Section of Ophthalmology

President H B Stallard MBE TD MCh

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Lang Lecture

A Characteristic Fluorescein Angiographic Pattern in Choroidal Folds

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To be invited to give a named lecture is always an honour, to be invited to give a named lecture in another country is an even greater honour, but to be invited to follow Sir Stewart Duke-Elder in this lecture is the greatest honour of which I can conceive. I am deeply grateful to your committee for selecting me and I trust that the material I present will be of as much interest to you as it has been to me in collecting it.

In any named lecture one desires to give proper recognition to the man who is being honoured. Unfortunately I never knew Mr William Lang nor have I ever worked closely with anyone who did. However, I have read the inaugural talks by Mr Whiting and Sir Stewart and the recent book by J Bernard Hutton entitled 'Healing Hands' and I cannot help but believe that Mr Lang was a master of the art of medicine.

Today the young ophthalmologist is often so highly trained in the science of our specialty that he forgets he is treating a total human being and not just a pair of eyes. We try our best to instil in our residents the importance of considering the entire needs of a patient, including his family, but I believe the true test of a physician comes when one observes how he handles his complications. Almost anyone can handle a patient with a successful result but how a physician handles his bad results – and anyone who does anything must have these – is probably the best indication of his skill in the art of medicine.

No matter how far we advance scientifically, the art of medicine as exemplified by Mr Lang and many great physicians must always be an integral part of our training and care.

In this Lecture I shall deal with a condition I have chosen to call choroidal folds.

Folds in the posterior fundus associated with space-occupying lesions in the orbit have been described by many authors (Birch-Hirschfeld & Siegfried 1915, Blegvad 1944, Kubik 1928, Kugelberg 1932, Löhlein 1927, Vedel-Jensen 1959, Wolter & Jampel 1957) and recently reviewed by Hedges & Leopold (1959) and Wolter (1962). In most of these reports the emphasis has been placed on retinal folds and in fact Reese (1963) states: 'These striæ are produced by wrinkling of the internal limiting membrane of the retina, which can be viewed as tension lines radiating from the point of contact with the sclera.' Wolter (1962) clearly demonstrates that in addition to the wrinkling of the inner layers of the retina, there is folding of the pigment epithelium and Bruch's membrane. It is the purpose of this paper to present the characteristic fluorescein angiographic pattern seen in these choroidal folds and to demonstrate their occurrence in patients with an orbital mass, scleral buckle, choroidal tumour, high hypermetropia, and without any clinical evidence of a space-occupying lesion.

Case Material

Case 1 A D, man aged 67

First seen April 14, 1967, with a chief complaint of slowly progressive exophthalmos of the left eye of one year's duration. Twenty-six years previously, the patient had battery acid splashed into his left eye and had noted decreased vision since

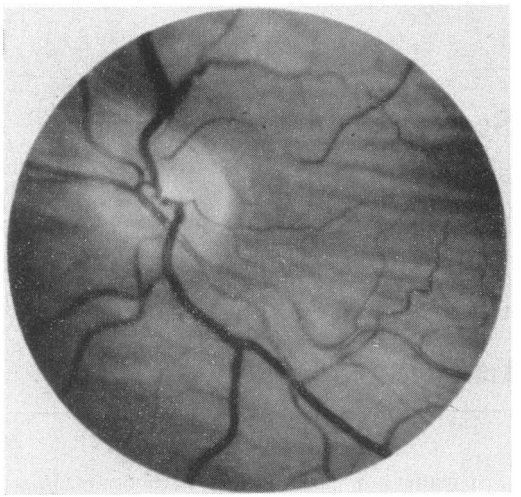

Fig 1A **Case 1**

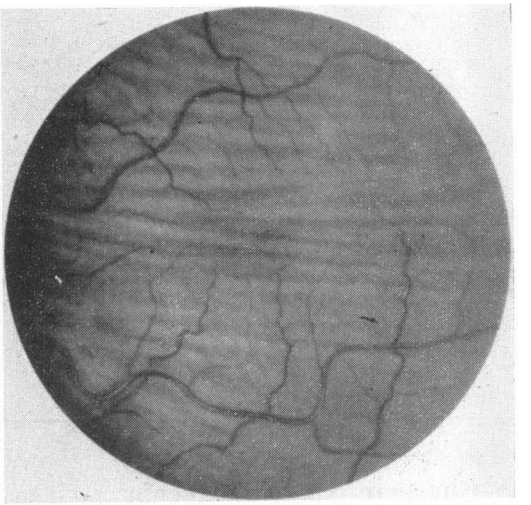

Fig 1B **Case 1**

then. During the preceding year he had noted diplopia, more at distance than near, associated with a prominence of the left eye. He had had pain in the left side of his head for the past two weeks. There was no history of sinus disease, thyroid disease, or other medical or surgical problems. A maternal grandmother had a goitre.

