UVEAL EFFUSION SYNDROME: A NEW HYPOTHESIS CONCERNING PATHOGENESIS AND TECHNIQUE OF SURGICAL TREATMENT*

BY *J. Donald M. Gass*, MD

EXUDATIVE DETACHMENT OF THE CHOROID AND CILIARY BODY MAY BE CAUSED BY A variety of disease states, eg, postoperative hypotony, scleral buckling procedures, scleritis, etc. In addition, it may occur spontaneously with no apparent cause in one or both eyes of healthy individuals, particularly middle-aged males. These patients are defined as having the idiopathic uveal effusion syndrome.[1,2] In this latter group of patients other findings which may be present include: dilation of episcleral blood vessels, blood in Schlemm's canal, normal intraocular pressure, few vitreous cells, nonrhegmatogenous retinal detachment with shifting of subretinal fluid, elevation of subretinal fluid protein to two or three times the normal plasma level, elevation of cerebrospinal fluid protein (50% cases), protracted clinical course with remissions and exacerbations, and poor response to treatment with corticosteroids, antimetabolites, drainage of subretinal exudate and scleral buckling.

In a previous report of nine patients with the uveal effusion syndrome studied at the Bascom Palmer Eye Institute, we concluded that its underlying cause was a congenital anomaly of the sclera and vortex veins that resulted in intermittent obstruction of the flow of the venous outflow of the posterior uveal tract.[2] A similar pathogenesis had been previously suggested by others.[3,4] In 1975, Shaffer[5] in discussing Calhoun's paper concerning uveal detachment occurring after surgery for narrow angle glaucoma in nan-ophthalmic eyes, suggested that vortex vein obstruction caused by the thickened sclera may be responsible for the detachment. Brockhurst[6] reported decompression of the vortex veins as a successful

*From the Bascom Palmer Eye Institute, Department of Ophthalmology, University of Miami School of Medicine, Miami, Florida. This investigation was supported in part by Public Health Research Grant No EY-02549-03 from the National Eye Institute, National Institutes of Health, Bethesda, Maryland.

treatment of postoperative ciliochoroidal detachment in nan-ophthalmic eyes. We attempted a similar procedure in both eyes of one of our patients with bilateral uveal effusion syndrome and long-standing bullous retinal detachment.[2] The eyes were slightly smaller than average. The sclera was abnormally thick. Only three vortex veins were present in one eye. Several veins in both eyes were hypoplastic. Our attempt to dissect the veins free of sclera resulted in amputation of four and rupture of the fifth vein operated. In spite of these complications, and a large suprachoroidal hemorrhage occurring intraoperatively and following surgery in one eye, the uveal and retinal detachment resolved in both eyes. It was this observation that prompted me to consider the possibility that it was the excision of large partial thickness scleral flaps at the sites of the attempted vortex vein decompression that might be responsible for the favorable outcome in our patient, as well as those in Brockhurst's cases with nan-ophthalmic eyes.

The two primary purposes of this report are: (1) to present a new hypothesis for the pathogenesis of this syndrome, and (2) to present results of a surgical procedure to test the hypothesis. Hypothesis—The primary underlying cause of uveal effusion syndrome is a scleral abnormality (probably congenital) which predisposes the eye to vortex vein obstruction, and which more importantly acts as an abnormal barrier to the transport of protein out of the eye.

To test the hypothesis that the barrier effect of the sclera is more important than vortex vein obstructing effect, I performed multiple segmental equatorial partial thickness sclerectomies and sclerostomies without drainage of subretinal fluid and without decompression of the vortex veins first in one eye and later in the second eye of a patient with long-standing bullous retinal detachment caused by the uveal effusion syndrome.

CASE REPORT

Details of the early clinical course and fundus photographs were reported previously (case 2).[2] The patient was a mildly myopic (-1.00 sphere OD, and -0.50 sphere OS) Hispanic man with an 8-year history of recurrent episodes of metamorphopsia and blurred vision in the right eye and a 4-year history of similar complaints in the left eye. He was initially misdiagnosed as having bilateral idiopathic central serous choroidopathy (ICSC). He developed bilateral ciliochoroidal and bullous retinal detachment that persisted for 2½ years prior to November, 1981, when at age 45 years surgery was recommended for his right eye. His medical and

