

## REFERENCES

- 1 **Resnikoff S**, Pascolini D, Etya'ale D, *et al*. Global data on visual impairment in the year 2002. *Bull World Health Organ* 2004;**82**:844–51.
- 2 **World Health Organization**. *International statistical classification of diseases, injuries and causes of death, tenth revision*. Geneva: WHO, 1993.
- 3 **McCarty CA**. Uncorrected refractive error. *Br J Ophthalmol* 2006;**90**:521–2.
- 4 **Taylor HR**, Livingston PM, Stanislavsky YL, *et al*. Visual impairment in Australia: distance visual acuity, near vision and visual field findings of the Melbourne Visual Impairment Unit. *Am J Ophthalmol* 1997;**123**:328–37.
- 5 **Dandona L**, Dandona R, Srinivas M, *et al*. Blindness in the Indian state of Andhra Pradesh. *Invest Ophthalmol Vis Sci* 2001;**42**:908–16.
- 6 **Jadoon MZ**, Dineen B, Bourne RR, *et al*. Prevalence of blindness and visual impairment in Pakistan: the Pakistan National Blindness and Visual Impairment Survey. *Invest Ophthalmol Vis Sci* 2006;**47**:4749–55.
- 7 **Ramke J**, du Toit R, Palagyi A, *et al*. Correction of refractive error and presbyopia in Timor-Leste. *Br J Ophthalmol* 2007;**91**:860–6.
- 8 **Bourne RRA**, Dineen BP, Noorul Huq DM, *et al*. Correction of refractive error in the adult population of Bangladesh: meeting the unmet need. *Invest Ophthalmol Vis Sci* 2004;**45**:410–17.
- 9 **Burke AG**, Patel I, Munoz B, *et al*. Population-based study of presbyopia in rural Tanzania. *Ophthalmology* 2006;**113**:723–7.
- 10 **Nwosu SN**. Ocular problems of young adults in rural Nigeria. *Int Ophthalmol* 1998;**22**:259–63.
- 11 **Nirmalan PK**, Krishnaiah S, Shamanna BR, *et al*. A population-based assessment of presbyopia in the state of Andhra Pradesh, South India: the Andhra Pradesh Eye Disease Study. *Invest Ophthalmol Vis Sci* 2006;**47**:2324–8.
- 12 **Patel I**, Munoz B, Burke AG, *et al*. Impact of presbyopia on quality of life in a rural African setting. *Ophthalmology* 2006;**113**:728–34.
- 13 **Limburg H**, Kumar R, Indrayan A, *et al*. Rapid assessment of prevalence of cataract blindness at district level. *Int J Epidemiol* 1997;**26**:1049–54.
- 14 **Dandona R**, Dandona L, Kovai V, *et al*. Population-based study of spectacles use in Southern India. *Indian J Ophthalmol* 2002;**50**:145–55.
- 15 **Dandona R**, Dandona L. Refractive error blindness. *Bull World Health Organ* 2001;**79**:237–43.

Subhyaloidal and macular haemorrhage

## Subhyaloidal and macular haemorrhage: localisation and treatment strategies

Stefan Mennel

### Deterioration of visual acuity as a result of haemorrhage

Haemorrhage at the macula causes deterioration of visual acuity within seconds or minutes. Biomicroscopy reveals a dome-shaped acute bleeding in the macular area, but the precise localisation of the blood—that is, subhyaloidal or macular—is mostly unknown. In this issue of the *British Journal of Ophthalmology*, De Maeyer *et al* (see page 869)<sup>1</sup> identify the sub-internal limiting membrane (ILM) cleavage plane as the site of haemorrhage in their patients, and present vitrectomy as an excellent treatment option for this pathology.