Examination showed a visual acuity in the right eye of 20/20-2 and in the left eye of 20/20-2. The palpebral fissure on the left was wider than that on the right. Hertel exophthalmometer: right eye 19 mm, left eye 22 mm. The globe was displaced forward and slightly down and to the left. There was increased orbital resistance and definite tenderness superonasally, but no mass could be felt. There was no bruit. Applanation tension was right eye 16, left eye 19 mmHg. Examination of the right eye was normal. The left fundus showed definite striæ in the posterior pole, extending horizontally from the disc through the macula (Fig 1A, B). With the Hruby lens these were seen to be folds deep to the retina at the level of the pigment epithelium and Bruch's membrane. Fluorescein angiography showed a characteristic pattern to be discussed.

The patient was admitted to Jackson Memorial Hospital where X-rays of the skull, orbits, sinuses, optic foramina, and chest were normal. PBI 5·0 μg/100 ml; ^{131}I uptake 13% in 24 hours. All blood and urine studies normal. Sigmoidoscopy, barium enema and gastrointestinal series normal.

On the third day in hospital a therapeutic trial of prednisone 60 mg per day was started and within forty-eight hours the patient reported marked decrease in the orbital pain and tenderness. He was discharged on this medication. Ten days after discharge he collapsed in church, and was found to have hæmatemesis and melæna, and the diagnosis of gastrointestinal bleeding secondary to prednisone was made.

The patient was last seen on March 9, 1968 and had no new complaints; in fact he felt quite well and desired no therapy for his proptosis. Vision in the right eye was 20/20 with a +1·50 sphere, left eye was 20/30 with a +4·00 sphere. Amsler Grid RE was normal, LE showed distortion of both vertical and horizontal lines. Colour vision (HRR test) was impaired in the left eye as compared with the right. Exophthalmometer readings: RE 14, LE 23. There was no bruit; the trigeminal nerve was intact, and there was slight limitation of movement of the left globe in all directions. There was increased orbital resistance and a suggestive mass superotemporally. Hruby lens examination revealed the inner layer of the retina to be quite smooth, and the prominent folds to be at the level of choroid, Bruch's membrane, and pigment epithelium. While there is little doubt that the patient has a mass in his left orbit, he will not consent to further investigation at this time.

Summary: This case shows classical folds which are located deep to the retina and associated with a proptosis of 9 mm, increased orbital resistance and a hypermetropia of 4·00 D. Stereophotographs confirm the location of the folds deep to the retina and fluorescein angiography shows a characteristic pattern. This consists of horizontal alternating light and dark lines corresponding to the folds (Fig 2A, B, C). The light portion corresponding to the crest transmits the choroidal fluorescence brilliantly, while the dark portion

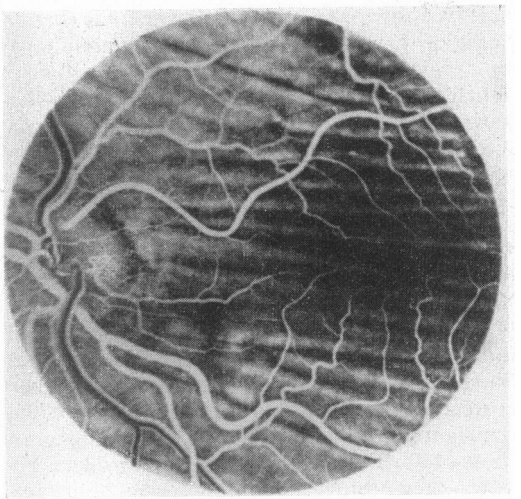

Fig 2A **Case 1** *Arterial phase*

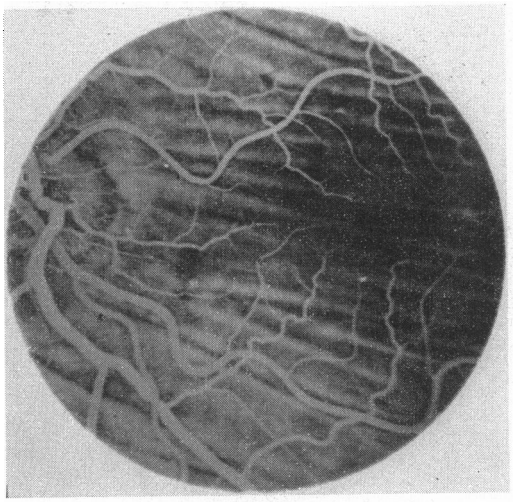

Fig 2B **Case 1** *Arterial-venous phase*

corresponding to the valley transmits the choroidal fluorescence poorly. There is no staining in the late pictures.

Case 2 A A, woman aged 53

First seen on March 15, 1964, with a history of blurred vision in the left eye of nine years' duration. In 1955 she was noted to have œdema of the left optic nerve. She was examined in St Luke's Hospital in New York where no evidence of a brain tumour was established. Five years before examination, central vision dropped to 20/50 and she was treated with steroids with an improvement to 20/30. Three months prior to admission the patient began having mild transient obscurations of vision lasting for a few seconds and a dull aching in the left eye. Her medical and family history are noncontributory.

Examination: Visual acuity in the right eye was 20/15 and in the left eye 20/30. Refraction: right eye, $-2{\cdot}00=+2{\cdot}00\times110$; left eye, $+2{\cdot}50=+1{\cdot}00\times75$. Visual field in the right eye was normal. The left visual field had a definite nasal cut with arcuate type defects radiating out from the disc superiorly and inferiorly.