family histories were unremarkable. Visual acuity in the right eye was counting fingers at 4 feet and in the left eye was 20/200. The episcleral blood vessels were dilated and blood was present in Schlemm's canal in each eye. The anterior chambers were of normal depth. The applanation intraocular pressure was 12 mm Hg in the right eye and 9 mm Hg in the left eye. There was a 1+ aqueous flare in the anterior chamber of the left eye. Two-plus vitreous cells were present bilaterally. There was bilateral 360° ciliochoroidal detachment and a bullous retinal detachment with shifting subretinal fluid in each eye (Fig 1A and B). There was prominent leopard-spot mottling of the pigment epithelium bilaterally. This was most evident angiographically (Fig 1C). There were several plaques of fibrous metaplasia of the pigment epithelium in the posterior pole of the right eye and temporal to the macula in the left eye. There was cystic degeneration of the retina in the right macula. The optic discs were normal. Ultrasonography revealed thickening of the posterior choroid and a peripheral ciliochoroidal and retinal detachment bilaterally. The axial length of each eye was normal ultrasonographically. His general physical examination was unremarkable except for his blood pressure of 150/100. He was placed on a low salt diet and propranolol hydrochloride (Inderal), 25 mg twice daily.

The right eye with no potential for central vision was operated on initially. At surgery the eye appeared to be normal in size. The episcleral vessels anterior to the recti muscle insertions were dilated. The orbital blood vessels were not congested. Normal caliber vortex veins exited posterior to the equator in the 1:30, 4:30, and 6:30 meridian. None were present in the superotemporal quadrant. Four rectangular 5 × 7 mm, one-half to two-thirds thickness sclerectomies were done in each quadrant taking care to avoid the areas anterior to exit sites of the vortex veins. These were centered 1 to 2 mm anterior to the equator with the long axis oriented circumferentially (Fig 2). The sclera appeared to be thicker than normal (1.5 to 2 mm). A linear 2 mm sclerostomy was made in the center of each of the sclerectomy sites. There was drainage of some watery suprachoroidal fluid from each of these sites. The 1-mm Gass punch was used to enlarge the scleral opening. No attempt was made to perforate the choroid. The first few days postoperatively the anterior chamber was of normal depth and the applanation intraocular pressure of the eye operated upon was 24 and 20 mm Hg. Three weeks postoperatively, the amount of subretinal fluid was unchanged. It was reduced by 50% at 4 weeks, and by the third month postoperatively all supraciliochoroidal and subretinal fluid was gone. In the left eye, the area and height of bullous retinal detachment had increased. Six months later the same operative proce-

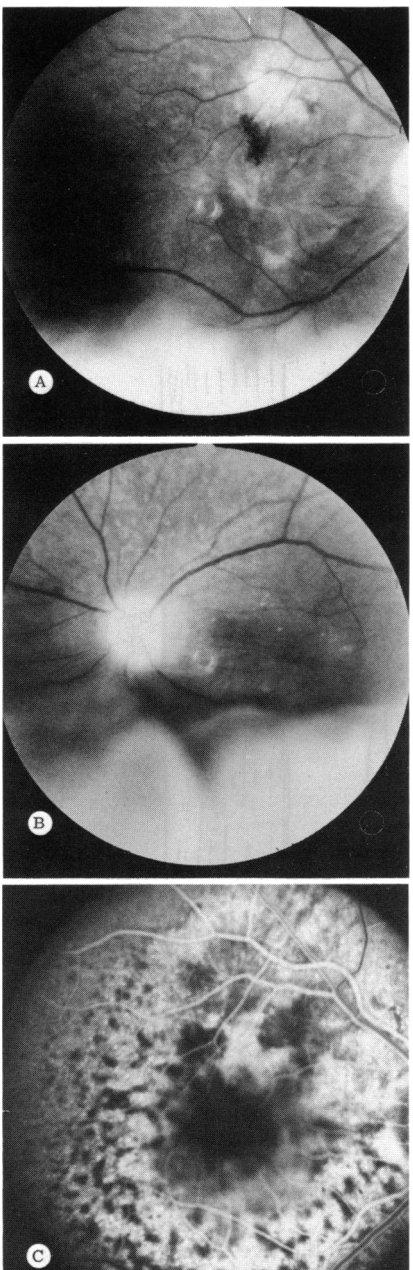

FIGURE 1
A: Right eye, preoperatively. B: Left eye, preoperatively. C: Right eye, fluorescein angiography.

