Retinal detachment surgery : pre and postoperative prognostic factors

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Voor Luuk en Nina

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Abbreviations

AAD	angle-to-angle distance
AC	anterior chamber
ACD	anterior chamber depth
ACW	anterior chamber width
AS	anterior segment
CV	coefficient of variation ((SD/mean cell area) x 100)
C_3F_8	perfluoropropane
D	dioptre
DD	disc diameter
ECD	endothelial cell density
ELM	external limiting membrane
F-U	follow-up
GAG	glycosaminoglycans
GP	general practitioner
GRT	giant retinal tear
IOL	intraocular lens
IOP	intraocular pressure
ILM	internal limiting membrane
NFL	nerve fibre layer
MRI	magnetic resonance imaging
OCT	optical coherence tomography
PDR	proliferative diabetic retinopathy
PPV	pars plana vitrectomy
pIOL	phakic intraocular lens
PVD	posterior vitreous detachment
PVR	proliferative vitreoretinopathy
RPE	retinal pigment epithelium
RD	retinal detachment
RRD	rhegmatogenous retinal detachment
RO	referring ophthalmologist
SB(S)	scleral buckling (surgery)
SD	standard deviation
SF_6	sulphur hexafluoride
SO	silicone oil
SOR	silicone oil removal
SRF	subretinal fluid
TRC	tertiary referral centre
VA	visual acuity

Chapter 1

General Introduction

Retinal detachment: history, pathophysiology and surgical treatment

Retinal detachment is a sight-threatening condition, which requires immediate treatment in order to prevent blindness. The photoreceptor of the retina becomes separated from the retinal pigment epithelium (RPE) with fluid accumulation in the intervening space. Sight deteriorates particularly when the centre, the macula[1], is involved in the retinal detachment. Treatment should take place within one week after the macula becomes detached to prevent further deterioration in sight.[1] The annual incidence of retinal detachment is 5 to 18.2 per 100,000 persons[2-10] and the average age is 70 years. Symptoms comprise flashes of light in the extreme periphery of the eye, floaters, visual field loss and veiled vision.[11] There are several treatment options, which all have advantages and disadvantages.[12-18]
In order to determine the best possible treatment, the aim of this study was to answer the following questions: Why do patients with retinal detachment delay consulting a doctor? What is the best treatment for patients with rhegmatogenous retinal detachment? Does the treatment have side-effects and what are the consequences for the rest of the eye?

Aims and outlines of the thesis

The **first chapter** describes the anatomy and physiology of the eye, with particular attention to the retina and retinal detachment. This is followed by a description of the various treatments available for retinal detachment.
Chapter 2 analyses the reason why patients with symptoms of retinal detachment, such as flashes of light, floaters or visual field loss, delay seeking medical treatment.
Treatment for rhegmatogenous retinal detachment with scleral buckling surgery is analysed in **Chapter 3**. Attention is paid to functional recovery and risk factors that hinder recovery.
Chapters 4 and 5 describe the anterior chamber depth, axial length and diplopia after scleral buckling surgery for rhegmatogenous retinal detachment.
Chapter 6 analyses vitrectomy with silicone oil tamponade as the best treatment for a giant retinal tear and addresses the question of whether an encircling element is necessary.
Chapter 7 describes the anatomical and functional results following removal of the silicone oil and the risks in the case of re-detachment and deterioration in visual outcome.
Chapter 8 investigates whether vitrectomy with silicone oil tamponade causes changes to the endothelial cell density of the cornea in different groups of patients in whom the lens had been left in situ, replaced or removed.
Chapter 9 contains a general discussion and a summary of the results.

THE ANATOMY OF THE EYE

The eye is a sensory organ that receives signals from the environment and transmits them to the brain where they are converted into perceptions and committed to memory. From the outside, particularly the eyelids, cornea, iris and sclera are visible (Figure 1.1).

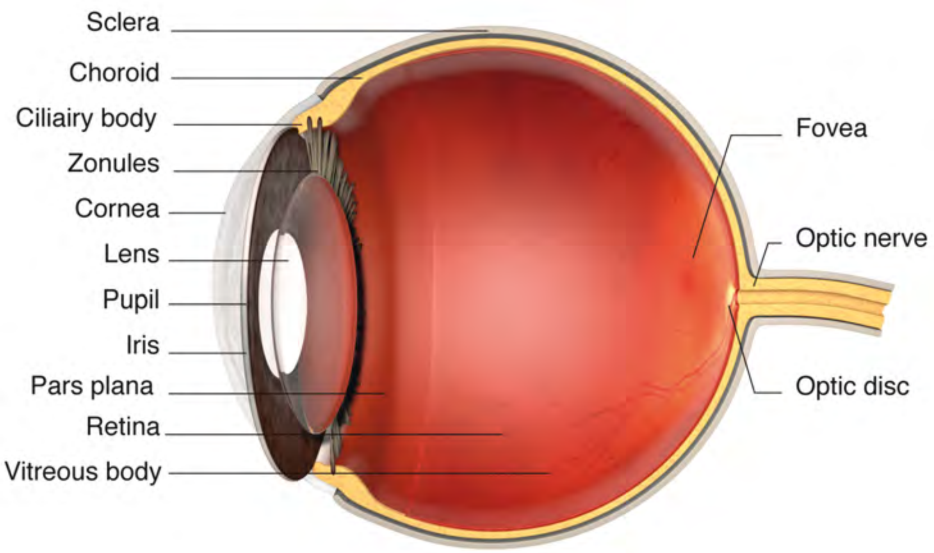

Figure 1.1: Anatomy of the eye

The cornea

The cornea is the transparent anterior part of the eye. It borders with the sclera that covers the rest of the eyeball. The cornea has a thickness of about 500 micrometres and is composed of five layers. The surface layer comprises thin epithelium.[19] Just below, lies Bowman's membrane. The middle layer is the stroma, which consists of a network of water and collagen fibres; it forms 90% of the thickness of the cornea. The next layer, the Descemet membrane, is tough and thin. Resting on this Descemet membrane is the inner layer, the endothelium (see Figure 1.2).

The endothelium forms the posterior side of the cornea and is one cell layer thick. Endothelial cells generally have a hexagonal shape. In addition, the endothelium contains pumps that eject fluid from the cornea to maintain a clear, transparent area where light can enter the eye.[20] With increasing age, the number of endothelial cells and the symmetry of their pattern decreases.[19] Very limited cell division occurs in endothelial cells and this too decreases with age by an average of 0.3-0.6% per year. After treatment

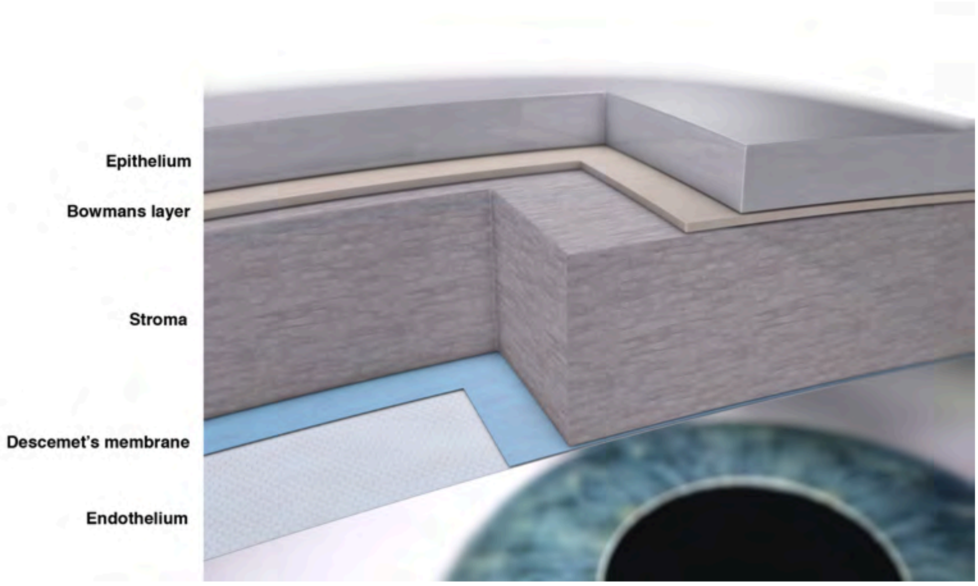

Figure 1.2: Corneal layers

for cataracts, the decrease is 2.5% per year. A normal cornea generally has about 2,500-3,000 cells per mm^2. If the number of cells decreases to 700-500 cells per mm^2, the pumps no longer function adequately and the cornea can become thickened by oedema.

The lens

The lens, or crystalline lens, lies immediately behind the iris. Within the eye, the lens is situated between the cornea and the retina, or more specifically behind the iris and in front of the corpus vitreum. The lens is a virtual barrier between the anterior and posterior chambers of the eye. It consists of a lens capsule, a cortex and a nucleus. With advanced age, the lens becomes opaque in many people, so-called cataract development.

Corpus vitreum

Behind the lens there is a cavity that is completely filled with gel-like vitreous humour (corpus vitreum, vitreous body) that fills about 80 per cent of the eye volume. The vitreous body is acellular apart from a few cells (hylocytes) and is composed of an extracellular matrix of 98% water and macromolecules. These macromolecules are structural proteins and glycosaminoglycans (GAGs).[21] The structural proteins comprise collagen fibres and non-collagen proteins. The fibres run parallel to each other and are connected together as a sort of branching meshwork by chondroitin sulphate bridges.

There are different types of collagen fibre. The vitreous body mainly contains type II fibres, but to a smaller extent also type V/XI (hybrid type) and type IX. The average protein concentration of the healthy corpus vitreum is 0.5 mg/mL, consisting largely of albumin (60–70%).[22] GAGs are molecules with a sponge-like function. Water sticks to them and this contributes to the pressure-volume relationship. The most important GAG is hyaluronic acid, followed by chondroitin sulphate and heparin sulphate; the latter two are proteoglycans that bind to protein.[23, 24] This vitreous body is contained within a thin membrane called the hyaloid membrane.

The vitreous body is known to have three divisions: a central part (core vitreous), a peripheral part and the vitreous base. The peripheral part lies against the retina and comprises the outer layer of the vitreous. At this location the vitreous body has a thicker consistency than in the core. The collagen fibres are more densely packed together. The thin layer of vitreous body is about 100-200 micrometres thick. On the anterior side, just behind the lens, lies the anterior boundary of the vitreous, the so-called anterior hyaloid membrane.[21] The vitreous base is an area at the equator where the vitreous is firmly anchored to the eyeball. On the posterior side, the posterior hyaloid membrane forms the boundary between the vitreous body and the retina.

The collagen fibres in the posterior part of the vitreous are connected to the inner layer of the retina (the so-called internal limiting membrane) by laminin and fibronectin. Clinically, this complex is referred to as the vitreoretinal interface.

For the greater part, the peripheral cortical vitreous is not attached firmly to the inner layer of the retina. However, at several locations there are foci of strong adhesions, for example around the optic disc. Weak adhesions are present around the fovea that are easily broken. In addition, there are weak adhesions around the blood vessels and very strong adhesions at the vitreous base. At this location, the vitreous cannot become completely detached due to the strong adhesions. The vitreous base lies over the ora serrata (the junction between the retina and ciliary body): 1 to 2 mm on the anterior side and 3 to 4 mm on the posterior side of the ora serrata.

In the case of abnormal pathological structures, such as scarring and weak retinal regions, the vitreous is more firmly attached.

With increasing age, there is decreasing elasticity of the fibres in the vitreous. The collagen fibres are partly broken down, they lose their structural pattern (unequal distances between the fibres) and they clump together. Diverse pockets of fluid develop in the vitreous. At the age of about 60 years, the vitreous body starts to condense and liquefy. The macromolecular structure of the vitreous body changes. Liquefaction of the vitreous is also known as synchysis.[25]

The retina

The inside of the eye is lined by the retina, the light-sensitive layer of the eye. The retina comprises about 126 million sensory cells. These cells absorb the light that enters the eye.

They can be divided into rods and cones: the former are sensitive to dark and light changes, while the latter can detect colour differences. During daylight hours, vision occurs with the central point of the retina, the macula lutea, where most of the cones are located. The macula enables us to perceive tiny details. In the dark, our vision moves slightly to the periphery of the central point where there are more rods and fewer cones. The retina outside the macula is commonly divided into a few general regions. The retina around the equator is called the equatorial retina, while the region anterior to this is called the anterior, or peripheral retina. In the far periphery, the border between the retina and the pars plana is called the ora serrata. The layers of the retina can be seen clearly in cross-sectional histological preparations. Their order from the inner to the outer retina is (Figure 1.3): internal limiting membrane (ILM), nerve fibre layer (NFL; the axons of the ganglion cell layer), ganglion cell layer, inner plexiform layer, inner nuclear layer, outer plexiform layer, outer nuclear layer (the nuclei of the photoreceptors), external limiting membrane (ELM), rod and cone inner and outer segments.

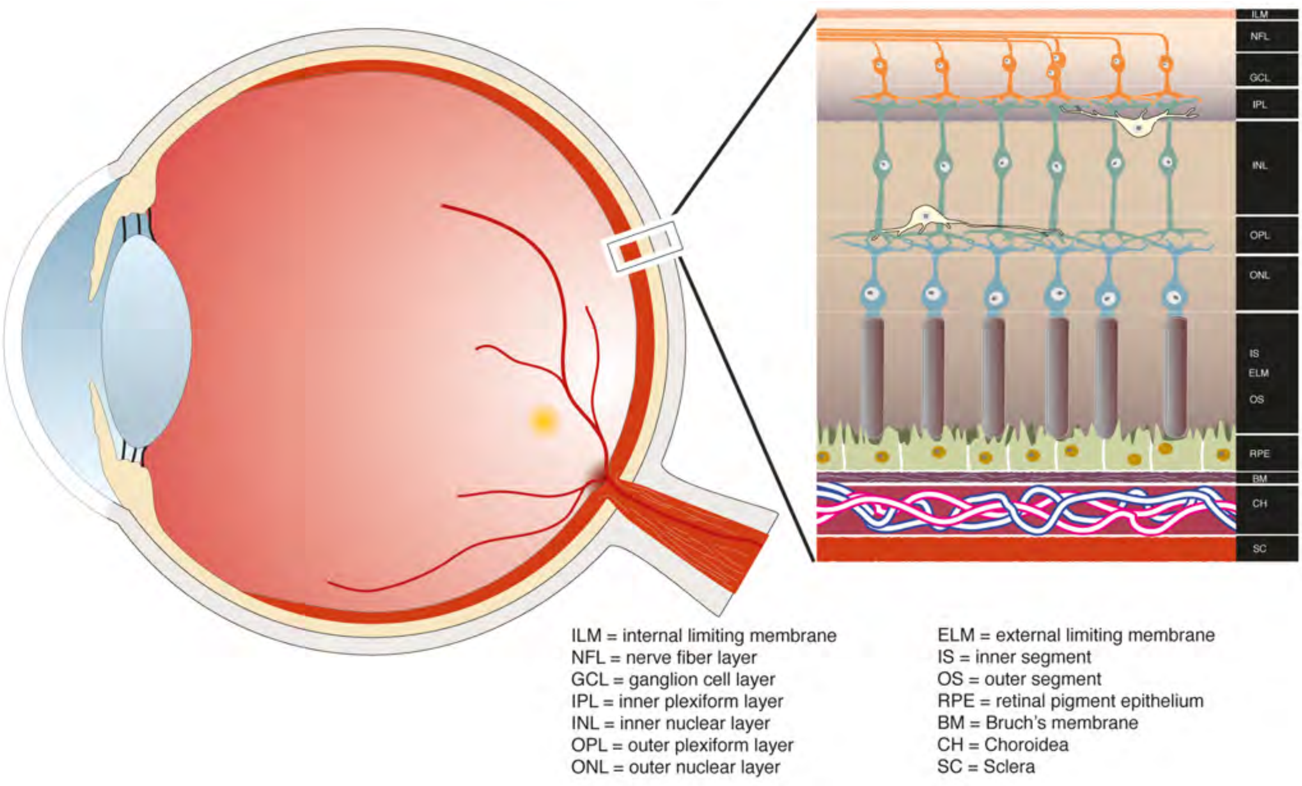

Figure 1.3: Retinal layers. Adapted from thesis Dr. L.P.J. Cruysberg, with permission.

The extraocular muscles

Each eye has six muscles: four recti and two oblique. The four recti muscles are attached to the superior, inferior, lateral and medial sides of the eyeball. They move the eye in superior, inferior, lateral and medial directions. The oblique muscles move the eyes obliquely upwards or downwards. One end of an extraocular muscle is attached to a ring (annulus tendineus) at the back of the orbit, while the other end is attached to the eyeball. Eye movements are generally well-conjugated, i.e. both eyes move simultaneously in the same direction, so that the image of the world is projected onto corresponding areas of both retinas. The muscles work in combination to coordinate sight in both eyes and prevent double vision. In addition, this produces deep sight that enables the estimation of speed and distance.

PATHOPHYSIOLOGY

Posterior vitreous detachment

With aging, the vitreous body shrinks and liquefied pockets develop. Other changes include cross-linkage between the collagen fibres, damage from free oxygen radicals and thinning of the network of collagen fibres. This decreases the stability of the vitreous body and causes the cortex to shrink away from the internal limiting membrane (ILM) of the retina. Once the gel has become liquefied, water can seep through the cortex and accumulate behind the vitreous membrane. The vitreous gel gradually shrinks and ultimately collapses. Detachment of the vitreous is referred to as vitreous collapse

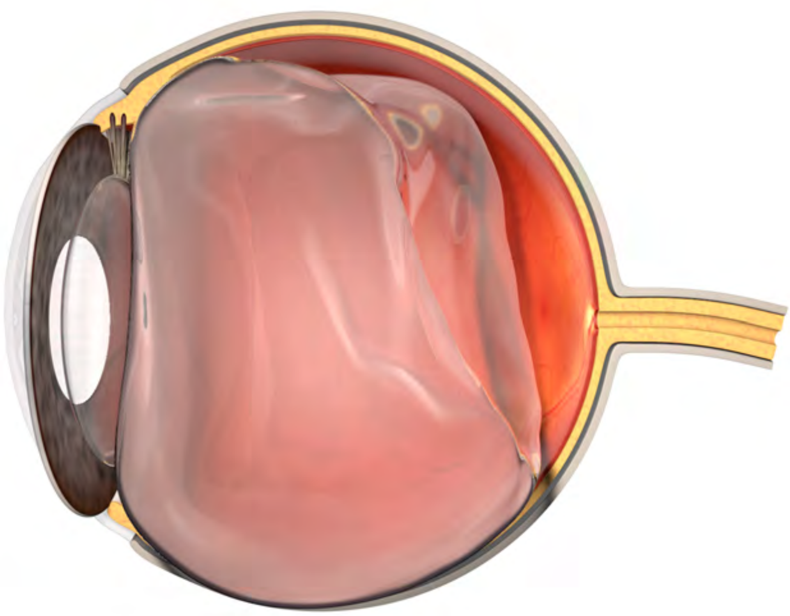

Figure 1.4: Posterior vitreous detachment

(syneresis), or acute posterior vitreous detachment (PVD)[26, 27] (Figure 1.4). The condition is acute and patients see floaters, which comprise the posterior side of the vitreous membrane (a Weiss ring). Due to traction of the vitreous on the retina, patients also see flashes of light.

Retinal tears and retinal detachment

If the vitreous gel sticks to the vitreous base, traction can occur due to spontaneous or traumatic PVD and cause retinal tears (Figure 1.5). About 15% of patients with acute symptomatic PVD have one or more retinal tears.[28, 29] Tears in symptomatic eyes run a high risk of rhegmatogenous (*rhegma* means tear in Greek) retinal detachment (RRD). In the case of RRD, the liquefied vitreous leaks through the tear into the subretinal space between the photoreceptor layer and the retinal pigment epithelium (RPE). If the fluid collects under the retina, this is referred to as subretinal fluid (Figure 1.6). Gradual sight loss occurs from the periphery to the centre. Further seepage of the fluid under the retina may cause detachment of the macula. Patients become acutely aware of losing their sight if the macula also becomes detached. The condition causes considerable irreparable visual acuity loss, possibly because the fovea loses its own retinal blood supply and becomes completely dependent on the blood vessels in the choroid. Tears differ from one patient to another. The most common tears are horseshoe tears. There are also wide differences in the number and size of the tears between patients. If a very large tear spans more than three clock hour positions, then this is referred to as a giant retinal tear.[30, 31]

Risk factors for developing rhegmatogenous retinal detachment are: myopia, trauma, cataract surgery,[32] family history and retinal detachment surgery to the contralateral eye.[32-34]

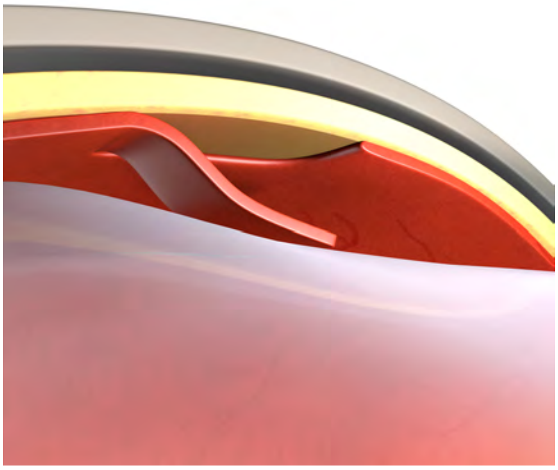

Figure 1.5: Retinal tear due to posterior vitreous detachment

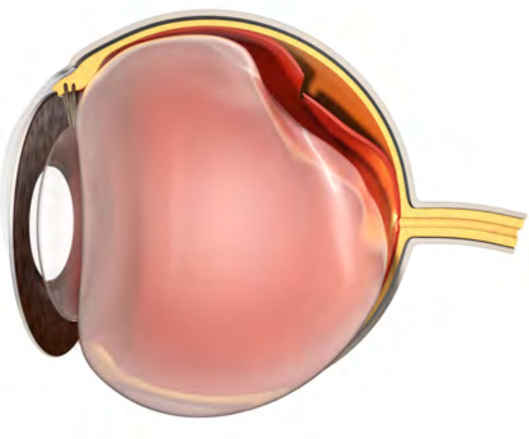

Figure 1.6: Retinal detachment. Fluid collection beneath the retina is referred to as subretinal fluid

Treatment for retinal detachment

Several treatments are available for retinal detachment. The choice of treatment is determined for each patient individually. Treatment options comprise: pneumatic retinopexy, scleral buckling surgery or vitrectomy.

Pneumatic retinopexy

In pneumatic retinopexy, a bubble of gas is injected into the eye and cryocoagulation is performed at the location of the tear in cases of highly localised retinal detachment without any other abnormalities.[16, 35-42] This procedure in a slightly different form was first performed by Vogt and Gonin in 1919 using electrodiathermy instead of cryocoagulation.[43] In 1933, cryocoagulation was introduced by Deutschman and Bietti.[43] Some years later, in 1952, Rosengren progressed with the injection of intravitreal air.[44] In 1980, surgeons started to use expandable gas, e.g. sulphur hexafluoride (SF_6).

Scleral buckling surgery

At the majority of clinics, scleral buckling is performed in patients with less complex retinal detachment.[45-49] Suitable candidates are, for example, patients with phakia who have retinal detachment in one or two quadrants, often with one horseshoe tear or one or two small tears. In this procedure, an encircling band is sutured around the circumference of the sclera, under the ocular muscles. A grooved buckle is placed under the band at the level of the tear to create an indentation in the eye wall that causes the underlying choroid and sclera to press against the retinal tear and close it. Explants are made of either solid silicone rubber or silicone sponges. Buckles come in various types, for example, the radial that compresses the tear, or the circumferential that indents a larger area (Figure 1.7). To reduce the volume of subretinal fluid, transscleral puncture can be performed. Adhesion of the retina is achieved by applying cryocoagulation around the tear. A bubble of expandable gas can be injected into the eye to achieve stronger adhesion. The current technique is derived from scleral resection that was first described by Müller in 1903.[50] Further variations of altering the globe wall to push the choroid closer to the retina have been used in retinal re-attachment procedures. In 1918, Jules Gonin made the revolutionary hypothesis that retinal tears caused by vitreous traction constitute the

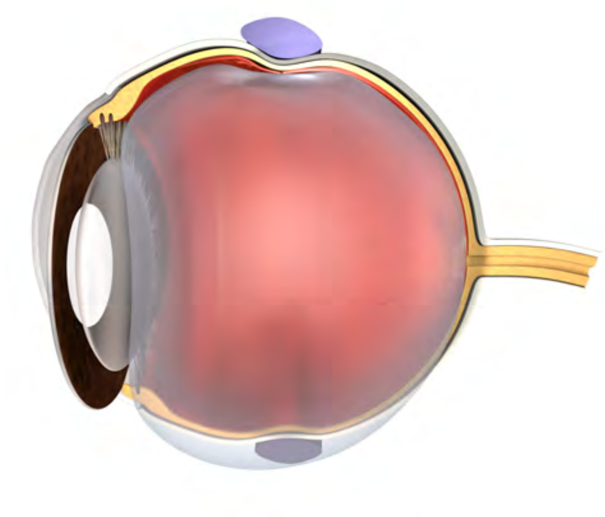

Figure 1.7: Scleral buckling surgery

pathogenic mechanism in retinal detachments.[51] This laid the groundwork for developing various techniques to release the vitreous traction by creating scleral indentation. These techniques include the use of polyethylene tubes, sutures, gelatine, human sclera, fascia lata and plantaris tendon.

Scleral buckling surgery was first performed by Jess in 1937. To create an indentation, he sutured material to the sclera, which was removed after 14 days. In 1952, Custodis presented his form of scleral buckling surgery, which comprised the creation of an indentation by placing a rod-shaped piece of the elastic material (polyviol, polymerized alcohol) on to the sclera and securing it with strong Supramid (non-absorbable synthetic) sutures. In accordance with his expectations, the tears were sealed mechanically by the protruding choroid. The subretinal fluid was resorbed so quickly that in most cases, the retina became re-attached within 12 to 24 hours, which made it possible to mobilize the patient on the second or third postoperative day. This approach revolutionized retinal detachment surgery and postoperative care.[52]

Vitrectomy

Pars plana vitrectomy (PPV) is generally performed in the more complicated cases, e.g. severe preoperative proliferative vitreoretinopathy (PVR),[53] giant retinal tears (GRT)[36 37] and vitreous haemorrhage. Currently, there are discussions about whether it should be applied more often to treat RRD.[54-58]

PPV is the surgical removal of vitreous through three small openings in the pars plana (Figure 1.8). The openings are made 3-5 mm from the corneal limbus.[59, 60] They can be of various sizes and entered with or without trocars; they can be self-sealing or non-self-sealing. One of the openings is used for fluid infusion to maintain pressure within the eye. The other two openings enable the surgeon to work bimanually. An implantable high intensity fibre optic light source is used to illuminate the inside of the eye during surgery. It can be inserted through one of the openings, or a so-called chandelier can be used if the surgeon wishes to use two instruments simultaneously. This high intensity fibre optic light source does not induce thermal damage or phototoxicity.[61] The surgeon uses a specialised surgical microscope with a contact lens or a wide viewing system, which provides increased

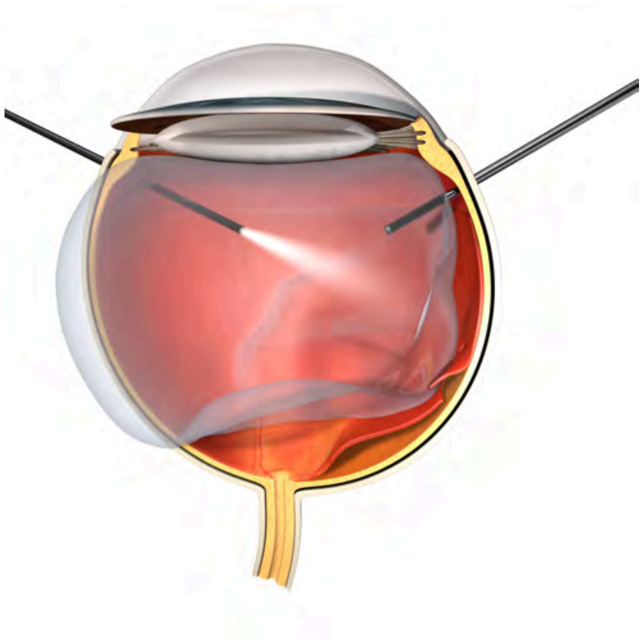

Figure 1.8: Vitrectomy

magnification of the retina and vitreous. During the operation, the retina can be flattened using gas or fluids, such as perfluorocarbon liquid. In order to re-attach the retina after the procedure and to create scar tissue, endolasercoagulation can be used, or cryocoagulation. To maintain some degree of pressure on the retina, a gas bubble or oil can be injected. Silicone oil is used to ensure continuous complete support of the retina.

Various intraocular tamponades can be used, which include air or expandable gases, such as short-acting or long-acting gas. The gases are gradually resorbed, so no further surgery is required. Silicone oil, however, does have to be removed surgically. This can be accomplished in various ways, but the pressure within the eye must be maintained. One method is to place an infusion via the pars plana, or via the anterior chamber. The latter is only possible in patients with aphakia.

Surgical instruments to perform PPV were invented by Machemer. He first used them in 1970 to operate on a patient with vitreous haemorrhage.[43, 44, 46-48] Since then, further developments have been made and in 1982, Grieshaber introduced a new instrument (*the Grieshaber instrument)* that simultaneously cuts and aspirates.[43, 48-55] Progress has continued with a succession of new vitrectomy instruments, while PPV[59, 60, 62, 63] has been followed by one of the most revolutionary developments in vitreoretinal surgery over the past few years: transconjunctival sutureless PPV. The introduction of transconjunctival microincision vitrectomy surgery with 23, 25 or 27 gauge instrumentation has potential advantages over traditional 20-gauge PPV. These advantages include faster wound healing, less conjunctival scarring, shorter surgical procedure, elimination of astigmatism, improved patient comfort and less postoperative inflammation.[64-70]

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Chapter 2

Patient ignorance is the main reason for treatment delay in primary rhegmatogenous retinal detachment in the Netherlands

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Abstract

Aims: To quantify and evaluate the causes for treatment delay in patients with rhegmatogenous retinal detachment who were scheduled to undergo retinal detachment surgery. Treatment delay must be kept to a minimum in progressive vision-threatening conditions.

Material and methods: Consecutive patients (n=205) with symptoms of primary rhegmatogenous retinal detachment were interviewed at the tertiary referral centre between June 2006 and June 2007. Five categories of delay were identified: "patient delay", "general practitioner delay", "referring ophthalmologist delay", "delay at the tertiary referral centre" and "delay until surgery at the tertiary referral centre". Overall delay was also calculated.

Results: 186 eyes were included in the analysis. Median overall delay between the initial symptoms and surgery was 10 days. Almost 60% of the overall delay was caused by the patient or the general practitioner. In more than 50% of the patients, the delay was due to unawareness and/or unfamiliarity with the symptoms. Median patient delay was significantly lower in the patients with vitreous haemorrhage and in the patients with a history of rhegmatogenous retinal detachment in the fellow eye.

Conclusion: The major cause for delay was the patients' unawareness and unfamiliarity with the symptoms of retinal detachment.

Introduction

Rhegmatogenous retinal detachment (RRD) is caused by a retinal tear and the subsequent leakage of intraocular fluid and liquefied vitreous into the subretinal space. This causes separation of the neuro-retina from the retinal pigment epithelium.[1] If left untreated, most RRDs will progress to complete detachment and vision loss in the affected eye.[1] The annual incidence of RRD is about 10 per 100,000 persons.[2, 3] Predictors of functional and anatomical success after primary RRD repair were found to be: more than 6 days of vision loss,[4] macular involvement and the size of the detachment area.[3-9] As the interval between the first symptoms noted by the patient and the timing of surgery is of critical importance to all these factors, our aim was to investigate the reasons for treatment delay.

Subjects and Methods

In this prospective study, we included all consecutive patients with primary RRD, who were referred to the Department of Ophthalmology at the University Hospital Maastricht between June 2006 and June 2007. This hospital department functions as a tertiary referral centre (TRC), especially for retinal detachment cases. Patients with secondary retinal detachment were excluded.

The study was approved by the institutional ethics committee. Patients were informed about the study procedure and gave informed consent. The study was conducted in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

Preoperative clinical characteristics of the patients were collected: age, sex, preoperative visual acuity, myopia (defined as spherical equivalent of -6 dioptres or more), previous intraocular surgery (i.e. cataract and retinal detachment in the fellow eye), the number of retinal quadrants detached, central macular (foveal region) involvement (measured by optical computer tomography (OCT)) and the number of days of macular involvement. The latter was defined as the subjective duration of the sudden deterioration in visual acuity and/or visual field loss. Clinical findings were also noted: the presence of proliferative vitreoretinopathy (PVR) graded according to the classification of RRD,[10] the type and localization of the retinal tear, the number of tears, the presence of vitreous haemorrhage and the type of surgery (scleral buckling or vitrectomy).

