

TITLE: PERIPHERAL RETINAL DEGENERATIONS IN

KENYAN AFRICAN MYOPES

UNIVERSITY OF NAIROBI LIBRARY

A thesis submitted in part fulfilment for the degree of Master of Medicine (Ophthalmology) in the University of Nairobi.

by

DR. H.S. ADALA, M.B., Ch.B. (MAKERERE), D.O.(LONDON)

DECLARATION

This thesis is my original work and has not been presented for a degree in any other University.

Signed H.S. Adala Date 30/4/80

Dr. H.S. Adala
Candidate

This thesis has been submitted for examination, with my approval.

Signed V. Klauss Date 6/5/80

(iii) Pigmentary
(iv) Cysto Dr. V. Klauss, M.D.

Supervisor
Signed W. Dechant Date 8/5/80

Dr. W. Dechant, M.D.
Supervisor

DECLARATION

This thesis is my original work and has not been presented for a degree in any other University.

(i). Tables I and II
Signed [Signature] Date 30/4/80

(ii). Tables III and IV 15

(iii). Table V 16

(iv). Tables VI, VII, VIII 17

Dr. H.S. Adala

(v). Statement of abstract and tables 18

Candidate

This thesis has been submitted for examination, with my approval.

(i). Lattice regeneration 45
Signed [Signature] Date 6/5/80

(iii). Pigmentary 49

(iv). Cysto 51

Dr. V. Klauss, M.D.

Supervisor

Signed [Signature] Date 8/5/80

(i). Cells 54

Supervisor

C O N T E N T S

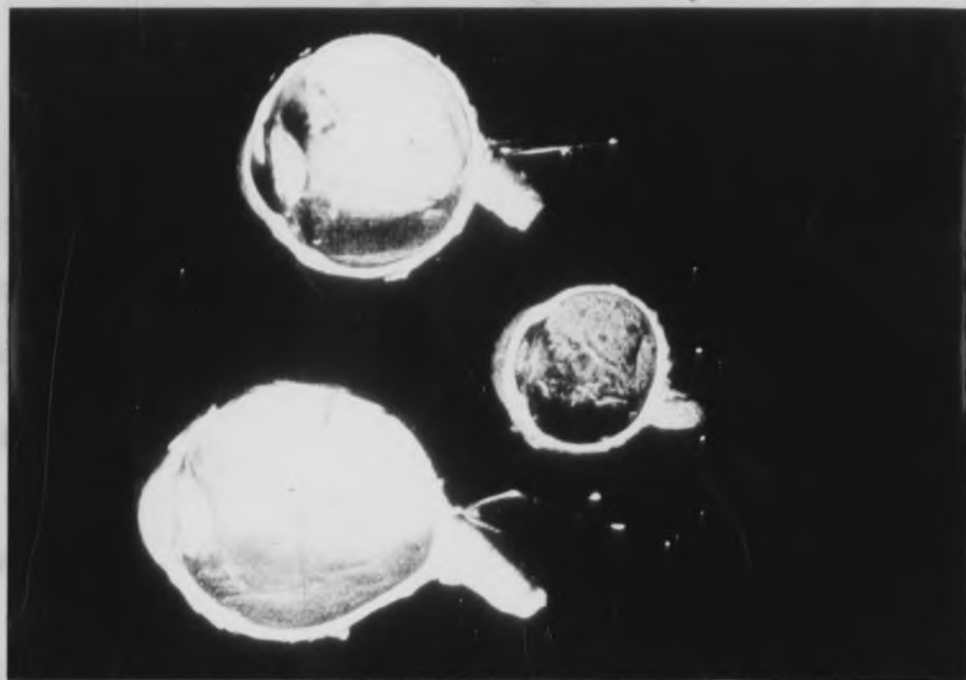
I. INTROUDCTION.....	1
2. SUMMARY.....	3
3. DEFINITIONS.....	4
4. METHODS AND MATERIAL	
(i). Methods.....	8
(ii). Material.....	11
5. RESULTS	
(i). Tables I and II.....	14
(ii). Tables III and IV.....	15
(iii). Table V	16
(iv) Tables VI,VII,VIII.....	17
(v). Discussion of histogram and tables....	18
6. DISCUSSION (Pre-degenerations and Degenerations)	
(a). Pre-degeneration	
(i). White without pressure.....	41
(ii). snow-flake.....	42
(b). Degenerations	
(i). Lattice Degeneration.....	45
(ii). Snail-track Degeneration.....	47
(iii). pigmentary	49
(iv) . Cystoid.....	51
7. TREATMENT.....	53
8. CONCLUSION.....	55
9. FUNDUS DRAWINGS (I -XII)	
(i). Colour and numeral code.....	59
(ii). Key.....	60
10. ACKNOWLEDGEMENTS.....	61
11. REFERENCES.....	62

I N T R O D U C T I O N

Retinal breaks have for long been associated with Retinal Detachment (Gonin 1921) and the closure of such retinal breaks results into reattachment of already detached retina, or prevention of retinal detachment in cases of flat holes. Prophylactic treatment of flat holes with symptoms, is practiced in many centres, however asymptomatic retinal breaks have been noticed to occur in both emmetropes and myopias. About $\frac{2}{3}$ of retinal detachments occurs in myopic eyes (Duke-Elder and Dobree, 1967).

Myopia results into both peripheral and central chorio-retinal degenerative changes. The posterior or central degenerative changes are noticed in very high myopia, referred to as pathological or progressive myopia. This is said to be due to uncontrolled increase in the axial length of the eyeball. Peripheral (Equatorial) degeneration are more common in myopes than in normal eyes. These degenerations can progress to retinal breaks with subsequent retinal detachment. It would therefore be useful to detect these degenerations early, follow them up and give prophylactic treatment where necessary.

Some of these degenerations are known to run in families and hence detection of a particular degeneration especially



Anterior-Posterior length of the Eyeball

Compared; Top - normal

Middle- Hypermetropic Eye

Below - Myopic Eye

if it is known to be highly associated with retinal breaks, would warrant a screening exercise of the fundi of that particular family. The treatment of such degenerations would also be influenced in some cases, by any positive family history.

A number of studies have been done and reported on this subject among the caucasians, however, literature is scanty on similar studies done among African patients. A significant number of cases of retinal detachment are seen at the Kenyatta National Hospital, the majority of which are rhegmatogenous detachments, with peripheral retinal degenerations. It would therefore be useful to know the incidence of peripheral retinal degenerations among our myopic patients. The Ophthalmic Surgeon treating such a group of patients, would therefore be prompted to examine carefully the periphery of the fundus and decide on the treatment of any degenerations if detected and thought to be dangerous. This would be a preventive step to curb the incidence of retinal detachment. It is for this reason that I have been prompted to conduct the following study.

S U M M A R Y

A total of 128 subjects (256 Eyes) had their eyes examined under full mydriasis. Indirect Ophthalmoscopy was done in every subject to determine the presence or absence of peripheral retinal pre-degenerations and degenerations. The subjects consisted of volunteers and patients who attended the refraction clinics. The results were analysed according to age groups, degree of myopia and also considered was the frequency of distribution in various quadrants of the eye.

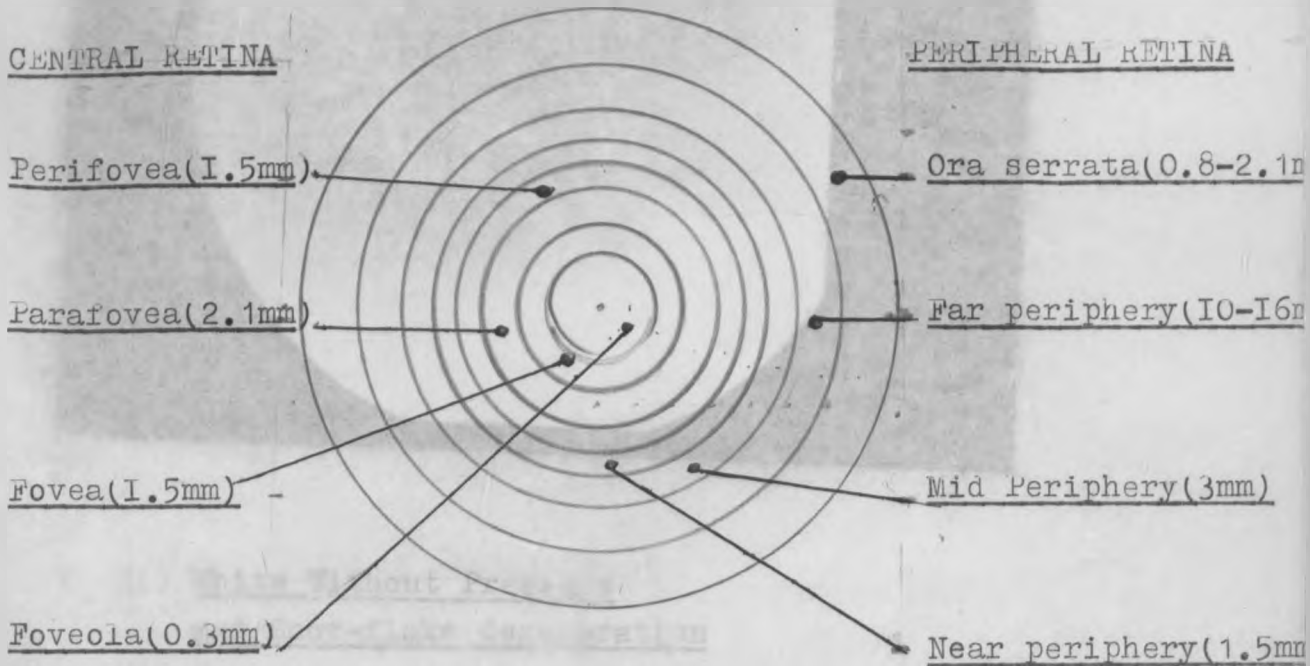
The results were compared in association with retinal breaks and retinal detachments. A review of the retinal detachment operations done within the last ten years at the Kenyatta National Hospital, was done and a graph constructed to determine the decline or increase in the numbers seen every year.

A significant incidence of pre-degenerations was noted, and there was a strong association between these degenerations and the retinal breaks and detachments seen during the period of the study.

In conclusion, considering the ever increasing number of retinal detachments seen every year at the Kenyatta National Hospital, it is necessary that every ophthalmic surgeon becomes more conscious of peripheral retinal degenerations and treat them as necessary, in order to prevent some, of the retinal detachments that would otherwise arise from them.

DEFINITIONS

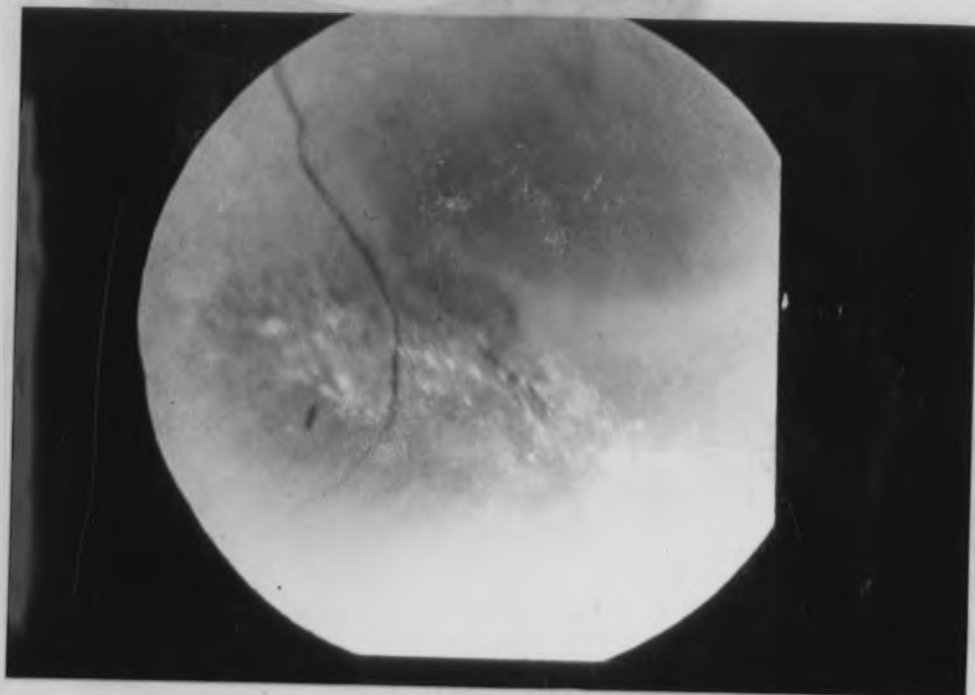
1. Peripheral Retina: See diagram below



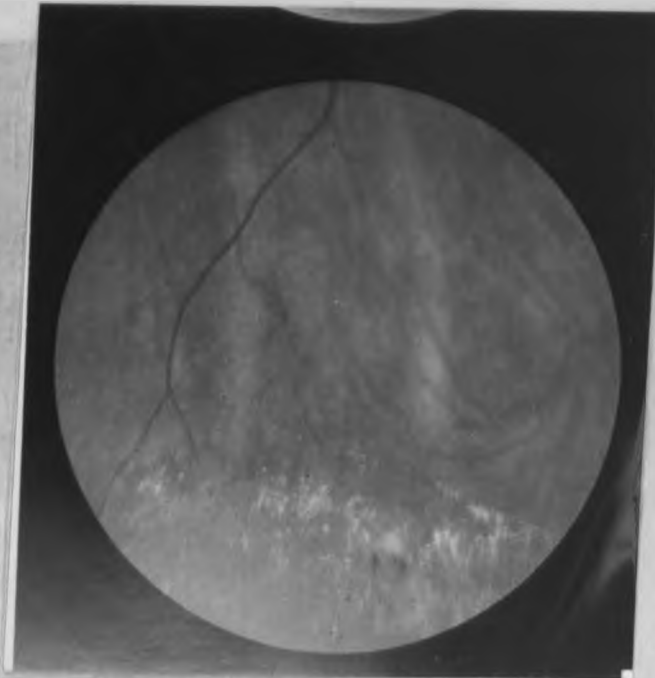
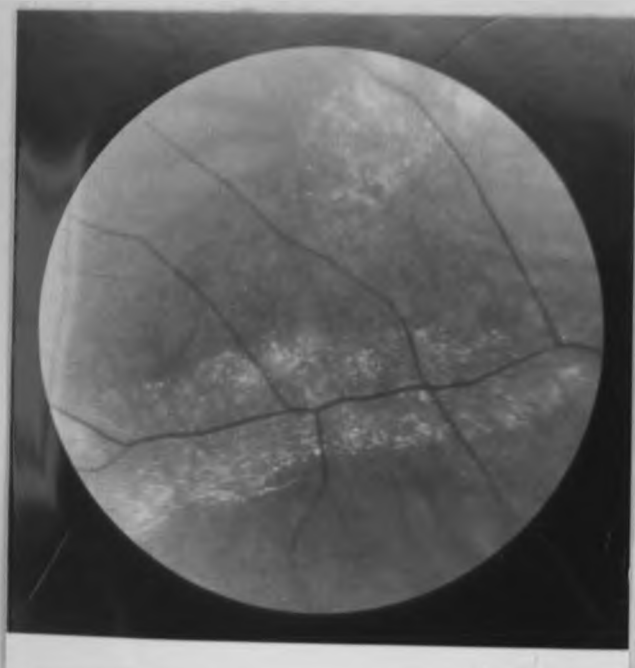
2. Pre-Degenerations

(i) White without pressure: Areas of vitreoretinal adhesion associated with retinal atrophy. clinically appears as white areas, usually with geographical outline posteriorly, occasionally seen in the presence of retinal breaks and/or retinal detachment. (See Photograph 2(i))

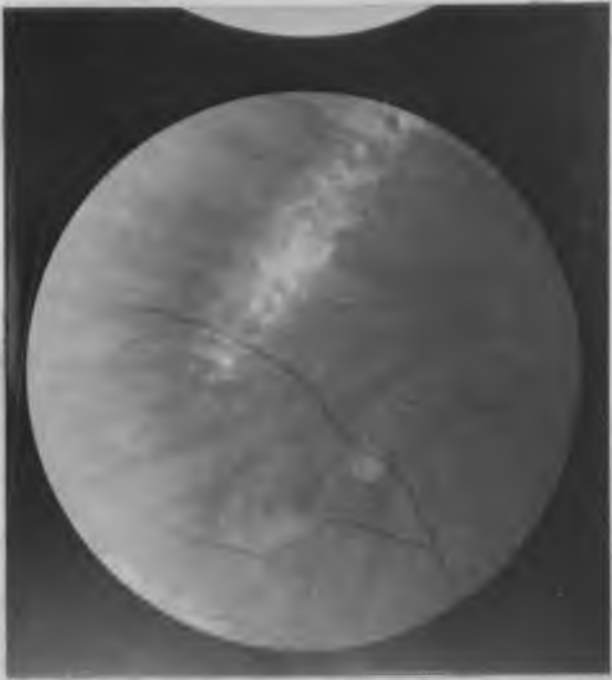
(ii). Snow-Flake Degeneration: Small, tiny glistening yellow-white dots usually found anterior to the equator. They can be diffuse with no demarcation or well circumscribed with definite posterior and



2. (i) White Without Pressure
and Snow-flake degeneration



36 Small-Tract Degeneration
2. (ii) Snow-Flake degeneration



also in micro-
 graph 3(ii). The
 structures which are
 the fibres.

3(ii) Lattice Degeneration

the structure
 of the lattice
 is shown
 in the
 photograph



3(iii) Snail-Track Degeneration

photograph 3(iii).

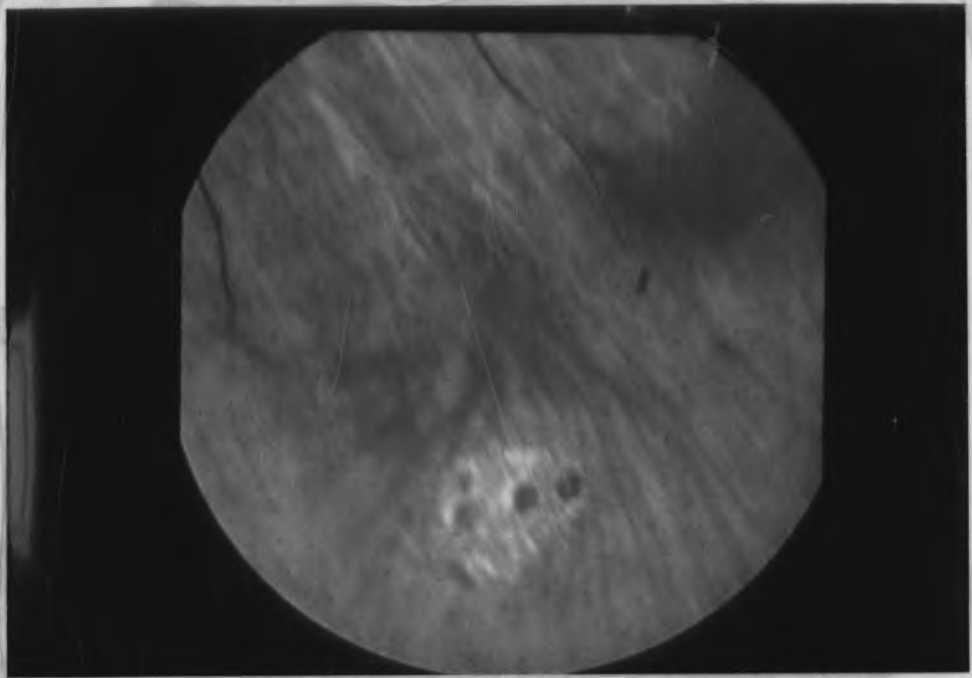
anterior borders. These lesions can also be distributed along blood vessels (Ref. Photograph 2(ii)). The white dots are lipid containing macrophages which are also seen in ganglion cells and muller's fibres.

3. Degenerations:

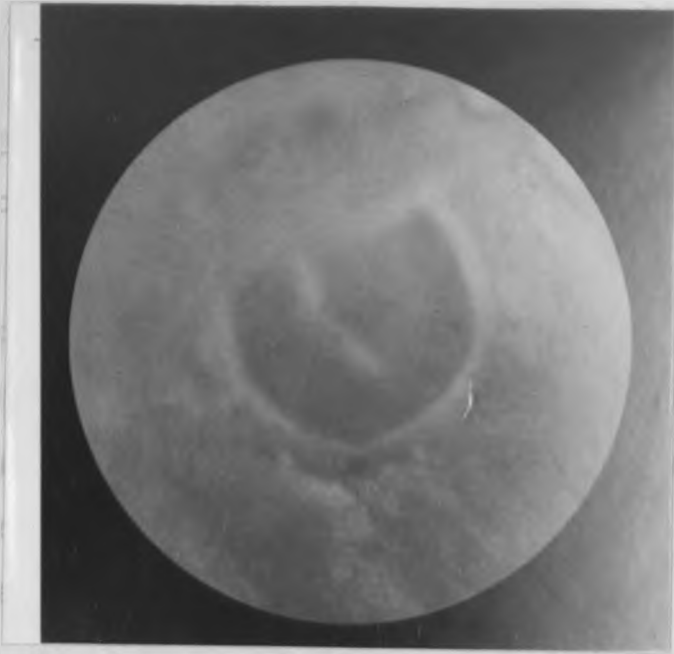
(i) Lattice degeneration: A typical lattice degeneration takes a criss-cross pattern of white lines (thickened or obliterated vessels) occasionally with pigmentation in-between the white lines. They are usually seen between the equator and the posterior border of the vitreous base (Ref. Photograph No. 3(i)).

(ii) Snail-track degeneration: Sharply demarcated bands of white crinkles or frost-like changes of the inner retinal surface. Along the margins, are vitreoretinal changes. (see photograph No. 3(ii)).

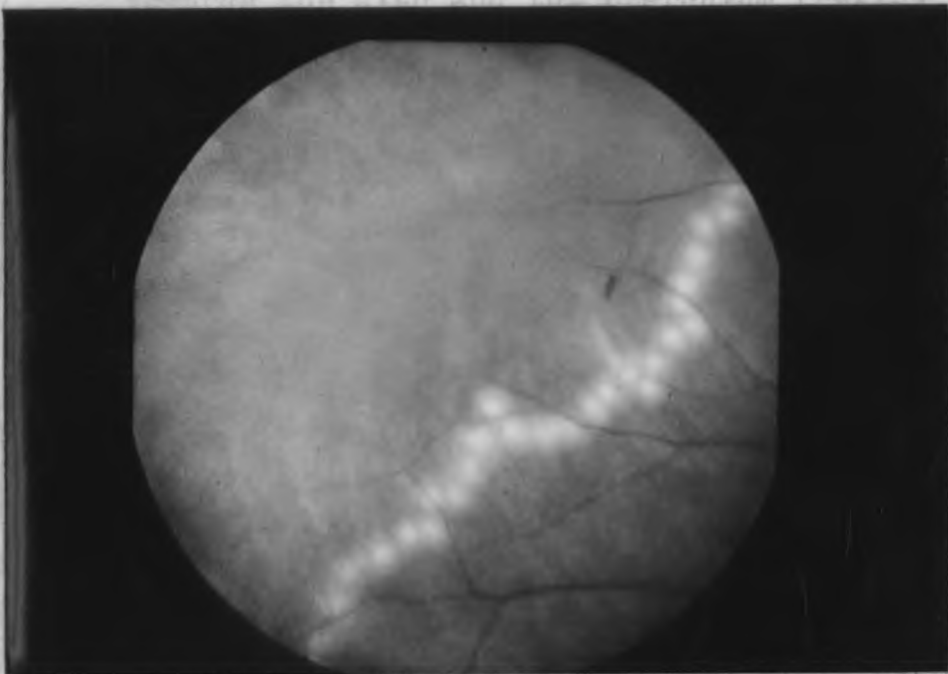
(iii) Pigmentary degeneration: Three types are seen, first the oral pigmentary degeneration which consists of hyperpigmented band running adjacent to the ora serrata, second the clumping type, where clumps of pigment are seen either in the periphery, or around retinal breaks and occasionally beneath a detached retina. The third type is where pigmentation is seen as a drusen with areas of depigmentation around them, sometimes giving a honeycomb appearance. Ref. to photograph 3(iii).



