropic astigmatism, left esotropia, amblyopia ex anopsia.

Pedigree No. 229.

III/1 Male, aged 3 years. Hypermetropic astigmatism, left esotropia, amblyopia ex anopsia.

III/3 Male, aged 8 years. Hypermetropia, esophoria. This patient also has congenital nystagmus.

Exotropia.

Pedigree No. 204.

I/2 Male, aged 39 years. Mixed astigmatism, exophoria.

II/2 Female, aged 7 years. Hypermetropia, exotropia.

Hyperphoria.

Pedigree No. 43.

I/1 Male, aged 52 years. Hypermetropic astigmatism, exophoria, right hyperphoria.

II/1 Male, aged 10 years. Right hyperphoria. No refractive error.

II/2 Female, aged 6 years. Hypermetropic astigmatism. Esotropia, amblyopia ex anopsia.

III/1 Female, aged 3 years. Hypermetropic astigmatism, exophoria. No amblyopia.

REFERENCES.

CANTONNET, A. (1934), Strabismus, London.
DOBSON, M. (1933), Binocular Vision and Modern Treatment of Squint, London.
FRANCESCHETTI, B. (1930), Kurze, Hand. d. Ophthal. Page 685.
HAMILTON, J. B. (1936), Med. Jl. of Aust. Dec. 12th. Page 815.
HAMILTON, J. B. (1940), Aust. and N.Z. Jl. of Surgery. Vol. IX. Page 378.
HAMILTON, J. B. (1941), Med. Jl. of Aust. Feb. 22nd. Page 233.
LYLE and JACKSON (1940), Practical Orthoptics. 2nd edit. London.
MAYOU, S. (1935), Brit. Jl. Ophthal. Vol. XIX. Page 37.
PARSONS, J. H. (1936), Disease of the Eye. 8th edit. Page 558. London.
PETERS, A. (1924), Zeitschr. f. Augenheilk. Vol. LII. Page 369.
PETER, L. (1928), The Extra-ocular Muscles. 1st edit. London.
PUGH, M. A. (1936), Squint Training, London.
RODIN, F. (1934), Arch. of Ophthal. Vol. XII. Page 874.
TRAVERS, T. A'b. (1936), An Essay on Concomitant Strabismus, London.
WILKINSON, O. (1928), Strabismus, Its Etiology and Treatment. Page 57.
WORTH, C. (1939), Squint. 7th edit. London.

The SIGNIFICANCE of HEREDITY IN OPHTHALMOLOGY

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A TASMANIAN SURVEY

BY

J. BRUCE HAMILTON

•

PEDIGREES

TABLE XII

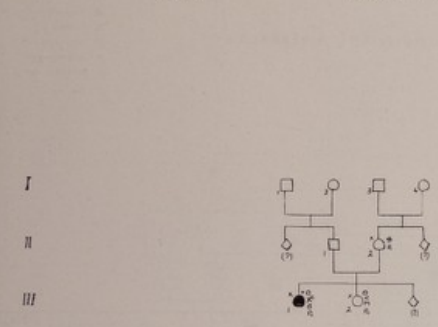
LIST OF PEDIGREES

(Angioid Streaks—Strabismus)

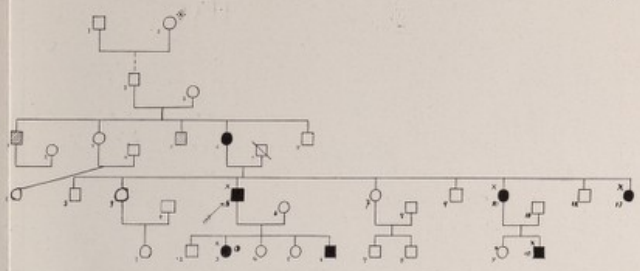
This table indicates the inherited eye diseases so far found in Tasmania, with the pedigree numbers against each disease. There are 18 diseases and 111 pedigrees.

DISEASE.	PEDIGREE NUMBERS.
ANGIOID STREAKS	No. 151
CATARACTS—CONGENITAL	Nos. 6, 7, 8, 9 18
CATARACTS—DYSTROPHIA MYOTONICA	No. 10
CATARACTS—SENILE	Nos. 87, 163, 217
COLOBOMA OF THE IRIS	No. 142
DACRYOCYSTITIS	Nos. 71, 107, 135, 143, 144, 145, 150, 176
GLAUCOMA	Nos. 11, 13, 14, 77, 91, 103, 108, 179, 203
KERATOCONUS	Nos. 15, 16, 222
MACULAR DEGENERATION—Senile Pigmentary	No. 118
NYSTAGMUS	Nos. 4, 27, 28, 29, 30
OPTIC ATROPHY—LEBER'S	Nos. 31, 33, 34, 88, 221
PTERYGIUM	Nos. 35, 141
PTOSIS	Nos. 36, 215, 216, 232
REFRACTIVE ERRORS—	
Hypermetropic Astigmatism	Nos. 5, 69, 91, 92, 96, 129
Mixed Astigmatism	Nos. 1, 2
Myopic Astigmatism	Nos. 20, 21, 22, 23, 25, 55, 63, 78, 95, 101, 124, 125, 140, 146, 148, 158, 162, 168, 169, 173, 180, 210, 213
RETINAL DETACHMENT	No. 37
RETINITIS PIGMENTOSA	Nos. 38, 39, 40, 41, 190
SARCOMA OF THE CHOROID	No. 89
STRABISMUS—	
Esophoria	Nos. 42, 198, 200
Exophoria	Nos. 44, 45, 79, 106, 152, 164, 166, 174
Esotropia	Nos. 46, 47, 49, 50, 51, 52, 157 177, 184, 191, 226, 229
Exotropia	No. 204
Hyperphoria	No. 43

No. 151 ANGEOID STREAKS



No. 6. CONGENITAL CATARACTS.



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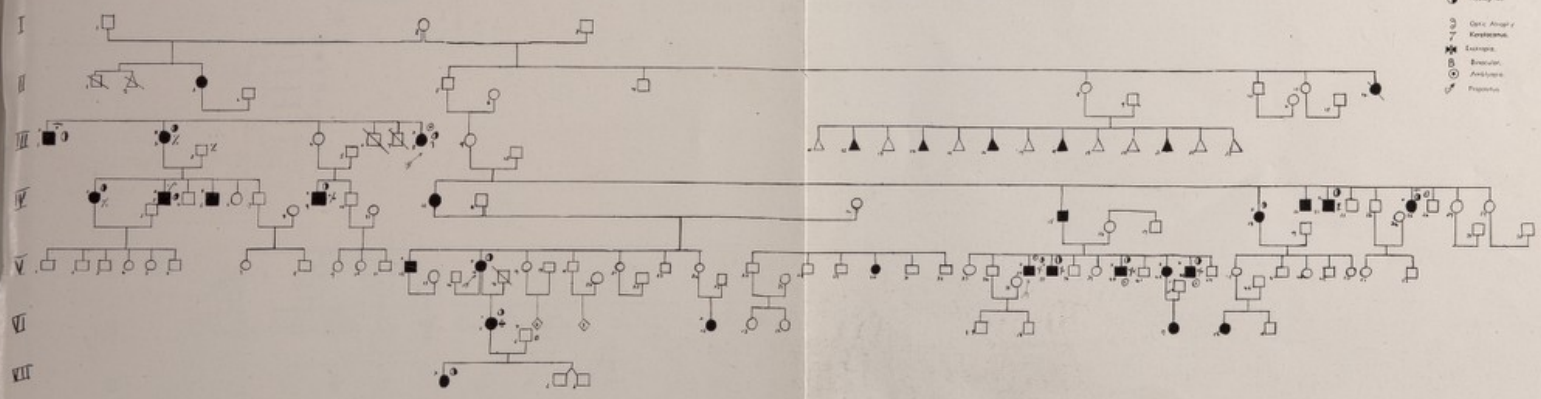
CONGENITAL CATARACT



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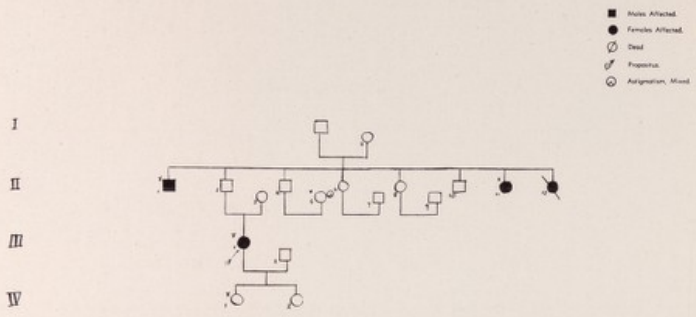
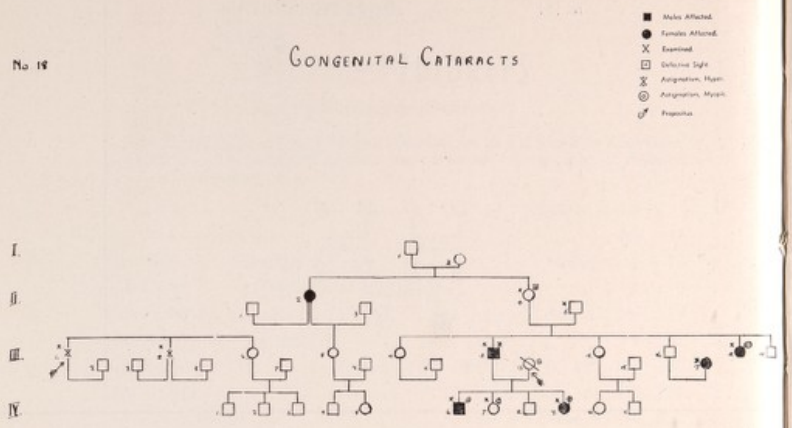
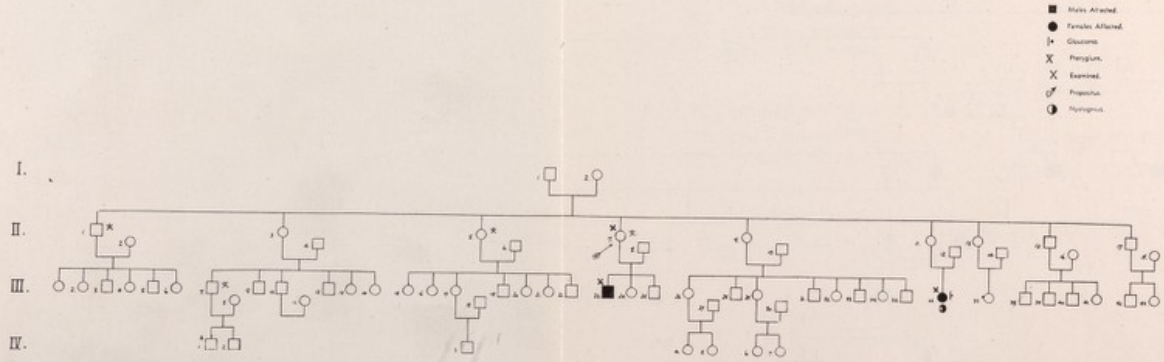


No 12

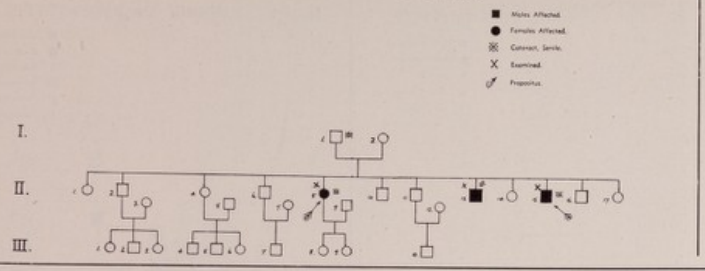
Congenital Cataracts



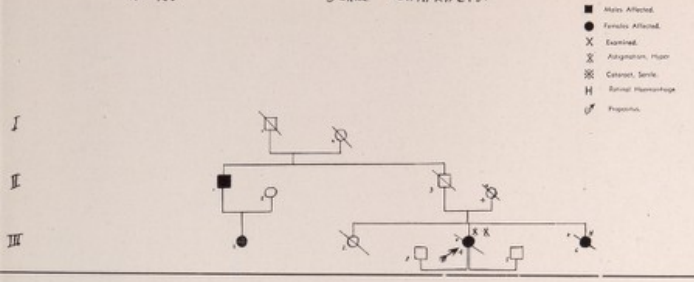
No 8 Congenital Cataract.