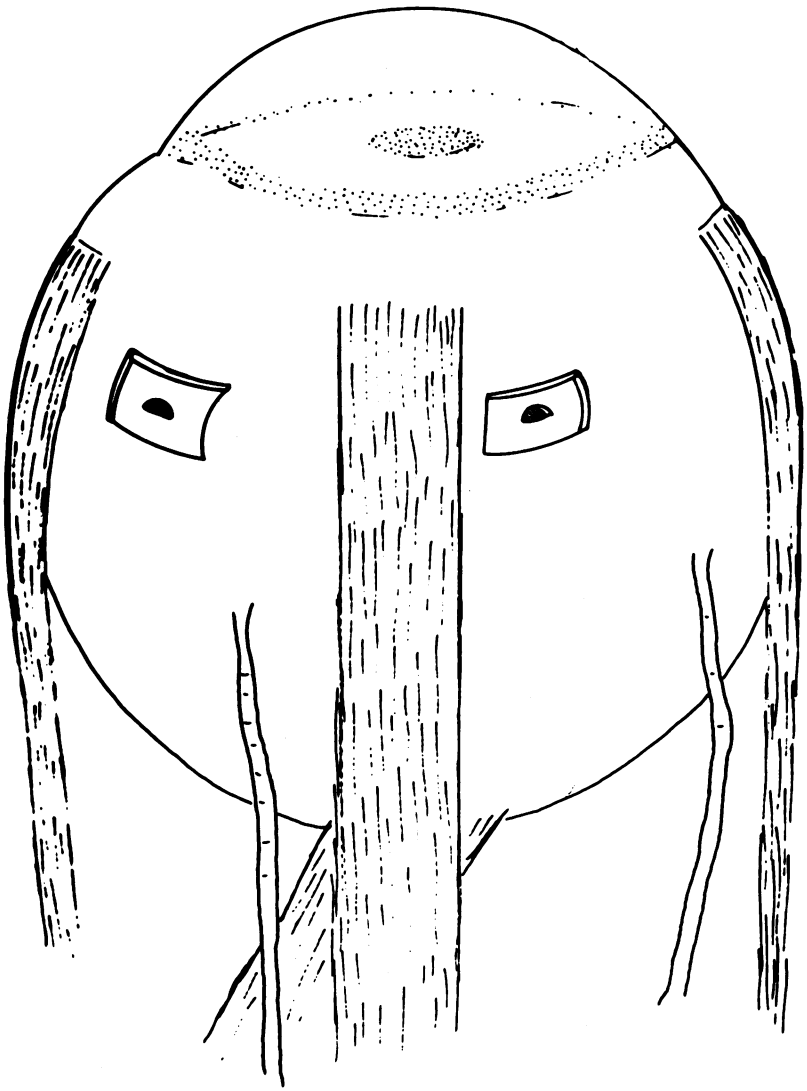

FIGURE 2
Diagram of operative procedure.

dure was performed on the left eye. Only two vortex veins were present, both located on the nasal side of the eye. Six weeks postoperatively, only a small area of serous detachment of the peripheral retina was present between 5:30 and 6:30 o'clock. All supraciliochoroidal and subretinal fluid

was gone at the 10-week postoperative examination (Fig 3A and B). Visual acuity in the right eye was 20/200, and in the left eye was 20/70. The episcleral vessel dilation was no longer present. The applanation intraocular pressures were 10 mm Hg in each eye. Gonioscopy revealed no evidence of blood in Schlemm's canal. Five months postoperatively, no detachment was present in either eye. Visual acuity in the right eye was 10/200 and in the left eye was 20/50.

Routine light microscopy and histochemical stains revealed no abnormality of the excised scleral flaps.

DISCUSSION

The disappearance of the long-standing large volume of subretinal exudation within 3 months in both eyes of this patient after equatorial sclerectomies and sclerostomies alone suggests that the thickened sclera was reponsible for the eye's inability to rid itself of the concentrated proteinaceous exudate in the supraciliochoroidal and subretinal spaces.

This concept is in keeping with knowledge concerning vascular pathophysiology. Unlike water and electrolytes, protein that escapes from normal, as well as abnormal, blood vessels into the interstitial tissues cannot easily re-enter the blood circulatory system directly because of the high protein concentration in the plasma. It must find its way into the regional lymphatics, whose primary function is to maintain a low protein concentration in the interstitial fluid.[7] Since there are no lymphatics in the eye, there are two primary pathways for removal of intraocular extravascular protein: (1) Schlemm's canal and aqueous veins anteriorly and, (2) transscleral diffusion directly through the sclera, as well as along its emissary canals into the orbital tissues posteriorly (Fig 4).[8,9] Protein once outside the eye enters the orbital and conjunctival lymphatics.[10]