Round hole in an area of Equatorial degeneration
after photocoagulation.



3(ii) Pigmentary Degeneration at the edge of a large retinal tear



5(iv) Cystoid degeneration after Photocoagulation

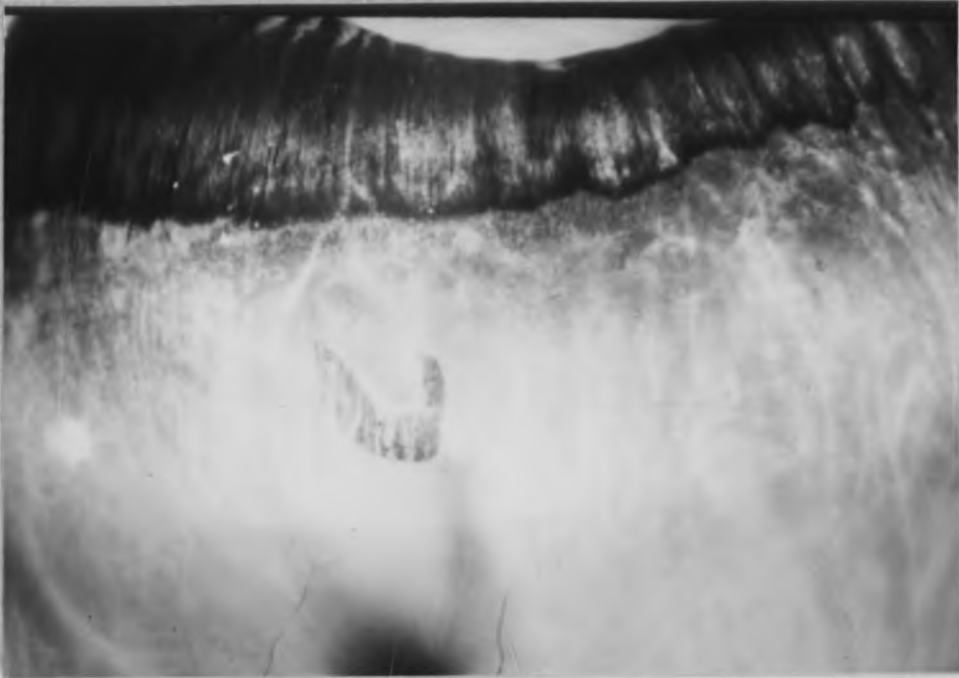
(iv) Cystoid degeneration: Two types exist, first the microcystoid which is a senile change and is seen in the ora serrata, commonly seen in adults. Second is retinoschisis, which is a split in the neuro-retinal layer at the level of outer plexiform layer. This gives a smooth raised area from the periphery with clinically an absolute scotoma. (Ref. to photograph No. 3(iv)).

4. Retinal breaks:

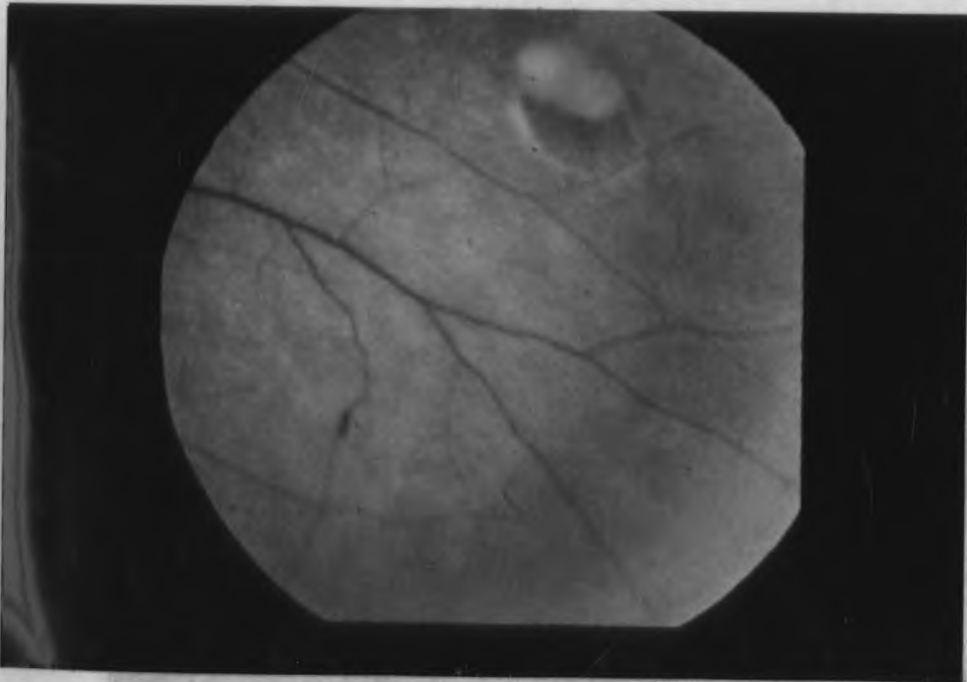
(i). Horse-shoe tear(Ref. to photograph 4(i)).

This is an arrow-head type of tear due to traction from vitreoretinal adhesions. Its convexity is, towards the disc and the operculum (retina in the concavity of the tear) pulled inward by a vitreous traction band on the distal (ora serrata) concave side. It is a common retinal break and leads to retinal detachment. The retina which forms the operculum is always the area of lattice degeneration.

(ii)Dialysis: (Ref. photograph 4(ii)). This is a disinsertion of the retina from its attachment to the ora serrata. It occurs either spontaneously or as a consequence of atrophy of the peripheral retina. It commonly occurs after trauma. It is seen in

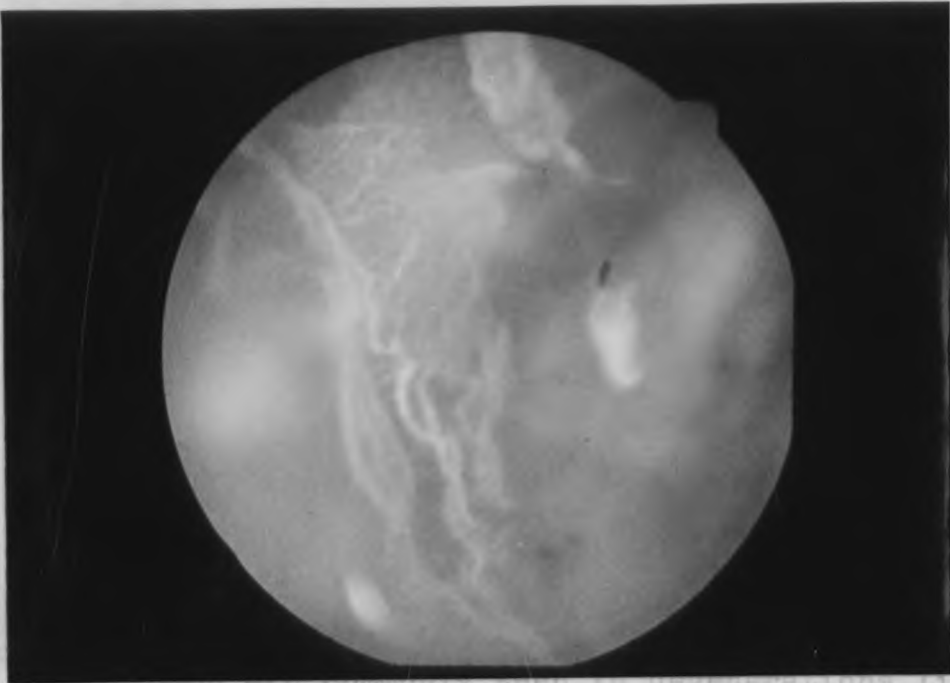


40) Horse shoe tear from a flat preparation
of the retina



Operculated Horse-Shoe tear.

40) DIALYSIS

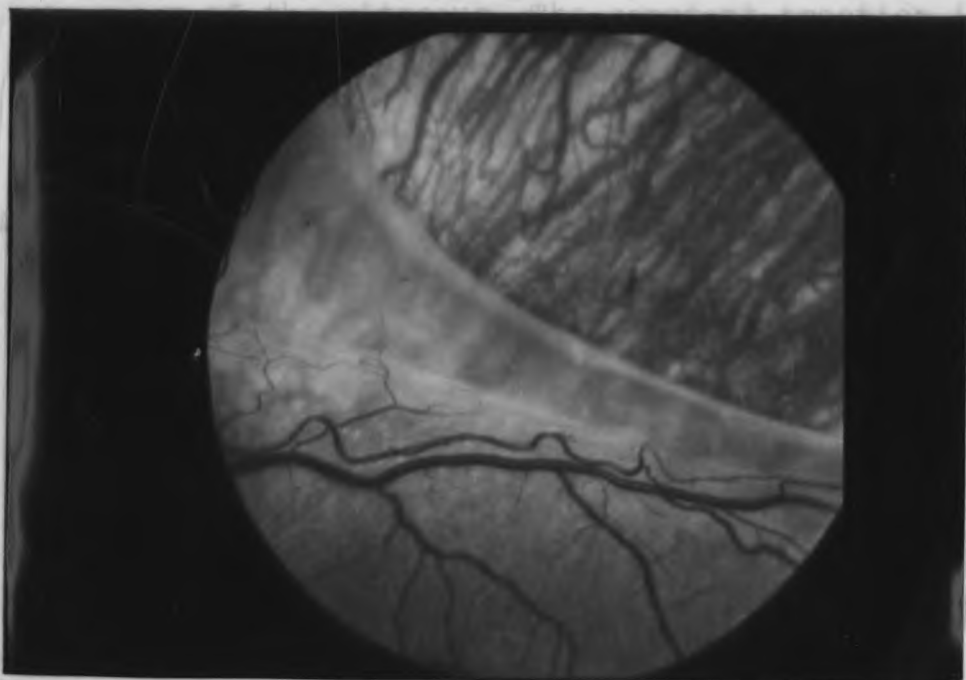


result
of the
at round
vitreous,
reaching
oor.

Extensive Vitreo-retinal traction

a high risk of vitreous haemorrhage and retinal detachment. (Ref. to photograph 4(iii)).

(ii). Operculated round hole: These tears have the detached piece of retina freely floating in the

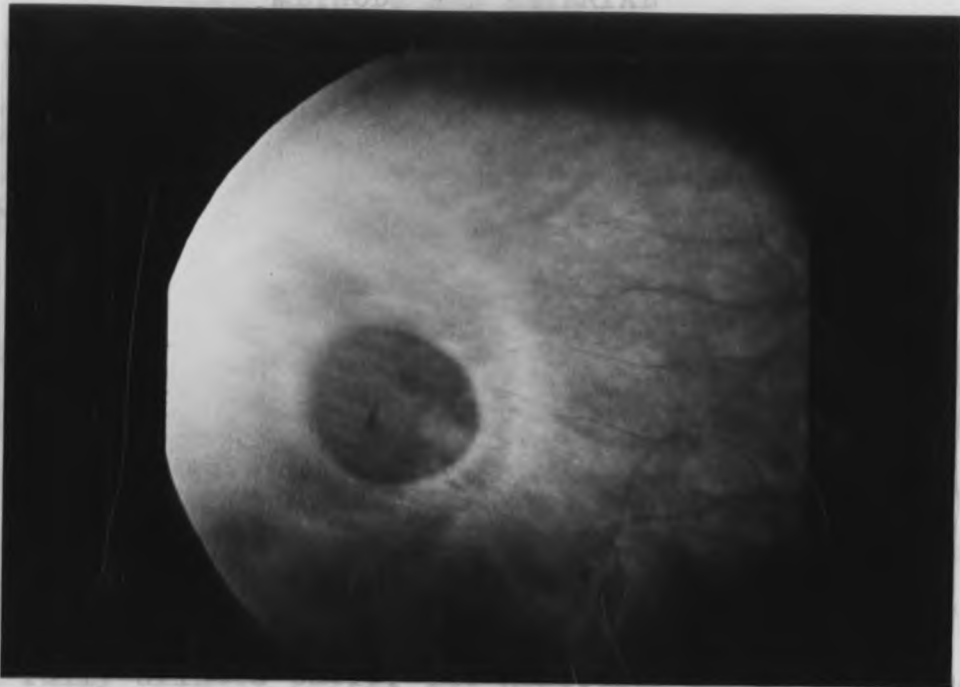


rich

4.(ii) DIALYSIS

children with retinal detachment and could result from congenital deformity of the insertion of the retina at the ora serrata.

- (iii). Operculated round hole: This is a punched out round retinal tear resulting from longstanding vitreoretinal adhesion with retinal atrophy. The remaining piece of retina becomes suspended in the vitreous, by a pedicle (sometimes a blood vessel) attaching it to the margin of the hole, like a trap-door. These are commonly seen in degenerations like 'Lattice' and 'Snail-Track'. These tears have a high risk of vitreous haemorrhage and retinal detachment. (Ref. to photograph 4(iii)).
- (b). Operculated round hole: These tears have the detached piece of retina freely floating in the cortex of the vitreous. The constant traction is thus reduced and the risk of retinal detachment is low in these cases.
- (iv). Non-Operculated holes (atrophy): Area of lattice degeneration in these cases become atrophic and disappear, leaving behind a punched out hole, which does not give a high risk of retinal detachment like in the preceding types of retinal tear.



Unoperculated large round hole

Round holes in an area of snail-track degeneration

METHODS AND MATERIAL

(a) Methods:

Two methods were used to examine the periphery of the fundus.

(i) The first method which was used in all patients, is monocular Indirect Ophthalmoscopy. The advantage of using indirect ophthalmoscope is that it exposes the periphery of the fundus upto the ora serrata even with a moderately dilated pupil. This is in contrast to the use of direct ophthalmoscope which does not show changes anterior to the equator, even with a fully dilated pupil. The monocular indirect ophthalmoscope also has the advantage that the patient can be examined in a sitting position. The disadvantage of the monocular indirect ophthalmoscope is that it does not give stereoscopic view of various lesions and hence the degree of elevation of lesions like retinal cysts would not be very easy to appreciate.

However, where stereopsis was necessary, the binocular Indirect Ophthalmoscope (Keeler) was used. A + 16 Daspheric lens was used with both the monocular and binocular ophthalmoscopes.

(ii) The second method which was not used primarily, but only to supplement the first one, was the slit-lamp biomicroscopy (Goldman's 3-mirror contact lens). This method was employed where the peripheral retinal changes were doubtful, or when it was necessary to determine an elevation in the periphery. The latter was possible because this method also gives stereopsis. The 3-mirror contact lens was used in the conventional way, with the cornea anaesthetised with guttae decicaine, while methocel was used for contact between the lens

The examination started with a brief ocular history to rule out any previous organic eye problems since such cases were excluded from the study. Ocular conditions like uveitis, glaucoma, trauma or any form of vascular retinopathy (diabetic retinopathy, hypertensive retinopathy, sickle cell retinopathy or Eales disease), did not allow the subject to qualify as a candidate. Any previous surgical procedure in the eye similarly disqualified one from the study.

Age limit for the study was 8-65 years. Younger patients i.e. less than 8 years are not co-operative enough during a prolonged funduscopy and would be even more difficult for 3-mirror contact lens examination. Patients over 65 years tend to have nuclear sclerosis and early lenticular opacities, and these would have made funduscopy difficult.

The subjects were then interrogated according to the questionnaire overleaf, stressing more on the history related to their refractive errors. The majority of the patients already had spectacles for correction of myopia. Patients who presented through the filter clinic, were specifically asked their presenting complaints and like the rest of the subjects they were asked if they had noticed any photopsies floaters.

Patients who needed a change of glasses were invited to a 2nd session at a later date, however for those who had to travel long distances, a quick refraction was done before.

the whole process of funduscopy started. Prescriptions were given accordingly. The same procedure was followed in patients who had not been refracted before and among them myopes who qualified, were examined for the study.

The examination proceeded to determining their visual acuities with and without glasses. A routine still-lamp examination was performed to determine the state of the refractive media (cornea, aqueous, lens and vitreous), rest of ocular tissues were also examined where possible. Patients qualified for the study, if they had clear media, normal posterior fundus especially the macular, and had V.A. of 6/12 (corrected) or better.

The subject then had 2 drops of Gutta Midriacyl in both eyes and funduscopy was started ½ hr. later, with the pupils widely dilated. Monocular Indirect Ophthalmoscopy was done with the patient seated, and all quadrants were examined in each eye. Examination of each eye lasted 5-10 minutes and both eyes were examined in one session. In cases where stereoscopic views were required the binocular indirect ophthalmoscope was used sometimes with indentation and the patients were examined on a couch. In a few cases, scrutiny of some peripheral cystic changes required the use of 3-mirror contact lens. The latter group had drops of decicaine and examined with Haag-strait slit lamp (patients seen during the early part of the study were examined with Nikon Slit Lamp).

M A T E R I A L

The material for the study, consisted of:-

- (i) Patients who were seen in the Eye Filter Clinic, with various complaints and were later suspected of having refractive error, (Such patients are usually referred to the Consultative Clinic for routine refraction) It was by performing refraction on these patients that myopes among them were obtained for the study.
- (ii) A second group consisted of medical students from the University of Nairobi. These were requested to report to the eye clinic if they had eye problems corrected with glasses. Among them myopes were detected and those who qualified for the study, had funduscopy done on them.
- (iii) A third group of subjects, included members of staff at the Kenyatta National Hospital (Doctors, Nurses and Radiographers). The majority in this group already had glasses for correction of myopia, and attended the clinic either on request for the study, or turned up for routine refraction check up.
- (iv) Patients admitted in the Ward with retinal detachments and had myopic corrections, were similarly examined. Degenerative changes were noted particularly in relation to the position of any retinal breaks. Second eyes of these patients were examined like all other subjects.

All patients from (i) - (iv) were Kenyan Africans.

The subjects described above (i) to (iii) and the 2nd eyes of (iv) were thus unselected group of myopes, asymptomatic with regard to any retinal disease. The outcome of the study would therefore be an unbiased profile of peripheral retinal degenerations as seen in Kenyan African myopes.

The detachment cases in (iv) were useful in determining any association of the degenerations, with retinal breaks and with retinal detachment.

Age	R E S U L T S		Total Eye
8-10			
11-20			
21-30			
31-40			
41-50	13(25.4)	3(5.4)	32
51-60	15(24.3)	1(1.6)	5
Total	168(45.7)	27(10.6)	255

The results are presented in forms of tables (absolute figures and percentages). Histograms have been constructed representing the incidence of various pre-degenerations and degenerations. The degenerations have been compared in various parameters like age, degree of myopia, and frequency of distribution in various quadrants of the eye.

Fundus drawings have been included on some selected cases to demonstrate various pre-degeneration and degeneration together with holes and detachments. Fundus photographs

Age	Degenerations				Total Eye
Years	Sattine	Ball-Track	Peripapillary	Peripheral	Total
8-10	1(1.6)	3(4.3)	1(1.2)	0(0)	5
11-20	1(1.7)	1(1.5)	1(1.4)	0(0)	3
31-40	2(4.2)	2(4.3)	4(9.5)	3(7.2)	11
41-50	4(12.5)	1(3.2)	3(9.4)	4(12.5)	12
51-60	5(21.8)	1(4.4)	6(26.1)	2(8.70)	14
Total	13(25.00)	6(0)	8(66.7)	3(25.00)	17
Total	17(6.6)	11(4.3)	23(9.6)	12(4.7)	255

would have been ideal for this purpose, however facilities for such fundus photography (demonstrating the periphery) are not available in our unit.

Table I Pre-Degenerations Vs Age

Age	Pre-Degenerations		Total Eye
Year	WWP	Snow-Flake	Examined
8-20	39(61.9)	8(12.7)	63
21-30	62(73.8)	10(11.9)	84
31-40	26(61.9)	5(11.9)	42
41-50	19(59.4)	3(9.4)	32
51-60	15(65.3)	1(4.4)	23
60+	7(58.4)	0(0)	12
Total	168(65.7)	27(10.6)	256

Table IV Incidence of Pre-Degeneration and degeneration

Table II Incidence of Degeneration in various age groups

Age	D e g e n e r a t i o n s				Eyes exam
Years	Lattice	Snail-track	Pigmentation	Cystoid	Total
8-20	1(1.6)	3(4.8)	2(3.2)	0(0)	63
21-30	2(2.4)	4(4.8)	2(2.4)	0(0)	84
31-40	2(4.8)	2(4.8)	4(9.5)	3(7.2)	42
41-50	4(12.5)	1(3.2)	3(9.4)	4(12.5)	32
51-60	5(21.8)	1(4.4)	6(26.1)	2(8.70)	23
60+	3(25.00)	0(0)	8(66.7)	3(25.00)	12
Total	17(6.6)	11(4.3)	25(9.8)	12(4.7)	256

Table III frequency of distribution by quadrants of pre-degenerations and degenerations

Quadrants	Pre-degenerations		Degenerations			
	'WWP'	Snow-flake	Lattice	Snail Track	Pigm.	Cystoid
UT	89(52.9)	15(55.6)	9(52.9)	6(52.9)	9(36.00)	3(25)
LT	46(27.4)	7(25.9)	4(14.8)	3(27.3)	6(24.00)	7(58.3)
LN	10(5.9)	0(0)	1(5.9)	0(0)	6(24.00)	1(8.3)
UN	23(13.7)	5(18.6)	3(17.6)	2(18.2)	4(16.00)	1(8.3)
	I68	27	I7	II	25	I2

Table IV Incidence of Pre-degeneration and degenerations compared in various degrees of myopia

Myopia	Pre-Deg.		Degenerations				Total
	'WWP'	Snow flake	Lattice	Snail track	Pig-ment	Cystoid	
1-2	74(44.1)	6(22.3)	2(11.8)	2(18.2)	14(56.00)	5(41.7)	10
2.25-4	56(33.4)	7(25.9)	3(17.7)	1(9.1)	6(24.00)	1(8.3)	7
4.25-6	31(18.5)	5(18.6)	5(29.4)	3(27.3)	7(28.00)	4(33.3)	5
6+	8(4.8)	9(33.3)	7(41.2)	5(45.5)	II(44.00)	2(16.7)	I
Total	I68	27	I7	II	25	I2	25

Table VI: Degeneration Vs. Retinal Detachment

Degenerations	Detaches	Small-Drum Sign.	Cystoid Total
Retinal holes	21(27.7)	3(7.7)	1(2.8)

Table V

Age (Yrs.)	Myopia in Diopters				Total
	1-2	2.25-4	4.25-6	6+	
8-20	37(35.9)	15(18.9)	9(15.3)	2(13.3)	63
21-30	33(32.1)	29(36.7)	21(35.6)	1(6.7)	84
31-40	18(17.5)	17(21.5)	5(8.5)	2(13.3)	42
41-50	6(5.8)	7(8.9)	14(23.7)	5(33.3)	32
51-60	7(6.8)	6(7.6)	8(13.6)	2(13.3)	23
60+	2(1.9)	5(6.3)	2(3.4)	3(20.00)	12
Total	103	79	59	15	256

Table VIII: Retinal detachment operations done within last 10 years - 1970-1980

Year	No.
1970	5
1971	4
1972	5
1973	15
1974	10
1975	11
1976	20
1977	27
1978	19
1979	33
1980 (April)	32
Total	162

Table VI: Degeneration Vs. retinal holes & Detachment

Degenerations	Lattice	Snail-Track Pigm.	Cystoid	Total
Retinal holes	II(64.70)	3(I7.7)	I(5.9)	2(II.8) I7
Retinal Detachment	5(50)	I(10.00)	I3(30.00)	I(10.00) IO

Table VII: Distribution of retinal breaks by quadrants

Quadrant	Dialysis	Horse shoe	Operculated round holes	Non-operculated round hole
UT	I(33.3)	3(75.00)	3(60.00)	2(33.3)
LT	I(33.3)	I(25.00)	I(20.00)	I(16.7)
UN	0(0)	0(0)	I(20.00)	2(33.3)
LN	I(33.3)	0(0)	0(0)	0(0.0)
Total	3	4	5	5

Table VIII: Retinal detachment operations done within last 10 years -K.N.H

Year	No.
1970	5
1971	4
1972	5
1973	15
1974	10
1975	12
1976	20
1977	27
1978	19
1979	33
1980 (April)	12
Total	162

Table I (Refer to Histograms 1,3 and 4 - Page 19 and 20)

The results presented here reflect the incidence of Pre-Degenerations in various age groups.

