MAJOR REVIEW

Suprachoroidal Hemorrhage

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Abstract. Suprachoroidal hemorrhage is a feared complication of all types of intraocular surgery. Although rare, it is typically associated with severe visual disability, and this has prompted efforts to better understand the pathogenesis of this condition, to identify the patients at risk for this event, and to improve treatment of patients who develop this condition either intraoperatively or postoperatively. Controversy still exists regarding the best course of treatment for these patients. Although the introduction of perfluorocarbon liquids as a surgical adjunct during vitrectomy surgery may assist in the removal of suprachoroidal hemorrhage, the visual outcomes still remain disappointing. (**Surv Ophthalmol 43**: 471–486, 1999.   © 1999 by Elsevier Science Inc. All rights reserved.)

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Suprachoroidal hemorrhage (SCH), typically an explosive accumulation of blood within the suprachoroidal space, can be a devastating complication of ophthalmic surgery. The initial case report of choroidal hemorrhage in the setting of ophthalmic surgery was in 1760, by Baron de Wetzel.[50] Terson first coined the term *expulsive hemorrhage* in 1894 to denote an acute hemorrhage of the choroid resulting in poor visual outcome and partial or total loss of vision.[67] The first successfully managed case of expulsive choroidal hemorrhage was reported in 1915 by Verhoeff.[69] Since then, much has been written regarding the etiology, risk factors, and management of this condition. A better understanding of SCH can help the ophthalmic surgeon to avoid this complication in patients at risk during surgery and to optimally treat patients in whom SCH develops.

I. Anatomic Considerations

The physiology of the suprachoroidal space, a potential space situated between the choroid and the sclera, has been well described elsewhere.[12] When filled with blood or fluid, it becomes a true space of which the boundaries are the scleral spur anteriorly and the optic disc posteriorly. The choroid is firmly attached to the sclera at the ampullae of the vortex veins. These attachments are responsible for the typical lobular appearance of a large choroidal detachment. The outer surface of the ciliary body and the choroid are closely attached to the sclera by a series of fine collagen fibrils arranged in tangential sheets.[12] The suprachoroidal space normally contains approximately 10 μL of fluid.[31]

II. Definitions

Choroidal detachment and SCH represent two distinct entities. A choroidal detachment is defined as a separation of the uvea from the sclera. Choroidal detachment is secondary to effusion of serous fluid within the suprachoroidal space. Both hypotony and inflammation appear to be causative factors responsible for the accumulation of fluid in the suprachoroidal space.[12,19]

Suprachoroidal hemorrhage is defined as blood, as opposed to serous fluid, within the suprachoroidal space. Suprachoroidal hemorrhages can be clas-

sified in several ways. They may be categorized with respect to size and the extent of hemorrhage. They may be classified by their relationship to intraocular surgery. Lastly, they may also be categorized by precipitating events.

When categorized with respect to size, SCHs can vary from a small area of involvement to massive involvement. Suprachoroidal hematomas represent small loculated collections of blood within the suprachoroidal space. These lesions are benign, usually resolve spontaneously, and are distinct from SCH associated with intraocular surgery.[5] Hoffman and coworkers have defined small areas of suprachoroidal blood in patients who have undergone intracapsular cataract extraction as *limited* choroidal hemorrhage.[32] These hemorrhages most likely represent the postoperative equivalent of spontaneous suprachoroidal hematomas. Massive SCHs represent the other end of the size spectrum. A massive hemorrhage into the suprachoroidal space can be sufficiently large to force the inner retinal surfaces into direct apposition, usually within the center of the posterior chamber. This extensive type of hemorrhage is commonly defined as a *kissing* SCH; an analogous term is a *massive SCH with central retinal apposition.*

The timing of development of SCH with relation to intraocular surgery is another method of classifying the condition. Suprachoroidal hemorrhage may develop at the time of intraocular surgery, representing intraoperative SCH. Typically, an intraoperative SCH is associated with a massive degree of hemorrhage, resulting in the expulsion of intraocular contents through the surgical wound. Such a forceful SCH is categorized as an *expulsive SCH*. Suprachoroidal hemorrhages that develop in the postoperative period are termed either *postoperative SCH* or *delayed SCH*. Delayed SCHs occur in a closed system and, therefore, are not typically associated with expulsion of intraocular contents. Nevertheless, they may be extensive enough to result in a kissing-type configuration.

Finally, SCHs can be categorized by precipitating events. In particular, they can occur in the setting of either penetrating or blunt trauma. Traumatic SCH behaves differently from SCH associated with intraoperative surgery and should be considered a distinct entity. Traumatic SCH will not be included in this discussion.

III. Pathophysiology

Several theories have been postulated to explain the mechanism whereby SCHs develop in nontraumatized eyes.[9,42,43,75] Hypotony appears to be the major precipitating factor, resulting in a rupture of a necrotic long or short posterior ciliary artery.[42] Another theory is that hypotony causes a choroidal effusion that stretches and ruptures a long or a short posterior ciliary artery.[43] Experimentally, hypotony has been linked to enhanced aqueous humor outflow from the anterior chamber into the suprachoroidal space.[48] Obstruction of venous outflow from the vortex veins may also be a precipitating factor that initiates the cascade of events leading to an SCH.[75]

Histopathologic studies on either experimental animal models or human autopsy eyes have been performed to try to help elucidate the precise cause of expulsive SCH. A rabbit model for the development of expulsive SCH has been developed.[9] Using histologic evidence from this rabbit model, Beyer and coworkers have suggested four sequential stages in the development of expulsive SCH: 1) engorgement of the choriocapillaris; 2) serous effusion into the suprachoroidal space, occurring mainly in the posterior pole; 3) stretching and tearing of the vessels and attachments at the base of the ciliary body as the effusion enlarges; and 4) resultant massive extravasation of blood arising from torn ciliary body vessels, which leads to SCH and expulsion of intraocular contents through the surgical wound.[9]

Human histopathologic studies appear to confirm the postulate that hypotony followed by ciliochoroidal effusion can ultimately lead to rupture of the long posterior ciliary arteries and, therefore, be responsible for the development of SCH.[43,73,74] This seems to be illustrated in a histopathologic study reported by Wolter and Garfinkel; in their study typical ciliochoroidal effusion is demonstrated in a perforated human eye in which a change from effusion to expulsive SCH is beginning to occur.[74] The long posterior ciliary arteries appear especially vulnerable to rupture during separation of the choroid from the sclera—from ciliochoroidal effusion—because their connections between the scleral exit and the outer choroid are short.[74]

IV. Incidence

Suprachoroidal hemorrhage, both during intraocular surgery and in the postoperative period, is a relatively rare event. It has been reported to occur in the setting of all types of intraocular procedures, including cataract extraction,[17,24,46,61,65,70] penetrating keratoplasty,[22,34,52,53] glaucoma filtering surgery,[14,27,30,47,57,61] and vitreoretinal surgery.[25,38,51,60,61,64] The actual incidence of SCH is somewhat difficult to reliably estimate, because it occurs so infrequently. Numerous authors have retrospectively studied the incidence of expulsive SCH during intraocular surgery for various surgical procedures. The results of these studies, as well as studies of the incidence of delayed SCH, are summarized in Table 1. With older methods of cataract surgery, the overall incidence of expulsive SCH

has been widely regarded to be approximately 0.2%.[21] Furthermore, secondary intraocular lens implantation surgery appears to carry a similar risk of expulsive SCH.[61]

The advent of phacoemulsification lens extraction, topical anesthesia, and clear corneal incision techniques has lowered the incidence of SCH secondary to cataract surgery to 0.03%[24] to 0.06%.[17] These newer techniques of cataract extraction can be carried out more rapidly with less manipulation of the globe. In addition, phacoemulsification cataract extraction can be performed with less pronounced fluctuations of intraocular pressure (IOP). One can postulate that fewer oscillations of IOP should circumvent the development of ocular hypotony during surgery, thereby preventing the inciting event in the development of SCH.