External examination revealed definite proptosis of the left globe. Hertel exophthalmometer: right eye 12·5 mm, left eye 15 mm.

Applanation tension on the right was 16 and on the left 17 mmHg.

The pupils were equal but with a definitely impaired direct response on the left as compared with the right.

Extraocular movements: Minimal limitation of eye movements in all directions in the left eye.

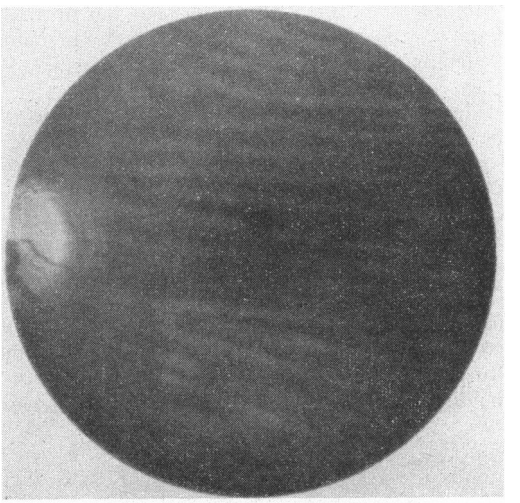

Fig 2C **Case 1** *Late picture (one hour)*

There was no bruit. The cranial nerves were otherwise intact.

Ophthalmoscopy of the right eye was normal. In the left eye the disc was elevated with distension of the veins and peripapillary œdema. At the posterior pole nasal and temporal to the disc there were many striæ which on slit-lamp examination appeared to be beneath the retina at the level of the pigment epithelium and Bruch's membrane. The whole posterior pole appeared to be pushed in and to have lost its normal concavity. The patient had an extensive laboratory work-up in hospital. X-rays of the skull, orbit, and optic foramen were normal. Pneumo-orbital tomograms were not very satisfactory but

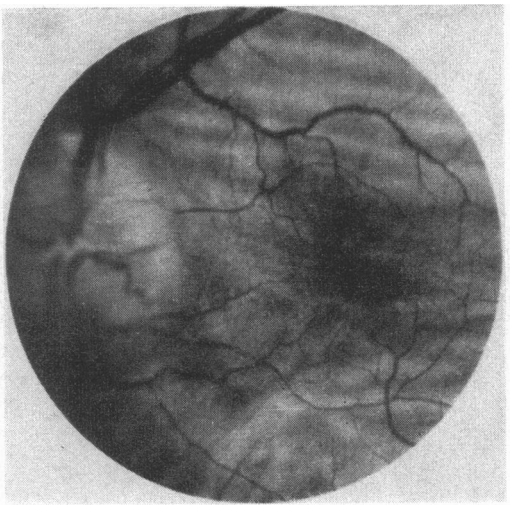

Fig 3 **Case 2**

were read as normal. The brain scan was normal. A spinal tap was normal for pressure and protein. Neurological examination was entirely normal.

The clinical diagnosis has been meningioma of the optic nerve and, in view of the excellent function and minimal discomfort for over nine years, it has been decided to follow the patient.

She was last seen on May 17, 1966, and there was essentially no change in her clinical picture.

Summary: This patient has a long history of papillœdema and exophthalmos progressing slowly with retention of good acuity and function. In addition to chronic papillœdema, the posterior pole has horizontal and curvilinear folds (Fig 3). Some of these have a fluorescein pattern similar to angioid streaks and suggest breaks in Bruch's membrane, but others have the alternating light and dark lines typical of choroidal folds (Fig 4A, B). These folds have shown no tendency to change in two years of observation.

Case 3 P K, woman aged 35
(Courtesy of Dr G M Shannon and Mr J Justice, Wills Eye Hospital, Philadelphia)
Noted the onset of painless left proptosis only six months before being seen at Wills Eye Hospital. She had no diplopia, but visual acuity with best correction was 20/40–3 in the left eye and 20/20 in the right eye. The posterior polar findings were those of obvious striæ and early papillœdema. The only other positive finding on complete evaluation was Hertel exophthalmometer measurements of 5 mm exophthalmos on the left.

The retrobulbar tumour, approached via a Kronlein incision, proved to be a large hæmangioma with ramifications of the III nerve lying on its surface. It was removed *in toto*.

Summary: This case of pathologically proven hæmangioma of the orbit shows oblique and curvilinear folds which were demonstrated by fluorescein angiography.

Case 4 R A, woman aged 74
This patient noticed a shadow in the temporal field of the right eye six months before being referred for evaluation of a suspected choroidal tumour.

