

## Acute vitreous haemorrhage: a clinical report

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Acute vitreous haemorrhage presents to the patient as a sudden onset of black spots, followed by rapid clouding of vision as the blood disperses through the media. The ophthalmoscopic picture varies from a loss of retinal detail to a loss of fundus reflex, depending on the quantity of blood released.

Biomicroscopy of the vitreous is revealing because blood cells collect on the surface of the vitreous membranes and membranelles and provide a kind of *in vivo* staining of vitreous structures. Vitreous membranes appear to be impermeable to red blood cells. When detachment of the posterior hyaloid provokes a retinal haemorrhage the post-hyaloid space fills with blood. Red blood cells coat the external surface of the hyaloid but the internal surface and the vitreous gel remain clear (Fig. 1).

The retrohyaloid space is an aqueous-filled chamber and eye movements maintain red blood cells in it in diffuse suspension. If the eye movements are diminished, however, by binocular patching and the patient is maintained with his head raised, the blood cells gravitate to the bottom of the space and collect as a meniscus at the inferior vitreo-retinal insertion (Fig. 2a, b). This occurs within hours. There is a small convection current, due to the relative warmth of the retinal surface as opposed to the hyaloid membrane, but it is too small to overcome the gravitational pull.

Blood eventually enters the vitreous gel by way of holes that develop in the posterior hyaloid, usually in the days immediately following the haemorrhage. Eisner (1973) has observed these holes in cadaver eyes. They start as a tearing of the hyaloid at the edge of the disc and become round as the membrane separates from the disc and the gel prolapses through the holes (Fig. 3a, b). Blood cells migrate into the gel with eye movements and collect on the membranelles, making the pattern of prolapse visible. More than one hole may form (Fig. 4) and they may enlarge and expand all the way to the bottom of the eye (Fig. 5).

The selection of patients who will respond to binocular occlusion and positioning may be based upon the biomicroscopic examination. If the blood

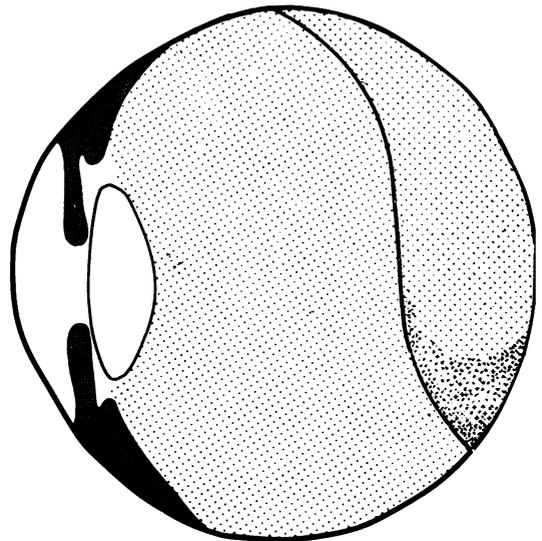


FIG. 1 *Blood diffused in retrohyaloid space after acute haemorrhage. Red blood cells coat external surface of hyaloid but internal surface and vitreous gel remain clear*

is confined behind the posterior hyaloid membrane and the vitreous gel is free of cells settling may be predicted with some certainty and a view of the retina will become available within hours. If, however, the gel is already infiltrated with cells and they are dense enough to obscure the hyaloid membrane occlusion and positioning will not result in significant clearing, for cells in the gel do not settle. If the hyaloid can be perceived at all a trial is indicated.

### Patients and methods

One hundred consecutive patients with acute vitreous haemorrhage who were treated with binocular occlusion and elevation from 1966 to 1974 at the New York Hospital were analysed. Forty-three were of rhegmatogenous origin and 57 bled from haemorrhagic vasculopathies (Table I). Eighty-two of the 100 patients were hospitalized. Eighteen diabetics, 15 of whom were recurrent bleeders, were treated as outpatients. All patients had both eyes covered. They spent most of their waking hours sitting in a chair where they were also fed. The dietetics department devised a menu of