Studies examining the risk of SCH in relation to glaucoma filtering surgery must be separated according to type of SCH. The incidence of expulsive SCH during glaucoma filtering surgery has been reported to be approximately 0.15%.[61] Moreover, several studies have reported the incidence of delayed

SCH after glaucoma filtering surgery. Givens and Shields examined 305 consecutive cases of glaucoma filtering procedures and found an incidence of 1.6%.[27] Ruderman and coworkers reported a similar incidence of delayed SCH of 2.0% after 500 consecutive cases of glaucoma surgical procedures.[57] Paysse and coworkers reported about a 6% incidence of delayed SCH in patients participating in Molteno implantation studies.[47] It is not surprising that the incidence of delayed SCH is approximately 10-fold greater than that of expulsive SCH. The opportunity to develop a delayed SCH after glaucoma surgery appears to be particularly great. Indeed, delayed SCH is believed to be precipitated by prolonged postoperative hypotony and inflammation, which are not uncommon after glaucoma procedures. Also, delayed SCHs vary in extent from small, limited, suprachoroidal hematomas to massive hemorrhages, whereas expulsive SCHs usually are severe and massive hemorrhages.

Expulsive SCH has been rarely reported after both penetrating keratoplasty and vitreoretinal surgery. The incidence of expulsive SCH during penetrating keratoplasty has been reported to vary between

TABLE 1

Incidence of Expulsive and Delayed Suprachoroidal Hemorrhage

Study	Surgery Type	Number of Procedures	Number of SCHs	Incidence (%)	SCH Type
Cataract surgery					
Taylor[66]	ICCE	58,735	115	0.2	Expulsive
Straatsma et al[63]	CE	8,285	4	0.05	Expulsive
Speaker et al[61]	ECCE/Phaco	22,262	34	0.15	Expulsive
	ICCE	6,440	12	0.19	Expulsive
	2° IOL	1,782	3	0.17	Expulsive
Davison[19]	Phaco	2,839		0.81	Delayed
Davison[17]	Phaco	3,096		0.06	Delayed
Eriksson et al[24]	ECCE	14,352		0.13	Mixed
	Phaco	23,213		0.03	Mixed
Corneal surgery					
Holland et al[33]	PK	115	1	0.087	Expulsive
Ingraham et al[34]	PK	830	9	1.08	Expulsive
Speaker et al[61]	PK	1,436	8	0.56	Expulsive
Price et al[52]	PK	2,011		0.45	Expulsive
Glaucoma surgery					
Givens and Shields[27]	Glaucoma	305	5	1.60	Delayed
Ruderman et al[57]	Glaucoma	500	10	2.00	Delayed
Speaker et al[61]	Glaucoma	1,329	2	0.15	Expulsive
Paysse et al[47]	Molteno	197	12	6.1	Delayed
Vitreoretinal surgery					
Speaker et al[61]	Vitreoretinal	2,210	9	0.41	Expulsive
Hawkins and Schepens[31]	Retinal	1,500	15	1.0	Expulsive
Piper et al[51]	Vitreoretinal	683	13	1.9	Mixed
Sharma et al[60]	Vitrectomy	6,971		0.17	
Intraocular surgery					
Cantor et al[14]	Intraocular	1,638	12	0.73	Expulsive

SCH: suprachoroidal hemorrhage; ICCE: intracapsular cataract extraction; ECCE: extracapsular cataract extraction; CE: cataract extraction; Phaco: phacoemulsification; 2°IOL: secondary intraocular lens; PK: penetrating keratoplasty; Mixed: expulsive and delayed suprachoroidal hemorrhage.

0.45 and 1.08%.[52,61] The cumulative incidence, incorporating the data from all reported studies, is approximately 0.75%. Penetrating keratoplasty typically involves a greater period of intraocular hypotony than cataract surgery. Furthermore, this type of surgical procedure affords a greater opportunity for scleral collapse and displacement of intraocular structures. This may account for the higher incidence of expulsive SCH after this type of surgery.

Several studies have systematically examined the relationship between expulsive SCH and vitreoretinal surgery; the reported incidence during this type of surgery varies from 0.17% to as high as 1.9%.[31,51,60,61] Typically, prolonged intraocular hypotony does not occur during vitreoretinal surgery. Expulsive SCH, however, may be related to direct trauma to the choroid during drainage of subretinal fluid or creation of pars plana sclerotomies, or to compression and trauma to vortex veins during placement of scleral buckling elements.

V. Patient Characteristics

Retrospective studies and anecdotal case reports indicate that certain patient risk factors are associated with the development of an SCH, both during and after intraocular surgery. These systemic, ocular, intraoperative, and postoperative risk factors are summarized in Table 2.

TABLE 2

Risk Factors for Suprachoroidal Hemorrhage

Systemic risk factors
   Advanced age
   Arteriosclerosis
   Hypertension
   Blood dyscrasia or coagulation defect
   Diabetes mellitus
Ocular risk factors
   Choroidal arteriolar sclerosis
   Glaucoma
   Myopia
   Aphakia or pseudophakia
   Choroiditis
   Recent intraocular surgery
   SCH in fellow eye
Perioperative risk factors
   Retrobulbar anesthesia without epinephrine
   Precipitous drop in IOP
   Valsalva maneuvers
   Vitreous loss
   Inraoperative systemic hypertension
Postoperative risk factors
   Postoperative trauma
   Ocular hypotony
   Valsalva maneuvers
   TPA administration

IOP: intraocular pressure; TPA: tissue plasminogen activator.

Numerous systemic findings have been implicated in the development of SCH. In particular, sclerosis and fragility of choroidal vessels associated with advanced age, systemic hypertension, and arteriosclerosis have all been frequently described as predisposing factors.[19,32,53,63,64] Furthermore, in an extensive case-control study of risk factors for expulsive SCH, Speaker and coworkers reported that generalized atherosclerosis is a significant systemic risk factor.[61] This study also reported an association between the development of SCH in the perioperative period with a history of liver disease and with preoperative use of digoxin.[61]

Various ocular conditions have been reported to be associated with SCH, including glaucoma, elevated IOP, aphakia, axial myopia, and inflammation.[1,14,16,27,30,57,61,64] In the most extensive case-control study of expulsive SCH, glaucoma, elevated IOP, and increased axial length were all reported to be highly significant.[41] The mechanism by which these ocular risk factors are believed to have an impact on the development of SCH is similar. These ocular conditions are presumed to weaken the integrity of the long posterior ciliary arteries by promoting vascular necrosis. This, in turn, would make these vessels more susceptible to rupture. In the case of surgical aphakia, the absence of the lens and zonular support is believed to allow more stretching and separation of the uvea from the sclera during ciliochoroidal effusions. Loss of scleral rigidity and/or choroidal vascular fragility are also believed to be responsible for the association between SCH and axial myopia.

Certain intraoperative maneuvers have been anecdotally implicated in the development of SCH. Numerous authors have cautioned that general anesthesia may be a risk factor for SCH.[13,66,71] Speaker and coworkers found an association between expulsive SCH and intraoperative pulse rate greater than 90 beats per minute.[61] Furthermore, they reported a protective effect when epinephrine was in the anesthetic mixture used for a lid block, and also when the eye was softened before surgery.[61]

Coughing, straining, nausea, vomiting, and Valsalva-type maneuvers have all been implicated in precipitating SCH, either from bucking during general anesthesia, or in the postoperative period.[4,13,14,26,34,57] All these maneuvers are believed to increase episcleral venous pressure, resulting in an increased pressure gradient across the wall of necrotic ciliary vessels, thereby promoting their rupture.

Hypotony in the postoperative period has been reported to help contribute to the development of delayed SCH.[30,57] As mentioned above, hypotony is believed to initiate the development of ciliochoroidal effusion, thereby starting the cascade of events end-

ing in SCH.[48] In addition, the hypotonous eye may be more susceptible to episcleral venous pressure fluctuations induced by Valsalva maneuvers and, therefore, be more vulnerable to rupture of ciliary arteries.[57] The Fluorouracil Filtering Surgery Study Group examined risk factors for SCH after filtering surgery and did not find postoperative hypotony to be statistically significant when comparing eyes with SCH versus eyes without hemorrhage.[1] The incidence of SCH in this study was 6%, however, and therefore this study may not have had the statistical power to detect a difference between these two populations of patients.