Patients were interviewed preoperatively by the author or co-authors (F.G., J.H., L.K.) using a questionnaire (see Table 2.1). If and when necessary, the questions were clarified by the interviewers. Patients were requested to carefully estimate the time interval in days since their initial symptoms. We helped them to relate their answers to a significant event, such as a birthday. This method enabled more precise estimation of the delay, at least to within a few days, even when the event had occurred more than two months previously. Patients who could not give clear answers, or were uncertain, were excluded.

Table 2.1: Questionnaire

Have you experienced any flashes?	Yes/No	For how long?.....................days
Have you experienced any floaters?	Yes/No	For how long?.....................days
Have you experienced any visual field loss?	Yes/No	For how long?.....................days
Have you experienced an acute sight loss?	Yes/No	For how long?.....................days
When did you first contact the general practitioner / ophthalmologist? .... days ago Reason for delay:	Patient-related delay ....days	
When did your general practitioner/ophthalmologist refer you? .... days ago Reason for delay:	General practitioner delay...days	
When did the ophthalmologist refer you to our hospital? .... days ago Reason for delay:	Referring ophthalmologist delay............................days	
When did the retinal surgeon at the University Hospital Maastricht (UHM) decide to operate (ask retinal surgeon) ? ……………………………. days ago Reason for delay:	Our out-patient clinic delay ….............................….days	
On what date did the surgery take place ? (this information was retrieved from the admission documents) ……………………………. days ago Reason for delay:	Delay before surgery ……….....................….............days	
	Total delay...days	

To be filled in after ophthalmological examination by the ophthalmologist or retinal surgeon:

Visual acuity……………………………………………………………….

Funduscopic examination: PVR A / PVR B / PVR C

Foveal involvement Yes/No? (how long).......................................days

Number of tears…………/ horseshoe/hole/oral dialysis/giant retinal tear

Ophthalmological history;……

Pseudophakia? Yes/No

Fellow eye retinal detachment Yes/No

Myopia? Yes/No dioptres: SE…………

Note: all interviews were conducted by the same three residents (FG, JH, LK)

Five different categories of delay were identified: "patient delay", "general practitioner (GP) delay", "referring ophthalmologist (RO) delay", "delay at the TRC" and "delay until surgery at the TRC". In addition, the overall (i.e. cumulative) delay was calculated per patient (see Table 2.2).

Information on three out of the five categories could be obtained from the patient (patient delay, GP delay, RO delay). "Delay at the TRC" was determined using admission documents. The exact date of surgery was derived from the surgical report. An example of a patient's time schedule (in days) is shown in Figure 2.1.

During the interviews, it became apparent that the patients had little or no knowledge and/or experience with the symptoms of posterior vitreous detachment (PVD) and/or retinal detachment (RD). For example, they explained to the interviewer that they had not considered the symptoms of flashes, floaters or visual field loss to be serious, so they had not made any immediate effort to consult a physician.

Statistical analysis was performed with the assistance of a statistician, using the SPSS software 13.0. The duration of delay (in days) was converted into a logarithmic scale to create an equal distribution. After this conversion, statistical analysis was performed using the independent sample T-test (significance was defined as $P < 0.05$).

Results

A total of 206 eyes in 205 patients with RRD (134 men (65.5%) and 71 women (34.5%)) were eligible for this study. No reliable answers could be obtained from seven out of the

Table 2.2: Definitions used in this study to specify the different categories of delay

Categories of delay	**Defined as number of days** ***from….until….***
Patient delay	*from* the first symptoms (flashes / floaters/ visual acuity loss and/or visual field loss) noted by the patient *until* the date of the first appointment with the GP, the RO, or the TRC
General practitioners (GP) delay	*from* the first date of the appointment with the GP *until* referral to either an RO or the TRC
Referring ophthalmologist (RO) delay	*from* the date moment the patient or GP contacted# the RO *until* the date the RO referred the patient
Delay at the tertiary referral centre (TRC)	*from* the date the patient, GP or RO first contacted# the department *until* the patient was scheduled for surgery
Delay until surgery at TRC	*from* the date was scheduled for surgery *until* the actual intervention
Overall delay	*from* the first symptoms noted by the patient *until* the actual intervention

Note: contacted# = GP or RO telephoned/patient visited

Table 2.3: Patients characteristics

Clinical variable		**N (n=186 eyes)**	**Percentage (%)**
Gender	Male	121	65.1
	Female	65	34.9
Eye	Right	99	53.2
	Left	87	46.8
Quadrants involved	1	57	30.5
	2	93	50.0
	3	22	11.8
	4	14	7.5
PVR	None	69	37.1
	PVR A	45	22.6
	PVR B	48	25.8
	PVR C	24	12.9
Macular involvement	Yes	103	44.1
	No	82	55.4
	Not known	1	0.5
Retinal tear	Horseshoe	96	51.6
	Hole	47	25.3
	Oral dialysis	4	2.2
	Tear >2 clock hours	10	5.4
	Missing	29	15.6
Cumulative size of the retinal tear in disc diameters	1	106	57.0
	2	33	17.7
	3	10	5.4
	>3	5	2.7
	Missing	29	15.6
Pseudophakic eye	Yes	69	37.1
	No	117	62.9
Fellow eye RD	Yes	15	8.1
	No	171	91.9
Myopia of > 6 Dioptres	Yes	33	17.7
	No	151	81.2
	Unknown	2	1.1
Type of surgery	Scleral buckle	138	74.2
	Pars plana vitrectomy	48	25.8

PVR: proliferative vitreoretinopathy, RD: retinal detachment

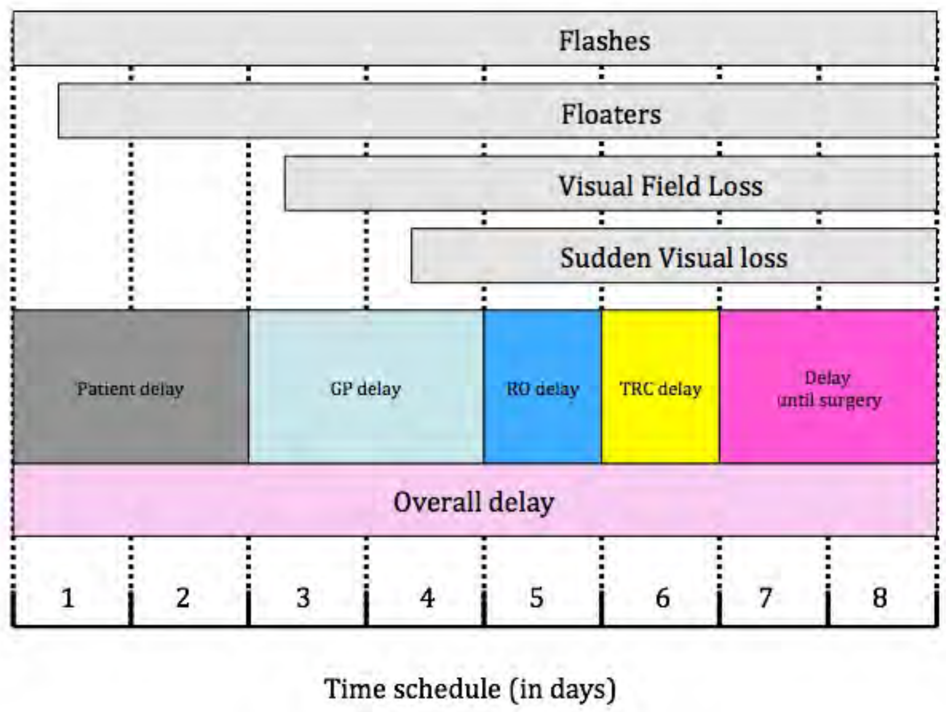

Figure 2.1: Example of the symptoms and different categories of delay in one patient over a period of 8 days

The first symptoms were flashes, followed by floaters, visual field loss and sudden vision loss.

"Patient delay" was two days; "general practitioner (GP) delay" was also two days; "Referring ophthalmologist (RO) delay" was one day; "tertiary referral centre (TRC) delay" was also one day; "delay until surgery at the TRC" was 2 days. Cumulative (or overall) delay was 8 days

TRC: our tertiary referral centre

205 patients. In two of these patients, the RRD was found by coincidence, so they were unable to recall the onset of their symptoms. In five other patients, the results of the interviews were unreliable due to dementia or confusion. Another 12 patients were also excluded (primary symptomatic PVD but no retinal tears; retinal tear treated with photocoagulation; no RRD present at initial visit – retinal tear appeared later). After excluding these 19 patients, a total of 186 eyes were included in the analysis. Clinical characteristics of the 186 patients are summarized in Table 2.3. There were 99 right eyes (53.2%) and 87 left eyes (46.8%). The male versus female pseudophakic ratio was 51 to 18 (3:1). Mean patient age was 59 years (range: 18-87 years). Fifteen fellow eyes (8.1%) had a history of RRD; 33 eyes (17.9%) were myopic. Surgical interventions comprised scleral buckling surgery in 138 eyes (74.2%) and primary pars plana vitrectomy in 48 eyes (25.8%).

The first symptoms experienced by the patients were: flashes (n=70; 37.6%), floaters (n=112; 60.2%), visual field defects (n=145; 78.8%) and vision loss (n=131; 70.4%). In patients complaining of flashes, floaters, visual field defects and sudden vision loss, the

Table 2.4: Reasons for delay

Categories of delay	Cause related to	Number n (%)	Median ± SD days	Mean delay	Min-max
Patient delay (n=186)	No delay	24 (13%)	0	0	0
	"Thought nothing serious"	104 (56%)	6 ± 31	17	1-230
	Wait until next working day	34 (18%)	1 ± 4	3	1-20
	Symptoms started on holiday	6 (3%)	7 ± 1	6	5-8
	Wait for appointment	6 (3%)	6 ± 4	6	1-11
	Other	11 (6%)	7 ± 17	15	2-50
	Total	186 (100%)	4 ± 25	12	0-230
General practitioner (GP) delay (n=119)	No delay	78 (66%)	0	0	0
	First available appointment	18 (15%)	1 ± 2	1	1-7
	Incorrect diagnosis	20 (17%)	14 ± 42	30	1-173
	Other	3 (3%)	1 ± 1	1	1-2
	Total	119 (100%)	0 ± 20	5	0-173
Referring ophthalmologist (RO) delay (n=127)	No delay	73 (57%)	0	0	0
	Next day	16 (13%)	1	1	1
	GP referral without urgency	17 (13%)	4 ± 12	9	2-45
	First had other treatment or additional diagnostic procedure ‡	9 (7 %)	21 ± 56	52	8-180
	Ophthalmology patient, made own appointment	6 (5%)	15 ± 12	18	7-35
	Patient was not able to come earlier	7 (6%)	4 ± 11	9	1-30
	Total	127 (100%)	0 ± 20	6	0-180
Delay at our tertiary referral centre (TRC) (n=186)	No delay	145 (78%)	0	0	0
	Appointment next day	18 (10%)	1	1	1
	Not referred as urgent	12 (6%)	1 ±15	7	1-53
	First had other treatment or additional diagnostic procedure ‡	1 (1%)	2	2	2
	Other	10 (5%)	3 ± 3	4	1-10
	Total	186 (100%)	0 ± 4	1	0-53
Delay before surgery at (TRC) (n=198)	No delay	5 (3%)	0	0	0
	Next day	83 (45%)	1 ± 0	1	1
	Over weekend	26 (14%)	2 ± 1	2	1-4
	Scheduled within……days	36 (20%)	7 ± 6	8	1-33
	Emergency surgery not possible on that day *	25 (13%)	2 ± 1	2	1-3
	Other	11 (6%)	2 ± 7	7	2-20
	Total	186 (100%)	1 ± 4	3	0-33

‡ Eyes with a PVD alone at first visit, which developed a RRD before next appointment
Eyes with retinal tears which were first photocoagulated, but later developed a RD
* operating theatre was not available, or patients were unsuitable for surgery under local and/or general anesthesia
For explanations and definitions of categories of delay see Table 2.2

Table 2.5: Delay in days in RD patients with or without concomitant vitreous haemorrhage

Categories of delay	With vitreous haemorrhage (n=7) Median ± SD (range) days	Without vitreous haemorrhage (n=179) Median ± SD (range) days
Patients delay‡	1 ± 1.1 (0-3)	4.0 ± 24.9 (0-230)
General practitioner (GP) delay	0.0 ± 0.4 (0-1)	0 ± 20.6 (0-173)
Referring ophthalmologist (RO) delay	0.5 ± 1.9 (0-4)	0.0 ± 20.5 (0-180)
Delay at TRC	0.0 ± 0.4 (0-1)	0.0 ± 4.3 (0-53)
Delay until surgery at TRC	2.0 ± 7.8 (1-20)	1.0 ± 4.0 (0-33)
Overall delay	3 ± 8.9 (3-22)	10 ± 34.9 (1-231)

‡ $p<0.05$ for patient delay between RD patients with (n=7) and without (n=179) concomitant vitreous haemorrhage
For explanations and definitions of categories of delay see Table 2.2

median patient delay was 6 days (SD ± 27), 15 days (SD ± 41), 5 days (SD ± 15) and 5 days (SD ± 16), respectively.

Median overall delay in all 186 eyes was 10 days (SD ± 35). Expressed as percentages, mean overall delay was 51% (SD ± 32), GP delay was 9% (SD ± 21), RO delay was 9% (SD ± 20). TRC delay was 5% (SD ± 15), delay until surgery was 26% (SD ± 25). Almost 60% of the overall delay was caused by patient delay and GP delay. Median patient delay was 4 days (SD ± 24.5; n=186); median GP delay was 0 days (SD ± 20.0; n=119). Median RO delay was 0 days (SD ± 20.2; n=127); median TRC delay was 0 days (SD ± 4.2; n=186). Median delay until surgery was 1 day (SD ± 4.2; n=186) (Table 2.4).

In 104 patients (55.9%) we found that patient delay was due to unawareness and/or unfamiliarity with the RRD symptoms. Median patient delay in this subgroup was 6 days (SD ± 31). Reasons for GP, RO and TRC delay are shown in Table 2.4.

Median patient delay was longer in the patients with a detached macula than in the patients without macular detachment: 4 days (SD ± 30 days) versus 4 days (SD ± 14 days), although this finding was not statistically significant (P=0.67). Patients with a history of retinal detachment in the fellow eye (n=15) sought medical treatment significantly earlier. Their median patient delay was 1.0 day (SD ± 8.0 days), whereas in the group without a history of retinal detachment (n=171) patient delay was 4 days (SD ± 25 days) (P=0.015). Median patient delay in the patients with vitreous haemorrhage (n=7) was significantly shorter than that in the patients without vitreous haemorrhage (P=0.032): 1 day (SD ± 1 day) versus 4 days (SD ± 25 days). This difference only applied to the category patient delay, but not to the other four delay categories (see Table 2.5).

No statistically significant differences in delay were found in relation with myopia, affected eye (right or left), age or symptoms.

Nine patients were delayed because the RO had made an incorrect diagnosis and the RRD had been overlooked. In one case, the initial decision at our centre was to apply alternative interventions (see Table 2.4), which were defined as ultrasonography. In one patient, the delay was probably due to a misunderstanding between the patient and the administration staff.

Discussion

Median overall delay between the first subjective symptoms and surgical intervention was 10 days. Almost 60% of this overall delay was due to patient delay and GP delay. In more than 50% of the patients, the delay was due to unawareness and/or unfamiliarity with the symptoms of PVD and/or RRD. For example, some patients explained that they did not consider the symptoms of flashes, floaters or visual field reduction to be serious, so they did not make an urgent appointment with a physician. Mean patient delay was significantly shorter in the patients with vitreous haemorrhage and in the patients with a history of RRD in the fellow eye. The acute vision loss (acuity and field) in the patients with vitreous haemorrhage was probably the reason why they sought immediate help. Patients who had a history of treatment for RRD in the fellow eye were more likely to recognize the symptoms of impending retinal detachment and go to a doctor straight away.

Our data were collected by interviewing the patients using a questionnaire. In our experience, this method results in more accurate information than if a questionnaire is used without an interview. Quinn et al (United Kingdom)[11] also used interviews to evaluate the referral route of patients with RRD from primary care to a tertiary care ophthalmic unit. They reported that more than 50% of the patients had significant delay due to ignorance, which was comparable with our percentage. The majority of their patients with RRD had been referred by optometrists and GPs (about two thirds of the referrals).[11] Contrastingly, 86% of the patients in the Netherlands first visited their GP. In our study, the GPs made an incorrect diagnosis in 10% of the cases (n=20) (see Table 2.4), which resulted in a median GP delay of 0 ± 20 days.

Although the median patient-related delay was 4 days, the median GP and RO delay was 0 days. We excluded all the patients who first presented to the retinal surgeon with symptomatic PVD alone, i.e. without RRD. A routine re-examination was scheduled for these patients within 6 weeks. The reason why we excluded these patients was because they had symptoms of PVD or retinal tears, but not RRD. These patients developed RRD in the interval before this scheduled visit, but found no reason to consult the retinal surgeon earlier.

Van Overdam et al[12, 13] found that 5% of their patients developed a new retinal tear after the initial examination. In the present study, 3% (n=6) of the patients (2 referred by an ophthalmologist and 4 from our centre) developed RRD after the initial examination in which PVD alone had been diagnosed. These patients were excluded from the study. Another six patients (4 referred by an ophthalmologist and 2 from our centre) were also excluded, because they had first undergone photocoagulation, but did not develop RRD until later. Inclusion of these patients would have resulted in a relatively long RO delay and biased the delay of the RO. We also excluded a group of patients who presented with vitreous haemorrhage (another form of symptomatic PVD), in whom successive ultrasound examinations had been performed by the RO or at the TRC; they did not develop RRD until a few days later.

Posterior vitreous detachment (PVD) is a common age-related degenerative condition,[14, 15] in which patients experience light flashes and/or floaters.[16] These symptoms are specific to PVD, but not all patients suffer from them. A study by Hikichi and Trempe[16] reported 89% sensitivity for floaters and flashes, with a specificity of 25%. This supports the statement made by Green and Sebag that *posterior vitreous detachment* is one of the least accurate diagnosis employed by ophthalmologists on a daily basis.[17] These findings also confirm our results that there were no significant differences in delay between the patients with or without flashes, or between the patients with or without floaters. Nevertheless, PVD is associated with an increased risk of developing retinal tears.[12] In the literature, 7.3-14% of patients with PVD had retinal tears.[6] Although RRD may be present or may develop following PVD, subjective flashes and/or floaters are often absent. Owing to the observation that many patients with RRD in the Netherlands initially consult their GP, it is important that GPs are familiar with the complexity and variations of symptoms associated with RRD and have adequate knowledge of the implications of these symptoms.

In this study, we found a striking male to female ratio of 134 men to 71 women. Our pseudophakic male to female ratio was 51 to 18 (3:1), which closely matched the ratio reported in a study by Sheu et al.[18] Their cumulative 6-year pseudophakic RRD rates were 1.9% in the male subgroup compared to 0.56% in the female subgroup at the end of follow-up.[18]

In summary, the median overall delay between the initial symptoms and surgery was 10 days. Almost 60% of the overall delay was caused by the patient or the general practitioner. In more than 50% of the patients, the delay was due to unawareness and/or unfamiliarity with the symptoms. Patient and GP education may therefore form primary goals to improve the functional outcome of RRD surgery. In every symptomatic patient, complete funduscopic examination should be performed by an ophthalmologist, including 3-mirror contact lens examination; re-examinations should take place within 6 weeks. Patients should also be well-instructed to return earlier if their symptoms deteriorate. It is vital that they realise the importance of the symptoms of flashes, floaters and visual field loss and contact an ophthalmologist promptly. The education of patients who are at risk of RRD (e.g. patients with myopia) can start at their first visit to an ophthalmologist (irrespective of the reason for consultation) and be backed-up by brochures. GPs must also be made aware of the different symptoms that can lead to PVD and/or RRD. Education can be provided, for example, in the form of articles in national or GP journals.

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Chapter 3

Incidence of re-detachment 6 months after scleral buckling surgery

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Abstract

Purpose: This study aimed to analyse the long-term effects of scleral buckling (SB) surgery for rhegmatogenous retinal detachment (RRD) by following patients for at least 6 months. In the literature, preoperative and intraoperative clinical variables associated with re-detachment and/or poor visual outcome have mainly been studied in the short-term.

Methods: In a retrospective survey, we evaluated data on 436 eyes after SB surgery. Postoperative data were collected at 3 monthly intervals.

Results: After a mean follow-up of 51 months, anatomical re-attachment was achieved in 76% after one SB procedure, with an ultimate re-attachment rate of 97% after additional vitreoretinal procedures. A total of 104 eyes developed re-detachment during follow-up. After more than six and twelve months follow-up, 32 eyes (7%) and 20 eyes (5%) developed re-detachment, respectively. Multivariate regression analysis showed that recurrent re-detachment and more than seven days of visual field loss were significant predictors of poor postoperative visual outcome at 12 months. A significant predictor of recurrent RRD was a cumulative tear size of more than 3 disc diameters.

Conclusion: Conventional SB surgery was a reliable procedure in a selected group of eyes with primary RRD. However, in eyes with a retinal tear that has a cumulative size of more than 3 disc diameters, primary vitrectomy is probably a better option. Based on our finding that 7% of the eyes developed re-detachment after 6 months, a longer follow-up period seems necessary to evaluate the anatomical and visual outcomes after SB surgery.

Introduction

With the introduction of improved vitreoretinal surgical techniques, there is on-going debate about which patients with rhegmatogenous retinal detachment (RRD) are good candidates for a simple scleral buckling (SB) procedure.[1-5] Although the majority of vitreoretinal surgeons still perform SB surgery in uncomplicated RRD,[1, 6-11] others propagate primary vitrectomy in such cases.[12-15] Those in support of primary vitrectomy (PPV) argue that PPV has potential advantages over SB surgery, including shorter surgery, more accurate diagnosis of tears, no postoperative axial length changes and a higher re-attachment rate after one procedure.[13, 16]

In a consecutive series of eyes that underwent SB surgery for primary RRD, we evaluated which preoperative and intraoperative clinical variables were associated with an increased risk of re-detachment and/or poor visual outcome after a minimum follow-up of 6 months. Our aim was to identify patients with good results after SB surgery, in order to improve the preoperative selection criteria.

Materials and methods

Medical records were reviewed of all patients with RRD who underwent primary SB surgery without vitrectomy. All the patients included in the study had been operated on by the same surgeon (ELH). In the period between January 1997 and October 2004, SB surgery was performed on primary clear media non-traumatic, non-uveitic retinal detachments with limited proliferative vitreoretinopathy (PVR), except for three eyes that underwent pneumatic retinopexy. None of the eyes in the study sample had primary vitrectomy. We excluded all the eyes with: a follow-up of less than 6 months (17 eyes), a history of uveitis or trauma, PVR grade C2 or higher[17, 18] and complicated RRD due to vitreous haemorrhage that obscured fundus details. The study was conducted in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki. When present, PVR was graded according to the Classification of the Retina Society Terminology Committee.[19]

All the patients were operated on by the same surgeon. The standard surgical technique was to fit an encircling band. In each patient, the decision of whether to fit a radial or segmental buckle was based on the size and location of the retinal tear. Drainage of sub-retinal fluid was performed when judged necessary by the surgeon. Cryocoagulation was applied to almost all the cases. Intravitreal gas tamponade was used only for RRDs in the superior segment.

The following preoperative clinical patient characteristics were collected for statistical analysis: age, sex, preoperative visual acuity, prior intraocular surgery, the number of detached quadrants of the retina, whether or not the central area of the macula (foveal region) was involved in the detachment, the presence and grade of PVR, the type and localization of the retinal tear, the number of tears and their cumulative size in disc diameters (DD). The type of tear was noted as an atrophic hole, a horseshoe tear, a combination of the two, or ora-dialysis. Pre- and postoperative Snellen visual acuities (also with pinhole correction) and pre- and postoperative Goldmann applanation tonometry were recorded. During slit-lamp examination, the presence of pseudophakia, or intravitreal pigment was identified. Funduscopy was performed using indirect binocular ophthalmoscopy in combination with a Goldmann three-mirror contact lens (without scleral depression) and with a pan fundus contact lens (Supersquad 160°). Fundus drawings of the RRD were made in clock hours. By carefully interviewing the patient, we determined the approximate time of onset of the detachment. The duration of detachment until surgery was categorized into the following groups: 0 to 7 days, or more than 7 days, as described by Diederen et al.[20]

Surgical SB reports were reviewed to retrieve details of the procedure used to repair the tear. The following surgical variables were recorded: radial or segmental silicone buckle, subretinal fluid drainage (yes/no), cryocoagulation (yes/no), air or intravitreal sulphur hexafluoride (SF_6) gas as internal retinal tamponade (yes/no). All intraoperative complications were noted, such as scleral perforation, vitreous incarceration, or retinal incarceration.

After SB surgery, we noted whether the patients had developed recurrent RRD and at what interval, the cause of the re-detachment and the subsequent clinical procedures. Anatomical success was defined as: complete re-attachment of the retina, or as a stable situation in which the retina was attached, with confined pockets of subretinal fluid. Recurrent retinal detachment was categorized as being due to: vitreous traction with PVR, new or missed retinal tears, persistent and/or progressive re-accumulation of subretinal fluid, or progressive "leakage" of subretinal fluid at the edge of the buckle due to lack of indentation. Re-detachment within 6 months of the primary SB procedure was defined as "early re-detachment".

Data were collected at 3-monthly intervals following SB surgery. After 12 months, many of the patients had returned to their referring ophthalmologist, who we subsequently approached for the relevant follow-up data. If patients had not returned for follow-up at our department or at the ophthalmology practice, data on anatomical success were obtained by telephone and interview by one of the authors (FG). We asked these patients whether their visual function had deteriorated and whether they had noticed any signs or symptoms of (re-)detachment. In the case of deterioration and/or symptoms, we noted the name of the ophthalmologist they had consulted and asked for their permission to retrieve the relevant data. Statistical analysis was performed using the SPSS software 13.0. Snellen visual acuities were converted into a logarithmic scale (LogMAR, i.e. the logarithm of the minimal angle of resolution), as described previously.[21] In the present study, poor visual outcome was defined as: visual acuity of less than 0.1 Snellen lines at 12 months follow-up. Missing data on co-variables or outcome variables (when the variable had been measured at least three times) were inferred with the multiple imputation technique described by van Buuren et al,[22] using STATA 8 software. Comparisons were made between preoperative and postoperative visual acuities using the Wilcoxon signed rank test. Univariate analysis was performed with the Chi-square test or Fisher's exact test to determine whether preoperative or intraoperative clinical variables were associated with recurrent RRD or poor visual outcome. A stepwise forward multiple logistic regression analysis was used to determine the strongest predictors of poor visual outcome from the following preoperative variables, using a probability for entry of < 0.1 or recurrent RRD: age older than 70 years, pseudophakia, number of days of visual field loss, the type of retinal tear, three or more tears, cumulative tear size of more than 3 DD, extent of total detachment, the presence of PVR (B or C) and macular involvement. A P-value of ≤ 0.05 was considered significant.

Results

A total of 436 eyes (263 men and 173 women, 60.3% versus 39.7%) in 424 patients were included in this study. These patients had a mean age of 58.2 years (range 15-90 years). Mean follow-up was 50.6 months (range 6-120 months). Separated into age groups, 356 patients (81.7%) were 70 years or younger, while 80 (18.3%) were older than 70 years. There were 339 phakic eyes (77.8%) and 97 pseudophakic eyes (22.2%). Preoperatively, 252 eyes (57.8%) had a detached macula and 184 eyes (42.2%) had an attached macula.

In this series of 436 eyes, anatomical success was achieved in 76.1% (N=332) after one SB procedure. Re-attachment was ultimately achieved in 424 (97.2%) of the eyes after multiple vitreoretinal procedures. A Kaplan Meijer survival curve of time versus re-detachment is shown in Figure 3.1.

Subsequent vitreoretinal procedures to re-attach the retina were performed in 100 out of the 104 eyes that developed re-detachment after SB surgery. In four out of the 104 eyes, the patient and/or the surgeon decided not to proceed with any further operations, because of the poor prognosis for the eye, or the poor physical condition of the patient. This decision was ultimately made in nine (2%) out of the 436 eyes, although five patients had undergone additional vitreoretinal procedures.

Data on the interval since SB surgery were available in 100 out of the 104 eyes (23.9%) that developed re-detachment. Data on the timing and the cause of the re-detachment were available in 87 eyes (Table 3.1).

Re-detachment occurred after a mean period of 54.4 weeks (median 12 weeks, range 0 days to 516 weeks) in all 100 eyes. Within the first 3 months, 55 (12.6%) eyes had re-detachment. At 6 months, 68 out of the 436 eyes (15.6% of total) had re-detachment; one year after the SB procedure, a total of 80 out of the 436 eyes (18.3%) eyes had recurrent RRD. Twenty out of the 100 eyes (4.6% of the total surgical series) developed re-detachment more than one year postoperatively. Details of the timing and causes of the re-detachments are described in Table 3.1.

We used telephone interviews to ask the patients whether their visual function had deteriorated, or whether they had noticed any signs or symptoms of (re-)detachment. With this method, we found that only one patient had received retinal re-detachment

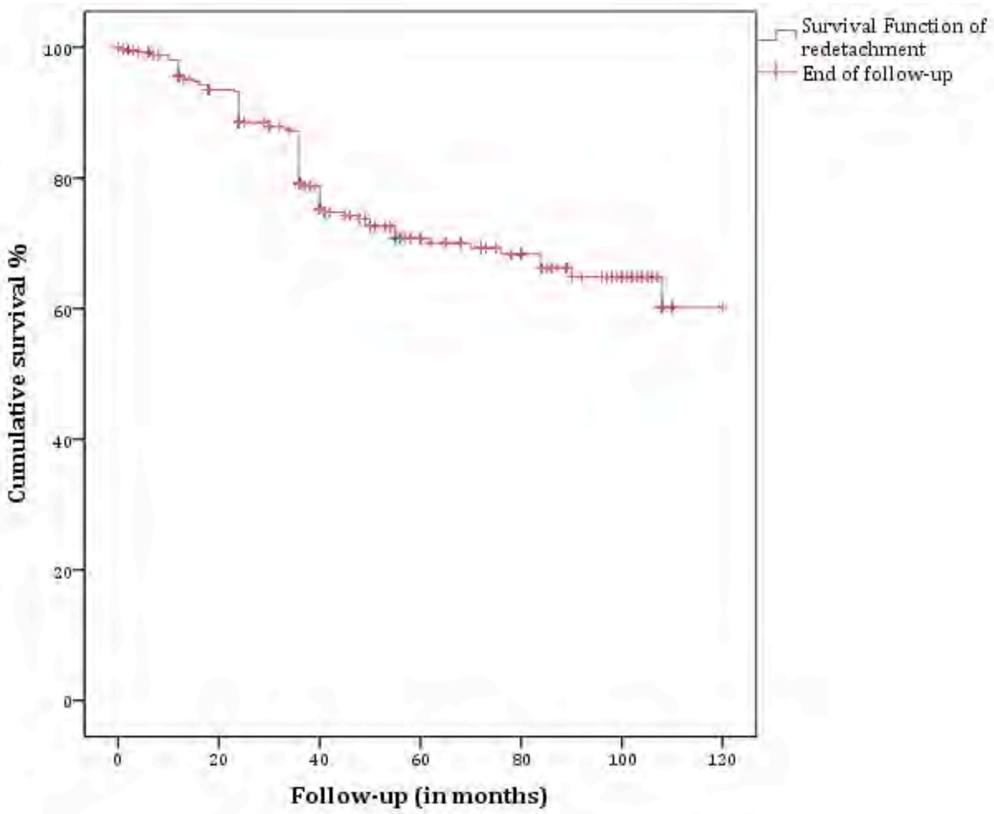

Figure 3.1: Kaplan Meijer survival plot of re-detachment after scleral buckling surgery (n=436)

Survival = number of eyes with an attached retina after one SB procedure

The curve shows a steep decline because two patients had re-detachment at 120 months follow-up

surgery at another hospital. Eight other cases, who stated that their visual acuity had changed, were seen at our hospital. However, none of these eyes had re-detachment.