Different primary causes of subhyaloidal or macular haemorrhage have been stated, the most common being Valsalva retinopathy and Terson syndrome. In addition, such haemorrhages may occur secondary to vascular diseases such as arteriosclerosis, hypertension, retinal artery or vein occlusion, diabetic retinopathy, retinal macroaneurysm, chorioretinitis, blood disorders as well as shaken baby syndrome, age-related macular degeneration, and can also occur spontaneously or as a result of trauma.<sup>2–9</sup>

In previous studies, the sharply demarcated, dome-shaped haemorrhage has been assumed to be in the subhyaloidal space, anterior to the ILM.<sup>6 10 11</sup> Although some authors identified a sub-ILM haemorrhage by glistening reflexes and surface striae,<sup>12 13</sup> others disputed the reliability of biomicroscopy in locating the plane of haemorrhage.<sup>2 10 14–18</sup> A definitive

sub-ILM haemorrhage had been demonstrated in selected cases, where the cleavage plane could be identified by ophthalmoscopy, because of the presence of previously detached vitreous at the area of the sub-ILM haemorrhage,<sup>3 10 12 19–21</sup> by echography,<sup>22</sup> by optical coherence tomography (OCT)<sup>15 16 23</sup> or by histological analyses of the surgically removed anterior wall of the haemorrhage.<sup>2 4 12 14 19 24–28</sup>

Premacular or preretinal haemorrhage and subhyaloidal haemorrhage were the commonly used synonyms for subhyaloidal and sub-ILM haemorrhages, although these terms are anatomically correct only if the haemorrhage is located anterior to the ILM. As the ILM represents the basement membrane of the mueller cells, a haemorrhage beneath the ILM is located within the neuroretina and the anatomically correct description would be macular or sub-ILM haemorrhage. Subhyaloidal haemorrhage has also been described as hyphema posterior,<sup>22</sup> whereas the terms submembranous haemorrhage, haemorrhagic detachment of the ILM<sup>29</sup> or “haemorrhagic macular cyst”<sup>19</sup> have been used for sub-ILM haemorrhage. However, because “cyst” describes a cavity lined by epithelium or endothelium, Schubert recommended the use of an established term, macular haematoma.<sup>30</sup>

The lack of a definitive biomicroscopic characteristic to differentiate subhyaloidal and macular haemorrhages clinically,

which may be important for treatment decisions in the future, emphasises the need to develop additional diagnostic techniques. In selected cases, OCT may be helpful. In general, an OCT scan through the centre of a haemorrhage at the macula does not illustrate whether the location is subhyaloidal or sub-ILM. Moreover, it does not allow differentiation between subhyaloidal and subretinal haemorrhage, because the haemorrhage severely attenuates the underlying structures.<sup>31</sup> Shukla *et al*<sup>23</sup> presented a technique to increase the effectivity of OCT by taking OCT scans just above the level of the sedimented blood. In a case of a partial detached vitreous and sub-ILM location of the haemorrhage, these scans displayed two distinct membranes; a single highly reflective band corresponding to the ILM, and an overlying patchy membrane with low optical reflectivity consistent with the posterior hyaloid. Meyer and colleagues<sup>15 16</sup> performed a selective A-scan analysis and identified numerous hyper-reflective spikes, of which a highly reflective band representing the anterior wall of the previous haemorrhage corresponded to the ILM.

Although treatment choices must consider the underlying disease, in clinical practice, the primary aim of treatment is removal of the haemorrhage.

Spontaneous reabsorption of the haemorrhage may occur, but this could take 1–2 months,<sup>7 13 21 25 26</sup> during which time the persistence of blood may irreversibly damage the retina and cause permanent visual loss as a result of the formation of preretinal tractional membrane and proliferative vitreoretinopathy.<sup>3 32</sup> The toxic effects of longstanding haemorrhage are even more destructive in macular than in subhyaloidal haemorrhage,<sup>3 26</sup> and haemorrhage beneath the ILM tends to remain longer than subhyaloidal haemorrhage.<sup>3 33</sup> Observation for up to 3 months for spontaneous clearing of haemorrhage is a clinically accepted practice,<sup>4 25</sup> but others advocate early surgery even for

these cases, as a prolonged persistence of haemorrhage may cause irreversible retinal damage.<sup>34</sup>