In times of transient compromise of the choroidal vascular integrity caused by a variety of ordinarily innocuous pathophysiologic changes, eg, viral infection, allergic reaction, minor trauma, elevated blood pressure, etc, this barrier of abnormally thickened or constituted sclera is sufficient to cause progressive concentration of extravascular protein in the choroid and ciliary body to levels equal to that in the blood plasma, as well as detachment of these structures because of increased tissue colloid osmotic pressure (Fig 4).[11,12] Encroachment on the unusually long emissary scleral canals by swelling of the sclera caused by high protein concentration may be responsible for either causing or aggravating the signs of vortex vein obstruction seen in these patients. This obstruction in turn causes congestion of the uvea and additional protein extravasation. Decompensa-

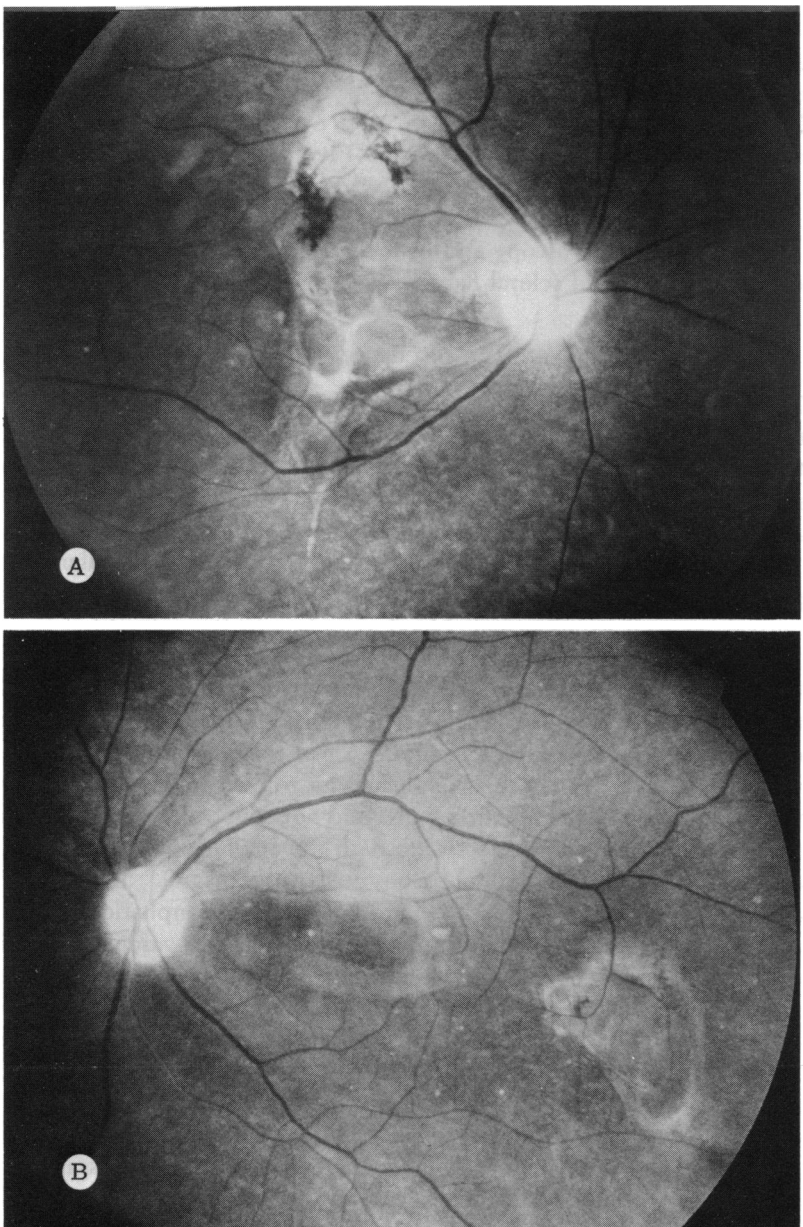

FIGURE 3
A: Right eye, postoperatively. B: Left eye, postoperatively.