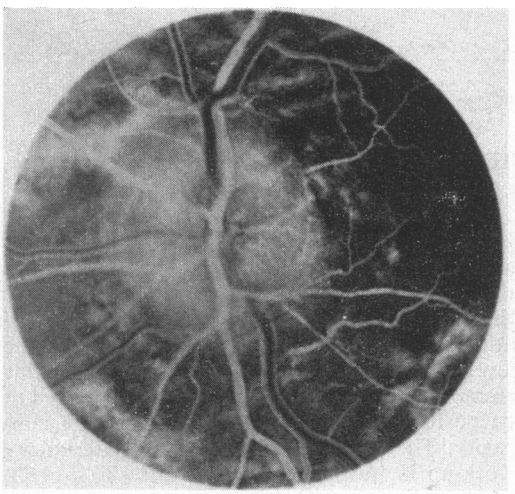

Fig 4A **Case 2** *Arterial phase*

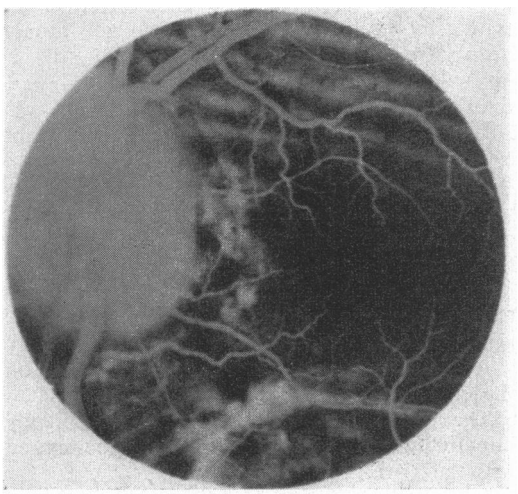

Fig 4B **Case 2** *Arterial-venous phase*

Past and family history are noncontributory. Vision: right eye 20/40, left eye 20/40. Tension (applanation) 14 and 18 mmHg. The disc appeared normal. Horizontal striæ were noted to run through the macular area and on slit-lamp examination they were seen to be folds deep to the retina and at the level of the choroid and pigment epithelium. Superotemporally, there was a large elevated lesion that had retina attached to its surface. The mass extended across the ora on to the pars plana. There was a detachment inferiorly with some blood in the subretinal space. In the macular area, in addition to the choroidal folds there was preretinal wrinkling and no evidence of vitreous separation. Transillumination revealed a large defect consistent with the mass. On slit-lamp examination of the left eye there was wrinkling of the inner layer of the retina, the so-called 'cellophane' appearance.

A clinical diagnosis of malignant melanoma of the choroid with secondary choroidal folds with retinal detachment was made.

The patient was admitted to the hospital and enucleation was carried out.

Summary: This patient presented with a large multilobulated choroidal mass with secondary retinal detachment. In the posterior pole typical horizontal folds were noted and were readily demonstrated with fluorescein. On slit-lamp examination the prominent folds were noted to be deep to the retina. Pathologically (Fig 5A, B, C, D) the choroid was congested and the prominent folds were shown to involve the choroid, Bruch's membrane, and the pigment epithelium. The pigment epithelium on the crests of the folds appeared normal, but it was thickened in the valleys. The retinal detachment was an artifact and could not be fully evaluated.

Case 5 Z M, woman aged 68
This patient developed a retinal detachment in 1961 operated upon with diathermy with a good result.

On November 21, 1967, the patient noted a blurring of vision and a shadow in the inferior nasal field and was found to have a recurrence of the detachment and was referred for treatment.
Past medical history: Coronary occlusion in 1962.

Her vision in both eyes was 20/25. Ophthalmoscopy in the right eye was normal while in the left eye a retinal detachment was seen extending from 10 to 1.30 o'clock and back to the upper pole of the disc. There was an area of lattice between 10 and 11 o'clock with an area of previous diathermy at 10 o'clock. There was a radial split in the retina at the upper border of the previously placed diathermy extending well posteriorly towards the disc. There was a smaller break superiorly to this at the equator.

At operation cryotherapy was applied to the break and a radial silastic sponge implant was placed under the hole extending back towards the disc. Fluid was not drained. Post-operatively the retina was attached on the buckle. All the fluid absorbed but the buckle was very high and extended back to the disc.

The patient was discharged three days post-operatively and was seen again two weeks later at which time the buckle was noted to be high. The visual acuity was 20/400 and many choroidal folds were seen running through the macular area. These gave a typical fluorescein pattern of light and dark lines. No definite subretinal fluid could be recognized under the macular area. Because of the very posterior location of the radial buckle, the poor visual acuity and the choroidal striæ, three weeks post-operatively the radial buckle was removed. Forty-eight hours later the choroidal folds were gone. The retina remained attached and the visual acuity improved to 20/50. Fluorescein studies at that time no longer showed the choroidal striæ but did show some irregular derangement in the pigment epithelium.

Summary: This patient developed typically appearing horizontal choroidal folds in the macula, following a large radial buckle in the superior nasal quadrant. This resulted in a marked drop in visual acuity. Following removal of the implant, the folds disappeared and the acuity improved. When the folds were present, a typical light and dark pattern was seen on fluorescein angiography which was replaced by irregular choroidal fluorescence when they disappeared.

Case 6 S M, woman aged 65
First seen on May 24, 1960, with an acute attack of glaucoma in the right eye and a history of a similar attack two years previously with spontaneous remission. Visual acuity: right eye with $+7{\cdot}50 = +2{\cdot}00 \times 125 = 20/40$; left eye with $+7{\cdot}75 = +1{\cdot}00 \times 70 = 20/100$.

