CURRENT TOPIC

Retinal haemorrhages

Baljit Kaur, David Taylor

Site and appearance of retinal haemorrhages

Retinal haemorrhages occur when there is extravasation of blood, either into the retina itself (intraretinal), between the retina and the retinal pigment epithelium (subretinal), or between the retina and the hyaloid face of the vitreous body (subhyaloid or preretinal). The shape and colour of the haemorrhages are determined by the level or levels of the retina affected, and they are often mixed. Often the hyaloid face ruptures and the haemorrhage spreads into the vitreous (intravitreal). Richman in 1936 classified retinal haemorrhages into four groups according to the morphological appearances,¹ and their location can be determined by their clinical appearance.

The ophthalmoscopic appearance of preretinal blood depends on its age and the size and shape of the blood filled space. Fresh preretinal blood usually looks like a dome shaped, semilunar, crescentic, or geographic accumulation of dark red blood overlying and obscuring its source. With time the same accumulation of blood can look semilunar or crescentic in shape, with a fluid level caused by gravitational settling of the formed elements. The plasma-erythrocyte interface may shift with changes in the position of the head. A faint line of residual blood usually marks the superior extent of the space originally filled with whole blood.

Intraretinal haemorrhages may be splinter shaped, flame shaped, or 'dot and blot' rounded haemorrhages. Splinter haemorrhages are small, linear, dull red, and lie superficially about the disc margin parallel to the major vessels, their shape being determined largely by the nerve fibre layer of the retina and the vessels. They may be single or multiple, and they clear rapidly.

Flame shaped haemorrhages are bright red, and lozenge shaped with a striated or serrated outline on at least one margin, and they follow the course of the retinal nerve fibre layer. They occur in the posterior pole of the fundus and are characteristically associated with disorders of the superficial radial peripapillary capillaries.

Dot and blot haemorrhages are dense, dark red, and sharply outlined. They are located within the compact middle layers of the retina and are seen in association with disorders that predominantly affect the deep retinal capillary system. Occasionally several discrete blot haemorrhages may coalesce and appear as an irregular geographic accumulation of blood.

Subretinal haemorrhages typically appear as irregular red plaques or opaque reddish circular lesions deep to the retina and retinal vessels (between the photoreceptors and pigment epithelial layer). On resolution they can result in a dis-

turbance of the underlying retinal pigment epithelium.

Haemorrhage beneath the retinal pigment epithelial layer is usually confined, and related to a rupture (for example, trauma or pseudoxanthoma elasticum) in Bruch's membrane, which is normally adherent to it. This results in diffuse, roughly linear haemorrhages of variable—sometimes great—extent.

Haemorrhage within the choroid expands freely and forms pool like areas the ophthalmoscopic appearance of which is governed by the density of the overlying pigmentation. Generally, choroidal haemorrhages appear as dark reddish blue areas, obviously deep in the fundus, and they usually absorb leaving a normal fundus.

Some intraretinal and preretinal haemorrhages have white centres. Such lesions (or Roth's spots) are not specific for any single underlying disorder. The white centre can be focal ischaemia, an inflammatory infiltrate, a colony of infective organisms, fibrin and platelets, or an accumulation of neoplastic cells. It is easy to mistake the bright light reflex from the internal limiting membrane over the apex of a retinal haemorrhage for the small white centre of a haemorrhage.

Intravitreal haemorrhages are relatively uncommon in children; they usually resolve slowly but completely,² but in young children may cause amblyopia warranting early vitrectomy.

Aetiology**NEONATAL RETINAL HAEMORRHAGES**

Jaeger was probably the first to have reported this condition.³ The exact mechanism of neonatal retinal haemorrhages is not clear, but it has been suggested that blood viscosity,⁴ prostaglandins in the fetal circulation,⁵ and the unequal pressure distribution during birth—the sudden release of pressure with a rapid change in intracranial pressure—may be partly responsible.⁶ Direct trauma is not implicated in most cases.