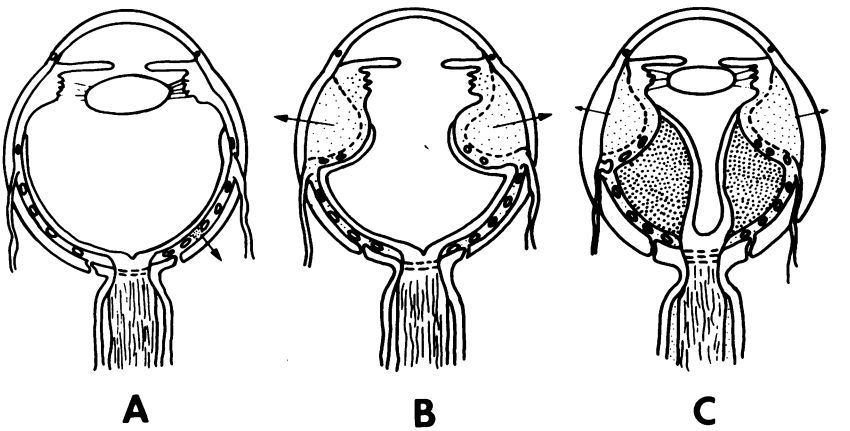

FIGURE 4
Diagrams of pathways (arrows) of transscleral movement of extravascular protein (stippling). A: Normal eye with focal vascular leak. B: Aphakic eye with transient postoperative ciliochoroidal detachment that may require several days or weeks for restoration. C: Uveal effusion syndrome with increased resistance to protein outflow and uveal venous outflow caused by abnormal sclera.

tion of the pigment epithelium caused by the uveal congestion and detachment permits water and protein to extend from the choroid into the subretinal space. There, the colloid osmotic forces of the subretinal protein are opposed by the dehydration forces of the pigment epithelial and retinal endothelial physiologic pump. As a consequence the subretinal protein concentration rises to levels equal to two or three times that found in the plasma.[1,2,11,12] This high subretinal and choroidal protein concentration may be responsible for diffusion of protein into the perioptic subarachnoid space in amounts adequate to elevate the cerebrospinal fluid protein level. The superconcentration of protein in the subretinal fluid is probably the cause of its rapid shifting with changes in position. It may also play a role in causing the characteristic leopard-spot pattern of derangement of the pigment epithelium in these patients.

Any hypothesis concerning the pathogenesis of uveal effusion must account for the normal intraocular pressure that is usually present in spite of the widespread ciliochoroidal detachment. In the past, the hypotony that persisted in patients following cyclodialysis and following spontaneous closure for postoperative wound leaks after cataract extraction was attributed to decreased aqueous production by the detached ciliary body. There is now evidence to suggest, however, that the hypotony is instead caused by increased uveoscleral outflow of aqueous humor via the ex-

panded suprachoroidal space (Fig 5).[13] In the normal eye this portion of outflow probably constitutes less than 15% of the total outflow.[14] In the uveal effusion syndrome the following counterbalancing forces may be responsible for maintenance of a normal intraocular pressure: (1) increased transscleral resistance to aqueous outflow in spite of the increased pathways to posterior flow through the expanded supraciliochoroidal space, and (2) increased resistance of anterior outflow by elevation of the episcleral venous pressure, associated with vortex vein obstruction (Fig 5).

Why, if idiopathic uveal effusion is caused by a congenital anomaly of the sclera, are most patients middle-aged males at the time of onset of their signs and symptoms? It is hypothesized that aging and hormonal changes in the collagen and ground substance of the congenitally abnormal sclera are responsible for reducing scleral permeability to protein to a level where the eye becomes incapable of handling even small amounts of extravascular protein occurring from minor insults to the uveal vasculature.

ICSC is another clinical entity that shares some features in common with the idiopathic uveal effusion syndrome. Both affect predominantly healthy middle-aged males. Both cause recurrent serous detachment of