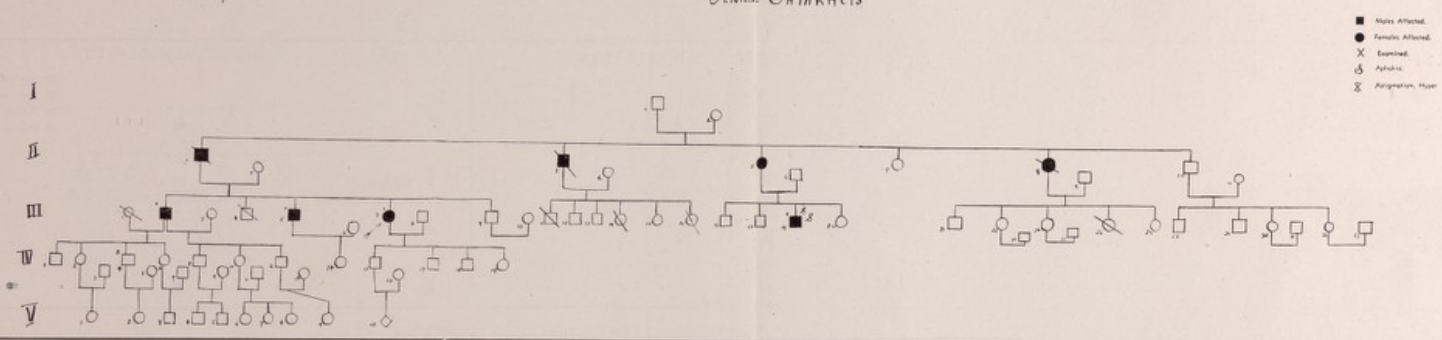
No. 10. DYSTROPHIA MYOTONICA.



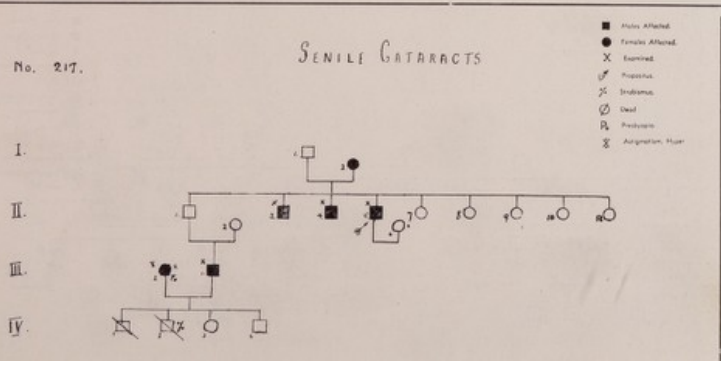
No 163 SENILE CATARACTS.



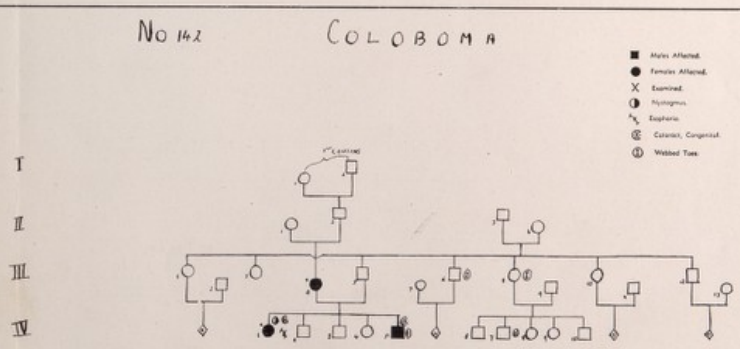
No 87 SENILE CATARACTS



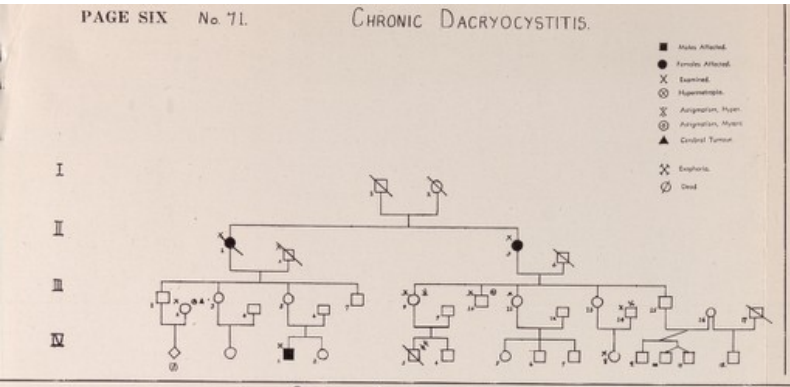
No. 217. SENILE CATARACTS



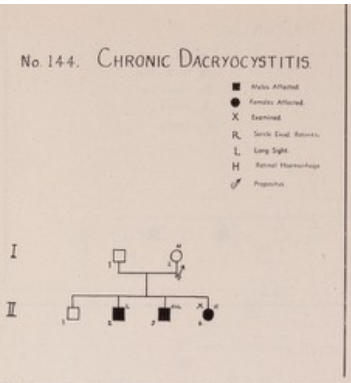
No 142 COLOBOMA



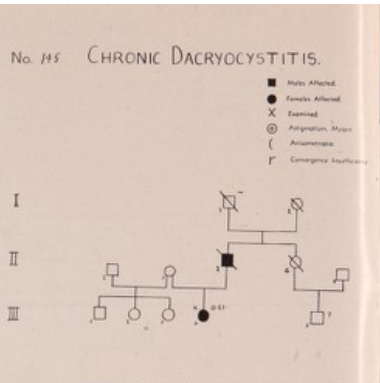
No. 71. CHRONIC DACRYOCYSTITIS.



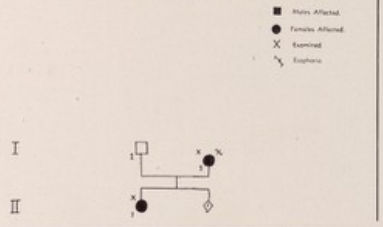
No. 144. CHRONIC DACRYOCYSTITIS.



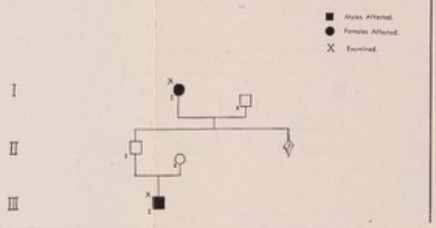
No. 145 CHRONIC DACRYOCYSTITIS.



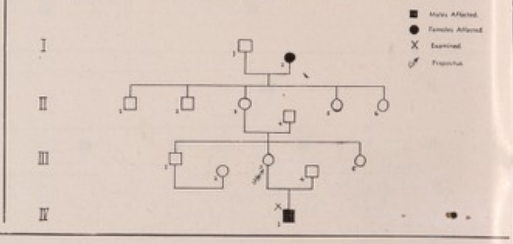
No. 143. CHRONIC DACRYOCYSTITIS.



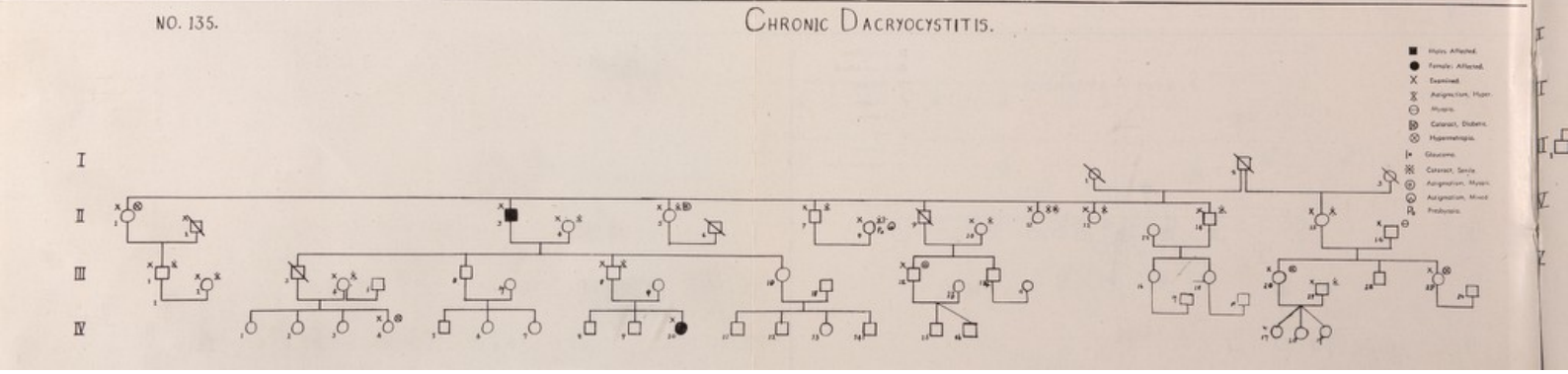
No. 107. CHRONIC DACRYOCYSTITIS.



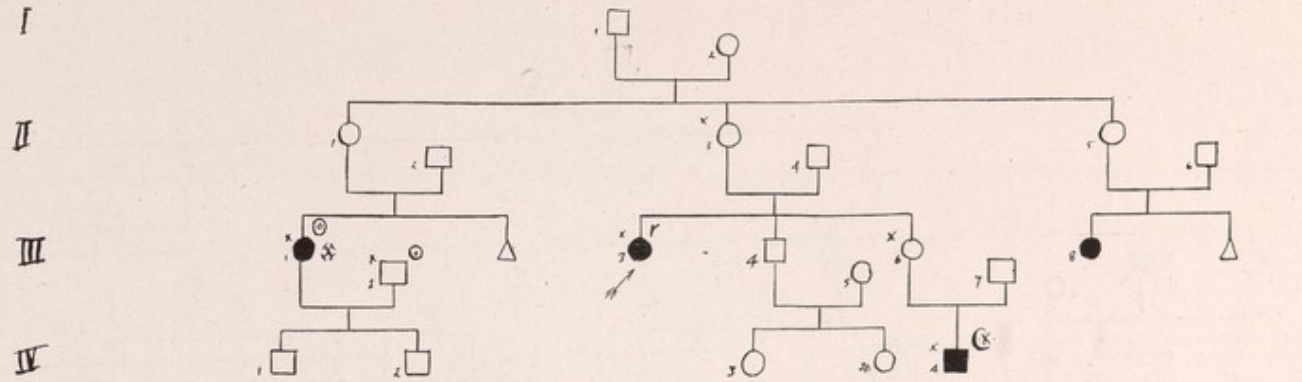
No. 150 CHRONIC DACRYOCYSTITIS.