Laser drainage, introduced in 1973 by Heydenreich<sup>7</sup> and Fechner,<sup>35</sup> gives the entrapped blood a focal opening into the vitreous cavity to accelerate clearing and visual improvement.<sup>3-8 11 35 36</sup> Synonyms are laser membranotomy and laser puncturing. Kroll and Busse<sup>5</sup> recommend this treatment within the first 3–4 days after the occurrence of haemorrhage. Serious complications of this procedure have rarely been reported (macular hole formation and retinal detachment). The formation of epimacular membranes is more common,<sup>14–16</sup> probably because of growth factors that are supposed to stimulate proliferation of entrapped cells along the ILM and retinal surface.<sup>2</sup> It has been assumed that this occurs mainly in cases of sub-ILM haemorrhage, in which laser drainage requires disruption of the basal lamina of the sensory retina, with a consequent gliotic wound-healing response.<sup>2 8 14 23 33 34</sup> Surgical removal of epiretinal membrane secondary to sub-ILM haemorrhage verified the ILM with adherent clumps of pigmented macrophages containing intracellular iron deposition, hemosiderin deposits and nuclei of a fine glial epiretinal membrane.<sup>14</sup>

Recombinant tissue plasminogen activator and gas are routinely used in the treatment of submacular haemorrhage secondary to age-related macular degeneration.<sup>37 38</sup> The same technique, resulting in separation of the vitreous and the promotion of the distribution of blood, was used successfully to treat subhyaloidal or macular haemorrhage.<sup>39 40</sup>

Vitreotomy allows the immediate removal of the haemorrhage and analysis of the surgically removed anterior wall of the haemorrhage cavity, as well as definitive location of the haemorrhage.<sup>2 14 24 27</sup> De Maeyer *et al*<sup>1</sup> treated five patients by vitrectomy after insufficient spontaneous visual recovery, and identified the sub-ILM location of the haemorrhage intraoperatively in all patients by ILM biostaining.<sup>41 42</sup> Excellent visual recovery occurred in all patients without any procedure-related complications. Timely surgical removal of the vitreous haemorrhage has the advantage of significant and immediate improvement of vision, and may also prevent complications of longstanding haemorrhage. However, vitrectomy, despite being a routine procedure, also has numerous risks and side effects. Formation of a nuclear sclerotic cataract is a well-known and relatively common complication, especially in patients aged >50 years.<sup>43–46</sup> Intraoperative retinal breaks and postoperative proliferative vitreoretinopathy

may result in retinal detachment or endophthalmitis, which may cause severe loss of visual function.

In summary, several successful methods have been established for the treatment of haemorrhage at the macula, and concurrent progress in scientific research has distinguished between subhyaloidal and macular haemorrhages, at least in most cases. This may be an important factor in therapeutic decisionmaking.

Despite this excellent scientific progress, however, fundamental questions still remain. Whereas laser drainage has generally yielded very good functional results, with the potential risk of secondary membrane formation reported mainly in cases with sub-ILM haemorrhage, De Maeyer *et al*<sup>1</sup> presented excellent visual recovery using vitrectomy in similar cases. Therefore, the suitability of laser drainage for subhyaloidal haemorrhages and vitrectomy for the sub-ILM location deserves study. In addition, the underlying disease, patient's age, duration since onset of haemorrhage and the size of the haemorrhage are highly relevant factors affecting decisions regarding awaiting spontaneous resorption or administering treatment, as well as regarding the type of treatment.

Furthermore, new therapeutic strategies introducing drugs to induce syneresis and sychysis of the vitreous may permit recovery of vision and rehabilitation of the patient by minimally invasive treatments, which also minimise possible side effects.

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REFERENCES

- 1 De Maeyer K, Van Ginderdeuren R, Postelmans L, *et al*. Sub inner limiting membrane hemorrhage: causes and treatment with vitrectomy. *Br J Ophthalmol* 2007;**x**:xxx.
- 2 Russell SR, Hageman GS. Hemorrhagic detachment of the internal limiting membrane after penetrating ocular injury. *Retina* 1992;**12**:346–50.
- 3 Iijima H, Satoh S, Tsukahara S. Nd: YAG laser photodisruption for preretinal hemorrhage due to retinal macroaneurysm. *Retina* 1998;**18**:430–4.
- 4 Meier P, Schmitz F, Wiedemann P. Vitrectomy for pre-macular hemorrhagic cyst in children and young adults. *Graefes Arch Clin Exp Ophthalmol* 2005;**243**:824–8.
- 5 Kroll P, Busse H. Therapy of preretinal macular hemorrhages. *Klin Monatsbl Augenheilkd* 1986;**188**:610–12.
- 6 Kroll P, Le Mer Y. Treatment of preretinal retrolental hemorrhage: value of early argon laser photocoagulation. *J Fr Ophthalmol* 1989;**12**:61–6.