Re-detachment occurred in 29 out of the 97 pseudophakic eyes (30%). There were no aphakic eyes in the present series. In 3 eyes, no further operations were performed, because of the poor prognosis for the eye, or the poor physical condition of the patient. A total of 26 eyes underwent subsequent vitrectomy procedures: in 20 eyes, one vitrectomy with gas tamponade (n=6) or oil tamponade (n=14); in 6 eyes two vitrectomies: 5 with oil tamponade, and one eye with gas tamponade followed in a later procedure with oil tamponade. In one of the eyes, a total of 3 re-attachment vitrectomies were performed with oil tamponade. The retina was ultimately attached in 100% (26/26) of the 26 eyes that underwent subsequent surgery. However, two eyes with attached retinas were enucleated because of severe pain.

At the end of follow-up, visual acuity was hand motion or less in 14% of the patients. A total of 225 eyes (52%) had 20/20 to 20/40 vision, 148 (34%) had 20/50 to 20/200, while 63 eyes had less than 20/200. In the group with 20/200 or less, 37 had re-detachment (59%). In nine eyes (14%), the patient and/or the surgeon decided not to proceed with any further operations, because of the poor prognosis for the eye, or the

Table 3.1: Causes of re-detachment during follow-up after the first scleral buckling procedure

Time since the first operation	PVR/ traction n	Leakage‡ n	Missed / New tears n	Persistent SRF n	Other #	Total cumulative n (cumulative % of total)	Total n (cumulative % of re-detachment and cause)
1 - 3 months	35	7	2	10	1	55 (12.6%)	55 (55%)
3 - 6 months	8	0	1	3	1	68 (15.6%)	13 (68%)
6 months-1 year	8	0	0	3	1	80 (18.3%)	12 (80%)
> 1 year	4	0	6	0	10	100 (22.9%)	20 (100%)
Total	55	7	9	16	13	104 (23.9%)	100 (100%)

PVR: proliferative vitreoretinopathy, SRF: subretinal fluid
‡ Leakage is defined as the accumulation of subretinal fluid at the edge of the buckle due to lack of indentation
# Other: other causes of re-detachment, such as removal of the scleral buckle in two patients after one year, or not noted, or cause unknown
Note: Data on the interval between SB surgery and re-detachment were available in 100 out of the 104 eyes (23.9%)

poor physical condition of the patient. In the subgroup of 63 eyes whose visual acuity was 20/200 or less, we were able to determine the cause in 16 cases: eight patients developed severe cataract, two patients had a vascular disease (one case of anterior ischaemic optic neuropathy and one case of central retinal vein occlusion), one patient developed corneal decompensation, two cases had amblyopia, one case developed phthisis bulbi, one patient had an epiretinal membrane and one patient developed neovascular glaucoma. Owing to the retrospective nature of the study and the fact that we only asked the referring ophthalmologists to provide visual acuity results, no data were available on the cause of low visual acuity in the other 38 eyes.

Univariate analysis was performed on all the preoperative, intraoperative and postoperative clinical variables to obtain odds ratios for poor postoperative visual outcome. Significant risk factors were found to be: preoperative Snellen visual acuity of less than 0.1, more than 7 days of visual field loss, more than three quadrants of retinal detachment, PVR B or C, macular involvement and re-detachment (Table 3.2). Multivariate analysis revealed that more than 7 days of visual field loss and recurrent retinal detachment were significant risk factors for poor visual outcome (odds ratios: 2.3, $P=0.006$ and 5.0; $P<0.001$, respectively).

Univariate analysis was also performed on all the preoperative and intraoperative clinical variables to obtain odds ratios for re-detachment. Significant risk factors were found to be: a cumulative tear size of > 3 DDs, more than three quadrants of retinal detachment and PVR grade B or C (Table 3.3). Multivariate analysis revealed that only the size of the tear was significantly associated with the occurrence of re-detachment ($P=0.009$) (Table 3.3). Further stepwise forward multiple logistic regression analysis on these variables, with the addition of the interval until re-detachment, showed a significant association

Table 3.2: Univariate and multivariate analyses on clinical variables associated with poor visual acuity (< 0.1 Snellen) at 12 months follow-up

Factors	Total no. of Eyes (N=436)	Univariate analysis			Multivariate analysis		
Preoperative		**Odds ratio**	**CI**	**p**	**Odds ratio**	**CI**	**p**
Age							
≤ 70 years	356						
> 70 years	80	1.56	0.65-3.70	0.29			NS
Pseudophakic eye							
Yes	97	1.57	0.89-2.78	0.12			NS
No	339						
Visual acuity preoperatively (Snellen)							
< 0.1	182						
≥ 0.1	254	0.42	0.24-0.74	0.004			NS
Days of visual field loss reported‡							
0 - 6 days	230	2.31	1.36-3.92	0.002	2.30	1.27-4.18	0.006
≥7 days	206						
Number of tears‡							
Missed tears	39						
1 - 3	358	1.26	0.43-3.67	0.67			NS
≥ 4	39	1.93	0.89-4.17	0.09			NS
Type of retinal tear							
Round	64	1.76	0.72-4.30	0.21			NS
Horseshoe	296	1.04	0.61-1.77	0.90			NS
Other	37	0.92	0.19-4.32	0.92			NS
None/missing	39	0.19	0.00-74.78	0.49			NS
Cumulative size of the tears *							
< 3 DD	290						
≥ 3 DD	107	1.64	0.86-3.16	0.13			NS
Detachment size							
1 - 3 quadrants	409						
>3 quadrants	27	4.19	1.75-10.06	0.001			NS
PVR							
B/C	119	2.38	1.02-5.56	0.0046			NS
Other	317						
Macular status							
Macular involvement	252	0.37	0.20-0.72	0.004			NS
Macula not involved	184						
Re-detachment							
Yes	104	5.02	2.57-9.82	<0.001	5.04	2.27-11.19	<0.001
No	332						

NS: not significant, DD: disc diameter, PVR: proliferative vitreoretinopathy, SF_6: sulphur hexafluoride
‡ variables were compared to the first variable
* Missed tears (n=39) were excluded

Table 3.2 (Continued)

Factors Preoperative	Total no. of Eyes (N=436)	Univariate analysis			Multivariate analysis		
		Odds ratio	CI	p	Odds ratio	CI	p
Intraoperative factors							
Type of buckle							
Segmental	257	1.35	0.71-2.58	0.35			NS
Radial	184	0.66	0.33-1.31	0.22			NS
Combination of the two	12	2.18	0.61-7.81	0.23			NS
Drainage of subretinal fluid							
Yes	364	1.80	0.57-5.65	0.29			NS
No	72						
Transscleral cryopexy							
Yes	341						
No	95	1.04	0.54-2.00	0.89			NS
Use of intravitreal gas (SF_6 gas)							
Yes	316						
No	120	0.78	0.38-1.59	0.48			NS
Peroperative complications							
Yes	21						
No	415	2.48	0.77-7.92	0.12			NS

NS: not significant, DD: disc diameter, PVR: proliferative vitreoretinopathy, SF_6: sulphur hexafluoride
‡ variables were compared to the first variable
* Missed tears (n=39) were excluded

between the cumulative size of the tear of more than 3 DDs and early re-detachment (P=0.007, data not shown in the Table).

Postoperatively, the scleral buckle or encircling band had to be removed from six eyes. This was due to intolerable pain in four eyes, infection in one eye and exposure of the buckle in one eye. During follow-up, re-detachment developed in two (33%) out of these six eyes.

Discussion

In this series of consecutive, non-selected patients, anatomical re-attachment of the retina was achieved in 76% after one scleral buckling (SB) procedure. Ultimate anatomical success was achieved in 97%. These percentages were comparable with those reported in earlier studies on RRD treated with SB surgery alone.[7-10, 18, 23-28]

Table 3.3: Univariate and multivariate analyses on clinical variables associated with re-detachment

Factors	Total no. of Eyes	Univariate analysis			Multivariate analysis		
Preoperative	(N=436)	Odds ratio	CI	p	Odds ratio	CI	p
Age							
≤ 70 years	356	1.59	1.16-2.19	0.143			NS
> 70 years	80						
Pseudophakic eye							
Yes	97	0.67	0.52-0.86	0.12			NS
No	339						
Visual acuity preoperatively (Snellen)							
< 0.1	182						
≥ 0.1	254	1.14	0.87-1.51	0.556			NS
Day of visual field loss reported‡							
0 - 6 days	230						
≥7days	206	0.79	0.62-1.00	0.293			NS
Number of tears‡							
missing	39						
1 - 3	358	0.41	0.15-1.12	0.082			NS
>3	39	0.47	0.15-1.50	0.183			NS
Type of retinal tear							
Round	64	0.79	0.26-2.43	0.473			NS
Horseshoe	296	0.74	0.28-1.95	0.197			NS
Other	37	1.26	1.11-1.43	0.773			NS
Missing	39	1.36	0.41-4.51	0.361			NS
Cumulative size of tears *							
< 3 DD	290						
≥ 3 DD	107	0.50	0.24-1.03	0.006	0.51	0.40-0.66	0.009
Detachment size							
1 - 3 quadrants	409						
> 3 quadrants	27	0.31	0.29-0.33	0.004			NS
PVR							
B/C	119						
Other	317	0.56	0.26-1.19	0.016			NS
Macular status							
Macular involvement	252	1.29	0.35-4.89	0.267			NS
Macula not involved	184						
Intraoperative factors							
Type of buckle							
Segmental	247	0.65	0.24-1.76	0.068			NS
Radial	177	0.57	0.38-7.19	0.036			NS
Combination of the two	12	0.62	0.33-1.15	0.439			NS

NS: not significant, DD: disc diameter, PVR: proliferative vitreoretinopathy, SF_6: sulphur hexafluoride
‡variables are compared with the first variable

Table 3.3 (continued)

Factors	Total no. of Eyes	Univariate analysis			Multivariate analysis		
Preoperative	(N=436)	Odds ratio	CI	p	Odds ratio	CI	p
Drainage of subretinal fluid							
Yes	364	813	0.26-2.51	0.511			NS
No	72						
Transscleral cryopexy							
Yes	341	1.10	0.33-3.60	0.715			NS
No	95						
Use of intravitreal gas (SF_6 gas)							
Yes	316	1.23	0.36-4.18	0.396			NS
No	120						
Peroperative complications							
Yes	21						
No	415	0.61	0.31-1.22	0.300			NS

NS: not significant, DD: disc diameter, PVR: proliferative vitreoretinopathy, SF_6: sulphur hexafluoride ‡variables were compared to the first variable

After more than six and twelve months follow-up, 32 eyes (7%) and 20 eyes (5%) developed re-detachment, respectively. Multivariate regression analysis showed that recurrent re-detachment and more than seven days of visual field loss were significant predictors of poor visual outcome at 12 months. In the case of recurrent RRD, a cumulative tear size of more than 3 DDs was a significant predictor. The predictors of poor visual outcome found in the present study corresponded with the risk factors reported in previous studies.[6, 20, 29, 30] However, they differed from a recently published study by Salicone et al,[9] who found that macular involvement was the main risk factor of poor visual outcome after SB surgery. This discrepancy may have been due to differences in the time intervals at which the visual acuity data were obtained. Several studies indicated that visual recovery after SB surgery increases over time.[31-34] Salicone et al[9] based their conclusions mainly on postoperative visual acuity data obtained after a follow-up of 2 months, whereas in our study, the conclusions were based on visual acuity data obtained at 12 months.

Following our patients for a longer period revealed that ultimately, 23.9% of the eyes developed re-detachment. Six months after SB surgery, 8.3% developed “late” re-detachment; after 12 months, another 4.6% of the eyes developed recurrent RRD. Foster et al[25] found recurrent RRD after one year in 2.2% (10/453 eyes).

Only a few studies have been published that had a relatively long mean postoperative follow-up period (Table 3.4).[11, 25, 27, 32, 35, 36] Schwartz et al[27] had a follow-up of 20 years, but they only included patients who responded to a follow-up call after 20 years and they

Table 3.4: Summary of long-term results of scleral buckling surgery in patients with rhegmatogenous retinal detachment

Author	Year	Number of patients n=	Initial surgery	F-U Mean (range)	Detached after one surgery	Detached after one year	Final success	VA	Note
Goezinne et al	2008	436	SB	4.2 years (6-120 months)	23.9%	4.6%	97.2%	NN	Operated on by same surgeon
Sasoh et al[32]	2005	205	SB	10 years	NN	NN	NN	20/15-20/20 76% in macula on 20/15-20/20 50% in macula of	Only patients with 10 years F-U (30.9% of total) amount of surgeon NN
Foster et al[25]	2002	453	NN	8.5 years (69 to 140 months)	7.8%	2.2%	NN	NN	Study on 10 eyes with late re-detachment, more than one surgeon
Schwartz et al[27]	2002	227	SB	20 years	18%	NN	95%	20/40 median 20 years	More than one surgeon
Greven et al[11]	1999	28	SB	mean 29 months (6-78 months)	3.6%	3.6%	100%	20/20 in 32% 20/25 in 18% 20/30 in 25% 20/40 in 18% 20/50 in 7%	Asymptomatic retinal detachment, operated on two surgeons
Kreissig et al[35]	1995	107	SB	15 years	NN	NN	92.6%	20/30 in group macula attached, 20/100 in group macula partially detached and 20/400 in group completely detached	Number of surgeons NN
Kreissig et al[36]	1992	107	SB	(11-11.5 years)	12.1%	6.5%	92.6%	NN	Numbers of surgeons NN

SB:scleral buckle, NN: not noted, F-U: follow up

did not retrieve any data from the referring ophthalmologists or by interviewing the patients, contrary to the present study. In the studies by Foster et al,[11] Greven et al[25] and Kreissig et al,[36] the late re-detachment rates ranged from 2.2% to 6.5%, which was supported by the results of the present study (Table 3.4).

A cumulative retinal tear size of more than 3 DDs was identified as a significant risk factor for recurrent RRD in the present series. This was in agreement with previous studies.[6, 9, 18, 23, 37] We also found that a large retinal tear was a significant risk factor for early re-detachment (within 6 months after SB surgery). Kreissig et al[35, 36] distinguished between early and late re-detachments and observed that early re-detachment (up to 6 months following SB surgery) was mainly caused by PVR, whereas late re-detachment was caused by new tears in half of the cases. Although PVR can be found in patients with late

recurrent RRD, we believe that vitreous base traction is one of the main causative factors in these cases, because the PVR may well be a secondary phenomenon.[25, 36]
The occurrence of late re-detachment is an argument in favour of following patients for at least 6 months after SB surgery. In addition, visual acuity may continue to improve for some time. Using foveal densitometry, Liem at al[31] showed that recovery of foveal cone photopigments could take several months after macular detachment. It was also found that subfoveal fluid may persist subclinically for several months following SB surgery, which may explain the slow visual recovery in some of the patients.[38-40]
A disadvantage of following patients over a longer period can be bias to the visual acuity results due to deterioration in the elderly patients who develop cataract or macular problems. Visual acuity results in the present study were therefore analysed at a fixed period of 12 months after primary surgery.
Le Rouic et al[41] investigated the outcomes of buckle removal. They found an overall re-detachment rate of 8.8% in a series of 90 eyes after the scleral buckles had been removed. Re-detachment occurred in 16% of the eyes after solid silicone buckle removal, compared to 7% in the eyes after silicone sponge removal.[41] In the present study, only six eyes underwent scleral buckle removal, which contrasted sharply with the larger series studied by Le Rouic et al.[41] Two out of these six eyes developed re-detachment after silicone sponge removal. In one case, re-detachment was due to perforation that occurred during removal from an eye with staphyloma. In the other case, there was no clear cause for the re-detachment.
Primary vitrectomy (PPV) has become a leading treatment for RRD. Several studies compared SB surgery to primary PPV in eyes with new RRD.[3, 42-46] A meta-analysis on pseudophakic retinal detachment by Arya et al[14] revealed that PPV was more likely to achieve favourable anatomical and visual results than conventional SB surgery alone.[14] However, an evidence-based analysis on uncomplicated RRD did not show any difference in retinal re-attachment or ultimate VA between the SB group and PPV group.[3] An argument in favour of PPV instead of SB surgery is that peripheral tears can be missed preoperatively, especially in pseudophakic eyes.[47] In the present study, in which 22% of the eyes were pseudophakic, missed tears were not significantly associated with poor visual or functional results.
In conclusion, conventional SB surgery was a reliable procedure in selected eyes with primary RRD. Primary PPV should be considered if the retinal tear has a cumulative size of more than 3 DDs. In studies that aim to predict how macular detachment affects visual outcome or recovery, it may be an advantage to plan a longer follow-up period after SB surgery in view of the high percentage of eyes that develop re-detachment after 6 months and the chance of further visual recovery over time.

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Chapter 4

Anterior chamber depth is significantly decreased after scleral buckling surgery

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Abstract

Objective: to evaluate the duration of changes in anterior chamber depth (ACD) in patients with rhegmatogenous retinal detachment (RRD) after scleral buckling (SB) surgery. The SB surgery itself may result in decreased ACD. Myopic patients are known to have an increased risk of developing RRD. Nowadays, they can opt to undergo correction of their refractive error by phakic intraocular lens (pIOL) implantation. After implantation, progressive endothelial cell loss can occur if the ACD is too shallow. This may form an important issue not only for the retinal surgeon in patients with combined RRD and a pIOL, but also for the refractive surgeon who has to consider the potential problems of pIOL implantation in an eye that has previously undergone SB surgery.

Design: A prospective controlled study.

Participants: Thirty-eight eyes with primary RRD treated with SB surgery, using an encircling element and a radial or segmental buckle; 31 fellow eyes served as controls.

Methods: ACD (in the horizontal meridian) and axial length were measured preoperatively and at 1 week, 1 month and 3, 6, 9 and 12 months postoperatively using an anterior optical coherence tomography method and an IOL master, respectively.

Main outcome measures: In all 38 SB surgery eyes, ACD was significantly decreased compared to the preoperative levels up to 9 months after SB surgery.

Results: ACD had returned to normal at one year follow-up. Axial length was significantly increased during the whole follow-up period. No significant differences in ACD or axial length were found between the use of radial or segmental buckles.

Conclusions: ACD may remain decreased after SB surgery for longer than has previously been reported.

Introduction

Many vitreoretinal surgeons consider SB surgery to be the first choice of treatment in patients with uncomplicated RRD.[1] Previously, scleral dissection was performed,[2] while the use of an encircling element was introduced by Schepens et al in 1957.[3] In some patients, these external techniques not only caused changes in axial length, but also decreased the ACD.[2, 4, 5] Earlier studies on ACD changes after RRD surgery reported a temporary decrease in ACD until 3 months follow-up, but no further ACD measurements were performed beyond that point.[2, 4]

Nowadays, a phakic intraocular lens (pIOL) can be implanted in an eye to correct refractive errors.[6, 7] Strict ACD criteria have been formulated by refractive surgeons to lower the risk of endothelial cell loss after pIOL implantation.[8] SB surgery for the treatment of RRD may also result in decreased ACD. Therefore, in patients who have previously undergone SB surgery, accurate preoperative ACD measurements are essential if they need pIOL implantation surgery. Conversely, when a patient with a pIOL presents with primary RRD (myopic patients are known to have an increased risk of developing RRD), the vitreoretinal surgeon must be aware that the ACD can become shallower after SB surgery, with a subsequent increase in endothelial cell loss.

Recently, a new non-contact anterior segment optical coherence tomography imaging method (AS-OCT) has become available that can provide high resolution cross-sectional images of the anterior chamber (Visante, Carl Zeiss, Meditec, Jena, Germany). Several studies have reported on its high reproducibility and repeatability, which were equal to other ACD measurement techniques, or even better.[9]

The goal of this study was to evaluate the nature and time frame of changes in ACD and axial length after SB surgery.

Subjects and methods

This prospective study included all the patients with primary RRD who had been treated with conventional SB surgery between July 2006 and January 2007. Eyes were excluded if they developed recurrent RRD during follow-up and if they had undergone previous glaucoma surgery in one or both eyes.

The fellow eye served as a control for all the measurements. We excluded fellow eyes if they developed RRD preoperatively or during the study period, or if they underwent subsequent intraocular surgery during follow-up.

Approval for the study was granted by the institutional ethics committee. Patients were informed about the study procedure and provided informed consent. All procedures were conducted in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

The following preoperative patient characteristics were collected for analysis: age, gender, myopia (defined as spherical equivalent of -6 dioptres (D) or more), previous intraocular surgery (i.e. cataract and/or retinal detachment in the fellow eye), number of retinal quadrants detached, foveal involvement (yes/no) confirmed by posterior optical coherence tomography (OCT) (Carl Zeiss, Meditec, Jena, Germany), number of days of foveal detachment, the presence and grade of proliferative vitreoretinopathy (PVR),[10] type and localization of the retinal tear and the number of tears.

Four vitreoretinal surgeons (FH, ELH, AL, IL) at our retina department performed the operations under general anaesthesia. Each patient received a silicone encircling element (ref. no. 240, MIRA Inc., Uxbridge, MA, USA) under the rectus muscles and either a segmental buckle (silicone grooved strip no. 506G, DORC International, Zuidland, the Netherlands) or a radial buckle (solid silicone wedge, G135, MIRA Inc Uxbridge, MA, USA or G137, Labtician Ophthalmics Inc., Oakville, Canada). The encircling band was fixed 10-12 mm posteriorly to the limbus with a mercilene 5.0 suture. Whether a radial or segmental buckle was used depended on the size and location of the retinal tear. Subretinal fluid was drained when judged necessary by the surgeon. Cryocoagulation was performed in some cases, according to the preferences of the surgeon. Intravitreal gas tamponade was used for RRDs in the upper quadrants, located between the 8 and 4 o'clock positions.

All the patients received routine postoperative treatment with antibiotic eye drops (chloramphenicol 5 mg/ml) 4 times daily for 14 days (Ratiopharm, Zaandam, the Netherlands and prednisolone eye drops (Pred Forte®, prednisolone acetate 10 mg/ml, Allergan BV, Nieuwegein, the Netherlands) 3 times daily for 2 months. Patients with phakic eyes also used mydriatic eye drops (atropine sulphate 1%) 2 times daily for 1 week after the operation (Chauvin Pharmaceuticals Ltd Surrey, UK). Patients with pseudophakic eyes used additional tropicamide 0.5% (Thea Pharma, Wetteren, Belgium) 2 times daily for 1 week.

The following SB-surgery-related variables were noted: radial or segmental silicone buckle, subretinal fluid drainage (yes/no), cryocoagulation (yes/no), intravitreal sulphur hexafluoride (SF_6) gas as internal retinal tamponade (yes/no), paracenthesis (yes/no) and postoperative acetazolamide (Diamox®, Goldshield Pharmaceuticals BV, Surry, UK).

All the patients were examined by two investigators (FG and DC). One investigator (FG) performed all the measurements. The analysis was performed later, without prior knowledge of patient history. IOL master (Carl Zeiss, Meditec, Jena, Germany) was used to measure pre and postoperative axial length in the RRD eyes and the fellow eyes; postoperatively, only the scleral buckling surgery (SBS) eyes that had an attached macula preoperatively (confirmed with posterior OCT) were re-measured.

During follow-up, we obtained the following data: intraocular pressure (IOP) (Goldman applanation tonometry), ACD in the horizontal meridian (AS-OCT), temporal and nasal anterior chamber angle and pupil diameter (AS-OCT) (Figure 4.1). Anterior chamber depth and angles were calculated (FG) with the AS-OCT software. Anterior chamber depth (ACD) was defined as the distance between the corneal endothelium and the anterior pole of the crystalline lens or IOL. The lens rise was measured as the horizontal distance from the angles to the lens (Figure 4.1). Angle-to angle distance (AAD) (also known as anterior chamber width (ACW) was defined as the distance from the temporal angle to the nasal angle along the 0-180° axis. Intra-investigator and inter-investigator differences in measurements were tested in a group of eight patients, differences were negligible.

All the measurements were performed on both eyes preoperatively, and on the SBS eyes on the first postoperative day, 1 week postoperatively and 3, 6, 9 and 12 months postoperatively. It was impossible to measure axial length on the first postoperative day due to the presence of intravitreal gas in most of the SBS eyes.

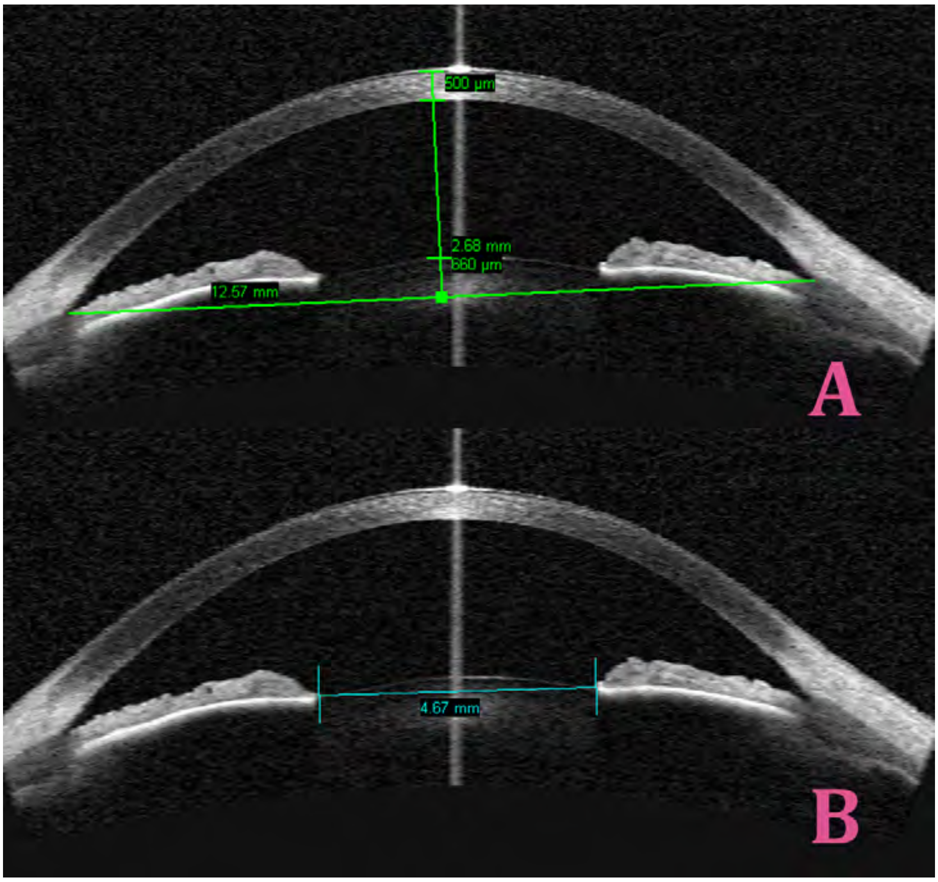

Figure 4.1: Anterior chamber depth using anterior segment optical coherence tomography (AS-OCT) Sectional plane is horizontal (from 0° to 180°). The white line in the middle shows that the image was obtained correctly.

A and B are the same OCTs.

A: anterior chamber depth (ACD) measured from the corneal endothelium to the anterior pole of the crystalline lens (2.68 mm). The angle to angle distance was measured from the nasal angle to the temporal anterior chamber angle (also known as the anterior chamber width) (12.57 mm). Lens rise is also marked (from the angle tot angle distance to the anterior pole of the crystalline lens (660 µm)

B: pupillary distance is 4.67 mm. Note that the pupil was not dilated with tropicamide and phenylephrine drops

Table 4.1: Before and after effects of mydriatic tropicamide 0.5% and phenylephrine 2.5% eye drops on anterior chamber depth in 11 postoperative patients

Patient no.	ACD before	ACD after	AAD before	AAD after	Pupil diameter before	Pupil diameter after
1	4.35	4.30	12.32	12.42	2.40	7.30
2	4.71	4.70	12.71	12.48	3.73	6.96
3	3.75	3.72	12.71	12.29	3.97	7.39
4	2.57	2.54	12.11	12.19	3.16	7.87
5	1.83	1.80	10.72	10.95	2.49	6.23
6	2.68	2.75	11.56	11.30	3.51	5.69
7	2.93	2.97	11.70	11.51	3.60	6.29
8	3.92	3.97	12.09	11.85	2.46	5.92
9	3.32	3.35	12.19	12.04	5.13	7.44
10	3.43	3.54	12.21	12.73	3.22	6.70
11	2.63	2.79	12.23	12.71	3.72	7.33
Mean ± SD	3.28 ±0.86	3.31 ±0.85	12.04 ±0.56	12.04 ±0.85	3.40 ±0.80	6.93 ±0.71
Significance		p=0.939		p=0.985		p<0.001

before: before instillation of eyedrops; after: 30 minutes after instillation of eyedrops; ACD: Anterior chamber depth; AAD: angle to angle distance; SD: standard deviation

Statistics: Independent sample T-test

Except for IOP, all the data were obtained after the instillation of tropicamide 0.5% and phenylephrine 2.5% (Thea Pharma, Wetteren, Belgium) in each eye. The effect of these mydriatic eye drops on the ACD was determined in 11 postoperative patients by taking measurements before instillation and 30 minutes afterwards (Table 4.1). No significant differences in ACD and AAD were found before and after instillation.
Statistical analysis was performed using the SPSS software 15.0 (SPSS for Windows, Rel. 13.0 Chicago: SPSS Inc. USA) using independent sample T-test. Significance was defined as $P < 0.05$.

Results

Between July 2006 and January 2007, 53 patients with RRD underwent SB surgery. We excluded patients with recurrent retinal detachment (n=13) or additional intraocular surgery (n=2) (i.e. cataract extraction) from our analysis. No significant differences were found between the patients included in the analysis and the patients who we excluded

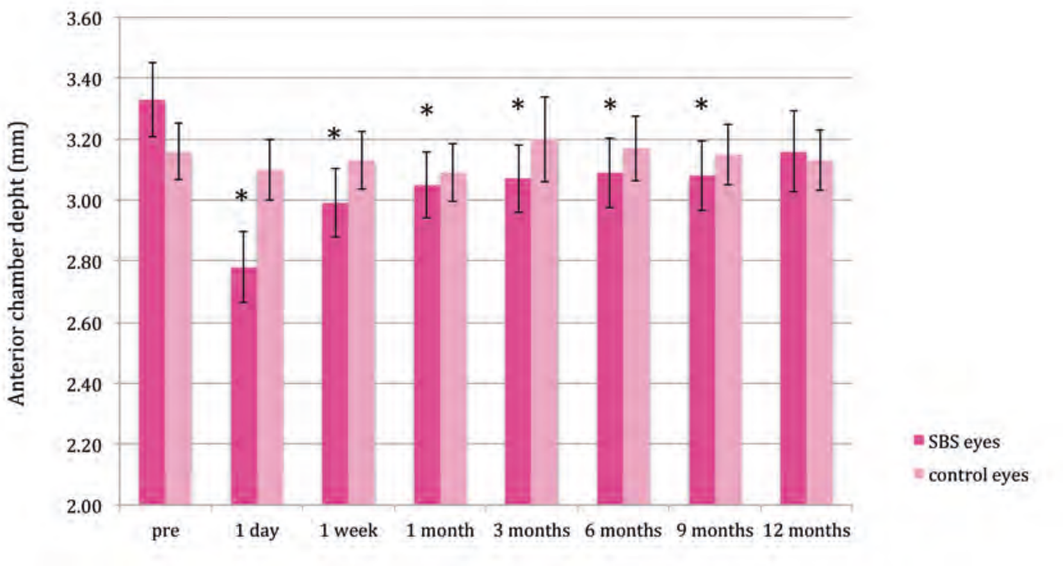

Figure 4.2: ACD measurements (anterior chamber depth) in SBS eyes and control eyes

Anterior chamber depth decreased significantly in the SBS eyes up to 9 months after scleral buckling surgery (P < 0.05) (*). Control eyes did not show any significant change compared to preoperative values. The vertical lines represent the standard error of the mean (S.E.M.).

Statistics: Independent sample T-test

(lost to follow-up), except for re-detachment (which was an exclusion criterion) and gender (Table 4.2). A total of 69 eyes in 38 patients with RRD (20 men (52.6%) and 18 women (47.4%) who underwent successful SB surgery were included in this study. We analysed 38 RRD eyes and 31 fellow eyes; the fellow eyes served as controls. Ultimately, seven fellow eyes were excluded, because of RRD development (n=3) or intraocular surgery (n=4) (i.e. cataract extraction) during the study period. Mean patient age was 58.5 years (range 36-74 years). The SB surgery group comprised 23 right eyes (60.5%) and 15 left eyes (39.1%); the control group comprised 13 right eyes (41.9%) and 18 left eyes (58.1%). Preoperatively, 12 eyes did not have PVR (31.6%), 13 eyes had PVR-A (34.2%) and 13 eyes had PVR-B (34.2%). Based on preoperative OCT imaging, 16 eyes (42.1%) had an attached fovea prior to surgery and 22 eyes (57.9%) had a detached fovea. Clinical characteristics of all 69 eyes are summarized in Table 4.3.