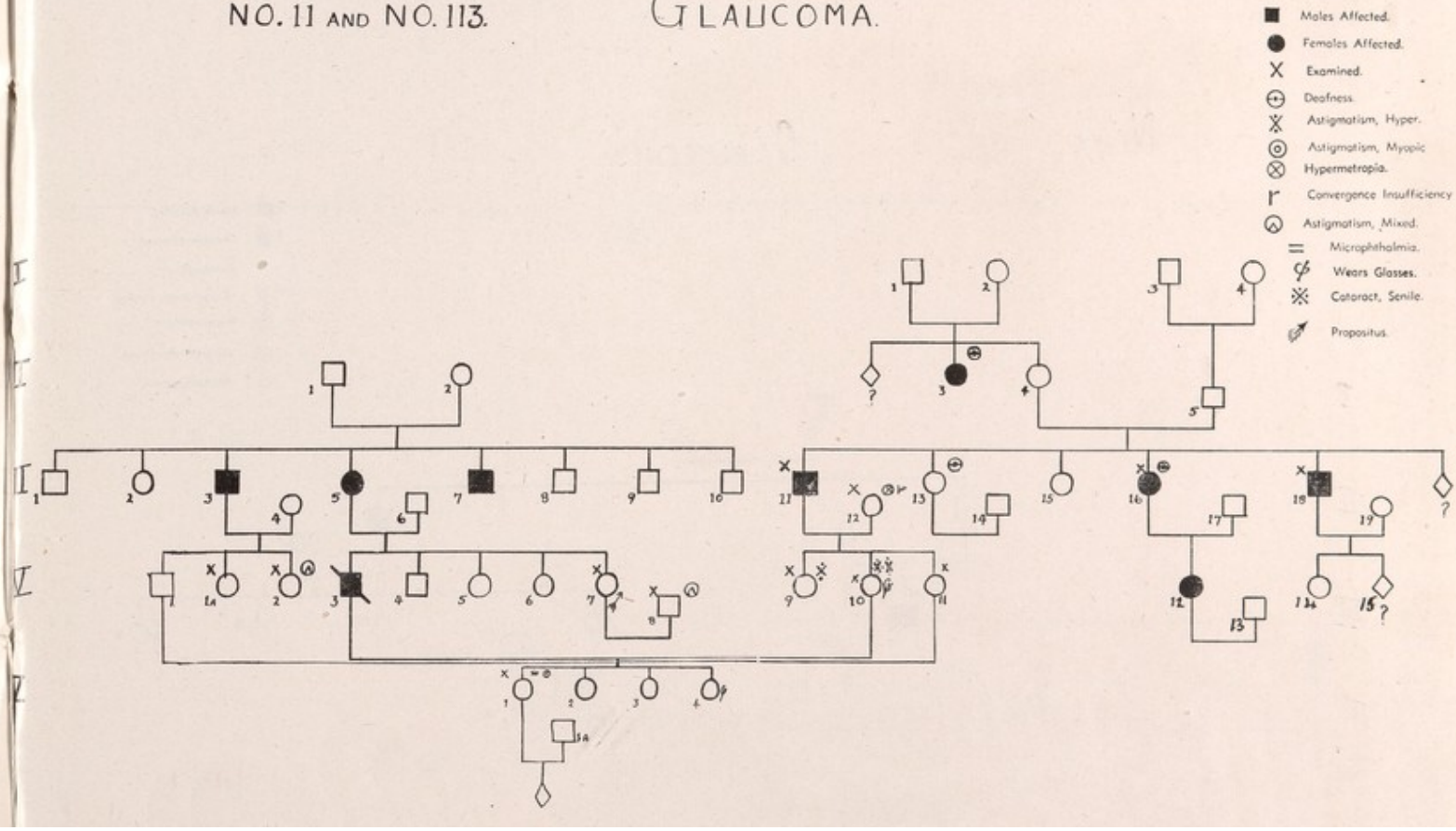
NO. 135. CHRONIC DACRYOCYSTITIS.



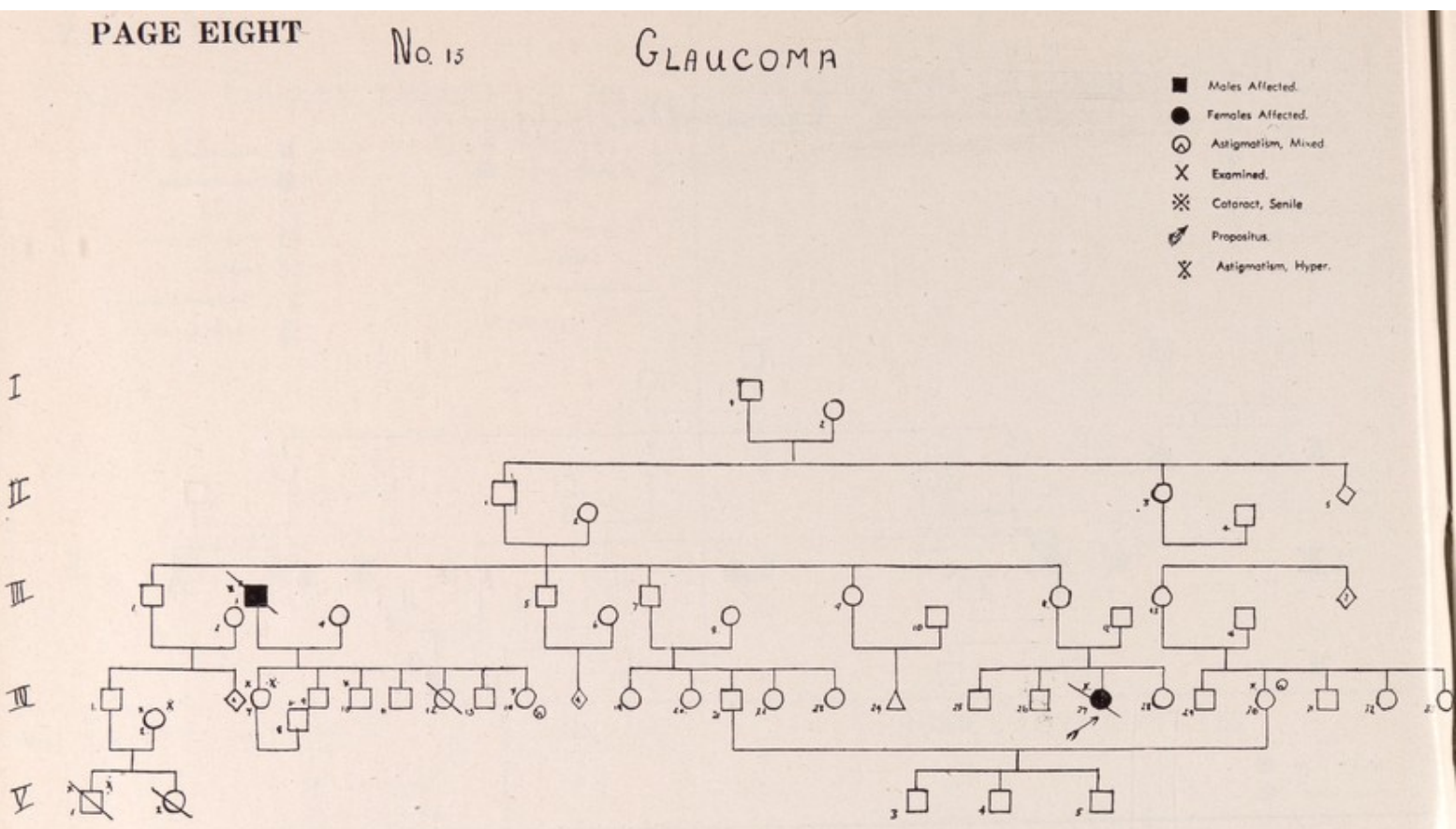
No. 176 Chronic Dacryocystitis



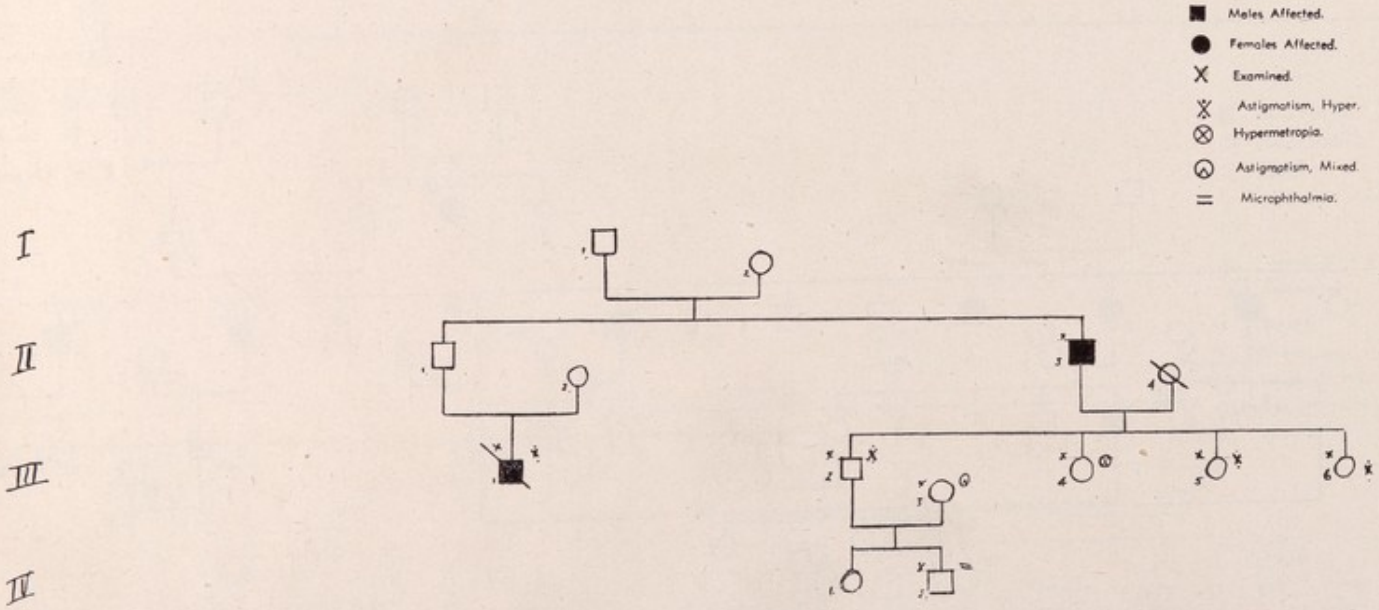
No. 11 and No. 113. Glaucoma.

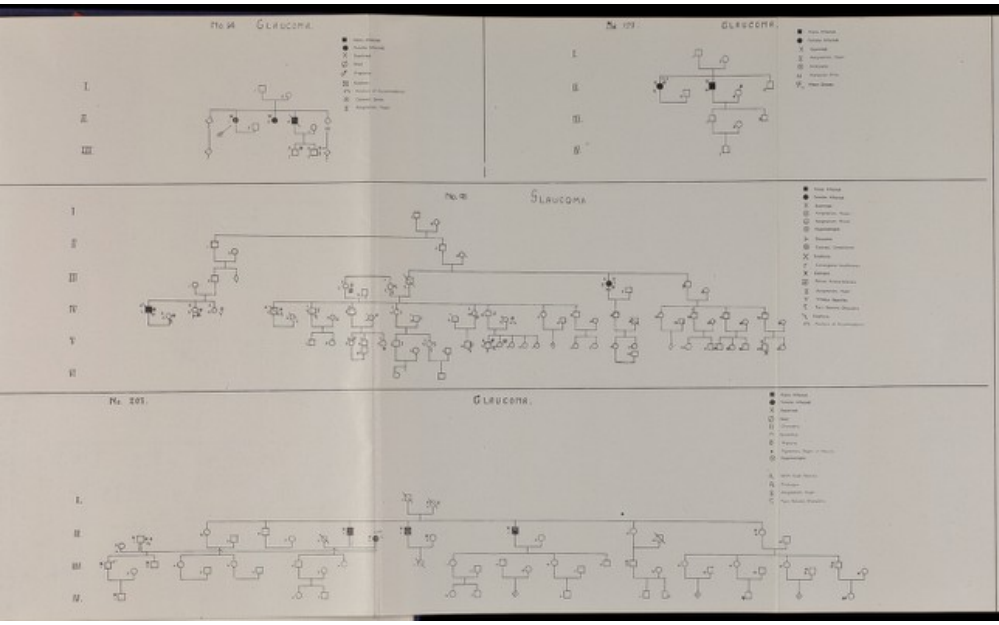


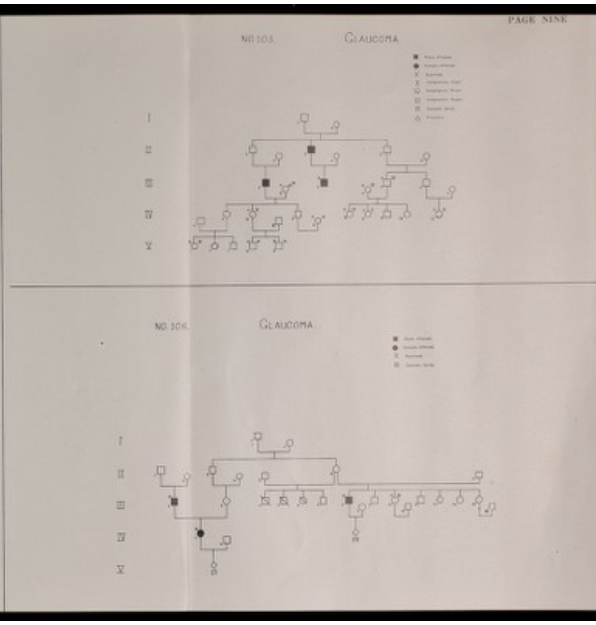
No. 15 GLAUCOMA



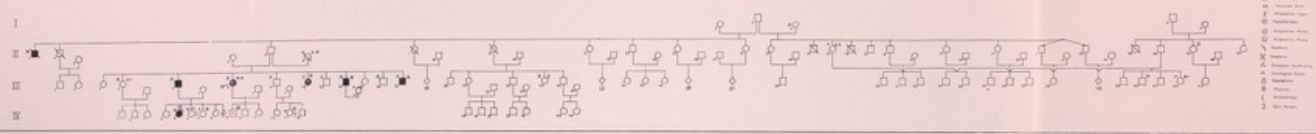
No. 77 GLAUCOMA



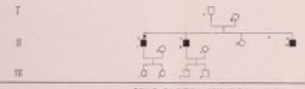

No 91 Glaucoma.
No. 101 Glaucoma.
No. 181 Glaucoma
No. 201. Glaucoma.