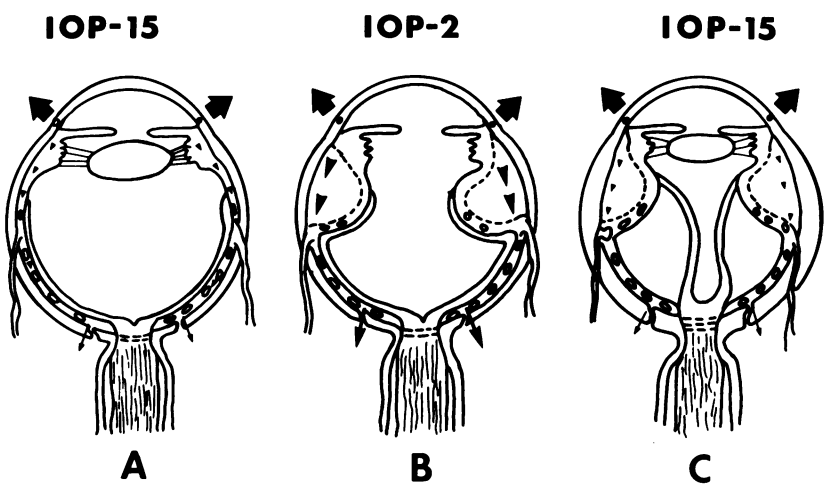

FIGURE 5

Diagram of uveoscleral outflow of aqueous humor. A: Normal intraocular pressure (IOP) in normal eye with 15% of aqueous outflow (arrows) via uveoscleral pathways. B: Hypotony caused by increased pathways for uveoscleral outflow (small arrows) in postoperative aphakic eye with ciliochoroidal detachment. C: Normal IOP in uveal effusion syndrome in spite of ciliochoroidal detachment because of increased transscleral resistance to aqueous outflow.

the retina that is unresponsive to corticosteroid therapy. Both occur infrequently in myopic eyes. Both have a relatively good visual prognosis except in those cases with long-standing recurrent detachment. While the initial retinal detachment in uveal effusion begins in the periphery, it may be confined to the macula. Vitreous cells, which are common in uveal effusion, and which are probably in response to the elevated protein concentration,[2] are not a feature of ICSC. Focal fluorescein leaks and focal pigment epithelial detachments are typical of ICSC, but occur infrequently in uveal effusion. The association of emotional stress and onset of ICSC noted by some authors is not a feature of uveal effusion. Ciliochoroidal detachment is a feature of uveal effusion, but in the past was noted in as few as one-third of cases.[1] Diffuse ciliochoroidal detachment is probably present in all of these patients, but may be detected in some only with the aid of ultrasonography.[2] Because of the similarities of these two diseases, an ultrasonographic study to look for evidence of mild ciliochoroidal detachment in patients with ICSC is in progress at the Bascom Palmer Eye Institute.

Although the disappearance of the detachment in the above patient would appear to have been caused by providing new pathways for transscleral outflow of protein from the eye, it is possible that the resolution was merely a coincidence or that drainage of supraciliochoroidal fluid triggered some other mechanism to cause disappearance of the subretinal exudate. Additional trials of the surgical procedure in patients with long-standing detachment are required to verify its effectiveness. Further histochemical, as well as transmission and scanning electron microscopic studies are needed to determine if the sclera in patients with uveal effusion is qualitatively and structurally different from that in normal eyes. Douglas Anderson, MD, is conducting animal experimentation to attempt to reproduce uveal effusion by altering the scleral pathway of protein outflow. If further investigation confirms the hypothesis, the surgical technique of posterior sclerectomies and sclerostomies may have other applications in management of patients with nan-ophthalmos or other causes of persistent secondary uveal and retinal detachment.

SUMMARY

It is hypothesized that the primary underlying cause of the idiopathic uveal effusion syndrome is a congenital anomaly of the sclera, and in some cases, the vortex veins. Superimposed aging and hormonal changes in the sclera and its emissary channels impair its permeability to protein and predispose the eye to vortex vein obstruction. The inability of the eye to

transport extravascular protein across the abnormal sclera is probably the cause of prolonged exudative detachment of the uvea and retina in these patients.

Partial thickness sclerectomies and sclerostomies without choroidal puncture in each quadrant near the equator caused prompt resolution of long-standing uveal and bullous retinal detachment in two eyes.

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DISCUSSION

Dr Robert J. Brockhurst. I am very pleased to open the discussion of this excellent paper suggesting a most intriguing and simple theory of the mechanism of a very complex disorder known as uveal effusion. First, I would like to make a few comments and then close with some questions.

I think we should define uveal effusion as a syndrome showing the accumulation of serous fluid in the choroid and/or subretinal space. As such, it is analogous to Coats' disease or Eales' disease, in that several entirely different etiologic

factors may bring about a similar clinical picture. At the present time I classify uveal effusions as inflammatory, hydrostatic, and idiopathic (Table I).

In retrospect, I realize that, 20 years ago in 1963, Doctor Schepens and I included some nan-ophthalmic eyes in our original 17 patients, 16 of whom were males. Moreover, at this time, I do not believe idiopathic uveal effusion is more common in males since in 16 idiopathic cases seen during the past 10 years, 8 were males and 8 were females. In 24 additional nan-ophthalmic patients 13 were females and 11 were males. The chief point I want to make is that we are slowly learning more about so-called "idiopathic uveal effusion" and gradually we will uncover more basic causes, thereby reducing the number of unknown types now lurking in the "idiopathic" category.