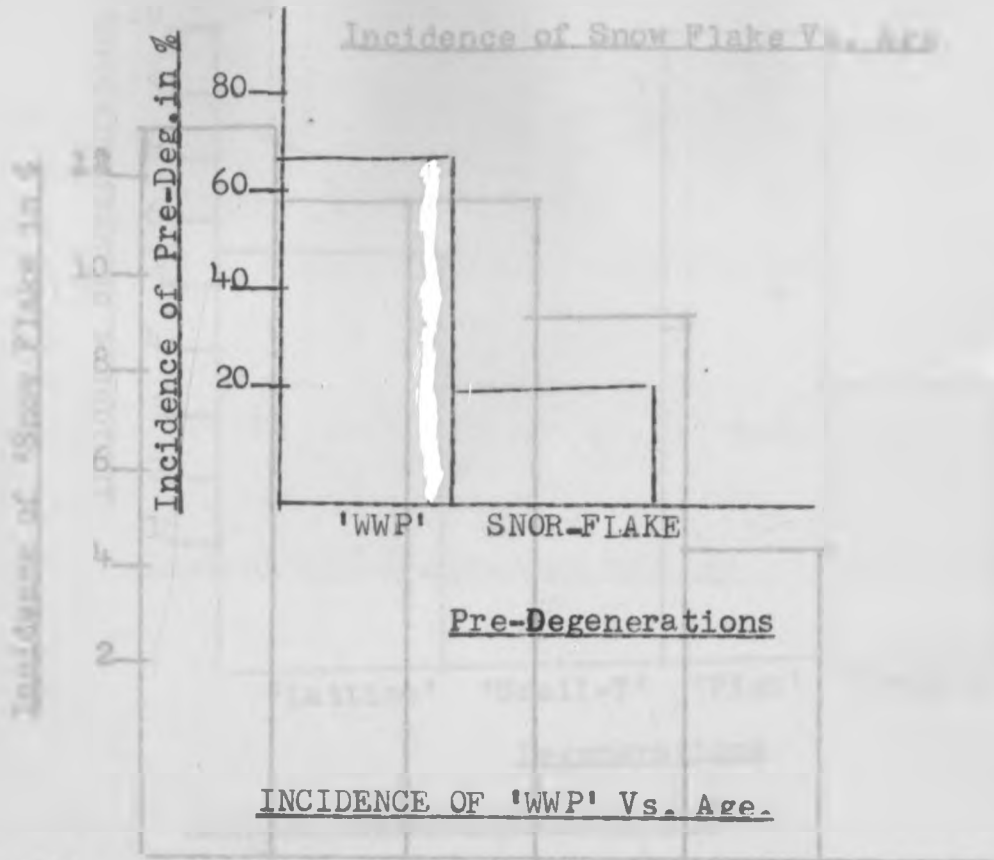
Histogram 1: demonstrates that "White without pressure" (WWP) has a very high incidence among myopes, compared to Snow Flake degeneration. The latter has an incidence of only (10.6%) compared to the former (65.7%), among the study group.

Histogram 3: represents the incidence of "WWP" as seen in various age-groups. It is apparent that age has very little influence on the incidence of white without pressure, the highest incidence was noted among the 21-30 age group i.e. (73.8%), while the lowest was among the 'above 60 years' age group (58.4%). The bias thus seems to favour the younger age group though not significantly. In any case the 51-60 age group showed the second highest incidence, demonstrating an irregular pattern of the incidence among the various age groups.

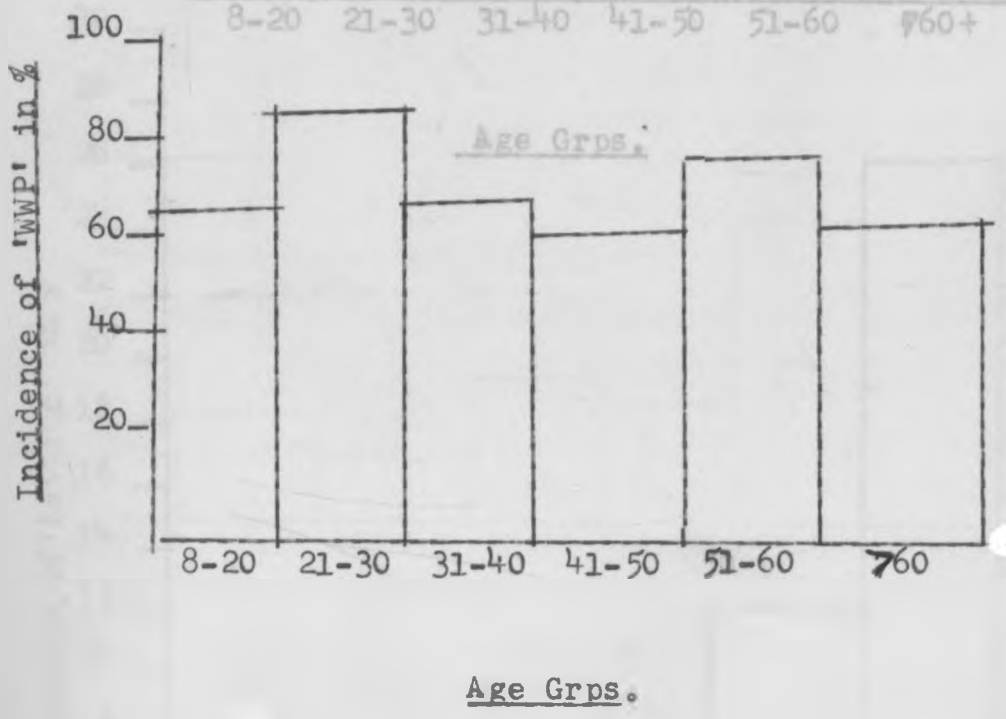
Histogram 4: This demonstrates the incidence of Snow-Flake degeneration among the various age groups. There seems to be a definite evidence of preponderance of this degeneration among the younger age group. The highest (12-7%) was seen in the 8-20 age group while **none** was seen after the age of 60 years.

INCIDENCE OF PRE-DEGEN.

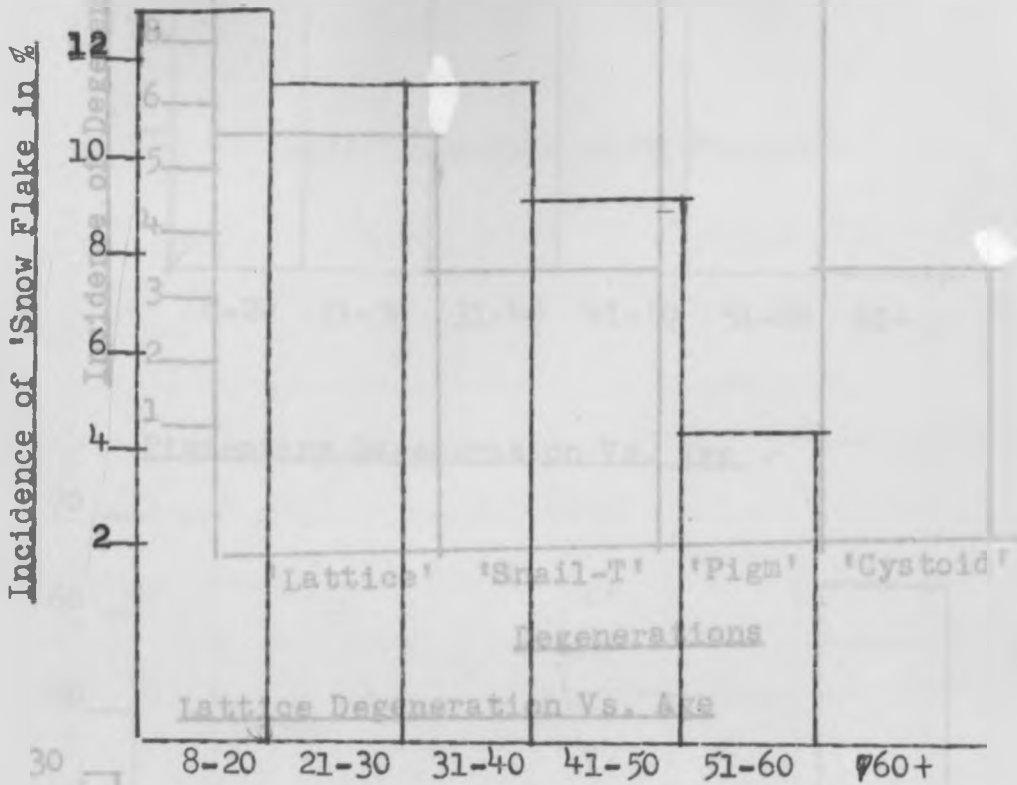
(1)



(3)



Incidence of Snow Flake Vs. Age

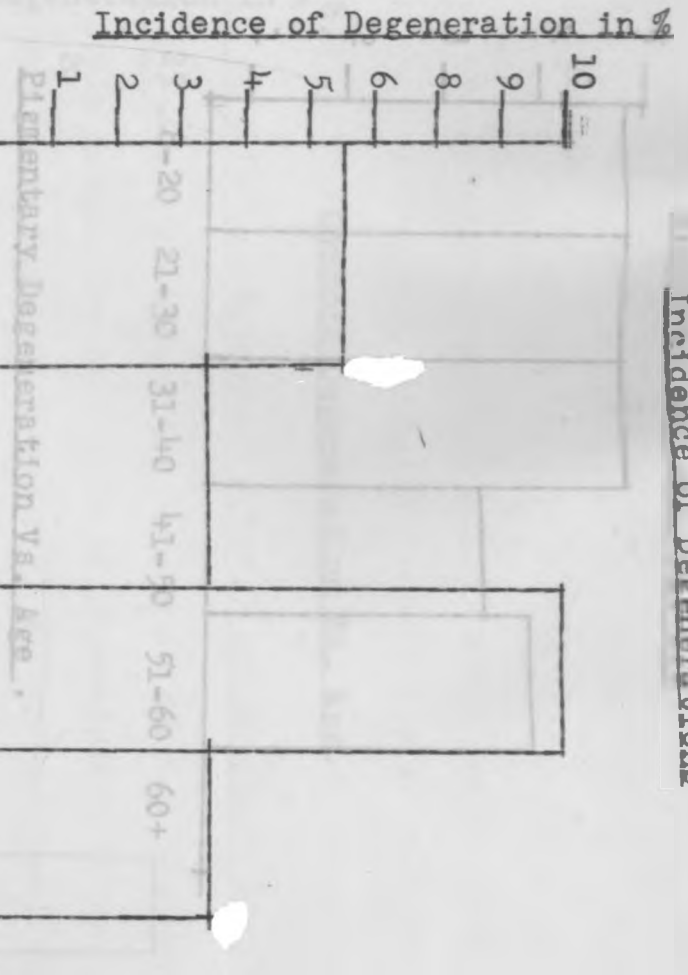


Age Grps.

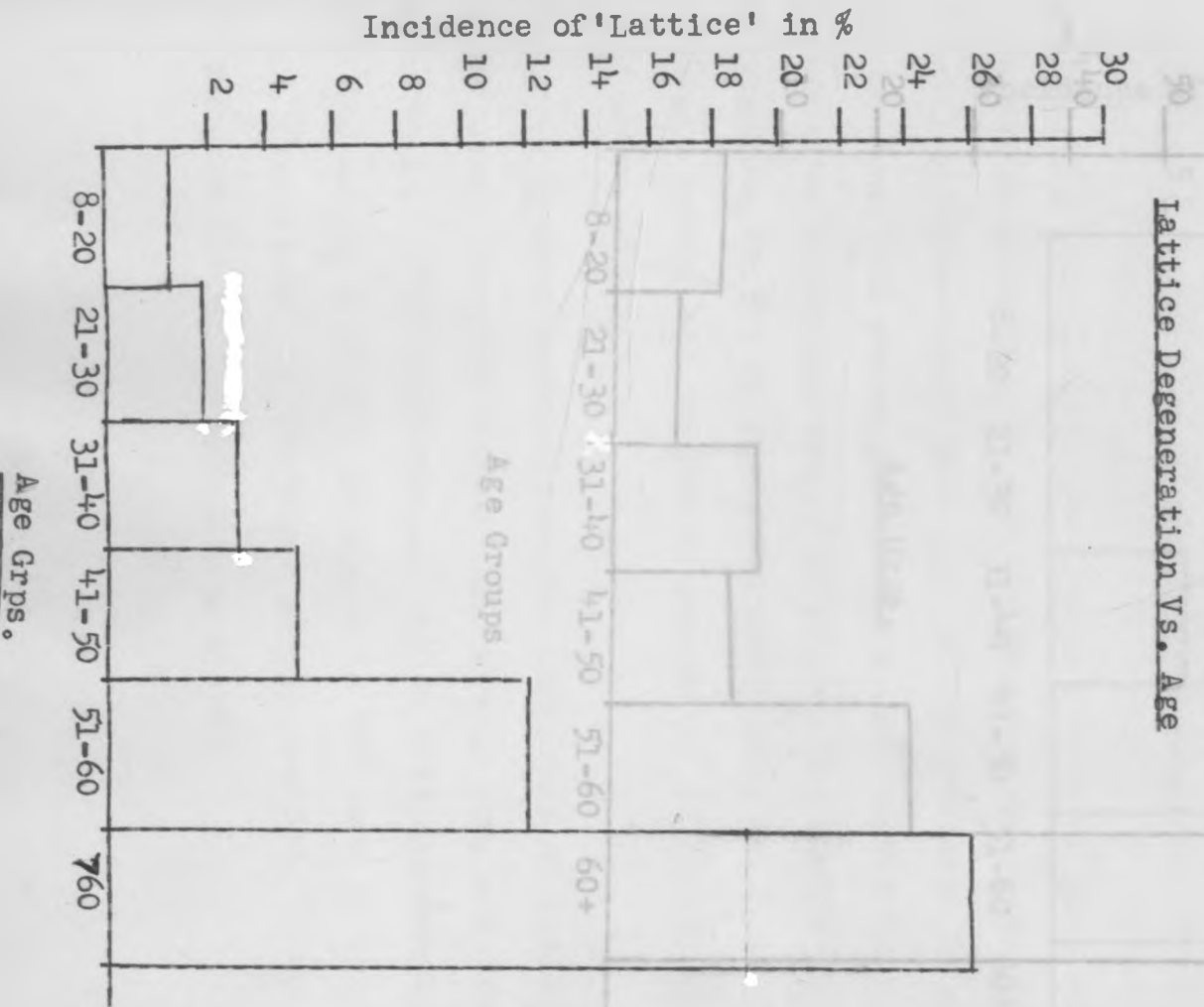


Incidence of Degenerations

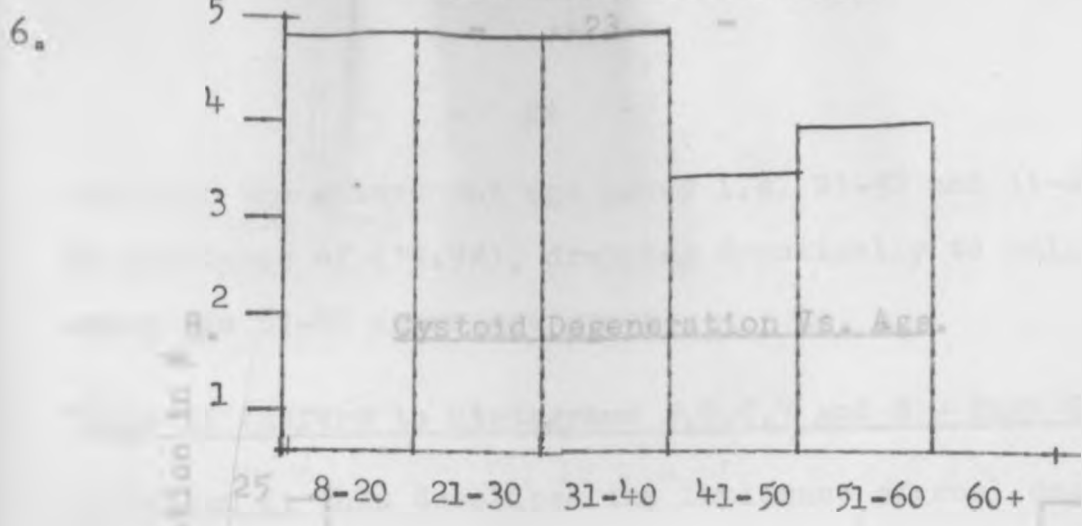
(2)



(5)

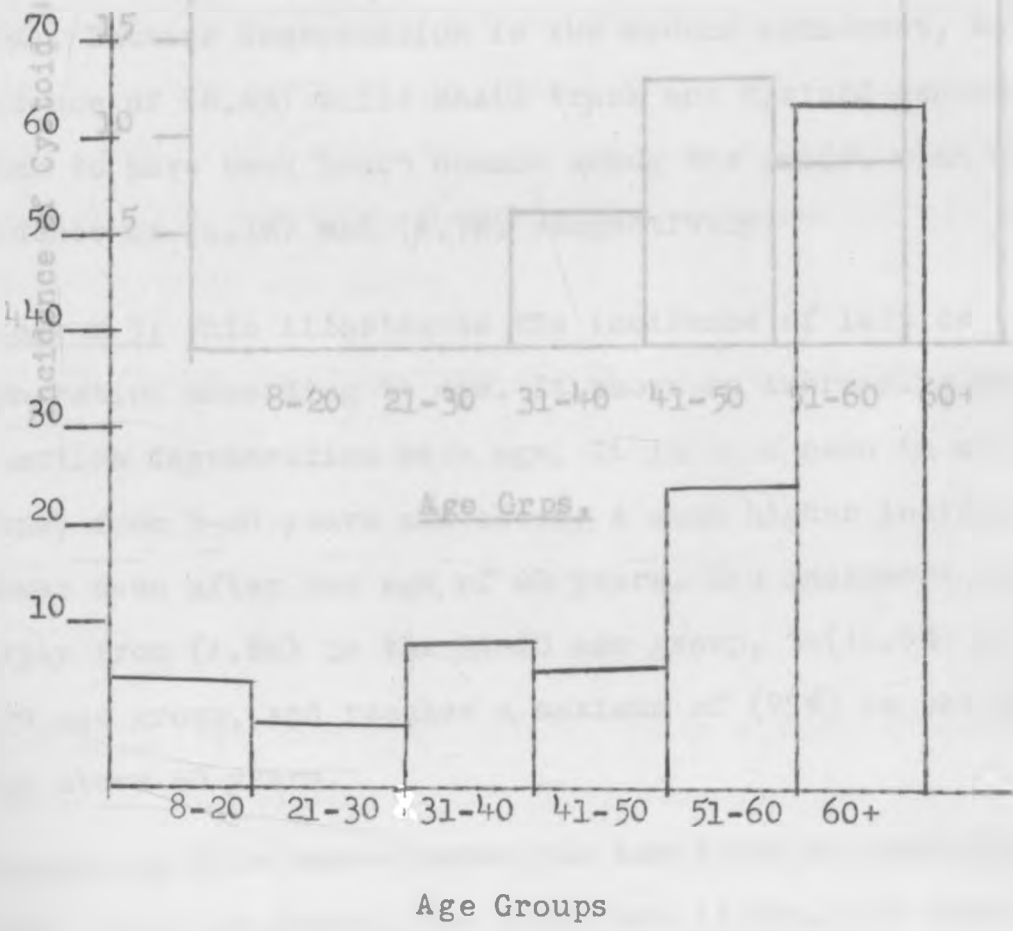


Snail Track Degrn. Vs. Age



Cystoid Degeneration Vs. Age

7. Pigmentary Degeneration Vs. Age .



and Justice Department with respect to the case, it is not clear that any

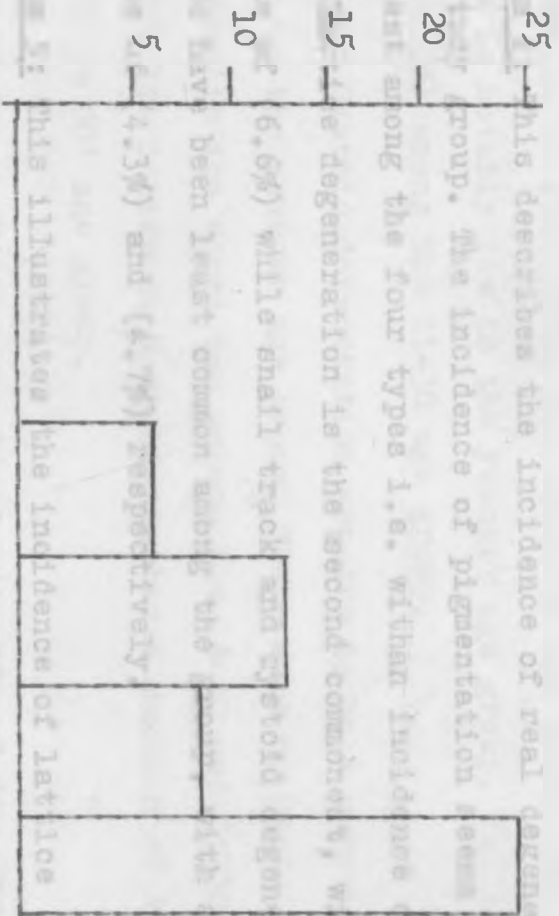
Age Grps.

A more detailed breakdown of the groups, from birth to 100 years, is available in the report. The data shows that the population is increasing in all age groups, but the increase is most rapid in the 15-20 age group, and is followed by the 20-25 age group, and the 25-30 age group. The 30-35 age group is also increasing, but at a slower rate. The 35-40 age group is decreasing, and the 40-45 age group is also decreasing, but at a slower rate. The 45-50 age group is also decreasing, but at a slower rate. The 50-55 age group is also decreasing, but at a slower rate. The 55-60 age group is also decreasing, but at a slower rate. The 60-65 age group is also decreasing, but at a slower rate. The 65-70 age group is also decreasing, but at a slower rate. The 70-75 age group is also decreasing, but at a slower rate. The 75-80 age group is also decreasing, but at a slower rate. The 80-85 age group is also decreasing, but at a slower rate. The 85-90 age group is also decreasing, but at a slower rate. The 90-95 age group is also decreasing, but at a slower rate. The 95-100 age group is also decreasing, but at a slower rate.