The patient gave a history of hypermetropia and an esotropia as a child and poor vision in the left eye since childhood. The rest of her medical history was noncontributory. A peripheral iridectomy was performed on the right eye at that

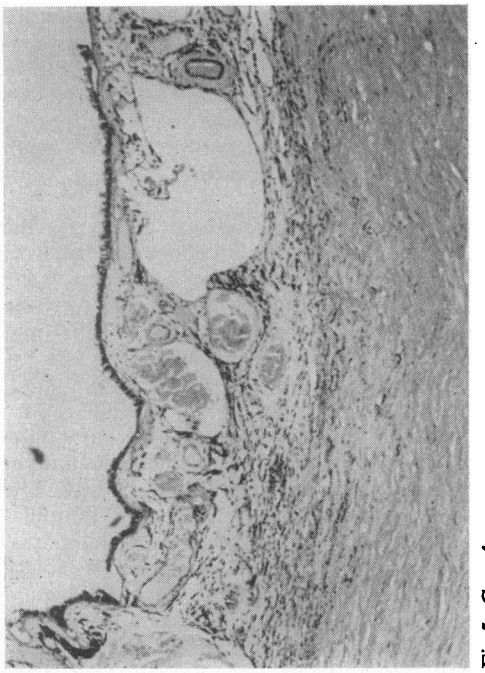

Fig 5C Case 4

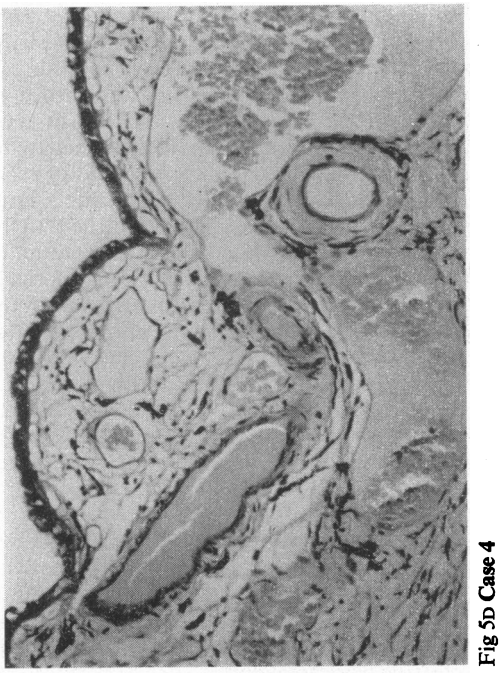

Fig 5D Case 4

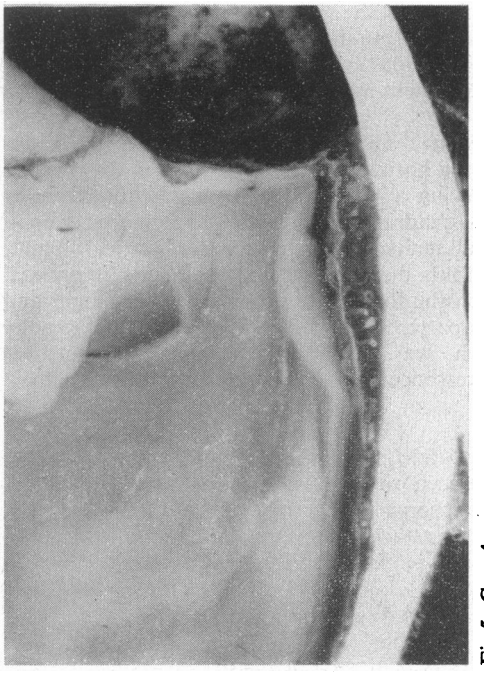

Fig 5A Case 4

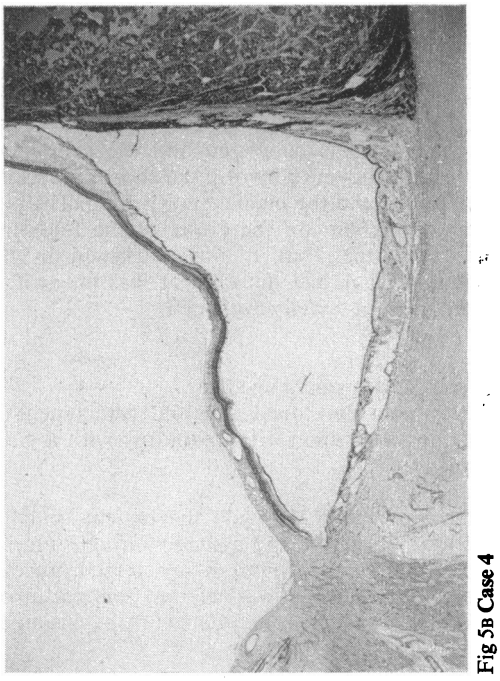

Fig 5B Case 4

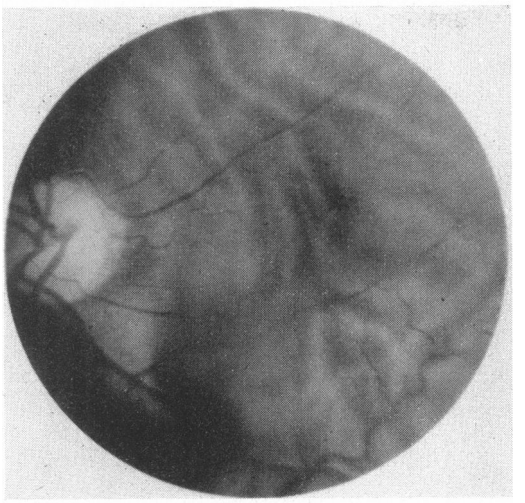

Fig 6A **Case 6**

time. Eight months post-operatively following dilatation of her right pupil horizontal and oblique folds deep to the retina at the level of the pigment epithelium were noted around the posterior pole. At the time of this initial observation it was noted that these folds were similar to those seen with orbital tumours. They were present in both eyes and tended to light up brilliantly with indirect illumination. There was no clinical evidence of an orbital mass on either side.

The patient was last examined on August 2, 1967, and, except for a slowly developing immature cataract, there has been no change in her status. There was no evidence of an orbital mass and the folds remain as previously noted.

Summary: This patient had known hypermetropia since childhood with amblyopia in the left eye. Following recovery from peripheral iridectomy in the right eye for angle-closure glaucoma, choroidal folds were noted in each fundus (Fig 6 A, B). These tended to run vertically and obliquely and with less regularity than in some of the orbital tumours. However, they did show a similar pattern on fluorescein studies. With the Hruby lens these folds were deep to the retina but tended to be seen better with indirect illumination, similar to some drusen.

Case 7 J P, man aged 49

The patient was first examined on May 18, 1967. He gave a history of having poor vision in the right eye since childhood. The left eye was asymptomatic except for hypermetropia until February 7, 1967, when he noted the onset of distorted vision. He has been followed since with a classical picture of central serous choroidopathy and was referred for evaluation of this.