Spontaneous SCH has also been reported in cardiac patients undergoing treatment of acute myocardial infarction with systemic thrombolytic agents.[15,35,62] These potent agents promote a systemic hemolytic state and thus greatly increase the risk of severe hemorrhaging.

VI. Clinical Considerations

A. PROPHYLACTIC MEASURES

Before surgery, certain preventive measures should be employed in patients at high risk for the development of SCH. As outlined in Table 3, these measures should be undertaken before, during, and after surgery.

A thorough preoperative examination should be performed with particular attention to both systemic and ocular risk factors for SCH (Table 2). In particular, a complete medical evaluation should be undertaken, looking for evidence of cardiovascular disease, such as hypertension and arteriosclerosis. Also, patients should be screened for evidence of liver disease or the use of digoxin. Any underlying blood dyscrasia or coagulation defect should be addressed.

TABLE 3

Prophylactic Measures

Preoperative
   Perform complete ophthalmic evaluation.
   Perform complete medical evaluation.
   Avoid aspirin and other anticoagulants.
Operative
   Use minimal preoperative phenylephrine to avoid
     systemic hypertension.
   Use epinephrine in lid blocks.
   Lower IOP before incision.
   Avoid rapid decompression of globe.
   Avoid Valsalva maneuvers.
   Recognize SCH early if it occurs.
Postoperative
   Avoid eye trauma or eye pressure.
   Avoid hypotony.
   Avoid Valsalva maneuvers.

IOP: intraocular pressure; SCH: suprachoroidal hemorrhage.

Patients should be encouraged to avoid the use of aspirin or other nonsteroidal antiinflammatory agents. Diabetic patients should have their blood glucose level under satisfactory control.

Preoperatively, the physician should be alerted to the high-risk patient by the presence of certain findings in the ophthalmic history. Indeed, patients at highest risk for the development of SCH usually have a history of chronic glaucoma and are either aphakic or pseudophakic. Other risk factors include severe myopia, choroidal arteriolar disease, recent intraocular surgery, and the presence of SCH in the fellow eye.

Prophylactic measures should be used in the perioperative period. Increased preoperative IOP and a sudden decompression of the globe during intraocular surgery have both been implicated as risk factors for the development of SCH.[61] It would seem prudent, therefore, to recommend that aggressive medical management of high IOP be undertaken before surgery. Softening of the eye with intravenous hyperosmotic agents or carbonic anhydrase inhibitors at the beginning of the operative procedure should be considered. Compressive maneuvers, however, should be avoided, and they may contribute to choroidal hyperemia and/or may facilitate the rupture of a weakened artery. Recently, an anterior chamber maintainer has been advocated to reduce intraoperative hypotony, which may lower the chance of SCH in high-risk cases.[11]

Hypertension and increased intraoperative heart rate have also been implicated as risk factors for SCH. In patients with hypertension and tachycardia, efforts should be made at the time of surgery to lower the heart rate and blood pressure. Labetalol, a rapidly acting intravenous agent with both alpha- and beta-adrenergic antagonist activity, has been suggested for use in such high-risk patients.[61] The use of preoperative phenylephrine should also be restricted to help avoid systemic hypertension.

General anesthesia may also be a risk factor for SCH. Performing surgery under monitored local anesthesia rather than under general anesthesia should be considered. A protective effect of epinephrine added to the lid block has been reported in one study[61] and therefore should be considered when this form of anesthesia is administered.

Both during and after surgery, Valsalva maneuvers should be avoided. Laxatives and antiemetics are therefore recommended for high-risk patients. Postoperatively, the patient must be instructed to avoid any eye trauma or eye pressure, as this may precipitate the rupture of ciliary arteries. Postoperative inflammation must be vigorously controlled because inflammation may contribute to serous fluid accumulation in the suprachoroidal space, thereby starting the cascade of events leading to SCH.

In cases of glaucoma filtering surgery, every effort should be made to avoid postoperative hypotony. In this regard, the use of a scleral flap rather than a full-thickness procedure may theoretically result in less marked hypotony. Also, tight wound closure with restoration of IOP to normal levels at the end of surgery is recommended regardless of the type of surgical procedure.

B. INTRAOPERATIVE DIAGNOSIS AND MANAGEMENT

A favorable outcome after expulsive or delayed SCH requires early recognition and expeditious management. If an expulsive SCH occurs during intraocular surgery, the surgeon must be prepared to react quickly and decisively. Clinically, early signs of an intraoperative SCH include a sudden increase in IOP with firming of the globe, loss of a red reflex, and shallowing of the anterior chamber with forward displacement of the iris and lens or lens implant, with or without vitreous prolapse. If an intraoperative SCH is suspected, immediate tamponade of the open globe is required. This can be accomplished by either direct digital pressure or rapid suturing of all surgical incisions. Closure of the eye allows the IOP to rise to a sufficient level to tamponade the bleeding vessel. If intraocular contents are expelling, they should be reposited as quickly as possible. If the intraocular contents cannot be replaced in the globe, the eye can be softened by performing posterior sclerotomies. The long-term benefit of performing posterior sclerotomies acutely at the time the SCH occurs remains debatable. Verhoeff originally advocated the use of emergence posterior sclerotomies to salvage such eyes.[69] Blood in the suprachoroidal space clots extremely rapidly, and often the SCH has already clotted by the time the emergency sclerotomy is performed. If the hemorrhage has not clotted, the eye will usually soften enough to allow for the repositioning of intraocular tissue. With the acute drainage of a SCH, however, the tamponading effect of increased IOP in a closed eye is lost and frequently the SCH will recommence hemorrhaging. The efficacy of drainage sclerotomies was tested in an experimental model of nonexpulsive SCH in rabbit eyes developed by Lakhanpal.[36] The creation of drainage sclerotomies in this experimental model, during the acute formation of SCH, resulted in a further increase in the size of the SCH with marked extension of the hemorrhage into the retina and vitreous.[36] Therefore, Lakhanpal concluded that creation of immediate sclerotomies during SCH is detrimental to eyes.

Several other maneuvers may be performed acutely. Reformation of the anterior chamber by saline or air injection is recommended. This can pre-vent entrapment of vitreous into the surgical wound, which increases the risk of development of retinal detachment. Intravenous hyperosmotic agents, sedation for agitated patients, and lowering of systolic blood pressure may be helpful. Removal of the lid speculum may also decrease direct pressure on the globe, preventing further extrusion of intraocular contents.

C. POSTOPERATIVE DIAGNOSIS AND MANAGEMENT

Delayed or postoperative SCH behaves somewhat differently from expulsive SCH. This type of SCH usually presents after uncomplicated glaucoma filtering surgery. Typically, patients experience a sudden onset of severe ocular pain with a subsequent loss of vision. Headache, nausea, and vomiting may also accompany the ocular pain. These symptoms may occur after a Valsalva-type maneuver or may be severe enough to arouse a patient from sleep. Clinically, patients with delayed SCH can have markedly decreased vision. The appearance of the eye can mimic that of an acute retrobulbar hemorrhage.[29] On slit-lamp examination, there may be shallowing of the anterior chamber, vitreous prolapse into the anterior chamber in aphakic and pseudophakic eyes, and loss of a red reflex (Fig. 1, left). On funduscopic examination, dark elevated dome-shaped lesions are seen occupying the equatorial fundus, but they may also be seen to extend posteriorly (Fig. 1, right). These lesions do not transilluminate well. Intraocular pressure may be low, normal, or elevated.

Regardless of the cause of the SCH, whether expulsive or delayed, the immediate postoperative management is similar. If the IOP is elevated, aggressive medical therapy with a topical beta-blocker and an oral carbonic anhydrase inhibitor is advocated. Inflammation should be controlled by liberal use of a topical steroid. Oral prednisone may be necessary in cases of severe intraocular inflammation. Pain can be considerable in this condition, because of stretching of ciliary nerves. Pain can be managed with adequate cycloplegia and analgesics. Aspirin and nonsteroidal agents, however, are contraindicated, as they may contribute to further hemorrhaging.