This usually benign form of haemorrhagic retinopathy usually presents as flame shaped or blot haemorrhages located in the posterior pole or periphery.⁷ This type of haemorrhage has been a subject of a monograph⁸ and the reported incidence varies enormously, from 2.6% to 50%.^{4–11} The reason for this is the variable time before fundus examination after birth, the technique of examination, and whether this is histological or by direct or indirect ophthalmoscopy. Von Barsewisch pointed out that whether the examination was done by an experienced or inexperienced ophthalmologist or a paediatrician or other, significantly influenced the results⁸; the method of

delivery of the baby is also important. Retinal haemorrhages occur in about one third of babies born by occipital presentation, increasing in incidence with prolonged labour and obstetric procedures,¹² and decreasing in breech presentations and birth by caesarean section.⁸ Retinal haemorrhages are more common in babies of elderly primiparas,⁷ and mothers with toxæmia.¹³

Retinal haemorrhages in newborn infants usually resolve within a few days.⁴ Follow up studies have reported normal visual development.^{14 15}

SUBARACHNOID HAEMORRHAGE

When bleeding from any cause occurs into the subarachnoid space there may be increased intracranial pressure, optic nerve sheath haemorrhage, and an increase in the pressure within the optic nerve sheath because of raised central retinal venous pressure and obstruction of the retinochoroidal anastomosis¹⁶; to this may be added raised central venous pressure as a result of epileptic seizures.¹⁷ The incidence of retinal haemorrhages in subarachnoid haemorrhage is between 20%¹⁸ and 32%,¹⁹ and they usually occur simultaneously or are noted within a few days.²⁰ Streak and preretinal haemorrhages occur mainly around the optic disc and often accompany papilloedema. These haemorrhages often resolve spontaneously.²¹

Occasionally large preretinal haemorrhages may break into the vitreous (Terson's syndrome). This is often a delayed phenomenon. The pathogenesis of haemorrhages in Terson's syndrome is controversial. One view is that blood from the subarachnoid space is forced into the optic nerve sheath and penetrates the lamina cribrosa to appear within the globe.^{22 23} Intraocular and optic nerve sheath haemorrhages have, however, been reported in cases of sudden intracranial hypertension in which there had been no intracranial bleeding.²⁴ Other authors^{16 18 25} believe that intraocular haemorrhage is the result of rupture of retinal veins secondary to retinal venous hypertension, which in turn is the result of obstruction of both the central retinal vein and the retinochoroidal anastomosis.^{16 18 25} For this to occur the intracranial pressure must rise much higher than it does to cause optic nerve sheath haemorrhage alone.²¹

In subdural haemorrhage the rise in intracranial pressure is less acute; they are usually post-traumatic,²⁶ but acute subdural haematoma has been described in association with Purtscher's retinopathy (which is normally associated with thoracic compression) in an infant with non-accidental injury,²⁷ and in infants with pre-existing asymptomatic chronic subdural haematoma or subdural effusion with occult craniocerebral disproportion.²⁸ Retinal and preretinal haemorrhages are consistently present in all infants with acute subdural haematoma.²⁹

RAISED CENTRAL VENOUS PRESSURE

Raised central venous pressure from crying, seizures, or squeezing of the chest, by any mechanism where there is forced contracture of the chest against a closed or partially closed glottis,³⁰ may cause the so called Valsalva's haemorrhagic retinopathy,³¹ which is usually associated with superficial retinal haemorrhages. Haemorrhages into all layers of the retina may be more common in non-accidental injury, at least in pathological studies.^{30 32}

Retinal haemorrhages from crush injuries to the chest are usually accompanied by retinal infarcts ('cotton wool spots') and exudates in the so called Purtscher's retinopathy,³³ the mechanisms for which may be air embolism,³⁴ an acute rise in intraocular venous pressure, or hypoxia from angiospasm of the retina.³³

The sudden rise in intrathoracic pressure as a result of forceful contraction of the thoracoabdominal muscles against a closed glottis causes an acute rise in central venous pressure, which in turn increases the intracranial pressure as the intracranial venous sinuses are in direct communication with the superior vena cava and there are no intervening valves in the large veins above the heart. This acute raised central venous pressure occurs with the Valsalva manoeuvre,³¹ in cases of trauma when there is forced compression of the chest (usually anteroposteriorly) against a closed glottis,³⁵ and also in epileptic seizures,^{36 37} vomiting, or spells of coughing.³⁷

Appreciable rises in cerebral venous pressure (10–20 cm H₂O) have been reported³⁸ in neonates during episodes of crying, abdominal straining, and convulsions.³⁸ Values of cerebral venous pressure above 100 cm H₂O have been shown experimentally during a tonic seizure.³⁹ The increase in muscle tone and intrathoracic pressure makes an important contribution to this. These transient rises in cerebral venous pressure are probably responsible for the small cerebral haemorrhages seen after experimental seizures.⁴⁰ Retinal haemorrhages may occur under these circumstances but only rarely do they have more than transient effect.^{37 41} The coincident rise in intraocular pressure at such times reduces the pressure difference across the vessel wall and may give a protective effect against more profuse bleeding.³⁷

This mechanism could explain the occurrence of retinal haemorrhages after clumsy attempted resuscitation of infants by inexperienced adults.