History of the Department The history of the Department of Social Security is a long and interesting one. It began in 1935 with the Social Security Act, which established the Social Security Administration. The Department was then known as the Social Security Administration. In 1946, the Department was renamed the Social Security Administration. In 1953, the Department was renamed the Social Security Administration. In 1961, the Department was renamed the Social Security Administration. In 1970, the Department was renamed the Social Security Administration. In 1978, the Department was renamed the Social Security Administration. In 1980, the Department was renamed the Social Security Administration. In 1982, the Department was renamed the Social Security Administration. In 1984, the Department was renamed the Social Security Administration. In 1986, the Department was renamed the Social Security Administration. In 1988, the Department was renamed the Social Security Administration. In 1990, the Department was renamed the Social Security Administration. In 1992, the Department was renamed the Social Security Administration. In 1994, the Department was renamed the Social Security Administration. In 1996, the Department was renamed the Social Security Administration. In 1998, the Department was renamed the Social Security Administration. In 2000, the Department was renamed the Social Security Administration. In 2002, the Department was renamed the Social Security Administration. In 2004, the Department was renamed the Social Security Administration. In 2006, the Department was renamed the Social Security Administration. In 2008, the Department was renamed the Social Security Administration. In 2010, the Department was renamed the Social Security Administration. In 2012, the Department was renamed the Social Security Administration. In 2014, the Department was renamed the Social Security Administration. In 2016, the Department was renamed the Social Security Administration. In 2018, the Department was renamed the Social Security Administration. In 2020, the Department was renamed the Social Security Administration.

Cystoid Degeneration Vs. Age.

Incidence of Cystoid Degeneration in %



8-20 21-30 31-40 41-50 51-60 60+

Moreover the subsequent age group i.e. 21-30 and 31-40 had an incidence of (11.9%), dropping drastically to only (4.4%) among his 51-60 years age group.

Table II (Refer to Histograms 2,5,6,7 and 8) - Page 21-23)

Histogram 2: This describes the incidence of real degenerations in our study group. The incidence of pigmentation seems to be the highest among the four types i.e. with an incidence of (9.8%). Lattice degeneration is the second commonest, with an incidence of (6.6%) while snail track and cystoid degeneration appear to have been least common among the group, with an incidence of (4.3%) and (4.7%) respectively.

Histogram 5: This illustrates the incidence of lattice degeneration according to age. It shows an increasing incidence of lattice degeneration with age. It is seen in all age groups, from 8-60 years and above. A much higher incidence is however seen after the age of 40 years. The incidence rises sharply from (4.8%) in the 31-40 age group, to (12.5%) in the 41-50 age group, and reaches a maximum of (25%) in the age group above 60 years.

Histogram 6: This demonstrates the incidence of snail-track degeneration. In general the incidence is low, and there seems to be little influence of age on this degeneration. From the age of 8-40 the incidence is the same in our study group, i.e. (4.8%) in the three groups covered. It is about the same in the 60 age group i.e. (4.4%).

Histogram 7: Illustrates the incidence of pigmentary degenerations among various age groups. There is a marked increase in the incidence after the age of 50 i.e. 51-60 age group shows an incidence of (26.1%) followed by a sharp increase to (66.7%) in the above 60 age group. The incidence is notably low in the younger age group, falling down to (2.4%) among the 21-30 age group.

Histogram 8: Cystoid degeneration appears to be a very rare condition in the younger age group. There were no cystoid changes seen in the first two groups i.e. 8-20 and 21-30 age groups. Its incidence however reaches a maximum of 25% in the 'above 60' age group.

Table III (Refer to Histograms 9, 10, 11, 12 and 14 - Page 27-30)

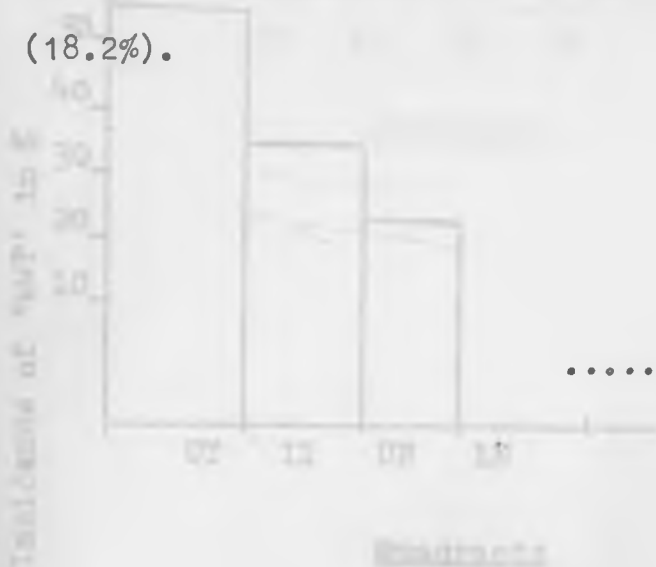
Table III refers to the frequency of distribution of the Pre-degenerations and degenerations within the four quadrants of the eye i.e. Upper Temporal (UT), Lower temporal (LT), Upper nasal (UN) and Lower nasal (LN).

Histogram 9: The Pre-degeneration 'White without Pressure' found to be more common in the upper temporal quadrant of the retina compared to other quadrants. The highest percentage (52.9%) were noticed in the UT, while second highest distribution was noted in the LT (27.4%) followed by UN (13.7%) and LN (5.9%).

Histogram 10: A similar distribution was noted for snow-flake degeneration. The quadrant with the highest distribution was found to be UT, (55.6%), followed by LT (25.5%). The distribution dropped to (18.6%) in UN while none of the patients examined had snow-flake degeneration in the lower nasal quadrant.

Histogram 11: The distribution of lattice degeneration was similarly found to be highest in the UT quadrant (52.9%), followed by UN quadrant (17.6%). The LT distribution was noted to be (14.8%) while like the preceding pre-degeneration the LN had the least frequency of distribution (5.9%).

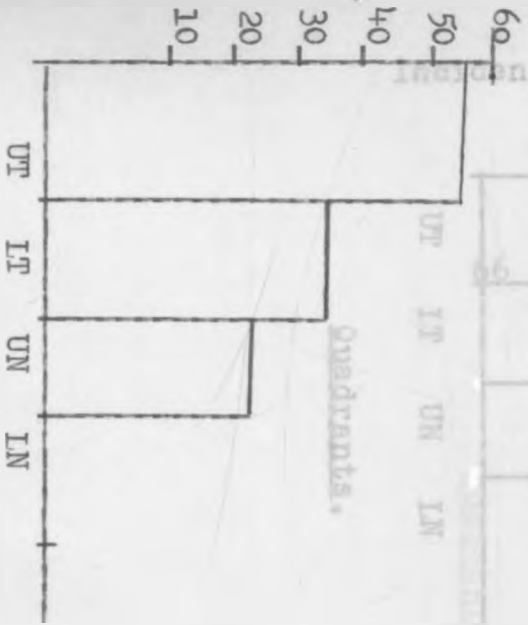
Histogram 12: Snail-track degeneration had its highest frequency of distributions in the upper temporal quadrant (54.6%), followed by lower temporal quadrant (27.3%) while none of the patients examined had this degeneration in the lower nasal quadrant. The upper nasal quadrant had a frequency distribution of (18.2%).



...../27

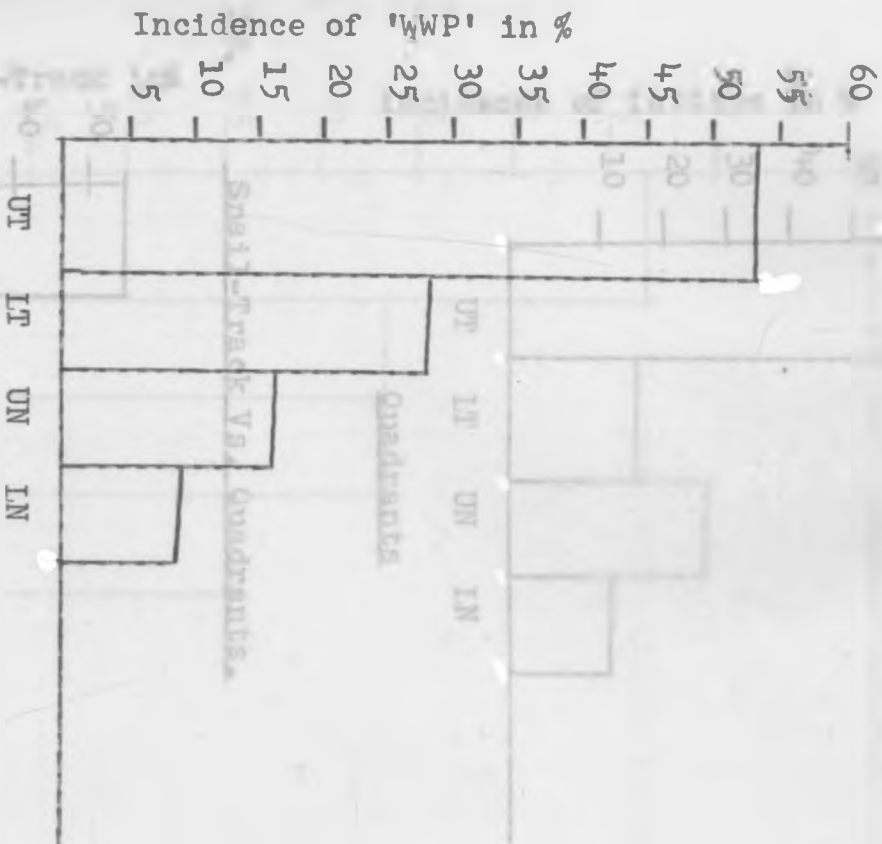
10. 'Snow-Flake' Vs. Quadrants

Incidence of 'WVP' in %



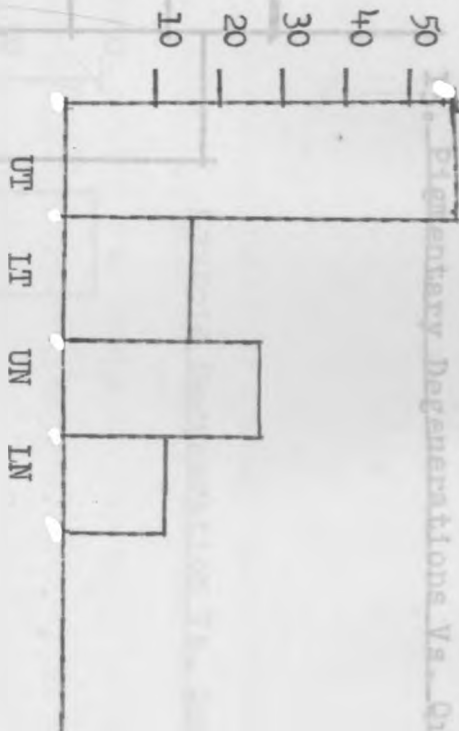
Quadrants

9. 'WPI' Vs. Quadrants



Quadrants.

Incidence of Lattice in %



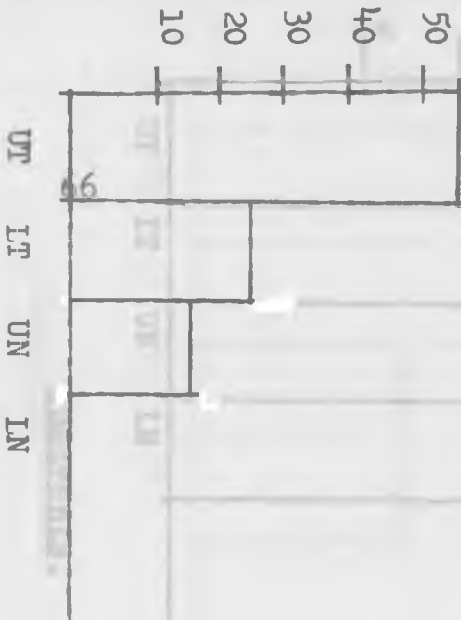
Quadrants

Incidence of Elementary Degn. in %

12.

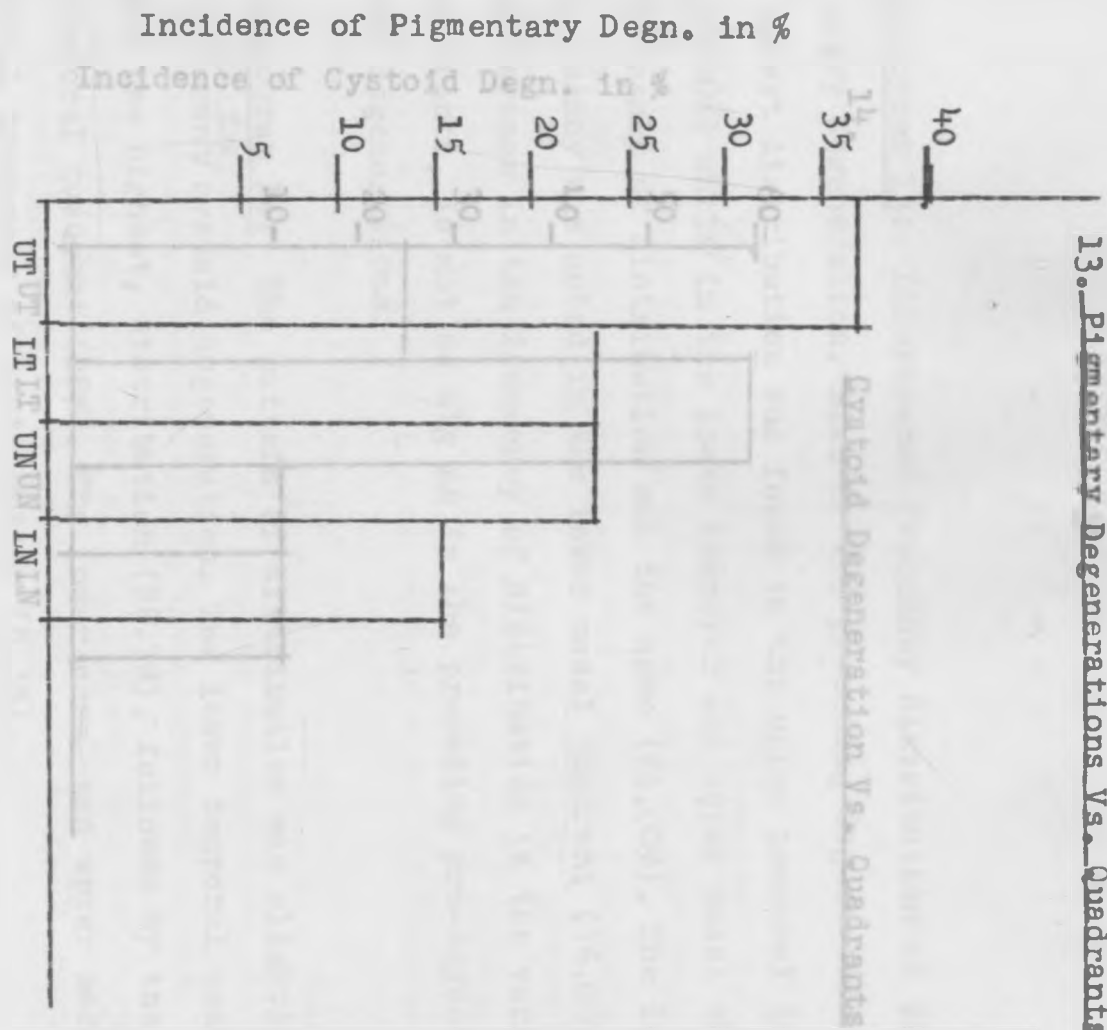
Snail-Track Vs. Quadrants

Incidence of Snail-Track in %



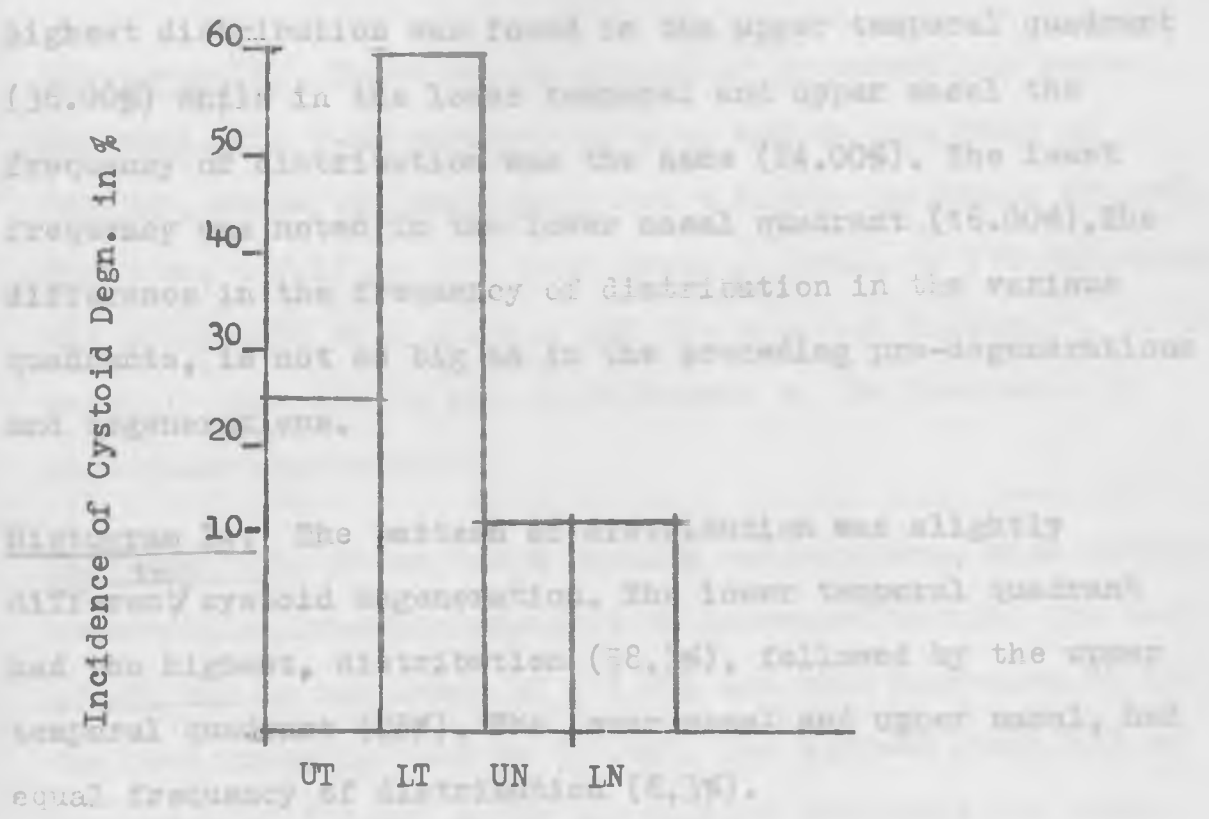
Quadrants.

13. Pigmentary Degenerations Vs. Quadrants



Histogram 13: Illustration frequency distribution of 302-

14. Cystoid Degeneration Vs. Quadrants



Quadrants

Table IV (continued from Histogram 13-36)

Table IV illustrates the incidence of various pre-degenerations and degenerations as seen in various degrees of myopia. The letter has been divided into four groups: 1-4, 1-20, 2.25-40, 4.25-60, 60+.

Histogram 15: The least myopic group i.e. 1-20 group had the highest incidence of 'CST' (44.1%), followed by the next group 2.25-40 which had (33.3%). There was a sudden fall of the incidence, to (16.3%) among the next group (4.25-60) and with a greater fall to (1.3%) among the group with more than 60

Histogram I3: Illustrates frequency distribution of pigmetary degeneration. Like in the preceding degeneration, the highest distribution was found in the upper temporal quadrant (36.00%) while in the lower temporal and upper nasal the frequency of distribution was the same (24.00%). The least frequency was noted in the lower nasal quadrant (16.00%). The difference in the frequency of distribution in the various quadrants, is not as big as in the preceding pre-degenerations and degenerations.

Histogram I4: The pattern of distribution was slightly different in cystoid degeneration. The lower temporal quadrant had the highest, distribution (58.3%), followed by the upper temporal quadrant (25%). The lower nasal and upper nasal, had equal frequency of distribution (8.3%).

Table IV (Refer to Histogram I5-20- Page 34-36)

Table IV illustrates the incidence of various pre-degenerations and degenerations as seen in various degrees of myopia. The latter has been divided into four groups. i.e. 1-2D, 2.25-4D, 4.25-6D, 6D+.

Histogram 15: The least myopic group i.e. 1-2D group had the highest incidence of 'WWP' (44.1%), followed by the next group 2.25-4D which had (33.3%). There was a sudden fall of the incidence, to (18.5%) among the next group (4.25-6D) and even a greater fall to (4.8%) among the group with more than 6D

It seems that myopia influences WWP i.e. the higher the myopia the less is the incidence of WWP.

Histogram I6: There was an irregular/in ^{pattern} snow-flake degeneration. The 1-2D group of myopia, had an incidence of (22.3%), while the next group had a higher incidence (25.9%). There was a fall in the third group 4.25-6D, to (18.6%) while the group with high myopia, had the greatest incidence. It is possible that myopia has no influence on the incidence of snow-flake degeneration.

Histogram I7: There was a regular pattern here, with lattice degeneration steadily rising with increased myopia. The 1-2D group, had an incidence of (11.8%) and this increased up in the next group 2.25-4D to (17.6%). The 4.25-6D group had an incidence of (29.4%) while the highest incidence was noted in the 6D+ group i.e. (41.2%).

(A possible explanation to this pattern is the degenerative changes (Chorioretinal) which accompany a higher degree of myopia might hasten the changes seen in lattice degeneration).

Histogram 18: Snail-track degeneration also seems to have been influenced by myopia. The least incidence was noted in the 2.25-4D group 9.1% while the highest incidence was noted in the 6D+ group. (45.5%). It is difficult to explain why the group with 1-2D had a slightly higher incidence than the second group, i.e. the first group had an incidence

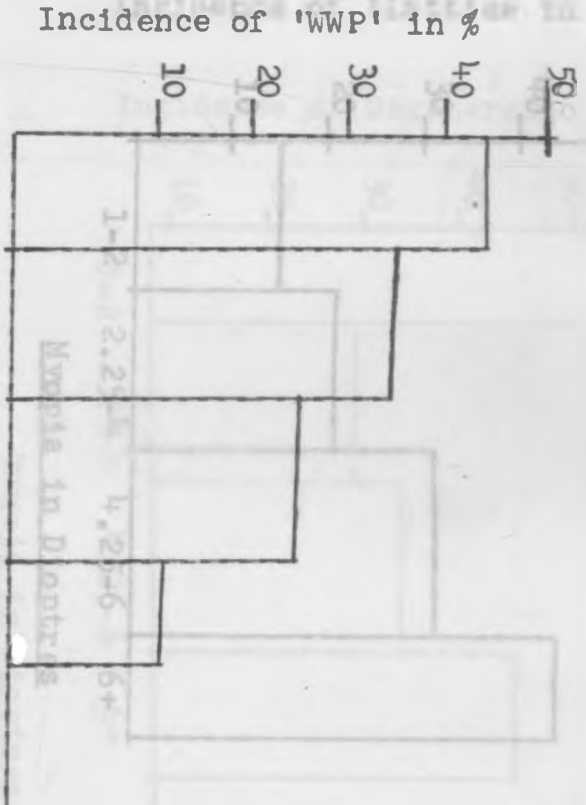
of 18.2% (c.f. 9.1% of the 2nd group). This could have been an error in observation. However the general rising pattern might have similar explanation to that of lattice degeneration. The changes in the vitreous and the chorioretinal degenerative changes in higher degree of myopia might hasten the appearance of snail-track degeneration.

Histogram 19: The highest incidence of pigmentary degeneration was noted among the 1-2D group (56.00%) while second highest was noted in the 6D+ group (44.00%). The least incidence was in the 2.25-4D group (8.3%). This irregular pattern possibly indicates that myopia might have no influence on peripheral, pigmentary degeneration.