- 7 Heydenreich A. Treatment of preretinal haemorrhages by means of photocoagulation. *Klin Monatsbl Augenheilkd* 1973;**163**:671–6.
- 8 Ulbig MW, Mangouritas G, Rothbacher HH, *et al*. Long-term results after drainage of premacular subhyaloid hemorrhage into the vitreous with a pulsed Nd:YAG laser. *Arch Ophthalmol* 1998;**116**:1465–9.
- 9 Kaynak S, Eryildirim A, Kaynak T, *et al*. Nd: YAG laser posterior hyaloidotomy in subhyaloid hemorrhage. *Ophthalmic Surg* 1994;**25**:474–6.
- 10 Spraul CW, Grossniklaus HE. Vitreous hemorrhage. *Surv Ophthalmol* 1997;**42**:3–39.
- 11 Schmidt JC, Nietgen GW. Argon laser treatment of subhyaloidal hemorrhage. *Lasermedizin*, 1998/99, **14**:94–8.
- 12 Gass JDM. Traumatic retinopathy. In: *Stereoscopic atlas of macular diseases: diagnosis and treatment*. St Louis: Mosby, 1997:737–74.
- 13 Keilhahn MA, Bennett SR, Cameron D, *et al*. Retinal folds in Terson syndrome. *Ophthalmology* 1993;**100**:1187–90.
- 14 Kwok AK, Lai TY, Chan NR. Epiretinal membrane formation with internal limiting membrane wrinkling after Nd: YAG laser membranotomy in vasa lva retinopathy. *Am J Ophthalmol* 2003;**136**:763–6.
- 15 Meyer CH, Mennel S, Rodrigues EB, *et al*. Persistent premacular cavity after membranotomy in vasa lva retinopathy evident by optical coherence tomography. *Retina* 2006;**26**:116–18.
- 16 Meyer CH, Mennel S, Rodrigues EB, *et al*. Is the location of vasa lva hemorrhages submembranous or subhyaloidal? *Am J Ophthalmol* 2006;**141**:231–2.
- 17 McCarron MO, Alberts MJ, McCarron P. A systematic review of Terson's syndrome: frequency and prognosis after subarachnoid haemorrhage. *J Neural Neurosurg Psychiatry* 2004;**75**:491–3.
- 18 Adel B, Israel A, Friedman Z. Dense subhyaloid hemorrhage or subinternal limiting membrane hemorrhage in the macula treated by Nd:YAG laser. *Arch Ophthalmol* 1998;**116**:1542–3.
- 19 Morris R, Kuhn F, Witherspoon CD. Hemorrhagic macular cysts. *Ophthalmology* 1994;**101**:1.
- 20 Srinivasan S, Kyle G. Subinternal limiting membrane and subhyaloid haemorrhage in Terson syndrome: the macular 'double ring' sign. *Eye* 2006;**20**:1099–101.
- 21 Messmer EP, Wessing A, Ruprecht K, *et al*. Solitary intraretinal macular hemorrhage. *Graefes Arch Clin Exp Ophthalmol* 1984;**222**:9–12.
- 22 Ossoinig KC. Echographic detection and classification of posterior hyphemas. *Ophthalmologica* 1984;**189**:2–11.
- 23 Shukla D, Naresh KB, Kim R. Optical coherence tomography findings in vasa lva retinopathy. *Am J Ophthalmol* 2005;**140**:134–6.
- 24 Arroyo JG, Bula DV. Immunohistochemical study of the internal limiting membrane in Terson syndrome. *Retina* 2004;**24**:155–7.
- 25 Kuhn F, Morris R, Witherspoon CD, *et al*. Terson syndrome. Results of vitrectomy and the significance of vitreous hemorrhage in patients with subarachnoid hemorrhage. *Ophthalmology* 1998;**105**:472–7.
- 26 Morris R, Kuhn F, Witherspoon CD, *et al*. Hemorrhagic macular cysts in Terson's syndrome and its implications for macular surgery. *Dev Ophthalmol* 1997;**29**:44–54.
- 27 Friedman SM, Margo CE. Bilateral subinternal limiting membrane hemorrhage with Terson syndrome. *Am J Ophthalmol* 1997;**124**:850–1.
- 28 Arroyo JG, Bula DV. Immunohistochemical study of the internal limiting membrane in Terson syndrome. *Retina* 2004;**24**:155–7.
- 29 Duke-Elder S, Dobree JH. *System of ophthalmology*, Vol 10. St Louis: C V Mosby, 1967:145–7.
- 30 Schubert HD. Hemorrhagic macular cyst or hematoma? *Ophthalmology* 1994;**101**:1477–8.
- 31 Blaise P, Duchateau E, Comhaire Y, *et al*. Optical coherence tomography in the diagnosis of premacular hemorrhage. *Retina* 2006;**26**:232–4.
- 32 Velikay M, Datlinger P, Stolba U, *et al*. Retinal detachment with severe proliferative vitreoretinopathy in Terson syndrome. *Ophthalmology* 1994;**101**:35–7.