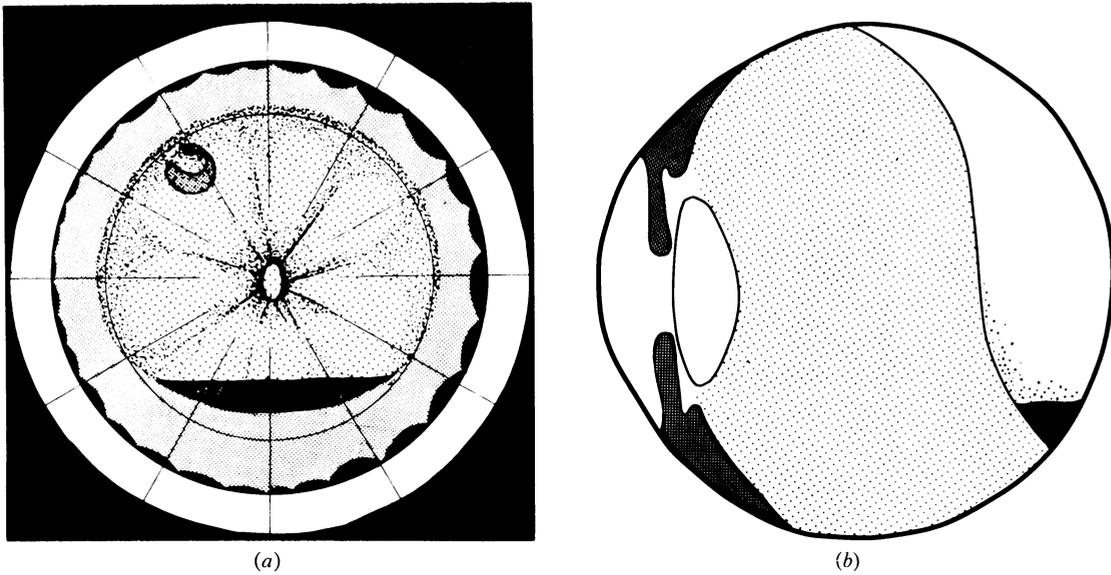


FIG. 2 *Acute retinal haemorrhage confined by hyaloid membrane to posterior hyaloid space. (a) Blood cells have settled to bottom of space in 14 hours to reveal retinal tear at 11 o'clock. (b) Sagittal section. Some cells still on external surface of hyaloid membrane and in aqueous fluid below*

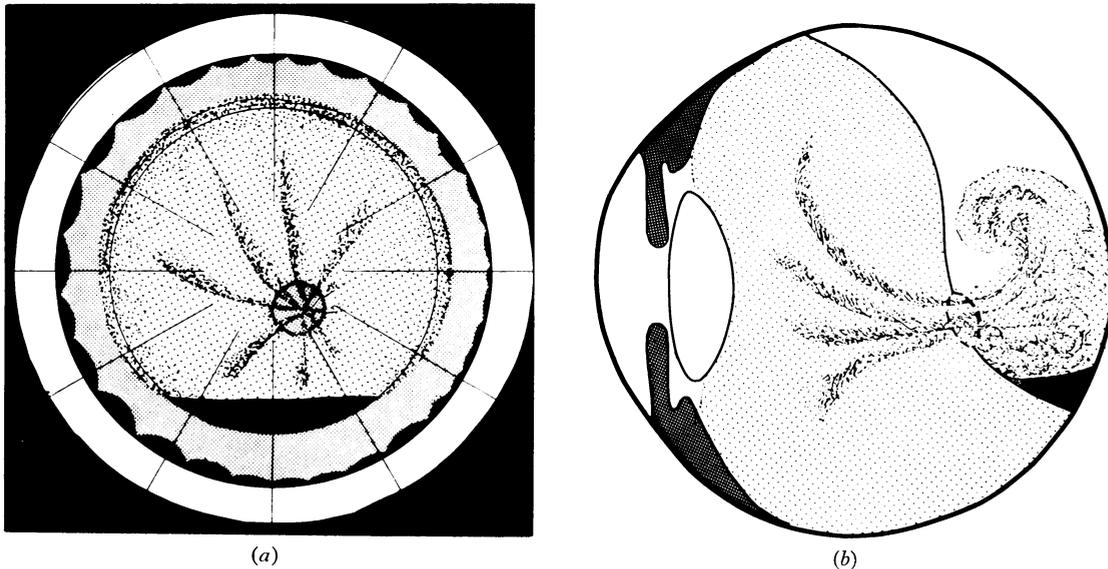


FIG. 3 *Prolapse of vitreous through hole in posterior hyaloid membrane. (a) Red cells dispersing into gel along membranellae. (b) Sagittal section shows vitreous gel in posterior hyaloid space*

finger foods consisting of sandwiches, raw vegetables and fruits, and other foods that required a minimum of aid. Sleeping with the head of the bed raised 30 degrees was found adequate to encourage and maintain pooling of the blood at the bottom of the eye. Mobilization for toilet and for walking in the corridor had no dispersing effect as long as patching was maintained. A few minutes of unpatching a day for washing and examination did

not provoke significant mixing. Pinhole glasses retarded dispersion but did not prevent it.