We were fortunate in being able to contact his childhood doctors through the courtesy of Dr Thomas Kerns at MacPherson Hospital, Durham, NC, and get his childhood records. He was seen at the age of 9 with a vision in the right eye of 20/200 and in the left eye of 20/20. Refraction: right eye +8·00, left eye, +5·00. At the age of 18 he underwent muscle surgery. He had a right lateral rectus resection and a right medial rectus recession. No mention was made of choroidal striæ.

Examination now shows right vision of 20/400 and a left of 20/50 with refractive errors similar to above. Applanation tension 18 and 15 mmHg. Ophthalmoscopy in the right eye revealed prominent horizontally orientated choroidal folds extending through the macular area. In the left eye there appeared to be a fold in the retina extending from the margin of the disc to the fovea with minimal evidence of serous detachment. There were numerous choroidal folds similar to those in the right eye. The fluorescein study showed a picture in the left eye consistent with central serous choroidopathy. Both eyes showed retinal folds passing through the macular area and, in addition, irregular lines radiating obliquely in the posterior pole.

Summary: This patient with known hypermetropia since the age of 9, with amblyopia in the right eye, recently developed central serous choroidopathy in the left eye. He showed typical choroidal folds running through the macula in

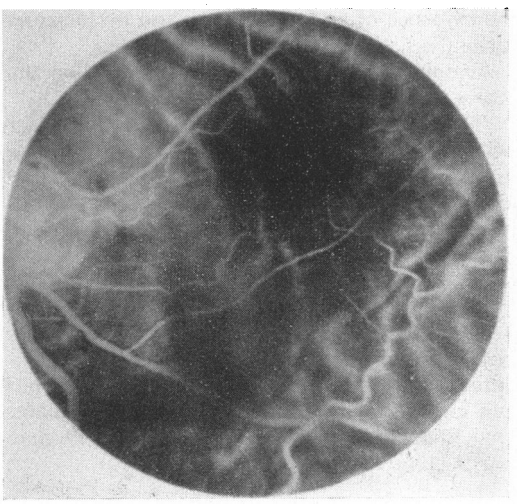

Fig 6B **Case 6**

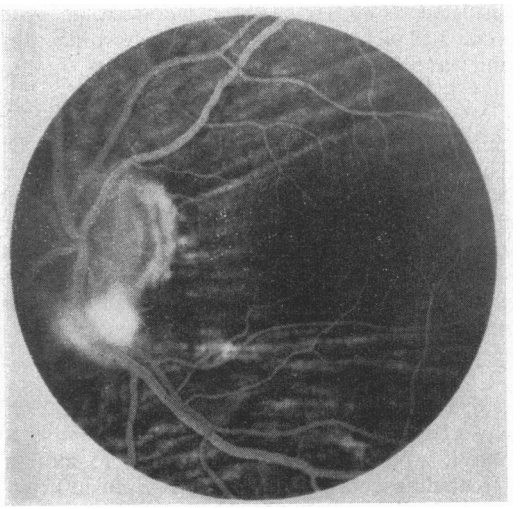

Fig 7A Case 8

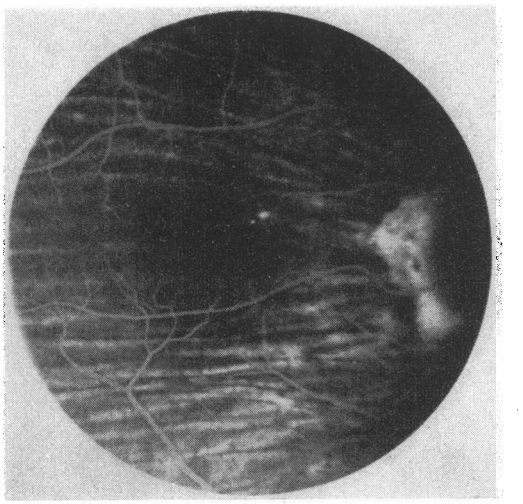

Fig 7B Case 8

the right eye, but in addition had many irregular lines at the same depth that were better seen on indirect illumination. These are narrower and less elevated than the typical choroidal folds.