No. 103. Glaucoma
No. 301. Glaucoma

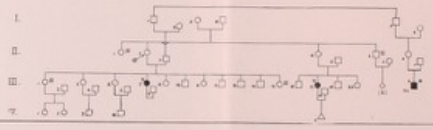
No. 15 KERATOCONUS.



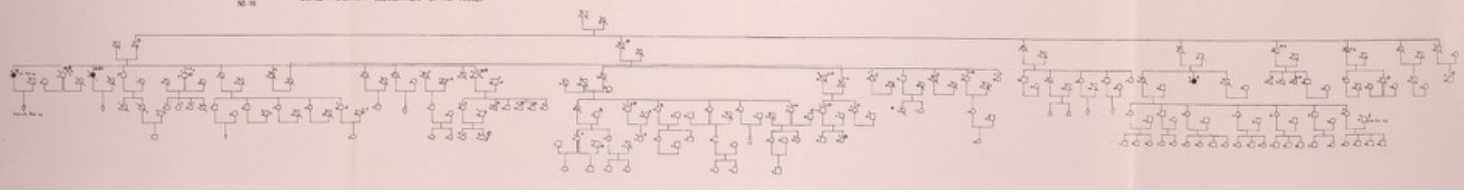
No. 222 KERATOCONUS.



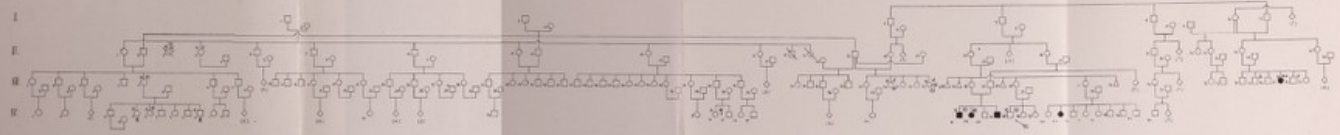
No. 16. KERATOCONUS.



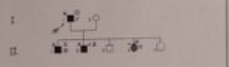
No. 19 SENILE PIGMENTARY DEGENERATION OF THE MACULA



No. 4. Nystagmus.



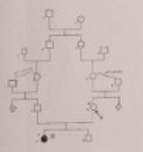
No. 22. Nystagmus.



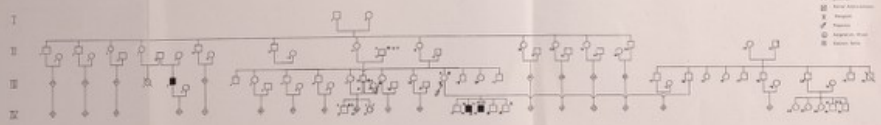
No. 21. Nystagmus.

Nystagmus.

No. 20

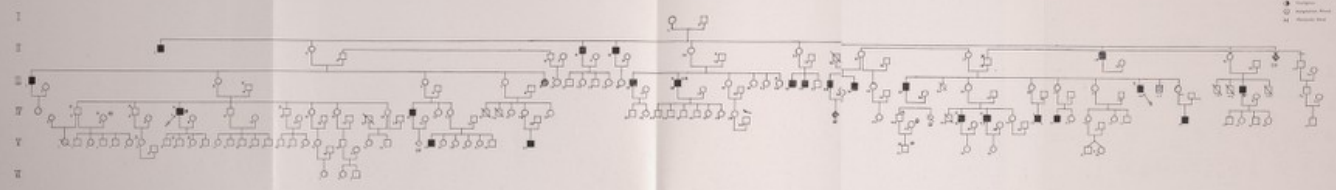


No. 19. Nystagmus.



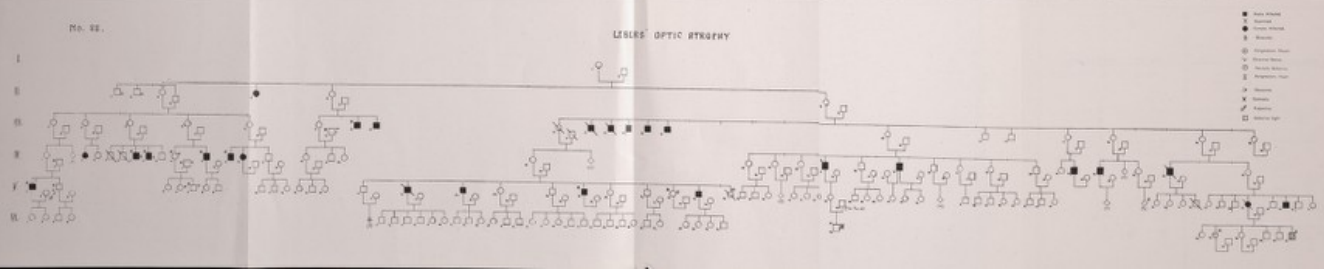
No. 31

Hereditary Optic Atrophy (Lebers Disease)

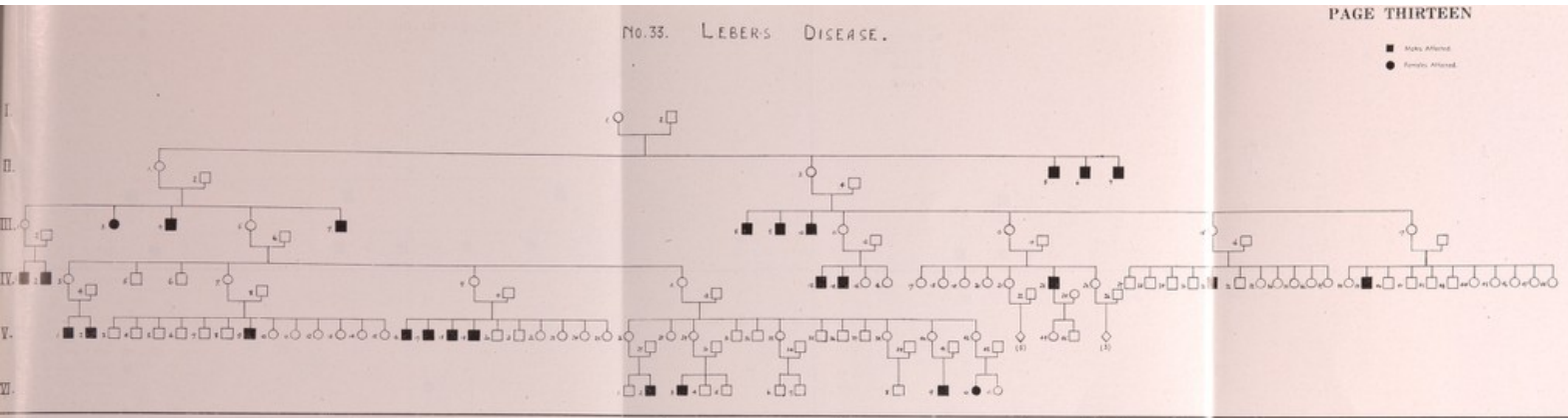


No. 32.

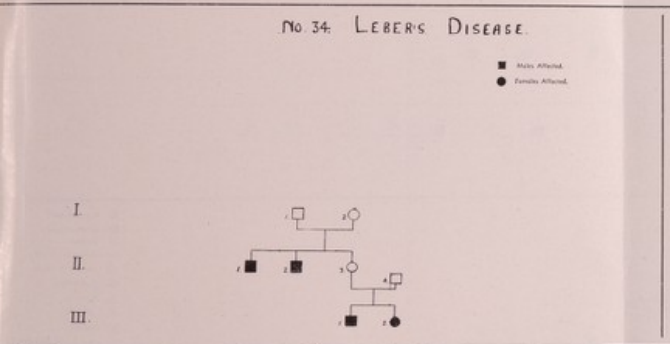
LESLERS OPTIC ATROPHY



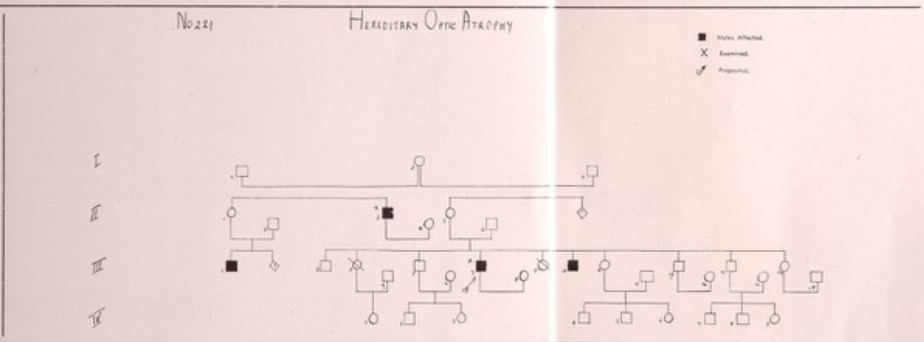
No. 33. Leber's Disease.



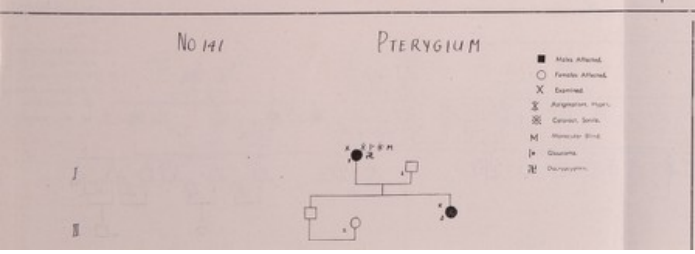
No. 34. Leber's Disease.