NON-ACCIDENTAL INJURY

Ocular complications of non-accidental injury were first described in 1964.⁴² The most common finding is retinal haemorrhage,^{43 44} which occur in up to 90% of such children.⁴⁵ These haemorrhages can last for several years, in contrast to those seen at birth.⁴³ The cause cannot be determined by fundal examination alone or by the morphology of the retinal haemorrhages.

In non-accidental injury there are several mechanisms that cause the haemorrhage.

Raised intraocular venous pressure

This occurs by two mechanisms, often occurring together. Firstly, raised intracranial pressure is transmitted to the subarachnoid space, either through clear cerebrospinal fluid or by haemorrhage.³⁰ The raised optic nerve sheath pressure compresses the central retinal vein, and the chorioretinal anastomosis thus raising the pressure upstream to the capillary bed.¹⁶

In non-accidental injury the intraocular haemorrhages are often accompanied by intracranial haemorrhages with associated damage.⁴⁶ The severity of intraocular haemorrhage correlates with the severity of acute neurological injury.⁴⁷ Diffuse fundal impairment, vitreous haemorrhage, or large subhyaloid haemorrhages are associated with more severe acute neurological injury.⁴⁷

The second mechanism is raised central venous pressure, as already discussed.

Rapid deceleration or cycles of rapid acceleration and deceleration

Ober and Lyle *et al* have implied that the shear forces generated by acceleration or deceleration, or both, have been a factor in the production of haemorrhages.³²⁻⁴⁸ Duhami *et al*, however, have reported that the shaken baby syndrome—at least in its most severe form—is not usually caused by non-impact acceleration-deceleration forces alone.⁴⁹ The tendency for more severe retinopathy to occur in infants than in older children could be explained by clinical and experimental evidence that suggests that intraocular haemorrhages may be caused by abrupt increase in retinal venous pressure secondary to an acute rapid rise in intracranial pressure.¹⁶⁻³⁰ Infants weigh less, have a greater head size in relation to body size, and have less developed neck musculature to support the head during shaking of the thorax. These factors may contribute to the susceptibility of infants to acceleration-deceleration forces and more severe increases in retinal venous pressure.⁵⁰ Commonly, however, there are no external signs of head injury, but bilateral infantile subdural haematomas and bilateral retinal haemorrhages are present.⁴⁶ The latter are seen particularly in the posterior interhemispheric fissure, presumably as a result of tearing of the immature and fragile bridging veins. In shaken infants, retinal haemorrhages typically extend through all the layers of the retina and into the subretinal space. In Purtscher's and haemorrhagic Valsalva's retinopathy, exudates and superficial haemorrhages, respectively, are seen.³⁰

BLOOD DYSCRASIAS

Blood dyscrasias with anaemia, pancytopenia, and coagulopathy may give rise to retinal haemorrhages—for example, in leukaemia and sickle cell disease.

Retinal haemorrhages occur in both acute (more common)⁵¹ and chronic leukaemia, but their presence does not necessarily imply a grave prognosis.⁵² The retinopathy is characterised by multiple intraretinal haemorrhages, subhyaloid haemorrhages, Roth's spots, exudates, and abnormalities of retinal vasculature.

In sickle cell disease retinal haemorrhages are the result of extravasation of blood through the damaged arteriolar walls after transient arteriolar occlusions and subsequent reperfusion.⁵³ Preretinal or intraretinal haemorrhages may evolve into retinal schisis cavities.