I want to make it clear that vortex vein decompression appears to be effective only in one type of uveal effusion, namely in nan-ophthalmic eyes with extremely thick sclera. Successful reattachment of the retina has been obtained in 14 of 17 eyes which have been operated upon. I have not experienced the horrendous difficulties in decompressing vortex veins as described by Doctor Gass. I note that his patient was not nan-ophthalmic and perhaps represents some unusual congenital weakness and abnormality of the vortex veins. This slide shows the desired appearance of a decompressed vortex vein at the time of surgery in typical nan-ophthalmic uveal effusion as I experience it (SLIDE).

Like Doctor Gass, encouraged by my results in nan-ophthalmic uveal effusion, I decided to try removal of sclera, but in the vortex vein area, in so-called "idiopathic uveal effusion" in the more involved eye of two patients with bilateral retinal detachments of 6 and 12 months' duration. One patient was emmetropic and one slightly myopic. The sclera was found to be of normal thickness in one patient and thin in the other. In addition, the thin sclera found in the second patient became wet soon after drying it with a cotton-tipped sponge, indicating that fluid was passing through the sclera from the inside of the eye. In both patients, both retinas gradually reattached in about 1 year, the unoperated and less severely detached retina spontaneously reattaching first. I have not pursued vortex vein decompression, or resection of sclera, in non-nan-ophthalmic cases of uveal effusion since that time.

TABLE I: UVEAL EFFUSION

INFLAMMATORY
1. Trauma, intraocular surgery
2. Uveitis, sympathetic ophthalmia
3. Vogt-Koyanagi-Harada
4. Scleritis, infected buckle
5. Pan retinal photocoagulation
HYDRODYNAMIC
1. Dural arteriovenous fistula
2. Hypotony, wound leak
3. Nan-ophthalmos
IDIOPATHIC

I suspect Doctor Gass' two patients may have undergone spontaneous remission. Over the years I have done bucklings, choroidal and subretinal fluid taps, and scleral resections on a fair number of idiopathic uveal effusions and have not observed thick sclera like that seen in nan-ophthalmos. However, it is possible Doctor Gass has uncovered another subtype in our idiopathic group wherein thick sclera, in essentially emmetropic eyes, results in uveal effusion. If this is true, I think idiopathic cases of uveal effusion should be explored to see how common thick sclera is found, and if the sclera is thick, to perform scleral resections in those patients to settle this question.

In regard to histochemical and electron microscopic study of sclera I would like to mention that Trelstad, Silbermann, and I have reported such studies in nan-ophthalmic sclera. The main differences include a less orderly arrangement of collagen bundles and increased amounts of presumed proteoglycans (SLIDE).

My questions are as follows: (1) Does the author postulate that all idiopathic uveal effusions result from decreased passage of proteins through the sclera, whether or not it is thick, or is one subtype of this interesting and perplexing condition due to thick sclera in essentially normal sized eyes? (2) If the scleral resistance to passage of proteins is responsible for the uveal effusion, why do spontaneous remissions occur? (3) In 1973 the author reported a group of patients with bullous non-rhegmatogenous detachments who showed large, multiple detachments of the retinal pigment epithelium (RPE) and felt these patients had idiopathic central serous choroidopathy. I have seen similar patients with uveal effusion showing these large detachments of the RPE as well as serous retinal detachment. (SLIDES) In my experience these large RPE detachments are much different from the small smokestack leaks characteristic of ordinary "idiopathic central serous choroidopathy." I wonder now, 10 years later, if he feels this entity is another subtype of our wastebasket group of so-called "idiopathic uveal effusion," or is uveal effusion still nothing more than a severe form of "idiopathic central serous choroidopathy?"

Dr Douglas R. Anderson. As befits a glaucoma specialist, I have been interested in the past several years in uveoscleral outflow, as well as the fluid dynamics in the uveal tract as it might relate to the choroidal detachment that sometimes follows filtration surgery. After instruction from Jonathan Pederson, MD, who advised me also to read the books of Guyton, I think I came to understand the relevant basic physiology (which has recently been reviewed by Drs Brubaker and Pederson, *Surv Ophthalmol* 1983; 27:281-289), in a nutshell, to be something like this:

When proteins leak into a tissue, they are in a lesser concentration than in the blood and do not re-enter the bloodstream. The protein molecules command the presence of a certain number of water molecules (the amount determined by the osmotic and hydrostatic forces present), constituting a certain bulk of fluid. In most tissues, this volume of fluid is drained away by the lymphatics and does not accumulate in the tissues as edema unless there is some pathologic condition, such as blockage of the lymphatic drainage, or some change in the osmotic and hydrostatic pressure relationships.