Up to 9 months after SB surgery, the mean postoperative ACD was significantly decreased (P = 0.007) compared to the mean preoperative ACD (Figure 4.2).

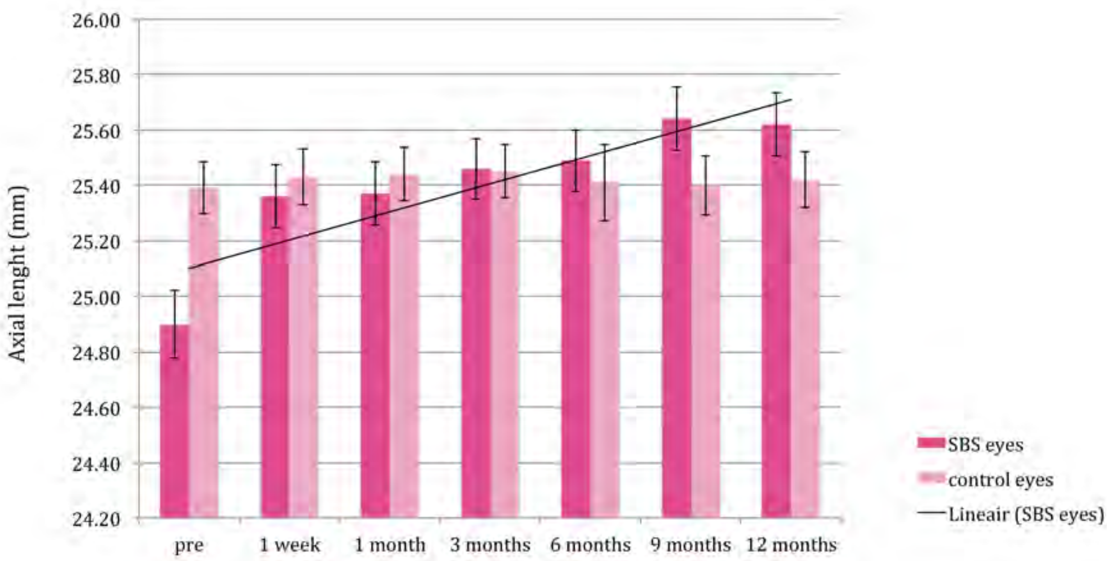

Figure 4.3: Measurements of the axial length in the SBS eyes and the control eyes

In the SBS eyes (n=14), axial length increased significantly during follow-up. Significant differences were found between 1 week and 3 months postoperatively (P = 0.024), between 1 and 6 months postoperatively (P = 0.020) and between 3 months and 9 months postoperatively (P = 0.027). No significant differences were found in the control eyes (n=31). The straight line shows a trend in the SBS eyes.

Statistics: Independent sample T-test

Preoperatively, the mean ACD was 3.33 ± 0.75 mm. On the first postoperative day, it was 2.78 ± 0.71 mm; at 1 week it was 2.99 ± 0.70 mm; at 1 month it was 3.05 ± 0.67; at 3 months it was 3.07 ± 0.68 mm; at 6 months it was 3.09 ± 0.71 mm; at 9 months it was 3.08 ± 0.70 mm; at 1 year it was 3.16 ± 0.81 mm (P = 0.144). Preoperatively, there was no significant difference in the mean ACD between the SBS eyes and the fellow eyes; (3.33 ± 0.75 mm versus 3.12 ± 0.52 mm, respectively) (P = 0.19). However, there was a significant difference in preoperative ACD between the phakic eyes (n=28) and the pseudophakic (n=10) eyes (3.04 ± 0.64 mm versus 4.13 ± 0.39 mm, P < 0.001).

Postoperatively, the mean ACD in the phakic eyes was significantly decreased up to 9 months follow-up (3.04 ± 0.64 mm versus 2.79 ± 0.37 mm) (P = 0.010). In the pseudophakic eyes, the mean ACD was significantly decreased even up to 1 year follow-up (4.13 ± 0.39 mm versus 4.04 ± 0.38 mm) (P = 0.007). On the first postoperative day, there was no significant difference in ACD between the eyes that received intraoperative gas (0.55 ± 0.60 mm) and the eyes that did not (0.53 ± 0.40 mm). In 16 out of the 32 eyes that had paracenthesis prior to intraocular gas injection, ACD was significantly decreased until 1 week after surgery, compared to the eyes without paracenthesis (n=22).

The segmental buckle group (silicone grooved strip n=25) and the radial buckle group (n=13) had significantly decreased mean ACD up to 9 months follow-up (difference of 0.29 ± 0.6 mm versus 0.15 ± 0.8 mm, P = 0.028 and P = 0.025, respectively). However, no

Table 4.2: Baseline data on patients lost to follow-up compared to study patients

Preoperative characteristics	Study eyes	Lost to follow-up	P value
Number of eyes	38	15	
Mean age (years)	58 ± 10	58 ± 18	P=0.435
Re-detachment	0	13	P<0.001
Gender male : female	20 : 18	14 : 1	P=0.05
Pseudophakic eye :phakic	10 : 28	3 : 12	P=0.376
Myopia of > 6 Dioptres (Yes : No)	10 : 28	3 :12	P=0.932
Macular involvement (Yes : No)	16 : 22	4 : 11	P=0.376
Type of Buckle (S:R)	25 : 13	12 : 3	P=0.484

S : R: segmental buckle : radial buckle

significant difference in mean ACD was found between the two buckle groups. All the patients received an encircling element of 360° around the eye; the segmental buckle extended from 90° to 270° with a mean of 108°. ACD was significantly decreased in all the SBS eyes, irrespective of whether they were myopic (>-6 D), or whether they had received cryocoagulation or scleral puncture. No significant decrease in ACD was found in the fellow eyes, even when they had received acetazolamide (n=14).

Mean preoperative axial length was 24.9 ± 1.5 mm (n=14) and was not significantly different from the mean value in the fellow eyes (n=14) (24.8 ± 1.2 mm). Mean postoperative axial length was significantly increased during the whole follow-up period (Figure 4.3). No significant differences between the preoperative and postoperative measurements were found in the fellow eyes during follow-up (n=31). At 3 months, the mean difference in spherical equivalent was 2.6 ± 1.2 D (range 0.75 - 4.75). No further increase in myopia was observed after 3 months follow-up.

Mean AAD in the SBS eyes was 13.1 ± 6.3 mm, which was not significantly different from the mean value in the fellow eyes (12.0 ± 0.5, P = 0.26). No significant difference was found between the mean preoperative AAD and the postoperative AAD measurements obtained during the whole follow-up period.

Lens rise was found to be significantly increased up to 1 year postoperatively (increase of 145.8 µm ± 161.2 µm, P < 0.001). Remarkably, a significant difference in lens rise was found preoperatively between the phakic eyes (n=10) and the pseudophakic eyes (n=28, 342.5 µm versus -830µm, P < 0.001). No differences in lens rise were found between the eyes that received a radial buckle and the eyes that received a segmental buckle.

Table 4.3: Patients characteristics before, during and after surgery

Clinical variable		SBS eyes (n=38)	Percentage (%)	Fellow/control eye (n=31)	Percentage (%)
Gender	Male	20	52.6	16	51.6
	Female	18	47.4	15	48.4
Eye	Right	23	60.5	13	41.9
	Left	15	39.5	18	58.1
Pseudophakic eye	Yes	10	26.3	4	12.9
	No	28	73.7	27	87.1
Myopia of > 6 Dioptres	Yes	10	26.3	9	29.0
	No	28	73.7	22	71.0
Quadrants involved	1	16	42.1		
	2	19	50		
	3	2	5.3		
	4	1	2.6		
PVR	None	12	31.6		
	PVR A	13	34.2		
	PVR B	13	34.2		
Macular involvement	Yes	16	42.1		
	No	22	57.9		
Type of retinal tear	Horseshoe	25	65.8		
	Hole	12	31.6		
	Tear	1	2.6		
Cumulative size of the retinal tear in Disc Diameters	1	25	65.8		
	2	7	18.4		
	3	4	10.5		
	>3	1	2.6		
	Missing	1	2.6		
Paracenthesis	Yes	16	42.1		
	No	22	57.9		
Use of intravitreal gas	Yes	32	84.2		
	No	6	15.8		
Type of buckle	Segmental	25	65.8		
	Radial	13	34.2		
Subretinal fluid drainage	Yes	33	86.8		
	No	5	13.2		
Transscleral cryopexy	Yes	23	60.5		
	No	15	39.5		
Use of Acetazolamide	None	24	63.2	17	54.8
	250 mg	1	2.6	1	3.2
	500 mg	5	13.2	5	16.1
	750 mg	2	5.3	2	6.5
	1000 mg	5	13.2	5	16.1
	1500 mg	1	2.6	1	3.2

PVR: proliferative vitreoretinopathy; RD: retinal detachment; SBS eyes: eyes that underwent scleral buckling surgery

On the first postoperative day, mean IOP in the SBS eyes was significantly higher than the preoperative value (n=37, 19.4 ± 5.4 mmHg versus 14.2 ± 3.7 mmHg, respectively) (P = 0.016). No significant changes in mean IOP were observed at 1 week or at 1, 3, 6, 9 and 12 months follow-up in the SBS eyes. None of the eyes developed secondary angle closure glaucoma.

In the eyes that received gas tamponade during SB surgery, mean IOP was significantly higher than the mean preoperative value only on the first day after surgery (P < 0.001); this situation did not apply to the eyes without gas tamponade (P = 0.08). Furthermore, on the first postoperative day, mean IOP was significantly higher in the eyes that did not receive acetazolamide than in the eyes that did (19.6 ± 4.9 mmHg versus 13.3 ± 3.9 mmHg) (n = 18; P = 0.001).

Discussion

In this study, we demonstrated that ACD was significantly decreased up to 9 months after SB surgery. ACD had returned to normal levels at one year follow-up.

Decreased ACD after SB surgery was also described by Fiore et al.[5] They found a depth difference of 0.44 mm one week after surgery (n=14), compared to 0.34 mm in our study. After 2 months, they found a mean ACD difference of 0.13 mm, compared to a mean difference of 0.26 mm after 3 months in our study. An explanation for the discrepancies between the results may lie in the measurement methods. Fiore et al used a slit-lamp technique, whereas we used AS-OCT in a large group of patients. Meinhardt et al[11] did not find any significant differences between slit-lamp, IOL-Master, AC-Master and Pentacam measurements,[11] but they did not include AS-OCT, which was used in our study. Cetin et al[4] monitored ACD with Orbscan II up to 3 months after SB surgery. They also found a significant decrease in ACD within the same time frame.[4] However, their measurements did not continue beyond 3 months follow-up. Burton et al[2] found a significant decrease in ACD up to 6 weeks after SB surgery using A-scan ultrasonography.[2]

Over the past 40 years, various techniques have been developed to measure ACD, such as A-scan ultrasonography, Orbscan II, IOL-Master and Pentacam rotating Scheimpflug camera.[11-20] In our study, we used AS-OCT, which is a non-contact anterior segment optical coherence imaging method. This choice was based on evidence of the high reproducibility and repeatability reported in previous studies.[9, 21] In the studies by Lavanya et al[21] and Nemeth et al,[9] it was shown that AS-OCT measurements were significantly deeper than those obtained with IOL-Master or US immersion A-scan. This implies that the decrease in ACD in our study may be even more profound. Elbaz et al[15] reported that measurements obtained with three different devices, such as Pentacam, A-scan ultrasonography and IOL Master, are not necessarily comparable or interchangeable. For this reason, we only used one device in the present study. Moreover, no significant intra-investigator and/or inter-investigator differences were found. Furthermore, all the measurements were performed by one investigator, which may have led to higher reproducibility.

Potentially, discrepancies between ACD results can be explained by differences in SB techniques: in the older studies by Fiore et al[5] and Burton et al,[2] the majority of eyes received hard silicone explants or scleral implants, whereas in our study, only scleral explants were used, without dissection of the sclera. We did not find any significant difference in postoperative ACD between the use of radial or segmental buckles. All our SBS eyes had an additional encircling band, which may itself have caused a decrease in ACD. A possible explanation is as follows: if encircling elements reduce the uveal or retinochoroidal circulation, ciliary body oedema is a likely consequence.[22] This ciliary body oedema with forward rotation of its body and forward shift of the iris-lens diaphragm, in combination with compression by the encircling element on the vitreous, might significantly decrease the ACD.[22] We believe that this could be the major reason for the decrease in ACD found in our study. The accompanying increase in lens rise lends support to this idea. Further potential evidence was provided by Burton et al, who found an increase in lens thickness for at least 6 weeks after SB surgery.[2]

Fiore et al[5] found a minimal change of 1 grade decrease in anterior chamber angle (according to the classification of Becker and Schaffer[23]) in the quadrant directly anterior to the intrascleral implant.[5] In a histopathological study on rhesus monkeys, Berler and Goldstein[24] showed that the ciliary body rotated around the scleral spur after SB surgery. This might explain the higher incidence of angle closure glaucoma reported after SB procedures. In the present series of 38 eyes, however, mean IOP was not significantly higher, except at 1 week after SB surgery. None of our cases presented with acute angle closure glaucoma.

We found a significant increase in axial length, which even continued to increase during follow-up. It resulted in a mean myopic shift of 2.6 D 3 months after SB surgery. Smiddy et al[25] observed a larger difference in axial length than ours. Possible explanations for these differences are inter-surgeon variance or variation between surgical techniques. Axial length may be influenced by the tension of the encircling element. Citirik et al,[26] however, did not find any increase in axial length. Burton et al[2] even found a decrease in axial length after surgery. A plausible explanation for the decrease in axial length is that Burton et al[2] used a technique with silicone implants under scleral flaps[2], which resulted in scleral shortening. Burton et al[2] only included patients with an attached macula, so the axial length measurements were probably more reliable, as was also the case in our study.

In our study, ACD was significantly decreased up to 9 months after SB surgery. Saxena et al[8] reported a significant negative correlation between ACD and endothelial cell loss. They suggested stricter inclusion criteria for pIOL implantation surgery: ACD should be at least 3.5 mm, measured from the corneal epithelium to the anterior pole of the crystalline lens or IOL, in order to prevent an excessive rate of endothelial cell loss after pIOL implantation. If a patient presents with an eye that has a combination of myopic pIOL, RRD and an ACD of less than 3.5 mm, the vitreoretinal surgeon may consider performing primary vitrectomy instead of SB surgery. Marigo et al[27] showed that uncomplicated pars plana vitrectomy did not induce any long-term changes in anterior segment morphometry. A potential disadvantage of performing vitrectomy in eyes with RRD and a pIOL is the progressive development of cataract, making it necessary to remove the pIOL.[28, 29] On the other hand, if a patient with a history of SB surgery wants to have a pIOL implanted and the ACD is too shallow, dissection of the encircling element is potentially a good option, as was described by Kreissig et al.[30]

A weakness of our study was that the measurements were performed in a non-masked fashion, with the use of AS-OCT alone. Therefore, the results may not be fully applicable to a clinical setting without this equipment.

In conclusion, ACD was significantly decreased up to 9 months after SB surgery, while the axial length increased. AS-OCT was an accurate and practical device for the follow-up of ACD after SB surgery. In patients with prior SB surgery who plan to have a myopic pIOL implanted, dissection of the encircling element may be considered if the anterior chamber has become too shallow, with the aim of preventing excessive endothelial cell loss.

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Chapter 5

Diplopia was not predictable and not associated with buckle position after scleral buckling surgery for retinal detachment

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Abstract

Background or Purpose: The risk of postoperative binocular diplopia is seen as an important drawback of conventional scleral buckling (SB) surgery for rhegmatogenous retinal detachment (RRD). The goal of this study was to evaluate the incidence of binocular diplopia and the pattern of progression after SB surgery in patients with RRD.

Methods: In retrospect, postoperative data on the occurrence of binocular diplopia were retrieved from the medical records of 1030 patients with primary RRD who underwent SB surgery between January 2001 and July 2008.

Results: Secondary strabismus occurred in 39 subjects (3.8%) during a mean follow-up of 6.4 ± 6.3 months. Twenty-eight patients (2.7%) developed strabismus due to mechanical restriction of one of the ocular muscles. No association was found between the position of the buckle, i.e. the muscle affected, and the incidence of diplopia. A moderately significant association was found between the involvement of two muscles and a higher incidence of diplopia. However, this did not apply to the involvement of three or more muscles. In 28 out of the 39 patients, binocular single vision was restored at the end of follow-up. In the majority of cases, this had been accomplished with conventional prism treatment.

Conclusions: Strabismus due to restriction of the ocular muscles after SB surgery was not predictable on the basis of buckle position. Conventional prism treatment proved successful in patients with minimal restriction of the muscles after SB surgery.

Introduction

For many vitreoretinal surgeons, scleral buckling (SB) surgery is the treatment of choice for primary rhegmatogenous retinal detachment (RRD) with limited PVR.[1, 2] Adversaries of SB surgery emphasize that diplopia is one of the most important postoperative complications, which can be avoided by performing primary vitrectomy instead.[3-5]
Diplopia was reported in 3% to 40% of the patients after SB surgery.[3, 4, 6-15] Multiple causes have been proposed.[3, 4, 14, 16-20] One mechanism that may play an important role is ocular muscle restriction due to leverage from the buckle. Therefore, an association might be expected between the affected ocular muscle and the buckle that is placed beneath it. The goal of this study was to investigate the incidence of binocular diplopia and the pattern of progression in a large group of patients who had recently undergone SB surgery. Secondary aims were to analyse whether strabismus was predictable after SB surgery and to identify the type of treatment that had mostly been used to resolve the diplopia.

Materials and methods

A retrospective review was made of the medical records of all the patients with RRD who had undergone primary SB surgery without vitrectomy between January 2001 and July 2008. Seven different surgeons had performed the operations (FH, ELH, AL, IL, VN, WJ and EB). We excluded any patients with a history of strabismus and/or thyroid disease. Medical records were matched with the data obtained during our orthoptic consultations. In 39 out of the 1030 patients, the orthoptic consultation included the measurement of visual acuity and the range of ocular movement. Patients had been referred to the orthoptist due to complaints of binocular diplopia. The orthoptist used the serial Hess charts to examine patients for muscle restriction and the prism cover-test to measure the angle of heterotropia. To calculate the mean angles, we used mean horizontal deviations from patients with horizontal diplopia and mean vertical deviations from patients with vertical diplopia. Absolute values were used in the statistical analysis to calculate the mean deviation. The study was conducted in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.
During SB surgery, a silicone encircling element (reference number 240, MIRA Inc. Uxbridge, MA, USA) was placed beneath the rectus muscles, with either a segmental buckle (silicone grooved strip no. 506G, DORC International, Zuidland, the Netherlands) and/or a radial buckle (solid silicone wedge, G135, MIRA Inc., Uxbridge, MA, USA, or G137, Labtician Ophthalmics Inc., Oakville, Canada). Data on the position of the buckle could easily be retrieved from the surgical reports, because all the surgeons had drawn a diagram or described in detail the exact location of the buckle. The encircling band was fixed 10-12 mm posteriorly to the limbus with a mercilene 5.0 suture. Whether a radial or segmental buckle was selected depended on the size and location of the retinal tear.

Subretinal fluid was drained when judged necessary by the surgeon. Cryocoagulation was performed in some cases, according to the preferences of the surgeon. Intravitreal gas tamponade was used for RRDs in the upper quadrants, located between the 8 and 4 o'clock positions.
All the patients received routine postoperative treatment with antibiotic eye drops (chloramphenicol 5 mg/ml) 4 times daily for 14 days (Ratiopharm, Zaandam, the Netherlands) and prednisolone eye drops (Pred Forte®, prednisolone acetate 10 mg/ml, Allergan BV, Nieuwegein, the Netherlands) 3 times daily for 2 months. Patients with phakic eyes also used mydriatic eye drops (atropine sulphate 1%) 2 times daily for 1 week after the operation (Chauvin Pharmaceuticals Ltd, Surrey, UK). Patients with pseudophakic eyes used additional tropicamide 0.5% (Thea Pharma, Wetteren, Belgium) 2 times daily for 1 week.
The following patient characteristics were noted: gender, age, right or left eye, re-detachment (yes/no), RRD in the fellow eye (yes/no), removal of the scleral buckle or encircling element (yes/no). SB-surgery-related variables were also noted: radial or segmental silicone buckle and muscle or muscles involved, subretinal fluid drainage (yes/no), cryocoagulation (yes/no), intravitreal sulphur hexafluoride (SF_6) gas tamponade (yes/no). Additional data were noted in patients with diplopia: macula involved in the retinal detachment (yes/no), duration of diplopia after surgery, visual acuity at the time of diplopia, cause of the diplopia and treatment received, treatment successful (yes/no). In our study, 'diplopia' refers exclusively to binocular diplopia. Mechanical strabismus

Table 5.1: Patient characteristics in the groups with and without diplopia

Total no. of patients n=1030	Patients without diplopia (total n=991) n (%)	Patients with mechanical strabismus (total n=28) n (%)	P value (T-test)
Age (years)	58.1±13.5	58.6 ± 10.6	P=0.861
Gender m : f (%)	644 : 347 (63 : 37)	22 : 6 (79 : 21)	P=0.133
Re-detachment (%)	185 (18.7)	9 (32.2)	P=0.085
Fellow eye detachment (%)	148 (14.9)	4 (14.3)	P=0.920
Buckle beneath superior rectus (%)	508 (50.6)	13 (46.4)	P=0.663
Buckle beneath lateral rectus (%)	473 (47.7)	13 (46.4)	P=0.888
Buckle beneath medial rectus (%)	193 (19.5)	8 (28.6)	P=0.234
Buckle beneath inferior rectus (%)	224 (22.6)	7 (25.0)	P=0.767
Punction	839 (84.7)	23 (82.1)	P=0.731
Cryocoagulation	546 (55.1)	15 (53.6)	P=0.866
Intravitreal gas	750 (75.7)	19 (67.9)	P=0.345
Radial buckle	216 (25.5)	10 (35.7)	P=0.179
Segmental buckle	716 (72.3)	17 (60.7)	P=0.459
Mean no. of muscles involved	1.4	1.4	P=0.699

was defined as diplopia due to mechanical restriction. Functional diplopia was defined as diplopia due to decompensation of former heterotopia and limited fusional amplitudes caused by decreased visual acuity.

Snellen visual acuities were converted into a logarithmic scale (LogMAR, i.e. the logarithm of the minimal angle of resolution) as described previously.[21] In this study, poor visual outcome was defined as less than 0.1 Snellen visual acuity at 12 months follow-up. Statistical analysis was performed using the SPSS software 15.0 (SPSS for Windows, Rel. 13.0 Chicago: SPSS Inc., USA) with independent sample T-test and $\chi 2$ test. Significance was defined as $P < 0.05$.

Results

Between January 2001 and July 2008, a total of 1030 patients with primary RRD underwent SB surgery. Mean age was 58.1 ± 13.4 years.

In the patients with diplopia (n=39) mean age was 59.3 ± 12.7 years (not significantly different from the whole group). The diplopia group comprised 24 right eyes (61.5%), 15 left eyes (38.5%), 28 men (71.8%) and 11 women (28.2%); 6 patients had retinal detachment in the fellow eye; 24 segmental buckles had been used compared to 14 radial buckles.

All the patients attended regular follow-up visits after SB surgery. Patients with an uneventful and uncomplicated postoperative course and the patients without complaints of diplopia returned to their referring ophthalmologist after approximately three to six months. The patients who complained of diplopia (n=39) were sent for orthoptic evaluation after a mean follow-up of 6.4 ± 6.3 months (range 1-24 months). Total duration of follow-up in this group was 41.2 ± 23.3 months (range 9-48 months). Within the group of 39 patients who complained of diplopia, 28 patients (2.7%) had mechanical strabismus due to mechanical restriction of the ocular muscles. These patients formed our diplopia study group. Their first orthoptic assessment took place 4.1 ± 3.7 months postoperatively. The macula was involved in the detachment in 15 patients. Superior rectus muscle restriction was present in 14 patients (confirmed by Hess chart) and in 9 of them (64.3%) the buckle lay beneath this muscle. Inferior rectus muscle restriction was present in 13 patients (confirmed by Hess chart) and in 5 of them (38.4%) the buckle lay beneath that muscle. Lateral rectus muscle restriction was present in one patient and the buckle lay beneath that muscle. Patient characteristics in the diplopia group and the non-diplopia group are shown in Table 5.1. Ten patients (1.0%) had functional diplopia due to decompensation of former heterophoria, low visual acuity and inefficient fusion. Paresis of the trochlear nerve was found in one patient (0.1%). None of the patients with diplopia had thyroid eye disease.

In the patients with vertical diplopia (n=25) mean vertical deviation at 30 cm was 6.0 ± 5.1 Δ versus 3.7 ± 1.5 Δ in the patients with functional diplopia (n=5) (P=0.06). At 6 m, the mean values were 6.7 ± 5.4 Δ and 7.0 ± 5.4 Δ, respectively (P=0.87). In the patients with

Table 5.2: Buckle placement beneath muscles in the groups with and without diplopia

No. of muscles involved	Non-diplopia (n=991)	Diplopia (n=28)	P value
0	208	6	0.955
1	314	7	0.453
2	342	13	0.037
3	124	2	0.395
4	3	0	0.771

horizontal diplopia (n=11) mean horizontal deviation at 30 cm was 6.7 ± 5.3 Δ versus 16.4 ± 13.4 Δ in the patients with functional diplopia (n=5); this was a significant difference (P=0.04). When measured at 6 m, the mean values were 8.0 ± 7.3 Δ and 15.2 ± 12.4 Δ, respectively (P=0.06).

Table 5.1 lists the positions of the buckle in the patients in the diplopia group and the non-diplopia group. No association was found between the buckle position or type of buckle and the incidence of diplopia. Similarly, no correlation was found between the position of the buckle and restriction of the muscle under which the buckle had been placed. In the majority of patients with mechanical strabismus, the buckle was not beneath the muscle that would be expected to be associated with the diplopia. Furthermore, in six out of the 28 patients (21%) in the diplopia group, the surgical reports revealed that no buckle had been placed beneath any of the rectus muscles. A buckle had been placed beneath one muscle in 7 patients (25%), beneath two muscles in 13 patients (47%) and beneath three muscles in 2 patients (7%). It was only when two muscles were involved that we found a moderately significant association with the incidence of diplopia. No significant associations were found when 3 or 4 muscles were affected (Table 5.2).

Visual acuity in LogMAR was 0.41 ± 0.47 (20/50) in the diplopia group versus 0.27 ± 0.32 (P=0.201) (20/40) in the patients with functional diplopia. In the fellow eye, visual acuity in LogMAR was 0.00 ± 0.09 (20/20) in the diplopia group versus 0.18 ± 0.41 (P=0.01) (20/30) in the functional diplopia group.

Treatment for diplopia

In the diplopia group, 17 out of the 28 patients regained binocular single vision after prism treatment: 16 patients had prisms for vertical deviation and one patient had a prism for horizontal deviation. Another two patients regained binocular single vision after a training programme with fusional exercises. One patient underwent surgical strabismus correction, but without success and was ultimately advised to occlude the eye. In two other patients, the diplopia disappeared after adjusting the refractive error. Four patients with minimal vertical deviation followed a “no treatment policy” and the diplopia resolved spontaneously after 6 months. Prism treatment was unsuccessful in two patients, who were ultimately advised to occlude the eye.

In the group of patients with functional diplopia who were considered for treatment for the diplopia (n=10) one patient was treated successfully with a prism and one patient recuperated after optimal refractive correction. It was necessary to occlude the eye in 4 cases. Three other patients declined further treatment. The scleral buckle and encircling element had to be removed from one patient, which resulted in resolution of the diplopia. Trochlear nerve paresis (n=1) resolved spontaneously within 6 months.

Ultimately, binocular single vision was restored in 28 (72%) out of the 39 patients at the end of follow-up.

Discussion

In our series of 1030 eyes that underwent SB surgery for RRD, secondary diplopia occurred in 3.8% after a mean follow-up of more than six months. Orthoptic assessment confirmed the presence of mechanical restriction in 2.7% of the total series. No significant correlation was found between the position of the buckle or the type of buckle and the incidence of diplopia or the pattern of strabismus. A moderately significant association was found between the involvement of two muscles and a higher incidence of diplopia. However, this did not apply to the involvement of three or four muscles. Therefore, we did not observe the expected trend of: the more muscles affected, the higher the incidence of diplopia. It cannot be ruled out that this was due to small group size. Based on our calculations and statistics, we can conclude that in the present retrospective study, diplopia was not associated with the position of the buckle and there was only a moderate association with the number of muscles affected. Moreover, in the majority of patients in the diplopia group, the buckle was not beneath the muscle that would be expected to be associated with the diplopia. Therefore, the incidence of diplopia was only predictable in a minority of the patients.

It has previously been suggested that transient diplopia after SB surgery can be attributed to cryocoagulation of a muscle.[3] In our series, this may explain the disappearance of the diplopia within 6 months in two patients who received conservative treatment with fusional exercises from an orthoptist. Another possible explanation for diplopia after SB surgery is scarring of the conjunctiva near the limbus, which would induce shortening, i.e. mechanical restriction, as was proposed in a study on pterygium excision.[22]

Kanski et al[4] published a study on 750 SB surgery patients in 1973. They reported that 25 out of the 30 patients who developed diplopia underwent a secondary procedure to mobilize the muscles, which was standard procedure in those days. In the present study, however, we did not mobilize any of the muscles or perform tendonectomies.