D. ECHOGRAPHY AND RADIOLOGIC EVALUATION

Suprachoroidal hemorrhage may be difficult to diagnose in the presence of opaque media. Corneal changes, breakthrough vitreous hemorrhage, and/or a kissing configuration may make adequate visualization of the posterior chamber impossible. Echography can be extremely useful in the diagnosis and in making decisions about management. Standardized echography can help establish an accurate diagnosis.[45] Echography can determine the location and
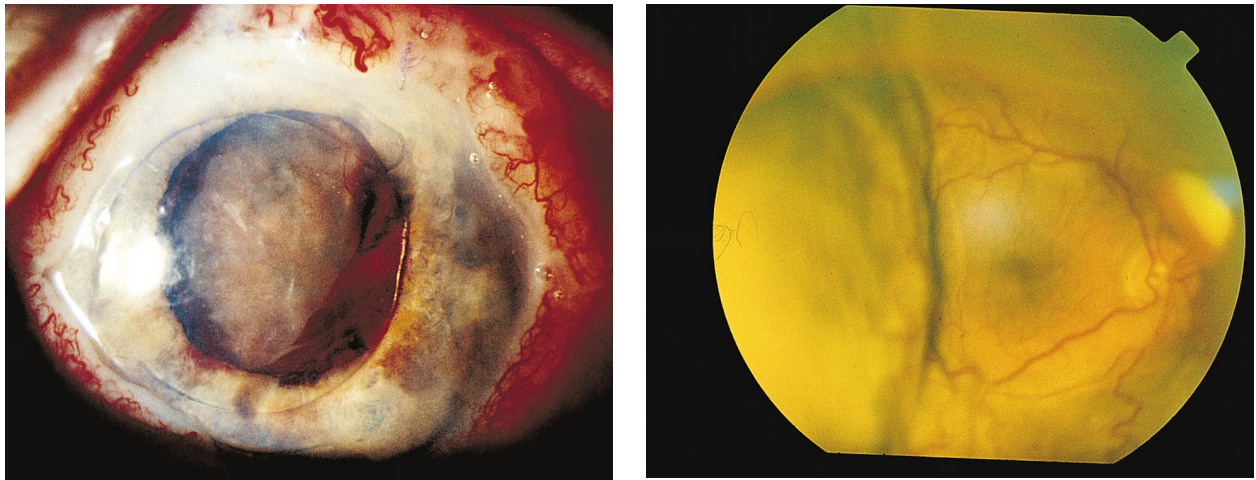
Fig. 1. *Left:* Suprachoroidal hemorrhage in an 89-year-old woman with a history of chronic glaucoma. She underwent uncomplicated cataract extraction, anterior chamber intraocular lens placement, and trabeculectomy. On the second postoperative day, she was awakened from sleep by severe eye pain. Notice the shallowed anterior chamber and forward displacement of the vitreous and anterior chamber lens. *Right:* Notice the large, darkly colored dome-shaped elevations arising from the peripheral retina extending toward the optic nerve.

extent of an SCH, as well as determine the status of the retina and vitreous. Furthermore, differentiation between hemorrhagic choroidal detachment and serous choroidal effusion can be made by A- and B-scans.[44]

Echographic evaluation should be performed as early as possible in the postoperative period in eyes with opaque media. This examination can be performed directly through the lids, with minimal direct pressure on the globe. Echographically, on B-scan, patients with SCH exhibit highly elevated choroidal detachments with a typical dome-shaped appearance (Fig. 2). In severe cases, broad central retinal apposition can be seen, illustrating a kissing-type configuration. The suprachoroidal space is typically filled with opacities denoting the presence of clotted blood. On A-scan evaluation, a steeply rising, double-peaked wide spike is seen, characteristic of choroidal detachment, with lower reflective spikes in the suprachoroidal space, indicating clotted hemorrhage.

The density of choroidal hemorrhage will vary with time. Echography is an excellent way to follow up patients, with or without opaque media, for the liquefaction of the SCH. On initial evaluation, most patients with SCH will have dense clotted hemorrhage seen on ultrasound. The fresh clots are seen echographically as a high-reflective, solid-appearing mass with irregular internal structure and irregular shape (Figs. 3 and 4). In subsequent follow-up, liquefaction of these blood clots occurs. A lower and more regular internal reflectivity can be noted on ultrasound (Fig. 4) because these clots have increased homogeneity of the internal structure. During dy-

namic echographic examination, fluid blood can be seen moving freely within the suprachoroidal space. Eventually, complete liquefaction of the SCH can be seen echographically, with the suprachoroidal space being filled with diffuse, low-reflective mobile opacities (Fig. 4). The time to liquefaction of such hemorrhages has been reported to vary from 7 to 14 days.[16,28,55,56] Once liquefaction of the hemorrhage has occurred, the height of the choroidal detachment will be seen to slowly diminish with time (Fig. 5). This reduction in height should be watched for in clinical follow-up.

Several reports have questioned the usefulness of ultrasound in the diagnosis and management of SCH.[30,57] In contrast, we found echography to be useful in the management of such cases.[16] With B-scan echography, blood clots can be readily identified in the suprachoroidal space (Figs. 3 and 4), as can the progression of clot lysis. In our patients, complete liquefaction of the clot occurred between 6 and 25 days.[16] If early surgical drainage of a massive suprachoroidal hemorrhage is warranted, echography may be a helpful adjunct in determining the optimal time for drainage. In our patients who did undergo surgical intervention, drainage was delayed until echographic evidence was obtained indicating liquefaction of the SCH.[16] By allowing for complete liquefaction of the SCH, one can minimize probing of the suprachoroidal space for residual clots, a maneuver that may cause further bleeding or retinal damage, yet facilitate the evacuation of the hemorrhage and restoration of normal ocular anatomy. It would appear that surgical intervention is most effective when clot lysis is near completion; however, whether com-
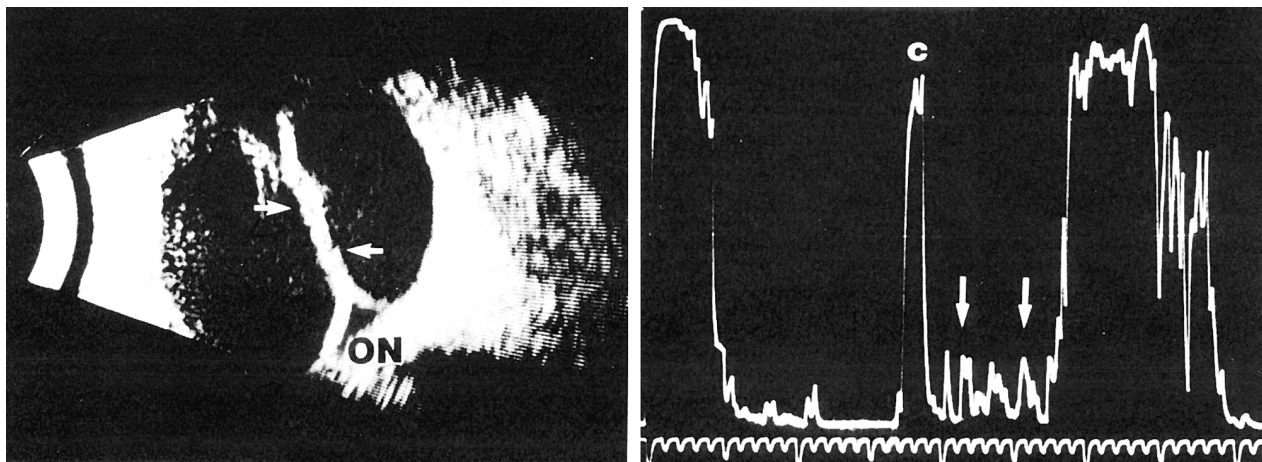
Fig. 2.   Left: B-scan echogram. Longitudinal view demonstrating a massive suprachoroidal hemorrhage with central retinal apposition. Note the typical dome-shaped appearance and broad central apposition of the detached choroid (arrows). ON indicates optic nerve. The suprachoroidal space is filled with opacities denoting the presence of blood.   *Right:* A-scan echogram demonstrating a steeply rising, double-peaked, wide spike (c) characteristic of a choroidal detachment; the lower-reflective spikes (arrows) in the suprachoroidal space represent hemorrhage. (Reprinted from Chu et al[16] with permission of the American Medical Association.)

plete liquefaction of the hemorrhage is absolutely necessary for successful drainage of the hemorrhage remains unknown. In our series, the mean time for clot lysis was 14 days, as determined by echography.[16] Gloor and Kalman also confirmed this finding with mean clot lysis between 10 and 15 days.[28] Welch et al,[70] Lambrou et al,[39] and Lakhanpal et al[37] all advocated delaying drainage of an SCH for 7 to 14 days.