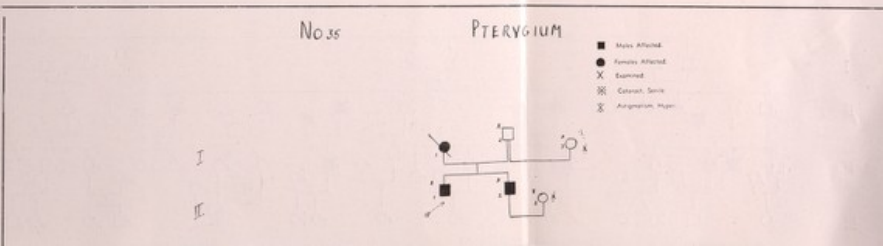
No 221 Hereditary Optic Atrophy



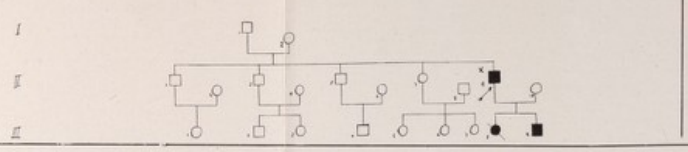
No 141 Pterygium



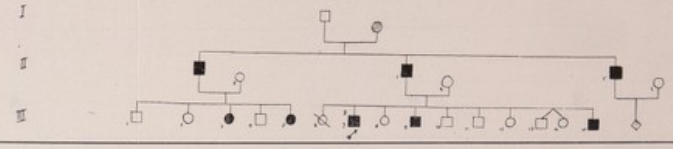
No 55 Pterygium



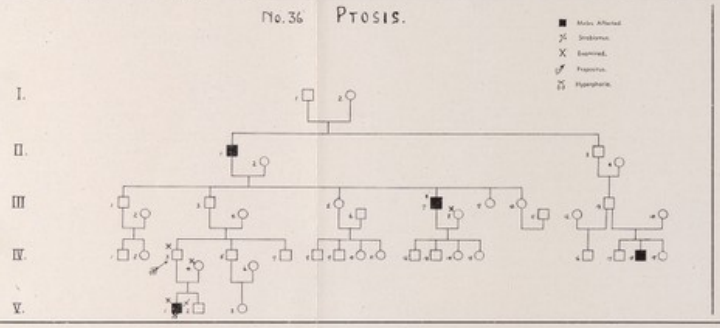
No 214 PTOSIS



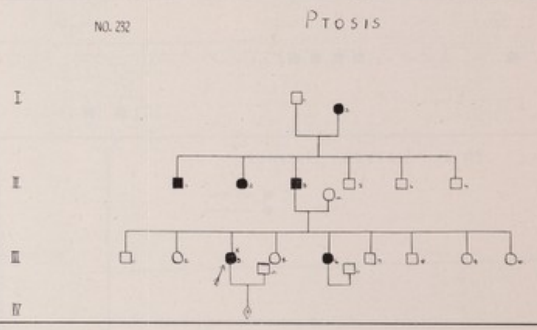
No 215 CONGENITAL PTOSIS



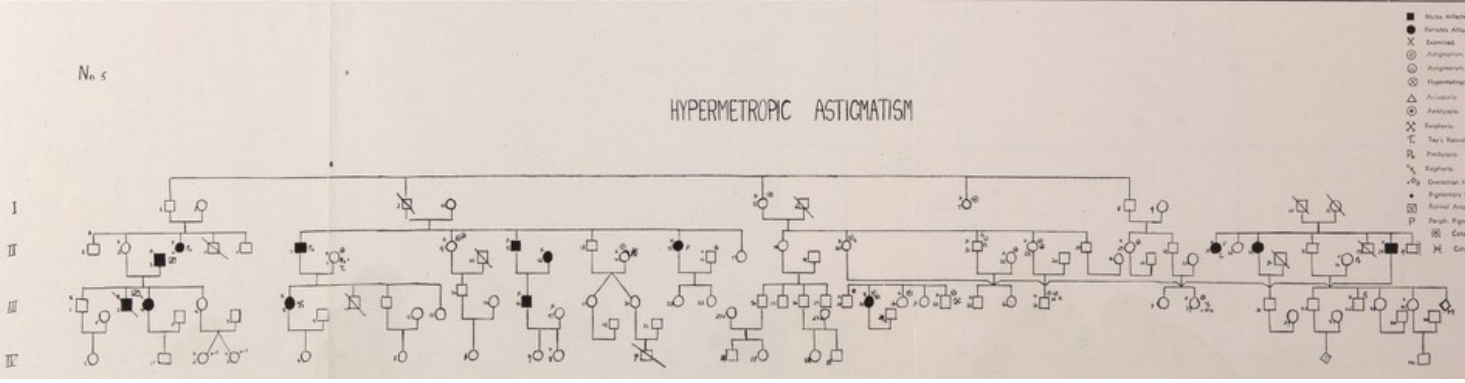
No. 36 PTOSIS.



No. 232 PTOSIS

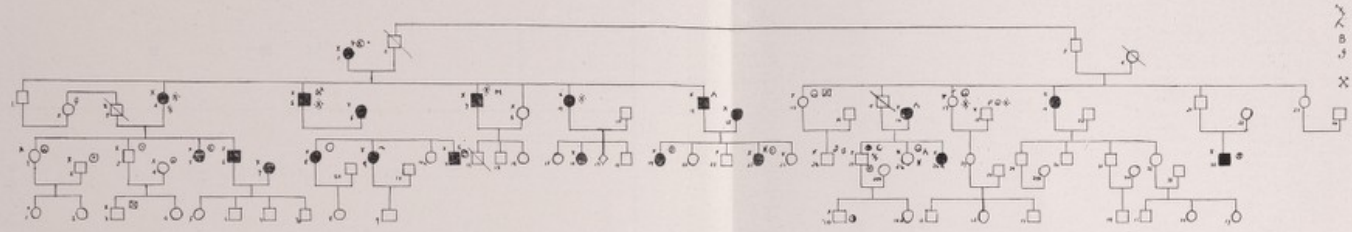


No. 5 HYPERMETROPIC ASTIGMATISM



No 69

Hypermetropic Astigmatism

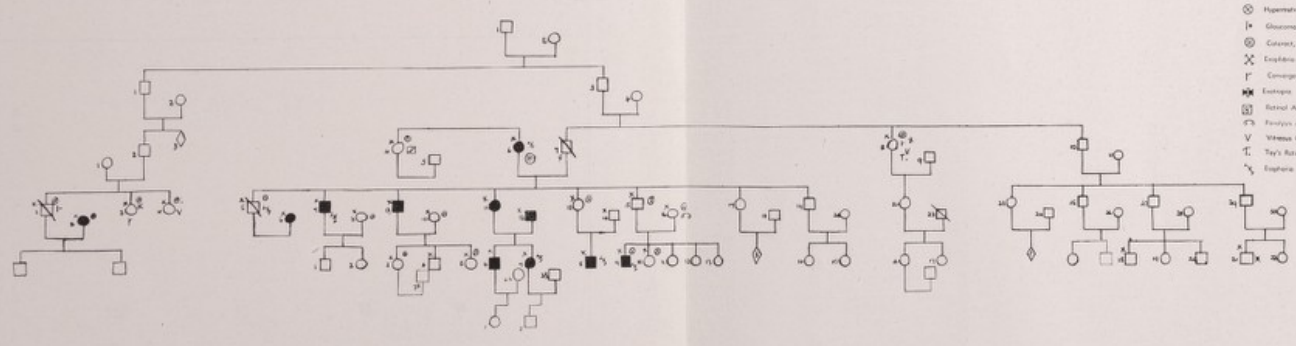


Males Affected.
Females Affected.
Examined.
Hypermetropia.
Astigmatism, Mixed.
Astigmatism, Myopic.
Glaucoma.
Strabismus.
Cataract, Senile.
Esophoria.
Wears Glasses.
Colour Blind.
Exophoria.
Divergence Insufficiency.
Binocular.
Im. Optic Atrophy.
Exophoria.
Hyperphoria.
Convergence Excess.
Retinal Arteria-Sclerosis.
Pigmentary Degen. of Macula
Amblyopia.
Monocular Blind.
Nystagmus.
Esotropia.
Periph. Pigmentary Degeneration.
Persistant Hyaloid.

NO 91

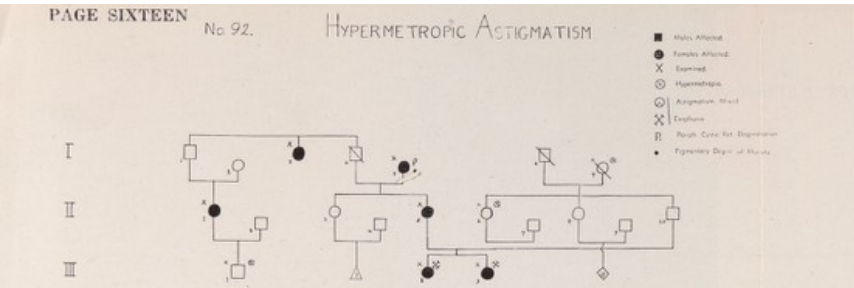
HYPERMETROPIC ASTIGMATISM

I
II
III
IV
V
VI

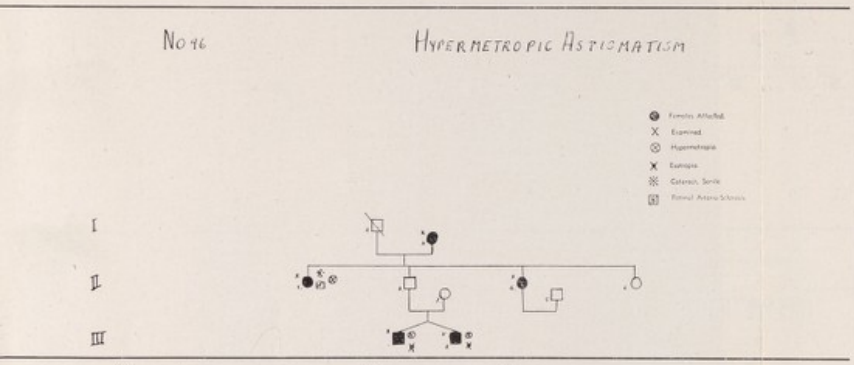


Males Affected.
Females Affected.
Examined.
Astigmatism, Myopic.
Astigmatism, Mixed.
Hypermetropia.
Glaucoma.
Cataract, Complicated.
Exophoria.
Convergence Insufficiency.
Esotropia.
Retinal Arteria Sclerosis.
Paralysis of Accommodation.
Vitreous Opacities.
Tay's Retinitis Chorioidea.
Esophoria.

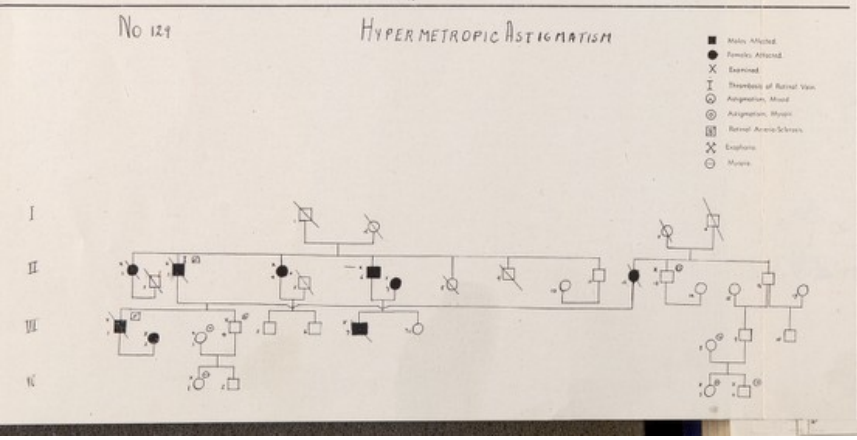
No 92. HYPERMETROPIC ASTIGMATISM



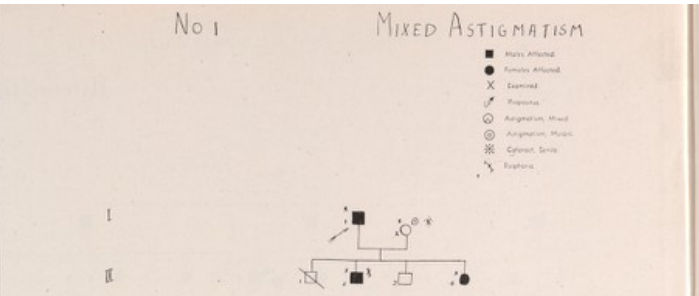
No 46 HYPERMETROPIC ASTIGMATISM



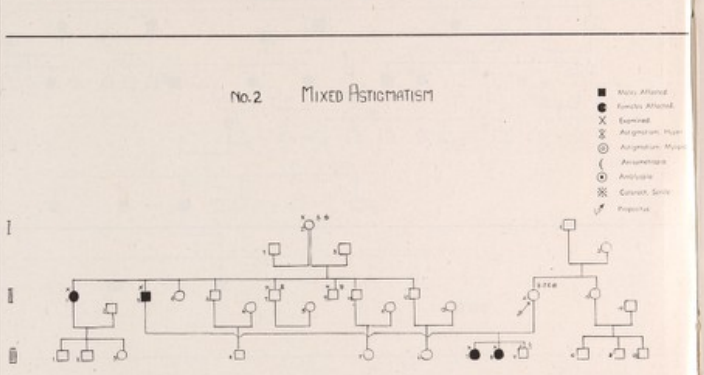
No 129 HYPERMETROPIC ASTIGMATISM



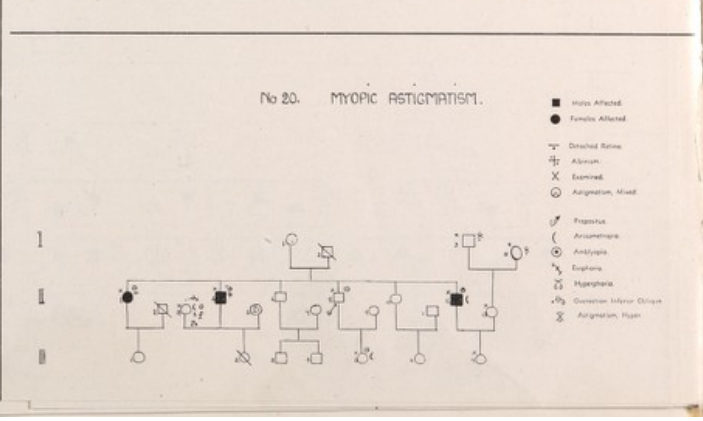
No 1 MIXED ASTIGMATISM



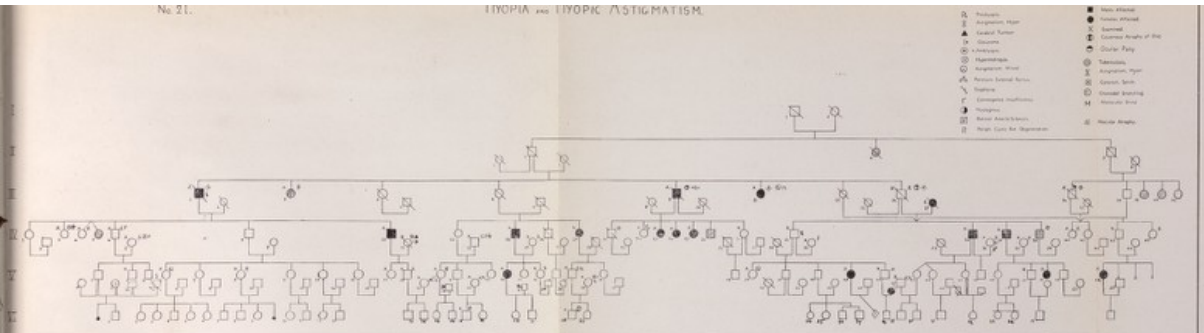
No. 2 MIXED ASTIGMATISM



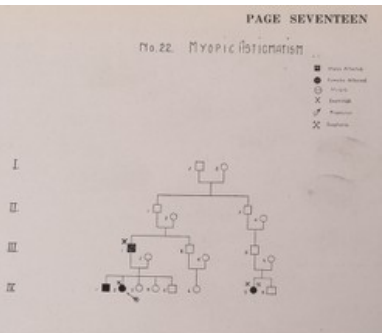
No 20. MYOPIC ASTIGMATISM.