Many causes for strabismus have been described in the literature (Table 5.3). Local anesthetics[7] sometimes cause anaesthetic myotoxicity, with temporary restrictive

Table 5.3: Summary of studies on patients with diplopia after retinal detachment surgery

Author	Year	N	% Diplopia	Δ Angle (horizontal)
Wu et al[23]	2005	6	NN	25- >90Δ
Langmann et al[24]	2003	18	7%	36.4Δ
Salama et al[7]	2000	36	NN	3-40Δ
Farr and Gyton[17]	2000	Review		
Wright et al[25]	1999	40		
Maurino et al[26]	1998	68	NN	2-70Δ
Cooper et al[27]	1998	37	NN	Horizontal 0-36Δ Vertical 0-32Δ Torsional 3-10Δ
Macleod et al[28]	1997	2		
Berk et al[15]	1996	44	13.6%	
Seaber et al[14]	1995		5-25%	
Schrader et al[8]	1995	45	15.5%	
Spencer et al[29]	1993	70	12%	NN
Hwang et al[30]	1992	31	NN	NN
Kalman et al[31]	1992	117	6%/11%/30%	NN
Petitto and Buckley[32]	1991	20	NN	10-60Δ
Lee et al[33]	1991	31	NN	18-50 Δ
Lawin-brussel et al[12]	1991			
Scott et al[34]	1990	21	NN	5-45 Δ
Eckardt et al[35]	1990	33	30%	NN
Smiddy et al[36]	1989	69	37.7% strabismus 4.3% diplopia	
Maillette de Buy Wenniger-Prick et al[5]	1988	18	4.5%	1-16 Δ
Munoz et al[22]	1987	33		4-40 Δ
Metz and Norris [23]	1987	4	NN	6-40 Δ
Fison and Chignell[3]	1987	14	4.8%	
Wright[6]	1986	7		15-70 Δ
Metser et al[38]	1986	60	80% strabismus 40% diplopia	
Mets et al[39]	1985	30	10% (at 6 months)	2-10Δ (at 6 months)
Peduzzi et al[10]	1984	8/3	3% muscle imbalance 2.5% diplopia	NN
Sewell et al[40]	1974	19	14% muscle imbalance	
Kanski et al[4]	1973	30	6% muscle imbalance 3.3% diplopia	NN
Arruga et al[41]	1973	90	10% with large comitant deviation, 13% medium size motility imbalance 16% small tropias, 51% orthophoric	Up to 8Δ vertically and 20Δ horizontally

Table 5.3 (continued)

Author	Treatment	Scleral buckling technique
Wu et al[23]	1. Reinsertion, 2. Declined by patient 3. Reinsertion and Δ 4. Reinsertion and SC 5. Reinsertion LR and SC 6. Reinsertion	1. EE + SGB, 2. NN 3. NN, 4. NN 5. EE, SB+PPV 6. 260° EE and SB +PPV
Langmann et al[24]	12 Δ, 5 OK 1 OC	EE alone
Salama et al[7]	9 Δ, 27 recommended SC, 17 operated on	NN
Farr and Gyton[17]		
Wright et al[25]		
Maurino et al[26]	17.6% SC 38.2% BT in muscle (2.9 average) 19.1%Δ 11.7% OC, 13.2% refused treatment	41 eyes 1 or more explants, 27 eyes EE, with or without explant
Cooper et al[27]	100% SC	EE and SB
Macleod et al[28]	SBR, SC	
Berk et al[15]	Mostly expectative	EE +RB or SGB
Seaber et al[14]		
Schrader et al[8]	Prisms	RB
Spencer et al[29]	6 prism 1 SBR +SC+BT	58 CFE 2 RB 10 CFE+RB
Hwang et al[30]	1. Bilateral recession 2. Recession resection procedures 3.Removal of Tenon's capsule 4. Advancement of slipped muscle 5. Repositioning of displaces superior oblique 6. Temporary traction suture 7 Removal of explant	NN
Kalman et al[31]	NN	54 SB 63 EE + RB or not
Petitto and Buckley[32]	BT in muscle	NN
Lee et al[33]	BT in muscle, 3 SC	NN
Lawin-brussel et al[12]		
Scott et al[34]	BT in muscle	NN
Eckardt et al[35]	4 SC or prisms	16 SB surgery 17 EE (all 33 PPV+ SOI)
Smiddy et al[36]	SC in one and prisms in two eyes	EE or SB CFB, EE was anchored with scleral tunnel
Maillette de Buy Wenniger-Prick et al[5]	4 SC, 4Δ, 5 was not restored, 2 CHP, 3 expectative	NN
Munoz et al[22]	20 SC	EE or 360° explant, RB, SGB
Metz and Norris [23]	SC and SBR	
Fison and Chignell[3]	1st Δ, 2nd SBR, 3rd SC	
Wright[6]	SC, OC	6 360° buckle, 5 mm radial buckle. During surgery 1 slipped muscle and 1 inadvertent section
Metser et al[38]		30 balloon buckle or 30 silicone sponge
Mets et al[39]	1 Δ, 2 expectative	EE with radial buckle or radial buckle alone
Peduzzi et al[10]	4 spontaneously recovery 2 Δ, 2SC	2/8 had muscle mobilization, 4 had the scleral buckle over the muscle insertion
Sewell et al[40]	½ of patient received tendontomy	
Kanski et al[4]	15 spontaneous and complete recovery, 3 control imbalance, 4Δ, 1 occlusion, 1 expectative, 1 SC	25/30 patients had muscle detached. 11 EE with 7 scleral resection and 4 buckle, 8 local procedure with 2 scleral resection and 6 buckle
Arruga et al[41]	NN	Light coagulation, retinopexy, scleral resection (with or without recession of lateral rectus) indentation methods and EE (n=29)

N: number , NN: not noted, EE: encircling element, RB: radiar buckle, SGB: segmental buckle, CFE: circumferential explants, SBR: scleral buckle removal, Δ:prisms, F-U: follow up SC: strabismus correction, OC:occlusion, EOM:extra ocular muscle, SB: scleral buckling, PPV: pars plana vitrectomy, SOI: silicone oil injection, BT: botulinum toxin, CHP: compensatory head posture

Table 5.3 (continued)

Author	Success	Cause of diplopia/ major
Wu et al[23]	All except 2 patients who declined treatment	Mechanical
Langmann et al[24]		
Salama et al[7]	88% of SC	Mechanical
Farr and Gyton[17]		
Wright et al[25]		
Maurino et al[26]	75% of SC. 96% of BT (although with repeated treatment) 77% of Δ	NN
Cooper et al[27]	9 mild residual deviation 1 no follow-up 6 successful	70% mechanical. 30% NN
Macleod et al[28]		Mechanical
Berk et al[15]		Mainly mechanical
Seaber et al[14]		
Schrader et al[8]		Mechanical
Spencer et al[29]		Mechanical Note: a second procedure is more likely to cause restricted motility
Hwang et al[30]		30 mechanical, 4 patients functional, 3 previous strabismus
Kalman et al[31]	NN	NN
Petitto and Buckley[32]	73% 1 or 2 injections	NN
Lee et al[33]	62%	NN
Lawin-brussel et al[12]		
Scott et al[34]	60%	NN
Eckardt et al[35]		NN
Smiddy et al[36]		Mechanical
Maillette de Buy Wenniger-Prick et al[5]	5/8 fusion was restored	NN
Munoz et al[22]	Specified per group	Mechanical
Metz and Norris [23]	25%	50% unknown, 50% mechanical
Fison and Chignell[3]	80%	
Wright[6]	20%	Mechanical
Metser et al[38]		Mechanical
Mets et al[39]		NN
Peduzzi et al[10]		Mechanical
Sewell et al[40]		NN
Kanski et al[4]	96%	Mechanical
Arruga et al[41]	NN	

Table 5.3 (continued)

Author	Duration of strabismus after SB surgery	F-U	Note
Wu et al[23]	8 -120 months	NN	Cause of diplopia found with MRI
Langmann et al[24]			Local anaesthetics
Salama et al[7]	NN	NN	
Farr and Gyton[17]			
Wright et al[25]			SB surgery compared to PPV
Maurino et al[26]	NN	SC 4-48 months BT 2-60 months	
Cooper et al[27]	≥6 months	6-56 months mean 8	
Macleod et al[28]			Case report
Berk et al[15]	0-6 months	6 months	Prospective study
Seaber et al[14]			Review
Schrader et al[8]		2-4 years	
Spencer et al[29]	After 6 months	Mean 20.1 months (9-32)	
Hwang et al[30]	NN	NN	
Kalman et al[31]	NN	NN	
Petitto and Buckley[32]	NN	NN	
Lee et al[33]	One year	NN	
Lawin-brussel et al[12]			
Scott et al[34]	4 months-20 years	5-96 months	
Eckardt et al[35]	NN	6-29 months	
Smiddy et al[36]		4-20 months	
Maillette de Buy Wenniger-Prick et al[5]	NN	NN	
Munoz et al[22]	More than 6 months	6 months to 17 years	
Metz and Norris [23]			
Fison and Chignell[3]		Mean 4 years	
Wright[6]		2 months to 2 years	
Metser et al[38]		26 weeks	Prospective study
Mets et al[39]	After surgery till 6 months	6 months	
Peduzzi et al[10]	NN	NN	
Sewell et al[40]			
Kanski et al[4]	NN	1-10 years	More vertical muscle imbalance 3:23
Arruga et al[41]	After 5 moths	5 months to 2 years	

N: number , NN: not noted, EE: encircling element, RB: radiar buckle, SGB: segmental buckle, CFE: circumferential explants, SBR: scleral buckle removal, Δ:prisms, F-U: follow up SC: strabismus correction, OC:occlusion, EOM:extra ocular muscle, SB: scleral buckling, PPV: pars plana vitrectomy, SOI: silicone oil injection, BT: botulinum toxin, CHP: compensatory head posture strabismus, as was reported by Salama et al.[7] This cause can be ruled out in the present series, because all the patients were operated on under general anaesthesia, without any additional locoregional supplementation. Ischaemia of the rectus muscles may also explain diplopia after SB surgery. An ischaemic muscle loses its function and/or strength. Ischaemia of the rectus muscles may develop due to muscle constraint, or arterial occlusion by the encircling element.[23] A study by Lincoff et al[23] showed that the encircling band significantly reduced the pulsatile ocular blood flow by a mean of 43% compared to the fellow eye. Subsequent cutting of the band resulted in a mean recovery of up to 85.6%.[23] In addition, when a muscle is constrained too aggressively, it may become seriously traumatized, resulting in haemorrhage and/or oedema of the muscle, but also ultimately to fibrosis and scarring, with restriction of its function.[14] Trauma with scarring was also held responsible for the ocular motility disturbances that occurred after the placement of Baerveldt glaucoma implants under or on top of the superior rectus muscle or lateral rectus muscle.[24] In some of our cases, this mechanism of trauma with scarring may be the explanation, as there was no apparent association between the position of the buckle and the pattern of diplopia. It was striking that none of the buckles were beneath any of the muscles in six of our patients with diplopia (21%). All six cases had a fusional problem as a result of de-compensation of former heterophoria caused by decreased visual acuity.

Magnetic resonance imaging (MRI)[25] identified various pathological characteristics in the extra ocular muscles 8-120 months after SB surgery. Deviation varied between 25Δ to more than 90Δ (n=6), whereas in the present study, a mean deviation of 6Δ was found. The MRI study[25] also reported exposure and anterior migration of the scleral buckle in three cases, which was caused by complete erosion of the extra ocular muscle tendon (Table 5.3). Therefore, it can be concluded that the MRI study included severe types of pathology and that the results cannot simply be extrapolated to other cases who undergo SB surgery.

It is well-known that MIRAgel buckles give the highest rate of complications after SB surgery (MIRAgel, Medical Instruments Research Associates, Waltham, Massachusetts, USA). Progressive diplopia[18, 19, 26] was reported up to 7-11 years after the placement of MIRAgel buckles. In the present series of patients, no MIRAgel buckles were used.

Binocular single vision was ultimately restored in 72% of the patients with diplopia in the present study. Re-detachment is also a risk factor for diplopia, because it diminishes or disables existing fusion due to further deterioration in visual acuity. In patients with prolonged sensory deprivation due to macular detachment, fusion could not always be restored.[27-29] Therefore, the only possible treatment in these cases was occlusion of the eye.

We found that prism therapy was a good treatment option in patients whose strabismus was caused by mechanical restriction. In a study by Sauer et al,[30] scleral buckle removal was a successful treatment in 50% of the patients with postoperative strabismus; prisms restored binocular vision in an additional 25%, while strabismus surgery was necessary in the remaining 25%. However, a major disadvantage of scleral buckle removal, besides

a second operation, is the increased risk of re-detachment.[31-33] After scleral buckle removal, re-detachment rates were as high as 10-25%.[1, 2, 27, 29, 34]
A limitation of the present study was the retrospective design. During follow-up, the patients were not asked explicitly whether they were experiencing diplopia. Thus, we assumed that patients would be referred back to our centre if they developed diplopia more than six months postoperatively, because we are the only referral centre for these complicated strabismus cases. This may have resulted in missed cases and underestimation of the incidence of diplopia. A second limitation of this study was that we did not exclude patients with poor visual acuity. Therefore, the incidence of diplopia may have been further underestimated. In the literature, the various studies on this subject are difficult to compare, due to differences in surgical techniques. For example, in the present study, we used an encircling element and a segmental or radial buckle made of the same material (in all the cases) and we did not mobilize any of the muscles. On the basis of our analyses, we could not establish an exact cause for diplopia after retinal detachment surgery, which was in line with previous studies (Table 5.3).

Treatment recommendations for postoperative diplopia

We recommend that treatment should start with a conservative approach using prisms. If this does not yield the desired results, the buckle can be removed, or - but only and as a last resort - the buckle and the encircling element. Strabismus surgery is an alternative option.[30] Although operating on the healthy fellow eye is contradictory to the general rule of only operating on a visually impaired or compromised eye, perhaps the best result will be obtained by performing strabismus surgery on the fellow eye. The muscles of the SB surgery eye may have such extensive scarring and fibrosis that surgery on such eyes is much more difficult and far less predictable.[25] A further consideration is that strabismus surgery may not be effective if the problem is truly restrictive; for example, release of conjunctival or muscle scarring may be the most effective means of curing the strabismus. Another form of treatment is botulinum injection into a horizontal or vertical ocular muscle. Scott[35] reported a success rate of 60% and only three out of the 20 patients needed an additional injection.[35]

In summary, 2.7% of our 1030 patients had diplopia due to mechanical restriction of one of the muscles after SB surgery.[3, 4, 14, 16-20] No significant correlation was found between the position of the buckle or the type of buckle and the incidence of diplopia or the pattern of strabismus. A moderately significant association was found between the involvement of two muscles and a higher incidence of diplopia. However, this did not apply to the involvement of three or more muscles. Patients with minimal restriction of the muscles after SB surgery can often be treated successfully with prisms.

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Chapter 6

Low re-detachment rate after vitrectomy with an encircling scleral buckle and silicone oil for giant retinal tears

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Abstract

Background: The goal of this study was to assess the long-term anatomical and functional results of pars plana vitrectomy (PPV) with or without an encircling scleral buckle in patients with giant retinal tear (GRT) retinal detachment.
Subjects and Methods: 30 eyes were analysed in retrospect following PPV for GRT retinal detachment between March 1998 and August 2003.
Results: Re-detachment occurred in nine out of the 30 eyes (30%) after one vitrectomy procedure. Ultimately, re-attachment was achieved in 29 eyes (96.7%). Multivariate analysis showed that the absence of an encircling scleral buckle (P=0.008) was significantly associated with re-detachment. Visual acuity improved in 54% of the eyes.
Conclusion: Placement of an encircling scleral buckle appears to be highly recommended in the treatment of GRT retinal detachment with PPV.

Introduction

A giant retinal tear (GRT) refers to a retinal tear that extends over three clock hours or more of the circumference.[1] Treatment of choice is pars plana vitrectomy (PPV)[2-8] with repositioning of the inverted central retinal flap using perfluorocarbon liquids,[9] endolaser,[4] or cryocoagulation and silicone oil tamponade.[5, 6, 10] Some surgeons elect to use an additional encircling scleral buckle, or a scleral buckle over a portion of the circumference. The goal of this study was to identify risk factors related to re-detachment and to assess the long-term anatomical and functional results of PPV for GRT retinal detachment.

Material and methods

A retrospective study was performed on all the patients with GRT retinal detachment who underwent vitrectomy (surgeons: FH, ELH or AL) at our clinic between March 1998 and August 2003.

The following preoperative clinical patient data were collected for statistical analysis: age, gender, preoperative trauma, size of the GRT in clock hours, number of detached quadrants of the retina, central macular (foveal region) involvement (yes/no) and presence and grade of proliferative vitreoretinopathy (PVR). Preoperative and postoperative best corrected Snellen visual acuity (also with pinhole correction) was measured. The following preoperative slit-lamp examination variables were noted: lens status and phakia, pseudophakia or aphakia. Funduscopy was performed using indirect binocular ophthalmoscopy with a Goldmann three-mirror contact lens (without scleral depression) and a panfundus contact lens (Supersquad 160°). Fundus drawings were made of the retinal detachment (RD) in clock hours. By carefully questioning the patients, we determined the approximate time of onset of the detachment (visual field loss).

During follow-up, we noted whether re-detachment occurred, the interval until re-detachment and any subsequent surgical procedures. In addition, we noted whether lensectomy or phaco-emulsifaction was performed. Anatomical success was defined as complete attachment of the retina. PVR was graded according to the Retina Society Classification of 1983.[11]

The following data were collected regarding the PPV procedure: placement of an encircling scleral buckle (yes/no), gas or oil tamponade (yes/no), endolaser coagulation (yes/no) and cryocoagulation (yes/no). Our vitrectomy technique with silicone oil or gas tamponade comprised: standard three-port vitrectomy with a trocar microcannular system (Grieshaber & Co, AG, Schaffhausen, Switzerland) using a non-contact wide angle panoramic viewing system (BIOM, Carl Zeiss, Meditec, Jena, Germany). As much vitreous as possible was removed, with shaving of the vitreous base as described previously.[12, 13]

All the operations were conducted with perfluorocarbon liquid (DK-line, Chauvin Opsia, France) to cover the ora serrata and flatten the retina. Endolaser coagulation or cryocoagulation was performed around the tears to re-attach the basal retina. At the end of the procedure, the perfluorcarbon liquid was exchanged for silicone oil (1000 centistokes) or gas (C_3F_8 gas). The decision to use oil or gas was made during vitrectomy, but mostly depended on the underlying PVR. If necessary and according to the surgeon's preference, an encircling scleral buckle was placed before vitrectomy. We used an encircling narrow element (Mira Inc., scleral buckle component, reference number 240). Our silicone oil removal technique was through two pars plana sclerotomies. When additional procedures were necessary, such as endolaser photocoagulation, lensectomy, removal of epiretinal membranes, or removal of ischaemic edges of former retinectomies, three-port vitrectomy was applied.

Statistical analysis was performed using the SPSS software 15.0. Snellen visual acuities were converted to a logarithmic scale (LogMAR, i.e. the logarithm of the minimal angle of resolution).[14] Comparisons were made between preoperative and postoperative visual acuities using the paired sample T-test. Univariate analysis was performed with the Pearson Chi-square exact test to determine whether the preoperative clinical variables were associated with postoperative re-detachment. Stepwise forward conditional multiple logistic regression analysis was used on the preoperative and intraoperative variables to identify the strongest predictors of re-detachment. Kaplan Meyer survival analysis was also included.

RESULTS

A total of 30 eyes (19 right eyes (63.3%) and 11 left eyes (36.7%); 26 men and 4 women, 86.7% vs 13.3%) in 28 patients were included in this study (Table 6.1). Mean age was 53.2 years (range 30-70 years). Mean follow-up was 49 months (range 13-101 months) (Table 6.2). There were 23 phakic eyes (76.7%), six pseudophakic eyes (20%) and one aphakic eye (3.3%). In 19 out of the 30 eyes (63.3%) the macula was detached, while in 11 eyes (36.7%) the macula was attached. Preoperative PVR grade A was present in 19 out of the 30 eyes (63.3%) versus grade B in 11 eyes (36.7%). Dense vitreous haemorrhage was found in six eyes (20%); four eyes (13.3%) had suffered preoperative trauma. Mean size of the GRTs was 3.75 clock hours (range 3-6 clock hours). In 20 out of the 29 eyes (69%) visual field scotoma was present for 10 days or less, while in nine eyes (31%) visual field scotoma existed for longer than 11 days. During vitrectomy, an encircling scleral buckle was placed in 21 eyes (70%); 29 eyes (96.6%) received silicone oil tamponade and one eye (3.4%) received C_3F_8 gas tamponade. Lensectomy was performed on two eyes during primary vitrectomy. All the retinas were attached after the first operation. Anatomical success was achieved in 21 out of the 30 eyes (70%) after one vitrectomy procedure. Ultimately, the retina was attached in 29 (96.7%) eyes. At the end of follow-up, silicone oil had been removed from all the eyes except one (follow-up: 80 months) (Table 6.1). Although the retina was attached in this case, corneal

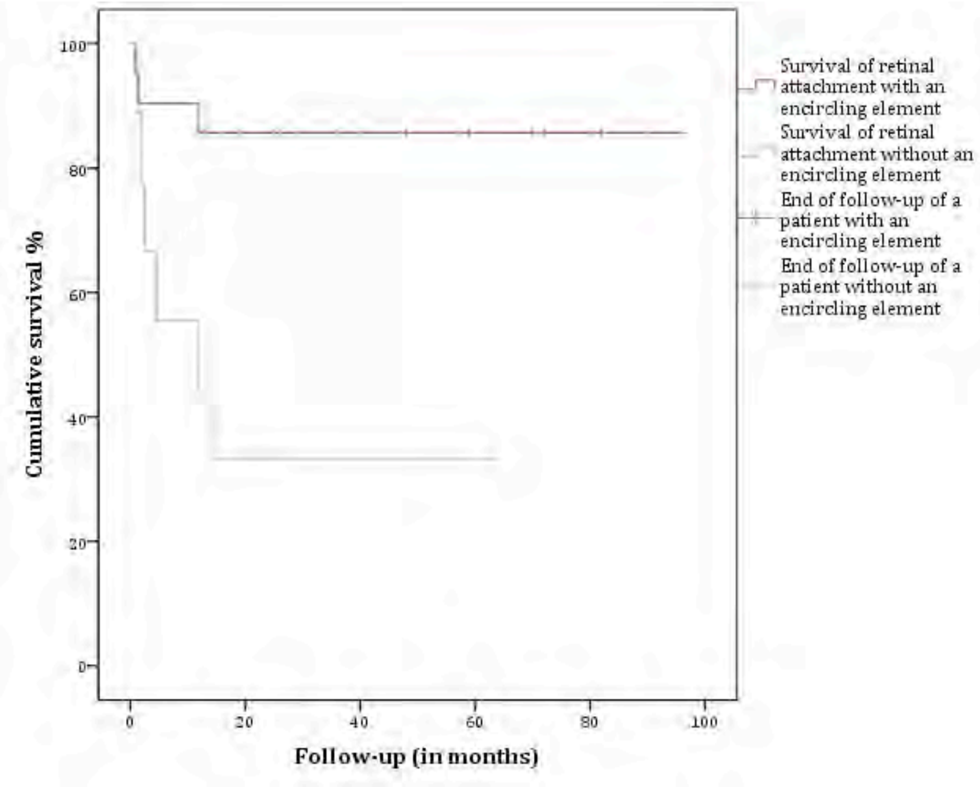

Figure 6.1: Kaplan Meyer cumulative survival curve of retinal attachment in patients with or without an encircling scleral buckle

Difference between groups was significant (P=0.004)

decompensation was present. Therefore, the patient and surgeon agreed not to undertake any further operations because of the poor prognosis.

Re-detachment occurred in nine eyes after PPV. All nine eyes underwent subsequent vitreoretinal procedures to re-attach the retina. Mean interval until re-detachment was 24.7 weeks (range 4-64 weeks, median 11 weeks). In five eyes, re-detachment occurred within three months; another eye re-detached in the following three months and three more eyes re-detached one year after PPV.

Univariate analysis showed that an age of older than 60 years (P=0.030), the absence of an encircling scleral buckle (P=0.008) and pseudophakia (P=0.049) were significantly associated with re-detachment (Table 6.3). Multivariate analysis revealed that the absence of an encircling scleral buckle (P=0.008) was significantly associated with re-detachment. Figure 6.1 shows a Kaplan Meyer survival plot of retinal attachment after PPV with and without an encircling scleral buckle (P=0.004). Six out of the nine eyes that re-detached did not have an encircling scleral buckle, versus three out of the 21 eyes that did have an encircling scleral buckle.

Table 6.1: Patient characteristics

Pt	Gender	Age (yrs)	Eye	Lens status pre	Size GRT	Macula	PVR grade	CVH	Trauma	Encircling scleral buckle	Detached after one PPV	F-U (mnths)	attached EF-U	SO EF-U	lens status EF-U
1	male	64	OD	phakic	3	off	A	yes	yes	yes	no	47	yes	no	psphakic
2	male	67	OD	phakic	3	off	A	no	no	no	yes	101	yes	no	psphakic
3	male	53	OS	phakic	6	on	A	no	no	yes	no	96	yes	no	psphakic
4	female	62	OD	psphakic	3	off	B	no	no	no	yes	80	yes	yes	aphakic
5	male	50	OS	phakic	5	off	A	yes	no	no	yes	24	yes	no	aphakic
6	male	65	OD	psphakic	3	on	A	no	no	yes	yes	50	yes	no	psphakic
7	male	70	OS	psphakic	3	off	B	yes	no	yes	yes	18	yes	no	psphakic
8	male	56	OD	phakic	3	on	B	no	no	yes	no	80	yes	no	psphakic
9	male	47	OS	psphakic	4	on	B	no	no	yes	no	90	yes	no	psphakic
10	male	41	OD	phakic	3	off	A	no	no	yes	no	19	yes	no	psphakic
11	male	33	OS	phakic	5	off	A	no	yes	yes	no	25	yes	no	psphakic
12	male	52	OD	psphakic	4	off	B	no	no	yes	no	72	yes	no	psphakic
13	male	47	OD	aphakic	3	off	B	no	no	yes	no	82	yes	no	psphakic
14	male	49	OS	phakic	4	off	A	no	no	yes	no	48	yes	no	psphakic
15	male	59	OD	phakic	4.5	off	A	yes	no	no	yes	62	yes	no	psphakic
16	male	46	OS	phakic	3	on	A	no	yes	yes	no	13	yes	no	psphakic
17	male	47	OD	phakic	6	on	A	no	no	yes	no	70	yes	no	psphakic
18	male	42	OD	psphakic	3	on	A	no	no	yes	yes	76	no	no	psphakic
19	male	46	OS	phakic	3	off	A	yes	yes	yes	no	59	yes	no	psphakic
20	female	30	OS	phakic	6	off	b	no	no	no	no	63	yes	no	psphakic
21	male	66	OD	phakic	3	off	A	no	no	no	yes	24	yes	no	psphakic
22	male	68	OD	phakic	3	on	A	no	no	no	no	24	yes	no	psphakic
23	male	42	OD	phakic	4	off	B	no	no	yes	no	57	yes	no	psphakic
24	female	63	OD	phakic	3	off	B	no	no	no	yes	24	yes	no	psphakic
25	male	70	OD	phakic	3	off	B	no	no	yes	no	16	yes	no	psphakic
26	male	67	OS	phakic	3	off	A	no	no	yes	no	36	yes	no	psphakic
27	male	49	OS	phakic	6	on	A	no	no	yes	no	40	yes	no	psphakic
28	female	53	OD	phakic	3	on	A	yes	no	yes	no	29	yes	no	psphakic
29	male	51	OD	phakic	4	on	B	no	no	yes	no	26	yes	no	psphakic
30	male	42	OD	phakic	3	off	A	no	no	no	no	20	yes	no	psphakic

Pt: patient, PVR: proliferative vitreoretinopathy, CVH: corpus vitreous haemorrhage, GRT: giant retinal tear, PPV: pars plana vitrectomy, F-U: follow-up, EF-U: end follow-up, mnths: months, SO: silicone oil, psphakic: pseudophakic

Table 6.2: Follow-up of 30 eyes with giant retinal tear retinal detachment

Follow-up (years)	No.	Eyes %
1	5	16.7
2	7	23.3
3	3	10
4	4	13.3
5 and more	11	36.7
Total	30	100

Postoperative visual acuity (VA) was significantly better than preoperative VA (paired sample T-test: P=0.028). At the end of follow-up, 13 eyes (43.3%) had a VA of 0.1 or less (Figure 6.2). Multivariate analysis did not identify any risk factors for poor visual outcome. Improvement of at least two Snellen lines was seen in 11 eyes (36.7%), while improvement of at least one Snellen line was seen in 16 eyes (53.4%). VA did not change in two eyes (6.7%). Deterioration of two Snellen lines or more was seen in nine eyes (30%). This deterioration was due to an epiretinal membrane of the macula with cystoid macular oedema in three eyes (33.3%). One patient developed postoperative endophthalmitis after PPV and silicone oil injection; visual acuity was 0.1 after silicone oil removal (SOR). Another patient with uncontrollably high intraocular pressure received a Baerveldt implant; one patient had corneal decompensation and three other patients showed deterioration of more than two lines without any obvious clinical cause.

At the end of follow-up, 28 (93.3%) eyes were pseudophakic and two (6.7%) were aphakic. Seven (23.3%) eyes had combined SOR and phaco-emulsification with intraocular lens (IOL) implantation; 14 eyes (46.7%) underwent phaco-emulsifation with IOL implantation after SOR. Another two eyes (6.7%) were aphakic at the end of follow-up: one due to lensectomy during primary PPV and one due to removal of the IOL during a second PPV procedure.

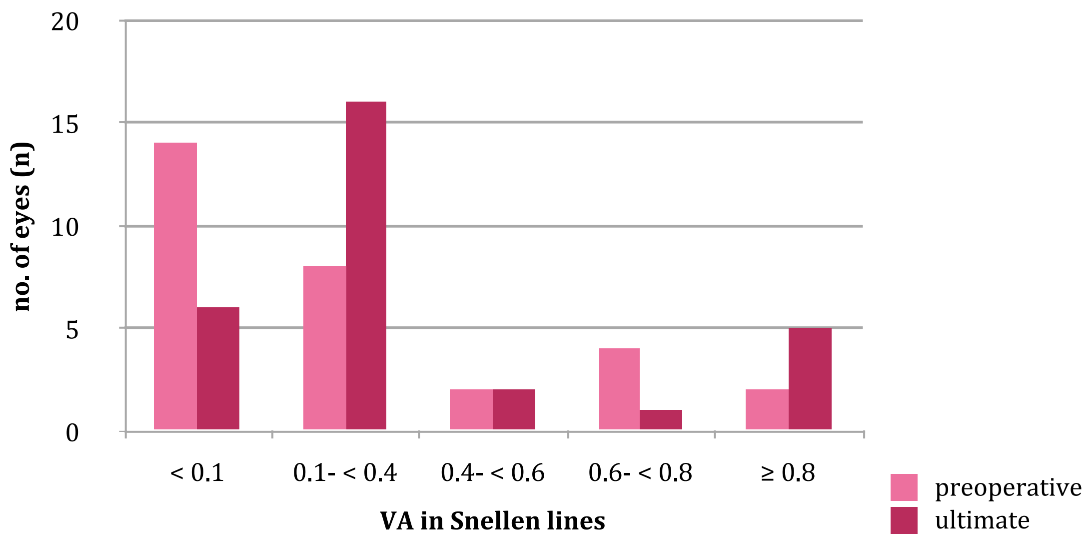

Figure 6.2: Preoperative and ultimate visual acuity (VA) in Snellen lines

Table 6.3: Univariate and multivariate analysis on clinical variables

Factors	Total	Recurrent retinal detachment		Univariate analysis	Multivariate analysis
	n=30	n=9	30.0%	P value	P value
Gender					
Male	26	7	26.9%	NS	NS
Female	4	2	50%		
Age (yrs)					
< 60	20	3	15%		
≥ 60	10	6	60%	P=0.030	NS
Duration of visual field loss					
0-10 days	20	6	30%	NS	NS
> 11 days	9	3	30%		
Preoperative visual acuity (Snellen)					
≤ 0.1	15	6	40%	NS	NS
> 0.1	15	3	20%		
Macular status					
Macular involvement	19	7	36.8%	NS	NS
Macula not involved	11	2	18.2%		
Size of GRT in clock hours					
3 clock hours	18	7	38.9%	NS	NS
> 3 clock hours	12	2	16.7%		
Pseudophakic eye					
yes	6	4	66.7%	P=0.049	NS
no	24	5	20.8%		
PVR					
A	19	6	31.6%	NS	NS
B	11	3	27.3%		
Vitreous haemorrhage					
Yes	6	3	50%	NS	NS
No	24	6	25%		
Trauma					
Yes	4	0	0%	NS	NS
No	26	9	34.6%		
Encircling scleral buckle present					
Yes	21	3	14.3%		
No	9	6	66.7%	P=0.008	P=0.008
Primary lensectomy					
Yes	2	1	50%	NS	NS
No	28	8	28.6%		

NS: not significant, GRT: giant retinal tear, PVR: proliferative vitreoretinopathy, yrs: years

Discussion

Many techniques have been described for the management of a GRT.[1-10, 12, 15-36] In the present study, we found that the absence of an encircling scleral buckle was significantly associated with re-detachment, which supported the results of the study (n=34) by Verstraeten et al.[34] In the latter study, the re-detachment rate in the eyes that received a scleral buckle was 14% versus 45% in the eyes without a scleral buckle.[34] In our series, the re-detachment rate in the encircling scleral buckle group was comparable with Verstraeten et al[34] (Figure 6.1) and also comparable with other studies in which a scleral buckle was used (Table 6.4).[27, 31, 34] However, we did not exclude patients with proliferative vitreoretinopathy (PVR) or traumatic GRT, which contrasts with various other studies and makes comparison difficult.

In agreement with the literature, the present study strongly indicated that the use of an encircling scleral buckle combined with PPV and gas or silicone oil tamponade, reduced the rate of re-detachment. In their GRT patients who did not receive an encircling scleral buckle, Rofail et al[15] found a low percentage of re-detachment after PPV, possibly due to the small number of patients with (only mild) PVR in their series. It is likely that the presence of some degree of PVR in most of our cases skewed the results towards greater success by adding an encircling scleral buckle.

Other authors have suggested that an encircling scleral buckle reduces traction on the remaining vitreous and exerts more pressure on the retina when silicone oil tamponade is used.[37, 38] An argument in favour of using an encircling narrow tyre is that if local re-detachment occurs in the "intact" retina, which is often the case, the 360° buckle may prevent any further detachment. If a wider tyre is used for a portion of the circumference, it can be placed over the GRT, or alternatively, over the "intact" retina, as was done by Aylward et al[36] in more than half of their patients.

In the present series, the re-detachment rate after one vitrectomy procedure was 30%. Ultimately, the retina was attached in 98% of the eyes. Over the past 10 years, primary anatomical success rates in eyes treated for GRT varied between 50% and 94%.[1, 3-5, 15, 24, 32, 34] Ultimate success rates were approximately 95% in most studies.[1, 3, 4, 15, 24, 32] Anatomical success rates depended on the surgical technique, the inclusion or exclusion of eyes with traumatic GRT or PVR, the duration of follow-up and whether the silicone oil was removed from the eye at the end of follow-up. Although most authors reported their results after a maximum of six months follow-up (Table 6.4), mean follow-up in the present series was four years and all of our patients had a follow-up of at least one year (Table 6.2). We found that after one year, another three re-detachments occurred, which was almost one third of the total number of recurrences.