HYPOTENSION AND ANOXIA

Sudden reduction of systemic blood pressure because of cardiac decompensation, or traumatic shock, or treatment for arterial hypertension⁵⁴ may cause retinal and optic nerve ischaemia and haemorrhages. Sudden surgical lowering of intraocular pressure (for example, in glaucoma filtering surgery) in the presence of a healthy retinal vessel seldom produces retinal bleeding, but anoxia together with increased transmural pressure is likely to produce bleeding—for example, after difficult breech deliveries or where the cord is strangulating the neck at birth.⁵⁵

INFECTIONS

Viral infections

In cytomegalovirus infection the retinal haemorrhages are usually flame shaped, and occur after the white lesions that look similar to 'cotton wool spots' and retinal necrosis; they may resemble retinal branch vein occlusions. The infective process spreads slowly along the course of the retinal blood vessels to encompass the entire fundus. Retinal haemorrhages are seen in acquired cytomegalovirus retinitis after cytotoxic and corticosteroid treatment in recipients of renal grafts and in patients with neoplastic disease.⁵⁶

Other infections

Rickettsial infections,⁵⁷ subacute bacterial endocarditis, and herpes simplex are other known causes of retinal haemorrhages.

FAMILIAL RETINAL ARTERIOLAR TORTUOSITY

The progressive autosomal dominant inherited syndrome of retinal arteriolar tortuosity may be complicated by spontaneous retinal haemorrhages.⁵⁸ The retinal arteriolar tortuosity is often subtly increased during adolescence and affects small retinal arterioles, mainly in the macular area. It is easily overlooked, but even when haemorrhages affect the fovea spontaneous clearing with recovery of normal vision is the rule.

MISCELLANEOUS CAUSES AND PREDISPOSING FACTORS

Retinal diseases

Coats' disease (telangiectasis of retinal vessels), Eale's disease (retinal periphlebitis), radiation retinopathy, juvenile diabetes mellitus, hypertension, and high myopia predispose to retinal or vitreous haemorrhage.

Blood hyperviscosity

Retinal haemorrhages occur in patients with hyperviscosity of the blood such as in cystic fibrosis,⁵⁹ macroglobulinaemia,⁶⁰ cryoglobulinaemia,⁶¹ and paraproteinaemia.⁶¹ The mechanism by which these scattered retinal haemorrhages develop is not clear. In cystic fibrosis it has been found in patients with moderate to severe pulmonary disease.⁵⁹

Carotid-cavernous fistula

Especially when traumatic in origin, this is considerably less common in infancy than adults.⁶² The fistula produces haemodynamic changes resulting in lowered arterial pressure and raised venous pressure in the eye and orbit. The resultant reduction in perfusion pressure for a long time, with ischaemia, results in widespread intraretinal and superficial haemorrhages.⁶³

Other predisposing factors

These include drugs (for example, aspirin and anticoagulants) and hypercapnia.

Conclusion

There are many causes of retinal haemorrhages in infancy, and there are several causative mechan-

isms. The presence, site, or physical appearance of the haemorrhages are not pathognomonic, and although their association with other clinical features could help with the differential diagnosis it is important—especially in suspected non-accidental injury—to avoid attributing causation to the presence of the haemorrhages alone.

We thank Miss Angela Tank for typing the manuscript.