Histogram 20: Cystoid degeneration, similarly had an irregular pattern, with the highest incidence being in the 1-2D group (41.7%), while the second highest was in the third group 4.25-6D group (33.3%). The least was the 2.25-4D group with an incidence of (8.3%).

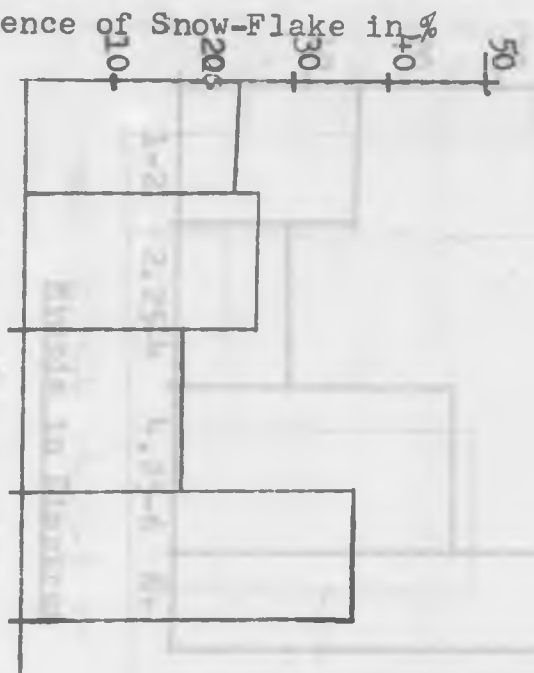
This pattern also indicates that myopia has no influence on cystoid degeneration changes.

15. 'WVP' Vs. Myopia



Myopia in Dioptres

16. Snow-Flake Vs. Myopia

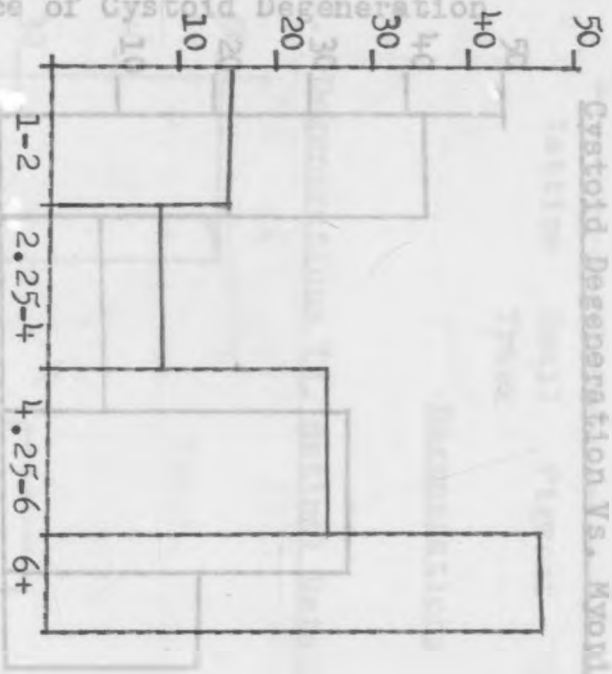


Myopia in Dioptres

'Snail-track' Vs. Myopia

Incidence of Snail-track in %

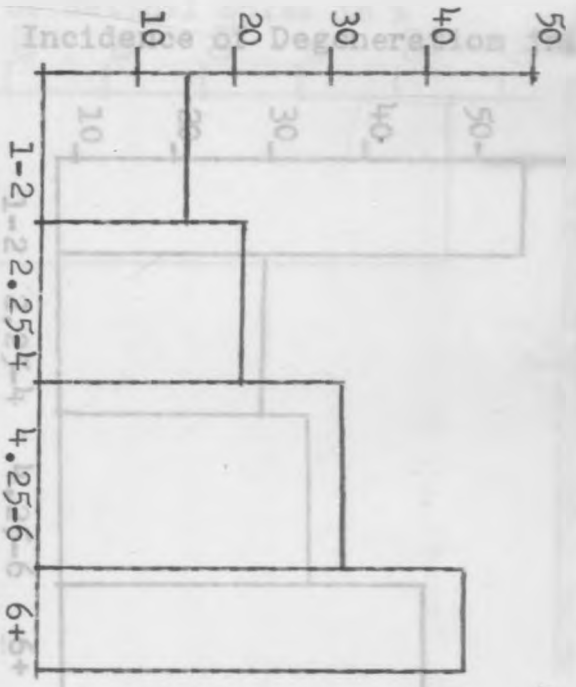
Incidence of Cystoid Degeneration



Myopia in Dioptres

Myopia in Dioptres

Incidence of 'Lattice in %

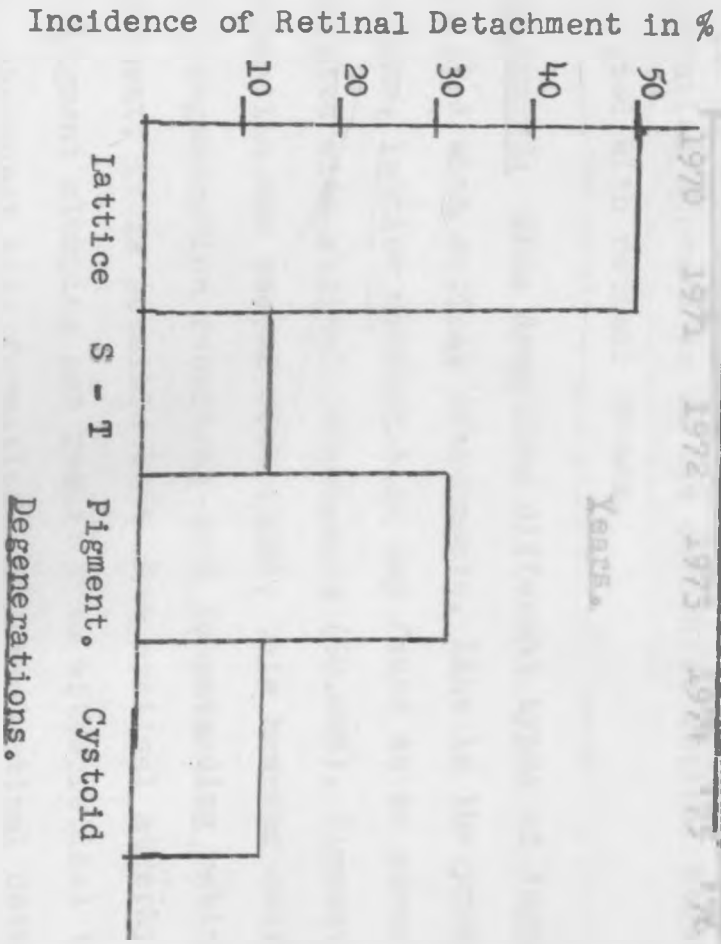


UNIVERSITY OF NAIROBI LIBRARY

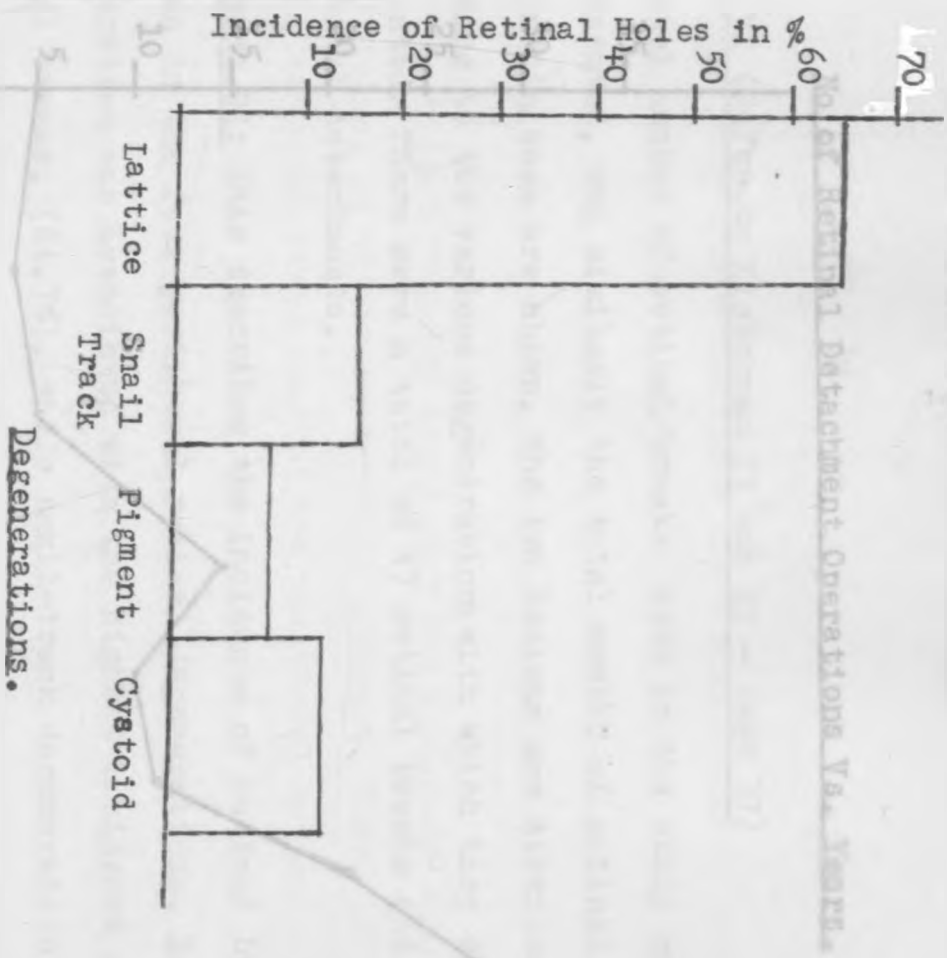
Myopia in Dioptres

Myopia in Dioptres

22. Degenerations Vs. Retinal Detachments.



21. Degenerations Vs. Retinal Holes



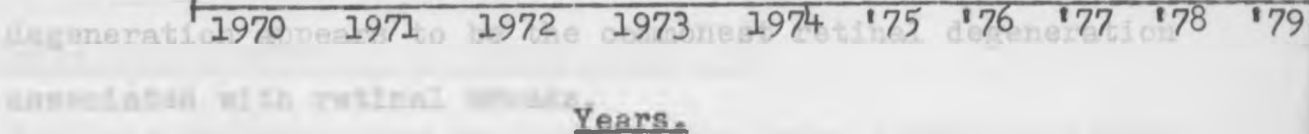
Degenerations.

No of Retinal Detachment Operations Vs. Years.

Table VI (Refer to Histogram 21 and 22 - Page 37)

The total number of retinal breaks seen in the study group is presented, and similarly the total number of retinal detachments seen are shown. The two lesions are distributed according to the various degenerations with which they were associated. There were a total of 17 retinal breaks and 11 detachments.

Histogram 21: This describes the incidence of retinal breaks as seen in the four peripheral retinal degenerations. Lattice degeneration was associated with the highest incidence of retinal breaks, (64.7%), while snail-track degeneration was associated with (17.7%) cystoid and pigmentary degenerations had an incidence of (11.8%) and (5.9%) respectively. Lattice



Histogram 22: This describes different types of degenerations associated with retinal detachments. Like in the preceding histogram, lattice degeneration was found to be commonly associated with retinal detachments (59.09%). Pigmentary degeneration was associated with (30%), this however could have been a degeneration resulting from longstanding retinal detachment. It is possible that choroidal atrophy in areas with pigment clumping can result into vitreoretinal traction with subsequent hole formation and later retinal detachment.

Table VI (Refer to Histogram 21 and 22 - Page 37)

The total number of retinal breaks seen in the study group is presented, and similarly the total number of retinal detachments seen are shown. The two lesions are distributed according to the various degenerations with which they were associated. There were a total of 17 retinal breaks and 10 retinal detachments.

Histogram 21: This describes the incidence of retinal breaks as seen in the four peripheral retinal degenerations. Lattice degeneration was associated with the highest incidence of retinal breaks, (64.7%), while snail-track degeneration was second with (17.7%) cystoid and pigmentary degenerations had an incidence of (11.8%) and (5.9%) respectively. Lattice degeneration appears to be the commonest retinal degeneration associated with retinal breaks.

Histogram 22: This describes different types of degenerations associated with retinal detachments. Like in the preceding histogram, lattice degeneration was found to be commonly associated with retinal detachments (50.00%). Pigmentary degeneration was second with (30%), this however could have been a degeneration resulting from longstanding retinal detachment. It is possible that chorioretinal atrophy in areas with pigment clumping can result into vitreoretinal traction with subsequent hole formation and later retinal detachment.

DISCUSSION

Table VII: This table describes the distribution of retinal breaks and dialyses by quadrants, and also breaks down the retinal tears into various types i.e. dialyses horse-shoe types, operculated and non-operculated holes.

It is evident from the table that the commonest site for retinal tears, is the upper temporal quadrant. This is probably expected since most of the peripheral retinal degenerations have been found to be commonest in this quadrant (Ref. table III). Second commonest was the lower temporal quadrant except the non-operculated holes which were found (33.3%) in the UN quadrant and (33.3%) upper temporal quadrant. The dialyses however seem to have had equal distribution in the three quadrants UT,LT and LN.

Table VIII (Refer to the graph - Page 38)

This is a retrospective analysis of the total number of retinal detachments operated upon at the Kenyatta National Hospital within the last 10 years. The general pattern of the graph, representing these figures, indicates an upward trend on the number of cases of retinal detachments being operated on at the Kenyatta National Hospital every year. The most likely explanation for this is the increasing availability of better diagnostic facilities.

D I S C U S S I O N

1. Pre-Degenerations

(i) White without Pressure(WWP)

This is the whitish opaque appearance which is seen at the periphery of the retina. In some eyes, it is only seen after indenting the retina, when it is referred to as 'white with pressure'. Referring to Table I on the incidence of this condition among our study group, it can be stated that it is the commonest type of pre-degeneration seen among African myopes of all degrees. It has been reported by J.J. Kanski (1975) that 'white without pressure' is a normal finding in Negro eyes. White without pressure is a more marked form of white with pressure. The high incidence in African myopes is therefore not surprising. It is however interesting to note that it's incidence tends to decline with higher degrees of myopia (Ref. to Table III Histg.9). It was seen in more than half of the 10 cases of retinal detachment seen and operated on during the period of study.

Although it was noticed present together with other forms of degenerations like 'lattice' or 'snail-track' there is no evidence that white without pressure progresses to other forms of degenerations. Large retinal tears have been noticed in the posterior edges of these lesions (Clinical signs in ophthalmology special edition by J.J.Kauski and Paul Henkind). It can be said that if the vitreous detaches itself from the site of vitreoretinal adhesion,

without causing a retinal tear, than the situation remains safe. If however the vitreoretinal traction persists, then the risk of retinal detachment remains high. The exact clinical significance of these lesions however has not been fully understood.

The pathology possibly lies in the adhesion between the cortex of the vitreous and the inner layer of the retina. It is common in retinopathy of pre-maturity and in areas with microcystoid degeneration in adults.

(ii). Snow-flake degenerations: This was the second pre-degeneration seen in our study group. It had a much lower incidence than 'WVP' i.e. (10.6%). Clinically this is seen in two forms, the DIFFUSE type and the CIRCUMSCRIBED type.

(a). The Diffuse Type: White minute or whitish-yellow spots scattered in the periphery on the retina mostly anterior to the equator. They have an irregular posterior border and some posterior extensions are only along the blood vessels. (Ref. Photograph 2(ii)).

(b). The circumscribed type: Glistening yellow-minute dots extending from the periphery, to the equator with well demarcated borders. They take different shapes. Some are oval, others are elongated and circumferential to the equator. Cortical vitreous becomes thickened over the craters.

In the present series, both types were observed and their distribution in the fundus had a higher frequency in the upper temporal-quadrant while none appeared in the lower nasal quadrant. The latter is in contrast to what others have found Hunter, D.M. (1974). Although there is no significant difference in its incidence in the younger age group (upto 40), the incidence is notably less after the age of 50, and in the present study group, none was seen after the age of 60. This is comparable to what had been reported earlier, Hunter D.M. (1974). It is possible that with time, some areas where snow-flake degeneration occurs, increased pigmentation takes place, the retina become atrophic, and white dots of 'snow-flake' become less apparent, and hence the low incidence in the older age group.

Myopia does not seem to have a significant influence on snow-flake degeneration, although there is a slight bias to a higher incidence with increased degree of myopia (Ref. Table IV Histogram I6). It was found in only three out of 17 retinal breaks recorded during this study and the three breaks had retinal detachment. It was however present together with other real degenerations 'lattice' and 'snail-track! It is not easy from such a small series to make a conclusion of progression from snow-flake to lattice or snail-track degeneration. There are specific features noted in lattice degeneration which help to differentiate it from snow-flake, for example lattice degeneration is rarely seen

at the ora serrata. Lattice also takes a lace-like pattern with a criss-cross network of fine white lines (occluded vessels) having small discrete yellow particles in-between. It is therefore not an early lattice degeneration. It has been suggested by Hunter, D.M. (1974) that snow-flake degeneration can be inherited as an autosomal dominant mutant gene.

Using the degeneration, lattice degeneration is most commonly associated with retinal holes and retinal detachment. Similar findings were observed in this study. Out of the 76 eyes examined only 17 eyes had lattice degeneration (22.37%), however, out of the retinal hole eyes, 11 had lattice degeneration (64.71%) associated with these (64.71%). There were 5 cases of retinal detachment arising from the 11 holes associated with lattice degeneration (45.45%). These figures indicate that although the incidence of lattice degeneration is low, where it occurs, it gives a higher risk of developing retinal holes and the latter carries as high as 45.45% risk of developing retinal detachment. It is therefore a dangerous retinal degeneration. These figures though slightly on the slight side, are comparable to what other authors have found. Byar (1967) stated that 50% of retinal holes are directly associated with lattice degeneration. Stanton and Allan (1962) found that 30% of patients with retinal detachment are associated with lattice degeneration. The present study

D E G E N E R A T I O N S

2. (i). Lattice Degeneration: This degeneration was first recognized by Gonin (1934), then described as palisade degeneration, and it was not until 1952, that Schepens suggested the term 'lattice' which has since, become a common terminology describing the degeneration. Lattice degeneration is most commonly associated with retinal holes and retinal detachment. Similar findings were observed in this study. Out of the 256 eyes examined only 17 eyes had lattice degeneration (6.6%), however, out of the retinal holes seen, 11 had lattice degeneration closely associated with them (64.7%). There were 5 cases of retinal detachment arising from the 11 holes associated with lattice degeneration (45.5%). These figures indicate that although the incidence of lattice degeneration is low, where it occurs, it gives a higher risk of developing retinal breaks and the latter carries as high as 45.5% risk of developing retinal detachment. It is therefore a dangerous retinal degeneration. These figures though slightly on the higher side, are comparable to what other authors have found. Byer (1967) stated that 55% of retinal breaks are directly associated with lattice degeneration. Staatsma and Allan (1962) found that 31% of patients with retinal detachment are associated with lattice degeneration. The present study

also confirms that lattice degeneration is associated more with retinal breaks, than with retinal detachment.

Age, seems to influence the incidence of lattice degeneration.

It is less common in the younger age group than in the older age group (Ref. Table II Histogram 5). A possible explanation is that senile changes at the periphery of

the retina, such as ischaemia due to arteriosclerosis chorio-retinal and vitreal changes might make lattice more apparent if not hasten its appearance. Lattice seen in childhood in reaches its peak in the 2nd decade (Teng C.C. and Katzin, H.M.

The pathology of lattice degeneration has been studied in flat preparations of the retina. The histology reveals a dense adherence of the vitreous to the vessels in the lattice lesions. There is a surrounding rim of vitreous adhesions present, with liquified vitreous overlying, the central portion of the lesion. The white lines often seen, are occluded vessels, and it has been noticed that arteries and veins are equally affected. The pathological process at first leaves the inner layers of the retina intact, in the more marked stages, some glial tissue and the outer limiting membrane alone, may remain. Pigmentary epithelium shows areas of clumping and rarefactions.

(ii) SNAIL-TRACK DEGENERATION

The description as given under the chapter of definitions, is that of sharply demarcated bands of white crinkles or frost-like change of the inner retinal surface. Vitreoretinal lesions are seen over the snail-track lesions especially at the margins.

The majority of the cases seen during the study showed features described above, however some cases showed a confluence of such single lesions giving an appearance of a large white sheet in the pre-equatorial regions, and some of these were seen adjacent to areas of definite lattice degeneration. The incidence in study group was only 4.3% and the majority of these were patients below 40 years. (Table II Histogram 6).

There were no cases of snail-track degeneration seen in patients more than 60 years old. The condition tends to be common in young myopes. This is in conformity with what other writers have found. Aaberg and Stevens (1972) reported a hereditary tendency and a predilection for young myopes.

It's association with retinal breaks, was only second to lattice degeneration i.e. (17.7%). The incidence of retinal detachment among this group was 33.3%, and all the patients

...../

were less than 40 years old. It can therefore be concluded that snail-track degeneration is a dangerous rhegmatogenous lesion i.e. a severe sign of retinal degeneration which gives trophic chorioretinal changes with subsequent vitreous traction leading to retinal breaks and later retinal detachment.

The frequency of distribution in various quadrants is similar to that of lattice degeneration and the influence of myopia similarly takes the same pattern as that of lattice degeneration. i.e. it increases with high myopia (comparable to findings of Aaberg, T.N., Stevens, T.R. 1972).

In the present study, a few points are notable about pigmentation. Snail-track degeneration has a familial tendency with autosomal traits in males and females, the possible mode of inheritance is autosomal dominant with low penetrance or recessive with both parents, heterozygous. (T.M. Aaber and T.R. Stevens 1972). The same workers have however remarked that these findings are questionable.

Peripheral pigmentary changes seem to have very little influence from degree of myopia.

There was no definite pattern, i.e. zones in the group

(iii) Pigmentary degeneration

Peripheral pigmentary degenerations are senile changes which can take either a diffuse or clumping pattern. In themselves, these are said to be innocuous. However, they are known to occur in association with retinal tears and even retinal detachment. Dumas and Schepens (1966) reported a relationship between U-shaped tears and pigment clumps. J.J. Kanski (1975) suggested that the formation of tears depends on the presence or absence of vitreous traction rather than the size, shape or degree of pigmentations.

In the present study, a few points are notable about pigmentary degeneration of the retina. The lesion was confirmed to be largely present in the older age group (Table II. Histo. 7). The incidence in the (8-20) age group was as low as 3.2% while in the 60+ age group it was as high as (66.7%). It's association with retinal breaks was seen in only 1 patient out 17 cases of retinal breaks (5.9%). It's incidence in association with retinal detachment however was much higher (30%) this could be because of chorioretinal atrophy with hyperplasia of pigment epithelium resulting from longstanding retinal detachment.

Peripheral pigmentary changes seem to have very little influence from degree of myopia.

There was no definite pattern, i.e. myopes in the group

1-2D had an incidence of (56%) while the 2.25-4D group had an incidence of 24%, and this rises again to 44% in the 6D+ group. A young patient with myopia of -8D was noted to have extensive peripheral pigmentation with some vitreous traction, more extensive temporally. Other features in the rest of the fundus were consistent with retinopathy of prematurity and this was confirmed from history.

The pigmentation seen in the periphery particularly those along the ora serrata are possibly due to ischaemic changes i.e. the ora being quite distal to the source of blood supply. The pigment epithelium therefore degenerates and these are what appear as pigment clumps along the extreme periphery of the retina.

noticed before, most of them were in the lower temporal. There seems to be an influence of the incidence from age (Table IV Histogram 20). It has a greater proportion in the older age group, and none of the subjects below the age of 30 had the cystoid degeneration.