At the end of follow-up, the silicone oil had been removed from all the eyes except one. In other studies, the percentage of eyes that still held the silicone oil at the end of follow-up was much higher (Table 6.4).[5, 24, 32] For example, Scott et al[32] performed a larger study (n=212) and reported that the silicone oil tamponade was present in 41% of their series at the end of follow-up (Table 6.4). They also mentioned many risk factors for re-detachment, including female gender, younger age, PVR, prior PPV, larger size of the giant retinal tear, absence of an encircling scleral buckle and relaxing retinotomy.[32] Our series only confirmed the association with an encircling scleral buckle. However, it is difficult to compare the results, because although Scott et al studied a larger series of eyes, their follow-up period was short and a high percentage of the eyes retained the silicone oil (Table 6.4).

In the present study, 43% of the eyes had postoperative VA of ≤ 0.1. Karel et al[24] followed the patients for a longer period and found that VA was ≤ 0.1 in 70% (Table 6.4). Postoperative VA was better than 0.1 in 57% of our patients, while 60% had equal or better postoperative VA than preoperatively, which was comparable with the results reported previously by other authors.[32]

PPV in combination with primary lensectomy, as described by Kreiger & Lewis[25] and Aylward et al,[36] is advocated to provide better access to the vitreous base and the extreme periphery of the retina.[25, 36] Without lensectomy, it can be fairly awkward to reach the vitreous base,[25, 36] but an encircling scleral buckle or indentation makes this less difficult. Primary lensectomy may be a traumatic event associated with endothelial cell loss. Earlier studies have shown that visual outcome was poor in aphakic eyes after surgery for complicated RD.[39, 40] We performed primary lensectomy in a minority of the eyes.

In conclusion, recurrent retinal detachment occurred in 30% of our cases. Further surgical interventions led to an ultimate attachment rate of 97%. The use of an encircling scleral buckle during vitrectomy with silicone oil tamponade was associated with a low re-detachment rate in patients treated for GRT retinal detachment.

Table 6.4: Summary of the literature on patients treated for giant retinal tear

Author	Year	N=	Detached after one PPV	Final attachment	VA	Tamponade	PVR grade	Trauma	Primary lensectomy	Scleral buckle	F-U mean (mnths)	F-U range (mnths)	SO EF-U
Sirimaharaj et al[3]	2005	62	77.4%	93.5%	54.8% improvement	6.5%BSS 67.7%SF_6 17.4 C_3F_8 8.1% SO	29% B & C	25.9%	21.3%	27.4%	24.5	8-69	NN
Rofail et al [15]	2005	16	93.7%	NN	68.8% improvement	100% PFO	18.8% mild	31.3% Mild	0%	0%	12	3-40	6.3%
Ambresin et al[4]	2003	18	88.8%	94.4%	50% improvement	94.4%SO 5.6% gas	38.9% A 44.4%B 16.7%C	11.1%	27.2%	0%	28.6	4.5-73	5.6%
Scott et al[32]	2002	212	70%	79%	59% improvement	1%air 7% SF_6 57% C_3F_8 34%SO	NN	0%	34%	62%	3.8	NN	41%
Batman et al[5]	1999	47	NN	89.4%	48.9% ≥5/200	46.8% C_3F_8 53.2% SO	44.7%C 55.3 D	23.4%	NN	0%	NN	48-60	36%
Kertes et al[1]	1997	162	49.4%	90.7%	48.8%	9.9% vitr 48.8% gas 35.2% SO 6.2% air	40.7% any PVR (32.1% C or D)	25.3%	37.8%	51.9% or 62.5%	13.7	6-48	NN
Karel et al[24]	1996	50	70%	78%	74% >0.02	96% SO 4% gas	14% C3-D3	38%	56%	98%	33	12-96	56%
Verstraeten et al[34]	1995	34	67%	88.3%	74% >20/80	100% gas	0% PVR	0%	NN	41.2% SB	NN	6-60	NN
Bottoni et al[7]	1994	11	81.8%	100%	64% > 20/40	100%PFD	100% PVR B	9%	NN	100%	17.3	12-21	9%
Ie et al[20]	1994	25	88%	100%	72% >20/80	100% gas	0% PVR	NN	69%	92%	12	6-24	0%
Millsap et al [30]	1993	50	74%	88%	52% > 20/400 48% improvement	16% Vitr 52% SO 43% gas	14% B 4% C 22% D	26%	NN	40%	8.6	6	NN
Leaver et al[10]	1993	39	NN	77%	51%> 6/60	100%SO	NN	NN	NN	NN	120	120	28%
Aylward et al[36]	1993	38	84.2%	89%	Mean 6/36 65.7%> 6/60	84% SO 13% gas	NN	100%	61%	58% ee 61% se	12	12	29%
Kreiger et al[25]	1992	11	91%	100%	73% improvement	45% SO 55% gas	0% PVR	27%	83%	0%	13	7-29	9%
Mathis et al[28]	1992	24	96%	96%	37.5% > 20/200	100% SO	16.6% C-D	25%	71%	75%	11	6-23	4%
Glaser et al[17]	1991	10	90%	90%	80% improvement	100% gas	100% D	25%	100%	100%	8.7	6-12	0%
Chang et al[9]	1989	17	71%	94%	53% ≥20/60	94% gas 6% SO	18% C-D	18%	NN	NN	12	6-20	6%

PPV: pars plana vitrectomy, PVR: proliferative vitreoretinopathy, F-UP: follow-up, SO: silicone oil, EF-UP: end follow-up, BSS:balanced salt solution; SF_6 sulphurhexafluoride; C_3F_8: perfluoropropane, NN:not noted, PFO: perfluoro-n-octane, vitr: vitreon, SB: scleral buckling procedure, PFD: perfluorodecaline, ee: encircling element, se: segmental explant

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Chapter 7

Risk factors for re-detachment and deterioration in visual outcome after silicone oil removal from eyes treated for complicated retinal detachment

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Abstract

Background: The goal of this study was to identify risk factors for re-detachment and/or deterioration in visual outcome after silicone oil removal from eyes treated for complicated retinal detachment.
Subjects and Methods: An unselected series of 287 consecutive eyes that underwent silicone oil removal (SOR) between January 1999 and December 2003 were analysed in retrospect.
Results: Anatomical success after SOR was achieved in 81% of the eyes. Overall anatomical success at the end of follow-up was 94%. Postoperatively, 8% of the eyes had ocular hypertension, 6% had hypotony and 29% had keratopathy. After SOR, visual acuity improved by at least two Snellen lines in 43% of the eyes. Multivariate analysis showed that male gender, preoperative rubeosis and proliferative diabetic retinopathy (PDR) were risk factors for re-detachment. Furthermore, male gender, preoperative visual acuity of <0.1 Snellen lines, PDR, three additional operations, retinectomy (irrespective of size) and hypotony were found to be associated with poor visual outcome (<0.1 Snellen lines).
Conclusions: Retinal detachment after SOR occurred in approximately 20% of the eyes in this unselected series, which was comparable with the rates in the Silicone Study Reports published approximately 20 years ago. However, in the previous studies, preoperative patient selection was made and SOR was performed on less than 50% of the eyes with silicone oil tamponade. Our higher overall anatomical success may have been due to improved vitreoretinal surgical techniques.

Introduction

Silicone oil is a commonly used tamponade agent in the surgical management of complicated retinal detachment (RD). The tamponade is often temporary, owing to the risk of complications. These include cataract, glaucoma, keratopathy and oil emulsification.[1-9] In the literature, visual outcome, recurrent RD and the incidence of these complications varies considerably.[1-3, 6-8, 10-13] One of the complications of silicone oil removal (SOR) is hypotony.[4] Furthermore, various preoperative and intraoperative factors have been reported as risk factors for recurrent RD, poor visual outcome and the development of complications.[6, 10-12, 14] To identify which patients have the best anatomical and visual results after SOR, it is important to analyse preoperative, intraoperative and postoperative clinical variables. Therefore, the goal of this study was to analyse the data on a consecutive series of eyes that underwent SOR after surgery for complicated RD and to identify which clinical variables were associated with re-detachment and/or deterioration in visual outcome. We also investigated which variables were associated with an increased risk of glaucoma, keratopathy and hypotony.

Subjects and methods

The medical files of all the patients who underwent SOR (n=287) at the University Medical Centre Maastricht between January 1999 and December 2003 were reviewed in retrospect. A total of 287 eyes were identified in 280 patients: 210 eyes had proliferative vitreoretinopathy (PVR), 27 eyes had suffered trauma, 26 eyes had a giant retinal tear and 13 eyes had traction retinal detachment due to proliferative diabetic retinopathy (PDR). We excluded eyes with retinopathy due to prematurity and eyes with a follow-up of less than 3 months.

The following data were gathered from the medical files: age, gender, duration of silicone oil tamponade, follow-up duration, diagnosis and original indication for silicone oil injection, ocular history including all previous surgery, intraocular pressure, trauma (yes/no) and a history of systemic disease (yes/no). We also noted the preoperative and postoperative best corrected Snellen visual acuity and preoperative and postoperative Goldmann applanation tonometry. An inventory was made of all possible causes for poor postoperative visual acuity: macular degeneration, macular hole, cystoid macular oedema, abnormal intraocular pressure or optic atrophy. Slit-lamp examination was used to record the following: keratopathy (yes/no), silicone oil in the anterior chamber (yes/no) and the amount of remaining oil, strong emulsification of the silicone oil (yes/no), neovascularization of the iris (yes/no) and the status of the lens. Funduscopy was performed using indirect binocular ophthalmoscopy with a Goldmann three-mirror contact lens and a panfundus contact lens (Supersquad 160°). The following postoperative data were collected: the presence of an encircling band, the cumulative size of all retinectomies performed on that eye and whether or not there was stable, but persistent peripheral RD at the time of oil removal. PVR was graded according to the Retina Society Classification of 1983.[15]

At our clinic, eyes with up to PVR grade C1 were operated on using a conventional scleral buckling technique.[6, 16] In the case of eyes with poor funduscopic view (e.g. with miosis and/or extensive vitreous haemorrhage), central and complex tears at various distances from the vitreous base, PVR grade C2 and higher and trauma with PVR, we applied primary vitrectomy. All the operations were performed by one of three surgeons (ELH, FH or AL). Our vitrectomy technique with silicone oil tamponade comprised the following: standard three-port vitrectomy with a trocar microcannular system (Grieshaber & Co, AG, Schaffhausen, Switzerland) with a non-contact wide-angle panoramic viewing system (BIOM, Carl Zeiss, Meditec, Jena, Germany). The scleral buckle was always left in situ, whereas the silicone oil was removed as completely as possible, including shaving of the vitreous base. The retina was mobilized by excising all the epiretinal and subretinal membranes and strings, or by performing relaxing retinectomy as a last resort if the retina remained rigid. Lensectomy was only carried out in eyes with anterior PVR, as described in Silicone Study Report 10,[17] to completely clean the peripheral retina and vitreous base. All the eyes in our series received perfluorocarbon liquid (DK-line, Chauvin Opsia, France) which was later exchanged for silicone oil (1000 centistokes). Re-operations were performed until stable attachment of the retina was achieved, centrally to the encircling element. Partial peripheral retinal detachment, i.e. anterior to the encircling scleral buckle, was treated locally with argon laser confinement, as described previously,[18] until the situation was stable enough to enable silicone oil removal (SOR).

SOR was indicated when a stable situation had been achieved with the retina attached posteriorly to the encircling scleral buckle, or if complications arose, such as ocular hypertension, keratopathy, or oil-corneal touch, as described previously.[6] Our SOR technique took place through two corneoscleral incisions in the case of aphakia, or through two pars plana sclerotomies. When additional procedures were necessary, such as endolaser photocoagulation, lensectomy by phacofragmentation or phaco-emulsification, removal of epiretinal membranes or removal of ischaemic edges of former retinectomies, three-port vitrectomy was performed.

The definition used for recurrent RD after SOR in the present study was either complete retinal re-detachment, or local detachment. Anatomical success was defined as attachment of the retina between the photocoagulation barrier at the end of follow-up. During follow-up, all the patients were seen at regular intervals of three to four weeks. A clinically significant change in visual acuity was defined as at least two lines of Snellen acuity (e.g. a change from light perception to hand movements, or from finger counting to 0.1, or vice versa) since the previous follow-up visit.

We used the same definitions for outcome and complications as those described previously.[6] However, in the case of glaucoma, we employed the following definition from Silicone Study Report 6:[19] an intraocular pressure of more than 25 mm Hg, or more than 20 mm Hg with antiglaucoma medication,[19] measured at any time during follow-up.[19] This was because, in this retrospective study, incomplete data were available on visual field defects and the presence of optic disc cupping. Owing to the fact that this definition was more applicable to "ocular hypertension" (i.e. it does not take into account whether there are any related morphofunctional optic nerve changes), we replaced the word "glaucoma" with "ocular hypertension", but adhered to the original definition in the previously mentioned Silicone Study Report 6.[19] Hypotony was defined as an intraocular pressure of less than 5 mm Hg.[19] Keratopathy was defined as bullous or band-shaped keratopathy, epithelial or stromal oedema, or localized opacities, in conformity with Silicone Study Report 6.[19]

Statistical analyses were performed with SPSS version 12.0. Snellen visual acuities were converted to a logarithmic scale (LogMAR, i.e. the logarithm of the minimal angle of resolution), as described previously.[20] Comparisons between preoperative and postoperative visual acuities were made using the Wilcoxon signed rank test. Univariate analysis was performed with the Pearson Chi-square exact test to determine which preoperative clinical variables were associated with postoperative re-detachment, complications (such as ocular hypertension, or hypotony) and the visual outcome. Stepwise forward conditional multiple logistic regression analysis was used on the following preoperative variables to determine the strongest predictors of postoperative re-detachment, complications and visual outcome: the probability of PDR, three or more operations on the same eye, PVR, trauma, previous retinectomy, giant retinal tear (GRT) and the presence of rubeosis.

RESULTS

Whole study group

The study included 287 eyes in 280 patients (199 men (69%) and 88 women (31%); mean age 56.6 years (SD ± 14.8)). Mean follow-up in the 287 eyes was 21.2 months (SD ± 16.6). Mean duration of silicone tamponade was 10.0 months (SD ± 6.9) and the mean number of surgical procedures before SOR was 1.2 (SD ± 0.5).

The causes of complicated retinal detachment were PVR in 210/287 eyes (73%), trauma in 27/287 eyes (9%), giant tear in 26/287 eyes (9%) and traction retinal detachment due to PDR in 13/287 eyes (5%). Eleven eyes (4%) had complicated RD associated with a previous intraocular surgical procedure, such as vitrectomy for retained lens fragments or macular hole surgery.

Anatomical success

Anatomical success was achieved in 233/287 eyes (81%). After silicone oil removal, re-detachment occurred in 54/287 eyes (19%). Data on the timing and cause of re-detachment were available in 38/54 eyes. Re-detachment occurred 0 to 108 weeks after SOR (Figure 7.1), with a mean of 14.3 weeks (SD ± 20.6). Within the first 3 months after SOR, re-detachment occurred in 26/38 eyes (68%); in 21 cases, re-detachment was due to the re-opening of old retinal tears, or the development of a new tear, because of membrane proliferation attributable to PVR. Within the first 6 months, re-detachment occurred in 33/38 eyes (87%). After one year, re-detachment occurred in another 3/38 eyes (8%). At the end of follow-up, five other eyes had re-detachment due to the re-opening of old retinal tears, or the development of new tears. After re-detachment, 21/38 eyes (55%) underwent vitrectomy, once again with silicone oil tamponade. The silicone oil was subsequently removed from 8/21 eyes (38%), whereas 13/21 eyes (62%) still had oil tamponade in situ at the end of follow-up. In 17/38 eyes (45%), the patient and/or the surgeon decided not to proceed with any further operations, because of the poor prognosis for the eye, or the poor physical condition of the patient. Three eyes needed enuclation because of intractable pain and function loss.

Ultimate anatomical success was achieved in 265/282 eyes (94%) at the end of follow-up. Table 7.1 shows the results of our univariate analysis on the association between re-detachment and all the preoperative and postoperative variables at the end of follow up. Multivariate analysis revealed that only the variables male gender, the presence of rubeosis and PDR were significantly associated with recurrent retinal detachment (P=0.034, P=0.007 and P=0.001, respectively).

Visual acuity

Preoperative and postoperative visual acuity measurements in the whole group are listed in Table 7.2. At 3 months and 12 months postoperatively, visual acuity was significantly better than preoperatively (P=0.022 and P=0.005, respectively) (Figure 7.2, Table 7.2).

An improvement of at least two Snellen lines was seen in 128/277 cases (45%) (Figure 7.3). Deterioration of two Snellen lines or more was seen in 19/277 cases (7%). This deterioration was due to retinal detachment in 15/18 eyes (83%), corneal decompensation in 6/19 eyes (32%), keratopathy in 9/19 eyes (47%), maculopathy in 4/19 eyes (21%) and a pale optic disc or optic atrophy in 5/19 eyes (26%). Other less frequent causes of visual loss were cataract and floaters. Although we performed cataract extraction in 48 eyes after SOR, four out of the 48 eyes still showed deterioration of two Snellen lines or more. In one of the five above-mentioned patients with a pale optic disc, this may have been caused by glaucoma, because high ocular pressures were present preoperatively and postoperatively. However, in the remaining four cases, we did not encounter any documented intraocular pressure increase during follow-up.

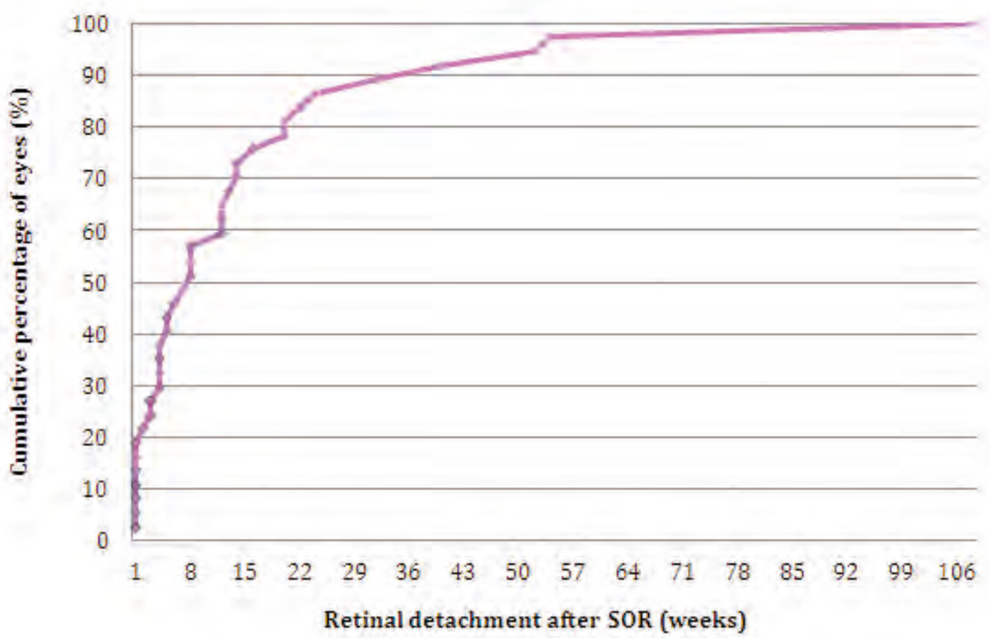

Figure 7.1: Cumulative percentage of patients (n=38) with retinal re-detachment after silicone oil removal (SOR).

Postoperative visual acuity data were available in 158 eyes at 12 months follow-up. Almost 60% of them had a visual acuity of ≥ 0.1 Snellen lines (Table 7.2). Multivariate analysis showed that the following variables were statistically significantly associated with poorer visual outcome (<0.1 Snellen lines) at 12 months follow-up: male gender (P=0.028), preoperative visual acuity of <0.1 Snellen lines (P<0.001), PDR (P=0.001), three or more operations (P=0.018), retinectomy (irrespective of size) (P=0.006) and hypotony (P=0.023) (Table 7.3).

Complications

Postoperative keratopathy developed in 82/287 eyes (29%). In 33 of these eyes (40%) the ultimate outcome was corneal decompensation. A history of three or more operations on the same eye (P<0.001) and aphakia (P=0.001) were significantly associated with the development of keratopathy.

Preoperative ocular hypertension was found in 46/271 eyes (17%). Postoperative ocular hypertension was present in 23/271 eyes (8.5%) (P<0.001); 12 of these 23 eyes had new onset ocular hypertension after SOR. Mean preoperative ocular pressure was 17.5 mmHg (SD ± 8.4); mean postoperative ocular pressure was 13.4 mmHg (SD ± 6.9). This difference was statistically significant (P<0.001). In 31/271 eyes (11%) we found a pale optic disc postoperatively, which was significantly associated with the presence of preoperative ocular hypertension (P<0.001) and the use of antiglaucoma medication

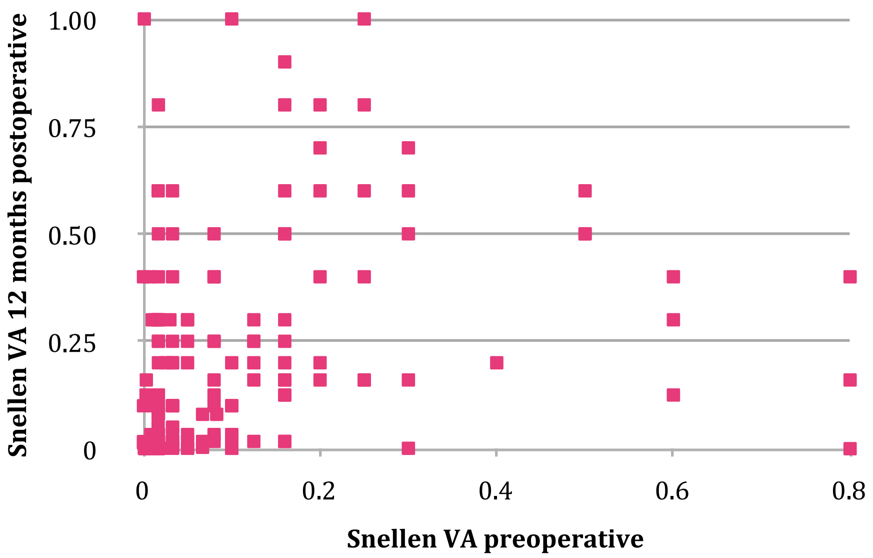

Figure 7.2: Snellen visual acuity preoperative versus visual acuity at 12 months F-U

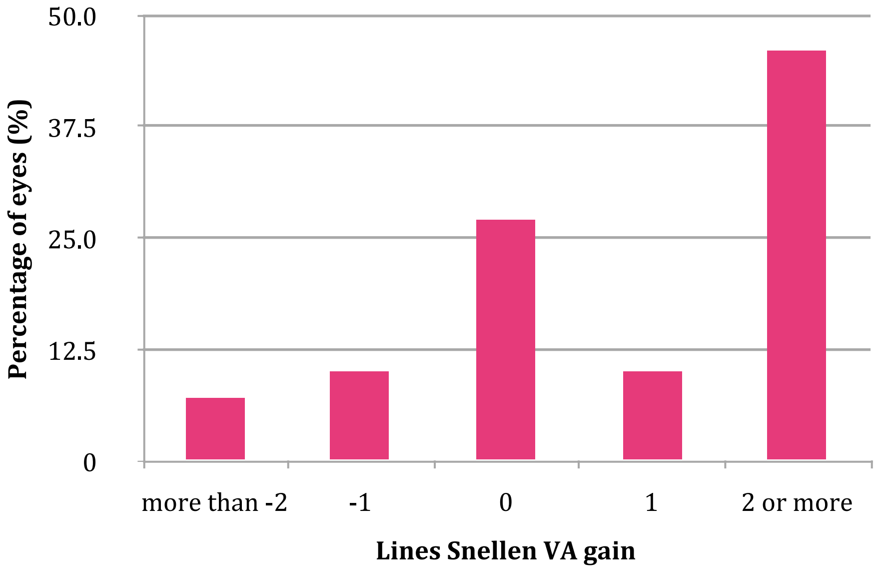

Figure 7.3: Visual acuity improvement noted in number of Snellen lines. Improvement of at least 2 lines (n=128), improvement of less than 2 lines (n= 30), equal visual acuity (n=72), deterioration less than 2 lines (n=28), deterioration of at least 2 lines (n=19).

Table 7.1: Univariate and multivariate analysis on clinical variables associated with re-detachment

Variables	Total	Retina re-detached		Univariate analysis P value	Multivariate analysis P value
	n=287	n=54	8.8%		
Age					
≤ 70 years	234	43	18.4%	NS	NS
> 70 years	53	11	20.8%		
Gender					
Male	199	31	15.6%	P=0.048	P=0.034
Female	88	23	26.1%		
Preoperative visual acuity (Snellen)					
< 0.1	187	40	21.5%	P=0.054	NS
≥ 0.1	100	12	12.1%		
Preoperative rubeosis					
Yes	21	8	38.1%	P=0.026	P=0.007
No	266	46	17.3%		
PDR pre SOR					
Yes	13	7	53.8%	P=0.004	P=0.001
No	274	47	17.2%		
Operations					
< 3	180	29	16.1%	NS	NS
≥ 3	107	25	23.4%		
No. of SOR					
< 3	278	53	19.1%	NS	NS
≥ 3	9	1	11.1%		
Tamponade					
< 6 months	79	15	19.0%	NS	NS
≥ 6 months	208	39	18.8%		
SOR procedure					
Pars plana	182	37	20.3%	NS	NS
Anterior chamber	105	17	16.2%		
Encirling band present					
Yes	214	36	16.8%	NS	NS
No	69	17	24.6%		
Retinectomy					
Yes	118	26	22.0%	NS	NS
No	169	28	16.6%		

SOR: silicone oil removal, PDR: proliferative diabetic retinopathy, NS: not significant

(P<0.001). There were no significant differences in the rates of all three above-mentioned complications and the presence of PVR, PDR, trauma, or a GRT. In the patients who did not undergo retinectomy in the present study, we found a significant association with postoperative ocular hypertension (P=0.049).

Hypotony was found in 17/287 eyes (6%). Univariate analysis showed that three or more operations on the same eye (P=0.020), RD at the end of follow-up (P=0.011) and retinectomy (irrespective of size) (P=0.003) were significantly associated with hypotony. Only two out of the 14 patients with preoperative hypotony also had postoperative hypotony. According to our univariate and multivariate analysis, this was not statistically

Table 7.2: Preoperative and postoperative visual acuity (Snellen) in the whole group (including eyes with retinal detachment)

	Total eyes measured n=287	Snellen VA <0.1 n (%)	Snellen VA ≥0.1 n (%)	Mean Sn VA (SD)	P value univariate, by Wilcoxon sign rank test
Preoperative	n=285	186 (65%)	99 (35%)	0.040 ± 0.73	
Postoperative:					
3 mnths F-U	n=262	133 (51%)	129 (49%)	0.05 ± 0.85	P=0.022
6 mnths F-U	n=199	97 (49%)	102 (51%)	0.05 ± 0.94	NS
12 mnths F-U	n=158	65 (41%)	93 (59%)	0.06 ± 0.95	P=0.005

VA:visual acuity, Sn:Snellen, F-U:follow-up, mnths:months, NS:not significant

significant. Multivariate analysis showed that RD at the end of follow-up (P=0.013) and retinectomy (irrespective of size) (P=0.012) were associated with hypotony.
SOR was performed on 18 eyes (6%) because of elevated intraocular pressure and/or keratopathy. After SOR, six eyes (33%) had corneal decompensation, while two eyes (11%) had persistent ocular hypertension. In one of these two eyes, Baerveldt implantation was necessary to successfully lower the intraocular pressure. One eye developed phthisis bulbi and was enucleated.

Discussion

In this study, retinal re-detachment occurred in 19% of the eyes after silicone oil removal (SOR). We did not make any prior selection in relation to SOR, which was contrary to Silicone Study Report 6,[19] in which only 45% of the eyes had SOR. They selected the better eyes, with better visual acuity, fewer previous surgical interventions and fewer complications.[19] Nowadays, we have more sophisticated equipment with improved surgical techniques,[21] such as wide-angle viewing systems[22] and perfluorocarbon liquids (DK-line).[18, 23-25] These developments have contributed to the overall improvement in vitreoretinal surgery outcome compared to 20 years ago when the Silicone Study Reports[19] were published. This was confirmed by the results of the present study.
Overall anatomical success in our study was higher (94%) than that reported in Silicone Study Report 6: 81%.[19] Although we did not make any preoperative selection and our postoperative follow-up was longer (mean follow-up 21.2 months (SD ± 16.6) versus 6 months total) our results in terms of re-detachment (19% versus 20%) and deterioration in visual acuity (16.4% versus 15.5%) were comparable with those in Silicone Study Report 6.[19]

Table 7.3: Univariate and multivariate analysis on clinical variables associated with poorer visual acuity after SOR at 12 months follow-up

Variables	Total	VA ≥ 0.1		Univariate analysis P value	Multivariate analysis P value
	n=158	n=93	58.9%		
Preoperative					
Age					
≤ 70 years	137	81	59.1%	NS	NS
> 70 years	21	12	57.1%		
Gender					
Male	117	76	65.0%	P=0.010	P=0.028
Female	41	17	41.5%		
Preoperative visual acuity (snellen)					
< 0.1	101	46	45.6%	P<0.001	P<0.001
≥ 0.1	57	47	82.5%		
Preoperative rubeosis					
Yes	144	98	68.1%	P=0.022	NS
No	14	4	28.6%		
PDR pre SOR					
Yes	9	1	11.1%	P=0.004	P=0.001
No	149	92	61.7%		
Operations					
< 3	95	68	71.6%	P<0.001	P=0.018
≥ 3	63	25	39.7%		
No. of SOR					
< 3	156	91	58.3%	P=0.028	NS
≥ 3	2	2	100%		
SOR procedure					
Pars plana	96	59	61.5%	NS	NS
Anterior segment	62	34	54.8%		
Retinectomy					
Yes	68	27	39.7%	P<0.001	P=0.006
No	90	66	73.3%		
Keratopathy					
Yes	50	20	40.0%	P=0.002	NS
No	108	73	67.6%		
Hypotony					
Yes	10	1	10.0%	P=0.002	P=0.023
No	148	92	62.2%		
Re-detachment					
Yes	32	12	37.5%	P=0.008	NS
No	126	81	64.3%		

SOR:silicone oil removal, PDR:proliferative diabetic retinopathy, NS:not significant

In the present study, the mean duration until re-detachment was 14.3. weeks (SD ± 20.6). Within the first 3 months after SOR, 26 eyes (68%) had re-detachment, which was mostly due to PVR. Within the first six months, 87% had re-detachment. In 8% of the eyes with recurrent RD, re-detachment occurred more than one year after SOR. Our results were comparable with previous studies, which reported re-detachment rates of between 9% and 25% after SOR.[6-8, 10-14, 26-28] It is likely that the variation between studies can mainly be attributed to differences in the duration of follow-up and preoperative patient selection. Most authors reported their results after a maximum follow-up of 6 months, whereas in the present study, eyes were included with longer follow-up.

Independent predictors of recurrent RD were male gender, PDR and the presence of rubeosis. Other studies, such as Jiang et al,[14] did not mention preoperative rubeosis as a risk factor for re-detachment, which may have been due to their smaller number of eyes (n=94).[14] Jonas et al[10] studied a larger series (n=221) and found the following risk factors for re-detachment: several previously unsuccessful operations for RD, the surgeon, visual acuity before SOR, incomplete removal of the vitreous base, the absence of an encircling scleral band in eyes with PVR and the duration of silicone oil tamponade.[10] Other studies reported that a GRT (n=92, n=58)[6, 14] age above 70 years (n=58)[6] and retinectomy of 180° or more (n=58)[6] were significant for risk factors for recurrent RD. Our analyses (n=287) did not confirm these findings.

In the present study, visual acuity improved significantly after SOR: 45% of the eyes improved by two or more Snellen lines, while 36% had equal visual acuity or improved by one or more Snellen lines, which confirms previous studies.[6, 8, 10, 11, 27, 29] Delayed natural recovery of the retina and additional procedures performed at the time of SOR, such as cataract extraction, may have contributed to the improvements in postoperative visual outcome. It also has to be kept in mind that preoperative and postoperative full refraction measurements were not performed or documented in all cases. Thus, the apparent improvements in visual acuity may be partly due to different standards of acuity measurement.