- 33 **Shukla D.** Is the location of Valsalva hemorrhages submembranous or subhyaloidal? Authors reply. *Am J Ophthalmol* 2006;**141**:231–2.
- 34 **Garcia-Arumi J,** Corcostegui B, Tallada N, *et al.* Epiretinal membranes in Terson's syndrome. A clinicopathologic study. *Retina* 1994;**14**:351–5.
- 35 **Fechner PU.** Premacular hemorrhage: a new indication for argon-laser-therapy. *Klin Monatsbl Augenheilkd* 1980;**177**:502–5.
- 36 **Raymond LA.** Neodymium: YAG laser treatment for hemorrhages under the internal limiting membrane and posterior hyaloid face in the macula. *Ophthalmology* 1995;**102**:406–11.
- 37 **Hesse L,** Schmidt J, Kroll P. Management of acute submacular hemorrhage using recombinant tissue plasminogen activator and gas. *Graefes Arch Clin Exp Ophthalmol* 1999;**237**:273–7.
- 38 **Hesse L,** Kroll P. Enzymatically induced posterior vitreous detachment in proliferative diabetic retinopathy. *Klin Monatsbl Augenheilkd* 1999;**214**:84–9.
- 39 **Schmitz K,** Kreutzer B, Hitzer S, *et al.* Therapy of subhyaloidal hemorrhage by intravitreal application of rtPA and SF<sub>6</sub> gas. *Br J Ophthalmol* 2000;**84**:1324–5.
- 40 **Yang CM,** Chen MS. Tissue plasminogen activator and gas for diabetic premacular hemorrhage. *Am J Ophthalmol* 2000;**129**:393–4.
- 41 **Burk SE,** Da Mata AP, Snyder ME, *et al.* Indocyanine green-assisted peeling of the retinal internal limiting membrane. *Ophthalmology* 2000;**107**:2010–14.
- 42 **Stalmans P,** Parys-Vanginderdeuren R, De Vos R, *et al.* ICG staining of the inner limiting membrane facilitates its removal during surgery for macular holes and puckers. *Bull Soc Belge Ophthalmol* 2001;**281**:21–6.
- 43 **Kroll P.** Passagere Linsenröbung bei intraokularer Gastamponade. *Ber Dtsch Ges* 1981;**78**:1067–8.
- 44 **Chang MA,** Parides MK, Chang S, *et al.* Outcome of phakoemulsifikation after pars plana vitrectomy. *Ophthalmology* 2002;**109**:948–54.
- 45 **Thompson JT.** The role of the patient age and intraocular gas use in cataract progression after vitrectomy for macular holes and epiretinal membranes. *Am J Ophthalmol* 2004;**137**:250–7.
- 46 **Cherfan GM,** Michels RG, de Bustras S, *et al.* Nuclear sclerotic cataract after vitrectomy for idiopathic epiretinal membranes causing macular pucker. *Am J Ophthalmol* 1991;**111**:434–8.