Inpatients were examined daily, outpatients every second or third day. Patches were maintained if there was perceptible clearing at each examination. Clearing time for the purpose of this report is defined as the day the retina could be seen well enough for diagnosis. Treatment consisted of cryopexy to retinal tears,

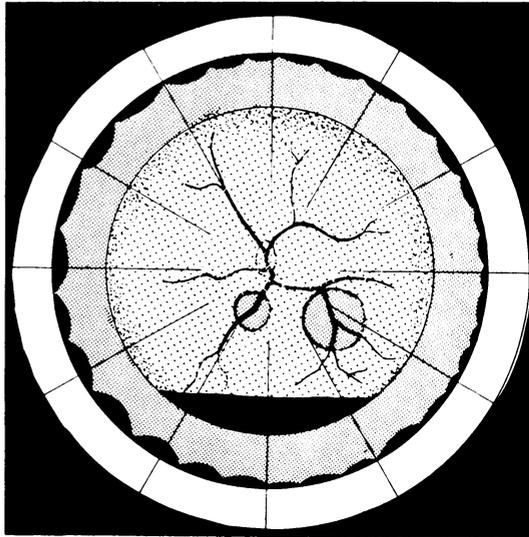


FIG. 4 Two round holes in posterior hyaloid that appeared on second day after acute vitreous detachment and haemorrhage. Blood has collected below as a result of occlusion and elevation

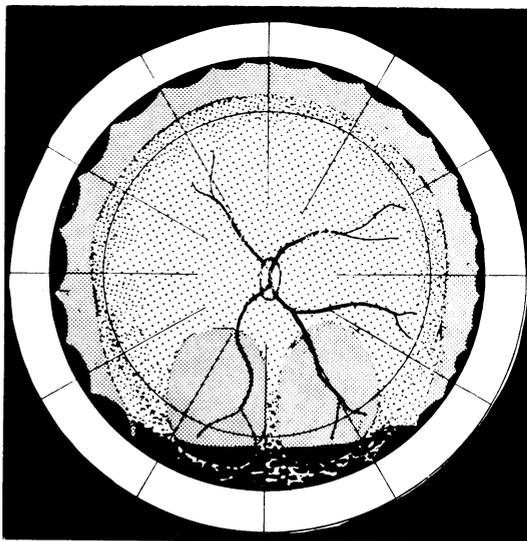


FIG. 5 Holes in posterior hyaloid have expanded to bottom of eye. Blood is still collected below but has spilled over the surface of ciliary body

scleral buckling for retinal detachment, and photo-coagulation for the vasculopathies.

## Results

### RHEGMATOGENOUS HAEMORRHAGE

In none of the 43 patients with acute vitreous haemorrhage of rhegmatogenous origin was there

evidence of a haemorrhagic vasculopathy in the second eye. These patients were treated with binocular occlusion with the expectation of finding a retinal break. Thirty-eight cleared—20 in one day, another 15 by four days, and three more by the end of a week (Table II). Acute retinal breaks were found in 25 cases, of which 16 were combined with retinal detachment. Four patients bled from bridging vessels across an operculated break that had been treated previously with cryopexy and scleral buckling. Nine patients had a posterior hyaloid detachment but no retinal break was found, and they were presumed to have bled from traction of the hyaloid on a retinal vessel.

Of the five patients in whom occlusion was abandoned because blood became diffuse in the gel two cleared over several months and were found to be without retinal breaks. Three did not clear before they showed signs of retinal detachment. One occurred in a woman aged 93 years, before the advent of ultrasound. The vitreous did not clear in the remaining four years of her life, but since she lost perception of light during this time she was presumed to have a retinal detachment. A second was a patient with a recent aphakia who reported,

**Table I** Results of ocular immobilization of 100 consecutive patients with acute vitreous haemorrhage

Aetiology	No.	Cleared	Not cleared
Rhegmatogenous	43	38	5
Bleeding vasculopathies			
Diabetes	50	45	5
Vein occlusion	5	4	1
Sickle cell	1	1	0
Leber's miliary aneurysms	1	1	0
<b>Total</b>	<b>100</b>	<b>89</b>	<b>11</b>

**Table II** Clearing time of 38 cases of rhegmatogenous vitreous haemorrhage that responded to ocular immobilization

Aetiology	No.	Clearing time (days)		
		1	2-4	5-7
Retinal tears	9	7	2	—
Tears with detachment	16	9	6	1
Bridging vessel on scleral buckle	4	2	1	1
Posterior hyaloid detachment without tear	9	2	6	1
<b>Total</b>	<b>38</b>	<b>20</b>	<b>15</b>	<b>3</b>

six weeks after his haemorrhage, a rising horizontal opacity superimposed on his vitreous density. Ultrasound confirmed the presence of a retinal detachment. A pars plana vitrectomy was done in combination with retinal detachment procedure. The retina reattached and vision recovered to 20/40. The third patient with a very dense haemorrhage had not cleared after 22 months. She had developed a defective light field and ultrasonography showed a retinal detachment. She refused surgical intervention.