An independent predicator of poor postoperative visual acuity (defined as <0.1 at 12 months in our study) was poor preoperative visual acuity, which was also found by Jiang et al.[14] Another significant risk factor was PDR, as has been described by others.[30] In the present study, we found that a history of three or more operations on the same eye before SOR, retinectomy (irrespective of size) and hypotony were also significantly associated with poor visual outcome. Eckardt et al,[28] in contrast, did not find any association between retinectomy size and visual outcome,[28] whereas Federman & Eagle[8] reported a significant association between extensive retinectomy of 360° and poorer visual outcome. It might be argued that the poor visual outcome in these eyes was related to advanced underlying pathology, which often necessitated large-scale retinectomy.

In our study, 19 patients (7%) experienced unexpected deterioration in visual acuity of two or more Snellen lines after SOR. Newsom et al[31] described seven patients with unexplained visual loss after SOR. Visual loss in these patients was not associated with re-detachment, macular oedema or epiretinal membrane formation, while optical coherence tomography (OCT) showed normal foveal anatomy.[31] Such unexplained sudden visual loss has also been reported by others.[32, 33] One possible explanation is retinotoxity of the silicone oil.[34] Toxicity may also be the explanation for the optic atrophy found in four of our eyes with a pale optic disc without any documented intraocular pressure rise during follow-up. Damage to the outer segment photoreceptors forms another feasible explanation,[35, 36] because this has recently been observed with ultra-high resolution

(UHR) OCT after successful anatomical repair. Similar microstructural alterations have also been discovered in eyes without macular detachment.[35, 36]

Different criteria and definitions have been used in the literature, which makes it difficult to compare studies. Therefore, we used the same definitions for complications as those formulated in Silicone Study Report 6.[19] Keratopathy was found in 29% of our cases. In other studies, this percentage varied between 4.5% and 100%,[1, 2, 11, 27, 37] which may have been due to contrasting definitions. In the present study, a history of three or more operations on the same eye and aphakia were identified as significant risk factors for the development of keratopathy, which confirms previous findings.[3, 13] We found significantly more keratopathy in association with aphakia and/or repeated surgery, including anterior segment procedures, in conformity with other studies.[38-41] These two variables are known to be related to (cumulative) damage to corneal endothelial cells,[41] which may lead to the keratopathy.

Hypotony is another frequent major complication. In our series, we found hypotony in 6% of the cases, which was in agreement with other research.[5, 11, 13, 26] Theories suggest that anterior PVR, traction and detachment of the ciliary body may result in diminished aqueous humor production.[4, 42-44] In a previous smaller study, we found that retinectomy of 180° or more was a significant risk factor for hypotony.[6] However, in the present larger series, we found a significant association between hypotony and retinectomy, irrespective of the size. In contrast, Eckhard et al[28] found no relation between intraocular pressure and retinectomy size.

The incidence of glaucoma varied depending on the definition used in each study. Owing to our retrospective design, data on visual field defects and the presence of optic disc cupping were incomplete. Therefore, we used the same definition for glaucoma as that employed in Silicone Study Report 6.[19] However, this definition does not distinguish between ocular hypertension and true glaucoma, because it disregards morphofunctional optic nerve changes. On the basis of this definition, we found prepolymegathismive ocular hypertension in 17% of the eyes and postoperative ocular hypertension in 8.5%. In most other studies, comparable or higher percentages were reported.[6, 8, 11, 26] The decrease in mean intraocular pressure after SOR was significant in the present study, in agreement with previous findings.[6, 8, 11, 14] It can be concluded that SOR may contribute to better intraocular pressure control. The mechanisms that underlie the development of chronic glaucoma after silicone oil tamponade are still poorly understood. It has been suggested that oil droplets in the anterior chamber, especially when the oil is emulsified, with infiltration and obstruction of the trabecular meshwork, is probably the most important causative factor in ocular hypertension.[45, 46] Van Meurs et al[7] found a significantly higher incidence of glaucoma in patients with oil droplets in the anterior chamber angle. The duration of oil tamponade may play a crucial role in the process of emulsification.[7] Although we could not find a significant association between ocular hypertension and the duration of oil tamponade or emulsification, there was a significant association between postoperative ocular hypertension and eyes that did not undergo retinectomy. This seems to support the hypothesis that retinectomy is a valuable treatment for therapyresistant glaucoma,[47] because it may help to lower the incidence of postoperative ocular hypertension in silicone-filled eyes.

In conclusion, RD occurred in approximately 20% of the present series of eyes after silicone oil removal, which was comparable with the rates in the Silicone Study Reports published approximately 20 years ago. An important difference in study design was that the present results were obtained in an unselected series of eyes, whereas the Silicone Study Reports employed patient selection so that less than 50% of the eyes had SOR. In comparison, we obtained a higher overall anatomical success rate, mainly due to improved vitreoretinal surgical techniques. The following independent risk factors were found for recurrent RD after SOR: male gender, rubeosis and PDR. Risk factors for poor visual outcome after SOR were: male gender, preoperative Snellen visual acuity < 0.1, PDR, three or more operations on the same eye, retinectomy (irrespective of size) and hypotony.

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Chapter 8

Corneal endothelial cell density after vitrectomy with silicone oil tamponade for complex retinal detachment

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Abstract

Background and purpose: To evaluate endothelial cell density (ECD) changes in eyes with silicone oil (SO) tamponade after vitrectomy for complex rhegmatogenous retinal detachment (RRD).
Methods: A prospective controlled study on 81 eyes with complex RRD that underwent vitrectomy and SO tamponade. Fellow eyes that fulfilled specific inclusion criteria served as controls. Preoperative ECD (cells/mm^2), coefficient of variance (CV, SD/mean cell area x 100), percentage of hexagonal cells and corneal thickness were documented and compared to values obtained at 3, 6 and 12 months follow-up. For the purpose of the analysis, the eyes were classified into five groups according to their lens status during the study period: group 1: phakic eyes that remained phakic; group 2: pseudophakic eyes that remained pseudophakic; group 3: phakic eyes that underwent phacoemulsification with IOL implantation; group 4: eyes that became aphakic; group 5: fellow eyes that served as controls.
Results: High endothelial cell loss was found in group 3 and group 4 at 12 months follow-up: mean cell loss was 19% and 39%, respectively ($P < 0.001$).
Conclusions: An intact natural or artificial lens-iris diaphragm seemed to provide a protective barrier against corneal endothelial cell damage from long-term SO tamponade.

Introduction

In more complex retinal detachment cases, pars plana vitrectomy (PPV) with silicone oil (SO) tamponade is recommended.[1-4] However, many complications have been reported with the use of SO tamponade,[5-11] such as glaucoma,[12-14] hypotony,[12] cataract formation[15] and corneal endothelial cell (EC) loss[6, 16] due to SO kerathopathy.[17, 18] SO keratopathy often leads to corneal decompensation, which necessitates keratoplasty. This was shown in a study by Beekman et al,[17] who performed keratoplasty on a series of 12 patients with silicone oil keratopathy after PPV with SO tamponade. Previous studies have demonstrated EC loss in eyes that underwent PPV, but they did not include cases with SO tamponade.[6, 16] However, in a smaller study on 10 eyes with permanent SO tamponade, high EC loss was reported.[7] To the best of our knowledge, no prospective studies have been published on EC counts and/or EC morphometry after PPV with SO tamponade.
The goal of this study was to measure changes in ECD by means of specular microscopy in eyes that underwent PPV with temporary SO tamponade. We also evaluated risk factors for EC loss after PPV with SO tamponade in phakic, pseudophakic and aphakic eyes.

Subjects and methods

This prospective study was performed on all consecutive patients who underwent PPV with SO tamponade for either complex RRD or recurrent RRD between November 2006 and November 2008. We excluded patients with trauma and/or uveitis. Fellow eyes served as controls if they did not undergo ocular surgery during the study period. The study was approved by the Maastricht University institutional ethics committee. All the patients gave informed consent and the tenets of the declaration of Helsinki were followed.

Surgical history

A comprehensive set of preoperative patient characteristics were noted: age, gender, prior intraocular surgery, prior scleral buckling surgery and the presence of proliferative vitreoretinopathy (PVR) graded according to the Retina Society Classification of 1983.[19] We also noted the cumulative duration of surgery and the presence of keratopathy, corneal oedema or SO emulsification.
All the patients were operated on by one of four experienced vitreoretinal surgeons (FH, ELH, AL, IL) at the Eye Clinic of the University Medical Centre Maastricht (tertiary referral centre). Indications for PPV in complex RRD cases were poor funduscopic view, miosis, extensive vitreous haemorrhage, central and complex tears near the vitreous base, PVR of at least grade C2, trauma with PVR and retinal re-detachment. Our PPV technique with SO tamponade comprised: standard three-port PPV with a trocar microcannular system (Grieshaber & Co, AG, Schaffhausen, Switzerland) using a non-contact wide angle

panoramic viewing system (EIBOS, Möller-Wedel, Wedel, Germany). In patients with existing scleral buckling surgery, the encircling band and buckle were always left in situ and as much vitreous as possible was removed, with shaving of the vitreous base. The retina was mobilized by excising all the epiretinal and subretinal membranes and strings, or by performing relaxing retinectomy as a last resort if the retina remained rigid. Lensectomy was carried out in eyes with anterior PVR, as described in Silicone Study Report 10,[20] to completely clean the peripheral retina and vitreous base.

All the eyes received perfluorocarbon liquid (DK-line, Chauvin Opsia, France) which was subsequently exchanged for SO (Dimeticon 1000 centistokes, Dimeticon 1000 cs is manufactured by Dow Corning Ltd, Coventry (United Kingdom) under number 0006488526, imported by Spruyt Hillen, Ijsselstijn (the Netherlands) and distributed to the University Medical Centre Nijmegen (UMCN), where it is sterilised at the Pharmacology Department. No further editing is performed at Spruyt Hillen or at the UMCN. The sterilised SO is transported to our hospital for use at the operating theatre).

During PPV, we sometimes placed a silicone encircling band (240, MIRA Inc. Uxbridge, MA, USA) beneath the rectus muscles. The encircling band was attached 10-12 mm posteriorly to the limbus with a mersilene 5.0 suture. The decision of whether or not to use an encircling band was based on the size and location of the retinal tear and on the lens status.

Re-operations were performed until stable attachment of the retina was achieved anteriorly to the encircling element. When partial peripheral retinal detachment occurred, i.e. anterior to the encircling scleral buckle, it was treated locally with argon laser confinement, as described previously,[3, 4] until a stable situation was obtained that enabled SO removal (SOR).

SOR was indicated when the retina was attached posteriorly to the encircling band, or if complications arose, such as ocular hypertension, keratopathy, or oil-corneal touch.[3] Our SOR technique took place through two corneoscleral incisions in the aphakic patients, or through two pars plana sclerotomies in the phakic and pseudophakic patients. When additional procedures were necessary, such as endolaser photocoagulation, lensectomy by phacofragmentation, removal of epiretinal membranes, removal of ischaemic edges of former retinectomies, or air-fluid exchange to remove surplus SO emulsification, three-port vitrectomy was performed.

All the patients received routine postoperative treatment with antibiotic eye drops 4 times daily for 14 days (chloramphenicol 5 mg/ml, Ratiopharm, Zaandam, the Netherlands). Patients with phakic eyes also used mydriatic eye drops (atropine sulphate 1%) 2 times daily for 1 week after the operation (Chauvin Pharmaceuticals Ltd Surrey, UK). Patients with pseudophakic eyes used additional tropicamide 0.5% (Thea Pharma, Wetteren, Belgium) 2 times daily for 1 week and prednisolone eye drops (Pred Forte®, prednisolone acetate 10 mg/ml, Allergan BV, Nieuwegein, the Netherlands) 3 times daily for 2 months.

Corneal endothelial cell density analysis

Two independent investigators (FG and DC) performed all the ocular examinations. These included best-corrected visual acuity (BCVA), slit-lamp examination, intraocular pressure (IOP), dilated funduscopy, pachymetry (corneal thickness) and non-contact specular microscopy of the corneal endothelium (Noncon Robo, SP 8000, Konan, Hyogo, Japan).

ECD calculation

At each examination, we evaluated endothelial cell density (ECD, cells/mm^2), coefficient of variance (CV, SD/mean cell area x 100), percentage of hexagonal cells and corneal thickness. ECD was determined from the average cell area with the following equation: cell density = 10^6 / average cell area with cell density (cell/mm^2), average cell area (μm^2); the value 10^6 was used to convert units of measure.
The cells in human corneal endothelium have various surface areas. Polymegathism value is the name given to a coefficient that describes the variation in cell area. With increasing standard deviation of the average cell area, the accuracy of the estimated true cell density decreases. Therefore, increases in the polymegathism value decrease the accuracy of the average cell area. The following equation was used to calculate the polymegathism value: CV = SD cell area / mean cell area, μm^2. CV is the coefficient of variation, while SD is the standard deviation of the mean cell area.[21, 22] We determined the figure coefficient, which is also a dimensionless index, using the equation: 4 A/P^2, where A is the cell area (nm^2) and P is the perimeter (μm). This value describes the degree to which the endothelial cells approach perfect hexagonality. A perfect hexagon has a value of 0.907, but deviations from hexagonality reduce this value. The number of apices of each endothelial cell describes the cell shape. To reduce the sampling error, 50 cells in the centre of each specular microscopy image were analysed and the ECD, CV and the percentage of hexagonality of three measurements were averaged in each eye. Intra-investigator and inter-investigator measurements were tested in a group of 39 healthy patients, which yielded a small CV (5.0% ± 3.6%).
All the examinations were performed on the RD and control eyes, preoperatively and at 3, 6 and 12 months follow-up. Measurements were obtained after the instillation of tropicamide 0.5% and phenylephrine 2.5% (Thea Pharma, Wetteren, Belgium), except for IOP.

Lens status and definition of variables

For the purpose of our analysis, the eyes were classified into five groups according to lens status: group 1: phakic eyes that remained phakic; group 2: pseudophakic eyes that remained pseudophakic; group 3: phakic eyes that underwent phacoemulsification with IOL implantation; group 4: aphakic eyes that were originally phakic or pseudophakic, but

underwent lensectomy or IOL removal; group 5: fellow eyes (control group) that did not undergo any intraocular procedures during follow-up.
Recurrent retinal detachment after SO removal referred to either complete retinal re-detachment, or local retinal detachment if progression could not be confined by laser treatment. Anatomical success was defined as attachment of the retina within the photocoagulation barrier at the end of follow-up. For glaucoma, we used the following definition from Silicone Study Report 6: an intraocular pressure of more than 25 mm Hg, or more than 20 mm Hg with antiglaucoma medication, measured at any time during follow-up.[23] Our patients received anti-glaucoma medication if their IOP was higher than 25 mm Hg and the treatment mostly continued until just after SO removal. Hypotony was defined as an intraocular pressure of less than 5 mm Hg.[23] Keratopathy was defined as bullous or band-shaped keratopathy, epithelial or stromal oedema or localized opacities, in conformity with Silicone Study Report 6.[23]

Statistical analysis

Statistical analysis was performed with the SPSS software 16.0 (SPSS for Windows, Rel. 13.0 Chicago: SPSS Inc., USA) using independent sample T-test for univariate analysis. Multivariate analysis was performed by applying a linear mixed model to our data, with ECD as independent variable, time as covariate and assuming a random intercept per eye. Significance was defined as $P < 0.05$. Snellen visual acuities were converted into a logarithmic scale (LogMAR, i.e. the logarithm of the minimal angle of resolution), as described previously.[24] In this study, poor visual outcome was defined as visual acuity of less than 0.1 Snellen at 12 months follow-up. The linear mixed model was applied to all the eyes, including those that were excluded from our analysis and the control eyes (total, n=168), with ECD as the independent variable, time as covariate and assuming a random intercept per eye (2354.8 ± 70.1 cells/mm^2). Glaucoma, SO in the anterior chamber (AC)

Table 8.1: Patients lost to follow-up

No. of patients (n=11)	Reason
3	Patient could not or did not want to participate in the study
5	Inadequate postoperative specular micrographs because of punctate Epitheliopathy or haze due to poor recovery of an erosion
1	Corneal oedema due to-cornea decompensation
2	Died during follow-up

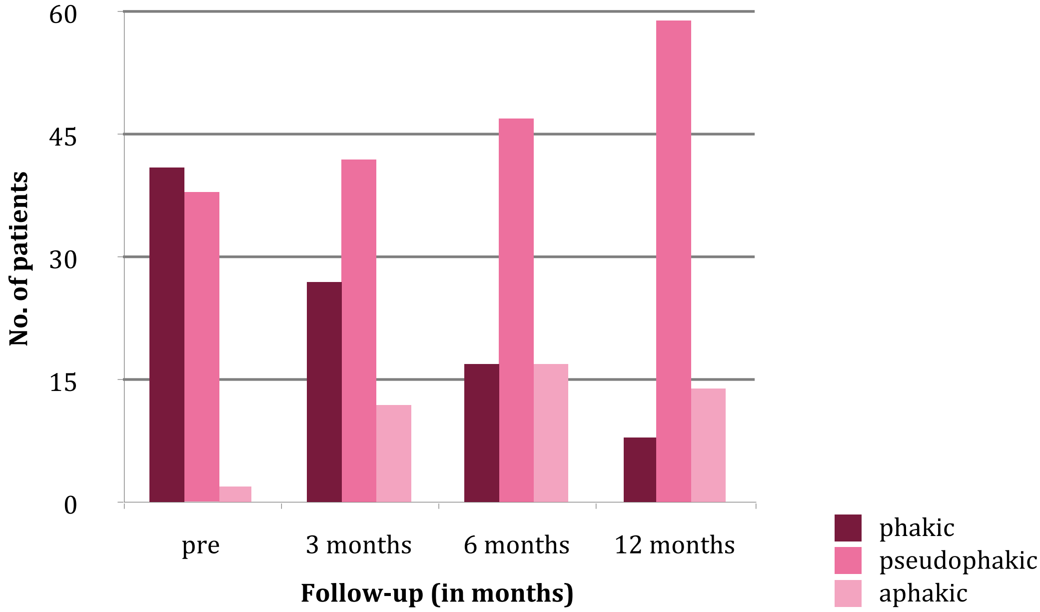

Figure 8.1: Lens status of the patients during the follow-up

and cataract extraction during follow-up were also included as fixed factors. We excluded various parameters from the linear mixed model that were not found to be significant in the multivariate analysis, such as cumulative surgical duration, emulsification, erosion during follow-up, IOP on the first day and perfluorocarbon liquid in the AC.

Results

Between November 2006 and November 2008, a total of 92 consecutive patients with RRD underwent vitrectomy and SO injection at our clinic. In 81 patients, follow-up data were available over a minimum period of one year. Mean age of the 81 patients was 61.1 ± 12.0 years; there were 70.4% men (n=57) and 29.6% women (n=24). At 3 months follow-up, 96.3% of the eyes (n=78) still had SO tamponade; at 6 months this rate was 58% (n=47) and at 12 months, this rate was only 23.5% (n=19). The eleven excluded eyes (Table 8.1) comprised 72.7% (n=8) men and 27.3% (n=3) women. Mean age was 66.2 ± 14.7 years; mean baseline ECD was 2056.8 ± 532.1 cells/mm^2. This was not significantly different from the mean value in the study group of 81 patients (2309.4 ± 417.8 cells/mm^2).

Ocular history of the 81 eyes at baseline was as follows: 32.1% of the eyes (n=26) did not have any ophthalmological ocular history, 46.9% (n=38) eyes had undergone phacoemulsification with IOL implantation, 21% (n=17) eyes had a history of scleral buckling surgery, 9.9% (n=8) eyes had undergone primary vitrectomy (SO or gas tamponade) for various reasons and 4.9% (n=4) eyes had undergone vitrectomy with phacoemulsification, or simultaneous scleral buckling surgery.

The eyes were classified into five different groups depending on their lens status (Table 8.2): group 1: phakic eyes that remained phakic; group 2: pseudophakic eyes that remained pseudophakic; group 3: phakic eyes that underwent phacoemulsification with IOL implantation; group 4: eyes that became aphakic; group 5: fellow eyes that served as controls.

No statistically significant preoperative / baseline differences were found between these five groups, except for intraocular pressure, which was significantly higher in the control eyes (15.4 ± 3.6 mm Hg) than in the study eyes (13.4 ± 4.1 mm Hg, P<0.001). Re-operation applied to: 25% (n=2) in group 1, 31% (n=10) in group 2, 46% (n=10) in group 3 and 44% (n=8) in group 4. No significant differences were found between the groups (P=0.566).

Lens status changed in some of the patients during follow-up (Figure 8.1). After one year, seven out of the eight phakic eyes had undergone phacoemulsification with IOL implantation; one patient refused cataract surgery because of her poor physical condition; one aphakic eye with an endothelial cell density of less than 1500 cells/mm^2 did not undergo secondary IOL implantation.

Endothelial cell density (ECD)

Mean postoperative ECD change at 12 months varied between 2.4% and -39.2% (Table 8.2). In group 1 (phakic eyes that remained phakic) and group 2 (pseudophakic eyes that remained pseudophakic) there was less than 5% EC loss 12 months after vitrectomy with silicone oil tamponade for complex RRD (see Table 8.2). In group 3 (phakic eyes that underwent phacoemulsification with IOL implantation) mean EC loss was 19% at 12 months (P<0.001). In group 4 (eyes that became aphakic after lensectomy or IOL removal) EC loss was even higher at 3, 6 and 12 months (P=0.002, P=0.001 and P<0.001) with a mean endothelial cell loss of 39% at 12 months (Figure 8.2, Table 8.2). No significant differences were found in the mean percentage of hexagonal cells or in the coefficient of variation between groups before or after surgery (Table 8.2).

During follow-up, SO was found in the AC in 10 eyes. Mean EC loss in these eyes at 12 months was 32% (712.7 ± 480.0 cells/mm^2, NS) compared to 13% loss in the 71 eyes without SO in the AC (311.8 ± 490.8 cells/ mm^2, P<0.001). In group 4 (eyes that became aphakic after lensectomy or IOL removal) EC loss in the four patients with SO in the AC at 12 months was 52% (1182.5 ± 365.6 cells/mm^2, P=0.007), whereas in the other 14 eyes that did not have SO in the AC, EC loss was 36% (857.3 ± 464.2 cells/mm^2, P<0.001).

linear mixed model analysis showed that ECD was significantly lower in glaucomatous eyes (β=-104, P<0.001) and also in eyes with SO in the AC (β=-336, P<0.001). ECD decreased significantly more rapidly in group 3 (β=-31 per month, P<0.001) and group 4

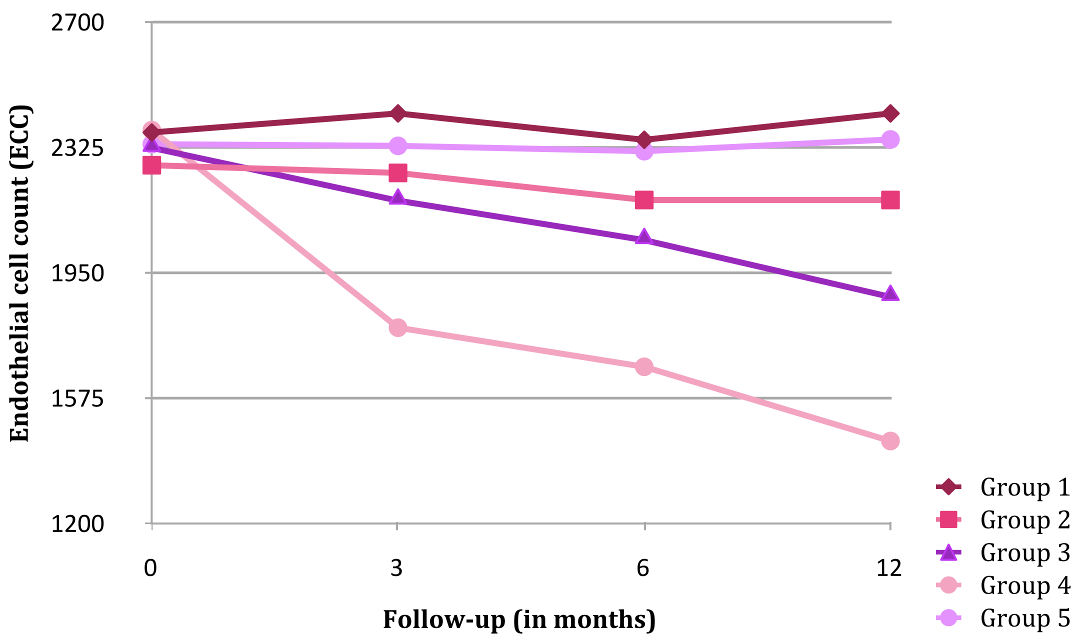

Figure 8.2: Endothelial cell count during follow-up

Group 1: phakic eyes that remained phakic; group 2: pseudophakic eyes that remained pseudophakic; group 3: phakic eyes that underwent phacoemulsification with IOL implantation; group 4: eyes that became aphakic; group 5: fellow eyes that served as controls

ECD was significantly lower in groups 3 and 4 at 6 months and 12 months (P=0.001 and P<0.001) and at 3, 6 and 12 months (P=0.002, P=0.001 and P<0.001)

(β=-66 per month, P<0.001) than in the controls (β=-2.7 per month, see Figure 8.2). Linear mixed model analysis did not reveal any significant correlation between ECD loss and hypotony, cumulative duration of surgery and the number of operations after primary SO injection. Furthermore, SO in the AC was not found to be associated with hypotony.

Intraocular pressure

Mean preoperative IOP in the whole study group of 81 eyes with complex RRD was 13.4 ± 4.2 mm Hg. Five patients (6.3%) had a history of glaucoma and were receiving treatment with local antiglaucoma medication before primary vitrectomy. In the first three months following surgery, 58% of the patients (n=46) were on temporary antiglaucoma medication. Between 3 months and 6 months, 40% (n=32) were using antiglaucoma medication. At 12 months, 30% (24 patients) were still on antiglaucoma medication. Compared to the preoperative values, EC loss at 12 months was 19% in the glaucoma group (P<0.001) versus 11% in the group without glaucoma (P=0.002).

Table 8.2: Corneal endothelial cell assessment after vitrectomy with silicone oil tamponade

Group	Preoperative	3 months	6 months	12 months	% change at 12 months compared to preoperative	P value
Endothelial cell density (cells/mm²)						
Group 1 (n=8)	2367 ± 268	2425 ± 312	2345 ± 309	2425 ± 340	+2.4%	NS
Group 2 (n=32)	2270 ± 433	2247 ± 440	2165 ± 419	2165 ± 422	-4.6%	NS
Group 3 (n=22)	2321 ± 467	2163 ± 556	2045 ± 471	1876 ± 515	-19.2%	<0.001
Group 4 (n=18)	2374 ± 386	1783 ± 691	1666 ± 649	1445 ± 437	-39.2%	<0.001
Group 5 (n=73)	2333 ± 409	2327 ± 470	2311 ± 503	2346 ± 400	+0.6%	NS
Percentage of hexagonal cells (%)						
Group 1 (n=8)	58.4 ± 10.0	61.8 ± 5.6	58.5 ± 6.1	54.1 ± 9.0	-1.6%	NS
Group 2 (n=32)	57.7 ± 9.6	58.1 ± 7.1	55.2 ± 8.7	59.3 ± 8.3	+2.8%	NS
Group 3 (n=22)	61.4 ± 10.5	60.4 ± 10.9	56.1 ± 7.9	55.6 ± 8.8	-9.4%	NS
Group 4 (n=18)‡	58.66 ± 8.6	55.5 ± 10.7	55.4 ± 9.1	56.5 ± 4.7	-3.6%	NS
Group 5 (n=73)	61.5 ± 9.3	59.4 ± 8.5	58.6 ± 10.1	59.6 ± 9.3	-3.1%	NS
Coefficient of variation (%)						
Group 1 (n=8)	29.5 ± 4.0	30.6 ± 4.0	31.1 ± 3.2	31.8 ± 5.1	+7.8%	NS
Group 2 (n=32)	38.2 ± 23.5	30.7 ± 5.0	33.4 ± 7.2	33.1 ± 22.3	-13.4%	NS
Group 3 (n=22)	41.7 ± 38.9	29.4 ± 4.0	32.0 ± 4.9	31.2 ± 5.4	-25.2%	NS
Group 4 (n=18)	30.2 ± 3.9	32.0 ± 6.6	33.8 ± 7.9	32.4 ± 7.9	-22.8%	NS
Group 5 (n=73)	31.6 ± 5.1	33.1 ± 16.3	31.2 ± 6.1	26.1 ± 22.4	-17.4%	NS

ECD: endothelial cell density (cells/mm²), NS: not significant‡ no significant difference was found with a non-parametric test (wilcoxon) also no significant difference was found P value: significance was defined as $P < 0.05$

Group 1: phakic eyes that remained phakic; group 2: pseudophakic eyes that remained pseudophakic; group 3: phakic eyes that underwent phacoemulsification with IOL implantation; group 4: eyes that became aphakic; group 5: fellow eyes that served as controls

Discussion

In the present study, EC loss was less than 5% at 12 months after vitrectomy with SO tamponade for complex RRD in group 1 (phakic eyes that remained phakic) and group 2 (pseudophakic eyes that remained pseudophakic). Highest postoperative EC loss was found in the eyes that underwent an additional phacoemulsification procedure with IOL removal. Glaucoma and direct oil-corneal touch had further negative effects on ECD.

In an earlier prospective study on ECD after PPV without SO tamponade, Friberg et al[6] reported significantly higher ECD loss in aphakic eyes and in eyes that underwent simultaneous lensectomy, with or without gas-fluid exchange, than in patients who underwent scleral buckling alone. They also found lower percentages of EC loss in their aphakic group (8.5-16.9%) than the levels in the present study. However, their ECD measurements were performed after shorter follow-up (3.9-6.7 months).[6] No EC loss was found in phakic eyes that underwent PPV without lens removal. These authors hypothesized that an intact lens may protect the corneal endothelium during PPV and that fluid-gas exchange may be harmful to aphakic eyes.[6] Rosenfeld et al[16] reported greater EC loss six months postoperatively in aphakic eyes (13%) and in eyes that underwent lensectomy combined with PPV (17%) than in phakic eyes (0.4%).[16] Although these authors did not study PPV with SO tamponade, they launched the idea that higher EC loss may be due to the use of SO. This was confirmed in the present study, because we found 39% EC loss in aphakic eyes and 52% EC loss in eyes with SO in the AC. An earlier study reported EC loss of up to 83% when there was SO in the AC and when SO had been in direct contact with the corneal endothelium.[25] However, the latter study involved only four eyes and was not prospective.[25] Although oil-corneal touch was a significant risk factor for EC loss in the present study, the cumulative duration of surgery was not a significant risk factor for EC loss.

Szaflik et al[26] observed significant EC loss in the upper parts of the cornea in patients with SO in the AC. A mean difference of 32% was found between the upper part and the central part of the cornea (985 cells/mm^2 and 1456 cells/mm^2, respectively).[26] All the patients had irregular cell shapes and EC lesions, while six patients showed aberrant ECs in the upper part of the cornea.[26] In contrast with Szaflik et al,[26] we performed our measurements in the centre of the cornea, according to a standard protocol described previously.[27-32]

Reduced frequency of hexagons with associated elevations in other cell shapes is indicative of increased cellular pleomorphism. Endothelial morphology is the best indicator of corneal endothelial stress or instability. As a general rule, stressed corneas have: % Hex <45 and/or CV >45. ECD changes indicate cell damage that has already occurred, while EC morphology represents ongoing corneal endothelial cell stress or instability.[22] The size and shape of corneal ECs provides objective information on damage levels and corneal endothelial functioning.[33-35] We did not find any significant difference in the CV or the percentage of hexagonal cells between the groups. The latter finding seems to indicate the absence of wound healing, despite high EC loss, which suggests unchanged EC function. On the other hand, there may not have been any variation in cell size (polymegathism) or irregularity in cell shape with a decreased percentage of hexagons (pleomorphism) at the central location where we took our measurements, in contrast with Szaflik et al.[26] In the study by Szaflik et al,[26] ten patients had direct silicone exposure to the upper part of the cornea. Confocal specular microscopy examinations were performed between 3 weeks to 10 months after detecting SO in the AC. Another difference between the latter study and our study was that they used the upper part of the cornea of the same eye as "control", whereas in the present study, in which EC loss was up to 52% in these cases, we obtained this percentage by comparing preoperative and postoperative values.