Computed tomography can also be helpful in the localization and differentiation of serous and hemorrhagic choroidal detachments. Serous and hemorrhagic choroidal detachment can be differentiated on computed tomography because of differences in attenuation values.[49] On average, fresh hemorrhagic lesions have higher attenuation values than serous choroidal detachments. In addition, the use of magnetic resonance imaging has been shown to be helpful in the evaluation of suprachoroidal hemorrhage.[41]

E. INDICATIONS FOR SECONDARY SURGICAL MANAGEMENT

The decision to reoperate on patients with a postoperative SCH is controversial. Many recent reports have advocated early surgical intervention in the management of SCH.[2,4,26,30,37,39,54] These reports encompass, however, different subgroups of SCH. In
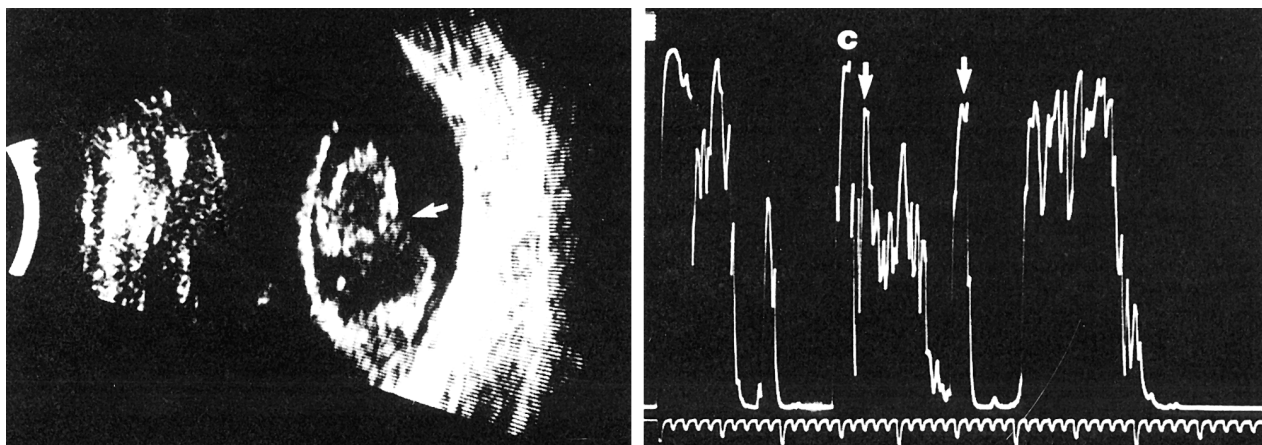

Fig. 3.   Left: B-scan echogram of a patient with a suprachoroidal hemorrhage 2 days after the hemorrhage occurred. The blood clot in the suprachoroidal space is easily identified (arrow).   *Right:* Corresponding A-scan shows the choroidal detachment (c) and the edges of the clot (arrows) with a low to medium, irregular internal reflectivity. (Reprinted from Chu et al[16] with permission of the American Medical Association.)
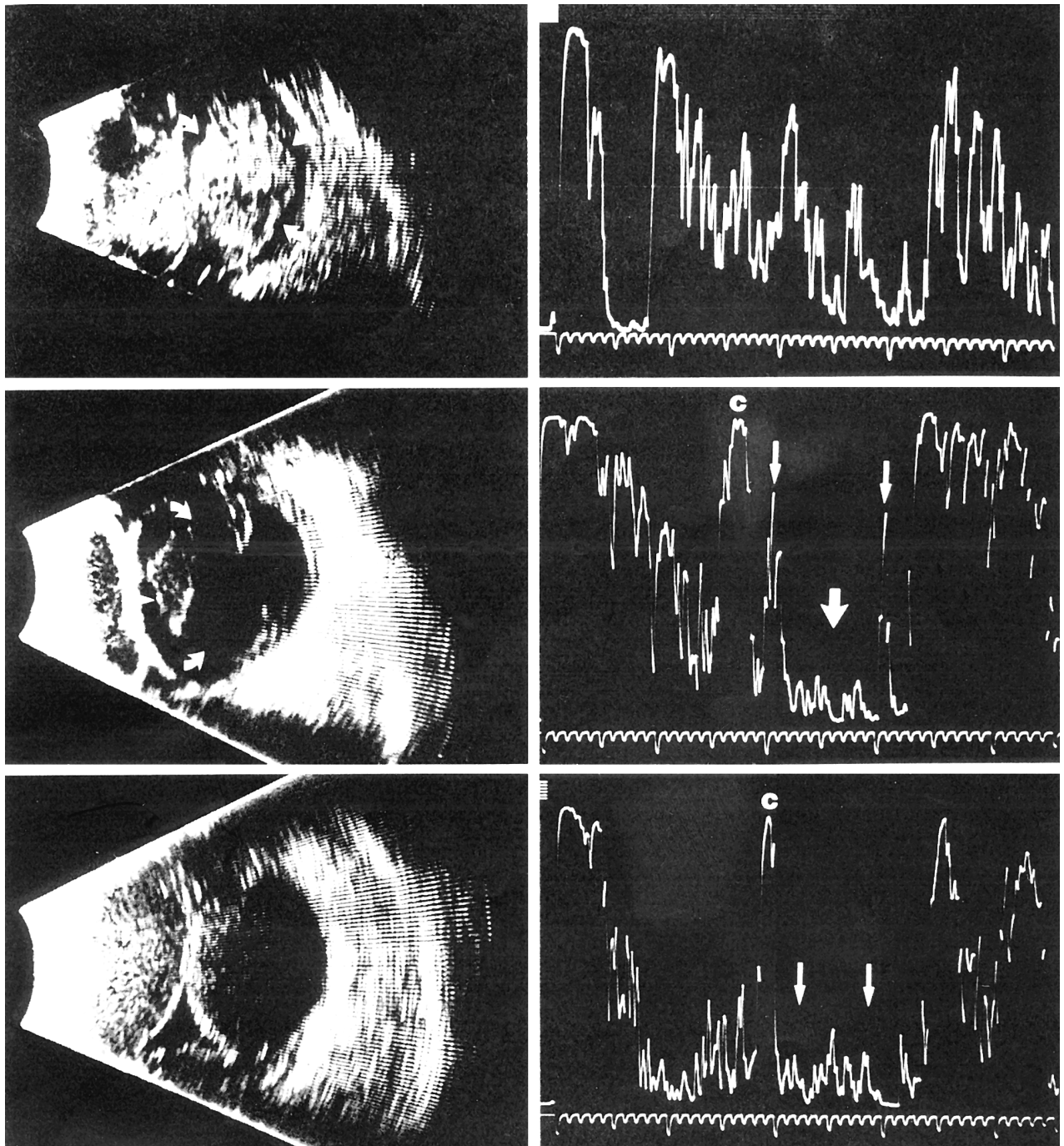
Fig. 4.   Top left: B-scan echogram 24 hours after repair of a cataract wound dehiscence. Note the massive suprachoroidal hemorrhage with central retinal apposition; the solid-appearing mass in the suprachoroidal space (arrows) represents a large blood clot.   *Top right:* A-scan showing the high and irregular reflectivity of the fresh clot.   *Center left:* B-scan echogram 5 days later. The large clot is still present (arrows); however, it now appears more homogeneous (echolucent).   *Center right:* A-scan spikes from the edges of the clot (small arrows). The internal reflectivity of the clot itself is lower and more homogeneous (large arrow).   *Bottom left:* B-scan echogram 2 weeks after occurrence of massive suprachoroidal hemorrhage. The suprachoroidal space is filled with fine diffuse opacities (mobile during dynamic examination) indicative of clot lysis.   *Bottom right:* Corresponding A-scan echogram 2 weeks after hemorrhage, exhibiting choroidal detachment (c) and low regular internal reflectivity of the liquefied blood (arrows). (Reprinted from Chu et al[16] with permission of the American Medical Association.)
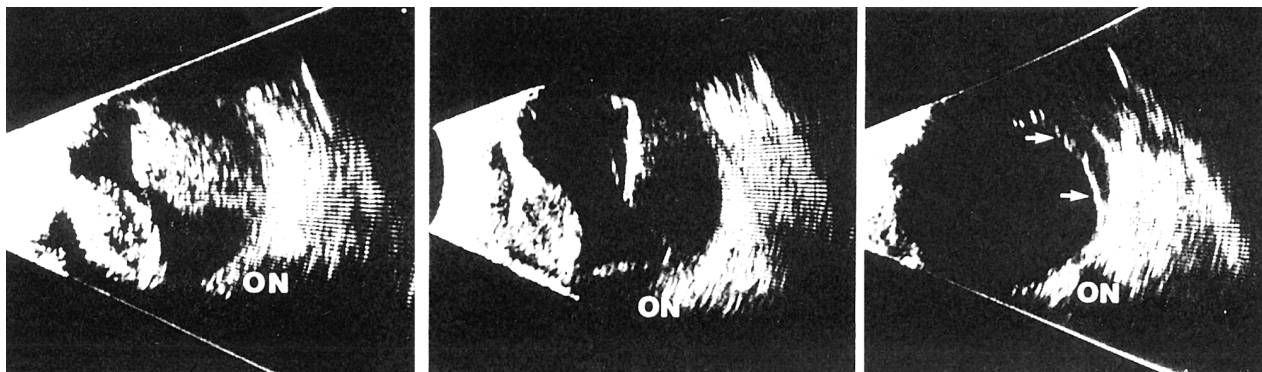
Fig. 5. *Left:* B-scan echogram demonstrating a massive suprachoroidal hemorrhage with retinal apposition 24 hours after the hemorrhage occurred. ON indicates optic nerve.   *Center:* Echographic examination at 2 weeks shows a decrease in the elevation of the choroidal hemorrhage.   *Right:* Resorption of the suprachoroidal hemorrhage is seen 5 weeks after hemorrhage. A shallow suprachoroidal hemorrhage is seen peripherally (arrows). (Reprinted from Chu et al[16] with permission of the American Medical Association.)