#### BLEEDING VASCULOPATHIES (Table III)

##### Diabetic

Fifty patients who were treated with occlusion had bled from retinopathy of diabetic origin. All had diabetic retinopathy in the other eye. Out of the 50, 45 cleared—seven in one day, 19 by four days, and 11 by seven days. Occlusion was sustained in six patients for one to 28 days and in two patients for one to three months in order to obtain clearing of a slightly hazy gel and to prevent a larger amount of blood that had collected at the bottom of the retrohyaloid space from also entering the gel. Twenty-nine patients had a history of previous haemorrhage into the vitreous. This proved not to affect clearing time; recurrent bleeders cleared as quickly as initial ones. In diabetics the posterior hyaloid membrane seems more resistant to hole formation. Tolentino, Lee, and Schepens (1966) reported holes in 10 out of 40 diabetic eyes that bled.

##### VENOUS OCCLUSION

Five patients without haemorrhagic vasculopathy in the other eye were treated for vitreous haemorrhage with occlusion and elevation. All had hyper-

tension, but the pertinent common factor was a history of branch vein occlusion in the bleeding eye 10–22 months previously. Four of the five cleared by day five and proved to have bled from secondary neovascularization in the occluded zone.

##### Sickle-cell disease

One patient bled from the vascular malformations of sickle-cell disease and had sickle-cell vasculopathy in the other eye. The eye cleared sufficiently for photocoagulation in three days.

##### Leber's miliary aneurysms

A single patient with Leber's disease cleared in one day. There was typical vasculopathy in the other eye.

#### Discussion

Acute intraocular haemorrhage in the absence of diabetic haemorrhagic retinopathy or haemorrhagic retinopathy of other systemic origin in the other eye is most likely to be the result of a retinal tear (Lincoff, 1967; Lincoff and Kreissig, 1975). Morse, Aminlari, and Scheie (1974) collected 200 cases of spontaneous vitreous haemorrhage. Analysing these it is evident that if only the diabetics (108) are excluded 76 per cent (69 of 92) of idiopathic vitreous haemorrhages were of rhegmatogenous origin. In our series, excluding patients with diabetic vasculopathy in the other eye, 86 per cent (43 of 50) of idiopathic haemorrhages were of rhegmatogenous origin. This would seem to justify emergency management of at least this group of patients with vitreous haemorrhage and to discourage the practice of sending them first for diabetic or hypertensive treatment.

Bleeding of diabetic origin in a patient without diabetic changes in the second eye did not occur in our series. Nor did we see vitreous haemorrhage due only to hypertension. The five hypertensives in the series all bled from secondary neovascularization and had a history of retinal vein occlusion. Delay in instituting ocular immobilization and positioning of the patient increases the likelihood of holes developing in the posterior hyaloid membrane and blood entering the gel, resulting in prolonged obscuration of the fundus.

#### Summary

In the first hours after a vitreous haemorrhage dense enough to obscure the retina, the blood is usually confined to the posthyaloid space in an aqueous

**Table III** Clearing time of 51 cases of bleeding vasculopathy that responded to ocular immobilization

Aetiology	No.	Clearing time (days)				
		1	2-4	5-7	8-28	>28
Diabetes	45	7	19	11	6	2
First						
haemorrhage	(16)	(3)	(8)	(2)	(2)	(1)
Recurrent						
haemorrhage	(29)	(4)	(11)	(9)	(4)	(2)
Vein occlusion	4	1	2	1	0	0
Sickle cell	1	0	1	0	0	0
Leber's miliary aneurysms	1	1	0	0	0	0
Total	51	9	22	12	6	2

phase. Binocular occlusion and elevation provides sufficient immobilization of the eyes in nine out of 10 patients for the blood cells to settle to the bottom of the space and make the retina available

for examination and repair. Blood enters the vitreous gel through holes that develop in the posterior hyaloid membrane. Blood in the gel does not settle and requires months to clear.

### References

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