The pathology of retinoblastoma involves splitting of the retina into two layers usually at the outer limiting layer, i.e. between the nuclei of the photoreceptors and the bipolar cells. There are two forms the diffuse and focal types, and the former results from degenerative changes in the elderly, while

(iv) CYSTOID DEGENERATION

Microcystoid degeneration are said to be present in all adults increasing in severity with age, Kanski(1975). They are not directly related to retinal detachment but their progression into retinoschisis might lead to retinal detachment if the schisis develops holes in the inner and outer leaves.

A total of 12 cystoid degenerations were seen and none of them had typical features of retinoschisis. These were cystoid changes with varying sizes. Two were seen in eyes with retinal holes, however the holes were not associated with the cystoid areas. Similarly the one case seen in association with retinal detachment, had no direct association since the retinal break was found in a different quadrant and was central to the equator.

The distribution of these cysts however confirmed what has been noticed before, most of them were in the lower temporal. There seems to be no influence of its incidence from myopia (Table IV Histogram 20). It has a greater proponderance in the older age group, and none of the subjects below the age of 30 had the cystoid degeneration.

The pathology of retinoschisis involves splitting of the retina into two layers usually at the outer plexiform layer, i.e. between the nuclei of the photoreceptors and the bipolar cells. There are two forms the senile and juvenile types, and the former results from degenerative changes in the elderly, while

TREATMENT OF RETINAL DEGENERATION

the juvenile type results from dystrophic changes (Duke-Elders) in the periphery of young persons. Central schisis involving the posterior pole have been noticed, but these are rare.

...the preceding chapters that lattice degeneration is the most dangerous form of degeneration with regard to retinal detachment. Indeed, it is said that lattice degeneration and scattered retinoschisis when it develops holes in its inner and outer layers, the treatment should therefore be aimed at preventing the separation of the retina in these areas.

The principle of treatment is therefore to create a firm adhesion of the retina to the choroid. The process involves an intraglobular chorioretinitis thereby starting an inflammatory process resulting into exudate formation from the choroidal capillaries. It is the exudate which initially creates the firm adhesion between the retina and the choroid. Eventually fibrous tissue develops eventually in the area, resulting into a scar tissue. The latter is the ultimate aim of the procedure, as it gives a very firm adhesion between the retina and the choroid, in such areas. This can be done either in a wide area covering the whole area of degeneration, or in several small areas surrounding the dangerous parts.

The chorioretinitis can be induced either from outside i.e. the scleral side, or from inside i.e. vitreal side. The

TREATMENT OF RETINAL DEGENERATIONS

The sequale of peripheral retinal degenerations which is most feared is the occurrence of retinal breaks, which subsequently result into retinal detachment. It has been stressed in the preceding chapters that lattice degeneration is the most dangerous form of degeneration with regard to retinal detachment. Second to, it is snail-track degeneration and sometimes retinoschisis when it develops holes in its inner and outer borders. The treatment should therefore be aimed at preventing the separation of the retina in these areas.

necrosis and might also lead to vitreous hemorrhage, from The principle of treatment is therefore to create a firm adhesion of the retina to the choroid. The process involves an iatrogenic chorioretinitis thereby starting an inflammatory process resulting into exudate formation from the choriocapillaries. It is the exudate which initially creates the firm adhesion between the retina and the choroid. Eventually fibroblastic tissue accumulate excessively in the area, resulting into a scar tissue. The latter is the ultimate aim of the whole procedure, as it gives a very firm adhesion between the retina and the choroid, in such areas. This can be done either in a wide area covering the whole area of degeneration, or in several small areas surrounding the dangerous parts.

The chorioretinitis can be incited either from outside i.e. the scleral side, or from inside i.e. vitreal side. The

employed to seal the hole and prevent retinal detachment.

former is accomplished by using one of the two methods, cryopexy or diathermy. Cryopexy involves applying excessive cold at approx. -70°C and this creates an inflammatory process in the choroid. It is the method commonly used from the scleral side. It is useful especially in cases with large areas of peripheral degeneration. The second method used from outside, is diathermy. The latter similarly creates an inflammatory process, but even though it is almost an obsolete procedure in most centres, as it causes scleral necrosis and might also lead to vitreous haemorrhage, from intense reaction which it creates.

The method used from inside is photocoagulation. Light is emitted in a strong beam, to create an inflammatory process. This can be done either from a Xenon arc photocoagulator which gives a wide beam, or from laser beam which gives a narrow intense beam, and can be used with a three mirror contact-lens, to treat even the most peripheral retinal degenerations. Light energy transformed into heat is absorbed by the pigment epithelium and blood vessels, and this causes an inflammatory process which results into exudation with subsequent scar formation.

Retinal holes can be prevented by these methods and where retinal breaks have already occurred, the same methods can be employed to seal them and prevent retinal detachment.

C O N C L U S I O N

This study confirmed some previous reports on peripheral retinal degenerations while some contrasting features have also been noted. The following conclusion can be drawn from the results of this study.

- (i). White without pressure (WWP) is a common finding in eyes with myopia among African subjects. There is no evidence that it progresses to real degenerations like lattice degeneration or snail-track degeneration. However it can occur together with other degenerations though not necessary associated with them. It is seen in some cases of retinal breaks, or retinal detachment although it has no direct association with the two lesions. It is therefore an innocuous pre-degenerative lesion which needs no treatment.
- (ii). Snow-flake degeneration is relatively rare (c.f. WWP) among African myopes. It has a higher incidence in the younger age group than in the elderly. Myopia seems to have very little influence on its incidence. Like WWP, it is also seen with other types of degenerations like 'Lattice and 'Snail-Track.' It does not progress to any of the real degenerations. Although it can be seen in cases of retinal breaks and retinal detachment, it has no direct association with these lesions. It is therefore similarly a

- (ii) benign lesion which requires no treatment.
- (iii). Lattice degeneration has the highest incidence of the dangerous retinal degenerations. Its incidence in the present series, is however generally low. It is commonly seen in the elderly and appears to get worse, with increasing myopia. It is strongly associated with retinal detachment and retinal breaks. It leads to formation of retinal holes which might progress to retinal detachments. It is therefore a dangerous degeneration, which should be treated in the majority of cases. This should be particularly done if the fellow eye has had retinal breaks, or retinal detachment. They should also be treated if there is a positive family history of retinal breaks or detachment. The incidence of retinal detachment has been shown to be quite low, compared to the incidence of lattice degeneration in the general population. This indicates that most of the lattice degenerations probably do not end up in retinal breaks or retinal detachment. Some people do not therefore treat these degenerations. The final decision however rests with the surgeon who makes on-the-spot judgement on the nature of the lesion and other relevant factors mentioned above. The treatment is either by cryopexy or by light coagulation.

(iv). Snail-Track degeneration has distinct features, which separate it from lattice degeneration. It has a tendency to vitreo-retinal changes and tractional breaks. It is therefore a potential danger which can result into breaks or retinal detachment. Although its association with retinal detachment is not as strong as that of 'Lattice' it is potentially dangerous and should be treated with either photocoagulation or cryopexy.



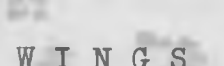



(v). Pigmentary degeneration^{is} a condition which is common in the elderly. It increases with age and seems to result from chorioretinal changes involving mainly the pigment epithelium. The latter undergoes a process of hyperplasia, resulting into clumping of pigments. There is no direct association with retinal breaks, however where the latter is the primary lesion, pigmentation occurs along its edges and possibly in some small retinal breaks, covering the holes with a pigment clump. longstanding retinal detachments also undergo hyperplasia of the pigment epithelium and hence pigment clumps are commonly seen in such cases. Pigmentary degenerative changes are not dangerous with regard to retinal breaks, and therefore need no treatment.

(vi). Cystoid degenerations leading to retinal detachment are rare. The microcysts commonly seen in adults do not require any treatment. In some cases however they might result into retinoschisis and when the latter develops holes in its outer and inner leaves, then a total retinal detachment occurs. Treatment of retinoschisis with no breaks is debatable however if it is progressing fast towards the posterior pole, then it should be treated with photocoagulation.

In general, it can be concluded that the pattern of pre-degenerations and degenerations in myopic Africans is more or less similar to that in caucasians. These degenerations as has been shown and discussed, are associated with retinal breaks and retinal detachment. The total number of retinal detachments seen annually at K.N.H. are on the increase and it is possible to reduce the incidence by performing careful fundus examination of both symptomatic and asymptomatic myopes. Patients with peripheral retinal degenerations which are commonly associated with retinal breaks or detachments, should have treatment as explained above, so that the incidence of retinal detachment can be reduced.




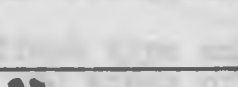
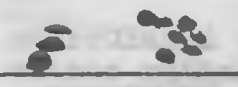





It would be interesting and useful for a similar survey to be conducted among emmetropes to determine the incidence of peripheral degenerations. This would give an insight as to what the majority of the population has in terms of degenerations, and it would also give figures with which to compare and contrast those of the present study.

TABLE SHOWING OCCURRENCE AND NUMERICAL CODE

Degenerative	Colour code	Numerical code
White without pressure		I
Snow-Flake		II
Lattice degeneration		III
Shell-track degeneration		IV
Flame-like degeneration		V
Cystoid degeneration		VI
Retinal holes		VII
Thin retina		VIII
Retina detachment		IX
Vitreo-retinal traction		X

FUNDUS DRAWINGS

FUNDUS DRAWINGS-COLOUR AND NUMERICAL CODE

Degenerations	Colour code	Numerical code
White without pressure		I
Snow-flake		II
Lattice degeneration		III
Snail-track degeneration		IV
Pigmentary degeneration		V
Cystoid degeneration		VI
Retinal Holes		VII
Thin retina		VIII
Retina detachment		IX
Vitreo-retinal traction		X

OD

OS

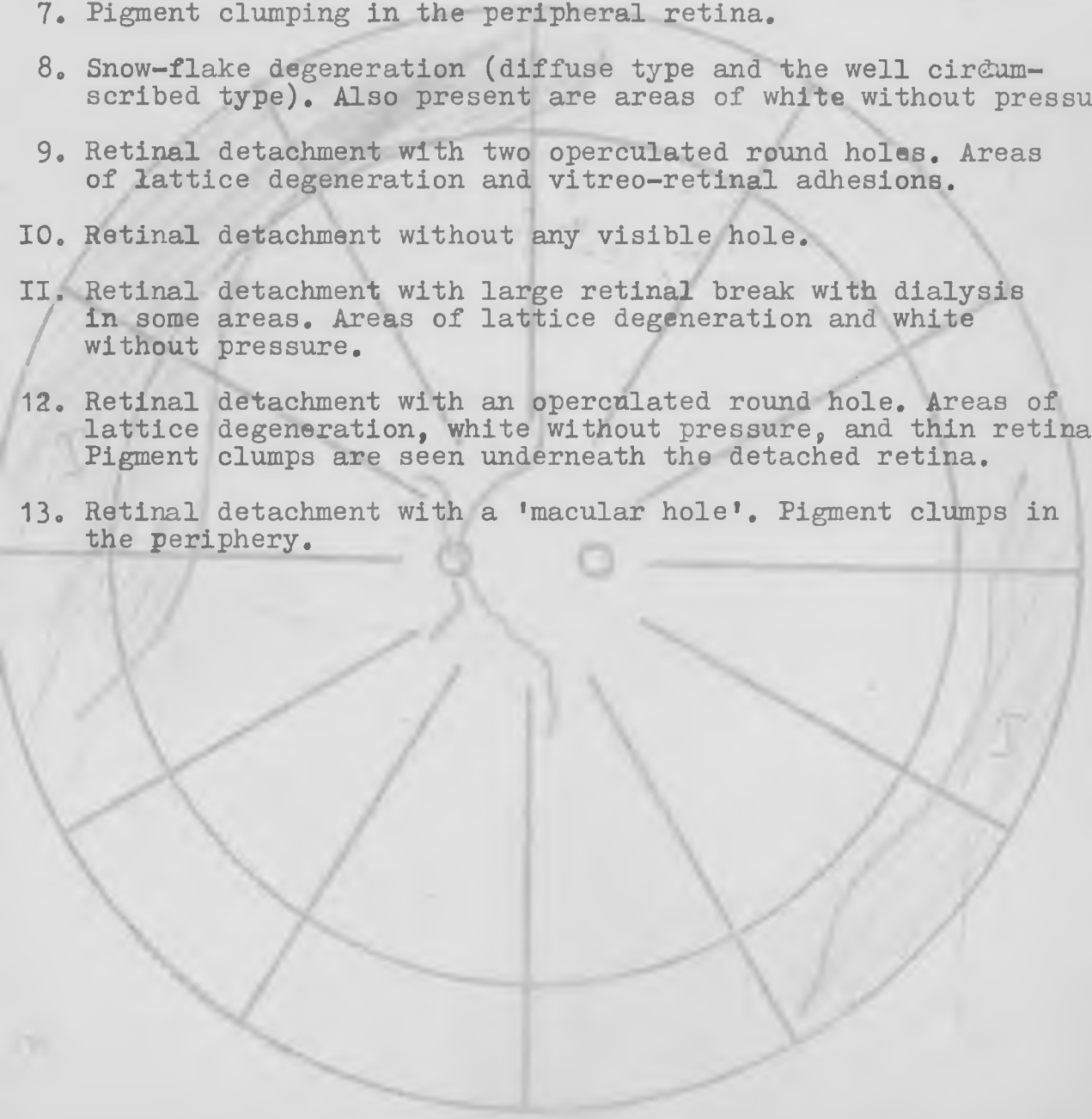
Zeichnung Nr.

KEY

60

1. White without pressure	Oct. 18/11/79	Dr. ADALA
2. White without pressure and two retinal unoperculated round holes with a small area of shallow retinal detachment.		
3. White without pressure and lattice degeneration having pigment deposit in-between the 'white lines'.		
4. Diffuse pigmentation extending to the posterior pole with light vitreous traction temporal side. Fundus of a patient with retinopathy of pre-maturity.		
5. White without pressure and cystoid degeneration.		

- 6. White without pressure and lattice degeneration, also present are cobblestone changes in the upper nasal quadrant.
- 7. Pigment clumping in the peripheral retina.
- 8. Snow-flake degeneration (diffuse type and the well circumscribed type). Also present are areas of white without pressure.
- 9. Retinal detachment with two operculated round holes. Areas of lattice degeneration and vitreo-retinal adhesions.
- IO. Retinal detachment without any visible hole.
- II. Retinal detachment with large retinal break with dialysis in some areas. Areas of lattice degeneration and white without pressure.
- 12. Retinal detachment with an operculated round hole. Areas of lattice degeneration, white without pressure, and thin retina. Pigment clumps are seen underneath the detached retina.
- 13. Retinal detachment with a 'macular hole'. Pigment clumps in the periphery.

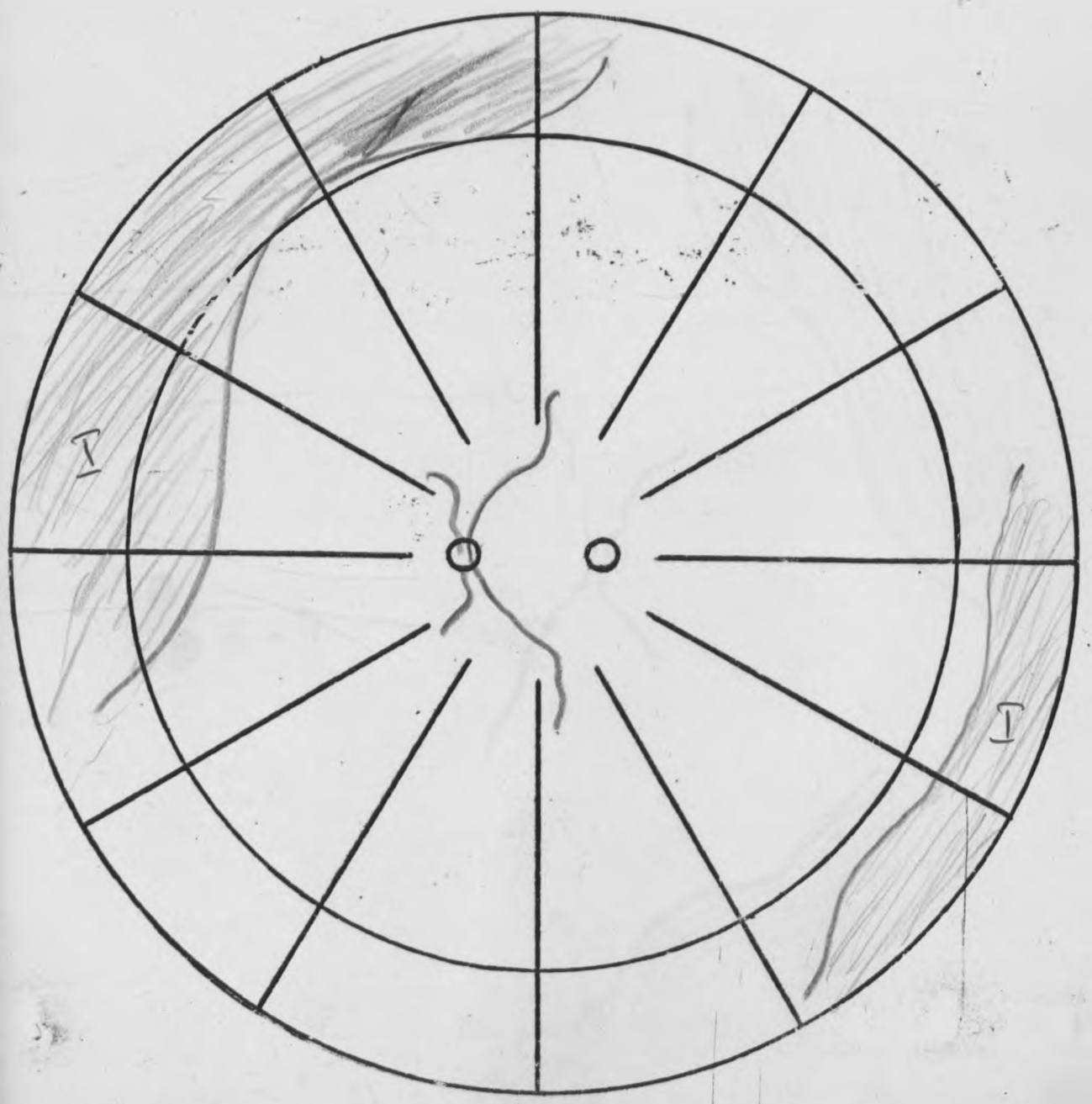


OD

OS

Zeichnung Nr.

Pat.	C.M. (23)	Dat.	18/11/79	Dr.	ADALA
V. OD	6/12 ε - 6.00DS 0.5x110	V. OS	6/9 ε - 5.00DS -1.50x10		
Lens	Clear	Corpus	floaters	Bes.	
Op.					
Dat.		Dr.		Prognose	

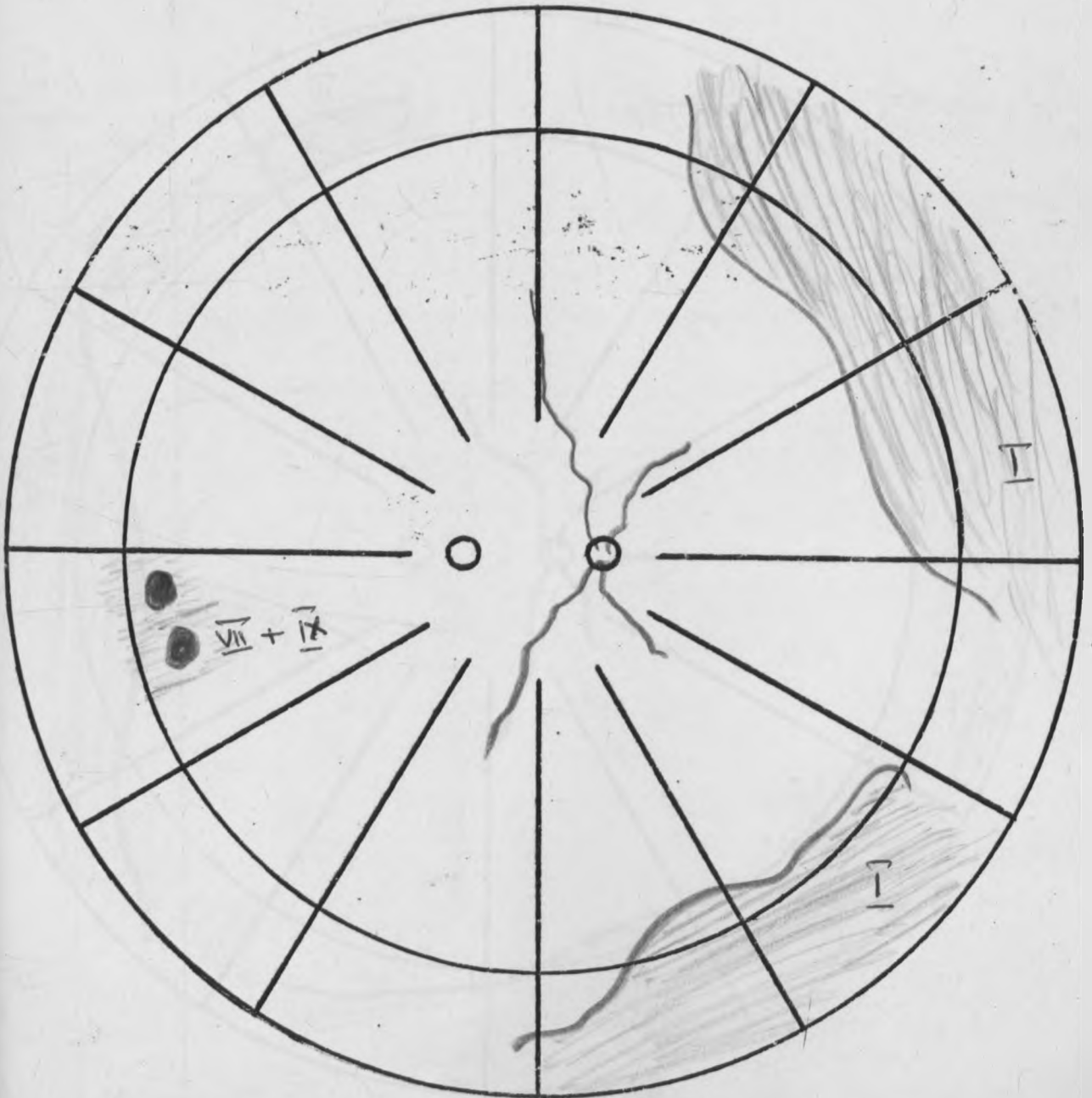


OD

OS

Zeichnung Nr.