In a more recent study, Friberg et al[7] showed that SO retention in the vitreous body made a significant contribution to EC loss. Compared to the fellow control eye, mean EC loss was 69% in 10 eyes with SO retention after a mean follow-up of 10 months. Mean EC loss was almost 95% in three eyes with SO in the AC.[7] Pseudophakic eyes had 52% EC loss compared to aphakic eyes (67%).[7] A major conclusion was that EC loss after vitreoretinal surgery may increase further due to long-term SO retention.[11] Previous studies reported that high EC loss occurred in eyes without a barrier between the anterior segment and the vitreous cavity,[6,7,16] but that EC loss was highest in eyes in which SO was in direct contact with the endothelium.[6, 7] The authors suggested that the original lens and artificial IOL may have protected the endothelium from turbulence and from prolonged exposure to the irrigation solution and SO during vitreous surgery.[16] It has been hypothesized that EC damage is cumulative over repeated intraocular operations.[7, 36] Although direct oil-corneal touch was a significant risk factor for EC loss in the present study, the cumulative duration of surgery was not. Emulsified oil droplets that pass through the zonular fibres of the lens may damage the corneal endothelium and cause long-term EC loss. However, in the present study, we did not find any association between EC loss and the degree of oil emulsification.

The eyes with glaucoma in our series had significantly higher EC loss at 12 months than the eyes without glaucoma (19% vs 11%). In addition, 30% of the patients were still on antiglaucoma medication at one year follow-up. A negative relation was reported between ECD and glaucoma in patients with a history of acute angle closure glaucoma.[37-40] High intraocular pressures, even for a minimal duration of 3 days, significantly lowered the central ECD.[39]

Boscia et al[8] studied EC loss in eyes that underwent combined phacoemulsification and SO removal (n=17).[8] They found that after 6 months, passive SO efflux from the AC caused significantly more EC loss (11.2%) than phacoemulsification as a single procedure without simultaneous SO removal (8.3%).[8] These percentages of EC loss were comparable with our findings after 6 and 12 months in the group with combined SO removal (through a pars plana approach) and phacoemulsification: 12% and 19% EC loss, respectively. However, Boscia et al[8] used a different SO removal technique via the AC with a 16-gauge cannula through posterior capsulorhexis, which was performed at the end of the phacoemulsification procedure.[8] However, in the present study, SO removal was always performed through a pars plana approach after the completion of phacoemulsification.

Mean preoperative ECDs were different in group 1 and group 2: 2367 ± 268.3 and 2269.6 ± 433.2, respectively. The difference was 4%, which was comparable with the literature.[41] Much greater ECD loss was found in group 3 and group 4. Group 3 underwent anterior segment surgery during follow-up and became pseudophakic. EC loss was smaller in group 3 (phacoemulsification without IOL implantation) than in group 4 (lensectomy). However, in some cases a secondary IOL was placed. Patients with low EC loss did not receive a secondary IOL. Faramarzi et al[41] found an ECD of 6% after phacoemulsification. This was comparable with our preoperative ECD in group 2 (pseudophakic eyes that remained pseudophakic). However, this did not explain why the group that underwent phacoemulsification during follow-up had much greater ECD loss.

One of the limitations of the present study was that the patient population who required SO was quite heterogeneous, with eyes in various states. The effects of these confounding factors were not controlled for in the final analysis, but their inclusion would probably dilute the results to the point of insignificance.

A further problem was that the number of ECs that need to be counted in order to obtain reliable specular microscopy data is still controversial. Benetz et al[42] suggested that at least 30 ECs should be counted to obtain maximum accuracy, whereas Doughty et al[43] recommended a total of 75 cells for the highest precision. In this study, the number of ECs counted was sometimes lower than 30, which should be taken into account when interpreting our results. We also determined the best case scenario with a single clinical site, single photographer and a single reader. Our reading precision was 2%. Thus, in group 1, the cell counts were within the noise of our measurement precision with non-contact specular microscopy and the results were probably representative of no change in ECD.

Another shortcoming was that this study did not incorporate a control group with gas tamponade and a comparable duration of surgery. Therefore, we could not establish whether the ECD changes were truly attributable to oil tamponade. It should also be kept in mind that EC loss may be even higher if patients are remeasured after longer follow-up.

In summary, less than 5% EC loss was measured at 12 months follow-up in eyes that had undergone vitrectomy with SO tamponade, without any additional surgical procedures to the anterior segment. Highest postoperative EC loss was found in eyes that underwent an additional phacoemulsification procedure (mean -19% change) or IOL removal (mean -39% change) during the study period. Glaucoma and direct oil-corneal touch had further negative effects on ECD counts. Our hypothesis is that the lens plays an important role in protecting the ECs in eyes that receive SO tamponade during the surgical management of complex retinal detachment. Apparently, the lens functions as a protective EC shield against turbulence and prolonged exposure to the irrigating solutions and SO. Lensectomy, i.e. removal of the barrier between the cornea and posterior segment during vitreoretinal procedures, possibly accelerates EC loss. It therefore seems justified to avoid lensectomy whenever possible.

To further protect the ECs and restrict the number of vitreoretinal procedures, primary retinectomy seems to be a good option for proliferative vitreoretinopathy.[44] In general, reducing the overall duration of surgery may decrease EC loss. To minimize intraoperative turbulence, high viscosity viscoelastics can be used, because they provide more EC protection than lower viscosity viscoelastics.[45] Owing to the fact that eyes with complex retinal detachment often need repeat vitreoretinal surgical procedures, the above-mentioned techniques deserve consideration to maintain long-term corneal clarity.

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Chapter 9

General Discussion
Summary
Samenvatting
Dankwoord
Curriculum vitae
List of publications

General Discussion

Retinal detachment

Posterior vitreous detachment (PVD) is a common age-related degenerative condition,[1, 2] in which patients experience light floaters and/or flashes.[3] These symptoms are specific to PVD, but not all patients suffer from them. In the studies described in this thesis, the first symptoms expressed by the patients were: floaters (60.2%) and flashes (37.6%). A study by Hikichi and Trempe[3] reported 89% sensitivity for floaters and flashes, with a specificity of 25%. This confirms the statement made by Green and Sebag that *posterior vitreous detachment* is one of the least accurate diagnosis employed by ophthalmologists on a daily basis.[4] Symptoms of rhegmatogenous retinal detachment (RRD) are flashes and visual field reduction.[5] We observed that the median overall delay between the patients' first subjective symptoms and the time of surgery was 10 days. Almost 60% of this overall delay was caused by patient-related delay and general practitioner (GP) delay. In addition, in more than 50% of the patients, the delay was due to unawareness and/or unfamiliarity with the symptoms of PVD and/or RRD. For example, patients explained that they had not considered the flashes, floaters or visual field reduction to be serious, so they did not seek prompt medical attention. Mean patient-related delay was significantly lower in the patients with vitreous haemorrhage and in the patients with a history of a RRD in the fellow eye. Acute visual function loss in the patients with vitreous haemorrhage was probably the reason why they sought immediate help. Patients who have been treated for RRD in the fellow eye are more likely to recognize the symptoms of impending retinal detachment and contact a doctor straight away.
PVD is also associated with an increased risk of developing retinal tears.[6] The literature reports an incidence of 7.3-14% in patients with PVD.[7] Although RRD may be present, or it may develop following PVD, not all the patients experience flashes and/or floaters. The annual incidence of retinal detachment is 5 to 18.2 per 100,000 persons.[8-16] There are several treatment options, which all have advantages and disadvantages.[17-23]

Results of scleral buckling surgery

Anatomical re-attachment of the retina was achieved in 76% of our series of consecutive, non-selected patients after one scleral buckling (SB) procedure. Ultimate anatomical success was achieved in 97%. These rates were comparable with earlier reports on RRD following SB surgery alone [24-34] after a follow-up of more than 10 years.
In our series, 32 eyes (7%) and 20 eyes (5%) developed re-detachment after more than six and twelve months follow-up, respectively. Multivariate regression analysis showed that recurrent re-detachment and more than seven days of visual field loss were significant predictors of poor postoperative visual outcome at 12 months follow-up. Furthermore, in recurrent RRD, a cumulative size of the tear of more than 3 disc

diameters was a significant predictor of poor outcome. The predictors of poor visual outcome found in the present study corresponded with the risk factors reported in earlier studies.[35-38] Late re-detachment forms an argument to follow patients for at least 6 months after an SB procedure. Moreover, visual acuity may continue to improve for several months after SB surgery.

Using foveal densitometry, Liem et al[39] showed that it may take several months for the foveal cone photopigments to recover after macular detachment. In addition, subfoveal fluid sometimes persists subclinically for several months following an SB procedure, which may explain the slow visual recovery in certain patients.[40-42]

The disadvantage of longer postoperative follow-up, however, may be bias caused by visual acuity degeneration in the elderly patients (cataracts or macular problems). In the present study, the visual acuity results were therefore analysed after a fixed period of 12 months following the primary operation.

Our studies demonstrated that anterior chamber depth (ACD) was significantly reduced until nine months after SB surgery. The ACD had returned to normal levels at one year follow-up.

In this thesis, we did not find any significant differences in outcome between radial and segmental buckles. All the eyes treated in our series received an additional encircling band. This encircling element itself may have caused the decrease in ACD, which can be explained as follows: encircling elements may reduce the uveal or retinochoroidal circulation and cause ciliary body oedema.[22] This ciliary body oedema with forward rotation of its body and forward shift of the iris-lens diaphragm, in combination with compression by the encircling element on the vitreous, may cause significant anterior chamber (AC) reduction.[22] In our opinion, this was probably the major reason for the decrease in ACD. This notion was supported by our finding of increased lens rise. The axial length was significantly increased and continued to increase during follow-up, which resulted in a mean myopic shift of 2.6 D three months after SB surgery.

If a patient presents with a combination of myopic phakic intraocular lens (pIOL), RRD and an ACD of less than 3.5 mm, the vitreoretinal surgeon may consider performing primary vitrectomy instead of an SB procedure. Based on the finding of a significant negative correlation between ACD and the loss of endothelial cells, Saxena et al[8] suggested stricter inclusion criteria for pIOL implantation surgery. They proposed that the ACD should be at least 3.5 mm,[8] measured from the corneal epithelium to the anterior pole of the crystalline lens or IOL, in order to prevent excessive endothelial cell loss after pIOL implantation. Marigo et al[27] showed that uncomplicated pars plana vitrectomy did not induce any long-term changes in anterior segment morphometry. A disadvantage of performing vitrectomy in eyes with RRD and a pIOL may be the progressive development of cataract, which requires removal of the pIOL.[28, 29] However, if a patient with a history of SB surgery wants to have a pIOL implanted, but the ACD is too shallow, it may be a good option to dissect the encircling element, as described by Kreissig et al.[30]

In our study on the occurrence and pattern of binocular diplopia, we found that 3.8% of the 1030 eyes that underwent SB surgery for RRD developed secondary diplopia during a mean follow-up of more than six months. Orthoptic assessment confirmed mechanical restriction in 2.7% of the cases. No significant correlation was found between the position of the buckle or the type of buckle and the incidence of diplopia or the pattern of strabismus. When two muscles were affected by the buckle, a moderately significant association was found with the incidence of diplopia. However, this association was not found when three or four muscles were affected, which was contrary to the expected trend of the more muscles affected, the higher the incidence of diplopia. It should be noted that we cannot rule out whether this was due to the small numbers in these groups. Based on our calculations and statistics, we concluded that in the present retrospective study, diplopia was only moderately associated with the number of muscles affected, whereas it was not associated with the buckle position. Furthermore, in the majority of cases in the diplopia group, the buckle was not positioned underneath the muscle that would be expected to be associated with the diplopia. Therefore, the incidence of diplopia was only predictable in a minority of the patients.

In 1973, Kanski et al[4] reported that 25 out of the 30 patients who developed diplopia after SB surgery (n=750) underwent a secondary procedure to mobilize the muscles, which was standard practice in those days. In the present study, however, we did not mobilize any of the muscles, or perform tendonectomy.

Many causes for strabismus are described in the literature. Local anaesthetics[7] may cause anaesthetic myotoxicity, with temporary restrictive strabismus, as was reported by Salama et al.[7] This can be ruled out in the present series, because all the patients were operated on under general anaesthesia without any additional locoregional anaesthetic. Ischaemia of the rectus muscles may also be an explanation for diplopia after SB surgery. An ischaemic muscle has lost its function and/or strength. Ischaemia of the rectus muscles may develop due to muscle constraint, or arterial occlusion caused by compression from the encircling element.[23] In addition, if a muscle is constrained too aggressively, it may become seriously traumatized and result in haemorrhage and/or oedema of the muscle, but also ultimately to fibrosis and scarring, with restriction of its function.[14] This mechanism may be the explanation in some of our cases, as there was no apparent association between the position of the buckle and the pattern of diplopia. Strikingly, none of the buckles in the present study were beneath the muscles in the six cases with diplopia (21%). These were all cases with a fusional problem as result of de-compensation of previous heterophoria caused by decreased visual acuity.

Binocular single vision was ultimately restored in 72% of the cases with diplopia in the present study. Re-detachment was an additional risk factor for diplopia, because it diminished or eradicated the existing fusion due to further deterioration in visual acuity. It was not always possible to restore this fusion if sensory deprivation was caused by long-term macular detachment.[27-29] The only possible treatment in these cases, therefore, was occlusion of the eye.

Results of vitrectomy in retinal detachment surgery

Many techniques have been described for the management of giant retinal tears (GRTs).[43-75] In the present study, we found that pars plana vitrectomy (PPV) with the absence of an encircling scleral element was a significant risk factor for re-detachment, which supported the results reported by Verstraeten et al.[68] Possible explanations are that an encircling scleral element reduces traction on the remaining vitreous and that the encircling scleral element exerts more pressure on the retina when silicone oil (SO) tamponade is involved.[76, 77] An argument in favour of using an encircling narrow tyre is that local re-detachment often occurs in the "intact" part of the retina and a 360° buckle may prevent any further detachment. If a larger tyre is used over a portion of the circumference, one option is to place it over the GRT, or alternatively, it can be placed on the "intact" retina, as was done by Aylward et al[73] in more than half of their patients.

In the present study, the re-detachment rate after one PPV procedure in patients with GRT was 30%; ultimately the retina was attached in 98% of the eyes. Over the past 10 years, primary anatomical success rates in eyes treated for GRT have varied between 50% and 94%.[43, 54, 55, 65, 66, 68, 72, 74] Ultimately, the success rate in most of the studies was approximately 95%.[43, 54, 55, 65, 66, 72] Anatomical success rates depend on the surgical technique, the inclusion or exclusion of eyes with traumatic GRT or proliferative vitreoretinopathy (PVR), the duration of follow-up and whether or not the silicone oil has been removed from the eye at the end of follow-up. Vitrectomy in combination with primary lensectomy, as described by Kreiger and Lewis[56] and Aylward et al,[73] was advocated to provide better access to the vitreous base and the extreme periphery of the retina.[56, 73] Without lensectomy, it may be difficult to reach the vitreous base.[56, 73] However, an encircling scleral element or indentation might improve access. Primary lensectomy can be a traumatic event associated with endothelial cell loss. Earlier studies have demonstrated that aphakic eyes have a poor visual outcome after surgery for complicated RD.[78, 79] In the present series, primary lensectomy was performed in a minority of the eyes.

In our study, re-detachment of the retina occurred in 19% of the eyes after silicone oil removal (SOR). We did not make any prior patient selection in relation to SOR. This is contrary to Silicone Oil Study Report no. 6,[80] in which the re-detachment rate was 20%, but only 45% of the eyes underwent SOR and patient selection did take place: the best eyes, with better visual acuity, fewer preoperative interventions and fewer complications.[80] Nowadays, we have far more sophisticated equipment and surgical techniques at our disposal,[81, 82] such as wide-angle viewing systems[83] and perfluorocarbon liquids (DK-line),[84-87] which have contributed to improving the overall outcome of vitreoretinal surgery compared to 20 years ago when the Silicone Oil Study Report was published.[80]

The overall anatomical success in our study was higher (94%) than that in Silicone Oil Study no. 6 (81%).[80] In the present study, re-detachment occurred after a median of 7.5 weeks (range 0 days to 108 weeks). Within the first three months after SOR, 26 eyes (68%) developed retinal re-detachment, which was mostly due to PVR. Within the first six months, 87% developed re-detachment. In the eyes with recurrent RRD, 8% developed the re-detachment more than one year after SOR. Our results were comparable with those of earlier studies, which reported a re-detachment rate of between 9% and 25% after SOR.[88-99] In the present series, a history of three or more operations and aphakia were identified as significant risk factors for the development of keratopathy, in agreement with earlier studies.[95, 96] We found significantly more keratopathy in patients with aphakia and/or a history of repeated ocular surgery, including anterior segment surgery, as has also been reported in the literature.[78, 88, 89, 99] These two variables are known to be associated with (cumulative) damage to the corneal endothelial cells,[78] which may lead to keratopathy.

This thesis also analysed postoperative endothelial cell (EC) loss after PPV with SO tamponade. The highest postoperative EC losses were found in the eyes that underwent an additional phacoemulsification procedure, with or without IOL removal during follow-up. Glaucoma and direct contact of the SO with the endothelial cells had further negative effects on endothelial cell density (ECD) counts. No EC loss was found in phakic eyes that underwent PPV without lens removal. Rosenfeld et al[78] reported greater reductions in ECD at six months follow-up in aphakic eyes (13%) and in eyes that underwent lensectomy combined with PPV (17%) than in phakic eyes (0.4%).[78] Although they did not study eyes after PPV with SO tamponade, they proposed the theory that higher EC losses may be due to the use of SO. The present study confirmed this hypothesis: in the aphakic eyes, EC loss was 39%, whereas in the eyes with SO in the AC, EC loss was as much as 52%. An earlier study reported an EC loss of up to 83% when SO filled the AC and when SO had been in direct contact with the corneal endothelium.[88] However, the latter study only involved 4 eyes and it did not have a prospective design.[88]

Recommendations for further research

Recurrence and complications: the treatment procedures for retinal detachment described in this thesis were not free from recurrence and complications. Therefore, further research is required into preventive measures for recurrence. It is believed that recurrence on the basis of proliferative vitreoretinopathy occurs due to excessive vitreous humour. Prior to surgery, measures could be taken to condense and liquefy the vitreous humour, such as the administration of ocriplasmin.[100-103] If vitreous humour is still in situ, PVR can develop.[104] Thus, some researchers are in favour of removing all the vitreous humour.[105]

Tamponade: it would be worthwhile to develop an alternative form of tamponade. Silicone oil is associated with complications such as EC loss, which can lead to bullous keratopahty, band-shaped keratopathy[106,107] or ocular hypertension due to emulsification.[95,96,108-110] A new tamponade must be a fluid with a large surface tension, such as perfluorocarbon liquid. [70,71,11-114] It should be clear, without a breaking index and resorbable within three months in the same way as an expandable gas, but not

retinotoxic. In addition, it must not emulsify in order to avoid ocular hypertension or endothelial cell loss in patients with aphakia. Medication may be added to the tamponade to prevent PVR for instance as a slow-release drug system.
Corneal decompensation due to EC loss after succesfull vitreoretinal surgery for complex detachments is a serious problem. The question of whether EC loss is caused by SO could be addressed by counting the endothelial cells in patients with retinal detachment who are scheduled for PPV with gas tamponade. Moreover, the development of more purified forms of endotamponade with less emulsification and /or less toxic effects to endothelial cells would diminish this complication.
The development of retinal chip implants may in the far future be of help in replacing deceased retinal neuronal cells in anatomic attached retinas with low visual outcome.

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SUMMARY

Retinal detachment is a sight-threatening condition, which requires immediate treatment in order to prevent blindness. This thesis aimed to determine the best possible treatment by answering the following questions: Why do patients with retinal detachment delay consulting a doctor? What is the best treatment for patients with rhegmatogenous retinal detachment (RRD)? Does the treatment have side-effects and what are the consequences for the rest of the eye?

In **Chapter 2**, we quantified the treatment delay in patients with RRD who underwent retinal detachment surgery and evaluated the causes for this delay. A total of 186 eyes were included in the analysis, with a follow-up of 1 year. Median overall delay between the patients' first symptoms and RRD surgery was 10 days. The major reason for patient delay was their unawareness and unfamiliarity with the symptoms of retinal detachment.

In **Chapters 3 to 5**, we analysed the results of scleral buckling (SB) surgery. In **Chapter 3**, we found that after a mean follow-up of 51 months, anatomical re-attachment had been achieved in 76% of the eyes after one SB procedure. The final re-attachment rate was 97% after additional vitreoretinal procedures. A total of 104 eyes developed re-detachment during follow-up. After more than six and twelve months follow-up, 32 eyes (7%) and 20 eyes (5%) developed re-detachment, respectively. Multivariate regression analysis showed that recurrent re-detachment and more than seven days of visual field loss were significant predictors of poor postoperative visual outcome at 12 months follow-up. Furthermore, a significant predictor of recurrent RRD was a cumulative tear size of more than 3 disc diameters.

Conventional SB surgery proved to be a reliable procedure in a selected group of eyes with primary RRD. However, in eyes with a retinal tear with a cumulative size of more than 3 disc diameters, primary vitrectomy should be considered. Based on our finding that 7% of the eyes developed re-detachment after 6 months, a longer follow-up period seems necessary to evaluate the anatomical and visual outcomes after SB surgery.

In **Chapter 4**, we analysed the anterior chamber depth (ACD) in 38 eyes after SB surgery. ACD was significantly reduced compared to the preoperative levels at up to 9 months follow-up. Depths had returned to normal one year after surgery. Axial length was significantly enlarged during the total observation period. No significant differences were found between the use of radial or segmental buckles. In patients with prior SB surgery who plan to have myopic pIOL implantation, dissection of the encircling element may be considered if the anterior chamber has become too shallow, with the aim of preventing high endothelial cell loss.

In **Chapter 5**, we analysed diplopia after SB surgery. In the 1030 eyes, secondary strabismus developed in 39 eyes (3.8%) during a mean follow-up of 6.4 ± 6.3 months; 28 patients (2.7%) developed strabismus due to mechanical restriction of one of the muscles. No association was found between the position of the buckle, i.e. the muscle affected, and the incidence of diplopia. In 28 out of the 39 patients, binocular single vision

was restored at the end of follow-up. This was accomplished with conventional prism treatment in the majority of cases.

In **Chapters 6 to 8**, we analysed the results of vitrectomy procedures with silicone oil (SO) tamponade for the treatment of retinal detachment. In **Chapter 6**, we analysed the functional and anatomical results in patients with giant retinal tear (GRT) retinal detachment who had been treated with pars plana vitrectomy (PPV) with or without an encircling element and SO tamponade. In a series of 30 eyes, the re-detachment rate was 30% (n=9) after one PPV procedure. Ultimately, the retina was attached in 29 (96.7%) eyes. Multivariate analysis showed that the absence of an encircling scleral buckle (P=0.008) was significantly associated with re-detachment. Visual acuity improved in 54% of the eyes. PPV with an encircling scleral buckle and SO tamponade seemed to be a successful treatment for GRT retinal detachment.

In **Chapter 7**, we analysed the anatomical and functional results after silicone oil removal (SOR) in patients who had undergone vitrectomy with SO. Anatomical success was achieved in 81% of the eyes after SOR. The overall anatomical success rate at the end of follow-up was 94%. Postoperative ocular hypertension was found in 8% of the eyes, hypotony in 6% of the eyes and keratopathy in 29% of the eyes. After SOR, visual acuity had improved by at least 2 Snellen lines in 43% of the eyes. Multivariate analysis showed that male gender, preoperative rubeosis and proliferative diabetic retinopathy (PDR) were risk factors for recurrent retinal detachment. Furthermore, poor visual outcome (i.e. Snellen visual acuity of <0.1) was associated with male gender, preoperative visual acuity of <0.1 Snellen lines, PDR, three or more surgical procedures, any size of retinectomy and hypotony.

In **Chapter 8**, we analysed the endothelial cell density (ECD) after vitrectomy with SO tamponade. Mean postoperative ECD change at 12 months follow-up varied between 2.4% and -39.2% compared to the preoperative values. Less than 5% endothelial cell (EC) loss was found in patients with a native lens or pseudophakia. In the eyes that underwent an additional phacoemulsification procedure and in the eyes that underwent lens and/or IOL removal, postoperative ECD was significantly reduced at 12 months follow-up, with a mean cell loss of 19% and 39%, respectively (P <0.001). ECD was also significantly lower in the eyes with glaucoma (β= -104, P<0.001) and in the eyes in which the SO filled the anterior chamber (β= -336, P<0.001). Mean postoperative percentage of hexagonal cells and the coefficients of variation did not differ significantly from the preoperative values.

SAMENVATTING

Netvliesloslating is een visus bedreigende ziekte waarbij behandeling noodzakelijk is om niet blind te worden. Om een zo goed mogelijke behandeling te realiseren beoogt dit proefschrift de volgende vragen te beantwoorden:

Waarom gaan patiënten niet eerder naar de dokter wanneer zij symptomen hebben van een netvliesloslating, zoals flitsen, vlekjes of gezichtsvelduitval?

Wat is de beste behandeling voor patiënten met een rhegmatogene netvliesloslating ?

Wat zijn de bijwerkingen van de behandeling en wat zijn de consequenties voor de rest van het oog?

In **Hoofdstuk 2** wordt de eerste vraag over het behandelingsuitstel besproken. Patiënten met een rhegmatogene netvliesloslating die netvlieschirurgie ondergaan werden gevraagd naar de redenen waarom zij niet direct naar de dokter gingen. Gedurende een periode van één jaar hebben wij 186 patiënten geïnterviewd. Het mediaan behandelingsuitstel tussen het moment waarop de eerste symptomen door de patiënten werden ervaren en de netvlieschirurgie bedroeg 10 dagen. Behandelingsuitstel veroorzaakt door de patiënten zelf kwam hoofdzakelijk door hun onwetendheid en onbekendheid met de symptomen van een netvliesloslating.

Hoofdstukken 3 t/m 5 onderzoeken de resultaten van een behandeling met cerclage plombe chirurgie bij patiënten met een netvliesloslating.

Hoofdstuk 3 toont aan dat gedurende een gemiddelde controle periode van 51 maanden anatomisch herstel werd bereikt bij 76% van de ogen na één operatie. Uiteindelijk werd anatomisch herstel bereikt bij 97% na extra vitreoretinale chirurgische ingrepen. Recidief netvliesloslating werd bij 104 ogen tijdens de controle periode gevonden. Na een controle periode van meer dan 6 maanden en meer dan 12 maanden ontwikkelde zich een recidief bij respectievelijk 32 ogen (7%) en 20 ogen (5%). Multivariate regressieanalyse werd gebruikt om risicofactoren voor een verminderd gezichtsvermogen 12 maanden na de operatie op te sporen. De volgende factoren bleken een significant voorspelbare waarde te hebben: een recidief netvliesloslating en een gezichtsvelduitval die langer dan zeven dagen bestond. Een significante risicofactor voor een recidief netvliesloslating bleek een defect in het netvlies van meer dan drie papil groottes te zijn. Conventionele cerclage plombe chirurgie bleek een betrouwbare procedure te zijn voor een bepaalde groep ogen met primaire rhegmatogene netvliesloslatingen. Voor ogen met een defect grootte van meer dan drie papil diameters in het netvlies dient primaire vitrectomie te worden overwogen. Op basis van onze bevinding dat 7% van de ogen een recidief netvliesloslating ontwikkelden na zes maanden, lijkt een langere controle periode nodig om anatomisch herstel en het gezichtsvermogen te beoordelen na cerclage plombe chirurgie.

Hoofdstuk 4 analyseert de diepte van de voorste oogkamer bij 38 ogen na cerclage plombe chirurgie. Na een controle periode van 9 maanden was de diepte van de voorste oogkamer significant minder diep dan voor de operatie. Een jaar na de operatie was de diepte van de voorste oogkamer bijna hersteld tot de normale waarde. De aslengte was

significant vergroot tijdens de gehele observatie periode. Er was geen verschil tussen het gebruik van een radiaire of een limbus-parallelle plombe.

Voor patiënten die cerclage plombe chirurgie hebben ondergaan en van plan zijn refractiechirurgie door middel van een fake kunststof voorste oogkamerlens te laten implanteren, kan, als de diepte van de voorste oogkamer te ondiep is geworden, dissectie van het cerclage element overwogen worden om het verlies van endotheelcellen tegen te gaan.

Hoofdstuk 5 onderzoekt hoe vaak dubbelbeelden voorkomen na cerclage plombe chirurgie. Secundaire dubbelbeelden werd gevonden bij 39 van de 1030 ogen (3,8%) tijdens een gemiddelde controle periode van 6,4 ± 6,3 maanden; 28 patiënten (2,7%) kregen dubbelbeelden door mechanische restrictie van een van de oogspieren. Er was geen relatie tussen de positie van de plombe (t.o.v. de aangedane spier) en het voorkomen van dubbelbeelden. Aan het einde van de controle periode was enkelvoudig binoculair zien hersteld bij 28 van de 39 patiënten. In het merendeel van de gevallen werd dit met een prismacorrectie bereikt.

Hoofdstukken 6 t/m 8 analyseren de uitkomsten van een vitrectomie behandeling met silicone olie tamponade voor patiënten met RRD. **Hoofdstuk 6** beschrijft de functionele en anatomische uitkomsten bij patiënten met een reuzescheur netvliesloslating na een pars plana vitrectomie procedure met of zonder een cerclage en silicone olie tamponade. Recidief netvliesloslating trad op bij negen van de 30 ogen (30%) na één vitrectomie. Uiteindelijk is het netvlies weer aanliggend bij 29 ogen (96,7%). Met multivariate regressieanalyse werd een significante (P=0,008) relatie aangetoond tussen het ontbreken van een cerclage en recidief netvliesloslating. Verbetering van het gezichtsvermogen werd gemeten bij 54% van de ogen. Vitrectomie met cerclage en silicone olie tamponade bleek een succesvolle behandeling te zijn voor reuzescheur netvliesloslating.

Hoofdstuk 7 inventariseert de anatomische en functionele uitkomsten bij patiënten na vitrectomie met silicone olie tamponade na het verwijderen van de silicone olie. Anatomisch herstel werd bereikt bij 81% van de ogen na het verwijderen van de silicone olie. Het uiteindelijke succespercentage was 94% aan het einde van de controle periode. Na de operatie werd een verhoogde oogdruk gevonden bij 8% van de ogen, een verlaagde oogdruk bij 6% van de ogen en keratopathie bij 29% van de ogen. Het gezichtsvermogen verbeterde met meer dan 2 Snellen lijnen bij 43% van de ogen na het verwijderen van de silicone olie. Multivariate analyse werd gebruikt om risicofactoren voor een recidief netvliesloslating aan te tonen. De volgende factoren bleken een significant voorspellende waarde te hebben: mannelijk geslacht, preoperatieve rubeosis en proliferatieve diabetische retinopathie. Ook werd er een relatie gevonden tussen een verslechterd gezichtsvermogen (Snellen <0,1) en mannelijk geslacht, preoperatief gezichtsvermogen van <0,1 Snellen lijnen, proliferatieve diabetische retinopathie, drie of meer chirurgische interventies, gedeeltelijke retinectomie (ongeacht de grootte) en verlaagde oogdruk.

Hoofdstuk 8 onderzoekt de endotheelceldichtheid in het hoornvlies na vitrectomie met silicone olie tamponade. Twaalf maanden na de operatie lag de gemiddelde endotheelceldichtheid verandering tussen de 2,4% en -39,2% vergeleken met de preoperatieve endotheelceldichtheid. Minder dan 5% verlies aan endotheelceldichtheid werd gezien bij patiënten met een eigen lens of een kunstlens. Bij de 12-maandelijkse controle was het gemiddelde endotheelceldichtheid verlies in de ogen met een gecombineerde phaco-emulsificatie procedure en de ogen met een (kunst)lens extractie significant hoger: respectievelijk 19% en 39% ($P < 0{,}001$). Ook was de endotheelceldichtheid significant lager bij de ogen met glaucoom ($\beta = -104$, $P<0{,}001$) en de ogen met silicone olie in de voorste oogkamer ($\beta = -336$, $P<0{,}001$). Het percentage hexagonale cellen en de variatie coëfficiënten veranderen na de operatie niet significant.

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CURRICULUM VITAE

De auteur van dit proefschrift werd geboren op 22 februari 1974 te Amsterdam als Fleur Goezinne. Ze groeide op in Vinkeveen. Het voortgezet onderwijs werd in Amsterdam gevolgd. Na het behalen van het examen voorgezet wetenschappelijk onderwijs aan het Amsterdamse Fons Vitae Lyceum in 1994 begon ze met de studie medische biologie aan de Universiteit van Amsterdam. Tijdens deze studie werd zij ingeloot voor geneeskunde, ook aan de Universiteit van Amsterdam. Na het behalen van haar artsexamen 23 mei 2003 en doctoraal medische biologie ging zij haar specialisatie oogheelkunde doen in Maastricht. In 2008 werd ze ingeschreven in het BIG register als oogarts, en volgde ze een fellowship vitreoretinale chirurgie eveneens in Maastricht. Tot op heden werkt ze daar als netvlieschirurg.

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