Case 8 C R, man aged 48

This patient has worn glasses for hypermetropia since childhood. He was seen for routine refraction in the Eye Clinic on April 13, 1965: Right eye $+6{\cdot}50 = +0{\cdot}75 \times 90 = 20/25$; left eye $+6{\cdot}50 = +0{\cdot}75 \times 90 = 20/25$. Detailed eye examination was negative except for myelinated nerve fibres at the upper pole of the right disc and extensive choroidal folds running horizontally from the disc through the macula in both fundi. There was no evidence of an orbital mass and no increased orbital resistance.

Exophthalmometer readings were well within normal limits and symmetrical.

The patient has been followed up regularly without any evidence of change. His last examination was January 31, 1968. His general medical condition has required eight admissions to hospital over the past seven years because of generalized vascular disease. He has had previous diagnoses of cerebral infarction in the right middle cerebral artery distribution, thrombosis of the left renal artery causing renal hypertension necessitating left nephrectomy, transient ischæmic attacks consisting of basilar artery disease which are currently being treated with anticoagulants, and ECG changes consistent with an old myocardial infarction.

Summary: A patient with severe generalized vascular disease and having worn glasses for years was found on routine examination to have extensive choroidal folds in both fundi. These are horizontal and show a typical pattern with fluorescein (Fig 7A, B). His refractive error is that of high hypermetropia. The choroidal folds are more horizontal and very similar to Case 1. During three years of observation no evidence of an orbital mass in either eye has developed.

Case 9 R C, man aged 32

(Seen through the courtesy of Dr Stanley Spielman)

The patient was first examined in March 1965 for a routine refraction. Right eye, plano $= +0{\cdot}50 \times 90 = 20/20$; left eye, plano $= +1{\cdot}00 \times 90 = 20/20$. On examination at this initial time both fundi were reported to be normal.

A year and a half ago the patient developed gradual painless loss of vision in his left eye. An examination at that time showed his vision to have dropped to 20/40 in the left with a cycloplegic refraction in the right eye of $-0{\cdot}25 = +0{\cdot}50 \times 90 = 20/20$ and in the left eye of $+0{\cdot}50 = +1{\cdot}25 \times 90 = 20/40$.

Skull X-rays were normal. The patient's vision has remained unchanged. His past history, except for a suggestion of hypertension, has been noncontributory.

Examination on March 5, 1968, showed the cycloplegic refraction in the right eye to be plano $= +0{\cdot}50 \times 90 = 20/20$ and in the left eye, to be $+2{\cdot}25 = +1{\cdot}50 \times 100 = 20/25$. The Amsler Grid was normal with the right eye. In the left eye he had a defect consisting of blurring just temporal to the macular area but the patient was unaware of any waving lines. Exophthalmometer reading:

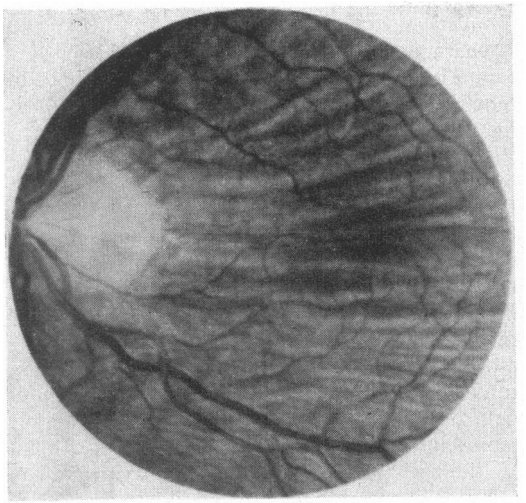

Fig 8A **Case 9**

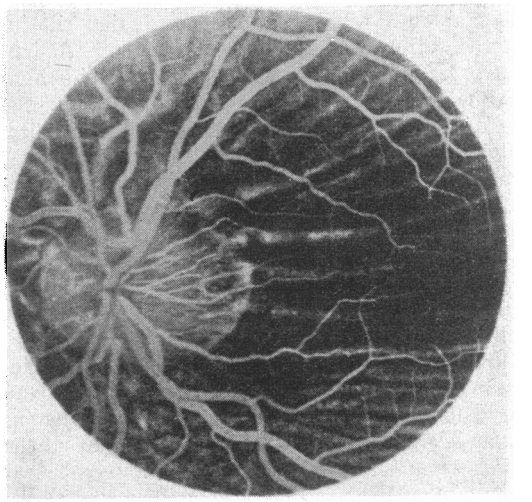

Fig 8B **Case 9**

right eye 16; left eye 16·5. Ophthalmoscopy: right eye entirely normal; left eye revealed horizontal choroidal folds which, on examination with the Hruby lens, were definitely under the retina at the level of the pigment epithelium. The inner layer of the retina had minimal irregularity. Fluorescein angiography showed the typical horizontal light and dark pattern and showed no change from the pattern of one year before.