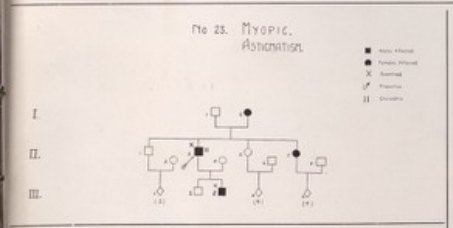
No. 21. MYOPIA and MYOPIC ASTIGMATISM.



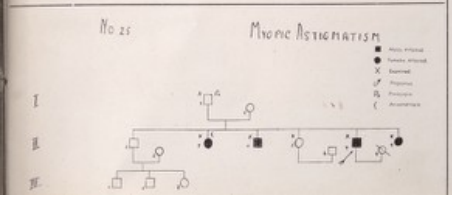
No. 22. MYOPIC ASTIGMATISM



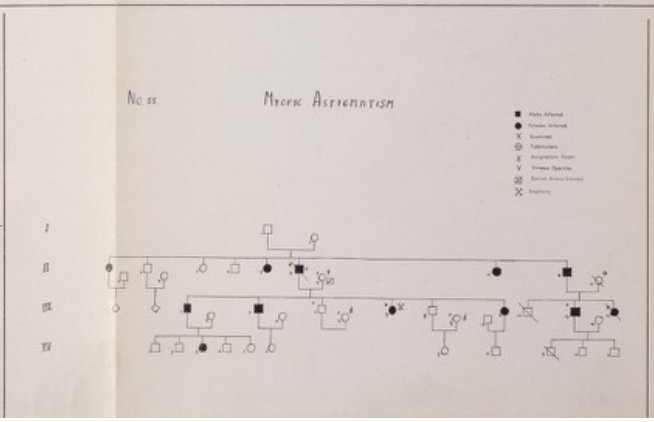
No. 23. MYOPIC ASTIGMATISM



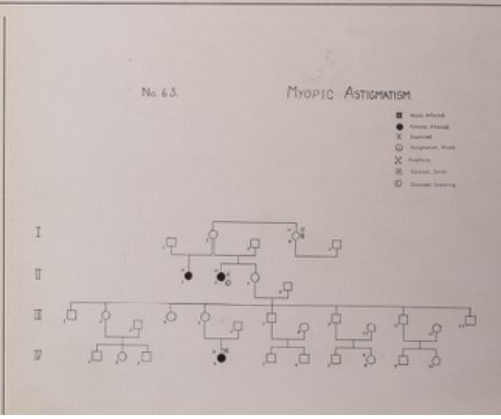
No. 25 MYOPIC ASTIGMATISM



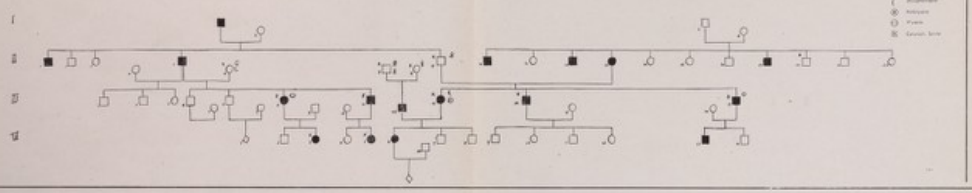
No. 55 MYOPIC ASTIGMATISM



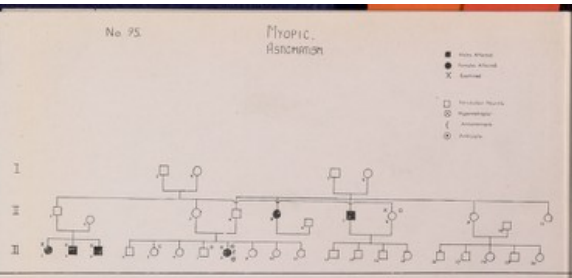
No. 63. MYOPIC ASTIGMATISM



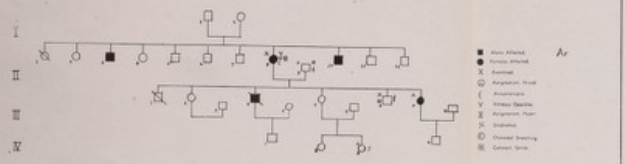
No 79 Myopic Astigmatism



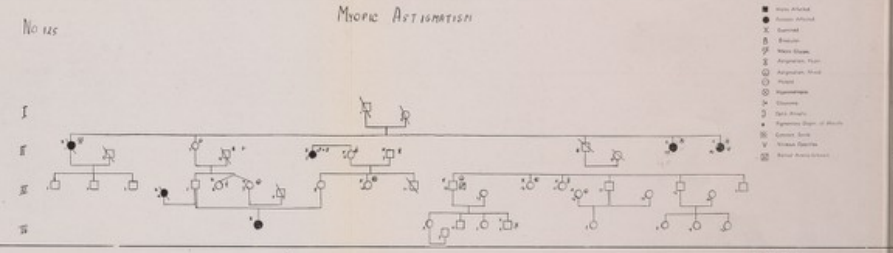
No 95 Myopic. Astigmatism



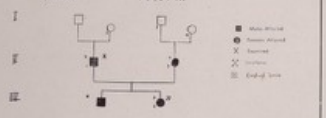
No 101 Myopic Astigmatism



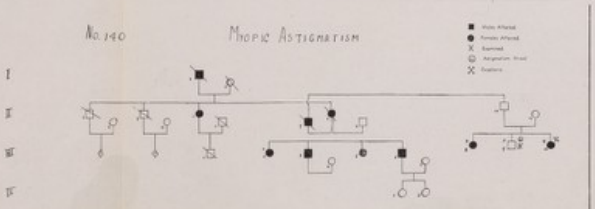
No 125 Myopic Astigmatism



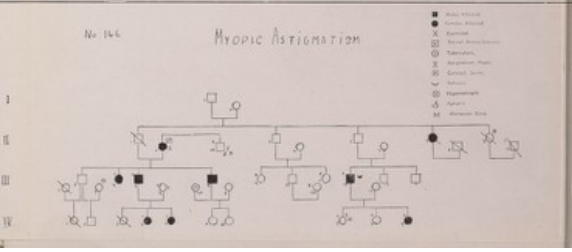
No 124 Myopia



No 140 Myopic Astigmatism

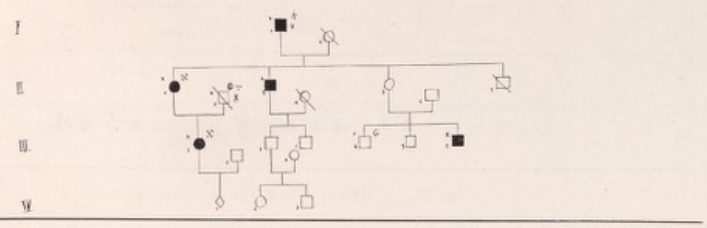


No 146 Myopic Astigmatism



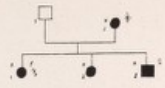
No. 148

Myopic Astigmatism



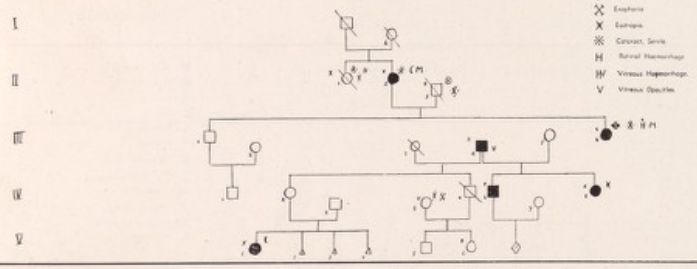
No. 115

Myopic Astigmatism



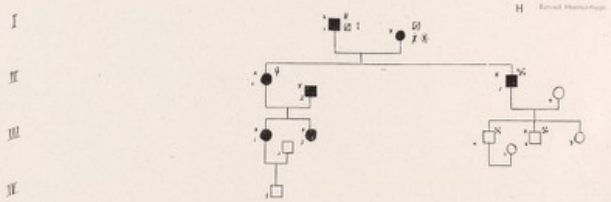
No 158

Myopic Astigmatism



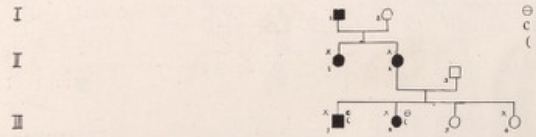
No 119

Myopic Astigmatism



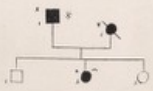
No. 162

Myopic Astigmatism.

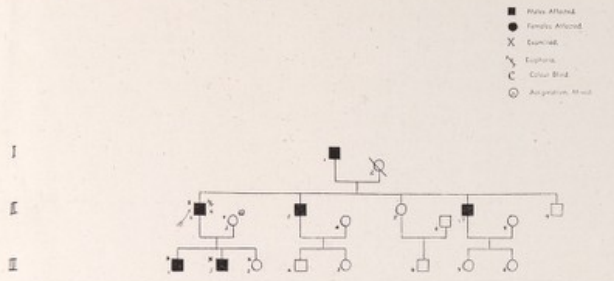


No 175

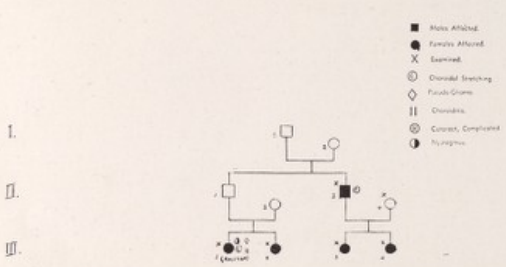
Myopic Astigmatism



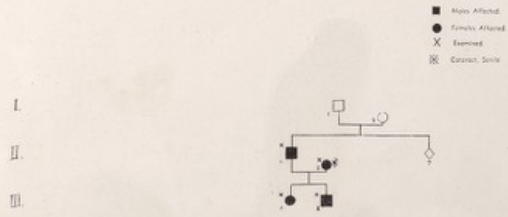
No. 150 Myopic Astigmatism



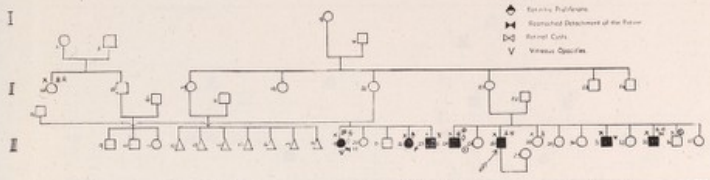
No. 210. Myopic Astigmatism.



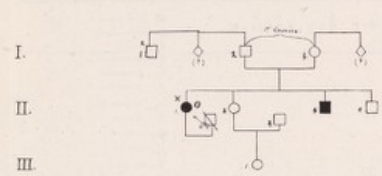
No. 219. Myopic Astigmatism.