Pat.	R.W. (22)	Dat.	29/8/79	Dr.	ADALA
V. OD	6/18 → 6/6 ε -4.50 DS	V. OS	6/18 ε -2.50 → 6/6 ε 5.25 DS		
Lens	Corpus	Bes.			
Op.	CRYOPEXY				
Dat.	Dr.	Prognose			

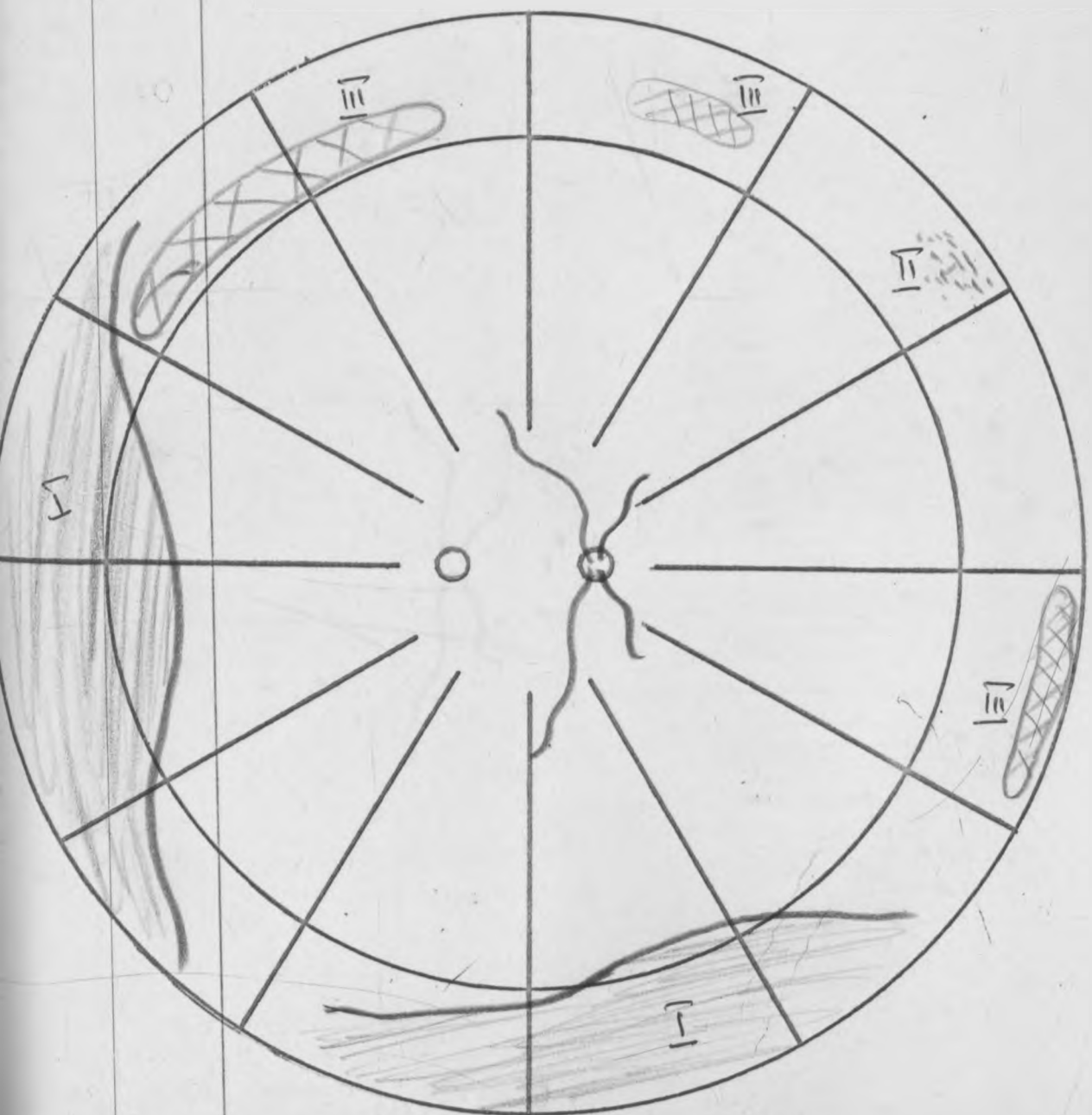


OD

OS

Zeichnung Nr.

Pat.	R.G. (31)	Dat.	28/12/79	Dr.	ADALA
V. OD	6/18 ε - 6.00 DS	V. OS	6/24 ε - 7.50 DS		
Lens	Clear	Corpus	Clear	Bes.	
Op.					
Dat.		Dr.		Prognose	

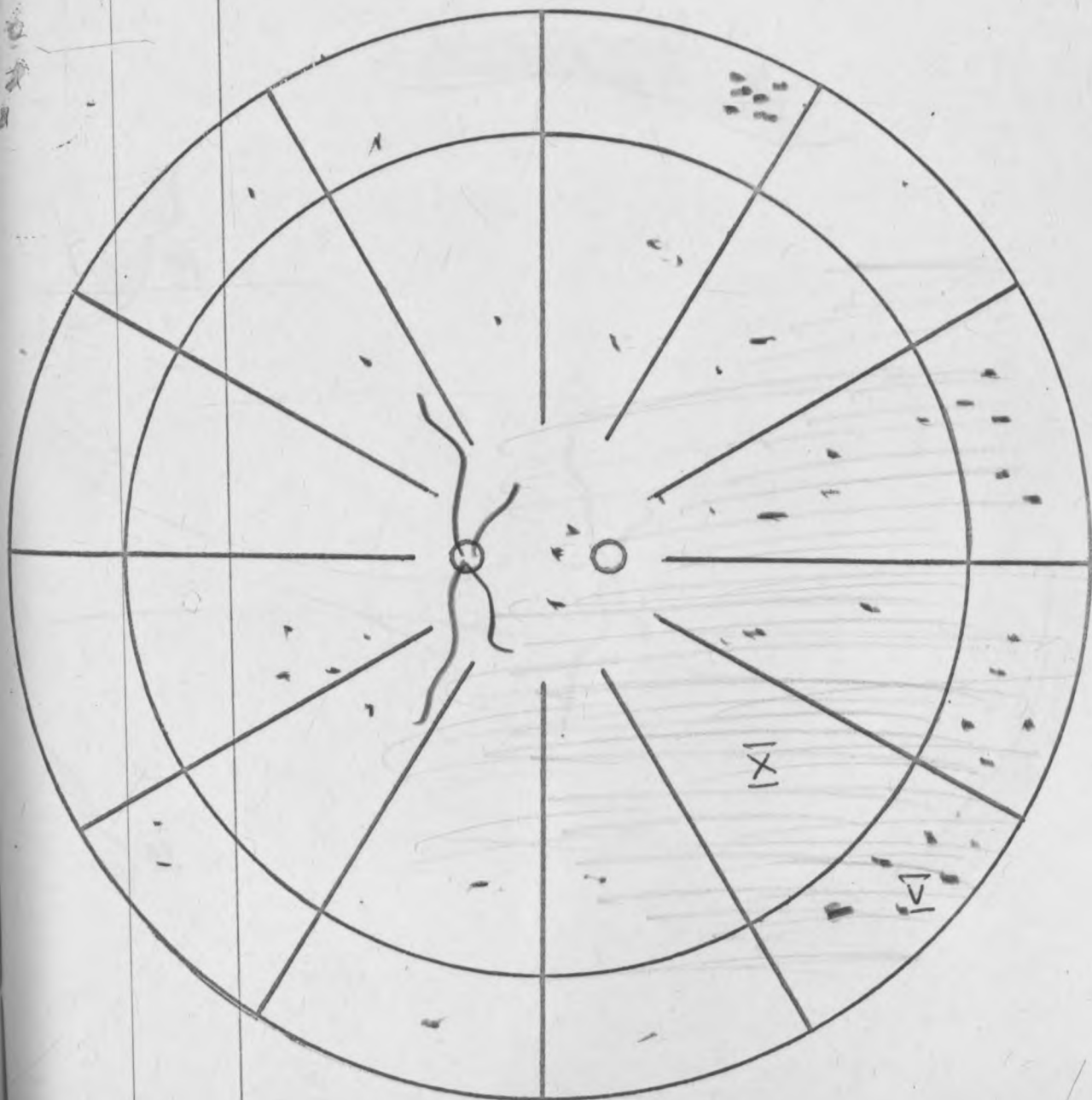


OD

OS

Zeichnung Nr.

Pat.	R.VI. (11)	Dat.	14/2/80	Dr.	ADALA
V. OD	6/18 \bar{c} -5.50 DS	V. OS	6/18 \bar{c} -5.00 DS		
Lens		Corpus		Bes.	
Op.					
Dat.		Dr.		Prognose	



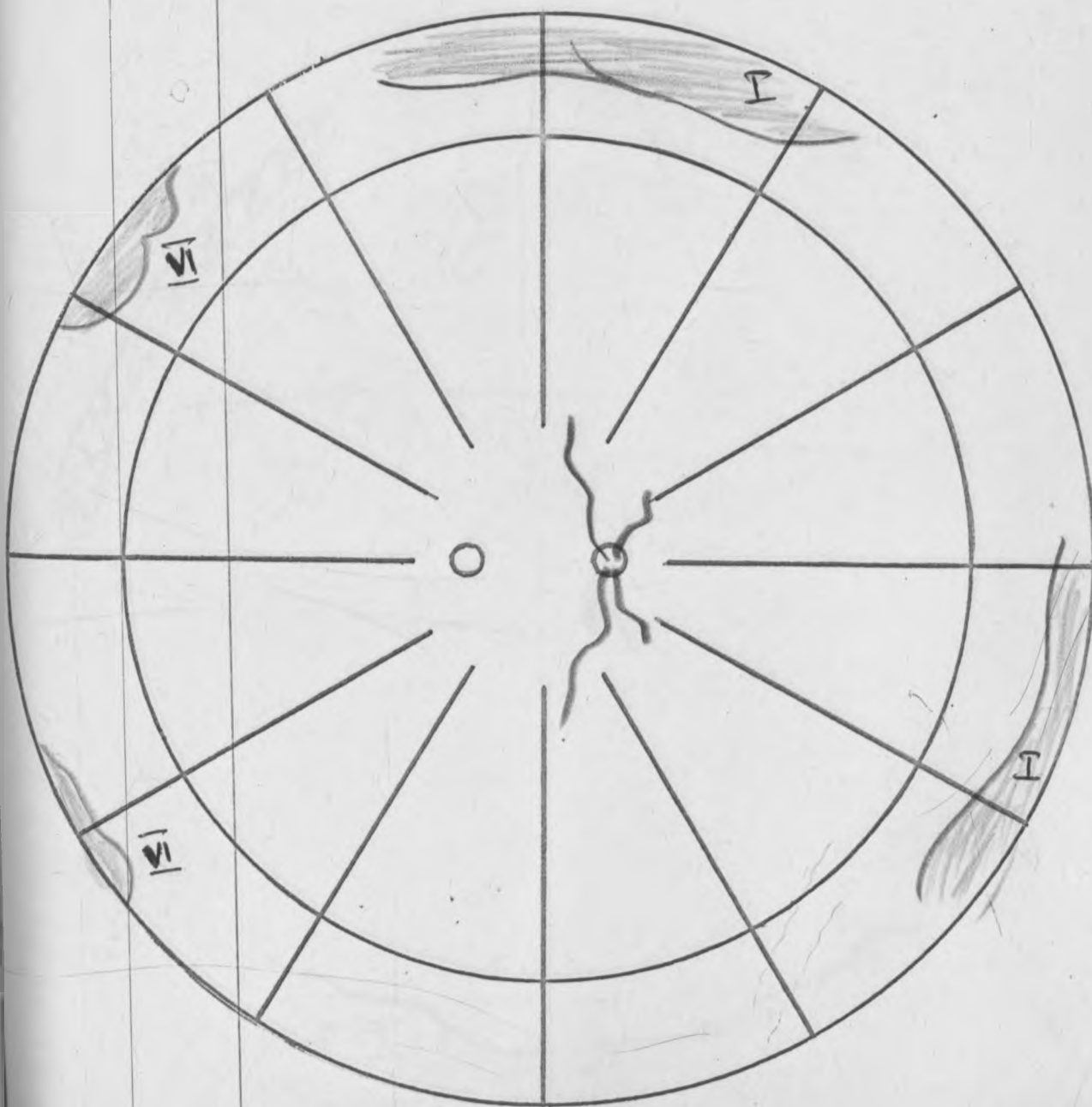
V

OD

OS

Zeichnung Nr.

Pat.	J.K. (51)		Dat.	19/3/80	Dr.	ADALA		
V. OD	6/9 \bar{c} -2.75 DS			V. OS	6/9 \bar{c} -3.25 DS			
Lens	Clear	Corpus	Clear	Bes.				
Op.								
Dat.	Dr.			Prognose				

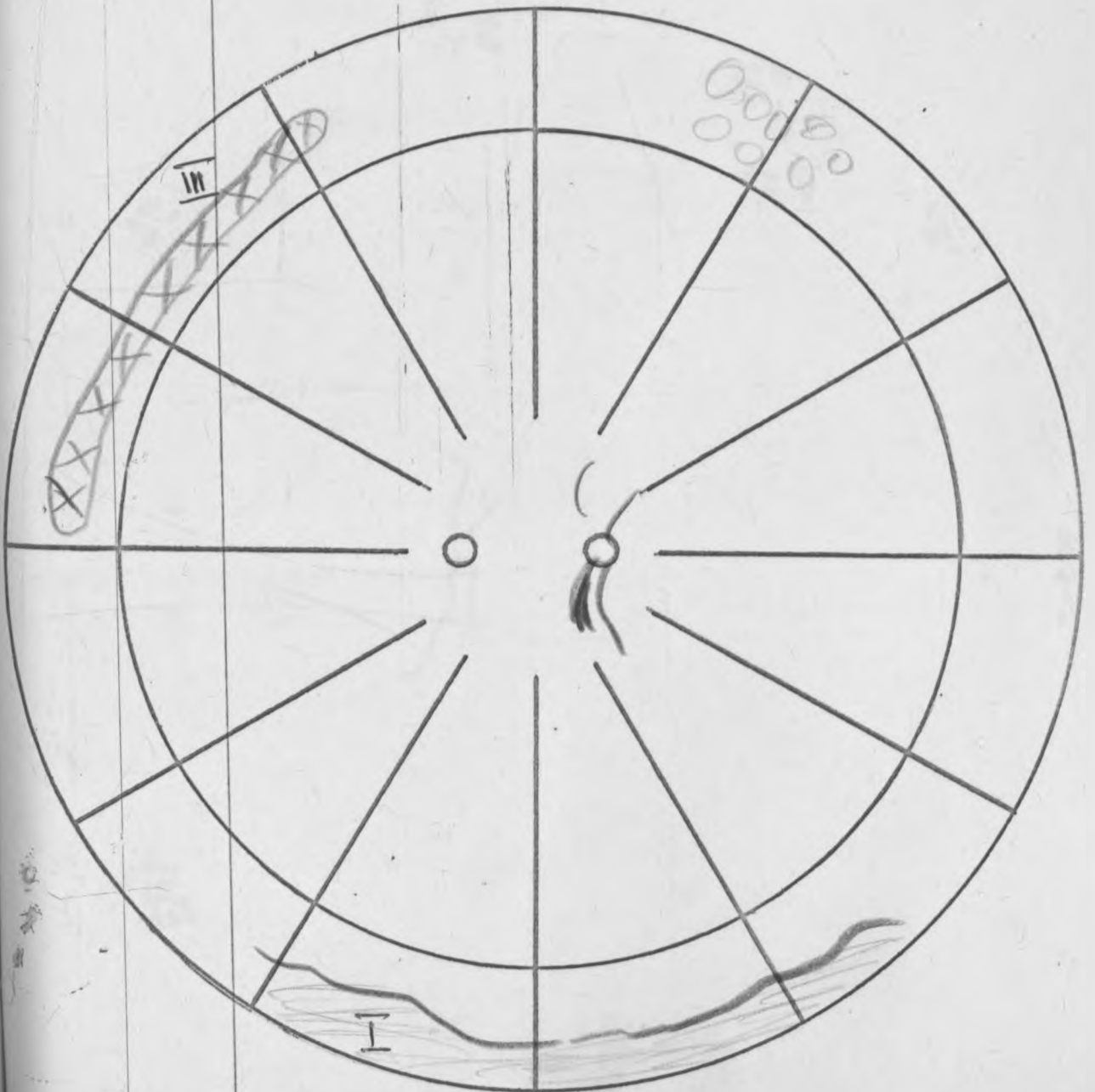


OD

OS

Zeichnung Nr.

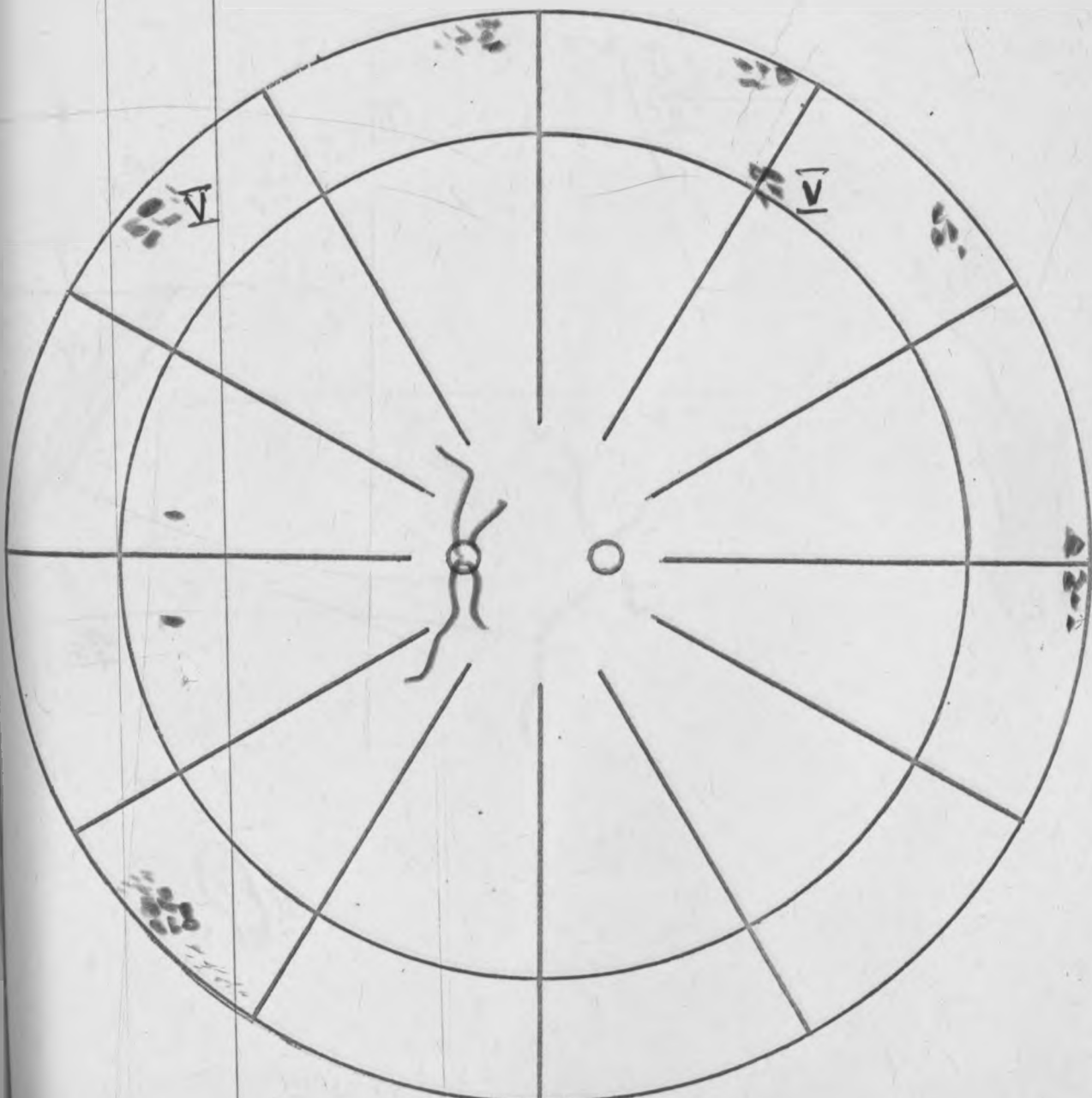
Pat.	V.O.	Dat.	10/12/79	Dr.	ADALA
V. OD	6/6 z -1.75 DS	V. OS	6/6 z -1.50 DS		
Lens		Corpus		Bes.	
Op.					
Dat.		Dr.		Prognose	



OD ^{VII} OS

Zeichnung Nr.

Pat.	J.M. (48)		Dat.	11/10/79	Dr.	ADALA		
V. OD	6/9 \bar{c} -3.50/-0.50x45			V. OS	6/9 \bar{c} -4.75/-0.50x110			
Lens	Corpus			Bes.				
Op.								
Dat.	Dr.			Prognose				

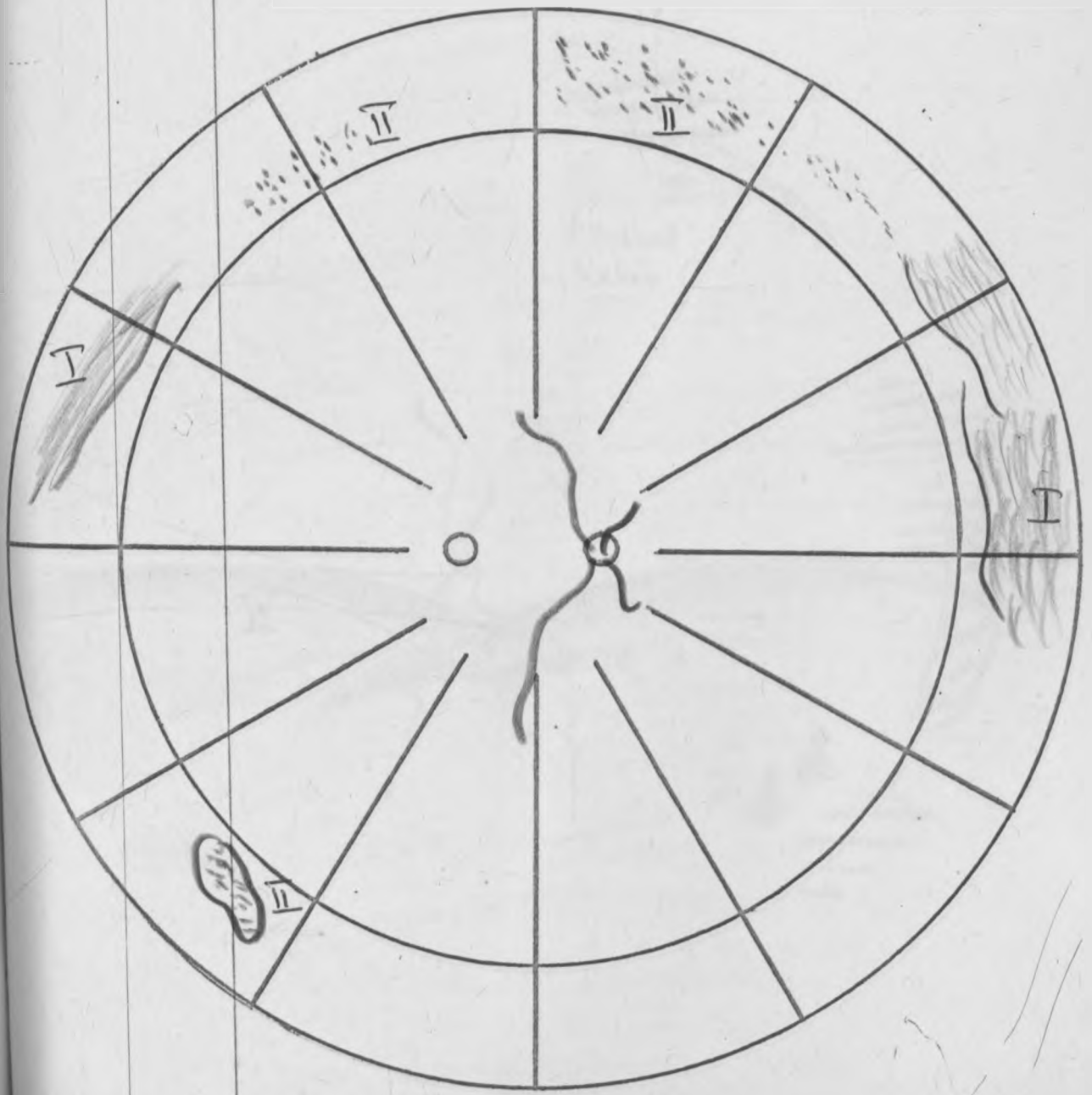


OD

OS

Zeichnung Nr.