analyzing these reports, it is imperative that a distinction be made between expulsive SCH and delayed SCH, as their long-term prognosis may differ. Regardless of the cause of SCH, however, several indications for early surgical drainage have been proposed.[2,37,58,72] Specific clinical features may influence the decision to consider surgical drainage, including the presence of a retinal detachment, central retinal apposition, vitreous incarceration into a surgical wound, or a breakthrough vitreous hemorrhage; increased IOP; retained lens material during cataract surgery; and intractable eye pain.

Quite frequently, a retinal detachment can be seen in the postoperative period, identified either

Fig. 6. B-scan echogram demonstrating a shallowly elevated, high-reflective membrane (large arrow) inserting into the optic nerve (ON) representing a shallow posterior retinal detachment. Note the moderate elevation of the choroidal detachment (small arrow). (Reprinted from Chu et al[16] with permission of the American Medical Association.)

on funduscopic examination or by echography. A distinction must be made, however, between retinal detachments of a serous origin and retinal detachments of a tractional or rhegmatogenous cause. Typically, serous retinal detachments are dome-shaped, low-lying, and situated over areas of choroidal hemorrhage. In contrast, traction retinal detachments are taut areas of retinal separation, with apparent areas of vitreoretinal traction. Rhegmatogenous retinal detachments are usually more elevated and bullous, and may not be overlying an area of choroidal hemorrhage. The presence of a rhegmatogenous retinal detachment in the postoperative period remains a common indication for surgical intervention. With regard to serous retinal detachments, we reported a series of 18 patients with massive SCH and central retinal apposition, including 12 patients with either an expulsive or a delayed cause.[16] Six of these 12 patients were noted to exhibit a shallow posterior serous elevation of the retina once the choroidal detachments began to decrease in elevation (Fig. 6). The retinal detachment in only one of these patients progressed to a more extensive rhegmatogenous retinal detachment; the other five detachments resolved spontaneously. Therefore, serous retinal detachments should be observed closely for regression or progression.

Central retinal apposition has traditionally been considered to be an absolute indication for surgical intervention. Berrocal reported that retinal surfaces in apposition can become fixed.[8] We reported the natural course of central retinal apposition without surgical intervention.[16] Echographically, all patients, including those who underwent intraoperative drainage, exhibited highly elevated hemor-

rhagic choroidal detachments with an appositional configuration. Follow-up examination revealed that the duration of central retinal apposition in SCH ranged from 10 to 25 days. A rapid decrease in elevation was noted after the third week, although slight choroidal elevation persisted peripherally for longer than 6 weeks in all these patients (Fig. 5). Despite this prolonged period of apposition of retinal surfaces, no evidence of persistent retinal adherence was noted either clinically or echographically. Therefore, central retinal apposition may be a relative rather than an absolute indication for early surgical intervention. In direct contrast to our findings, Reynolds et al had good results with early surgical intervention in patients with central retinal apposition.[56] In their study, 13 of 20 eyes with SCH with central retinal apposition underwent a secondary surgical procedure. Of these 13 eyes, six had a visual acuity outcome of 20/200 or better compared with none of the seven eyes that were observed. Several factors may be responsible for the discrepancy between the findings of Reynolds et al and ours. They did not specify the cause of SCH in their patients with central retinal apposition. If the unoperated eyes suffered an SCH after trauma, the prognosis would be poor regardless of surgical intervention. Also, they did not specify whether any of their patients with central retinal apposition had concomitant rhegmatogenous retinal detachments. Certainly, if the eyes that were simply observed had a concomitant retinal detachment, the final visual outcome would be expected to be poor. Finally, Reynolds et al followed up their patients for only 3 months, whereas we followed up patients from 6 months to 2 years.

Scott et al reported a large series of 51 patients with SCH and central retinal apposition of various causes.[58] Included in this study were patients who developed SCH intraoperatively, postoperatively, and after ocular trauma. Scott et al found no statistically significant difference in final visual acuity among patients who were observed versus patients who underwent secondary surgical intervention. An additional risk factor of poor visual outcome noted in this study was duration of central retinal apposition for longer than 2 weeks. Their data showed no difference in surgery versus observation; however, the authors suggest that early surgical drainage may be warranted in patients in whom the retinal apposition does not resolve promptly, because outcomes are uniformly poor in patients who have central retinal apposition for 2 weeks or more. The conclusions of this study are limited because of the small number of patients in each subgroup, the nonrandomized study design, and the fact that different surgical techniques were used by the treating physicians. Therefore, central

retinal apposition may be a relative, and not an absolute, indication for surgical intervention.

Becquet and coworkers studied 13 patients with delayed SCH. Eleven of 13 eyes underwent a secondary surgical procedure.[7] On the basis of their results, the authors concluded that not all SCHs need to be drained surgically. They did, however, advocate the use of gas or silicone oil tamponade if a secondary surgical procedure is performed.