Summary: This patient apparently developed choroidal striæ in his left eye over a period of one and a half to three years. During this time he experienced blurring of vision and his refractive error became significantly more hypermetropic. Up to now he has shown no clinical evidence of an orbital mass. Fluorescein studies show a similar pattern to Cases 1 and 8 (Fig 8A, B).

Case 10 J M D, man aged 43
(Reported through the courtesy of Dr Richard Ruiz)
Examination in June 1967 because of rather sudden blurring of vision in the left eye three months before, without any change since.

Refraction: right eye $-0{\cdot}25=+0{\cdot}25\times 15=$ 20/20, left eye $+2{\cdot}00=+0{\cdot}50\times 160=20/25$.

Ocular tensions 14 mmHg in each eye (applanation).

The right eye was entirely normal to examination. The left eye revealed choroidal folds radiating between the macula and disc. The foveal reflex was poor and the macula was thought to be œdematous.

Amsler Grid in the right eye was normal; in the left eye there was marked distortion of the lines.

Fluorescein angiography revealed the typical pattern of choroidal folds. There was no evidence of exophthalmos or increased orbital resistance. X-rays of the orbits were entirely normal.

The patient was last examined in February 1968 and no change had been noted.

Summary: This patient at the age of 43 suddenly developed blurred vision in the left eye, has a hypermetropic refraction in that eye, and on examination was found to have choroidal folds in the posterior pole with a typical fluorescein pattern. There has been no change during eight months of observation and the patient shows no other evidence of a retrobulbar mass.

Discussion and Differential Diagnosis

A series of cases has been presented, each of which demonstrated folds in the posterior pole deep to the retina, at the level of the choroid, Bruch's membrane and pigment epithelium, and showed a similar pattern of alternating light and dark lines on fluorescein angiography. In addition, some of these patients had decreased visual acuity, distortion of the Amsler Grid, and hypermetropic refractive errors.

The fluorescein pattern is typical and in some cases the folds were not recognized clinically until their presence was noted on fluorescein studies. The usual folds are recognized by the early passage of fluorescein through the choroid and its late disappearance without staining. The light portion of the fold corresponds to the crest and transmits the choroidal fluorescence brilliantly, while the dark portion corresponds to the valleys and transmits the choroidal fluorescence poorly.

We assume, but have not proved, that the increased transmission of the crests is related to atrophy of the overlying pigment epithelium while the decreased fluorescence of the valleys is related to their inclination which does not reflect light well and effectively gives an increased thickness to the pigment layer. It is also possible that the crests overlie a pool of fluorescein in the choroid.

As is evident from this material, such folds are not diagnostic of a retrobulbar mass. Certainly this diagnosis must be considered in each case; however, folds are found in association with long-standing hypermetropia and may have developed unilaterally in two patients with increasing hyperopia without any other clinical signs that, at the present at least, would point to a retrobulbar mass. Another possibility, at least in one of the cases (Case 10), is that there may have been a pre-existing anisometropia which presented as a visual problem with the development of presbyopia.

The exact mechanism of fold formation is obscure but certainly its high correlation with orbital tumours and its occurrence with melanoma of the choroid, and with a large scleral buckle, indicate either a mechanical disturbance or congestion of the choroid in the posterior pole as a possible mechanism. Since clinically these do appear similar to 'choroidals' in the posterior pole, what is their relationship to these?

We have several patients with high degrees of hypermetropia apparently dating back to childhood with typical choroidal folds. Why do these patients have folds and other hypermetropes not?

Finally, what is happening in the two patients who apparently have developed these folds unilaterally along with hypermetropia? Has there been scleral shrinkage and a shortened globe or has their choroid thickened and pushed the retina forward? Only continued observation and pathological study of these cases will resolve these problems.

Summary

A characteristic fluorescein angiographic pattern found in choroidal folds is presented. The occurrence of these folds in retrobulbar masses, hypermetropia and other conditions is discussed.

Conclusion

I have presented in this lecture certain changes that occur in the fundus and occur frequently enough so that most ophthalmologists have seen them even if they did not recognize them. I am certain these changes do not represent new conditions despite the fact that I failed to recognize some of them a decade ago and some I cannot find described in the literature. They merely represent observations that are readily possible if all the techniques of examination available are utilized. In this respect I would like to emphasize biomicroscopic examination of the retina and choroid with both the Hruby and contact lenses, stereo fundus photography and fluorescein angiography. I am certain that with the routine use of these techniques, and new ones as yet undiscovered, we shall come to a better understanding of pathological processes in the fundus.

It is only through the use of new techniques and a questioning mind that we will *prevent* ourselves from becoming the type of physician to whom my Professor of Neurology, Dr Harold G Wolff, used to refer to as 'a man who can make the same mistake a thousand times and call it experience'.

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