No. 37 Retinal Detachment

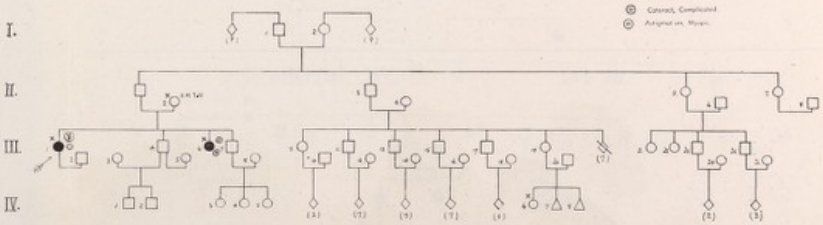


No. 38. Retinitis Pigmentosa



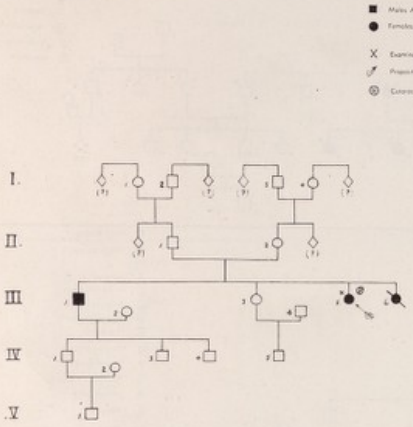
Consanguinity.

No. 39. Retinitis Pigmentosa.

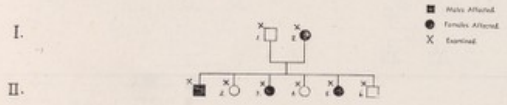


No Consanguinity

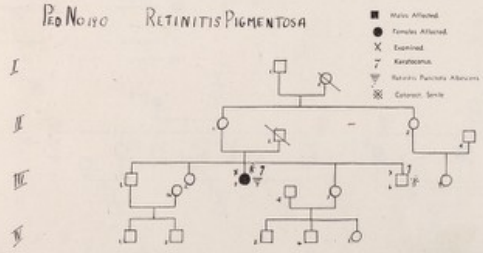
No. 40. Retinitis Pigmentosa.



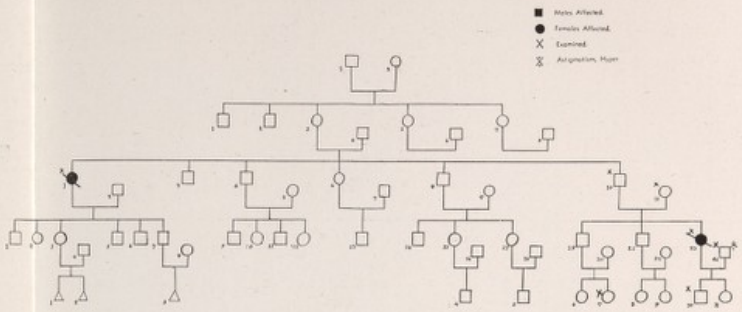
No. 41. Retinitis Pigmentosa.



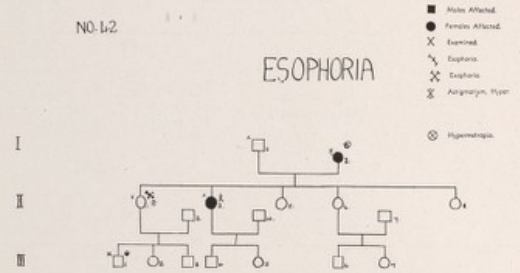
Ped No 190 Retinitis Pigmentosa



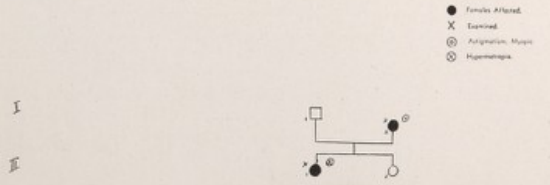
No. 89. Sarcoma of the Choroid.



No. 42 Esophoria

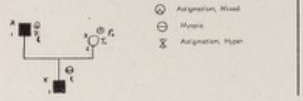


No 198 Esophoria

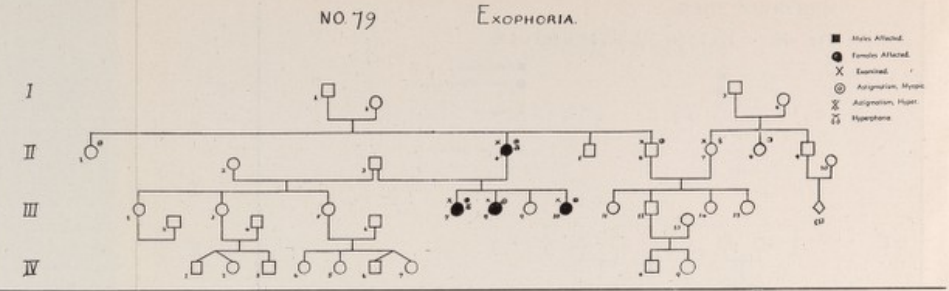


No. 200

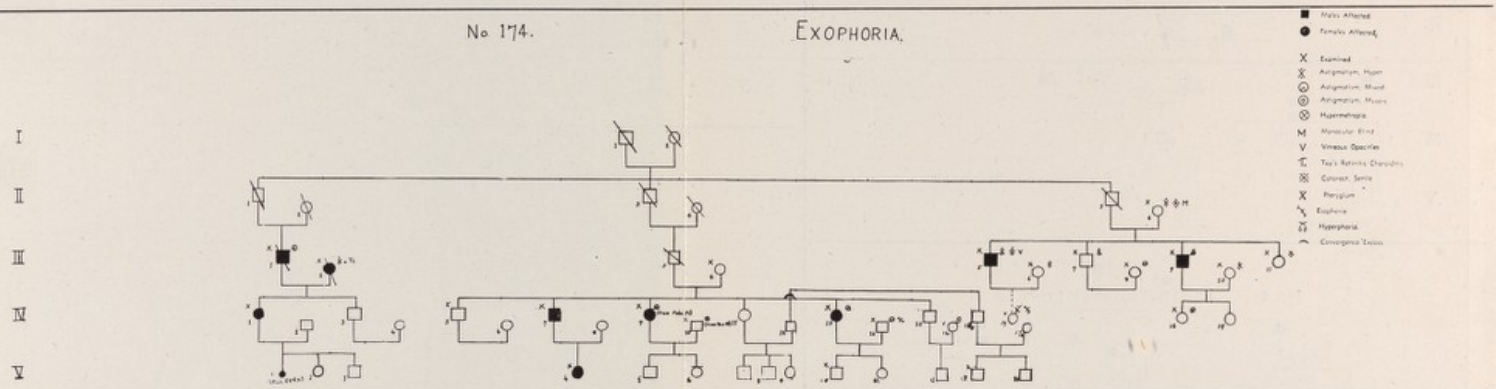
ESOPHORIA



NO. 79 EXOPHORIA.

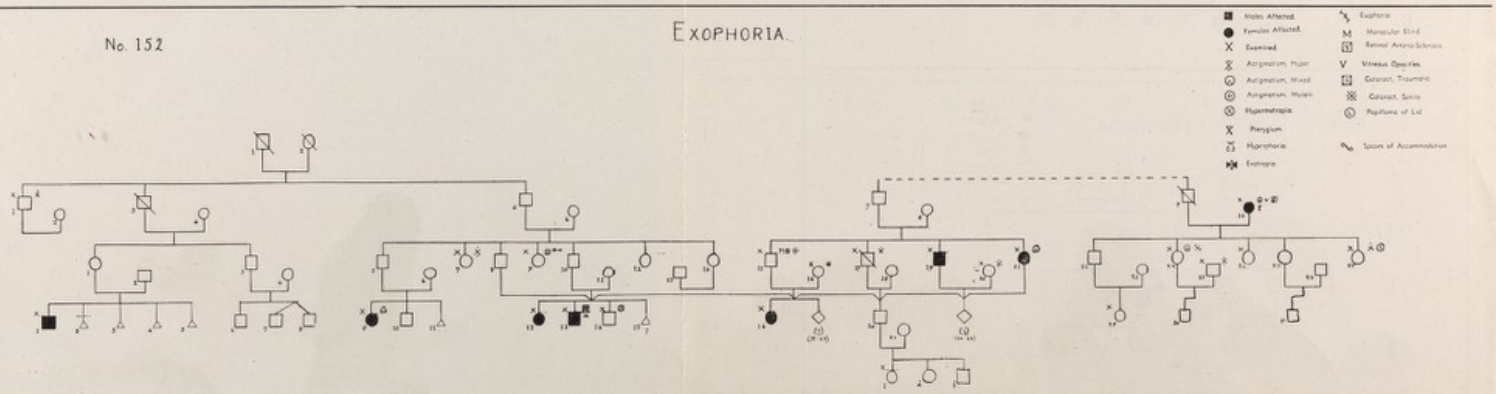


No. 174. EXOPHORIA.



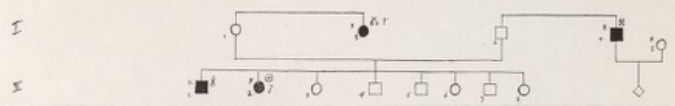
No. 152

EXOPHORIA.



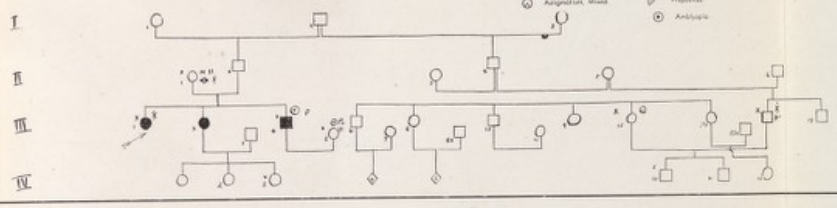
No. 164

EXOPHORIA.



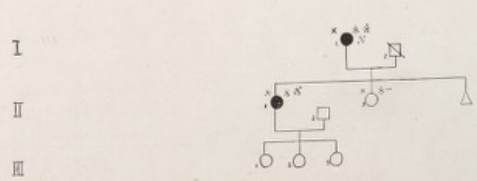
No. 166

EXOPHORIA



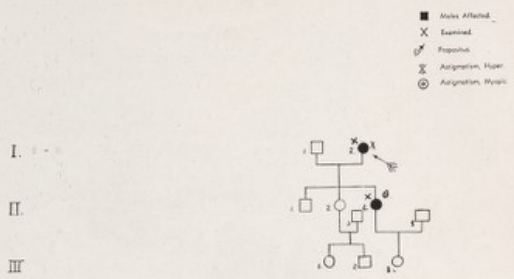
No. 106.

EXOPHORIA.



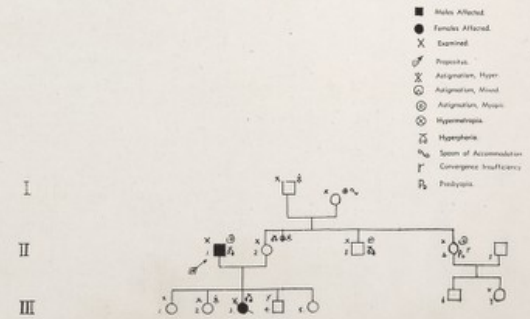
HETEROPHORIA.

No. 44. EXOPHORIA.

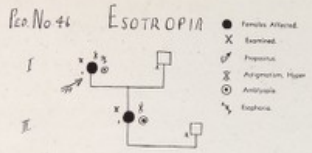


HETEROPHORIA.

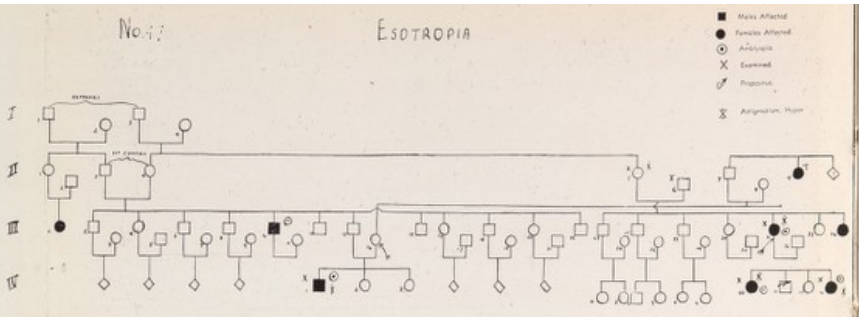
No. 45. EXOPHORIA.