Recently, Wirostko and coworkers studied the outcome of surgical management based on a new classification scheme of SCH severity.[72] Wirostko et al reported on 48 eyes undergoing surgical drainage of SCHs. Included in this study were patients who developed SCH intraoperatively, postoperatively, and after ocular trauma. The eyes were further subdivided into additional categories, based on whether central retinal apposition, vitreous incarceration in the wound, and/or retinal incarceration in the wound was present. Not surprisingly, poorer visual outcome was associated with increasing complexity of the SCH. Retinal incarceration was strongly associated with a poor prognosis. Of eyes with retinal incarceration, 63% progressed to visual acuity of no light perception compared to only 15% of eyes without retinal incarceration. In addition, 50% of eyes with retinal incarceration possessed an irreparable retinal detachment compared to 20% of eyes without retinal incarceration.

VII. Surgical Techniques

When reoperation is considered in patients with SCH, the surgical approach can be one of two choices: 1) drainage procedures to remove the SCH and to reestablish normal IOP; and 2) vitreoretinal surgery in combination with a drainage procedure to remove vitreous hemorrhage and/or retained lens material, to relieve vitreoretinal traction, and to reestablish the normal anatomic configuration of the posterior segment. Even when contemplating only a surgical drainage procedure, the anterior segment surgeon should generally seek consultation with a vitreoretinal surgeon. Commonly, posterior segment complications such as retinal dialysis and retinal detachment can arise during anterior segment approaches to drain SCH.

A. DRAINAGE PROCEDURES

Anterior segment approaches are usually restricted to drainage procedures with or without anterior vitrectomy. When a drainage procedure is considered, the optimal time for intervention can be critical for success. As previously mentioned, mean clot lysis time for an SCH has been reported to be between 7 and 14 days. Attempts to drain an SCH

before some degree of clot lysis has occurred are usually unsuccessful. Furthermore, vigorous attempts to remove clotted blood from the suprachoroidal space can result in additional injury to the globe. It is therefore recommended that drainage procedures in patients with SCH be deferred for 1 to 2 weeks, preferably with clot lysis confirmed by echography.

Verhoeff in 1915 recommended the use of drainage sclerotomies at the time of expulsive SCH to enable closure of the surgical wound and to reduce IOP.[69] He advocated the use of scleral punctures with a Graefe knife; furthermore, he suggested that scleral punctures by made as quickly as possible at the time of expulsive SCH, and that the scleral opening be V-shaped with excision of the apex of the "V" to facilitate postoperatively the continued escape of blood. The technique of postoperative drainage of SCH suggested by Vail in 1938 was not unlike the method first recommended by Verhoeff for intraoperative management of SCH.[21,68] Shaffer advocated the use of a T-shaped posterior sclerotomy, with diathermy to the lips of the wound to cause scleral shrinkage.[59] He also recommended that the anterior chamber be reformed with either an air bubble or saline injection. Recently, Lakhanpal challenged the conventional wisdom that drainage sclerotomies are indicated in the acute management of SCH.[36] He suggested that immediate sclerotomy during SCH is detrimental rather than helpful.

Several authors have suggested the use of drainage procedures in the postoperative management of SCH. The surgical principles of all these methods are essentially the same. Drainage sclerotomies are created in the quadrant(s) of the involved SCH. The IOP is then maintained by continuously injecting a vitreous substitute into the globe. Usually, an anterior chamber approach is recommended, as the majority of these eyes are aphakic or pseudophakic. The reestablishment of IOP by these methods facilitates the egress of lysed blood through the drainage sclerotomies in a controlled fashion. These methods are ideally suited for management of SCH in which there is little remaining vitreous in the eye, when vitreoretinal traction is absent, and no retinal detachment exists. In many cases of expulsive SCH, the vitreous at the time of initial surgery is expelled by the force of the SCH. The remaining vitreous in these eyes may cause vitreoretinal traction, increasing the risk of retinal detachment. In these cases, care must be taken when IOP is reestablished by instilling balanced saline solution or a vitreous substitute, because significant retinal traction can be created. In contrast, vitreous incarceration into the surgical wound is usually absent in delayed SCH. Therefore,

vitreoretinal traction is usually absent if the surgical wound is not violated in delayed SCH.

Several vitreous substitutes have been recommended for reestablishing IOP, all with distinct advantages as well as disadvantages. Both balanced saline solution and viscoelastic solutions have been advocated as vitreous substitutes.[6,18] Balanced saline solution can be instilled via a limbal approach with a gravity infusion system. Drainage sclerotomies are created posteriorly, in a radical fashion, before engagement of the infusion system. The anterior and posterior chambers are maintained with balanced saline solution as the choroidal blood is drained from the sclerotomies. The sclerotomies are then held open with forceps, allowing drainage of the suprachoroidal space as the eye is reformed with solution. A cyclodialysis spatula can also be gently introduced into the suprachoroidal space to facilitate removal of persistent blood clots. If a viscoelastic solution is used, it can be injected into the globe from a limbal approach via a syringe. These methods afford some visualization of the posterior chamber during drainage of suprachoroidal blood. The disadvantage of these methods, however, is the potential of uncontrolled IOP. Syringe injection of viscoelastic agents is not ideal because maintenance of IOP is imprecise. A gravity infusion system for balanced saline infusion is certainly preferable; however, care must be taken not to allow the globe to become hypotonous (which may lead to recurrent SCH) or for the IOP to become too great (which may result in retinal incarceration). Eller et al[23] and Birt and Berger[10] advocated using an anterior chamber maintainer to form the anterior chamber during drainage of suprachoroidal blood, while maintaining a uniform IOP.

Instead of balanced saline solution or viscoelastic agents, sterile air can be used to hydraulically aid in the draining of suprachoroidal blood.[2,27] Again, radial incisions are made in the sclera into the suprachoroidal space. The eye is then insufflated with sterile air by using a continuous-infusion air pump through a 25-, 27-, or 30-gauge needle inserted through the limbus. The air pump insufflation pressure is preset to 20 to 30 mm Hg. The sclerotomies are then held open with forceps, allowing drainage of the suprachoroidal space as the eye is reformed with air. Again, a cyclodialysis spatula can be gently introduced into the suprachoroidal space to facilitate removal of persistent blood clots. When drainage is complete, the insufflation needle is withdrawn and the sclerotomies can be closed with sutures. A major advantage of this technique is the use of a continuous-infusion air pump rather than a syringe. The IOP can be maintained at a predetermined level, thereby preventing both hypotony and exces-

sive pressure. A potential disadvantage of this technique is the loss of detailed visualization of the posterior segment because of air-fluid interface reflections. This method may therefore make it difficult to identify peripheral retinal tears and persistent vitreoretinal traction at the time of surgery.

B. VITREORETINAL SURGICAL APPROACHES

When retinal detachment, vitreoretinal traction, vitreous hemorrhage, and/or dislocated lens fragments are present in the setting of SCH, vitreoretinal surgery at the time of the SCH drainage procedure is usually advisable. Although no randomized prospective studies are available, this surgical approach may be preferable to drainage-only procedures, since vitreoretinal surgery can directly address many of the underlying pathologic changes in this condition. Indeed, vitreoretinal surgery will reestablish the normal anatomic configuration of the globe by controlled removal of vitreous and vitreous debris (i.e., blood, lens remnants), relief of vitreoretinal traction, and reattachment of detached retina.

When vitreoretinal surgery is planned in combination with a drainage procedure, the sequence of surgical maneuvers is extremely important. Typically, in the presence of an SCH, the normal anatomic location of the pars plana, anterior retina, and vitreous base is distorted. Entry into the posterior segment of the globe via a pars plana approach can be dangerous and can result in iatrogenic damage to the anterior retina. Drainage of hemorrhage from the suprachoroidal space is therefore initially required before any pars plana incisions are created. The choice of the vitreous substitute used to reestablish IOP during this drainage procedure is usually limited to clear liquid solutions to enable good visualization during vitrectomy. Both balanced saline solution and viscoelastic agents can be used to accomplish drainage of suprachoroidal blood.