## CATARACT SURGICAL RATES

# Cataract surgical rates: is there overprovision in certain areas?

John M Sparrow

Healthcare providers should ensure that cataract surgical rates are beneficial to all

Cataract surgery rates in developed countries have increased dramatically over the past two decades. In England, the crude surgical rate in 1990 was around 2/1000,<sup>1</sup> by 1997 this had risen to around 3/1000<sup>2</sup> and by 2005 peaked at around 6/1000,<sup>3</sup> an increase of close to 300% over 16 years. In Sweden, rates have been even higher; over a 9-year period, the demand for cataract surgery rose steadily from 4.5/1000 in 1992 to 7.3/1000 in 2000.<sup>4</sup> Encouraged by the "Action on Cataract" initiative in 2000, National Health Service units streamlined practice and massively increased surgical throughput. For a time, health policy became so obsessed by capacity building that fixed and travelling independent sector treatment centre facilities were set up irrespective of local need. Publicity and political hype far outstripped the actual capacity of these treatment centres, which contributed a mere 3% to national cataract surgical throughput. Inappropriate and politically enforced contracting at a local level, however, undermined a number of established high-quality ophthalmological units, illustrating a profound lack of insight and planning behind these politically driven initiatives. This surgical bonanza, handed down by a government keen to gain popularity by cutting surgical waiting times, has paid scant attention to the possibility that surgery for early cataract may carry unacceptable risks. Misleading of the

public by trivialisation of cataract surgery has occurred, with its presentation in the media as a quick and easy operation with a high success rate, and patients with minor visual symptoms frequently seek surgery in the belief that it is (virtually) risk free. The evidence, however, indicates otherwise. Following cataract surgery, up to 8% of patients may be dissatisfied with the outcome of their operation,<sup>5</sup> with 7% reporting no change and 9% reporting increased difficulty at 6 months post-operatively in a sample of over 10 000 operations in the Swedish register.<sup>4</sup> These figures are at odds with the "technical success" rates frequently quoted, which typically note the posterior capsular and/or vitreous loss benchmark rate of  $\leq 2\%$ .<sup>6</sup> However perfect though, an operation on an eye that does not really need surgery is unlikely to provide much visual benefit and carries an unjustifiable risk.

Improvements in technology, higher expectations by the public, greater confidence of surgeons in their ability to deliver a quality outcome and politically driven initiatives to reduce waiting times have contributed to this phenomenon in the UK and elsewhere. In the National Health Service (NHS), thresholds for listing for surgery have become increasingly lenient in visual acuity terms; in 1990 under 9% of eyes for surgery had an acuity 6/12 or better,<sup>7</sup> by 1997 this had risen to 31%<sup>8</sup> and in 2003 had reached 45% in an 8-centre audit of over 16 000 cases.<sup>9</sup>

In this issue, Keenan *et al*<sup>10</sup> (*see p 901*) provide an impressive and detailed review of cataract surgical rates across England from the 1960s onwards. The exponential rise in surgery is catalogued separately from Hospital Episode Statistics (and its precursor, the Hospital Inpatient Enquiry) and the Oxford Record Linkage Study, the latter capable of separate identification of "people" as opposed to "eyes" undergoing surgery each year. Data are aggregated for three separate periods and further broken down by age and gender. Recent data are mapped and graphed to illustrate large geographical variations in surgical rates between local authorities, and these are correlated against the social deprivation score for that locality. Interestingly, higher levels of social deprivation are correlated with higher surgical rates, suggesting that access to care seems not to be significantly compromised in socially deprived local authorities, although, as the authors correctly comment, other socioeconomic forces may be influencing these observed gradients.

Few questions have been asked about the appropriateness of this exponential rise in surgical rates, which have generally been packaged and received as good news. Following on this tide of promotion and surgical confidence, a reality check may now be necessary to ensure that patients are protected from unnecessary harm when seeking assistance for minor visual symptoms from early cataract. Overprovision, should this now be occurring, is wasteful and potentially damaging. Patient-reported outcome and health-gain data are urgently needed to better our understanding of the risk-to-benefit balance of surgery for early cataract. This will serve to inform patients, surgeons and commissioners on how best to use an undoubtedly excellent surgical procedure. Healthcare providers are duty bound to ensure that cataract surgery is appropriately applied with optimisation of benefit and minimisation of harm.