Pat.	E.M. (27)	Dat.	12/7/79	Dr.	ADALA
V. OD	6/36 → 6/6 c - 2.00 DS	V. OS	6/60 → 6/6 c - 2.75 DS		
Lens		Corpus		Bes.	
Op.					
Dat.		Dr.		Prognose	



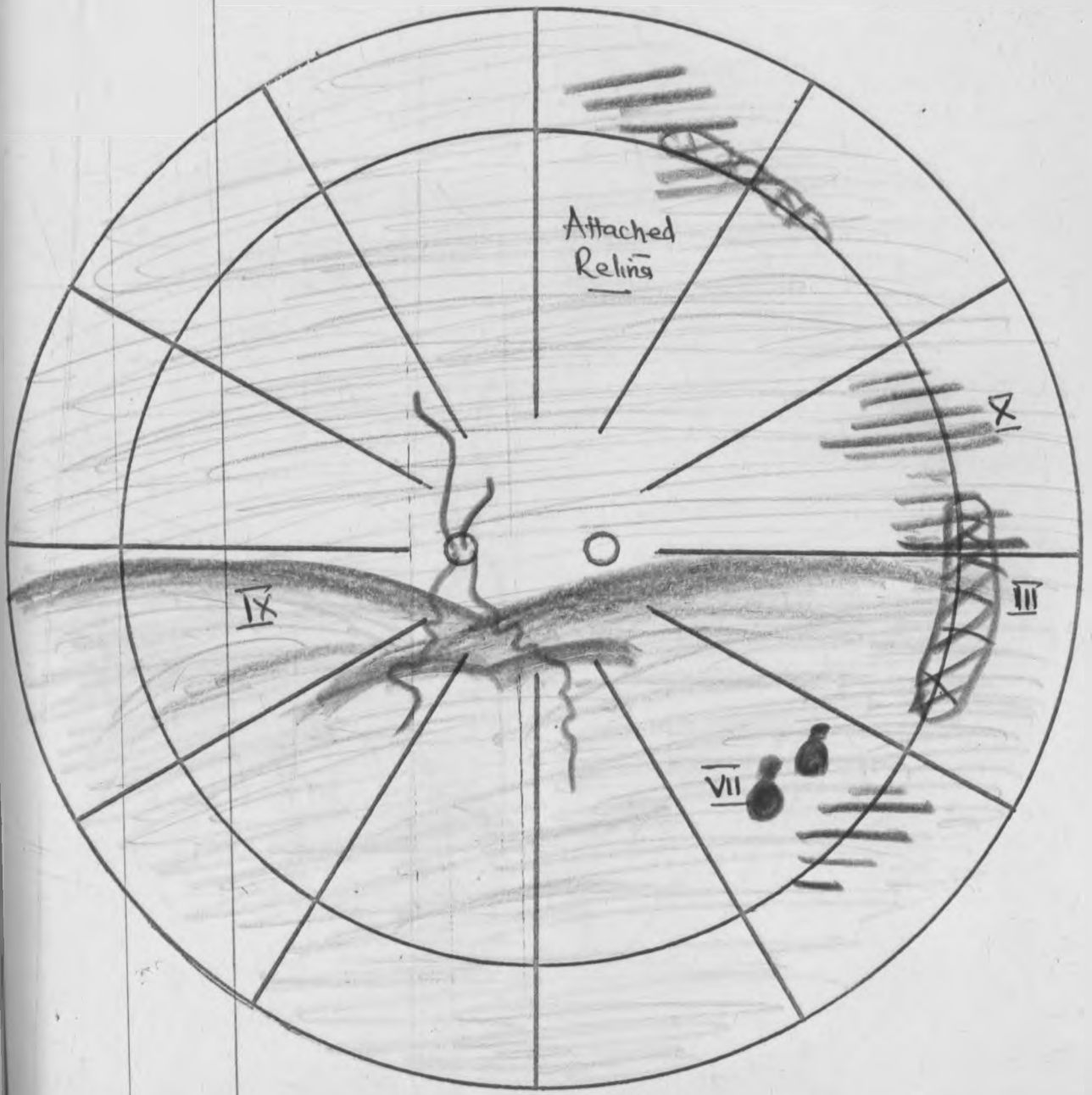
IX

OD

OS

Zeichnung Nr.

Pat.	R.N. (30)	Dat.	11/12/79	Dr.	ADALA
V. OD	6/18 \bar{c} -2.50 DS	V. OS	6/60 \bar{c} -1.25 DS		
Lens		Corpus		Bes.	
Op.					
Dat.		Dr.		Prognose	



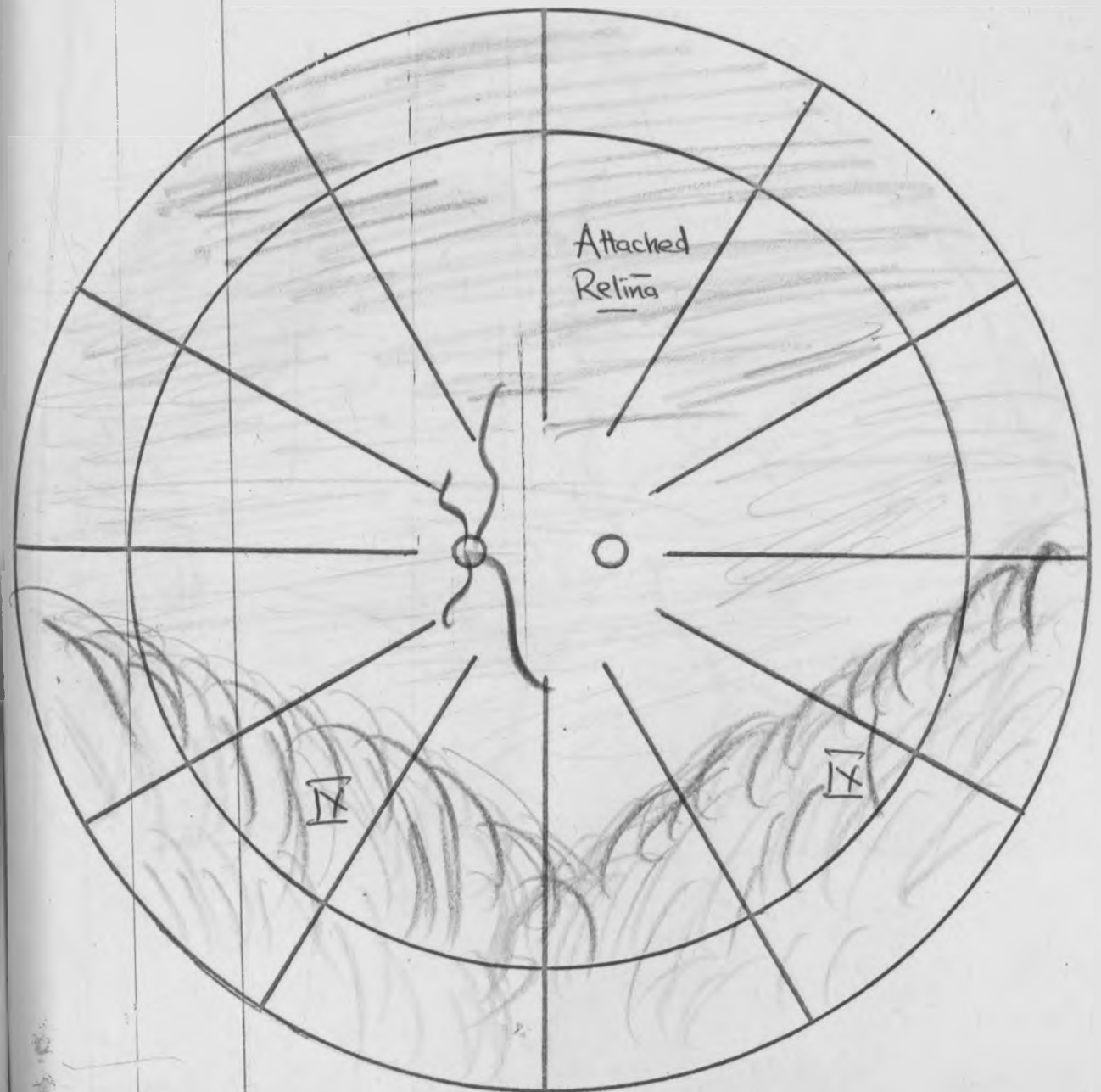


OD

OS

Zeichnung Nr.

Pat.	R.N.W. (37)	Dat.	7/3/79	Dr.	ADALA
V. OD	6/6 \bar{a} -1.25 DS	V. OS	6/36 \bar{a} -1.25 DS.		
Lens		Corpus		Bes.	
Op.					
Dat.		Dr.		Prognose	



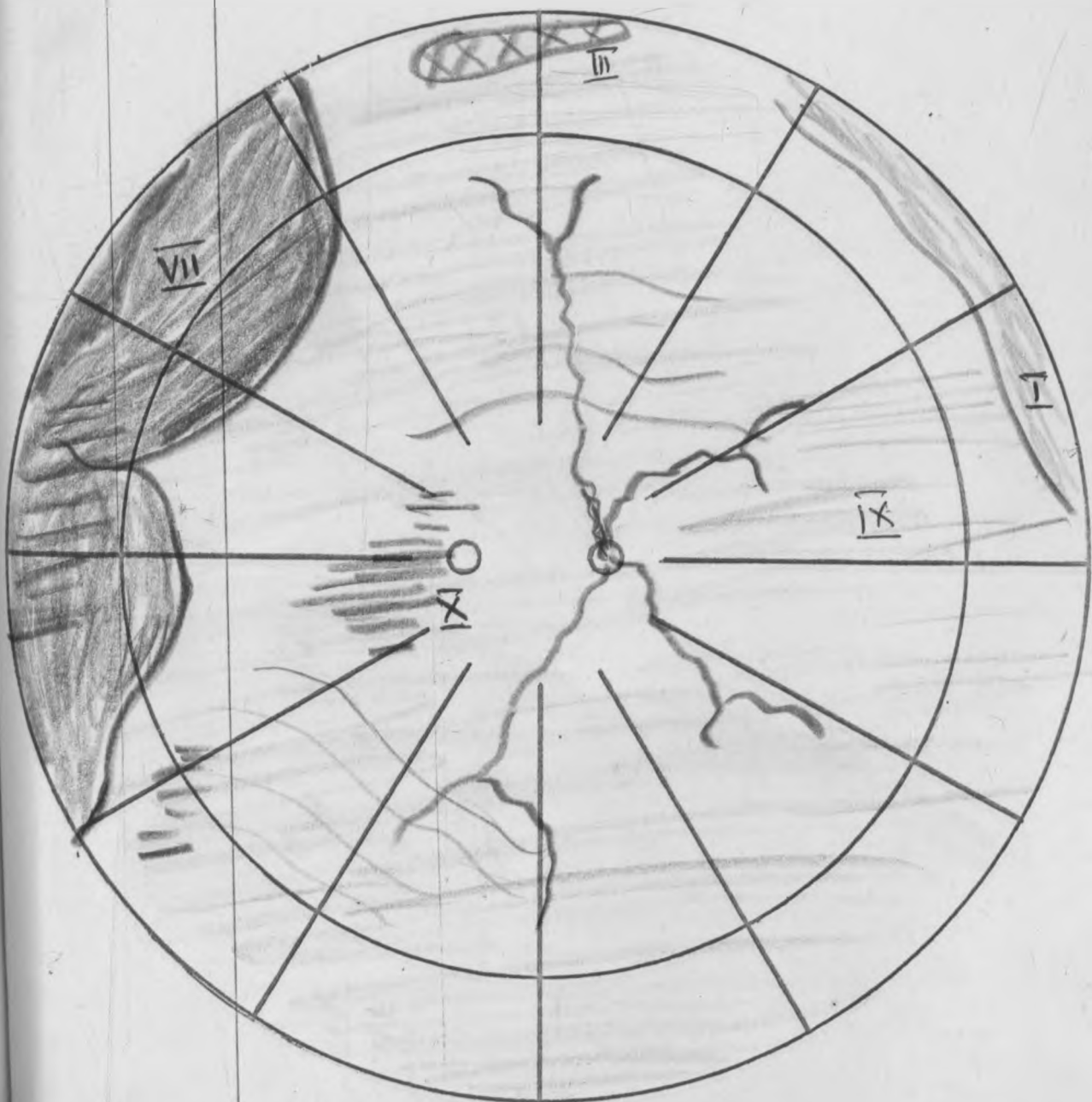
XI

OD

OS

Zeichnung Nr.

Pat.	J. M. W. (15)	Dat.	2/11/79	Dr.	ADALA
V. OD	3/60 ± 2.00 DS	V. OS	6/6 ± -1.25 DS		
Lens	Clear	Corpus	Tobacco dust.	Bes.	
Op.					
Dat.		Dr.		Prognose	

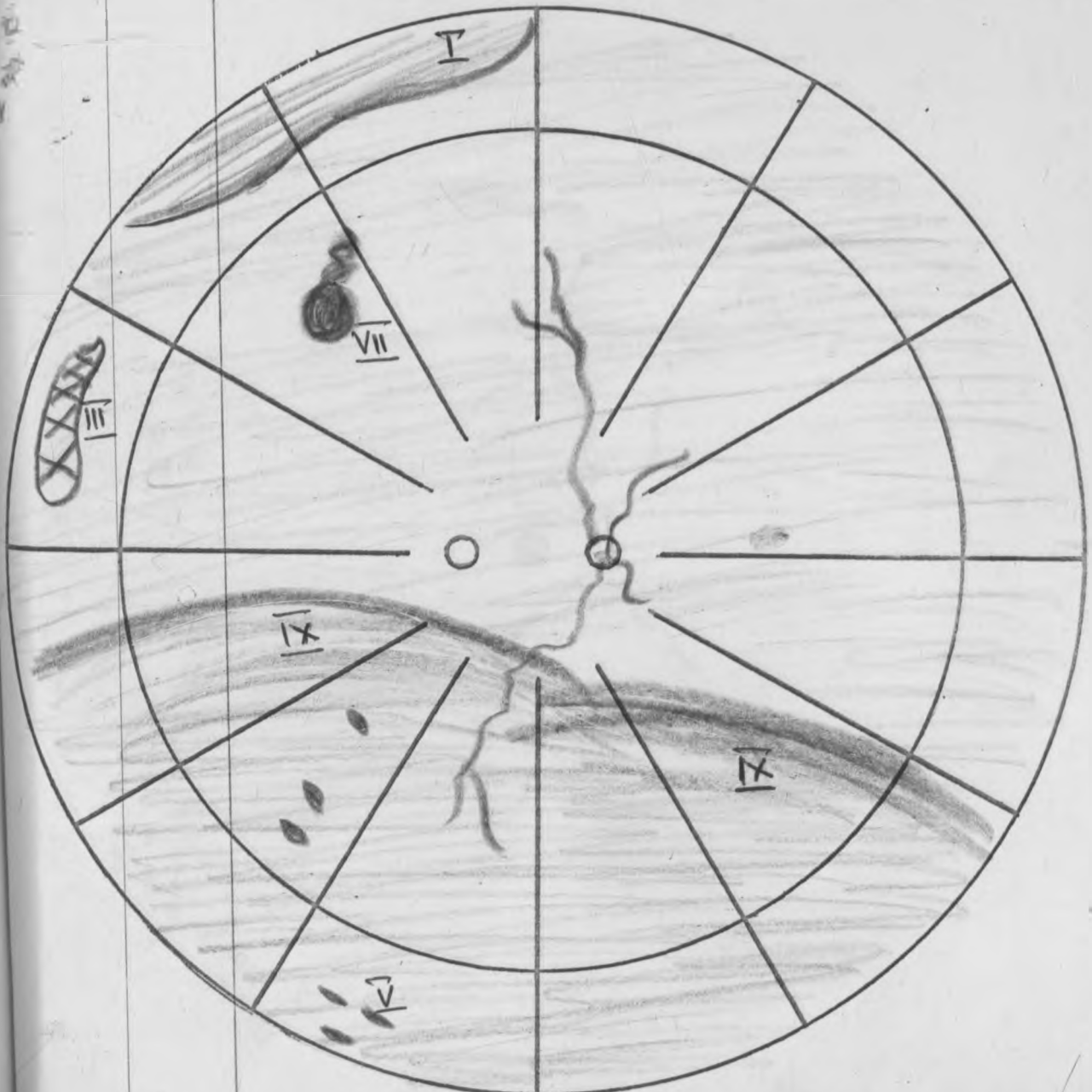


OD

OS

Zeichnung Nr.

Pat.	K.N.	Dat.	14/9/79	Dr.	ADALA
V. OD	6/60 c -2.25 DS	V. OS	6/36 c -2.00 DS		
Lens		Corpus		Bes.	
Op.					
Dat.		Dr.		Prognose	

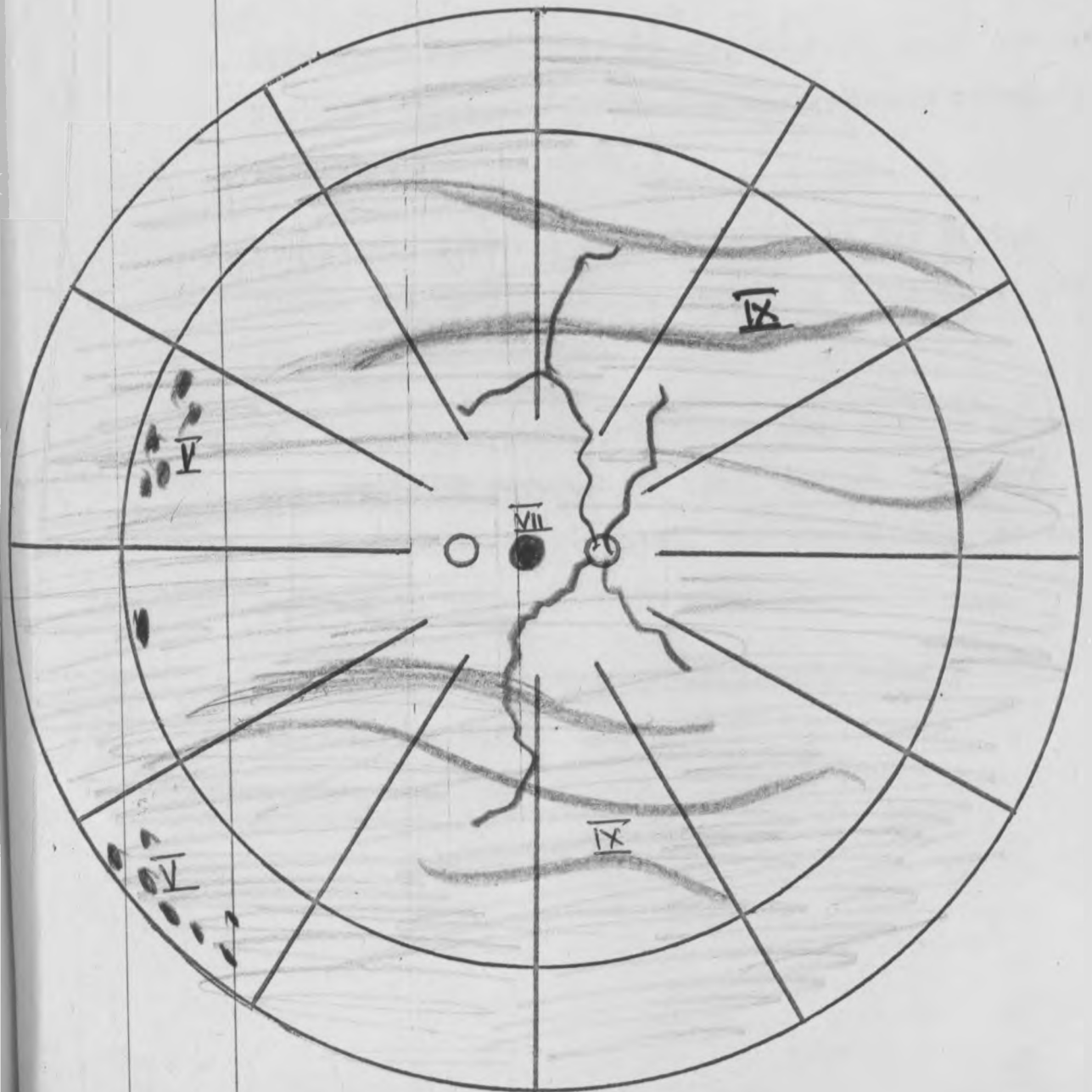


OD

OS

Zeichnung Nr.

Pat.	G.G.	Dat.	7/12/79	Dr.	ADALA
V. OD	1/60 ε -1.75 DS	V. OS	6/18 ε -2.50 DS.		
Lens		Corpus		Bes.	
Op.					
Dat.		Dr.		Prognose	



A C K N O W L E D G E M E N T S

I am most grateful to the following for having contributed to the completion of this thesis.

1. Dr. V. Klauss and Dr. Dechant for their supervision and encouragement during the entire period of the study, and for their patience in reading through the draft and giving useful criticisms.
2. Ophthalmic specialists at the Eye Department, Kenyatta National Hospital, for having availed some of their patients, for the study.
3. Ophthalmic Clinical Assistants in the Eye Filter Clinic who co-operated by referring a number of patients for the study.
4. All volunteers particularly 3rd year Medical Students, University of Nairobi and those members of staff Kenyatta National Hospital, who kindly accepted the request to be part of the study group.
5. Miss Beatrice Wanjiku and Rhoda Akech for excellent secretarial work.

R E F E R E N C E S

- I. Aaber T.M. and Stephens, T.R., Snail-Track degeneration of the retina 73,270-276 (1972)
2. Byer, N.E., Trans. Am. Ac. Ophthalm. Otolaryng, 69, 1064.
3. Dumas, J. and Schepens, C.L. Am. J. Ophthalm. 61, 620(1966)
4. Duke-Elder, Systems of Ophthalmology Vol. 10, page 55, Henry Kimpton.
5. Eisner G., Chorioretinal lesions predisposing to retinal breaks, Am. J. Ophthalm. 61,620-630 (1966).
6. Gonin J. (1935) le decollement de la retne, Payot Lausanne.
7. Okun, E. Pigmentation and retinal breaks Am. J. Ophthalm. 79, 190-193 (1975) Am. J. Ophthalm. 51,369.
8. Rutnin, U. Schepens, The standard peripheral fundus and developmental variation Am. J. Ophthalm. 64, 840-852(1967).
9. Hyams S.W., Neuman, Lattice Degeneration Am. J. Ophthalm. 67, 973 (1969).
10. Iwanoff, A. Peripheral retina in myopia, B.J.O. 53, 300-306
11. Kanski, J.J. Peripheral retinal degeneration Trans. Ophthalm. Soc. U.K. Vol. XCV. Part I, 1975.
12. Michaelson and Ballantyne Text of the Fundus of the eye 2nd. Ed. Churchill Livingstone Page 385.
13. Staatsma, B.R., Zeegen, P.D., Foos, R. Fomens S., Shabo, S.L. Typical and reticular degenerative retinoschisis Am. J. Ophthalm 75, 551-575 (1973).
14. Teng. C.C. and Katzin, H.M., the retinal surface in Lattice degeneration of the retina Am. J. Ophthalm. 1201-1209(1972).
15. Wazke R.C. The Ophthalmoscopic sign, white without pressure, clinicopathologic correlection, Arch. Opth., 66,812-823(1961).
14. Degeneration of the peripheral retina by Aaberg T.M. and Stephens T.R. (1972) Am. J. Ophthalm. 73, 370.
17. Amster M. and Kloti R. (1961) Ophthalmologica (Basel) 141,329.
18. Boniuk, M. and Buttler F. (1968) "New and Controversial Aspects of retinal detachment 59 Harper and Row, New York.
19. Rutnin, V. and Schepens C.L. (1967) Ibid 64, 1063