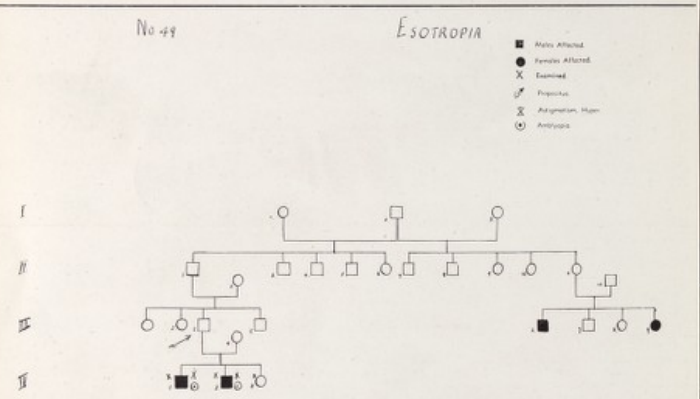
Ped. No 46 ESOTROPIA



No. 47 ESOTROPIA

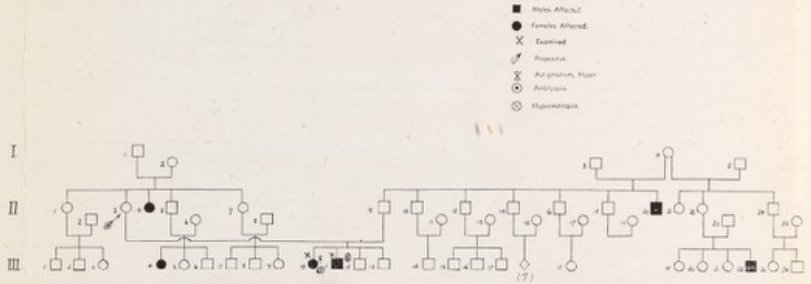


No. 49 ESOTROPIA



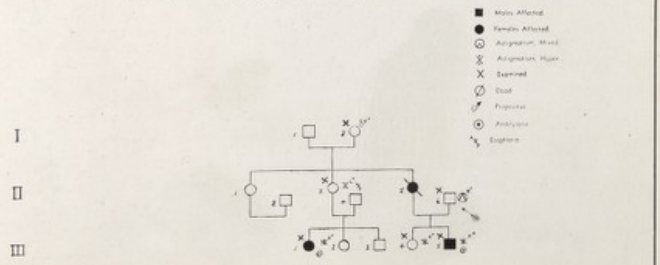
HETEROTROPIA.

No. 50. CONVERGENT CONCOMITANT STRABISMUS.

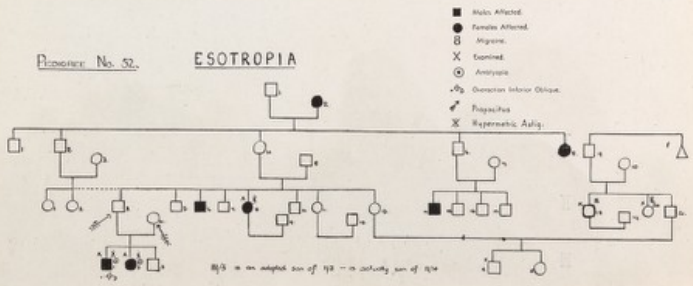


ESOTROPIA.

No. 51. CONVERGENT CONCOMITANT STRABISMUS.

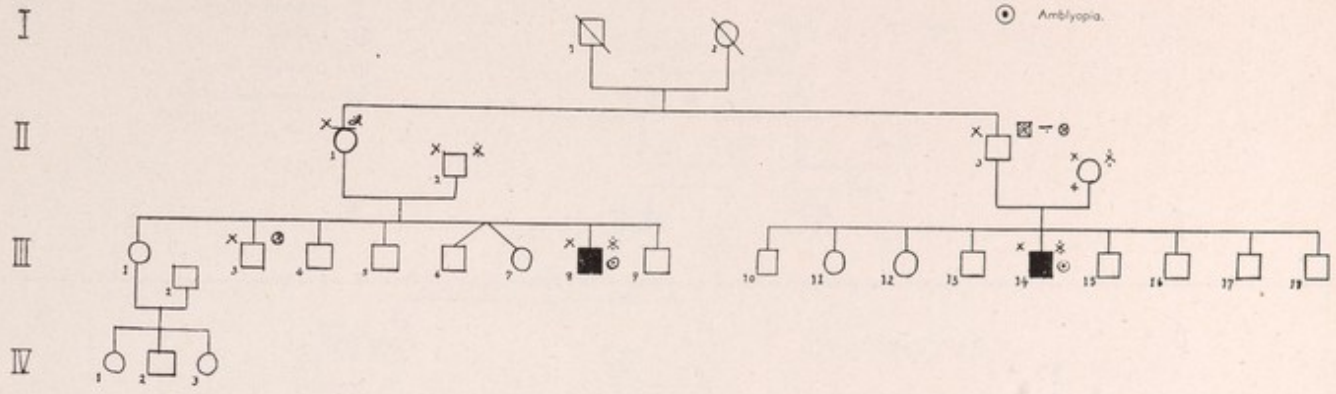


Pedigree No. 52. ESOTROPIA



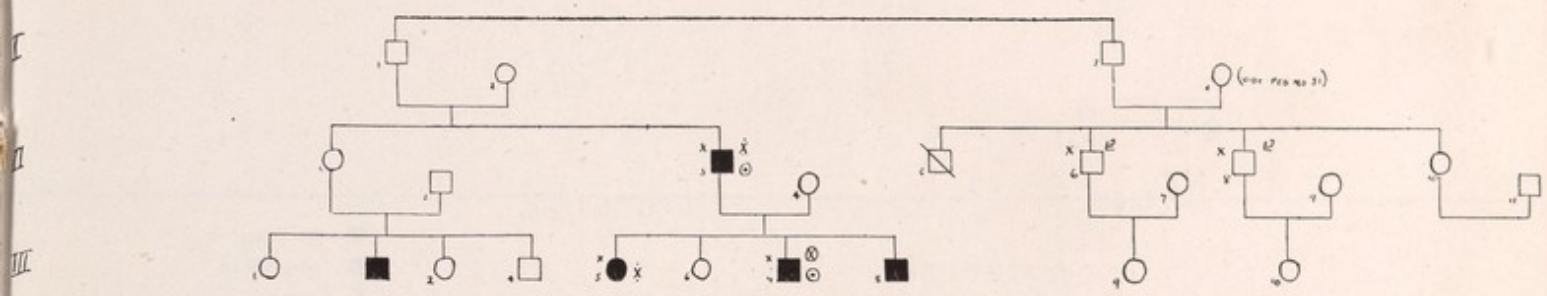
No. 157 ESOTROPIA.

■ Males Affected.
● Females Affected.
X Examined.
Astigmatism, Hyper.
⊗ Hypermetropia.
Detached Retina.
Sec. Optic Atrophy.
Cataract, Traumatic.
⊙ Amblyopia.



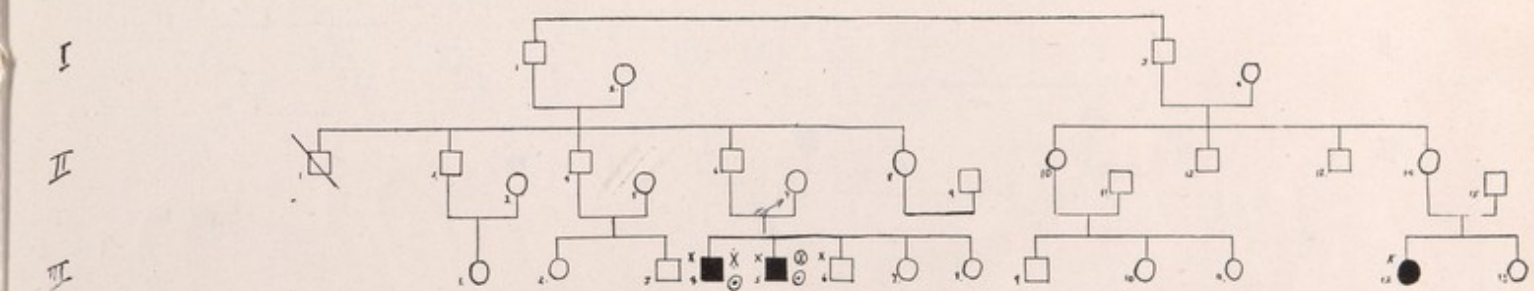
No. 177. ESOTROPIA

■ Males Affected.
● Females Affected.
X Examined.
⊗ Hypermetropia.
Astigmatism, Hyper
Leber's Optic Atrophy.
⊙ Amblyopia.



No 184 ESOTROPIA

■ Males Affectrd.
● Females Affected.
X Examined.
Astigmatism, Hyper.
⊗ Hypermetropia.
⊙ Amblyopia.



No. 191

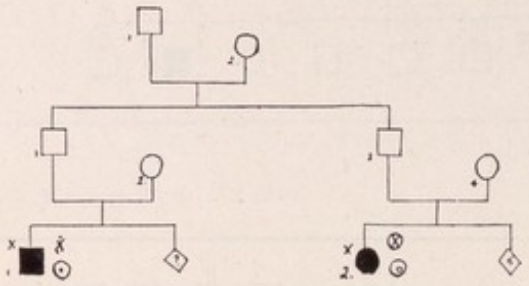
ESOTROPIA

- Males Affected.
- Females Affected.
- Examined.
- Astigmatism, Hyper.
- Amblyopia.
- Astigmatism, Myopic
- Hypermetropia.

I

II

III



No. 226

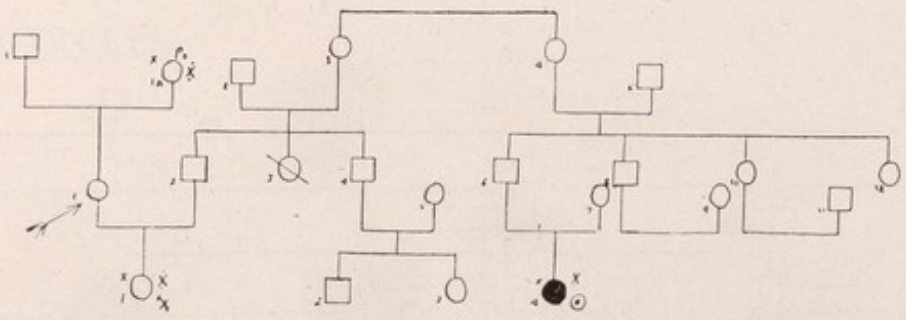
ESOTROPIA

- Males Affected.
- Females Affected.
- Examined.
- Propositus
- Astigmatism, Hyper.
- Amblyopia
- Esophoria.
- P_o Presbyopia

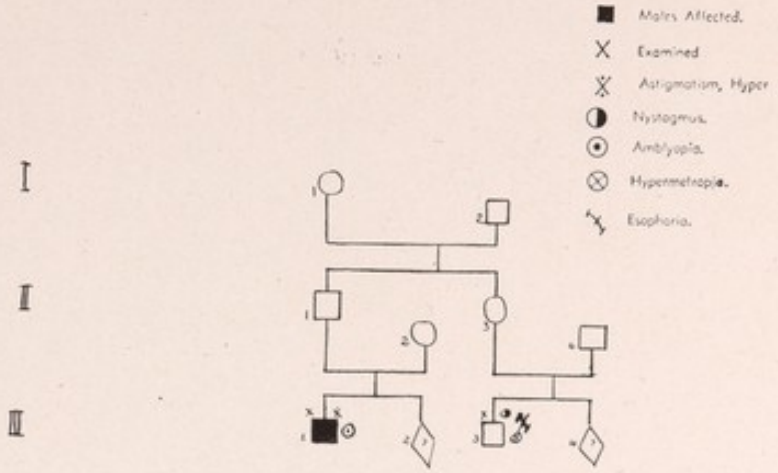
I

II

III

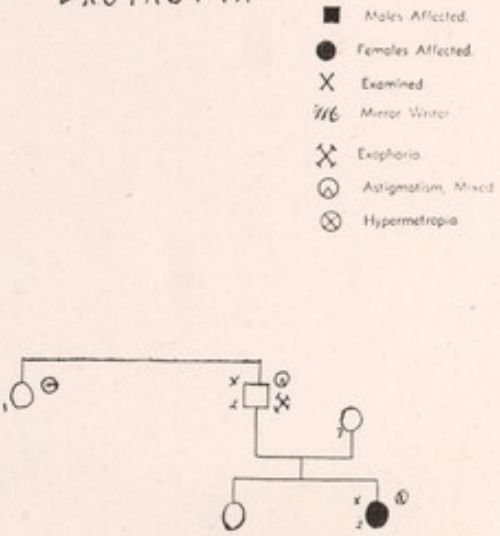


ESOTROPIA



No. 204

EXOTROPIA



No. 43.

HYPERPHORIA