In the choroid, there are no lymphatics. The protein escaping from the choriocapillaris commands a certain volume of water (some that diffuses from the choroidal vessels and some that enters the choroid from the anterior chamber by "uveoscleral outflow" of aqueous humor). The bulk of fluid thus created by protein leakage must somehow exit. This is evidently accomplished as a bulk flow across the sclera with the intraocular pressure as the driving force. Under the conditions of inflammation (with an increase in protein leakage) and hypotony (with a reduced force for transscleral outflow), the fluid collects in the choroid. This edema constitutes what we call choroidal detachment. Important factors include inflammation and hypotony. When the intraocular pressure is restored, the collected fluid is pushed out through the sclera over a period of time.

Some time after I had come to understand this pathophysiology, I suddenly realized one day that the thick sclera of nan-ophthalmic eyes might well explain why they tend to develop more severe and more prolonged choroidal detachments after surgery. With a postoperative increase in protein leakage, the edema would collect more rapidly and take longer to resolve—simply on the basis that the thick sclera constitutes a high resistance to the bulk outflow of the proteinaceous fluid.

I was particularly excited during a hallway discussion with Doctor Gass to learn that he had similar thoughts with reference to patients with choroidal effusion that were under his care, based on the observation he reported today that the effusion cleared following a scleral dissection that had not accomplished a vortex vein decompression.

Within the last year, I was forced to perform a surgical iridectomy on a nanophthalmic eye with acute angle closure glaucoma, because an iridectomy had not been achieved with a laser. Encouraged by Doctor Gass' experience, I removed lamellar patches of sclera between the muscle insertions at the time of iridectomy, leaving behind a thinned sclera over at least a portion of the anterior segment of the globe. There was some shallowing of the anterior chamber, presumably from uveal swelling, during the first week; but this cleared without incident, unlike the longer, stormier course that had followed an iridectomy in the fellow eye years before.

None of this discussion is meant to deny that an elevated venous pressure or other factors might lead to or contribute to uveal edema in some tissues. However, the insight that a thickened sclera can produce or contribute to edema by impeding the transscleral outflow of proteinaceous fluid is a new idea. It seems a very satisfying explanation for the occurrence of choroidal edema in eyes with thick sclera, and is particularly interesting if surgery aimed at this underlying pathophysiology (by thinning or perforating the sclera) is helpful in cases that until now have been difficult to manage.

DR J. DONALD M. GASS. First of all, I would like to thank Doctor Brockhurst for discussing the paper. I will try to answer the questions. Do all patients with idiopathic uveal effusion have the same disease? If you confine the diagnosis to those patients with evidence of ciliochoroidal detachment with or without retinal

detachment and with no apparent cause, then I believe we are dealing with a relatively homogeneous group of patients who probably have the same underlying pathogenesis. Patients with bullous retinal detachment without clinical or ultrasonic evidence of ciliochoroidal detachment should not be diagnosed as idiopathic uveal effusion. I do not know whether all patients with idiopathic uveal effusion have abnormally thick sclera. All four patients that I have operated upon have had thick sclera. It is not possible to determine scleral thickness with current ultrasonographic methods.

Why do spontaneous remissions occur if the primary problem is caused by the eye being unable to rid itself of protein and sclera? It is postulated that these patients unlike normals have a limited capacity to handle extravascular protein in the uvea because of abnormalities in the sclera that apparently increase with age. Under the stress caused by a variety of ordinarily innocuous stimuli, this limited capacity is temporarily exceeded and protein and fluid accumulate first in the uvea, and secondly in the subretinal space. Once the stimuli subside, the uveal and retina reattaches and the protein slowly escapes from the eye. Most eyes will recover function spontaneously. It is only those patients with chronic detachment that require surgery.

Why do you think that this disease may be related to idiopathic central serous choroidopathy? The predominantly middle-aged male involvement, the frequency of bilateral involvement, the recurrent secondary retinal detachment, and failure to respond to corticosteroid therapy are features common to both diseases. There are, however, some important differences, but nevertheless, I believe it is worthwhile to study a group of central serous patients ultrasonographically to determine if they have any evidence of posterior choroidal detachment or edema that might implicate the sclera in the pathogenesis of that disease. Finally, I would like to thank Doctor Anderson for clarifying for you as he has done for me on many occasions the problems concerning the physiology of the eye.