- Richman F. Retinal haemorrhages in the newborn. *Proceedings of the Royal Society of Medicine* 1936;30:277–80.
- Augsburger JJ, Goldberg RE, Magargal LE. Retinal and choroidal vascular abnormalities. In: Harley RD, ed. *Pediatric ophthalmology*. 2nd Ed. Philadelphia: WB Saunders, 1983:664–5.
- Jaeger E. *Ueber die Einstellung des Dioptrischen Apparates im menschlichen Auge*. Giles CL, ed. Vienna: LW Seidel, 1960. (Originally published 1861.)
- Baum JD, Bulpitt CJ. Retinal and conjunctival haemorrhage in the newborn. *Arch Dis Child* 1970;45:344–9.
- Schoenfeld A, Buckman G, Nissenkorn I, Cohen S, Ben-Sira I, Ovadia J. Retinal hemorrhages in the newborn following labour induced by oxytocin or dinoprostone. *Arch Ophthalmol* 1985;103:932–4.
- Critchley ER. Observations on retinal haemorrhages in the newborn. *J Neurol Neurosurg Psychiatry* 1968;31:259–62.
- Sezen F. Retinal haemorrhages in newborn infants. *Br J Ophthalmol* 1971;55:248–53.
- Von Barsewisch B. *Perinatal retinal haemorrhages: morphology, aetiology, and significance*. Berlin: Springer Verlag, 1979: 1–184.
- Besio R, Caballero C, Meerhoff E, Schwarcz R. Neonatal retinal haemorrhages in newborn infants. *Br J Ophthalmol* 1979;87:74–6.
- Chase RR, Merritt KK, Bellows M. Ocular findings in the newborn infant. *Arch Ophthalmol* 1950;44:236–42.
- McKeown H. Retinal hemorrhages in the newborn. *Arch Ophthalmol* 1941;26:25–37.
- Kauffman ML. Retinal hemorrhages in the newborn. *Am J Ophthalmol* 1958;46:658–60.
- Sihota R, Bose S, Paul AH. The neonatal fundus in maternal toxemia. *J Pediatr Ophthalmol Strabismus* 1989;26:281–4.
- Jacobs MW. The frequency in the newborn of intraocular hemorrhages and their relationship to intracranial trauma. *Transactions of the American Academy of Ophthalmology* 1928;33:218–26.
- Rowland WD. Ocular pathology of the newborn; an end-results study. *Am J Ophthalmol* 1935;18:647–50.
- Muller PJ, Deck JHN. Intraocular and optic nerve sheath hemorrhage in cases of sudden intracranial hypertension. *J Neurosurg* 1974;41:160–6.
- Alexander EG. A case of stasis cyanosis following an epileptic seizure, simulating traumatic asphyxia. *Ann Surg* 1909;49: 762–6.
- Manschott WA. The fundus oculi in subarachnoid haemorrhage. *Acta Ophthalmol* 1944;22:281–99.
- Fahny JA. Symptoms and signs of intracranial aneurysm. *Acta Ophthalmol* 1972;50:129–36.
- Manschott WA. Subarachnoid hemorrhage: intraocular symptoms and their pathogenesis. *Am J Ophthalmol* 1954; 38:501–5.
- Vanderlinden RG, Chisholm LD. Vitreous haemorrhages and sudden increased intracranial pressure. *J Neurosurg* 1974; 41:167–76.
- Paton L. VII. Diseases of the nervous system: ocular symptoms in subarachnoid haemorrhage. *Transactions of the Ophthalmological Society of the United Kingdom* 1924;44: 110–26.
- Symonds CP. Spontaneous subarachnoid hemorrhage. *J Med* 1924;18:93–122.
- Weaver RG, Davies CH. Subarachnoid hemorrhage. *Am J Ophthalmol* 1961;52:257–9.
- Smith DC, Kearns TP, Sayre GP. Preretinal and optic nerve sheath haemorrhage: pathologic and experimental aspects in subarachnoid hemorrhage. *Transactions of the American Academy of Ophthalmology and Otolaryngology* 1957;61: 201–11.
- Newton RW. Intracranial haemorrhage and non-accidental injury. *Arch Dis Child* 1989;64:188–90.
- Tomasi LG, Rosman NP. Purtscher retinopathy in the battered child syndrome. *Am J Dis Child* 1975;129:1335–7.
- Ikedo A, Sato O, Tsugane R, Shibuya N, Yamamoto I, Shimoda M. Infantile acute subdural hematoma. *Childs Nerv Syst* 1987;3:19–22.
- Aoki N, Masuzawa H. Infantile acute subdural hematoma. Clinical analysis of 26 cases. *J Neurosurg* 1984;61:273–80.
- Lambert SR, Johnson TE, Hoyt CS. Optic nerve sheath and retinal hemorrhages associated with the shaken baby syndrome. *Arch Ophthalmol* 1986;104:1509–12.
- Duane TD. Valsalva hemorrhagic retinopathy. *Am J Ophthalmol* 1973;75:637–42.
- Ober RR. Hemorrhagic retinopathy in infancy: a clinicopathologic report. *J Pediatr Ophthalmol Strabismus* 1980; 17:17–20.
- Marr WG, Marr EG. Some observations of Purtscher's disease: traumatic retinal angiopathy. *Am J Ophthalmol* 1962;54:693–705.