Recently, perfluorocarbon liquids have been recommended as a new surgical adjunct in cases of complex vitreoretinal pathology.[20] These agents are clear liquids with a specific gravity higher than that of water. When instilled into the vitreous cavity, these liquids will "sink" to the posterior pole of the eye. They thus force all other ocular fluids, both within the vitreous cavity (i.e., aqueous, vitreous, and saline) and within the suprachoroidal space (i.e., hemorrhage) anteriorly. These physiochemical properties make them ideally suited for use in drainage of SCH. Indeed, perfluoroperhydrophenanthrene, a perfluorocarbon liquid, has been used successfully in the drainage of SCH.[20,55] Unlike the above-mentioned methods, when a perfluorocarbon liquid is used, the drainage sclerotomies should be placed anteriorly, approximately 4 mm off the limbus. A 30-gauge needle attached to a syringe containing perfluorocarbon liquid is then introduced through the limbus. This liquid material is then slowly injected, whereby it will immediately move posteriorly. As it fills the posterior chamber, the perfluorocarbon liquid will flatten the posterior pole while displacing suprachoroidal blood anteriorly, thereby allowing more complete removal of liquified blood through anteriorly placed sclerotomies. An added benefit of using perfluorocarbon solutions under these circumstances is that this liquid will tamponade the retina, while forcing vitreous and vitreous debris anteriorly. This allows for easier removal of vitreous and/or retained lens fragments during vitrectomy, while protecting the retina from iatrogenic damage.

After the SCH has been drained with the use of perfluorocarbon liquid, the normal anatomic relationships can be reestablished. A conventional three-port pars plana vitrectomy configuration can then be created. The settled perfluorocarbon liquid is left undisturbed, and residual anterior vitreous can be removed by vitrectomy. Vitreous strands causing vitreoretinal traction can be severed. If a rhegmatogenous retinal detachment is present, the retinal break can then be treated with retinopexy (photocoagulation or cryopexy). Finally, the perfluorocarbon liquid can be removed either via a fluid-fluid exchange with infusion of balanced saline solution or via a perfluorocarbon liquid-air exchange. A scleral buckling procedure can then be performed if residual vitreoretinal traction persists, or if support to areas of retinal breaks is required. Internal tamponade with a long-acting intraocular gas or silicone oil may also be required.

VIII. Prognosis

A. EXPULSIVE SUPRACHOROIDAL HEMORRHAGE

In cases of expulsive SCH, in the absence of a rhegmatogenous retinal detachment, both early surgical intervention and observation and medical management have been advocated. Taylor reported severe visual loss in four of five patients with expulsive SCH.[65] Despite early surgical drainage of the hemorrhage in a study conducted by Lakhanpal et al, only one of five patients with an expulsive SCH regained visual acuity greater than 20/200.[37] Lambrou et al also reported early surgical drainage in six patients with SCH; only one patient, however, recovered visual acuity to the 20/200 level.[39] Welch et al were somewhat more encouraged by early surgical drainage in expulsive SCH in their group of patients with vitreous, but not retinal, incarceration.[70] Five of

seven patients who underwent early surgical drainage recovered visual acuity of 20/200 or better, although some patients required subsequent vitreoretinal surgery for retinal detachments. In the series by Scott et al, 27.5% of patients with expulsive SCH and central retinal apposition had visual acuity of no light perception on final follow-up, and only 29.4% of patients achieved their prehemorrhage visual acuity or a final visual acuity of 20/200 or better.[58] Nine of 18 patients with expulsive SCH in this study by Scott et al underwent secondary pars plana vitrectomy; however, this additional surgery did not have statistically significant visual benefit.[58] Four of our 18 patients developed SCHs intraoperatively.[16] All four patients underwent intraoperative placement of sclerotomies to facilitate drainage and wound closure, as previously described. All of these patients, however, had rapid reaccumulation of the hemorrhage. The reaccumulated hemorrhages were allowed to resolve spontaneously in all of these cases. All four patients obtained good initial visual acuity after resolution of the hemorrhage; however, they all had a poor visual outcome because of subsequent retinal detachment without repair or progression of their underlying ocular disease. Therefore, it does not appear that all patients suffering from an expulsive SCH should be subjected to a secondary surgical procedure. Secondary surgery, however, should be contemplated on an individual level, based on clearly defined indications for intervention.

B. DELAYED SUPRACHOROIDAL HEMORRHAGE

Abrams et al,[2] Lakhanpal et al,[37] Gressel et al,[30] and Frenkel and Shin[26] all believed that early surgical drainage may be beneficial in patients with delayed SCH. Gressel et al reported five cases of delayed SCH, with only one patient undergoing early surgical drainage.[30] Interestingly, this patient was the only patient in their study who maintained a visual acuity better than hand motions. Ruderman et al described nine patients who underwent surgical drainage within 72 hours of an SCH after filtration surgery.[57] Of these nine patients, only two regained prehemorrhage visual acuity. Ariano and Ball presented five cases of delayed SCH, with three patients undergoing surgical drainage.[4] Two of these three patients recovered their prehemorrhage visual acuity. Of the two remaining patients in that study, one patient maintained prehemorrhage visual acuity without the need for surgical drainage. Abrams et al reported the greatest success with early surgical drainage in patients with delayed SCH.[2] In their study, three of seven patients maintained prehemorrhage visual acuity, and two of seven patients actually had a final visual acuity that was better than their prehemorrhage visual acuity. Frenkel and Shin described two patients who recovered baseline visual acuity after prompt surgical drainage.[26] In direct contrast to the above studies, we observed that good initial visual outcome can be attained without early surgical drainage in delayed SCH, and that echography may be a helpful adjunct in the treatment of patients in whom surgical drainage is warranted.[16] Scott et al reported that only two of 20 patients with delayed SCH progressed to a visual acuity of no light perception, and 10 of these patients achieved their prehemorrhage visual acuity or a final visual acuity of 20/200 or better.[58] Eleven of the 20 patients underwent a secondary procedure to drain the SCH.

Eight of our 18 patients developed a delayed SCH.[16] In this group, all five patients who were followed up without surgical drainage maintained their prehemorrhage visual acuity. Two patients underwent early surgical drainage, and both maintained preoperative visual acuity. Despite surgical drainage and sophisticated vitreoretinal surgery at 1 month, the eye of another patient developed phthisis bulbi. Our findings suggest that not all patients with delayed SCH need to undergo early surgical decompression of the choroidal hemorrhage. Interestingly, both Gressel and coworkers[30] and Abrams et al[2] mentioned that delayed SCH may be more serous than expulsive SCH and therefore easier to drain. We did not find this to be the case either clinically or echographically.

IX. New Frontiers

Despite our increased knowledge of the causes of SCH, both expulsive and delayed SCH continue to be devastating conditions. Several new treatment prospects hold promise in the treatment of SCH. As mentioned above, the use of perfluorocarbon liquids to help drain suprachoroidal blood is a tremendous surgical tool for the vitreoretinal surgeon.[20] Another new treatment modality is the use of intravenous tissue plasminogen activator to accelerate clot lysis.[40] This form of treatment is experimental, and its clinical benefit remains to be proved. Finally, the use of silicone oil tamponade after vitreoretinal surgery for SCH has been recommended[3], but should be reserved for only the most desperate cases.

X. Summary

Suprachoroidal hemorrhage, both expulsive and delayed, is a rare, but severely debilitating complication of intraocular surgery. A thorough understanding of the pathophysiology, risk factors, and clinical outcome of patients who develop SCH can help the ophthalmic surgeon successfully avoid this complication in patients at risk, as well as help in the postoperative management in those patients who develop SCH during or after intraocular surgery.

Methods of Literature Search

MEDLINE/PubMed and Ovid databases were searched, covering the years 1970 through 1998. Search words were *choroidal hemorrhage, ocular surgery, risk factors,* and *cataract extraction/adverse effects.* Articles involving the development of SCH in the perioperative period were included in this article. Articles involving the development of choroidal hemorrhage secondary to trauma or age-related macular degeneration were excluded, as were articles that combined SCH with other entities, such as choroidal effusions.

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Outline