- Burton TC. Unilateral Purtscher's retinopathy. *Ophthalmology* 1980;87:1096–105.
- Fred HL, Chandler FW. Traumatic asphyxia. *Am J Med* 1960;29:508–17.
- Meldrum BS. Pathophysiology. In: Rickens LJ, ed. *A textbook of epilepsy*. New York: Churchill Livingstone, 1982:456–87.
- Henkind P, Wise GN, Dollery CT. Clinical and microscopic features of retinal lesions. *The retinal circulation*. Philadelphia: Harper and Row, 1971:167–78.
- Vert P, Monin P, Sibout M. Intracranial venous pressure in newborns: variations in physiologic state and in neurologic and respiratory disorders. In: Stern L, Fris-Hansen B, Kildeberg P, eds. New York: Masson, 1975:185–96.
- Hendley CD, Spudis EV, De La Torre E. Intracranial pressure electroshock convulsions in the dog. *Neurology* 1965; 15:351–60.
- Krushinsky LV. A study of pathophysiological mechanism of cerebral hemorrhage provoked by reflex epileptic seizures in rats. *Epilepsia* 1962;3:363–80.
- Kanter RK. Retinal hemorrhage after cardiopulmonary resuscitation or child abuse. *Pediatrics* 1986;108:430–2.
- Kiffney GT. The eye of the 'battered child'. *Arch Ophthalmol* 1964;72:231–3.
- Jensen AD, Smith RE, Olson MI. Ocular clues to child abuse. *J Pediatr Ophthalmol Strabismus* 1971;8:270–2.
- Friendly DS. Ocular manifestations of physical child abuse. *Transactions of the American Academy of Ophthalmology and Otolaryngology* 1971;75:318–32.
- Aron JJ, Marx P, Black FM, et al. Ocular signs observed in the syndrome of Silverman. *Annales Oculistes* 1970;208: 533–46.
- Caffey J. On the theory and practice of shaking infants: its potential residual effects of permanent brain damage and mental retardation. *Am J Dis Child* 1972;124:161–9.
- Wilkinson WS, Han DP, Rappley MD, Owings CL. Retinal hemorrhages predict neurological injury in the shaken baby syndrome. *Arch Ophthalmol* 1989;107:1472–4.
- Lyle DJ, Stapp JP, Button RR. Ophthalmologic hydrostatic pressure syndrome. *Am J Ophthalmol* 1957;44:652–6.
- Duhaimi A, Gennarelli TA, Thibault LE, Bruce DA, Margulies SS, Wiser R. The shaken baby syndrome. A clinical, pathological, and biomechanical study. *J Neurosurg* 1987;66:409–15.
- Caffey J. The whiplash shaken infant syndrome: manual shaking by the extremities with whiplash-induced intracranial and intraocular bleedings, linked with residual permanent brain damage and mental retardation. *Pediatrics* 1974;54:396–403.
- Rosenthal AR. Ocular manifestations of leukaemia. A review. *Ophthalmology* 1983;90:899–905.
- Jolt JM, Gordon-Smith ED. Retinal abnormalities in diseases of the blood. *Br J Ophthalmol* 1969;53:145–60.
- Talbot JF, Bird AC, Mande GH, Acheson RW, Moriarty BJ, Serjeant GR. Sickle cell retinopathy in Jamaican children: further observations from a cohort study. *Br J Ophthalmol* 1988;72:727–32.
- Pears MA, Pickering GW. Changes in fundus oculi after hemorrhages. *Q J Med* 1960;29:153–78.
- Naunton WJ, Forrester RM. Severe vitreous haemorrhage in a premature infant. *Br J Ophthalmol* 1955;39:563–5.
- Berger BB, Weiberg RS, Tessler HH, Wyhinny J, Vygantas CM. Bilateral cytomegalovirus panuveitis after high-dose corticosteroid therapy. *Am J Ophthalmol* 1979;88:1020–5.
- Giraud P. Altérations vasculaires rétinienne d'origine rickettsienne. *Bulletins et Memoires de la Societe Francaise d'Ophthalmologie* 1959;72:621–34.
- Wells CG, Kahna RE. Progressive inherited retinal arteriolar tortuosity with spontaneous retinal hemorrhages. *Ophthalmology* 1985;92:1015–24.
- Rimsza ME, Menried LS, Kaplan AM. Hemorrhagic retinopathy in a patient with cystic fibrosis. *Pediatrics* 1978;62: 336–8.
- Orellana J, Friedman AH. Ocular manifestations of multiple myeloma, Waldenstrom's gammopathy. *Surv Ophthalmol* 1981;26:157–69.
- Carr RE, Henkind P. Retinal findings associated with serum hyperviscosity. *Am J Ophthalmol* 1963;56:23–31.
- Carmel PW. Head injury. In: Kelly VC, ed. *Practice of pediatrics*. Philadelphia: Harper and Row, 1987:9–18.
- Sanders MD, Hoyt WF. Hypoxic ocular sequelae of carotid-cavernous fistulae. Study of the causes of visual failure before and after neurosurgical treatment in a series of 25 cases. *Br J Ophthalmol* 1969;53:82–97.